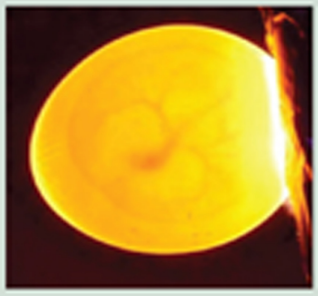
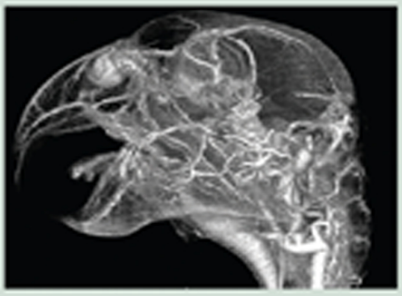


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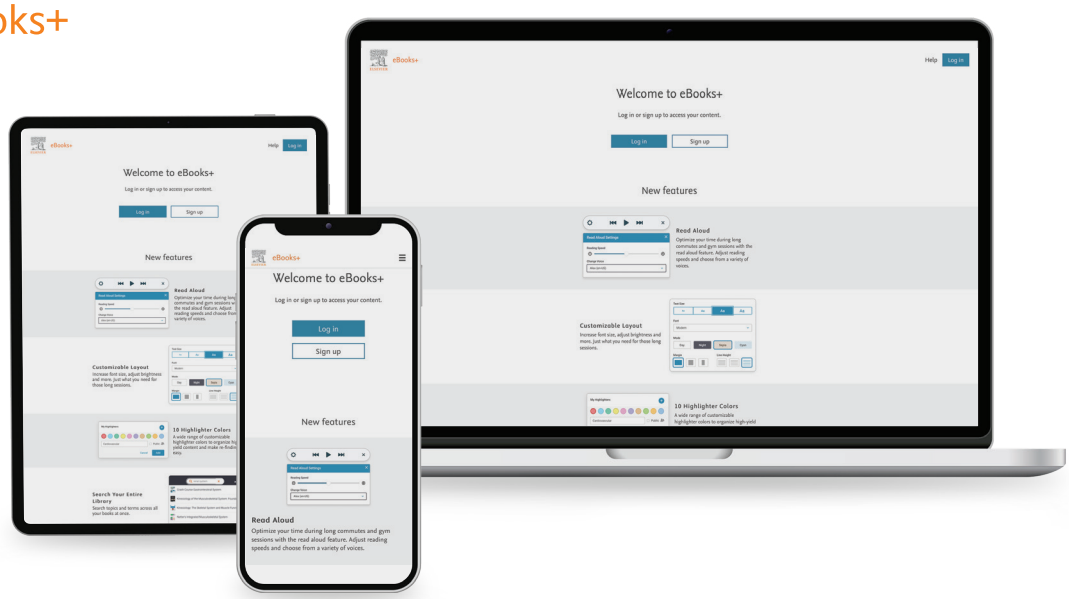
VOLUME II

BRIAN L. SPEER
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CURRENT THERAPY IN

AVIAN MEDICINE AND SURGERY

VOLUME II

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To our immediate and extended families,
those that touched our lives and stay in our hearts forever.
At the end of the day, you are what matters most.

And to the birds. They always continue to inspire us to soar to new heights,
both on a personal and professional level.
They truly are magical and wonderful creatures that we are privileged not only to share this world with,
but to also have the opportunity to do better by and for, in health and illness.

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Foreword

Drs. Brian Speer and Yvonne van Zeeland have invited many of the leading experts in avian medicine to share with you the latest advances in avian medicine in this second volume of *Current Therapy in Avian Medicine and Surgery*.

As I read through the various chapters of subject material included in this volume, I was awestruck by how far avian medicine and surgery has come in the 50 plus years since my first interest in avian medicine began. The literature at that time consisted of a handful of books, written by amateur avian enthusiasts, anatomists, or physiologists, some representing significant inaccuracies such as the book *Diseases of Canaries* by Robert Stroud (the Bird Man of Alcatraz). The only journal focusing on avian species at that time was the *Journal of Avian Diseases* (first published in 1957), but this largely focused on (commercial) poultry. Treatment protocols were anecdotal and rigid scientific information was basically nonexistent. Medications and equipment were adapted to the best of our knowledge. The state of avian practice was probably comparable to small animal practice in the late 1940s.

Simultaneously, pet birds were becoming "the pet" of the 1970s to 1990s. Millions of birds were being imported into the United States, often in some strikingly deplorable conditions. Those that survived were often malnourished and/or had viral or bacterial diseases. Many of the infectious pathogens responsible for causing disease in these birds were either not known or poorly understood. Nutritional recommendations primarily referred to diets consisting of mixtures of seeds, cuttle bone, maybe some fruits and vegetables, and more seeds, which did little to improve their health.

While the general public and the veterinary communities did not initially recognize avian medicine as being of value, an increasing number of bird "owners" ("caregivers" today) who were committed to the care of their birds began to seek out veterinarians who also shared a passion for birds. At the same time, we as veterinarians began treating birds using the limited resources available and established a community focusing on avian medicine, the Association of Avian Veterinarians (founded in 1980). Through this organization, we continued to more formally share our passion for and knowledge about birds and develop strategies for diagnosing and treating disease in nondomestic avian species. The growing information gained through clinical practice and research in 1989 also resulted in the transformation of our initial *A.A.V. Newsletter* to a more professional journal, the *Journal of the Association of Avian Veterinarians*, which in 1995 was renamed to the *Journal of Avian Medicine and Surgery* that we know today. These publications allowed researchers and clinicians with a desire to share and disseminate information to publish peer-reviewed case studies, treatment protocols, procedures, and techniques to advance the care of our avian patients. Simultaneously, the growing need for specialized care for birds resulted in the establishment of avian veterinary specialty colleges within the framework of the American (ABVP) and European (EBVS) Board of Veterinary Specialization, and the Australian and New Zealand College of Veterinary Scientists (ANZCVZ), securing global recognition and continuing advancement of avian medicine.

Societies have valued birds in many ways, and the international trade in birds, both legal and illegal, was having a significant negative impact on avian species globally. With the introduction of the Migratory Bird Treaty Act (1918) and the Endangered Species Act (1973), a rising societal awareness of and desire to preserve and protect (native) wild birds and their natural habitats developed. As conservation efforts became more refined, they began to have increasingly significant impacts on avian conservation. The mass importation of wild birds into the United States in the mid twentieth century became a societal issue in the 1990s and ultimately resulted in the Wild Bird Importation Act of 1994. This dramatically changed both the demographics and practice of avian medicine. Caregivers increasingly sought out "avian veterinarians" for the improved veterinary care for their birds, ultimately supporting a drive for the continual elevation of the standard of care for all birds. Veterinarians, working with ornithologists, biologists, and conservationists, became integral members of recovery programs for endangered avian species. Largely fueled by knowledge and advances gained from companion avian medicine and surgery, zoological, avicultural, and conservational aspects of avian medicine matured and evolved, benefiting both wild and captive avian species to this date.

As you open these pages of Volume 2, *Current Therapy in Avian Medicine and Surgery*, you will be amazed how thoroughly and competently the content has been compiled by its editors, Drs. Speer and van Zeeland, and the authors. The contents of this volume, alongside the first volume of this textbook, reflects the amazing growth in avian medicine and surgery from its infancy in the 1960s to today. As a small animal/avian practitioner in the 1980s, 80% of the types of cases we treated at that time are no longer seen or at least are seen much less frequently, including diseases and conditions such as scaley leg mites, vitamin A deficiencies, Pacheco's disease, metabolic bone fractures, and so on. If we were to plot the trajectory of care, the curve would almost certainly be at 90 degrees, with many technological, pharmacological, medical, and surgical advances helping to raise the standards of care and therapeutic opportunities for avian patients. And that just relates to the physical care of our patients. Consideration for the psychological or emotional wellbeing of our patients now ranks equal to their physical wellbeing as also reflected by the chapters dedicated to enrichment and minimizing stress in this book.

However, what has remained consistent in avian medicine throughout time is the passion and love for birds within our avian veterinary community. This passion drives researchers to work tirelessly, and authors and lecturers to disseminate

and share their knowledge, which can then be put to use by practitioners and support staff in the daily care of their avian patients. It is also our clients' love for their birds and their trust in our profession that allow us to treat their birds and share these advances. Volume 2 of *Current Therapy in Avian Medicine and Surgery* marks a point in the trajectory of avian medicine. Society has always been and will continue to be passionate about and value birds and it is this passion that will continue to drive the future of avian medicine, which you as a reader are an important part of and contribute to by applying the knowledge presented in this book while caring for your patients.

Bob Groskin, DVM

Preface

Following the publication of Volume 1 of *Current Therapy in Avian Medicine and Surgery* in 2016, there had been a plan to release a new volume about every 5 years. Obviously, this second volume does not follow that anticipated schedule. Complicating matters, including a global pandemic, the challenges of maintaining clinical practice, its management and overseeing new building construction duties, and a host of other personal matters all collectively raised their heads to provide a perfect storm to account for the delay. Nevertheless, we have endured the storms and have made it! We are proud and grateful to have this second volume come out as a joint effort by two editors. The combined skillsets, experience, and efforts of both coeditors have greatly helped to streamline the process, spread out the workload, coordinate, and assemble this second volume. Drafting the initial outline to its final version, recruiting the various international contributors, and working with them to review and edit their manuscripts represents an obvious large editorial effort for the publication of this book. Tying in the individual contributors' information with the overall envisioned framework of the current, previous, and future volumes is and will remain an editorial task that we both look forward to continue as coeditors while moving forward.

In keeping with Brian's original vision—a vision wholeheartedly shared by Yvonne—*Current Therapy in Avian Medicine and Surgery* continues to serve in a key text reference position as a leading source of current material within the broad scope of avian medicine and surgery. Both volumes of this text are written with the target audience of colleagues who are beginning their careers as well as those who are continuing to broaden their understanding and expertise. This second volume contains 12 sections, each exploring advancements in their respective areas, the vast majority bringing forward new, relevant, and in-depth information to add to the content brought forward in Volume 1. The interconnections between topics should be apparent to readers. While each chapter by itself provides important pieces of the whole, it also relies on others to help build a more complete picture of the delivery of avian healthcare, as also indicated by the various cross-references included in the text. Paired with the first volume, readers are bound to recognize the vision of *Current Therapy in Avian Medicine and Surgery*, each subsequent volume being complementary to the prior ones, and neither of them attempting to present the full depth and breadth of the field of avian medicine and surgery in its entirety between two covers. In this light, the outlines for the third, fourth, and fifth volumes of *Current Therapy in Avian Medicine and Surgery* are already shaping, aiming to add valuable new content to our current body of knowledge, and addressing relevant, current topics for the reader.

In the Section 1 of Volume 2, Advances in Practice Management, concepts in practice management are again explored as they were in the first volume. To develop a successful career in avian medicine, awareness of these concepts is important, as is your knowledge of the art and science of what we do. Section 2, Establishing a Diagnosis, explores common imaging modalities and how to get the most out of them, thermal imaging, and the value of necropsy as a part of practice, while Section 3, Therapeutic Intervention, offers a comprehensive overview for practitioners treating avian patients, fundamental guidelines, and a review of general pharmacologic principles, pharmacokinetic modeling approaches, and interpretation of antimicrobial results. This is a much more in-depth review for those colleagues who really want to understand where we currently stand in relation to applying these principles directly to avian practice while planning our therapeutic intervention strategies. Antimicrobial, antibacterial, antifungal, antiparasitic, and antiviral drugs are separately addressed with these same goals. Section 4, Infectious Disease Prevention, reviews the interactions of host, agent, environment and disease, hygiene, and disinfection, as well as current vaccination strategies. Perhaps stating the obvious, infectious disease does not just occur in a vacuum—it typically happens for reasons that can be managed, and is far more than a quest for pathogens only. Section 5, Updates on Infectious Diseases, updates our current understanding of important infectious diseases, including disease caused by avian bornavirus, poxvirus, West Nile Virus and other flaviviruses, campylobacteriosis, chlamydiosis, erysipelas in wild birds, mycoplasmosis, cryptococcosis, trichomoniasis, diplomonad protozoal diseases including giardiasis and spironucleosis, histomoniasis, and cochlosomosis. With the exception of bornavirus, none of these infectious diseases were presented in Volume 1, and as such these chapters will add to those presented in the first volume in a collective manner for your reference. We also dedicated a section to the respiratory system (Section 6), which—similar to the Cardiology chapter in the first volume—provides a focused, system-based in-depth review of avian respiratory anatomy and physiology, diagnostic work-up and diseases of the respiratory tract, complemented by chapters on surgery, endoscopy, and endosurgery of the respiratory system. Section 7, Beaks, Feathers, and Feet, aims to provide the reader with a review of the normal anatomy and physiology of these structures, as well as the etiology and risk factors that can lead to pathology, thereby aiding the reader in gaining an in-depth insight and understanding of the diseases and conditions affecting these anatomic regions. In Section 8, the trauma patient is presented, with individual chapters focusing on triage and stabilization, pain management updates, wound management, and fracture management. Many of the patients initially presented for veterinary assessment are the victims of traumatic injuries, and this is often where our doctor-patient relationship begins in practice. Keeping our abilities current in order to keep these patients alive, stabilize them, and move them toward ultimate release or long-term wellness

management is immensely important in what we do in practice. Section 9, dedicated to embryology, neonatology and pediatrics, is a long-standing area of Brian's personal interest and provides an excellent overview of egg and embryonic development, natural and artificial incubation, and pediatrics in psittacine and other commonly kept birds. Section 10, the welfare section, which was an important part of the first volume and is intended to be an integral part of any volume of *Current Therapy in Avian Medicine and Surgery*, brings two chapters directly applicable to practice: minimizing stress and fear during the physical examination, and the strategic use of enrichment. These chapters are immensely important to the art and science of how we can do best-practice in providing care for our patients. Likewise, Section 11, the conservation section, another standing part of both volumes, focuses on a timely and current topic: anthropogenic and environmental threats to avian wildlife. Included in this section are chapters dedicated to biotoxins, hazards related to man-made structures, petroleum products, anthropogenic toxins, wildfires, and an important one health subject: wild birds and cats. These topics are directly applicable to various aspects of healthcare for the individual bird and conservation medicine, and should be important to us all. Section 12, the final section is dedicated to specific taxonomic groups of birds, that is, the broad group of toucans, hornbills and aracaris, and birds of prey, the latter not specifically dealing with diseases common to these birds, but rather attempting to provide a global view of falconry, its past, present, and future, as we believe it is important for veterinarians dealing with these birds to have a better in-depth understanding of this ancient yet unique cultural sport. As editors, we are quite excited to see these taxonomic groups be presented in this manner for your reading pleasure. This second volume finishes with a series of appendices that include updated information on drugs and approximate doses (with several new drug groups added for your convenience), normal clinical pathological data, and normal biological data, as well as a new appendix presenting chick growth data compiled from previously published reference materials that many colleagues may not have had easy access to, and which have therefore been "brought back from the past" so that their information is not lost to time. As a result, *Current Therapy in Avian Medicine and Surgery* remembers the past, focuses on the present, but also keeps a clear and changing vision of the future in mind.

Acknowledgments

There are many colleagues to whom we are grateful for their influence in the development, guidance, and maturation of our current views of avian medicine and surgery. This volume of *Current Therapy in Avian Medicine and Surgery* would simply not be what it is, without the countless hours of hard work and sacrifice of personal time that each and every one of the contributors devoted to this project. As you will see, their expertise, work, and dedication are the building blocks for the past, present, and future volumes of *Current Therapy in Avian Medicine and Surgery*. Thank you, sincerely, to every single one of you.

Thanks are also needed for many others who have been directly and indirectly involved. Greg Harrison, who authored the first text on *Clinical Avian Medicine and Surgery* in 1986 and who was, in so many ways, a visionary and considerably ahead of his time in helping meet the need to unify medical knowledge and surgery. To this day, Greg continues his solid support, providing a guiding light for so many of us avian veterinarians. Walter Rosskopf, who helped pave the way so many years ago when Brian started his career in avian medicine and began to see how much could be medically considered and done for birds. For Yvonne, Gerry Dorrestein and Nico Schoemaker both had foundational roles as friends and mentors, sparking her fire for avian medicine and showing her the endless possibilities of what can be, if you do it well. James Harris, who shared the excitement and passion for learning and the scientific methods that serve as the foundations for moving our profession forward, but also had a big impact on a personal level. We sometimes get so caught up in things that we do not always realize that the personal relationships we continue to develop with colleagues and clients are among the most important things to cherish and keep close to our core, but James emphasized the value and importance of these relationships. So thank you James for demonstrating for us these important things, and teaching, by example, how you can grow as an individual while nurturing these personal relationships. The entire staff of our daily work environments, the Medical Center for Birds and Division of Zoological Medicine, must also be acknowledged and thanked, as their support was instrumental in enabling us to pursue this and other projects in the past. Other friends and colleagues should certainly also be named, those who have had formative roles and impacted our lives, personally and professionally, including—but not limited to—David Phalen, Jamie Samour, Bob Groskin, Tom Tully, Joanne Paul-Murphy, Murray Fowler, Michelle Hawkins, Neil Forbes, Scott Echols, Susan Orosz, Marina Krautwald-Junghanns, Bob Doneley, James Carpenter, Nigel Harcourt-Brown, Michael Lierz, John Chitty, Lauren Powers, Brenna Fitzgerald, Susan Clubb, Branson Ritchie, Helene Pendl, Pat Redig, Avery Bennett, Julie Murad, Irene Pepperberg, Chris Davis, Ted Lafeber, Keven Flammer, Helga Gerlach, Petra Zsivanovits, Deborah Monks, Tom Dutton, and Susan Friedman. Not all of you are still with us, but your words, your examples shown and shared, your thoughtful comments, tokens of kindness and support, constructive critiques and coaching, and your willingness to share your approaches to avian medicine and life in general, have guided us and continue to guide us constantly. Every single day.

A project of this size and scope represents innumerable hours of effort to coordinate, edit, and bring to fruition. This would not be possible without those who have supported us personally throughout this effort. We both agree that without a doubt, our spouses, Denise and John, must be acknowledged. They deserve a gold medal for their friendship and willingness to share life's journey with us, to always be there as an anchor in our lives. The practice of veterinary medicine is so much more than just a job; it is a passion and a true career for most of us. Our veterinary careers, and where they take us within the veterinary profession, are undeniably important, but without family and friends to share our journey in life with, our lives simply are not complete. Thank you, Denise, for completing, patiently mentoring, and guiding me, and helping to keep me on track and see what is needed to maintain and nurture those ever-changing and delicate balances needed in our lives together. Thank you, John, for teaching me how meaningful those little things are in life, that two are always stronger than one, and that love really does help overcome the most difficult times. Thank you as well to Robin and Cody, for being there with your dad through this all, and for the little things that you do that are the essence of what family is. From the day you were born, you have been, and will always remain, Daddy's little girl and Dad's favorite (and only) son.

Both of us have a clear understanding of the importance of our early childhood memories and how these shape our personalities and lives. Our parents have played a key role in this process and must therefore be recognized. So thank you, Verl and Lucille, Harrie and Mieke, for supporting us in achieving our goals and dreams; for raising us to be passionate, honest, and fair and do what we can to do the right thing; for teaching us that perseverance goes a long way in helping to achieve your goals, even if that is the harder road to take. And it's not just our immediate family, but also our extended families, our friends, that complement, enrich, and help to shape our lives. Nico, Frederique, and Raymon, you have proven yourselves to be true friends-for-life, offering helpful advice, providing a listening ear or shoulder to cry on whenever needed, and helping me (and John) get through what, undoubtedly, have been the toughest times of our lives. Fenna, Patricia, Melina, Aylis, and Ensar, I am blessed to have had Kees and still have you as my in-law family, and continue to be grateful for the time we spend together. After losing John, you have helped me to appreciate and enjoy life again, and I consider you as important as my own flesh and blood.

Wrapping up the final portions for the book and writing these acknowledgments gave us some time to reflect on the

journey that resulted in the creation of this second volume. I, Yvonne, still vividly remember the day that you, Brian, contacted me to invite me as coeditor for *Current Therapy*. It was the best Christmas present ever! I very much enjoyed working alongside someone that I consider one of my biggest role models and mentors, and would never have imagined the joy that this privilege could bring. Thank you so much for taking me on this journey, for the great teamwork, the many inspiring discussions, and the equally large number of bad jokes, which always bring a smile to my face! But most of all, thank you for your friendship and guidance throughout the years, which helped me grow, both as a person and professional. Likewise, I, Brian, am equally grateful for your, Yvonne's, friendship, and admire your hard work, incredible organizing skills, contagious passion, and relentless dedication to the profession, which have only grown stronger as we have worked on this project. In the many years we have known each other, you have grown to become a role model and mentor for me too, exemplifying that this role not necessarily has to be fulfilled by your senior. I am excited to see what the future will bring, planning the next volumes of *Current Therapy* together.

Looking back, we never imagined at the start of this project how rough the journey was going to be, with life throwing many challenges at us to pull us off course, and with many losses incurred along the way. John, Kees, and mom: we must continue life's journey without you, but promise to stay strong, never give up, take care of each other, and try and make our lives the best they can be. Dad and Taylor, we will carry on and do our best to get organized and stay on schedule. Dona, we know that you are still with us at every family meal, helping to plan the next, even before dessert is served. You are missed greatly, but forever in our hearts and thoughts. Thank you all for touching our lives.

Brian & Yvonne

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CHAPTER 1

Practice Management: Developing a Career as an Avian Veterinarian

Len Donato • Marianne Donato • R. Eric Miller • Bruce J. Nixon

INTRODUCTION

A career in avian clinical practice can be quite rewarding. In addition to what is seen in private practice, there are times that avian clinicians will play a role in public health, work to help preserve endangered species, aid government programs, and perform research. Avian medicine and surgery is a highly specialized field that fills a need not only for our society but also on a day-to-day basis for our clients and patients. Despite the great personal rewards that go with private practice and avian medicine, there are many challenges. For example, there are concerns about maintaining and continually improving the level of patient care, successfully generating meaningful revenue and income, preserving longevity as a clinician in the field, and possibilities for effective retirement planning. Successful management of all of these aspects can bring greater personal, professional, and financial rewards to the practitioner pursuing a career in avian medicine.

When making the decision to pursue a career in avian medicine, certain questions have to be answered:

1. *What kinds of birds do you want to see?* Some practitioners elect to see any bird that can be kept as a pet. Others prefer to work with wild birds, hence working with wildlife rehabilitators and falconers may be the mainstay of a veterinarian's avian practice. Providing healthcare and management recommendations for zoologic collections and aviculturists is another potential pathway that fits under the broad umbrella of avian medical practice. Poultry healthcare is yet another option, whether that becomes only backyard chickens or production flock management.
2. *What percentage of the caseload do you want to be birds?* Some practitioners may aim to only treat birds owned by their previously established dog- or cat-owning clients. Other practitioners choose to focus all of their energy on birds, and rarely if ever see any other species, or choose to treat birds as a component of a larger variety of other exotic species in their practices.
3. *What level of medicine and surgery do you want to provide?* There are many practitioners who are happy with providing service for general health evaluations, nail trims, and wing trimming procedures. In these practices, birds with medical issues are usually referred to a facility that sees birds regularly. Clinicians that are interested in treating medical and surgical cases should ensure to clearly outline the level of diagnostic and surgical capabilities for potential clients. Similarly, it is important to decide whether hospitalization services will be available, whether the hospital will cover overnight care, and if so, whether it will be open to the public for 24-hour emergencies.

Answering these questions will help define at least the beginning of your career working with birds in clinical practice. After outlining the parameters, be sure to record them. Referring back to them regularly will keep you on track to achieve those goals. This exercise will also help you plan for continuing education, equipment acquisition, physical plant design, staffing needs, inventory management, and marketing strategy. The answers to these questions may change as your career advances. That is very normal, as healthcare providers typically evolve throughout their careers.

GAINING KNOWLEDGE

There are many opportunities for continuing education in the field of avian clinical medicine and surgery. Defining the goals as previously outlined will aid to determine which type of education will best serve and fit your needs.

Independent Study

Having good, current resources available to help with everyday practice is crucial. There are many quick reference guides to have on hand, as well as more detailed textbooks as outlined in Box 1.1. Having easy access to hard or digital copies of your reference material is paramount. There are veterinary online forums through social media or paid subscription that can help keep a practitioner up-to-date on current trends in

BOX 1.1 AVIAN VETERINARY REFERENCE MATERIALS

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Journals

Avian Diseases
Avian Pathology
British Poultry Science
Journal of Avian Medicine and Surgery
Journal of Exotic Pet Medicine
Journal of Wildlife Diseases
Journal of Zoo and Wildlife Medicine
Poultry Science
Veterinary Clinics of North America: Exotic Animal Practice

avian medicine, follow thought patterns through a case, communicate with colleagues, or get access to scientific papers. As with any online forum, be mindful of the information and check your sources. Peer-reviewed journals and articles are available and should be a part of any avian library you have assembled.

Continuing Education

Continuing education allows veterinarians to gain detailed knowledge for credit in a focused area of study, in this case avian medicine and surgery. There are many online and in-person opportunities, as detailed in Box 1.2. Many states have restrictions on the amount of continuing education a veterinarian can receive from online seminars. Be sure to check state regulations. Conferences for in-person opportunities also allow for networking with colleagues, which is crucial in nontraditional species medicine. The Association of Avian Veterinarians (AAV), the European Association of Avian Veterinarians (EAAV), and the AAV Australasian Committee (AAVAC) are international organizations of veterinary professionals that offer various educational opportunities and information on national and international meetings that focus on avian medicine and surgery.

BOX 1.2 AVIAN VETERINARIAN CONTINUING EDUCATION (CE)

Veterinary Information Network
- Individual courses
- Portal for access to other veterinary organizations CE

Association of Avian Veterinarians (AAV)
- Competency-Based Medical Education (CBME) on website
- ExoticsCon or AAV Annual Conference
- Online CE on website
- Announcements of local, national, and international avian conferences posted on website

American Board of Veterinary Practitioners (ABVP)—annual conference
Association of Avian Veterinarians Australasian Committee (AAVAC)—annual conference
European Association of Avian Veterinarians (EAAV) conferences—ICARE conference
American College of Poultry Veterinarians (ACPV)—annual conference
World Veterinary Poultry Association (WVPA)—biennial conference
American Association of Zoo Veterinarians (AAZV)—annual conference
European Association of Zoo and Wildlife Veterinarians (EAZWV)—annual conference
Some avian pet food companies will host online CE
Many popular companion animal conferences will have exotic pet sessions featuring avian medicine and surgery

Academic Opportunities

There are a number of opportunities to gain exposure and receive advanced training in avian medicine in the academic environment. Veterinary students can volunteer in their extracurricular time to explore areas of interest in veterinary medicine. For avian medicine, that may be at a species-focused practice at least including a significant portion of birds as their caseload at their school or a nearby veterinary hospital. It can also include wildlife facilities, zoos, laboratory animal settings, or even poultry farms. In the clinical years of veterinary school, there may be an in-house service that offers a rotation for the students to get further exposure to avian medicine. Students may also elect to schedule rotations outside of the university, referred to as externships. Externships can take place in private practice, zoos, wildlife rehabilitation centers, veterinary poultry practices, and laboratory animal facilities. Veterinary schools usually have a list of externships for students to review when scheduling their clinical year. Selecting an externship outside of the approved lists will typically require working with student affairs.

Internships and Residencies

If one decides to specialize in avian medicine and surgery, internship and residency opportunities are available and a common path is taken. They are available in academia and private practice. Internships are typically one year in duration and prepare a candidate, primarily through clinical training, literature reviews, and research, for a residency or clinical practice. A residency is advanced training that lasts two to three years and prepares candidates for board examinations that lead to specialization. Internships are usually required to obtain a residency position. In Europe, some training facilities allow application for a residency position following a minimum of two years of work in a highly equipped veterinary clinic. In the United States, the Veterinary Internship and Residency Matching Program (VIRMP) and independent research will help identify the right place for advanced training in avian medicine. Note: Not all internship and residency opportunities are found through VIRMP.

Specialty Board Certification

You can obtain specialty board certifications in avian medicine and surgery. There are several certifying organizations recognized by the AVMA. The American Board of Veterinary Practitioners (ABVP) certifies veterinarians for avian practice. There are also board certifications through the American College of Zoological Medicine (ACZM), the European College of Zoological Medicine (ECZM), and the Australian and New Zealand College of Veterinary Scientists—Avian chapter. The details of these varied options for achieving advanced specialization including avian species are discussed in Volume 1 of this series. The American College of Poultry Veterinarians (ACPV) and the European College of Poultry Veterinary Science (ECPVS) provide certification specifically for poultry flocks and their management. These organizations require either completion of an internship and an approved residency or a number of years of clinical practice with the species of birds for that category. All specialty boards have their own requirements, but most involve writing articles for a peer-reviewed journal or preparing case reports that are reviewed by the certifying entity. An examination is also administered to candidates testing in-depth knowledge of birds in the specialty category. In addition, various countries offer national recognition in avian and/or exotic animal medicine and surgery through certification at an intermediate level (e.g., Germany, Spain, United Kingdom).

AVIAN HOSPITAL DESIGN

Avian/exotic exclusive practices are becoming increasingly popular, especially in large metropolitan areas. The majority of avian practitioners work in practices that also see other species, often including dogs and cats. Design for these clinics should be highly specialized and often there is an emphasis on incorporating avian design elements into multispecies facilities.

Waiting Room Design

Species segregation is a priority in a hospital that sees birds, and many, if not most, multispecies hospitals already have this incorporated into their design element. Unfortunately, however, these areas are usually segregated into dog and cat, without a designated area for birds, let alone a separate area for bird species. This creates a potentially dangerous situation in which birds may be exposed to either loud and often very large predators or small yet incredibly efficient predators with a predisposition for bird prey. Therefore, waiting rooms in mixed species practices should be designed to have a specific bird area. This begins with clear signage before the client enters the lobby and should be reinforced by receptionists strictly directing clients into designated areas (Figure 1.1). Despite all efforts to have a segregated waiting room, there will always be emergencies and walk-in appointments, but even with those, every effort should be made to reinforce species segregation. All clients should be instructed prior to visits to keep their birds confined to a carrier, as many avian clients tend to carry their birds on their shoulder without proper, safe confinement. Veterinarians in the position of designing or remodeling a waiting room should consider a segregated space for birds. For existing lobbies, the only practical option will be to add a vertical divider in whatever part of the lobby seems to be the most inconspicuous. Receptionists will need to remain alert to clients if they are out of direct line of sight.

Ideally, birds should not be in waiting rooms beyond initial greetings. Rather, they should be directed immediately into an examination room with closed doors to reduce stress, ensure patient safety, and reduce the risk of infectious disease transmission to other birds and clients (in case of a possible zoonosis). Check-in and check-out procedures should be accomplished while client and patient are within the confines of that safe space. This of course assumes that the hospital has the capabilities to efficiently maintain an appointment schedule with a surplus of examination rooms.

Ceiling fans pose a threat to flighted birds and therefore should be removed completely. It is not sufficient to keep any such fans turned off as many bird owners will notice them immediately and question either your commitment to or



FIGURE 1.1 External signage directing clients to species-specific areas. (Courtesy B. Nixon.)

knowledge of avian patients. Ceiling height can also become a major frustration if a bird escapes from its enclosure or the handler. Many hospitals use very tall ceilings and numerous windows in the waiting area as a design element. Any practice that sees even a moderate number of birds will inevitably suffer the anxiety of an escaped, flighted bird. Having escapees perched 18 feet off the ground or flying into windows is not an amusing situation, thus reception staff must be relentless in their insistence that all birds brought into the hospital must be in an enclosure of some kind. Receptionists should offer towels or blankets to cover the cage or carrier to reduce anxiety. Offering possibilities to elevate the carrier rather than placing it on the ground as well as playing music in the background to reduce anxiety from sudden noises may be helpful as well (see Chapter 46).

Examination Rooms

Most examination room designs work quite well with avian patients. Simplicity in design elements leads to simplicity of maintenance. Built-in benches are preferred to free-standing chairs as birds—once released—will often perch on the backs of chairs. Chairs or other furniture may be stained with droppings from birds perching on them or become ragged from the ravages of anxious or bored psittacines nibbling chunks out of them. As a result, furniture in the examination room should be made of material that is chew-resistant and is easy to clean, like metal.

Upper cabinets that extend to the ceiling preclude flighted birds from perching on top (Figure 1.2). If existing cabinets do not extend to the ceiling, a cabinetmaker can easily install blanks atop existing millwork to achieve this effect (Figure 1.3). For new construction, laminated millwork is a superior choice to stained wood for ease of maintenance and resistance to beak damage. Stainless steel is often used as a tabletop and although some may not appreciate the visual appeal of bare metal, it is indisputable that the material is practically indestructible and easy to clean. Screen or mesh can be inserted inside heating, ventilation, and air conditioning vents to prevent small, flighted birds from escaping into the ductwork. Windows in the examination rooms may be problematic for birds, potentially serving as a means of injury should the birds fly into them. As a result, these should preferably be covered with opaque window films, blinds, screens, or similarly functional alternatives.



FIGURE 1.2 Patient out of reach on top of a cabinet in the examination room. (Courtesy B. Nixon.)

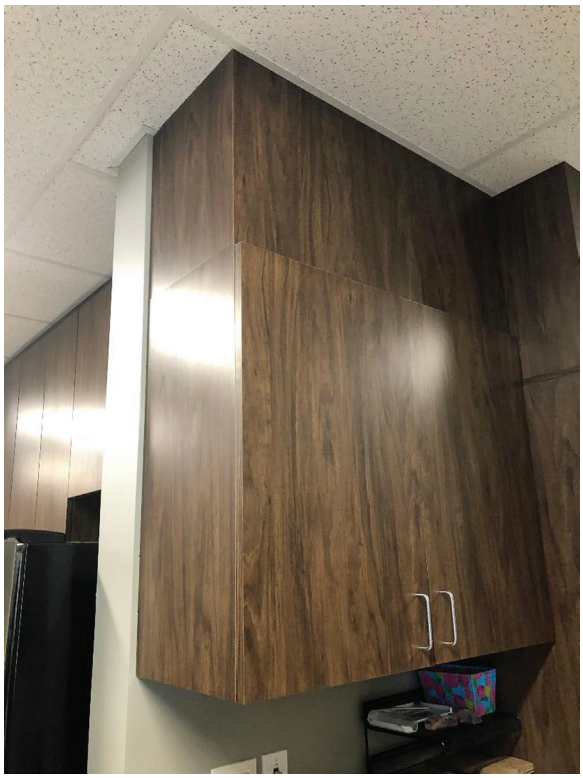


FIGURE 1.3 False panel placed on top of a cabinet to prevent flighted birds from perching out of reach. (Courtesy B. Nixon.)

Overhead light emitting diode (LED) lighting has largely replaced incandescent and fluorescent lights and is a superior choice in virtually every way (color, brightness, and energy efficiency). Having an electrician replace existing fluorescent lights with LED fixtures is surprisingly inexpensive and fast. While the electrician is in the building, you can also have them install motion-activated wall switches to increase energy efficiency even more. In new construction, both LED lighting and motion-activated wall switches will usually be required by local ordinance. Adding a dimmer switch can be handy for easing capture of freed birds in the examination room.

Some avian practitioners prefer to do thorough examinations and even some diagnostic sampling in the examination room itself to enable clients to see as much as possible of what is done with their bird compared to transporting the patient into a separate treatment area. In these situations, it may be wise to install specialty examination lights that can be used as a spot or flood and positioned where needed.

Treatment Areas

If possible, it is best to have at least one designated treatment table in an area segregated from the bustle and noise of most multispecies animal hospitals. The lack of such a space in hospitals is what may lead some practitioners to do much of their hands-on work in the examination rooms. In new construction and most remodeling projects, a segregated avian treatment table can usually be fit in without much difficulty, as it requires very little space. In fact, a small treatment space can be used to the clinician's advantage with flighted birds to keep them contained. As birds may overheat easily when handled for some procedures, it is convenient to be able to set them on the floor immediately afterward or on the table in a well-ventilated container. A small, quiet, separate space allows them to safely calm and cool down.

In many hospitals, an existing isolation room can be used as the treatment area for birds. These rooms are often empty and underutilized. They can be readily customized into becoming a combined isolation/avian treatment area. Even small practices with closet-sized isolation rooms can consider having only one large cage on the ground floor with a countertop built upon it (Figure 1.4).

Incubators are indispensable items in practices that see birds as being self-contained units that can control heat and deliver oxygen. Most can also monitor and adjust humidity and deliver nebulization. Unfortunately, these units require a great deal of counterspace in the treatment area and many of these units cannot be stacked. Counterspace is usually premium real estate in most practices already, particularly in the treatment room. There is no magical fix for increasing counterspace. Even in new construction, you will usually be limited by the length of your walls, less doorways. This frustration can sometimes be solved by recruiting the services of an interior designer who specializes in commercial buildings. Some of these professionals are expert organizers and can free up counterspace in imaginative and functional ways, such as installing wall shelving to place incubator units.

Avian Wards

A completely segregated avian ward is the ultimate in hospitalized patient housing, if space permits. New construction should be able to efficiently locate an avian-specific ward (ideally multiple wards) with an observation window off the treatment room. If an observation window is not possible, then consider installing webcams that can allow observation without having to enter the room. Even some remodels may be able to carve space for an avian ward, as the space requirements are not unreasonable. The ward will require at least 5 feet of width to accommodate cages and countertops and leave free workspace for clinic staff (Figure 1.5). The purpose of the ward is to provide a quiet, controlled environment for convalescing birds, easy access for staff, and to provide strict isolation for birds that may have contagious disease. Because of the possibility of contagious diseases, it is not a good idea to combine the avian treatment area into the avian ward. Doing so will force the practitioner into identifying yet another space to hospitalize birds with known or suspected contagious disease.

Controlling air quality in an avian ward can be accomplished in several ways. In most situations, the avian ward will

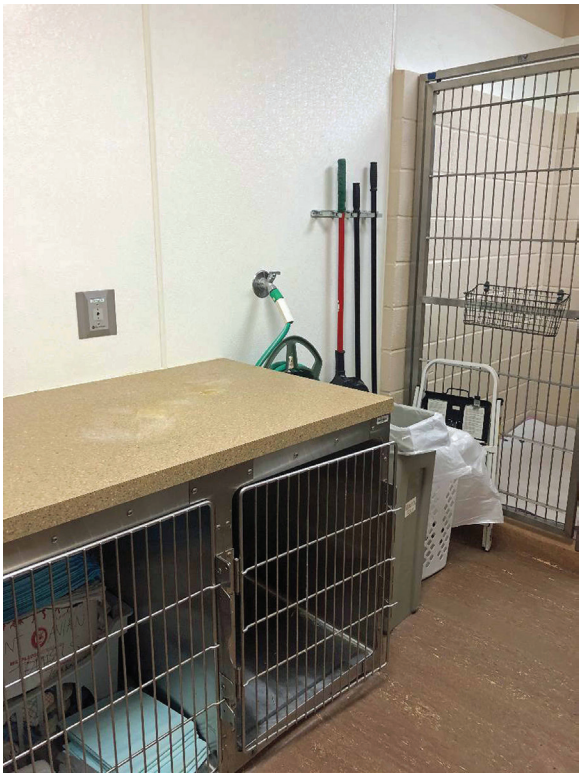


FIGURE 1.4 Improvization of a treatment space. (Courtesy B. Nixon.)



FIGURE 1.5 Avian ward with an observation window. (Courtesy B. Nixon.)

be connected to the heating/air conditioning system being used throughout the building. Many bird species shed large amounts of dander that can quickly clog air filters. Thus, it is essential to have routine replacement of these filters included in your hospital or clinic maintenance plan. Initially, you can aim for quarterly replacement, and monitoring the condition of the replaced filters will help determine whether maintenance needs to be scheduled more or less frequently. Free-standing *HEPA* (high-efficiency particulate air) filters can also be used within avian wards, especially those that do not *have high air* change rates. When choosing this option, do not make the mistake of choosing small or inexpensive units as they will not be able to handle the volume of bird dander or volume of air to fully filter the air. New construction will almost certainly require the use of a mechanical engineer to design your heating and cooling system. Remodels or retrofits can use the services of a commercial heating/air conditioning company experienced with hospital, dental, physician, and/or veterinary facilities. Take the time to study and understand your system including the temperature ranges needed within each zone, air change rates, air velocity at head level, the use of ERVs (energy recovery ventilators), and the number and types of air filters. Mistakes are costly, time-consuming, and frustrating for the practice owner and the staff.

The ambient temperature needed in avian wards is typically warmer than in other areas of the hospital. It is not reasonable to have working areas of the hospital be as warm as the ward. Have a separate thermostat (zone) for the avian ward or use a supplemental heating unit as a means of maintaining temperature that is safe for your patients. Consider wall units or split air conditioning systems as the room can be separate from all other systems and provide safe temperature control. Monitor temperature with the unit's thermostat and a separate stand-alone thermometer. Most avian wards can be kept at 80°F (27°C) depending on species; if tighter temperature regulation is needed, then incubators should be used.

Other Considerations

Flooring

Hospital flooring is often the number one question asked at hospital design continuing education events. There are simply no right or wrong answers; the choices are determined by cost, aesthetics, owner preferences, and functionality. Most hospitals have multiple types of flooring throughout the facility. What is offered here is the opinion of one of the authors based on several decades of working in, visiting, designing, and building veterinary facilities, both large and small.

Waiting rooms and lobbies should be the most aesthetically pleasing area within your hospital. As such, natural stone such as slate is quite beautiful and durable. Porcelain tiles are available in an astonishing variety of styles, textures, and colors. Stained concrete should be strongly considered in new construction.

For working areas of the hospital, one of the author has found rubber sheet flooring to be almost ideal. This material is thick and has some give, which makes it forgiving to walk upon and also deadens sound. It is available in a variety of colors and patterns that are distributed through the entire thickness of the material, meaning that even a deep gouge into the material is almost invisible. The product is heat welded so that you have effectively one solid piece of flooring with no gaps. Even more importantly, it can be flash coved 4 to 6 inches up the wall (Figure 1.6). This eliminates the need for baseboards, which collect all manner of liquid and other debris under and behind them and inevitably become disgusting foci of decay. Rubber sheet flooring is maintained with simple mopping, and no stripping, buffing, or annual top layer applications are needed. The biggest downside to rubber sheet flooring is cost; however, the material should last the lifetime of the building with no need for replacement. Another potential problem with rubber flooring is that moisture migration upwards from a concrete slab could cause bubbles to develop.



FIGURE 1.6 Rubber flooring flash-cove up the wall in lieu of baseboards. (Courtesy B. Nixon.)

Sheet vinyl is commonly used in working areas of veterinary hospitals, primarily due to its lower cost. It can be flash coved, and should be, in lieu of baseboards, but the material stiffens and cracks over time and the coving eventually fails, as do the seams that connect the sheeting.

Epoxy flooring is widely used in kennel and run areas and throughout a clinic or hospital. The material is certainly durable and the incorporation of "chips" within the material can give both texture and pattern. It can be spoon coved up the wall to eliminate the need for baseboards. In the experience of one of authors, this flooring becomes dingy or unevenly discolored with time regardless of scrubbing or pressure washing. It can be unforgiving on feet and is noisy. A newer process using polyurea basecoat and polyaspartic clear coat appears to be a superior alternative to traditional epoxy.

Noise

Minimizing loud noises for our avian patients will reduce stress and promote a friendlier environment. Behavioral changes by the staff to reduce noise will be reviewed later in the chapter.

Changes to the physical plant can help reduce noise. Increased drywall thickness and insulation between walls should be considered throughout the hospital, but especially for avian-specific areas. If possible, bird wards and bird-only examination rooms can be located out of the flow of main traffic and away from barking dogs if the practice is a multispecies facility. Select surfaces that absorb sound. Ceiling tiles have a noise reduction coefficient (NRC) of 0 to 1.0, which represents a percentage of noise absorbed. If the NRC of a tile is 0.2, 20% of the noise is absorbed and 80% is reflected. Decorative tiles can also be put on walls to reduce noise and have the same rating system as ceiling tiles. Furniture, paintings, and other fixtures also reduce noise as is evidenced by the echo in an empty house. Use any physically possible tactic to reduce noise or use soft ambient music to dampen sudden noises.

Permits/local ordinances

When remodeling or starting a new construction project, be sure to follow all local ordinances and have proper permitting and inspections. This cardinal rule is commonly broken with remodeling. While it may seem like a simple project, construction frequently goes awry and another set of experienced eyes on the project is crucial. Proper inspections may save a big disaster later.

EQUIPMENT

To practice quality avian medicine and surgery, it is essential to have proper equipment available. Whether and which equipment is necessary will be determined by your pre-set goals of percentage of the practice that is composed of bird care and to what medical and surgical level you want to practice avian medicine. As an example, an endoscope is not needed if the goal is to see healthy birds and refer ill patients to specialists. An existing multispecies practice will have many of the items on hand already. Regular maintenance should be scheduled to ensure that equipment is maintained in good working order and is replaced if it is malfunctioning and cannot be repaired.

Here is a comprehensive list of equipment by potential goals that may be defined.

Examination and Routine Care

- Gram scale: Have a scale that goes up to 2000 g and one that can accommodate larger birds, if you are seeing those. Preferably, birds should not be weighed in pounds or ounces; grams and kilograms are much more accurate. Perch stands for the scale can make weighing easier if the bird is comfortable using it. Human baby scales are inexpensive and very helpful for weighing many larger, non-perching species. Otherwise, clear plastic containers with securable lids and air circulation can be used to keep a patient restricted but monitored and observed safely while being weighed.
- Nail clippers of varying sizes: You may also want to have a handheld cautery pen to prevent bleeding for small birds weighing less than 120 g.
- Scissors of varying sizes: Used to cut feathers for wing trims (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 22, Advancements in Management of the Welfare of Avian Species).

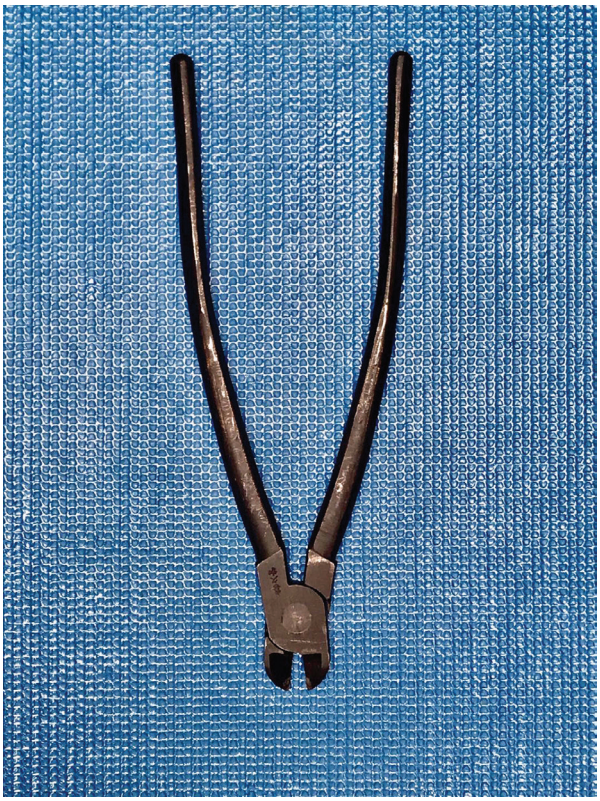


FIGURE 1.7 Band removal cutting tool. (Courtesy L. Donato.)

- Rotary hand tool: These are very helpful for large bird nails or for rhamphothecal shaping procedures (see Chapter 31).
- Band cutters: Used to cut and remove leg bands, when indicated (Figure 1.7).
- Dental high-speed drill: This is used to remove leg bands that are too thick for standard band cutters. This drill can also be used to remove leg bands that have embedded in swollen tissue from trauma.
- Paper towels, washcloths, hand towels, bath towels: These are used to hold birds for examination or other procedures.
- Oral speculum: Can be used to help maintain an open mouth for oral examination, sample collection, or tube feeding if necessary (Figure 1.8).
- Pediatric or neonatal stethoscope/ophthalmoscope/otoscope: These are important tools for any avian physical examination, particularly the more common, small species seen in companion bird practice.
- Net: This will aid in catching escaped birds. Nets can be modified to have a soft, supportive ring that holds the net webbing so birds are not injured during the recovery process. Foam or insulating pipe material are options for this modification. A telescopic handle to extend the net is advised (Figure 1.9).
- Microchip reader: When placing microchips or if lost birds are brought to your facility, a reader will be needed to confirm the microchip number.
- Microtransponder chips: These are also known as p-Chips. They are small enough to implant in a small bird, as the standard microchip is sizable and will damage pectoral muscles of smaller birds.



FIGURE 1.8 Oral speculum. (Courtesy L. Donato.)



FIGURE 1.9 Soft ringed capture net. (Courtesy Y. van Zeeland.)

Advanced Medical Care and Hospitalization

- Red rubber catheters: These are used for tube feeding for nutritional support, obtaining samples from the crop, and some surgical procedures.
- Metal crop feeding tubes with rounded tips: These are also used for feeding nutrient-deficient birds.
- Bird collars: There are times that some birds may require placement of a response-block mechanism to prevent access to wounds as a part of their treatment plan. Collars can be purchased or homemade. The patient should be monitored for adequate time following placement to obtain assurance that it can eat, drink, and adequately function prior to discharge.
- Small- and large-tipped culture swabs, Gram stain and modified Wright stain, frosted slides: These are for laboratory processing. Use a reference laboratory that specifically works with birds. Call their specialist to discuss best practices for sample collection and processing.
- Needles and intravenous catheters from 18 to 30 gauge: Bird species seen in private practice can vary in size greatly.
- Nebulizer: Medication can be delivered to the respiratory tract with a nebulizer. This is commonly used as an adjunct in treatment for aspergillosis (see Chapter 8) but has many indications.
- Thermal support and oxygen supplementation: Heat lamps and incubators are excellent heat sources for hospitalized birds. Incubators have the option to be used as a nebulization or oxygen chamber. Circulating air thermal blankets are great additions for maintaining temperature during surgery.
- Housing: As previously discussed, a separate avian ward should be available. There are many types of housing possibilities for birds while hospitalized. All caging should be avian specific, as other modified types may have spaces too large so birds can escape or areas where the patients can get injured. Depending on space available, countertop incubators may be used or smaller bird cages. A heat source should be able to be used safely with the caging or have one incorporated. Any type of housing should be easily cleaned and maintained in good working order.
- In-house chemistry analyzer: Emergencies are common and waiting for lab work may not be in the best interest of the bird. A system that is able to use a small amount of blood is best. Some patients may not have great blood volume due to size or illness.

Anesthesia and Surgery

- Small anesthetic face masks: They come in many sizes, but commonly they need to be modified with bandage wrap and small hole cut in the center for the smaller patient to efficiently receive inhalant anesthetic gas or oxygen.
- Uncuffed endotracheal tubes: Sizes typically range from 1.0 mm to 7.0 mm (3 to 22 French). Larger and smaller sizes can be fashioned from catheters of any size, or cuffed endotracheal tubes can be used without inflating the cuff. Inflation of cuffed endotracheal tubes can cause necrosis if inflated against the complete tracheal rings present in avian patients (Figure 1.10).

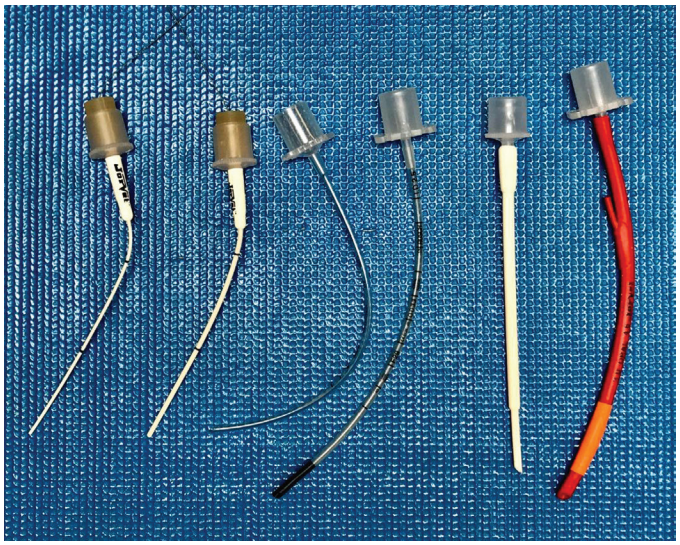


FIGURE 1.10 Uncuffed endotracheal tubes. (Courtesy L. Donato.)



FIGURE 1.11 Therapeutic laser. (Courtesy L. Donato.)

- Therapeutic laser: This can help with pain and inflammation associated with trauma, wound management, osteoarthritis, and other medical conditions deemed appropriate by the treating veterinarian (Figure 1.11).
- Nonrebreathing anesthesia circuit: Using these for anesthesia allows us to make rapid changes to concentrations of inhalant gas.
- Isoflurane and/or sevoflurane vaporizers: These are the best inhalant anesthetics for use in birds.
- ECG: Monitors the electrical activity of the heart during anesthesia.
- Doppler probe: This will give an audible reading of blood flow through peripheral vessels. Pressures can also be obtained in larger patients.
- Capnography equipment: This typically attaches to the endotracheal tube to measure levels of carbon dioxide. This is an important parameter for birds that was reviewed in the anesthesia section (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 19, Anesthesia).
- Magnifying head loupes: During surgery, these help to provide an improved field of view through magnification; some also have a separate light source.
- Radiosurgery/electrosurgery unit: This is an excellent tool to have during surgery to control hemostasis, particularly in smaller patients.
- Surgical laser: Some surgeons like a surgical laser for birds as it can help with hemostasis similarly to electrocautery. If doing endosurgery, a surgical laser can be used in conjunction with a telescope and operating sheath.
- Sterilized cotton-tipped applicators and surgical hemostatic clips: These items will help identify and control bleeding during surgery. Hemostatic clips come in many sizes. The surgeon can establish the sizes they would use most commonly.
- Clear surgical drapes: Seeing our smaller birds can be challenging with the surgeon and potentially an assistant present. A clear drape gives the anesthetist the opportunity to visualize respiration, blink response, mucous membrane color, and be sure there is no endotracheal tube migration.
- Surgical instruments: This can range from basic surgical packs used for dogs and cats to very specialized instruments for microsurgery. It will depend on the type of surgery being performed and the comfort level of the surgeon (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 21, Surgery).
- Endoscopic equipment: There are varied sizes and types of endoscopes, but the most commonly used for birds is a 2.7-mm rigid scope with a 30-degree angle at the end for improved viewing. The telescope with sheath and instrumentation can be used for viewing, obtaining samples for culture, cytology, and biopsy, retrieving gastrointestinal foreign bodies, or performing surgical procedures (Figure 1.12).

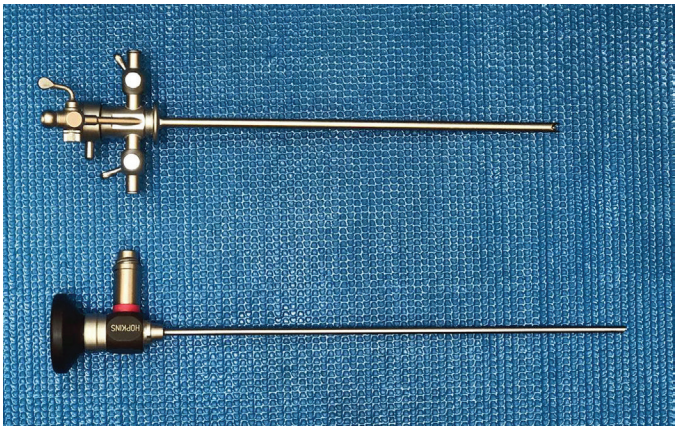


FIGURE 1.12 A 2.7-mm rigid endoscope with operating sheath. (Courtesy L. Donato.)

Imaging (See Also Chapter 2)

- Digital radiography (DR): This produces high-quality images in small avian patients that other types of radiography would not provide. Having dental digital radiography available is also quite helpful.
- Ultrasound: Because air can block ultrasonic waves, this imaging modality requires an absence of air in the region of the examined anatomy. Ultrasonography is commonly useful in cases with coelomic fluid or organomegaly; ultrasound is also of great value for cardiac examination.
- Fluoroscopy: This imaging modality is being increasingly utilized, with its availability increased through the increased popularity of computed tomography (CT) imaging that has a fluoroscopic function. Helpful in evaluating real-time function of the gastrointestinal system, and it is also of benefit in some orthopedic procedures.
- CT: This has become more readily available for the everyday practitioner. The image quality is incredible, and areas of interest can be viewed three dimensionally. Skeletal and respiratory tracts are most easily evaluated; however, soft



FIGURE 1.13 Computed tomography scanner. (Courtesy L. Donato.)

tissue can be imaged very well with the use of intravenous contrast agents (Figure 1.13).

STAFF

Staffing for an avian practice or multispecies practice that sees birds is a crucial part of the puzzle, if not the most important. Everyone from owner to veterinarian to receptionist to technician has to be interested and excited about incorporating avian medicine into the practice. If their interest is not present, it will show in client and patient interactions, which is not an impression the client should be left feeling (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 24, Practice Management and Risk Management). It can also affect care where uninvolved technicians may avoid handling, cleaning a cage, or performing treatments. Staff interested in avian medicine may view the ones not interested in a negative way as well. Making a plan to identify the right candidates, outlining expectations at time of hire, establishing a proper training protocol, and understanding what it takes to keep good staff will minimize staffing concerns at an avian practice. Associate veterinarians that have an interest in avian medicine should ask about the steps of this process when selecting a practice that supports their interests and therefore is a good fit for them.

Profile of Candidates

Candidates will include veterinarians, office staff, technicians, and kennel employees. Posting available positions is done mainly online, and the most popular sites should be identified for your locale. When advertising for a position, be sure the posting mentions the percentage breakdown of the different species in the practice. This will begin the process of whittling down the candidates that will be the best fit.

When you receive a resume, follow a basic screening process to identify normal initial concerns such as proper organization, grammar, large gaps of unemployment, and longevity at prior jobs. Then look into previous experience that correlates with the main duties the new employee would be performing. Previously having worked with birds can be a significant advantage when reviewing resumes to identify the next staff member. It demonstrates interest, which can be a big hurdle in finding the best candidate to hire. It is not all important though, so do not make the mistake of hiring for experience over personality and attitude. If someone has great interpersonal skills and a willingness to learn, along with a personality that will fit in well with the culture and client demographic, that person is uncommon and should be hired. In the practice of one of the authors, almost no new hires have had any significant avian veterinary experiences prior to their hire, but have excelled with their work on birds.

Hiring Candidates

Once a set of candidates has been selected for the interview process, it is time to identify if your mission and a potential new employee will be a match. Follow all interview processes that normally take place. With regard to birds and a candidate's interest, this is a good opportunity for a tour of the facility. During the tour of the hospital, the candidate will be shown all areas of the facility, which will include the segregated area of reception for birds, the avian wards, and they will likely see equipment for avian patients around the facility. This will aid in getting your message across that birds are important, just like the other species seen at the practice. Having a clear and concise mission statement that spells out all of the species that come as patients clearly defines how a hospital feels about birds. This mission statement should be in the handbook as well as posted in the common area for employees to see on a daily basis. This signage should be brought to the candidate's attention during the tour.

Once the right candidate is found and both parties believe this is a great fit, hiring can take place. Have the proper onboarding procedures and associated paperwork, which would include signing an acknowledgment of reading and understanding the employee handbook. This is where the mission statement and commitment to birds will be found. All other hiring procedures are to be followed. A lawyer should review the employee handbook yearly to be sure any changes comply with the law, and the same goes for a contract for an associate veterinarian. Be sure the employee handbook and any contracts clearly outline expectations for all parties.

Training

Working with birds can be both challenging and immensely rewarding. It requires specialized knowledge that is ever changing. A consistent and sustained training program is all important. For staff beginning in avian medicine, in-house seminars are a great way to build confidence. They are specific to the staff member's everyday job and give them a feeling of competence while learning on the job. Some examples of in-house continuing education seminars include proper diets and

diet conversion, low stress handling, restraint and treatment methods, wing and nail trims, and common normal and abnormal behaviors (see Chapter 46 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). These sessions can get much more complex, including topics such as phlebotomy, radiographic positioning, anesthesia, and common medical issues. Sending staff members to continuing education at local and national meetings is beneficial and depends on their longevity and value to the practice. Staff members are an investment and having a written plan on how to invest in them is crucial for their development, as well as the development of the avian portion of your practice. The important part is to not let the staff become stagnant and to stimulate their curiosity about avian medicine and their roles in providing this care for your practice. It will pay off in the long run for patient care and staff fulfillment with their careers.

Employee Retention

Retaining good employees starts with identification of the right employee that, as mentioned, is not necessarily about experience, but willingness and desire by the candidate. Identifying the right candidate is the most important step to retaining that employee. Spell out expectations so there is no misunderstanding of the goals at hand. Keeping them engaged and getting them to be confident in their daily tasks through ongoing education and training is more important than pay and benefits. With that being said, stay competitive with compensation and benefits because you want to take care of the people that take care of you, your patients, and your business.

AFTER-HOURS EMERGENCIES

As a veterinarian that has birds as part or all of the practice, accommodations have to be made for after-hour emergencies. Most state governments have regulations about providing care for patients after closing. That could include being on-call, having an in-house emergency hospital, or providing phone numbers for emergency hospitals. There are overall few veterinarians in many locations who prefer to handle their own after-hour emergencies. Being on-call may be that one is available to talk, but then sends to an emergency facility if deemed necessary. It may also be an option that the doctor meets the owner at the hospital, but the hospital is not officially open. Sometimes a hospital may elect to be open all night in addition to the day practice. Typically, when a hospital dedicates resources to having their own emergency service, they hire additional staff to work overnight or they may be available by being on-call. Practices successful enough to have internships or residencies, and which rely on them to handle emergencies for their clients, are in an enviable position to accept avian emergencies. Some avian veterinarians may pool resources with other veterinarians in your area to spread out the emergency workload. The group can rotate nights and weekend availability or even designate a location and fully staff overnight if the average number of cases seen is high enough.

Most of the avian veterinarians practice on other species and most would relish the opportunity to pass emergencies along to a competent dependable emergency room (ER). This opportunity will rarely just land in your lap, as it almost always requires considerable investment of your time to get such a luxury initiated and maintained. If your practice is located in a metropolitan area, then there may already be avian-boarded specialists or avian/exotic specialty practices in your area. This will usually be your best starting point. If they are using their own interns or residents to handle emergencies, they may be willing to see yours as well. The price of going in this direction is that you will inevitably and permanently lose some clients to the practice where you are referring your emergencies. This will happen regardless of the good intentions of the referral practice. If you cannot tolerate some small shrinkage of your client base, then it is wise to take this option off the table *a priori* and not risk creating a rift with your colleagues.

This leaves you with the final option of identifying an ER in your area that will at least consider accepting birds. The key to this strategy is getting an ER to make a long-term commitment. You will need to identify the one person, the Chief of Staff, Director, or owner, who has the ability to make a decision for the ER as a whole and who will agree to meet with you. Assuming you have cleared this hurdle, which is no small feat in itself, your work has just begun. First, you should determine why you were even granted a meeting. An owner may be interested in the additional income, or a director may have personal interest or experience with birds. Your colleague may just have been polite and did not want to risk losing seeing your dog and cat patients if they had given you a gruff "no way." Knowing their motivation to agree to meet with you will help determine how to get them on board.

Your next hurdle will likely be some version of "I have some ER staff who are willing to see birds, but I can't force my doctors to see them if they don't want to." This will be your opportunity to ask that if you are successful in getting their current staff to agree to see birds, will the ER commit to accepting birds now and in the future as they add new staff and doctors. In other words, you will promise to get their current staff up to speed if they promise to disclose to future employees that the ER treats bird emergencies.

Obviously, the next step is convincing all the existing ER doctors to see birds. This is going to be your most formidable challenge. Most ER doctors will have either no interest or no experience with birds, and some may outright loathe seeing them. The emphasis to motivate those doctors should be the following:

1. You are asking them to provide first aid, not become an expert in bird medicine. Most bird emergencies fall into the categories of trauma (including blood feathers), reproductive (especially dystocia), respiratory (tail bobbing and dyspnea), gastrointestinal (regurgitation and diarrhea), and the catch-all phrase of "bird at the bottom of the cage" (see also Chapters 28 and 34).
2. You can provide algorithms that can clearly and quickly follow through a diagnostic path and result in a treatment plan. You will also provide the ER with some reference books at your expense to keep in their library. Direct the ER to appropriate forums on the VIN (Veterinary Information Network) and supply them with an avian/exotic formulary. Make a Quick Reference chart of the most used drugs, their indications, and dosages.
3. You will be available by phone to answer questions, review radiographs or other diagnostic information, and develop

a treatment plan. You must not shirk this responsibility. The first time an unstable bird arrives at the ER and the admitting doctor is in need of help and cannot contact you, that may as well be the last bird emergency they will ever admit. Take this seriously. If you are going to be indisposed for any amount of time, call the ER first. If you are going on vacation, make sure an associate or colleague is available. If you commit to this part, then you will earn their trust and eventual loyalty to the mission of seeing bird emergencies. Over time, texts can be used preferentially, with phone calls being used for more complicated cases.

4. You must provide a series of in-person, hands-on wet labs for the entire ER staff including receptionists, technicians, and doctors. Bring birds to these demonstrations or request that ER staff bring any birds they own personally, if they are comfortable doing so. To instill confidence, see if different species are available with differing handling challenges, so staff are not surprised when numerous species come in the door. This is for basic history taking, handling, and working through each of the algorithms you have provided. You will need to provide refresher courses at least annually as new personnel rotate into the ER. There are many online resources that have excellent training resources.
5. You must become familiar with the ER facility and may need to assist with acquisition of essential equipment such as incubators. Provide vendor information to be sure they have medications commonly used in birds. Help assemble a minor surgery pack for birds. Provide the ER with brochures, handouts, or other literature that they can provide to owners. Remember that the AAV has an abundance of informational material designed for clients.

Every practitioner is going to have to make a calculation of whether the above venture is worthwhile. A practice that sees one or two birds a day may well decide to provide bird owners with a special number, possibly even the veterinarian's personal cell number, and see their own patient's emergencies themselves. Otherwise, you will have to identify that particular ER willing to help and then nurture them essentially for the life of your practice.

There is one stellar upside to taking the time to train an ER to see avian emergencies; you will have a new source of referrals to your practice. Once an ER has established a reputation in your area as always accepting birds, then other ERs are usually eager to refer them to your preferred location. The ER's website can include an invitation to bird owners with emergencies. An ER thus established can quickly see a remarkable number of patients. Many, if not most, of these clients will have no regular veterinarian. You can bet that if you have taken the time to train and encourage your ER, they will be more than happy to refer all of these people to your practice for follow-up. You should not underestimate what a potential asset this can be to the growth of your own avian practice.

LOW STRESS INTERACTION

After the decision making of how to set up your avian practice based on your predetermined goals, let us outline how to make your avian patients comfortable in the hospital environment. Typically, birds are comprised of mainly prey species and do not handle stress very well. Even predator species, like raptors, undergo significant stress when brought to the veterinary office. There is a certification for avian practice for those focusing on making the environment as low stress as possible. Benefits as listed by the Fear Free certifying organization are in Box 1.3. Fear Free principles can be applied to everyday practice to provide the most calming environment for our avian patients. Physical plant adaptations, aware and informed staff, minimizing noise, and behavior intervention strategies all can help birds tolerate our facilities and hopefully prevent significant effects of stress (see also Chapters 46 and 47).

BOX 1.3 BENEFITS OF A LOW-STRESS ENVIRONMENT

- Reduce or remove anxiety triggers that can cause pets to become fearful at home, in transport, and at the veterinary hospital
- Help owners deliver calm pets to your hospital
- Enhance the quality of medicine in your practice
- Increase compliance
- Improve safety for the veterinary team

From Fear Free Veterinary Certification Program website https://fearfreepets.com/

PRIVATE PRACTICE: MANAGING INCOME AND EXPENSES

Whether you are an owner or associate, you need to be more business savvy than veterinarians who exclusively treat dogs and cats. The avian practitioner has a few barriers to becoming profitable. The average transaction fee can be lower than for canine and feline patients, as birds do not regularly receive vaccinations, flea and tick preventatives, or heartworm products.[1] In addition, the number of patient visits per year may be less than what is seen in canine and feline practice. If diets are sold, they cost less than large bags of dog or cat food. Ancillary services and products for dogs and cats can be up to 12.5% of revenue, with an average of 10%.[2] This may include boarding, daycare, grooming, and products such as shampoos, skin care products, over-the-counter foods, collars and leads, litter, chew products, and toys. Pet birds may not always require these products or services to the same extent as their canine and feline counterparts. Some practitioners furthermore believe that they should charge less because the patients are smaller, which contributes to a lower average client transaction fee (ACT). However, generally more rather than less time is scheduled for bird appointments in order to obtain their more complex husbandry information, thus actually resulting in higher costs associated with the visit, although those are rarely charged to the owner.

It has also been found that over 40% of dog and cat owners indicated that cost prevented them from getting care for their pets and many seek out lower cost options.[3] Bird clients will also take advantage of lower costs provided though bird stores and breeders using routine grooming services, boarding, and obtaining diets from them. Potential clients may also look to online forums without veterinarian moderators for medical advice. All these factors erode client and patient numbers and revenue generated per client. To make private practice more

profitable for avian veterinarians, effective management strategies are needed.

Strategy Versus Tactics

By defining goals of how you desire to practice avian medicine, decisions can be made on how to become profitable. By answering questions about what our avian practice should look like and developing a written plan, we can then develop a strategy. It is important to differentiate strategy versus tactics. The overall action or plan is what is referred to as strategy. Tactics are the smaller steps one takes to achieve parts of the plan.

Before discussing techniques to become more profitable, there must be an understanding that patient care and client service is the most important goal. If we do what is best for the patient, profitability is not far behind. When the patient comes first, trust is built between you and the client. This trust is invaluable and results in returning clients and referrals. Importantly, this is why we went to veterinary school in the first place. When reviewing the strategy in your plan and the management tactics, it will become obvious that the patient is thought about each step of the way.

Managing Income

Although managing expenses is important, it is not the main road to success. Historically, expenses are what owners worry about most, but one cannot save one's way to retirement. Managing income has the greatest impact on your profitability. To understand income, there is a simple formula:

Number of transactions (n) × average client transaction fee (ACT) = gross revenue[1]

It is important to understand the components that make up both factors in the equation and the best ways they can be affected to produce higher income or gross revenue.

Increasing the number of transactions (n)

The first part of the equation reflects how many transactions occur in a defined time frame. If a practice wants to see more birds, that is an overall strategy that is part of the defined plan. Several different tactics can be used to achieve the goal of increasing "n," which include using efficient time management techniques, taking advantage of reminder systems, setting up recall systems for patient care, and implementing a solid marketing plan.

Time management. This is one of the biggest problems for avian veterinarians. Time is a precious commodity and should not be wasted. Having extra time allows opportunities to see additional cases, which increases "n." Additional benefits of effective time management also give us time to focus on marketing strategies, train staff, research clinical or management topics, and have meaningful time off. Three significant ways to optimize time include executing proper delegation, also known as staff leveraging, implementing proper scheduling techniques, and having a paperless or "paper-light" practice.

Staff leveraging. Delegation is defined in Webster's Dictionary as "the act of empowering to act for another." Within the veterinary profession, it is referred to as staff leveraging. Whether you are an associate veterinarian or owner, staff leveraging can make your life easier. Veterinarians are the only ones who can examine patients, obtain diagnoses, and perform surgery. Additionally, advanced follow-up cannot be done by other staff members, including discussion of abnormal laboratory results and complex treatment plans. Delegating properly also allows the avian veterinarian to see more patients during the day, which increases the "n" in the gross revenue equation. It can also increase the ACT by giving the veterinarian the opportunity to build trust with the client, contemplate possible etiologies of disease, and choose helpful diagnostics pertinent to cases. All other tasks should be delegated to staff. Empowering the staff allows your hospital to run smoothly and makes your staff feel that you trust and need them. It gives them a sense of team and prevents burnout, which is quite prevalent in veterinary medicine.[4] A happy staff is a productive staff. It boosts team members' morale and helps reduce turnover. Turnover can also affect profit, which has been estimated to cost approximately 6 to 9 months of the employee salary per year.[5] There is an obvious benefit to both the practice and the staff when delegation is properly implemented.[6]

There are many barriers to implementing delegation. Veterinarians tend to be the type of people who want to do everything themselves and can be perfectionists. It may also be because we feel that a staff member is not adequately trained, or that we feel better when we are "there." We may tell ourselves, "I'm already here" or "Everyone else is doing other things." We can get in our own way and it is a habit that has to be broken.[7] The differences between doctor-centered practices and client-centered practices are explored in *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 24, Practice Management and Risk Management. These excuses are used in a variety of practice settings daily but are quite common when diagnostics for birds are needed, including phlebotomy, taking radiographic images, and administration of anesthesia. Overcome these barriers with an attitude of empowering your staff, educating them, and fighting the urge to want to be involved in everything. Delegate only what you are comfortable with, but you should get comfortable with a lot. Even teach your staff members to delegate in an appropriate way.

As a hospital owner or an associate veterinarian, we know how to give staff tasks, but do we do it effectively? There is a fine balance, and delegation has steps to follow just as any medical or surgical procedure. Ask these questions each time you delegate and leverage the staff.[8,9]

1. Does the person have knowledge of the task?
   If the staff member does not know the basics behind the task, they will be sure to fail. Do they know the practice standard for the project they have been given? Education is the key here. This can take the form of a quick discussion, an in-house seminar, or a continuing education conference. Something as simple as getting together with your staff for 10 to 15 minutes can have tremendous rewards. A common example is that many practices that treat bird patients often task staff to discuss husbandry with the clients. Hospitals and clinics that have avian patients will routinely have in-house seminars to educate staff on dietary requirements for different types of birds so that communication is clear and consistent between staff members and clients.
2. Is the staff member interested in the job?
   This may seem like an obvious statement but is a concept that is commonly overlooked. Keeping the task related

to their position generally avoids this problem. Typically, one would not have a veterinary technician have bookkeeping responsibilities. Also, personality can play a role in delegation of tasks. Having an introvert in charge of client communication may not be the best fit. The reason that staff members still work with your hospital is that they get satisfaction from their everyday tasks. Take advantage of their interests and strengths. When identifying a task to delegate, meet with the staff member to gauge interest. Getting their input will be extremely valuable.

3. Are the expectations clear?
   Communication is the key here. Review what you want to have happen and put it in writing, whether that is in formal meeting notes or a quick memo for the staff member to review. That is done in the form of a treatment sheet for hospitalized patients. Why should other important tasks not be in written form? Too many times, we ask for things to get done and expect everyone to know what we want. Our expectations are high. If there is poor communication, the expectations can never be met.
4. What is a reasonable timetable?
   This is a skill that comes with experience and knowledge of the task and the person performing the task. Setting unrealistic expectations will only set up the team members for failure. If there is a husbandry issue, give the staff members enough time for a thorough discussion to take place with the owner. Shortening the staff members' time ultimately makes them fail at their task, which hurts the patient. In the end, this requires more time to be dedicated because the situation has to be revisited.
5. Did I give the proper authority necessary and let them own the project or task?
   This empowers an employee and allows them to achieve the goal. They become proud of their work and then want to take on more. This does not mean that the employee should not be monitored, but you can step back if the other points of delegation have been met. Depending on the project, it may require communication with others in the hospital, so they know the plan of action and who is responsible.
6. Will I be available if help is needed?
   This can be challenging because there is a fine balance of giving the staff member help and completing most of the project for them. This is when we may start to make excuses to just do the task ourselves. Fight that urge. This is another opportunity to educate and empower your staff members. This is what training is all about, train and train again.
7. Was there proper follow-up?
   Ask the staff member if everything went well with the task. This is a chance for you to make sure the work was completed and to learn what problems were encountered. Input is crucial. This is not only an evaluation of the staff member but also the process and end result. Evaluate objectively if there are metrics that can be measured. The process can be measured by direct observation, survey, or even by testing depending on the level of task.
8. Did I praise?
   This can be the most important part of the process of delegating. Team members want to be acknowledged. Just remember, it is not the "reward," but more the recognition. It can be as simple as a "thank you," giving a gift certificate, or baking some cookies. Just be genuine.

Now that we know how to delegate, who do we utilize? As an associate, it will typically be the internal staff that was hired by the hospital owner or administrative staff. As an owner, there will also be external support for the overall functioning of the clinic or hospital.

Internal staff

MANAGEMENT. One of the more important keys to this puzzle is the manager. This person can free a veterinarian from paperwork and provide the time and freedom to practice quality medicine and surgery. Hospital size will determine if a bookkeeper, office manager, hospital administrator, or someone in between is needed. It is generally accepted that management staff is paid approximately 3% of the gross revenue.[10] That number will define the type of person or people needed. Take time to research your needs and then choose the candidate. This person will change the quality of your practice, and your life. With someone else doing the office work, you are left to focus on doctoring your patients and informing your clients. Veterinarians who do not use their managers efficiently also have lower transaction fees.

The most delegated duties are accounts receivable, scheduling, hiring and firing employees, updating fees, and developing marketing materials. Other delegated tasks include ordering medicines and supplies, payroll, managing benefits, monitoring client and traffic flow, accounts payable, computer maintenance, problem-solving, staff meetings, ensuring regulatory compliance, and staff training. For office personnel to be more efficient for the hospital, they too should learn how to delegate tasks.

TECHNICAL STAFF. The number of technicians needs to be considered. Having too few will make a veterinarian's life very stressful at work. Patients will not be seen in a timely manner or appointment schedules will have to be adjusted to accommodate. Either way the number of patients seen will decrease. To evaluate your current situation, ask yourself these questions: Is an assistant putting the next bird in an examination room? Is the assistant getting a complete history and weight? Are the technicians getting the wings trimmed and collecting the laboratory samples as needed? Is the technician or assistant talking to the other owner about diet and then filling medications? The answers to all of these questions should be "yes." If there is a "no," evaluate the number and knowledge of the staff. Also, evaluate your ability to delegate technical tasks. A great way to insult your team is to make them feel as if they should not do a grooming procedure or obtain a blood sample. Technicians trim nails and get blood from dogs, why not birds? Train them. Trust them.

RECEPTIONISTS. The receptionist plays a crucial role in avian practice. They are the first contact for the client and the point of sale, which can make reception a common bottleneck in veterinary practices. Ensure they are properly trained so that they will ask questions and demonstrate genuine interest in the client and their bird rather than just answering the question that is posed. They should be able to do this in an efficient, but caring way. This builds trust, and the phone call

is more likely to turn into an appointment or other service. Reception handles phone calls so that the veterinarian is not swamped with calls about basic husbandry or simple medical issues. Good receptionists know how to delegate calls to the technicians, office staff, and finally the veterinarian. As receptionists are the main guardians of the schedule, their skills will lead to efficient scheduling. That in turn increases the number of patients helped and transactions per hour. Reception can make or break your quality of life at your practice.

ASSOCIATE VETERINARIANS. If enough well-trained, nonveterinary staff is available, evaluate if another associate is needed. Determine if a full- or part-time veterinarian is appropriate, and if he or she will practice on dogs, cats, or all species. Typically, if 80% of the schedule is full consistently, that is a good time to add an associate. A single doctor practice may need to add one before that so there is adequate coverage for any missed time by the owner. The addition of an associate veterinarian will allow the hospital to increase its number of transactions. Again, this has to be a careful, well-thought-out addition to the team. Over time, with good training and mentoring, the long-term associate will hone their skills and provide consistent care that reflects the practice's medical philosophy. The benefit of this is less management, which leaves the practice owner time for other responsibilities. A less obvious benefit is that a long-term associate provides balance. With a dependable associate, you can afford to exercise your entrepreneurial spirit. Rather than focusing on day-to-day effort of providing services, you can step back and look at the practice's direction. With this big picture view, you may find more creative solutions to persistent challenges. This associate should also be trained in the practice's protocols for delegation of tasks.

External support. There are veterinarians who elect to do much of their own financial and legal duties without realizing they do not have the expertise needed. A little research is done and then they believe it will be fine. This sounds like some of our clientele. This process takes considerable time in and out of the office and the best outcome is unlikely to be achieved. Payroll companies, accountants, financial consultants, and lawyers should be used to their potential. An office manager can organize this team. The veterinarian can see patients, while the office manager is getting information to the outside support staff. The time and headaches saved far outweigh the seeming cost benefits.

While staff can be used for some building upkeep, weekly cleaning services, maintenance, anesthesia, and microscope checks can be delegated to outside companies. When these duties are thrust upon the busy staff, this can distract them from taking care of our patients.

Scheduling. A schedule has to be built in the proper manner to fit your clinic or hospital doctor's schedule so that time is managed properly. Without a very good schedule, your day can turn into a mess. Good scheduling improves time management, which will allow you to see more patients, increasing your "n." The schedule has to fit your style. Do you work well with multiple cases going on at the same time, or do you need to have your head clear for the next case? Do you tend to be very chatty, or are you moving along with urgency? We all can answer yes to these questions but know your tendencies so you can make the best plan for a schedule as possible.

Building a schedule starts with knowing when your clients need services. Typically, patients are seen in the morning until noon, then there is some sort of gap to accommodate breaks for veterinarians and staff, and then appointments start for the afternoon or evening. Surgery will be added in the gap between appointments or there may be a surgery day for each doctor. Whatever style fits your caseload, client need, and number of doctors and staff should be adopted.

Within the general blocks of surgery and clinical appointments, individual time slots have to be defined. The number of surgeries that are scheduled will depend on how many surgeries are performed on a weekly basis, number of doctors in the practice, and skilled leveraged staff. As the avian component of your practice is growing, these factors will change, and the schedule should be adjusted for growth accordingly.

Individual clinical appointments also have to be outlined. Each appointment has to have a placeholder in the schedule. Is that going to be every 15, 20, or 30 minutes, or some other defined time frame that works for you? Do you want to consider if new or sick birds get longer appointments? Do you want to have emergency or "catch-up spots" in the schedule? One strategy that leverages staff that practices can implement is a layered or high-density scheduling. This is when, at the beginning of an appointment, the technical staff will spend 5 minutes with the client getting basic information and some history. The next step is that the doctor will spend 10 minutes with the client and patient getting more detailed history, performing an examination, and developing a plan. After that the doctor passes the appointment back to the technician to get outpatient diagnostic samples collected or discuss with the client behavior change recommendations or diet changes. Layering starts when the doctor is with the client and patient, the technician starts the process with the next client and patient in another room by getting basic information and history. Then the doctor comes into that room with the second patient, and the technician goes back into the room with the first patient (Figure 1.14). Time frames can be extended to accommodate appointment length that the hospital has adopted as their norm. More patients can be seen when working in this efficient manner.

Paperless or paper-"Light" practice. One way to increase efficiency and lower costs associated with human resources and office supplies is to convert paper patient files to a paperless system. This system will utilize your practice management software (PMS) along with other process flow tools to cut out the need for handwritten records. Ideally, anything handwritten to a client and/or patient paper folder is now recorded in your PMS. The real efficiency with converting to this new way to manage your information process flow is seen around time management. If you choose a paper-light method to manage your patient files, this means that you will still utilize your PMS for your client database and most record keeping, but you may choose to keep some items in a paper file as well.

To illustrate the efficient benefit of going paperless, as an experiment one of the authors asked reception staff of a four-doctor hospital to start a timer when they were asked to look for a patient file in a paper record practice. When they found the patient file, they stopped the timer. It was discovered that within a 12-hour day, receptionists spent 3 hours looking for files. This does not include the time a doctor or technician

TIme	Exam room 1	Exam room 2
9:00		Blocked off
	Cockatoo: Exam, break, wing nail trim	
9:20		Macaw: Initial exam, poss broken leg
9:40		
	Budgie: Exam, not eating, on bottom of cage	
10:00		
		Cockatiel: Recheck liver values
10:20		
	African grey: Exam, feather picking	
10:40		
		Conure: CT scan consult
11:00		
	Chicken: Exam, wellness	
11:20		
11:40	Catch up	Catch up
Key:	Veterinary assistant: Take history, trim nails, provide general treatments	
	Doctor1: Exam and specific treatment protocols	

FIGURE 1.14 Layered scheduling of patient visits can be helpful to increase practice efficiency.

spent looking for files. After switching to a paperless system, customer service improved dramatically because there was less time looking for files on desks or in obscure places within the hospital. Receptionists could get answers to client concerns of the moment, as the information could be called up on their computer screen in real time. Another wonderful benefit to going paperless is that records become legible. Templates make filling out an examination much faster and more efficient. Electronic records can be gathered to share with specialty hospitals, which can be sent by email rather than having to spend time and money using a fax machine that requires more paper, not to mention the price of toner. In the above example, paper waste was significantly reduced resulting in fewer trips to the recycling dumpster, which can account for savings in trash removal.

Additional benefits to utilizing PMS to go paperless extend to your lab work. Clinical pathology results can be imported from in-house laboratory equipment or external reference labs as soon as the results are ready. Signature pads for numerous electronic forms can be incorporated into the system, as well as credit card processing. Veterinary software or applications can help manage inventory by accurate counts, automating shopping or "need" lists, and giving alerts when an item is low. Time clocks are also included with PMS, so staff can clock-in when they log into the system upon arrival. In most cases, those same clock-in features extend to security tracking where you are able to generate reports on what functions users are performing within your software. This is beneficial if you are suspecting that someone is inappropriately using the clock-in system, or even in a case of suspected theft. Veterinary paperless applications typically have an audit trail to identify changes that were made within the system. Additionally, generating financial reports by inventory, treatment procedures, doctor activity, or area of the business such as boarding becomes very easy and allows you to focus on important details of your practice to achieve your goals.

Having a paperless or paper-light practice saves time for all staff and doctors, but what will be needed? Must-haves include a server that is located either in-house or offsite, or a cloud-based system that utilizes the internet to deliver your PMS as an "app"; examination room stations to enter data while with clients ranging from desktops, laptops, or even tablets; a wired network, reliable internet access particularly when utilizing cloud technology; and onsite and offsite backup capabilities. Getting whatever data in the current system to the new system is simplified with the veterinary software company converting any necessary data, so their help will be paramount.

If you run your practice with paper files and are thinking of going paperless, a consideration to the overall management

of the conversion will be if a decision is made to scan all previous records for a patient or "draw a line in the sand" and start entering all data anew. If the decision is made to scan all previous paper records to your new paperless system, consider which key staff members will dedicate hours of time to this project. Depending on the number of files, the completion of the work may take quite some time. Training for existing staff members is crucial when making the switch from paper to paperless, as well as for new staff members who may be learning the system for the first time. Some team members may have used the system at a previous job but will need to learn how your hospital integrates the system into everyday practice. As each PMS suite is different and so are your individual practice's goals, it will be helpful for you to map out what areas of the software you plan to utilize in relation to the way your clients and patients actually flow through your healthcare and customer service processes within your hospital. In addition, because you are an avian veterinarian, it would be important to keep in mind that when shopping for software, some packages may not cater to the healthcare delivery systems for birds. Customization is key in regard to species and breed lists, as well as the ability to work with lab-specific data within the software.

Overall, the conversion to paperless or paper-light record keeping is a lengthy process that takes time to plan and execute. The results of the project include freeing up many hours per week where staff can redirect their time to more efficient activities. Rather than the employee spending 3 or more hours a day looking for misplaced or misfiled patient records, a receptionist can redirect their energy to making appointment confirmation or follow-up calls, and other revenue-generating activities. Customer service and patient care will improve. Ultimately, the expense and time required to go paperless is well worth the increased efficiency, time, and monetary savings that your hospital will enjoy once the conversion is complete.

Client reminders. Client reminders will increase "n" by creating another point of contact with our current bird owners. This can take form of an email, text, personalized newsletter, or traditionally, a postcard. Your PMS should have a section to build regular reminder templates for yearly examinations, vaccination schedules, behavior programs, senior wellness, or other medical plans that you have tailored for the patient. In addition, you may prefer to set up semi-regular reminders for grooming appointments for birds if a client regularly requests certain services or you feel are required. This is becoming more commonplace in avian medicine, and continually reviewing and applying reminders for your avian patients will boost revenue by increasing the number of visits. An annual screening program can uncover diseases in apparently healthy dogs and cats, thus boosting your level of medical care and your revenue. The same concept should be explored, applied, and maximized for avian patients whereby regular reminders for these programs are sent to the client.

There are several ways to send reminders. Among the most efficient is having a company that links to the veterinary software application for your practice and have them send the reminders by email, text, or postcard. This not only gets the reminders out in a timely manner but also delegates a time-consuming job to an outside source. Some service providers bundle reminders with other digital marketing plans to maximize their partnership with your hospital and provide aggressive pricing for their competitors. Alternatively, this job can also be carried out by your staff. The benefit of doing them on your own is determined by weighing all costs, time involved, PMS capabilities, and timeliness of delivery.

Recall system. One expense that your veterinary practice incurs each month is your phone bill. The following statement sounds counterintuitive, but as we continue on this topic, you will see the logic: you want your phone bill to increase! A higher phone bill equates to clients returning to your practice for ongoing care. If you see a new client for an initial wellness examination and identify a specific concern, would you just have the client check out without a plan to manage their concern? You would be proactive and follow-up with them, likely with a call. Most veterinary software applications have a built-in system that allows the users to input callbacks for their patients. This means a veterinarian or staff member will have access to a list of clients that need follow-up each day. Do not just call a client when they call you. If you find yourself saying, "Let me know" or "Give me a call if there is a problem," you are missing an opportunity. Recalls by design are meant to know what is happening with any given patient. There is no guess work. We have all talked to clients who do not truly assess their bird's health until we start asking questions.

Most veterinarians will make follow-up calls for sick patients, but a call for any issue is a good call. Call for the ill cases, but also for rhamphothecal shaping procedures, annual or semiannual laboratory testing or imaging studies, weight checks, 1 day postsurgery, diet conversions, medication administration, chronic medical conditions, and behavior modification monitoring. Any concern, no matter how small, should be called to be sure things are going the way we as veterinarians would want. Technicians and receptionists can call less critical cases, but if you as the veterinarian have the opportunity, make the calls yourself. While delegating is important, this is one task that will be very rewarding all around if you are able to make the call personally. "I wish my doctor would call me" and "Can you be my doctor?" are commonly said by clients when we call.

Recalls increase "n" by a few different means, one of which is you are showing you care when you call. Not only is it a great follow-up for the patient, but also it builds a trust between your hospital and the client that will last for years. When there is trust and a bond, you will get the call the next time there is an issue with their bird. If the patient is not progressing as well as expecte d, you will know and get a recheck scheduled. Many clients will not call and wonder why their bird is not improving or they may not recognize that it is not going well until detailed questions are asked. This will lead to more appointments, diagnostics being performed, and medicine being dispensed, all while doing what is best for the patient. The other way recalls help is that when you first start working with birds, you know technically what should work according to the books, but that may not be the reality. Client input is imperative to your learning process. If you are not receiving the input, you will not know if the medicine or surgery worked well. You will learn what challenges the owners have while administering medication or changing diet. We tend to be teachers to our clients, but we should not forget that they teach us as well.

Your practice should generate daily follow-up call lists. The cost of a higher phone bill is a welcome expense when you consider the increased patient visits and positive perceived value of your practice. Clients really appreciate it when their doctor calls to check on them, and it shows your commitment to providing the most comprehensive and compassionate care. These calls build trust, education for the client and veterinarian, healthier birds, and more patients for the long term.

Marketing. Managing a successful veterinary practice involves strategic planning within and between several functional areas of the business; just a few of those areas include finance, human resources, inventory, operations, and marketing. The hospital's marketing strategy should provide a clear depiction of the specific elements that set your practice apart from the crowd. The marketing strategy's key elements ideally include defined internal and external marketing components, brand messaging, print- and digital-based marketing tools, word-of-mouth communications, and social media. All these marketing mediums work together to reach and engage different types of consumers that would benefit from your services. The success of these marketing efforts depends in part on the practice owner's understanding of the spending power they have with which to dedicate to campaigns, as well as expenditures in other functional areas, relative to gross revenue. Having a general understanding of the different types of marketing tools and their costs will help a practice owner determine specific marketing mediums to focus on relative to their overall return on investment.

Internal marketing. Marketing your veterinary practice starts with internal efforts that define the standout features and beneficial attributes of your hospital or clinic. When we internally market, we treat our internal staff as the client, in effect "selling" the significance of our products and services to our team as a means to create a unified marketing foundation that will be communicated by your practice as one message.[11] The internal marketing effort is ongoing so that any changes to products, services, or new capabilities are communicated to clients through your staff. Internal marketing seeks to communicate and unify the message of what your practice is at the core of its belief system, specific abilities, and its process of executing animal care. The continual effort should highlight your core mission, methods, and tools you will use to deliver your services. Hospital design, process and procedure protocols, technology, operations management, your team's expertise, and the company's culture and values all contribute to how you deliver your care; therefore, these internal attributes can and should be highlighted as positive features that set your business apart from other practices. Whether you have an ABVP-certified avian practice diplomate on your team or your nursing team is highly skilled in treatment and handling of birds of all sizes, the overall marketing strategy should emphasize these key offerings as a means to elevate the perception of expertise of your practice in comparison to others in your local demographic area. Internal marketing seeks to ensure that your team understands the value that your hospital's unique attributes provide and contribute to the staff's ability to perform services. Their understanding that your practice's unique qualifier as an avian veterinarian surrounded by a skilled team builds the well-defined mission and vision focus that the staff will adapt as its cultural foundation. This unified core value system will help staff execute the overall strategy of the practice owners.

The first step to building an Internal marketing scheme can start with defining your mission statement. What is the purpose of your practice beyond the obvious goal to provide healthcare to birds? It goes without saying that you are in business for pet healthcare; but what is important to you as a service provider? What do you want clients to know about your beliefs as a veterinarian? Whether it is your compassionate care, your specific medical philosophy, or your respect for client concerns, the mission statement is a few sentences defining your commitment to your clients and pets. An example could include a statement like, "We at 'x' hospital strive to provide complete and compassionate avian medical services that focus on proper client education to support happy and healthy lives for both the birds and their owners." Once you have this statement finalized, you now have a philosophical foundation on which to build your internal marketing scheme for your team where you outline how you plan to fulfill your mission statement's intended goal. Continual review of your mission and values will achieve and maintain a cultural understanding and acceptance of your vision, mission, and capabilities. This is vital to a clinic's overall marketing strategy, because ultimately the staff is acting on your behalf to broadcast this unified message to your clients.

External marketing. External marketing efforts seek to bring awareness of your hospital or clinic capabilities to the consumer. Just as you sold your mission, vision, and level of capability to your staff through internal marketing, you will sell those same concepts to your current and future client base. Several tools help build a successful external marketing strategy that includes brand recognition, print marketing, phone on-hold programs, referral programs for clients, word of mouth through local veterinarians and pet organizations, postcard and/or e-reminder systems, recall programs, and digital marketing efforts. These external efforts seek not only to broadcast the attributes that set the practice apart, but also, perhaps more importantly in today's technology-driven world, to establish consumer loyalty and to solicit feedback about services, experiences, and opinions that can lead to further enhancements for the practice. Utilizing marketing tools that work to boost client engagement can solidify the doctor-client relationship and increase the client's perception of your value. That client's loyalty will create a bonus marketing tool where the client will become an advocate for your practice!

Brand recognition. Creating a solid brand foundation helps to build a visual representation of your hospital. Your color scheme, logo, and printed templates provide brand recognition for your clients when you apply them to all visual marketing efforts throughout your business. It is therefore important to create these templates and logos with the goal of utilizing them uniformly throughout all practice marketing efforts. Educational materials, signage, fonts, logos, staff uniforms, and even paint color schemes within your hospital should all be consistent to create a homogeneous and familiar feel to your service delivery, with additional care to create a clear differentiation of your clinic as an avian veterinary practice. For example, an obvious way to define your brand would be to craft your logo with birds depicted. With a solid visual representation, your marketing material will highlight your specialization as an avian practice. There are several ways to

effectively showcase your brand and the unique traits of your practice. Utilizing both visual and word-of-mouth tools will provide a thorough representation. Some of these tools include the following.

Print marketing. Use your logo, streamlined font, and color schemes across all printed material, including:

- Brochure about your practice's history and service offerings, with hours, contact information;
- Business cards that include all doctor contact information and brief description of services;
- Signage that uses your colors and fonts;
- Scrubs or other uniforms that marry with the visual representation of your practice; and
- Pet handouts about husbandry that are formatted using your marketing template.

Inexpensive options for creating these materials exist online. If you are comfortable enough designing them on your own, there are many websites that help with inexpensive bulk pricing for printed materials. If you prefer, you can hire a print marketing company to draft content for you, but obviously, this will be at a higher cost.

Phone on-hold programming. Incorporating an on-hold recorded messaging system to help to call out specific services, events, or other offerings unique to birds is a useful way to educate clients on your unique capabilities while they wait. On-hold messaging services are provided on a monthly basis where a recorded message can be created with looping content that you can customize to your unique offerings and concerns of the moment. For example, if you provide boarding, you can call out specific holiday times in your on-hold message to remind owners to make sure they book their reservation before you fill up. You can also call out specific services such as bird grooming or that you provide international travel documentation for pets. This service is low cost and easy to customize. The company will provide you with a sample menu of topics to choose from, and you can also make sure your website, social media pages, or other online content are called out in the recording.

Referral programs. Referral programs are a great way to engage clients in supporting your practice by word of mouth and are a large source of new clients.[12] Creating a referral program is simple: come up with a reward for clients to receive if they refer someone to your practice and create a tracking process for referrals. For example, a great way to learn if a client was referred to you is to place the question on a new client form that gets filled out at the first visit.

> *How did you hear about our practice?*

The new client enters either the name of the person who referred them, or another source like your website, or a search engine. Once you have the name of the person who referred them, you can gift them with a credit on their account or something else, like a coffee shop gift card of a small amount. Little gestures of thanks can be a great way to further engage your client base. The bonus of asking this question is that you can create an ongoing study of real trending data on how clients find you.

Local veterinarians. Being an avian practitioner will afford you opportunities to become the trusted veterinarian where colleagues will be willing to refer bird cases. Offering your expertise and services to surrounding vets will elevate your practice as a unique service provider to your local veterinary community. A great way to market your practice in this arena is to periodically send out branded newsletters with any important updates or to drop off your brochures and business cards to other practices that may have an occasion to send bird cases elsewhere. Spreading the word about your unique capabilities will establish your practice as the go-to veterinarian for referrals.

Bird stores/bird clubs/wildlife rehabilitators. Partnering with local pet stores and rescue organizations that handle birds will generate referrals. Becoming a trusted veterinarian to pet stores by providing general care at perhaps a slight discount (i.e., 10% off services) will help pets receive proper healthcare. While it is not recommended to discount services, most corporate pet stores will ask for one. It can be considered an advertising expense or part of the referral program that is in place. Local bird clubs are a valuable source of clients, and the members look for any educational opportunities that will help keep their pets healthy. Spend time speaking at these clubs, and you will see the benefits by helping more families with birds and an increase in clients. The same is true for wildlife rehabilitation organizations. These facilities receive countless wild animals and need access to healthcare to nurse patients back to health and release them back into the wild. Your expertise as an avian practitioner means that hawks, eagles, and other wild birds will benefit from your services when partnered with a rescue group that rehabilitates raptors and wild birds. This is another marketing opportunity for the practice highlighting goodwill, sense of community, and environmental awareness.

Digital marketing tools. The communication tool with arguably the widest reach is technology-based through use of the Internet. Gone are the days of taking out a color advertisement in the Yellow Pages. Today's most successful marketing tools reside online and provide living, real-time statistical data to help practice owners understand who is looking for you, when, how often, and why. A website is the grown-up version of that phone book advertisement where veterinarians can showcase their unique services and capabilities and communicate any other specific information such as the addition of a doctor or piece of diagnostic equipment that increases perceived value to target clients. Websites can incorporate search engine optimization (SEO) tools, which increase visibility in searches by using keywords and code to elevate your website on search result listings. For example, by utilizing *bird* or *avian vet* along with your town name as keywords that are coded within your website, you elevate your webpage to a higher spot in search results when a client enters those words *<town>*, *<bird veterinarian>*, in their search bar. As an avian veterinarian, your website will naturally contain these and other species and location-specific keywords that will aid in utilizing SEO to elevate your practice and create visibility to bird owners in your local market. Some built-in website management tools even include call-tracking analytics to help you understand where calls are coming from and show peak call time data. If you are reading this and becoming overwhelmed at all of the potential work this means for you, do not worry—you already have enough to do! While SEO does generate demographic and consumer activity reports for your review, you otherwise will not be adding more work to your pile if you employ these digital tools because they are automated

and run without your input. You can choose to review regular reports through website tools, though it is probable that the web service will generate that data for you on a continual basis.

In addition to your own website, other sites that support growth of your online presence include popular review sites, as well as social media sites. These online partners can contribute to statistical data or analytics, by providing details about click-throughs, which occur when a client visits any of these sites and clicks on a link to be redirected to your content, or clicks on your phone number to place a call to your hospital from their mobile phone. It is useful to review these data to see where clients are located and how often they are clicking-through to gain an understanding of the percentage of calls and subsequent visits that result from your internet presence. Some light maintenance is required from your side where you need to respond to reviews and monitor client feedback to handle any concerns presented for public viewing. Engaging with clients online in this way is very valuable to positively managing your online reputation. It is recommended to delegate this task to one of your office personnel. Once the data are collected, analyze them to determine if they fit with your current marketing strategy or if there is a need to make changes to achieve your overall goals.

Social media. In addition to having an electronic "billboard" in the form of your website, it is important to recognize social media as a major avenue for direct communication between you and your clients. Social media sites and your website's blog section all provide additional ways for you to deliver specific content and engage with your target audience. Avian-specific messaging can elevate your perceived value in your local veterinary marketplace as the first choice for treatment of birds. Also, the social media content allows you to broadcast custom subject matter to your audience where you can post about your staff, special pets, equipment upgrades, changes to hours due to weather, and other need-to-know information. Although social media is generally a free service, you can elect to pay a small fee to run ads that may achieve a greater reach to current and potential clients who may engage with the ad and seek out your services. Some social media sites also provide insights into visitor engagement by reporting on page visits, "Likes," and click-throughs. To properly monitor these tools, do recognize that you or a staff member will have to set aside a few hours per week to regularly post content, reply to client engagements, and review analytics on social media, which goes back to budgeting for marketing costs. Social media only works to engage clients if you are actively tending to the content and analytics results, and you must consider your time's value and payroll costs associated with this in relation to the return on investment that your newfound client engagements will bring. These sites are very client facing and they perform for your business, but you must pay regular attention to them. Less is more with regard to posting content—you want to engage scrolling clients with a picture or video and then post your comments with a brief explanation. Whether it is an employee promotion or the addition of a bird-specific laboratory testing capability, you should grab attention with a visual item and then follow through with your commentary. Remember, engagement is everything. Posts that ask questions or request feedback are a great way to increase your social media presence.

Other client engagement e-tools. Taking advantage of an e-marketing suite of services helps create a level of online engagement between you and your clients. This engagement is really important to some groups of people who through technology are seeking a more involved level of communication with their practitioners because it really enhances their perception that they are an active partner in their pet's healthcare. E-marketing tools can include a mobile phone app that provides access to pet records with notifications; a client survey tool to invite feedback; a reminder tool that sends out appointment notifications on your behalf that can be via mobile phone text and/or email in real-time capability; and regular newsletters that can include stock prewritten or custom content. The client survey tool has an additional benefit—if the client fills out the survey and it submits a positive rating, the client will automatically be invited to rate your practice on popular review sites, which increases a more favorable online reputation.

It can be daunting to think about the digital side to marketing, particularly if you are not technologically savvy. The important thing is not to be overwhelmed. There are plenty of reputable digital marketing companies who cater to veterinarians with packaged offerings for these services. Tailored plans can be created to suit just what you need, and payment for these bundles can be managed over a short-term or longer-time span. It would be best to receive quotes and demonstrations from a few different companies to make sure you are building an e-marketing package that best fits your needs and budget. For example, you may not want to employ their services to manage your social media content, which will lower your package cost, but require more weekly maintenance from you.

Marketing budgeting. With an understanding of the different types of marketing tools available, it is necessary to create a marketing budget that is comfortable for your practice. Cost analysis for these activities should include sending out monthly reminder postcards and associated postage, print marketing, on-hold messaging, your website hosting company fees, the electronic suite of services that include the mobile phone app, the survey feedback tool, automated newsletter capabilities, and wage costs of staff to manage social media and other tools as necessary. Most of the technology-based bundled offerings are available as a monthly or annual pricing structure with a signed contract, and most of these marketing organizations will offer a considerable discount for prepayment. Your goal is to spend within your budget's means in order to increase client awareness and utilization of the specialized services available at your avian practice. Typical costs for advertising and promotion of services would be 7% of total revenue (Table 1.1).[13]

Ultimately, communicating your range of service offerings and avian healthcare special interests through multiple avenues will reach several different types of clients to increase perceived value and thereby contribute to increased revenue and client/patient counts. Planning a marketing strategy starts with your own goals. Defining how you want your practice to grow as well as communicating that goal to your staff is the first step in your marketing plan. Spreading awareness of those goals comes next where you and your staff present a homogeneous message of your brand to current and potential clients through in-house communications, print marketing,

TABLE 1.1

Practice Expenses as a Percentage of Gross Revenue

Promotion of Services/Advertising	0.7%[13]
Medical Supplies	17.5%[13]
Other COGS	5.9%[13]
Laboratory Fees	3.6%[13]
Rent (Location Dependent)	7.8%[22]
Total Labor Expense	39.6%[22]
Other Employee Benefits	0.5–3.4%[22]
Other Operational Fees	4.0%[13]
Net Profit	20–30%[18]

word of mouth, and digital avenues. Examining your revenue relative to expenses in other areas will help determine how much you can comfortably spend on marketing efforts and should also help you decide what tools to focus efforts on when creating and maintaining a marketing scheme that continually contributes to positively trending client and patient statistics and a favorable return on investment.

Increasing average client transaction fee

An increase in ACT makes a big difference in gross revenue without seeing more cases. In our equation, number of transactions (n) × average client transaction fee (ACT) = gross revenue[1], ACT is easier to increase by $5.00 than what it takes to get five new clients. Price increases, charging appropriately, practicing quality medicine, addressing missed fees, and adding in ancillary services are efficient and productive ways to increase ACT.

Price increases. Increasing the fees will directly boost revenue. If this is done appropriately, it can be easy and effortless for both veterinarian and client. Increases should be scheduled minimally yearly for services like examinations and surgery. Laboratory fees should be increased yearly or if the labs used increase their fees more frequently. Inventory items should be increased as the distributor or wholesaler increases them. Medications can increase dramatically from one bill to the next because of a manufacturing issue, supply chain disruption, or demand has changed. Be sure to monitor your expenses all around to manage price increases properly.

When increasing fees, it is crucial to educate employees of how and why this increase occurred, so that they can relay this to the client if questioned. When educating employees, it is important to explain how the fees are determined and what costs the practice incurs to offer patients high-quality care.[14] This will aid in comforting clients when they are well informed of the high-quality care they are receiving for their payment.

Services should be based on value, and this works very well when pricing surgery, examinations, and boarding.[14,15] Ask yourself, "What is my time and skill worth"? Do not feel guilty, as it is expensive to practice quality avian medicine. Review your bills to remind yourself of that fact. Typical fee structure for laboratory tests is the cost of the test at least doubled, with the possible addition of a nominal blood draw fee. Inventory and medications are usually priced around two to two-and-a-half times the hospital cost.[15] There is usually a pharmacy packaging fee to cover costs such as ordering time, filling the medication, labels, drams, or other containers. Others may elect to have a minimum pharmacy fee because some products may be very inexpensive, but still require work to have in the hospital and deliver to the owner.

Charge appropriately. It is important to remember that it is expensive to practice quality avian medicine and surgery. Many avian veterinarians will have lower fees, or feel they need to charge less for a budgerigar than a grey parrot than a cat or dog. Technician time, radiographs, anesthesia, surgery, and appointment times are essentially equal for those two birds and can easily be greater than for a cat or dog. The fees should reflect that fact. You are charging for your materials, knowledge, and time. Remember that a smaller patient does not equal a smaller fee. Discounts are detrimental, as they minimize our value as veterinarians as well as the profit we work so hard to maintain and grow. Charging proper fees is not only appropriate given your skills and education, but also necessary to properly invest in your staff, hospital, and building to provide the best for your patients.

Practice quality medicine. We all strive to practice quality medicine and surgery. This is the greatest part of our careers; not only does that satisfy our desire to be an avian veterinarian but it is also what allows us to be profitable. Having a strong knowledge and skillset base for avian practice allows us to offer a variety of services like preventative care, inhospital diagnostics, radiology, ultrasound, CT, endoscopy, soft tissue surgery, orthopedics, behavior consultations, and senior wellness programs. These can function to increase the average transaction fee dramatically and provide opportunities for the exciting, career-enhancing problems and procedures we seek. To gain this knowledge, attend and participate in meaningful continuing education. Do not just meet the minimum requirements. There are many opportunities as discussed earlier in this chapter and elsewhere in this book.

Address missed fees. Missed invoice items occur at every practice. For one reason or another, fees are missed. By conducting an audit of what fees are missed and identifying the reason why they are missed, the average fee per transaction can easily increase. Once the cause is determined, policies can be instituted to reduce or eliminate missed fees. Veterinary software can help track and reduce human error, which is another advantage to becoming paperless or paper "light." You should implement a system, whether paperless or not, that you and your staff are comfortable using to track missed fees.

Additional services and products. Any program or service that is offered can add to the bottom line. In avian medicine, best practices include postpurchase screening, routine blood testing, and comprehensive senior programs. Another service may include behavior modification. Generally, consultation for behavior is more costly than a regular appointment as it is more time consuming. These services will increase not only the number of transactions but also the average transaction fee. Many avian veterinarians will have formulated avian pellets in the hospital for sale. This is many times added onto a final bill, which increases the average transaction. Adding ancillary services is where you can pursue other interests or get creative. Grief counselors, boarding with additional services like play time, and the sale of toys, grooming equipment, and diets are some examples of services you can offer your

clients that are not only good for their birds but also will add to your profit.

Managing Expenses

As mentioned previously, increasing income is the best way to make your hospital profitable. This should be the main area of focus, but do not forget about expenses. The main goals when watching expenses are to not be wasteful and to be responsible with spending. This sounds very obvious, but when reviewing practice expenses, it is common to find a lot of waste and unnecessary spending. When evaluating expenses, they should be compared to gross revenue to get an expense ratio or percentage. Table 1.1 outlines expense percentages that a veterinary practice should maintain. If you can improve upon those percentages, profits will increase. Review of these percentages monthly will keep a hospital on track.

Many costs are relatively fixed, and there is not much you can do to change them, such as utilities, waste removal, and postage. Have a manager shop for the best prices for these and other relatively fixed expenses. Activities like these are not a cost-effective use of owner time. There are two main expenses that can be changed through effective management to become more profitable: inventory control and payroll costs. Rent is another expense you can exert some degree of control over. Once set, rental expenses can be challenging to change, so this should be well thought out prior to signing a lease.

Inventory management

There are many veterinarians that want what they want with regard to medication in the hospital, but this must be managed so as not to have excess inventory and spending. There are several systems in place that practices use to monitor inventory. Those systems can be automated through veterinary software, or they can be manual counts and physically tracking inventory. Whatever system is used, it should be monitored to minimize waste in the form of expired medication, having excess on hand, or loss through theft. It is best to assign a staff member and a backup to manage inventory.[16] Controlled substances require additional documentation that meets government standards and licensing requirements, which tends to get veterinary hospitals to manage them very tightly. Note that there are numerous medications that will be important to have on hand when seeing avian patients that will likely include compounded forms. These will likely require special attention to source these items at reasonable costs and shipping lead times.

Whatever systems are used to track inventory, consider implementation of just-in-time (JIT) ordering. JIT was recognized by Honda Corporation to have parts for vehicles ordered as they were needed.[17] The ideal situation is that as soon as something is needed, it "appears" and then it is used or sold. Because pharmacy items and medical supplies do not just appear, the systems in place should account for ordering and shipping lead times. With online ordering systems and overnight shipping that most vendors have available, minimal stock needs to be kept on the shelves. The rapid ordering and delivery allow us to act like a big corporation, just the way Honda has developed for managing inventory.

JIT prevents excess inventory, which has a cost. That cost is the amount equal to the physical product that is constantly sitting on the shelves unused. Extra inventory also takes up space and the money generated per square foot decreases for what a hospital or clinic can produce. Order what you need by checking history for sales of an inventory item, which can be done through your PMS, as well as through your distributor accounts.[16] See how often you sell those products and then order accordingly. Financially responsible practices typically spend roughly 17.5% of gross revenue on medical supplies (Table 1.1).[13] Continue to check your percentage and alter ordering habits appropriately. This also includes regularly reviewing product pricing and sources to ensure you are purchasing the most cost-effective product. It is also worth negotiating discounts with distributors. Many will also offer discounts for paying invoices by automatic debit from a business bank account.

The use of online pharmacies will also decrease what is on hand. For medications uncommonly dispensed, consider working with an online pharmacy that is aligned with your hospital where some profit can be realized by the practice. There are also compounding pharmacies that will mail directly to the owner's home. Take advantage of these resources to minimize stock on hand that does not rotate with regularity, while still earning revenue for the hospital.

Payroll

Payroll expenses are a factor of how efficiently a practice runs and the pay rate of employees. The percentage of gross revenue for all of payroll should be 40% or lower, with 20% going to veterinarians and 20% dedicated to all other staff. Many practices have too many employees or nonproductive employees. Following the rules of staff leveraging will help manage both of those issues. A starting point to evaluate the number of staff is a doctor-to-staff ratio of 1:4.1.[13] Identifying excess, like inventory, is vital to having a productive, fully functioning, and happy staff. Proper scheduling prevents overtime and burnout, both of which increase payroll.

To establish the pay rate per employee, there are resources available that evaluate pay by year, position, and area of the country. You can find these with the Veterinary Hospital Managers Association by having someone in the practice as a member of the organization. It is very worthwhile to be a member as an owner, interested associate, hospital administrator, or office manager.

Rent

Rental expense can be one of the more complicated calculations to determine. A quick calculation for rent would be 5% to 6% of gross revenue. Major metropolitan areas may command a higher rent closer to 8% or more. If you own the property and the veterinary practice, you may go above that percentage to pay your property loan and have an additional distribution to you through rent. In this scenario, one must keep in mind reasonable rent when you go to sell the veterinary practice goodwill. At the time of practice sale, excess rent will either count against your profitability and subsequently value, or it will be added back to the profits, and you will have offered a less valuable lease as the building owner.

Another more accurate way to calculate rent is to have a commercial real estate professional establish a proper rental rate. They will collect per square footage data from the surrounding areas and match that to your hospital.

If you own the real property and want to sell or establish value for the asset, the rental rate is used to calculate the value of the property. A 7000 sq ft building that has a veterinary hospital rents space at $15/sq ft as researched in the surrounding markets. That equals a yearly rent of $105,000 (7000 × 15). An investor purchasing the property would like a return on the investment of a certain percent or capitalization rate. A starting capitalization rate for veterinary real estate can be 8%, although that number can vary greatly depending on the length of lease, guarantees, coverage of taxes and repairs, and many other factors. The value to the investor in this example is the yearly rent of $105,000 and divides by 0.08 (8%). That equals a value of $1,312,500 for the real property. If another investor finds the terms of the lease very favorable, they may offer a 7% capitalization rate, which gives a value for the real property of $1,500,000 (105,000/0.07).

The impact of having a proper lease and paying reasonable rent not only affects the profit of the veterinary practice on a monthly and yearly basis but also impacts on the value of the practice and the building, if also owned by you. Involving real estate professionals, lawyers, and accountants is paramount when establishing a lease between other parties or your own entity.

PRIVATE PRACTICE: VETERINARIAN CAREER PLANNING

When starting to work with birds in private practice, veterinarians are looking for a fulfilling career of caring for patients, relieving their suffering, and hopefully earning an acceptable income while doing so. This goes for associates and owners alike, even though they can have very different roles. Each veterinarian will have their own challenges, but there are some commonalities among the individuals in each group.

Associate Challenges

Job selection

Associate veterinarians have the responsibility to find the perfect fit for them. When wanting to work with birds, the options start to become limited. Not every small animal hospital wants to work with birds. Some may think they do until it starts happening, as the execution of medicine and surgery for birds can become quite challenging managerially. Some hospitals may not fully realize that until they start working with them. The questions discussed earlier in the chapter should be asked again. What percentage of birds do you want to see at your practice relative to other types of patients? What level of medicine and surgery do you want to perform? These are important questions that should be answered prior to the job search, and then apply to hospitals that meet your criteria. Invariably you will have many criteria like location, hours, pay scale, and many others.

It is best to have a place established in the broad and general avian community. These owners are committed to working with birds. When an inexperienced associate is asked to start the avian part of the practice, roadblocks present themselves. Owners may not persevere through those tough times and may abandon the idea of seeing bird patients altogether. Starting on your own in a practice may be the only option, but it is generally not the preferred way.

It is important to prioritize what is important to you and be sure to listen to yourself as you look for employment. Fortunately, if it is not a great fit, you can do something different. If birds are a priority, spend time in the interview asking about how you can develop your career doing just that.

Proper compensation

Typically, associates are paid in a few different ways. They can receive a salary that is based on the area of the country of the practice and experience of the associate. There are survey publications that have that information for veterinarians from the previous year(s) so all parties know a competitive salary range.

Production is another way to be paid, which includes everything generated by the associate, but considers any benefits received like paid time off, continuing education, any licensing, continuing education, and other agreed-upon items. An associate may be paid 18% of production, but benefits could increase that percentage to 25%. A calculation is completed at the end of the contract year to determine what monies are paid. In this scenario, there is a base salary throughout the year with 18% to 25% of production as the total compensation. Usually, the associate is owed money in this scenario, but if there is excessive time off, money could potentially be owed back to the practice.

ProSal is a scenario where the associate is offered a base salary with a percentage of production beyond the base. This is a positive for the associate, as they will never owe money to the practice. It also rewards hard work and good medicine and surgery. Remember that if we do what is right for the patient, money and profit will follow. Standard ProSal is calculated monthly so the second pay for the month is adjusted based on the previous month.[18] Several permutations of ProSal remuneration exist, so it is important to perform calculations ahead of signing a contract. A lawyer and an accountant can not only help estimate yearly pay but also use a veterinary salary resource guide to be sure the pay is fair.

Retirement

The goal is to be able to live comfortably during retirement. Because associates often will not make salaries similar to practice owners, they have to make sure to start early and contribute often to proper investments. Also, there can be large student loans that prohibit what seems like a meaningful contribution to retirement investments. There are numerous aspects to retirement planning that require a written plan. Retirement is not just about saving money. It is also about looking at investment sources of income to aid you during retirement. Eliminating debt, having a proper estate plan, and protecting your assets are also important and will help make retirement much more enjoyable. Choose a financial planner to assist you in your retirement planning process; this is not a process that should be managed yourself.

Owner Challenges

The day you decide to become an owner of a veterinary practice is both exhilarating and terrifying at the same time. The exhilaration comes from the fact that you are now in charge of making decisions and can practice medicine the way you see fit. The terrifying part comes into play when you realize that you did not learn any of the necessary skills that you need to become a successful owner in veterinary school. They did

not teach how to market and promote, manage finances, handle people, hire personnel, understand business and legal documents, or set up business systems so the organization can run efficiently. There is great joy in creating a business and having that business financially serve your household. Ownership also provides you the opportunity to accelerate your personal wealth building and become financially free much earlier than if you were to remain an associate. But you first must understand each of the roles you play to navigate the pitfalls of practice ownership.

The three roles

In every business, there are three roles that must be worn. These roles would be those of the owner, the executive, and the practitioner.[19] Any business owner must know how to calmly wear these three roles and ensure they are being worn in their organization. The success of the business is directly proportional to these three roles being played well by individuals within the organization.

The practitioner role. Most veterinary owners started off as practitioners in their practice. They went to school for an ungodly number of years to learn the skills necessary to treat and help patients. This is the most natural role you play in your business. The importance of this role cannot be overstated. If you are not a good practitioner, you will not be in business very long. The list of responsibilities is as follows:

- Delivers the care and medical expertise to the public
- Develops standardized protocols of how to deliver the company product
- Provides consistent results with patients
- Provides high-quality service to the patients
- Is highly educated in their field
- Strives to innovate and make their field better

If you plan on continuing to treat patients, you must calmly and confidently wear this role. In addition, you need to ensure the other associate doctors are acting as professional practitioners. While no two people treat the same, there needs to be consistency in how you treat the public from associate doctor to associate doctor. The protocols need to be standard. If an associate wants to treat a certain way that is not in alignment with your philosophy, they need to start their own practice. You should always be looking for ways to improve your technical skills and innovate. True professionals are constantly looking for methods to make things better.

The executive role. An executive could be said to be someone who has the responsibility to manage others to ensure they perform the work to produce the company's product or service. This is probably the least liked role for most practice owners. Most practice owners simply do not like managing others. The reason they do not like the executive role is they have had the least amount of training in how to perform this function. Being a good executive is not thoroughly taught in medical training. Even in residency and specialty training programs in avian practice, this function in an executive role is not required as part of the training and is therefore not commonly taught. Yet, the executive role and having competent executives in your business is the make or break of whether your organization will expand or contract. Among the responsibilities of the executive role are the following:

- Organizing and carrying out the intentions of the owner(s)
- Establishing policy and procedure for all staff
- Training all staff on a standard management system
- Being responsible for the gross revenue and profit
- Being responsible for marketing, promotion, and sales
- Being responsible for execution of strategic plan for expansion and production

While all these functions seem overwhelming to most solo practitioners and one-doctor practices (and it is), having competent and cooperative executives can take a large amount of pressure off the owner to "do everything" and allow for actual business growth to occur. The beautiful part of this is you do not have to create a "perfect" job description for every executive. If you hire the right people from the top down and give them just a little direction and purpose, they will create things you never even thought of. A lot of business owners are afraid to hire a full-time CEO or other executives for fear of eating into their profit margins. This is short-sighted and a myth. The most profitable and well-run practices one of the authors has seen operating with over a 25% profit margin all have done executive training and have hired executives to get others to get work done.

You will see a very large resistance wave come your way when you decide to really wear this executive role in your business, but if you can blast through it, you will see massive growth and increased time for you to pursue other life goals and ambitions.

The owner role. By far the most fun role you can wear is that of the owner. When you first had the idea and a goal for what you wanted your business to look like, you were playing the owner role. The owner's role is the visionary. This is the role where you get to create and dream. It is the role that many practice owners unfortunately neglect to wear properly because the practitioner and executive roles eat up all of your time and attention. But if you do not carve out time to do this role, you will find yourself stuck in second gear and will likely never achieve the financial wherewithal you dreamed about when you decided to start your business. Wearing the owner role correctly means you set up the business to serve the goals, purposes, and needs of the household, and creating a business that provides maximum value to your household. Value does not just mean money; it means time, wealth, relationships, friendships, acknowledgments, and achievement. A business must bring all of these "value" items to the household, and it is the owner's responsibility to do so. Among the responsibilities of owner role are the following:

- Establishing goals and purposes, mission, and vision
- Establishing standards and culture
- Establishing legal and regulatory compliance
- Creating maximum enterprise value in the business
- Acting as a professional investor
- Using household financial planning to meet the financial objectives of the household members.

When you are truly wearing your "owner" hat, you are focusing most of your time on expansion activities, ensuring the business compliance and regulatory activities are up-to-date and secure, and ensuring the business is providing the proper value to the household based upon the risk you took for putting the business in existence.

Of the three roles, focusing on your owner and executive roles will always provide for more compensation and expansion of revenue than focusing on your practitioner role. The reason most veterinary owners never get their practice

revenues to the $2- to $5-million-dollar revenue range is they focus too much on the practitioner role and never get out of treating on a day-to-day basis.

From a practicality standpoint, it is very difficult to play all three roles in your business. You will need to delegate one to two of these roles in your organization to others if you want to become "unstuck." You must always wear the "owner hat," and you cannot give that responsibility away if you own the business. Therefore, the decision comes down to whether you want to be an owner-executive or an owner-practitioner. If you want to be an owner-executive, then you must ensure the associates are excellent practitioners and deliver the products of the organization very well. If you choose to be an owner-practitioner, you must ensure you build an executive team that can motivate, plan, and execute the strategic plan of the organization to expand while you focus on treating full time. Pick two of the roles for best success.

Your Practice Is an Investment, Not a Job

For most veterinary owners, their practice is the main income source for their household. While you never want to rely on one income source forever, it is an economic reality that the business needs to provide a tremendous amount of value to the household. This comes in the form of business profits, rental income, and eventually the proceeds from the practice sale. To get maximum value from the business, you need to treat it as an investment and not just a place to work.[20]

Warren Buffet is a master investor. He has a mindset that if he is going to invest in a company, it needs to produce enough value to exceed the risk he took in utilizing his own money. As a practice owner, you must maintain a similar point of view. Many owners have poured enormous amounts of money to either acquire their practices or start them from scratch. Either way, there was no guarantee of success when starting down this road. Unlike investing in the stock market, it is possible to control the returns you get from your practice. Most practice owners have the belief that they can expect only 10% to 12% profits from their veterinary practice and some think 15% to 20% is reasonable. Elite practice owners demand a 25% to 30% profit margin from their business. This is done through a combination of excellent marketing and promotion, strong sales, excellent delivery, and strict money management where the profits are considered a necessary expense. Elite practice owners understand how important their business profits are to the overall value of their business. To illustrate, here is an example of a poorly run, well-run, and extremely well-run veterinary practice (Figure 1.15).

As you can see, the difference in cash flow and overall investment value from a poorly run and extremely well-run practice is staggering. These elite owners do not have some secret knowledge that was locked in a vault. They just understood they needed to invest their money in certain areas in order to ensure the business could achieve maximum value for themselves, their family, and their community. These areas include the following:

1. Practice management and business consulting
   Investing in increasing your ability to run a business is probably the best investment you can make. Having a proven business consultant who understands business systems and can help prevent you from making bad business decisions is worth potentially millions of dollars.

Valuation scenarios of $1,000,000 annual revenue practice

Not well managed	Well managed	Very well managed
10% profit margin = $100,000	15% profit margin = $150,000	20% profit margin = $200,000
@2–3x multiple **$200k–$300k value**	@2–3x multiple **$300k–$450k value**	@3–4x multiple **$600k–$800k value**

Difference of not well managed and very well managed: Loss of $400–$500k

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FIGURE 1.15 Difference in value of not well-managed and very well managed practice. (Eric Miller 2024, © 2024 Sage Assets LLC. Published with permission.)

2. Staff training
   No one can have a sustainable business by doing it alone. You need to invest in your people. That means spending money on training your staff so they can do their jobs better. This is the *only* way you will truly be able to step away from your practice and have it run profitably without you having to monitor it full time. If you want to be able to take a 2-month vacation and come back to a practice that is growing, train your staff.
3. Real estate
   Patients and employees want a safe and aesthetic place to come to every day. It is imperative that you invest in your facility to make it a warm and inviting place. For most owners, it is highly advisable to own your practice building in an established or growing area. By owning the building, you can get certain tax benefits and can continue to collect the rent even after you have sold the practice to another veterinarian or corporate buyer.
4. Tax, financial, and legal management
   The most elite owners have a coordinated team of financial professionals who help them navigate the financial pitfalls one can encounter. This means having a top-notch accountant and tax strategist who minimize your taxes, an industry-specific financial planner who shows you how to channel the business profits to your household to increase your personal net worth, and an attorney who helps with your legal affairs and keeps your business compliant and your assets protected.

The most elite owners are willing to spend hundreds of thousands of dollars on the above because they understand they are likely to get a return of 10 times of their investment. To build a profitable, sustainable, and transferable practice, it is vital that you invest heavily in these four areas.

Exit and Retirement

Every practice owner must confront the reality that there will come a time when they will need to exit their business. Your exit can either be a positive experience or it can be a stressful and sad event. The choice is mostly yours. The key factors are how well you plan your exit and diligently work toward the event no matter how many years in the future it might be. You

do not start planning your exit 2 months before it happens; you start now, even if you are just starting ownership. The first step is exploring the seven different ways in which you can exit your practice and figuring out the strategy that makes the most sense for you.[21]

1. Die with your boots on
   As it sounds, this is where you simply keep working up until the day you die. Some practice owners simply cannot walk away. If this is your exit of choice, just be sure to ease the burden of disposing the practice from your heirs by having your estate plan in place, which provides a detailed and step-by-step process for them to follow.
2. Close the doors
   Utilizing this strategy, you simply close the practice down. This is the saddest method and least valuable manner of exiting your practice because it provides no value for your household. This method is only doable if you have accumulated enough wealth outside of the business that can make up for the loss of income from the practice.
3. Sell to a competitor
   It is unlikely that your practice is the only practice in town. If this is the case, you may be able to sell your practice to a competitor. You can get a relatively fair market value price for your practice. The downside is that you are likely to be out of a job and there is no future value of the business available.
4. Associate or management buy-in or buy-out
   With this method, you are choosing to sell to your upper management or a successor associate. In most cases, the people buying your practice will not have all the resources necessary to buy the practice outright. You will most likely need to use the business as collateral, and they will pay you over a period of time through the business profits. The upside is that you can minimize your tax liability and still maintain control of the business until the purchase is complete. The downside is that if your successors are not well trained on how to run a business, you could be forced to reassume full control at a time where you were looking to reduce hours and retire.
5. Private equity or corporate groups
   The veterinary industry is a very lucrative and profitable industry. Good businesses in good locations with multiple doctors and high-profit margins will always be the target of corporate and private equity groups. These groups will pay relatively high multiples of EBITDA (earnings before interest, taxes, depreciation, and amortization) for well-run practices. They will generally require the owner to stay on for some time to make sure the transition runs smoothly. In addition, they will pay a lump sum for the practice, which reduces the risk to the seller. The seller may also get an option to leave some of their value in the corporation and take advantage of future growth. These groups are sometimes painted in a bad light for their "greed" and "profit-first" approach. This is not always the case. There are numerous examples of corporate groups who enhance business and make the environment better for their employees and patients.
6. Employee stock ownership plan
   This is a highly specialized type of strategy where a practice owner will set a legally qualified retirement plan that will allow their employees to purchase the company stock and eventually take over. The owner will still maintain strategic control of the business and keep their salary and perks. The downside is the expense to administer this type of approach. You would need to have over $1,000,000 of net income (not gross revenue) to justify the expense of setting up this type of plan.
7. Gifting strategies
   There may be circumstances where an owner simply wants to gift company stock to their family or employees. This is generally done where the owner has a high level of financial resources and is not dependent on the sale proceeds to fund retirement. Gifting is done as an effort to minimize certain types of taxes and to ensure the practice legacy continues.

Whatever exit strategy you choose, it is imperative that you build your practice to transition for maximum value. That value should be created for you and your family, your staff, the successor owner, the community, and your patients. This is not a zero-sum game, meaning everyone should win in this transaction. Those owners who only look out for themselves come to regret their choices later on in life. Make it a win-win proposal for everyone and you will be rewarded!

Lastly, it is possible for you to retire with dignity and enough resources, so you never have to worry about money again. For this to occur, you cannot assume the sale of the business will fund your retirement 100%. The single most important action you can take to ensure your business sale is not the only funding of your retirement, is to set up an automatic and systematic payment to your household of 10% of the business revenue. This means, if you are doing $1,000,000 a year in gross revenue, that $100,000 is coming right off the top and being allocated into a wealth storage account for the benefit of the household. This is simply an account that will be used to store money that is designed to create other income streams for the household and act as an emergency fund during a financial crisis. A competent financial advisor will help you prudently invest that money to ensure it is not wasted or subject to huge losses. This one action is the most powerful financial action you can take to provide financial security for your family. Do not underestimate the power of putting your profit first. It will allow you to create financial freedom.

SUMMARY

The practice of avian medicine and surgery is present in every corner of the globe. The medicine and the management that allows delivery of services are not necessarily handled differently by comparison in different geographic areas. While there are nuances in many areas of management between states, provinces, and countries, the basic business principles still apply. The topics discussed are not difficult concepts, and understanding them seems inherent. Developing an overall strategy, communicating that philosophy, and implementing the management tactics are the most challenging parts of having a wonderfully rewarding and profitable career in avian medicine. Having a written plan along each step of the way and continually reviewing that plan with your team and yourself will allow you to achieve your professional and financial goals whether you are an owner or an associate veterinarian.

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CHAPTER 2

Common Imaging Modalities: Selecting the Right Modality and Getting the Most Out of Your Imaging

Sophie E. Dennison

Visit eBooks.Health.Elsevier.com for a complete set of references.

GENERAL CONCEPTS OF DIGITAL IMAGING

In the last decade, there has been significant progression in veterinary medical digital imaging, with increased availability and diminished cost to the practitioner for almost all digital imaging modalities. This availability has been accompanied by more reasonable costs associated with digital media storage. Digital images are part of the medical record, and all images must be saved according to local veterinary medical board or equivalent governing body requirements. Hard film records of ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI) images can be made and stored in the same way as screen-film radiographs. Diagnostic images acquired as digital media should be saved as DICOM (Digital Imaging and COmmunications in Medicine) files, and studies must be carefully labeled with unique patient identifiers (name and/or ID) prior to starting the study as well as ensuring that the date and time of acquisition are correct. Most digital imaging systems do not (and should not) permit alteration of patient identifiers after the study has been started due to potential medicolegal ramifications.[1] Digital image resolution and DICOM guidelines for digital imaging have been published by the American College of Veterinary Radiology (ACVR) and are available at www.acvr.org. DICOMs are large data files that are the accepted standard for medical imaging storage and interpretation.[1] Interpretations from compressed image files such as jpeg are discouraged as some data are irreversibly lost. This can lead to errors of interpretation particularly in very small patients where compression may affect resolution.[1] The use of cataloged image storage on a PACS (Picture Archiving and Communication System) is recommended to ensure that studies can be quickly identified.[2] Many PACS are now available as cloud systems that remove the cost of hardware and need for additional backup systems, as well as increasing accessibility for authorized users without exposing internal IT networks. Data corruption can occur at many levels and quality assurance is a necessity to ensure that studies are of diagnostic quality at the time of imaging, that the entire study is sent to the PACS, and that an adequate PACS system is in place to protect data. DICOM imaging software is used to view DICOM images and both open-source and license-based software are available for use on Mac and PC.[3] Examples of popular open-source DICOM software for Mac include Horos (www.horosproject.com) and for PCs Radiant Viewer (www.radiantviewer.com) and MicroDicom (www.microdicom.com). All digital imaging, regardless of the modality used, should be evaluated on a high-resolution screen in an environment optimized for image evaluation. Ideally, a dedicated high-resolution workstation in an area that can be darkened/shaded and that is quiet and away from distraction is suggested. In many busy hospital situations this can be challenging to achieve, but bright background lighting, noise, and distraction can contribute to misinterpretation.

Diagnostic imaging will always be limited if patient positioning and modality technique are not optimized for every study. Standardized patient positioning and technique reduce frustration, help remove iatrogenic artifacts, and improve the accuracy of the interpretation, as studies from the same patient or another patient being used for comparison can be compared side by side.

INTRODUCTION TO RADIOGRAPHY

Diagnostic radiography has been available since the early 20th century following the discovery of x-rays in the late 19th century. Radiographs are the images produced after an image detector is exposed to an x-ray beam that has been passed through a patient. X-ray photon attenuation varies by tissue resulting in the five radiographic opacities: metal, bone, soft tissue/fluid, fat, and gas. Screen-film has been used for the

production of radiographs from the discovery of x-rays to beyond the advent of digital radiography. Digital radiography became available for medical imaging in 1980 although screen-film radiography prevailed as the main form of radiography used in veterinary medicine until the last decade and screen-film imaging remains in veterinary use today in some practices.

Screen-Film Radiography

Screen-film radiography refers to the traditional emulsion film system used to capture, store, and present radiographic images after exposure to an x-ray beam. Double-sided or single-sided emulsion film is placed in a cassette that contains intensifying screen(s) that reduce the amount of radiation needed for a diagnostic image. Single emulsion film is preferred for very small patients as it has higher spatial resolution and will produce sharper images than double emulsion film where the two superimposed emulsions on either side of a transparent base can cause very slight blurring of margins. When x-rays, gamma rays, heat, or light interact with silver bromide in the film, it results in the release of bromide into the emulsion gel and deposits of ionized silver known as "sensitivity specks" form the latent image. The film is then developed and fixed producing a visible image using manual, semi-automatic, or automatic developer systems. Developing is the conversion of the silver ions in the emulsion to black metallic silver that is visible to the eye. Fixing is the removal of unexposed silver halide crystals from the film making the image permanent and hardening of the emulsion to prevent damage.[4]

Nonexposed radiographic film must be protected from light and heat to prevent accidental activation of sensitivity specks that result in fog on the final radiograph. Loaded cassettes and film storage boxes should be opened carefully in red light, temperature-controlled dark rooms and film handled gently. When replacing film in the cassette, ensure that dirt, dust, hair, or other debris are not trapped inside the cassette as these will also create artifacts on the film.[4]

Digital Radiography

Two main forms of digital radiography technologies exist: computed radiography (CR) and direct radiography (DR). The image detector in digital radiography is used to capture the latent image which is then processed and stored and presented via digital medium. Technique charts used for digital radiography are extremely simplified compared to screen-film, making the systems very appealing and more user-friendly. Digital radiography images have a wide dynamic range that allows manipulation of the image post-processing and greatly reduces the number of retakes needed due to technique.

The theory behind CR is like traditional screen-film radiography. In CR, the image detector is an image plate contained within a cassette that contains photostimulable crystals similar to the intensifying screen in traditional cassettes. When exposed to x-rays, the image plate temporarily absorbs and stores the x-ray photon energy as a latent image that is read out pixel by pixel via a laser beam in a scanner. The laser beam releases the stored energy as light that is detected by photodiodes within the scanner and converted into a digital image. The time from image detector exposure to image presentation takes at least 45 seconds and is slightly faster than traditional screen-film using a fully automated developer system. The image plate should be processed quickly after exposure to prevent image degradation, and image plates should be passed through the scanner between exposures to remove all remaining latent image, even if a long period of time has passed. Damage to CR cassettes and image plates can occur during handling, and dirt may enter the cassettes leading to artifacts on the final image. CR image plates and screens are more expensive to replace than screen-film but less expensive than DR flat panel detectors.[5,6]

DR is the alternative form of digital radiography to CR. X-ray photons that reach the image detector (usually a flat panel) are converted into electrical charges which are then converted into an image via direct or indirect technology. Direct technology is based on a photoconductor, typically selenium, converting x-ray photons into electrical charges stored in a thin-film transistor array and processed to form the image. Indirect technology systems utilize a light sensitive system that has a layered construction comprising a scintillator layer (usually gadolinium), amorphous silicon photodiode layer, and thin-film transistor array. Indirect technology has better optical properties and better quantum efficiency than direct technology and is more widely available. The production of an image using a DR system is essentially real time with only a few seconds lag between image detector exposure and presentation of a digital image. Contrary to screen-film systems and CR image detectors, DR flat panel detectors are quite fragile and expensive to replace.[5,6]

Clinical images acquired in medium-sized birds via any digital radiography technology are at least equal to and often supersede perceived conventional screen-film image quality, despite medical physics studies showing that spatial resolution of CR is slightly less than screen-film.[5,7] This perception of improved image quality is due to the wide dynamic range that means all tissues can be optimally displayed on the same image after adjustment of window width (WW) and window level (WL) (brightness and contrast). This and other manipulations of the image such as enlargement or inversion result in greater user satisfaction.[3] Over- and underexposure do still occur but have a very different appearance on digital imaging compared to screen-film techniques (see section on Selected Radiographic Artifacts). Digital radiography has removed some of the technical skills required to produce diagnostic images and with that operators may be less conscientious about technique selection and less aware of the radiation risk to themselves. Continued training in and use of the ALARA principle (as low as reasonably achievable) for radiographic technique as well as enforcement of local requirements for personal radiation safety is encouraged.

Patient Positioning for Radiographic Imaging

Patient rotation can affect assessment of some coelomic organs, and optimal and repeatable positioning permits the most accurate assessment and comparison, especially in birds that undergo serial studies.[8] The use of radiolucent restraints and positioning aids such as tape, rope, or foam wedges/troughs, can be used to prevent motion and optimize positioning, and chemical restraint (either sedation or anesthesia) can be utilized to minimize stress. On the lateral view projections, the coracoids and femoral heads should be very close or superimposed on each other (Figure 2.1), whereas on the dorsoventral (DV)/ventrodorsal (VD) view, the spine should be superimposed on the sternum/keel.[8] Studies have shown that an erect (standing) DV view may be adequate for some anatomic evaluations in the critically ill bird where sedation and anesthesia or restraint to permit a VD projection are contraindicated.[9] If acquiring standing DV views, clinicians should be aware that measurements of the heart will be larger

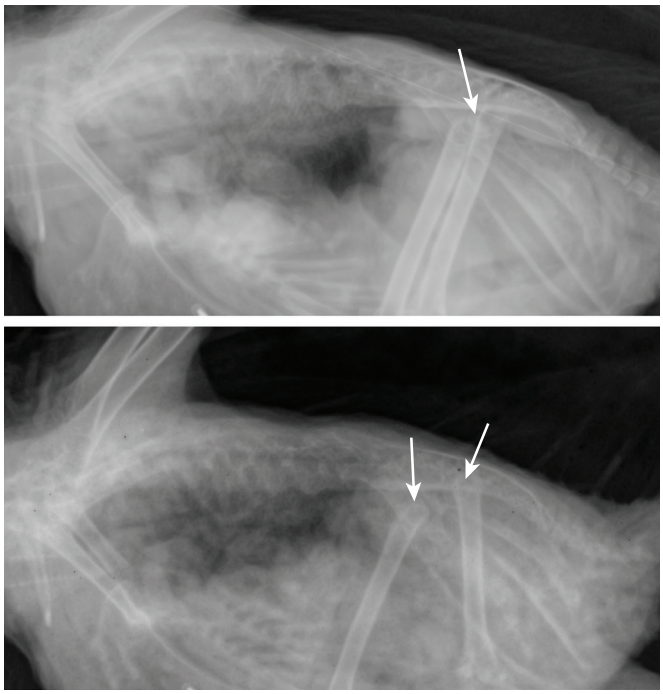


FIGURE 2.1 Lateral radiographic projections acquired in lateral recumbency showing optimized positioning with near superimposition of the coxofemoral joints (*arrow,* top image) and separation of the coxofemoral joints (*arrows,* bottom image) when rotation is present.

on the DV projection due to magnification effects, and evaluation of the respiratory tract is more limited than on the VD view. The erect DV is considered adequate for evaluation of the heart and upper gastrointestinal (GI) tract.[9]

Historically, radiographic units were fixed in position for vertical beam image acquisition, where the x-ray tube sits over a table pointing to the ground and the image detector is placed either on or under the table perpendicular to the primary beam. The patient is positioned on the table and then repositioned for the different projections. However, more recently, many systems have a movable x-ray tube and a movable or removable image detector that permits vertical and horizontal beam image acquisition (Figure 2.2). Horizontal beam lateral projections are beneficial for avian patients as they remove iatrogenic organ displacement (Figure 2.3) that could result in misinterpretation.[10] Horizontal beam radiographs are acquired by positioning the patient in sternal recumbency or standing with the x-ray tube positioned on one side of the patient and the image detector on the other, parallel to the table (Figure 2.2) using radiolucent tubes or containers. Interpreters should be aware of variations in appearance of organs in images acquired in standing compared to ventral/sternal recumbency (Figure 2.4).

Peer-reviewed texts are available that demonstrate normal avian radiographic anatomy in some species including the cockatiel (*Nymphicus hollandicus*),[11] Amazon parrot (*Amazona* sp.),[12] galliform species,[13] anserine species,[14] diurnal raptors,[15] and the barn owl (*Tyto alba*).[16]

Advantages of Radiography

Radiographic equipment is available in most veterinary hospitals making it extremely accessible. The vast majority of companion bird species will fit within the margins of the radiographic cassette/image detector, thus requiring only two exposures and making radiography a very rapid and inexpensive test. In the case of severe respiratory disease, fractures/luxations, some coelomic effusive disease processes, or GI tract ileus, a diagnosis can often be made immediately.



FIGURE 2.2 Photograph demonstrating use of a horizontal beam technique to acquire orthogonal views in a standing penguin. The penguin is restrained inside a radiolucent cylinder. The same technique is used for restraint for C-arm fluoroscopy or CBCT-incorporated fluoroscopy. *CBCT,* Cone beam computed tomography.

Disadvantages of Radiography

Patient size can be challenging due to the very small size of many avian species requiring examination. Superimposition of structures combined with relatively low inherent patient contrast especially in the caudal coelom, can make radiographic interpretation of avian patients challenging. Digital radiography and the ability to enlarge images have helped but do not escape the limitations of superimposition that can obscure pathology. For very large birds, multiple overlapping exposures may be needed to cover the entire body for each projection. Anesthesia or sedation may be necessary to achieve optimized diagnostic quality studies and minimize patient stress.

Direct Clinical Applications of Radiography

- Evaluation of the axial and appendicular skeleton
- Evaluation of respiratory tract
- Evaluation of the coelomic cavities for evidence of fluid, egg binding, or organomegaly
- Evaluation for radiopaque foreign material (gunshot/trauma, ingested)
- Contrast studies (anatomical and functional studies)[17]

Selected Radiographic Artifacts

Several texts are available that explain screen-film radiography artifacts[18] and digital radiography artifacts[19,20] in detail. The most observed artifacts in digital radiography are overexposure, underexposure, incorrect placement of the imaging detector/digital plate, and lines or stripes across the image.

Overexposure may be due to excessively high kVp (peak kV), mAs (the combination of mA and time of exposure), or both and occurs when x-ray photons saturate the image

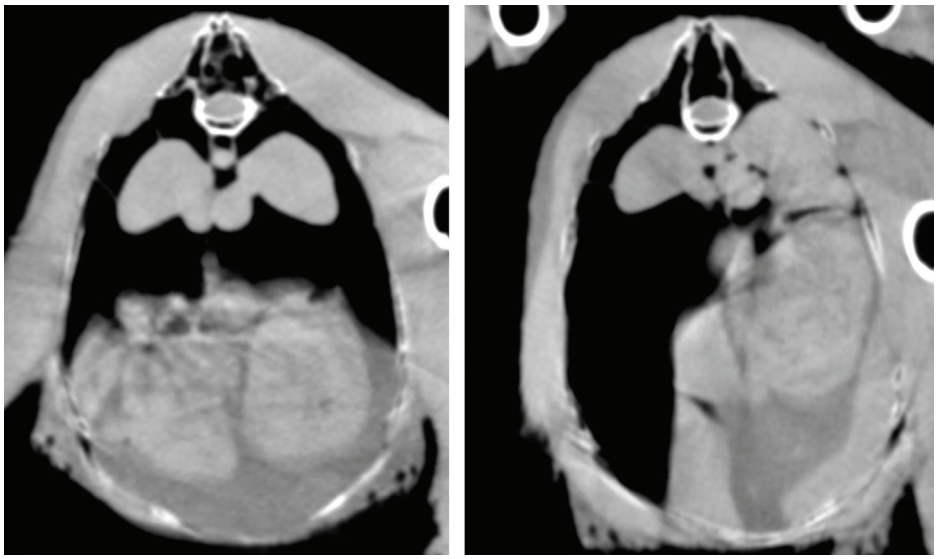


FIGURE 2.3 CT images demonstrating the varying position of internal organs when positioned in sternal (left image) and left lateral (right image). *CT*, Computed tomography.

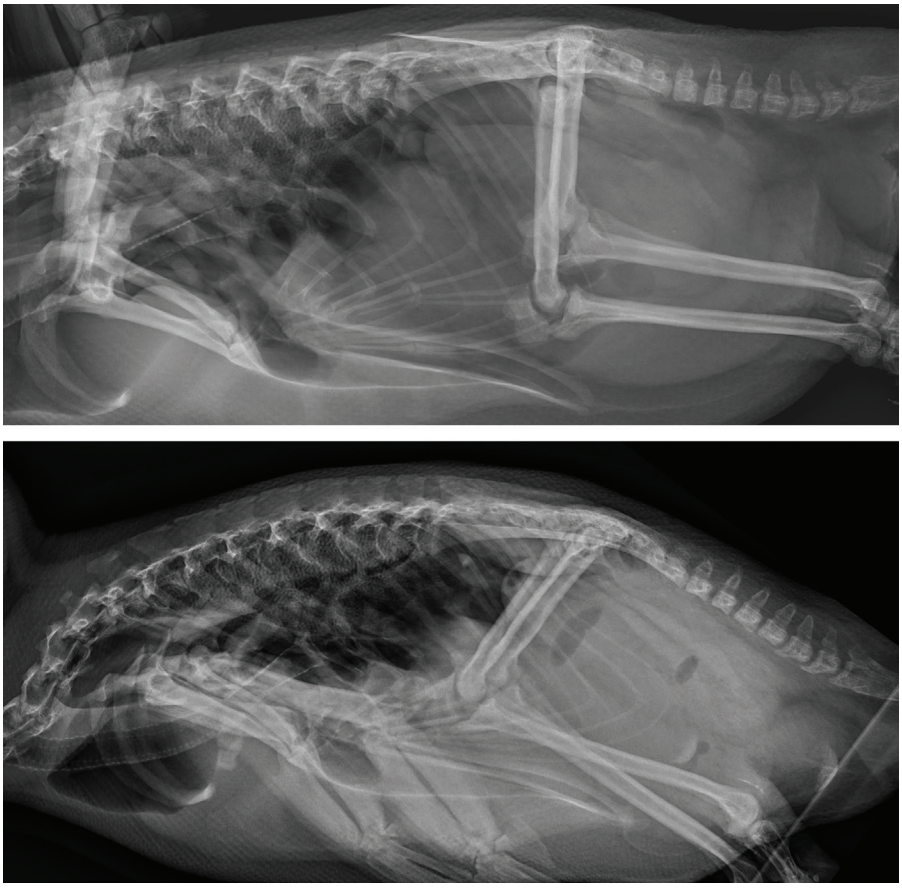


FIGURE 2.4 Lateral projections of a penguin. The top image was acquired in lateral recumbency and gives the impression of increased soft tissues in the caudal coelomic cavities. The bottom image is the same penguin with the lateral view acquired standing via horizontal beam.

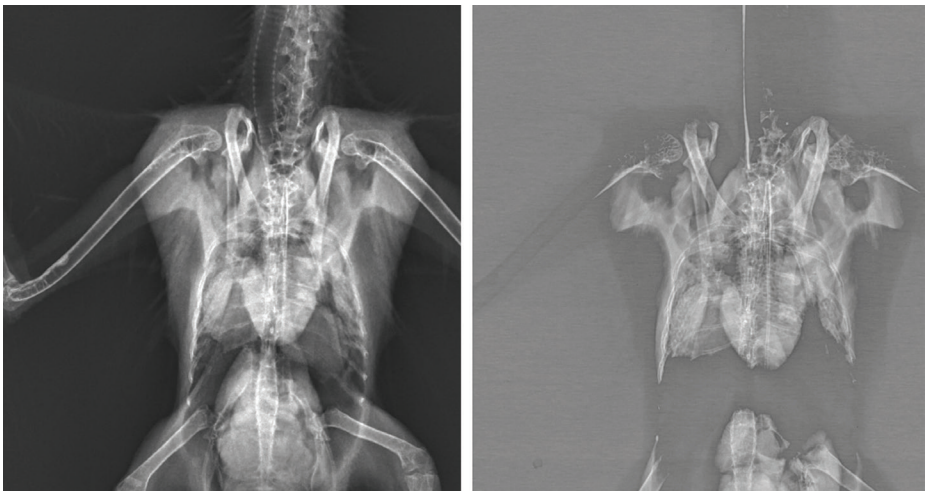


FIGURE 2.5 Optimized radiographic technique is shown on the left. The right image demonstrates burn out of soft tissues, and in this case also bone, due to higher than necessary technique and saturation of the image detectors. Regions with burn out cannot be adjusted and evaluated, as the data do not exist.

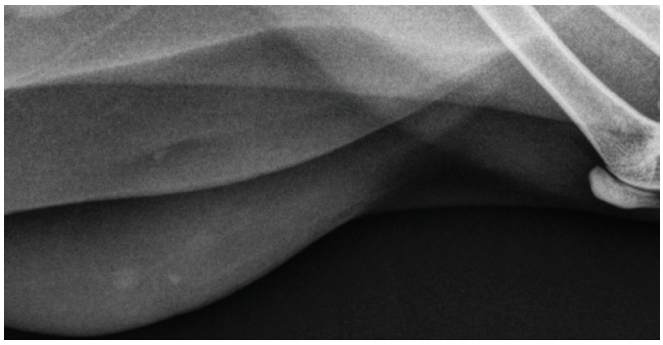


FIGURE 2.6 Underexposure is recognized as a grainy appearance extending across the soft tissues, demonstrated here in a domestic cat.

detector, resulting in areas with little or no detail (Figure 2.5). Overexposure will affect the entire image but is most evident by its effect on anatomy and may not appear uniform. This artifact is corrected by reducing the technique (kVp and/or mAs) to a level appropriate for the patient.

Underexposure or noise occurs when inadequate technique is used to overcome the electronic noise present within the system. This can result in diminished image quality with the overall appearance of speckling or graininess (Figure 2.6) that can obscure subtle pathology. Underexposure will affect the entire image and can be identified in regions with and without. This artifact is corrected by increasing the technique.

Incorrect image detector placement (Figure 2.7) can occur when image detectors are accidentally flipped so that the back of the plate faces toward the x-ray generator. In this case, the x-ray photons pass through the electronics of the system before hitting the scintillator and contribute to the image. This artifact is rectified by orientating the detector correctly toward the x-ray tube.

CR readout errors occur most frequently when dirt or dust is present on the light path assembly within the scanner (Figure 2.8) creating a perpendicular line through the image. This may resolve itself or require cleaning.

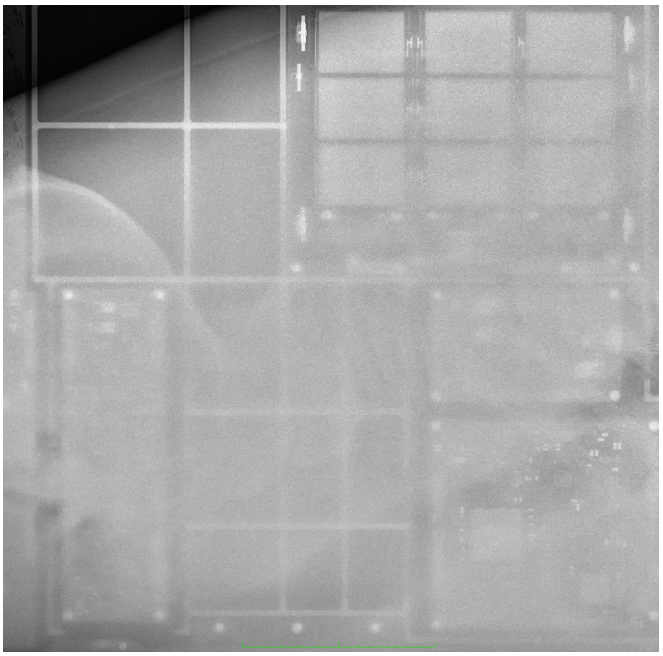


FIGURE 2.7 The plate was placed upside down with the backside facing the x-ray tube resulting in the electronics creating part of the image as the beam passed through them before reaching the detectors.

INTRODUCTION TO FLUOROSCOPY

Fluoroscopy development progressed alongside radiography and came into clinical use after the development of image intensifiers in 1937, with greater use once images could be viewed via television in 1948. The first C-arm fluoroscopic machine was developed by Philips in 1955. Digital fluoroscopy was first introduced in the late 1970s and became more widely available in 1980 enabling the use of masking or subtraction imaging, tools which greatly improved dynamic angiogram, pyelogram, and GI tract contrast studies.[21] Digital fluoroscopy

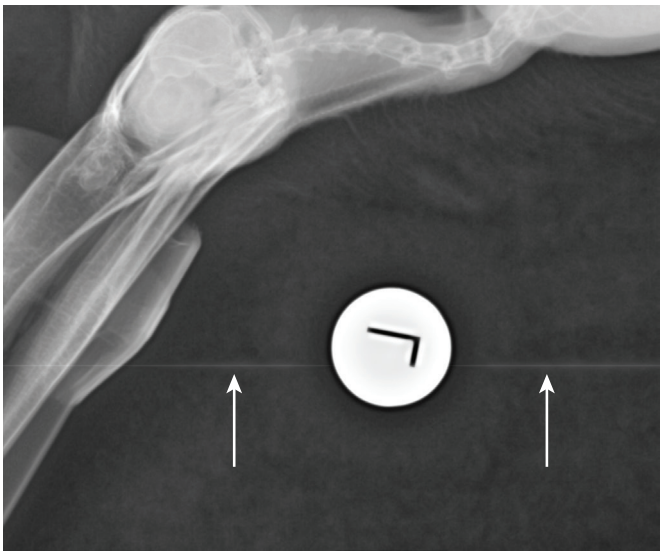


FIGURE 2.8 The white streak seen on this CR image and highlighted by the two arrows is due to dirt within the scanner lying in the light path. *CR,* Computed radiography.

has been recently incorporated into some high-definition volume imaging computed tomography units (HDVI CT) available on the veterinary market, resulting in easier addition of this imaging modality for veterinarians to utilize.

Positioning for Fluoroscopy

Small birds undergoing upper GI contrast studies are usually restricted in a radiolucent box rather than specifically positioned and restrained, as for standing radiographs.[22,23] C-arm fluoroscopic units are then moved around the box to acquire multiple projections, similar to standing radiography (Figure 2.2). Birds being imaged with fluoroscopy using CT units with this capacity can similarly be positioned in small radiolucent boxes on the CT gantry, and the imaging unit is similarly moved around the patient.

Advantages of Fluoroscopy

The main use of fluoroscopy in avian medicine is for evaluation of GI tract motility where fluoroscopy permits real-time dynamic evaluation of the GI tract and the quality, character, and direction of GI tract contractions and contents can be determined. Physical restraint, conscious sedation or anesthesia are often not needed for many studies. Fluoroscopic imaging removes any negative effects of dorsal or lateral recumbency on GI motility and respiration by not requiring any form of patient handling or stress-evoking procedures to optimize patient positioning. Anesthesia is often required for angiography studies. Subtraction techniques may be helpful in some studies.

Disadvantages of Fluoroscopy

Fluoroscopic imaging is similar to radiography in that it is a two-dimensional (2D) representation of three-dimensional (3D) structures, and the problems associated with superimposition of structures therefore remain present, although as for radiography, acquisition of multiple projections is beneficial for interpretation to help counteract the effects of superimposition. Fluoroscopic equipment has lower inherent spatial resolution than can be achieved with radiography or CT, which may limit its use in very small patients, and patients are exposed to relatively large radiation doses. Patient movement may be problematic. GI tract contrast studies may require an empty GI tract before the study is started to eliminate filling defects from food that could mimic pathology and because food in the GI tract will affect the transit time of contrast medium.

Direct Clinical Applications of Fluoroscopy

- Upper GI contrast studies, particularly evaluation of motility[22]
- Intraoperative evaluation of implant placement for fracture repair
- Intravenous (IV) pyelogram
- Angiography
- Quick assessment of patients for the presence of metallic foreign materials without necessitating restraint

INTRODUCTION TO CONTRAST STUDIES

Contrast studies have been used in avian medicine to delineate the GI tract and cloaca and for imaging of the kidneys and ureters. Images can be captured using radiography, fluoroscopy, or CT following administration of contrast medium. In addition to observation of anatomical abnormalities and determining GI transit times, fluoroscopic evaluation has the benefit of dynamic observation of GI contractility and motility following oral administration of contrast medium, and of vasculature anatomy and subjective renal clearance/ureteral opacification following IV or intraosseous administration of contrast medium. CT has the benefit of acquiring images without superimposition of structures and requires a lower concentration of positive contrast medium, but dynamic real-time observation is more limited. Texts are available that describe the details of acquiring a diagnostic contrast study as well as providing time ranges for normal GI tract transit of both barium and iohexol.[17,23,24]

Upper Gastrointestinal Tract Contrast Studies

Upper GI contrast studies typically involve monitoring of contrast traveling from crop to cloaca and can be used to help determine if a mass identified within the intestinal peritoneal cavity is GI in origin, to determine if mechanical obstruction of the GI tract is present, to determine if proventriculus distension is present, and to evaluate GI tract motility times. Studies are performed using positive contrast medium and both barium sulfate and iohexol have been used. Barium dosage of 20 to 30 mL/kg of 25% to 30% weight to volume barium suspension has been described as administered via crop gavage. The volumes administered for some GI fluoroscopic contrast studies may be less. The GI tract should be allowed to empty prior to the study by withholding food for approximately 4 hours, although this may not be necessary in birds who are actively regurgitating, as food present in the GI tract may cause artifacts of filling defects and can affect transit times for the contrast medium.

Average normal GI transit for barium from crop to cloaca is 3 hours. Iohexol 240 mgI/mL diluted 1:1 with water and a total final dosage of 20 to 30 mL/kg has also been used successfully. Iohexol has a more rapid GI transit time compared to barium of 1 hour from crop to cloaca. Iohexol is preferable if GI perforation is a concern or if there is a high likelihood

of proceeding to surgery or endoscopy following the study. Iohexol is hypertonic and will move fluid into the intestinal tract and gradually dilute through the duration of the study, and patients should be appropriately hydrated prior to use. Anecdotally, injectable anesthetic and sedative protocols may cause delayed gastric emptying, while isoflurane has not been noted to significantly affect transit times. Detailed texts are available describing the techniques and timing of serial radiographic and fluoroscopic image acquisition.[17,23,24] If CT is to be used for image capture, iodinated contrast medium is suggested and should be diluted to 25% to 50% of the doses suggested here to help prevent detrimental beam hardening artifact. Beam hardening artifacts on CT are unavoidable with barium suspension even if diluted.

Cloacagrams

Cloacagrams are useful to evaluate for cloacal masses or ulceration/mural defects associated with cloacitis. These studies may be performed using positive contrast (barium or iohexol) or negative contrast (room air or CO_2) administered retrograde directly into the cloaca or by extending out a positive contrast upper GI study or IV pyelogram until the cloaca is filled. If a retrograde cloacagram is selected, only sufficient contrast to fill the cloaca is needed as excessive pressure may result in retrograde movement of contrast and cloacal contents.

Intravenous Pyelogram

Intravenous pyelograms (IVPs) can be performed via IV injection of contrast at the same 600 to 800 mgI/kg dose rate as used for CT. If the medial metatarsal vein is used, the ipsilateral kidney and ureter will contrast enhance preferentially, rapidly, and with greater opacity via the renal portal system, which can be very useful if unilateral disease is suspected. If bilateral disease is suspected, an ulnar vein injection is suggested so the renal and ureteral opacities can be compared to each other as part of the interpretation. The same contrast medium dose is suggested for radiographic, fluoroscopic, or CT capture of the study in this case.

Angiography

Angiography can be used to identify vascular and perfusion abnormalities. As with contrast CT, IVP, and some fluoroscopic imaging procedures, contrast administration methodology is similar. Anesthesia is typically required. A dose of 2 mL/kg of iohexol (240 mg/mL) (Omnipaque, GE healthcare Inc., Princeton, NJ) is administered through an IV catheter at a rate of 1 to 2 mL/kg per second during video acquisition at a rate of 30 frames per second for best resolution.[25]

Sinusography

Sinusography has been used to identify structural abnormalities in the nasal passageways, choana, and infraorbital sinus.[26] With the bird anesthetized and intubated, choanal atresia was confirmed by radiographic imaging in an umbrella cockatoo and grey parrot by administering 0.2 to 0.5 mL of iohexol 300 (Omnipaque, Winthrop, New York).

INTRODUCTION TO ULTRASOUND

Ultrasound was first discovered in 1880 when a piezoelectric effect was identified in certain crystals. Ultrasound in the medical field was first developed for therapeutic effects in the late 1920s and diagnostic use began in 1942. The first B-mode scanner was developed in 1965 and Doppler became available in 1976.

Ultrasound uses the transmission and reflection of sound in the range of 1 to 50 MHz to create images. The interaction of the sound beam with soft tissues is complex, and reflection, scatter, and absorption of sound all contribute to the final image. Higher sound frequencies undergo greater absorption in tissues than lower frequencies and have higher axial spatial resolution. Thus, higher frequencies are used for evaluating tissues closer to the transducer and lower frequencies used for tissues at greater depth, further from the transducer. Most cart-based or portable ultrasound system transducers have a broad range of frequencies and can be used to varying depths. The internal array of piezoelectric transducers dictates the use. Linear array transducers are typically used for B-mode (brightness) imaging, which is the typical grayscale mode used for anatomical/structural evaluation of organs, and phased array for M-mode (motion) imaging, which is the mode used during echocardiography. Transducers can have varying footprints/shapes and sizes. Microconvex transducers that have a small, curved footprint are most amenable to avian patients and frequently have a frequency range of approximately 3 to 10 MHz (Figure 2.9). Some ultrasound systems have software that facilitates Doppler studies. These can be used to estimate blood flow velocities, confirm that a structure is vascular or intravascular, and determine the direction of blood flow within a vessel, and are most useful for but not exclusive to echocardiography.[27]

Recently, new hand-held ultrasound technologies have become available in addition to cart-based and portable ultrasound machines. Hand-held systems use USB-based transducers that contain either piezoelectric crystals or newer chip-based technology and attach to a smart phone or other hand-held smart device. Images are displayed and stored via brand-specific software downloaded as an app. Many of these hand-held point-of-care devices have been developed and are FDA-approved for use in the human medical field and are gradually filtering into the veterinary market. Chip-based ultrasound technology permits a much wider range of frequencies and the ability to select B-mode and M-mode applications in a single transducer. Anecdotally, images from hand-held devices are predictably perceived as inferior to cart-based or portable machines; however, peer-reviewed published clinical comparisons in diagnosis of clinical disease are not yet available.[28]

When performing an ultrasound study, the depth of the field should be adjusted to optimize each organ or area as is evaluated. Focal points define the level at which axial spatial resolution is most accurate. The optimal focal point position varies slightly by ultrasound system but as a rule of thumb should be positioned just below or at the level of the deep margin of the structure/region of interest. The depth of tissue displayed on the screen should also be optimized so that the patient's anatomy is seen across the screen rather than all in the very near field. For very small patients, the use of a standoff may be useful or necessary to increase the distance between the organ(s) of interest and the transducer so the focal point and depth can be appropriately adjusted. Commercially available standoffs made of gel can be purchased; however, anything that will transmit the sound beam without significant



FIGURE 2.9 Three commonly available transducers are shown. Curvilinear (left), linear (middle), and microconvex (right). The microconvex is often the most useful scanner in companion birds as the footprint is smaller than the linear and curvilinear transducers, and the shape of the head is the easiest to angle into acoustic windows when they are available. The numbers on the transducers represent the range of frequencies in MHz each transducer can emit.

interaction such as a bag of IV fluids or a water bath can also be used, as the sole purpose of the standoff is to move the patient's anatomy away from the transducer. Gain and time gain compensation (TGC) can be adjusted to make the entire image (gain) or specific regions of the image (TGC) brighter via amplification of returning sound; however, this will not create image data where none is available. If the sound beam is not adequately penetrating tissues, the size of the electrical pulses (power) can be increased but this will affect thermal index (TI) and mechanical index (MI). These indices describe the likelihood of thermal (tissue heating) and mechanical (most frequently described as cavitation or hemorrhage) hazards in vivo due to the ultrasound beam and are displayed on the active screen.[29] Ultrasound is generally considered to be a very safe imaging modality; however, TI or MI greater than 1.0 combined with extended duration studies focused on a small area should be avoided.[29] Organs should be evaluated as entirely as acoustic windows permit and whenever possible in two planes: the short and long axes of the organ. Recording cine loops of the examination can be very helpful for later review, and at a minimum, representative static images or loops of the study should be stored as part of the medical record.

Patient Positioning for Ultrasound Imaging

In avian patients, acoustic windows to the coelomic cavities can be found ventrally between the sternum and pubis and laterally through the flank (Figure 2.10). The size of the patient and the size of the transducer footprint will determine which acoustic windows are most useful, and not all windows will be available in all patients. Patients are usually restrained



FIGURE 2.10 Ultrasound examination in an anesthetized pigeon using a microconvex transducer via the ventral acoustic window. (Image courtesy Dr. Shachar Malka, Long Island Bird and Exotics Clinic, NY.)

in an upright position or in dorsal recumbency (Figure 2.11) but can be adjusted as needed. Sedation or anesthesia may be necessary in some patients to prevent unnecessary stress and permit positioning. Ultrasound coupling gel or alcohol is needed to optimize contact between the transducer and the



FIGURE 2.11 Ultrasound examination in an awake patient using the ventral acoustic window and a high-resolution linear transducer. (Image courtesy Dr. Shachar Malka, Long Island Bird and Exotics Clinic, NY.)

patient but should be used cautiously to prevent hypothermia especially in smaller patients.

Advantages of Ultrasound Imaging

Ultrasound permits further evaluation of individual coelomic organ internal architecture and identification of internal architectural changes, when organs are accessible, than does radiography and survey CT. Ultrasound may help determine the origin of a mass when a mass effect is suspected radiographically and can be used to confirm the presence of coelomic effusion.[30–32] Ultrasound guidance for tissue sampling is invaluable in ensuring that representative samples are acquired, and that other structures are not inadvertently sampled or traumatized.[33] Ultrasound is the only imaging modality currently that can provide functional cardiac data (echocardiography) in birds.

Disadvantages of Ultrasound Imaging

The acoustic windows for ultrasound imaging in some species can be small due to skeletal anatomy and the presence of air sacs. This small size reduces further when air sac hyperinflation is present or in the presence of pneumocoelom or subcutaneous emphysema. Gas-filled GI structures may further limit evaluation of coelomic organs. Sedation or anesthesia may be necessary for patient compliance and to reduce stress. The use of coupling agents may cause hypothermia in smaller birds.

Direct Clinical Applications of Avian Ultrasound

- Structural and functional studies of the heart
- Evaluation of the coelomic cavities for the presence of fluid
- Evaluation of the liver[32,34]
- Evaluation of the urogenital tract[31,34]
- Evaluation of the intestinal tract[34]
- Evaluation of superficial subcutaneous masses
- Guidance for tissue sampling[33]
- Ocular and retrobulbar examination[35]
- Intracranial evaluation in thin-skulled birds or incompletely ossified skulls/open fontanelles

Selected Ultrasound Artifacts

Artifacts produced on grayscale, B-mode ultrasound images may occur due to physics, the ultrasound transducer, technical selections, or from the patient. The most frequently observed artifacts are those associated with gas due to poor contact at the skin or the effects of gas as a true reflector. Other artifacts such as mirror image artifact or partial volume artifact occur because of the physics of the sound beam.

Partial volume averaging occurs because there is thickness to the sound beam and it can result in slight misplacement of sound reflections, for example, giving the impression of echogenic fluid when an organ is partially captured in the same image as anechoic fluid. This can often be resolved by adjusting focal zone to the level of the artifact narrowing the beam and often removing the artifact, or by turning the transducer 90 degrees.

Mirror image artifact (Figure 2.12) occurs when the sound beam hits a highly reflective surface but the beam is redirected increasing the return journey. This causes the data to be placed further away from the transducer in the image. This is most observed where the craniolateral liver lies adjacent to

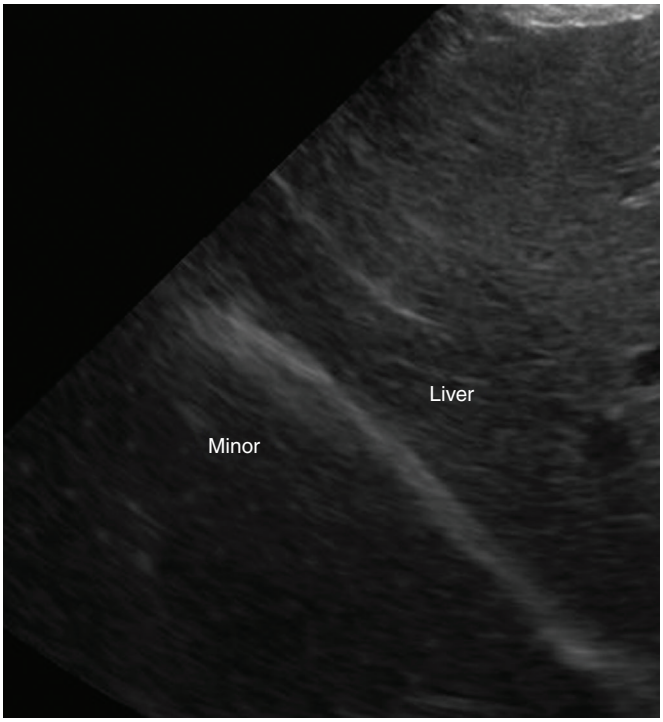


FIGURE 2.12 Mirror image artifact. A portion of the liver is duplicated due to an indirect return of the sound beam to the transducer, and liver tissue is displayed deep to the true liver margin.

air sacs and can result in both the liver and the liver's reflection being displayed.

Reverberation artifacts (Figures 2.13 and 2.14) occur when the sound beam hits a strong reflector. If there is incomplete contact at the skin and gas lies between the transducer and the patient, reverberation artifact will be observed, seen as recurrent horizontal lines through the image with uniform spacing and caused by the beam repeatedly bouncing between the transducer and reflector. Small gas bubbles present within, and trapping small volumes of fluid causes ring-down artifact, which occurs because of constant reverberation of the sound beam between the bubbles, and the constant return of sound to the transducer. Ring-down artifact is seen as a thin ladder-like artifact and is distinguished from comet tail artifact as the artifact extends to the depths of the image and does not taper. Comet tail artifact is a specific form of reverberation artifact that occurs when the sound beam meets highly reflective structures in close proximity and bounces between them. It is most frequently seen with crystalline structures such as pieces of grit within the GI tract or pieces of eggshell retained within the oviduct. Comet tails are distinguished from ring down by their tapering shape.

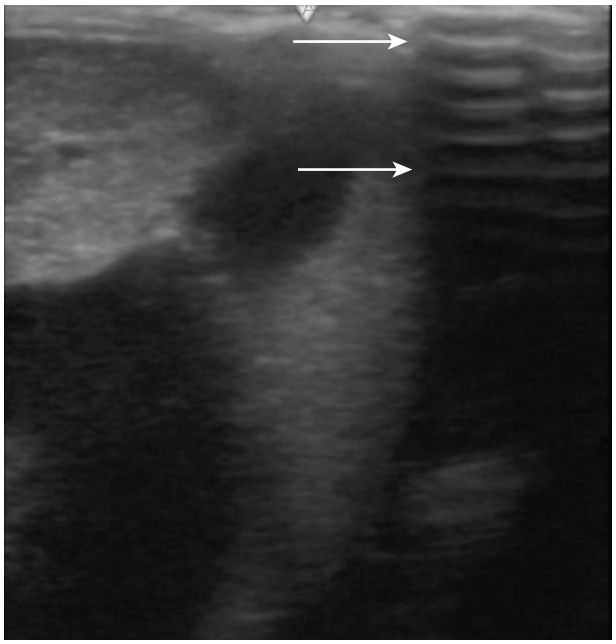


FIGURE 2.13 Reverberation artifact due to poor contact between the transducer and patient's skin is shown, highlighted by the two arrows. The sound bounces between the transducer and air trapped between the transducer and patient resulting in bright horizontal lines at regular intervals being displayed as part of the image.

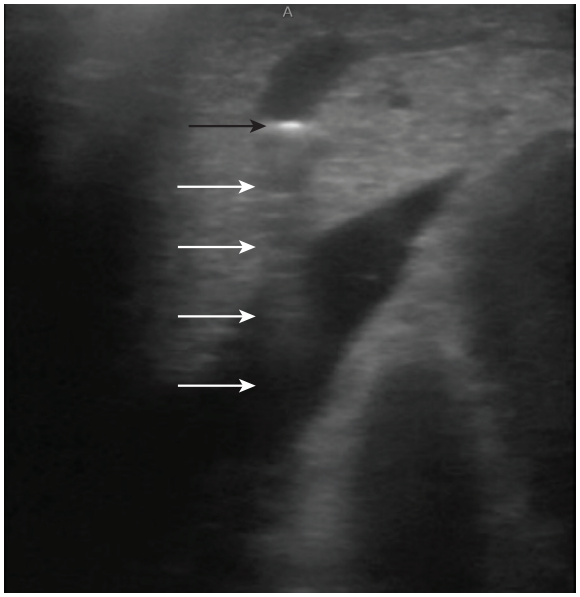


FIGURE 2.14 Ring-down artifact is a form of reverberation artifact and in this case is due to intravascular gas bubbles *(black arrow)*. The artifact occurs because of the sound beam bouncing between the reflectors (gas bubbles) with a constant release of sound back to the transducer.

INTRODUCTION TO COMPUTED TOMOGRAPHY

CT was first developed in the late 1960s and the first images of a brain in vivo acquired in 1971. CT became more widely available in human medicine around 1980 and really started to grow in veterinary medicine in the late 1990s. Slip ring technology was introduced in 1990 allowing continuous gantry rotation. Helical scanning with multirow CT scanners followed with greatly reduced scan time and improved spatial resolution. The first 16-row scanners were available in 2002, 64-row scanners in 2004, and 320-row detector scanners in 2010.[36] These CT scanners were all developed based on the use of a traditional fan-shaped x-ray beam and a table that moves through a gantry. For the purposes of this chapter, this technology is referred to as "fan beam."

Cone beam CT (CBCT) was originally developed in 1996 for dental and extremity imaging applications. In this technology, the data are acquired as a volume or stack rather than image by image by utilizing a cone beam–shaped x-ray beam. Volume acquisition means a large area can be covered with fewer rotations of the gantry, and multiple stacks of data can be acquired and merged together to cover an entire area of interest. Data are reconstructed by the system to provide a series of images. There is very high spatial resolution possible with these systems with excellent imaging of the bone and gas-filled structures, but the quality of soft tissue imaging is diminished compared to fan beam CT due to a relative increase in scatter radiation.[37] Recently, in the veterinary medical market, an offshoot technology has been developed from CBCT technology. Bone and respiratory tract definition is excellent on these scanners and CBCT developers continue to make efforts to improve soft tissue imaging capabilities. CBCT soft tissue capabilities lag behind fan beam image quality in general and are very scanner- and software-dependent. A basic comparison of traditional fan beam and CBCT scanners is provided in Table 2.1 and is summarized from publicly available marketing data.

CT permits superior evaluation of internal structures compared to radiography by removing superimposition of structures and permitting distinction between fluid and soft tissues via differences in attenuation.[38] CT may be preferable to ultrasound in some patients that lack adequate acoustic windows. Despite the improvement over radiography, evaluation of soft tissues on survey CT remains limited and can be greatly

TABLE 2.1

Basic Comparison Between Cone Beam Computed Tomography and Traditional Fan Beam Computed Tomography

	CBCT	Fan Beam CT
Power requirements	Standard single-phase 240 V	Three-phase 480 V
Dedicated lead lined room	No	Yes
Footprint size of scanner	Small	Large
Table weight limits (patient)	204 kg	136 kg older scanners; 204 kg is now standard
Gantry functional size	50 cm	Up to 90 cm megabore; 50–60 cm is standard
Maximum display field of view	32 cm	Up to 60 cm megabore; 40–50 cm in standard scanners
Maximum resolution	Isotropic 0.09 mm	8 rows or less anisotropic 1 mm 16+ row scanners isotropic 0.6 mm High-resolution CT (Siemens) isotropic 0.25 mm**
Relative scan time	Short for one stack. Long when multiple stacks needed.	Generally shorter than HDVI for a whole-body study.
Visualization of soft tissues	Generally poorer than fan beam CT due to more scatter, but varies greatly by vendor. Improves with IV iodinated contrast medium administration.	Generally better than HVDI but still limited. Improves with IV iodinated contrast medium administration.
Visualization of dense structures (bone, teeth)	Excellent. Better than fan beam (better resolution).	Excellent
Visualization of air-filled structures	Excellent. May be better than fan beam (better resolution).	Excellent

CBCT, Cone beam computed tomography; *CT*, computed tomography; *HDVI*, high-definition volume imaging; *IV*, intravenous.
**The high resolution package for Siemens to achieve this slice thickness is not automatically included on older Siemens CT machines and has to be purchased as specific additional software. So trying to make the point its available but may require an additional (not cheap) purchase.

improved via the use of IV nonionic iodinated contrast medium administered into the cutaneous ulnar or medial metatarsal veins. A study evaluating renal enhancement post-IV contrast in pigeons using a traditional fan beam CT scanner determined that a dose of 0.5 mL/kg of 370 mgI/mL contrast medium provided adequate soft tissue opacification.[39] However, few other studies have been performed to determine the optimal dose of contrast medium or optimal timing of the post-contrast scan specific to birds. Extrapolating from other veterinary species, a minimal dose of 400 mgI/kg and a preferable dose of 600 to 800 mgI/kg contrast medium is suggested so that enhancement is adequate on post-IV contrast images. That dose is simplified to 2 mL/kg when using commercially available formulations that range from 240 mgI/mL to 370 mgI/mL. Recent conference proceedings have suggested a total volume of up to 4 mL of contrast medium, injected continuously over a 2-minute period, which effectively increases the duration of circulating contrast medium at a diagnostic concentration. No immediate adverse effects have been described in medium-sized birds, although toxicologic studies at this dosage have yet to be performed.[40] This technique requires either the use of an infusion pump or that personnel remain within the CT scanner room or in close proximity to the CT scanner during scanning. Contrast medium should be injected directly into the IV catheter without dilution. Renal clearance of iodinated contrast medium is rapid in birds, and post-IV contrast scan acquisition should be started at the end of or immediately on completion of the injection without delay. Examples of optimized post-contrast images are shown using fan beam technology CT in Figure 2.15.

Contrast medium injected into the metatarsal vein will pass first through the ipsilateral renal portal system, and asymmetrical renal and ureteral opacification will be seen, with earlier excretion compared to injections given via the ulnar vein (Figure 2.16).

The contrast enhancement seen on CBCT technology images is often relatively less than seen on fan beam technology images due to scatter, longer scan times using CBCT, and rapid excretion of contrast medium that occurs in birds (Figure 2.17).

Severe reactions attributed to iodinated contrast medium are unusual in birds and where possible it is suggested that nonionic formulations should be used, as these tend to cause fewer reactions across species. Cardiovascular fluctuations or abnormalities may be seen immediately following contrast medium administration as well as respiratory depression/apnea and are usually transient. Sudden death has been reported in a small number of sick birds undergoing CT with a temporal association to contrast medium administration and tends to be seen more in smaller patients.[41] The exact cause of death (anesthesia-related, contrast media–related, or due to preexisting disease) is unfortunately often not determined.

Positioning for Computed Tomography

The direction of scan, head or tail first, does not tend to matter in avian species but should be consistent to permit easy linking of serial studies on the workstation for side-by-side comparison. Both sedation and general anesthesia are viable options for patients undergoing fan beam CT.[42] Patients should be placed in sternal recumbency. Dorsal and lateral recumbency should be avoided as it will reduce air sac inflation and relative organ position (Figure 2.3), which will affect interpretation of the air sacs themselves and make distinction and assessment of other coelomic structures more challenging. Wings can be positioned folded against the body wall or used to help support

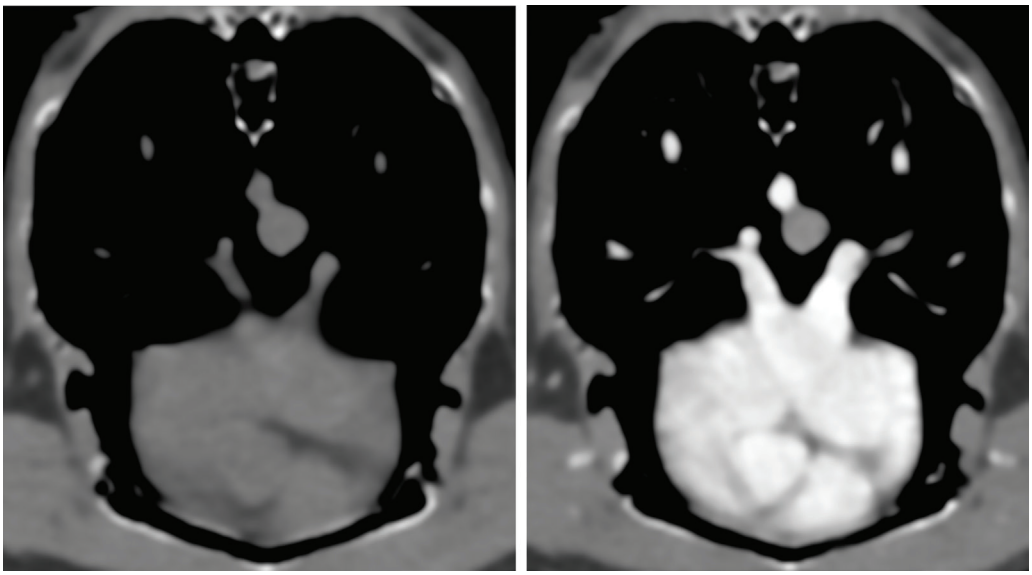


FIGURE 2.15 Pre- and post-IV contrast images acquired using a fan beam CT scanner with a soft tissue or medium-frequency reconstruction algorithm and timed to optimize the post-contrast series. Images are displayed using appropriate soft tissue window width and window level (WW 40, WL 350). *CT*, Computed tomography; *IV*, intravenous.

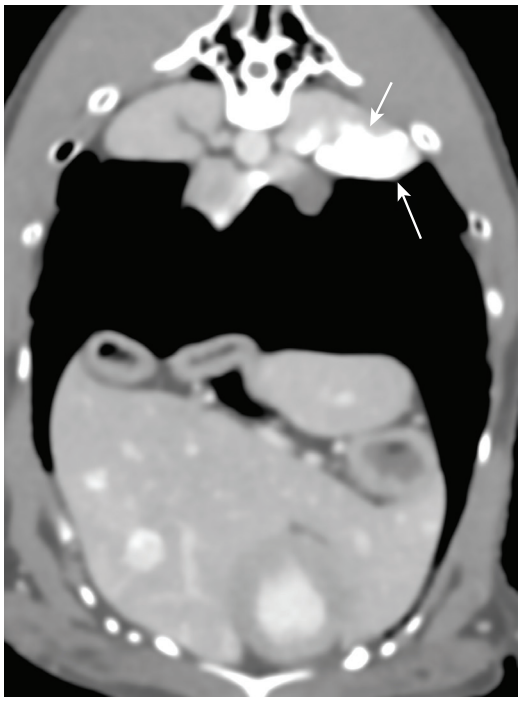


FIGURE 2.16 In this post-IV contrast medium image, the asymmetry seen in the kidneys with increased accumulation in the left kidney (*arrows*, shown on the right side of the image) is due to injection using the ipsilateral metatarsal vein.

the body in a sternal position by placing over the edges of the trough or on top of foam wedges (Figure 2.18).

The head is positioned straight with the beak parallel to the gantry table using radiolucent aids. Localizers are used to define the field of view and data that will be displayed on the final images. Localizers should be set as close to the body as possible to optimize the x- and y-axis resolution without cropping any of the soft tissues (Figure 2.19).

Localizer position should be checked both left to right and dorsal to ventral relative to the patient with the goal of left to right symmetrical positioning. The smaller the field of view, the higher the resolution. Slice thickness selection optimizes the z-axis, and for medium-sized avian patients, it should be no more than 1.5 mm, preferably less to optimize resolution. All three axes (x, y, z) must be optimized for high-resolution native images to be produced (Figures 2.20 and 2.21).

If wing joint or osseous pathology is present or suspected, a standard study is encouraged first with the wings folded against the body as this allows evaluation of wing and pectoral girdle symmetry. This can then be followed by a dedicated wing scan. For the dedicated wing scan, the patient is moved so their body lies perpendicular to the gantry table and the wing is positioned extended along the z-axis/parallel to both the long axis of the gantry table and the table surface to produce a high-resolution study of the wing. As with all studies, localizers and slice thickness should be adjusted to optimize the x, y, and z axes.

Multiplanar reconstructions (MPRs) can be performed post-processing from high-resolution images. These are very useful for localizing challenging lesions, evaluating joints, and can be used to correct unavoidable suboptimal positioning. When suboptimal positioning is present, MPRs can be very time-consuming and laborious to achieve, taking much longer than the time needed initially to correct positioning of the patient. MPRs made from lower resolution images may demonstrate pixilation, blurring, and stepping depending on parameters that were used to acquire the native images, and these artifacts may render them unhelpful (Figure 2.22).

Measurements made from MPRs produced from high-resolution isotropic data are accurate but MPR image measurements lose accuracy when low-resolution or anisotropic

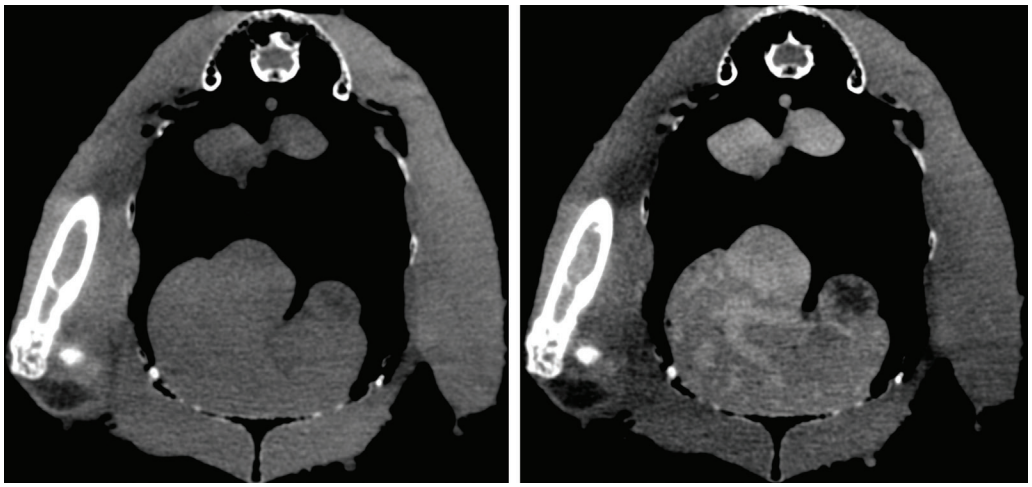


FIGURE 2.17 Examples of CBCT images acquired pre-IV contrast (left) and post-IV contrast (right) and optimized for evaluation. *CBCT*, Cone beam computed tomography; *IV*, intravenous.

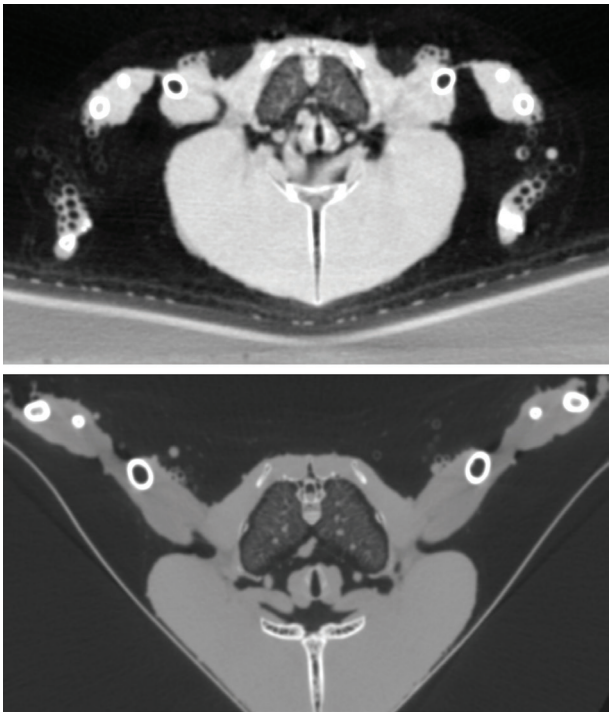


FIGURE 2.18 Transverse CT images with the wings alongside the body (top image) and suspended over the edge of a radiolucent trough (bottom image) to aid positioning. *CT*, Computed tomography.

data are used or if curved MPRs are needed to adjust for suboptimal positioning.

Advantages of Computed Tomography

The main advantage of CT is removal of superimposition of structures compared to radiography which improves conspicuity of structures that may not otherwise be accurately and thoroughly assessed.[43] Beyond that, CT can provide high-resolution studies with excellent evaluation of osseous and air-filled structures/respiratory tract with better evaluation of soft tissues than possible on radiographs, including staging of infectious or neoplastic processes.[43] CT is a rapid advanced imaging modality and scan times are very much faster than MRI; thus, anesthesia time can be kept very short. Any anesthesia or monitoring equipment can be used around CT scanners although monitoring equipment should be kept outside of the scan region. CT supersedes MRI when acute intracranial hemorrhage less than 24 hours old is a concern, for example, in acute head trauma. Acute hemorrhage is indistinguishable from other fluids on MRI but is slightly hyperattenuating to soft tissues on CT until the chemical composition alters around 24 hours post-leakage. CT images can be enlarged, WW and WL adjusted, and data reconstructed in multiple planes to aid interpretation and help plan approaches for surgery or tissue sampling. Images can be displayed as 3D images (3D slab, surface rending, and volumetric reconstructions) which aid with client education and provide an overview of the issues present for people less experienced with 2D CT images. Lastly, CT data can be used for 3D printing under certain circumstances, which may allow complex orthopedic procedures to be tested and prosthetic design and construction (see Chapters 31 and 37).

Disadvantages of Computed Tomography

The quality of soft tissue studies varies depending on the CT technology being used, patient size versus study resolution, and the availability of a post-IV contrast study. Fan beam technology scanners require dedicated rooms and electrical upgrades. The relatively short lifespan of many veterinary species compared to humans means that concerns regarding radiation exposure are generally low; however, as CT use increases, the effects of repeated radiation exposure may become evident and more applicable. CT studies contain a wealth of data, and some understanding and experience are needed to optimize the images displayed post-processing for the most accurate evaluation as well as understanding of CT anatomy. Publications are available that help correlate CT anatomy to gross anatomy[44] but not all avian species variations have been documented to date. High-resolution studies will generate many images, which require high-speed Internet for timely transfer out of the console to the PACS and beyond.

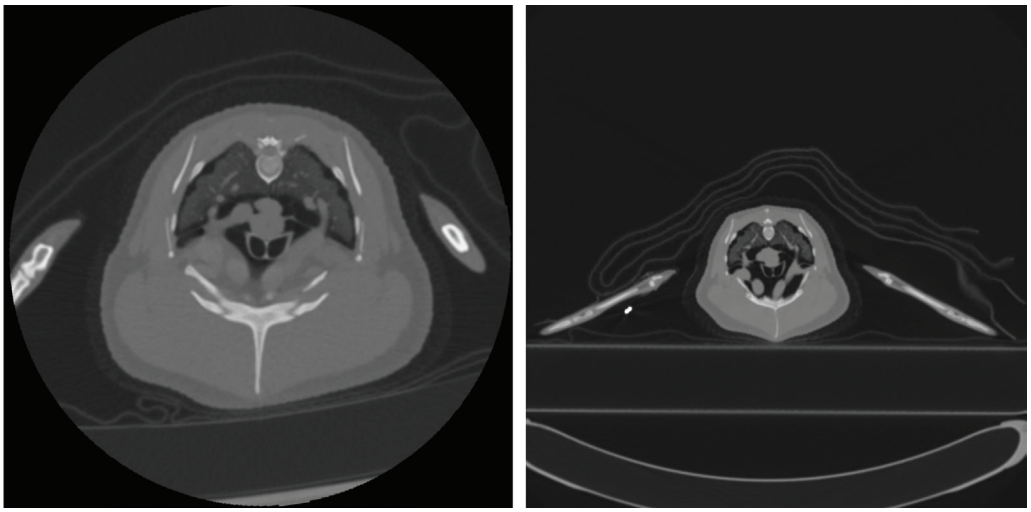


FIGURE 2.19 On the left image, the display field of view localizers (shown by the *gray circular margin*) have been set close to the body wall without cropping soft tissues. This will permit higher resolution images than the setup in the right image where a much larger field of view has been selected. The same total number of pixels is used for both images, so with fewer pixels covering the anatomy on the right, resolution will be lower than on the left when the image is zoomed or reconstructed in alternative planes.

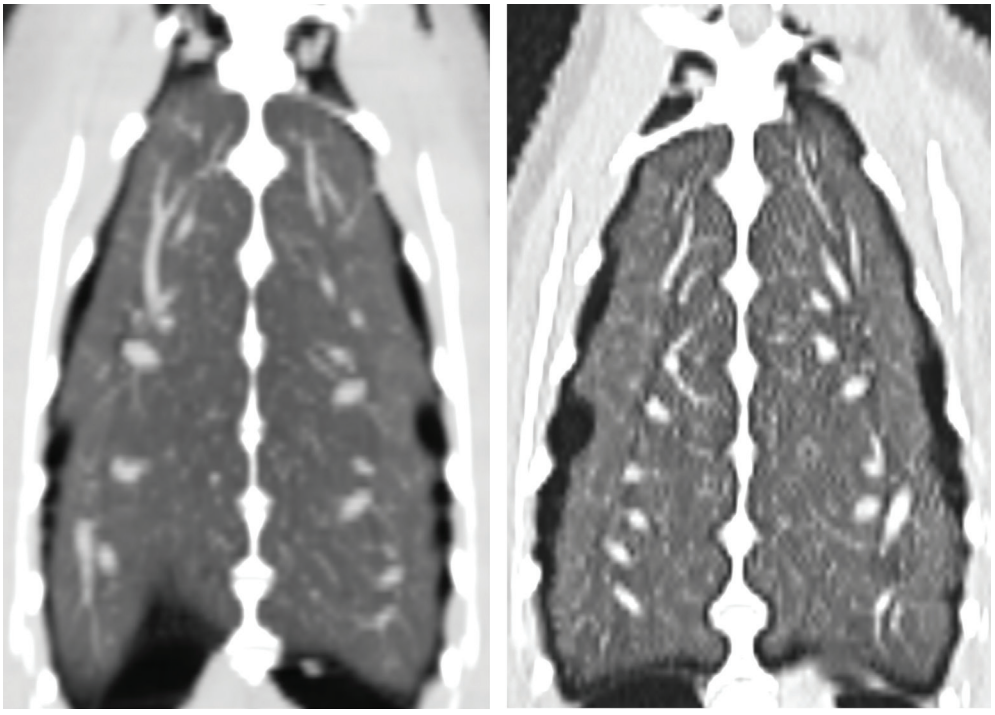


FIGURE 2.20 Dorsal reconstructions made post-processing from the same penguin as in Figure 2.19, with images acquired at 0.75 mm on the left and 2 mm on the right. The vasculature and lung margins have a pixelated or stepped appearance on the right with smoother margins seen on the left.

Direct Clinical Applications of Computed Tomography

- Imaging of axial and appendicular skeletal structures, particularly valuable for imaging of the head[45,46]
- Imaging of the respiratory tract[44,47]
- Imaging of the structures of the coelomic cavities (this may be limited on noncontrast CT but is improved with IV or intraosseus (IO) contrast medium administration)[44]
- Excretory CT urograms
- Angiography and evaluation for atherosclerosis[48]

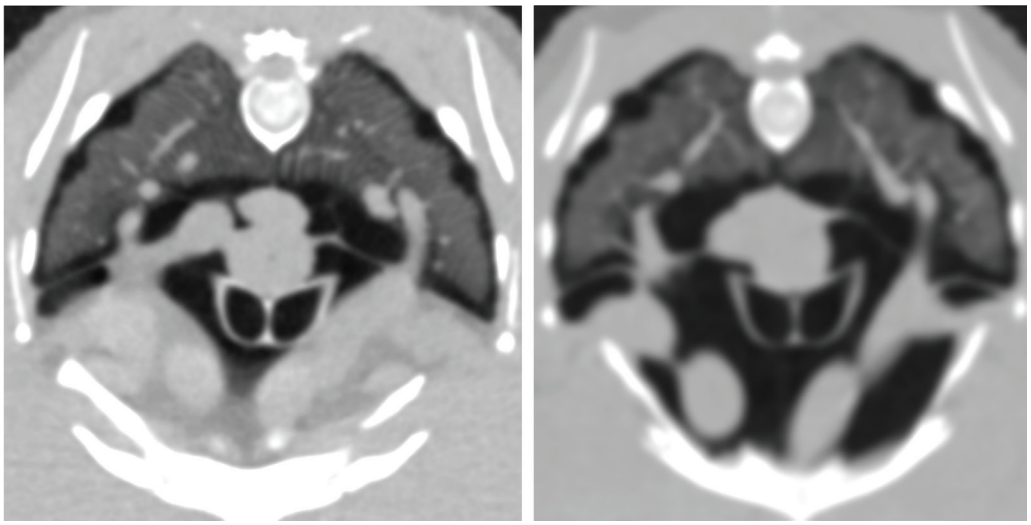


FIGURE 2.21 Transverse images from Figure 2.19. The left image demonstrates better resolution once zoomed in for evaluation because all three planes were optimized during setup. The image on the right from the lower resolution image in Figure 2.19 is blurred in comparison when zoomed to the same size.

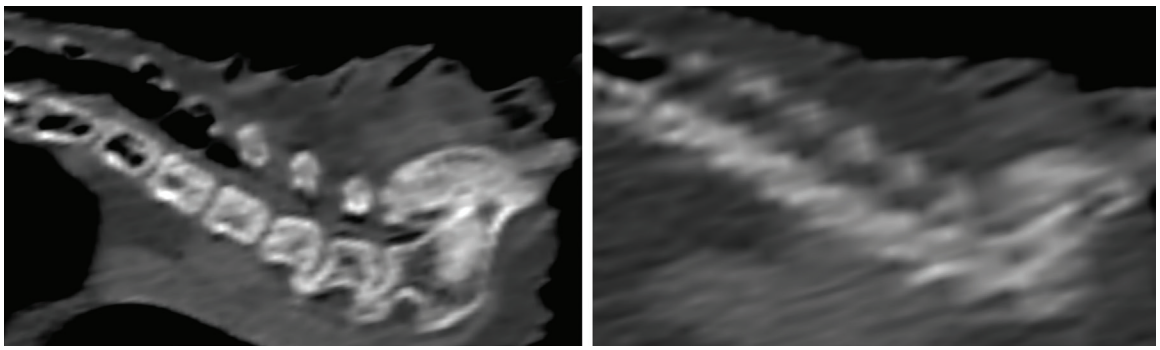


FIGURE 2.22 Sagittal reconstruction performed to evaluate the spine with adequate resolution (left image) and inadequate resolution (right image) to be helpful in the interpretation. The resolution of multiplanar reconstructions is limited by the original resolution acquired.

- Identification of intracranial hemorrhage in acute trauma <24 hours
- Guidance for tissue sampling, surgery, and radiation therapy planning

Interpreting Computed Tomography Images

Initial selection of the appropriate reconstruction algorithm and slice thickness for processing of data into images is key for subsequent optimization and accurate interpretation. Fan beam technology images are produced using operator-selected reconstruction algorithms. The names of the reconstructions vary by manufacturer, and in general, soft tissue and bone reconstruction algorithms (medium and high frequency) pre-IV contrast and soft tissue (medium frequency) post-IV contrast are needed for thorough evaluation. As most companion birds are very small in size, thin slice thickness is necessary for optimized resolution relative to body size, and sub–1 mm images are a must. Many fan beam CT scanners can provide 0.5 to 0.625 mm slice thickness images as standard and thinner images with additional high-resolution software when available. CBCT will reconstruct images in the range of 0.15 to 0.3 mm as standard. It should be noted that when using fan beam CT in helical mode, fan beam high-resolution software, or CBCT, the final thin slice images are a mathematically derived predicted representation of what anatomy looks like in each image slice based on the larger data volume, rather than an absolutely true depiction of the anatomy present. Once the reconstructed images are available, they are windowed and leveled by the interpreter to optimize tissue evaluation. Window and level can be repeatedly adjusted, but the algorithm used to create the images cannot be changed once images are created. Bone reconstruction algorithm images are optimized for evaluation of both highly attenuating structures, such as bone, surgical implants, or ingested mineral/metal, and high-contrast, air-filled respiratory tract, and other air/gas-filled structures. Appropriate initial algorithm selection and subsequent window and level adjustment are critical for accurate interpretation of the images. For example, soft tissue or medium-frequency reconstruction algorithms have a softening effect that is used to enhance small differences in attenuation between different

soft tissues. Even if windowed and leveled appropriately for bone, the softening algorithm causes blurring of bone, artificial thickening of cortices, and poor conspicuity of trabecular bone pattern. This means that subtle cortical and trabecular bone lesions can be missed if this reconstruction is used for osseous evaluation. Likewise, a bone reconstruction is a high-frequency reconstruction algorithm that sharpens margins resulting in a large amount of noise when windowed for soft tissue, and soft tissue lesions may be obscured (Figure 2.23).

Hounsfield units (HU) are a quantitative measurement used in fan beam technology and are calibrated relative to water (0 HU) and air (−1000 HU). This means that quantitative HU measurements of tissue can be made and are accurate across time and between scanners. It also means that preset WW and WLs can be created. A general guide for WW and WL for images created via fan beam technology is:

- Soft tissue reconstruction algorithm (pre- and post-IV contrast): WL 40, WW 350
- Bone reconstruction algorithm for bone: WL 300, WW 1500
- Bone reconstruction algorithm for respiratory tract: WL −500, WW 1400

CBCT technology treats the raw data slightly differently, and while there often are operator-selectable options for image processing, usually only a single pre- and post-IV contrast series is reconstructed that can be windowed and leveled to optimize the different tissues. CBCT technology has more latitude within the images than fan beam technology, and while attenuation follows the same trends as fan beam and can be qualitatively compared within a study, absolute values do

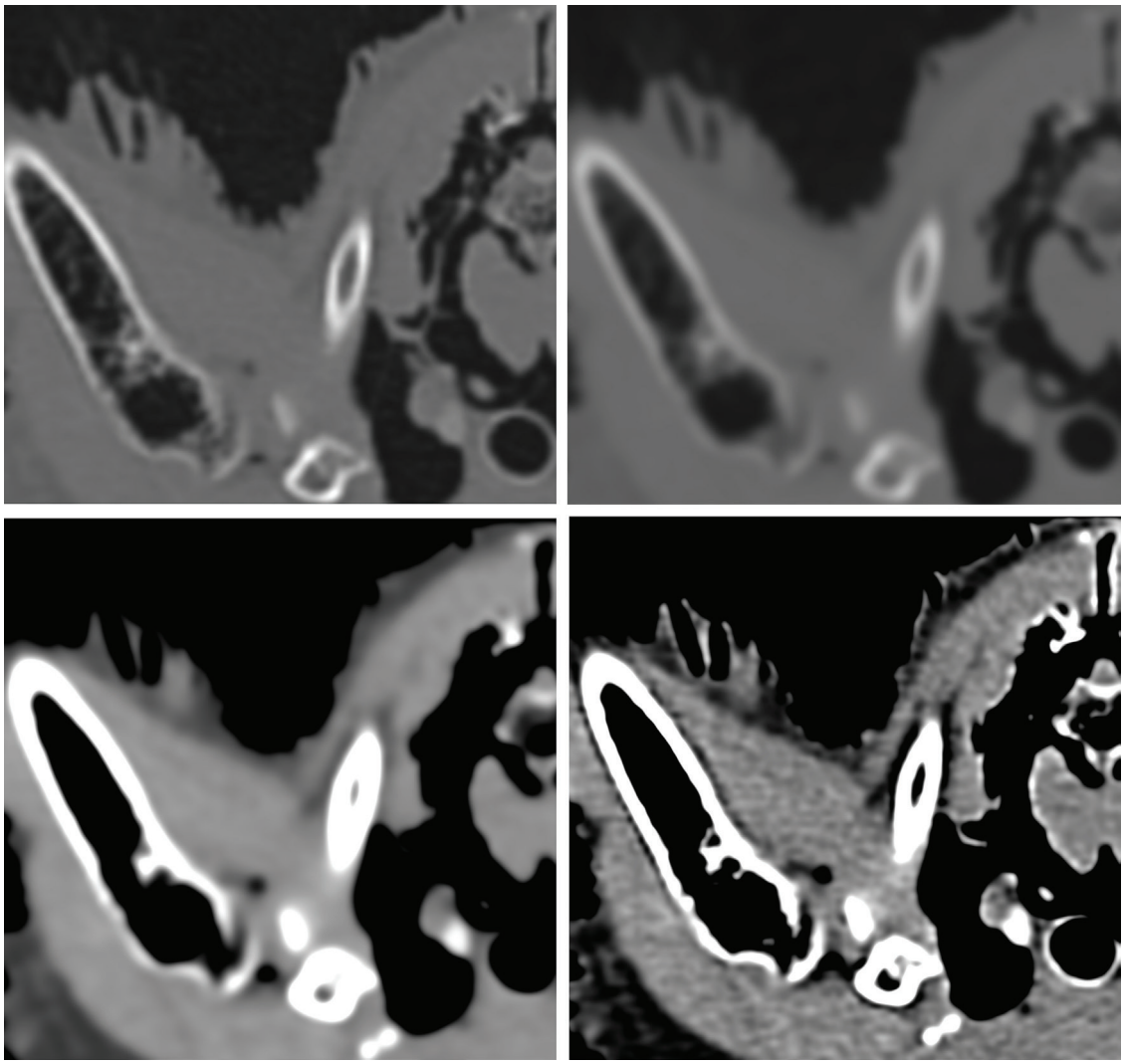


FIGURE 2.23 Fan beam CT technology images. The top row shows images windowed and leveled for assessment of bone. Top left is a bone reconstruction algorithm and shows superior detail of the bone compared to top right which is a soft tissue reconstruction algorithm. On the bottom row, the left image is a soft tissue reconstruction algorithm appropriately windowed and leveled for soft tissue evaluation. Bottom right is the same image acquired using a bone reconstruction algorithm, then windowed and leveled for soft tissue. The soft tissue detail is superior on the left with the right image demonstrating noise and mottling through the soft tissues. *CT,* Computed tomography.

not carry across different studies or different scanners. CBCT images demonstrate less soft tissue distinction and less post-contrast enhancement when compared to fan beam technology (Figure 2.17); however, optimized bone and respiratory tract images are quite similar in appearance between technologies with superior spatial resolution available on CBCT.

Internal anatomy varies among species of birds; however, several excellent texts that correlate anatomical and CT anatomy of specific species are available of the structures of the coelomic cavities,[41] head,[45,46] and respiratory tract,[47] and can be used as helpful references even if not specific to the species being evaluated.

Selected Computed Tomography Artifacts

There are many CT artifacts that can occur and the origin may be physics-based, patient-based, or scanner-based artifacts. Very detailed texts that explain the artifacts according to each origin are available.[49] When recognizing and evaluating artifacts, most clinicians and radiologists see the artifact on the final images and then work backward simply with the desire to resolve the issue. Artifacts that tend to have the same or similar final appearance also tend to have the same resolution regardless of the origin, and as such, artifacts are simply grouped here according to their appearance on the final image. The most frequently observed artifacts are image graininess due to noise (physics, scanner) or photon starvation (physics); streaks due to beam hardening (physics) or metal (patient), undersampling (physics), out-of-field anatomy/incomplete projections (patient), or windmill artifact (scanner, physics); blurriness due to partial volume averaging or algorithm selection (physics); ring artifacts (scanner); and malpositioned images due to patient motion (patient) or a sticky gantry/table (scanner).

A grainy image on CT occurs when inadequate photons reach the image detector after passing through the patient. There are many causes for this, including inadequate technique, allowing system electronic noise to affect the data generated, patient anatomy/size, heating, positioning, or monitoring equipment attenuating the beam. The artifact can also be created when fan beam bone reconstruction algorithm images are inappropriately windowed for soft tissue. In the case of inadequate photons, the artifact significantly degrades image quality (Figure 2.24). An example where this frequently occurs is when a fan beam CT scanner is set to acquire images at a thick slice thickness and then retrospectively thin slices are made. The technique the scanner automatically selected for the thicker images is now inadequate for the thinner slice images and noise occurs. This artifact can be corrected by increasing the technique (kV and/or mA) and by ensuring that the smallest end scan image thickness that will be required is the slice thickness that is natively acquired using an appropriate kV and mA. In the case of fan beam images where graininess is noted on bone reconstruction images but not soft tissue reconstruction images, appropriate windowing and leveling will resolve the appearance.

The blurring of structure margins due to partial volume averaging occurs in both technologies and it occurs in two ways. The first is when tissues of different attenuation both fall into the same voxel resulting in an averaging of the attenuation (Figure 2.23). This may affect conspicuity particularly of margins of lesions or give normal anatomy an abnormal appearance. The second is failure to distinguish margins of closely located structures, for example, distinguishing vasculature on angiography or distinguishing between liver lobe margins. In both cases, blurring of structure margins is noted and will worsen if MPRs are attempted. Partial volume averaging is removed by decreasing the slice thickness to an appropriate size for patient size.

Stripes may be seen on images due to several causes that include beam hardening, undersampling, out-of-field anatomy

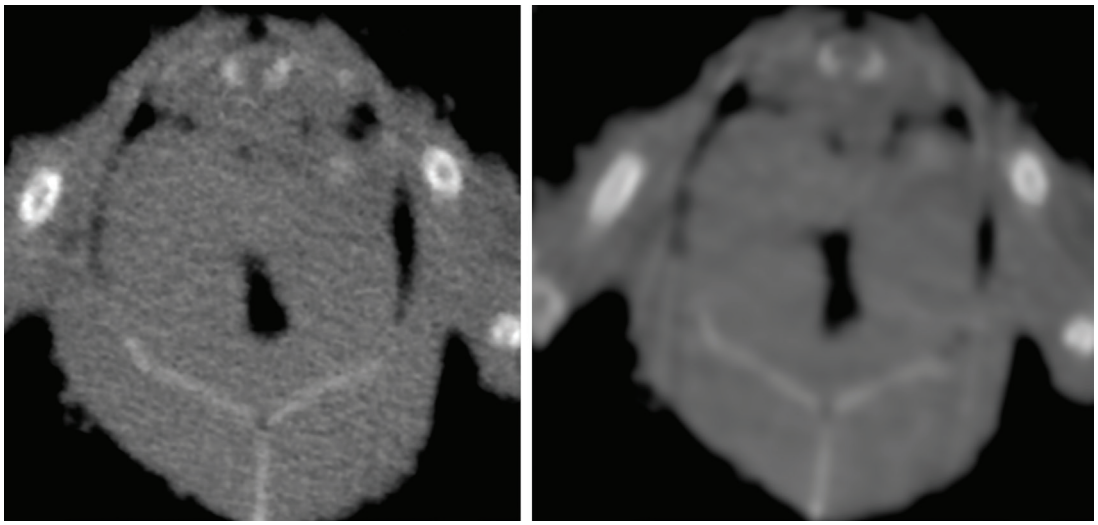


FIGURE 2.24 CBCT images of the femur. The image on the left demonstrates noise resulting in a mottled and grainy appearance to the image compared to the right image. Partial volume averaging is also demonstrated in this image with the image on the left acquired at 0.3 mm slice thickness and on the right at 0.6 mm slice thickness. The reduced distinction between the femoral cortex and medullary cavity is due to inclusion of more tissues in each voxel. *CBCT,* Cone beam computed tomography.

(incomplete projections), and windmill artifacts. Frustratingly, all artifacts can occur simultaneously and may be seen in some form regardless of scanner technology. Beam hardening occurs when a very attenuating structure prevents passage of adequate x-rays to image the adjacent tissue. This can occur due to patient anatomy, internal pathology such as metal ingestion, or when metal-based equipment remains adjacent to the patient in the gantry during scanning or large sandbags are used for positioning. In fan beam technology, the effect will only be observed in the x and y planes at the level of the origin and will not extend through the z-axis beyond the margins of the object causing the artifact. In CBCT, the cone shape of the beam means the effects of the artifact may be seen extending through the volume in the x-, y-, and z-axes depending on where within the volume the object lies. The amount of metal present and its location will determine how much effect it has on the image quality (Figure 2.25). The solution for beam hardening is to remove the offending object from the scan area inside the gantry if at all possible. When the artifact is due to internal material that cannot be removed (shrapnel, surgical implants), increasing the technique (kV and/or mA) and/or increasing the slice thickness slightly will diminish the effect of beam hardening on image quality. In smaller patients, rescanning the patient after repositioning by 90 degrees to lay perpendicular to the gantry table may be feasible and can help produce diagnostic quality images through anatomy most affected by beam hardening. Daily scanner calibration is needed in scanners with built-in filtration hardware or software to help combat beam hardening. This enables them to work to the best of their ability and it is crucial that nothing is in the gantry during these calibration events for the software to work appropriately.

Undersampling artifacts have a slightly different appearance to beam hardening, creating streaks that slightly diverge and widen as they course from their origin to the periphery.

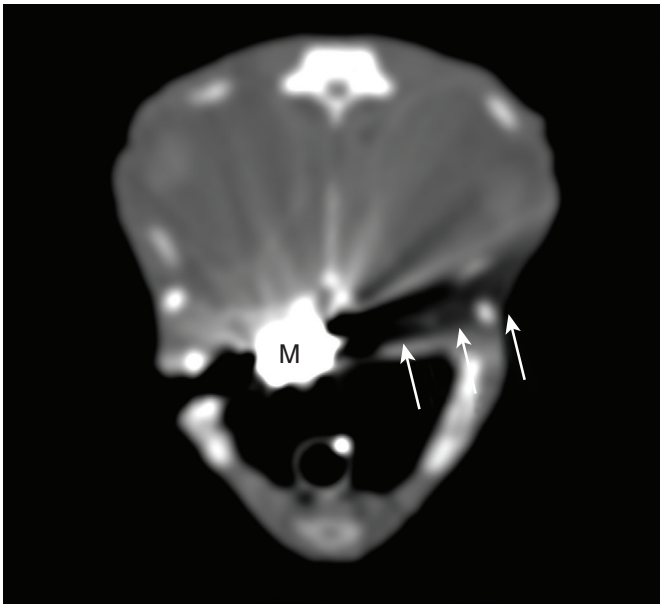


FIGURE 2.25 Beam hardening artifact (*arrows*) due to metal (M) positioned within the oral cavity. The metal greatly attenuates the beam resulting in streaks across the images.

These artifacts occur when the beam angle is nonperpendicular and interacts with highly attenuating structures and at margins between structures with significantly different attenuations. Concurrent partial volume averaging can also occur due to the oblique photon course in larger patients (demonstrated by the widening of the artifact toward the periphery). Figure 2.26 demonstrates some examples of undersampling. These artifacts occur in both technologies but are seen most frequently in CBCT and are commonly referred to as cone beam artifact. Undersampling artifacts are, however, also seen in fan beam technology when very thick slice thicknesses are acquired or if the scan field of view is too small and a portion of the patient is outside of designated scan region. In the latter case, the artifact is more correctly referred to as incomplete projection or out-of-field artifact. Incomplete projection is only an issue in fan beam technology as CBCT scanners automatically scan everything within the gantry. In fan beam technology, both the scan region and the display field of view (final image) can be adjusted. Treating undersampling artifacts in the same way as beam hardening and increasing technique (kV or mA) to increase the number of photons reaching the image detector will improve the appearance if it is significant enough to affect image quality. Ensuring all of the patient is inside the scan field of view will also reduce streaking in cases where anatomy is outside the scan field of view on fan beam CT scanners.

Windmill artifact can be seen on both technologies. It occurs during fan beam technology helical scanning when high pitch results in several detectors intersecting the plane of reconstruction (slice thickness). Equally distanced bright streaks are seen diverging out toward the periphery from focal high-density structures such as the ribs and the streaks "rotate" as the image stack is scrolled through. This is unlikely to occur in many avian CT studies given the small size of the majority of patients and use of low-pitch values and can be mitigated by using a noninteger pitch, for example, selecting a pitch of 3.5 rather than 4 on a four-row CT scanner.[46] Newer helical multislice CT scanners use z-filter helical interpolators to reduce the artifact. A similar image artifact is seen on CBCT around long bones that lie parallel to the z-axis (Figure 2.27).

Another form of beam hardening that was seen occasionally in older scanners is called cupping artifact. This is best demonstrated by a solid cylindrical phantom. The center of the cylinder attenuates the beam more than the edges, and so the center is assigned lower Hounsfield units and a darker shade of gray. It is very unusual to see cupping on images from modern scanners as they have a beam hardening software built in to correct for this. In addition, avian patients' pneumatization of the bones of the head, where the artifact was most typically observed, causes far less attenuation of the beam than in other species. This artifact is now more a testing tool to ensure a beam hardening software is working appropriately using phantoms, than a concern in clinical studies.

A common scanner-based artifact that occurs is ring artifact. This is observed as eccentric circles that partially obscure anatomy with the center of the circle set at the epicenter of the scanner. This artifact occurs due to image detector calibration issues or inhomogeneous image detector sensitivity (Figure 2.28) and can occur in both fan beam and CBCT technologies. Calibration errors in both CBCT and fan beam technology can usually be reduced or

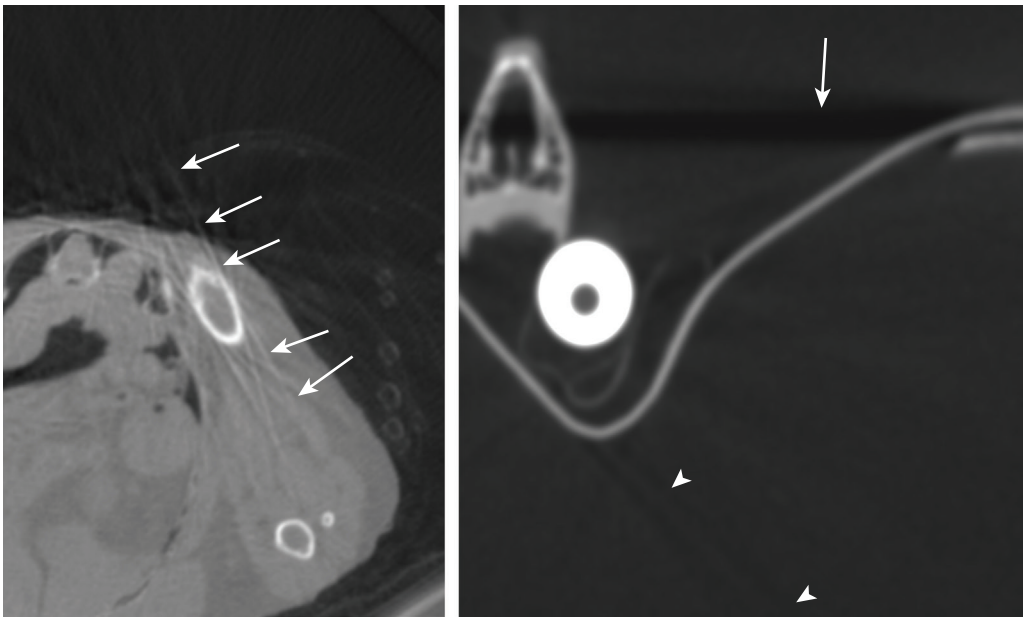


FIGURE 2.26 Examples of streak artifacts (*arrows*) seen on CBCT images due to undersampling. On the left, streaks extend out from the femoral cortices. On the right, streaks are seen coursing horizontally due to the extended edges of the trough and obliquely due to the inner margins of the trough. *CBCT*, Cone beam computed tomography.

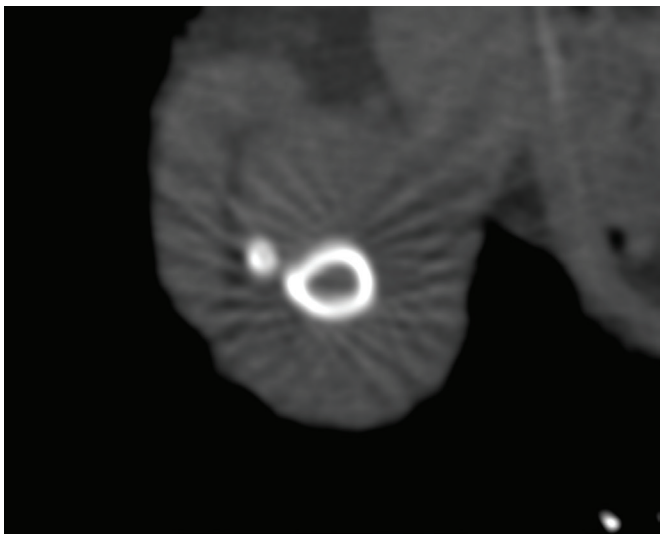


FIGURE 2.27 Windmill-like artifact shown here on a CBCT image as rotating striations or stripes extending outward from a high-density structure. *CBCT*, Cone beam computed tomography.

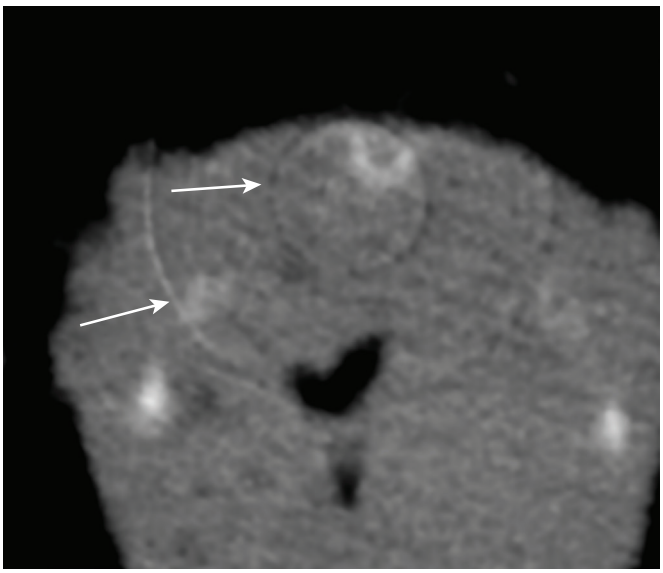


FIGURE 2.28 Ring artifact or detector calibration artifact is seen as *circles* through the image(s) and is highlighted by *arrows*.

prevented by performing the complete daily warmup and calibration test indicated on the individual scanner and by ensuring nothing is present within the gantry while calibration tests take place. If the artifact persists despite calibration, detector(s) may require servicing or replacement.

Motion artifact can occur when the patient moves (breathing, twitching, or gross movements) or when debris gets caught in the CT scanner resulting in irregular or jolting movement of the gantry table (fan beam) or gantry (CBCT) during the scan. The higher the study resolution, the more effect a smaller amount of motion will have on the final image. The likelihood of motion also increases with longer scan times. Motion may be identified most commonly as streaks, varying attenuation, blurring, or duplication or malalignment of anatomy, as shown in Figure 2.29. Some motion, for example, cardiac, respiratory, or GI movement, is unavoidable while other motion is reduced by adequate restraint, sedation, or anesthesia during the study, by minimizing scan times (i.e., faster gantry rotation and/or more x-ray sources) and by ensuring the gantry and table runners are kept clean.

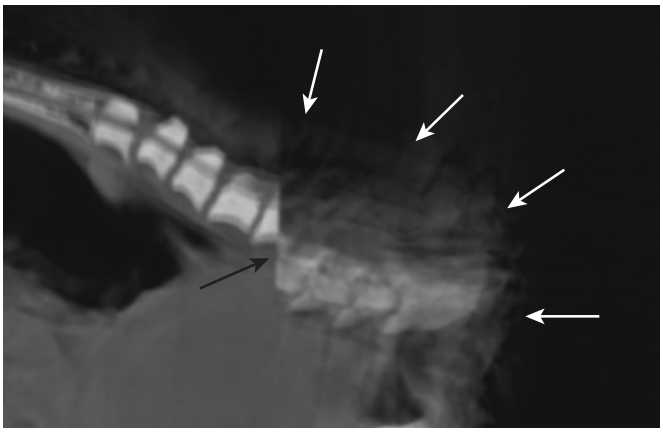


FIGURE 2.29 Motion artifact shown in a stack acquired on CBCT. There is a false step in the anatomy *(black arrow)* and blurring of the distal spine/tail *(white arrows)* because of motion occurring during scanning. *CBCT,* Cone beam computed tomography.

INTRODUCTION TO MAGNETIC RESONANCE IMAGING

MRI was first developed in the late 1930s but was not used for medical purposes until the late 1970s. In the last two decades, there has been increased accessibility and availability of MRI to veterinarians with the development of dedicated outpatient veterinary MRI imaging centers and installation of MRI units in veterinary specialty hospitals. There are several factors to consider when working with or considering installation of MRI scanners. All MRI scanners require a dedicated imaging room with shielding to limit radiofrequency (RF) interference, as well as very efficient air temperature control. Only certified MRI-safe equipment should enter the MRI scanner room including anesthesia equipment. Ferrous metal objects can become high-velocity projectiles once they enter the magnetic field, and nonferrous, non–MRI-certified equipment can affect the scan quality via interference. MRI scanners in clinical use are typically in the range of 0.15 to 3 Tesla (T). MRI scanners up to 1 T are considered low field and over 1 T high field. 1 T and higher MRI scanners have a closed design with a standard bore diameter of 60 cm. MRI scanners in the range 0.15 T to 0.6 T have an open design with a wider functional diameter of 70 cm. Open scanners are permanent magnets that have lower operating costs associated with them compared to closed magnets but may have a more limited selection of sequences. A developing consideration for high-field MRI scanners is that most are resistive or superconductive magnets, and many require helium as a coolant to function efficiently. Helium needs periodic replenishment and helium reserves in the United States are depleting, which may affect long-term availability and cost. MRI scanners with lower helium volumes and lower helium losses as well as helium-free scanners have been developed more recently.

MRI images are produced when protons in water are excited and the energy emitted (signal) is captured by a coil, data collected and converted into an image. Each sequence and each orientation/axis are acquired independently; thus, multiple scans are needed for a complete study contrary to image acquisition in CT. Avian patients have relatively less water/soft tissue compared to similarly sized mammals due to pneumatized bone and a large volume of air present within the respiratory tract. This causes a relatively diminished signal production compared to similarly sized mammals. Open low-field scanners have lower image quality due to poorer signal-to-noise ratio (SNR) when compared to closed high-field scanners using the same scan parameters. Veterinary-specific, low-field models have the benefit of coils that have been developed specifically for veterinary species. These tend to fit animals far better than coils designed for human body parts resulting in a relative improvement in SNR ratio and image quality compared to human coils used in the same system, but image quality will still be lower than a high-field scanner study.[50] In human shoulder studies that compared low- and high-field scanner images, small lesions evident on high-field MRI images that were confirmed at surgery and considered clinically significant were frequently not identified on low-field MRI images.[51] In veterinary-based studies, low-field scanners produced poorer image quality compared to high-field scanners, but images remained diagnostic provided scan time was increased.[52,53] However, it should be noted that those veterinary studies were performed on horse cadaver limbs where extended scan times were inconsequential. SNR ratio in MRI can be improved on both low- and high-field scanners at the expense of spatial resolution (increasing slice thickness and/or field of view) and scan time (increasing the number of excitations [NEX]). Lowering resolution runs the risk of missing pathology. Lengthening scan times increases the likelihood of motion artifacts and requires a longer duration of anesthesia.

Diagnostic MRI studies have been acquired in psittacine birds with chronic sinusitis using a high-field magnet. In that study, the time taken to acquire diagnostic images was not reported and the authors state that only enough sequences (rather than a complete study) were acquired to identify that pathology was present, and that three planes that are considered necessary for a complete study were not acquired.[54]

When metallic implants, identification microchips, ingested metallic material, or shrapnel are present within a patient, there will be susceptibility artifacts and artifacts associated with induced eddy currents that are evident on images. These will vary in their effect on image quality among sequences used and tend to be fewer and less severe when using low-field MRI compared to high-field MRI. MRI-safe metallic implants and equipment do not directly result in image artifacts but can heat within the MRI scanner and result in tissue damage. Nonsurgical metal such as shrapnel or ingested metal may be ferrous and susceptible to both migration and heating. Metal that has been present in tissues for at least 3 months tends to secure in place due to bone proliferation and fibrosis and is typically considered safe for MRI.[47] Rarely, superficial burns have been reported that occurred due to heating of items in contact with the patient such as fluid bags or cords from monitoring equipment.

Gadolinium-based contrast medium is used in MRI and can be used to help characterize lesions and identify active lesions. The dose of gadolinium extrapolated from other veterinary species is 0.1 mmol/kg injected IV via the cutaneous ulnar or medial metatarsal veins. The effects of gadolinium contrast medium persist for longer than iodinated contrast medium and can be expected to be evident on MRI images for at least 20 to 30 minutes after administration. Increased signal intensity/contrast enhancement of lesions within the brain and spinal cord parenchyma following gadolinium administration

is indicative of disruption of the blood-brain barrier.[50] No adverse reactions to gadolinium have been reported in birds.

Patient Positioning for Magnetic Resonance Imaging

Patients must be motionless with limited accessibility during scanning, and general anesthesia is necessary under most circumstances. Patient positioning will be primarily determined by the scanner and coil system being used combined with patient size. The anatomic area of interest needs to be positioned as close to the coil as possible to maximize SNR ratio and with a straight spine so that midline sagittal images include the entire length of the spinal cord.

Advantages of Magnetic Resonance Imaging

The superior soft tissue contrast resolution produced in MRI images often outweighs the reduced spatial resolution compared to CT. MRI is the modality of choice for central nervous system evaluation. Among sequences, lesions of adequate size compared to slice thickness will be easily identified on MRI.

Disadvantages of Magnetic Resonance Imaging

MRI is very susceptible to artifacts from environmental, hardware, software, and inherent patient causes. Relatively long scan times are to be expected in avian species regardless of the scanner used due to small patient size compounded by fewer water-containing tissues relative to body size than other species. The many air-filled structures, and in particular the air-tissue interfaces around the central nervous system, may limit the use of gradient echo (GRE) sequences which are otherwise helpful for identifying hemorrhage older than 24 hours. The scan time for MRI is long under any circumstances especially when compared to CT, and this not only increases the likelihood of motion artifact but also increases the time the patient is under anesthesia. A full set of sequences for a penguin-sized avian brain study on a high-field scanner should be expected to take at least 35 to 40 minutes without post-contrast sequences and around an hour with post-contrast sequences, while the same study on a low-field magnet will take approximately 30% to 50% longer and may have more limited sequences. General anesthesia is needed for avian patients undergoing MRI to prevent movement. Only MRI-specific equipment can be in the scanner room, and options are quite limited and optimized for human-sized patients rather than small birds. This may add challenges to anesthesia, patient monitoring, and safety. The use of MRI is limited in small patients, as the typical slice thickness selected in MRI to keep reasonable scan times is 2.5 to 3 mm, which is very large relative to all birds and limits use to medium-sized birds or larger. Few MRI acquisitions permit MPRs, and a component of the long scan time is the need to acquire multiple sequences in multiple planes for the most accurate assessment.

Direct Clinical Application of Magnetic Resonance Imaging

- Imaging of the central nervous system[55–57]
- Imaging of the eye[57]
- Imaging of the inner ear[57]
- Imaging of the solid soft tissues of the coelom[56]
- Imaging of chronic sinusitis[54]
- Limited evaluation of the heart (due to motion), respiratory tract, and osseous structures (due to lack of signal). Pathological lesions including granulomas or neoplasia in the bone or lung may be identified on MRI due to the increased soft tissue and increased signal at those locations

Selected Magnetic Resonance Imaging Artifacts

Many MRI artifact peer-reviewed texts[58] as well as online resources providing extensive reviews and examples of artifacts are available. MRI artifacts are frequently sequence-dependent and so their relevance depends on the sequences available and selected. The most observed artifacts that can affect all scanners and sequences to some degree include susceptibility artifact, motion, and aliasing (wrap around) artifact.

Susceptibility artifact can be used as a diagnostic advantage when trying to determine if hemorrhage or other paramagnetic material is present. However, susceptibility artifacts from metal or air-tissue interfaces can obscure anatomy and pathology (Figure 2.30). In some cases, moving the skin and

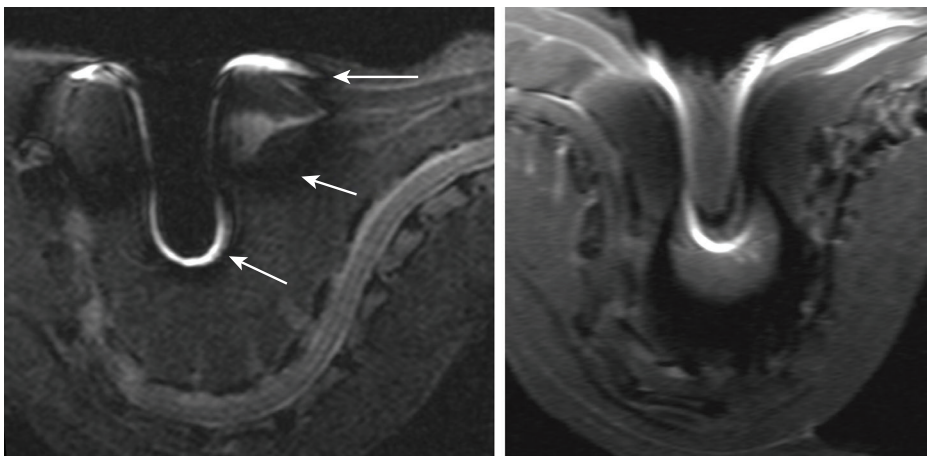


FIGURE 2.30 Examples of susceptibility artifact (highlighted by arrows) from a microchip. In the image on the left, the resultant artifact is significant but does not affect the images of spinal anatomy. On the right, the spinal anatomy is affected by the artifact.

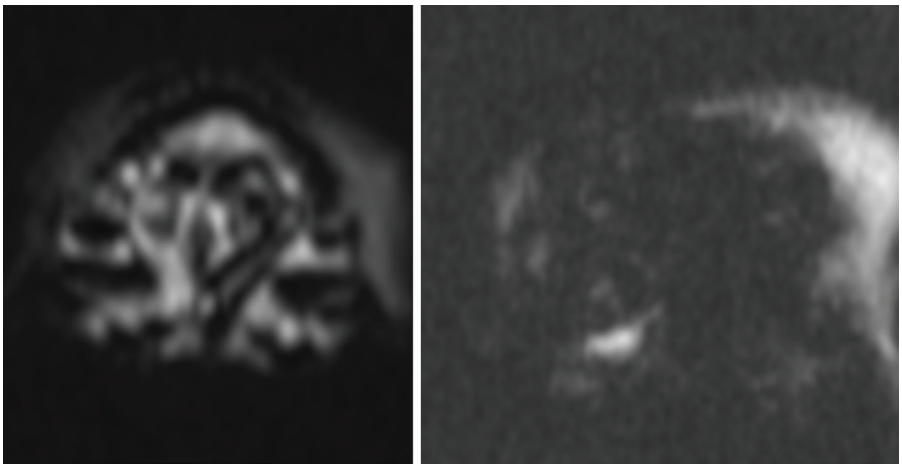


FIGURE 2.31 Transverse (left) and sagittal (right) MRI images acquired using GRE sequences demonstrating the lack of signal observed in part because of the large volume of air/gas present in the scan region. *GRE,* Gradient echo; *MRI,* magnetic resonance imaging.

subcutaneous tissues containing metal can help reduce its effect or move it out of an area of concern. Susceptibility artifacts (Figure 2.31) are enhanced on GRE sequences, and as a result, GRE tends to have more limited use in avian species.

Aliasing as shown in Figure 2.32 occurs when there is anatomy present inside the coil that is outside the selected display field of view. The excluded anatomy may be superimposed on the anatomy of interest or seen along the edge of the image. It is correctable via internal software or by opening out the field of view to include all tissues.

Motion artifact is demonstrated in Figure 2.33 and may occur voluntarily or involuntarily, even under adequate chemical restraint. The most common artifacts seen are eye movement, heart motion, respiratory motion, swallowing, and pulse artifact from arteries. Rapid imaging techniques with or without cardiac or respiratory gating can be used to help reduce this artifact but are challenging to use in avian patients. Changing phase and frequency direction may help move artifacts away from regions of interest.

CONCLUSIONS

Diagnostic imaging can greatly enhance diagnostic capabilities in a multitude of cases. However, appropriate modality selection with optimization of data collection and interpretation is key to gaining as much as possible from each imaging study. In addition, an understanding of the limitations of each modality will help direct the clinician to the most appropriate modality/study for the case in hand. When artifacts occur on diagnostic imaging, an understanding of what may have contributed to them to guide adjustments to resolve or improve them, will greatly enhance the image quality acquired and overall veterinarian satisfaction.

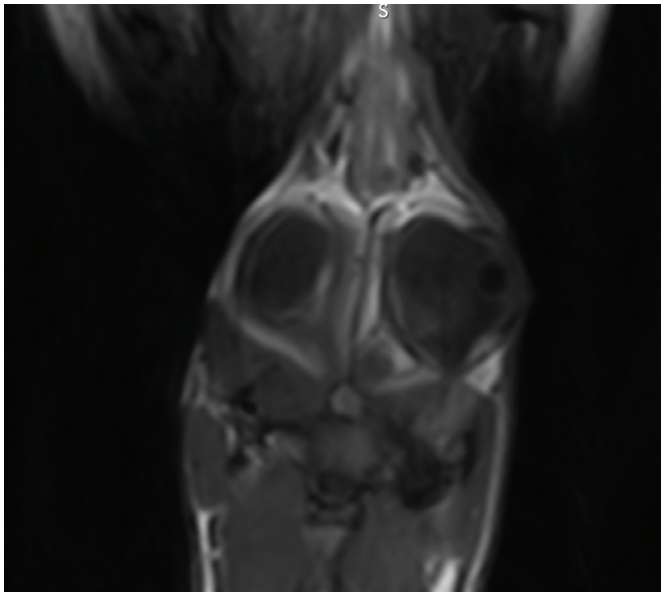


FIGURE 2.32 Aliasing is seen here as anatomy from caudal to the head wrapping around the image and incorrectly superimposed on the rostral head.

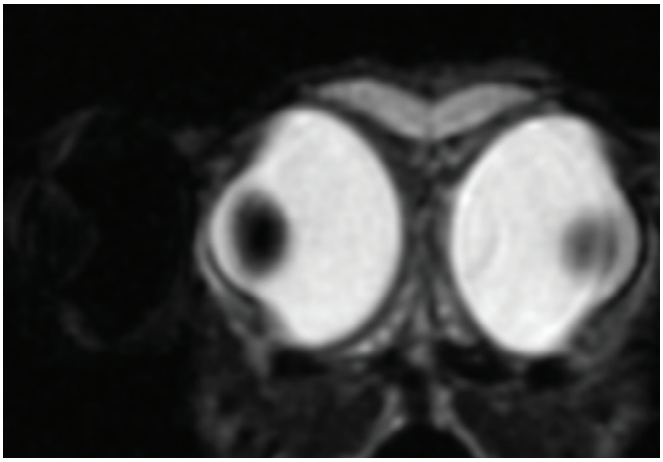


FIGURE 2.33 Motion artifact shown here in the eyes with artifacts both superimposed on anatomy and extending out through the periphery of the image.

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CHAPTER 3

Thermal Imaging

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INTRODUCTION TO VETERINARY AND AVIAN THERMOGRAPHY

Infrared thermography is an imaging modality that measures infrared radiation (IR) and digitally converts it into grayscale images (artificial color palettes are commonly applied for practicality).[1] As any object above absolute zero (0°K, −273.15°C, −459.67°F) emits IR as a function of its temperature, infrared thermography allows to map variations in temperature across an object surface.[2,3] Biological relevant temperatures generate IR in the long-wave wavelength (8–12 μm), which is invisible to the human eye; thus specific thermal cameras are employed.[2] It is a noncontact, stress-free, and safe imaging method that maps heat in real time. Medical thermography is the use of infrared thermography in a medical context. As heat is generated during inflammatory processes and is affected by the amount of regional vascular perfusion (controlled by vasomotor tone), it can be used as a diagnostic aid for some diseases. Medical thermography only measures surface temperature (or a few mm deep); it will therefore be affected by various factors such as insulation, wetness (water absorbs IR and cools surface down, alcohol also decreases surface temperature), emissivity of the surface, and environmental factors (wind and outside temperature).[2,4] Emissivity is an important parameter and is defined as the ability of an object to emit and absorb IR. An emissivity of 1 means the object is a perfect emitter (black body), and the emissivity of biological tissues, including avian tissues, is close at 0.96 to 0.98.[2]

The American Academy of Thermology (https://aathermology.org) has set forth some veterinary guidelines for infrared thermography.[5] Recommended infrared camera for veterinary examination should have a minimum IR spatial resolution of 320 × 240 pixels (ideally 640 × 480), a thermal precision lower than 0.05°C (50 mK), a thermal sensitivity lower than 0.05°C (50 mK), a temperature coverage encompassing physiological temperatures (20°C–45°C: 68°F–113°F), real-time capture capability, and a software for measurements. Thermal camera requirements are similar for research in avian biology.[2] Thermal cameras are considered low resolution at or below 160 × 120, medium resolution at 320 × 240, and high resolution at 640 × 480. The IR spatial resolution is the main factor affecting IR camera cost. High-resolution cameras are onerous ($15k–70k), especially when marketed for medical use. Medium-resolution cameras or low-resolution cameras marketed for nonmedical uses are more affordable and still give images of diagnostic quality in most circumstances. There are also some low-resolution, smartphone-based cameras that may be used. The author has used a low-resolution camera (FLIR E6, 160 × 120) on multiple cases and found it useful for veterinary purpose and general screening as it still allows detection of clinically significant temperature changes. Besides, the investment in a high-resolution IR camera may not be warranted considering that IR imaging is mainly an adjunct diagnostic tool and rarely diagnostic in itself, so the return on investment may be low for avian practices. The main advantage of using low-resolution cameras is their low cost ($400–2000). However, the disadvantages of using them for diagnostic purpose should be kept in mind. A low resolution means that temperatures will be less accurate because temperature is averaged over larger pixel areas. Consequently, tiny hotspots may not be detectable and the ability to discriminate temperature differences will be lower. However, studies in chickens have shown that low-resolution cameras were still providing biologic information close to what would be achieved with high-resolution cameras.[1]

Interpretation of thermal images requires a good knowledge of thermal biology of the species being imaged, scanned anatomical area, and the thermal pattern of common diseases. First, it is important to standardize the scanning protocol (see AAT guidelines)[5] by selecting a constant environmental temperature (20°C–22°C) and limiting the sources of variation (distance from the camera, wetness, wind, airflow). All examinations need to be performed in a thermoneutral environment (indoors preferentially) prior to restraining the bird (if possible) and prior to touching or manipulating the examined surface (to limit thermal transfer). It is also recommended to scan symmetrical areas for reference (e.g., both legs at the same time, both wings). There are several limitations to thermal imaging to be aware of. First, only the surface temperature can be measured (or a few mm below the feathers).[1,2] Therefore, to be detectable, lesions will have to be on the surface and in poorly insulated areas. The accuracy is also dependent on the equipment if one is interested in absolute temperature measurement. The accuracy of most commercial nonmedical infrared cameras is ±2°C (±3.6°F) or ±2% of temperature reading. The threshold of surface temperature increase or decrease that is considered clinically significant will also affect the correct interpretation of thermal images. For instance, the author considered a difference of 5°C to 7°C

between legs/feet, which is likely a conservative cutoff value and significant in most cases. It is also important to be aware that glass is opaque to IR, meaning that animals cannot be imaged in incubators. Birds that have been in incubators for a certain period of time may also undergo surface thermal changes, which may decrease differences between lesions and normal tissues and make it more difficult to identify lesions. Another way that the interpretation of results may be confounding is during a physical examination and touching of the bird which may lead to heat transfer from the handler to the surface of the skin of the bird. Finally, stress may cause cutaneous vasoconstriction that may confound thermographic interpretation, and prolonged handling may lead to patient overheating,[6–8] which may reduce previously observable temperature differences. Consequently, in pet birds, it is best to perform the thermographic examination before restraint, on a calm animal (it can be performed in the dark for instance), and before placing it in an incubator.

Thus far, veterinary thermography has mainly been employed in mammals, including humans, to monitor fever, overall body temperature, local inflammation, circulatory disorders, pain, detection of tumors (which have a higher metabolism than normal tissue and therefore appear warmer), detection of pregnancy in zoo animals, and assisting in the diagnosis of lameness and musculoskeletal stress in horses. A book chapter devoted to veterinary thermography in zoological medicine is available, but it has limited information on birds.[4]

AVIAN THERMAL BIOLOGY AND THERMOGRAPHIC IMPLICATIONS

Avian thermal biology differs from mammals in several aspects; therefore, it is important for veterinarians or researchers who use this technology to be aware of these, prior to using thermal imaging in birds. Furthermore, species-specific differences in thermal biology may lead to misinterpretation of thermographic patterns. This is a significant limitation as thermography may potentially be applied to hundreds of different species of birds. Aquatic species in particular pose increased challenges to the application of veterinary thermography.

Body Temperature

Like mammals, all birds are endothermic and need to keep their body temperature within a limited range. In general, birds have higher core body temperatures than mammals (approximately 1.9°C–2.4°C higher on average than mammals)[9]. At rest and depending on the species, core body temperature is approximately 35°C to 40.8°C (95°F–105.4°F) in most birds with a mean temperature of about 38.5°C (101.3°F).[9,10] Active birds may have body temperatures up to 44.6°C (112.3°F), and with extreme activity, the temperature can climb as high as 47.7°C (117.9°F) in some birds.[9,11] The average temperature depends on the species and other biological variables. For instance, neonates of altricial species tend to be poikilothermic. Ratites and some aquatic species (Sheniciformes, Procellariiformes) generally have lower temperatures than other birds.[12] Contrary to popular belief, birds also experience fever when having an infection, which leads to increased body temperature.[13,14]

Many avian species are not homeothermic, but instead heterothermic (body temperature is not constant). In most bird species, body temperature varies by 2.5°C to 2.8°C during the day.[11] Avian heterothermy is a metabolic adaptation to conserve energy when not active (at night) or when the outside temperature is low (i.e., in the winter). For instance, nocturnal hypothermia is seen in doves, pigeons, turkey vultures (*Cathartes aura*), Japanese quails (*Coturnix japonica*), and many passerines.[12] Facultative hypothermia in small birds in the winter or boreal environment is common.[15] For example, a common North American bird, the black-capped chickadee (*Poecile atricapillus*), is able to considerably lower its body temperature at night in the winter by as much as 10°C.[16] Hummingbirds, nightjars, swifts, and mousebirds go beyond heterothermy and are able to fall into torpor at night decreasing their metabolism and body temperature even further, as low as 4°C to 7°C in some species.[9,12]

Most animals, including birds, have obligate thermal windows (areas that reflect core body temperature such as the eyes or the cloaca) and facultative thermal windows (areas with varying thermal biology such as the feet, face, skin folds, beak, underside of the wings, and so on) (Figure 3.1). The eye-auricular region is the main obligate thermal windows in birds, and thermography of this region has been used to estimate core body temperature in birds.[2]

Generalities on Insulation and Heat Dissipation

Birds predominantly use feathers for insulation. Most of the avian feathered area has a surface temperature closer to environmental temperatures than to core body temperature.[2] The feathering over the body allows to adjust the thickness of the insulating layer by ptiloerection.[11] The amount of feathering, especially for down feathers, is highly correlated to the species' ecology and habitat, and each bird species has a specific molting pattern. Because of this fact, the use of thermal imaging is limited over feathered areas, especially in arctic species for which most of the body and extremities are feathered. However, thermal imaging

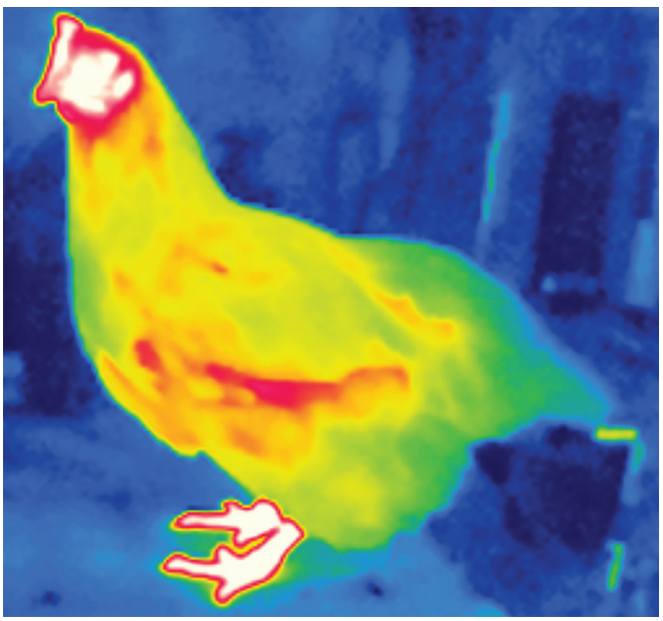


FIGURE 3.1 Normal chicken (*Gallus gallus domesticus*) thermograph. The thermal windows include the head (note the face, beak, comb, wattles, and ear lobes), the feet, and under the wings.

can be used to detect a loss of insulation over the body. Dark feathers and dark feather patterns may also be associated with increased heat detection due to increased absorbance of solar radiation, especially when birds are imaged outside. Birds also shiver like mammals to produce heat. When increasing heat loss is necessary, birds use evaporative water loss through respiratory surfaces, skin, or cloaca. Other species-specific heat dissipation mechanisms include gular fluttering, panting, radiative loss through the beak, and thermal dissipation across areas of bare skin, skin folds, skin protuberances, as well as flippers and feet in aquatic species (see later).[2,12]

Head and Neck

In chickens, the face, comb, wattles, ear lobes, and beak radiate heat directly and may be used for thermoregulation (Figure 3.1).[1] In species with bare head and neck such as ratites, turkeys, vultures, guineafowl, and bald ibises (*Geronticus* spp.), a significant amount of heat can dissipate through these large feather-free areas (Figures 3.2 and 3.3).[2,17–19] In dark birds inhabiting hot environments, such as the bald ibis, it has been hypothesized that the bald head coupled with control of skin blood flow (facial flushing) is associated with the ability to dissipate heat.[20] Facial flushing is also common as a thermoregulatory (and social signaling) mechanism in other bare-faced birds. The lappet-faced vulture (*Torgos tracheliotos*) also has two folds of skin on the neck that are likely used to increase the surface area for thermal exchange. *Gyps* vultures, which have long unfeathered necks, may regulate heat exchange from their neck by changing their neck posture in cold and hot conditions.[17] Some parrots such as macaws (*Ara* spp.) and grey parrots (*Psittacus erithacus*) also have large facial featherless areas that dissipate more heat than other psittacine species (Figure 3.4).

Most birds use their beak as a thermal radiator, and it is particularly marked in toucans and hornbills. The beak morphology and size is not only determined by feeding strategies but is also strongly associated with environmental temperature and thermal ecology in many species of birds.[21–23] Thermal imaging studies in toco toucans (*Ramphastos toco*) have shown that as much as 30% to 60% of body heat is dissipated by blood flow through their large beak.[24] In hornbills, their large bill may also have a similar function depending on species and biotope.[25] This mechanism may be particularly important for species inhabiting hot humid environment, such as toucans, where evaporating heat loss mechanisms are less effective. The role of the beak in heat dissipation is also paramount as a thermal radiator independent of evaporative water loss in species for which water conservation is important.[22] Seabirds with large beaks such as puffins also use their beak as a thermal radiator.[26] Parrots also have large beaks which, while it has not been investigated, may also serve as thermal radiators. In species with a well-developed bill tip organ (such as in Anseriformes), the bill may appear warmer on thermal images (Figure 3.5), which should not be confused with a lesion. Therefore, thermal imaging of the avian beak must be interpreted in the context of individual thermoregulatory status and physiology, especially in species with beak thermoregulatory

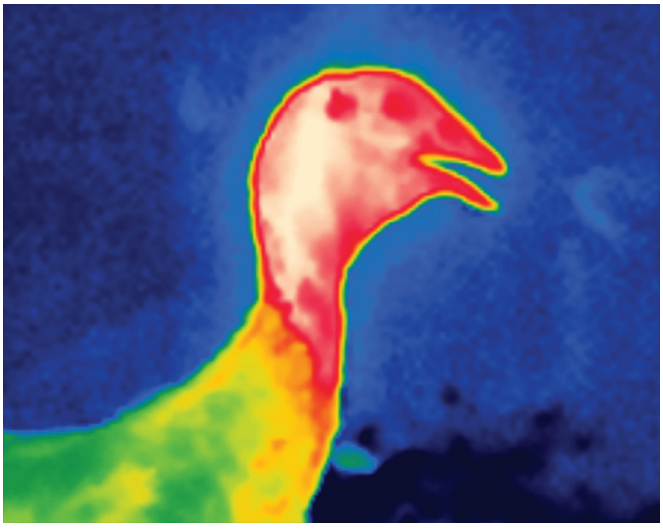


FIGURE 3.2 Normal turkey (*Meleagris gallopavo*) head/neck thermograph. In turkeys, the head and distal neck are featherless and readily radiate heat. This turkey was panting, which is common in meat breed poultry birds that have a low thermotolerance.

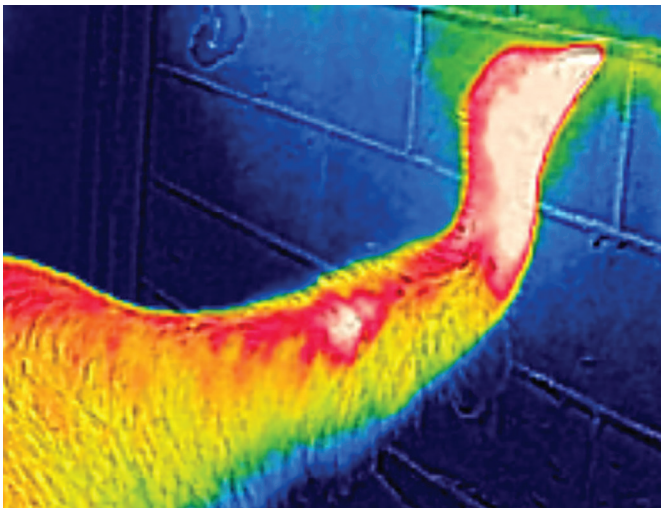


FIGURE 3.3 Normal head and neck thermograph of an emu (*Dromaius novaehollandiae*).

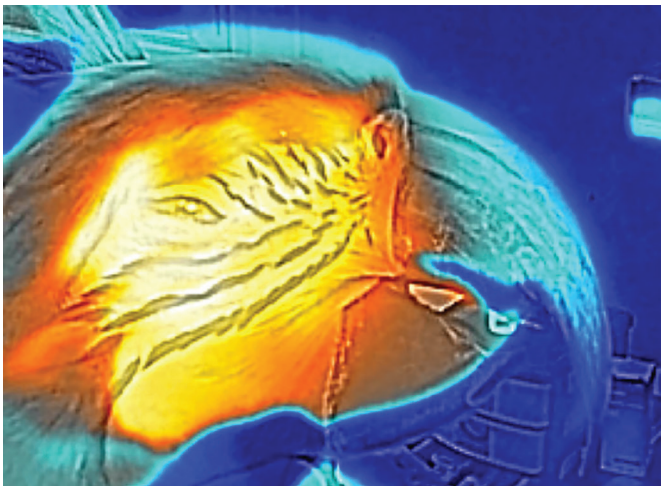


FIGURE 3.4 Normal facial thermograph of a blue and gold macaw (*Ara ararauna*). In macaws, a large featherless track is present on the face.

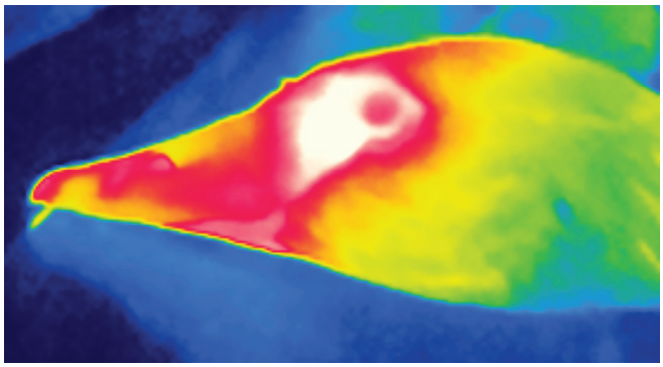


FIGURE 3.5 Normal facial thermograph of a trumpeter swan (*Cygnus buccinator*). Note the bill tip organ and the nasal cavities (*red area* immediately caudal to bill tip organ).

adaptations. The casque present in some species may also have a thermoregulatory function, as demonstrated in cassowaries,[19,27] and potentially in other species such as hornbills.

Gular fluttering, as a method of thermoregulation, is predominantly seen in Pelecaniformes (cormorants, pelicans) but is also seen in turkey vultures, quail, nightjars, pigeons, owls, and mousebirds.[12] In doves and pigeons, for instance, the esophagus is well vascularized to increase the efficiency of gular flutters and upper esophageal pulsations and a subcutaneous plexus of veins are present (*plexus venosus intracutaneous collaris*).[28] Gular fluttering is controlled by movements of the hyoid apparatus and is energetically more efficient than panting.[12] The throat area will appear warm on thermal imaging during gular fluttering in these birds, especially if unfeathered. Gular fluttering may occur concurrently to panting.

The Legs and Feet

As the feet of most birds are unfeathered, they will usually appear warmer during thermal imaging (Figure 3.1). However, a good knowledge of normal leg thermal biology in birds should be known as there are several notable exceptions. First, many boreal and arctic birds have feathered tarsi and toes. For instance, most owls, some hawks and eagles, and grouses have feathered tarsi. Snowy owls (*Bubo scandiacus*) and ptarmigans have feathered feet. Thermal imaging of the legs and feet in these species will therefore be severely hampered. Most birds tend to sleep with one foot tucked inside their plumage (unipedal sleeping/resting), which will increase its temperature and lead to noticeable differences between the feet on thermal imaging.

Furthermore, most birds use their legs for thermoregulation.[11,12,19,29] In numerous bird species, there are extensive counter-current heat exchange systems in the legs and feet (called regional heterothermy) at the feather line to decrease heat loss in water or during low environmental temperatures. This is especially pronounced in aquatic birds, wading birds, gulls, and some northern species, in which the temperature of the legs and feet is close to the ambient temperature. There are two main mechanisms that allow this to occur: in some species such as ducks, geese, storks, cranes, penguins, and flamingos, the heat exchange system takes the form of a rete system of arteriovenous anastomosis, the *rete tibiotarsale*, at the level of the tibiotarsus and intertarsal joint.[30] In flamingos, in which it is the most developed, this *rete* includes more than 60 arteries and 40 veins and is therefore very efficient (Figure 3.6). It is also present in chickens, owls, and a number of short-legged birds, but is a more simplistic system in these species. In other species such as herons, gulls, and other Charadriiformes (most small wading birds), the heat exchange system is not a rete but rather simply composed of counter-current veins along arteries.[30] Species with a rete may be able to control blood flow within the rete, whereas non-rete species do not have such control. Consequently, the legs and feet of these birds may appear cooler in thermal images than the rest of the body and the feet of other birds. Even in warmer conditions, the feet of these birds may be closer to environmental temperature than body temperature, especially in non-rete species.

In penguins and other birds such as ratites,[19] the opposite occurs, where the feet may appear warmer in thermal images, as the feet are important thermal radiators in warm conditions. In addition, some birds such as New World vultures (Cathartiformes) and some storks urinate on their feet as a



FIGURE 3.6 Thermal image of a flock of flamingos. Note that the legs are of similar temperature of the environment due to their highly efficient rete tibiotarsale.

cooling mechanism; this is called urohidrosis.[12] To reduce leg thermal loss, many birds may also assume a squatting position, thereby covering their legs with body contour feathers.

The Special Case of Poultry

Poultry are a special case as they have been selected over decades for increasing metabolism and growth. This came at the expense of thermoregulation and cardiovascular function. Therefore, chickens and turkeys have a low capacity to regulate their body temperature in extreme conditions and have an overall low thermotolerance.[1] Overheating chickens and turkeys typically pant heavily (called thermal tachypnea), which is accompanied by significant evaporative water loss (Figure 3.2). Thermal imaging has been found by the author to have a lower sensitivity for detecting inflammatory changes in poultry for this reason when compared to other species of birds.

Other Adaptations

The females of some bird species develop a brood patch after egg laying that is used for thermal transfer during egg incubation. It is typically located over the ventrum and is an area with reduced feathering. This area is readily visible on thermal imaging (Figure 3.7) and should not be confused with a lesion. Just as some species of birds have arteriovenous anastomosis in the legs to conserve heat, penguins have a similar mechanism in their flippers.

Behavioral adaptations may also be thermally important for some species such as when using communal roosts, huddling (passerines, penguins), and selecting microclimates and shelters. Postural adjustment is also employed by many birds in addition to ptiloerection such as hunching, head retraction (see *Gyps* vultures earlier, but many other birds too), and tucking the head beneath the scapular feathers (as both the head and beak are thermal windows).[12] Furthermore, many birds reduce requirement for thermogenesis by "sunbathing" during which they frequently spread their wings. In addition to reducing ptiloerection and panting, overheated birds frequently slightly open their wings to increase surface area. This behavior is frequently seen in hospitalized birds in incubators when the temperature is too high. Wild birds that need to cool down will also seek shade and water.

AVIAN VETERINARY THERMOGRAPHY

Infrared thermography has not been as widely used in avian medicine yet as it has in mammals. Of the avian medicine articles that have been published, the great majority deal with poultry species,[31–33] aquatic species (penguins, flamingos),[34–37] and birds of prey.[38,39] There is also extensive nonveterinary literature on the use of thermography in the poultry industry[1] and for avian metabolism studies.[2]

In birds, the main indications and reported uses for thermal imaging are listed as follows:

- The use of thermography for unfeathered areas such as the pelvic limbs and the head (in some species): Because feathers cover most of the avian body and thermography only measures surface temperature, this technology does not allow examination of the underlying skin and associated pathologic thermal patterns.
- Feather loss/insulation disorders: A loss in insulation is readily spotted on thermal imaging.
- Flock screening: Individuals within a flock may show different thermographic patterns associated with diseases and lesions.
- Research on avian metabolism, thermal physiology, and in the chicken industry (thermal stress, welfare, building insulation, egg incubation): This topic will not be covered in this publication.

Leg Thermography

By far, the legs are the easiest part of the avian body for using thermal imaging as they are unfeathered in most birds. Furthermore, a range of common diseases occur on the legs and feet such as bumblefoot, wounds, trauma, infection, frostbite, and avascular necrosis. Several studies have investigated the usefulness of infrared thermography in the detection of foot diseases in chickens, turkeys, penguins, flamingos, and raptors.[32,34,35,38,40]

In the poultry industry, pododermatitis is a major health problem and is responsible for significant economic loss. For this reason, several studies have been performed in chickens using infrared thermography. In one study, thermography was used to correlate the development of pododermatitis 2 weeks after thermographic examination (>80% correlation), which was higher

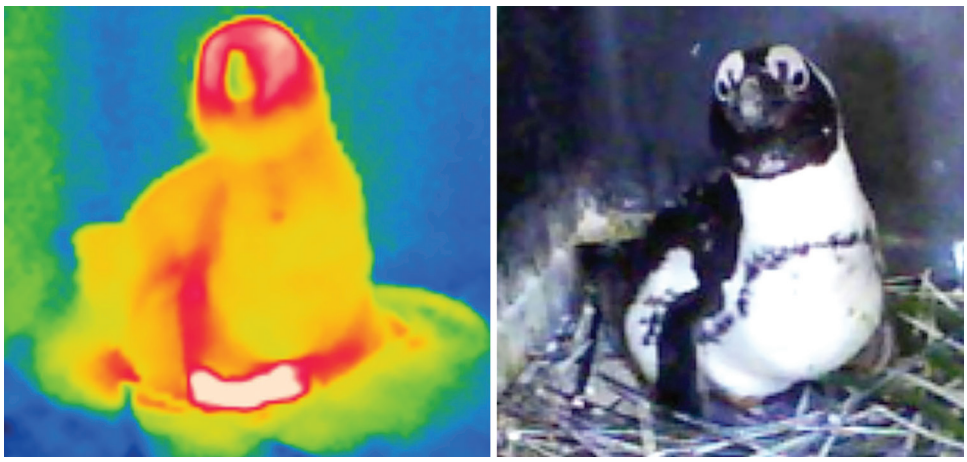


FIGURE 3.7 Black-footed penguin (*Spheniscus demersus*) incubating an egg. Note the increased temperature around the brood patch. Side-by-side thermography and regular image.

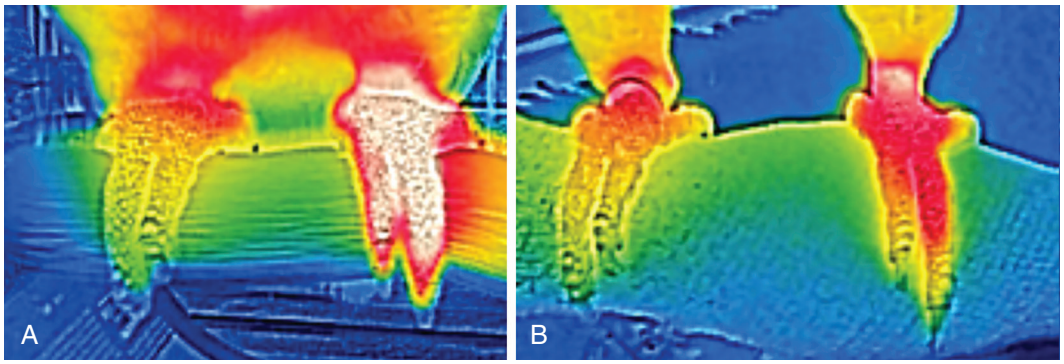


FIGURE 3.8 (A) A grey parrot (*Psittacus erithacus*) that had walked over pieces of glass and had an inflamed left foot; (B) 24 hours after meloxicam (1 mg/kg PO) administration.

than visual inspection alone in subclinical cases.[40] In the same study, bumblefoot was experimentally induced with *Staphylococcus aureus* injection and thermal imaging was also found to be useful in that model.[40] In a different study, still in chickens, the temperature of the plantar lesion was found to decrease with increasing bumblefoot severity because of the development of surface necrosis and scabbing.[32] In turkeys, thermal imaging was found to be a reliable method to detect subclinical footpad dermatitis. However, as observed in chickens, higher degrees of dermatitis were associated with lower surface temperature because of hyperkeratosis or early ischemia.[33]

As mentioned earlier, thermal imaging was expected to be more challenging for the feet of aquatic species because of thermal leg adaptations such as the counter-current heat exchange system and thermal heat dissipators (penguins, ratites, others). As a matter of fact, this was somewhat confirmed in two studies on pododermatitis with mixed results, one in penguins and one in flamingos.[34,35] In both studies, differences were found between feet with active lesions compared with inactive lesions or normal feet, but there was too much variability and overlap between conditions to be clinically applicable.

Information in the literature is scarce on the use of leg thermography in other species, even though they are more commonly seen in practice. A study in raptors found that thermography was useful to detect a decrease in temperature of electrocuted feet over time, which was correlated to the likelihood of avascular necrosis.[39] Another recent study in hawks found that thermographic measurements on the tarsometatarsal region correlated well with the temperature of the plantar surface of the feet.[38] Therefore, this area may be able to be used as a surrogate for inflammation located on the plantar surface (such as with active bumblefoot or wounds) when assessing raptors from a distance.

While the lead author has not published any formal research on this topic, he has extensively used thermal imaging in birds in clinical practice, mainly for conditions affecting the pelvic limb and face. It was found that temperature differences higher than 5°C to 7°C were generally clinically significant. Differences between legs/feet were harder to assess for chickens, as even normal chickens had a tendency to have one leg warmer than the other. However, it was not the case in parrots and raptors. It is also worth mentioning that any difference in feathering between legs or feet will result in temperature differences, reflecting insulation differences.

Inflammatory lesions (infection or trauma) were readily detectable through higher temperatures in a variety of cases such as a grey parrot that walked over pieces of glass (Figure 3.8), active bumblefoot lesions in raptors (Figure 3.9), leg wounds (Figure 3.10) and leg cellulitis in raptors (Figure 3.11), toe wounds (Figure 3.12), and frostbites (Figure 3.13). While some of these diagnoses were still obvious without the use of thermal imaging, what was found to be the most useful with this imaging modality was the ability to monitor the lesion, especially without restraining the birds. The use of antibiotics and/or meloxicam initially, as well as lesion resolution several days later, was associated with sharp decreases in foot temperature (Figure 3.8) allowing to assess therapy efficacy. While the scientific literature had mixed results about the use of thermal imaging for bumblefoot (see earlier), the author has found it useful for differentiating active lesions (active infection, more severe case) (Figure 3.9) from chronic ones (chronic scab formation and low-grade inflammation). Scabs on

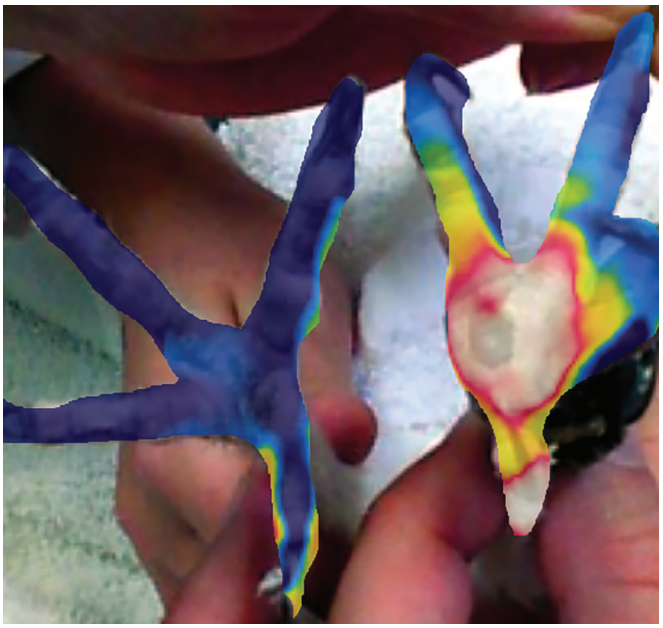


FIGURE 3.9 Bumblefoot lesion in a barn owl (*Tyto alba*). Thermograph overlay on regular picture. The left foot had an active infected lesion that was much warmer than the contralateral foot.

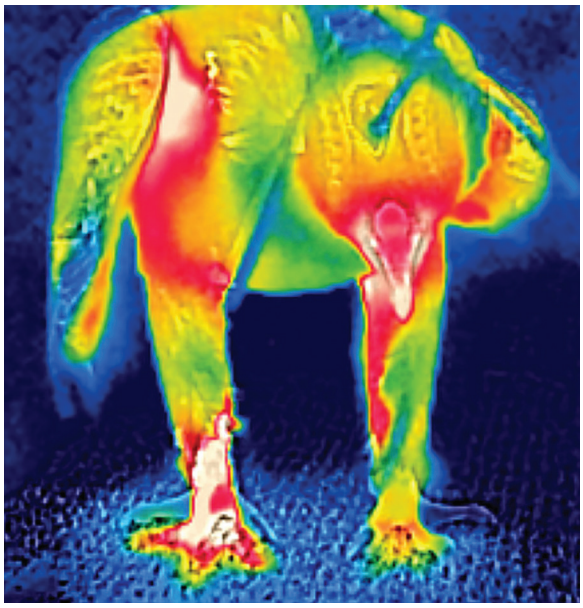


FIGURE 3.10 Golden eagle (*Aquila chrysaetos*) that was imaged in its carrier prior to physical examination. Note the warm left foot. A distal tarsometatarsal deep wound was found on physical examination.

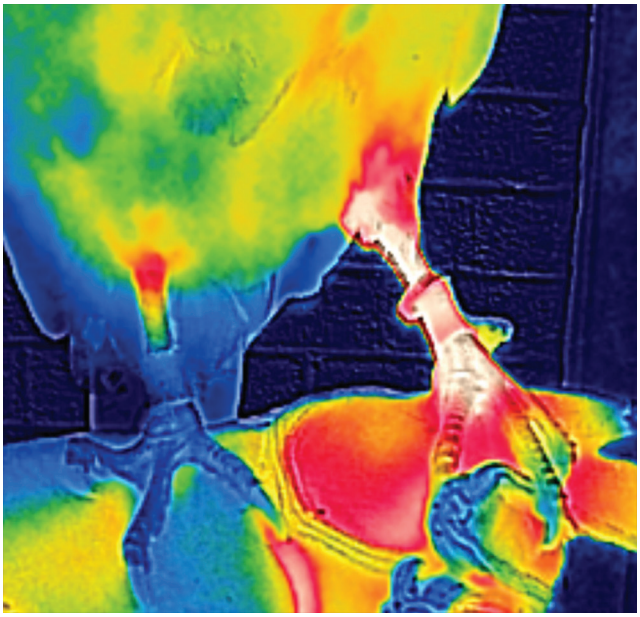


FIGURE 3.11 Cellulitis of the left leg in a Harris hawk (*Parabuteo unicinctus*) presented for lameness. The physical examination and radiographic images of the leg were unremarkable.

the plantar surface of the central metatarsal pad of chronic lesions had a tendency to be cooler than the contralateral foot, as described in the literature. In one case of a unilateral bumblefoot lesion in a peregrine falcon, the lesion was of a similar temperature than the contralateral foot, but after anesthesia leading to a mild decrease in core body temperature, the lesion was evident on thermal imaging. Most frostbite lesions, when mild, lead to an increase in temperature of the affected foot consecutive to inflammation (Figure 3.13). In several cases of lameness or decreased leg

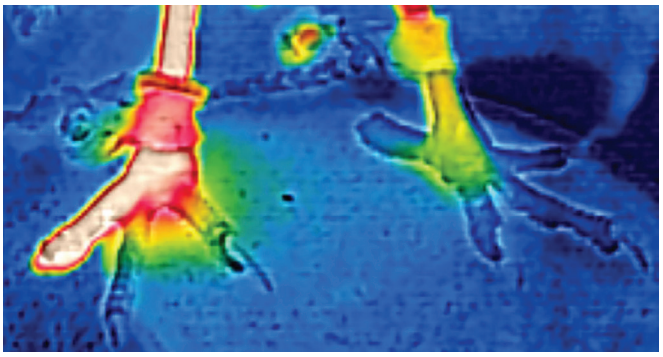


FIGURE 3.12 Distal phalangeal luxation on digit IV of right foot in a Harris hawk (*Parabuteo unicinctus*). Only the affected toe is warm with heat coming with the increased blood flow to the area from the more proximal leg.

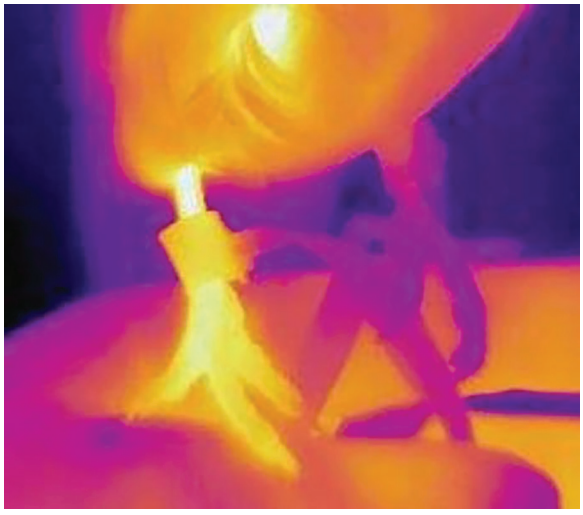


FIGURE 3.13 Frostbite in an American kestrel (*Falco sparverius*). The left frostbitten leg is warmer than the right due to inflammation from the acute phase of the injury.

use in raptors, thermal imaging readily detected increased temperature of the affected leg or foot when the physical examination and radiographs were normal (Figure 3.11). These cases were interpreted as cellulitis (especially with concomitant inflammatory leukogram) or soft tissue trauma. Thermal imaging was found to be useful to assess the evolution of these issues as physical examination and other imaging modalities were unremarkable and could not be used for monitoring.

Several cases of avascular necrosis were also diagnosed with thermal imaging. These were mainly secondary to leg band constriction, other types of constriction (constricting wound, hair, nest materials) (Figure 3.14), self-mutilation (Figure 3.15), severe frostbite, or trauma. The method is particularly useful as it may be difficult to assess tissue viability solely on physical examination. Similar findings were reported in cases of electrocution in raptors (see earlier). In a case of toe infection in an American kestrel (*Falco sparverius*), repeated thermal imaging revealed that the foot first became warmer consecutive to the infection-induced inflammation, but later one of the toes progressed to be cold as necrosis developed, likely due to the subsequent

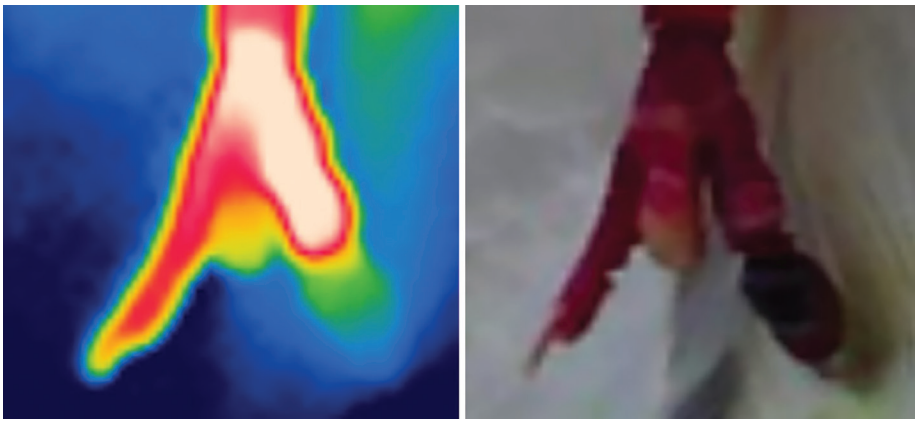


FIGURE 3.14 Toe constriction in a pigeon (*Columba livia domestica*) caused by a constricting wound. Side-by-side thermographic and regular pictures. While no heat from blood flow can be evidenced on the toe distal to the constriction, the proximal aspects of the toe and the leg are warmer indicating inflammation.

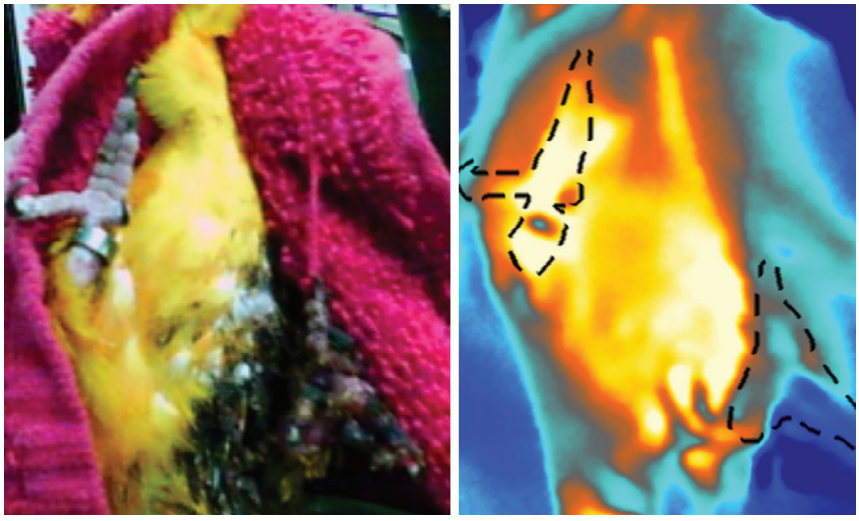


FIGURE 3.15 Avascular necrosis secondary to self-mutilation in a conure. Side-by-side regular and thermographic picture. The feet have been outlined for ease of interpretation.

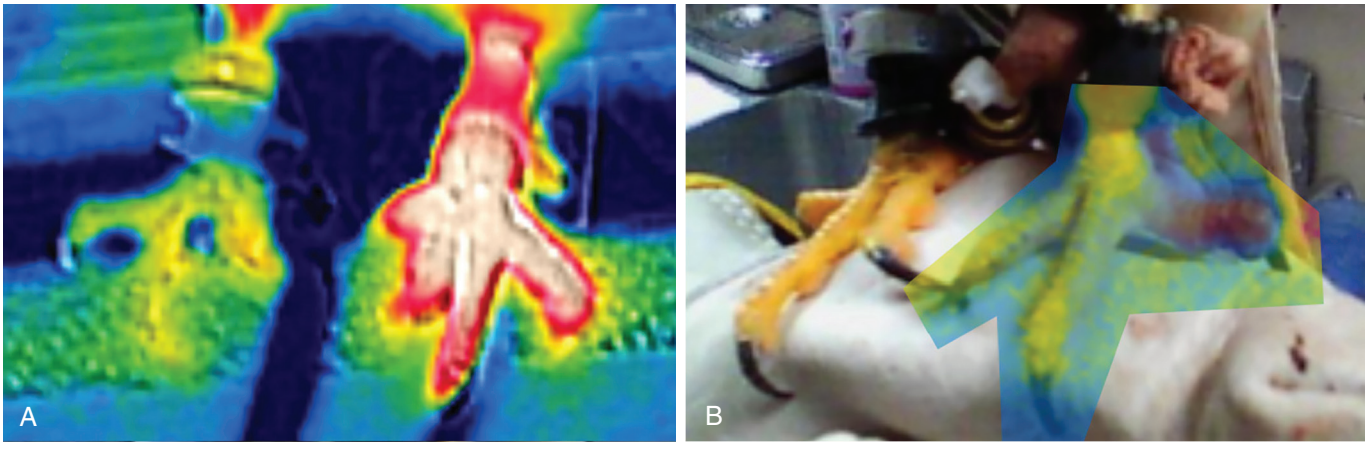


FIGURE 3.16 (A) Foot inflammation likely due to an infection in an American kestrel (*Falco sparverius*). (B) While the foot healed, the fourth digit showed a loss of vascularization likely due to avascular necrosis.

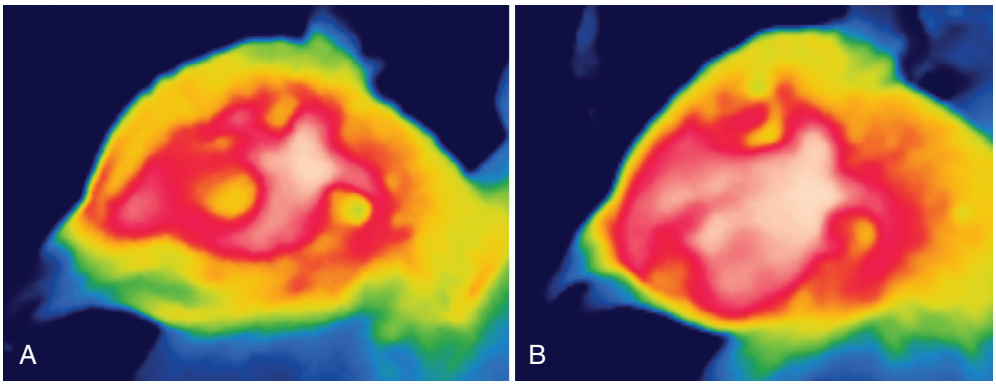


FIGURE 3.17 Peacock (*Pavo cristatus*) with sinusitis. Side-by-side comparison of the normal side (A) and the affected side (B). Note that the affected side was also swollen.

thrombosis (Figure 3.16). This also underscores the usefulness of thermal imaging as a lesion-monitoring tool.

Head Thermography

Head thermography is less useful overall than for the pelvic limb and depends on the level of head feathering as well as the presence of skin folds and facial protuberances (see earlier). Thermography of the head has been used to estimate body temperature noninvasively in several avian species. In chickens, facial temperature measured using a thermal camera strongly correlates with core body temperature.[1] Thus, thermal imaging can be used to detect thermal stress in chickens. In a study in mallards, internal body temperature was estimated by plucking a small area on top of the head and performing thermography on the scalp.[41] In most of these cases, infrared thermography accurately estimated core body temperature within 1°C. In Arctic species housed in zoos in the spring and summer, infrared thermography of the head could also be potentially useful to screen for thermal stress.

In parrots, some species have small bare skin patches on the face such as grey parrots or large ones such as macaws (Figure 3.4). They seem to act as facultative thermal windows in various circumstances in these species. Thus, facial temperatures may be more easily performed on these species. Since the infraorbital sinus in birds is immediately below the skin, sinusitis may manifest as increased temperature in the subocular area. Infraorbital sinusitis readily leads to increased temperatures on the affected side (Figure 3.17). However, reduced feathering and dermatitis on the affected side (from exudate and scratching) may also lead to an ipsilateral increased observable temperature.

As the beak is such an important thermoregulatory organ in birds (see earlier), it can theoretically be used to assess thermal status or stress in birds. In overgrown rhamphothecas, thermal imaging may be used to get a general idea of where significant vascularization stops and may assist in corrective beak trimming (Figure 3.18). However, this should be used with caution as thermography only measures surface temperature. Beak and soft tissue trauma are generally associated with warm lesions on the thermographic images. With beak tip fractures or other fractures, thermography may be used to assess the viability of the distal fragment (Figure 3.19).

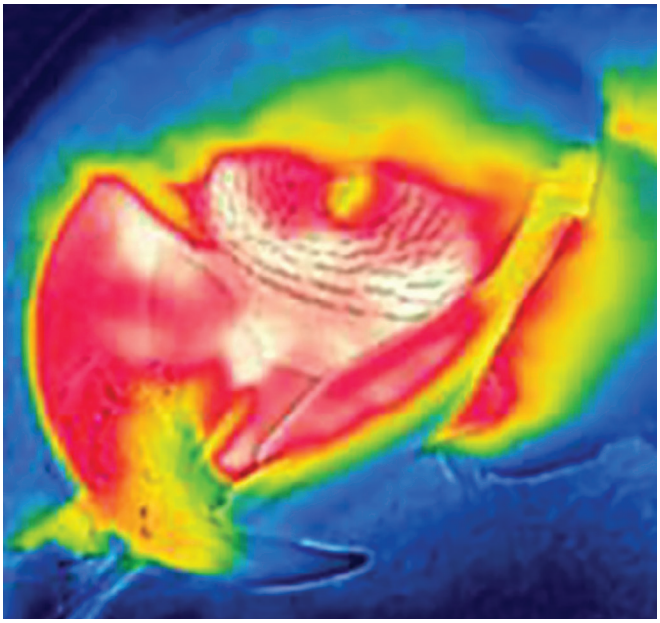


FIGURE 3.18 Green-winged macaw (*Ara chloropterus*) with rhamphotecal overgrowth. See the potential demarcation between the overgrown keratin and the rest of the beak.

Wing Thermography

Wing thermography is not commonly used because of the intense feather coverage. However, the underside of the wing tends to be less feathered and significant heat may be released from that site. It has in fact been used for metabolic studies in flying birds.[2]

Thermography was used on a case report in a Chilean flamingo (*Phoenicopterus chilensis*) with avascular wing necrosis following acute trauma.[36] A thermographic camera was used to image the underside of the wing. Vascular compromise was confirmed with fluorescein angiography. A similar use was reported in a Spanish imperial eagle (*Aquila adalberti*) with wing electrocution and radial fracture.[42]

In penguins, flippers were found to be much warmer in molting individuals.[37]

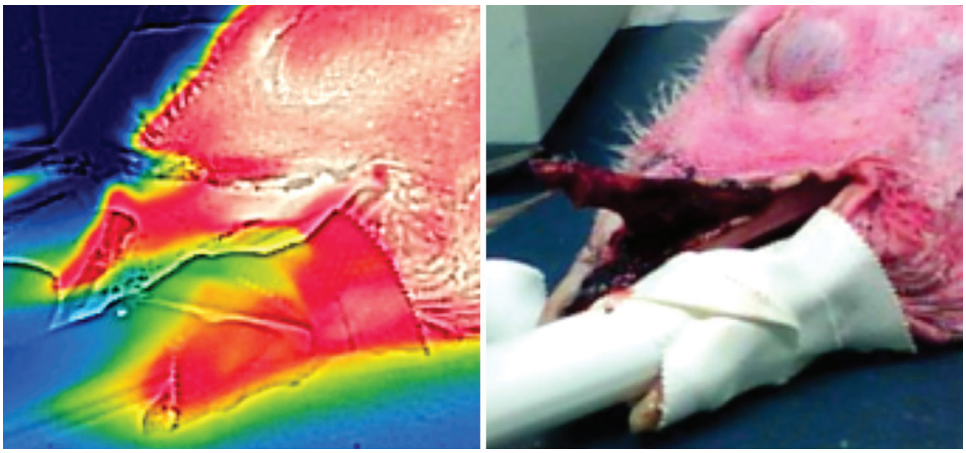


FIGURE 3.19 Turkey (*Meleagris gallopavo*) with beak fracture following an animal bite. Side-by-side thermographic and regular picture. Note that the avulsed part (part of palate with some soft tissue) does not seem to have any vascularization; it was thus excised prior to debridement and reconstruction.

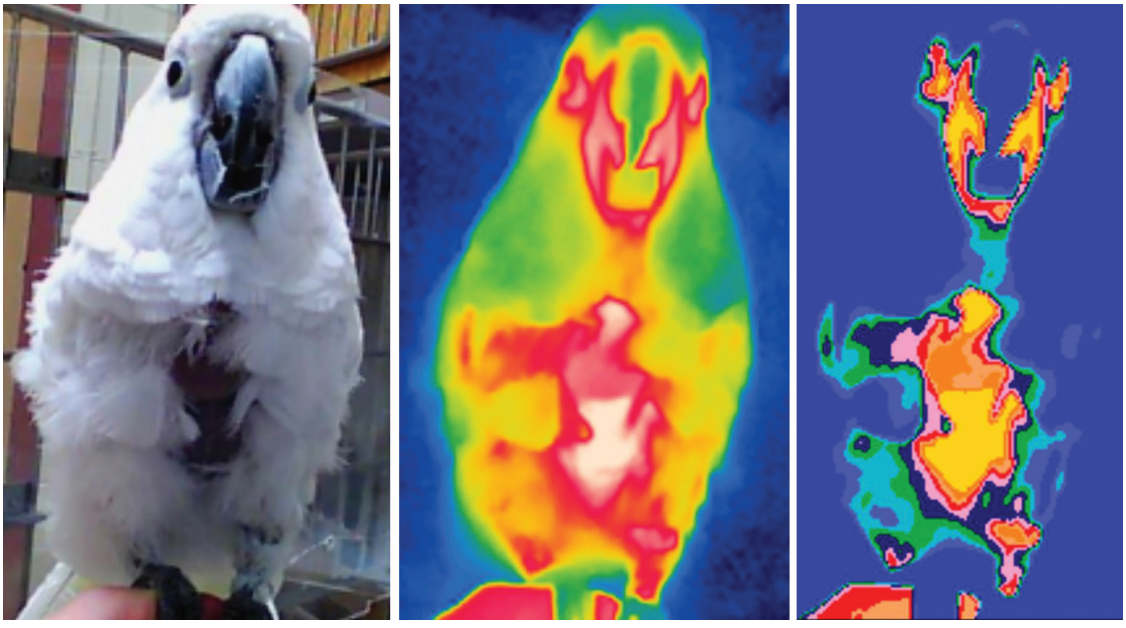


FIGURE 3.20 Umbrella cockatoo (*Cacatua alba*) with mild-to-moderate feather damaging behavior over the pectoral and ventral areas. Side-by-side comparisons of a regular picture, a conventional thermal image, and a thermal image after thresholding that could potentially be used in place of a feather score.

Plumage Thermography

Another indication for infrared thermography in avian medicine is assessment of feather covering and insulation. This modality has mainly been used in birds affected by oil and prior to release, to screen for focal areas of insulation and waterproofing loss, typically associated with residual oil (see Oiled Bird Response).[37]

In parrots, a thermographic picture may be used as a proxy for plumage scoring as hotspots are correlated to feather loss (Figure 3.20). Feather damaging behavior is common in a number of psittacine species and thermal imaging may provide a quick and easy way to monitor progress noninvasively. However, this approach has not yet been compared to plumage scoring[43] and assessed for repeatability. It will also likely have to be standardized across examinations for consistency and comparability. Interestingly, infrared thermography has been used in chickens for that exact purpose. In a study assessing the effect of different housings on feather damage, the feather damage scoring system was replaced by a thermal image of the body.[44]

Oiled Bird Response

The use of infrared thermography for research and response in wildlife exposed to oil, although in its infancy, has shown great promise. The ability to map external heat without capturing the animals is especially beneficial in an oiled wildlife

rehabilitation setting. Since the commercial availability of infrared thermography cameras, they have been used in several research projects evaluating their potential for remote assessment of waterproofing in seabirds and sea otters. The primary cause of acute debilitation and mortality in seabirds impacted by petroleum and other oils is the loss of waterproofing and subsequent heat loss. Removal of oil through washing/rinsing and rehabilitative care can lead to the restoration of the waterproofing properties of feathers, although frequent handling of the animal post-wash to evaluate waterproofing can increase stress to an already stressed animal. In addition to adding stress, assessing waterproofing is time-consuming and can be somewhat subjective. The ability to track the return of water repellency can be extremely beneficial for the animal and for those providing care for oiled wildlife. Infrared thermography can be used to detect, quantify, and photographically display the body's surface temperature from a distance and has the potential to be a more sensitive indicator of waterproofing than behavioral observations alone.[37,45] With large areas of oiled plumage, thermography may detect widespread insulation and heat loss over the body. Smaller areas of waterproofing loss may also be readily detected using this technique (Figure 3.21).

In addition to being used for identifying waterproofing issues, infrared thermography images can also be useful for revealing areas of inflammation and healing in aquatic birds. An infrared thermography camera was used for this purpose to examine the ventral midline incision on a Western grebe (*Aechmophorus occidentalis*) after surgery.[46] The heat loss from this area on the bird (indicative of inflammation) was not evident on day 9 post-surgery when it is evident from a thermograph that the incision site had healed.

Since so little information was available on the effects of chemical dispersants and chemically dispersed oil on bird waterproofing, the Oiled Wildlife Care Network (University of California, Davis) conducted a research study to examine the physical effects of chemical dispersant and chemically dispersed oil on the feathers of the common murre (*Uria aalge*).[47] The main goals of this research were to expose birds to chemical dispersant and dispersed oil and assess structural changes to these feathers (Figure 3.22). Results suggest that chemical dispersants could potentially cause significant waterproofing issues in

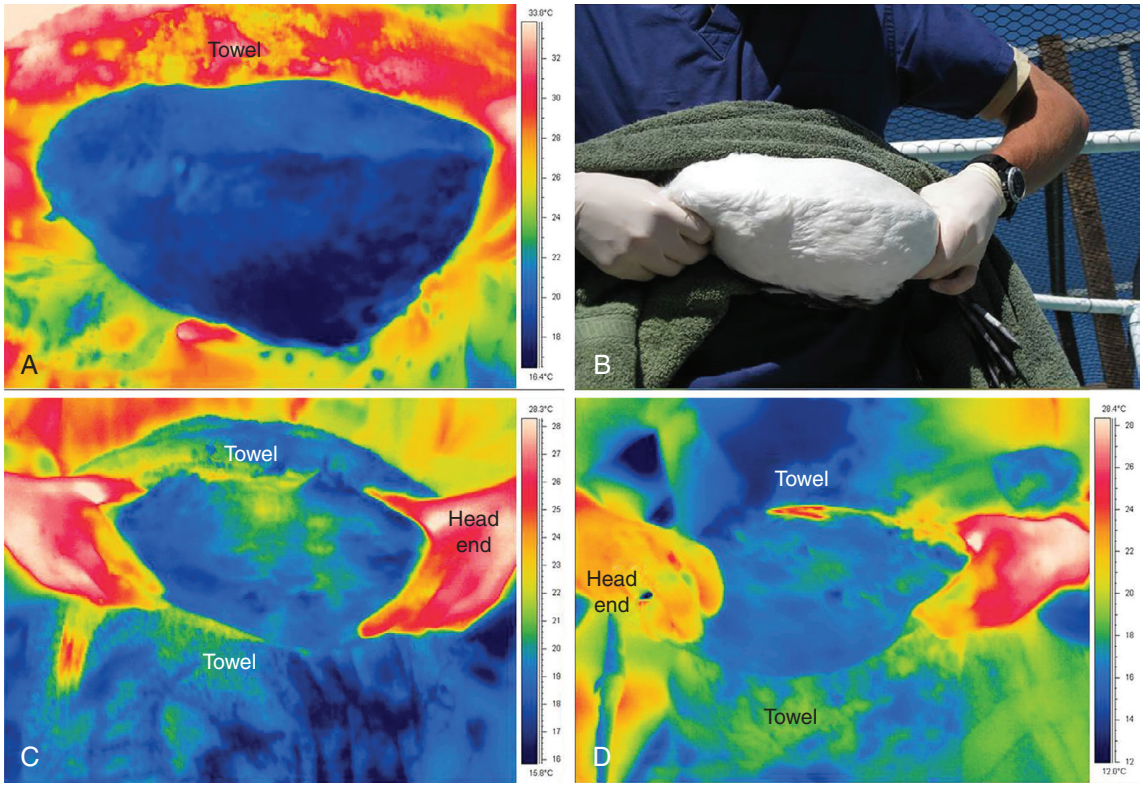


FIGURE 3.21 Assessment of waterproofing on the ventral surface of seabirds during an oil spill response using infrared thermography. (A) Thermographic photo of the ventral surface of a Pacific loon (*Gavia pacifica*) after spending its first post-wash night in a pool. Upon physical examination, no wet spots were found (100% waterproof). (B) Paired digital photo of the bird on photo A being held for an infrared thermographic waterproofing check. (C) Infrared thermographic photo of the ventrum of the same bird during a different waterproofing check. No wet spots were found; the *green areas* on the ventrum are actual residual warmth from heat conductive transfer from the handler's hands during restraint. (D) A different bird, a Western grebe (*Aechmophorus occidentalis*), is being imaged after being in a pool for 2 hours. Warmer spots (*greens and yellows*) on the ventrum matched the "wet-to-skin" areas (nonwaterproof) observed clinically. (Reproduced with permission from OWCN, University of California, Davis.)

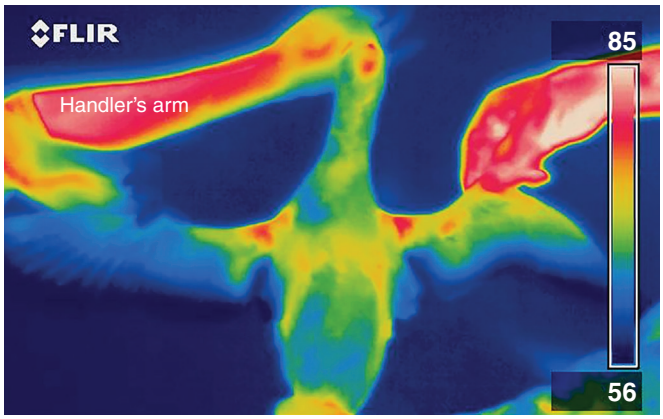


FIGURE 3.22 Thermographic image of a common murre (*Uria aalge*) being examined for feather waterproofing following exposure to chemically dispersed oil. The areas in the image in *orange* and *red* indicate areas with loss of waterproofing (heat escaping indicated by the "warmer" colors).

aquatic birds by disrupting the feather structure. Infrared thermography was used to determine loss of waterproofing following exposure to dispersants and chemically dispersed oil, and the return to waterproofing.

Flock Screening

When imaging a flock of birds, individual birds may stand out because of different thermal signature than the rest of the flock (Figure 3.23). These birds can be further assessed by physical examination without having to physically examine the entire flock. However, molting birds may also display different thermographs than nonmolting birds and individuals may try to dissipate heat for various reasons (after exercise, stress, recent flying, etc.).[37] In aquatic birds, recent swimming may also lead to colder individuals than the rest of the flock. Thus, these differences may not always be related to clinical abnormalities.

Infrared thermography may also be considered useful to screen a flock for thermal stress as it is done in the poultry industry. Conversely, for tropical species housed indoors during the winter, thermal imaging can be used to assess signs of thermal struggle or even the insulation of the building and aviary.

CONCLUSION

In conclusion, while infrared thermography may only have value as an adjunct diagnostic modality, to the author's opinion, it is useful as a monitoring tool for inflammatory or necrotic lesions as well as for monitoring treatment efficacy. For medical thermography of the pelvic limbs, a temperature increase over 5°C appears to be clinically significant. However, the avian veterinarian should be mindful of the multiple thermal adaptations of birds, especially regarding their feet. For these reasons, it is best to perform the thermographic examination in a controlled indoor environment and prior to any stress, restraint, and placing birds into incubators. The use of thermal imaging to monitor feather damaging in psittacine birds and captive flocks of birds should also be further explored.

Disclosure statement: Parts of this chapter were published in the ABVP 2019 symposium proceedings.

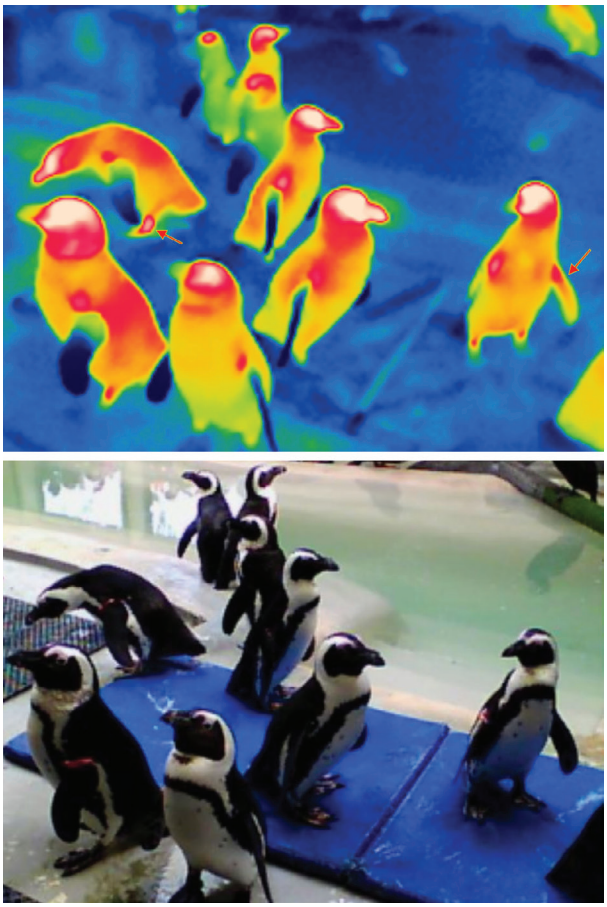


FIGURE 3.23 Thermographic image of a flock of black-footed penguins (*Spheniscus demersus*). Note that compared to the rest of the flock, one penguin shows a warmer flipper and another one a warmer foot *(red arrows)*. Note that the top of the flipper in penguins is always warmer than the body, likely related to a heat exchanger mechanism for the flipper.

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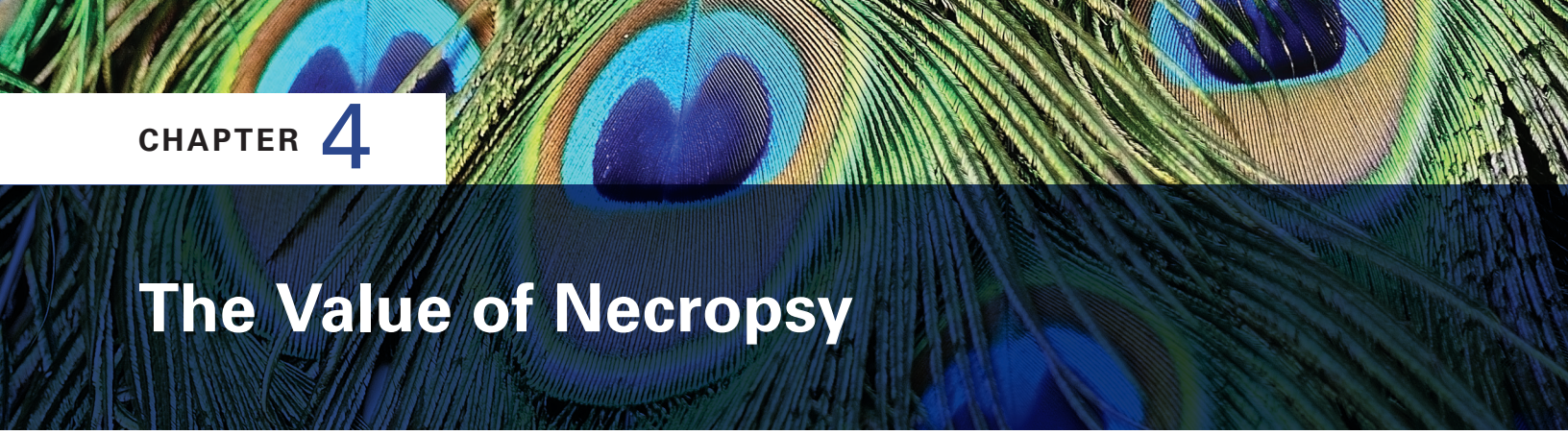
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CHAPTER 4

The Value of Necropsy

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INTRODUCTION

The value of diagnostic testing for the living patient is clear—the results establish diagnosis, direct treatment, suggest a prognosis, and provide information about the patient, contact animals and people, and even overall management. A complete blood count, serum chemistry, and various cytologic evaluations are the foundations of clinical pathology; bacterial and fungal cultures and sensitivities identify pathogens and direct treatment; polymerase chain reaction (PCR) testing is routine for various infectious and parasitic diseases and directs both prognosis and therapy. How can a necropsy provide value for avian clinicians?

The necropsy not only tells us why a bird has died but also defines the extent of the disease, identifies concurrent or predisposing issues, and directs actions to protect or improve the health of cohort birds. Furthermore, the necropsy can provide valuable closure for an owner or keeper as well as the veterinary staff who has suffered the loss of the bird, when it is offered and performed in a caring, compassionate manner. An important component of the process of accepting and processing loss can be provided with a factual understanding of the cause of death through necropsy. Veterinary health care providers can also benefit from this information, and review of their clinical and diagnostic thought processes that were utilized during the provision of health care prior to death. This chapter aims to provide an overview of the importance of necropsy as a tool, and provides important details on how veterinarians can work best with their pathologist to help them obtain the most out of a necropsy.

WHY A NECROPSY IS VALUABLE

There are many reasons for a necropsy to be considered part of complete medical care. The top 10 list is provided in Box 4.1. In essence, these reasons can be categorized into three categories: (1) patient focused when the aim of the necropsy is to learn more about the specific patient; (2) professional focused when the value of the necropsy is most obvious for the professional team; and (3) owner focused when the necropsy mostly aims to help the owner obtain critical information for either legal or management actions.

The value of a necropsy in the diagnostic evaluation of the patient that has died is self-evident. First and foremost, it establishes a cause of death. This examination may be requested from an owner, breeder, or wildlife rehabilitator interested in evaluating a bird found dead. The client may call with questions about what to do after a bird has died. This is an opportunity to suggest a necropsy evaluation. History may be suggestive—something like one or more birds suddenly dying while the owner inadvertently burned something while using a nonstick cooking item. A necropsy can confirm (or refute) such assumptions. Not all birds that are candidates for necropsy examinations die suddenly. A thorough necropsy can also be beneficial in confirmation or refinement of the clinical diagnosis. Clinical pathology may identify an illness as renal or hepatic disease in the live patient. Necropsy can refine and better characterize the type of liver or renal disease, and if there are other organs involved as well. For example, serum protein electrophoresis may show chronic inflammation with a spike in gamma proteins. Such a change can be related to fungal infections like aspergillosis, malaria infections, and sometimes even chronic issues like pododermatitis (bumblefoot). In penguins and birds of prey, differentiating these conditions is important for both individual management and flock management. Identifying comorbidities is another important role for necropsy examinations. It may be clear and evident that a young bird has died from a bacterial infection, but histologic review of the bursa or spleen may identify underlying circovirus or polyoma viral agents that lead to the fatal concern. Such cases are ones where the findings from a dead bird can impact decisions that affect the remaining living birds in an aviary.

Necropsy examinations provide important opportunities for training for practitioners and staff. Consider the opportunities they afford clinicians, technicians, and students. Regular internal examinations on deceased patients create a familiarity with anatomy that is difficult to obtain in any other manner. Regularly examining the internal viscera of various bird species has a direct and significant impact on clinical interventions such as radiology, endoscopy, advanced imaging such as computed tomography (CT) examinations, and surgery. Necropsy examinations provide opportunities to consider anatomy in relation to imaging results. Necropsies should be performed in a manner that helps to improve tissue handling; a valuable opportunity for the clinician with which to develop and hone their skills for the living patients with similar potential pathology suspected to be present. Approaches for interventions including endoscopy, surgical access to the coelomic cavities, and vascular access can be practiced or visualized during a necropsy examination. Suture skills can be perfected.

BOX 4.1 SELECTED REASONS TO PERFORM AN AVIAN NECROPSY

Patient Focused:

1. Determine the cause of death
2. Confirm or expand on the clinical diagnosis
3. Identify comorbidities (concurrent conditions)

Professional Focused:

4. Improve the accuracy of clinical diagnoses
5. Evaluate the effectiveness or medical and surgical interventions
6. Improve knowledge of anatomy

Owner Focused:

7. Obtain documentation for legal or forensic cases
8. Identify emerging diseases/flock issues
9. Screen for zoonotic disease issues
10. Identify and evaluate disease trends and management systems



FIGURE 4.1 At the base of the heart, there is a bulbous dilation of the trachea, the syringeal bulla, at the syrinx in the adult duck *(Anas platyrhynchos)*. The pericardium is mottled with urate deposits.

Even surgical techniques can be practiced without the interference of active bleeding. Every necropsy affords the clinician an opportunity to become better acquainted with the three-dimensional anatomy and anatomical pathology of birds. Anatomical variations such as the syrinx of ducks, the elongated gall bladder of toucans and toucanets, and even the grinding pads and stones found in the ventriculus of the Nicobar pigeon *(Caloenas nicobarica)* can be appreciated and their existence confirmed on necropsy examination (Figures 4.1–4.3).

Occasionally, patients do not respond to case management as expected. In these cases, a necropsy provides information on disease severity, which may explain the response to therapy. For example, birds affected by air sacculitis and/or pneumonia due to aspergillosis commonly require long-term treatment. When these infections are especially severe (Figure 4.4), death can result from tracheal occlusions (Figure 4.5) even if the air sac damage is resolving (see also Section 6: The Respiratory System, and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).

What value can an examination on a dead bird provide to remaining household birds or to the owner/caregiver? If



FIGURE 4.2 The naturally elongated gallbladder of a toucanet (*Aracari* spp.). The mottled appearance to the liver is due to a bacterial hepatitis, but the gallbladder is normal.

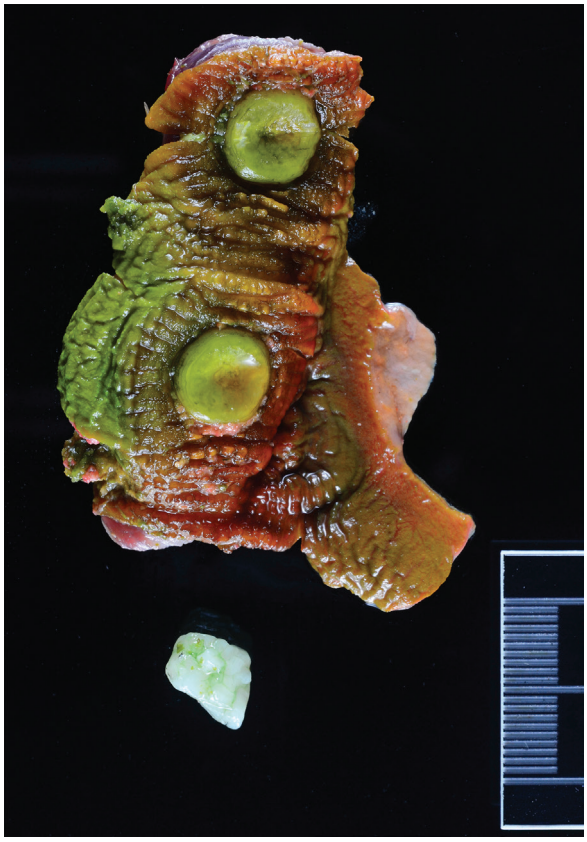


FIGURE 4.3 The ventricular mucosa and grinding stone from a Nicobar pigeon *(Caloenas nicobarica)*. The pads within the koilin are a normal feature in this species. The stone assists in grinding food. (Photo courtesy Disease Investigations, San Diego Zoo.)

infectious disease is a possibility, cohort animals can be treated or managed in ways to reduce the spread of infection. Often, these necropsies include cultures and cytologies collected at the time of necropsy. A case of enteritis or hepatitis could be due to a variety of bacterial or viral pathogens. A simple gross examination will not identify the culprit to a



FIGURE 4.4 Severe clavicular air sacculitis due to aspergillosis in a Magellanic penguin *(Spheniscus magellanicus)*. The clavicular air sac has a thick film of fungus and inflammation. The thoracic air sac has multifocal distribution of fungal plaques.

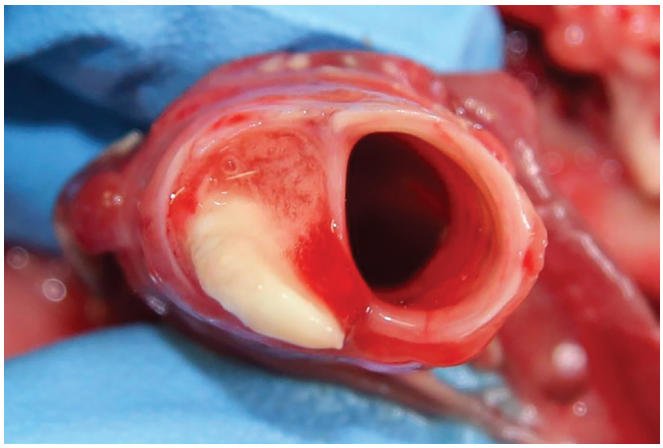


FIGURE 4.5 Cross section of the trachea of a penguin, showing complete occlusion of one of the tracheal lumens. Partial occlusion also occurs.

level of value in addressing the health of poor nutritional condition and contact animals. As a case example, a relatively new bird to a collection died suddenly while being treated for gram-negative enteritis based on fecal cytology. Necropsy with cytologies could show that this bird had a much more concerning coinfection with *Chlamydia psittaci* resulting in classical signs of poor nutritional condition, hepatitis, splenic enlargement with necrosis, enteritis and air sacculitis. As such, the necropsy cytology, resulted in the entire collection being treated for chlamydiosis and prevented a simple infection from becoming a disaster. Similarly, the necropsy results could lead to the owner being diagnosed and treated by their physician as well. Therefore, enhanced diagnostics from the necropsy can be useful to allow quick resolution of a disease and help prevent a large outbreak with spread of the disease to other birds, animals and/or humans.

Zoonotic conditions like chlamydiosis are well known to avian practitioners (see Chapter 18 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Obviously, screening for *Chlamydia* spp. on post-mortem examinations is indicated. In these cases, a positive finding suggests a need to screen or treat cohort birds and people. However, chlamydiosis is not the only zoonotic condition of concern. Other infectious agents such as influenza, West Nile virus (see Chapter 16), *Escherichia coli*, *Salmonella*, *Campylobacter* (see Chapter 17), cryptosporidia (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease), and *Giardia* (see Chapter 23) can impact multiple birds and owners.[1] When these agents are identified, further screening of live birds is indicated for direct treatment. Additionally, owners should be advised to consult their physician if they have concerns about zoonoses.

Unusual mortality events occur in collections and wildlife populations. The typical scenario is that one or two birds die, and then suddenly, lots of birds become sick and/or die. At this time, the clinician can be faced with desperate phone calls from one or more clients. In free-ranging birds, etiologies are often environmental, infectious, or toxic. Emaciation events due to fish population declines are not uncommon in seabirds like puffins (*Fratercula* spp.) and fulmars (*Fulmarus* spp.).[2] "Wrecks" due to migration during bad weather (where hundreds or thousands of birds can be found dead and dying because of uncontrolled landings), as well as massive die-offs due to infectious diseases like salmonellosis, West Nile virus, and avian influenza, or intoxications like avian botulism or ingestion of Avitrol (4-aminopyridine)[3,4] are all well documented. In these cases, it is not uncommon for the clinician to be presented with birds in various stages of decomposition. To best manage this, two or three of the best-preserved (freshest) specimens should be selected for further examination and sample collection while the rest of the carcasses are frozen for later evaluation, if needed. In rare cases, complete documentation of findings in all birds will be valuable.

A published report of lories and lorikeets (Loriinae) with salmonellosis causing an unusual mortality event in a zoo follows this pattern to a tee. First, two birds died. Then over the next 10 days, eight more birds died. Necropsy examinations identified hepatitis and splenitis, bacterial emboli, and enteritis indicative of overwhelming bacterial septicemia. *Salmonella typhimurium*, with the same antibiotic sensitivity pattern, was isolated from four birds. The same pathogen was isolated from a cohort snake and the environment, but not the food preparation areas. The follow-up of removing snakes that likely shed the agent into the environment was important in outbreak control.[5]

In private collections, conditions such as weather extremes and infectious disease concerns like psittacine herpesvirus-1 (Pacheco disease), paramyxovirus-1 and -3, avian polyoma virus in young birds, and chlamydiosis are common agents of unusual mortality events (see Chapter 18, Chapter 41, and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2 Infectious Disease). Toxic conditions like polytetrafluoroethylene (PTFE—Teflon) toxicosis and toxic plant ingestion are sadly still seen in both pet bird and breeder bird collections (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 18, Advancements in Diagnosis and Management of Toxicologic Problems).[6] Some infectious diseases that normally impact only individual birds like aspergillosis can present as unusual mortality events if predisposing conditions affect a large number at the same time. For example, if polar penguins are exposed to warm temperatures, they will often break out with aspergillosis as a colony issue within a few days to weeks. The goal of the necropsy examination in unusual mortality events is to determine both the cause of death and the predisposing conditions or

events that led to the deaths. In doing so, the necropsy is highly relevant to the remaining living birds.

A great example of the value in unusual mortality investigations is an event involving a collection of breeding psittacines in southern California that died from heat exhaustion. While these birds were acclimated to hot temperatures, a mix of high temperatures and reduced aviary shade resulted in seven adult birds dying over a 2-day period. Presumptive concerns were for an infectious disease or a toxicosis. The necropsy examinations showed high internal body temperatures and tissue congestion with nothing more. The environmental history and lack of lesions suggesting other typical causes for sudden death were key to the diagnosis. Shade clothes and sprinklers were installed and the remaining 55 adult birds in this collection survived continued temperature extremes.

Necropsy examinations are not only to determine the ultimate cause of death but also to determine the pathogenesis of disease to address primary problems. For example, many birds that die from acute visceral gout (and presumptive hyperuricemia) have predisposing renal amyloidosis, which may have resulted from chronic inflammation due to initiating conditions such as self-mutilation of pododermatitis (bumblefoot, see also Chapter 33; Figure 4.6). Management changes to reduce the incidence and severity of the initiating concern in the population can reduce or delay deaths.

Not only do thorough necropsy examinations consider how the story of the animal's illness and death evolved, but they also screen for concurrent health conditions that may impact other birds in the household or the collection. For example, nutritional issues such as vitamin A deficiency (identified histologically by squamous metaplasia of glands such as the esophageal glands) or vitamin D deficiency (identified both grossly and histologically by soft, bendable bones with disorganized cellular arrangement) may be present in birds that die for a completely unrelated issue. Regardless, managing these conditions in the remaining birds will elevate the overall health of the birds in the home or collection. Incidental viral inclusions can be identified histologically indicating viral presence in the bird or home or collection. Finding indication of the presence of agents such as avian polyomavirus or avian bornavirus (see Chapter 14 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease) can have an impact on management and/or vaccination considerations and future management decisions for a flock. Parasitic conditions (both internal and external) can be identified via necropsy examinations, and treated accordingly in the remaining live birds (see Chapter 9). While these infections may be incidental, they do reflect health management and may suggest a need for screening or treatment in cohort birds, or changes in management strategies and philosophy (see also Section 4: Infectious Disease Prevention).



FIGURE 4.6 Bilateral ventral hyperkeratosis and ulceration (pododermatitis, bumblefoot) in a gentoo penguin *(Pygoscelis papua)*.

BOX 4.2 ORGAN SYSTEMS AND THEIR REPRESENTATIVE TISSUES

Skin: skin, feathers, conjunctiva, uropygial gland
Nervous: brain, pituitary gland, spinal cord, sciatic nerve, crop or proventricular wall
Cardiovascular: heart, heart valves, major vessels
Respiratory: nares, sinuses, trachea, larynx, air sacs, lungs, pneumatic bones
Gastrointestinal: oral cavity/tongue, crop, esophagus, proventriculus, ventriculus, pancreas, duodenum, jejunum, ileum, ceca, colon, cloaca, vent, liver, gall bladder, Meckel diverticulum
Renal: Kidneys, ureters
Hematopoietic: spleen, thymus, bursa, bone marrow
Endocrine: thyroids, parathyroids, adrenal glands
Reproductive: ovaries, testes, oviduct (multiple levels), phallus
Musculoskeletal: skeletal muscle, bone, joint
Special sense: eye (globe), ears

There are certain cases where you may want to refer to a necropsy examination instead of performing it yourself. Legal cases associated with animal abuse, injury, or death, and concerns over malpractice are unfortunately common. These examinations, their write-up, and subsequent follow-up are often extensive. Working closely with an avian pathologist who can perform or suggest board-certified specialists for these types of forensic examinations is very valuable. Options for board-certified avian pathologists include state diagnostic laboratories and a few commercial pathology services. If you must complete the examination, you must focus on thorough, detailed documentation and extensive tissue collection. Box 4.2 lists sampling for extensive histopathology. The table is designed to assist as a reminder for day-to-day examinations. The list can be used as a check off list to identify what samples are collected and which are submitted for histopathology. In cases with legal concerns, collecting as many of the listed organs as is feasible is recommended. If you are aware of (or suspect) legal issues related to a case that you cannot refer to for some reason, it will be valuable to identify an hourly rate upfront for follow-on work after the necropsy is completed.

LOGISTICS: ASKING, SCHEDULING, AND CHARGING FOR NECROPSY EXAMINATIONS

Many veterinarians feel awkward asking owners about a necropsy. Hesitancy can result from unease with adding to the bill for a patient that has died or worries that a necropsy will provide information that was not obtained or uncovered during live animal care. Occasionally, owners are uncomfortable

with the carcass being cut open. The death of a bird can be an emotional time. Neverteless, the multiple levels of value from necropsy examinations should help to overcome these hurdles. Just as it may once have been difficult to suggest interventions like biopsies and advanced imaging, post-mortem examinations should be considered a regular part of complete standard of care provided to avian patients. Given that avian practitioners are often working with wildlife, multiple-bird households and collections, and new pet owners, the necropsy may indeed be the most valuable diagnostic investigation to offer. Post-mortem findings can be critical to managing contact birds, avian collections, and even humans.

First, set expectations ahead of the death of a bird. Many practices use new pet/new client onboarding to present information of the diagnostic opportunities for the practice. Sure, you may want to tout your new digital radiographic imaging, surgical equipment, or advanced imaging that is available. Why not also include a paragraph about complete examinations including necropsies so that clients understand that this is a diagnostic service that your practice provides? Explaining the value of a necropsy is much easier when it is a discussion about a distant future event in the face of a healthy and thriving pet. Necropsy examination results can provide a sense of closure. It can reassure the client and clinician that efforts were well spent. Most importantly, it can provide information that may benefit remaining birds or sometimes even people at home. If owners are concerned about desecration to the body, reassure them that a cosmetic examination can be performed with a single, long incision that is subsequently sutured closed. With all of this value, and all of these solutions, does it not make sense to recommend a necropsy?

Once you define a necropsy examination as an examination, scheduling becomes easier. Do not try to sneak a peek during your lunch break. Doing so shortchanges you, the examination itself, and your lunch hour. You are charging for the procedure; schedule it as you would any other procedure. Time in the schedule for necropsies should be scheduled just like it is for surgeries. Many clinicians prefer to perform necropsy examinations at the end of the day to manage potential spread of disease. Fair enough. If you save the last 30 minutes to an hour of the daily schedule for necropsy examinations, you will have time to do the job well.

Like surgeries, you need to charge for both the procedure itself and for tests—your time and knowledge have a value (see also Chapter 1 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 24, Practice Management and Risk Management). After working hard to treat a bird, death is an unwanted result. If you view it as a failure, you will miss opportunities to enhance your abilities and to provide the top level of care. You will miss an opportunity to understand more fully what happened in the case. Necropsy examinations can be billed as surgeries—there may be a cost based on time or a flat fee based on the procedure itself. If the necropsy also includes cytologic evaluations, cultures, and molecular diagnostics, these should be charged at clinical examination rates. Once a necropsy is incorporated into care as though it were a standard examination, it becomes a standard examination.

Occasionally, you will have a strong reason to perform a necropsy, but a client who is not willing to pay for the service. Some clients will provide permission but only if the examination is at no cost to them. The next step comes down to your investment in yourself. Will you perform the examination without pay? If yes, will you let the owner know what you find? These are difficult questions that need to be based in the philosophy of the practice. This is a great time to establish a theoretical "personal investment account." Your time is free to you—but we all only get so many hours in a day. Are you willing and able to invest some of your time to better understand this case? Post-mortem examinations often need follow-up like tissue histology, cultures, and cytologies. These evaluations have real costs associated with them. Before progressing with a necropsy, it must be determined who will pay for these costs. By keeping track of the costs associated with these educational opportunities, they become more like small professional workshops. Do not expect your diagnostic laboratory to underwrite these evaluations. They usually will not. But your pathologist may be willing to discuss your gross findings and images to help with interpretation. We will explore this further in the section How to Get the Most from Your Pathologist. No matter how or why you approach a necropsy, first and foremost, *get permission in writing from the owner*. Informed consent is required for post-mortem evaluations, just as it is for any procedure for a living patient.

GETTING THE MOST OUT OF A NECROPSY

The techniques of a necropsy examination are well described and often included in the vet school curriculum.[7] Rather than focusing on these techniques, this section provides a few tips that will make your necropsy examination more satisfying and successful.

A necropsy should always be performed in a specific area that is well lit, removed from the general patient traffic, and regularly cleaned and sanitized. Many practices have a small necropsy area in a storeroom near cadaver freezers. These areas, while remote from living patients, are often poorly lit and not pleasant to work in. Alternatives must be identified. If a necropsy examination is analogous to a surgical procedure, the area should be comparable. An isolation area treatment or examination room works well. In many practices, a corner of the regular treatment room is used. In any case, the area should be designated as the necropsy area and stocked with tools and supplies to reflect the work. There should be a counter or cabinet containing tools and supplies as well as materials to record notes—either paper or a computer or tablet terminal.

Basic necropsy tools should not be derived from tools that are considered too dull to serve in surgical packs. The tools that are considered essential for performing an avian necropsy are few. A basic inventory is listed in Box 4.3.

Having a good sterile set or two will make it much easier to perform necropsy examinations. Consider having specific

BOX 4.3 STANDARD NECROPSY TOOLS

Small Birds	Medium-to-Large Birds
1. Iris scissors	1. Poultry shears
2. Jewelers' forceps	2. Scissors
3. Iris forceps	3. Scalpel blade and handle
4. Magnification	4. Thumb forceps

Modified from Schmidt R: Forensic necropsy. In Speer BL, editor: *Current Therapy in Avian Medicine and Surgery*, St. Louis, 2016, Elsevier, pp 769–775.

necropsy examination packs to assure that the tools you need are all set out before the examination. Adequate forms, writing utensils, or access to a computer terminal to enter data should be readily available in the necropsy area. In addition to the tools for necropsy, the area should have adequate cleaning supplies and bags (and labels) for carcasses after the examination.

No tubes, bags, or swabs should leave the necropsy area without a proper label. Every label should have the patient identification (ID), the organ/contents, and the date. This rule will save you hours of confusion, investigation, and possible sadness. You may think you will remember later what went into that tube. You likely will not. A downside of performing necropsies on the fly or as the last examination of the day is the tendency to shortcut steps. Labeling is not a worthwhile shortcut.

Necropsy is about more than collecting tissues in formalin for histology. Tissues and fluids should also be collected and frozen for potential follow-on examinations such as microbiology and toxicology. Recording notes soon after the examination is important. As with surgical notes, the nuances of the procedure can easily be forgotten. Necropsy notes should include both what was seen and what was collected. It is often helpful to review the necropsy images to confirm that all of the important findings are recorded.

TRICKS TO SIMPLIFY THE NECROPSY PROCESS

Although we are all taught the basics of a necropsy in vet school, multiple avian necropsy protocols are available in the literature. However, there are a few simple tricks that will make the job easier and improve results. These tricks are generally not highlighted in the standard "how to" documents. In addition to the standard avian necropsy, evaluations on eggs and babies are slightly different. Information on those examinations can be found in the following section: Examinations of Eggs, Embryos, and Babies or Nestlings.

Standard tool packs or an image of the standard tools and supplies for the examination should be readily available in the necropsy area. Placing a copy of Figure 4.7 in the area gives a simple visual reminder of the complete setup. Many sections of the examination can be performed by support staff. Training a technician in normal avian anatomy will permit the clinician to evaluate the carcass, perform the initial examination, and indicate the tissue collection and sampling plan. The educated technician can finish the examination. Establishing a necropsy workflow plan like this is like managing clinical examinations. The trade-off for this sort of time management is that the ancillary benefits like tissue handling and surgical approach experience are not realized by the veterinarian.

Imaging may be helpful in evaluating a carcass prior to the direct examination of the body. Consider the value of radiographs in documenting injuries such as fractures and metal foreign bodies and directing where to search for items. If ultrasound or CT technology is available to your practice, images can be helpful, not only for evaluating patients but also for developing interpretive skills in this area. A necropsy examination provides a rare opportunity to check your answers in real time.

Because the feathers of birds can contaminate the internal tissues, control is critical to necropsy success. Feathers can be plucked from the carcass to provide a clean skin surface. Alternatives to plucking include wetting the feather coat with water and a small amount of dish soap to permit separating the feathers along feather tracks and reducing the number of downy and semiplume feathers floating about during the examination. The addition of soap helps to control the natural oils and powder down on the feathers that would otherwise repel the water. If neither plucking nor wetting is a realistic option, the body incision site can be managed with a layer of K-Y or ultrasound jelly to separate feathers from the incision area. In a cosmetic necropsy, the jelly can later be removed



FIGURE 4.7 Basic tools for a necropsy examination. Using sharp, dedicated tools will simply improve the experience of the necropsy. Have materials for sample collection, labeling, and taking notes. Not pictured is a camera, a critical tool to ensure that important findings are adequately captured.

with water and the feather coat blown dry with a hair drier to reduce incision visibility.

A picture is worth a thousand words. To get the most out of necropsy images, they need to document lesions and capture all the important findings in your examination. Having an assistant take images during your examination eliminates the need to glove and unglove repeatedly throughout the procedure. Take images with and without size markers. Take images with a wide angle and a close-up perspective. Images can (and should) be deleted so that you have an accurate but not overly extensive bank of images from each necropsy. However, if there is any possibility of a legal issue related to the case, save all images—you can always pare the number down in the future. In the end, you want to capture any identifying features, bands, tags, scars, or injuries. Weigh and photograph all patients prior to the examination to record the patient condition—both nutritional and post-mortem. Nutritional condition is often best captured after feathers have been removed or wetted to better expose the keel. Necropsy images may include a ruler for scale and should regularly have a marker of the patient ID to assure that images can be linked back to the patient. The scale/ruler should be at the margin of the image, not in the center covering tissues. In gross images (such as the whole coelomic cavities), the organs can be used for relative size indicators. If a ruler is not readily available, a known-sized object such as a pen or a coin can serve as size markers. As a rule, it is helpful to take images of lesions in situ and then of the affected organ or organ system alone. If images might be used for presentations or publications, think about how they best illustrate the lesion of interest. Often, including an image of a lesion as seen helps with the clinical correlates for radiographs or palpation. A second image showing the inside of a change then clarifies the finding. In Figure 4.8, a ventricular foreign body is imaged as seen on first examination and then after the ventriculus has been opened. A smooth, neutral background is a good start for compositions. While image background can now be manipulated with processing software, reducing background distractions and glare makes medical images easier to work with in the long run.

You will get most out of images without distractions like blood, feathers, fingers, or foreign objects. However, if coelomic cavities are full of blood, obtain an image of it before removing the offending material. When the cause of death is something like an aortic rupture, the coelomic cavity accumulation of blood tells the story. Similarly, peritonitis from omphalitis is a messy presentation. Before cleaning up the opened intestinal peritoneal cavity, consider what the findings are showing you. If tissue needs to be retracted to better show a deep lesion, use the necropsy forceps or scissors as a retractor. If you must have hands in your image, try to be sure that they are sporting clean gloves. Performing necropsy examinations on a table with integral plumbing makes spraying away blood or intestinal content much easier. Paper towels can be used to blot organs to remove excess blood or debris. Images of the intestinal mucosa should have as much ingesta and blood removed as is feasible.

If clinical radiographs identified pathology, a whole-body image or image of the affected site dissected that illustrates the change is often very helpful. For example, air sac thickening is a common change in cases of air sacculitis from aspergillosis. Post-mortem images of the air sac wall help to illustrate the changes identified on clinical imaging (Figure 4.4).

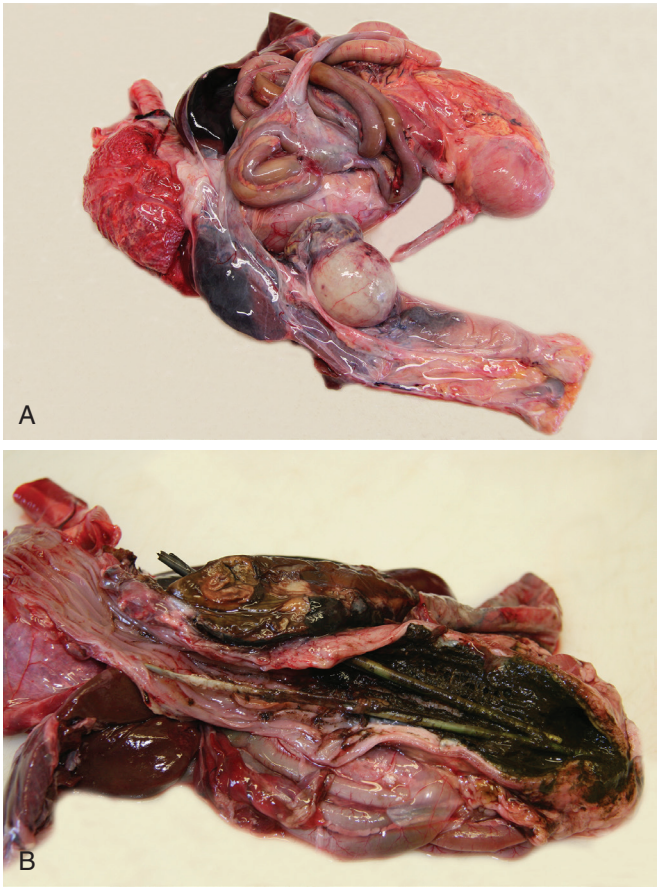


FIGURE 4.8 (A) Viscera from an Adelie penguin *(Pygoscelis adeliae)* demonstrate a linear protrusion from the ventriculus. (B) On cut section, there are feather shafts coursing through the ventricular wall. Inflammation is closely associated with the intestinal peritoneal cavity location of the shafts.

Certain organs are easiest to evaluate at early in the examination or with a specific approach. Upon opening the coelomic cavities, examination of the gonads and adrenals is easiest if performed early in the gross examination. The author recommends collecting the adrenals, gonad, and cranial kidney in one small section. The spleen in some birds can be very small and difficult to locate unless enlarged. In these species, identifying the spleen early in the coelomic examination is essential to organ identification. For example, penguins have a notoriously small spleen. In this species group, the author recommends opening the intestinal peritoneal cavity and reflecting the proventriculus and ventriculus to the left to identify the gastrosplenic region as the first step in the gross examination of the coelomic cavities. If the spleen is very easy to identify in penguins, it is enlarged. In young birds, locating and examining the bursa and thymus should be an early part of the examination to assure that these elusive organs are not forgotten. The thyroids and parathyroids are generally small. They are easily located along the great vessels at the base of the heart when the sternum is first reflected (Figure 4.9). Because they can be more difficult to identify once the heart has been removed, these organs should be identified and sampled prior to removal of the heart.

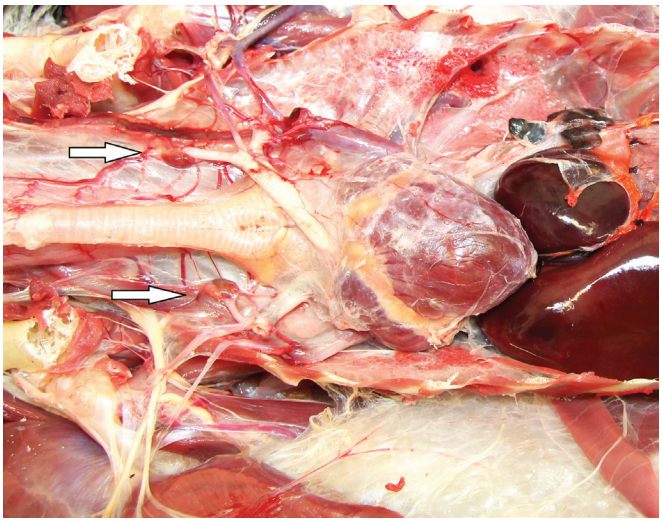


FIGURE 4.9 Thyroids from a California brown pelican *(Pelecanus occidentalis)*. The organs *(arrows)* are easiest to identify before the heart is removed.

The isthmus between the proventriculus and ventriculus is a common site for ulceration, neoplastic proliferation, and growth of some bacteria and fungi. It is therefore better to submit the proventriculus and ventriculus as a unit rather than as two separate samples to facilitate review of the isthmus. Sinuses are the final area where knowing a trick can make necropsy sampling more productive. In passerines, the best approach to sinus evaluation is to slice the head longitudinally and place the half head (brain included) directly into formalin. In psittacine and larger birds, the best sinus exposure is accomplished by a cross-sectional cut at or just behind the nares to expose the bony structures of the sinus and associated soft tissues. This cut is especially helpful in evaluating cockatiels *(Nymphicus hollandicus)* with clinical lockjaw (see Chapter 31 and Chapter 41). Small tissue samples have a way of getting lost in the formalin. Just as you might do with a biopsy, place small tissue fragments into a cassette to keep them more easily identifiable.

A necropsy examination may include sampling for microbiology. Collecting sterile swabs of organs and lesions is not difficult. If the organ of interest is exposed in a clean manner, direct collection with a needle or swab is feasible. When contamination is a concern, one additional step can make a big difference in getting pure cultures. A small spatula and a crème brûlée torch (Figure 4.7) provide a quick and simple way to sear tissue surfaces. Those preferring not to acquire veterinary tools at the cooking store can use a propane torch for the same purpose. The spatula is flamed with the torch until hot. The hot flat surface is pressed against the tissue to kill surface contaminants in a small section. For sampling soft solid organs like the liver and lungs, swabs can be directly plunged into the tissue in the seared area. For tougher organs like the heart and intestines, a scalpel blade can be used to cut the surface so that a swab can be introduced in a sterile manner.

A shortcut to get quick answers from a necropsy is sometimes tissue preps for cytology. Tissues such as lung, liver, kidney, and spleen can routinely be blotted to remove excess blood and touched to a slide surface (Figure 4.10). One

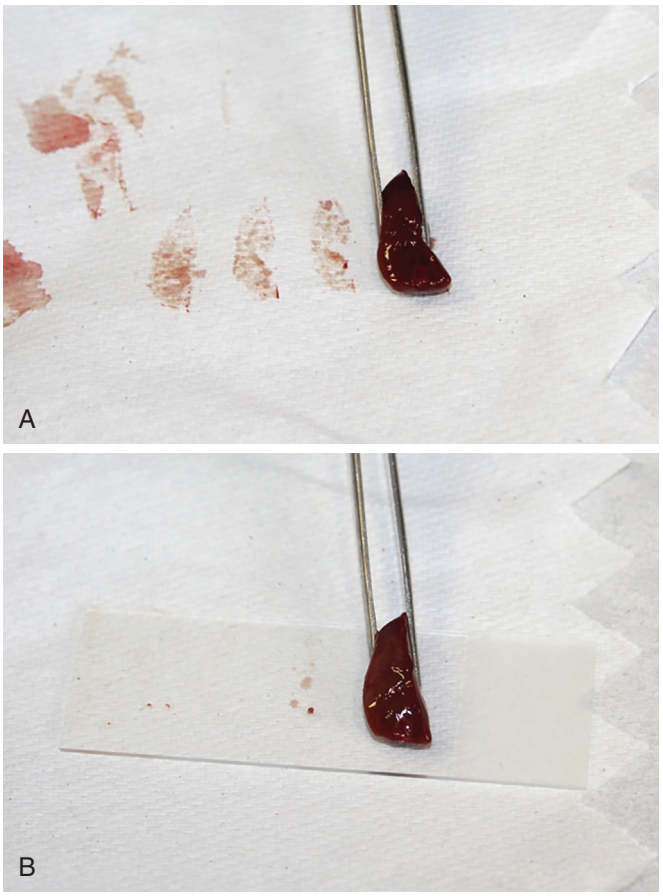


FIGURE 4.10 Touch preps for cytology. (A) Blotting tissue until very little blood remains. (B) Simple touch to a glass slide for cytology. By preparing slides with liver, lung, kidney, and spleen, each slide represents four major organ systems for a quick microscopic examination.

slide can be used for the entire organ set. If three slides are produced (each with the set of four organs), these can be stained with a modified Wright stain (Diff-Quick), Gram stain, and acid-fast or Giemsa stain (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 13 Advances in Clinical Pathology and Diagnostic Medicine). Some protozoal infections such as coccidiosis and atoxoplasmosis are better identified with these cytologies than with histologic review (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Often, when infectious agents are a concern, the speedy early results can direct therapy for the remaining collection while histopathology and bacteriology are pending.

WHAT TO COLLECT

Collecting tissues in a necropsy examination is a complicated decision. The first goal is to determine the cause of death. An important secondary goal is to get an idea of the holistic condition of the animal. These secondary, concurrent, or predisposing concerns are often where necropsy results can guide management efforts. The simplest solution is to collect everything. But reality jumps in all too often. Tissues may be collected to be cultured, frozen for later screening by

molecular or toxicologic evaluations, or fixed in formalin for histology. The amount of information gleaned from a necropsy is often related to the tissues collected for screening. Because necropsy examinations on hand-fed babies and eggs are different from routine necropsies, they will be separately addressed in the next section: Examinations of Eggs, Embryos, and Babies or Nestlings (see also Section 9: Embryology, Neonatology and Pediatrics).

Tissue preparations for ancillary testing beyond the examination and histology include cytologies, fluid analysis, and collection of tissues for molecular screening, toxicology, viral culture, microbiology, gamete rescue, and fibroblast or stem cell culture. Cytologies and fluid analysis rely on freshly collected (not frozen) tissues. Toxicology is usually performed on the liver, kidney, or adipose or gastrointestinal content. Brain tissue can be diagnostic if exposure to pesticides is a possibility. Microbiology materials depend on the clinical and gross findings. Typical sites for culture include the intestinal peritoneal cavity, liver, lung, air sacs, spleen, and intestines. Numerous samples can be collected for fibroblast culture and molecular archiving. In most cases, skin and/or adipose is the preferred sample. Molecular screening for infectious agents is often performed on the liver, kidney, spleen, heart, brain, and intestines. Some avian species have specific species survival programs run by zoos. (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 23, Conservation of Avian Species). A few owners and breeders support these projects and will request specific organs collected for archiving.

The most difficult necropsies are those with no specific gross findings. The tissues that are most collected for these cases are heart, lung, liver, kidney, spleen, and intestines. Please consider adding the brain to this list. While the basic set is often diagnostic, the questions often unresolved include histologic changes that might be present in the brain. Brain tissue can be collected cosmetically. The skin can be reflected to expose the dorsal surface of the skull. A window is cut through the skull cap, the brain is removed, and the skull cap and skin are replaced. The skin can then be sutured closed.

Even cases where the cause of death is identified benefit from a wide tissue review. Decisions on which tissues to submit for histology should be based on the pricing program at your diagnostic laboratory. Some laboratories charge a flat fee for a "whole-bird" necropsy set of tissues for histologic evaluation. If this is the case with your laboratory, the "more is more" philosophy of tissue collection should apply. Box 4.2 presents the full tissue collection list. Some laboratories charge per tissue. If this is the case, fewer tissues for evaluation may be selected—but unanswered questions may remain after histologic review. A solution to dealing with the "less is more" billing system is to collect a complete set of tissues, submit only the tissues critically needed for histology, and archive the additional formalin-fixed tissues. If histology suggests a need for more materials, they will be at hand. Admittedly, this tactic requires more time upfront on the part of the clinician. Some doctors deal with this challenge by performing a cursory examination and having students or technicians drive the thorough sampling plan. The time to collect additional materials is during the necropsy of the fresh carcass. Collecting tissues from a frozen carcass is less than optimal. One final diagnostic laboratory pricing scheme is a charge for grouped numbers of tissues—for example, one to four (small), five to seven (medium), and eight to ten (large). If this is the billing style of your diagnostic laboratory, you can maximize the histologic review by selecting the higher number of tissues from each grouping—for example, in this case, four, seven, and ten, respectively.

EXAMINATIONS OF EGGS, EMBRYOS, AND BABIES OR NESTLINGS

Veterinarians are often requested by aviculturists to investigate cases of reproductive failure. Knowledge of normal reproductive rates and an ability to perform an egg evaluation/embryo necropsy can provide tremendous value in this arena.

Population-level data on fertility and hatchability are essential for proper interpretation of any hatching failure. A useful rule of thumb for species with good incubation parameters is that roughly 80% of the total eggs laid should be fertile and hatch. Of those that fail to hatch, about half should be infertile and half embryo mortalities (see Section 9: Embryology, Neonatology and Pediatrics). Any changes to hatching success above background levels indicate a problem that warrants investigation.

Nutritional deficiencies and imbalances and almost any disease in breeding birds can significantly affect fertility and hatchability of eggs. Suggestive history for nutritional problems includes high or increased infertility, poor hatchability, thin eggshells, and egg binding in females, as well as poor plumage and poor bone quality in chicks. Evaluation of the breeder diet should include a review of the main diet and supplements.

Incubation issues can and often do result in embryonic mortality. Parameters that can result in failed embryonic development or hatch include temperature (dry bulb temp), humidity (wet bulb temp), incubator ventilation, and egg rotation frequency and manner (see also Chapter 40). Determining the stage of embryo development at which death occurred (and comparing that to the number of days of incubation) enables evaluation of developmental progress and facilitates formation of a differential diagnosis. If the embryo stage is retarded in relation to the days of incubation, it could indicate that the incubation temperature is too low.

Most embryos are nearly fully developed at the time of death. In these cases, the first step is to evaluate embryo position to evaluate potential hatching success. The embryo assumes the proper pip position 2 to 3 days before hatching. The normal position for hatching is head under the right wing, with the body oriented so that the beak is pointed toward the lower slope of the air cell. If the head is over the right wing, this is not typically a mortal malposition. Common malpositions associated with mortality include head under the left wing rotation away from the air cell, leg over the head, upside down (head away from the air cell), and head between the legs[8] (see Chapter 40).

Once the egg is opened, culture the yolk for bacterial pathogens. Yolk sac and umbilical infections are common, but post-mortem interval is important to rule out contamination. Infections can come from the maternal oviduct, the egg surface at laying or during incubation. Embryos do not produce much inflammatory response until very late in incubation.

For this reason, cultures without white blood cell reaction can still indicate pathogens. Changes such as necrosis may be the extent of the inflammatory change.

The embryo may have malformations. These can result from genetic or toxic issues with parents but can also reflect incubation issues early in development. Inbreeding is a common concern. In these instances, an overall evaluation of reproductive success and flock management may be needed to fully evaluate these findings.[8]

Mortalities are often recognized in nestlings and fledglings in a breeding collection (see Section 9: Embryology, Neonatology and Pediatrics). Necropsy evaluations in young birds should have a thorough history including brooder temperatures, hand-feeding records, and cohort health concerns. Necropsies should start with an assessment of nutritional condition and hydration as these are common issues with hand-fed babies. If a yolk sac is present, culture upon opening the intestinal peritoneal cavity is recommended. While yolk sac absorption varies by species (altricial species tend to resorb their yolk sacs more rapidly than precocial species), any yolk sac remaining after 13 days posthatch should be diagnosed as yolk sac retention.[8] Additional cultures and/or cytology of the mouth, crop, and intestines should be considered. Aspiration of formula is a common concern. Diagnosis is often seen with formula present within air sacs. The subacute condition typically results in a mixed bacterial infection that can lead to sepsis. Because terminal regurgitation can lead to formula at the larynx and in the proximal trachea, findings of formula limited to this region should be noted but not considered diagnostic until all other etiologies are ruled out.

Congenital malformations may be present that do not result in embryonic mortality but do cause death in hand-fed chicks. Cardiac malformations are the most common issue in the author's experience. For this reason, close examination of the heart is strongly recommended in neonatal bird necropsies (Figure 4.11). Ascites, either in fluid or gel form, can be the first finding suggestive of cardiac issues. Beyond cardiac concerns, leg and beak deformities can arise from management and nutritional issues. These conditions are often not fatal, but their recognition can suggest broader management issues worthy of investigation in the nursery (see Section 9: Embryology, Neonatology and Pediatrics).

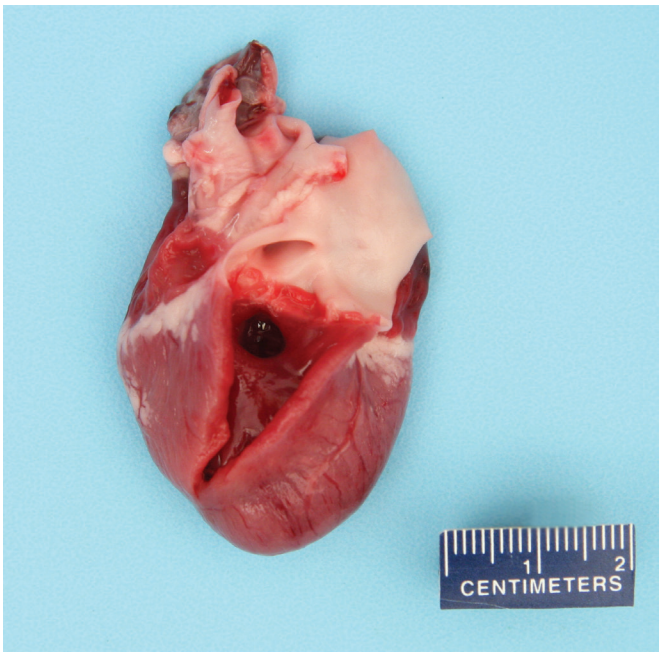


FIGURE 4.11 Subaortic ventricular septal defect in a neonatal Adelie penguin *(Pygoscelis adeliae)*.

Infectious diseases can rapidly cause mortality in many hand-fed birds. Primary viral concerns of psittacine and passerine babies include avian polyoma virus, circovirus, and avian adenovirus (see Section 9 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Avipox infections predominate in the immune-incompetent neonates (see Chapter 15). These infections are often more of a clinical nuisance than a cause of fatality, but they certainly represent a condition of neonatal concern. *Candida albicans* can proliferate and result in clinical disease. It is the most common fungal infection in the young bird and can result in thickening of the crop mucosa associated with delayed crop emptying or crop stasis (see Chapter 41). Associated with this condition, the crop contents often coagulate and present with a sour or fetid odor associated with overgrowth of yeast and bacteria. Bacterial sepsis is a major concern in baby birds. The incompletely developed immune system and immunosuppressive viruses make bacterial infections a more serious concern in neonates compared to adults. Common portals of entry include the umbilicus and yolk sac (omphalitis), as well as the gastrointestinal and respiratory tracts. The agents of greatest concern are like those in adult birds. They include *E. coli*, *Klebsiella*, *Salmonella* spp., *Pseudomonas* spp., and *Chlamydia* spp. (see Chapter 41). "Lockjaw syndrome" is a specific clinical entity in neonatal cockatiels. The condition is generally associated with infection by *Bordetella avium*, but a variety of bacterial agents have been isolated from cases. Some reported cases have had *Chlamydia psittaci* as a coinfection associated with *B. avium*. The lesions in this condition include rhinitis, sinusitis, and fibrosis of the prokinetic musculature in all terminal cases of lockjaw (see Chapter 31 and 41).[9]

HOW TO GET THE MOST FROM YOUR PATHOLOGIST

Selecting an avian pathologist is no small task. Done well, you will have a partner interested in your cases and your professional success. Most board-certified pathologists can do a very good job describing lesions in avian tissues. The value of an avian pathologist is in their specific knowledge relative to the diseases of birds and appropriate management. The avian pathologist understands your questions as a clinician and can rank lesions as to their significance in the "big picture" facing you and your client. Thankfully, good avian pathologists can be found at state and university diagnostic laboratories, in exotic animal pathology businesses, and even in large standard commercial laboratories. Once you have found one, be sure to ask for them when you send in samples. How can you find this magical pathologist?

Be on the lookout for pathologists who present at avian and exotic animal veterinary meetings or who present avian material at zoo and wildlife meetings. Look for their names on publications or in the membership roster of organizations like the Association of Avian Veterinarians (AAV), Wildlife

Disease Association (WDA), and the American (or European) Association of Zoo Veterinarians (AAZV or EAZV). Ask other avian clinicians who they use and trust with their histology. Important characteristics are that the pathologist can address questions you present either in writing with a case submission or on the phone call. They should be able to provide results in a timely manner. They should be able to direct you as needed with follow-up diagnostics, therapeutics, or management.

You can directly impact the quality of your pathology reports with the information you provide/share with your pathologist. Provide clinical pathology information (titers, culture results, etc.) when it reflects topics you would like addressed. For example, if bornavirus molecular diagnostic testing results were positive on the patient, and you submit organs like the proventriculus, ventriculus, adrenal, and brain, the pathologist should be looking at those organs specifically to identify lesions of mononuclear inflammation. (see Chapter 14 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). This should happen even if the cause of death is unrelated. If a previous biopsy has been performed, providing those results can help to guide the pathologist—you are not swaying them; you are helping them to provide context for their findings. Case material for necropsy submissions should include information about the individual, the flock or collection, the working diagnosis, gross findings, and specific questions you want to know the answers to from your report. With this degree of background information, the pathologist can create a more holistic evaluation of the tissues and the case. They can tell you what they know and share information about diagnostic limitations based on the tissue available.

Beyond single-case evaluations, having a good relationship with your pathologist means that they can help you with integrated investigations. If you are interested in cases of a specific neoplasia or infectious disease, your pathologist should be able to work with you through their database. By working with a single laboratory or a specific pathologist, you get the added value of their database as a resource for you and your practice. Most avian pathologists are delighted to work with you on presentations and publications related to important or interesting findings. Pathologists can assist with collaborations on study design and even identify additional clinicians with related interests. In short, a good pathologist is a great resource.

TALKING THE SAME LANGUAGE

It will be easier for you to write more like a pathologist than to convince your pathologist to write more like a clinician. Capturing gross findings without overinterpreting takes a little practice. But once you have established a routine for descriptive terms you will be able to comfortably describe what you see in a manner that will be very clear to the pathologist. The first step in every necropsy is to examine the patient as a whole and to evaluate each organ system. After observing the tissues, you can identify any abnormalities. Occasionally, special features like smell will be important. The most employed descriptive terms are for visually identified lesions. They should include five features:

1. Location and distribution: This can be the region in an organ such as a focal hemorrhage in the liver or it can describe the section of the organ such as thickening of the mucosa of the crop due to candidiasis. Typical terms include focal, multifocal, locally extensive, and diffuse.
2. Size and extent: Size can be an actual measure (length or weight) or a comparative statement like "the right kidney is twice the size of the left." Extent describes the portion of the organ or tissue that is affected.
3. Shape, demarcation, and contour: Shape is typically something like round, irregular, square, or pinpoint. Contour describes if a lesion is raised or depressed. Raised lesions usually indicate something added, like inflammatory cells or neoplasia. Something depressed usually indicates loss from necrosis or atrophy.
4. Color: Certain colors suggest different processes of the pathology. Red to purple often suggests hemorrhage or congestion. White or gray is often seen with necrosis or white blood cell infiltration. White is also seen in urate and calcium deposits, lipid, bone, fibrosis, and scarring. Yellow can be seen due to bile (icterus), degeneration of adipose (Figure 4.12), or associated with yolk. Black suggests melanin. Often, organs such as the gonads can be displaying natural melanosis. Other areas of normal pigmentation include the thyroid, oral mucosa, crop, and the meninges. Pathologic black changes can arise due to melanomas (Figure 4.13), excessive inhaled carbon, or in some cases trematode migrations and pigment deposition in the liver and spleen secondary to malaria infections. Green is common in some fungal infections. In addition to colors, pathologists often describe modifiers for distribution like linear, mottled, or stippled.
5. Texture: This describes how the organ feels and what the surface looks like. Typical words are soft (like your cheek), firm (like your nose), and hard (like a bone). Shiny tissues are usually wet. Crumbly tissues are usually dry. Surfaces can be regular or irregular, rough, or smooth. Tissues expanded with little bubbles are said to have crepitus. A gritty texture suggests mineralization. Fluids can be watery, viscous, gelatinous, creamy, or clotted.

Your description should be such that you can picture the case findings from words used in your gross report. You can either describe things system by system. Or, more typically,

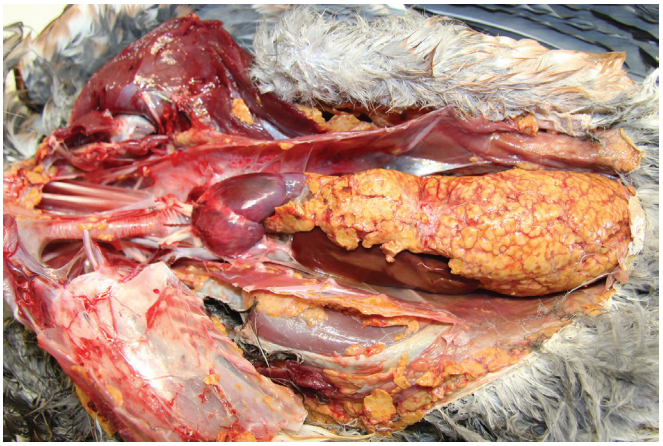


FIGURE 4.12 Degeneration of adipose in a nestling great blue heron chick *(Ardea herodias)*. Steatitis in this species is associated with pronounced accumulation of hard, yellow fat.

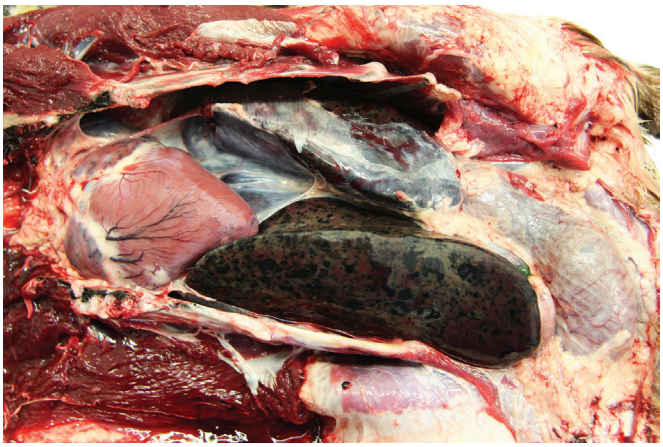


FIGURE 4.13 Hepatic melanoma in a Humboldt penguin *(Spheniscus humboldti)*. The extensive black pigment in the liver represents metastatic melanoma cells throughout the organ. The primary neoplasm was identified associated with skin.

describe the most important changes first and describe artifacts last, if at all. As you become more comfortable with necropsy examinations, you will learn what changes are artifacts and what are real. If you cannot decide, describe them. Pathologists can sort out what they mean based on histology. Typical artifact changes in avian patients are the product of decomposition. These include bile staining from the gall bladder on the liver, intestines, and even the muscle and skin over the abdominal region, an overall redness to intestines and intestinal content, dilation of the intestines, sloughing of the ventricular koilin, and congestion of the skull or meninges in the region of the cerebellum.

Getting your reports clear and concise for the pathologist is the first step in a common language for necropsies and pathology. Communication and transfer of information goes both ways. Proper interpretation of necropsy reports will increase the information you can glean from these documents. A necropsy report generally consists of three sections—description, diagnosis, and comments. To get more information inserted into the comment section, make sure to submit specific questions with your tissues. Questions can vary from "What is the change I saw in the kidney?" to "What should we do to manage the remaining household birds based on your findings?" A skilled pathologist can interpret results to answer your questions even if the histology findings are limited. Indeed, sometimes, negative findings are very helpful. Infectious diseases can be ruled out by a lack of changes in many cases. If additional tissues or special stains might address questions left unanswered, these too may be presented in the comments section.

Within the diagnosis section, there are multiple ways to present findings. Typically, the most important changes are noted as the first diagnoses. Down the line, significance becomes less. Generally, the cause of death is the first diagnosis. However, if the cause of death was known clinically but not shared on the histopathology submission form, the first diagnosis will only reflect the histopathology findings and not the entirety of available case information. This is not a shortcoming of the histopathology report, but it can sometimes be modified to fit your system as desired. It is a good idea to discuss how you prefer these reports to reflect the post-mortem findings.

The tissue description section will have specific descriptions of all abnormalities examined. A review of this section confirms which tissues were reviewed and where lesions were (and were not). It is important to remember that the diagnosis and description terminology is flexible. There are many ways to say the same thing. For example, "acute necrotizing hepatitis" and "acute hepatic necrosis with heterophilic infiltrates" can both serve as a diagnosis for similar histologic changes in the liver. The histologic descriptions can help you to better understand the variations in morphologic diagnoses.

WHAT TO DO WITH NECROPSY/ PATHOLOGY RESULTS

As with all laboratory results, the necropsy report is entered or attached to the individual patient's record—be it paper or electronic. But this should not be the end of what happens with this information. Avian veterinarians are unique in that many of our clients have collections of birds. Individual results provide a snapshot view of the collection at any time. Having a paper or digital folder for these clients in addition to individual records allows the clinician to keep track of "collection"-based results. Many practitioners have such folders to file flock-screening results and treatments. If the necropsy had ancillary testing, say cultures or PCR, the results should be filed in both the individual bird file as well as in the flock file. Also, into this file, should go the photos collected during the necropsy examination. Data management is where using a single pathologist from a single laboratory has tremendous value. Reference laboratories generally track submissions and diagnoses. Preparing a matrix of cases to address questions about facilities, species, and even specific diagnoses is only a simple search.

Some veterinarians use a variety of services for clinical and post-mortem examinations. When this is the case, the client should be encouraged to develop a matrix of mortalities to allow the same sort of evaluation that you can provide including cases that you have not seen.

If a case has a specifically good illustrating image from the gross or histologic examination, these images should get labeled and stored in yet another folder. I like to call this folder the "Good Pix" folder. Images here can be sorted by etiology or affected organ system. They should be well labeled as to bird species, identification, organ system, and diagnosis. This folder is where you can go for images for presentations and publications and to produce illustrations for client information. If a necropsy image specifically relates to a clinical image such as the live bird, a radiographic image, or an endoscopic view, including this image in the Good Pix folder enhances the value of the necropsy images. Indeed, the Good Pix folder can serve as an indexed repository for all images from clinical examinations, clinical pathology, imaging, and necropsy examinations. The index for this folder should be a spreadsheet that captures the bird species, identification, image organ system (or whole body), description, and diagnosis.

In addition to data and results from a necropsy, there is fair chance you will also have residual samples either frozen or in

formalin. Having these on hand is helpful, but keeping them forever is not. All stored or retained samples should be clearly labeled with a case and specimen ID and the date collected. This should be logged onto a simple spreadsheet. A monthly review and sample management (read tossing) will keep this archive under control while allowing you to hold onto samples that might prove useful in the future.

CONCLUSIONS

There are many reasons for viewing the necropsy as a standard examination in avian medicine. Working in concert with an avian pathologist, you can advance your abilities and expand your practice in a manner that adds not only value but also satisfaction.

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CHAPTER 5

Pharmacologic Intervention

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Avian medicine encompasses treatment of a broad range of species across a variety of sectors, so it is no surprise that while some overarching principles of treatment may be similar, other considerations vary widely. Differing sectors include companion exotics, zoological institutions, backyard or commercial poultry and game birds, free-ranging, and hobby/sport species just to name a few. In some cases, the therapeutic focus might be on treating an individual patient or commercial flock, while in other situations release of the patient into a free-ranging environment might impact the treatment plan since the bird might be hunted and consumed by humans. Additionally, in some cases veterinarians might have to consider broader economic impacts and consequences of treating avian patients. For example, with a commercial flock, choosing to treat with a medication may drastically alter the return on investment and affect the treatment course, while simultaneously necessitating being mindful of any potential impacts on human food safety. As new research is published and therapies are discovered, practitioners need to evaluate relevant findings and establish an optimal therapeutic plan for their patients. The aim of this chapter is to provide a comprehensive overview for practitioners considering pharmacologic intervention for treating avian patients. Fundamental guidelines that are foundational to developing and monitoring a treatment plan will be covered, in addition to a review of general pharmacologic principles, pharmacokinetic modeling approaches, and interpretation of antimicrobial results. Lastly, useful resources are provided for current and future use.

EVALUATING DRUG THERAPY OPTIONS

Lisa A. Tell, Krysta L. Martin, Emily D. Richards

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Therapeutic intervention for an ill avian patient can be challenging due to limited scientific data that are bird specific. As with any other patient, thorough husbandry and medical assessment of the individual or flock and documenting problem and differential diagnosis lists are essential. Subjective and objective physical examination findings, pertinent husbandry practices, past medical history, clinical pathology, microbiology, and/or imaging results are all critical for identifying medical conditions needing to be addressed with treatment. Determining if clinical signs could be resolved with management or nutritional changes is essential for avian patients. Avian patients commonly have husbandry and management issues; therefore even if clinical pathology results suggest treatment, the disease condition might be secondary. For example, avian patients might have a gram-negative bacterial enteritis where contaminated food or water might be the source of the problem versus a true primary bacterial pathogen. Committing to a detailed assessment before instituting pharmacologic intervention could help avoid repeated and unnecessary treatment. In addition, identifying the exact medical condition that warrants treatment will help narrow the proposed medication list. If treating the bird is instigated, monitoring treatment outcome, whether symptom solution or restoration of clinical pathology laboratory values, is essential.

If pharmacologic therapy is warranted, a starting point for determining specific drug options would be drug formularies that have been compiled specifically for avian patients. Drug formularies for avian patients can be found in the "Resources for Practitioners" table at the end of this chapter. When choosing a therapeutic regimen based on a formulary listing, it is important to critically review references, check for evidence-based practices (Veterinary Information Network[1] is a commonly used resource in the United States), and confirm the applicability of the planned treatment regimens for the specific medical condition and

case. A drug therapy plan is complete when the practitioner has decided on the medication, dose to be administered, route of administration, frequency of administration, treatment duration, therapeutic monitoring, and how treatment success will be defined.

When a drug therapy plan is being constructed, best practice questions to consider include the following:

- *What nonpharmacological interventions can be utilized as an alternative?*

  While medications are often the first line of defense for "treatment," nonpharmacological interventions should be prioritized and could be a more favorable alternative without compromising the patient's health. Nonpharmacologic options minimize unnecessary medication use, especially with antimicrobials, and prevent adverse reactions. Even simple approaches, such as isolating sick patients, preventing the spread of disease, changing the food/water more frequently, or evaluating where the food/water bowls are relative to where the bird perches, can minimize or eliminate the need for antibiotic treatment. In some cases, establishing protocols for treating patients in multibird or multianimal homes is helpful since separating treated from nontreated animals might be warranted, especially to avoid cross-contamination through coprophagia. Further details on nonpharmacological interventions will be discussed later in this chapter.

- *What patient factors should be considered?*

  There are many patient factors that can affect selection of a medication, including breed/species, age, general health, renal/hepatic function, temperament/behavior, and reproductive status. Some breeds or species are more sensitive to certain drugs than others and should be avoided. For example, bone marrow toxicosis following fenbendazole treatment has been documented in Columbiformes, storks, and vultures, thus other anthelmintics should be considered.[2–4] Some drugs may have deleterious effects on certain age groups, such as fluoroquinolones causing cartilage damage in some young animals. The general health or concurrent issues of a patient may also play a role in drug selection. If a particular drug is known to exacerbate or have additive effects to current issues (such as a side effect of vomiting in a patient already presenting for regurgitation), risk versus benefits of the drug should be evaluated. The reproductive status of the patient may also be of value when determining what medication to prescribe.

- *Is the treated bird a food animal species or does it have the potential to produce food products for human consumption?*

  This is a vital question to answer prior to treating a bird because of the regulations and prohibitions related to medication use in food-producing animals. In some countries, there are specific drugs and drug classes that are prohibited from use in food-producing animals (Table 5.1).[5–8] This is due to the concern for drug residues being present in food products intended for human consumption. Additionally, in certain countries, such as the United States and Europe, if a drug is used in an extra-label manner in a food-producing animal, scientific data documenting drug residue depletion are required in order to establish an extended withdrawal interval. If such data do not exist, other medications have to be considered. If an animal has already been treated and there are no scientific data to support a withdrawal interval, the animal cannot enter the human food chain. Withholding animals for extended periods following treatment or having them condemned following illegal drug use poses economic losses for producers.

- *Will the proposed medication treat the disease or alleviate clinical signs?*

  It is important to utilize medications that will be effective, but equally critical is selection of antibiotics with a narrow killing spectrum to mitigate antimicrobial resistance. Drug selection based on culture and susceptibility is the gold standard. Best practice recommendations for using susceptibility results to guide therapeutic selection are discussed later in this chapter and in Chapter 7.

  One must also consider if the selected medication will be effective mechanistically and/or reach the site of action or infection at appropriate concentrations. These concepts are especially important for antimicrobials. For skin infections, consider topical therapy as drug concentrations at the infection site will likely be higher compared to systemic administration. With avian patients, it is also important to consider ingestion of a topical medication and secondary disruption of the gastrointestinal (GI) flora. Caseous abscesses can be challenging to resolve, since antibiotic penetration will be limited, and therefore surgical debridement should be performed prior to treatment. When therapeutic concentrations of medications need to be achieved in the central nervous system, it is imperative to determine if the medication's properties will allow it to cross the blood-brain barrier. Generally, drugs that are small, lipid soluble, unionized, along with low plasma protein binding are more likely to cross the blood-brain barrier.[9,10] For example, aspergillosis typically originates in the avian respiratory tract and the antifungal agent, itraconazole, is commonly prescribed.[11,12] However, if the infection disseminates to the central nervous system, treatment with itraconazole is not ideal since poor blood-brain barrier penetration has been documented.[10,13]

- *What will be the route of administration?*

  There are many ways medications can be delivered to avian patients—orally (via medicated water, medicated feed, or direct administration to oral cavity or ingluvies), intravascular, extravascular (intramuscular [IM], subcutaneous [SC], intracoelomic [IC]), inhalation, topically, ophthalmic, and so on. This is an important aspect to consider for avian patients, but especially imperative for medications that are concentration dependent to be therapeutic (see also Chapter 7). A classic example would be concentration-dependent antimicrobials like fluoroquinolones. If therapeutic concentrations cannot be achieved, it is likely that the infection will not resolve, but there is also increased potential for antibiotic resistance.

  For drugs proposed to be administered by intravascular or extravascular route to the caudal portion of a bird's body, one should assume that the drug will pass through the kidneys first via the renal portal system before being delivered to the rest of the body. This is especially important for renally excreted drugs. The renal portal

TABLE 5.1

Drugs Prohibited From Use in Food-Producing Animals in Various Countries

A

Drug Classes Prohibited From Extra-Label Drug Use

Fluoroquinolones (all avian species)
Indexed drugs (all avian species)
Medicated feeds (specific to chickens and turkeys)
Cephalosporins (specific to chickens and turkeys)

Drugs Prohibited From Use Entirely

Chloramphenicol (all avian species)
Clenbuterol (all avian species)
Diethylstilbestrol (all avian species)
Nitroimidazoles (dimetridazole, ipronidazole, metronidazole, etc.) (all avian species)
Nitrofurans (furazolidone, nitrofurazone, etc.) (all avian species)
Glycopeptides (all avian species)
Gentian violet (all avian species)
Adamantane and neuraminidase inhibitors (specific to chickens, turkeys, and ducks)

B

Carbadox
Chloramphenicol
Clenbuterol (except for tocolysis as per approved label)
Diethylstilbestrol (hexoestrol, dienoestrol, etc.)
Fluoroquinolones
Nitrofurans (furazolidone, nitrofurazone, etc.)
Nitroimidazoles (except dimetridazole as per approved label)
Phenylbutazone
Salbutamol
Thyrostats (thiouracil, etc.)

C

Thyrostatic substances
Stilbenes (stilbene derivatives, their salts, and esters)
Oestradiol-17β and ester-like derivatives
Beta-agonists (with derogations)

D

Chloramphenicol (or its salts or derivatives)
5-Nitrofuran compounds
Diethylstilbestrol (or other stilbene compounds)
Clenbuterol (or its salts or derivatives)
5-Nitroimidazole compounds

A: Drugs prohibited by the US Food and Drug Administration—avian specific.[5]
B: Drugs prohibited by the Australian Pesticides and Veterinary Medicines Authority.[6]
C: Drugs prohibited by the European Union.[7]
D: Drugs prohibited by Health Canada.[8]

system could eliminate the drug before it circulates to other parts of the body, which could lead to treatment failure. In some cases, the effect of the renal portal system on drug concentration may not necessarily be clinically significant, but it is still important to evaluate the literature before choosing to administer renally excreted drugs to the caudal half of the body. In addition, administering nephrotoxic drugs to the caudal portion of a bird's body is contraindicated.

For oral medication delivery via medicated water or feed, it is necessary to estimate the amount of food and water the patient consumes to calculate the necessary concentration of medicated feed or water so that the patient receives the proper dose. In sick patients, consumed amounts are likely to be decreased compared to healthy birds. Using average consumption estimates based on healthy individuals has the potential to result in underdosing and treatment failure. Additionally, medication delivery via feed or water is likely to provide a sustained drug concentration but at a lower maximum concentration, as the consumption of the medicated feed or water is continuous throughout the day versus a one-time event. Therefore, drug administration via medicated feed or water might not be appropriate for concentration-dependent medications. Another point to consider with orally administered drugs is the potential for crop retention. In most poultry species, GI absorption occurs in the small intestines.[14] This is likely true for most other avian species as well. Depending on the feeding system utilized or GI diseases, crop retention may occur, thus delaying the passage of orally administered medications to the small intestine and delaying absorption.[15]

If there are a several medication options for oral administration, consider whether administration with food or without food is necessary. For some medications, absorption may be better when administered with food, while others have better absorption when the patient is fasted. For example, voriconazole and doxycycline have been shown to have increased absorption when administered to fasted patients.[16,17] Because there are limited data for avian species regarding the effect of food on oral bioavailability of medications, effects of prandial state for other species and/or medications should be considered when treating birds, but further studies are necessary to guide definitive practices for birds. Information regarding optimal prandial states when taking medications can be found in the package insert and full details about the medication are outlined in the prescribing information handout. Alternatively, this information may also be found in a comprehensive veterinary drug information reference such as Plumb's Veterinary Drug Handbook.[18]

Reviewing the drug's mechanism of action, published pharmacokinetic data for the species to be treated or a related class of birds, and deciding on a route of administration are important before initiating therapy. With any treatment plan, administration compliance is key

for successful therapy. While the veterinarian may prefer a specific route of administration due to a specific drug's properties and treatment effectiveness, it is important to consider the client's or caregiver's ability and willingness to administer the medication(s) compliantly, especially in situations where there are multiple options for drug selection.

- *If an antibiotic is required, does it have time-dependent or concentration-dependent pharmacodynamics?*

  Ideally, antibiotic selection should be based on culture and sensitivity. When choosing a medication, it is important to know if the pharmacodynamics are time dependent or concentration dependent (see also Chapter 7). Efficacy of time-dependent antibiotics depends on the percentage of the dosing interval that an antibiotic is above the minimum inhibitory concentration (MIC), while concentration-dependent antibiotics target a serum/plasma/tissue concentration that is above the MIC. It is important to evaluate the literature to ensure that the pharmacokinetics of the desired drug, dose, and route of administration provides antibiotic concentrations in the serum/plasma/tissue or at the desired treatment site adequately, with respect to time or concentration above the MIC. These characteristics will impact the frequency of drug administration, so it is important to evaluate the pharmacokinetic literature for scientific guidance on how often to dose the medication to maintain concentrations above MIC. Such practices will help ensure treatment efficacy and help prevent the development of antimicrobial resistance. Knowing administration frequency may also impact the drug choice as some antibiotics require more frequent dosing compared to others. Typically, frequency of drug administration and treatment compliance are inversely related.

- *Does the patient have concomitant medical conditions or coadministered medications that can affect this drug?*

  It is always important to evaluate a patient's current medication list for potential drug interactions. If data do not exist for the species and drugs of interest, look to similar species, for potential or documented interactions as guidance. Plumb's Veterinary Drug Handbook[18] offers potential drug interactions based on data from small animals and humans. While this resource does not specifically focus on avian species, it does provide guidelines that should be considered when establishing a treatment plan. If veterinary-specific drug information is not available, the use of a human drug resource can be used to gain insight into potential drug-drug or drug-disease interactions. Some human drug information resources include Clinical Pharmacology, Lexicomp, and Micromedex.[19–21] Additional resources can be found in the "Resources for Practitioners" table at the end of this chapter.

  In addition to drug-drug interactions, evaluate the patient's medical status to reduce the incidence of drug-disease interactions. For example, patients with renal compromise should not receive drugs that have a relatively low threshold for being nephrotoxic or medications that are metabolized by the liver might accumulate in patients with hepatic disease.

- *Do the benefits of a chosen treatment outweigh the potential side effects or complications?*

  After the previously mentioned considerations have been addressed, it is vital to critically evaluate potential negative consequences that can occur from treating the patient with a particular medication. This could include negative consequences in terms of side effects directly related to the medication or economic losses if the bird or their by-products cannot enter the human food chain if a prohibited drug is administered. With any treatment, it is important to ensure that therapeutic benefits and desired outcomes for the patient, owner, producer, etc., outweigh the potential negative consequences.

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REVIEW OF PHARMACOKINETICS AND PHARMACODYNAMICS

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Drug administration in avian medicine requires selection of safe and effective dosage regimens. Unfortunately, in avian practice, this information is often limited or not available and therapy is further complicated by variability within and between avian species. However, a basic understanding of fundamental concepts associated with drug administration to birds[1] and the pharmacokinetics and pharmacodynamics of drugs in avian species will enable the clinician to optimize therapy while minimizing the risk of adverse drug effects.

Pharmacokinetics describes the effect the body has on a drug, including drug absorption, distribution, metabolism, and elimination. Pharmacodynamics, in contrast, describes the effect a drug has on the body and may be affected by an individual bird's or subspecies of bird's pharmacogenetic profile. This information, when available, can be applied to avian patients to determine dosage regimens in healthy animals and optimize treatment of diseased animals.

From a practitioner's standpoint, pharmacokinetic studies that detail drug absorption, distribution, metabolism, and elimination might seem esoteric but understanding these fundamental key components is essential for successful application in clinical practice.

DRUG ABSORPTION

A drug must get to the site of action at an effective concentration for a sufficient period of time in order to exert a therapeutic effect. This requires absorption into the system circulation, which is affected by the route of administration. The route by which a drug is administered also has economic and feasibility concerns, particularly in avian

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medicine where treatment can be for an individual bird or a flock.

Oral Drug Administration

This is a common route of drug administration in birds, especially if patients are to be sent home and treated by the owner. Compared to mammals, there are several anatomic differences in avian species that might affect drug absorption via this route. One of these differences is the presence of a crop in several avian species, such as some birds in the Galliformes, Psittaciformes, Columbiformes, and Accipitriformes orders.[2] The crop is an expanded area of the cervical esophagus used to store food and, by extension, orally administered medications.[3] As little absorption occurs in the crop itself in most species, this can result in a more rapid or delayed drug absorption, depending on transit time through the crop. Crop-emptying rate is dependent on fill; therefore birds fasted prior to drug administration might have rapid drug absorption. Sick birds that are anorexic may also have faster crop-emptying times. However, avian patients with diseases that slow crop emptying and young hand-fed birds being gavaged with commercial liquid food products might have delays in drug absorption. Animals on dry feeds have slower crop emptying compared to those on wet feeds. For chickens (broilers in particular) crop emptying can be delayed up to 19 hours or more depending on the amount of food ingested.[1] The pH of the crop is neutral to acidic, varying with the species of bird and the presence and type of feed. This pH variability can affect drug absorption by resulting in drug degradation, precipitation, or increased solubility of the dosage form in the crop itself.[4] The crop may also contain a normal bacterial flora (e.g., *Lactobacillus* sp.) that can break down some drugs (e.g., macrolides) prior to absorption.[4]

After leaving the crop, orally administered drugs will move to the proventriculus and ventriculus or "gizzard." These two chambers are somewhat equivalent to the mammalian "stomach." In carnivorous and piscivorous birds, the proventriculus can store food due to the presence of multiple extensible folds.[5] In some species of birds (such as granivorous species), the ventriculus will have a high mineral content (including calcium and magnesium) due to the presence of grit and may therefore chelate certain drugs like fluoroquinolones or tetracyclines. This grit, however, can help with the grinding and dissolution of solid dosage forms such as tablets. The efficiency of grinding may depend on the diet of the individual species; carnivorous and piscivorous birds have a poorly differentiated gizzard compared to other birds. The low pH of the proventriculus/ventriculus can also degrade some acid-labile drugs (e.g., penicillin and erythromycin). Ingesta mixes back and forth between the proventriculus and the ventriculus to optimize mechanical and enzymatic digestion.[5] There is also reflux of ingesta between the duodenum and ventriculus. This retrograde movement of ingesta (and drug) may further delay absorption, as many drugs are absorbed in the small intestine. The remainder of the GI tract is relatively short in most birds, compared to mammals, particularly with respect to the large intestine. The cecum, however, may be well developed in species such as ducks, chickens, and ratites. Although the large intestines are typically not a common site for drug absorption, carrier-mediated absorption or permeability of lipophilic drugs can occur at this site, and therefore these anatomical differences may have an effect on overall absorption as well as elimination throughout the GI tract.

Administration of solid oral dosage forms has issues related to dissolution, as discussed previously, but also has issues related to stress on the bird being treated, and this method is not amenable to dosing large numbers of birds from a practical standpoint. Direct oral administration of solutions and suspensions decreases the issues with dissolution, but drugs may precipitate out of solution at the varying pH in the upper GI tract. Aspiration following regurgitation of drugs in liquid form may also occur.[1,6] For the treatment of large flocks of birds, this method is also not practical; therefore more commonly dosing is done via medicated water or feed.

Administration of drug in water or feed has the advantage of being a low-stress method suitable for treatment of multiple birds. However, this is also a disadvantage, as it may expose normal, healthy birds to medications that are not necessary. For antibiotics, this can lead to the development of resistant bacteria. Additionally, it is not possible to determine how much drug an individual bird receives. Sick animals tend to have a decrease in feed and water intake and may therefore not get the required dosage. Drugs in water and feed may also change the taste of either, resulting in aversive behaviors. Day length and ambient temperature can affect consumption. To increase drug exposure, medication of both feed and water simultaneously can be performed. In birds intended for food in the United States, many feeds medicated with antimicrobials are regulated under the Veterinary Feed Directive (VFD) and any administration requires the oversight of a veterinarian. Extra-label use of VFD drugs is prohibited. However, in order to address the lack of approved medicated feeds for minor avian species (such as chukar, quail, pheasants, etc.), the US Food and Drug Administration (FDA) codified a compliance policy guide[7] that allows inspectors to have regulatory discretion when a veterinarian writes a VFD for minor avian species.

Medicated water and feed result in ingestion of smaller quantities of drug more frequently throughout the day. These administration routes are optimal for treatment of large numbers of birds but are also used for cases where handling and restraint of individual patients is not possible. In some cases, sick birds will reduce the amount of food or water ingested; in extreme cases some birds will not drink medicated water. Additionally, absorption of drugs in medicated feed may be further delayed due to storage of drug in the crop or proventriculus. Drugs administered via medicated feed or water have the potential for decreased absorption and subsequently lower plasma concentrations compared to direct oral administration. This is an important consideration when a maximal drug concentration or a percentage of time above MIC is being targeted to ensure treatment efficacy.

Parenteral Drug Administration

Parenteral drug administration will overcome many of the issues associated with oral drug administration. Intravenous (IV) administration bypasses the absorption phase and delivers 100% of the drug directly to the systemic circulation. However, this route is technically the most difficult, particularly in avian species. The subcutaneous and intramuscular routes are technically less difficult but can have specific disadvantages.[6] Subcutaneous drug administration is most commonly performed in the inguinal, axillary, or interscapular regions. This route of administration can have decreased absorption in

dehydrated birds. Intramuscular drug administration is most commonly performed in the pectoral muscles, mid-keel region. Injections into the cranial pectoral muscle are more likely to result in hemorrhage. Repeated injections in the pectoral muscle, particularly with irritating substances, can eventually lead to muscle necrosis and atrophy. This can have long-term effects on the bird's ability to fly and also result in carcass rejection in animals intended for food. From a welfare standpoint, repeated intramuscular injections can also be painful, especially if the vehicle is viscous or administration of large volumes is required. Other factors to consider with intramuscular injections include differences in myoglobin concentrations in muscle fibers and the degree of muscle vascularization. These differences can be substantial between flighted and nonflighted birds and might influence drug pharmacokinetics, but no definitive studies have been published to date. In general, intramuscular injections administered in the thigh muscles are not advised in birds. Drugs administered at this site may be absorbed first into the external iliac vein, and then into the renal portal vein, where it is delivered to and immediately excreted via the kidneys. This can result in a renal "first-pass" effect and potentially decreased amounts of drug reaching the systemic circulation via this route of administration, particularly water-soluble drugs that undergo minimal metabolism such as gentamicin.[8,9]

Drug Administration via Inhalation

Nebulization has been commonly used in avian medicine to administer medications directly to the upper respiratory tract, pulmonary parenchyma, and air sac membranes. Experimental studies have shown that nebulization is not an efficacious method for delivering medications to the avian respiratory tract given the challenge of getting enough particle sizes that are also small enough for distribution to the air sac membranes and pulmonary parenchyma.[10] Direct tracheal delivery via ventilation[11] and using a commercial atomizer[12] has been experimentally shown to successfully deliver 0.5 μm particles of amphotericin B, but further work remains to be performed before such techniques can be applied clinically. Systemic absorption of drugs originally delivered directly to the avian respiratory tract is unknown and will most likely depend on the physiochemical properties of the medication. However, therapeutic drug concentrations of some antibiotics may persist locally in the lungs and air sac membranes for 4 to 6 hours after administration.[13] Drug persistence can also be impacted by the presence or absence of ciliated epithelium that would functionally remove inhaled particles. The distribution of ciliated epithelium in the upper airway of birds is similar to mammals but is sparse in the lower respiratory tract where pulmonary macrophages dominate. For the air sac membranes, ciliated epithelium is present at the ostia,[2] but not in the distal portions of the air sac membranes where drug removal would be minimal. Birds only utilize a small percentage of their respiratory tract when at rest, therefore drug absorption or distribution from the lung parenchyma might be influenced by the degree of activity and flight.

Pharmacokinetic Parameters Associated With Absorption

The fraction of drug absorbed after administration is represented by bioavailability (F%). Bioavailability is calculated by comparing the area under the curve (AUC) concentration versus time curves generated from plasma concentration data collected over a period of time. Because IV administration of a drug is presumed to have 100% absorption, the AUC of the IV dose is compared to the AUC via other routes of administration to get absolute bioavailability. When IV dosing is not feasible, due to lack of formulations, toxicity, or other factors, two extravascular routes of administration can be compared to calculate a relative bioavailability. Reasons for low bioavailability include incomplete absorption from the GI tract or injection site, instability of the drug in the vehicle, or first-pass metabolism (hepatic or renal in birds).[14,15]

Both the rate and extent of absorption affect bioavailability. The rate of absorption is represented by the absorption rate constant (k_a) or the time to maximum concentration (T_{max}). The extent of absorption includes AUC estimation and is also assessed using the maximum plasma concentration achieved (C_{max}).

DRUG DISTRIBUTION

Drug distribution is a key factor to evaluate when trying to extrapolate drug dosing regimens from mammals to birds or from birds to birds. Once a drug reaches the systemic circulation, it must be distributed from the central (blood) compartment to the peripheral tissues to exert an effect at the site of action. Distribution to organs of metabolism and elimination is also required for drug to be cleared from the body. There are multiple factors affecting a drug's ability to exit the plasma and therefore distribute to the site of action. All other factors remaining equal, well-perfused tissues take up a drug much more rapidly than do poorly perfused tissues. For tissues of greater mass (such as muscle), more drug-binding sites are present, which increase the capacity of that tissue for the drug. These two factors are dependent on each other and the ratio of blood flow to tissue mass is used to categorize drugs as either high or low-distribution drugs. For example, the brain has a high perfusion rate, but a relatively small tissue mass, resulting in a higher ratio. Not all drugs will distribute to the brain, however, due to the presence of protective barriers at this site that prevent drug diffusion between cells (paracellular). Only drugs that can diffuse through cells (transcellular) or those that have tissue-specific transport across the membrane will reach therapeutic concentration in these protected sites. Other sites in avian species with these protective barriers include the eye and atria (equivalent to mammalian alveoli). A drug's ability to diffuse transcellularly is dependent on its physicochemical characteristics: small weight, lipophilic drugs that are unionized at physiologic pH will cross membranes and typically have a higher distribution. Protein-bound drugs are too large to pass through or between cells and therefore have limited distribution. Protein binding is a property of the drug, but also the species to which the drug is administered. Proteins such as albumin, the main protein that binds weak acids, have species-specific affinities and a number of binding sites.[16] P-glycoprotein efflux pumps present at protective barriers pump drugs back out of cells, resulting in decreased drug diffusion into these sites. These pumps may be present in other organs, including the liver and GI tract, and may prevent drug absorption from and distribution into those sites.

Pharmacokinetic Parameters Associated With Distribution

The volume of distribution (Vd) is a theoretical proportionality constant relating the mass of drug in the body (dose) to the concentration measured in a sample (usually the plasma):

$$Vd = Dose/Concentration$$

It should be referred to as the "apparent" Vd because it represents a theoretical volume not any actual physiologic or anatomic space or volume. Low-Vd drugs remain in the blood vasculature and extracellular fluids whereas high-Vd drugs also distribute into the intracellular and transcellular fluid compartments. Drugs with a high Vd may have higher and more persistent tissue concentrations compared to plasma concentrations.

Since plasma concentrations are determined by absorption, Vd is typically only calculated for an IV dose, where absorption is 100%. If Vd is reported for extravascular routes of administration, it should be reported as Vd/F, or Vd relative to bioavailability. However, if bioavailability is low following extravascular administration, the Vd will be falsely elevated. Once the Vd for a specific drug is known for a specific species, this information can be used to design a dose that will result in the desired plasma concentration using the following equation (which is a modification of the previous equation):

$$Dose = Vd \times Concentration$$

DRUG METABOLISM AND ELIMINATION

Metabolism

Compared to mammals, information regarding drug metabolism is limited in birds with the majority of the literature focused on poultry species. Drug metabolism is the chemical alteration (biotransformation) of drugs by the body, and is most often accomplished by the liver, although other organs can play a role, including the lungs, kidney, and GI epithelium. There are two primary phases in drug metabolism. Phase I reactions are carried out by the cytochrome P450 (CYP450) enzyme system and include oxidation, reduction, or hydrolysis reactions. The purpose of these reactions is to add polar groups or remove nonpolar groups from the drug compound so that the drug is either directly excreted by biliary or renal processes or moved on to phase II reactions. Phase II reactions are conjugation reactions that couple a parent drug or its metabolite to a polar endogenous substrate (glucuronic acid, acetic acid, an amino acid, or an organic substrate such as sulfate). Phase II products are larger and more polar than the parent drugs, which restricts their distribution, and thus activity, in the body and allow for biliary or renal excretion/secretion. Biotransformation generally results in a metabolite with reduced pharmacological activity and effectively blocks drug effects. However, in some cases, the metabolite has enhanced activity compared to the parent drug. This phenomenon can be manipulated in the drug manufacturing process where a drug with enhanced stability and absorption is then cleaved in the body to an active form. These drugs are referred to as prodrugs.

Drug metabolism is not always a static process, since induction or inhibition of metabolism can occur, mainly due to changes in production of phase I CYP450 enzymes. This may occur as a result of administration of the drug itself (autoinduction or autoinhibition) or secondary to coadministration of a second drug that inhibits or induces the same CYP450 isoenzyme. There are limited data for avian species, and species differences may occur. For example, voriconazole, a triazole antifungal agent, is known to induce its own metabolism with multiple doses in humans and some avian species (e.g., grey parrots [*Psittacus erithacus*]), but multiple doses do not result in alterations in metabolism in horses.[17–19] In the case of enzymatic autoinduction or autoinhibition, single-dose studies might not reflect the pharmacokinetics of multiple dosing; therefore multiple-dose studies are essential. Diet, age, gender, and health, particularly with regard to liver disease, can also have an effect on drug metabolism and subsequent elimination.

Both phases I and II metabolic pathways have been shown to occur in avian species. Results of mRNA expression tests suggest CYP2C genes may have an important role in xenobiotic metabolism in chickens. The CYP2C45 gene was the most highly expressed isoform in chicken liver, while the CYP2C23b gene was the most highly induced by phenobarbital.[14] Responses to three separate classes of inducers of CYP450 are conserved in chickens and mammals, and CYP2R1 and CYP2U1 are fully conserved between avian and mammalian species, which reflect their essential functions in the metabolism of endogenous compounds.[20,21] Piscivorous birds show lower hepatic microsomal CYP450 activity relative to omnivorous or herbivorous terrestrial birds. For phase II metabolism, conjugation pathways present in mammals are also present in birds; however, only birds and reptiles form ornithine conjugates.[4]

Drug metabolism varies not only between species but also within species. The greatest source of variability between species relates to isoenzyme makeup of both cytochrome and noncytochrome enzyme systems, and in the availability of substrates for conjugation. Within a species, there are groups of individuals classified as ultrarapid or poor metabolizers of certain drug classes. The study of this phenomenon is called pharmacogenomics. Polymorphisms are well documented with certain reactions in humans and rats; however, very little information is available in avian or other veterinary species. Polymorphisms in CYP450 are speculated to be behind the severe toxicity seen in vultures exposed to nonsteroidal antiinflammatory drugs such as diclofenac and ketoprofen.[22] Drug metabolism can be influenced by disease processes and reproductive status as well as body composition, gender, age, and light cycles as has been reported for major veterinary species,[23] but these influences have not been well elucidated for avian species.

Drug-metabolizing enzymes obey classic Michaelis-Menten enzyme kinetics and may therefore display saturation with exposure to high drug concentrations, leading to zero-order (nonlinear) pharmacokinetics. This results in a constant rate of drug elimination, independent of dose, meaning the eliminated fraction of the administered dose has a relative decrease at higher doses. This can result in an enhancement of drug effect, or even toxicity, at high doses. Voriconazole is an example of a medication used in avian medicine to treat aspergillosis that has zero-order kinetics.[24]

ELIMINATION

Like mammals, the kidneys are the main route for drug elimination for birds. Renal elimination occurs passively through

glomerular filtration or actively through tubular secretion. Drug can also be passively reabsorbed in the kidney; therefore, net renal elimination of a drug will be the sum of all these processes.

Glomerular filtration is a relatively low-efficiency process and is restricted to protein-unbound drugs dissolved in plasma water. Renal clearance of a drug by passive diffusion is approximately one-sixth the corresponding value of renal blood flow. Tubular secretion is an active, carrier-mediated process that is relatively specific for organic acids and bases and is also remarkably rapid and efficient; the renal clearance of a drug excreted by active secretion approaches the value of renal blood flow. Protein binding does not restrict secretion because affinity for the active transport systems is greater than affinity for the protein. Tubular secretion is a saturable process. The final determinant of a drug's renal disposition is the mechanism of passive tubular reabsorption, which is dependent upon urine flow rate and pH, as well as lipid solubility of the nonionized drug. At low urine flow rates, as in states of dehydration, there is greater time for drug molecules to diffuse from the tubular fluid back into the blood.

There are numerous unique characteristics of a bird's renal system that may affect drug elimination.[8] The avian kidney consists of three lobular divisions (cranial, middle, caudal), each of which has cortical and medullary regions. The nephrons located in the more superficial, cortical area are simple nephrons, like those found in reptiles. These reptilian nephrons make up most of the nephrons in the bird, although mammalian-like nephrons may make up to 10% to 30% of the nephrons found in different bird species, the concentration of which depends on the bird's environment. In general, glomerular filtration rates in birds are lower than in mammals of similar body weights, and it is intermittent in birds whereas it is constant in mammals.[24,25] This may result in more variable drug elimination by glomerular filtration in birds, particularly in gallinaceous birds that have a more marked intermittent filtration compared to Passeriformes. Less is known about tubular secretion and reabsorption in avian species compared to mammals. The avian renal system is less efficient at concentrating urine and therefore may be less capable of tubular reabsorption. Urine pH, which can also affect tubular reabsorption, varies in female birds between 4.7 and 8 and is affected by egg-laying status; in male birds the pH is more stable and approximately 6.4.[26] The renal portal system and its role in excreting drug prior to absorption have been discussed previously.

Hepatobiliary excretion can also be a major route of drug elimination for some drugs and many metabolites, particularly lipophilic anionic drugs with larger molecular weights. The molecular weight cutoff for biliary excretion may vary by species, with 375 to 400, 394, and 475 Da reported for rats, dogs, and humans, respectively.[27,28] Assuming a similar range for the cutoff in birds, this may include drugs such as azithromycin and doxycycline. This process is relatively slow compared to renal excretion. Bile is an excretory product continuously produced by hepatocytes, stored in the gall bladder, and then secreted through the common bile duct into the duodenum. Not all birds have gall bladders (i.e., pigeons, most parrots, ostriches); therefore bile storage does not occur.[2]

Hepatobiliary excretion is an active process, utilizing p-glycoprotein efflux pump transporters, and is therefore saturable and competitive. Diffusion of drugs passively into the bile is not of major significance.

Compounds secreted in bile have a high degree of polarity, inhibiting reabsorption in the small intestines, and drugs are therefore subsequently excreted in feces. In some instances, however, drugs are metabolized by the intestinal microflora back to parent drug and are then eligible for reabsorption back into the systemic circulation. This process is termed enterohepatic recirculation and may result in greater overall exposure to the drug (increased bioavailability).

Pharmacokinetic Parameters Associated With Metabolism and Elimination

Clearance

Clearance is defined as the volume of blood cleared of a substance (drug) per unit time and is typically adjusted for body weight (volume/time/kg). Most reported clearance rates are the sum of the individual organ clearances, resulting in the total body clearance (Cl) of the drug from the animal:

$$\text{Total body clearance} = Cl_{Renal} + Cl_{Hepatic} + Cl_{Other}$$

Although other organs may function as eliminating systems (i.e., lung), the kidney and liver play the major role. In mammals, sweat glands can function as an eliminating system but birds do not have sweat glands.[2] The liver also has a metabolic role in drug elimination. Specific renal and hepatic drug clearance can be determined by measuring drug concentrations separately in urine and feces. However, the unique excretory system of the bird results in "hepatorenal" drug elimination, as it is not possible to separate out the different renal or biliary drug excretion without invasive testing methods. The individual contribution of the kidneys versus the liver in avian species is not often known.

Total body clearance can be calculated using the equation:

$$Cl = \text{Dose}/AUC$$

Similar to the volume of distribution, since this equation uses a measure of drug absorption, AUC, clearance can only be predicted accurately following IV administration. Clearance from extravascular administration should be reported as Cl/F and may not be accurate.

Half-life

The elimination half-life of a drug is a hybrid parameter that is related to the primary drug parameters of volume of distribution and clearance.

$$\text{Elimination half-life} = (0.693 \times Vd)/Cl$$

The plasma elimination half-life is defined as the time it takes for plasma drug concentration to decrease by 50% of the previous value. Since some drugs distribute outside of the plasma, a plasma elimination half-life does not necessarily represent the time it takes to eliminate 50% of the drug from the body. In first-order (nonsaturable) kinetics, the elimination half-life is a fixed value and is independent of the drug concentration. In zero-order (saturable) kinetics, the half-life value changes when plasma drug concentrations reach the point of saturation.

Half-life becomes very useful in the creation of multiple-dose regimens and is used to predict the time to steady-state concentration, the time when the rate of drug input is equal to the rate of drug elimination. It takes approximately four to

five drug half-lives to reach a steady state. For predicting withdrawal intervals following drug administration in animals intended for food, half-life can be used as well. For drugs with first-order kinetics, 99.9% of the drug is eliminated after 10 half-lives. This means that for most drugs, plasma concentrations will be below the tolerance (or maximum residue limit). However, this is not useful unless plasma concentrations and tissue or egg concentrations are parallel, which only occurs with drugs that have a small volume of distribution. For drugs with a large volume of distribution, tissue or egg half-lives cannot be applied using this rule.

PHARMACOKINETICS OF DRUGS IN EGGS

The principles of absorption and distribution can be applied to drugs that reach the matrices of eggs (egg yolk and white) of treated animals. Distribution of drugs to and within the egg will depend on physiochemical characteristics of the medication[29] and the composition of egg components, which might differ between avian species. Drug distribution to eggs and within the egg is of concern, as exposure of the developing egg to drugs can lead to toxic effects in the oocyte, as well as potential drug residues in eggs used for human consumption. When discussing the kinetics in eggs, it is important to consider the different components of the egg, as each one may have different kinetic properties. The yolk consists primarily of lipoproteins, whereas the egg white (albumen) is made up of water-soluble proteins (albumins) and the shell is mainly made of calcium carbonate. Each of these matrices will therefore absorb, bind, and eliminate drugs at different rates. A comprehensive review of drug residues studies summarizes these differences.[30]

In theory, lipophilic drugs would distribute more to the egg yolk and water-soluble drugs to the egg white. Additionally, drugs that chelate to calcium may bind to the shell components.[29] This does not often correlate to drug concentrations in vivo, however. Relatively lipophilic drugs such as sulfonamides, enrofloxacin, and doxycycline/minocycline reach higher concentrations in egg whites than in the egg yolk. Other studies have attempted to correlate drug distribution within the egg with other physicochemical properties. The drug acid-based dissociation constant (pKa) has also been investigated to try and discern its role in the differential distribution of drugs into egg yolks and whites. The pH of the yolk and egg white are approximately 6 and 7.6, respectively. This difference in pH can lead to differences in drug ionization based on drug pKa. However, no direct correlation has been found with drug pKa alone.

Traditional pharmacokinetic modeling techniques have not been successful in predicting or explaining drug kinetics in eggs. Newer research has centered on physiologically based pharmacokinetic (PBPK) models.[31] Most avian PBPK models have focused on meat products, but models focusing on the reproductive tract have the potential for predicting drug kinetics in egg yolks and whites.[32,33] These models incorporate plasma drug concentrations in the female and the physiology of egg development into the equations as each component forms at different rates and stages of egg development.[34] Drug absorption and subsequent concentrations reached in eggs are a function of the drug concentration in the plasma of the female bird. This is thought to be a passive diffusion into the egg, and therefore nonsaturable. As previously described, direct oral administration or injection will result in higher plasma concentrations in the bird, and therefore higher concentrations in the egg matrices, compared to medicated water and feed. The duration of exposure also plays a role in drug absorption into the eggs and is linked to the stage of egg production. Prolonged exposure can be either from repeated dosing or from environmental exposure and drug recycling from excreted waste.[35] Drug disposition into egg yolks occurs mainly during the rapid growth phase, which lasts 8 to 10 days prior to ovulation in a nonlinear fashion, taking 8 to 10 days to reach equilibrium. Egg white formation is much more rapid than yolk formation, occurring over an 8- to 10-hour period after ovulation, with albumen deposition occurring in the first 2 to 3 hours. Drug deposition therefore occurs first in the egg whites and reaches equilibrium more rapidly (within 2–3 days).[36]

Incorporating the rate of egg component formation into the equation along with the plasma concentration to which the egg is exposed allows for approximate simulation of drug concentration in eggs.[34] Further refinement of the model incorporates both pKa and protein binding of the drug.[36] In this model, lipophilicity had little effect, confirming previous studies. However, the drugs for which the PBPK models have been applied are all moderately lipophilic. Highly lipophilic drugs, such as chloramphenicol, are known to reach higher concentrations in yolks versus egg whites.[37] Moreover, they persist in eggs for an extended period, particularly following multiple dose exposures. This is likely due to persistence of these drugs in the tissue of the female bird (which could include ovarian tissue) and therefore extended exposure in the forming eggs.

These models currently have some limitations. They assume passive transfer of drug into the egg, without the potential for active/facilitated transport.[36] They also assume uniform egg production, number of oocytes in the fast-growing stage, egg weight, and proportional weights of egg yolk and albumen.[34] Additionally, any factors that affect drug pharmacokinetics in the female can also affect the extent and rate of drug deposition in the eggs.

There is little-to-no drug metabolism or elimination from nonfertilized eggs. "Clearance" of drugs from eggs is therefore expressed as number of days a drug is detected in eggs following administration to the female. This information can be used to determine egg withholding times for eggs sold or used for human consumption.

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OVERVIEW OF MODELING OPTIONS: ESTABLISHING AND OPTIMIZING AVIAN DOSING REGIMEN

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Before administering a new medication that has never been used in birds, some baseline pharmacokinetic parameters should be estimated, even if it is in a domestic poultry species, to help provide insight regarding therapeutic targets and relative safety. To establish and optimize dosing regimens, PK modeling is the gold standard. PK modeling is a subdiscipline of pharmacology that studies the characteristics of absorption, distribution, metabolism, and excretion of drugs and their metabolites in the body using mathematical equations. The basic principles behind PK modeling have been summarized by JE Riviere and is an excellent review of the fundamental core concepts.[1] PK models play an important role in optimizing dosing regimens as they can relate the administered external dose to the internal dose (e.g., plasma or tissue dose metric), which can be further correlated with the efficacy of a drug to establish pharmacokinetic/pharmacodynamic (PK/PD) relationship. Due to increasing antimicrobial resistance and the shortage of new antibiotics in veterinary medicine, the use of PK models in dosing regimen optimization of antibiotics to improve clinical outcomes of infections is becoming increasingly important.[2,3]

There are different PK modeling and analysis methods that have been used with varying degrees of frequency and for different sectors of birds. Depending on the assumption of a specific method, PK analysis methods can be separated into classical PK (i.e., noncompartmental and compartmental approaches) and PBPK methods. Depending on whether the method considers population variability, PK analysis methods can be distinguished between individual and population approaches. Classical PK methods are typically individual approaches, whereas population approaches include population PK (PopPK) and population PBPK (PopPBPK) methods. This section will outline the three modeling approaches that have been used in avian medicine. Application of modeling techniques to guide dosing protocols will also be discussed.

CLASSIC PHARMACOKINETIC MODELING (NONCOMPARTMENTAL AND COMPARTMENTAL)

The simplest classical PK method is noncompartmental analysis (NCA). Compared to classical compartmental PK methods, NCA is unique and versatile as it does not rely on an assumption of compartmental models to the data, but just connects the observed individual concentrations by linear interpolation to calculate accurate results and the results are also acceptable for bioequivalence studies. NCA is mainly used to estimate area under the curve (AUC) of the plasma or blood concentration versus time plot that represents the kinetic profile of a drug upon administration. In general, NCA is a useful first step of PK analysis to explore available data and calculate regular PK measures, such as clearance, volume of distribution, AUC, and C_{max}. NCA is the most used modeling method to estimate basic PK parameters for drugs used in avian medicine.[4–6]

Classical compartmental PK models can be divided into one- or single-compartmental and multiple (two-, three-, and so on) compartmental models (Figure 5.1). Compartmental

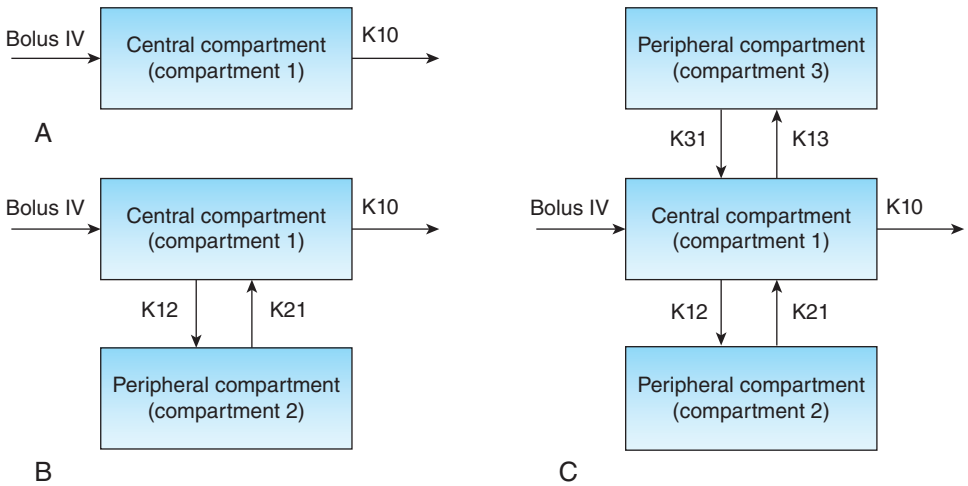


FIGURE 5.1 Schematics of common compartmental pharmacokinetic models following an IV bolus administration of a drug. (A) One-compartmental model; (B) two-compartmental model; (C) three-compartmental model. The unit of rate constants is typically per hour or per minute. *IV*, Intravenous; *K10*, elimination rate constant; *K12*, transfer rate constant from compartment 1 to compartment 2; *K13*, transfer rate constant from compartment 1 to compartment 3; *K21*, transfer rate constant from compartment 2 to compartment 1; *K31*, transfer rate constant from compartment 3 to compartment 1. The unit of rate constants is typically per hour or per minute.

PK methods assume that upon administration a drug is introduced into a central compartment, distributed throughout the central compartment for a one-compartmental model or between the central and one or more peripheral compartments for multicompartmental models, followed by elimination via metabolism and/or excretion from the central compartment.[7]

The simplest compartmental PK model is the one-compartmental PK model with IV bolus injection and first-order elimination. This model assumes that the entire body is a single kinetically homogeneous unit and upon injection a drug gets distributed throughout the body instantaneously, reaches equilibrium between the blood and tissues rapidly. In multicompartmental models, it is assumed that the body is composed of heterogeneous groups of tissues each with a different degree of blood flow and affinity for a drug and therefore different rates of distribution or elimination. The central compartment represents a combination of blood and highly perfused tissues (e.g., liver, lungs, and kidneys) that equilibrate with the blood rapidly, whereas the peripheral compartment comprises poorly perfused tissues (e.g., muscle, skin, and adipose) that equilibrate with the blood slowly. Compared to NCA, the advantage of a compartmental PK model is the ability to simulate or predict the concentration at any time following a specific exposure scenario where some PK data points are available and were used to fit the compartmental PK model. Compartmental modeling has been used to estimate PK parameters for avian species but given the study design and data-driven limitations this method has fallen out of favor. Classical compartmental modeling is less optimal for nondomestic avian species and small birds (limited blood volume) due to the need for numerous sampling times to accurately reflect drug distribution and elimination to/from various compartments.

While both NCA and compartmental PK models have merits, the common drawback of these classical PK methods is that they are data-driven empirical methods and thus have limited extrapolation capability. It is also important to note that in classical compartmental PK models, a compartment represents a virtual compartment that does not correspond to a physiological organ or tissue; thus the volume of a PK compartment is termed apparent volume of distribution rather than a physiological volume. This is a key distinction between compartmental PK and PBPK model that will be introduced further in the following sections.

PHYSIOLOGICALLY BASED PHARMACOKINETIC MODELING

PBPK Modeling and analysis is a computational simulation method that describes the absorption, distribution, metabolism, and excretion of a substance in the body based on the interrelationships among key physiological, biochemical, and physicochemical determinants using mathematical equations.[8,9] In a PBPK model, each compartment corresponds to a specific organ or tissue or a group of tissues; thus the volume of each compartment has a specific physiological meaning. PBPK models are a mechanism-based method and many species- and chemical-specific parameters can be incorporated into the model. As such, PBPK models are generally more complex and robust than classical compartmental PK models. The advantages of PBPK models include the great extrapolation capability across species, administration doses, routes, and duration, and the ability to predict concentrations of a chemical in the target organ at any time following different exposure scenarios. The shortcoming of PBPK models is that they are generally more complex, have more parameters, require more data to calibrate or validate, and are computationally more intensive. For birds, PBPK models have been developed most commonly for chemicals for domestic avian species[10–20] since the physiological parameter values (cardiac output, regional blood flow for organs/tissues, organ/tissue weights, etc.) needed to build the model can be obtained from terminal experiments and/or carcasses. In contrast, these physiological parameters are difficult to obtain for nondomestic species as a reasonable number of study subjects are needed, invasive methods are necessary for obtaining the physiological data necessary for building the model, and organ weights are obtained from healthy animal carcasses.

POPULATION-BASED PHARMACOKINETIC MODELING

The abovementioned classical compartmental PK and PBPK methods are individual approaches or deterministic methods as each model parameter has a fixed value, and the variability of model parameters within a population is not considered. Once population variability is considered, these PK and PBPK methods become population approaches and are termed PopPK and PopPBPK models, respectively.

PopPK methods analyze concentration-time datasets for a drug within a population as a whole. Traditionally, PopPK parameters were estimated by a standard two-stage "individual" approach, which fits PK data of each individual separately with a compartmental model and then combines individual parameter estimates to generate mean (population) parameters and standard deviations.[21,22] However, this traditional approach does not describe nor explain the types of population variability. This issue can be well addressed by more sophisticated PopPK methods that involve the development of nonlinear mixed-effects (NLME) models. These models are termed "nonlinear" because nonlinear kinetic equations are used and "mixed effects" implies the consideration of both fixed (for parameters that have the same values for every subject) and random (for parameters whose values are subject specific) effects. PopPK models can generate more types of results than classical compartmental PK models, including mean PopPK parameters (e.g., volume of distribution), variability between subjects (interindividual or between-subject variability, BSV), and explanations of observed variability by covariates (e.g., individual characteristics such as age, body weight, or sex), as well as residual variability (e.g., experimental uncertainty or variability). A PopPK approach is more applicable to nondomestic avian species versus a PopPBPK approach since only time versus concentration and covariate data are needed for a PopPK approach. For reasons that were mentioned previously, a PopPBPK approach is more applicable to domestic avian species.

APPLICATION OF PHARMACOKINETIC MODELING METHODS TO GUIDE DOSING REGIMEN

Classical PK, PBPK, and PopPK methods can all be used to guide dosing designs. While classical noncompartmental and compartmental PK methods are relatively simple, they are sufficient to calculate essential PK parameters, such as AUC and C_{max} following a specific dosing regimen. These parameters are useful to design an improved dosing strategy. The adequacy of different dosing regimens of antibiotics can be evaluated by different PK/PD indices (e.g., AUC/MIC, C_{max}/MIC, and time > MIC) that represent the relationship between a PK measure of exposure to the antibiotic (e.g., AUC and C_{max}) and a PD measure of bacterial susceptibility to the drug (usually the MIC).[21] For example, cephalosporins are time-dependent antibiotics and the PK/PD index of T > MIC (i.e., the percentage of a time period where the unbound drug concentration exceeds the minimum inhibitory concentration) is the best predictor of clinical success of this class of drugs.[23] Previous studies have shown that the optimal duration of T > MIC should be at least 50% of the dosage interval.[23,24] Based on this information, a recent study showed that an extra-label use of intramuscular injection of cefquinome in black swans at 2 mg/kg every 24 hours, the dose typically used in labeled species, would not be an appropriate dosing regimen in black swans to treat *Escherichia coli* as the probability of attaining T > MIC of at least 50% of the dosage interval was not attained[25]; this recommendation was based on Monte Carlo simulated data for classical PK analysis.[25] Note that while classical PK methods are useful to guide dosing, their usefulness is limited to the dose level at which PK data were collected, and it cannot be used to evaluate the appropriateness of other dosing regimens at other dose levels. While linear extrapolation can be done, this is usually of high uncertainty as it is not supported by empirical data or PK mechanisms.

Compared to classical PK methods, PopPK methods are more versatile and have various clinical applications, such as dosing evaluation/optimization of antibiotics, setting clinical breakpoints, and therapeutic drug monitoring.[21] Another strength is that PopPK methods can analyze sparse datasets from a population of animals, thereby reducing the number of samples needed for each animal thus mitigating the stress from frequent sampling on animals.[26] To develop a reliable and robust PopPK model for a particular drug in a certain species, available relevant data from a diverse source, including from different studies using different dosing regimens from different laboratories, can be included. Provided that the quality of data is acceptable (usually the richer the data are), the more robust the PopPK model is. Also, a reliable PopPK model usually undergoes an extensive validation process using different methods, such as objective functions based on likelihood (e.g., Akaike Information Criterion [AIC]), graphical comparisons, bootstrapping to estimate parameter precision, and simulation-based diagnostics (e.g., Visual Predictive Check [VPC]), as well as external validation when a new dataset becomes available after the model has been developed.[27,28] As a result, the inference range of the PopPK model is usually wider as it includes multiple datasets in the development process. Therefore, PopPK models can be used to run simulations of different dosing regimens (e.g., the same dose level with different dosing intervals, and different dose levels with same dosing interval) and predict the probabilities of target attainment (PTA) of specific PK/PD targets of different dosing regimens and a range of MICs.[29,30] These results can be used to design the optimal therapeutic regimen to treat an infectious disease against a particular bacterial agent with a certain MIC.

PopPK methods have been increasingly used to guide dosing designs in avian medicine.[26,31–34] In a recent study, a sparse-sampling design was used to collect plasma samples (one to four samples each animal) from California brown pelicans *(Pelecanus occidentalis californicus)* after a single intramuscular injection of danofloxacin at 15 mg/kg (27.45–55.95 mg each animal depending on body weight), and a PopPK model with NLME modeling was developed to characterize the PK properties of the animal population.[26] Since typical therapeutic targets for fluoroquinolones are a C_{max} greater than or equal to 10× the MIC or AUC greater than or equal to 100× the MIC,[35] this study showed that the typical value for AUC in the studied population (13.75 μg/mL × hour) was sufficient for bacteria with MIC values less than or equal to 0.12 μg/mL, and the typical C_{max} value (2.5 μg/mL) from this study was sufficient for bacteria with MIC values less than or equal to 0.25 μg/mL.[26] Based on these results, the authors concluded that the danofloxacin dose used in this study produced plasma drug concentrations that would meet traditional therapeutic targets for bacteria with MIC values less than or equal to 0.25 μg/mL in pelicans.[26]

While the PopPK method is useful to guide dosing, it is still a data-driven empirical method and thus is limited in usefulness to extrapolate beyond the inference range of the experimental data, although the range is typically wider as data from many sources with different study designs can be integrated during model development. Specifically, PopPK methods cannot be used to extrapolate across species or between different subpopulations of the same species of animals as population- and species-specific physiological information is not incorporated in the PopPK model. This issue can be ideally addressed by PBPK models.

PBPK models have many applications, including dose optimization, bioequivalence analysis, drug-drug interaction studies, drug residue predictions, food safety, and risk assessment.[36,37] In human medicine, PBPK models are useful to guide dosing special subpopulations, such as neonates, children, and pregnant women.[38–40] Typically, a PBPK model in healthy adults is developed first as there are usually more data available in adults than other age groups, and then age-specific physiological information is incorporated into the model to extrapolate it to a special subpopulation to guide dosing designs. For example, in a recent study a full PBPK model of oxycodone following IV and oral administration was developed in adults, and then was extrapolated to children by incorporating age-appropriate anatomy and physiology, such as protein binding, clearance, metabolic enzyme expression and activities, as well as metabolic rates, and this model was validated with available clinical PK datasets.[40] According to this age-dependent PBPK model, the authors found that different IV dose regimens should be given to children of different age groups compared to a standard 0.1 mg/kg in adults, and a progressive increasing dose level with age growth following oral administration is recommended for children. This approach of using PBPK models to guide dosing for subpopulations of birds has not been studied to date but holds promise for evaluating subpopulations such as breed cohort production classes.

In the field of veterinary medicine, there are several PBPK models for different chemicals in different avian species, including chickens, turkeys, pheasants, and quail.[10–20] However, these models were developed and mainly applied to predict chemical residues in poultry meat products for food safety and risk assessment. None of these models has been reported to guide dosing designs. A review of published physiological parameters for birds has been published and can serve as the basis for developing generic PBPK models for common avian species.[41] The use of PBPK models to guide dosing and drug residue avoidance in veterinary medicine, including in avian species, has not been explored and remains a gap in the application of PBPK models.

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PHARMACOLOGICAL INTERVENTION: INTERPRETATION OF ANTIMICROBIAL SUSCEPTIBILITY RESULTS

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INTRODUCTION

Antimicrobial therapy is the cornerstone of treating infectious diseases in avian species. Antimicrobials should only be used when there is evidence of an infection and ideally based on culture and susceptibility results. However, sometimes collection of a suitable sample is not possible; in these cases, one should use historical and regional susceptibility data reflecting the likely organisms for the anatomical location of the infection for empirical antimicrobial selection. In either case, response to therapy should be monitored and duration of treatment be limited as much as possible. Culture and susceptibility results, when carried out carefully, help to guide therapy through identification of causative organisms and provide in vitro measurements of the MIC in the case of broth dilution methods or zone diameter in disk diffusion that are then used for interpretation to predict in vivo efficacy.

PREANALYTICAL CONSIDERATIONS

Preanalytical considerations for culture and susceptibility testing include selection of the appropriate sample and conditions for sample storage and transport to the laboratory. These variables can impact interpretation and testing of isolates obtained. Ideally, a sterile site should be sampled that reflects the disease condition and sufficient material should be collected to allow detection of pathogens.

Isolation of pathogens from sterile sites, particularly when only one or two bacterial species are detected, likely reflects true infection rather than contamination if care was taken during sampling and transport to the laboratory. When multiple bacterial species are detected from a sample, it can be difficult to determine which, if any, are causative. In these cases, resampling may be more appropriate than susceptibility testing of all isolates. Interpretation of culture results from nonsterile sites is more challenging. If flora normally found in that site are isolated, it is difficult to distinguish whether they are causing disease or merely reflecting normal colonization. For example, isolation of streptococci or staphylococci from the nares or choanae could be considered normal flora and it would be difficult to determine if they were responsible for disease at that site or in the lower respiratory tract. Cytological findings such as the Gram stain and hematologic stains to distinguish heterophils can be helpful to determine the contribution of specific pathogens to a disease process when sampling sites with normal flora. For example, observation of bacteria and inflammation on cytology, especially if bacteria are found intracellularly, likely indicates the presence of an infection for a sterile site. The laboratory must determine which isolates to test or not test for antimicrobial susceptibility and which to consider as normal flora based on clinical signs, cytological findings, suspected condition, avian species, age, and other signalments. Optimization of sampling helps in this process.

Specimen size and storage conditions can also affect culture results. Small samples taken from normally sterile sites with few bacteria or fungi may not be adequate to detect pathogens. In general, as large a sample as can be safely collected is best. Swabs can be useful for sample collection in many avian species but if larger samples are available, they would be preferred. If anaerobic culture is desired, the swab or small (<1 mL) sample should be placed in prereduced transport medium that maintains the anaerobic environment. These media generally maintain aerobic, facultative anaerobic, and anaerobic bacteria, so a single swab, tissue, or fluid sample should be adequate for testing. However, if one or more enrichment steps are required, such as for isolation of *Salmonella* or other enteric organisms from a colonized site such as feces, more than one swab should be submitted.

Freezing of samples is not recommended as this will decrease the number of organisms in the sample and fastidious bacteria may be lost altogether. Fungi are highly susceptible to freezing so samples intended for fungal culture should never be frozen.

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Samples should be placed in a sterile container for transport to the laboratory. If samples are small, for example, a drop or two of fluid or tissue less than 0.5 cm,[1] the sample should be placed in a transport medium or a small amount of sterile water or saline should be added to maintain moisture during transport. Samples should be kept cool and shipped overnight, if possible, to the laboratory following local transportation guidelines.

The laboratory should be notified as to the suspected class(es) of pathogens since many may require specialized media to isolate. For example, both fungi and *Mycoplasma* require specific medium formulations and culture conditions for optimal isolation and the laboratory can utilize them if informed. Likewise, specific pathogens or samples from certain anatomic locations may require specialized medium or culture conditions.

SUSCEPTIBILITY TESTING

Once a culture yields one or more isolates, the laboratory has the responsibility to select the most appropriate isolates, considered causative, for susceptibility testing based on the information provided by the submitting veterinarian. Similarly, the laboratory must carry out proper testing with appropriate routine quality control testing when reporting susceptibility results.

Methods for susceptibility testing can vary between laboratories and some laboratories may use multiple techniques depending on the antimicrobial drug tested. Common methods for susceptibility testing include disk diffusion (also known as Kirby-Bauer testing), broth dilution or microdilution, and MIC gradient strips. Each method has both pros and cons and each laboratory must determine the best fit for their circumstances (Table 5.2).

In disk diffusion testing, a paper disk saturated with a standard amount of antimicrobial is placed on a standardized lawn of bacteria on Mueller-Hinton agar. The agar is then incubated up to 24 hours and the zone of inhibition is measured (Figure 5.2A). The zone diameter determines the interpretation of the isolate as susceptible, intermediately susceptible, or resistant. Disk diffusion testing does not require any specialized equipment or training and can be performed by most microbiology laboratories. It is also relatively inexpensive, and several different antimicrobials can be tested on a single agar plate depending on the plate diameter. New or different antimicrobial drugs can be incorporated easily. Although the results can be interpreted as susceptible, intermediate, or resistant, this test does not determine an MIC, so it cannot be used to refine therapy. Also, if zone diameter interpretations are not available for a particular drug:microorganism combination, it is difficult to extrapolate to estimate if a therapy would be effective. Slow-growing or some fastidious bacteria cannot be tested using this method.

For the broth dilution method, a standard number of bacteria is suspended in Mueller-Hinton broth and added to tubes (dilution) or wells (microdilution in a 96-well plate) containing different concentrations of antimicrobial drug reflecting expected plasma or tissue levels. The tube or plate is covered, incubated, and then examined for growth (Figure 5.2B). The lowest concentration of drug that inhibits visible growth of bacteria (or 80% of growth in the case of potentiated sulfa drugs) is the MIC. Advantages of the broth dilution method include the ability to determine an MIC and to test more fastidious organisms. The disadvantages of this technique are that more specialized equipment and training are needed, and it is more expensive to run. There are several commercially available panels that have set concentrations and antimicrobial drugs. Flexibility would require the creation of custom panels or in-house test development.

The MIC gradient strip also utilizes Mueller-Hinton agar inoculated with a standard number of bacteria. However, for

TABLE 5.2

Advantages and Disadvantages of Different Susceptibility Testing Methodologies

	Disk Diffusion (Kirby-Bauer)	Broth Dilution (Broth Microdilution)	Minimum Inhibitory Concentration Strips
Advantages	Inexpensive	A minimum inhibitory concentration value is generated to refine therapy	Can be used for slow-growing or some fastidious bacteria
	No specialized equipment or training necessary	Can be used for slow-growing or some fastidious bacteria	No specialized equipment or training necessary
	Easy to perform	Can test multiple antimicrobial agents on a single 96-well plate	Easy to perform
	Can test multiple antimicrobial agents on a single agar plate	Several commercially available panels available	Can test multiple antimicrobial agents on a single agar plate
	New or different antimicrobial drugs can be easily incorporated		New or different antimicrobial drugs can be easily incorporated
Disadvantages	No minimum inhibitory concentration value generated to refine therapy	More expensive to perform than disk diffusion or minimum inhibitory concentration strip method	More expensive to perform than disk diffusion
	Cannot be used for slow-growing or some fastidious bacteria	Some fastidious organisms cannot be tested	Some fastidious organisms cannot be tested
		Hard to modify commercially available panels for new or different antimicrobial drugs	

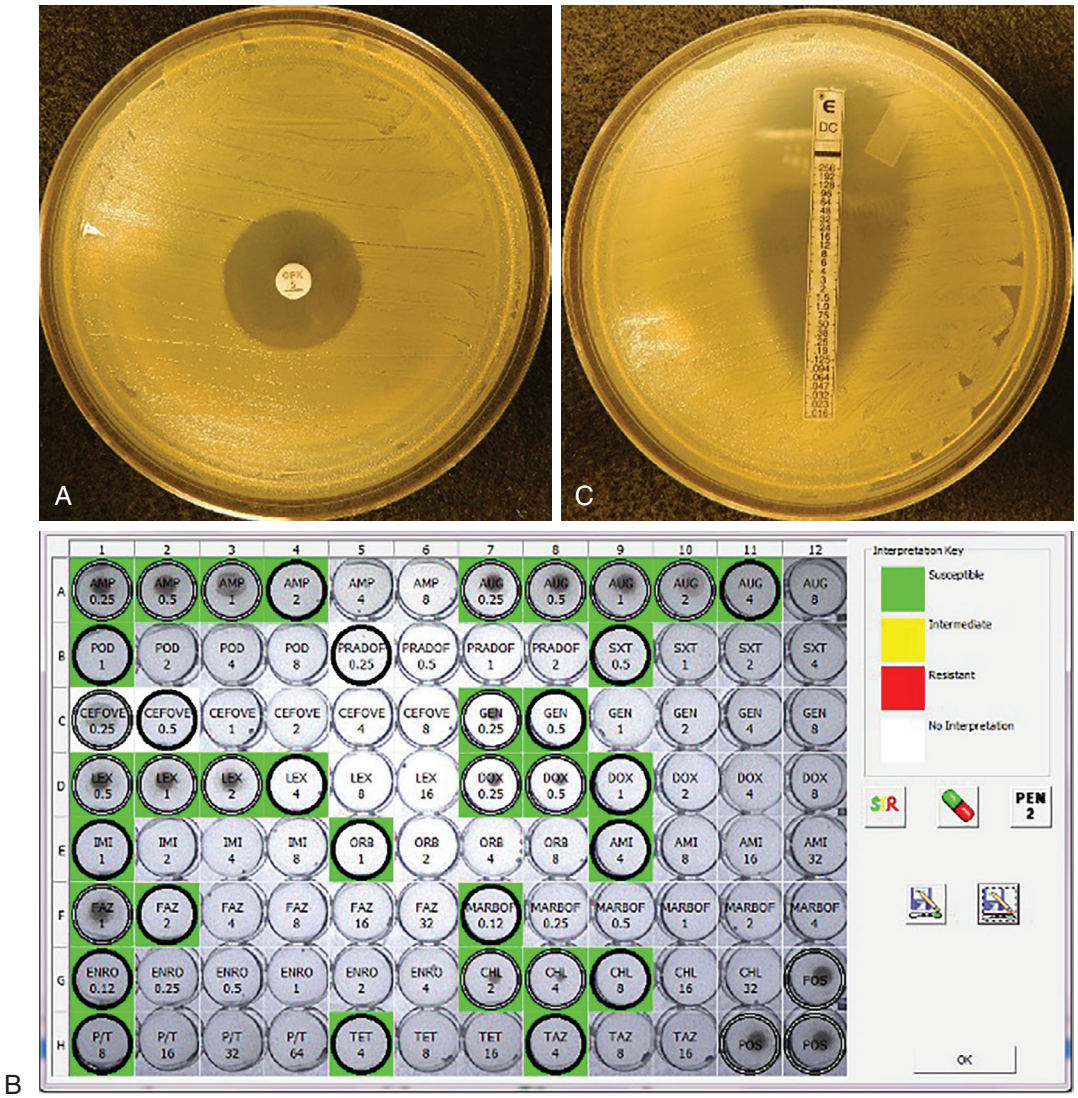


FIGURE 5.2 Examples of antimicrobial susceptibility tests. (A) Disk diffusion—*Staphylococcus aureus* tested for susceptibility to ofloxacin. The zone of inhibition is 24 cm. (B) Broth microdilution—canine *Escherichia coli* isolate showing inhibition of growth at different concentrations (numbers in μg/mL) of drug. *Green* indicates concentrations where the organism is considered susceptible to the agent. *Dark areas* within wells indicate bacterial growth. There are several antimicrobial drugs for which there is no interpretation (*no color*). (C) Minimum inhibitory concentration strip—doxycycline and *S. aureus* showing a minimum inhibitory concentration of 0.125 micrograms/mL.

this testing a strip impregnated with a gradient of antimicrobial is placed on the lawn and the agar incubated. The region where the bacterial growth contacts the strip determines the MIC (Figure 5.2C). Similar to the disk diffusion method, testing using an MIC strip is relatively easy to perform, and multiple strips can be tested on a single agar. It has the added advantage of providing an MIC. However, it is more expensive to perform than disk diffusion.

When testing is performed in a standardized manner using appropriate controls, medium, and incubation conditions according to methodology described by the manufacturer and the Clinical and Laboratories Standards Institute (CLSI)[2] or European Committee on Antimicrobial Susceptibility Testing, results can generally be compared between laboratories. Keep in mind that the MIC value may vary by a 2-fold dilution for the same isolate tested on different days.

INTERPRETATION OF SUSCEPTIBILITY RESULTS

Once a diameter of inhibition or an MIC is obtained an interpretation of these results can be made. In the context of this chapter, interpretation is a clinical interpretation reflecting whether an MIC or inhibitory zone diameter is likely to predict clinical efficacy. The three most common interpretive categories for antimicrobials used in veterinary medicine are susceptible, intermediate, and resistant. Categorization or interpretation of a zone diameter or an MIC is dependent on a

breakpoint or cutoff value. Interpretation of susceptible is "a category defined by a breakpoint that implies that isolates with an MIC at or below or a zone diameter at or above the susceptibility breakpoint are inhibited by the usually achievable concentrations of antimicrobial agent when the dosage recommended to treat the site of infection is used, resulting in likely clinical efficacy."[3] Intermediate is "a category defined by a breakpoint that includes isolates with MICs or zone diameters within the intermediate range that approach usually attainable blood and tissue levels and for which response rates may be lower than for susceptible isolates."[3] Finally, resistant is "a category defined by a breakpoint that implies that isolates with an MIC at or above or a zone diameter at or below the resistant breakpoint are not inhibited by the usually achievable concentration of the agent with normal dosage schedules and/or that demonstrate MICs or zone diameters that fall in the range in which specific microbial resistance mechanisms are likely, and clinical efficacy of the agent against the isolate has not been reliably shown in isolates with similar phenotypes."[3]

Breakpoints are determined using several criteria. The first is the pharmacokinetics (PK) of the drug at specific dosages, routes of administration, and animal species. The drug must achieve levels capable of inhibiting or killing bacteria at the site of the infection. The second is the pharmacodynamic (PD) relationship between a particular drug and pathogen where there is an antimicrobial effect on the pathogen over time. The relationship between PK and PD determines the outcome of therapy. The third input into determining the breakpoint is the epidemiological cutoff value (ECV). The ECV is determined by looking at the distribution of the MICs in a population of bacteria. Often this distribution frequency will fall into a bimodal distribution with some of the population with low MICs and some having higher MICs (Figure 5.3). The bacteria with higher MICs are presumed or proven to have acquired a resistance determinant to the drug whereas those with lower MICs have not. The drug concentration(s) between these two modes is(are) considered the ECV.

Antimicrobial drugs can be categorized by their PK/PD relationship. For example, beta-lactam drug effects on a microorganism depend on the time the drug is above the MIC and they are said to have "time-dependent" antimicrobial activity. For time-dependent inhibition the concentration of drug should exceed the MIC for at least 50% of the dosing interval.[3] On the other hand, drugs such as aminoglycosides or fluoroquinolones are considered "concentration dependent" where the maximum concentration above the MIC (C_{max}:MIC ratio) or the area under the concentration curve over 24 hours (AUC_{24}):MIC ratio determines clinical outcome. Concentration-dependent drugs such as aminoglycosides should have a C_{max}:MIC ratio of 8 to 10 whereas fluoroquinolone activity can be predicted by the 8- to 10-fold C_{max}:MIC ratio or AUC_{24}:MIC ratio of 100 to 125.[3]

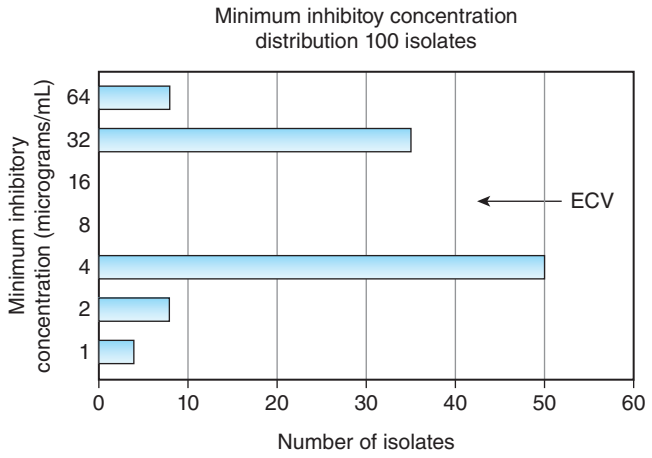


FIGURE 5.3 Theoretical example of the distribution of minimum inhibitory concentrations of 100 isolates. *ECV,* Epidemiological cutoff value.

Breakpoints are made for specific hosts (e.g., dog), drug:bug combinations (e.g., amoxicillin/clavulanic acid: *E. coli*), and often sites of infection (e.g., urine). In early CLSI publications with breakpoints, distinctions were not always made based on the site of infection, whereas now, they are more limited in scope where a breakpoint might be available for interpretation of an MIC from *Pasteurella multocida* isolated from the respiratory tract of cattle but not for an isolate from the urine. When a breakpoint is not available for one or more of these criteria, an interpretation cannot be made and may appear as "no interpretation" in a susceptibility report. Unfortunately, no interpretation categorization is now fairly common in veterinary medicine, particularly for isolates from avian species or for drugs used in an extra-label manner.

An MIC categorized as "susceptible" implies that clinical efficacy is likely when using that drug, provided that the drug reaches the site of infection. There are several reasons why an antimicrobial drug may not be as effective as predicted by a breakpoint. These include the presence of pus or necrotic debris that may bind drugs like aminoglycosides. A low tissue pH can decrease the activity of macrolides and fluoroquinolones. The presence of a surgical implant or other foreign material such as an IV or urinary catheter may provide a surface for biofilm formation by bacteria that prevents penetration of the antimicrobial agent to the organism. Decreased blood supply or the presence of a capsule in chronic infections may result in decreased concentration at the site of infection. Each of these is a consideration when planning antimicrobial therapy.

Extrapolations beyond established breakpoints should be approached cautiously. However, if a breakpoint is not available, a limited informed estimation of efficacy may be made. If there are data or breakpoints available for similar bacteria in the same host species these might be used. Unfortunately, the only avian breakpoint published in the current CLSI guidelines VET08 document is for enrofloxacin and *E. coli* in poultry.[1] However, in the United States, this drug was withdrawn for use in poultry in 2008. Still, some antimicrobial susceptibility software will report isolates as susceptible or resistant based on extrapolation from other veterinary species or humans. A general strategy is to first utilize breakpoints for related bacteria in the host species, then same bacteria in similar host species, then unrelated bacteria in the host species, and finally PK/PD relationships along with the measured MIC. Because so few breakpoints are established for any avian drug:bug combinations, one must have knowledge about the PK and PD in their avian species of interest to utilize MICs found in a susceptibility report.

Some bacteria will have intrinsic resistance to specific antimicrobial drugs or classes. For example, *Enterococcus* spp. are intrinsically resistant to cephalosporins, aminoglycosides (except high-level aminoglycoside concentrations), clindamycin, fluoroquinolones, and trimethoprim-sulfa. However, in some cases, the isolate will have a low MIC and appear susceptible in vivo.[1] The laboratory should elect to not report these results for drugs to which the organism has intrinsic resistance to avoid this pitfall and inappropriate use of antimicrobials.

COMPONENTS OF A SUSCEPTIBILITY REPORT

An antimicrobial susceptibility report will have several components including organism(s) tested, drugs reported, MIC in μg/mL, interpretation of the results when available, and any comments regarding the isolate or interpretation. Most laboratories will report the MIC when available, but in general do not report the zone diameter when disk diffusion is used. An example of a susceptibility report can be found in Figure 5.4. Generally, a report includes information regarding the animal identification, species, and owner. The report itself will detail the types of bacteria isolated and their relative amount(s) (e.g., 1+, 2+, etc., corresponding to the area of streaking where colonies are identified). Some reports will provide guidance as to drug dosages used for interpretation. The <= or ≤ symbol before the MIC indicates that there was no growth in the lowest concentration of drug tested and the > sign means that there was growth at the highest concentration of drug tested. Since the laboratory only tests concentrations that surround levels (and breakpoints) found in the blood or tissue of the host animal, a greater than symbol demonstrates that the bacterium was not inhibited at concentrations exceeding those found in the animal and it would be unlikely to be efficacious even if no interpretation can be provided. The less than or equal to symbol is more difficult to utilize unless you know something about the expected drug concentrations at the site of infection (drug distribution), duration of concentration above the MIC, and/or the AUC_{24} or C_{max}. If these are known, then the appropriate PK/PD indices can be calculated to estimate clinical efficacy.

	E. coli hemolytic		
Amoxi/Clav	Suscept		4.00
Ampicillin	Suscept		2.00
Cefazolin	Suscept		2.00
Cefovecin	No Int P		0.50
Cefpodoxime	Suscept	<=	1.00
Ceftazidime	Suscept	<=	4.00
Cephalexin	Suscept		8.00
Doxycycline	Suscept		1.00
Imipenem	Suscept	<=	1.00
Orbifloxacin	No Int P	<=	1.00
Pipera/Tazob	Suscept	<=	8.00
Tetracycline	Suscept	<=	4.00
Trim/Sulpham	Suscept	<=	0.50

FIGURE 5.4 Example of a susceptibility report of *Escherichia coli* isolated from an avian wound. The numbers represent the minimum inhibitory concentrations measured for this isolate in μg/mL. No Int P is no interpretation published, meaning that a breakpoint for that drug:bug combination has not been determined and susceptibility interpretation cannot be assigned. There are interpretations for many drugs based on host species other than avian, many of these drugs would be considered extra-label use or illegal to use in poultry.

WHAT IF YOU DO NOT HAVE CULTURE AND SUSCEPTIBILITY RESULTS?

Not uncommonly, clinical situations or economics do not allow the veterinarian to collect a sample for culture and susceptibility testing. In these cases, historical susceptibility testing results can be utilized. An antibiogram is a summary of susceptibility of organisms to specific antimicrobial drugs. It can be stratified as to animal host, anatomical location of the infection, and type of clinic. It is useful to compile an antibiogram using testing from the same hospital, or regional data if available. Ideally, information from the hospital where the animal is seen should be used for compiling. However, a single practice, particularly those seeing pet birds of many species, may not generate enough data to prepare an appropriate antibiogram. In these situations, regional data from a microbiology laboratory or compilation of results from multiple area practices can be used. If regional information is unavailable, then published studies reflecting regional or even national geographic regions can be utilized. However, one should keep in mind that these broad national data may not reflect regional antimicrobial resistance trends or practice types.

Antibiograms should be compiled at least annually utilizing clinical isolates rather than surveillance isolates,[4] although if insufficient data are available, several years can be used and a minimum of 30 isolates should be included. They should include only antimicrobial drugs routinely reported for a bacterial species and they should not utilize duplicate data; only data from the first instance of a bacterial species from a patient should be included. Isolates designated as intermediate should not be included in the percent susceptible category. The antibiogram should include information about the percentage of staphylococci that are methicillin resistant. If possible, supplemental information on the antibiogram by practice type can be useful as isolates from referral institutions may demonstrate increased resistance when compared to general practice.

ANTIMICROBIAL STEWARDSHIP

Antimicrobial resistance is a global health concern that can be attributed to overuse and misuse of antimicrobials,[5] in the human and veterinary world alike. Antimicrobial stewardship is an effort to optimize antimicrobial prescribing in such a way to improve overall outcomes while minimizing adverse consequences by considering antimicrobial, patient, and bacterial factors (see Chapter 6). In addition to minimizing antimicrobial resistance, antimicrobial stewardship is essential given the lack of novel antimicrobial development and potential for zoonotic diseases. In human medicine, the focus is using the right drug at the right dose for the right duration.[6] However, antimicrobial stewardship in veterinary species emphasizes the prevention of infection, judicious use of antimicrobials, and preventing the emergence of resistance to drugs considered important for use in human medicine.[7,8] Although human and veterinary strategies are implemented differently, it is important to recognize that both have similar goals: to use antimicrobial drugs

only when needed, to have the best outcome, and to limit harm, including development of resistant organisms.

In the United States, implementation of antimicrobial stewardship in the veterinary setting has historically led to a separation between companion animals and those that produce food for human consumption. The US Food and Drug Administration (FDA) has been taking measures to improve judicious use, such as requiring a defined duration of use on antimicrobial drug labels and requiring resistance risk evaluation of all new drugs since 2003. More recently there has been a strong push to advance progress by requiring veterinarian oversight of antimicrobials considered important in human medicine when used in livestock, which has led to reductions in the amount of antimicrobials used.[9,10] Although these regulations have been successful in limiting antimicrobial use in livestock, it may not go far enough, which is why the FDA has a 5-year target for this policy to include antimicrobials used in companion animals.[11]

Regardless of the type of animal being treated, there is a responsibility of veterinarians to limit antimicrobial drug use to situations when there is a high suspicion for or proven infection and to only use prophylactic antimicrobial regimens in cases where there is a proven historical benefit rather than purely for growth promotion. According to the Centers for Disease Control and Prevention (CDC), the *Core Elements of Antibiotic Stewardship* outlined for human medicine require practitioner commitment to implementing policies aimed at optimizing antibiotic prescribing, assessing the outcome and modifying as needed, and monitoring prescribing practices and access resources to stay up to date on prescribing practices.[12] In veterinary medicine, the International Society for Companion Animal Infectious Diseases (ISCAID) publishes guidelines for treating common infectious diseases.[13] Even though the current guidelines do not include recommendations for birds, they contain useful concepts that can be applied to avian medicine.

One aspect of optimal prescribing relies heavily on culture and susceptibility testing because these results drive the most appropriate drug selection with the highest likelihood of treatment success. Important considerations for culture and susceptibility testing include proper sample selection, judicious susceptibility testing, and interpretation of results to ensure they are consistent with the overall clinical picture. If culture and susceptibility testing are not options, before prescribing empiric antimicrobial treatment one must consider other reasons for observed physical examination findings and changes in clinical pathology. For example, an inflammatory leukogram might reflect a primary infection but rather inflammation secondary to a noninfectious process. Empiric antimicrobial therapy can delay diagnosis of the underlying process, confound further diagnostics, and promote resistance. These factors are components of risk assessment when employing empiric therapy.

To have an effective stewardship program, key elements revolve around culture and susceptibility testing, use of an appropriate antimicrobial drug at the correct dose for the shortest duration as possible, monitoring of outcomes, and de-escalation of therapy as appropriate. Additional resources regarding antimicrobial stewardship goals and implementation strategies can be found in the "Resources for Practitioners" table at the end of this chapter.

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CONSIDERATIONS WHEN PHARMACOKINETIC OR TREATMENT DATA DO NOT EXIST

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Given the large number of avian species and the paucity of pharmacokinetic data in these species, the practitioner is often unable to find published doses of drugs for a particular patient. This results in the need to interpolate or extrapolate information available in other species to that patient. Figure 5.5 depicts a pyramid approach for estimating a medication dose for avian patients utilizing different types of available data. Data available for nonavian species must be used with extreme caution, as mammalian species metabolize and eliminate drugs in a very different manner compared to birds. However, there are drugs considered "well-behaved" from a pharmacokinetic standpoint. This typically means that the drug exhibits linear kinetics over a range of doses, is not overly lipophilic, has low to moderate protein binding, undergoes minimal hepatic metabolism, and is eliminated by the kidneys using glomerular filtration. These drugs are most amenable to across and within species extrapolations. The pharmacodynamic effects also need to be similar across species. Fortunately, this means that several antibiotics are amenable to extrapolation across multiple species, as they are well-behaved and the pharmacodynamic effects relate to the concentration needed to affect the microbe being treated (MIC) at the site of infection rather than any specific receptor interaction in the animal, which may be species specific.[1] Examples of drugs that were determined to be well-behaved across veterinary and laboratory species include tetracycline, oxytetracycline, chlortetracycline, erythromycin, diazepam, prednisolone, cephapirin, ampicillin, gentamicin, apramycin, and carbenicillin.[1]

Determination of whether or not a drug is well-behaved can be done by researching available literature to determine if it demonstrates first-order kinetics, the presence of active or toxic metabolites, and knowledge of common elimination routes.

Options for finding dose information involve searching the literature as well as both domestic and foreign drug databases to discover data regarding pharmacokinetics, safety, and efficacy of the drug in the avian species of interest. If this is not available, data from other avian species, or lastly, data in mammalian species can be used. Literature should be evaluated preferentially as well. If meta-analysis or systematic reviews are available, they should be considered to have the highest quality of information, followed by randomized controlled trials, case-control studies, case reports or series, and lastly, anecdotal evidence. The drug approval process provides a vast

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% susceptible	Amikacin	Amoxi/Clav	Ampicillin	Cefazolin	Cefpdoxime	Ceftazidime	Chloramphenicol	Clindamycin	Doxycycline	Enrofloxacin	Erythromycin	Gentamicin	Imipenem	Marbofloxacin	Minocycline	Methacillin	Penicillin
By Organism																	
All Organisms (n=2651)	89.4%	65.1%	42.8%	48.2%	58.3%	89.3%	81.0%	53.4%	60.9%	69.6%	55.5%	80.1%	76.3%	73.6%	74.3%		32.4%
Gram Positive (n=1371)	80.5%	62.1%	31.7%	49.3%	46.0%		85.7%	53.4%	55.1%	56.1%	55.5%	66.9%	60.1%	60.5%	74.2%	58.7%	32.4%
Streptococcus (n=58)	43.1%	100%	98.3%	100%	96.4%						81.0%	72.4%	98.3%	95.5%	69.6%		92.3%
Corynebacterium spp. (n=104)	97.1%	53.8%	34.2%	33.0%	18.6%		93.3%	48.1%	90.4%	36.7%	53.8%	94.2%	54.8%	31.9%	94.0%		51.0%
Enterococcus spp. (n=218)		74.8%	75.2%				92.7%		39 4%		33.2%		58.3%	28.6%	46.5%		75.1%
Enterococcus faecalis (n=124)		99.2%	100%				91.1%		46.8%		40.7%		84.7%	50.0%	54.5%		100%
Enterococcus faecium (n=78)		27.4%	27.4%				93.2%		23.3%		8.2%				30.1%		27.4%
Staphylococcus spp. (n=981)	98.9%	57.6%	17.4%	58.5%	56.3%		83.3%	63.2%	55.9%	60.0%	58.9%	78.4%	58.5%	64.0%	78.5%	58.7%	17.3%
Gram Negative (n=1280)	98.9%	68.4%	54.5%	47.0%	71.0%	89.4%	76.2%		67.0%	76.4%		94.3%	93.5%	85.5%			
All Enteric organisms (n=812)	99.1%	71.4%	53.3%	56.3%	77.9%	85.2%	85.2%		82.0%	81.4%		94.8%	99.8%	82.1%			
E. coli (n=706)	99.0%	75.0%	60.8%	59.8%	78.1%	85.9%	86.3%		83.4%	82.4%		96.4%	99.7%	82.7%			
Pasteurella spp. (n=129)	100%	97.7%	87.6%		97.7%	99.2%	84.4%			94.3%		93.8%	82.9%	97.6%			
Proteus sp. (n=55)	96.4%	98.2%	87.3%	7.3%	94.5%	94 5%	76.4%		9.1%	92.7%		89.1%	69.1%	94.5%			
Pseudomonas spp. (n=149)	97.3%					97.3%				58.2%		89.3%	65.1%	78.5%			

FIGURE 5.5 Hierarchical approach for estimating a medication dose for avian patients. When extrapolating between species, it is recommended to use data from species that are most similar based on their primary food source (carnivorous, omnivorous, or herbivorous), especially for oral medications.

amount of information on efficacy, toxicity, or safety, and also pharmacokinetics, which may include dose escalation data and an assessment of linearity. Both domestic and foreign drug databases should be searched for this information as countries often have different drugs available for use. Additionally, even if the drug is not approved in an avian species, examination of approval documents may reveal preapproval clinical testing in avian species and be a useful source of information.

MULTIPLE DOSE REGIMENS

If data are reported in the literature, they are typically based on the administration of a single dose only, and to healthy animals only. However, clinical use usually involves multiple drug administrations to sick animals. For drugs that exhibit linear kinetics, multiple-dose regimens can be predicted from single-dose data. The two main parameters to adjust for a multiple dosage regimen are the drug dose and the frequency of drug administration (tau [τ] or the time interval between doses). Doubling the dose will produce higher plasma drug concentrations that are detectable for an additional half-life of the drug. Using a dose interval that is short compared to the time required for drug elimination will result in accumulation of the drug in the plasma; therefore extending the dose interval will change the degree of accumulation. The drug accumulation ratio (AR) can be calculated based on the equation:

$$AR = 1/(1 - e^{-k\tau})$$

where k is the elimination rate constant, determined by single-dose studies.

The principle of superposition assumes that early doses of drug do not affect the pharmacokinetics of subsequent doses.[2] Therefore, each dose can be considered as an independent event, and the sum of all these dosing events provides the total concentration of drug in circulation. This information can then be used to generate a concentration-time curve of any dosing regimen by calculating the total drug concentration in the body (C_{Total}) using the concentration from each individual dosing event. You can generalize this equation as the following:

$$Cn(t') = C_1(t) + C_1(t) \times [(1 - e^{-(n-1)\beta\tau})/e^{-\beta\tau} \times e^{-\beta\tau'}]$$

where t′ is the time after administration of the nth dose, t is the time after administration of the first dose, n is the number of doses, and β is the elimination rate constant. This equation relates to one-compartment, IV data. Also, if only plasma data are available, it may not accurately represent tissue concentrations. Drugs with a large Vd may have persistence and accumulation in tissues that are not reflected in the plasma.

ALLOMETRIC SCALING

Allometry is the study of size and its consequences. Allometric scaling is a method that predicts pharmacokinetic data for a drug in one species through interpolation or extrapolation from data available in other species.[3] This is possible because most pharmacokinetic parameters depend upon physiologic functions (heart rate, glomerular filtration rate [GFR], etc.) that are ultimately relatable to basal metabolic rate (BMR). Allometric scaling assumes that BMR is related to size, with larger animals having slower metabolic rates. Therefore, the equation used to mathematically define allometric scaling for a given pharmacokinetic parameter (Y) is:

$$Y = a \times BW^b$$

where a is the allometric coefficient, BW is body weight, and b is the exponent of the equation. Data can be log transformed to stabilize variance and minimize deviations from the regression line.

In order to obtain the best results from allometric scaling, interspecies comparisons should be done on animals with similar physiology that are closely related in aspects other than size, and data should be available from three or more species over three orders of magnitude in size (some smaller, some larger).[3] Therefore, when considering using allometric scaling for a specific avian species, data from other avian species over a range of weights are preferred and mammalian data are useful only for a limited number of drugs.[4] Differences in diet can also influence drug metabolism and renal elimination; therefore the best result within avian species would be obtained with birds within the same dietary group (e.g., piscivorous vs herbivorous).

Clearance is the most common parameter subjected to simple allometric scaling and is appropriate for drugs that are renally excreted by filtration (nonsaturable). These would be considered well-behaved drugs with linear kinetics over a range of doses. Drugs that are highly metabolized in the liver, particularly those with a low hepatic extraction ratio, do not scale as well due to the heterogeneity of metabolic capacity between species. Volume of distribution can also scale but is best for drugs that are confined to the plasma and extracellular space, as the volume of total body water and extracellular fluid scale with weight, whereas other fluid volumes may not. Increases in body weight typically lead to decreases in fluid volumes. Clearance and volume of distribution are both primary pharmacokinetic parameters and are affected by dose administered and absorption. Therefore, IV data must be available. Half-life is a hybrid parameter involving both Cl and Vd and not directly correlated to physiologic processes. It is sometimes used in allometric scaling and may be a more robust parameter as it is less sensitive to occasional differences in individual parameters caused by nonphysiologic reasons (e.g., laboratory variations).[1]

Both Cl and Vd can be affected by protein binding and using pharmacokinetic data derived from the unbound fraction of drug can help increase correlations between species, particularly for drugs with high hepatic extraction ratios.[3] The percentage of plasma protein binding of drugs can be a source of variability between species. There are several other factors to account for when considering using allometric scaling for dose determination. Pharmacogenetic differences in drug metabolism, saturation of metabolism or elimination processes (nonlinear kinetics), differences in urine pH, drug-induced alterations in physiologic processes (e.g., drugs that alter heart rate or renal blood flow and fluid balance), and the presence of species-specific drug transporters all influence (typically decrease) the usability of allometric scaling.

There are little published data regarding the use of allometric scaling to predict doses for avian species. Riviere et al.[1] examined allometric scaling of 44 drugs across mammalian (including laboratory animals) and avian species. A very limited number of drugs scaled across all species using half-life as the tested pharmacokinetic parameter, including ampicillin, apramycin, erythromycin, oxytetracycline, and tetracycline. Subsequent papers by this group removed avian species from analyses due to the difficulty of finding drugs that would scale based on the high degree of physiologic variation between mammals and birds.

The predictive power of allometric scaling increases when only data from avian species are included.[5] Analysis of commonly used antibacterial drugs in birds showed a significant correlation with body weight for Cl, Vd, and, in some cases, elimination half-life for fluoroquinolones, penicillins, aminoglycosides, potentiated sulfonamides, tetracyclines, and florfenicol.[6] This study looked at several avian species; however, they represented a relatively small variation in size from quail and pigeon to turkeys. Another study looked at the validity of scaling the chemotherapeutic drug carboplatin in avian species from different orders (parakeets, pigeons, chickens, ducks).[7] A significant correlation was found between body weight and elimination half-life in that study.

In summary, data from the literature can be used to determine drug doses for avian species in some instances. Well-behaved drugs that follow first-order, linear kinetics are most amenable to extrapolation across species. Drugs with nonlinear kinetics are harder to predict, particularly with multiple-dose regimens. If these drugs must be used in the absence of species-specific data, close and careful monitoring of patients is required, as well as a knowledge of the inherent toxicity associated with that drug in other species.

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ALTERNATIVES TO PHARMACOLOGICAL TREATMENT

Emily D. Richards, Lisa A. Tell



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Because the majority of the current literature and regulations regarding drug use in food-producing animals focuses on the reduction of antimicrobial medication, this section also focuses on alternatives to antimicrobial medication use; however, the concept of using medication therapy only when necessary should ideally be applied to any situation, regardless of medication class.

A useful resource describing ways to reduce antimicrobial use in animals is from the European Medicines Agency and European Food Safety Authority, which describes in detail nonpharmacological interventions and alternative therapies that can be utilized on a flock scale.[1] Nonpharmacological interventions include optimizing husbandry and maintaining biosecurity. These can be accomplished through adequate light and heat sources, providing nutritional feed, allowing for adequate space and a clean environment (regular and proper disinfection, removal of fecal material), as well as maintaining adequate protections from transmitting pathogens from one bird to another (especially between farms or homes).

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In poultry, current practices of utilizing alternatives to antimicrobials with strong scientific evidence center around the use of pre- and probiotics, organic acids, in-feed enzymes, and vaccines.[2] Other options utilized in poultry species, with some scientific evidence supporting efficacy, include phytochemicals, immune modulators, antimicrobial peptides, and bacteriophages.[2] An important consideration for these methods is the timing and duration of use, as some are only efficacious if used prior to exposure while others can be utilized continuously.[2]

COMPOUNDING CONSIDERATIONS FOR AVIAN PATIENTS

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According to the US Food and Drug Administration (FDA), compounding is the term used for "any manipulation of a drug formulation to produce a dosage form other than that manipulated and provided for in the directions for use on the approved drug product." Practically, compounding is the combining, mixing, or altering of ingredients to create a medication that is tailored to the needs of an individual patient. Oftentimes, compounded products lack safety, efficacy, and food residue data that are required for commercially approved products. Due to this lack of data, the potential exists for treatment failure and adverse reactions in treated animals or consumers of their food products. Before deciding to use a compounded product, it is important to consider your governmental regulations regarding compounding, especially for use in avian species that are food producing such as chickens and turkeys, regardless of their intended use. Although compounding may provide cost savings in some situations, compounded products should be reserved as a last line of therapy due to lack of supporting data, compared to commercially available products.

In nonproduction avian medicine, there is a shortage of FDA-approved veterinary products for avian species. Due to this, drugs are often utilized in an extra-label manner, including the use of compounded medications. Compounding allows the preparation of medication specific to the needs of the patient, such as preparation of liquid formulations, which are often times preferred for oral administration; ability to choose flavoring to increase palatability and compliance; and preparation at a concentration that is suitable given the patient's body weight.

Below are aspects to consider when utilizing compounded medications for avian patients.

1. It is important to consider whether or not the patient is a food-producing animal species or has the potential to become a food-producing animal. This is also a concern when treating wildlife where the fate of the animal is unknown or in backyard operations where antimicrobial resistance is still a potential and human exposure is a risk. Food-producing animal species tend to have stricter governmental regulations in regard to drug use in order to protect human health and avoid violative drug residues from entering the human food chain. In the United States, estimating a recommended withdrawal interval following extra-label drug use in food-producing animals is only possible if scientific data are available.[1] Since pharmacokinetic data are scarce for compounded medications, veterinarians need to consider the possibility that estimation of a withdrawal interval recommendation might not be possible following use of a compounded medication in a food-producing animal. Furthermore, if a withdrawal interval cannot be estimated, it would be best practice for the veterinarian to document the recommendation that the animal and its edible by-products never enter the human food chain.
2. When deciding to use a compounded product from an outsourced pharmacy (i.e., the veterinarian is not preparing the product in-house), it is important to use a reputable compounding pharmacy that is utilizing best compounding practices. In the United States, the *United States Pharmacopeia* (USP) establishes guidelines for best practices for both sterile and nonsterile compounding.[2] These guidelines discuss formulations, preparation techniques, storage and handling of ingredients and products, as well as establishing a beyond-use date (BUD). Veterinarians should be familiar with these guidelines when preparing compounded medications in-house, and ensure that an outsourced pharmacy follows these industry standards.
3. It is recommended that the "recipe" (or "formula") used to prepare a compounded medication has been evaluated at a minimum for potency and stability. This is to help ensure that the final product contains the desired drug concentration and will be stable for a given period of time. As a gold standard, every batch of compounded medication should be tested for potency and stability, since variability between batches is expected. However, even though interbatch variability is expected, it is likely cost and time prohibitive to test each individual batch for potency and stability; therefore, using a standard formulation that has been tested for stability and potency is the next best practice to provide for a minimum consistency between batches.
4. The source of the active ingredient is also important to consider. It is recommended that the prescribing veterinarian ask about this if the compounding is being outsourced to a pharmacy. There are generally two sources from which to obtain the active ingredient to compound from: a commercially available finished product (e.g., tablet, capsule) or a "bulk powder," or raw chemical, also known as the active pharmaceutical ingredient (API). APIs lack excipients, which can be beneficial in situations where excipients of a commercially available product have been known to cause adverse reactions in the intended patient or species. Due to the differences in purity, compounding from bulk powder runs the risk of the drug concentration differing from a commercial product. This is problematic when there is a small margin of safety between the therapeutic dose and a toxic dose. Additionally, dosing errors can occur due to potential differences in the salt forms between commercially available products and an API. For example, metronidazole is commercially formulated as the base form (~171 g/mol) and the hydrochloride salt (~207 g/mol)—both are known to be bitter, which is why the more palatable metronidazole benzoate salt form (~275 g/mol) API is preferred for compounding. However, due to the increased molecular weight

of the benzoate form, a 1.6 times dose increase is required when using the benzoate salt form in order to administer an accurate metronidazole dose.[3] Some compounding pharmacies will account for these differences in their formulations and the compounded metronidazole benzoate concentration will be listed on the label as equivalent to metronidazole hydrochloride. Although this example refers specifically to a medication that has regulatory concerns for some birds in certain countries, this concept applies to many other medications. Referring to compounding governmental regulations is necessary to confirm that use of bulk powder is permissible. Preferentially compounding from bulk powder rather than a commercially available product has been utilized to aid in cost savings, but this is not considered the best practice, and may even be considered illegal in some countries. Ideally the commercial product should be utilized as the source of active ingredient unless there are studies to show pharmacokinetics and benefit of using bulk powder. A prime example of the importance of knowing the supportive data for use of a compounded product involves the use of compounded itraconazole from bulk powder. Multiple studies in various species, including some avian species, have shown compounded itraconazole from bulk powder may not be well absorbed and therefore unable to reach therapeutic concentrations,[4–6] causing treatment failure. Unfortunately, in the United States, there are no set regulations that require a compounder to disclose up front the source of the active ingredient in the compounded preparation; however, they should provide this information upon request.

Compounded products play an important role in treating avian species as there are limited commercially approved products. The overarching message is that just because a compounded product can be prepared at the concentration/formulation/combination desired, it does not mean that it should be or that it will be effective, safe, and/or stable. Reviewing the literature on compounded formulations, pharmacokinetic data on compounded products, governmental regulations, and best compounding practices should not be overlooked in the best interest of the patient.

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MONITORING ESTABLISHED TREATMENT PLAN

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Once a treatment plan has been established and initiated, it is important to monitor the patient through the entire treatment course and illness resolution. By doing so, compliance as well as treatment effectiveness and drug safety can be evaluated. Being familiar with the characteristics of the drug(s) chosen for treatment, such as how it is metabolized, where the site of action is, and common side effects will help guide monitoring. For drugs that have a renal site of action, a downstream effect by the kidneys, or are known to be nephrotoxic (i.e., aminoglycosides), practitioners may consider checking renal biochemical parameters during treatment, especially if the patient already has some renal compromise. Renal dysfunction prior to treatment may even warrant consideration to dose adjust compared to patients with no impairment. Having knowledge that a drug might increase renal function biochemical parameters or liver enzyme activities, justifies recommendations for clients to monitor for renal or hepatic disease clinical signs, and could alter the treatment course or justify supportive care, such as addition of fluids to ensure proper hydration and support renal function.

Published resources (ideally referred or authored by experts in the field) detailing information about veterinary drugs are helpful with gathering information about key characteristics and side effects of commonly used medications. Examples of some resources can be found in the "Resources for Practitioners" table at the end of this chapter. For all medications, it is important to be aware of common side effects as well as less frequent or serious side effects that may require further evaluation. The occurrence of common side effects does not always require modification of treatment selection or plans, but serious side effects warrant further investigation. Not only is it important for the practitioner to be aware of potential complications, but it is also necessary to relay this information to the client/caregiver as they will be responsible for monitoring and observing for clinical signs of adverse side effects. If information available regarding drug side effects is not specific for the species being treated, look for information in a related species. If necessary, keep broadening the search even if you have to use an unrelated species to provide general guidance for advising the client.

In addition to monitoring for potential negative side effects of the medication(s), monitoring clinical signs, symptoms, and clinical pathology laboratory values for improvement is recommended to evaluate if treatment goals are being met. Depending on the length of the prescribed treatment course, evaluating the effectiveness of the therapeutic plan prior to completion and potentially multiple times during the treatment is optimal. This will allow necessary changes to be made sooner than later, which would achieve meeting therapeutic goals sooner and minimizing deleterious patient effects.

When monitoring an antimicrobial treatment plan, if the patient obtains the desired response and the antimicrobial(s) is/are confirmed to be susceptible, treatment de-escalation could be considered. For example, if two antibiotics (one with broad and one with narrow spectrum) are prescribed for initial treatment until culture and sensitivity results become available; if the cultured organism is found to be sensitive to both antibiotics, then reducing treatment to just the narrow spectrum antibiotic might be warranted. If the desired response is not obtained, but the antimicrobial is confirmed to be susceptible, evaluation if pharmacodynamic properties of the antimicrobial are being maximized would be important. For example, determining if a time-dependent antimicrobial is prescribed in a manner that provides sufficient time above MIC. Additionally, one should

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also consider whether the drug is reaching the site of action or assessing if another problem, that was not previously identified, is contributing to treatment failure. If the desired response is not obtained and the antimicrobial is confirmed to be resistant, then the therapeutic plan should be changed.

In certain situations, such as when drugs have a narrow therapeutic window, meaning there is little difference between the drug being effective and toxic, therapeutic drug monitoring (TDM) should be considered. TDM is the clinical practice of measuring blood/serum/plasma drug concentrations at specific time points during treatment in order to optimize drug dosage, frequency of administration, and to maintain therapeutic efficacy and safety. Ideally, TDM is used after the patient has achieved a steady-state concentration, which is generally time equivalent to three to five elimination half-lives.[1] In addition to TDM being useful for drugs with a narrow therapeutic window, TDM can also help identify therapeutic failure due to client noncompliance or a poorly prepared or ineffective compounded medication.[1,2] While TDM is not as commonly used in veterinary medicine as it is in human medicine, it has been used in animal patients for monitoring medications such as anticonvulsants (e.g., phenobarbital, bromide), antimicrobials (e.g., aminoglycosides), cardioactive drugs (e.g., digoxin, theophylline), immunosuppressive medications (e.g., cyclosporine), and thyroid supplementation.[1] In veterinary medicine, and particular avian medicine, there are multiple challenges with utilizing TDM. The biggest limitation is a lack of confirmed therapeutic concentrations for various drugs and species and unknown targeted concentrations for which above and below pose increased risks for severe side effects or treatment failure, respectively. Population pharmacokinetic studies are required to have this range defined, which are often lacking in avian medicine. Other challenges are limited availability of validated assays for the species of interest and analytical methods with minimum sample volumes that are small enough for avian patients. These considerations are important to allow proper interpretation of results. For more information and details regarding TDM refer to the "Resources for Practitioners" table for helpful references.

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Resources for Practitioners

Veterinary Drug Information Resources	*Plumb's veterinary drug handbook* (book and online reference)	Budde JA, McCluskey DM: *Plumb's veterinary drug handbook*, ed 10, 2023, Wiley Blackwell. https://plumbs.com/.
	Papich handbook of veterinary drugs (book)	Papich MG (editor): *Papich handbook of veterinary drugs*, ed 5, 2020, Elsevier, St.Louis, Missouri.
Avian Drug Formularies	*Current therapy in avian medicine and surgery* (book section)	Carpenter JW, Hawkins MG, Barron H: Appendix 1 - table of common drugs and approximate doses. In Speer BL, editor: *Current therapy in avian medicine and surgery*, 2016, W.B. Saunders, St. Louis, Missouri, pp 795–824.
	Exotic animal formulary (book)	Carpenter JW, Harms CA (editors): *Carpenter's exotic animal formulary*, ed 6, 2022, Elsevier, St.Louis, Missouri.
Compounding Resources	United States Pharmacopeia (USP) (US compounding standards website)	https://www.usp.org
	Veterinary compounding: regulations, challenges, resources (journal article)	Davidson G: Veterinary compounding: regulation, challenges, and resources, *Pharmaceutics* 9(1):5, 2017.
	International Journal of Pharmaceutical Compounding (website)	https://ijpc.com
	Society of Veterinary Hospital Pharmacists statement on compounding (website)	http://svhp.org/position-statements/
Therapeutic Drug Monitoring	Therapeutic drug monitoring (book chapter)	Boothe DM: Therapeutic drug monitoring. In *Small animal clinical pharmacology and therapeutics*, edited by DM Boothe, ed 2, 2012, Elsevier, St. Louis, Missouri, pp 385–394.
	Therapeutic drug monitoring in veterinary medicine (journal article)	Neff-Davis CA: Therapeutic drug monitoring in veterinary medicine, *Vet Clin North Am Small Anim Pract* 18(6):1287–1307, 1988.
	Monitoring recommendations (downloadable handout)	https://www.vetmed.auburn.edu/wp-content/uploads/2016/03/Recommendations-for-TDM-2016.pdf
Foreign Veterinary Drug Approvals	United States Food and Drug Administration (website)	https://animaldrugsatfda.fda.gov/adafda/views/#/search
	Canada Health (website)	https://www.canada.ca/en/health-canada/services/drugs-health-products/drug-products/drug-product-database.html

Continued

Resources for Practitioners—cont'd

	Australia Pesticides and Veterinary Medicine Authority (website)	https://portal.apvma.gov.au/pubcris
	European Union (website)—lists all countries, user will need to click on individual country's page	https://www.hma.eu/nationalcontacts_vet.html
Poultry-Specific Resources	Alabama Cooperative Extension (publication on pest management)	https://www.aces.edu/blog/tag/anr-0483/
	PoultryMed (website); international information for poultry veterinarians	http://www.poultrymed.com/Poultry/index.asp
	Poultry Science Association (website)	http://www.poultryscience.org
	US regulatory considerations for medication use in poultry (book chapter)	Tell L, Marmulak T, Martin K: Regulatory considerations for medication use in poultry. In Greenacre CB, Morishita TY, editors: *Backyard poultry medicine and surgery*, ed 2, Hoboken, NJ, 2021, Wiley Blackwell, pp 535–561.
	Alternatives to antibiotics in poultry (article)	Gadde U, Kim WH, Oh ST, et al: Alternatives to antibiotics for maximizing growth performance and feed efficiency in poultry: a review, *Anim Health Res Rev* 18(1):26–45, 2017.
Scientific Literature Search	FARAD bibliographic database (website): searchable citations for time versus concentration, food animal focused	https://cafarad.ucdavis.edu/citationsearch/
	United States safety and effectiveness studies for approved veterinary drugs	https://animaldrugsatfda.fda.gov/adafda/views/#/foiDrugSummaries
Antimicrobial Resistance	Food and Agriculture Organization of the United Nations (website)	http://www.fao.org/antimicrobial-resistance/en/
	European Medicines Agency (website)	https://www.ema.europa.eu/en/veterinary-regulatory/overview/antimicrobial-resistance-veterinary-medicine
	Australian Pesticides and Veterinary Medicines Authority (website)	https://apvma.gov.au/node/1013
	World Health Organization (website)	https://www.who.int/health-topics/antimicrobial-resistance
	World Health Organization (website): critically important antimicrobials for human medicine	https://www.who.int/publications/i/item/9789241515528
Laboratory Testing	Clinical Pharmacology Laboratory: Auburn University (website)—TDM monitoring veterinary specific	https://www.vetmed.auburn.edu/veterinarians/clinical-labs/
	University of Texas Health San Antonio (website): fungal identification, susceptibility testing, and testing of biological samples for antifungal concentrations	https://lsom.uthscsa.edu/pathology/reference-labs/fungus-testing-laboratory/

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CHAPTER 6

Antimicrobial Agents

Isabelle Langlois • Ingeborg M. van Geijlswijk • Christal G. Pollock

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The use of antimicrobial drugs made a wide range of life-threatening infections readily treatable in the 20th century. Unfortunately, any use of antimicrobial drugs can contribute to the emergence and spread of resistance. Today, antimicrobial resistance is recognized as one of the greatest global public health challenges of our time. Antimicrobial stewardship programs describe responsible measures that can help mitigate the public health crisis of antimicrobial resistance while preserving the effectiveness of available antimicrobial agents.

CORE ELEMENTS OF ANTIMICROBIAL STEWARDSHIP

Christal G. Pollock

THE "WONDER DRUG" ERA

Returning to his lab after summer vacation in 1928, Scottish microbiologist Sir Alexander Fleming noticed that dots of mold had somehow found their way into a petri dish in which he was growing *Staphylococcus* spp. Surrounding the mold was a halo in which the bacteria had died. Fleming's research on the antibacterial effects of this *Penicillium* mold was largely forgotten until the beginning of World War II when investigators at Oxford University took up this work.[1] Penicillin, the extract from this mold, was used to combat infections during the war, saving the lives of untold numbers of servicemen (Figure 6.1).[2]

Although penicillin was considered remarkable, the dawn of the "wonder drug" era technically began between 1941 and 1945 with the discovery of the first aminoglycoside, streptomycin.[3] Between 1945 and 1955, a variety of antimicrobial drugs were discovered, including bacitracin, chloramphenicol, erythromycin, neomycin, nitrofurans, polymyxin, streptogramins, and tetracyclines.[3] These antimicrobial drugs transformed both human and animal health, making a wide range of life-threatening infections readily treatable.[3]

ANTIMICROBIAL RESISTANCE CRISIS

Antimicrobial resistance mechanisms are defense strategies that microbes develop, or randomly evolve during erroneous DNA replications which are automatically selected during exposure to antibiotics, to help them survive and avoid the inhibitory or killing effects of antimicrobials.[4,5] Resistance was already recognized by Fleming in the late 1940s but became clinically important between 1961 and 1965 with the advent of methicillin and other penicillinase-resistant penicillins.[3] Increasing resistance was observed when methicillin-resistant *Staphylococcus aureus* (MRSA) emerged in human medicine between 1986 and 1990.[3] Vancomycin-resistant enterococci then surfaced in humans between 1991 and 1995 and penicillin-resistant *Streptococcus* between 1996 and 2000.[3] Between 2005 and 2010, methicillin-resistant *Staphylococcus pseudintermedius* and MRSA began to emerge in animals.[3] However, even in 30,000-year-old permafrost samples containing ancient DNA, genes encoding for resistance to beta-lactams, tetracyclines, and glycopeptide antibiotics were identified, showing that natural antimicrobials were already selected for genetic resistance information.[6]

Antimicrobial resistance is now recognized as one of the greatest global public health challenges of our time.[4,5,7,8] Rising levels of antimicrobial resistance pose serious dangers to patients, population health, food security, the environment, and economic stability worldwide.[4,9] Resistance has been found in human and veterinary communities and agriculture in every US state and every country across the globe.[4] The threat of antimicrobial resistance is most dire for natural and synthetic antibacterial antimicrobial agents, but the efficacy of antifungals, antiparasitics, and antivirals is also threatened.[7,8]

The world may now be entering a "post-antibiotic era".[3,10] We now live in a time when people around the world die from untreatable infections because of the emergence and spread of antimicrobial resistance.[4,8] A committee commissioned by the UK Prime Minister in 2014 concluded that worldwide human deaths due to antimicrobial resistance had already reached 700,000 people per year and deaths were estimated to eventually rise to 10 million annually.[7,11] In addition to increasing morbidity and mortality, resistant infections also add significant costs to healthcare systems.[7]

MECHANISMS OF ANTIMICROBIAL RESISTANCE

There are multiple mechanisms of antimicrobial resistance.[3,7] Any use of antimicrobial drugs, in people, veterinary medicine, or agriculture, can contribute to the development or selection of antimicrobial resistance,[7,11,12] particularly in common human enteric pathogens or commensals like *Salmonella* spp., *Campylobacter* spp., *Enterococcus* spp., and *Escherichia coli*.[7,11,13] Microorganisms faced with antimicrobial selection pressure enhance their

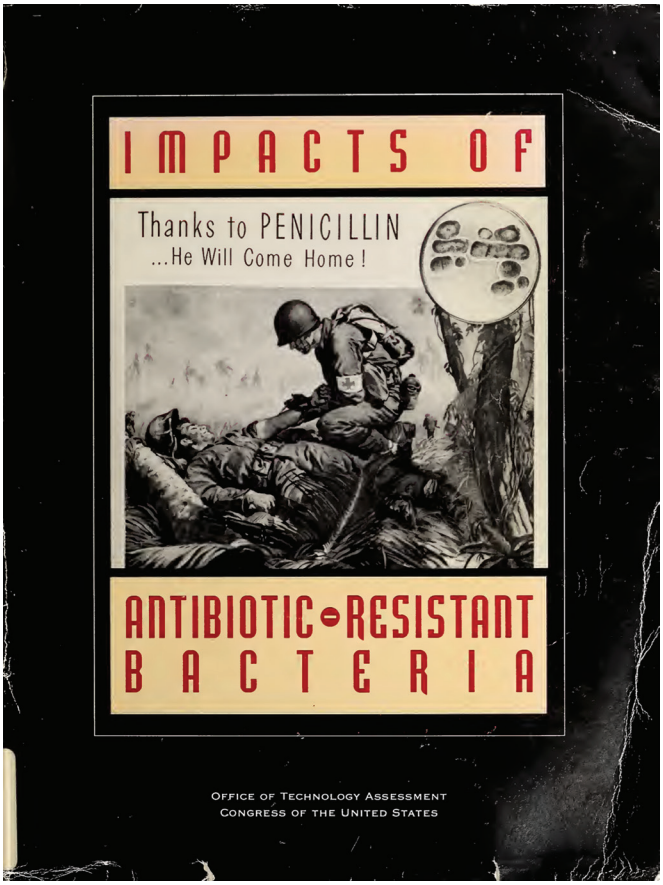


FIGURE 6.1 A century ago, clinicians had few effective medicines to treat infectious diseases. Penicillin became the foundation of a new era in treatment, but over time bacteria have demonstrated the ability to "fight back," and some antibacterial drugs are no longer effective against infectious diseases they defeated only a few years ago. Today, an understanding of antimicrobial resistance and antimicrobial stewardship is imperative to ensure most antimicrobials retain their efficacy (US Congress, Office of Technology Assessment).[10] (Original source: Life magazine advertisement, August 14, 1944. Image source: Wikimedia Commons via United States Congress, Office of Technology Assessment.)

fitness by acquiring, selecting, and expressing resistance genes and then share those genes via plasmid exchange with other microorganisms.[7,13] There is also concern that exposure to heavy metals and biocides, such as disinfectants and antiseptics in animals and environmental niches, may co-select for resistance to antimicrobials.[7,14] Furthermore, wherever antimicrobials are used, a resistant subpopulation may already be present, and selection pressure will allow these resistant microorganisms to increase in number.[13] Therefore, reservoirs of resistance exist within humans, animals, the local environments of hospitals, farms, and aquaculture environments, as well as water, soil, wildlife, and many other ecological niches, due to pollution by sewage, pharmaceutical industry waste, and manure runoff from farms (Figure 6.2).[7,15–17]

This is a true One Health issue.[8,11,18] Bacteria and their genes move relatively easily within and between humans, animals, and the environment.[6] There is evidence indicating human-to-animal transfer of resistance, particularly in horses and household pets, but also in livestock.[13,19–22] This problem is exemplified by the occurrence of human hospital-associated and community-associated MRSA infections in animals, particularly in domestic pets.[8,11,18] Resistant bacteria can also be transferred from animals to humans. Although in general one could safely state that antimicrobial use in companion animal practice is much lower than that in farm animals, the close relationship between treated animals and humans increases the risk of transfer of resistance and infection.[11,23]

ANTIMICROBIAL RESISTANCE IN VETERINARY MEDICINE

Veterinarians have come under increasing scrutiny for their antimicrobial prescribing practices.[11,12] Emergence of resistance among livestock and companion animals, and the risk of its transfer to humans, is an issue of increasing concern.[11]

Large-scale use of antimicrobials was widely adopted during the intensification and scaling-up of agriculture that occurred post–World War II.[7] By 1950, there was widespread, largely empirical, and preventive use of antimicrobials in animals, particularly food animals.[3] This practice is still considered necessary for efficient food production in some circles. In the veterinary/agricultural sectors, most antimicrobials were authorized for use before there was any systematic evaluation of the potential adverse effects on antimicrobial resistance.[7] In the United States, veterinary oversight has only been required since 2017 for the use of medically important antimicrobials in the feed and drinking water of food animals for treatment, control, or prevention of infection.[11] Globally, not all antimicrobial drug use in food animals is under veterinary prescription, despite repeated recommendations by the World Health Organization (WHO) and other responsible organizations.[3]

ANTIMICROBIAL RESISTANCE IN BIRDS

Detection of antimicrobial-resistant pathogens in birds is an important concern in the animal-human-ecosystem interface of One Health.[24–26] Among avian species, domestic chickens have been found to have the most antimicrobial resistance. This finding may partially reflect the large number of studies that have focused on antibacterial therapy in chickens, as well as the risk for resistance associated with intensive farming practices.[27–29] Multidrug-resistant bacteria and antibacterial-resistant genes have been isolated from live poultry (Table 6.1), as well as chicken carcasses and chicken meat.[27–29]

Wild birds, which inhabit a wide variety of environments and can migrate large distances, are also important reservoirs and vectors for antimicrobial resistance in the environment (Table 6.1).[37,118,121] Birds of prey are important indicators for the presence of multidrug-resistant bacteria within the ecosystem because of their predatory and/or scavenging behavior.[38,40,122,124] Urban pigeons (*Columba livia domestica*) are also an important reservoir of pathogenic and multidrug-resistant bacteria, as their close association with humans can facilitate the transfer of zoonotic disease.[26,76,78,79,81–84] Genes encoding for antimicrobial resistance have also been found in aquatic birds in diverse locations all over the world, from penguins in Antarctica to ducks in a wastewater treatment plant in Australia.[118] Bacteria resistant to beta-lactam and tetracycline drugs are also commonly found in the gut microbiome of birds, especially in scavenging and aquatic species, such as waterfowl, gulls, and waders,[45,51,52,55,59,68,118] and resistant bacteria have also

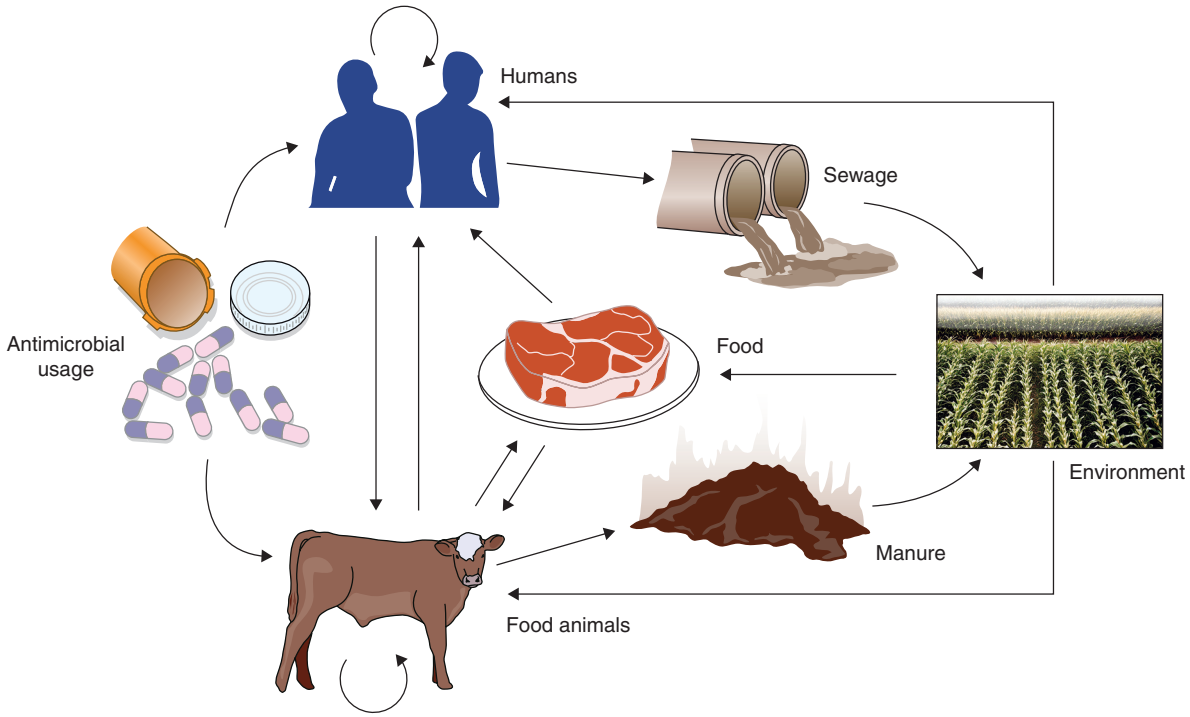


FIGURE 6.2 Diagrammatic representation of the routes of transmission of antimicrobial resistance between farm animals, the environment, and humans. (From Woolhouse ME, Ward MJ: Microbiology. Sources of antimicrobial resistance *Science* 341:1460–1461, 2013. doi: 10.1126/science.1243444.)[19]

even been isolated from songbirds that visit garden feeders.[125] Close contact with these birds, their feces, other bodily fluids, or soiled bedding material puts animal health care workers at risk for exposure to organisms with antimicrobial resistance.[121] It is imperative that workers practice sensible personal hygiene precautions when feeding or handling birds.[125]

Although the possibility of water contamination by bird feces is considered an important risk factor for the transmission of resistant bacteria,[51] exposure of migratory birds to swine waste or human waste (even if it goes through sewage treatment) is an important method for exposure and dispersal of a multidrug-resistant bacteria and resistance genes into waterways and other environmental sources.[44,45,51,118,121,126]

WHAT IS ANTIMICROBIAL STEWARDSHIP?

Antimicrobial stewardship describes responsible measures that can help mitigate the public health crisis of antimicrobial resistance while preserving the effectiveness of available antimicrobial agents.[11,24] Antimicrobial stewardship programs have been principally developed, implemented, and studied in human hospitals, but most global and national action plans to reduce antimicrobial resistance have also called for implementation of antimicrobial stewardship programs in veterinary practices.[11,12,25,26,127]

Additionally, legislative measures can support selective use of important antimicrobials. In the European Union, as a consequence of the new Veterinary Medicines Regulation (EU) 2019/6, implementing regulation (EU) 2022/1255 designating antimicrobials or groups of antimicrobials reserved for treatment of certain infections in humans has come into force on 9 February 2023. Eighteen substances/groups of substances of antibacterial drugs, 18 antiviral substances, and 1 antiprotozoal substance are specified in an Annex, which can be revised when necessary. The use in animals of medicinal products for human use that contain any of the antimicrobials or groups of antimicrobials listed in the Annex is prohibited.

No single intervention can address the problem of antimicrobial resistance. Instead a combination of interventions and approaches are used that have the potential to impact the antimicrobial resistance crisis.[3] The collective actions involved in antimicrobial stewardship have been conceptualized in a variety of ways: as a set of coordinated interventions, a strategy or program, or even as a philosophy or ethic.[26,121] Regardless of what it is called, antimicrobial stewardship requires a multifaceted approach that includes policies, guidelines, surveillance, prevalence reports, education, and audit of prescribing practices (Table 6.2).[26]

Stewardship programs typically entail a variety of strategies to prevent and control common diseases.[5,11,13,25,38] After all, if the incidence of disease can be reduced, the pressure to use antimicrobials can be similarly reduced.[13,25] When disease does occur, avoid the use of empirical antimicrobial agents whenever possible.[11] Early, appropriate diagnostic testing is an important substitute for the empirical use of antimicrobials.[13] When the use of antimicrobials cannot be circumvented, use an evidence-based approach to optimize antimicrobial selection, dosing, route, and duration of therapy (see Chapter 5). This approach will maximize clinical cure while minimizing conditions that select for resistant strains of microorganisms.[25,26,121] In some regions of the world, veterinary prescription and use of certain antimicrobial classes

Text continued on page 133

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds[30–32]

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
Accipitriformes	Cinereous vulture (*Aegypius monachus*) (F)	Spain (Huesca, Ciudad Real, Madrid, Valencia, Castellón, Cádiz)	7	*Staphylococcus aureus, Staphylococcus delphini* (tracheal swabs)
	Griffon vulture (*Gyps fulvus*) (F)	Spain (central)	1 isolate	*Enterobacter* spp. (cloacal swab)
	Griffon vulture (F)	Spain (central)	4 isolates	*Escherichia coli*
	Griffon vulture (F)	Spain (northern)	14	*E. coli* (feces)
	Griffon vulture (F)	Spain (central)	2 isolates	*Klebsiella pneumoniae*
	Griffon vulture (F)	Spain (northern)	40	*S. aureus* (nasal swab)
	Bald eagle (*Haliaeetus leucocephalus*) (F)	USA (Georgia, Alabama)	8 birds	*E. coli* (cloacal swab)
	Bonelli's eagle (*Aquila fasciata*) (F)	Spain (central)	1 isolate	*E. coli* (cloacal swab)
	Bonelli's eagle (F)	Spain (central)	1 isolate	*Enterobacter* spp. (cloacal swab)
	Bonelli's eagle (F)	Spain (central)	6 isolates	*Hafnia alvei* (cloacal swab)
	Bonelli's eagle (F)	Spain (central)	1 isolate	*Proteus mirabilis* (cloacal swab)
	Booted eagle (*Hieraaetus pennatus*) (F)	Spain (central)	2 isolates	*E. coli* (cloacal swab)
	Golden eagle (*A. chrysaetos*) (F)	Northern Spain	3 isolates	*E. coli* (feces)
	Golden eagle (F)	Spain (central)	1 isolate	*E. coli* (cloacal swab)
	Golden eagle (F)	USA (Georgia, Alabama)	1	*E. coli* (cloacal swab)
	Golden eagle (F)	Spain (central)	1 isolate	*Enterobacter* spp. (cloacal swab)
	Black kite (*Milvus migrans*) (F)	Spain (central)	2 isolates	*E. coli* (cloacal swab)
	Black kite (F)	Spain (northern)	5	*E. coli* (feces)
	Black kite (F)	Germany	13	*E. coli* (cloacal swab)
	Black kite (F)	Mongolia	19	*E. coli* (cloacal swab)
	Red kite (*Milvus milvus*) (F)	Spain (northern)	2	*E. coli* (feces)
	Red kite (F)	Germany	73	*E. coli* (cloacal swab)
	Red kite (F)	Spain	59	*Enterococcus* spp. (cloacal/rectal samples)
	Red kite (F)	Spain (Huesca, Ciudad Real, Madrid, Valencia, Castellón, Cádiz)	1	*S. delphini* (tracheal swabs)
	Broad-winged hawk (*Buteo platypterus*) (F)	USA (Georgia, Alabama)	8 birds	*E. coli* (cloacal swab)
	Common buzzard (*Buteo buteo*) (F)	Spain (central)	6 isolates	*E. coli* (cloacal swab)
	Common buzzard (F)	Germany	68	*E. coli* (cloacal swab)
	Common buzzard (F)	Portugal	33 samples	*Enterococcus faecium, E. durans* (feces)

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
PEN (6), FOX (6), ERY (1), CLI (1), TET (1)		*blaZ, tet(K), lnu(A), mecA, erm(C), vga(A), tet(M), str, fexA, tet(L)*	Six MRSA isolates	33
	cAmpC			34
	$bla_{CTX\text{-}M\text{-}1}$ (2) $bla_{CTX\text{-}M\text{-}9}$ (1) CIT pAmp (1)			34
CTX, CAZ, NA, C, CIP, GM, TB, TET, SXT	pAmpC, bla_{TEM}	SHV-12, CTX-M-1		35
	$bla_{CTX\text{-}M\text{-}1}$			34
TET			2 MRSA isolates (5%). 8 isolates were resistant to CIP (61.5%); 5 were resistant to ERY, CLI (38.5%); however, other species were surveyed including mammals and these results were not reported byspecies.	36
AMP, C, TET		*cat, tet(M)*	1 resistant isolate	37
	ESBL	CTX-M-1		34
	cAmpC ACC cAmpC CIT pAmpC			34 34 34
		CTX-M-1		34
CTX, CAZ, C, NA, CIP, TET	SBL, ESBL, pAmpC	SHV-12 (1)		35
	ESBL	CTX-M-1		34
AMP, C, TET		*Cat*		37
	cAmpC			34
	ESBL (1), $bla_{CTX\text{-}M\text{-}1}$ $bla_{SHV(1)}$			34
NA, TET (2), SXT, C, CIP	ESBL pAmpC	CTX-M-14a (1) TEM-1a (1) CTX-M-1 (1) TEM-1b (1)		35
	ESBL (22%)	CTX-M-1		38
	ESBL (7.6%)	CTX-M-9		38
CTX. CAZ, C, NA, CIP, TET, SXT	ESBL pAmpC	SHV-12		35
	ESBL (18.8%)	CTX-M-1		38
TET, ERY, KM (22.9%) STR (11.5%) GM (9%) CIP, AMP, SXT		vanCI, vanA, vanC2	VRE-i isolates (71.2%)	39
CLI		lnu(A)		33
AMP, TET		CTX-M-1	1 resistant isolate	37
	ESBL, $bla_{OXA\text{-}48}$	CTX-M-1 OXA-48		34
	ESBL ($n = 1$) 6.6%	CTX-M-1		38
TET, ERY, VAN, C, KM, quinupristin-dalfopristin		*vanA, vanC1 tet(M), tet(L) ermB, vat(E), vat(D), cat(A), aph(3)-IIIa*	VRE with an acquired mechanism of resistance (vanA genotype) was detected in 9% of samples analyzed. 27% of samples contained VRE with an intrinsic mechanism of resistance (*E. gallinarum*, vanC1)	40

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	Common buzzard (F)	Spain (central)	1 isolate	*Klebsiella pneumoniae* (cloacal swab)
	Common buzzard (F)	Spain (central)	1 isolate	*H. alvei*
	Common buzzard (F)	Portugal	16	*S. aureus* spp. (nasal)
	Cooper's hawk *(Accipiter cooperii)* (F)	USA (Georgia, Alabama)	7 birds, 6 isolates	*E. coli* (cloacal swab)
	Cooper's hawk (F)	USA (Georgia, Alabama)	4 isolates	*E. faecalis* (cloacal swab)
	Eurasian sparrowhawk (*Accipiter nisus*) (F)	Spain (central)	1 isolate	*E. coli* (cloacal swab)
	Eurasian sparrowhawk (F)	Portugal	1	*E. coli* (feces)
	European honey buzzard (*Pernis apivorus*)	Spain (central)	1	*E. coli* (cloacal swab)
	Northern goshawk (*Accipiter gentilis*) (F)	Spain (central)	1 isolate	*E. coli* (cloacal swab)
	Red-shouldered hawk (*Buteo lineatus*) (F)	USA (Georgia, Alabama)	11 birds, 9 isolates	*E. coli* (cloacal swab)
	Red-tailed hawk (*Buteo jamaicensis*) (F)	USA (Georgia, Alabama)	37 birds, 36 isolates	*E. coli* (cloacal swab)
	Red-tailed hawk (F)	USA (Georgia, Alabama)	37 birds, 28 isolates	*E. faecalis* (cloacal swab)
	Roadside hawk (*Rupornis magnirostris*) (C)	Brazil	6	*E. coli* ($n = 3$), (oropharynx)
	Roadside hawk (C)	Brazil	6	*Klebsiella oxytoca* ($n = 1$)
	Roadside hawk (C)	Brazil	6	*Salmonella* spp. ($n = 2$), *Proteus mirabilis* ($n = 2$)
	Roadside hawk (C)	Brazil	6	Coagulase-positive *Staphylococcus*
Anseriformes	Canada goose (*Branta canadensis*) (F)	USA (North Carolina)	90	*E. coli* (cloacal swabs or feces)
	Canada goose (F)	USA (Georgia)	72	*E. coli*
	Canada goose (F)	USA (Maryland)	Large flocks ($n > 200$)	*Enterococcus* spp., *E. coli* (feces)
	Canada goose (F)	USA (Ohio)	182	*S. aureus* (feces)
	Greater white-fronted goose (*Anser albifrons*) (F)	Germany (Hesse)	1	*E. coli* (feces)
	Eurasian wigeon (*Anas penelope*) (F)	Pakistan	15	*E. coli* (feces)
	Mallard duck (*Anas platyrhynchos*) (F)	Pakistan	20	*E. coli* (feces)
	Mallard (F)	Spain (central)	2 isolates	*E. coli* (cloacal swab)
	Mallard (F)	Spain (central)	1 isolate	*H. alvei* (cloacal swab)

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
	ESBL AmpC *bla*$_{OXA-48}$	CTX-M-1 OXA-48		34
	ESBL	CTX-M-1		34
PEN, TET, C		blaZ	Six birds carried *Staphylococcus* spp. (37.5%)	41
TET			1 resistant isolate	37
TET		*tet*(M), *tet*(L)	1 resistant isolate	37
	bla$_{CTX-M-9}$			34
	bla$_{CTX-M-14}$			42
	CIT pAmpC			34
	CIT pAmpC			34
AMP, C, CIP, TET		TEM-1, CTX-M-1, *cmlA, cat, gyrA, tet(M)*	2 resistant isolates	37
AMP, C, CIP, TET		TEM-1, CTX-M-1, cmlA, cat, gyrA, tet(M)	4 resistant isolates	37
TET		Tet(M), tet(L)	2 resistant isolates	37
CAZ, CTX, CPD, ATM, IPM, MEM				43
CAZ, CTX, CPD, ATM, IPM, MEM				43
CAZ, CTX, CPD, ATM				43
VAN, TEC, MET				43
TET, STR, SXT, AMP		intI1 (n = 9)	Geese lived near a swine housing facility and used swine waste lagoons and adjacent surface waters and farm fields. Most isolates (72%) recovered exhibited resistance to >1 abx 45% of these isolates (n = 25) possessed >1 abx-resistant determinant	44
FOX, AMC, CEPH			Resistant *E. coli* recovered from geese on agricultural land with no apparent contact to livestock waste had a lower proportion of resistance (19%) and only exhibited resistance to β-lactams	44
CEPH, STR, SZL			>95% of *E. coli* isolates were resistant	45
CLI, ERY, OXC, PEN			7/182 (3.8%) were MRSA and 6/182 (3.3%) were MSSA. One (7.7%) MSSA isolate was multidrug-resistant	46
	bla$_{CTX}$ *bla*$_{TEM-1}$ ESBL	CTX-M-15 *tet(A), tet(B), strA, strB* class I integron *drfA17-aadA5*		47
CTX, CAZ, AMP, DC, TET, SXT	*bla*$_{CTX-M-15}$ *bla*$_{TEM-1B}$	CTX-M-15, TEM-1B, *strA, strB, tet(A), sul2, QnrS1, dfrA14-like*		48
CTX, CAZ, SXT, AMP, DC, TET, ENR, MRB		CTX-M-15, TEM-1B, *strA,strB sul2 qnrS1, tet(A) dfrA14-like*		48
	bla$_{CTX-M-1}$ (1) *bla*$_{SHV}$ (1)			34
	ACC cAmpC			34

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	Mallard (F)	Spain (central)	1 isolate	*K. pneumoniae* (cloacal swab)
	Red-breasted merganser (*Mergus serrator*) (F)	USA (California)	1	*K. pneumoniae* (cloacal swab)
	Red-headed pochard (*Aythya americana*) (F)	Pakistan	10	*E. coli* (feces)
	Shoveler duck (*Anas clypeata*) (F)	Pakistan	15	*E. coli* (feces)
	White-face whistling duck (*Dendrocygna viduata*) (C)	Germany (zoo)	1	*S. aureus* (small intestine)
	Shoveler duck, Cotte duck, green-winged teal duck (*Anas carolinensis*) (F)	Egypt (Giza, Cairo, El-Sharkia, El-Ismailia, Port Said)	20 of each species	*E. coli, K. pneumoniae, K. oxytoca, P. aeruginosa* (feces)
	Domestic duck (*Anas platyrhynchos*), common teal (*Anas crecca*), tufted duck (*Aythya fuligula*) (C)	Bangladesh	41 wild ducks, 23 ducks, 3 geese	*E. coli* (feces or cloacal swab)
	"Farmed ducks" (C)	China	310 isolates	*Salmonella enterica* serovar Indiana
	Canada geese, mallard ducks, domestic/hybrid (free-living in a zoological setting)	USA (Ohio)	450 samples	*E. coli* (feces)
	Canada geese, mallard ducks, domestic/hybrid (free-living in a zoological setting)	USA (Ohio)	450 samples	*Salmonella typhimurium* (feces)
	"Waterfowl" (F)	Portugal	72	*E. coli* (feces)
Apodiformes	Common swift (F)	Spain (central)	1 isolate	*Citrobacter freundii*
	Common swift (*Apus apus*) (F)	Spain (central)	1 isolate	*H. alvei* (cloacal swab)
Cathartiformes	Black vulture (*Coragyps atratus*) (F)	Spain (central)	7 isolates	*E. coli* (cloacal swab)
	Black vulture (F)	USA (Georgia, Alabama)	4 birds	*E. coli* (cloacal swab)
	Black vulture (F)	Mongolia	30 birds	*E. coli* (cloacal swab)
	Turkey vulture (*Cathartes aura*) (F)	USA (Georgia, Alabama)	4 birds, 1 isolate	*E. faecalis* (cloacal swab)
Charadriiformes, Alcidae, Laridae	Common murre (*Uria aalge*) (F)	USA (California, Washington)	26	*Enterobacter cloacae* (cloacal swabs)
	Black-headed gull (*Chroicocephalus ridibundus*)	Czech Republic	157 birds, 257 isolates	*E. coli* (cloacal swabs)
	Black-headed gull (F)	Spain (central)	4 isolates	*E. coli* (cloacal swab)
	Franklin's gull (*Leucophaeus pipixcan*) (F)	Chile	370 samples	*E. coli* (feces)

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
	$bla_{CTX-M-1}$			34
AMP				49
CTX, CAZ, AMP, DC, TET, SXT		CTX-M-15, TEM-1B, *strA, strB, sul2, qnrS1, tet(A) dfrA14-like*		48
CTX, CAZ, AMP, SXT, GM, TB, ENR, MRB, TET, DC		CTX-M-15, TEM-1B, *aac(3)-IId-like, aadA2, tet(A) strA, strAlike, strB sul1, sul2 dfrA12 qnrS1, dfrA14-like*		48
TET		*tet(L)*	Zoo bird with catarrhal enteritis	50
		mcr-1 (37.5%) *mcr-2* (1.4%–12.5%)		51
TET, AMP, SXT, NA	ESBL	CTX-M CTX-M-9	53% of isolates were resistant to ~1 abx. MDR was found in 22.7% of isolates and 13.6% were resistant to 4/5 abx classes The overall prevalence of ESBL carriage was 30%	52
AMK, CTX, CIP		*rmtB, armA*		53
ERY (99.5%), PEN, LCM, BAC, ERY, vancomycin			300 *E. coli* isolates, susceptibility was tested in 194. Resistance to ERY in 99.5%	54
PEN, LCM, VAN, OXY, ERY, BAC, FEP			Eight *Salmonella* isolates were resistant	54
TET, STR, AMP, SXT (19%–35%), NA (14%), CIP (9%), AMC, GM, TB, C (4.5%–7%), CTX, ATM, CAZ	bla_{TEM-52} $bla_{CTX-M-14}$	TEM *aac(3)-II aac(3)-IV aadA, aadA2, tet(A), tet(B) sul1, sul2, sul3*	*E. coli* was recovered from 56 isolates (78%) A TEM gene was found in 22/25 AMP-resistant isolates	42
	cAmpC			34
	ACC cAmpC			34
	$bla_{CTX-M-1}$ (1) $bla_{CTX-M-9}$ (3) bla_{SHV} (1) pAmpC (1)	*mcr-1* (1)		34
TET			2 resistant isolates	37
	ESBL (27.3%)	CTX-M-9		38
TET		*tet(M)*		37
AMP				49
TET, AMP, CEPH, STR, SU, C		TEM *tetA, tetB, strA, aadA, sul1, sul2, cat*	High prevalence of ARGs and integrons in *E. coli* isolates. Total occurrence of abx resistance in isolates: a total 75/257 (29.9%)	55
	$bla_{CTX-M-1}$ (1) CIT-type pAmpC (3)			34
AMP (10%), TET (8%), STR (6%), CEF (1.1%)	ESBL	CTX-M-1 (39) CTX-M-15 (8) CTX-M-3 (2) SHV-12 (1) TEM-1 (8)	Resistance for other agents ranged between ~0.4% and 5% resistance with TGC, C, SXT, mecillinam	56

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	Franklin's gulls (*Leucophaeus pipixcan*) (F)	Canada (Winnipeg)	400 samples, 364 isolates	*E. coli* (feces)
	Glaucous gull (*Larus hyperboreus*) (F)	USA (Alaska)	150 samples, 137 isolates	*E. coli* (feces)
	Glaucous gull (F)	USA (Alaska)	7 isolates	*E. faecium* (feces)
	Glaucous gull (F)	USA (Alaska)	35 isolates	*K. pneumoniae*
	Glaucous-winged gull (*Larus glauscens*) (F)	USA (Alaska)	160 samples	*E. coli* (feces)
	Glaucous-winged gull (F)	US (Washington)	16	*K. pneumoniae, P. aeruginosa* (cloacal swab)
	Great black-backed gull (*Larus marinus*) (F)	US (northeastern)	2	*Pseudomonas* spp. (coelom)
	Herring gull (*Larus argentatus*) (F)	Lithuania	160	*E. coli* (cloacal swab)
	Herring gull	USA (northeastern)	1	*E. coli* (cloacal swab)
	Herring gull (F)	USA (northeastern)	1	*Proteus vulgaris* (oral swab)
	Herring gull (F)	USA (northeastern)	1	*Proteus penneri* (oral swab)
	Herring gull (F)	USA (northeastern)	1	*Pseudomonas* spp. (oral swab)
	Herring gull (F)	USA (northeastern)	1	Nonenteric gram-negative rod (cloacal swab)
	Herring gull (F)	USA (northeastern)	1	*Burkholderia cepacia* (cloacal swab)
	Lesser black-backed gull (*Larus fuscus*) (F)	Spain (central)	10 isolates	*E. coli* (cloacal swab)
	Lesser black-backed gull (F)	Spain (central)	1 isolate	*P. mirabilis* (cloacal swab)
	Lesser black-backed gull (F)	Spain (central)	1 isolate	*Morganella morganii* (cloacal swab)
	Ring-billed gull (*Larus delawarensis*) (F)	USA (Florida)	53 samples	*E. coli, E. cloacae* (feces)
	Silver gull (*Chroicocephalus novaehollandiae*) (F)	Australia (NSW)	504 samples	*E. coli* (cloacal swabs)

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
AMP (30%), CEF (15%)		CTX-M	364 samples yielded bacterial growth. 72 isolates (47.4%) were resistant to 1 agent; 16 (9.2%) were resistant to 3 agents	57
AMP (45%), TET (7.3%), CEF (5.8%), SXT (4.4%), STR (3.6%), NA (2.9%), C (0.7%)	ESBL bla_{CTX-M} bla_{SHV} bla_{TEM}	CTX-M SHV TEM	Population of adult gulls residing close to a landfill site. Nearly 48% of isolates were resistant to at least 1 of the drugs tested. Resistance to 2 abx was found in 13%, and resistance to >3 was found in 3% of isolates	58
		vanA esp	All isolates harbored both *vanA* and the enterococcal surface protein gene *(esp)*	58
	ESBL bla_{CTX-M} bla_{SHV} bla_{TEM}	CTX-M SHV-12, SHV-102 TEM		58
TET, SXT, CIP, GM	bla_{CMY-2} $bla_{CTX-M-15}$ bla_{TEM-1} AmpC		55% of isolates were resistant to ≥1 abx and 22% were resistant to ≥3 abx. 16% of *E. coli* isolates had ESBL and pAmpC	59
C, AMP, CEPH				49
AMC, AMP, CAR, CTF, CEPH, C, TIC, TRI			58% incidence of AR in isolates. 3 isolates resistant to 1 abx; 5 MDR isolates	60
AMP, CO, ampicillin/sulbactam		*mcr-1*	*mcr-1* gene was detected in non–ESBL-producing *E. coli* The incidence of *mcr-1* gene were very low (0.85%)	61
AMC, AMP, CAR, CEF, CEPH, PIP, TET, TIC				60
AMC, AMP, CAR, CEF, CEPH, PIP, TET, TIC			9 isolates resistant to 1 abx 13 MDR isolates 2 isolates sensitive to all abx	60
AMP, CAR, CEPH, C, PIP, TET, TIC, TRI				60
AMC, AMP, CAR, CEF, CEPH, C, PIP, TIC				60
AMC, AMK AMP, CAR, CEPH, TET, TIC, TRI				60
AMC, AMP, CAR, CAZ, CEF, CEPH, TET, TIC				60
				34
	$bla_{CTX-M-1}$ (3) $bla_{CTX-M-9}$ (3) bla_{SHV} (2) pAmpC (2)			
	$bla_{CTX-M-9}$			34
	cAmpC			34
CAZ (100%), CTX (100%), NA (90%), SXT (80%), TET (70%), CIP (70%), GM (60%), C (10%)	AmpC (29%) ESBL (14%)	CTX-M-15 CTX-M-32 CTX-M-124	Feces from 10 pelicans were also analyzed. In reporting results, no distinction was made between isolates from gulls or pelicans	62
SU (100%), AMP (99%), AMC (99%), CAZ (98%), GM (93%), STZ (93%), C (73%), TET (69%), NA (67%), STR (53%), CIP (46%), MEM (25%), IPM (6%)	bla_{IMP-4} bla_{IMP-38} bla_{IMP-26}		*E. coli* carrying ARG were obtained from 80 (40%) of gull chicks All bla_{IMP}-positive isolates showed resistance to at ≥2 abx	63

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	Silver gull	Australia (all states, excluding the Northern Territory and Capital Territory)	562 samples	*E. coli* (feces)
	Silver gull (F)	Australia	100 samples	*E. coli* (feces)
	Yellow-legged gull (*Larus michahellis*) (F)	Spain (northern)	1	*E. coli* (feces)
	Yellow-legged gull (F)	France (Marseille)	37 birds, 3 isolates	*E. coli* (feces)
	Yellow-legged gull (F)	France (Marseille)	37 birds, 1 isolate	*Cronobacter sakazakii* (feces)
	Yellow-legged gull (F)	France (Marseille)	37 birds, 4 isolates	*H. alvei* (feces)
	Yellow-legged gull (F)	France (Marseille)	37 birds, 1 isolate	*Proteus hauseri* (feces)
	Yellow-legged gull (F)	France (Marseille)	37 birds, 1 isolate	*Serratia marcescens*
	Yellow-legged gull (F)	Spain (Barcelona)	132 samples	*E. coli* (feces)
	Gulls (*Larus* spp.) (F)	Europe (Denmark, England, Latvia, the Netherlands, Poland, Portugal, Spain, Sweden)	3158 samples	*E. coli* (feces)
	Gulls (*Larus* spp.)	USA (California, Washington)	6	*E. coli* (cloacal swabs)
	Larus spp., black-headed gull (F)	Sweden (Malmø, Gothenburg)	96	*E. coli* (feces)
	Larus spp., black-headed gull (F)	Sweden (Stockholm)	283 samples	*E. coli* (feces)
	"Gulls," species unspecified (F)	Portugal (Lisbon)	88 samples, 2 isolates	*Citrobacter freundii* (feces)

ANTIMICROBIAL RESISTANCE

Phenotype	Enzymes	Genotype	Comments	Ref
AMP, CIP, CRO, FOX		*mcr-1* blaTEM (41%) CTX-M-15 (29%) CMY-2 CTX-M-27 CTX-M-14 *qnrB4, qnrB6, qnrS1*	Extended-spectrum cephalosporin-resistant (21.7%) and fluoroquinolone-resistant (23.8%) *E. coli* Single isolates of carbapenem-resistant *E. coli* and colistin-resistant *E. coli* were also detected	64
AMP (86%), CRO (55%), TET (52%), SXT (50%), CIP (48%), STR (46%), C (21%), AMC (20%), FOX (16%), GM (11%)		*qnrS* CTX-M-15	A range of ARG was present, likely representing the diverse origins of the isolates Resistant *E. coli* was detected in 53%. A small subset of isolates carried the plasmid-mediated quinolone resistance genes qnrS ($n = 4$)	65
NA, CIP, TET	*bla*$_{CMY-2}$ ESBL pAmpC	CMY-2 TEM-1b		35
AX, AMC, CRO, FEP, STX, CIP	*bla*$_{CTX-M}$ *bla*$_{TEM-1}$	CTX-M-15 CTX-M-1 TEM-1		66
AX, AMC, CRO, FF	*bla*$_{TEM-1}$	TEM-1		66
AX, AMC, CEPH, CO, FF, F				66
F, CO				66
F, CO				66
CIP (62.5%), GM (30.6%), AMK (12.5%)	*bla*$_{SHV}$ *bla*$_{CTX-M}$ *bla*$_{VIM}$ *bla*$_{KPC-2}$	SHV-12 CTX-M-15 SHV-2 TEM-1 TEM-84 CTX-M-1 CTX-M-14 VIM-1 KPC-2 CMY-2	72 samples (54.5%) were positive for *E. coli* producing extended-spectrum B-lactamase (ESPL) (51.5%), carbapenemase (1.5%), or cephamycinase (1.5%) SHV was the most prevalent group ARG (28.8%)	67
TET (19%), AMP (18%), NA, SXT			31.5% of isolates were resistant to ≥1 abx, but there was considerable variation between countries. Highest levels of resistance were observed in Spain and lowest in Denmark Highest levels of resistance observed in Spain, lowest in Denmark	68
AMP				49
CIP, GM, TET, SXT, AMC, NA, STR, C, CO, FLO, SU, TRI, KM, CAZ, FOX, TB, TGC, piperacillin/tazobactam	*bla*$_{CTX-M-15}$ (13) *bla*$_{CTX-M-14}$ (5) *bla*$_{CTX-M-55}$ (3) *bla*$_{SHV-12}$ (3) *bla*$_{CTX-M-1}$ (2) *bla*$_{CTX-M-32}$ (2) *bla*$_{CTX-M-27}$ (1)		The incidence of ESBL-producing *E. coli* ranged from 14% to 22% by region. ESBL-producing *E. coli* occurred over 3× more frequently in gulls than in humans in the same region 83% of ESBL-producing *E. coli* isolates were MDR Resistance to CIP, GM, TET, SXT common.	69
AMP, C, F, FF, NA, TET, CEF, SXT, STR	*bla*$_{CTX-M-1}$ (16) *bla*$_{CTX-M-14}$ (1) *bla*$_{TEM-1}$	CTX-M-1 CTX-M-14 SHV-12 TEM-1	*E. coli* isolated from 69% of the collected samples. One strain showed TEM-1 and CTX-M-14 resistance genes; nine strains carried only CTX-M-1, and one strain carried only SHV-12	70
TIC, CAZ, AMC, ETP, TEM, FOX, FF, GM, TB	*bla*$_{OXA-48}$ *bla*$_{GES-5}$ *bla*$_{KPC-2}$	OXA-48 KPC-2		71

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	"Gulls" (F)	Portugal (Lisbon)	88 samples, 39 isolates	*E. coli* (feces)
	"Gulls" (F)	Portugal (Berlengas Island)	57 samples	*E. coli* (feces)
	"Gulls" (F)	Portugal (Lisbon)	88 samples, 2 isolates	*Enterobacter cloacae* (feces)
	"Gulls" (F)	Portugal (Lisbon)	88 samples, 1 isolate	*Klebsiella oxytoca* (feces)
	"Gulls" (F)	Portugal (Lisbon)	88 samples, 20 isolates	*K. pneumoniae* (feces)
Burhinidae	Eurasian stone-curlew (*Burhinus oedicnemus*) (F)	Italy (Sicily)	61	*Proteus* spp., *Citrobacter* spp., *Enterobacter* spp., *Pseudomonas, Chryseobacterium* spp., *Leclercia* spp., *Hafnia* spp., *E. coli*, *Aeromonas* spp., *Serratia, Stenotrophomonas* spp. (cloacal, conjunctival, and oral swabs)
	Lesser yellowleg (*Tringa flavipes*) (F)	USA (Iowa)	1	*S. aureus* (nasal and wound samples)
Ciconiiformes	White stork (*Ciconia ciconia*) (F)	Spain (northern)	9	*E. coli* (feces)
	White stork (F)	Spain (central)	10 isolates	*E. coli* (5) (cloacal swab)
	White stork (F)	Spain (central)	1 isolate	*Enterobacter* spp. (cloacal swab)
	White stork	Spain	81	*Enterococcus* spp. (cloacal/ rectal samples)
	White stork (F)	Spain (central)	2 isolates	*P. mirabilis* (cloacal swab)

ANTIMICROBIAL RESISTANCE

Phenotype	Enzymes	Genotype	Comments	Ref
TIC (8/8), AMC (8/8), TEM (8/8), TET (4/8), ETP (2/8), CAZ (1/8), CTX (1/8), FOX (1/8), CIP (1/8), TM, GM, SXT	bla_{OXA-48} $bla_{OXA-181}$ $bla_{CTX-M-15}$ bla_{KPC-3} bla_{KPC-2} or bla_{GES-5} bla_{GES-6} bla_{CMY}	OX1-181 (54%) OXA-48 (25%) KPC-2 (n = 4, 17%) KPC-3 (n = 1) GES-5 (n = 1) GES-6 (n = 1) CTX-M-15 CTX-M-1 SHV-12 CTX-M-65 CTX-M-32 CTX-M-55 CTX-M-8 CTX-M-14 CTX-M-27	8/14 (57%) carbapenemase-positive fecal samples grew >1 type of isolate leading to a total of 24 carbapenemase-producing isolates Only two MRSA and two VRE were detected	71
TET, C, NA, CIP, STR, SXT		*cmlA* *tetA, tetB, aadA sul1, sul2, sul3* blaTEM-52 OXA-1 CTX-M-1 CTX-M-14a CTX-M-32	*E. coli* isolates were detected in 11/57 (19.3%) samples 73% of the ESBL-positive isolates of seagulls harbored the blaTEM-52 gene; 27% of isolates harbored the CTX-M	72
TIC, CAZ, AMC, CTX, FOX, CIP, SXT, GM, TEM	bla_{GES-6}	CTX-M-15		71
TIC, CAZ, AMC, ETP, MEM, TEM, FOX, CIP, SXT	bla_{KPC-2}	KPC-2		71
TIC, CAZ, CTX, CIP, SXT, TET, TM, GM, FF	bla_{OXA-48} $bla_{OXA-181}$ bla_{KPC-2} bla_{KPC-3} $bla_{CTX-M-15}$	GES-5, GES-6, OXA-48, KPC-2 CTX-M-15 CTX-M-55 SHV-2	12 isolates showed AR (50%) Only a single isolate was resistant to FF	71
CTX (90%), PEN (81%), LCM (35%), ERY (26%), GM (20.9%), VAN (18.7%), ENR (6.6%), IPM (4%), TET (2%)			A high percentage of MDR strains was found both in gram-positive (49.4%) and gram-negative isolates (34.9%). MDR levels were higher among strains isolated from the beak or eye than among fecal and cloacal strains The most abundant MDR pattern observed in gram-negative bacteria was combined resistance to aminoglycosides, penicillins, polymyxins while in gram-positive was combined resistance to cephalosporins, penicillins, lincosamides	73
OXC, ERY, CLI, LEV			MRSA isolate obtained	74
CTX, CAZ, TET, NA, C, CIP, SXT, GM, TB	SBL pAmpC	SHV-12 (3), CTX-M-1 (1) *sul3, tet(A) aac(3)-II*	This study revealed a high percentage of birds as carriers of ESBL/pAmpC *E. coli* isolates in Spain, highlighting the elevated rate among storks, kites, and vultures	35
	bla_{CTX-M}-1 (5) $bla_{CTX-M-9}$ (3) ACC pAmpC (2) CIT pAmpC (2) bla_{SHV} (1)			34
	cAmpC (1)			34
VAN			VRE-i isolates in white storks (46.9%)	39
	CIT pAmpC (2)			34

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	White stork (*Ciconia ciconia*)	Spain	92	*S. aureus* (trachea)
Columbiformes	Pigeon (C/F)	Poland (Lublin)	108 homing pigeons, 72 wild birds	*Campylobacter* spp. (cloacal swabs from homing pigeons, feces from wild birds)
	Pigeon (*Columba livia* f. *urbana*) (F)	France (Marseille)	71 birds, 1 isolate	*E. coli* (feces)
	Pigeon (F)	Australia	16	*E. coli* (feces)
	Pigeon (F)	Brazil (Juiz de Fora)	100	*E. coli* (feces)
	Pigeon (F)	South Africa (Pretoria, Johannesburg)	30 samples, 130 isolates	*E. coli* (feces)
	Pigeon (F)	Brazil (São Paulo)	107	*E. coli* (cloacal swabs)
	Pigeon (F)	Czech Republic (Brno)	247 samples	*E. coli* (cloacal swabs)
	European turtle dove (*Streptopelia turtur*) (F)	Spain (central)	1 isolate	*E. coli* (cloacal swab)
	Pigeon (F)	Germany (Hesse)	1	*E. coli* (heart)
	Pigeon (C/F)	Brazil	30 isolates	*E. coli*
	Pigeon (C)	Poland	Birds originated from 634 flocks	*E. coli*
	Pigeon (F)	Czech Republic	247 samples	*Enterococcus* spp. (cloacal swabs)
	Pigeon (F)	France (Marseille)	71 birds, 1 isolate	*H. alvei* (feces)
	Pigeon (F)	France (Marseille)	71 birds, 1 isolate	*Panteoa ananatis* (feces)
	Pigeon (C)	Egypt (Sharkia)	50	*Salmonella* spp.
	Pigeon (F)	USA (Iowa)	2	*S. aureus*
	Pigeon (C)	Poland	Birds originated from 634 flocks	*Staphylococcus* spp.
	Pigeon (F)	Costa Rica (Tres Ríos, San José)	141	Bacteria not isolated (feces)

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
PEN, TET, C, ERY, STR, CLI, FUS, OXC, FOX		mecA	*S. aureus* was identified in 32 samples (34.8%) from nestlings from two landfill-associated and two natural habitat colonies. Prevalence was higher in nestlings from landfills (55.8%). Three birds from landfill colonies carried MRSA	75
STR (>50%), AMP (40%), AX (37%), ERY, TET			35 strains were isolated: *Campylobacter jejuni* (27), *Campylobacter coli* (8)	76
AX, CEPH, CRO, FEP	bla_{CTX-M}	CTX-M-15		66
AMP (87.5%), CRO (62.5%), CIP (62.5%), STR (56%), TET (56%), SXT (56%), C (18.7%), GM (6%)		CTX-M-15 (25%)	Resistant *E. coli* detected in 9.8% of pigeons	65
AMK (36.8%), AMP (7.8%)		CTX-M-15	*E. coli* detected at an overall rate of 12.1%; resistance to ≥1 of abx in 37.9% Four CTX-M-15–producing strains from four different birds	77
AMK (24%), GM (23.6%), AMP (22.8%), TET (17%), AX (16.9%)			A relatively higher proportion of isolates from pigeon feces (67%) were AR than those from roof-harvested rainwater (53%) The most abundant resistance phenotype: AK-GM-CIP-TET (6.8%)	78
		CTX-M-2 CTX-M-8		79
STR, TET, SU, CIP		*qnrA, qnrB, qnrC, qnrS, qnrD*	*E. coli* isolates were found in 203 samples. AR was recorded in 3 isolates. No ESBL-producing *E. coli* isolates were detected	80
	$bla_{CTX-M-1}$ group ESBL			34
	$bla_{CTX-M-15}$	CTX, TEM-1 *tet(A), tet(B) strA, strB,* class I integron *drfA17-aadA5*		47
		CTX-M-8	The frequency of MDR *E. coli* in captive pigeons was relatively high	81
TET, AX				82
TET (20%), GM (9%), ERY (9%), AMP (3%), VAN (3%)		*tetM, tetK, tetL, tetO, ermB* *vanA, vanC1, ant(4′)-Ia, aac(6′)aph(2), aph(3″)-IIIa, pbp5*	A total of 143 enterococci were isolated: *E. faecalis* ($n = 36$), *E. faecium* (27), *E. durans* (19), *E. hirae* (17), *E. mundtii* (17), *E. gallinarum* (12), *E. casseliflavus* (12), *E. columbae* (3). Resistance to abx (1–4) was detected in 45 isolates (31%)	80
AX, AMC CEPH, FF, F, CO				66
AX, AMC, KF, FF, F, CO				66
STR, AX, AMC, AMP, CAZ	bla_{TEM-1}	Class1 integron	Samples were collected from 50 freshly dead squabs, 40 freshly dead adults, 60 diseased adults, and 100 apparently healthy adults Class1 integron were characterized in 70% of squab isolates, 42.9% in adults, and 14.3% in the pigeon environment	83
TET			Resistance was detected in one bird	74
OXY, quinolones, MET (5%)			Beta-hemolytic coagulase-positive staphylococci	82
		sulI (30%), *sulII, tet(Q)* (13%–17%) TEM (4%) *tet(A), catI, qnrS*	The average of antimicrobial resistance genes detected per pigeon was 2	84

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
Cuculiformes	Common cuckoo (*Cuculus canorus*) (F)	Spain (northern)	250	*E. coli* (feces)
	Great spotted cuckoo (*Clamator glandarius*) (F)	Spain (central)		*K. pneumoniae* (cloacal swab)
Falconiformes	American kestrel (*Falco sparverius*) (F)	USA (Georgia, Alabama)	1	*E. coli* (cloacal swab)
	Caracará (*Caracara plancus*) (C)	Brazil	6	*E. coli, Salmonella* spp., *K. oxytoca, K. pneumoniae* (oropharynx)
	Caracará (C)	Brazil	6	Coagulase-positive *Staphylococcus*
	Common kestrel (*Falco tinnunculus*) (F)	Portugal	1	*E. coli* (feces)
	Common kestrel (F)	Spain (central)	1 isolate	*E. coli* (cloacal swab)
	Common kestrel (F)	Spain (central)	2 isolates	*Enterobacter* spp. (cloacal swab)
	Lesser kestrel (*Falco naumanni*) (F)	Spain (central)	2 isolates	*K. pneumoniae* (cloacal swab)
	Peregrine falcon (*Falco peregrinus*) (F)	Spain (central)	1 isolate	*H. alvei* (cloacal swab)
Galliformes	Chicken (C)	China (Shandong)	146	*Acinetobacter lwoffii* (cloacal swabs)
	Chicken (C)	The Netherlands	16	*Campylobacter* spp.
	Chicken (C)	Czech Republic	189 samples	*Campylobacter* spp. (cloacal swab)
	Chicken (C)	Europe		*Campylobacter coli*
	Chicken, turkey (C)	USA	294 isolates	*C. coli*
	Chicken (C)	USA	100	*C. jejuni* (feces)
	Chicken (C)	USA (Georgia)	175	*C. jejuni, C. coli*

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
CTX, CAZ, C, NA, CIP, GM, TB, TET	*bla*$_{SHV}$ ESBL pAmpC	SHV-12 (3) CTX-M-1 (1)		35
	bla$_{CTX-M-1}$ (1)			35
AMP, C, CIP, TET		TEM-1, CTX-M-1 *cmlA, cat, gyrA, tet(M)*		37
CAZ, CTZ, CPD, ATM	ESBL		Resistance profile: ESBL in three birds; resistance to third-generation cephalosporins in a fourth bird	43
MET, TEC, VAN				43
	bla$_{CTX-M-14}$			42
	CIT pAmpC (1)			34
	bla$_{CTX-M-1}$ (1)			34
	bla$_{CTX-M-9}$ (1)			
	bla$_{CTX-M-1}$ (1)			34
	bla$_{OXA-48}$ (1)			
	AmpC			
	ACC cAmpC			34
IPM, most beta-lactams		bla$_{NDM-1}$		85
ENR		*gyrA*	1-day-old Ross broiler chicks treated with ENR (50 ppm in drinking water for 21 days) rapidly reduced the fecal counts of *E. coli* below the detection limit and did not induce resistance With *C. jejuni,* ENR treatment quickly selected for high incidence of fluoroquinolone-resistant strains	86
CIP (72%), AMP (26%), STR (9%), ERY (6%), oxolinic acid			92 isolates of *Campylobacter* spp. were obtained (48.7%). *C. jejuni* in 87 cases (46.0%) and *C. coli* in 5 cases (2.7%)	87
Macrolides			Macrolide resistance, ranging from 15% to 80%, was observed in *C. coli* isolated from chickens and swine	88
Resistance among conventionally raised turkeys: ERY (80%), KN (76%), AMP (31%)			>40% of *C. coli*, isolated from turkey (and swine) in the United States were resistant <2% of *Campylobacter* strains isolated from organically raised poultry were resistant to fluoroquinolones, while 46% and 67% of *Campylobacter* isolates from conventionally raised broilers and turkeys were resistant to these abx	88,89
CIP			ENR or sarafloxacin given at 40 μg/mL in drinking water for 5 days and then AR developed rapidly and persisted	90
ERY			Across *Campylobacter* spp., ERY resistance in broiler chicks was observed at a higher frequency when tylosin was administered at subtherapeutic levels (62.7%) than at therapeutic levels (11.4%) Subtherapeutic administration resulted in the recovery of 83.3 and 56.1% ERY-resistant isolates compared with only 33.3 and 7.9% of the isolates expressing ERY resistance following administration of therapeutic concentrations for *C. coli* and *C. jejuni*, respectively	91

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	Chicken (C)	North America		*C. jejuni, C. coli*
	Chicken (C)	USA (Iowa, Tennessee)	20	*C. jejuni, C. coli*
	Domestic chicken (*Gallus gallus domesticus*) (C)	Bangladesh	29	*E. coli* (feces or cloacal swab)
	Chicken (C)	Portugal	20	*E. coli* (feces)
	Chicken (C)	China (Shandong)	78 isolates	*E. coli* (feces)
	Chicken (C)	France (Marseille)	28 birds, 1 isolate	*E. coli* (feces)
	Chicken (C)	Algeria	124 isolates	*E. coli* (feces)
	Chicken (C)	China (Shandong)	1	*E. coli, Cronobacter sakazakii*
	Chicken (C)	France (Marseille)	28 birds, 2 isolates	*H. alvei* (feces)
	Chicken (C)	Egypt	100	*K. pneumoniae* (feces, necropsy)
	Chicken (C)	France (Marseille)	28 birds, 1 isolate	*P. auruginosa* (feces)
	Chicken (C)	France (Marseille)	28 birds, 1 isolate	*Providencia alcalifaciens* (feces)
	Chicken (C)	Argentina (Entre Ríos)	657 birds, 4 isolates	*Salmonella* spp. (cloacal swab)
	Chicken (C)	Germany (Bavaria)	799 swabs, 800 eggs	*Salmonella* spp., *Campylobacter* spp., *E. coli* (cloacal swabs, egg contents, and shells)
	Chicken (C)	Algeria	503	Bacteria not isolated (feces)
	Red-legged partridge (*Alectoris rufa*) (F)	Spain	127	*Enterococcus* spp. (cloacal/rectal samples)

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
ERY			Prevalence of ERY resistance among *Campylobacter* strains isolated from broiler chickens has been reported at 10% or lower	88
ERY			Macrolide use (single or multiple treatments with medicated water containing TY 0.53 g/L) did not qualify for ERY-resistant mutations in treated birds TY in feed at a growth-promoting dose (0.05 g/kg feed) caused ERY-resistant mutants to emerge after prolonged exposure to abx	92
TET, AMP, SXT, NA		CTX-M	Free range chickens	52
STR (65%), SU (51%), TRI (40%), SPT, TET, neomycin		TEM-52, SHV-2 CTX-M-1	Enterobacteriaceae (mostly *E. coli*) were identified in 10% of feces from healthy chickens. Resistance to at least 1 abx was observed in 113 isolates	29
CTX (100%), cefquinome (100%), CTF (98.7%), DC (97.4%), TET (97.4%), NA (97.4%), KM (96.2%), FLO (93.6%), ENR (89.7), CIP (83%), STR (83%), GM (78%), FF (78%), LEV (74%), CO (73%), MEM (47%), AMK (34.6%)		*mcr-1*	Isolates were collected from diseased chickens showing signs of diarrhea. About 53 isolates (67.9%) harbored *mcr-1*	93
AX, AMC, CRO, FEP, STX, GM	bla_{TEM-1}	TEM-1	*E. coli* was isolated from 25% of birds. MDR bacteria were also isolated	66
DC (97%), SU (94.9%), ENR (92%), AMP (89.7%), ofloxacin (84.6%), FLO (82%), SPT (61.5%), TET, SXT			About 92 avian pathogenic *E. coli* (APEC) isolates were recovered from broiler chickens with clinical signs and lesions of colibacillosis	94
CBP		*mcr-1* NDM-9	CBP-resistant *C. sakazakii* and *mcr-1* in *E. coli* were present in the same "diseased" chicken	95
AX, AMC, CEPH, CO				66
CBP		*kpc* OXA-48 NDM	*K. pneumoniae* was isolated from 35% from broilers with signs of respiratory disease. Fifteen of these isolates were CBP-resistant	96
AX, CEPH, CRO, F, STX	*bla*TEM	TEM-1		66
F, CO				66
ERY			Four serovars were isolated (*Salmonella ser.* Lille, *S. ser.* Newport, *S. ser.* Enteritidis, and *S. ser.* Rissen)	97
SPT			Laying hens were evaluated in both organic and conventional flocks *E. coli* from organic layers showed significant lower resistance rates All tested *Salmonella* ($n = 23$) proved to be resistant to SPT	98
		TEM ($n = 128$, 25.4%) SHV-128 ($n = 83$, 16.5%) CTX-M ($n = 46$, 9%) OXA-58		99
TET (66%), ERY (46%), KM (22.9%), STR (11.5%), GM (9.4%), VAN			Incidence of VRE-i isolates identified in red-legged partridges: 7.9%	39

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	Coturnix quail, (*Coturnix japonica*) (F)	USA (Georgia)	1 isolate	*Salmonella enterica* serovar Typhimurium (Copenhagen) (necropsy)
	Quail	USA (Georgia)	1 isolate	*Salmonella enterica* serovar Typhimurium (Copenhagen) (necropsy)
	Turkey (F)	USA (West Virginia)	1 of 264 bacterial cultures	*Salmonella enterica* serovar Java (necropsy)
	"Poultry"	Bangladesh (Savar, Dhaka)	106 isolates	*Enterobacter* spp. (cloacal swabs)
	"Poultry"			*E. coli, Salmonella* spp., *Acinetobacter* spp.
Gaviiformes, Gaviidae	Pacific loon (*Gavia pacifica*) (F)	USA (California)	1	*Enterobacter cloacae, Acinetobacter baumannii, P. aeruginosa* (cloacal swab)
Gruiformes	Eurasian coot (*Fulica atra*) (F)	Pakistan (Punjab)	60	*E. coli* (feces)
	Demoiselle crane (*Anthropoides virgo*) (F)	Mongolia	13	*E. coli* (cloacal swab)
Passeriformes	Finch (C)	Japan	53 strains	*E. coli*
	"Common bengal" (C)	Japan	6 strains	*E. coli*
Corvidae	American crow (*Corvus brachyrhynchos*)	Canada (5 roosting sites across Canada)	449 samples	*E. coli, Klebsiella* spp. (feces)
	Common raven (*Corvus corax*)	Spain (central)	1 isolate	*E. coli*
	Rook (*Corvus frugilegus*) (F)	Austria	54 migratory birds, 102 "resident" birds	*Enterobacter* spp., *E. coli*, *S. aureus* (feces)
Fringillidae	Common chaffinch (*Fringilla coelebs*) (F)	Scotland (Dumfries and Galloway)	1	*S. aureus*
	American goldfinch (*Spinus tristis*) (F)	USA (Kentucky)	1 isolate	*Salmonella enterica* serovar Typhimurium (Copenhagen) (necropsy)
Hirundinidae	Red-rumped swallow (*Cecropis daurica*) (F)	Spain (central)	1 isolate	*Enterobacter* spp. (cloacal swab)
Icteridae	Carib grackle (*Quiscalus lugubris*)	Guadeloupe and nearby islands	4 birds	*E. coli*

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
AMP, KM, STR, SU				100
		intI1 *aadA1*		
AMP, KM, STR, SU, TET			In a previous study, 32% of the *Salmonella* isolates from wild birds submitted to the Southeastern Cooperative Wildlife Disease Study were resistant to SU, and 18.1% were resistant to SU and STR	100
		intI1 *merA* *aadA1*		
		intI1		100
PEN (100%), rifampicin (100%), AMP (94%), CLI (94%), ERY (94%), VAN (88.9%), SU (72%), IPM (66.6%), STR (55.6%), F (33%), DC (33%), TET (33%), FEP (11%), GM (5.6%)				101
		vanA *sull*		
	bla_{VIM-1} bla_{OXA-23} bla_{NDM-1}		Carbapenemase-producing *E. coli*, *Salmonella* spp., and *Acinetobacter* spp. have been reported in poultry	102
CAR, C				49
CTX (12), CAZ (12), AMP (12), DC (11), TET (11), SXT (11), CO (1), GM (1), TB (1), C (1), PO (1), ENR (1), MRB (1)		CTX-M-15, TEM-1B *strA,strB sul2, qnrS1 dfrA14-like tet(A), tet(A)-like mcr-1*	First detection of *mcr-1* was in CO-resistant, ESBL-producing *E. coli*	48
	ESBL $n = 1$ (16.6%)	CTX-M-9		38
OXY, C, SA, SM, APC, KM			Imported into Japan from Thailand, Senegal; tested in quarantine	103
OXY, C, SM, SA			Imported into Japan from Taiwan; tested in quarantine	103
		qnrS1, qnrB19, qnrC, qnrB19	Low prevalence of *E. coli* containing QNR (1.3%, 6/449) and other ARGs	104
		oqxAB aac(6')-Ib-cr	One isolate of *K. pneumoniae* contained the plasmid-mediated ARG, qnrB19	
	$bla_{CTX-M-9}$			34
TET (85%), SXT (62%), C (48%), CTX, AMP, PIP	pAmpC bla_{TEM-1} bla_{OXA-1} bla_{CMY-2}	CTX-M-1 (15) CTX-M-3 (3) CTX-M-15 (3)	24 CTX-resistant isolates from migratory birds; 3 CTX-resistant isolates from resident birds MRSA isolated from 5 migratory bird samples	105
CXM, PEN, OXC, FOX		*mecA*	One of two birds submitted for testing after ~12 finches were found dead over a 6-week period. Both birds died with severe necrotic esophagitis, most likely caused by *Trichomonas gallinae*, but *S. aureus* was isolated from the liver and intestine of one bird	106
SU			Multifocal, necrotizing esophagitis and ingluvitis	100
		cAmpC		34
	ESBL (1)		1 isolate resistant, MDR in 1 isolate. Two genetically unrelated isolates found in one bird	107

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	Cowbird (*Molothrus ater*) (F)	USA (Georgia)	2 isolates	*Salmonella enterica* serovar Typhimurium (necropsy)
Passeridae	English sparrow (*Passer domesticus*) (F)	USA (Wyoming)	1 isolate	*Salmonella enterica* serovar Typhimurium (necropsy)
Sturnidae	Common magpie (*Pica pica*) (F)	Spain (Huesca, Ciudad Real, Madrid, Valencia, Castellón, Cádiz)	7	*S. delphini* (tracheal swabs)
	Common mynah (*Acridotheres tristis*) (C)	Japan	148 strains	*E. coli*
	Rosy starling (*Pastor roseus*) (F)	Pakistan	15	*E. coli* (feces)
	Spotless starling (*Sturnus unicolor*) (F)	Spain (northern)	1	*E. coli* (feces)
Turdidae	American robin (*Turdus migratorius*) (F)	USA (Kansas)	22 samples, 38 birds	*Enterococcus* spp. (saliva, oral swab)
	Eurasian blackbird (*Turdus merula*) (F)	Germany (Hesse)	2 birds	*E. coli* (feces)
Vireonidae	Black-whiskered vireo (*Vireo altiloquus*) (F)	Guadeloupe and nearby islands	17	*E. coli*
Pelecaniformes	Cattle egret (*Bubulcus ibis*) (F)	Spain (central)	3 isolates	*E. coli* (cloacal swab)
	Cattle egret (F)	Spain (central)	2 isolates	*Enterobacter* spp. (cloacal swab)
	Pelican	USA (Florida)	10 samples	*E. coli* (feces)
Phoenicopteriformes	Flamingo	Japan	8 strains	*E. coli*
Piciformes	Great spotted woodpecker (*Dendrocopos major*) (F)	Spain (central)	1 isolate	*K. pneumoniae* (cloacal swab)
Podicipediformes	Pied-billed grebe (*Podilymbus podiceps*) (F)	USA (California)	4	*K. pneumoniae, Acinetobacter baumannii* (cloacal swabs)
Psittaciformes	Blue-fronted parrot (*Amazona aestiva*) (C)	Brazil (Rio de Janeiro)	88 birds, 231 strains	*Enterococcus* spp. (cloacal swabs)
	Blue-fronted parrot (C)	Brazil (Sao Paulo)	3	*Salmonella enterica* serovar Enteritidis
	Budgerigar (*Melopsittacus undulatus*) (C)	New Zealand	1	*K. oxytoca* (crop swab)
	Cockatiel (*Nymphicus hollandicus*) (C)	Brazil (São Paulo, Niterói)	94	*E. coli, Salmonella* spp. (cloacal swabs)
	Grey parrot (*Psittacus erithacus erithacus*) (C)	Germany (Luxembourg)	1	*Acinetobacter baumannii* (choanal swab)
	Grey parrot (C)	USA (North Carolina)	1 isolate	*Salmonella enterica* serovar Typhimurium (necropsy)

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
		intI1	ARG identified in 2 of 3 birds with multifocal, necrotizing esophagitis and ingluvitis	100
		intI1 (1)	ARG identified in 1 of 3 sparrows with multifocal, necrotizing esophagitis and ingluvitis	100
PEN (7), FOX (7)		*mecC, mecXI* blaZ-SCC	Coagulase-positive staphylococci	33
OXY, SM, SA, APC, C, KM			Imported into Japan from Thailand; tested during quarantine	103
CTX, CAZ, AMP, TET, SXT, C, DC, ENR, MRB	$bla_{CTX-M-15}$ bla_{TEM-1B}	*sul3, sul2-like* *qnrS1* *dfrA14-like, dfrA12* *cmlA1-like*		48
CTX, CAZ, C, NA, CIP, GM, TB, TET		SHV-12		35
VAN (8)		*vanA, vanB,vanC*		108
	$bla_{CTX-M-15}$ bla_{TEM-1}	CTX-M-15 TEM-1 *tet(A), tet(B)* *strA, strB,* *drfA17-aadA5* class I integron		47
			1 resistant isolate, 1 MDR isolate	107
	$bla_{CTX-M-1}$ (1) bla_{SHV} (1) cAmpC (1)			34
	$bla_{CTX-M-1}$ $bla_{CTX-M-15}$	CTX-M-1 (1) OXA-48 (1)		34
CAZ, CTX, TET, SXT, NA, CIP, GM, C	AmpC		Feces from 53 seagulls was also analyzed. Results reported make no distinction between isolates from gulls or pelicans All ESBL-producing isolates were resistant to CAZ, CTZ	62
OXY			Imported into Japan from Tanzania; tested during quarantine	103
	$bla_{CTX-M-1}$			34
AMP (1), CAR (1)				49
TET, GM, STR, macrolides, quinupristin/dalfopristin		*tet(M), tet(L)* *erm(B), erm(A)* *mefA* *aac(6')-Ie-aph(2")-Ia* *vat(D)*	48% of strains were MDR. The two strains presenting high-level resistance to STR were negative for *ant(6')-Ia, ant(3')-Ia, ant(9')-Ia,* and *ant(9')-Ib* genes	109
			Three samples were resistant to >4 abx	110
MDR			Bacterial ingluvitis associated with squamous cell carcinoma	111
AX (82%), AMP (79%), STR (67%), TET (41%)		*strAB* (65%) TEM (54%) *tetA, tetB* *aadA, aphaA* *sul1, sul2, sul3*	*E. coli* was isolated in 10%. 96% displayed resistance and MDR was verified in 59% of isolates. The most frequently observed MDR profile was a combination of penicillins, cephalosporins, and aminoglycosides	112
CBP, PIP, MEM, IPM, N, CTF, piperacillin/tazobactam, cefpirome	bla_{OXA-72} $bla_{OXA-343}$ ampC	OXA-72		113
SU				100

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	Grey parrot (*Psittacus erithacus erithacus*) (C)	USA (New York)	1	*S. aureus* (swab of axillary ulcerative dermatitis lesions)
	Grey parrot (C)	USA (Pennsylvania)	1	*S. aureus*
	Lorikeet, Senegal parrot (*Poicephalus senegalus*) (C)	USA (Georgia)	2 isolates	*Salmonella enterica* serovar Typhimurium (Copenhagen) DT104
	"Macaw"	Japan	89 strains	*E. coli*
	"Psittacine birds"	Brazil (Paraíba)		*E. coli* (cloacal swabs)
	"Parrots"	Brazil (Goiás)	300 samples	*E. coli* (feces)
	"Parrot"	Japan	8 strains	*E. coli*
Sphenisciformes	Gentoo penguin (*Pygoscelis papua*) (F)	Antarctica		
	Little penguin (*Eudyptula minor*)	Australia	9	*E. coli* (feces)
Strigiformes	Barn owl (*Tyto alba*) (F)	Spain (northern)	1	*E. coli* (feces)
	Barred owl (*Strix varia*) (F)	USA (Georgia, Alabama)	21 birds, 19 isolates	*E. coli* (cloacal swab)
	Eurasian eagle-owl (*Bubo bubo*) (F)	Spain (central)	3 isolates	*E. coli* (cloacal swab)
	Eurasian eagle-owl (F)	Spain (central)	3 isolates	*K. pneumoniae* (cloacal swab)
	Great horned owl (*Bubo virginianus*) (F)	USA (Georgia, Alabama)	6	*E. faecalis* (cloacal swab)
	Eastern screech owl (*Megascops asio*) (F)	USA (Georgia, Alabama)	5 birds	*E. coli* (cloacal swab)
	Screech owl (*Megascops* spp.) (F)	USA (Iowa)	1	*S. aureus*
	Tawny owl (Strix aluco) (F)	Spain (central)	1 isolate	*K. pneumoniae* (cloacal swab)
	"Owl" (F)	USA (Georgia)	1 isolate	*Salmonella enterica* serovar 4,5,12:l-monophasic
Suliformes	Double-crested cormorant (*Phalacrocorax auritus*) (F)	Prince Edward Island	100	*E. coli* (cloacal swabs)
Mixed taxa	Bald eagles, gulls, including glaucous-winged, herring, hybrids (F)	USA (Alaska)	20 eagles, 56 gulls	*E. coli*
	"Migratory birds"	Bangladesh (Baojani Baor)	66 samples, 55 isolates	*E. coli* (feces)

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
AMK, CEPH, CTX, CIP, ENR, PEN, PIP orbifloxacin			Coagulase-positive MRSA with marked antibiotic resistance in a Congo African grey parrot with dermatitis, septicemia	114
MET			Congo African grey parrot	115
AMP, C, STR, SU, TET		*intI1* *aadA1* *fex*	Birds were from the same household. The *Salmonella* isolates from the lorikeet and Senegal parrot had the FLO resistance gene, a marker for MDR *S. enterica*	100
OXY, SA, SM, C, APC, KM			Imported into Japan from Thailand, India, Paraguay, the Netherlands, Ghana, Ecuador; tested during quarantine	103
CIP (11.7%), ETP (9.3%), CAZ (9.3%)		*kpc*	Birds rescued from trafficking	116
AMP (75.58%), SU (71.51%), AX (70.93%), TET (69%), DC (64.53%), ENR (41%), C (33%), CIP (23%)			AR to 3 groups of abx occurred in 40 samples (23.25%) and to 4 groups of abx in 4 samples (2.32%)	117
OXY, C, SM			Imported into Japan; tested during quarantine	103
TET, C, ERY		TEM, *msr(A), catA1, tet(C), erm, aac(6)-Ib-cr*	Samples collected from two localities next to research bases in Antarctica. Samples collected near the research base with the largest human population contained more ARG than those from the more remote site	118
AMP (100%), CIP (66.7%), FOX (66.7%), TET (55.6%), SXT (44%), C (22%), GM (22%), STR (22%), AMC (11%)		CTX-M-15 (50%)	Resistance to fluoroquinolones and/or extended spectrum cephalosporins *E. coli* was detected (10.5%) A range of ARG were present, but CTX-M-15 occurred most frequently	65
CTX, NA, CIP	ESBL pAmpC	*qnrS1* CTX-M-14a TEM-1b		35
AMP, C, TET, CIP		TEM-1, *cat, tet(M)* CTX-M-1, *gyrA*		37
	$bla_{CTX-M-1}$ (2) bla_{SHV} (1) bla_{OXA-48} AmpC			34
	$bla_{CTX-M-1}$ (2) bla_{OXA-48} (1)			34
TET		*tet(M)*		37
AMP, C, CIP, TET		CTX-M-1, *cat,* TEM-1		37
TET			MSSA isolate	74
	$bla_{CTX-M-1}$			34
SU (32%), TET (18.%), STR		*intI1 merA*	32% of isolates were resistant to sulfamethoxazole, and 18.1% were resistant to both sulfamethoxazole and streptomycin	100
AMP, AX, GM, STR, TET			≤ 5% were resistant to any of the 12 antibiotics tested Tree-nesting chicks sampled	119
AMP, CEF			27 cephalosporin-resistant isolates were recovered. Most isolates were MDR, resistant to between 4 and 7 abx. One isolate was resistant to 13 abx	25
ERY (100%), AMP (100%), STR (74.55%), TET (63.6%), CIP (50.9%), C (43.6%)				120

Continued

TABLE 6.1

Select Reports of Antimicrobial Resistance in Birds—cont'd

Order/Family	Species	Geographic Location	Animals or Samples	ARB Isolated (Source)
	Cattle egret (*Bubulcus ibis*) Hooded crow (*Corvus cornix*) Rock pigeon (*Columba livia*) Laughing dove (*Spilopelia senegalensis*)	Egypt (Giza, Cairo, El-Sharkia, El-Ismailia, Port Said)	20 each	*E. coli, K. pneumoniae* (feces)
	Raptors, waterbirds, and passerines (F)	Italy (Sicily)		*E. coli, Proteus mirabilis, P. vulgaris, Citrobacter freundii, E. cloacae, K. oxytoca, Salmonella typhimurium, Escherichia vulneris, Enterobacter amnigenus* biogroup 2, *Salmonella* Duesseldorf, *H. alvei*
	Raptors (F)	Spain (Catalonia)	663	*E. coli* (35%), *P. aeruginosa* (7%), *Staphylococcus* spp. (5%)
	Three mountain caracaras (*Phalcoboenus megalopterus*) Four Egyptian vultures (*Neophron percnopterus*) Two red-headed vultures (*Sarcogyps calvus*) Two Eurasian eagle-owls (*Bubo bubo*) Three snowy owls (*Bubo scandiacus*) (C)	Italy (Verona)		*E. coli* (14), *Klebsiella* spp. (7), *Proteus* spp. (6), *Staphylococcus* spp. (6) as well as *Escherichia fergusonii. S. odorifera, Citrobacter youngae, Providencia stuartii, Raoultella ornithinolytica* (cloacal swabs)
	Herons, egrets, cormorants	Israel	47 isolates	*Vibrio cholerae* serogroups O1 and O139
	Two mountain caracaras (*Phalcoboenus megalopterus*) Two Eurasian eagle-owls (*Bubo bubo*) Three snowy owls (*Bubo scandiacus*) (C)	Italy (Verona)		Coagulase-negative *Staphylococcus* spp., *S. aureus* (conjunctival swabs)

Antimicrobial Resistance Phenotypes

AMC, Amoxicillin/clavulanic acid; *AMK*, amikacin; *AMP*, ampicillin; *APC*, aminobenzyl-penicillin; *ATM*, aztreonam; *AX*, amoxicillin; *BC*, bacitracin; *CAR*, carbenicillin; *CAZ*, ceftazidime; *CBP*, carbapenem; *CEF*, cefadroxil; *CEPH*, cephalothin; *C*, chloramphenicol; *CLI*, clindamycin; *CIP*, ciprofloxacin; *CO*, colistin; *CPD*, cefpodoxime; *CRO*, ceftriaxone; *CTF*, ceftiofur; *CTX*, cefotaxime; *CVN*, cefovecin; *DC*, doxycycline; *ENR*, enrofloxacin; *ERY*, erythromycin; *ETP*, ertapenem; *F*, nitrofurantoin; *FEP*, cefepime; *FF*, fosfomycin; *FLO*, florfenicol; *FOX*, cefoxitin; *FUS*, fusidic acid; *GM*, gentamicin; *IPM*, imipenem; *KM*, kanamycin; *KZ*, cefazolin; *LCM*, lincomycin; *LEV*, levofloxacin; *MEM*, meropenem; *MET*, methicillin; *MRB*, marbofloxacin; *NA*, nalidixic acid; *OXA*, oxacillin; *OXY*, oxytetracycline; *PEN*, penicillin; *PIP*, piperacillin; *PO*, polymyxin; *SA*, sulfadimethoxine; *SM*, dihydrostreptomycin; *SPT*, spectinomycin; *STR*, streptomycin; SU, sulfonamide, sulfamethoxazole; *SXT*, sulfamethoxazole/trimethoprim; *SZL*, sulfathiazole; *TB*, tobramycin; *TC*, oxytetracycline; *TEC*, teicoplanin; *TEM*, temocillin; *TET*, tetracycline; *TIC*, ticarcillin; *TGC*, tigecycline; *TM*, tobramycin; *TRI*, trimethoprim; *TY*, tylosin; *VAN*, vancomycin.

Antimicrobial Resistance Genotypes

aac(3)-II, aac(3)-IV, aadA1, aadA2, aadA5, aminoglycoside resistance genes; *aac(6)-Ib-cr*, associated with multidrug resistance plasmids; *AmpC*, class C beta-lactamase; *bla*, beta-lactamase; *CTX-M*, producing ESBL; *ESBL*, extended-spectrum beta-lactamase; *SBL*, serine β-lactamase; *SHV*: class A beta-lactamase; *TEM*, class A beta-lactamase; *pAmpC*, plasmid-encoded class C beta-lactamase.

Comments

abx, antibiotic; *ARB*, antibiotic-resistant bacteria; *ARG*, antibiotic resistance gene; *C*, captive; *F*, free-ranging; *MDR*, multidrug-resistant (at least three classes of antibiotics); *MRSA*, methicillin-resistant *Staphylococcus aureus*; *MSSA*, methicillin-susceptible *Staphylococcus aureus*; *VIM*, Verona integron-encoded; *MBL*, metallo-β-lactamase; *VRE-i*, vancomycin-resistant enterococci with intrinsic resistance.

ANTIMICROBIAL RESISTANCE				
Phenotype	**Enzymes**	**Genotype**	**Comments**	**Ref**
CO, PO		*mcr-1, mcr-2*	Isolates from 4 taxa grouped together as "resident birds": *E. coli* (12 isolates), *K. pneumoniae* (4 isolates), *K. oxytoca* (1 isolate), *P. aeruginosa* (1 isolate) The prevalence of *mcr-1* in resident birds was 10.4% while the prevalence of *mcr-2* was 1.4%	51
STX, STR, AMC, AMP, TET			Isolates showed significant frequencies of AR. MDR also occurred. The most frequently displayed resistances were to SXT. There were also significant levels of resistance to STR, AMC, AMP were found. Lower levels of AR to TET were shown	121
CLI, AMP, TET, ENR, SXT, CXM			Retrospective study evaluating 108 abx sensitivity profiles: >50% of isolates (34 different bacterial species) demonstrated resistance. Among *E. coli* strains, 71% (27/38) presented MDR	24
SXT, PEN, AMP, A, AMC, CEF, KZ, OXY, DC, STR, KM, GM, AMK, ERY, TY, LCM, CLI, ENR, CIP, MRB, CVN			Gram-positive bacteria were isolated from 3 Strigiformes and 3 Falconiformes; gram-negative bacteria were cultured from all birds. All isolates were MDR	122
SXT (44%), azithromycin (22%), DC (11%)			The percentage of AR was highest in birds when compared to isolates from chironomids or fish	123
A, KZ, GM, TY, CIP, MRB, PEN, AMP, LCM, C, ERY, SXT, STR, OXY, ENR			Seven out of 14 birds (50%) had positive culture. The most common bacteria isolated was *S. xylosus* ($n = 4$), while *S. chromogenes, S. lentus,* and *S. aureus* were only isolated once	122

TABLE 6.2

Core Principles of Judicious Use of Antimicrobial Agents[7,12,16,28]

Category	Principles	
Pretreatment	Disease prevention	Reduce the incidence of infection through proper animal care and husbandry, including appropriate biosecurity measures, effective sanitation, hygiene, infection prevention measures, and health monitoring.[6,12]
	Professional intervention	Over-the-counter access is contrary to the basic concepts of prudent and effective antimicrobial use and could presumably increase the risk of resistance.[13] Ensure that the use of antimicrobials meets all the requirements of a valid veterinarian–client–patient relationship.[12,13] Use flock surveillance programs and educational programs that promote responsible and prudent use of antimicrobial agents.[12]
	Alternatives to antimicrobial agents	Efficacious, evidence-based alternatives to antimicrobial agents can be an important adjunct to good husbandry practices.[12] For example, curettage of focal granulomas or local therapy with biocides or antimicrobial drugs may be effective in some cases.
Diagnosis	Accurate diagnosis	Use antimicrobials only after a general or preliminary diagnosis has been made, which indicates that antimicrobial therapy is appropriate.[5] Avoid antimicrobial therapy for nonseptic inflammatory conditions and uncomplicated viral infections, even when mucoid discharge is thick, yellow, or green.[5,13,26] Also remember that it is uncommon for many infections to develop without a predisposing condition. Therefore, repeated treatment with antimicrobials without attention to the underlying cause can ultimately be futile clinically while also increasing the risk of antimicrobial resistance.[13]
Therapeutic objective and plan	Develop outcome objectives and an implementation plan, including consideration of therapeutic choices, supportive therapy, host, environment, infectious agent, and other factors.[9]	
Drug selection	Justification of antimicrobial use	Antimicrobials should not be used to compensate for or mask poor farm or veterinary practices.[12] Use informed professional judgment balancing the potential benefits for animals, humans, and the environment against the risks, particularly the risk of antimicrobial resistance selection.[12]
	Guidelines for antimicrobial use	Consult disease- and species-specific guidelines to inform antimicrobial selection and use (Tables 6.10A–E). Although antimicrobials must be prescribed in accordance with existing laws and regulations,[12] there is a common misconception that using antimicrobial agents according to label recommendations is equivalent to prudent use of antimicrobial agents.[16] This is not true. The label dose, dosing interval, and indications are not always consistent with current principles for responsible antimicrobial use, particularly for some older drugs like penicillin.[16]
	Critically important antimicrobial agents	Antimicrobial agents should be used only after careful review and reasonable justification; however, there is a particular concern regarding the use of agents of special importance to human (and veterinary!) medicine (Table 6.3).[12]
	Culture and sensitivity testing	When clinically relevant, use culture and antimicrobial sensitivity testing to guide the selection of antimicrobials, especially when initial treatment has failed.[5,12] Selective reporting of sensitivity testing is another form of stewardship intervention designed to restrict inappropriate or unnecessary antimicrobial use.[29]
	Spectrum of activity	Whenever appropriate, select narrow-spectrum antimicrobials, as opposed to broad-spectrum antimicrobials.[12] Never hesitate to transition a patient to a more targeted antimicrobial for longer-term therapy based on culture and sensitivity test results, additional diagnostic information, and/or clinical progression.[16,33]
	Extra-label (off-label) therapy	Confine extra-label therapy to cases in which medications used according to label instructions were ineffective or unavailable and where there is scientific evidence.[12]
Drug use	Dosage regimens	Where possible, optimize regimens for therapeutic antimicrobial use following current pharmacokinetic and pharmacodynamic guidance.[12] Suboptimal dosing can lead to resistance.[33]
	Duration of treatment	As the duration of patient exposure to antimicrobials increases, the likelihood of selection and colonization with resistant organisms also rises.[28] Therefore, minimize exposure to antimicrobials by treating for only as long as needed to meet the therapeutic objective.[5,12] Higher doses for shorter periods of time are generally preferable.[31] Never continue antimicrobials once there is clinical and microbiological evidence that an infection has been eliminated or once an alternate diagnosis has been made.[16] Timely planned therapy evaluation is a prerogative for adequate stop or switch decisions. It is a common misconception that there is a need to complete a minimum duration of an antimicrobial drug to prevent the emergence of resistance.[13] There is no prescribed minimum duration of administration of an antimicrobial drug.[16]

TABLE 6.2

Core Principles of Judicious Use of Antimicrobial Agents—cont'd

Category	Principles	
	Labeling and instructions	The veterinarian should provide written instructions on drug use, with clear details on the method of administration, dose rate, frequency and duration of treatment, as well as precautions and withholding period.[12]
	Target animals	Limit antimicrobial treatment to ill or at-risk animals, treating the fewest animals possible.[5,12]
		The prudence of treating moribund birds with antimicrobial drugs must be questioned. Antimicrobial drugs, especially those of critical importance to human health (Table 6.3), should never be used without reasonable expectation that they will favorably impact the outcome.[16]
		Patients that require invasive therapies and intensive care are at increased risk for developing difficult-to-treat, resistant infections. Veterinarians must take every precaution to reduce the risk of infection in these animals.[16]
	Record keeping	Maintain accurate records of therapy and outcome to allow therapeutic regimens to be evaluated.[5,12]
	Compliance	Confirm that drug use instructions are accurately followed.[12]
Post treatment activities	Environmental contamination	Avoid antimicrobial contamination of the environment whenever possible.[5,12] Unused medication should never be disposed of in the sink or garbage. Instead medication should be returned to the veterinary office. Many communities also offer drug take-back programs, and some pharmacies and hospitals also have permanent disposal sites.
	Continuous evaluation	Continually monitor and evaluate veterinary prescribing practices as well as the effectiveness of the antimicrobial selection program.[12,15] Participate in benchmarking initiatives whenever available.[127]
		Interventions directed at driving behavioral change include practitioner education, reinforcing factors, such as audits and feedback, as well as enabling factors like decision support.[15]

are also restricted or banned based on residue or antimicrobial resistance concerns.[11] The outcome of therapy must also be continually evaluated.[5,25] Although these coordinated interventions are designed to improve and measure the appropriate use of antimicrobials, antimicrobial stewardship cannot target individual prescriptions alone because stressing the judicious use of antimicrobials does not explicitly consider the need to balance individual and societal needs.[3,26,40]

RESISTANCE TO RESPONSIBLE ANTIMICROBIAL STEWARDSHIP

There are many challenges to improving antimicrobial stewardship, including lack of awareness for the need for all prescribers and users to be good antimicrobial stewards, lack of controls on antimicrobial availability in many countries, pressures on prescribers to use antimicrobials even when they are unnecessary, and lack of oversight.[7] There are many online resources available that are intended to guide and assist clinicians (Table 6.3).

CLIENT EDUCATION

One of the prime challenges that veterinarians face are demands for antimicrobial therapy from clients, as owners may immediately assume that "every debilitated bird requires antibiotics".[7] Although this can be a difficult conversation, often the first and most crucial step is to educate owners about the concepts of antimicrobial resistance and responsible antimicrobial stewardship. Whenever possible, disseminate this information before a problem arises. Just as it is valuable to have a valid veterinarian-client-patient relationship before a problem arises, it is also extremely helpful for the owner to understand these concepts before a cherished companion animal becomes ill. Websites that target consumers, such as "Antibiotics and You", can be particularly helpful in explaining these concepts in layman's terms (Table 6.3).

Counseling clients and producers on the importance of diagnostic testing can be another major challenge for veterinary health professionals.[13] It sometimes helps to emphasize that the time and money spent on diagnostic testing can potentially reduce morbidity, mortality, and even overall treatment costs.[13]

When antimicrobial agents are prescribed, encourage and ensure that directions are accurately followed.[11] Provide clearly written instructions on drug use, with clear details on the method of administration, dose rate, frequency, and duration of treatment, as well as precautions and withholding period.[11] When antimicrobial drug therapy is discontinued, encourage clients to return unused antimicrobial drugs for disposal.[13] The educated consumer will have a clear understanding of why holding onto leftover antimicrobials "just in case" is a poor idea or why disposing of drugs by flushing them potentially contaminates the environment with antimicrobials.[5,11]

ANTIMICROBIAL DRUG SELECTION AND USE

Isabelle Langlois

The goal of antimicrobial therapy is to assist the defense mechanisms of the avian host to contain and eliminate the

TABLE 6.3

Online Resources on Antimicrobial Stewardship[3,7,11,13,122]

Source	Comments	Hyperlink(s)
Antibiotics and You	This consumer-oriented website is designed to explain responsible antimicrobial use and antimicrobial resistance in laymen's terms. The site is a collaboration between the Antibiotic Resistance Action Center at the Milken Institute School of Public Health at George Washington University and the Urgent Care Association.	antibioticsandyou.org
European Centre for Disease Prevention and Control (ECDC) European Food Safety Authority (EFSA)	Annual summaries of antimicrobial resistance (as well as zoonotic infections and foodborne outbreaks) in Europe are published by the ECDC and EFSA.	ecdc.europa.eu/en/antimicrobial-resistance efsa.europa.eu/en/topics/topic/antimicrobial-resistance efsa.onlinelibrary.wiley.com/doi/10.2903/j.efsa.2019.5598
European Platform for the Responsible Use of Medicines in Animals (EPRUMA)	Established in 2005, EPRUMA works at the European Union level facilitating and promoting coordinated and integrated action, including the production of framework documents providing guidance on responsible antimicrobial use in food-producing animals.	epruma.eu/
Responsible Use of Medicines in Agriculture Alliance (RUMA)	Established in the United Kingdom, RUMA brings together organizations involved in all stages of the food chain with the objective of promoting food safety, animal health, and animal welfare. Established in 1997, it publishes guidelines on antimicrobial use and vaccination in food animals, including poultry.	ruma.org.uk/
Task Force on Antibiotic Resistance in Production Agriculture	In 2014, the Association of Public and Land Grant Universities (APLU) and the Association of American Veterinary Medical Colleges (AAVMC) joined forces to create the Joint APLU/AAVMC Task Force on Antibiotic Resistance in Production Agriculture, which developed a comprehensive national strategy for diminishing the role of antimicrobial use in food animals in the broader antimicrobial resistance problem.	https://www.aavmc.org/assets/Site_18/files/Reports/APLU_AAVMC%20Task%20Force%20Report%20Final.pdf
US Food and Drug Administration	In 2018, the US Food and Drug Administration's Center for Veterinary Medicine released a multifaceted strategy designed to limit or reverse resistance arising from the use of antimicrobials in food-producing animals. Sources include their list of Antimicrobial Resistance Guidances.	fda.gov/animal-veterinary/safety-health/antimicrobial-resistance fda.gov/animal-veterinary/guidance-industry/antimicrobial-resistance-guidances
Veterinary Antimicrobial Stewardship Course Modules	A collaborative project of the Veterinary Schools of Australia and New Zealand funded by the Australian Government created five learning modules that explore the development of antimicrobial resistance and the important role that veterinarians play as prescribers in reducing selective pressure on microbes.	vetams.org
World Health Organization (WHO) List of Medically Important Antimicrobials World Organization for Animal Health (OIE) List of Antimicrobial Agents of Veterinary Importance	The first WHO *f*es, most recently in 2018. Antimicrobial classes used in human medicine are categorized into three groups: critically important, highly important, and important. Antimicrobials used in food-producing animals have also been listed and categorized by the OIE as critically important, highly important, and important together with the animal species to which they are applied, the indications for their use, and whether they are essential or have few alternatives.	https://www.who.int/publications/i/item/9789241515528 https://www.woah.org/app/uploads/2021/06/a-oie-list-antimicrobials-june2021.pdf

invading infectious agent. This action is facilitated when the therapeutic concentration of the selected drug is rapidly reached at the site of infection and maintained for sufficient length of time. Of course, the selective toxicity of the drug for the offending pathogen should occur prior to any toxic effect of the drug on the avian host. Selection of the most appropriate drug will also depend on the severity of illness, availability of pharmacokinetic and pharmacodynamic data in the treated species, cost of the drug, and routes of administration that can be accomplished by the owner or the veterinary hospital staff.

ANTIMICROBIALS ACCORDING TO THEIR IMPORTANCE IN HUMAN MEDICINE

The WHO has developed criteria to rank antimicrobials according to their importance in human medicine,[128] and several countries have drawn inspiration from this.[129–131] Table 6.4 categorizes antimicrobials according to their importance in human medicine. Several antimicrobial classes are commonly used in avian species. At first, the relative importance of antimicrobials was determined for use in developing risk management strategies related to antimicrobial use in food production animals.[128,132]

TABLE 6.4

Classification of Antimicrobial Drugs According to Their Importance in Human Medicine (WHO: https://www.who.int/publications/i/item/9789241515528)[128]

Because multidrug-resistance is mostly observed in bacterial pathogens, this list primarily focuses on antibacterial drugs.

Category I: Very High Importance (Critically Important Antimicrobials)	Category II: High Importance (Highly Important Antimicrobials)
◆ Essential for the treatment of serious infections ◆ Limited or no availability of alternative antimicrobials for effective treatment in case of emergence of resistance to these agents ◆ Aminoglycosides (except topical agents) ◆ Carbapenems (imipenem) ◆ Cephalosporins (third and fourth generation) ◆ Fluoroquinolones (enrofloxacin) ◆ Glycopeptides (vancomycin) ◆ Glycylcyclines (tigecycline) ◆ Macrolides and ketolides (azithromycin) ◆ Lipopeptides ◆ Monobactams ◆ Oxazolidinones ◆ Penicillins with penicillin-beta-lactamase inhibitors (amoxicillin/clavulanic acid) ◆ Phosphonic acid derivatives (fosfomycin) ◆ Polymyxin (colistin, polymyxin B) ◆ Therapeutic agents for *Mycobacterium* spp. (ethambutol, isoniazid, pyrazinamide, rifampicin)	◆ Used to treat a variety of infections, including serious infections, and for which alternatives are generally available ◆ Microorganisms resistant to drugs of this category are generally susceptible to Category I drugs ◆ Amphenicols (chloramphenicol, florfenicol) ◆ Cephalosporins (first and second generation) ◆ Fusidic acid ◆ Lincosamides (lincomycin, clindamycin) ◆ Penicillins ◆ Quinolones (except fluoroquinolones) ◆ Streptogramins (virginiamycin) ◆ Sulfonamides (sulfamethoxazole) ± trimethoprim ◆ Tetracyclines
Category III: Medium Importance (Important Antimicrobials)	**Category IV: Low Importance**
◆ Used for the treatment of infections for which alternatives are generally available ◆ Infections caused by microorganisms resistant to these drugs are generally susceptible to Category I or II drugs ◆ Aminocyclitols (streptomycin) ◆ Aminoglycosides (topical agents) ◆ Bacitracin ◆ Nitrofuran derivatives ◆ Nitroimidazoles (metronidazole) ◆ Pleuromutilins	Antimicrobials in this category are currently not used in human medicine ◆ Flavophospholipols (bambermycin) ◆ Ionophores (monensin) ◆ Amprolium

Later, it was expanded to companion animals,[13,133] and as such, this classification may also be used for avian species, knowing that ultimately the One Health concept applies in all spheres of veterinary medicine to minimize the risk of transmission of resistance to the human population. As previously stated, the correlations between antimicrobial usage in human and animals, and prevalence of resistance in each group, respectively, is well-documented.[132,134,135]

PRINCIPLES OF ANTIMICROBIAL SELECTION

The avian veterinarian's first choice for an antimicrobial drug should be an approved veterinary product, used in an on-label or, if need be, an extra-label manner. A drug that has been shown to be efficacious and is categorized as being of lower importance in human medicine should be prioritized unless medical requirements dictate otherwise. For situations in which extra-label drug use (ELDU) might result in the use of a drug of lower importance to human medicine, ELDU may in fact be preferable to on-label use of a drug of higher importance to human medicine.

Optimal antimicrobial therapy is based on identifying the microorganism involved and—in case of bacterial infections—culture and sensitivity testing to help select the most appropriate drug (Table 6.5; see also Chapter 5). Cytology (e.g., Gram stain) of an appropriately collected sample may provide insight as to a pathogen involved while awaiting culture results.[139,140] Gram-negative bacterial infections are most common in several species groups such as Psittacidae (*E. coli*, *Klebsiella* spp.) and Columbidae (*Salmonella* spp.). Knowing gram-negative bacteria readily acquire resistance genes,[141] the need to submit a sample for culture and sensitivity testing must be emphasized when they appear to be involved in the disease process. Gram-positive bacterial infections (*Staphylococcus aureus*, *Enterococcus* spp.) are seen less frequently in psittacine birds. However, in Passeriformes, *Enterococcus faecalis* is a major pathogen of the respiratory system, and *Streptococcus gallolyticus* causes septicemia in pigeons. Gram stain coloration may also help provide precise and prompt diagnoses if a mycotic organism is involved, as fungal cultures may take a few weeks.

Empirical antimicrobial treatment is appropriate in two clinical situations: life-threatening infection and mild infection managed on an outpatient basis.[139] Birds often hide clinical

TABLE 6.5

Pharmacokinetic/Pharmacodynamic Indices and Mechanism of Action of Select Antibacterial Agents[136–138]

Antibacterial Class	Representative Antibacterial Drugs	Mechanism of Action	Spectrum	"Cidal" or "Static"	Predictive PK/PD Parameter
Aminoglycosides	Gentamicin, amikacin, neomycin, spectinomycin	Protein synthesis inhibitor	Some gram positives, most gram negatives including *Pseudomonas*	Bactericidal	C_{max}/MIC
Beta-lactams	Penicillins, cephalosporins, carbapenems	Cell wall synthesis inhibitor	Many gram positives and negatives, most anaerobes	Bactericidal	Time > MIC
Glycopeptides	Vancomycin	Cell wall synthesis inhibitor	*Clostridium*, coagulase-negative staphylococci, beta-hemolytic streptococci, MRSA	Bactericidal (slowly)	Time > MIC, C_{max}/MIC
Fluoroquinolones	Enrofloxacin, ciprofloxacin, danofloxacin, marbofloxacin, pradofloxacin	DNA replication/transcription inhibitor	Most aerobic gram negatives, some gram positives, *Chlamydia*, *Mycoplasma*, *Mycobacterium*, *Campylobacter*	Bactericidal	AUC/MIC, C_{max}/MIC
Macrolides/azalides/lincosamides	Erythromycin, clarithromycin, azithromycin, clindamycin	Protein synthesis inhibitor	Many gram positives, most *Chlamydia*, *Mycoplasma*, *Campylobacter*, *Mycobacterium*	Bacteriostatic	AUC/MIC
Tetracyclines	Oxytetracycline, chlortetracycline, doxycycline, minocycline	Protein synthesis inhibitor	Many gram positives, some Gram negatives (except *Pseudomonas*), *Mycoplasma*, *Chlamydia*, anaerobes	Bacteriostatic	Time > MIC
Trimethoprim-sulfonamides	Sulfadimethoxine, sulfamethazine	Folic acid synthesis inhibitor	Many gram positives and negatives (except *Pseudomonas*)	Bactericidal	AUC/MIC

AUC/MIC, Area under the curve to MIC ratio; *C_{max}/MIC*, peak plasma concentration to MIC ratio; *T > MIC*, time above MIC; *MRSA*, methicillin-resistant *Staphylococcus aureus; PD,* pharmacodynamic; *PK,* pharmacokinetic.

signs of illness; therefore, they often present at an advanced critical stage of disease. In the later situation, if infection recurs or fails to respond to initial antimicrobial selection or if the clinical situation involves many animals presenting the same clinical signs, appropriate samples for culture and sensitivity testing should be obtained.[139,142]

Successful antimicrobial therapy requires reducing pathogen numbers to reach a point where host defenses are effective. The distinction between bactericidal-fungicidal-virucidal and bacteriostatic-fungistatic-virustatic agents appears to be clear using in vitro techniques but can be inconsistent for a particular drug against all microorganisms.[143] The supposed superiority of bactericidal-fungicidal-virucidal agents over bacteriostatic-fungistatic-virustatic agents is of little clinical relevance when treating the vast majority of infections, especially in patients with uncomplicated infections and competent immune system.[143] Bactericidal-fungicidal-virucidal drugs may be preferred for life-threatening infections, infections of vital tissues (central nervous system, cardiovascular, and bones), and in immunocompromised individuals.[139] If sensitivity testing has been performed, avian practitioners should use an antimicrobial drug to which the microorganism is susceptible regardless of whether it is bacteriostatic-fungistatic-virustatic or bactericidal-fungicidal-virucidal (Table 6.5).[144]

Narrow-spectrum drugs may be preferred to broad-spectrum antimicrobials when appropriate to decrease the likelihood of interfering with commensal microbial flora[139] while minimizing the selection of resistant organisms.[142] The narrower the spectrum, the less likely resistance might develop. Table 6.6 presents the examples of antimicrobial drugs classified according to their spectrum of activity.

Generally, drug combinations should be considered for their synergistic effects where individual agents have failed,[145] or when the identified clinical condition of the patient dictates such therapeutic approach, and their efficacy is established. The most important exception to this rule is the combination of sulfonamides with trimethoprim, as in these combinations, sulfonamides are much more potent (potentiated), and therefore lower doses are needed with less toxicity. Further, the inhibition of two sequential steps in folic acid production leaves less room for resistance due to escape routes. Combining antimicrobial agents may have disadvantages: (1) a bacteriostatic-fungistatic-virustatic drug may neutralize bactericidal-fungicidal-virucidal effects of another agent; (2) additive or synergistic toxicity may occur; and (3) superinfection may occur after destroying normal microbial flora.[139] Clinical indications for the use of antimicrobial combinations are presented in Table 6.7.

TABLE 6.6

List of Antimicrobial Drugs Used in Avian Medicine According to Their Spectrum of Activity

Spectrum of Activity	Examples of Antimicrobial Drugs
Narrow	Metronidazole
	Aminoglycosides (gentamicin)
Moderate	Macrolides (azithromycin)
	Lincosamides (clindamycin)
Broad	Amoxicillin ± clavulanic acid
	Cephalosporins
	Tetracyclines (doxycycline)
	Potentiated sulfonamides (trimethoprim-sulfamethoxazole)
	Fluoroquinolones (enrofloxacin)

Modified from Battersby I: Using antibiotics responsibly in companion animals, *In Pract* 36(3):106, 2014.

When applicable, local administration of the antimicrobial should be considered, as this method of delivery has the potential to achieve higher and more persistent drug concentration than systemic administration at the site of infection. When using antimicrobials for local delivery, ensure that the antimicrobial directed against the causative agent and vehicle are suited for this use and do not have a detrimental effect on the tissue while being compatible with the delivery system used.[146]

LOCAL ADMINISTRATION OF ANTIMICROBIALS IN AVIAN PATIENTS

In avian patients, local administration of antimicrobials may include local application or instillation, direct intralesional local delivery, nebulization, and regional perfusions. Topical hydrosoluble creams may be used sparingly on the skin to treat localized infections, such as dermatitis.[146] Topical ointment or drops may be preferred to treat eye infection. Antimicrobial ophthalmic preparations (ointments and drops) may be instilled intranasally to treat nasal infections,[146,147] in addition to their classical use to treat eye infection. Powdered and aerosolized antimicrobials may be useful to treat ectoparasites.[147]

Direct intralesional administration of antimicrobials may be achieved using antibiotic-impregnated beads.

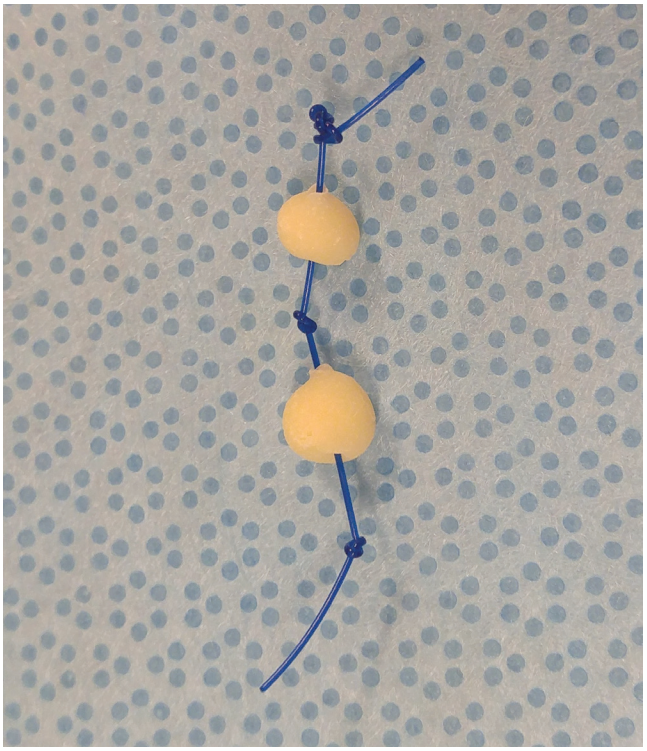


FIGURE 6.3 Beads fashioned around a piece of nonabsorbable suture material so that they are strung together to facilitate removal from the infection site. (Photo courtesy of Zoological Medicine Service, Université de Montréal.)

Antibiotic-impregnated polymethyl methacrylate (AIPMMA) beads are most often used in avian osteomyelitis and soft tissue infections.[148,149] Available authorized medicinal products like cement and beads containing gentamicin, tobramycin, and clindamycin may be used.[150] There is rapid release during the first 24 hours after implantation followed by prolonged sustained release that can last for weeks.[150] The antibacterial drug concentrations achieved at the site of infection are far above the minimal inhibitory concentrations (MICs) for most common bacterial pathogens,[151] without relying on vascular supply and tissue integrity. Beads can be fashioned around a piece of nonabsorbable suture material so that they are strung together, making it easier to remove them from the infection site (Figure 6.3).[146] Antibacterial drug–impregnated

TABLE 6.7

Clinical Indications for the Use of Antimicrobial Combinations[139,145]

Indications	Rationale
Antimicrobial synergism beneficial in other species with similar conditions	◆ Combination of antimicrobial agents has proved superior to monotherapy in other species (i.e., treatment of endocarditis in humans). ◆ Patients with impaired host defenses as synergistic combinations are advantageous in such patient.
Polymicrobial infections	One agent cannot address all the pathogens causing the infection.
Decrease the emergence of resistant isolates	Microorganisms that may develop resistance by various mechanism will have less chance of doing so if treated with multiple antimicrobials (e.g., *Mycobacterium* spp.).
Decrease dose-related toxicity	Combined therapy may allow dosage reduction of a toxic drug.

calcium sulfate beads were used in conjunction with regional limb perfusion (see later) in a Pekin duck (*Anas platyrhynchos domesticus*) to treat septic tenosynovitis and osteoarthritis.[152] Clinical use of calcium sulfate beads in veterinary medicine was first reported in 2008 with a case series of six dogs with osteomyelitis.[153] Other reported uses include the management of deep surgical site infection associated with orthopedic surgery and local carboplatin delivery.[154,155] In human medicine, they have been used in the treatment of osteomyelitis in the long bones, arthroplasty, periprosthetic joint infection, and open fractures.[156] Calcium sulfate is a bioresorbable material. Therefore, a second procedure is not required for removal as opposed to AIPMMA beads.[153,156] Pharmacokinetic studies have shown its superiority as 100% of its loaded antibacterial drugs are released, compared to around 10% in AIPMMA, which results in much higher concentrations for an extended period.[156] Finally, this biomaterial allows the use of a larger range of antimicrobials because setting occurs at room temperature. Complications reported with the use of calcium sulfate beads in human medicine include aseptic wound drainage, infection reoccurrence, transient hypercalcemia, and heterotropic ossification.[156]

The administration of antimicrobial agents by nebulization may be used as an adjunct to systemic administration in severe avian respiratory disease (see Chapter 5 and Appendix 1). Nebulization time is typically 15 to 30 minutes and frequency varies from one to three times daily. Administration is mostly performed without handling, in a closed cage or induction chamber, thus reducing stress for the patient (Figure 6.4). Nebulization enables to reach diseased tissue with poorly perfused respiratory structures such as the avian air sacs.[157] Pharmacokinetic studies of nebulized drugs have been published in various species[158–162] and have confirmed reduced systemic absorption with short-lasting low plasma concentrations.[159,160] Effective nebulization depends on the particle size. Use of a nebulizer generating microspheres smaller than 3 μm is recommended.[163,164] In pigeons, with an increasing time of exposure to aerosolized particles, the degree of particle deposition into the respiratory system is maximized, until a balance is reached with approximately uniform particle distribution to each of the air sacs.[165] However, nebulization had to be performed for at least 2 hours in this study to result in readily observable deposition of the 1.0 μm fluorescent microspheres in the air sacs.[165] The suspending agent for drug nebulization influences drug deposition. A study showed that saline resulted in more diffusion into the air sacs compared to propylene glycol, with some air sac diffusion being detectable after only 30 minutes of nebulization.[166] The addition of dimethyl sulfoxide (DMSO) to a nebulized solution of tylosin was shown to result in higher concentrations in lung and air sac tissues.[161] Overall, when using a lipophilic agent, current nebulization session time may be suboptimal and may need to be adapted according to the patient's clinical progress.



FIGURE 6.4 Nebulization of birds is readily performed using an acrylic box and human nebulizer. Dependent on the drug that is nebulized, a compressor or ultrasonic nebulizer can be used. Nebulization should always be performed in a well-ventilated room with as little human presence as possible to minimize exposure. (Photo courtesy Yvonne van Zeeland.)

Intravenous regional limb perfusion (IVRLP) and intraosseous regional limb perfusion (IORLP) appear to be safe and effective for local antimicrobial administration in avian patients with severe pododermatitis or osteomyelitis involving multiple joints of the distal extremities (Figure 6.5; see also Chapter 33). This method may be used as an alternative or adjunctive therapeutic option. In a chicken model, the mean plasma and synovial fluid ceftiofur concentrations exceeded the therapeutic threshold for most common bacterial pathogens (>1.0 μg/mL).[167] The mean synovial fluid ceftiofur concentration for the IVRLP group was significantly greater than that for the IORLP, making intravenous administration preferable. Clinically, the therapeutic use of amikacin along with flunixin meglumine administered through IVRLP yielded dramatic improvement of mobility and a noticeable decrease in the swelling of the right foot of the chicken suffering from cellulitis.[168] Also in chickens, regional limb perfusion of amikacin at high doses produced no discernable renal pathology in healthy euhydrated chickens.[169] IVRLP using ampicillin/sulbactam has also been used successfully in brown pelicans (*Pelecanus occidentalis*) presented for rehabilitation. Birds were released 3 to 4 weeks after initial perfusion therapy with no observed adverse effects.[170]

Recently, a simple novel surgical method (n = 8) involving custom-made drains to resolve septic arthritis of the intertarsal and interphalangeal joints in ducks has been presented.[171] Drains were regularly flushed with dilute 2.5% chlorhexidine solution, and seven of the eight ducks enrolled in the study received concurrent antibacterial treatment. Approximately a year post-surgery, 75% of the ducks had no detectable limp (25% were lost to follow-up). According to these authors' experiences, systemic and regional treatment with antibacterial drugs may slow the progression or stop early cases of septic arthritis in waterfowl, while successful treatment of chronic septic arthritis is difficult at best.

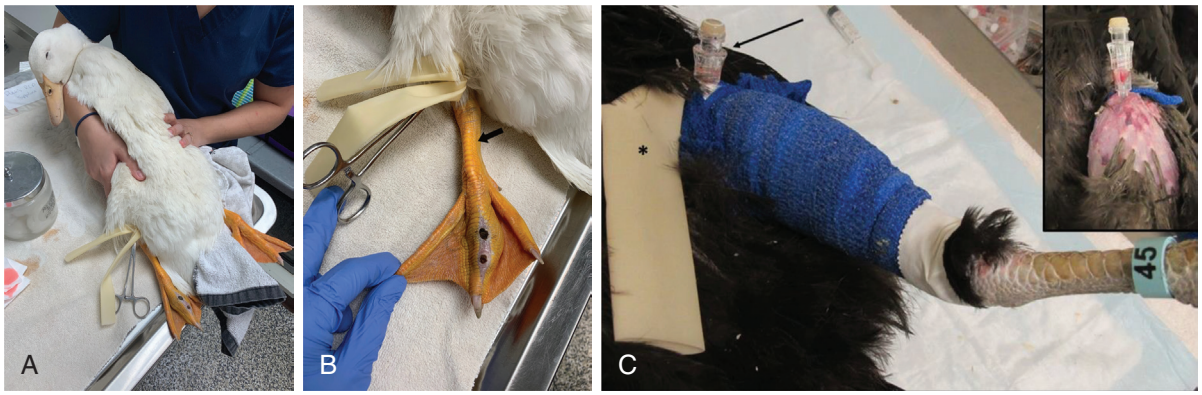


FIGURE 6.5 Regional limb perfusion (RLP) allows for the delivery of high concentrations of antimicrobial drugs into the synovium and osseous and soft tissue structures of the distal limbs in birds. For the procedure, a Penrose drain, which is used as a tourniquet, is positioned around the leg at mid-tibiotarsal level and secured with hemostat (A). Following disinfection of the skin, a 25-G butterfly catheter is aseptically placed in the medial metatarsal vein *(arrow)* to administer the drugs (B). Once the catheter is flushed with a small amount of sterile saline solution, the catheter is removed, and a bandage is placed over the injection site. To optimize perfusion of the drug into the distal tissues, the tourniquet is left in place for 10 minutes. To prevent hematoma formation, it is recommended to leave the bandage in place until after the tourniquet is removed. Aside from intravenous regional limb perfusion (IVRLP), the intraosseous route (intraosseous regional limb perfusion; IORLP) is also feasible. For this purpose, an intraosseous catheter is placed in the proximal aspect of the tibiotarsal bone. The procedure follows similar steps as those described for IVRLP, except for placement of the tourniquet, which should be positioned at mid-femoral level to allow for optimal perfusion of the distal extremities (C). (Images courtesy Jennifer Graham and Emi Knafo.)

IMPROVED PRESCRIBING

Isabelle Langlois, Ingeborg M. van Geijlswijk

Antimicrobials are regularly prescribed in avian practice, and it is not uncommon for avian veterinarians to prescribe them without having first documented an infection. As such, the prescription of antimicrobials is not always appropriate. Table 6.8 presents a list of tips to improve prescribing habits in avian medicine.

KEEP IN MIND THE ONE HEALTH CONCEPT

The problem of emerging multiresistant bacteria is well recognized in human medicine.[172] *S. aureus*, a major human pathogen, possesses virulence factors and the ability to acquire resistance to most antibacterial drugs. Clinical use of methicillin has led to the appearance of MRSA and the past few decades have witnessed the existence of new MRSA.[172,173] Some of these new clones can invade community settings and infect

TABLE 6.8

Tips to "Be Wise" When Prescribing Antimicrobials to Avian Patients[139,142,175]

B	Build practice policies	◆ Practice policies for empirical prescribing (while awaiting culture and/or other test results) and prophylactic use. ◆ Optimize therapy while taking into consideration the One Health concept.
E	Encourage/evaluate culture and sensitivity testing	◆ Culture and sensitivity testing are always indicated and essential when empirical dosing has failed.
W	Weigh pros and cons of delivery methods	◆ Use of topical preparation or loco-regional administration reduce selection pressure on commensal flora.
I	Inform yourself	◆ Consider which microorganisms are likely to be involved (viral, fungal, bacterial, gram positive, gram negative, aerobic/anaerobic). ◆ Assess the distribution and the penetration in relation to the clinical presentation. ◆ Search for recent pharmacokinetic/pharmacodynamics studies in a closely related species. ◆ Identify any potential side effects and how to ensure their early detection.
S	Select proper spectrum	◆ Narrow-spectrum antimicrobials have fewer side effects on the patient's microbiome.
E	Educate your clients	◆ Repeated culture may be indicated to confirm resolution of the infection. ◆ Dispose of remaining medication appropriately.

people without predisposing factors. This trend continues with the buildup of the MRSA reservoir in companion and production animals.[172] Shared population of MRSA can infect both humans and companion animals without undergoing host adaptation.[174,175] Furthermore, some clones of human origin have adapted to new animal hosts by losing useless virulence factor or acquiring new mobile genetic elements.[173]

There is a correlation between close contact with humans and increased risk of colonization of pets with *S. aureus*. This has been demonstrated in pet birds.[176] In a study to characterize *Staphylococcus* spp. isolated from commensal cutaneous microflora of privately owned birds and birds from a sanctuary for unwanted psittacines in the American desert southwest, companion birds were twice as likely to have positive results for staphylococci than sanctuary birds (71% vs 35%), and the species of staphylococci isolated from captive parrot were more commonly isolated from people.[177] Intimate contact between pets and their owners creates favorable conditions for MRSA transmission.[173] Concordantly, the owners of pets are more often colonized by MRSA than the general population.[178] Moreover, cases of MRSA infection in avian species have been documented.[114,180,181] Therefore, it is crucial that avian veterinarians adopt a responsible and unified approach to usage of antibacterial drugs (see also Chapter 7). Responsible prescribing of antibacterial drugs is also part of the strategy to minimize nosocomial multiresistant bacterial infection in veterinary facilities.

ESTABLISH PRESCRIBING PRACTICE POLICIES

Veterinary prescribing guidelines have been developed for many fields of veterinary medicine with the objective to confer benefits to animal and public health while preserving and maintaining the therapeutic efficacy of antimicrobials.[13,182] While contributing to educate veterinarians on the prudent use of antimicrobials, these guidelines do not provide specific directions regarding appropriate use of specific antimicrobials in a specific clinical situation. The development of individual practice policies regarding specific use of antimicrobials is likely to be more effective,[183] despite not being available in most practices.[184] Development of an antimicrobial policy was recently developed in a zoological medicine service after internal analysis revealed most antimicrobial prescriptions were not indicated or inappropriate.[185]

Charts that display strategies for selecting antimicrobials for common avian clinical conditions based on the organ system involved are tailored to assist veterinarians improve prescribing of antimicrobials in outpatient settings and inpatient care (Tables 6.10A-E). In addition, avian practices should develop a prescribing policy that includes a list of conditions for which antimicrobials are unnecessary and clinical situations for which antimicrobial prophylaxis is required. Studies in veterinary medicine have shown that establishing practice policies has a positive impact on prescribing habits, leading to a reduction in the frequency of antibacterial prescribing, such as fluoroquinolones, over the years.[175]

ANTIMICROBIAL SELECTION

As mentioned in the previous section, selection of antimicrobials should ideally be based on a definitive diagnosis of the microorganism involved and, in case of a bacterial pathogen, results from culture and sensitivity testing (see also Chapter 5). If this is not possible, then consider the most likely pathogens involved and the ability of the drug to achieve adequate tissue concentration at the infection site (Tables 6.10A-E). Beware that granuloma formation, commonly associated with subchronic and chronic infection in birds, inhibits drug penetration. Therefore, lipophilic drugs may be preferred and local treatment (debridement), when possible, will improve drug tissue penetration.

SELECT PROPER DOSE, FREQUENCY, AND ROUTE OF ADMINISTRATION

It is vital to use antimicrobials at an appropriate dose.[13] However, choosing a dose can be challenging as formularies often list a wide range of dosages, and pharmacokinetics of antimicrobials may not be available in the species treated or a closely related species. It is important to keep in mind that selection of resistant mutants takes place in concentration ranges above the MIC, when the wild types can no longer survive, but mutants can (Figure 6.6).[186] For some (but not all) antimicrobials, concentrations are achievable that also inhibit these mutants, as long as the dosing is high enough to exceed this mutant preventive concentration (MPC). During the period of the dosing interval that concentrations are between MPC and MIC, resistant mutants are selected; this concentration range is called the mutant selection window (MSW).[187] This mutant selection mechanism will also take place in the microbiota of the patient! Furthermore, published pharmacokinetic data represent mean data obtained from a limited number of healthy birds, while the immune status of the patient may influence therapeutic outcome. Dosages may also have to be modified in neonates and in animals with impaired liver or kidney function. Nevertheless, basic pharmacodynamic data of antimicrobials can be considered when selecting a dosing regimen. Concentration-dependent antimicrobial agents such as fluoroquinolones and aminoglycosides may be administered parenterally once daily if high peak concentrations are achieved, whereas time-dependent antimicrobials such as beta-lactams, sulfonamides, tetracycline, and macrolides must be given more frequently to maintain plasma concentration above the target plasma concentration. Of note, penicillins and cephalosporins have short duration of action in birds compared to mammals and must be administered orally at least three to four times daily.[188]

The frequency and route of administration of a drug are key elements to consider when choosing an antimicrobial regimen in an avian patient. The clinical status of the patient and its familiarity with human handling must be taken into consideration. In critical patients and those not familiar with human handling, a treatment regimen that can be administered with minimal handling through a vascular access or with longer dosage interval is preferred.

DETERMINE PROPER DURATION OF TREATMENT

Generally, the duration of treatment should be as short as possible. In human medicine, several studies have suggested that shortening duration of antimicrobial treatment may be helpful to limit the emergence of resistance, without negatively affecting clinical outcome.[189] Hence, there has been a shift toward much shorter courses of antimicrobial therapy.

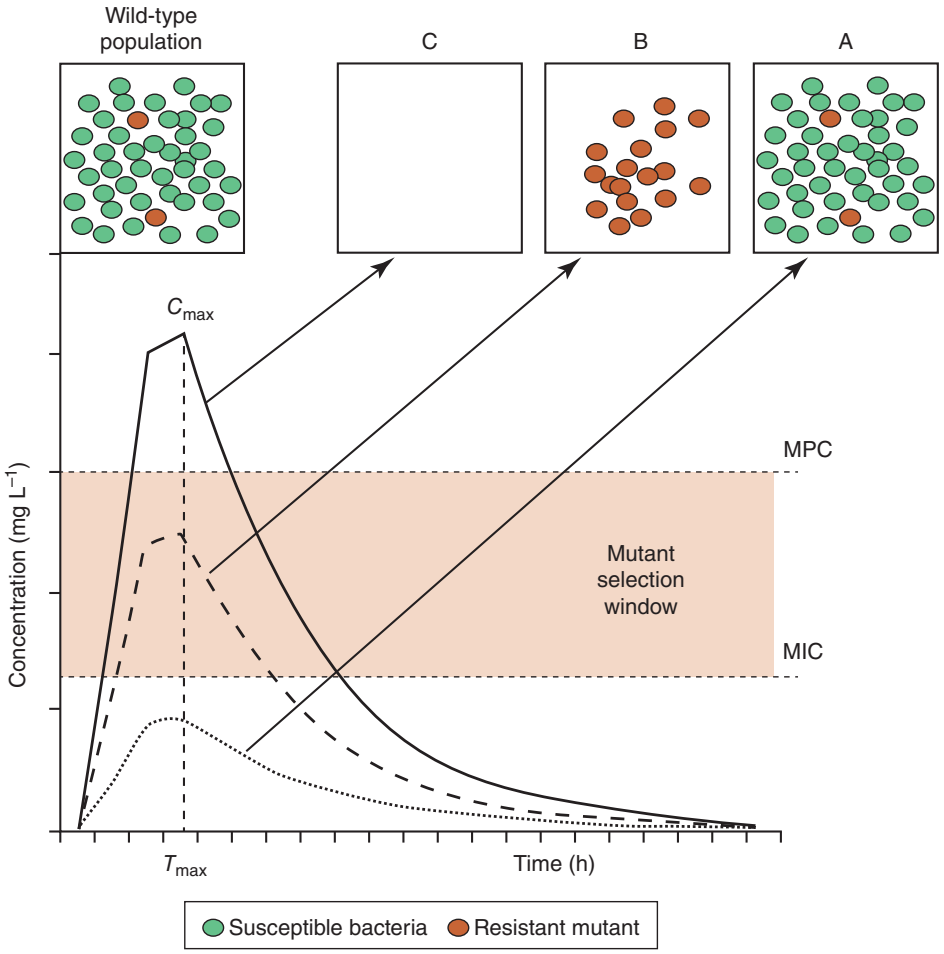


FIGURE 6.6 Mutant selection window (MSW) and mutant prevention concentration (MPC). The figure illustrates three different situations where an antimicrobial drug is administered. *Curves* represent the pharmacokinetics (concentration over time) of the antimicrobial agent and *square boxes* represent the bacterial population. (A) The pharmacokinetic curve is below the MIC; thus, no selection of a resistant mutant subpopulation within the wild-type population is expected. (B) The pharmacokinetic curve is mainly within the MSW; therefore, the resistant mutant subpopulation within the wild-type population can be selectable. (C) The pharmacokinetic curve surpasses the MPC; thus, the susceptible microorganisms are inhibited and selection of a resistant mutant subpopulation is potentially avoided. (From Canton et al: 2011, FEMS,[186] with permission.)

For example, septic arthritis in children is treated for 10 days, as there is no difference in outcome between a 10-day and a 30-day course of antibiotics.[190] In veterinary medicine, a prospective study in 47 dogs with uncomplicated pneumonia provides preliminary data (dogs not randomized, bacterial pneumonia not confirmed in all cases) following the same trend. There were no significant differences in radiographic resolution and relapse rates between dogs treated with a short course of antibacterial drugs (<14 days) compared to those treated with a longer course (>14 days).[191] The American College of Veterinary Internal Medicine (ACVIM) Consensus Statement on antimicrobial use in animals advises that antimicrobials should never be continued once there is clinical and microbiological evidence that an infection has been eliminated.[13] In avian medicine, the data to determine the shortest effective period of therapy are very limited. In 2010, a 21-day course of either doxycycline (35 mg/kg q24h) or azithromycin (40 mg/kg q48h) was effective in eliminating *Chlamydia psittaci* infection in experimentally inoculated cockatiels.[192] Historically, the recommended treatment period for chlamydiosis was 45 days, except in budgerigars, where 30 days of treatment can be effective.[193] Until further studies in avian species are performed comparing different treatment durations of the same antimicrobial agent for a specific condition, selection of the shortest treatment duration will remain challenging.

The clinical experience of avian veterinarians with diverse infections is helpful in assessing response to treatment. Indirect monitoring techniques can be used to evaluate the drug's efficacy such as repeated physical examination, complete blood count (CBC), plasma biochemical analysis, and repeated culture and sensitivity testing.[194] Direct therapeutic drug monitoring

can involve establishing or maintaining a patient within a therapeutic window to decrease the risk of adverse effects and ensure efficacy, or calculating a dosing interval.[194] However, in the absence of species-specific therapeutic intervals and therapeutic windows, direct methods are rarely used in avian medicine.

ANALYZE THERAPEUTIC FAILURE

Therapeutic failure when using antimicrobials has many causes. Table 6.9 presents a list of potential reasons avian practitioners may consider in understanding why undesirable results occurred. Antimicrobial therapy alone is no substitute for an integrated approach to the patient in the event of an infection. Appropriate management of the infection site by debriding ischemic tissue, removing foreign material, blood clots or fluid pockets, and/or flushing out caseous exudate should be performed when applicable.

Figure 6.7 provides a flowchart in which most of the above-discussed concepts have been integrated to help assist avian veterinarians in using antimicrobial agents judiciously until evidence-based treatment guidelines become available for common infections seen in birds.

TABLE 6.9

Potential Causes of Antimicrobial Therapeutic Failure[139,142]

Causes Related to the Pathogen	Causes Related to the Antimicrobial	Causes Related to the Patient
◆ Incorrect diagnosis (e.g., viral disease misdiagnosed as bacterial infection) ◆ Inaccurate results of laboratory tests ◆ Antimicrobial resistance ◆ Metabolic state of the pathogen (biofilm)	◆ Inappropriate dose, frequency, and route of administration ◆ Inadequate antimicrobial selection ◆ Poor diffusion at the target tissue (e.g., cross the blood-brain barrier) ◆ Poor performance inactivity of the selected drug at the site of infection (e.g., abscess)	◆ Underlying neoplastic process ◆ Presence of foreign material (foreign body, necrotic tissue) ◆ Depressed host immunity (concurrent debilitating illness, severe, and/or chronic malnutrition) ◆ Owner compliance

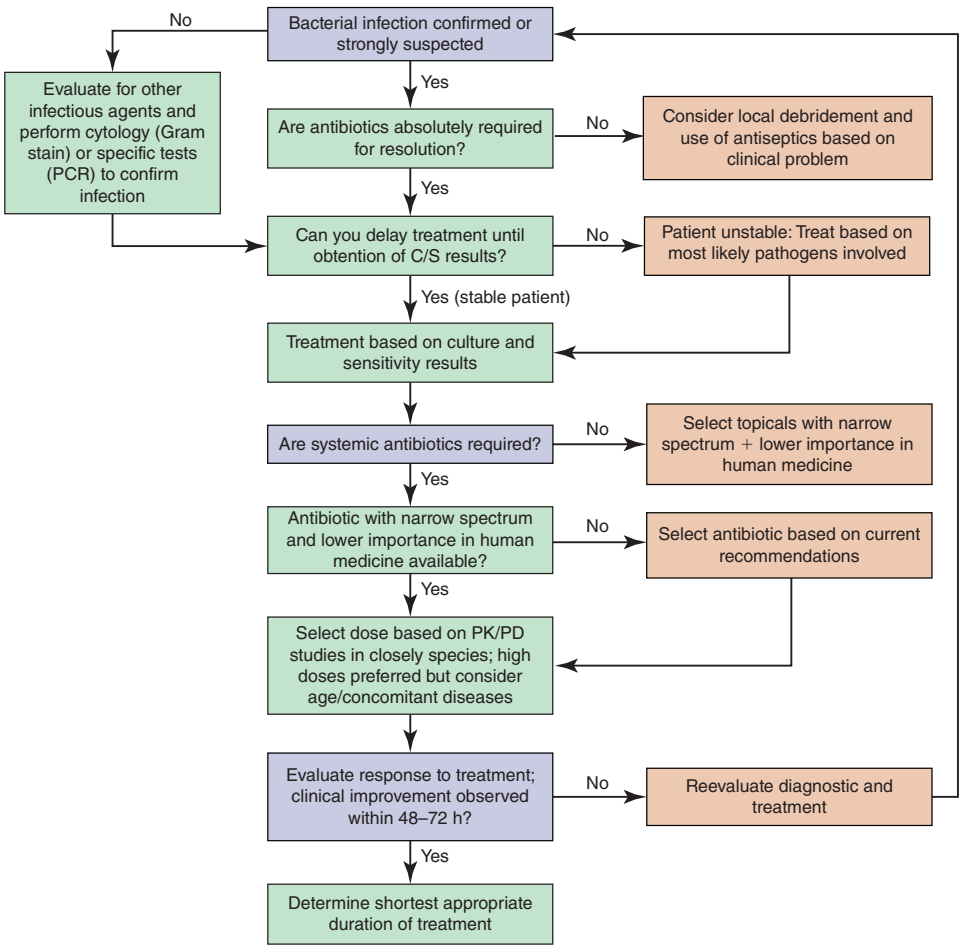


FIGURE 6.7 Flowchart for judicious use of antimicrobial drugs in avian medicine. *C/S*, Culture/sensitivity; *PCR*, polymerase chain reaction; *PK/PD*, pharmacokinetics/pharmacodynamics.

COMPOUNDING AND EXTRA-LABEL DRUG USE (SEE ALSO CHAPTER 5)

Ingeborg M. van Geijlswijk

Only a few veterinary medicinal products (VMPs) are authorized for birds; even in production animals, the number of antimicrobial VMPs (AVMPs) is limited. And even when AVMPs are available, most of the time the formulation and packages are not suitable for pet avian patients. Drinking water medication is common in food-producing animals, but the ill pet bird might refrain from drinking, and for that reason this route of administration is not preferred. Individual administration by the owner is preferably oral, and a potion is preferred over tablets for several reasons, above all the dosing accuracy. Due to large dosing differences between cats/dogs and birds for beta-lactam antibiotics, broadly available potions of amoxicillin for companion animals are suitable for use in (much smaller) birds, when needed with other excipients like taste correction with fruity aromas. When dosing regimens are more comparable (e.g., metronidazole 50 mg/kg/24 hours), the strength of the potion for cats/dogs may be too high to allow (accurate) dosing in small birds, or additives for disguising taste (e.g., poultry liver) might be unfavorable. For some antimicrobials, human medicinal products are available with a suitable concentration and formulation, for example, in case of metronidazole (Flagyl 40 mg/mL, which contains metronidazole as benzoate to prevent dissolution and metallic taste), TMS (Co-trimoxazole 48 mg/mL), and azithromycin (Azithromycin 40 mg/mL). Some products for drinking water, like tylosin 100%, can be compounded into capsules by a pharmacist, enabling the owner to make an ad hoc solution at a favorable concentration. Note that it is important to realize that for oral use the tartrate ester of tylosin is applied, while tylosin in most injectables is not suitable for oral use due to a much lower bioavailability.

For other substances, most importantly doxycycline, compounding might be necessary in the absence of commercial formulations (see also Chapter 5). Major questions in relation to compounding of antimicrobials and other drugs are as follows:

1) What volume is acceptable to administer (which determines the concentration)?
2) In which solvent is the active substance stable for at least one treatment course (e.g., water or oily solvents), or should it be formulated in a suspension to prevent degradation (e.g., doxycycline)?
3) Is the taste acceptable, is adding a taste excipient necessary and sufficient, or is it necessary to formulate a suspension for this reason (e.g., metronidazole)?

Another aspect is the exposure of the professional to the antimicrobial drug while compounding it and the safety of the patient owner during administration. Exposure to antimicrobials should be reduced to a minimum for all that have nothing to gain. For this reason, breaking or crushing of tablets is to be avoided at all times, unless performed by a professional with adequate personal protection. In EU member states, the Annex of Regulation (EU) 2022/1255 should be consulted to check whether the chosen antimicrobial is not reserved for treatment of infections in humans.

TREATMENT OF COMMON ILLNESSES

Table 6.10A: Psittaciformes—Isabelle Langlois;
Table 6.10B: Passeriformes—Christal Pollock;
Table 6.10C: Falconiformes/Strigiformes—Christal Pollock;
Table 6.10D: Columbiformes—Christal Pollock;
Table 6.10E: Anseriformes—Christal Pollock;

TABLE 6.10A

Recommendations for Judicious Antimicrobial Use in Psittaciformes[139,142,157,188,189,193]

	Body Systems				
	Integument			**Respiratory**	
Common conditions	Dermatitis/folliculitis	Bite wound	Beak/skin trauma	Rhinitis/sinusitis	Tracheitis, pneumonia, airsacculitis
Cytology and culture	Cytology: imprint smear, skin scraping, tape strips Culture: swab, skin biopsy	Deep culture if evolution is unfavorable	Imprint smear Culture if systemic signs are present or evolution is unfavorable	Choanal swab, nasal flush, sinusal wash Sample collected by biopsy in chronic cases	Patient stability may interfere w/ investigations Tracheal lavage, lung/air sac imprint from biopsy sample, lung/air sac biopsy
Most likely pathogens	*Staphylococcus*, *Enterobacter*, *E. coli*	Aerobes Anaerobes	Variable pending origin and location of lesion	Aerobic gram-negative bacteria (*E. coli, Klebsiella,* etc.) *Chlamydia psittaci*	*Aspergillus* spp. Aerobic gram-negative bacteria (*E. coli, Klebsiella,* etc.) *Chlamydia psittaci*
Empirical antimicrobial suggestions	Avoid empirical choice when possible TMS or Amox/Clav	Amox/Clav (stable patient) Consider first-generation cephalosporin and amikacin or cefotaxime or piperacillin (septic shock)	TMS Consider addition of antifungal drug (fluconazole) with beak lesion	Consider local treatment with ophthalmic preparation (tobramycin) Gram-negative infection: TMS Chlamydiosis: doxycycline	Aspergillosis: itraconazole (use low dose in Grey parrots) or terbinafine with amphotericin B nebulization Aerobic gram negative: parenteral TMS or enrofloxacin or cefotaxime Chlamydiosis: doxycycline
Comments	Consider topical therapy with antiseptic solution, dressings, antibacterial cream Treat until recheck evaluation confirms clinical resolution	Always combine with topical therapy Proper wound care (cleansing, debridement, irrigation) reduces infection more than antibiotics (see Chapter 36) Cat bite, deep puncture wound, severe crushing injury; systemic therapy indicated (see Chapter 36)	Proper wound care (cleansing, debridement, irrigation, closure if indicated) is often sufficient, especially if granulation is ongoing (see Chapter 36) Restore beak function using prosthetic or surgical reduction if indicated (see Chapter 31)	Perform nasal/sinusal flush to remove debris in the initial phase of treatment (see Chapter 27 and 28) If *C. psittaci* is likely, perform specific tests (see Chapter 18); adjust treatment duration based on shortest documented course in the species Further diagnostic tests indicated if recurrence or suboptimal response to treatment	Treat for 1 month post-resolution of clinical signs or documented lesions for Aspergillosis (see Chapter 8) If *C. psittaci* is likely, perform specific tests (see Chapter 18); adjust treatment duration based on shortest documented course in the affected species

Amox/Clav, Amoxicillin/clavulanate; *TMS*, trimethoprim-sulfonamides.

Ophthalmic	Gastrointestinal			Reproductive	Orthopedic	Multisystemic
Conjunctivitis, keratitis, uveitis	Ingluvitis	Proventriculitis, ventriculitis	Enteritis, cloacitis	Salpingitis	Septic arthritis, osteomyelitis	Septicemia, multiple organ infection
Conjunctival swab	Crop swab or wash	Fecal sample for direct smear evaluation Proventricular wash for cytology and culture	Fecal sample	Oviductal sample collected through cloacoscopy may be considered	Fine-needle aspirate of affected joint for cytology and culture Bone biopsy for impression smear, culture, histopathology	Patient stability may interfere with investigations Cytology and culture may be performed on fine-needle aspirate, biopsy of various organs
Various bacteria *Chlamydia psittaci*	*Candida albicans* Aerobic gram-negative bacteria (*E. coli, Klebsiella,* etc.)	*Macrorhabdus ornithogaster*	Aerobic gram-negative bacteria (*E. coli, Klebsiella,* etc.) Anaerobic bacteria (*Clostridium*)	Aerobic gram-negative bacteria (*E. coli* most common)	*Staphylococcus* spp. *Streptococcus* spp.	Aerobic gram-negative bacteria (*E. coli, Klebsiella,* etc.) *Coxiella*-like bacteria *Chlamydia psittaci* *Mycobacterium avium* and *M. genavense*
Tobramycin or gentamicin Chlamydiosis: tetracycline topically and doxycycline systemically	Candidiasis: nystatin Gram-negative infection: TMS	Amphotericin B orally for 1 month	Gram-negative infection: TMS or chloramphenicol Anaerobic bacteria: clindamycin	Amox/Clav or TMS or enrofloxacin (severe cases)	Septic arthritis: clindamycin or first-generation cephalosporin or Amox/Clav Osteomyelitis: clindamycin pending culture	Gram-negative infection: enrofloxacin or cefotaxime Coxiella-like: doxycycline and enrofloxacin Chlamydiosis: doxycycline Consider pros and cons of treating mycobacteriosis; if treatment is elected, ensure to use multiple drugs concurrently to prevent the emergence of resistance
Clean eye(s) with eyewash solution twice daily in the initial phase of treatment If *C. psittaci* is likely, perform specific tests (see Chapter 18); adjust doxycycline treatment duration based on shortest documented course in the affected species	Nystatin requires direct contact with *Candida* spp.; if fungal hyphae are seen suggesting deep tissue infection, prefer fluconazole	May not be possible to clear *Macrorhabdus* infection in all birds (see Chapter 2, Infectious Disease in Current Therapy in Avian Medicine and Surgery, Vol. 1) Improve husbandry practices Consider adding sodium benzoate to the drinking water (anecdotally successful; still experi-mental)	*Clostridium* spp. are difficult to isolate and cultivate; diagnosis often relies on cytology	TMS has poor diffusion in granuloma; consider alternative if suspected Check for retained or ruptured egg Salpingectomy may be required (see Chapter 21, Surgery in Current Therapy in Avian Medicine and Surgery, Vol. 1)	*Mycobacterium* spp. rarely cause osteomyelitis (see Chapter 2, Infectious Disease in Current Therapy in Avian Medicine and Surgery, Vol. 1)	Consider specific diagnostic tests for chlamydiosis, *Coxiella*-like bacteria, mycobacteriosis and aspergillosis (see Chapter 18, and Chapter 2, Infectious Disease in Current Therapy in Avian Medicine and Surgery, Vol. 1) Prefer IV/IO administration to rapidly achieve high plasma and tissue concentrations

TABLE 6.10B

Recommendations for Judicious Antimicrobial Use in Passeriformes[125,195–209]

	Body Systems						
	Integument		**Respiratory**			**Ophthalmic**	
Common conditions	Dermatitis, proliferative skin lesions	Cat bite wounds	Rhinitis/ sinusitis	Tracheitis	Pneumonia, airsacculitis	Panophthalmia, iridocyclitis	Conjunctivitis, blepharitis, keratitis
Cytology and culture	Cytology: imprint smear, skin scraping, tape preparation	Deep culture if progression is unfavorable	Cytology: choanal swab, nasal flush, sinus flush	Patient stability may interfere with antemortem diagnostic testing	Patient stability may interfere with antemortem diagnostic testing		Conjunctival swab
	Culture: swab, skin biopsy		Culture: Collect sample via biopsy in chronic cases	Diagnostic necropsy to collect samples or demonstrate the presence of parasites	Lung/air sac imprint, biopsy sample for cytology/culture: include acid-fast staining		
					Diagnostic necropsy to collect samples or demonstrate the presence of mites within the air sacs	Diagnostic necropsy to collect samples or demonstrate the presence of parasites	
Most likely pathogens	*Staphylococcus aureus, S. epidermidis, Streptococcus* spp.	*Pasteurella multocida,* anaerobes	*Staphylococcus* spp., *Streptococcus* spp., *E. coli*, and other Enterobacteriaceae, *Chlamydia psittaci*	*Enterococcus faecalis*	*Enterococcus faecalis, Pseudomonas, Staphylococcus* spp., *Streptococcus* spp., *Mycoplasma gallisepticum* (C/F), *Mycobacterium* spp. (RHS),	*Yersinia pseudotuberculosis*	*Staphylococcus* spp., *Streptococcus* spp., *E. coli* and other Enterobacteriaceae, *Mycoplasma gallisepticum* (HF), *Chlamydia psittaci*
	Knemidokoptes pilae (F)		*Trichomonas* spp.	*Sternostoma tracheacolum* (F/C)	*Sternostoma tracheacolum* (F/C), *Toxoplasma gondii*	*Toxoplasma gondii*	*Toxoplasma gondii*
	Microsporum gallinae, Trichophyton spp.				*Aspergillus fumigatus* (M, F)		

	Gastrointestinal				Musculoskeletal	Multisystemic Infection, Septicemia	Neurologic
	Oropharyngeal plaques, stomatitis	Ingluvitis, esophagitis	Proventriculitis, ventriculitis	Enteritis	Septic arthritis	Multiple organ infection, septicemia	Encephalitis
	Oral swab, wet mount and Gram stain cytology	Crop wash or swab, wet mount and Gram stain cytology	Fresh fecal sample for wet mount or Gram stain cytology Proventricular wash for cytology and culture	Fresh fecal sample for fecal wet mount, flotation, cytology, including acid-fast stain Impression smears of several organs to demonstrate *Atoxoplasma*	Fine-needle aspirate of affected joint for cytology and culture	Patient stability may interfere with investigations Impression smears of several organs to demonstrate *Atoxoplasma*	*Atoxoplasma, Toxoplasma*: Impression smears to demonstrate *Atoxoplasma*
		Aerobic culture		Aerobic culture		Aerobic fecal or tissue culture: fecal excretion of *Salmonella* spp. is intermittent and therefore serial sampling of feces from individual birds is recommended	
				Diagnostic necropsy to collect samples		Diagnostic necropsy to collect samples	Diagnostic necropsy to collect samples
		Salmonella typhimurium		*E. coli* and other Enterobacteriaceae (C/F), *Pseudomonas, Salmonella typhimurium, Yersinia pseudotuberculosis, Campylobacter fetus var jejuni* (TF, E), *Mycobacterium avium* (RHS), *M. intracellulare, M. genavense*	*Staphylococcus* spp., *Streptococcus* spp., *Mycobacterium* spp.	*Salmonella typhimurium*, *Pasteurella multocida* (see cat bite wounds), *Yersinia pseudotuberculosis*, *E. coli* (F/E/N), *Erysipelothrix rhusiopathiae*, *Listeria monocytogenes* (C), *Mycobacterium avium* complex, *Chlamydia psittaci*	
	Trichomonas gallinae	*Trichomonas gallinae*		*Atoxoplasma* (C/F/M), *Cochlosoma* (BF), *Isospora* spp. (C), *Cryptosporidium spp.* (GF)		Atoxoplasma (gf) (FL)	*Toxoplasma gondii*, *Atoxoplasma*, (*Trichomonas* spp.)
	Candida albicans	*Candida albicans* (F)	*Macrorhabdus ornithogaster*				

Continued

TABLE 6.10B

Recommendations for Judicious Antimicrobial Use in Passeriformes—cont'd

	Body Systems						
	Integument		**Respiratory**			**Ophthalmic**	
Common conditions	Dermatitis, proliferative skin lesions	Cat bite wounds	Rhinitis/ sinusitis	Tracheitis	Pneumonia, airsacculitis	Panophthalmia, iridocyclitis	Conjunctivitis, blepharitis, keratitis
Empirical antimicrobial suggestions	*Knemidokoptes:* macrocyclic lactones, pyrethrins (see Chapter 9)			*Sternostoma:* macrocylic lactones (see Chapter 9)	*Sternostoma:* macrocylic lactones (see Chapter 9)		
	TMS, doxycycline	Stable patient: TMS, Amox/Clav	TMS, water-soluble doxycyciline, Amox/Clav	TMS, water-soluble doxycycline	TMS, water-soluble doxycycline	TMS, doxycycline	TMS, water-soluble doxycycline
	Topical, dilute povidone-iodine solution	Sepsis: 1st generation cephalosporin and amikacin or cefotaxime or piperacillin	Amox/Clav	*Enterococcus:* later gen cephalosporin injectables			Mycoplasmosis: tylosin, tetracyclines, topical ophthalmic aminoglycosides
							Tobramycin or gentamicin
			Chlamydiosis: doxycycline	Chlamydiosis: doxycycline		Chlamydiosis: doxycycline	
	Dermatophytosis: nystatin, imidazoles, triazoles				Aspergillosis: triazole or terbinafine with amphotericin B nebulization		

	Gastrointestinal				Musculoskeletal	Multisystemic Infection, Septicemia	Neurologic
	Oropharyngeal plaques, stomatitis	Ingluvitis, esophagitis	Proventriculitis, ventriculitis	Enteritis	Septic arthritis	Multiple organ infection, septicemia	Encephalitis
	Trichomoniasis: nitroimidazoles (see Chapter 9)	Trichomoniasis: nitroimidazoles (see Chapter 9)		Cochlosomosis: nitroimidazoles (see Chapter 9)			
				Atoxoplasmosis: sulfachlorpyridazine, toltrazuril, diclazuril			Atoxoplasmosis: sulfachlorpyrazine or toltrazuril in the drinking water
				Coccidiosis: sulfachlorpyridazine, toltrazuril, or amprolium in the water, oral TMS, sulfadimethoxine			
				Cryptosporidiosis: Treatment may be attempted with paromomycin, azithromycin, roxithromycin			
		TMS		TMS, water-soluble doxycycline	Septic arthritis: Clindamycin or 1st generation cephalosporin or Amox/Clav	Gram-neg: fluoroquinolones or advanced-spectrum penicillin or cephalosporins	
				Campylobacteriosis: erythromycin			
					Chlamydiosis: doxycycline	Chlamydiosis: doxycycline	Chlamydiosis: doxycycline
	Candidiasis: Nystatin	Candidiasis: Nystatin	Macrorhabdus: amphotericin B (nystatin, fluconazole)				

Continued

TABLE 6.10B

Recommendations for Judicious Antimicrobial Use in Passeriformes—cont'd

	Body Systems						
	Integument		**Respiratory**			**Ophthalmic**	
Common conditions	Dermatitis, proliferative skin lesions	Cat bite wounds	Rhinitis/ sinusitis	Tracheitis	Pneumonia, airsacculitis	Panophthalmia, iridocyclitis	Conjunctivitis, blepharitis, keratitis
Comments	Dermatomycosis: HS, American robin blue tit bullfinch	Always combine with topical therapy	Perform nasal/ sinus flush to remove debris in the initial phase of treatment (see Chapters 27 and 28)	*Sternostoma:* an antemortem diagnosis can sometimes be made by transillumination of the trachea (see Chapters 27 and 28)			Clean eye(s) with eyewash solution twice daily in the initial phase of treatment
	Poxvirus is an important DDX (see Chapter 15) Treatment is primarily supportive, although surgical debulking of cutaneous masses may speed healing	Cat bite, deep puncture wound, severe crushing injury; systemic therapy indicated					Mycoplasma: treated birds remained culture or PCR+ for up to 6 months after Rx was ended. Treated birds may become asymptomatic carriers. Clinically affected birds should be euthanized or kept in strict quarantine (see Chapter 2, Infectious Disease in Current Therapy in Avian Medicine and Surgery, Vol. 1)

Amox/Clav, Amoxicillin and clavulanate; *BF*, Bengalese or society finch (*Lonchura striata*); *C*, canaries (*Serinus canaria*); *E*, Estrildidae (Gouldian finches, munias, mannikins, firefinches, parrotfinches, waxbills); *F*, finches; *gf*, greenfinch, goldfinch; *GIT*, gastrointestinal tract; *HF*, house finches (*Haemorhous mexicanus*); *M*, mynah birds (*Acridotheres tristis*); *RHS*, red-hooded siskins (*Spinus cucullatus*);TMS, trimethoprim-sulfonamides.

Gastrointestinal				Musculoskeletal	Multisystemic Infection, Septicemia	Neurologic
Oropharyngeal plaques, stomatitis	Ingluvitis, esophagitis	Proventriculitis, ventriculitis	Enteritis	Septic arthritis	Multiple organ infection, septicemia	Encephalitis
Candidiasis: nystatin requires direct contact with *Candida* spp. The presence of fungal hyphae suggests deep tissue infection; therefore, consider fluconazole. Correct predisposing factors.			Some reports of toxicity with metronidazole or dimetronidazole in some finch species, like owl finches		Salmonellosis, mycobacteriosis: Consider euthanasia *Salmonella* spp. can often be recovered from the feces even after prolonged and apparently effective treatment	Atoxoplasma: disinfection and prevention of fecal contamination are important methods to reduce transmission; however, oocysts are stable for months to years. Bali mynah most seriously impacted
			Enterobacteriaceae: neomycin and spectinomycin were historically used because they are effective and are not absorbed from the GIT			

TABLE 6.10C

Recommendations for Judicious Antimicrobial Use in Falconiformes/Strigiformes[24,210–223]

	Body Systems			
	Integument	**Respiratory**		**Ophthalmic**
Common conditions	Pododermatitis	Tracheitis	Pneumonia, airsacculitis	Conjunctivitis, keratitis
Cytology and culture	Cytology: impression smear Culture: swab, biopsy	Fresh fecal sample for direct smear, flotation; microscopic examination of oral swabs Tracheal lavage if clinical status allows	Lung/air sac imprints or squash preparations from biopsy sample, lung/air sac biopsy, if clinical status allows	Conjunctival swab
Most likely pathogens	*Staphylococcus* spp., mainly *S. aureus, Escherichia coli*	*Trichomonas gallinae* *E. coli* *Aspergillus* spp.	*Serratospiculum* spp. *E. coli, Pseudomonas aeruginosa* *Aspergillus* spp.	*Staphylococcus hyicus, S. intermedius, (Chlamydia buteonis)*
Empirical antimicrobial suggestions	See comments below		Serratospiculosis: IVM, FBZ Aspergillosis: amphotericin B, azoles (clotrimazole, enilconazole, itraconazole, ketoconazole, voriconazole), terbinafine	Tetracycline, erythromycin
Comments	Resistance is of concern; therefore base abx choice on C/S whenever possible. In addition to systemic abx, management can include direct intralesional antibiotic delivery, surgical debridement, and postoperative protective foot casting. Also, modify husbandry and perches as needed (see Chapter 33)		Falcons (*S. seurati* in tropical countries; *S. amaculata* in North America Chapter 2	

abx, Antibiotic; *FBZ*, fenbendazole; *IVM*, ivermectin

Gastrointestinal				Musculoskeletal	Multisystemic Infection, Septicemia
Oropharyngeal plaques, stomatitis	Ingluvitis	Proventriculitis, ventriculitis	Enteritis	Septic arthritis	Septicemia, multiple organ infection
Oropharyngeal swab for wet mount cytology, Gram stain cytology	Crop swab, crop wash for wet mount cytology, Gram stain cytology	Fresh fecal sample for direct smear, flotation	Fresh fecal sample for direct smear, flotation	Fine-needle aspirate of affected joint for cytology and culture	Fine-needle aspirate, biopsy of various organs for cytology and culture
		Proventricular wash for cytology and culture		Bone biopsy for impression smear, culture, histopathology	Blood culture
Trichomonas gallinae	*Trichomonas gallinae*	*Porrocaecum* spp., *Contracaecum* spp.	*Porrocaecum* spp., *Contracaecum* spp.	*Trichomonas gallinae*	*Serratospiculum* spp.
Capillaria	*Capillaria*	*Capillaria*	*Capillaria, Eimeria* spp., *Caryospora* spp.		
E. coli, Pseudomonas aeruginosa	*Candida* spp.			*S. aureus, S. hyicus*	*Pseudomonas aeruginosa*, *E. coli*, *Mycobacterium avium* complex
Trichomoniasis: metronidazole, carnidazole	Trichomoniasis: metronidazole, carnidazole	Nematodiasis: IVM, Drontal Plus (praziquantel + pyrantel pamoate + febantel)	Coccidiosis: sulfadimethoxine, toltrazuril	See comments below	
Trichomoniasis: poor prognosis for severe cases however, successful treatment has been reported with metronidazole × 3–7 days followed by surgical removal of granulomas (see Chapter 22)	Trichomoniasis: birds of prey can develop disease by feeding on infected prey, particularly rock pigeons (*Columba livia*) and mourning doves (*Zenaida macroura*; see Chapter 22)	*Contracaecum* spp. are particularly common in fish-eating birds			

TABLE 6.10D

Recommendations for Judicious Antimicrobial Use in Columbiformes[224–240]

	Body Systems					
	Integument		**Respiratory**		**Ophthalmic**	
Common conditions	Proliferative skin lesions	Dermatitis	Rhinitis/ sinusitis	Tracheitis, pneumonia, airsacculitis	Panophthalmia, iridocyclitis	Conjunctivitis, blepharitis, keratitis
Cytology and culture	Cytology: imprint smear, skin scraping, tape preparation Culture: swab, skin biopsy	Cytology: imprint smear, skin scraping, tape strips Culture: swab, skin biopsy	Cytology: choanal swab, nasal flush, sinus flush Culture: Collect sample via biopsy in chronic cases	Patient stability may interfere with antemortem diagnostic testing	Fine-needle aspirate for cytology and culture	Conjunctival swab
Most likely pathogens	*Knemidokoptes mutans*	*Dermanyssus gallinae, Ornithonyssus sylviarum* *Candida albicans*	*Trichomonas gallinae*	*Sarcocystis calchasi*		

Gastrointestinal				Reproductive	Musculoskeletal	Multisystemic Infection, Septicemia	Neurologic
Oropharyngeal plaques, stomatitis	Ingluvitis	Proventriculitis, ventriculitis	Enteritis	Oophoritis, orchitis	Septic arthritis	Multiple organ infection, septicemia	Encephalitis meningitis
Oral swab, wet mount, and Gram stain cytology	Crop wash or swab, wet mount, and Gram stain cytology Aerobic culture	Fresh fecal sample for wet mount or Gram stain cytology Proventricular wash Dlagnostic necropsy: include proventricular/ventricular scraping	Fresh fecal samples, cloacal swabs, or intestinal contents from very fresh cadavers: wet mount cytology, flotation, direct microscopic examination using Giemsa and acid-fast stains	Biopsy samples can be collected via laparoscopy but diagnosis is typically made at necropsy	Arthrocentesis of swollen joints for cytology and culture	Patient stability may interfere with investigations; however, cytology and culture can be performed on fine-needle aspirate or biopsy of various organs, particularly the liver and spleen Blood culture Diagnostic necropsy: collect fresh tissue samples from lungs, liver, intestines, heart, spleen, kidney	Collection of CSF by lumbar puncture is virtually impossible in birds. Instead, fluid is aspirated from the subarachnoid space or fourth ventricle. The presence of venous sinuses makes hemorrhage and death a significant risk of this procedure Diagnostic necropsy to collect samples
	Aerobic culture	Proventricular wash for cytology and culture	Aerobic and anaerobic cultures: pooling feces over 5 days may be useful because *Salmonella* spp. are shed intermittently				
		Diagnostic necropsy: include proventricular/ventricular scraping	Diagnostic necropsy: include intestinal scraping				
Trichomonas gallinae	*Trichomonas gallinae*	*Trichomonas gallinae* *Tetrameres* spp., *Dispharynx* spp.	*Capillaria obsignata, C. caudinflata* *Hexamita columbae* (*Spironucleus columbae*), *Eimeria columbarum, E. labbaena, Isospora* spp., *Ascaridia columbae, Ornithostrongylus* spp.				*Sarcocystis calchasi*
Candida albicans	*Candida albicans*						

Continued

TABLE 6.10D

Recommendations for Judicious Antimicrobial Use in Columbiformes—cont'd

	Body Systems					
	Integument		**Respiratory**		**Ophthalmic**	
		Salmonella typhimurium var. Copenhagen (paratyphoid) *S. arizonae, S pullorum*	*Pasteurella* spp., *Mycoplasma* spp., *Chlamydia psittaci*	*Mycoplasma* spp.	*Salmonella typhimurium* var. Copenhagen *S. arizonae, S pullorum, Chlamydia psittaci*	*Pasteurella multocida, Hemophilus gallinarum, Chlamydia psittaci, Mycoplasma* spp.
Empirical antimicrobial suggestions	Knemidokoptes: IVM, moxidectin, pyrethrin-based products	Ectoparasites: IVM	Trichomoniasis: carnidazole, ronidazole, metronidazole (Dimetridazole has a low margin of safety)			
		Candidiasis: nystatin, (fluconazole)				
			Chlamydiosis: Doxycycline can be given to individual birds, or chlortetracycline-medicated feed or oxytetracycline-medicated water can be used (see Chapter 18)	Chlamydiosis: doxycycline		Chlamydiosis: doxycycline
		TMS, Amox/Clav, ±florfenicol	TMS, tetracyclines, amoxicillin		TMS, Amox/Clav, ±florfenicol	

Gastrointestinal			Reproductive	Musculoskeletal	Multisystemic Infection, Septicemia	Neurologic
		Salmonella typhimurium var. Copenhagen *E. coli, Klebsiella, Chlamydia psittaci, C. perfringens*	*Salmonella typhimurium* var. Copenhagen	*Salmonella typhimurium* var. Copenhagen *Streptococcus gallolyticus, Pasteurella multocida*	*Salmonella typhimurium* var. Copenhagen *E. coli, Streptococcus gallolyticus, Streptococcus bovis, Klebsiella, Yersinia pseudotuberculosis, Pasteurella multocida, M. avium* complex, *S. arizonae, S pullorum, Chlamydia psittaci*	*Salmonella typhimurium* var. Copenhagen *Streptococcus gallolyticus, S. arizonae, S pullorum*
	Nematodiasis: IVM, FBZ (see caution below)	Nematodiasis: IVM, FBZ; pyrantel pamoate is better for ascarids				
	CAUTION: Most reported cases of benzimidazole toxicosis in birds are in Columbiformes: bone marrow suppression, direct GIT damage					Sarcocystosis: amprolium, sulfamethazine, chlortetracycline was ineffective in chickens
		Coccidiosis: amprolium, toltrazuril, clazuril, sulfas				
Candidiasis: nystatin, (fluconazole)	Candidiasis: nystatin (fluconazole)					
		Chlamydiosis: doxycycline			Chlamydiosis: doxycycline	
		TMS, Amox/Clav, ±florfenicol	TMS, Amox/Clav, ±florfenicol	TMS, amoxicillin, Amox/Clav, ±florfenicol	TMS, Amox/Clav, ±florfenicol	TMS, Amox/Clav, ±florfenicol

Continued

TABLE 6.10D

Recommendations for Judicious Antimicrobial Use in Columbiformes—cont'd

	Body Systems			
	Integument		**Respiratory**	**Ophthalmic**
Comments	Poxvirus is also an important DDX (see Chapter 15).	Debridement of severe trichomoniasis lesions may be indicated	Avoid intramuscular injections in racing pigeons	
		Sinus flushing of abx Consider euthanasia for paratyphoid (Salmonella typhimurium var. Copenhagen): Salmonella spp. can often be recovered from the feces even after prolonged and apparently effective treatment.		

ABX, Antibiotics; *IVM*, ivermectin; *TMS*, trimethoprim-sulfonamides.

TABLE 6.10E

Recommendations for Judicious Antimicrobial Use in Anseriformes[241–247]

	Body Systems			
	Integument	**Respiratory**		**Ophthalmic**
Common conditions	Pododermatitis	Rhinitis/sinusitis	Tracheitis, pneumonia, airsacculitis	Conjunctivitis, keratitis, uveitis
Cytology and culture	Cytology: impression smear	Cytology, culture: pharyngeal swabs, nasal flush, sinus flush, sinus aspirate	Lung/air sac imprint from biopsy sample, lung/air sac biopsy if clinical status allows Tracheal lavage if clinical status allows	Conjunctival swab

Gastrointestinal			Reproductive	Musculoskeletal	Multisystemic Infection, Septicemia	Neurologic
	Nematodiasis: prevents exposure via foraging to intermediate hosts, like pill bugs	Dimetridazole has a low margin of safety and can be toxic (see Chapter 9)				
		Recommend wire flooring and regular cleaning of the loft. Vitamin B supplementation after therapy is also beneficial because sulfas reduce thiamine absorption				*Sarcocystis:* trapping opossums and relocating birds to clean, new premises may reduce flock mortality (see Chapter 2, Infectious Disease in Current Therapy in Avian Medicine and Surgery, Vol. 1)
		There is evidence of growing resistance to sulfas and amprolium (see Chapter 9)				

	Gastrointestinal		Reproductive	Musculoskeletal	Multisystemic Infection, Septicemia	Neurologic
Proventriculitis, ventriculitis	Enteritis	Cloacitis, phallic prolapse	Salpingitis	Septic arthritis, osteomyelitis	Multiorgan infection, septicemia	Encephalitis, meningitis, myelitis
Fresh fecal sample for direct smear evaluation, flotation	Fresh fecal sample for direct smear evaluation, flotation, acid-fast stain	Cytology: impression smear	Samples can potentially be collected via cloacoscopy	Fine-needle aspirate of affected joint for cytology and culture	Fine-needle aspirate, biopsy of various organs for cytology and culture	Biopsy for impression smear, culture, histopathology

Continued

TABLE 6.10E

Recommendations for Judicious Antimicrobial Use in Anseriformes—cont'd

	Body Systems			
	Integument	**Respiratory**		**Ophthalmic**
	Culture: swab, biopsy	Collect sample via biopsy in chronic cases		
Most likely pathogens	*Staphylococcus, Enterobacter, E. coli*	*Riemerella anatipestifer, Hemophilus* spp., *Pasteurella* spp., *E. coli, Pseudomonas* spp., *Erysipelothrix rhusiopathiae, Mycobacterium* spp., *Mycoplasma* spp., *Chlamydia psittaci*	*Riemerella anatipestifer, Pasteurella multocida, Pseudomonas* spp., *Aeromonas hydrophila, Mycoplasma* spp., *(Mycobacterium avium)*	*Riemerella anatipestifer, Pasteurella multocida, Chlamydia psittaci*
		Theromyzon spp.	*Cytodites nudus,* Cyathostoma	*Theromyzon* spp.
			Syngamus trachea, Cyathostoma, Theromyzon spp. *Aspergillus fumigatus*	
Empirical antimicrobial suggestions	TMS, Amox/Clav	Mycoplasmosis: tylosin, tetracyclines, lincomycin, and spectinomycin, valnemulin, tiamulin, tyvalosin	TMS, sulfaquinoxaline, chloramphenicol, florfenicol, penicillin and streptomycin, or sulfaquinoxaline	Tobramycin, gentamicin
		R. anatipestifer: Amox/Clav, cefotaxime, cefuroxime sodium		Chlamydiosis: doxycycline
		Chlamydiosis: doxycycline		

	Gastrointestinal		Reproductive	Musculoskeletal	Multisystemic Infection, Septicemia	Neurologic
Proventricular wash for cytology and culture		Culture: swab, biopsy		Bone biopsy for impression smear, culture, histopathology	Blood culture	*Eimeria* truncata can be found in the urine
Riemerella anatipestifer	*Riemerella anatipestifer, Pasteurella multocida, E. coli, Hemophilus* spp., *Pseudomonas* spp., *Aeromonas hydrophila, Salmonella* spp., (*Mycobacterium avium*)	*Neisseria* spp., *Mycoplasma* spp.	*Riemerella anatipestifer, Mycoplasma* spp.	*Riemerella anatipestifer, Pasteurella multocida, Yersinia pseudotuberculosis, Actinobacillus, E. coli, Erysipelothrix rhusiopathiae, Mycoplasma synoviae, (Mycobacterium avium)*	*Riemerella anatipestifer, Pasteurella multocida, E. coli, Actinobacillus, Pseudomonas* spp., *Aeromonas hydrophila, Erysipelothrix rhusiopathiae*	*Riemerella anatipestifer, Salmonella* spp.
Echinuria, Tetrameres, Eustrongyloides, Amidostomum spp., *Epomidostomum* spp.	*Cryptosporidium avium,* coccidiosis, (acanthocephalans)	*Cryptosporidium avium*				*Eimeria truncata*
					Aspergillus fumigatus (young birds can die peracutely)	
TMS, sulfaquinoxaline, chloramphenicol, florfenicol, penicillin and streptomycin, or sulfaquinoxaline	TMS, sulfaquinoxaline, chloramphenicol, florfenicol, penicillin and streptomycin, or sulfaquinoxaline	Mycoplasmosis: tylosin, tetracyclines, lincomycin, and spectinomycin, valnemulin, tiamulin, tyvalosin	Amxo/Clav, TMS, enrofloxacin (severe cases)	Septic arthritis: clindamycin or first-generation cephalosporin or Amox/Clav	TMS, sulfaquinoxaline, chloramphenicol, florfenicol, penicillin and streptomycin, or sulfaquinoxaline	TMS, sulfaquinoxaline, chloramphenicol, florfenicol, penicillin and streptomycin, or sulfaquinoxaline
			Check for retained or ruptured egg(s), salpingectomy may be required	Osteomyelitis: clindamycin, pending culture	*R. anatipestifer*: Amox/Clav, cefotaxime, cefuroxime sodium	*R. anatipestifer*: Amox/Clav, cefotaxime, cefuroxime sodium
				Pasteurella multocida: beta-lactam antibiotics, oxytetracycline	*Pasteurella multocida*: beta-lactam antibiotics, oxytetracycline or enrofloxacin Recommend euthanasia for *Mycobacterium* spp.	

Continued

TABLE 6.10E

Recommendations for Judicious Antimicrobial Use in Anseriformes—cont'd

Body Systems			
Integument	**Respiratory**	**Ophthalmic**	
	Syngamus: IVM, thiabendazole		
	Aspergillosis: azole drug, amphotericin B		
Comments			
Mycoplasmosis: Antibiotic therapy does not eliminate the organism, but it can resolve clinical signs. It is equally important to assess and treat any underlying stressors. Consider euthanasia for the well-being of the flock (see Chapter 20)			
	Cyathostoma: Treatment with IVM was unsuccessful in a Hawaiian goose *Syngamus:* It is possible to retrieve the worms endoscopically		

IVM, Ivermectin; *TMS*, trimethoprim-sulfonamides.

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	Gastrointestinal	Reproductive	Musculoskeletal	Multisystemic Infection, Septicemia	Neurologic
	Nematodiasis: IVM, FBZ; pyrantel pamoate is better for ascarids				*Eimeria truncata:* sulfonamides
		TMS has poor diffusion in granuloma; consider alternative if suspected.	Pasteurellosis: The organism is easily destroyed by common disinfectants, sunlight, drying, and heat. Therefore, good sanitation practices are an important part of disease prevention and control Mycoplasmosis: Surgical intervention may be necessary but the prognosis is guarded. It is also important to move the bird to softer ground or grass. Antimicrobial treatment does *not* eliminate *Mycoplasma* spp. (see Chapter 20)	*Riemerella anatipestifer*: The management of disease relies upon prompt removal and incineration of carcasses as well as cleaning the environment Free-ranging swans and geese seem to be predisposed to *Pseudomonas* and *Aeromonas* infections, particularly during the summer months	

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CHAPTER 7

Antibacterial Drugs

Isabelle Langlois • Ingeborg M. van Geijlswijk • Christal G. Pollock

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CULTURE RESULTS AND INTERPRETATION

Isabelle Langlois

Laboratories used by veterinary practices should be able to provide information regarding the pathogen's identification and susceptibility within 48 hours of receiving the collected sample. Laboratory results may be misleading for several reasons, summarized in Box 7.1. Laboratories may use selective culture media to enhance the growth of pathogens or inhibit the growth of contaminants, for example, cefsulodin-irgasan-novobiocin (CIN) agar for the selection of *Yersinia* spp. or brilliant green agar (BGA) for the selection of *Salmonella* spp. This might be helpful in a selection of samples and potential pathogens.

Knowledge of commensal microbial flora of avian system is helpful in interpreting culture results. The most frequently identified bacterial organisms isolated from the skin of psittacine birds belongs to the genus *Staphylococcus*, followed by the genus *Corynebacterium*.[1] *Acinetobacter* spp., *Curtobacterium* spp., *Microbacterium* spp., and *Streptococcus* spp. are also commonly isolated. The normal gastrointestinal flora of healthy psittacine birds is predominately composed of gram-positive bacteria. This finding has been recently confirmed by a study investigating the level of agreement between results from Gram stain and bacterial culture from healthy Hispaniolan Amazon parrots *(Amazona ventralis)* fed a pelleted diet.[2] Most bacteria (97.2%) identified were gram-positive. Interestingly, gram-negative organisms were identified in 30% of birds on Gram stains, representing 3% to 15% of the observed bacterial population. *Escherichia coli* was the only gram-negative organism identified on culture. Agreement between results of Gram stain and culture was considered fair, with culture tending to underestimate the true diversity of bacteria colonizing the gastrointestinal tract. Clinical application of this study suggests that Gram stains may be of higher value than culture as screening tests for health assessment.[3] A parallel testing strategy may limit the likelihood of misclassifying the microbial flora of psittacine patients.

Scientific findings in one species may not be extrapolable to other species. Host bird species is the most important factor in determining microbial community composition, although sampling site, diet, and captivity status also contribute to gut bacterial communities.[4] As such, a study investigating gut microbiota in Asian herbivorous, long-distance migratory Anseriformes showed the dominant bacterial phyla across all samples were Firmicutes, Proteobacteria, and *Actinobacteria*, but significant variations were detected among different goose species and sampling sites.[5] In captive Falconiformes and Strigiformes, gram-negative bacteria were the most common isolates, and birds fed chicken had more varied gram-negative bacterial species and in greater numbers per fecal samples.[6] This outlines that interpretation of culture results must take into consideration the patient's species and diet.

ANTIBACTERIAL SUSCEPTIBILITY TESTING AND INTERPRETATION

Christal G. Pollock

Antibacterial susceptibility testing (AST) can be a powerful tool in the judicious selection of antibiotic therapy.[7] For individual patients, AST can assist in identifying the optimal antibiotic regimen by determining the susceptibility and resistance of a specific pathogen to a range of antibacterial agents.[7–11]

On a larger scale, AST can aid in the evaluation of treatment provided by hospitals or even national infectious disease control programs.[7] The data gathered can be used to create a cumulative antibiogram that indicates the proportion of bacterial isolates in a population susceptible (or resistant) to a specific drug.[12,13] A survey on antibiogram use in veterinary medicine showed antibiogram use to be almost systematic in poultry, frequent in pigs, and rare in both bovine and equine medicine.[14] Box 7.2 summarizes the clinical uses of antibiograms.

INDICATIONS

Empirical therapy continues to be an effective choice in simple infections or for known pathogens, such as beta-hemolytic *Streptococcus* spp., *Pasteurella* spp., and *Chlamydia* spp.[11,15,16] Susceptibility testing is used to guide drug choice for difficult-to-treat infections or for isolates that possess acquired resistance mechanisms, such as Enterobacteriaceae, *Pseudomonas* spp., *Staphylococcus* spp., and *Enterococcus* spp.[11,16] Special consideration should also be given to isolates retrieved from osteomyelitis, protected sites, like the eye, immunocompromised patients, or patients recently given antibiotics.[12,17] Even a single dose can be associated with emergent resistance.[12]

BOX 7.1 KEY ELEMENTS TO REMEMBER WHEN INTERPRETING CULTURE RESULTS

- A negative culture does not rule out infection. Failure to isolate the causative agents may be due to poor sample collection, poor sample transport (e.g., anaerobes transported in aerobic transport media), or additional/specific requirements for culture not routinely used by most laboratories (e.g., mycobacteria, Chlamydiales, *Mycoplasma* spp.).
- Identification of microbial agents does not necessarily implicate these organisms as the precipitating cause of the infection as they may be part of the normal flora or secondary invaders or contaminants.
- Bacterial organisms that are part of the normal avian microbiota in a normal physiologic state may also be opportunistic pathogens. Therefore, isolation of a commensal organism does not rule out infection.
- Bacteria isolated may represent local bacterial population in the bird's environment.

Modified from Langlois I: Medical causes of feather damaging behavior, *Vet Clin North Am Exot Anim Pract* 24(1):119–152, 2021.[86]

BOX 7.2 FUNCTION OF ANTIBIOGRAMS

Antibiograms are used to:
Assess local susceptibility rates
Monitor resistance trends over time
Guide selection of empiric antimicrobial treatment
Guide decisions about which antimicrobials are included in hospital formularies

From Wieczorkiewicz S, Sincak C: *The pharmacist's guide to antimicrobial therapy and stewardship*, ed 1, Bethesda, MA, 2016, American Society of Health-System Pharmacist Publications.[13]

SPECIMEN COLLECTION

A pure bacterial culture, containing approximately three to five well-isolated colonies of the organism causing infection, is needed for AST.[8,18] Therefore the clinician must submit samples that are representative of the disease process while minimizing contaminant growth. A surgical preparation should be performed before sample collection, whenever possible.[12] Blood, fluid, or tissue samples are preferable to swabs.[12] Consult your diagnostic laboratory for specific recommendations on appropriate specimen collection, handling, and transport.[16]

SUSCEPTIBILITY TEST TECHNIQUES

For all phenotypic AST, a standardized inoculum is prepared from well-isolated colonies taken from a bacterial culture.[7,8,12] A standard amount of the inoculum is then used to inoculate the testing media.[12] There are several methods available that can be divided into two distinct categories: diffusion and dilution based (see also Chapter 5).[8] Diffusion techniques are based on the inhibition of bacterial growth by an antibacterial agent diffusing through solid media from filter paper disks impregnated with various antibiotics (disk diffusion) or an elongated, absorbent strip containing a known gradient of antibiotic (Epsilometer or E-test) (Figure 7.1).[9,16,19] In dilution-based susceptibility testing, such as broth dilution or agar disk dilution, a standard concentration of the inoculum is combined with increasing concentrations of the antibiotic in a series of tubes or microdilution plates (Figure 7.1).[7,9]

Most veterinary laboratories use agar disk diffusion and/or the broth microdilution method.[7] Specific drug panels are used for each taxonomic group. There are also separate gram-negative and gram-positive panels for diagnostic and surveillance purposes.[7] Although some fastidious organisms, such as *Mycobacterium* spp., may be sent to specialized facilities, routine susceptibility testing is generally not performed for other difficult-to-grow organisms, such as obligate anaerobes and intracellular bacteria.[18]

The checkerboard assay allows laboratories to evaluate the activity of antibiotic combinations on pathogens (Figure 7.2).[7] Although not considered a standardized technique, this difficult and time-consuming assay can be used to evaluate synergy, indifference, or antagonism between two antibacterial agents when they are combined.[7]

ALTERNATIVE METHODOLOGIES

Alternative tests with the potential to reduce the standard processing time associated with AST (36 hours plus transport) include direct susceptibility testing by disk diffusion, polymerase chain reaction (PCR), matrix-assisted laser desorption ionization–time of flight, next-generation sequencing, and whole-genome sequencing.[7,20] Susceptibility tests using genotypic methods do not always require bacterial colonies from culture because resistance genes to certain antimicrobial drugs can be detected using molecular techniques.[8]

SUSCEPTIBILITY TEST RESULTS

Diagnostic laboratories frequently utilize "class testing," in which a particular drug is used to predict the susceptibility of other agents within the same antibiotic class.[7] For example, ampicillin, tetracycline, and clindamycin are routinely tested as the representatives for their respective antibacterial classes.[7] There are certain antibacterial agents, however, for which class testing cannot be performed, such as many macrolides, aminoglycosides, and some cephalosporins.[12]

QUANTITATIVE DATA

The lowest concentration of drug that inhibits visible growth of the organism is reported as the minimum inhibitory concentration (MIC),[8–11] which is expressed in milligrams or micrograms per milliliter.[12,13] A high MIC value means that more drug is needed to affect the organism's function or replication.[12,14] A low value means that less drug is needed to affect the organism.[16] MIC is currently the simplest and best measure we have of the antibacterial effect in vitro.[21]

For each organism-antibiotic pair, there is a particular cutoff MIC that defines susceptibility called the breakpoint (Figure 7.1).[10,12,16] Breakpoints or interpretative criteria differ not only based on organism-antibiotic pairs but also by the

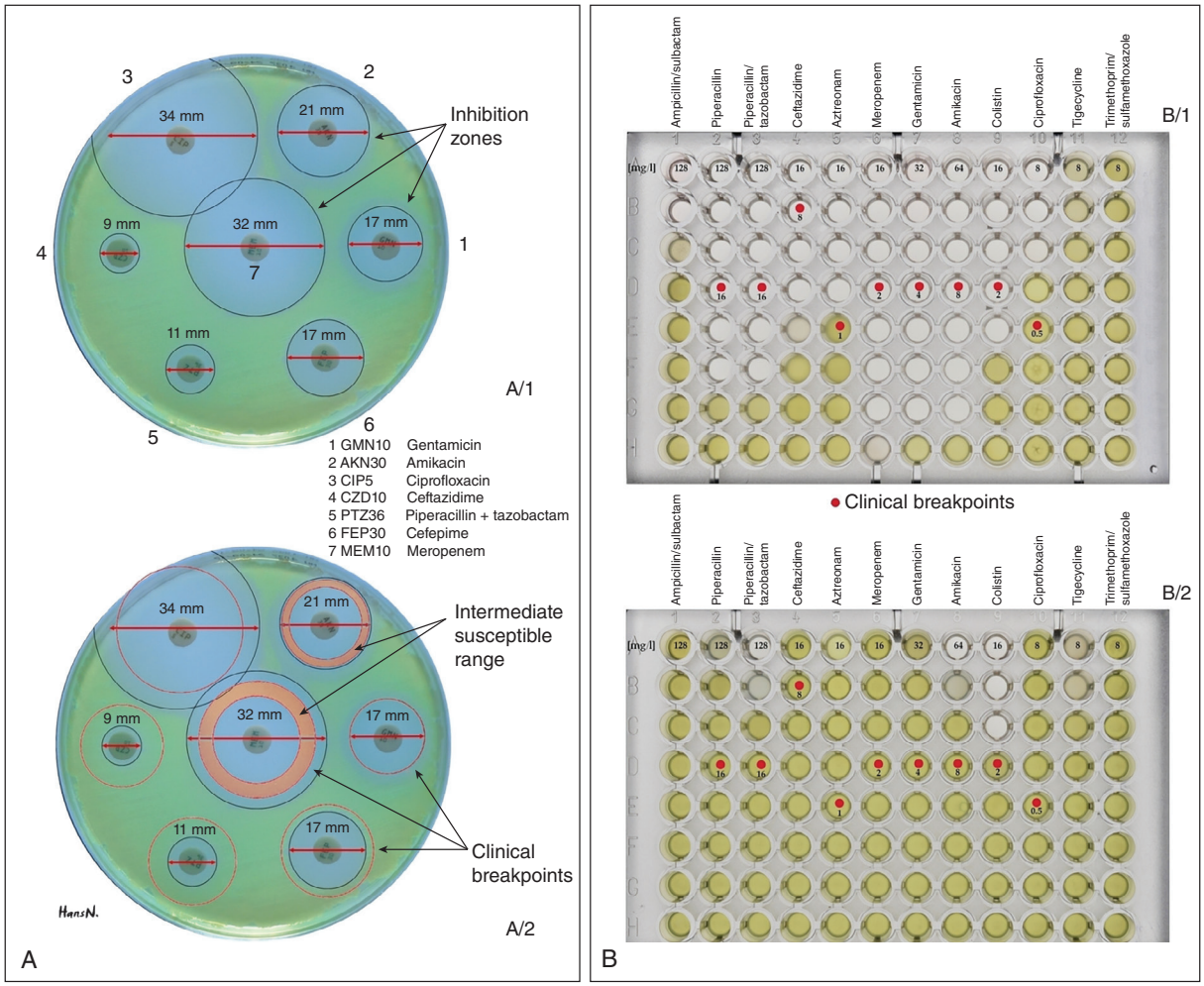


FIGURE 7.1 (A) Agar disk diffusion, also known as the Kirby-Bauer test. (B) Broth dilution using a microdilution plate. *Red dots* indicate clinical breakpoints. (Photo courtesy HansN via Wikimedia Commons.)

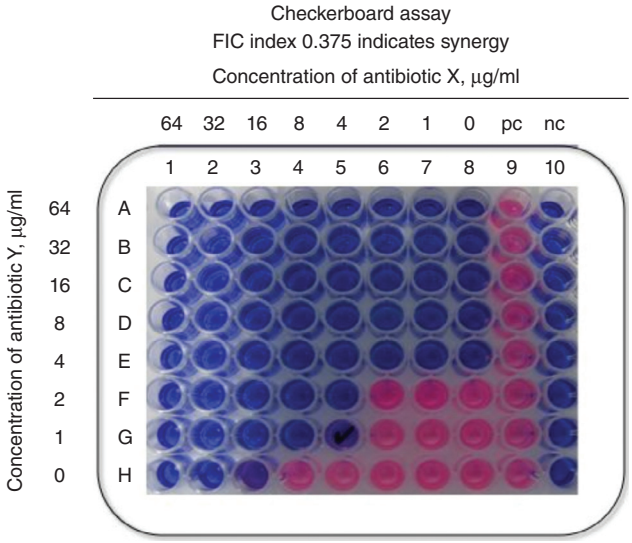


FIGURE 7.2 In the checkerboard assay, one antimicrobial agent is added vertically at two-fold dilutions to a 96-well plate while another agent is added horizontally to produce a "checkerboard" of various combined drug concentrations.[7] (Photo courtesy Dr. Marina Berditsch, IBG-2/IOC, KIT.)

site of infection.[10] Breakpoints are determined after careful and exhaustive review of pharmacodynamics (PD) and pharmacokinetics (PK), and they are regularly reviewed, particularly when mechanisms of bacterial resistance to a specific drug have been described.[12,16,21]

Initially, veterinary diagnostic laboratories had to rely heavily on human standards; however, the veterinary subcommittee of the Clinical and Laboratory Standards Institute has created a growing number of breakpoints for antibacterial agents in companion animal species, like dogs, cats, horses, cattle, and swine.[7,16] In chickens and turkeys, a veterinary-specific breakpoint has been identified for enrofloxacin.[7] Fortunately, similarities in PK and pathogen susceptibilities between humans and animals allow human breakpoints to be extrapolated for many drugs.[16]

QUALITATIVE DATA

Using available clinical breakpoints, an organism can also be categorized as susceptible ("S"), resistant ("R"), or intermediate ("I").[10] Susceptible is associated with a high probability of success following treatment and resistant is associated with a low probability of success.[9,13] Intermediate is a poorly defined range that is used when results fall into a buffer zone between susceptible and resistant strains.[10,16] Although successful therapy is possible in some circumstances, an intermediate result can mean that certain variables in the AST were not properly controlled (see the following section on Limitations of Susceptibility Testing).[10,16] Some isolates can also be reported as susceptible dose dependent ("S-DD"), which means the microbe may be susceptible to higher and/or more frequent dosing but is likely to be resistant to low doses (see the section on Integration of Pharmacokinetic/Pharmacodynamic Data).[10,13,16] If you are unclear about what information was used to determine "S"/"I"/"R" on your patient's report, clarify this point with your laboratory.[7]

LIMITATIONS OF SUSCEPTIBILITY TESTING

Pitfalls in susceptibility testing can occur at every stage of the AST process,[7,12,16] beginning with sampling, continuing through identification and testing, and concluding with interpretation and dose design.[12,18]

- Was the culture pure? Or does it consist of mixed colonies?[8]
- Was the microbe cultured truly a pathogen? Neither contaminants nor normal bacterial flora should be selected for AST.[7,17] This is why cytology often complements AST, since findings such as phagocytosis of the organism can be indicative of pathogenicity.[12]
- Was the isolate speciated? It is particularly important to speciate genera such as *Enterococcus*, *Streptococcus*, and *Staphylococcus* to identify the source of infection and/or determine proper treatment.[12]
- How was growth or absence of growth detected? Human observation versus computer-enhanced images or spectrophotometry can contribute to variability in results.[12]
- Are proper PK/PD data available? This crucial information is lacking for many antimicrobials used in companion birds, and extrapolation of data among different species is questionable, particularly for drugs that are orally administered and lipid soluble. Oral bioavailability can differ markedly among species.[12]

Another limitation of susceptibility testing is detection of acquired resistance that is rapidly induced by the presence of the drug.[12] This might be best exemplified by gram-negative organisms that produce extended-spectrum beta-lactamases.[12] These enzymes are induced at the site of infection by the presence of the drug and generally are not expressed by the isolate cultured in vitro.[12]

INTERPRETATION OF TEST RESULTS

Susceptibility testing measures the direct interactions between the antibacterial agent and the organism in vitro, but this is only one aspect of a very complicated clinical scenario.[21] Just because an antibiotic has the lowest MIC for a pathogen does not mean it is the best choice for an individual patient.[10] Susceptibility involves complex in vivo factors related to dose and dosing schedule, PK of the antibacterial in the individual, adequacy of host defenses, the site of infection, and other numerous factors that can influence the clinical outcome.[7,9,12,15,16,22] Therefore it is the job of the clinician to determine which "susceptible" drugs are most likely to achieve therapeutic success by considering a variety of drug, microbial, and host factors.[7,12,22]

INTEGRATION OF PHARMACOKINETIC/PHARMACODYNAMIC DATA

Breakpoints are related to achievable drug levels in target tissues.[7] Since drug concentrations vary in different body sites or fluids, PK studies are required to determine if therapeutic levels are reached in target tissues.[9]

The PD properties of an antibiotic refer to the manner in which the antibacterial drug exerts its effects on the target organism.[9,10] As previously stated, for most infections, outcomes using appropriate bacteriostatic or bactericidal drugs are similar; however, bactericidal antibiotics are preferred in immunocompromised patients and possibly those with osteomyelitis (see Table 6.5).[10] In these cases, it can be useful to know the lowest concentration of an antibacterial agent required to kill the organism or the minimal bactericidal concentration (MBC).[9] The MBC is determined by subculturing a small sample from the MIC assay onto growth medium and examining it for bacterial growth. Serial dilutions are made of the antimicrobial in bacterial growth media. After a standard incubation, the lowest concentration that has reduced the number of colonies by 99.9% is the MBC.[9] Bactericidal antibiotics usually have an MBC equal or similar to the MIC, whereas bacteriostatic antibiotics usually have an MBC significantly higher than the MIC (see Table 6.5).[9] While MIC testing is standard in most laboratories, MBC testing is more difficult to perform and is not routinely done in clinical practice.[10,16]

Antibiotics are also classified as either time or concentration dependent (Table 7.1). This information will help determine if it is better to administer a high dose of antibiotics all at once or achieve lower concentrations for a longer time. Table 7.2 illustrates the intersection of breakpoints, MIC, and PD data.[10]

TABLE 7.1

Concentration- Versus Time-Dependent Drugs[9,12,21–23]

Parameter	Concentration-Dependent Drugs[a]	Time-Dependent Drugs[b]
Examples	Fluoroquinolones,[c] aminoglycosides, metronidazole, doxycycline, vancomycin	Beta-lactams,[c] some macrolides, tetracyclines, trimethoprim-sulfa combinations, chloramphenicol, carbapenems, vancomycin, linezolid, lincosamides
Factor that increases drug efficacy	Drug concentration increases several-fold above the minimum inhibitory concentration (MIC) of a given pathogen[d]	Length of time that serum levels exceed the MIC of a given pathogen
Optimal dosing strategy	Use highest effective dose (without toxicity) at long dosing intervals	Frequent drug administration

[a]Also known as "dose dependent."
[b]Also known as "dose independent."
[c]Some fluoroquinolones, beta-lactams, tetracyclines, and glycopeptides possess characteristics of both time- and concentration-dependent classes.[12,23] Their best predictor of efficacy is the 24-hour area under the serum concentration versus time curve (AUC)/MIC ratio.
[d]It is not necessary or even beneficial to maintain drug levels above the MIC between doses, as it might increase toxicity.

TABLE 7.2

Examples of Antibiotic Susceptibility Breakpoints to *Escherichia coli*[10]

Antibiotics	Susceptible (Breakpoint)	Susceptible, Dose Dependent	Intermediate	Resistant
Ampicillin	≤8 mg/L		16 mg/L	≥32 mg/L
Cefepime	≤2 mg/L	4–8 mg/L		≥16 mg/L
Levofloxacin	≤2 mg/L		4 mg/L	≥8 mg/L
Trimethoprim/sulfamethoxazole	≤2/38 mg/L			≥4/76 mg/L

Breakpoints differ for various pathogen-antibiotic combinations. In this example, the breakpoint for levofloxacin against *E. coli* is 2 mg/L and 8 mg/L for ampicillin. If an isolate of *E. coli* from the patient's bloodstream is found to have an MIC of 1 mg/L for levofloxacin but 2 mg/L for ampicillin, it does not mean that levofloxacin is a better choice for that patient. Levofloxacin is a concentration-dependent drug typically dosed much less frequently than the time-dependent drug, ampicillin. The much higher concentration of ampicillin achieved in the body (due to higher doses) means that organisms with a higher MIC to ampicillin are still susceptible to this antimicrobial agent. In other words, the MIC values for levofloxacin and ampicillin are not directly comparable.[10]

The postantibiotic effect (PAE) is a period of suppressed bacterial growth seen after brief exposure of the organism to the antimicrobial.[12] The PAE is generally longer in vivo than in vitro.[23] Clinically, this translates to inhibited bacterial growth even though the drug is no longer present and thus is below the MIC of the infecting microbe.[12] The impact of the PAE on antimicrobial efficacy can be profound, particularly for concentration-dependent drugs.[12] It is the PAE that allows some drugs to be administered at long intervals despite short half-lives.[12] The mechanisms of action of the PAE are varied. For beta-lactams, it is the length of time the organism takes to synthesize new penicillin-binding proteins.[23] Beta-lactams only express PAE for gram-positive bacteria, with the exception of carbapenems which may also have a gram-negative PAE.[23] Antimicrobial drugs that inhibit DNA or protein synthesis, such as macrolides, fluoroquinolones, and aminoglycosides, tend to exert PAE against gram-negative bacteria.[23]

HOST FACTORS

The penetrability of host target tissue must also be considered.[9,12,16] If the target is a protected site, such as the central nervous system or eye, or perhaps a granuloma that cannot be accessed surgically, then a lipid-soluble drug is preferred (Box 7.3).[12] If there are no significant tissue barriers to penetrate and there is adequate blood supply, then a water-soluble antibacterial drug, such as beta-lactams or aminoglycosides, is recommended.[12,16]

Also consider the patient's clinical status.[12] A bactericidal drug is recommended if the patient is immunocompromised or severely debilitated (see Table 6.5).[12] If there is marked inflammation, a "susceptible" drug that accumulates in phagocytic heterophils, such as fluoroquinolones, macrolides, or lincosamides, should be considered.[12] Also consider the risk of altered drug disposition if the patient suffers from comorbidities.[9] For

BOX 7.3 LIPID-SOLUBLE ANTIBIOTICS

- Trimethoprim-sulfa
- Doxycycline
- Macrolides
- Clindamycin
- Chloramphenicol
- Fluoroquinolones

From Boothe DM: Interpreting culture and susceptibility data in critical care: perks and pitfalls, *J Vet Emerg Crit Care (San Antonio)* 20(1):110–131, 2010.[12]

instance, nephrotoxicity is a risk with the administration of aminoglycosides or sulfa drugs.

ANTIBACTERIAL PROPHYLAXIS

Isabelle Langlois, Ingeborg M. van Geijlswijk

Antibacterial prophylaxis consists of initiating antimicrobial chemotherapy in a patient considered to be at risk of infection in order to prevent an infection from developing. Patients considered at risk of infection include those undergoing surgical procedures or those with immunosuppression for a variety of reasons including cancer chemotherapy. When elected, antibacterial prophylaxis should have the smallest impact on the patient's microbiome and the microbiologic ecology of the hospital to prevent detrimental effect on the patient and work environment with increased risk of superinfection, development of bacterial resistance, additional cost of hospitalization, and increased incidence of adverse effects for the patient.[24]

PROPHYLACTIC ANTIBIOTIC USE IN THE SURGICAL PATIENT

Prophylactic use of antibacterial drugs is indicated when there is clearly an increased risk of infection or when infection would be catastrophic. The aim of antibacterial prophylaxis in the surgical patient is the reduction of microbial burden and the risk of intraoperative contamination such that normal host defenses can prevent infection.[25] The first, and of utmost importance, decisions avian clinician have to make regarding their surgical patient is whether or not antibacterial prophylaxis is indicated. Of course, antibacterial drug use does not replace proper surgical technique (atraumatic tissue handling, appropriate use of sutures, adequate hemostasis, elimination of dead space) and strict adhesion to aseptic techniques.[26,27]

Drug Selection

The drug selected must be present at the surgical site during the time of potential contamination.[28] Antibiotic prophylaxis should be selected based on expected microorganisms most likely to contaminate the surgical site and their anticipated resistance patterns as determined by recent laboratory data. These microorganisms are different in different species. While in mammals the main bacteria at the surgical site will consist of *Staphylococcus* spp. and *Streptococcus* spp. (which are covered by the surgical site infection [SSI] prophylactic antibiotic cefazolin), in reptiles *Pseudomonas* spp. are abundant, and in most avian species the feather microbiota were dominated by the phyla Proteobacteria, Actinobacteria, and Bacteroidetes.[29] These bacteria are known for producing antibiotics themselves.

Low incidence of adverse effects, available PK of the drug in the species of interest, PD indices of favorable outcome, and cost are other pertinent elements to consider. The use of newer broad-spectrum medication should be avoided in surgical prophylaxis to decrease the emergence of resistant isolates.[30] The goal is to use an antibiotic that is effective at least against 80% of the probable pathogens.[31] This recommendation remains a challenge with the increasing widespread resistance. Many bacterial isolates now have MIC approaching the threshold of susceptibility.[31] Therefore, the higher end of the dosing should be used, and for time-dependent drugs, intervals may need to be shortened.

Method of Administration

Intravenous or intraosseous prophylactic antibiotics should be given no earlier than 30 to 60 minutes prior to surgical incision, and they should ideally be discontinued at or within 24 hours of the end of the surgery.[26,31] The use of oral antibacterial drugs for prophylaxis may not be prudent since peak concentrations are likely to be lower due to bioavailability.[26] Surgical infection rates in dogs undergoing orthopedic implant surgery were similar when antibacterial prophylaxis was administered perioperatively over 3 hours or as a course continued for 6 days.[31] A study in small animals noted that individuals that received antibiotics without following the aforementioned protocol and those that received only postoperative antibiotics had a higher rate of infection than the group that did not receive any antibiotics.[32] This underscores that prophylactic antibiotics must be used correctly. However, a recent survey showed the use of perioperative antimicrobials in small animal surgery was suboptimal with respect to timing, duration, choice of antibiotic, and selection of surgical cases requiring prophylaxis.[33] Despite the absence of studies regarding prophylactic use of antibiotics in avian surgery, conclusions are likely to be similar. The administration of antibacterial drugs should be repeated intraoperatively if the surgical procedure lasts beyond two half-lives after the first dose.[30] This will ensure adequate drug concentration at the surgical site until wound closure is completed.

The use of prophylactic antibiotics postoperatively is not recommended by both the American Centers for Disease Control and Prevention (CDC) and the European Center for Disease Prevention and Control due to conflicting studies regarding their efficacy and their potential contribution to antibiotic resistance.[31] The administration of the first dose intravenously prior to surgery to obtain a quick efficacious peak at decreasing microbial population and a short course is key to minimizing emergent resistance.[34,35] Recent researches in dogs demonstrated prophylactic use of antibiotics postoperatively is beneficial in reducing SSI following clean procedures where a medical device was implanted.[36,37] Therefore, postoperative prophylactic antibiotics should be limited to this clinical situation with implants. A dosing regimen of 4 to 5 days is sufficient with proper dosing regimen, whereas dosing regimens extending to 10 days facilitate therapeutic failure due to emerging resistance.[34,35]

Factors Involved in the Development of a Surgical Site Infection

Numerous factors determine the likelihood of the development of SSI such as classification of type of wound, factors related to the nature of the procedure to be performed, and patient-related factors.[27]

Wound classification

Recommendations for antibacterial prophylaxis perioperatively in veterinary medicine are based on the classification scheme developed by the CDC.[38] Wounds are classified by degree of contamination to help predict the likelihood that infection will develop (Table 7.3). This scheme is helpful, but

TABLE 7.3

Surgical Wound Classification Based on Centers for Disease Control and Prevention Classification[38]

Classification	Description	Examples
Class 1: Clean	Nontraumatic, noninflamed operative wounds in which the respiratory, gastrointestinal, genitourinary, and oropharyngeal tracts are not entered	♦ Cutaneous mass excision ♦ Constricting toe syndrome surgical correction ♦ Reduction of closed fracture
Class 2: Clean-contaminated	Operative wounds in which the respiratory, gastrointestinal, or genitourinary tract is entered under controlled conditions without unusual contamination	♦ Ingluviotomy ♦ Proventriculotomy ♦ Salpingotomy ♦ Perforation of a surgical glove
Class 3: Contaminated	Open, fresh, accidental wounds; procedures in which gastrointestinal contents or infected oviductal content is spilled or a major break in aseptic technique occurs	♦ Skin laceration ♦ Proventricular spillage during proventriculotomy
Class 4: Dirty or infected	Old traumatic wounds with purulent discharge, devitalized tissue or foreign bodies; procedures in which a viscus is perforated or feces contamination occurs	♦ Chronic open fracture ♦ Abscess excision ♦ Septic coelomitis ♦ Ventral tail laceration

its accuracy in veterinary surgery may vary according to the procedure. Prophylactic antibiotics are not indicated for most clean procedures,[26,39] except if an implant (nonabsorbable suture, polypropylene mesh) is used.[26,31] In those circumstances, their use is seemingly warranted. Antibiotic prophylaxis is indicated for clean-contaminated and contaminated procedures.[31,32] Dirty wounds are considered to be infected. Therefore, in this clinical situation, antibacterial drugs are used therapeutically rather than prophylactically.

Procedure-related factors

Phagocytosis and humoral immunity are significantly diminished when tissue integrity is interrupted during surgery.[31] Therefore, inexperienced surgeons may create more tissue trauma leading to greater susceptibility to infection compared to experienced surgeons.[31] Total surgery time and total anesthesia time are well-established risk factors of SSI in small animals.[32,40,41] The proposed mechanism is by suppression of the immune system associated with a transient decrease in lymphocytes that has been correlated with the duration of the surgery.[31] The risk of infection for animals undergoing a 90-minute procedure is twice as high as that of animals undergoing a 60-minute procedure.[32] Of note, in small animals, anesthesia time is a risk factor for postoperative wound infection independent of the duration of surgery.[40] Therefore, efforts should be made to expedite avian patient preparation and postoperative diagnostic procedures such as radiography following orthopedic surgery, for example.

Patient-related factors

In human medicine, advancing age, obesity, chronic inflammation, depressed immune system, the use of braided/multifilament suture material, and severe concurrent illnesses are cited as risk factors for SSI.[25] Veterinary studies with sufficient statistical power to identify patient-related factors in the development of SSI are scant. A study showed that dogs suffering from hypotension during surgery were 27 times more likely to develop SSI potentially secondary to a decrease in oxygenated blood flow to the surgical site.[42] Some of these factors may also apply to birds and warrant antibiotic prophylaxis.

PROPHYLACTIC USE IN THE CANCER CHEMOTHERAPY PATIENT

Bacterial infection following cancer chemotherapy-induced neutropenia is a serious cause of morbidity and mortality both in human and veterinary patients. Comprehensive guidelines exist to assist human oncologists in prescribing antibacterial prophylaxis. Still, there is an ongoing debate among human oncologists as any decreased incidence in bacterial infections is countered by patient adverse effects and the development of antibiotic resistance. In veterinary medicine, there is a paucity of available resources.[43–45] This is even truer in avian medicine as oncology is in its infancy and there is no established standard of care. This section will review current knowledge in human and small animal veterinary medicine while providing insights on the prophylaxis practices described in the avian literature.

Potential Benefits of Antibacterial Prophylaxis

Control trials evaluating antibiotic prophylaxis in human cancer chemotherapy patients showed there was a statistically significant reduction in fever, microbiologically documented infections, and hospitalization, although there was no significant difference in mortality between the two groups.[46,47] Humans with neutropenia of great severity and duration seem to benefit from prophylactic therapy compared to humans with mild to moderate neutropenia.[48] In veterinary medicine, a recent double-blinded, placebo-controlled study in dogs with lymphoma (n = 39) or osteosarcoma (n = 34) showed administration of trimethoprim-sulfonamide (30 mg/kg PO q12h) for the first 14 days after the dogs' first doxorubicin chemotherapy to result in modest but significant reduction in nonhematologic toxicity (gastrointestinal toxicity, hospitalization, suspected infection).[49] Other veterinary studies have shown no beneficial evidence regarding the efficacy of antimicrobial prophylaxis.[50,51] Globally, the potential advantages of prophylactic therapy include a reduction in infection rate, a reduction in the time to onset of infection, and a reduction in the speed in which an incipient infection develops into

overwhelming sepsis.[24] Ultimately, these benefits may facilitate the home management of neutropenic animals, or heteropenic birds, and improve quality of life.

Potential Disadvantages of Antibacterial Prophylaxis

Antibacterial prophylaxis can exert a selection pressure on the composition and function within the gastrointestinal microbiome of an individual patient, which may result in the emergence of antibiotic-resistant strains. This undesirable effect is well documented in human literature but has yet to be evaluated in the veterinary field. Human studies show increasing reports of fluoroquinolone-resistant bacteria isolated from patients receiving chemotherapy prophylaxis.[52,53] Moreover, there is increasing evidence that disruption of the microbiome can ultimately alter the efficacy of chemotherapy. Mice treated with antibacterial drugs prior to chemotherapy showed decreased tumor necrosis, and mice with lymphoma showed reduced response to cyclophosphamide when receiving antibacterial drugs with gram-positive spectrum.[54,55] Adverse drug reactions and additional expense for the owner are other elements to consider.

Patients at Risk

In human medicine, antibacterial prophylaxis is indicated in patients most at risk of developing sepsis, such as patients with acute leukemias, those on high-dose chemotherapy, and those in the first cycle of chemotherapy.[56,57] In veterinary medicine, the latter was also demonstrated in dogs, with 48.7% of the dogs that became septic after receiving the drug for the first time and 71.8% that were in the induction phase when they became septic.[58] The presence of concomitant diseases (renal failure, heart conditions, etc.) renders human patients more likely to have septic complication.[59]

Timing for Initiating Antibacterial Prophylaxis

In febrile neutropenia cases, treatment with empirical antibiotics is recommended by both veterinary and human texts.[45] In patients that are not septic, starting prophylaxis at the time of chemotherapy protocol initiation is more common in human studies while administration of prophylaxis is more common on documentation of a neutropenia in veterinary patients.[45] Neutropenia is far more likely to occur in human chemotherapy protocols warranting the administration of prophylaxis in anticipation.[45]

The decision to give antibacterial and/or antifungal prophylaxis implies to determine what significant neutropenia is. For most chemotherapy drugs, the neutrophil nadir is at 5 to 7 days postadministration.[45] Therefore, complete blood count (CBC) is performed 1 week postchemotherapy. Heteropenia happens most commonly at approximately 1 week after chemotherapy in avian species as well and warrant weekly monitoring of blood cell counts.[60] According to the American Society of Clinical Oncology, antibacterial and antifungal prophylaxis are only recommended for patients expected to have an absolute neutrophil count (ANC) count of 0.1×10^9/L for >7 days, unless other factors increase risks for complications.[61] The Veterinary Cooperative Oncology Group provides a grading system based on the levels of neutropenia following chemotherapy administration in dogs and cats (Table 7.4).[62] This classification has been used to characterize heteropenia in birds.[63] According to this classification, veterinary patients with an ANC of below 0.5×10^9/L suffer from severe neutropenia. A grade 3 neutropenia ($<1 \times 10^9$/L) is generally considered clinically significant, especially if there is likely to be a decline in neutrophil numbers in the next 24 to 48 hours.[24]

Most veterinary texts therefore recommend antimicrobial prophylaxis in the asymptomatic patient whenever an ANC of ≤ 0.5 to 1.0×10^9/L is present or anticipated.[24,43] Bisson and colleagues recommend using an ANC cutoff 0.75×10^9/L to state prophylactic antimicrobial use is indicated.[45] Use should be considered if an ANC is 0.75 to 1.0×10^9/L with one or more risk factors (hematological malignancies, concomitant disease) and antimicrobial prophylaxis should not be used with an ANC $>1 \times 10^9$/L or with an ANC $>0.75 \times 10^9$/L with no risk factors.[45] These guidelines can be used to assist avian veterinarians in their decision process until research can provide a clearer answer on their application in birds. Routine prophylactic therapy during chemotherapy is not recommended if the owner can closely observe the animal for signs of illness and if the anticipated neutropenia is of short duration.[24] Chemotherapy has been performed successfully in avian patients without prophylactic use of antibiotics.[64,65] Among chemotherapeutic drugs, cyclophosphamide is known to cause significant heteropenia in Galliformes under experimental conditions.[66,67]

Duration of Administration

Most veterinary references advise a 3- to 7-day course of antimicrobials on documentation of neutropenia, although this recommendation is not supported by peer-reviewed literature.[43] If no infection is documented, Bisson and colleagues favor the administration of prophylactic antimicrobials until the ANC has increased back to the aforementioned original

TABLE 7.4

Grading Based on the Levels of Neutropenia Following Chemotherapy or Following Biological Antineoplastic Therapy in Dogs and Cats[62]

Grading	Absolute Neutrophil Count	Clinical Significance
1	1500/μL to lower limit of normal	Mild; observation; intervention not indicated
2	1000–1499/μL	Moderate; outpatient or noninvasive intervention indicated
3	500–999/μL	Severe or medically significant; hospitalization or prolongation of hospitalization indicated
4	<500/μL	Life-threatening; urgent intervention indicated

value used to initiate the therapy (0.75×10^9/L).[45] To monitor the ANC, measurement of CBC 3 days after starting antimicrobials should be performed. This approach appears to be most appropriate since there is no evidence that stopping antibiotic treatment early encourages antibiotic resistance, while taking antibiotics for a longer period than necessary increases the risk of resistance while contributing to antimicrobial overuse.[68]

Choice of Antibacterial Drugs

In humans and small mammals, prophylactic therapy is directed at the intestinal flora to reduce aerobic gram-negative organisms most often responsible for severe infections.[24,31] This principle probably applies to psittacine birds as well since their normal gastrointestinal flora is composed predominately of gram-positive bacteria and pathogenic bacteria are usually gram-negative.[1] The majority of infections occurring in cancer chemotherapy patients results from bacterial translocation from the patient's own gastrointestinal tract.[31,45] Prophylaxis should not be directed toward anaerobic bacterial population since this group contributes to prevent fungal overgrowth and colonization by exogenous organisms.

Extensive and unnecessary antibacterial coverage should be avoided. In human oncology, use of a single antibacterial is recommended unless there is a patient-specific indication.[59] In human medicine, the lack of adequate coverage for gram-positive bacteria when using quinolones has been investigated and showed that while bacteremia episodes were decreased, there was a significant increase in side effects in the patients receiving an additional drug compared to quinolones alone.[57] The most frequently used antibacterial for prophylaxis is levofloxacin, a second-generation fluoroquinolone, because they have minimal side effects, high concentration in feces, and little activity against anaerobic bacteria.[57] TMP-Sulfa (trimethoprim/sulfamethoxazole) have similar broad spectrum and spare anaerobic gastrointestinal flora, but they have been associated with more adverse effects than quinolones in humans.[57]

In small animal oncology, veterinary texts advise oral antibacterials similar to those used in humans such as enrofloxacin and TMP-Sulfa.[24,43] However, unlike TMP-sulfa, fluoroquinolones are listed by the World Health Organization as critically important for human medicine.[69] Therefore, prophylactic use of fluoroquinolones should be highly discouraged and veterinary clinicians have a key role to play in implementing this recommendation.

CHEMOTHERAPY PROPHYLAXIS IN AVIAN MEDICINE

Case reports describing the use of chemotherapy in different avian species have been described in the literature.[63–65,70–82] Among these, some describe the prophylactic use of antibiotics,[70,73,75,76,78,79] while others mention the use of antibiotics in an unspecified manner.[81] Table 7.5 provides a summary of cases for which antibiotic treatments were given prophylactically in association with chemotherapy.

Looking at the timing for prophylaxis administration, contrary to what is typically done in veterinary medicine, that is, to start antibiotic prophylaxis when neutropenia is documented, the administration of prophylaxis in birds is more in line with what is done in human medicine, that is, starting prophylaxis at the time of chemotherapy protocol initiation. The classification provided by the Veterinary Cooperative Oncology Group was not used and antibacterial prophylaxis was administered while absolute heterophil count (AHC) was within or superior to the reference interval for the affected bird in many cases.[73,74,78,80] When a clinically significant decrease in the AHC was identified, follow-up hematology was not performed to consider discontinuation of antibiotics.

The duration of administration of antibacterials was much longer in some cases than the typical a 3- to 7-day course of antibacterial drugs cited in small animal veterinary references when neutropenia is confirmed. In some birds, prophylaxis was started several days prior to chemotherapy,[78,79] and sometimes pursued weeks after chemotherapy is completed.[70]

Monotherapy with a fluoroquinolone or penicillin coupled with beta-lactamase inhibitors was mostly used. While monotherapy is in accordance with current human and veterinary recommendations for antibiotic prophylaxis in chemotherapy patients, the selection of antibiotics of very high importance in human medicine should be abandoned unless a culture and antibiotic susceptibility test supports its use with no other options. Further, penicillins are not ideal choices because they possess activity against anaerobic bacteria.[24] TMP-Sulfa is a commonly used antibiotic to treat bacterial gastrointestinal diseases in birds and should be considered to follow small animal recommendations.

Antifungal prophylaxis was used along with chemotherapy in some avian species known to be susceptible to aspergillosis.[70,76,78] While routine antifungal prophylaxis is not recommended in veterinary medicine,[24] this recommendation may not be appropriate in some avian species and merits further investigations. Along these lines, the use of antimicrobials may be considered in birds receiving corticosteroids. The hypothalamic-pituitary-adrenal system of pigeons was reported to react to exogenous glucocorticoids by early delayed feedback and to be more sensitive to suppression by glucocorticoids than that of mammals.[83] More recently, the safety or side effects of long-term oral steroid administration (prednisolone 1 mg/kg PO q24h for 29 days) were investigated in adult unknown sex Carneau pigeons *(Columba livia)*.[84] There was no evidence of infectious disease on gross or histologic postmortem examination. Overall, no adverse clinical, clinicopathologic, or histopathologic changes were noted in the treated birds compared to the control birds in this study. Along the same lines, a study in ring doves *(Streptopelia risoria)* suggested the presence of a melatonin-corticosterone coupling with the possibility that melatonin released in situations of stress counteracts the adverse effects of glucocorticoids on the organism.[85] Clinical acumen should be used in managing birds receiving corticosteroids in light of the aforementioned information.

The information presented here shows that prophylactic antibiotic administration to birds undergoing chemotherapy can be optimized. Current recommendations in veterinary and human medicine can be adapted to birds while awaiting research to provide answers as to their application and associated results.

TABLE 7.5

Antibacterial Prophylaxis in Avian Chemotherapy Patient

Species	Neoplasm	Chemotherapy	CBC	AHC	Prophylaxis
Mallard duck *(Anas platyrhynchos)*[70]	Sertoli cell tumor	◆ Carboplatin 15 mg/kg diluted in 25 ml 5% dextrose, q4–5 weeks for 4 doses	◆ 2 weeks after each carboplatin dose	◆ After first dose: 1.28 × 10^9/L ◆ After second dose: 0.84 × 10^9/L ◆ After third dose: 0.72 × 10^9/L	◆ Amoxicillin-clavulanic acid 125 mg/kg q12h initiated with chemotherapy and continued for 5 months postchemotherapy
Double yellow-headed Amazon parrot *(Amazona ochrocephala oratrix)*[74]	T-cell chronic lymphocytic leukemia	◆ Chlorambucil 2 mg/kg PO twice weekly for 40 days	◆ Every 1 week	◆ Remained within reference intervals (3.3–8.8 × 10^9/L)	◆ Amoxicillin-clavulanic acid 125 mg/kg q12h initiated with chemotherapy; duration not specified
Green-winged macaw *(Ara chloropterus)*[79]	Chronic lymphocytic leukemia	◆ Chlorambucil 1 mg/kg PO twice weekly with prednisone 1 mg/kg PO q24h (chlorambucil stopped at 6 weeks due to thrombocytopenia) Then 6 weeks later: ◆ Cyclophosphamide 5 mg/kg PO for 4 days q21d, then weekly for 29 weeks	◆ 1, 3, and 6 weeks post initiation of chemotherapy ◆ 7 and 16 days postcessation chlorambucil ◆ Every week after starting cyclophosphamide	◆ AHC above reference interval at 1 (45 × 10^9/L) and 3 (16.5 × 10^9/L) postinitiation of chlorambucil ◆ AHC not presented thereafter	◆ Enrofloxacin 10 mg/kg PO q12h began 5 weeks prior to chemotherapy; duration not specified
African penguin *(Spheniscus demersus)*[78]	Squamous cell carcinoma	◆ Intralesional cisplatin (0.2 mg/kg then 0.9 mg/kg) q7d for 3 doses	◆ Prior to second and third cisplatin injection	◆ AHC within reference intervals	◆ Prophylactic antibiotic (not specified) administered for 28 days prior to chemotherapy ◆ Enrofloxacin 10 mg/kg IM at first injection of cisplatin
Blue-and-gold macaw *(Ara ararauna)*[73]	Fibrosarcoma	◆ Radiation (40 Gy delivered over 22 days q48h) ◆ Intralesional cisplatin 0.3 mg/cm^3 of tissue	◆ Every week during treatment	◆ AHC above or within reference intervals	◆ Enrofloxacin 15 mg/kg PO q12h; duration not specified
Kori bustard *(Ardeotis kori)*[75]	Myxosarcoma	◆ Carboplatin (4.6 mg) impregnated, calcium-based matrix beads (12 beads) surgically placed locally Then 1 month later: ◆ Cyclophosphamide 10 mg/m^2 q24h and meloxicam 0.1 mg/kg q24h for 17 weeks	◆ At beads placement ◆ 1 month post beads placement	◆ AHC (1.7 × 10^9/L) at beads placement ◆ AHC within reference intervals 1 month post–beads placement	◆ Cefazolin 50 mg/kg IV at beads placement ◆ 30-day course of enrofloxacin 10 mg/kg PO q24h started with cyclophosphamide and meloxicam

AHC, Absolute heterophil count; *CBC*, complete blood count; *IM*, intramuscular; *IV*, intravenous; *PO*, per os.

CONCLUSION

Antibacterial drugs are a key treatment component for many bacterial diseases in veterinary and human medicine. Avian veterinarians need to be prudent, responsible, rational, and judicious in their antibacterial drug selection and use. They also need to educate owners to recruit them into partnership in this endeavor. Antibiotic resistance is affecting patient care. It is critical to implement ways to improve antibacterial uses in our daily practice to reduce antibiotic resistance selection and preserve the efficacy of antibacterial agents. Part of antimicrobial stewardship is timely therapy evaluation done within a couple of days, which can stimulate the owners of patients to therapy adherence and minimize the length of antibacterial treatment courses.

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CHAPTER 8

Antifungal Drugs

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An antifungal drug is a pharmaceutical fungicide or fungistatic used to treat and prevent mycosis such as aspergillosis, macrorhabdosis, and candidiasis. The use of antifungals in birds is characterized by interspecies and interindividual variability in the pharmacokinetics, affecting drug efficacy and toxicity. Rational usage of antifungal drugs in avian species is much more than only selecting an appropriate dosage. Different factors such as the chemical structure, resistance, species-specific toxicity, antifungal drug formulation, and optimal route and frequency of administration should be considered.

MECHANISM OF ACTION

Three major groups of antifungals involved in the inhibition of synthesis or direct interaction with ergosterol are frequently used in avian practice: 1) polyene macrolide antibioitcs or polyenes; 2) azole derivatives; and 3) allylamines (Figure 8.1).

Polyene macrolide antibiotics or polyenes, including, amphotericin B, nystatin, and pimaricin (or natamycin), act primarily by binding to ergosterol, a sterol that resides on the cell membranes of fungi and acts to maintain membrane integrity. Following binding to ergosterole polyenes form pores in the plasma membrane, causing leakage of sodium, potassium, and hydrogen ions, which eventually leads to cell death (Figure 8.1). Polyenes have a broad antifungal spectrum, including a variety of yeasts (e.g., *Macrorhabdus ornithogaster*, *Candida* spp., *Rhodotorula* spp.) and molds (e.g., *Aspergillus* spp., *Mucor* spp., *Penicillium* spp.), and can be fungistatic or fungicidal, depending on the dosage.[1–4]

Azoles derivatives or azoles inhibit the enzyme cytochrome P450–dependent 14-α-sterol demethylase, a key enzyme in ergosterol biosynthesis (Figure 8.1). Consequently, exposed fungi become depleted of ergosterol and accumulate 14-α-methylated sterols. This causes disruption of membrane structure and function, thereby inhibiting fungal growth. Azoles are classified into two groups: the imidazoles which have two nitrogens in the azole ring (e.g., clotrimazole, miconazole, enilconazole, and ketoconazole), and triazoles which have three nitrogens in the azole ring (e.g., itraconazole, fluconazole, voriconazole, isavuconazole, and posaconazole).[5–8] The lower host toxicity of triazoles compared to imidazoles has correlated with their lower affinity for avian cytochrome P450 enzymes and their lesser propensity to inhibit avian sterol synthesis. Most azole antifungal agents have fungistatic and broad-spectrum activity against both yeasts and filamentous fungi. At doses used in birds, voriconazole can be fungicidal, but several days are needed to reach steady-state concentrations.[1–4]

Allylamines, such as terbinafine, inhibit the enzyme squalene epoxidase, which converts squalene to ergosterol (Figure 8.1). Inhibition of this enzyme causes squalene, a substance toxic to fungal cells, to accumulate intracellularly and leads to rapid cell death. Allylamines have fungicidal activity against dermatophytes, molds, and certain dimorphic fungi and fungistatic activity against yeasts.[1–4]

In addition to the antimicrobial agents affecting fungal sterols, inhibitors of the glucan synthesis, such as echinocandins (e.g., caspofungin, micafungin, and anidulafungin) also affect the integrity of the fungal cell wall (Figure 8.1). Echinocandins act as specific, noncompetitive inhibitors of the fungal β-(1,3)-glucan synthase complex, an essential component of the cell wall. Inhibition of the β-(1,3)-glucan synthesis leads to cytological and ultrastructural changes in fungi, osmotic instability, and cell death. Echinocandins exhibit fungicidal activities against *Candida* spp. but are fungistatic for *Aspergillus* spp.[4]

Finally, fluoropyrimidines such as 5-fluorocytosine have with inhibitory activity against many yeasts, and fungi, including *Candida* spp., *Cryptococcus neoformans*, and against *Aspergillus* spp. Following uptake by the susceptible fungal cells, 5-fluorocytosine is converted into 5-fluorouracil, which is further converted to metabolites that inhibit fungal RNA and DNA synthesis (Figure 8.1).[4]

Toxicity

The development of safe new antifungal therapies has been constrained by fungal organisms being eukaryotic pathogens that colonize a eukaryotic host, and the narrow range of physiologic differences between the host and the pathogen may represent hurdles to developing safe and wide-ranging antifungal agents. Furthermore, antifungal therapy is typically prolonged and can last several months.

In birds, drug distribution patterns differ between avian species due to anatomic and physiologic differences such as differences in crop and/or gastric pH between avian groups. This may result in species differences in antifungal drug toxicity.

Parenteral amphotericin B administration can induce a dose-dependent nephrotoxicity in mammals, as a result of renal vasoconstriction, reduced glomerular filtration rate, and direct toxic effect on the membranes of the renal tubule cells. Renal

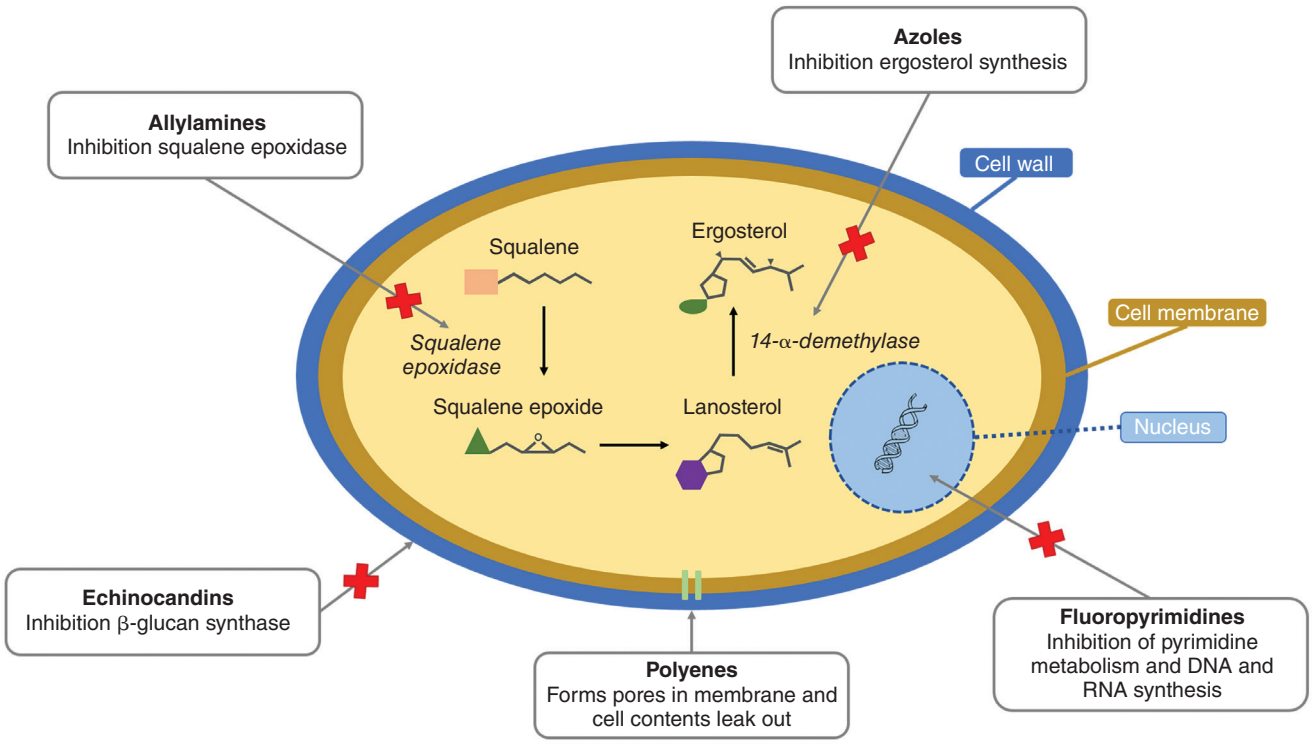


FIGURE 8.1 Mechanisms of action of the major classes of antifungal drugs.

toxicity is related to binding of the drug to the sterol-rich cell membranes in kidney tubules. In contrast to mammals, nephrotoxicity is lessened in birds due to their faster elimination rate. However, due to aforementioned risks, systemic use of amphotericin B deoxycholate should be limited and reserved for severe cases of aspergillosis. Lipid formulations of amphotericin B should be considered in settings in which azoles are contraindicated (e.g., in case of cardiac insufficiency) or not tolerated. Nystatin is poorly absorbed from the gastrointestinal tract, hence having limited-to-no side effects. However, when used parenterally, nystatin may cause high systemic toxicity and thus parenteral administration should be avoided. Therefore, nystatin is used primarily orally or topically to treat *Candida* infections of mucosa, skin, and the gastrointestinal tract. However, use of nystatin should be avoided if the gastrointestinal epithelium is not intact as systemic absorption might occur.[1,4]

The most commonly observed side effects associated with azole administration in avian patients are gastrointestinal signs, such as anorexia and vomiting, and liver dysfunction. Toxicity of azoles is associated with the drug-specific binding to the fungal cytochrome P450 enzyme instead of the vertebrate analog. In general, azoles are well tolerated in birds; however, remarkable species differences are observed. The apparent sensitivity to azoles experienced by different bird species might be explained by differences in the drug's pharmacokinetics. Although undocumented in avian species, genotypic polymorphism of cytochrome P450 isoenzyme CYP2C19 has been linked in humans with a variable sensitivity to voriconazole toxicity.[1,9,10] Currently, pharmacokinetic studies investigating the higher sensitivity to itraconazole toxicity of grey parrots *(Psittacus erithacus)* and Timneh parrots *(Psittacus timneh)* are still lacking. Grey parrots appear to be very sensitive to itraconazole and may exhibit adverse reactions, such as distinct anorexia and depression, at normal dosage levels. Anecdotally, similar adverse reactions have been seen in Senegal parrots *(Poicephalus senegalus)* and birds with impaired liver function. Anecdotally, drowsiness and apathy have also been reported in African fish eagles *(Haliaeetus vocifer)* following administration of 10 mg/kg itraconazole, but scientific evidence is lacking to confirm these observations. Nevertheless, it is advised to be cautious when using itraconazole in (potentially) sensitive species, ensuring not to exceed a dose of 5 mg/kg body weight (BW) in grey parrots, and carefully monitor for occurrence of adverse effects. If these are observed, treatment should be stopped immediately. However, it might be advisable to avoid use itraconazole in these species unless no alternative treatment is available.[1,2]

A prolonged elimination half-life of voriconazole has been observed in pigeons *(Columba livia domestica)* and African penguins *(Spheniscus demersus)*, which might potentially induce drug accumulation with extended dosing and toxicity.[11–13] Following oral administration of voriconazole (10 and 20 mg/kg BW q12h) to pigeons, Beernaert et al. observed hepatotoxicity, demonstrated by hepatomegaly and military hepatic necrosis and, on histology, vacuolization up to apoptosis of hepatocytes and heterophilic and lymphocytic infiltration.[11] Similarly, toxicity was observed in multiple penguin species after administering voriconazole (6.1–22.2 mg/kg BW q12-24h), resulting in clinical signs such as anorexia, lethargy, weakness, change in mentation, ataxia, paresis, apparent vision changes, seizure-like activity, and generalized seizures.[13] However, in most species, including grey parrots, voriconazole is generally characterized by a good tolerability when orally administered. Observed adverse effects during

treatment are slight and rare, and usually present as depression, hepatopathy, and polyuria. Similarly, limited adverse effects have been reported for fluconazole and posaconazole.

Of the imidazoles, enilconazole and clotrimazole rarely cause side effects when used topically. However, in humans it has been reported that clotrimazole induces edema, erythema, vesication, desquamation, pruritus, and urticarial after cutaneous application. Prudent use of ketoconazole is recommended since adverse liver, renal, gastrointestinal, and circulatory side effects have been reported following parenteral administration.[2] Furthermore, caution is advised when azoles are coadministered with drugs being potent cytochrome P450 inhibitors, such as midazolam, enrofloxacin, and clindamycin.[2,13]

The use of terbinafine is generally associated with limited side effects, such as mild gastrointestinal symptoms and hepatobiliary dysfunction. In red-tailed hawks *(Buteo jamaicensis)*, oral administration of a high-dose terbinafine (120 mg/kg BW) induced regurgitation.[14] Anecdotally, mild gastrointestinal toxicity and hepatobiliary dysfunction were observed in some psittacine species such as a grey parrot, a blue-fronted Amazon parrot *(Amazona aestiva)*, and a Senegal parrot *(Poicephalus senegalus)* following long-term administration of terbinafine (10–15 mg/kg BW q12h) alone or in combination with itraconazole or voriconazole (van Zeeland, personal communication). Likewise, regurgitation was anecdotally reported in macaws (*Ara* spp.) treated solely with either voriconazole or itraconazole (van Zeeland, personal communication).

No clinical studies are available describing the usage of echinocandins in avian patients; however, limited toxicity might be observed in humans, such as cardiovascular toxicity.

DRUG FORMULATION

Stability, solubility, absorption, and severe toxicity are among the major challenges of antifungal drugs compromising optimal treatment. Most of the polyene, allylamine, azole, and echinocandin derivatives are very slightly soluble in water and sparingly in organic solvents. Different formulations and drug delivery strategies have been developed to improve the antifungal pharmacokinetics with targeted delivery, rapidly followed by sustained release and prolonged retention of high drug concentration localized at the infection site.[1] To reduce the intrinsic toxicity and to enhance efficacy of amphotericin B, different lipid formulations have been developed, that is, amphotericin B lipid complex, liposomal amphotericin B, and amphotericin B colloidal dispersion. In budgerigars *(Melopsittacus undulatus)* and cockatiels, *(Nymphicus hollandicus)*, more than half of birds stopped shedding *M. ornithogaster* following treatment with amphotericin B colloidal dispersion via drinking water at 0.1 mg/mL for 28 days.[15] Nevertheless, in-depth information on the pharmacokinetics, pharmacodynamics, and clinical use of amphotericin B lipid formulations in birds is lacking. Based on *in vitro* and *in vivo* studies in rodents, dogs, and humans, lipid formulations of amphotericin B are suggested to generally have a slower onset of action since they require dissociation of free amphotericin from the lipid vehicle. The incorporation of amphotericin B into a lipid vesicle will enhance drug uptake by the liver and spleen and cause accumulation of the drug by the mononuclear phagocyte system at sites of capillary damage and inflammation.[1,4] Topical treatment of a deep infection with *Aspergillus fumigatus* of the pectoral muscle in a goliath heron *(Ardea goliath)* with liposomal amphotericin B (1.35 mg/kg q24h) (mixed with sterile, water-soluble, gelatin lubricant for more than 1 month) led to the complete resolution of the wound infection. No signs of nephrotoxicity were observed in this bird based on clinical examination and plasma uric acid levels.[16] Following intratracheal aerosol administration of a single dose (3 mg/kg BW) of liposomal amphotericin B to healthy mallard ducks *(Anas platyrhynchos)*, drug concentrations in pulmonary parenchyma exceeded the targeted minimal inhibitory concentration (MIC) for avian isolates of *Aspergillus* spp. of 1 μg/mL, while systemic levels of amphotericin B in plasma and kidney were low.[17] In human medicine, differences in pharmacokinetics and toxicity between conventional amphotericin B deoxycholate and lipid formulations are reflected in the recommended dosages: amphotericin B deoxycholate 0.25 to 1.5 mg/kg q24h, amphotericin B lipid complex 5 mg/kg BW q24h, liposomal amphotericin B 1 to 5 mg/kg BW q24h, and amphotericin B colloidal dispersion 3 to 5 mg/kg q24h.[18] Comparatively, the conventional amphotericin B deoxycholate is administered at a dose of 1 to 1.5 mg/kg BW q8h to birds.[1,19] Nanotechnology holds great promise to overcome the limitations of antifungal drug solubility and toxicity. Nanoparticles are characterized by their small particle size ranging from 1 to 1,000 nm and can be classified into phospholipid vesicles (e.g., liposomes), nonphospholipid vesicles, polymeric nanoparticles, polymeric micelles, solid lipid nanoparticles, nanostructured lipid carriers, nanoemulsions, and dendrimers. In falcons (*Falco* spp.), it was demonstrated that itraconazole-loaded nanostructured lipid carriers penetrate deeply in the lungs and air sacs, which is a prerequisite for pulmonary treatment of aspergillosis.[1,20] Innovative antifungal drug formulations are mostly only available as human-approved products. Because of the high cost of this type of product, attempts to use more cost-effective formulations in veterinary medicine, such as compounded formulations, have been attempted. However, these formulations are mostly characterized by a low systemic absorption; hence, their usage is not recommended.

DRUG RESISTANCE

Antifungal susceptibility testing may be helpful for monitoring therapy in avian species. Standardized in vitro antifungal susceptibility testing methods for yeast and molds have been developed by the Clinical and Laboratory Standards Institute (CLSI) and the European Committee on Antimicrobial Susceptibility Testing (EUCAST), whereby the MIC is considered as the lowest concentration giving rise to an inhibition of growth of 50% or more and referenced to a clinical breakpoint. Additionally, the minimal fungicidal concentration (MFC) is defined as the lowest concentration showing approximately 99.0% to 99.5% of killing activity. Acquired resistance of *A. fumigatus* strains, isolated from companion and wild birds, is reported for amphotericin B, itraconazole, voriconazole, and caspofungin.[21,22] Some isolates have been described with resistance to more than one antifungal drug (amphotericin + itraconazole, itraconazole + voriconazole, and caspofungin + amphotericin).[21,22] Although historically

used, significant resistance of *Candida albicans* to itraconazole has been reported.[23] Finally, also evidence of nystatin and of amphotericin B resistance in *M. ornithogaster* have been reported.[24]

Combined Antifungal Therapy

Treatment of fungal diseases is often complicated by poor response and high toxicity; therefore, double-drug combinations might be promising to enhance efficacy. Unfortunately, data on the effect of combining different types of antifungals in avian patients are lacking; however, data from human medicine demonstrate some interesting findings. *In vitro* studies have shown that combined use of antifungals can broaden the coverage, increase the fungicidal effect, and decrease the risk of development of resistance. Furthermore, combined agents can have synergistic activity with decreased toxicity. Nonetheless, different concentrations of each drug combination can be associated with results that range from antagonism to synergy, and host factors can greatly affect the clinical outcome. Synergy might occur by simultaneous inhibition of different fungal cell targets, for example, amphotericin B and echinocandins.[25] Similarly, a synergistic interaction between terbinafine and itraconazole has been observed *in vitro* for *A. fumigatus*, resulting in a reduced MIC and MFC. The synergistic effect of the combination terbinafine-itraconazole was even observed for an itraconazole-resistant *A. fumigatus* strain.[26] Amphotericin B and azoles damage the cell membrane, which might increase the uptake of other agents like 5-fluorocytosine. Since amphotericin B and azoles target the same site, simultaneous administration might result in an antagonistic interaction, where azoles prevent the synthesis of ergosterol, and render amphotericin B, which binds the ergosterol in the cell membrane, inactive. In avian medicine, the use of certain aerosolized disinfectants such as F10 (a blend of quaternary ammonium and biguanide compounds) (Health and Hygiene [Pty] Ltd, Johannesburg, South Africa) and lufenuron has been reported to enhance efficacy of inhalation therapy with azoles (e.g., enilconazole), but scientific studies on the safety and efficacy of these substances are lacking.[27] Anecdotally, the combination of nystatin and fluconazole increases drug efficiency and decreases the required volume to be administered for successful treatment of severe cases of gastrointestinal candidiasis compared to separate use of the substances. Likewise, the alternate combination of amphotericin B and nystatin (distributed across three to four time points) has been used successfully in birds with macrorhabdosis (van Zeeland, personal communication). Different routes of administration can also be combined, such as systemic triazole therapy (itraconazole or posaconazole) combined with inhalation therapy or topical intranasal clotrimazole or enilconazole.[25]

PRACTICAL GUIDELINES ON ADMINISTRATION AND TREATMENT

Drug administration and choice of drugs and additional measures taken to treat fungal infections depend mainly on the course and the severity of the disease. Depending on the drug formulation, antimycotic drugs need to be given regularly, i.e., once (SID, q24h) or twice (BID, q12h) daily, to achieve a systemic effect. In most cases, treatment needs to be continued for at least 21 to 28 days in milder cases, up to 3 to 4 months or even longer in severe cases, and a combination of several approaches is usually required. Antimycotic drugs may be administered orally, via nebulization, intratracheally, intramuscularly, subcutaneously, intravenously, and topically onto fungal lesions, for example, inside the air sac during endoscopic examination (Figure 8.2).[2] Consultation of pharmacokinetic studies is recommendable prior to making decisions on the use of particular antifungal agents and formulation of the drug in the individual avian patient.

Oral Therapy

Oral treatment with antifungal drugs is the most common route of drug delivery for systemic treatment of aspergillosis or microsporidia infections (e.g., *Encephalitozoon* spp. or *Enterocytozoon* spp.) as well as for the treatment of gastrointestinal yeast such as *Candida* spp. (most common *C. albicans* but also *C. parapsilosis*, *C. rugosa*, *C. krusei*, and *C. tropicalis*) and *M. ornithogaster*. Gastrointestinal yeast infections are usually treated with formulations that act locally and are not absorbed from the gastrointestinal tract.[28] For the treatment in passerines and psittacines, especially when dealing with treatment of a multi-bird aviary, powdered formulations of nystatin from small mammal medicine (e.g., RodiCare Nystatin, Alfavet, Neumünster, Germany) can be used and sprinkled over the food. Additionally, 28-day treatment regimens with amphotericin B via the drinkwater (alone or combined with individual oral treatment) have been described using a powder for the preparation of an infusion solution (amphotericin B 50 mg Pulver, Bristol-Myers Squibb GmbH & Co, KGaA, Monheim am Rhein, Germany) mixed at 0.1 mg/mL.[15] For the treatment of aspergillosis, oral formulations of azoles (mainly itraconazole and voriconazole) are most commonly used, which are absorbed from the gastrointestinal tract following oral administration. Oral suspensions are preferably dosed and administered using syringes. Because suspensions are not available for all antimycotic drugs, as an alternative, tablets can be dissolved in sterile water; however, demixing of these self-made drug solutions and unequal distribution of active ingredients must be considered. Microsporidia such as *Encephalitozoon* spp. (e.g., *E. hellem* or *E. cuniculi*) or *Enterocytozoon bieneusi*, which were initially regarded as protozoal parasites but are now recognized as fungi, require a different approach. These microsporidial organisms can be associated with renal, hepatic (e.g., granulomatous hepatitis), intestinal, and ocular infection, and are commonly treated with albendazole, sometimes combined with fumagillin, for a minimum duration of 4 weeks.[29] Medical training of the avian patient helps to facilitate (long-term) oral medication (see Chapter 46). When birds are trained to take medicine voluntarily from spoons or syringes, this is an immense advantage for targeted and stress-free treatment. To increase acceptance, drugs (suspensions, solutions, beats, or tablets) may be hidden in food (e.g., in mice for raptors), soaked up in treats or mixed with preferred porridge, meshed fruits, yogurt, or juice. Soaking of millets and grain feed with antifungal amphotericin B suspensions can provide a suitable option for treating avian gastric yeast and candidiasis. However, it should be kept in mind that any mixture of drugs carries a risk for altered resorption due to interaction of the drug and the carrier substance. Nevertheless, administration with food is usually more promising than mixing of drug solutions in the

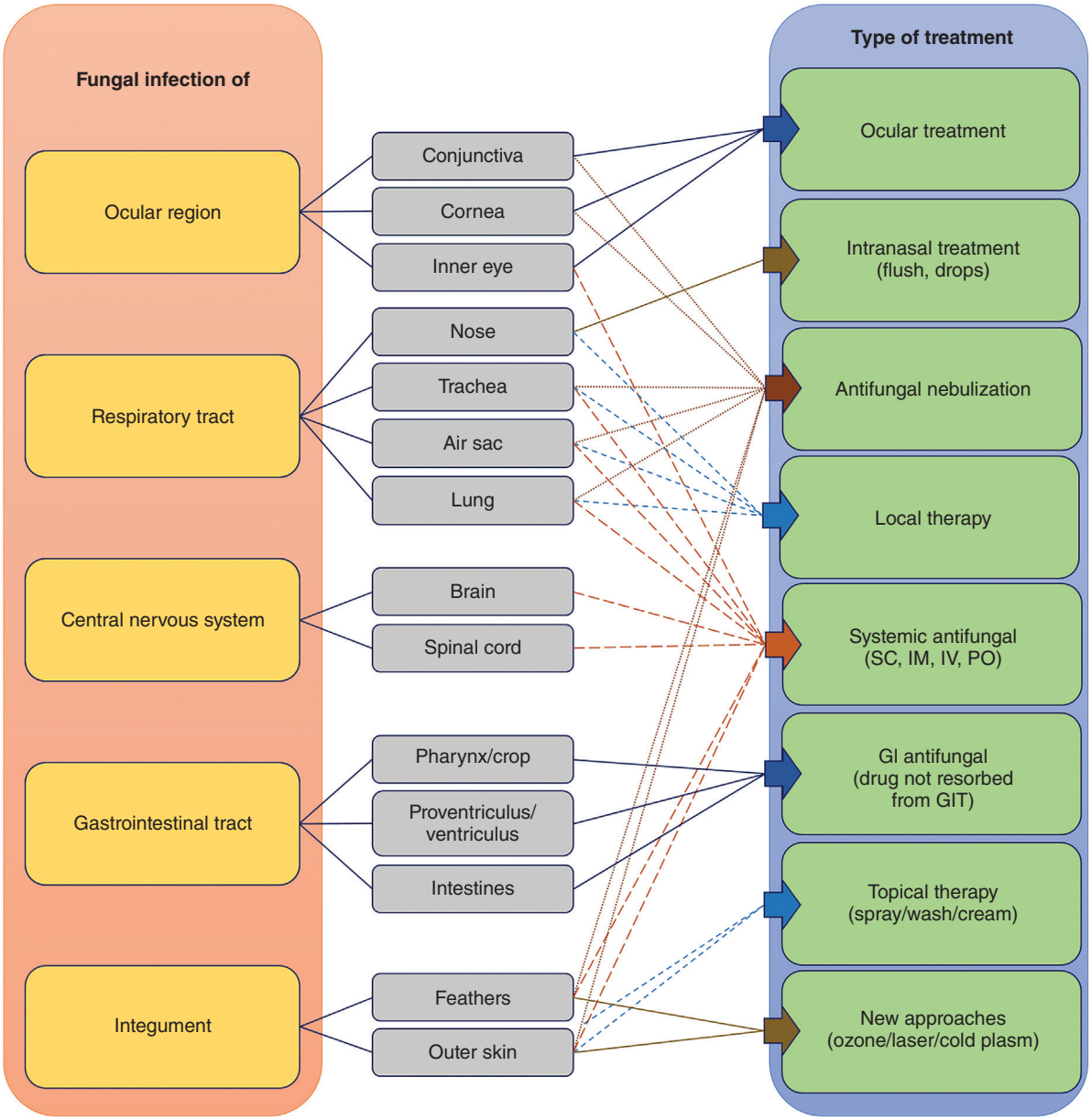


FIGURE 8.2 Antifungal treatment decision tree. *GI*, gastrointestinal; *GIT*, gastrointestinal tract; *IM*, intramuscular; *PO*, oral; *SC*, subcutaneous.

drinking water, unless specific formulations for the preparation of an infusion solution are used.[15] In other cases, demixing of drug solution may occur, as well as unequal or insufficient water uptake by individual birds, especially in case of xerophilic species that are adapted to deserts and hot climates such as budgerigars. Nevertheless, sodium benzoate at 500 to 1000 mg/L of drinking water has been suggested to successfully treat candidiasis in budgerigars.[30] Concerns have been raised with toxicity when using these high dosages, particularly in parent-fed nestlings or species that have higher fluid requirements than budgerigars. When birds do not ingest drugs with a small amount of food voluntarily and are not trained to drug administration, manual restraint is usually necessary to facilitate dropwise drug instillation, administration of tablets, or gavage/tube feeding. Independent from potential interactions with the food contents, bioavailability after oral administration may vary between avian species, dependent on the pH within the gastrointestinal tract, which needs to be considered by the veterinarian.

Inhalation Therapy

Inhalation therapy is preferably combined with oral medication, offering a valuable and 'stress-free' addition to the therapeutic regimen in the absence of major side effects.[31,32] Nebulization of antimycotic drugs (e.g., amphotericin B, terbinafine, or triazoles such as enilconazole and clotrimazole) has been regularly reported for the treatment of aspergillosis, despite the lack of antifungal drugs specifically designed or licensed for nebulization in veterinary or human medicine.[2] The antifungal medication is mixed with

saline solution and nebulized using a commercial inhalation device. Aerosol droplets produced should be sufficiently fine and respirable (droplet size smaller than 3–5 μm) to be able to enter parabronchial structures and atria. Ultrasonic nebulizers will generally suffice for hydrophilic substances; however, to accommodate the large lipophilic antifungal molecules, compressor nebulizers will often be required.[1] Fogging using a commercially available "smogger" unit has been used inside aviaries in the Middle East to treat a larger number of individuals at the same time.[27] Birds inhale the aerosolized antifungal agent via the respiratory route, enabling the drug to reach lesions in the upper respiratory tract, air sacs, and lungs; however, the timing of nebulization and the type of drug involved are important factors to consider (see also Chapters 5 and 6). Handling of birds and associated stress can be limited with inhalation therapy. Patients may be placed in specifically designed nebulization chambers or in transport boxes that are sealed off with towels or blankets to ensure concentrated delivery of aerosolized medication for at least 20 to 30 minutes (Figure 8.3).[27] From an occupational safety perspective, nebulization chambers/boxes should be placed in a well-ventilated room where no people are present for the duration of the nebulization process to minimize the human exposure to antifungal substances. It can be advantageous to the patient and overall success of the antifungal treatment if stress is minimized, e.g. by training the patient to voluntarily enter and stay inside the nebulization chambers, or by hooding a raptor and leaving it hooded while inside the nebulization box.

Parenteral Administration

In severe cases of systemic fungal infections, antifungal drugs may be administered parenterally via the subcutaneous, intramuscular, or intravenous route. Likewise, parenteral injections are an alternative administration route to oral medication in patients with vomitus or regurgitation or those with acute courses of aspergillosis or unusual localizations of fungal infections (e.g., in joints, bones, or the spinal cord). As with any oral drug delivery, medical training will facilitate stress-free medication in birds (Figure 8.4). Injectable solutions are available for amphotericin B, fluconazole, voriconazole, and posaconazole, but the therapeutic efficacy of fluconazole against *Aspergillus* spp. appears low.[2] Voriconazole has been used successfully in various species in recent years,[33–39] with relatively few side effects and larger clinical utility compared to amphotericin B. However, a faster onset of therapeutic action has been reported for amphotericin B compared to the triazole drugs. This might explain why this agent is often used for a limited time during the initial phases of aspergillosis treatment. For treatment of (potentially zoonotic) infections with *Cryptococcus neoformans*, which may result in ocular and nasal discharge, sinusitis, respiratory distress, CNS signs, and death (see Chapter 21), systemic therapy using fluconazole combined with amphotericin B or miconazole has been reported.[40–43] If central nervous system involvement is suspected, fluconazole is preferred due to its ability to cross the blood-brain barrier into cerebrospinal fluid.[43] For intravenous therapy, the installation of a peripheral venous catheter in the basilic or the tibiotarsal vein is recommended, as this allows parallel administration of fluids (intravenous drip infusion) and prevents phlebitis resulting from repeated venepuncture. Careful fixation and padding of the peripheral catheter using surgical suture material, appropriate adhesive tape, and bandage material are required in order to prevent inadvertent catheter removal by the patient.



FIGURE 8.3 A hooded gyrfalcon *(Falco rusticolus)* is placed in a nebulization chamber during inhalation therapy. (Courtesy Dr. Dominik Fischer.)



FIGURE 8.4 Medical training facilitates stress-free oral and parenteral antifungal drug administration in trained birds. (*Left:* Courtesy Dr. Elisa Wüst. *Right:* Courtesy Clinic for Birds, Reptiles, Amphibians and Fish, JLU Giessen.)

Topical Treatment

Topical administration of antimycotic drugs directly to the fungal lesion is suggested in addition to nebulization for the treatment of fungal lesions inside the trachea or the syrinx. For this purpose, injectable solutions such as amphotericin B or voriconazole can be administered intratracheally (IT), using a (plastic) catheter that is inserted via the glottis into the trachea in an orthograde manner. Alternatively, if the glottis is not easily accessible (e.g., in small-sized birds), small-sized needles may be used for transtracheal drug delivery after thorough cleaning and disinfection of the injection site. As birds with tracheal aspergillosis usually suffer from inspiratory dyspnea caused by restricted airflow, the installation of an air sac cannula is an important step for emergency treatment of patients (see Chapter 29 and 34), while enabling respiration to be maintained despite of tracheal administration of liquid medicine, or while performing tracheoscopy or tracheal surgery. In some cases of tracheal aspergillosis, tracheal plugs can be removed in toto or in piecemeal fashion using biopsy forceps, cotton swabs, or suction catheters under visual guidance of an endoscope (see Chapter 30). Alternatively, if tracheal lesions are extensive, surgical tracheotomy and debridement may be required (see Chapter 29).

Topical treatment and surgery may be required for the treatment of air sac aspergillomas. Similar to the inratracheal route, injectable solutions may be instilled dropwise on top of lesions using plastic tubes or flexible injection needle instrumentation that are inserted via the incision (during surgical procedures) or working channel (during endoscopic procedures). Prior to instillation of the antifungal medication, initial (endo)surgical debridement of the fungal lesions is recommended. There are several endo surgical procedures described to access the air sacs using a single-, a double-, or a triple-entry technique (see Chapters 29 and 30).[44] Aside from using a biopsy or grasping forceps and/or endoscopic scissors to debulk the lesions, diode lasers have also been used successfully for endoscopic removal of aspergillomas. However, caution is warranted when using this equipment inside the well-ventilated air sacs due to the risk of burning. If thick, caseous encapsulated air sac lesions are too extensive to achieve endoscopic removal, classical surgical intervention via lateral laparotomy approach may be performed (see Chapter 29). Stabilization of the patient and initiation of medical treatment are commonly required, prior to initiating (endo)surgical intervention.

Topical antifungal treatment may be applied to the oropharyngeal mucous membranes in case of candidiasis, to the outer skin and feather follicles in case of fungal dermatitis and/or folliculitis, or to eyes or nose in case of ocular fungal infection, rhinitis, or sinusitis. Samour and Naldo applied miconazole gel to the mucous membranes of falcons to treat clinical candidiasis in the oral cavity and crop.[45] Fungal dermatitis, especially in the axillary and inguinal regions, and associated feather destructive behavior may be caused by more than 20 fungal species including *Aspergillus* spp., *Candida* spp., *Trichophyton* spp. (e.g., *T. gallinae*, *T. rubrum*, *T. simii*, and *T. megnini*), *Malassezia* spp., *Mucor* sp., *Microsporum gallinae*, *Microsporum gypseum*, *Cladosporium* spp., *Helminthosporium* spp., *Paecilomyces* spp., and *Rhodotorula mucilaginosa.* Targeted therapy of these infections includes topical antifungal treatment using nystatin, terbinafine, or azole creams (e.g., clotrimazole or bifonazole) in addition to the application of enilconazole washing solution or chlorhexidine-miconazole shampoo as established for dogs and cats. However, bathing of the entire body in a washing solution might be less advisable because it is usually stressful and may alter feather condition in birds. Thus gentle dapping the affected skin with soaked gauze swabs or lightly spraying the lesions with a spray flask are generally preferred for topical delivery of the drug. Topical spray disinfection using chlorhexidine, iodine, or F10 solution may also be helpful in the treatment of fungal skin lesions. The use of creams and ointments for topical treatment in birds is controversial. While creams can cover the skin lesions and ensure targeted delivery of the antifungal agent they also stick to the feathers, thereby altering their structure and function, and possibly result in feather destructive behavior (see Chapter 32). Especially oily and lipid-based products can mat the plumage and lead to excessive preening; hence, these should be avoided in birds. If using creams, these should be restricted to featherless areas or areas where feathers have been pre-emptively removed. In severe cases of dermatitis, topical treatment should be combined with systemic antifungal therapy (e.g., administration of azoles such as fluconazole) to increase antifungal action. Moreover,

surgical debridement of infected skin, including removal of crusts, excoriated skin, and surgical excision of damaged tissues may be required. The same applies for bumblefoot lesions from which fungal organisms such as *Aspergillus* spp. or *Candida* spp. may be isolated in rare cases, hence requiring topical and systemic antifungal therapy (see Chapter 33). Soft laser therapy and alternative treatment options from human medicine such as cold plasma therapy using atmospheric pressure plasma jet devices (Figure 8.5) or ozone (trioxygen) therapy (Figure 8.6) seem promising for the topical treatment of chronic wounds and fungal infections of the skin and accessible mucous membranes. These therapeutic options might offer an effective fungistatic to fungicide supplement to standard wound management in avian patients in the future, especially facing increasing antifungal drug resistance in recent years.



FIGURE 8.5 Plasma therapy using atmospheric pressure plasma jet devices (kINPen VET, neoplas GmbH, Greifswald, Germany) for the treatment of fungal dermatitis in a peregrine falcon *(Falco rusticolus)*. (Courtesy Dr. Dominik Fischer.)



FIGURE 8.6 Use of ozone (trioxygen) via flat probe (OzoneDTA, Apoza Enterprise Co. Ltd, New Taipei City, Taiwan) for the treatment of a fungal bumblefoot lesion in a peregrine falcon *(Falco rusticolus)*. (Courtesy Dr. Dominik Fischer, Clinic for Birds, Reptiles, Amphibians and Fish, JLU Giessen.)

Ocular fungal infections including diseases of conjunctiva, cornea, and/or anterior eye chamber can be treated using antifungal eye drops. Since these are usually not commercially available, a pharmacist may prepare them on request. Similar formulations may be used for the treatment of fungal rhinitis and sinusitis in addition to inhalation therapy and systemic antifungal medication. However, nasal flushes using amphotericin B (intravenous formulation), enilconazole, or F10 SC solutions (Health and Hygiene, Florida Hills, South Africa) have been reported as beneficial addition to the treatment of fungal rhinitis and sinusitis.[27] In chronic or severe cases, surgical opening and thorough cleaning of the infraorbital sinus is required (see Chapter 29).[22] As some fungi such as *Aspergillus* spp. and *Exophiala (Cladosporium) werneckii* are known to be able to spread from the eye to the brain, ocular mycosis may carry a guarded prognosis.[46]

Supplemental Therapy

In addition to specific antimycotic therapy, elimination of predisposing factors (e.g., stress, suboptimal hygiene, husbandry deficiencies) and supportive treatment are required for effective treatment and prevention of relapses of fungal infections. Supportive treatment consists of supplementation of vitamins (especially vitamin A), support of liver function (e.g., by administration of silymarin/milk thistle), and fluid therapy. In inappetent patients, tube feeding and the addition of antiemetic drugs or antacids may be advised. Oral administration of thyme tea or sucralfate (25 mg/kg PO q8h) have been reported as supportive therapy in budgerigars suffering from macrorhabdosis. In case of respiratory fungal disease, oxygen supplementation will generally benefit the patient. Likewise, mucolytic drugs or bronchodilators might be beneficial additions to the treatment of respiratory fungal infections (see Chapter 28). As in most cases of fungal disease a bacterial coinfection cannot be ruled out, antibiotic therapy might be initiated parallel to initiating antifungal treatment. To manage disruption to the intestinal flora following antibiotic use, administration of probiotics may be considered. However, it should be taken into consideration that any of the aforementioned treatments, especially long-term antibiotic treatment, might lead to stress and immunosuppression (as do steroidal antiphlogistics), hence any additional treatment should be carefully assessed for its true need.[2]

Conclusion

To treat avian aspergillosis, the combined use of antifungal inhalation therapy (e.g., with enilconazole) with oral and/or parenteral medication (e.g., voriconazole) and supportive therapy, as needed, is advised.Therapeutic success needs to be monitored regularly, based on which treatment may be adjusted accordingly. Follow-up examinations should at least include a full clinical examinations, combined with imaging (especially endoscopy), hematology, blood chemistry, and measurement of oxygen saturation to allow for monitoring of clinical improvement in the patient's condition, anti-infective progress, and adequacy of respiratory function. In some avian species, specific tests such as the measurement of fungal antigens or specific antibodies are available besides plasma/serum electrophoresis and acute-phase protein determination.

(see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease, and Chapter 13, Advances in Clinical Pathology and Diagnostic Medicine). For emergency treatment, oxygen supplementation and air sac cannulation should be considered (see Chapters 28 and 29). In severe cases, surgical intervention may be necessary (see Chapter 29).[47,48]

Treatment of macrorhabdosis and candidiasis requires oral administration of amphotericin B and/or nystatin, administered over a period of 4 to 6 weeks twice daily (Table 8.1 ; see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). If antifungal resistance to amphotericin B is suspected, a therapy using voriconazole orally might be attempted. *Candida* spp. usually respond adequately to treatment using nystatin or nystatin-fluconazole mixtures; hence targeted therapy of candidiasis is mostly accomplished by orally administering these agents over a period of 7 to 10 days.

For the treatment of systemic fungal infections, parenteral formulations are available (Table 8.1).

Fungal infections of the skin, mucous membranes, the eye, the nose, and the sinus may be treated using topical antifungal medication (Table 8.1). In any case, fungal culture and sensitivity testing are advisable for diagnosis and targeted antifungal therapy.

TABLE 8.1

Overview of Antifungal Treatment Schedules[49,50]

TOPICAL OCULAR ANTIFUNGAL TREATMENT		
Amphotericin B *(polyene macrolide)*	0.025 mg/mL sterile water subconjunctival: q24h[51] 4% ointment: q24h[51]	Treatment of candidiasis of the third eyelid in ornamental ducks; often combined with systemic antifungal treatment
Fumagillin	1 drop (0.114 mg/mL in sterile water) q2–3h until 1 w post clinical signs[52]	Treatment of fungal and microsporidial keratoconjunctivitis in Amazon parrots (*Amazona* spp.); used in combination with oral albendazole; solution should be filtered prior to application
Miconazole *(azole derivative)*	2% cream: q24h × 7 d[52] IV solution: 1 drop q2h[52]	Most species; treatment of candidiasis of the third eyelid
Pimaricin/natamycin *(polyene macrolide)*	1 drop q2–8h × 14–21 d[53]	Gradually taper off after treatment
ANTIFUNGAL NEBULIZATION		
Amphotericin B *(polyene macrolide)*	0.1–1 mg/mL sterile water over 15–60 min, q6–24h[27,53–56] 10 mg/20 mL sterile water over 30 min, q12h[27]	Most species; minor systemic absorption with aerosol administration; may lead to hypokalemia, especially when given with corticosteroids; mix with sterile water due to risk of precipitation with electrolytic solutions and saline; low efficacy and possible weight loss in hummingbirds at 0.25 mg/mL × 15 min q12h; Falcons
Amphotericin B liposome *(polyene macrolide)*	1–4 mg/mL sterile water over 15–60 min, q12–24h; 7–10 mg/mL sterile water over 15 min, q12h[49]	Most species; mix with sterile water due to risk of precipitation with electrolyte and saline solutions
Clotrimazole *(azole derivative)*	10 mg/mL solution, e.g., with propylene glycol or polyethylene glycol over 30–60 min, q24h[49,53,54]	Most species; potentially toxic to psittacines at this dose; use in combination with systemic antifungals; in raptors, sometimes given over 3 d, followed by 2 d off and repeated for up to 4 months q8–12h[27]
Enilconazole *(azole derivative)*	0.2 mg/5 mL saline q12h × 21 d[53] 10 mg/mL saline or sterile water[53]	Most species; birds of prey are sometimes treated using a different regimen, i.e., 50 mg/25 mL saline × 30–45 min q8–12h[27] or 0.1 mL/kg in 5 mL sterile water × 30 min q24h 5 d on/2 d off[49]; in the authors' experience 10 mg/5 mL saline × 30 min q12h × 28 d is well accepted by most species
F10 *(blend of quaternary ammonium and biguanide compound)*	1:250 dilution over 30–45 min, q12h[27,57]	Dose given to raptors; in the authors' experience this dose is well tolerated in psittacines, also when combined with 10 mg/5 mL enilconazole[27,57]
Itraconazole *(azole derivative)*	1%–10% nanoparticulate suspension over 30 min, q24h[58,59]	Evaluated in Japanese quail *(Coturnix japonica)* in experimental aspergillosis, with 10% being most effective
Miconazole *(azole derivative)*	15 min q8h × 10 d[53]	Dose given to raptors to treat aspergillosis
Terbinafine *(allylamine)*	1 mg/mL in sterile water over 15 min; q12h[60]	Used in most species to treat aspergillosis; dose evaluated for Hispaniolan Amazon parrots *(Amazona ventralis)*; higher doses may be required to maintain therapeutic levels;[60] dosages used in raptors are similar but longer (30 min) and more frequent (q8–12h) intervals are advised; may also be used in combination with N-acetyl-L-cysteine[27]
Voriconazole *(azole derivative)*	10 mg/mL saline solution over 15–30 min, q8–12h[11,54]	Most species; low plasma concentration and low pulmonary drug concentration in pigeons *(Columba livia domestica)*

Continued

TABLE 8.1

Overview of Antifungal Treatment Schedules—cont'd

LOCAL THERAPY—TRACHEA, LUNG, AIR SAC, SINUSES OR NASAL CAVITY		
Amphotericin B *(polyene macrolide)*	1 mg/kg IT q8–12h × 3–12 d[49,61–63] 1 mg/kg IAS/IP inside fungal lesions[49,61–63] 0.05–1 mg/mL sterile water for nasal flush[49,61–63]	Most species including psittacines and raptors; dilute in sterile water or 5% glucose solution (e.g., 0.3–0.5 mL/kg BW) prior to administration; may cause tracheitis after IT administration; after initial IT therapy over 12 d may be continued q48h over 5 w
Clotrimazole *(azole derivative)*	nasal flush 10 mg/mL[49,64]	Effective against yeast and *Aspergillus*; used in most species for nasal flush;
	2 mg/kg IT q24h × 5 d[49,60]	In psittacines; applied to syringeal aspergilloma using catheter or endoscope working channel
Enilconazole *(azole derivative)*	1 mg/kg IT q24h of a 1:10 dilution with sterile water[49]	Treatment of aspergillosis in falcons (*Falco* spp.)
Miconazole *(azole derivative)*	5 mg/kg IT of a 10 mg/mL solution diluted with saline q12h × 5 d[49]	Treatment of aspergillosis in psittacines
Voriconazole *(azole derivative)*	12.5 mg/kg q24h[65]	Treatment of aspergillosis in raptors
SYSTEMIC ANTIFUNGAL TREATMENT—ORAL ADMINISTRATION		
Fluconazole *(azole derivative)*	10–20 mg/kg PO q24h × 30 d	Used in psittacines and pigeons *(Columba livia domestica)* to treat aspergillosis; doses ≥ 10 mg/kg might be toxic to budgerigars *(Melopsittacus undulatus)*;
	15 mg/kg PO q12h × 30 d[49]	Used in raptors to treat systemic mycoses—doses to treat candidiasis and GI fungal infections are much lower (see below)
Griseofulvin	10 mg/kg PO q12h × 21 d[49]	Used in pigeons *(Columba livia domestica)* to treat dermatophytosis;
	30–50 mg/kg q24h in drinking water[49]	Used in ostriches *(Struthio camelus)* to treat mycotic dermatitis
Itraconazole *(azole derivative)*	5–10 mg/kg q12–24h × 10–14 d[49] higher doses reported in: raptors: 15 mg/kg PO q12h × 4–6 w[66,67] penguins (Sphenisciformes): 20 mg/kg PO q24h[49,69]	Most species; first-choice drug for the treatment of aspergillosis, also used to treat candidiasis and dermatophytosis; treatment may be prolonged to 60 d in finches[67,68] and to 90 d in psittacines;[31,64] toxicity reported in grey parrots *(Psittacus erithacus)* at doses ≥ 5 mg/kg; anecdotal reports of drowsiness/apathy in African fish eagles *(Icthyophaga vocifer)*[49]
Ketoconazole *(azole derivative)*	15–30 mg/kg PO q8–24h × 7–14 d[49,64,67,70,71] 30 mg/kg q12h × 7–30 d[49,71]	Dose recommendations variable in avian species to treat aspergillosis and candidiasis; Dose used in most species; lower doses used in ratites: 5–10 mg/kg PO q24h; higher doses given to pigeons *(Columba livia domestica)*: 20–40 mg/kg PO q12h × 15–60 d; higher doses also used in raptors and toucans: 60 mg/kg PO q12h;
	200 mg/L drinking water[49,56,72]	Dose administered to canaries *(Serinus canaria)*, Gouldian finches *(Chloebia gouldiae)*, and hummingbirds (*Trochilus* spp.) to treat candidiasis
Terbinafine *(allylamine)*	10–30 mg/kg PO q12–24h[14,49,73,74] 15 mg/kg PO q24h (penguins)[73] 22 mg/kg PO q24h (raptors)[14]	Doses provided to most species to treat yeast and mold infection; limited action against *Aspergillus* spp. (questionable therapeutic potential), may be combined with itraconazole to increase effectivity; Higher dosages (60 m/kg PO q24h) given to Amazon parrots (*Amazona* spp.)[75] and common shelducks *(Tadorna tadorna)*;[76] Authors frequently have success using dosages of 15–20 mg/kg PO q12h × 30 d
Posaconazole *(azole derivative)*	12.5–20 mg/kg PO (falcons)[77]	Should be administered together with food or dissolved in corn oil

TABLE 8.1

Overview of Antifungal Treatment Schedules—cont'd

Voriconazole *(azole derivative)*	10–18 mg/kg PO q12h[11,19,21,49,65,78,79] or 20 mg/kg PO q24h × 21 d[80]	Used in most avian species to treat aspergillosis; efficacy against most yeasts; prolonged elimination half-life has been observed in pigeons *(Columba livia domestica)* and African penguins (*Spheniscus demersus*; Note: increased anecdotal reporting of toxicity in penguins); pigeons treated with low dose of 10 mg/kg PO q12h or 20 mg/kg PO q24h;[11,19,21] 20 mg/kg PO q8–12h has also been used in ducks;[81] doses in raptors vary from 10 mg/kg PO q8h (red-tailed hawks; *Buteo jamaicencis*) to 12.5 mg/kg PO q12h (falcons; *Falco* spp.);[65,82,83] Dose in parrots is 12–18 mg/kg PO q12h (Grey parrot; *Psittacus erithacus*)[78] or 18 mg/kg PO q8h (Amazon parrots; *Amazona* spp.)[79]
SYSTEMIC ANTIFUNGAL TREATMENT—PARENTERAL TREATMENT		
Amphotericin B *(polyene macrolide)*	1.5 mg/kg IV q8h × 3–7 d[49,53,61]	Most species
Miconazole *(azole derivative)*	10–20 mg/kg IM or IV q8–24h × 6–12 d[49,53,70]	Raptors and psittacines to treat generalized aspergillosis;
	20 mg/kg IV q8h[70]	Psittacines with candidiasis/cryptococcosis
Voriconazole *(azole derivative)*	12.5 mg/kg IM q24h[77] 20 mg/kg IV q8–12h[81]	Raptors to treat aspergillosis; Ducks
GASTROINTESTINAL ANTIFUNGAL TREATMENT—TO TREAT GASTROINTESTINAL FUNGAL DISEASES		
Amphotericin B *(polyene macrolide)*	100–109 mg/kg PO q12h × 10–30 d[49,84] 1,000 mg/L drinking water × 10 d[49,85] 0.1 mg/mL drinking water × 28 d (usage of a formulation to prepare infusion solution)[86]	Oral suspension with limited/no absorption from the GI tract is the first choice for the treatment of *Macrorhabdus*; Doses given for treatment of avian gastric yeast in budgerigars *(Melopsittacus undulatus)*; treatment of candidiasis in neonatal raptors; Authors' experience: 0.1 mL oral suspension (100 mg/mL) q12h × 28 d or 1 mL oral suspension (100 mg/mL) in 50 mL drinking water (requires frequent remixing/stirring to prevent demixing) to treat *Macrorhabdus/Candida* infections; for flock treatment marinate food with 1 mL/kg (use total body mass of all individuals) of oral suspension (100 mg/mL), mixed 1:4–1:10 with water
Fluconazole *(azole derivative)*	2–6 mg/kg PO q24h × 7 d; 5–15 mg/kg PO q12h × 14–60 d[87–89]	Treatment of candidiasis in psittacines, finches, and raptors; systemic activity; may also penetrate into CNS and eye; Death observed at 10 mg/kg q12h in budgerigars *(Melopsittacus undulatus)*;
	8 mg/kg PO q24h × 30 d[90]	Treatment of cryptococcosis in psittacines;
	50 mg/L drinking water × 14–60 d[49]	Most species; in cockatiels *(Nymphicus hollandicus)*: 100 mg/L drinking water × 8 d; in Gouldian finches *(Chloebia gouldiae)* up to 100 mg/L drinking water or 100 mg/kg soft food; Authors' experience: 50 mg mixed with 20 mL nystatin oral solution (100,000 IU/mL) and use 1 mL/kg PO q12h for the treatment of *candidiasis*
Nystatin *(polyene macrolide)*	300,000–600,000 IU/kg PO q8–12h × 7–14 d[49,67]	First choice for the treatment of GI candidiasis; may be effective against other yeast or fungi but is not systemically absorbed from GI tract; should not be given if GI epithelium is not intact; Dose given to most species; author's experience: 200,000–300,000 IU/kg PO q12h × 28 d
	20,000–100,000 IU/kg PO q12h × 7–14 d[67]	Dose given to raptors and pigeons *(Columba livia domestica)* to treat candidiasis;
	100,000 IU/L drinking water[72,89]	Dose given to canaries *(Serinus canaria)* and finches to treat candidiasis
TOPICAL TREATMENT		
Amphotericin B *(polyene macrolide)*	10% solution[67] 3% cream q12h[63]	Direct application in the oropharynx; Topical application to infected skin
Enilconazole *(azole derivative)*	1:10 dilution q12–72h × 21 d[62]	Topical treatment of cutaneous aspergillosis/candidiasis

Continued

TABLE 8.1

Overview of Antifungal Treatment Schedules—cont'd

F10 *(blend of quaternary ammonium and biguanide compound)*	q8h	Treatment of cutaneous fungal infections
Iodine 1%	q8–12h[61]	Treatment of oral or cutaneous candidiasis
Miconazole *(azole derivative)*	q12h	Treatment of cutaneous fungal infections such as cutaneous aspergillosis/dermatophytosis
Nystatin *(polyene macrolide)*	q6h[91]	Topical administration onto lesions in hummingbirds (*Trochilus* spp.)/candidiasis, e.g., by using a cotton swab for direct application
Silver sulfadiazine	q12–24h[63,92]	Topical administration to affected areas; bandage application is preferred
STA solution (salicylic acid 3 g, tannic acid 3 g, ethyl alcohol to 100 mL)	q24h[70]	Topical administration in case of fungal dermatitis
Terbinafine *(allylamine)*	q12h	Treatment of cutaneous fungal infection
SPECIAL CASE: TREATMENT OF MICROSPORIDIA/*ENCEPHALITOZOON HELLEM*		
Albendazole	25 mg/kg PO q24h × 30–90 d, then repeat × 120 d in case of relapse[29,93]	Dose recommended for the treatment of keratoconjunctivitis in cockatoos (Cacatuidae); may be combined with fumagillin;
	50 mg/kg PO q24h × 5 d[29,94]	dose given to treat microsporidial keratoconjunctivitis in Amazon parrots (*Amazona* spp.)
NEW APPROACHES FOR THE TREATMENT OF MUCOSAL OR DERMAL FUNGAL INFECTIONS		
Cold plasma	HF Plasmajet 5 ± 1 slm argon gas N48 (99.998%), temperature 38°–40°C, moved with a speed of 5 mm/sec resulting in a contact time of 20–30 sec/cm² × q48h × 14–30 d	
Ozone therapy	Sessions of 40–60 sec q12–24h × 3–6 d per spot, power setting 8 of 12	
Soft laser	980 nm in the continuous-wave mode (CW mode) in noncontact mode with a power of 2–2.5 W using a defocusing handpiece	

BW, Body weight; *°C*, degree Celsius; *d*, days; *GI*, gastrointestinal; *h*, hours; *IAS*, inside the air sac; *IM*, intramuscular; *IP*, intrapulmonary; *IT*, intratracheal; *IV*, intravenous; *min*, minutes; *PO*, oral; *q*, interval; *SC*, subcutaneous; *sec*, seconds; *slm*, standard liter per minute; *w*, weeks; *W*, watts.

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CHAPTER 9

Antiparasitic Drugs

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INTRODUCTION

To control parasitic infection, avian veterinarians have several classes of antiparasitic drugs in their arsenal, each with a different mode of action. In order to prescribe these drugs effectively, it is necessary to understand not only the parasiticide in question but also the life cycle of the parasite of interest. Although the focus of this text is exotic and companion birds, it is impossible to properly and completely discuss antiparasitic drugs in birds without exploring their impact on poultry production, and therefore this taxonomic group will also be mentioned frequently.

ANTHELMINTIC AGENTS

Macrocyclic Lactones

Macrocyclic lactones (MLs) are broad-spectrum drugs, referred to as endectocides because of their activity against both endo- and ectoparasites.[1,2] MLs are used not only in animals but also for pest control of crops because of their high activity against arthropod and nematode pests, low toxicity to birds and mammals, and their unique mode of action.[1] MLs are gamma-aminobutyric acid (GABA) agonists that also bind to glutamate-gated chloride channels in the nerve and muscle cells of invertebrates.[3,4] MLs include two types of compounds: avermectins and milbemycins.[3,4] Most members of the ML family belong to the avermectin subfamily, which includes abamectin, ivermectin (IVM), doramectin, eprinomectin, and selamectin.[3–5] Examples of milbemycins include moxidectin and milbemycin oxime, which have a similar mechanism of action to avermectins but a longer half-life (Table 9.1).[1,3,4]

Ivermectin

IVM is the most commonly used avermectin.[1,3] Although IVM is not licensed for use in any avian species, oral, topical, and injectable formulations licensed for cattle or sheep are widely used off-label to control parasites in various bird species.[4] Multiple studies have indicated that IVM is highly effective against nematode infections in poultry.[5] There have also been pharmacokinetic studies in gallinaceous birds (Table 9.2).[5,36] Regardless of the route of administration, IVM is widely distributed to tissues[5]; however, the high lipophilicity of IVM means that much more of this agent accumulates in egg yolk and fat compared to other tissues.[5,52] Therefore IVM should not be used in laying hens (Table 9.1).

Benzimidazoles

Benzimidazole (BZD) anthelmintics are a large class of broad-spectrum anthelmintics that includes albendazole (ABZ), mebendazole (MBZ), fenbendazole (FBZ), flubendazole (FLBZ), and thiabendazole (TBZ) (Table 9.1).[6] BZDs act by directly destabilizing tubulin, a system of proteins present in plasma and mitochondrial membranes and microtubules. Microtubules impact the cell's ability to undergo mitosis or regulate intracellular transport.[6,53] Helminths die as energy reserves are progressively depleted and excretion of cellular waste is inhibited.[53]

Much is known about the pharmacokinetic parameters of BZDs in the chicken.[52] Following oral administration to chickens, FLBZ and ABZ are absorbed relatively quickly, reaching peak plasma concentrations in 2 to 4 hours.[52] Despite a rapid rate of elimination, detectable residues of FLBZ and ABZ can be found in eggs laid up to 1 week or more after treatment.[52] BZD anthelmintics require extensive hepatic metabolism for excretion.[54] A study investigating FLBZ metabolism found that there were very significant differences in the pattern of metabolism observed among tested species.[54] Therefore caution should be applied in extrapolating data on metabolism, efficacy, and safety of drugs from one species to another.[54]

Benzimidazole toxicosis

Confirmed or suspected BZD toxicosis has been reported in pigeons and doves, two vulture species, two stork species, the American white pelican *(Pelecanus erythrorhynchos)*, and a harpy eagle *(Harpia harpyja)*.[9–15,55] Since BZDs affect cellular division, this can lead to extreme suppression of rapidly dividing cells in the host, such as intestinal crypt cells and bone marrow precursors.[10,14] Bone marrow dysplasia can rapidly result in anemia, marked leukopenia or heteropenia, septicemia, and secondary infection.[9,10,14,15] Affected birds may display nonspecific signs of illness and even sudden death within 72 hours of drug administration.[9–12,14] The overall mortality rate is typically high in affected birds, but some individuals can recover with aggressive supportive care.[10] Feather dystrophy has also been observed in surviving birds that were treated during active molt.[15,25]

TABLE 9.1

Anthelmintic Agents for Use in Birds

	Benzimidazoles	Macrocyclic Lactones
Examples	Albendazole (ABZ) Cambendazole (CBZ) Fenbendazole (FBZ) Flubendazole (FLBZ) Mebendazole (MBZ) Oxfendazole (OFZ) Thiabendazole (TBZ)	Doramectin (DOR) Eprinomectin (EPR) Ivermectin (IVM) Milbemycin oxime (MO) Moxidectin (MOX) Selamectin (SEL)
Clinical applications	Helminths, particularly nematodes like *Serratospiculum* Some BZDs, like FBZ, ABZ, and MBZ, are also active against *Giardia*[6]	Broad-spectrum, widely used to control endo- and ectoparasites IVM: Dilute with water or saline for immediate use; dilute with propylene glycol for long-term use[7,8]
Potential adverse effects or toxicity	Toxicosis or suspected toxicosis has been reported in a variety of avian species (see Benzimidazole Toxicosis) but is most often reported in pigeons and doves[8–14] ABZ may be toxic in keas *(Nestor notabilis)*[8]	No adverse effects have been reported in the chicken[5]; however, there are sporadic, anecdotal reports of IVM toxicosis in exotic birds, including finches[15] Risk of IVM toxicity may be higher if the drug is injected, particularly via the IM route[16] IVM (3 mg/kg IM) in falcons (*Falco* spp.) led to blindness, anorexia, vomiting, and ataxia that lasted up to 48 hours[17]
Contraindications, precautions	Avoid BZD use during breeding season as embryotoxic effects have been observed[16] Avoid use in birds molting primary feathers because it will cause weakness and breakage of growing remiges[16,25] Shake suspension products thoroughly before each use to minimize the risk of toxic effects	Avoid IVM in laying hens due to high residues found in eggs[5] Budgerigars *(Melopsittacus undulatus)*, African finches, and European finches may be more sensitive[16]
Dosage in most species	FBZ 10–50 mg/kg PO[8] FBZ 25 mg/kg PO, repeat in 14d[7] OFZ 10–40 mg/kg PO once[7] TBZ 40–100 mg/kg PO q24h × 7d[8,16]	IVM 0.2–0.4 mg/kg PO, topically, SC, IM (see Potential adverse effects or toxicity, earlier); can repeat in 10–14d[8,16,25,26]
Anseriformes	FBZ 5–15 mg/kg PO q24h × 5d[21] MBZ 5–15 mg/kg PO q24h × 2d[30] MBZ 1.2 mg/kg feed × 14d[30]	—
Columbiformes	CBZ 75 mg/kg PO q24h × 2d[7] FBZ 10–12 mg/kg PO q24h × 3d[25] FBZ 20–50 mg/kg PO once, repeat in 10d (ascarids)[7]	—
Galliformes	FBZ 50 mg/kg PO q24h × 3d[20] FBZ 100 ppm feed × 7d—peafowl, Chinese ring-necked pheasants *(Phasianus colchicus)*[31] FBZ in the water 5 mg/kg BW for 1d[32] FLBZ 1.43 mg/kg administered in drinking water × 7d[33] FLBZ 60 mg/kg feed × 7d (partridges, pheasants)[30]	MO 2 mg/kg PO, repeat in 28d[34] SEL 20 mg/kg topical (helmeted guineafowl *[Numida meleagris]*, PK)[35]
Passeriformes	CBZ 60–100 mg/kg PO q24h × 3–7d[16] FBZ 8–10 mg/kg PO q24h × 3–4d[16] FBZ 50 mg/kg PO q24h × 3–5d[38] FBZ 25 mg/L drinking water[39] FBZ 25 mg/kg soft food[39] MBZ 10 mg/kg PO q12h × 5d (canaries [*Serinus canaria*])[7] MBZ 10–25 mg/kg PO q12h × 5d)[16] MBZ 10–20 mg/L drinking water × 3–5d[16] OFZ 10–40 mg/kg PO once[16] TBZ 100–500 mg/kg once[16]	IVM 9 mg/L drinking water[39]
Piciformes	ABZ 15–20 mg/kg PO once (toucans)[7] MBZ 25 mg/kg PO q12h × 5d (toucans)[7,41]	MOX 0.2 mg/kg IM once[41]

Pyrazino-Isoquinoline	Imidathiazole	Tetrahydro-Pyrimidine
Praziquantel	Levamisole	Pyrantel embonate Pyrantel pamoate Pyrantel tartrate
Trematodes, cestodes	Adult nematodes of the lungs and intestinal tract (i.e., *Capillaria*)	Broad-spectrum activity against GI nematodes, most commonly used for ascarids
Injectable form toxic in finches and associated with depression, death in some other species[7,18] Death occurred in Gouldian finches *(Erythrura gouldiae)* at 45 mg/kg[19] Collapse, loss of balance observed in chickens *(Gallus gallus domesticus)* at 100 mg/kg Signs resolved within 3 hours[20]	Relatively narrow margin of safety. Adverse effects can include regurgitation, dyspnea, anorexia, diarrhea, ataxia, head tilt, torticollis, limb paralysis, and death[7,16,21,22] Respiratory distress, death in a North Island brown kiwi *(Apteryx mantelli)* within hours of one dose (25–43 mg/kg PO)[23] IM administration may cause severe toxicity[7]	Reported effects in humans in case of overdose include GI disturbance, CNS effects, and superficial skin reactions[24]
Caution with IM administration in finches; sudden death has been reported[16] Very unpalatable[16]	Not recommended in finches, lories, white-faced ibis *(Plegadis chihi)*, debilitated birds[7,16] Withhold food before administration to prevent regurgitation[7]	Does not penetrate the intestinal wall and therefore does not kill encysted strongyles[6] Not recommended for use in debilitated animals due to potential for pronounced cholinergic effects
10–20 mg/kg PO, repeat in 10–14d prn[7,18,27,28]	10–20 mg/kg SC once[18,29] 20–40 mg/kg PO once[16] 100–200 mg/L drinking water × 3d, repeat in 2 weeks[16] 264–396 mg/L drinking water × 1–3d[7]	7 mg/kg PO, repeat in 14d[7]
10 mg/kg PO, SC, IM q24h × 14d (trematodes)[21] 10–20 mg/kg SC, IM; repeat in 10d (cestodes)[21]	20–50 mg/kg PO once[21] 25–50 mg/kg SC once[18]	7 mg/kg PO, repeat in 14d[21]
10–20 mg/kg PO; repeat in 10–14d[25]	7.5 mg/kg IM once; can repeat in 7d[7] 20 mg/kg PO once[7] 300–400 mg/L drinking water × 24 hours, repeat in 7d (pigeons, *Capillaria*, ascarids)[7,18]	20–25 mg/kg PO[25]
8.5 mg/kg IM (chickens)[36] 10 mg/kg PO once (effective against *Raillietina tetragona*)[28] 11 mg/kg SC once (chickens)[36]	8 mg/kg BW in the water × 2d[32] 265–525 mg/L drinking water × 24 hours, repeat in 7–14d (game birds, poultry)[30]	—
Caution with IM administration in finches; sudden death has been reported[16] 5–10 mg/kg PO, repeat after 2–4 weeks[7] 10 mg/kg PO once a week for 4 weeks[40] 10–20 mg/kg PO, IM once[7] 20 mg/kg IM once weekly for 4 weeks[40] 25 mg/kg PO, IM; repeat in 10–14d[38] Nebulization of PZQ (56.8–85.2 mg in 100% oxygen) for 5, 10, and 15 minutes q24h × 3d (reduced air sac trematode egg counts)[40]		7–25 mg/kg PO once[16]
10 mg/kg IM q24h × 3d, then PO q24h × 11d (toucans, trematodes)[7,42] 10 mg/kg PO, SC, IM q24h × 14d; follow with 6 mg/kg PO q24h × 14d (toucans, trematodes)[36,41]	—	—

Continued

TABLE 9.1

Anthelmintic Agents for Use in Birds—cont'd

	Benzimidazoles	Macrocyclic Lactones
Psittaciformes	ABZ 10–50 mg/kg once[16] ABZ 25 mg/kg PO q24h × 90d, then repeat × 120d when signs returned (cockatoos [*Cacatua* spp.], *Encephalitozoon hellem* keratoconjunctivitis)[43] ABZ 50 mg/kg PO q24h × 5d (Amazon parrots [*Amazona* spp.], microsporidian keratoconjunctivitis)[44] CBZ 60–100 mg/kg PO q24h × 3–7d[16] FBZ 8–10 mg/kg PO q24h × 3–4d[16] FBZ 20–50 mg/kg PO once, repeat in 10d (ascarids) × 3d (trematodes, microfilaria) × 5d *(Capillaria)*[7] MBZ 10–25 mg/kg PO q12h × 5d[16] MBZ 10–20 mg/L drinking water × 3–5d[16] OFZ 10–40 mg/kg PO once[16] TBZ 100–500 mg/kg once[16]	SEL 23 mg/kg topically, repeat in 3–4 weeks (budgerigars [*Melopsittacus undulatus*], *Knemidokoptes*)[45]
Raptors	ABZ can be toxic at doses >25 mg/kg in birds of prey[16] FBZ 20 mg/kg PO q24h × 14d *(Serratospiculum)*[17] FBZ 30 mg/kg PO q24h × 5–8d (*Serratospiculum* spp. in falcons)[46] MBZ 20 mg/kg PO q24h x 10–14d[17,29,47] TBZ 100–200 mg/kg PO q12h × 10d[47]	IVM 0.2–1 mg/kg PO, SC, IM every 14d × 2–3 treatments[29] IVM 1 mg/kg SC once (effective for controlling *Capillaria*)[17] IVM 1 mg/kg SC, repeat in 7–14d (falcons, *Serratospiculum*)[17] IVM 2–3 mg/kg SC (falcons, *Serratospiculum*)[48,49] DOR 1 mg/kg SC, IM, repeat in 2 weeks[27,29] MOX 0.2 mg/kg PO once[17,41] MOX 0.5 mg/kg PO once[18,29] MOX 0.5–1 mg/kg PO *(Capillaria)*[7,27]
Ratites	ABZ 5.2 mg/kg PO q12h × 3d, repeat in 14d (flagellates, cestodes)[50] ABZ 6 mg/kg PO once (ostriches [*Struthio camelus*], *Libyostrongylus* spp.)[7,51] FBZ 15–45 mg/kg PO (ostriches)[7] MBZ 5–7 mg/kg PO (ostriches)[50] TBZ 50 mg/kg PO, repeat in 14d[7]	

BZD, Benzimidazole; *CNS*, central nervous system; *d*, day(s); *GI*, gastrointestinal; *IM*, intramuscular; *PO*, per os; *prn*, "*pro re nata*" or as needed; *SC*, subcutaneous.

Pyrazino-Isoquinoline	Imidathiazole	Tetrahydro-Pyrimidine
5–10 mg/kg PO, repeat after 2–4 weeks[7] 9 mg/kg IM, repeat in 10d (cestodes)[7,18] 10–20 mg/kg PO, IM once[16]	2–5 mg/kg SC, IM; repeat in 10–14d × three treatments (immunostimulant)[18] 20–40 mg/kg PO once[16] 100–200 mg/L drinking water × 3d, repeat in 2 weeks[16]	7–25 mg/kg PO once[16]
5–10 mg/kg PO, SC q24h × 14d[7] 10 mg/kg PO, SC once; repeat in 7d[26] 30–50 mg/kg PO, SC, IM; repeat in 14d[7]	10–20 mg/kg PO, SC q24h × 2d[47] 20 mg/kg PO once[47] 20–40 mg/kg PO once[29] 40 mg/kg PO once *(Capillaria)*[7] 100–200 mg/L drinking water × 3d, repeat in 2 weeks[27]	7–20 mg/kg PO, repeat in 14d[7] 20 mg/kg PO once[18,29,47]
7.5 mg/kg PO (ostriches [*Struthio camelus*])[7]	7.5 mg/kg PO, SC (ostriches [*Struthio camelus*])[7] 30 mg/kg PO q10d	5–7 mg/kg PO (ostriches [*Struthio camelus*])[50]

TABLE 9.2

Mean (±SD) Pharmacokinetic Parameters of Ivermectin in Plasma Following IV, SC, or Oral Administration to Laying Hens (*Gallus gallus domesticus*) at a Dose of 0.2 mg/kg Body Weight[5]

Parameters	IV	SC	PO
$T_{1/2}$ (days)	0.37 ± 0.06[a]	1.45 ± 0.45[b]	0.23 ± 0.11[c]
C_{max} (ng/mL)	316.0 ± 192.4	82.9 ± 12.4[b]	10.2 ± 7.2[c]
T_{max} (hours)	N/A	6.0 ± 0.00[b]	3.36 ± 0.96[c]
AUC (ng day/mL)	37.2 ± 18.6	49.2 ± 8.7[b]	4.4 ± 3.6[c]
MRT (days)	0.31 ± 0.06[a]	1.05 ± 0.24[b]	0.38 ± 0.14[c]
MAT (days)	N/A	0.78 ± 0.26[b]	0.11 ± 0.09[c]
Clearance (L/day/kg)	6.5 ± 2.7	N/A	N/A
Vd_{ss} (L/kg)	2.0 ± 0.9	N/A	N/A
Bioavailability (%)	N/A	132.3[b]	11.9[c]

[a,b,c]Values with different superscripts in same row differ ($P < 0.01$).

AUC, Area under the curve from time zero to infinity; C_{max}, maximum concentration; *IV*, intravenous; *MAT*, mean absorption time; *MRT*, mean residence time; *N/A*, not applicable; *PO*, per os; *SC*, subcutaneous; *SD*, standard deviation; $T_{1/2}$, elimination half-life; T_{max}, time to reach maximum concentration; Vd_{ss}, volume of distribution at steady state.

The toxic effects of BZDs are also highly species specific. Therefore every use of a BZD in a novel avian species should be approached with caution. Morbidity and mortality induced by FBZ are known to be dose related in pigeons and doves.[10] Use of FBZ (50 mg/kg PO q24h for 5 days) caused death in doves.[12,25] In a study evaluating FBZ use in day-old common peafowl *(Pavo cristatus)*, there were no morbidities or mortalities with up to 300 mg/kg fed for 21 days.[56] Therefore the proposed dose of 100 mg/kg in the feed for 7 consecutive days is well within the margin of safety for this species.[56] The risk of toxic effects from a BZD suspension may potentially be reduced by thoroughly shaking the suspension before administration.

Levamisole and Pyrantel

Cholinergic agonists, such as the synthetic imidazothiazole derivative, levamisole, and the pyrimidine anthelmintic, pyrantel pamoate, are widely used to treat nematode infestations (Table 9.1), including ascarids, *Capillaria*, and *Heterakis* (Table 9.3).[57] These cholinergic agonists target a restricted subgroup of nematode acetylcholine receptor ion channels in parasite nerves and muscles causing spastic contraction paralysis, followed by expulsion of worms by normal gut peristalsis, and death.[6,53,57] Levamisole has also been shown to enhance immunity in chickens and ducklings.[6,58,59]

Praziquantel

Praziquantel (PZQ) is a synthetic isoquinoline-pyrazine derivative that is active against a wide range of cestodes as well as trematodes (Table 9.1).[6] PZQ increases cell membrane permeability to calcium by disrupting ion transport, which damages the parasite tegument and causes spastic, paralytic muscle contractions of the parasite, followed by their expulsion and death.[6,53]

Niclosamide

The salicylamide derivative, niclosamide, is highly effective for the treatment of cestode infections.[6,20,53,61] Its proposed mechanism of action involves uncoupling of oxidative phosphorylation, but there is also mounting evidence that niclosamide has potential antineoplastic activity.[53,62] Use caution when handling this chemical as it is highly toxic to aquatic life with long-lasting effects,[61] which should be taken into consideration when birds are housed in mixed aquatic zoo exhibits. Niclosamide was voluntarily withdrawn from the US market in 1996.[53]

Therapy for Specific Helminthic Concerns

Ascarids

MLs are important for the control of nematodes, such as *Tetrameres* and *Dispharynx* (Table 9.3),[1] and studies have confirmed the high efficacy of IVM against nematodes in poultry (Table 9.3).[5] Most use of MLs in exotic birds is based on anecdotal experience; however, a study in ostrich *(Struthio camelus)* did confirm that IVM was effective against *Libyostrongylus* spp.[51]

BZDs are also frequently selected to treat helminthic diseases. In fact, only FLBZ and FBZ are currently approved in the European Union (EU) for treatment of nematodiasis in laying hens.[33] Due to a ban on battery cages, infection with the poultry roundworm *Ascaridia galli* has increased in European countries; however, FLBZ (1.43 mg/kg administered in the drinking water for 7 days) was highly effective against both adult worms and larvae although birds were reinfected within 1 week.[33]

Syngamus/Cyathostoma

The nematode *Syngamus trachea* (gapeworm or redworm) is primarily seen in gallinaceous birds and waterfowl; however, it has been reported in a variety of avian species, including birds of prey, passerine birds, particularly starlings and crows, as well as emus *(Dromaius novaehollandiae)*.[38,39,56,63–67] *Syngamus* spp. have a direct life cycle (Box 9.1), but transmission can involve a paratenic host, such as earthworms, snails, slugs, or insects, consumed by the host bird (Table 9.3). Adult worms are typically found within the trachea or lungs. Rapidly growing worms can cause inflammation or obstruction leading to open-mouth breathing, cough-like sounds, sneezing, blood in the oral cavity or at the commissure of the beak, head shaking, and severe respiratory distress.[38,39,63] Young birds are most seriously affected; adult birds rarely develop heavy infections.[68]

Closely related *Cyathostoma* spp. are found in the trachea, air sacs, or orbital sinuses of birds.[64] Cyathostomes have been reported quite commonly in raptors, like merlins (*Falco columbarius*), hawks, and owls. These birds do not feed directly on invertebrate paratenic hosts but it is thought they acquire infections by eating rodents or small birds with such prey in their gastrointestinal tract.[64]

TABLE 9.3

Life Cycle of Common Helminths[25,60]

Parasite	Life Cycle	Comments
Ascarids	Direct intestinal	Larvae may encyst in lungs and liver
Capillaria	Direct intestinal	
Cyathostoma	Direct	Infection can also be transmitted through ingestion of a paratenic host, the earthworm
Dispharynx	Intermediate host required	Pill bug
Heterakis	Direct	Eggs are ingested in contaminated food or water. All larval development occurs within the cecal lumen.
Syngamus	Direct	Infection can also be transmitted through ingestion of a paratenic host, such as earthworms, snails, slugs, and insects like flies, cockroaches
Tetrameres	Intermediate host required	Pill bug

BOX 9.1 DIRECT LIFE CYCLE OF *SYNGAMUS* SPP.[69]

- Birds become infected through ingestion of food or water contaminated with infective eggs or through ingestion of invertebrate paratenic hosts contaminated with larvae.
- Earthworms are particularly important in maintaining as paratenic hosts because third-stage larvae (L3) can survive for an extended period (months to years).
- After ingestion, *Syngamus* L3 hatch, cross the intestinal tract lining, and are carried in the blood stream to the lungs where they molt twice.
- Male and female worms migrate to larger airways where they permanently copulate.
- The pair then migrates to the trachea at about 7 days postinfection where they attach to the wall and suck blood, which causes the worms to appear reddish.
- Adult females lay eggs within the airways between 7 and 20 days later.
- Eggs are coughed up, swallowed, and then passed in the feces.
- Infective larvae develop within the egg beginning at about day 3 and hatch in 9 days when they are subsequently ingested by the host directly or by the host eating an earthworm or snail that ate the infective larva.

Birds with gapeworms often present as emergent cases requiring supplemental oxygen.[39] Dexamethasone can be administered intramuscularly or intratracheally to reduce airway inflammation and mucosal swelling.[39] Anthelmintics that have demonstrated efficacy against gapeworm/cyathostome infections include MLs, like IVM, and BZDs, such as FBZ, FLBZ, MBZ, and TBZ.[39,56,64] In fact, TBZ is registered for the control of *S. trachea* in pheasants in the United States.[70] Use of these anthelmintics will kill adult worms, which are then coughed up and swallowed.[39] If patient size allows, worms can also be removed endoscopically or with biopsy forceps. Restriction of young birds' access to soil, and potential transport hosts, can also reduce the impact of these parasites.[64]

Cestodes

PZQ, niclosamide, and the acridine derivative, quinacrine (see Antiprotozoal Agents), have been effective for the treatment of cestode infections.[6,53,71]

Trematodes

Although PZQ is reportedly effective against schistosomes,[6] most cases described in the literature have been less than promising. A study evaluating treatment of the eye fluke *Philophthalmus gralli* in specific-pathogen-free chickens *(Gallus gallus domesticus)* using PZQ (10-50-100 mg/kg IM once) or FBZ (50 mg/kg PO q24h for 3 days) found that neither treatment was effective for complete elimination of *P. gralli*.[20] Similarly, ineffective results were seen in greater rheas *(Rhea americana)* naturally infected with *P. gralli* and treated with 1% PZQ ointment twice daily.[20,72] Levamisole (1.5 mg/kg PO) was only partially effective in the treatment of ostriches infected with *P. gralli*.[20,73] Additionally, no differences in total counts of trematodes, cestodes, or *Capillaria* were noted in black stilts *(Himantopus novaezelandiae)* treated with PZQ (~20 mg/kg).[74]

ANTIPROTOZOAL AGENTS

Several drugs and drug classes demonstrate activity against protozoan parasites (Table 9.4).

Ionophores

Polyether ionophorous antibiotics or ionophores are fermentation products of *Streptomyces* spp. or *Actinomadura* spp. that act by disrupting ion gradients across the cell membrane of the parasite.[6,78,121] Monovalent ionophores, such as monensin, narasin, salinomycin, maduramicin, and semduramicin, preferentially bind to the monovalent ions sodium and potassium, affecting the transport of cations through biological membranes and leading to cytotoxicity.[6,52,121] Lasalocid has the ability to bind divalent cations, like calcium and magnesium, and is therefore termed a divalent ionophore.[6] Ionophores act on the intestinal free forms of coccidian stages when the drug comes into contact with them within the intestinal lumen.[6] This drug class does not completely suppress parasite development, which allows immunity to develop in the host.[78] Both monensin and salinomycin have been described as possessing broad-spectrum anticoccidial activity (Table 9.4).[6,121]

Ionophores are most widely used in the EU; however, monensin is approved for prevention of coccidiosis in chickens, turkeys, and bobwhite quail *(Colinus virginianus)* in the United States. Monensin has also demonstrated efficacy in the ring-necked pheasant *(Phasianus colchicus)* and Japanese quail *(Coturnix japonica)*.[77,78] Lasalocid is approved for use in the chukar partridge *(Alectoris chukar)*.[122]

Ionophores have a rather narrow safety margin; however, monensin is considered safe in poultry when fed at approved dosages.[77,78] Toxic effects can include neuropathy, manifested by myelin degeneration and ataxia, muscle degeneration, and cardiotoxicity.[78] Maduramicin is the most toxic of all the ionophores, potentially causing severe cardiovascular defects. Monensin has greater toxicity for turkeys than chickens.[6] Semduramicin is also well tolerated by chickens.[78] Lasalocid has relatively low toxicity, and salinomycin is the least toxic of all the ionophores; however, severe toxicity has been described in turkeys, manifesting as paralysis of the head and legs and death.[6]

Triazines

Triazines, such as toltrazuril, ponazuril, and diclazuril, have been used globally for years to combat protozoal parasites (Table 9.4). Unlike other anticoccidial drugs, triazines are effective against all intracellular stages although preformed oocysts do not appear to be susceptible.[75] Their mechanism of action is not completely understood but triazines inhibit cytokinesis of schizonts and in so doing prevent complete nuclear division.[122] Triazines may also promote the development of a strong humoral immune response, thereby preventing reinfection.[75,76,90,94,123]

In one study evaluating clazuril in pigeons (*Columba livia domestica*; 2.5 mg/bird), no adverse effects or changes in reproductive physiology or body weight were detected in birds dosed weekly for 6 months.[75] The pharmacokinetics of diclazuril and toltrazuril have also been studied in the chicken (Table 9.5).[75,123] Bioavailability after oral diclazuril has been described as approximately 5%.[75] Additionally, in a study evaluating triazines in broiler chickens, the clinical efficacy of

TABLE 9.4

Antiprotozoal Agents for Use in Birds

	Nitroimidazoles	Triazine Derivatives	Ionophores
Examples	Metronidazole (MTZ) Carnidazole (CRZ) Dimetridazole (DMZ) Ronidazole (RNZ) Tinidazole (TNZ)	Clazuril (CZ) Diclazuril (DIC) Ethanamizuril Nitromezuril (NTM) Ponazuril (PON) Toltrazuril (TOL)	Monensin (M) Narasin (N) Salinomycin (S) Maduramicin (MAD) Semduramicin (SEM) Lasalocid (L)
Clinical applications	Broad-spectrum antiprotozoal and anticoccidial activity, including *Giardia*, *Trichomonas*, *Cochlosoma*. Also active against anaerobic, microaerophilic bacteria[6] Former drug class of choice for *Histomonas*, *Spironucleus* in turkeys and game birds (see Potential adverse effects or toxicity, later)	Broad-spectrum anticoccidial and antiprotozoal activity, but primarily used for *Eimeria*, *Isospora*. Also used for refractory coccidiosis (e.g., *Atoxoplasma*)[6,75,76]	M: Approved for use in turkey, bobwhite quail *(Colinus virginianus)* in the United States, effective in the ring-necked pheasant *(Phasianus colchicus)* and Japanese quail *(Coturnix japonica)* but not the chukar *(Alectoris chukar)*[77] M, S: Broad-spectrum anticoccidial activity[6] SEM: Highly effective against *Eimeria*[78]
Contraindications, precautions	Nitroimidazole use is prohibited in food animals in many nations, including the United States and European Union[30,51] *Candida* overgrowth can occur after prolonged therapy, especially in cockatiels *(Nymphicus hollandicus)*[16] CRZ must be given on an empty stomach, otherwise vomiting can occur[16] DRZ: do not use in Pekin robins *(Leiothrix lutea)* and finches. Use lower dose with caution in lorikeets and mynahs[16]	DIC: some *Eimeria* resistance in poultry[8] Baycox 2.5% (TOL) should not be administered directly into the crop via gavage tube because of its harsh, alkaline properties[7,81]	Most ionophores are incompatible with several ABX, including tiamulin, chloramphenicol, erythromycin, certain sulfonamides, and some antioxidants[6,67,78] M (100 mg/kg) reduced fertility when given to broiler breeders but had no effect on egg production, egg weight, or hatch.[77] There are also anecdotal reports of anorexia with M administration during hot weather[77]
Potential adverse effects or toxicity	In some animal studies, high-dose, long-term MTZ has shown mutagenic or carcinogenic effects[80]; therefore nitroimidazole use has been suspended in many countries[6] Reversible CNS signs (ataxia, seizure) have been observed with MTZ or DMZ overdose[16,38,84–86]	Minimal adverse effects, such as GI problems, have been reported in mammals when given at approved dosages[75] Mild-to-severe feather damage has been reported in an 8.5-wk-old offspring of adult pigeons treated with TOL[81]	M: Relatively narrow safety margin[66]; however, safe in poultry when fed at approved dosages[67] M: Toxic effects include neuropathy, muscle degeneration, cardiotoxicity,[78] which appears as anorexia, decreased growth rate and weight gain, depression, ruffled feathers, recumbency, ataxia, leg weakness, paralysis, and death[77,87] N: Toxic to turkeys[36]
Dosage in most species	CRZ 3–7 mg/kg PO[8] MTZ 10–30 mg/kg PO q12h[8] MTZ 30–50 mg/kg PO q24h[8,16] RNZ 6–10 mg/kg PO q24h × 6–10d[8,16] RNZ 60–600 mg/L drinking water × 7d[16,25,91]	CZ 3–7 mg/kg PO[8] CZ 7 mg/kg PO × 3d, off 2d, on 3 d[7,18] PON 10–20 mg/kg PO; repeat in 10–14d[8]	—
Anseriformes	—	CZ 5–10 mg/kg PO q72h q3 trmts[47] CZ 5–10 mg/kg PO q24h × 3d, off 2d, on 3d[30] TOL 12.5 mg/L drinking water × 2d[30] TOL 25 mg/L drinking water × 2d[30]	—

Thiamine Analog	Aminoglycoside	Sulfonamides
Amprolium hydrochloride	Paromomycin	Sulfachlorpyridazine (SCP) Sulfadiazine-trimethoprim (TMP/SDZ) Sulfadimethoxine (SDM) Sulfamethazine (SMT) Sulfadimidine (SDI) Sulfamethoxazole (SMX) Sulfaquinoxaline (SQX)
Coccidiostat, poorly active against some *Eimeria* spp.[78] Largely used in combination products to extend its spectrum of activity, such as amprolium with ethopabate[78]	Antiprotozoal activity against *Entamoeba*, *Trichomonas*, *Giardia, Balantidium*, *Cryptosporidium*. Also active against gram-negative bacteria[6] Largely unabsorbed resulting in high drug levels within the GI tract[79,80]	Broad-spectrum activity against protozoa as well as aerobic bacteria; coccidiostatic at low doses, coccidiocidal at higher doses[6] Long-acting sulfonamides (e.g., sulfamethoxine) are highly active against avian malaria and coccidia when given in combination with pyrimethamine and other diaminopyrimidines
Resistance is common[16] Treatment periods >2 wks may require folic acid supplementation[16] Efficacy can be reduced by high doses of thiamine[7]	May result in secondary bacterial or fungal infections[7] Use with caution if ulcerative intestinal lesions are suspected because renal toxicity may occur[7]	Contraindicated with dehydration, liver disease, renal disease Sulfa drugs reduce pyridoxine and B6 absorption. Begin vitamin B supplementation for 6d after short-term therapy has been completed[25,82] A vitamin supplement containing vitamin B6 should also be used during long-term trmt (>2 wks)[16,83]
Large safety margin of at least 5-fold when used at the recommended level in feed (125 ppm)[78] Seizure activity responsive to thiamine supplementation described in a merlin *(Falco columbarius)* given amprolium (22 mg/kg PO q24h × 6d, then 7.5 mg/kg PO q24h × 4d)[88]	If absorbed systemically, paromomycin can induce ototoxicity and nephrotoxicity; however, it is less ototoxic than other aminoglycosides[80]	Can be associated with nausea, anorexia, diarrhea[6,89] In birds, sulfas can cause reproductive disorders, inhibit vitamin K synthesis, thus impairing blood coagulation, and contribute to bone marrow hypoplasia, agranulocytosis, hematoma, and necrotic changes in the spleen[90]
15–30 mg/kg PO q24h × 1–5d, rest for 5d then repeat trmt[16] 50–100 mg/L drinking water × 5–7d; rest 5d, then repeat trmt[7,16]	100 mg/kg PO q12h × 7d[16]	SCP 100–400 mg/L drinking water × 3–5d, rest × 5d then repeat trmt[16] SCP 400–500 mg/L drinking water × 5d, rest 2d, on 5d[8] SDM 20–50 mg/kg PO q12h × 3–5d, rest × 5d then repeat trmt[16] SDM 250–500 mg/L drinking water × 5–7d, rest × 5d then repeat trmt[16] SDI 50–150 mg/kg PO q12h, rest × 5d then repeat trmt[16] SDI 3330–6660 mg/L drinking water × 5d, rest × 5d then repeat trmt[16] SMT 75–185 mg/kg PO q24h × 3d, rest × 5d then repeat trmt[16] SMT 125 mg/L drinking water × 3d, rest for 5d then repeat trmt[16] SQX 100 mg/kg PO q24h × 3d, rest × 5d then repeat trmt[16] SQX 250–500 mg/L drinking water × 5–7d, rest for 5d then repeat trmt[16]
0.5–1.0 mL/L of 9.6% solution PO in drinking water × 5d[21]	—	—

Continued

TABLE 9.4

Antiprotozoal Agents for Use in Birds—cont'd

	Nitroimidazoles	Triazine Derivatives	Ionophores
Columbiformes	CRZ 10 mg/kg PO (adult pink pigeons [*Nesoenas mayeri*], squabs >18d old, *Trichomonas*)[92] CRZ 20 mg/kg PO once[30] MTZ 25–50 mg/kg PO q12–24h × 5d; use lower dose if twice daily[25] MTZ 1057 mg/L drinking water × 2d[25] CRZ 20 mg/kg PO once[25,91] CRZ 20–30 mg/kg PO q24h × 1–2d[18,27] DMZ 400 mg/L drinking water × at least 3d[93] RNZ 2.5 mg/kg PO × 6d[25] RNZ 10–20 mg/kg PO q24h × 7d[91]	CZ 2.5 mg tablet per pigeon once[6,25,75,81,94] CZ 7 mg/kg PO q24h × 2–3d; rest for 5d then repeat trmt[16] TOL 20 mg/kg[94] TOL 20 mg/kg in drinking water × 2d[25,81]	—
Galliformes	DMZ 200 mg/kg feed *(Histomonas)*[95] MTZ 25 mg/kg PO q12h × 10d[36] MTZ 30 mg/kg PO q12h (poultry, PK/PD)[96] MTZ 110 mg/kg PO q12h (poultry, *Histomonas*)[36] MTZ 200–400 mg/kg feed[95] MTZ 400 mg/L drinking water × 5–15d (game birds)[36] DMZ 800 mg/L drinking water (poultry, game birds)[36] DMZ 185–187.5 mg/kg feed (poultry, game birds)[36] DMZ 200–400 mg/kg feed (chickens [*Gallus gallus domesticus*], *Histomonas*)[95] DMZ 200–400 mg/L drinking water × 5d (game birds)[36] TNZ 200–400 mg/kg feed (chickens, *Histomonas*)[95]	CZ 3 mg/kg PO once or × 5d (chickens [*Gallus gallus domesticus*], PK)[97] DIC 2.5 mg/L drinking water[98] DIC 5–10 mg/L drinking water[8] DIC 1 mg/kg diet[99] DIC 5 mg/L drinking water[100] NTM 3 mg/kg feed (coccidia prophylaxis in broiler chickens, see Box 9.10)[76] PON 20 or 40 mg/kg PO once (peafowl [*Pavo cristatus*])[101] TOL 5–20 mg/kg PO q24h (suggested based on PK)[102] TOL 7.5–24.5 mg/kg PO in a variety of species or in drinking water × 2d (Japanese quail [*Coturnix japonica*])[90] TOL 25 mg/L drinking water × 2d (chickens, *Eimeria*)[100] TOL 75 ppm in drinking water (chickens)[103]	L 67–125 mg/kg feed (game birds, chickens)[36] L 125 mg/kg feed (Chinese ring-necked pheasants [*Phasianus colchicus*])[104] M 59.5–99 mg/kg (turkey [*Meleagris gallopavo*])[77] M 80.5 mg/kg feed (bobwhite quail [*Colinus virginianus*])[77] M 99–121 mg/kg feed (chicken [*Gallus gallus domesticus*])[77] MAD 5–6 mg/kg feed (chicken, turkey; coccidia)[36] N 20–80 mg/kg feed (chicken, prophylaxis; see Box 9.10)[36]
Passeriformes	CRZ 20–30 mg/kg PO q24h × 5d[38] CRZ 33 mg/kg PO, repeat in 14 and 28d (society finches [*Lonchura striata domestica*], Gouldian finches [*Erythrura gouldiae*]; flagellates)[7] DMZ 100 mg/L drinking water × 5 days *(Cochlosoma)*[39] MTZ 20 mg/kg PO q24h × 5d (canaries [*Serinus canaria*], *Trichomonas*)[106,a] MTZ 30 mg/kg PO once *(Cochlosoma)*[38] MTZ 100 mg/L drinking water[39] MTZ 100 mg/kg soft food[39] MTZ 200 mg/L drinking water × 5d (canaries, partially effective against *Trichomonas*)[106] RNZ 50 mg/L drinking water and 50 mg/kg in soft food × 7d *(Cochlosoma)*[39] RNZ 350 mg/L drinking water or 350 mg/kg soft food[39]	DIC 2.5 mg/L drinking water and sulfadiazine (400 mg) - trimethoprim (80 mg) (0.2 mL/L) added to the drinking water × 5d (mynah, *Atoxoplasma*)[98] DIC 5 mg/L drinking water *(Toxoplasma)*[7] DIC 10 mg/kg PO q12h on days 0, 1, 2, 4, 6, 8, 10 (Hawaiian crows [*Corvus hawaiiensis*], *Toxoplasma*)[7] TOL 7–15 mg/kg PO q24h × 3d[16] TOL 12.5 mg/kg PO q24h × 14d *(Atoxoplasma)*[38] TOL 12.5 mg/kg PO q24h × 2d, stop for 5d, repeat 7d cycle over 3 months (blue-crowned laughing thrush [*Pterorhinus courtoisi*], reduced *Atoxoplasma* oocysts in feces)[82] TOL 20–35 mg/kg PO once[16] TOL 25–75 mg/L drinking water × 5d[16] TOL 25 mg/kg in the food q24h × 2d for 10 wks (blue-crowned laughing thrush, *Atoxoplasma*)[107] TOL 180 mg/L drinking water[39]	—

Thiamine Analog	Aminoglycoside	Sulfonamides
200 mg/L × 5d[25] 25 mg/kg/d PO[25]	—	SMT 50–65 mg/pigeon PO × q24h × 3d, 2–3d rest, then a 2–5d trmt period[74] SMT 50–65 mg/pigeon PO × 5d[25] SMT 400 mg/L drinking water × 1d followed by 200–270 mg/L × 4d[25] SDM 330–400 mg/L drinking water × 1d, followed by 200 mg/L × 4d[25]
3 mL/L drinking water (using a 9.6% solution) × 3–5d[30] 115–235 mg/kg feed (poultry, pheasants; lower dose prophylaxis, higher dose therapeutic, *Sarcocystis*, see Box 9.10)[36]	100 mg/kg PO q12h or 50 mg/kg PO q8h × 1–2 wks[105]	SDM 250 mg/L drinking water × 5d (turkeys [*Meleagris gallopavo*])[36] SDM 500 mg/L drinking water × 6d (chickens [*Gallus gallus domesticus*])[30]
50–100 mg/L drinking water × 5–7d[38]	SDI 1000 mg/kg soft food or hulled millet for Gouldian finches *(Erythrura gouldiae)*[19]	SDI 150 mg/L drinking water[39] SCP 150–300 mg/L drinking water[39] SCP 300 mg/L drinking water × 5d, off 3d (repeat cycle 4 times) *(Atoxoplasma)*[38,83] DIC (2.5 mg/L) and SDZ (400 mg)—TMP (80 mg) (0.2 mL/L) added to drinking water × 5d (mynah, *Atoxoplasma*)[98] Sulfachloropyrazine (ESB3) (n/a in the United States)—mix 1 g of 30% powder in 1 L drinking water × 5d, off 3d, then repeat the 5d trmt. Repeat the cycle 4 times (Bali mynah [*Leucopsar rothschildi*] and other passerine birds)[83]

Continued

TABLE 9.4

Antiprotozoal Agents for Use in Birds—cont'd

	Nitroimidazoles	Triazine Derivatives	Ionophores
Psittaciformes	CRZ 20–30 mg/kg PO once[16,18] CRZ 30–50 mg/kg PO, repeat in 10–14d (cockatiels [Nymphicus hollandicus]/*Giardia*)[7] DMZ 200–400 mg/L drinking water × 5d[7,16] MTZ 50 mg/kg PO q12–24h[16] MTZ 40–80 mg/L drinking water × 3d[16] RNZ 6 mg/kg PO q12h and RNZ 2000 mg/L drinking water (budgerigar [*Melopsittacus undulatus*], *Giardia*)[109]	DIC 1 mg/kg PO q24h (TBP, *Sarcocystis* prophylaxis mixed in mashed pine nuts; see *Sarcocystosis* and Box 9.10)[110] PON 20 mg/kg PO q24h (part of a triple-drug combo with pyrimethamine and SMX in TBP; see *Sarcocystosis*)[110] TOL 2 mg/L drinking water × 2 consecutive days per wk[7] TOL 5 mg/L drinking water × 2d, repeat in 14–21d (10 mg/L administered during second course of trmt)[8]	
Raptors	CRZ 20–25 mg/kg PO once[29] CRZ 20 mg/kg PO q24h × 2–5d (Strigiformes)[114] CRZ 25 mg/kg once[47] CRZ 30 mg/kg PO q24h × 1–3d[26] CRZ 120 mg/kg PO once or divided over 2–5d (American kestrels [*Falco sparverius*], screech owls [*Megascops* spp.], resistant *Trichomonas*)[7] DMZ 50 mg/kg PO q24h × 10d (falcons, *Enterocytozoon*)[115] MTZ 50 mg/kg PO q24h × 5–7d (vulture, *Trichomonas*)[116,b] MTZ 50 mg/kg PO q12h × 5d (ABE, *Trichomonas*)[117] MTZ 50 mg/kg PO q12h and toltrazuril 20 mg/kg PO q24h × 2d, then on day 5 (sparrowhawk [*Accipiter nisus*], *Sarcocystis*)[118] MTZ 50–100 mg/kg PO q24h[29] MTZ 100 mg/kg PO q24h × 3d (for severe cases with large granulomas, tissue destruction, follow with surgical removal)[26]	CZ 5–10 mg/kg PO q72h for 3 cycles (coccidiosis)[47] PON 20 mg/kg PO q24h × 7d (falcons, respiratory *Cryptosporidium*)[119] TOL 10 mg/kg PO q8h every other day for three cycles OR one dose per wk for 3 wks[47] TOL 10 mg/kg PO q24h × 2d, repeat in 14d[26] TOL 10 mg/kg PO q24h × 2d *(Caryospora)*[7] TOL 15–25 mg/kg PO q24h × 2d or EOD for 3 doses; repeat in 2 wks[29]	—
Ratites	DMZ 250 mg/kg feed (ostrich [*Struthio camelus*], *Trichomonas*)[18] MTZ 20–25 mg/kg PO q12h[50] MTZ 40 mg/kg PO q24h (rheas)[7] MTZ 1250 mg/L drinking water × 7–10d[7]	—	—

[a]A trichomoniasis outbreak in a flock of canaries (*Serinus canaria* f. *domestica*) was initially managed with metronidazole (200 mg/L) in drinking water for 5 days. This regimen was only partially effective; however, no further clinical signs were observed in the flock after the oral course described was completed.[106]

[b]Used to successful treat *Trichomonas gypaetinii* in a juvenile Cinereous vulture *(Aegypius monachus)*; however, a new plaque was observed in the bird's oropharynx 10 days after completing MTZ administration, and a second 5-day course of MTZ was provided.[116]

ABE, American bald eagle; *ABX*, antibiotics; *CNS*, central nervous system; *d*, day(s); *EOD*, every other day; *GI*, gastrointestinal; *IM*, intramuscular; *PD*, pharmacodynamics; *PK*, pharmacokinetics; *PO*, per os; *SC*, subcutaneous; *TBP*, thick-billed parrots; *trmt*, treatment; *wk*, week.

Thiamine Analog	Aminoglycoside	Sulfonamides
250 mg/L drinking water × 7d (keas [*Nestor notabilis*], *Sarcocystis*; use in combination with pyrimethamine and primaquine)[7]	100 mg/kg PO q24h × 5–7d diluted in dextrose by crop gavage (black cockatoos, *Cryptosporidium*)[111] 100 mg/kg PO q12h × 7d (macaw [*Ara* spp.] chicks)[7]	Combination 2 wk regimen of TMP/SDZ (30 mg/kg IM 12qh) with pyrimethamine (0.5 mg/kg PO 12qh) (3 psittacine birds relapsed within 1 wk trmt was renewed and extended for an additional 30d, *Sarcocystis*)[112,113] SCP 400 mg/L drinking water × 30d (budgerigars [*Melopsittacus undulatus*], cockatiels [*Nymphicus hollandicus*])[36] SDM 50 mg/kg PO q24h × 5d, off 3d, on 5d[36]
30 mg/kg PO q24h × 5d[7,18,29]	100 mg/kg PO q12h *Cryptosporidium* (gyrfalcons [*Falco rusticulos*])[120] 100 mg/kg PO q12h × 7d (falcons)[7,108]	SDM 25–50 mg/kg PO q24h × 3d[36] SDM 25–50 mg/kg PO q24h × 3d, off 2d, then q24h × 3d[36] SDM 50 mg/kg PO once, then 25 mg/kg PO q24h × 3–7d[26]
–	–	–

TABLE 9.5

Pharmacokinetic Parameters of Clazuril Following Single and Multiple Doses (3 mg/kg) in Laying Hens (*Gallus gallus domesticus*)[97]

Parameter	Single Dose	Multiple Doses
AUC (μg hour/mL)	129.9 ± 43.1	223.8 ± 13.7
K_{01} (L/hour)	3.4	0.3
K_{10} (L/hour)	0.6	0.1
K_{01} $t_{1/2}$ (hours)	3.6 ± 1.9	2.0 ± 0.7
K_{10} $t_{1/2}$ (hours)	7.4 ± 4.3	16.4 ± 4.2
T_{max} (hours)	7.3 ± 2.3	6.8 ± 4.21
C_{max} (μg/mL)	6.2 ± 0.8	7.1 ± 0.7

Data are shown as mean + SD.
AUC, Area under the concentration-time curve; C_{max}, peak plasma concentration; K_{01}, constant for the absorption phase; K_{01} $t_{1/2}$, half-life of the absorption phase; K_{10}, constant of the elimination phase; K_{10} $t_{1/2}$, half-life of the elimination phase; T_{max}, time to reach peak plasma concentration.

nitromezuril (3 mg/kg) was superior to diclazuril (1 mg/kg) based on comparison of anticoccidial indices, such as weight gain and lesion scores.[75,76]

Amprolium

The pyrimidine derivative, amprolium, is structurally similar to thiamine and is therefore able to competitively inhibit thiamine uptake by schizonts and prevent many essential metabolic reactions.[78] Amprolium demonstrates a large margin of safety, but it is poorly active against some *Eimeria* spp.[78] Amprolium is largely used in combination products or mixtures to extend its spectrum of activity (Table 9.4).[78]

Azithromycin

The macrolide antibiotic, azithromycin, acts through inhibition of protein synthesis.[6,79] Azithromycin has shown some efficacy against *Cryptosporidium*, *Giardia*, and *Entamoeba*,[6,80,105,124]

as well as variable degrees of schizontocidal activity against avian malaria.[125–129]

Paromomycin

The aminoglycoside, paromomycin, has also shown some efficacy against *Giardia* and *Entamoeba* (Table 9.4).[6,79,80,105,124] Paromomycin has also been used for resistant *Trichomonas* spp. in human patients.[130] Like azithromycin, this antimicrobial acts through inhibition of protein synthesis.[6,79,80]

Nitroimidazoles

Nitroimidazoles are a class of drugs that have remarkable broad-spectrum activity against a wide range of protozoa, including *Giardia*, *Trichomonas*, *Spironucleus*, *Histomonas meleagridis*, and *Entamoeba* spp., as well as a few nematode worms, *Mycobacteria* spp., and anaerobic bacteria (Table 9.4).[6,53,131,132] Metronidazole is a prototype of the 5-nitroimidazole group and the most widely used drug of its class.[132] Close structural analogs include dimetridazole, ornidazole, ronidazole, tinidazole, carnidazole, and nimorazole. The mechanism of action for nitroimidazoles is not completely understood, but the target organism likely preferentially reduces the 5-nitro group on the molecule.[6,131] This creates active metabolites that cause a loss of helical DNA structure, strand breakage, and eventual cell death.[6,131]

In many countries, some nitroimidazoles are unavailable due to toxicity concerns.[6] Nitroimidazoles, like dimetridazole and metronidazole, are suspected mutagens and carcinogens, and their use in food animals is completely prohibited in both the United States and the EU.[52] Adverse effects specifically described in birds have been neurological. Reversible central nervous system deficits have been observed in some avian patients following metronidazole or dimetridazole administration.[39,84–86] Ronidazole neurotoxicity has also been described in three society finches *(Lonchura striata)*.[84]

Quinacrine

Quinacrine hydrochloride, also known as mepacrine, is an acridine derivative that is chemically and clinically very similar to 4-aminoquinolines, like chloroquine and amodiaquine (see Therapy for Specific Protozoal Concerns).[133] Quinacrine was initially the drug of choice for prevention and therapy of malaria and other tropical infections,[71,133] but today it is used to treat giardiasis and amebiasis.[79,133,134] Quinacrine has also been used as an immunomodulator, a form of contraception/sterilization, and recent research has shown potential antineoplastic activity.[71,135] In human patients, quinacrine is usually reserved for treatment of refractory protozoal disease, due to the potential for severe, acute hepatotoxicity, especially with high doses.[7,79,135] The most serious toxicity reported in human patients is acute aplastic anemia.[71] The mechanism of action is not fully understood[53]; however, quinacrine may intercalate with parasite DNA.[71,79] Quinacrine is unlicensed in the United Kingdom and it has also been discontinued in the United States since 1992,[134] although the drug could be accessed through compounding pharmacies until very recently.[136]

Therapy for Specific Protozoal Concerns

Intestinal coccidiosis

Coccidiosis is an infectious disease of the intestinal tract of wild and domestic birds caused by *Isospora* spp. and *Eimeria* spp. (Box 9.2). Coccidia are widespread, particularly where intensive production systems are used, making coccidiosis the most important parasitic disease of the poultry industry.[77,78,123] Signs of coccidiosis can range from reduced growth, impaired performance, and increased susceptibility to secondary infections, to weight loss, severe diarrhea, and even death.[78,81] Drug therapy is not indicated for mild cases.[74] Disease is self-limiting when proper management is instituted that focuses on strict attention to hygiene, sanitation, and biosecurity.[78,81]

BOX 9.2 DIRECT LIFE CYCLE OF *EIMERIA* spp.[77,78,106]

- The host animal ingests sporulated oocysts, an infectious stage that contains sporozoites.
- Once ingested, the oocyst wall degrades, releasing first-generation sporozoites into the small intestine.
- Activated sporozoites enter intestinal epithelia where they become trophozoites, acquiring nutrients from host cells.
- Both asexual and sexual reproduction (schizogony and gametogony) then occur within the host.
- After about 1 week, noninfectious, unsporulated oocysts are shed in the feces into the environment.

Anticoccidial drugs belong to one of two categories: ionophores and synthetic compounds. In large poultry flocks, ionophores are the principal choice for controlling coccidiosis.[78] Synthetic compounds include amprolium, decoquinate, clopidol, sulfonamides, halofuginone hydrobromide, robenidine, and triazines, like toltrazuril and diclazuril (Table 9.4).[78,90,123] The carbanilide, nicarbazin, can also be used; however, this broad-spectrum agent has only a small safety margin.[78] Nicarbazin disrupts ion and water equilibrium. Therefore medicated birds are at increased risk for heat stress in hot, humid conditions.[78] Nicarbazin also has an adverse effect on layers, causing mottling of yolks, reduced hatchability, and decreased egg production.[78] Combination products, consisting of a synthetic compound and an ionophore, such as nicarbazin and narasin or two synthetic compounds, like clopidol and methyl benzoquate, are also available.[78] All anticoccidial drugs are effective during only the asexual stage, except for triazines, which are effective against all intracellular stages although preformed oocysts do not appear to be susceptible.[75]

In a recent pharmacokinetic study evaluating a single dose of ponazuril (20 or 40 mg/kg PO) in peafowl, coccidial shedding ceased in all birds by the end of the study and no adverse effects were observed.[101] In a study investigating the efficacy of toltrazuril in pigeons against heavy experimental infections with *Eimeria* spp. infections, the minimum required dose of toltrazuril to completely suppress oocyst excretion was 20 mg/kg for 1 day.[75,94] After treatment with toltrazuril, there was also a dose-dependent period during which pigeons remained negative.[75,94] A period of persistent toltrazuril activity ranging from 7 to 28 days has been documented in both pigeons and poultry.[75,94] Nevertheless, toltrazuril is not a cure-all. Despite recent administration of toltrazuril (25 mg/kg PO), an abundance of *Eimeria apteryxii* and *E. kiwii* oocysts were identified in droppings from three brown kiwi *(Apteryx mantelli)*.[137]

Disseminated visceral coccidiosis

Disseminated visceral coccidiosis caused by *Eimeria* spp., particularly *E. reichenowi*, was first recognized in captive sandhill

BOX 9.3 PROBABLE LIFE CYCLE OF *EIMERIA* sp. IN CRANES[77,87,138]

- A sporulated oocyst is consumed and ruptures in the gut releasing sporocysts
- Sporocysts rupture to release sporozoites
- Sporozoites penetrate mucosal epithelium of the distal jejunum
- Sporozoites undergo asexual merogony for one or more generations
- Merozoites reinfect intestinal cells OR are taken up by mononuclear phagocytes, moving to the lungs and other organs, including the liver, heart, and spleen
- In chronic disease, merozoites survive in granulomas in various tissues

BOX 9.5 LIFE CYCLE OF *CRYPTOSPORIDIUM* spp.[105,143]

- Thick-walled, sporulated oocysts in contaminated food, water, or materials are ingested or inhaled by a new host
- Sporozoites are released and parasitize epithelial cells of the gastrointestinal tract or other tissues, undergoing asexual multiplication (schizogony or merogony) and sexual multiplication (gametogony)
- Two types of oocysts develop that sporulate within the host
- Thin-walled oocysts are autoinfective (sporozoite > trophozoite > meronts)
- Thick-walled oocysts are excreted in feces or coughed up from the respiratory tract

cranes *(Grus canadensis)* and whooping cranes *(G. americana)*.[87,138] These organisms demonstrate an unusual proliferative phase that can develop within the respiratory tract and disseminate to the viscera (Box 9.3).[77]

Reports describe the successful use of monensin (99 mg/kg feed), offered continuously or for 2 months before and 2 months after the chick-rearing season, and trimethoprim-sulfamethoxazole (16–24 mg/kg trimethoprim PO q12h) to successfully control visceral coccidiosis in *G. canadensis*.[77,138,139] Some anticoccidial drugs, such as amprolium and clazuril, have shown poor efficacy against disseminated visceral coccidiosis.[139]

Atoxoplasmosis

Atoxoplasmosis, also known as systemic isosporosis or visceral coccidiosis, is caused by *Isospora* spp. that invade circulating lymphocytes affecting internal organs and intestines (Box 9.4).[140] *Isospora* spp. are most commonly described in passerine birds.[98,140] These organisms are highly host specific, with approximately 500 *Isospora* spp. reported within the order Passeriformes.[140] There is no effective treatment to completely clear extraintestinal stages of isosporoid coccidia from a host species, and the variety of regimens described is a testament to the fact that no single treatment has proven consistently effective.[82] Drug regimens that have been tried include toltrazuril paired with sulfachlorpyrazine or sulfachlorpyridazine, trimethoprim-sulfamethoxazole alone or in combination with the folic acid antagonist, pyrimethamine, toltrazuril alone, as well as diclazuril and sulfadiazine-trimethoprim.[38,82,98]

Cryptosporidiosis

Cryptosporidiosis is one of the most common parasitic infections in domesticated, caged, and wild birds (Box 9.5),[141] and infections caused by this small, coccidian parasite have been observed in over 30 avian species.[142–144] Cryptosporidia are typically considered secondary, opportunistic pathogens more likely to impact young and/or immunocompromised birds.[105,124,142] Several different species and genotypes have been described in birds including *Cryptosporidium meleagridis*, first discovered in turkeys, *Cryptosporidium baileyi*, first described in broiler chickens, and *C. galli*. *Cryptosporidium baileyi* is the most commonly reported cryptosporidial pathogen in birds.[141–143] Among the species using avian hosts, *C. meleagridis* presents the known zoonotic risk.[142]

Management of cryptosporidiosis is challenging, requiring treatment of affected birds, as well as prevention of spread through the flock.[105] It is also important to identify the source of contamination and to implement proper hygiene and quarantine protocols.[105] Therapeutic agents that have shown activity against *Cryptosporidium* include paromomycin, ionophores, halofuginone, nitazoxanide, and toltrazuril.[6,105,124,145] Paromomycin (100 mg/kg PO q12h or 50 mg/kg PO q8h for 1–2 weeks) was the first drug that proved effective for treatment of intestinal cryptosporidiosis in poultry; however, paromomycin (100 mg/kg PO q12h) was ineffective in falcons with otitis, tracheitis, upper respiratory signs, and central nervous signs caused by *C. baileyi*.[105] Azithromycin (50–80 mg/kg q24h PO) has also been recommended for cryptosporidiosis in poultry.[105] The low end of this dose range was partially effective in treating scops owl (*Otus* spp.) in a rescue center, where 11 of 16 birds fully recovered clinically; however, this dose did not halt excretion of the protozoa in feces or improve endoscopic findings.[124] Azithromycin (50 mg/kg PO q24h for 1 month) given to two falcons with atypical cryptosporidiosis caused by *C. parvum* did not reduce excretion of protozoa or improve respiratory signs.[144]

BOX 9.4 LIFE CYCLE OF *ATOXOPLASMA*[140]

- The host animal ingests sporulated oocysts.
- Both sexual (gametogenesis) and asexual (merogeny) reproduction occur within the parasitized host, forming an environmentally resilient oocyst.
  - Merozoites in lymphocytes are disseminated via the circulation to the viscera where merogeny continues.
  - Gametogenesis occurs within intestinal epithelial cells.
- Oocysts are then released into the environment where they sporulate and become infective after ~5 to 7 days.

Trichomoniasis

Trichomonas gallinae is a flagellated protozoan that parasitizes the upper gastrointestinal tract (see Chapter 22). This organism has a direct life cycle and reproduces by binary fission. Under favorable conditions, trichomonads exist at the trophozoite stage in vitro.[146] Cyst-like stages or pseudocysts may survive up to 20 months during unfavorable environmental conditions.[116,146] Pigeons and doves serve as the reservoir hosts for *T. gallinae*.[116,146] Clinical disease is most common in Accipitriformes, Falconiformes, and Strigiformes.[116,146,147]

Trichomonas gallinae is also found in Psittaciformes, particularly budgerigars *(Melopsittacus undulatus)*, and trichomoniasis is also an emerging infectious disease of wild finches, mainly in Europe and the United Kingdom.[146,148]

Nitroimidazoles are the treatment of choice for avian trichomoniasis,[25,91,146,147] although dimetridazole and ronidazole were associated with only limited success in one subpopulation of wild pink pigeons *(Columba mayeri)*.[92] Some nitroimidazoles, such as dimetridazole, metronidazole, and ronidazole, can be administered in drinking water. To successfully suppress *T. gallinae* infection in the homing pigeon *(Columba livia)*, medicated drinking water containing dimetridazole (400 mg/L) had to be administered for at least 3 days.[93] There is a risk for overdose and toxicity when drugs are added to drinking water.[81] Base the dosage on the daily volume of water consumed by the individual pigeon loft, which should ideally be measured 1 day before treatment is initiated.[81] Water intake can vary considerably with the age of the bird and weather conditions.[81] Nestlings consume up to five times more water than adult birds.[81] Birds also tend to drink more during the warm summer months. Therefore, it is recommended to select a lower dose during hot weather and a higher dose during cool weather.[7]

Some nitroimidazoles can also be administered as tablets by gently advancing the pill into the crop with a digit or a curved hemostat.[149] The bioavailability of a sustained-release ronidazole tablet was studied in the homing pigeon.[149] The tablet exhibited prolonged drug release in vitro and drug absorption was nearly complete after oral administration of the tablet. When given on an empty stomach, the tablet failed as a prolonged release system.[149] Evaluation of a 2-day treatment with a dimetridazole tablet (20 mg/tablet) in both fasted and fed pigeons was shown to be ineffective.[93] The absolute bioavailability of the tablet in fasted pigeons was 83.8%. Bioavailability was reduced by 20% when the tablet was administered with food.[93]

Giardiasis

Giardia spp. is a flagellated protozoan capable of colonizing the small intestine of many vertebrates, including birds (see Chapter 23).[5] *Giardia psittaci* is relatively common in budgerigars.[150] Transmission of *Giardia* occurs through the fecal-oral route and may be either direct or indirect via contaminated food or water (Box 9.6).[34]

There is no ideal drug for treatment of giardiasis; however, nitroimidazoles, particularly metronidazole, are most commonly used for first-line treatment.[79,80] Nitroimidazoles attack the trophozoite form of *Giardia* effectively; however, they demonstrate poor penetration through the cyst wall.[80] Azithromycin has also demonstrated in vitro activity comparable to that of metronidazole.[80] Other drugs used to treat giardiasis include chloroquine, which reduces *Giardia*'s ability to attach to intestinal epithelium, the nitrofuran derivative, furazolidone; the synthetic benzamide, nitazoxanide; quinacrine, and BZDs, like ABZ and MBZ.[6,79,80] In some human studies, BZDs were more active against *Giardia* than metronidazole and quinacrine, even at low concentrations.[80] ABZ was also associated with fewer adverse effects in human patients when compared to metronidazole.[80] Know that ABZ's efficacy against *Giardia* is dose-dependent, ranging from 25% to 90%.[80] Paromomycin has also demonstrated antigiardial activity, but its effects were lower than with nitroimidazoles, quinacrine, or furazolidone, and it is therefore rarely used in clinical practice.[79,80] Combination therapies are frequently employed against *Giardia* and generally incorporate a nitroimidazole, like metronidazole.[79] For instance, in a survey of human patients, metronidazole and ABZ demonstrated a higher cure rate in vivo than ABZ alone.[79] Recent investigations have also found that cysteine-modifying agents, such as omeprazole and auranofin, had activity against *Giardia* resistant to first-line drugs.[80,151,152]

Sarcocystosis

Sarcocystis spp. is a coccidian parasite with an indirect life cycle. Predator species serve as definitive hosts and prey species serve as intermediate hosts (Box 9.7).[112,143] At least 12 *Sarcocystis* spp. use birds as definitive hosts and at least 22 *Sarcocystis* spp. use birds as intermediate hosts.[143] Two main species of *Sarcocystis* occur in birds: *S. rileyi*, between ducks and skunks, and *S. falcatula*, with a variety of passerine birds serving as intermediate hosts and the Virginia opossum *(Didelphis virginiana)* serving as the definitive host.[112,143,153] Old World psittacine birds are considered highly susceptible to *S. falcatula* infection.[112] Infection in psittacine birds typically occurs through ingestion of food contaminated with opossum feces, but insects like cockroaches can also serve as paratenic hosts or mechanical vectors.[112,153] *Sarcocystis calchasi* is typically reported in Germany but has also been found in the muscle of columbiforms in North America.[112]

Although sarcocystosis is generally fatal, resolution of clinical signs is possible with extensive supportive care and long-term treatment.[112] Recommended treatment protocols often include pyrimethamine with potentiated sulfonamides.[60,112] Supplementation with yeast and folinic acid

BOX 9.6 LIFE CYCLE OF *GIARDIA* spp.[80]

- The host ingests a viable and mature cyst, the infectious form, which is typically found in fecal-contaminated water, food, or soil.
- When the cyst reaches the duodenum, its wall dissolves, resulting in trophozoites, which bind to the intestinal epithelium.
- Trophozoites feed and grow and eventually encyst within the intestinal wall.
- Newly formed (infectious) cysts are shed in the feces, immediately starting the cycle over again.

BOX 9.7 INDIRECT LIFE CYCLE OF *SARCOCYSTIS* spp.[143,153]

- The intermediate host is infected through the fecal-oral route by ingesting sporocysts.
- The organism then invades various tissues where it undergoes schizogony or merogony, forming large, distinctive cysts in cardiac or striated muscles.
- When the intermediate host is ingested by the definitive host, the organism undergoes sexual multiplication within the intestinal tract.
- Oocysts sporulate internally and are shed in the feces.

(leucovorin) can also aid in the treatment.[112] Prevention and control of infection relies upon keeping definitive and paratenic hosts or their feces away from susceptible birds.[60,112]

A treatment protocol consisting of amprolium with either sulfamethazine or chlortetracycline did not appear to reduce mortality in a flock of chickens infected with *Sarcocystis* spp.[112] Toltrazuril administered at the onset of neurologic signs (25 mg/kg PO q24h for 3 days) was similarly ineffective in pigeons experimentally infected with *S. calchasi*,[154] although the researchers hypothesized that a longer treatment period could warrant further investigation. A triple-drug combination of pyrimethamine (1 mg/kg PO q24h), trimethoprim-sulfamethoxazole (60 mg/kg PO q24h), and ponazuril (20 mg/kg PO q24h) administered for up to 6 months achieved clinical resolution in a captive flock of thick-billed parrots *(Rhynchopsitta pachyrhyncha)*.[110] Also, a 2-week regimen of pyrimethamine (0.5 mg/kg PO q12h) and trimethoprim-sulfadiazine (30 mg/kg IM q12h) was used to successfully treat three psittacine birds.[112] The regimen was reinstated after 1 week and extended for an additional 30 days.[112] All birds recovered clinically after the second regimen; however, birds that recover from sarcocystosis are at risk for recurrence of the disease.[112]

Hemoparasites

Vector-borne haemosporidians are commonly found in birds worldwide.[155–157] Most hemoparasites in raptors are associated with subclinical infection[157,158]; however, Strigiformes show significantly higher prevalence of infection by blood parasites than diurnal raptors.[158] Some fatal infections have been reported in captive psittacine birds; however, free-ranging parrots are infrequently infected.[158] In an interesting survey of 19 Psittaciformes species from a range of habitats, hemoparasites were only found in two of the species sampled.[159] All species that consumed at least one food item known for its secondary metabolites with antimalarial, trypanocidal, or general antiparasitic properties were free from hemoparasites.[159]

Hemoproteus. *Hemoproteus* spp. are transmitted by biting midges, hippoboscid flies, or tabanids (horseflies).[156] Infections are usually relatively benign in birds with asymptomatic or mild clinical findings predominating, such as anemia and splenomegaly.[156,160] Some *Hemoproteus* spp. can cause severe disease and mortality, especially in captive birds removed from their original habitat.[160] Fatal cases have been described in a lorikeet *(Trichoglossus haematodus)*, plum-headed parakeet *(Psittacula cyanocephala)*, snowy owls *(Bubo scandiacus)*, and exotic passerine birds housed in California.[157,158,160–162]

Treatment for *Hemoproteus* is seldom indicated, but when necessary it is generally identical to that for avian malaria (see Plasmodium).[156] Treatment of a goshawk *(Accipiter gentilis)* relied upon a combination of atovaquone and proguanil hydrochloride (Malarone, GlaxoSmithKline, Mississauga, ON, Canada) administered orally.[162] Specifically, half of a Malarone tablet was given with food once a day for 3 days.[162] After 1 week the treatment course was repeated for another 3 days. After 2 weeks, the *Hemoproteus* infection was completely cleared and no parasites were observed in a blood smear stained with Wright solution.[162] A study evaluating *Hemoproteus* treatment with primaquine (0.1 mg SC) in free-ranging blue tits *(Cyanistes caeruleus)* showed increased local survival in female birds but not males.[163] Because few studies are available on experimental infection with *Hemoproteus*, more work is needed to assess the effect of sex on the course of disease.

BOX 9.8 INDIRECT LIFE CYCLE OF *PLASMODIUM* spp.[6,156,157,165–167]

- Gametocytes are ingested during a blood meal by a female mosquito.
- Sporogony occurs within the invertebrate host.
- The parasite reproduces and multiplies within the mosquito midgut. Oocytes eventually rupture releasing sporozoites.
- Sporozoites migrate to the salivary glands of the mosquito.
- Sporozoites are injected into a new avian host when the female mosquito takes a blood meal.
- Sporozoites migrate to the liver and other tissues like the lungs, spleen, kidney, and brain (tissue stage).
- Schizogony occurs within avian erythrocytes; the parasite multiplies in and destroys erythrocytes (blood stage).
- Merozoites are released from the destroyed erythrocytes and they continue the cycle by invading more erythrocytes.

Leucocytozoon. *Leucocytozoon* sporogony occurs in black flies (*Simulium* spp.) or *Culicoides* midges.[6,164,165] These parasites infect many species of avian hosts, with merogony in tissues, like the liver, and multiplication of sexual stages (gametocytes) within erythrocytes.[164,165] Most *Leucocytozoon* infections are subclinical,[156,158,164,166] although nonspecific signs of illness and death have been reported in some captive birds.[156,158,166] *Leucocytozoon* has also been associated with heavy economic losses in the poultry industry due to decreased egg production, anemia, and even death.[165]

Various antimalarial and anticoccidial compounds have shown activity against *Leucocytozoon* parasites including clopidol, primaquine, and halofuginone.[164] Sulfamonomethoxine and ormetoprim combinations have also been reported to prevent or reduce *Leucocytozoon* infection.[164]

Plasmodium. This cyclical infection involves avian hosts and female mosquitoes (Box 9.8).[89] There are over 30 *Plasmodium* spp. that affect birds, and many of these organisms can complete life cycles in a variety of avian species that belong to different families and even different orders.[6,165] Free-ranging birds are most commonly affected; however, avian malaria is occasionally found in domestic fowl and captive birds.[6,156] The clinical picture is often asymptomatic but *Plasmodium* infection can cause severe, acute disease in species that have not evolved with the parasite, such as captive penguins.[89,167]

The complex *Plasmodium* life cycle means that no single drug can eradicate all forms of the organism.[89,125,162,168] Antiplasmodial activity against the malaria life cycle can be divided into the following (Table 9.6)[169]:

- Tissue schizonticides are active against the tissue form of *Plasmodium* spp.
- Blood schizonticides are active against asexual erythrocyte forms of *Plasmodium*. In a study evaluating five antimalarial drugs in broilers infected with *P. gallinaceum*, the two most effective drugs against the asexual erythrocyte stage were chloroquine and doxycycline.[170]
- Gametocidal drugs destroy gametocytes of the malaria parasite, thereby inhibiting the transfer of *Plasmodium* from an infected host to an uninfected female mosquito.

TABLE 9.6

Antimalarial Drugs Used in Birds, Their Parasite Stage's Target, and Mechanism of Action[89,125,165,171,172]

Drug	Target(s)	Drug Class	Mechanism of Action	Comments
Amodiaquine	BS, G	Aryl aminoalcohol, 4-aminoquinoline compound	Not completely understood, but like other quinolone derivatives, likely able to inhibit heme polymerase activity in the body resulting in the accumulation of free heme, which is toxic to parasites[173]	Similar to chloroquine in structure and activity, but effective against some chloroquine-resistant strains[173]
Artemisinin (ART)	BS, G	Artemisinins are a new class of antimicrobial drugs	This prodrug is activated by intraparasitic heme or iron, which catalyzes the cleavage of this endoperoxide, resulting in the release of free radicals that kill the parasite by alkylating and poisoning one or more essential malarial protein(s)[174]	Rare instances of liver injury have been described in human patients
Atovaquone	BS, G, SP	Hydroxynaphthoquinone or an analog of ubiquinone	Inhibition of electron transport by atovaquone will result in indirect inhibition of these enzymes leading to metabolic effects that may include inhibition of nucleic acid and ATP synthesis of the parasite	The mitochondrial toxicity of atovaquone is enhanced by proguanil The adult Malarone (GlaxoSmithKline) tablet is a 250 mg atovaquone/100 mg proguanil combination
Chloroquine	BS, G, SP	Aryl aminoalcohol, 4-aminoquinoline compound	Inhibition of hemozoin formation, hemozoin being a biocrystal synthesized as a disposal product by the parasite[175]	A neuronal storage disease developed in Humboldt penguins *(Spheniscus humboldti)* treated prophylactically with chloroquine.[176] Chloroquine has been shown to cause similar effects in rats and miniature pigs under experimental conditions, therefore caution is advised when employing high and long-term treatment with this drug
Lumefantrine	BS	Aryl aminoalcohol	Unknown, but believed to inhibit nucleic acid and protein synthesis through the inhibition of hematin formation[177]	Currently used only in combination with artemether under the trade name Coartem[177]
Mefloquine	BS	Aryl aminoalcohol, quinoline-methanol derivative of quinine	Unknown, but believed to interfere with the transport of hemoglobin from the erythrocyte to the food vacuole	
Mepacrine				See quinacrine
Piperaquine	BS	Aryl aminoalcohol	The mechanism of piperaquine inhibition of the heme detoxification pathway is unknown but is expected to be similar to that of chloroquine	Currently used in combination with the artemisinin derivative, artenimol, as Eurartesim
Primaquine	TS, G	Aryl aminoalcohol, 8-aminoquinoline compound	Unknown, believed to act as oxidation-reduction mediator, inhibits electron transport processes by inhibiting pyrimidine synthesis[6]	Although primaquine has activity against blood-stage parasites, the levels required to achieve blood schizonticidal action are toxic
Proguanil hydrochloride	BS, SP	Antifolate compound	Selective inhibition of folate biosynthesis, making DNA synthesis impossible and depleting folate cofactors	Also known as chloroguanide See atovaquone, earlier

TABLE 9.6

Antimalarial Drugs Used in Birds, Their Parasite Stage's Target, and Mechanism of Action—cont'd

Drug	Target(s)	Drug Class	Mechanism of Action	Comments
Pyrimethamine	TS, BS, SP	Antifolate compound	Selective inhibition of folate biosynthesis	Similar action to proguanil but with greater efficacy (although with less efficacy on tissue stages). Effects on folate biosynthesis increase when used in combination with sulfonamides In addition to avian malaria, pyrimethamine is also used in the management of *Toxoplasma*, *Atoxoplasma*, *Sarcocystis* infections[16,47]
Quinine	BS, G	Aryl aminoalcohol	Interferes with the parasite's ability to break down and digest hemoglobin. Consequently, the parasite starves and/or builds up toxic levels of partially degraded hemoglobin in itself	An alkaloid derived from the bark of the cinchona tree. Since it was first isolated in 1820, quinine has been the fundamental medical tool for the treatment of malaria, particularly severe disease
Quinacrine hydrochloride	BS, weakly G	Aryl aminoalcohol, 4-aminoquinoline derivative	The exact mechanism of antiparasitic action is unknown; however, quinacrine binds to DNA by intercalation between adjacent base pairs	Also known as mepacrine, quinacrine was formerly used widely as an antimalarial but it is now superseded by chloroquine. Quinacrine has also been used to treat giardiasis as well as some inflammatory diseases, like lupus Overdosage may cause hepatotoxicity[135]
Sulfonamides	BS	—	Used in combination with pyrimethamine to enhance the inhibition of folate biosynthesis	
Tafenoquine	TS, BS, G	Aryl aminoalcohol, 8-aminoquinoline compound	Poorly understood; however, tafenoquine produces hydrogen peroxide and hydroxyl radicals that lead to the parasite death Also inhibits heme polymerase in blood stage of parasites	
Tetracyclines and derivatives such as doxycycline	BS	—	Tetracyclines act by inhibiting bacterial protein synthesis.[129] Their mechanisms of action on *Plasmodium* have not been as well described; however, doxycycline inhibits the synthesis of nucleotides and deoxynucleotides in *P. falciparum*[129]	

BS, schizonticide—active against the asexual blood-stage parasite; *G*, gametocytocide—kills the sexual stage of malaria parasite infective to mosquitoes; *SP*, sporontocide—inhibits the development of oocysts in the mosquito stage of the *Plasmodium* life cycle; *TS*, schizonticide—active against the tissue form of *Plasmodium* spp.

- Sporontocide drugs inhibit the development of oocysts in the mosquito stage of the *Plasmodium* life cycle, thereby preventing transmission of the disease.

In human patients with acute malaria, the complex life cycle of *Plasmodium* spp. also means that complete eradication of a patient's parasite load requires therapeutic drug levels to be present for four cycles to effect a cure.[125]

Antimalarials fall into three broad drug categories: aryl aminoalcohol, antifolate, and artemisinin compounds.[125,169] There is also atovaquone, an antimalarial in its own class, as well as antibiotics with antimalarial activity. Treatment protocols can vary widely (Table 9.7). Combination therapy is frequently used to overcome the limitations of each individual drug group.[169]

Aryl aminoalcohol compounds. The quinoline alkaloid, quinine, was first isolated from cinchona bark in 1820.[125,175,176] Since that time, a number of other natural and synthetic quinoline-based compounds have been developed, including quinidine,

TABLE 9.7

Select Hemoparasite Treatment Protocols

Drug	Species	Dosage	Combination Therapy	Comments	References
Atovaquone	Snowy owls *(Bubo scandiacus)*	½ of an atovaquone-proguanil tablet PO q24h with food × 3d, break for 1 week, then repeat treatment *(Hemoproteus)*		Malarone (GlaxoSmithKline) is a 250 mg atovaquone/100 mg proguanil tablet	162
Chloroquine	Sphenisciformes	5 mg/kg PO q24h for 10d	Primaquine 0.3 mg/kg PO q24h × 10d	*Plasmodium*	178
	Sphenisciformes	10 mg/kg PO q24h × 10–14d, then 5 mg/kg q12h PO × 3d	Primaquine 1.25 mg/kg PO q24h × 10–14d	Some institutions stop therapy at this point and others continue chloroquine 5 mg/kg PO q24h) concurrently with primaquine	89
	Sphenisciformes	10 mg/kg PO on day 1, then 5 mg/kg PO q24h × 9d	Primaquine one 26.3 mg tablet PO q24h × 10d	*Plasmodium*	178
	Broiler chicks *(Gallus gallus domesticus)*	10 mg/kg PO q24h × 5d		Significantly lowest *Plasmodium* infectivity rate (66.67%) for experimentally infected broiler chicks	170
	Psittaciformes and Passeriformes	10–25 mg/kg PO, then 5–15 mg/kg at 6, 18, and 24 h	Primaquine 0.3–1 mg/kg PO q24h × 3–10d	*Plasmodium* and other hemoparasites	16
	Most species, including raptors	25 mg/kg PO followed after 12 h by 15 mg/kg PO followed after 24 h by 15 mg/kg PO followed after 48 h by 15 mg/kg PO	Primaquine 0.75–1 mg/kg PO once	*Plasmodium*	155
	Raptors	Chloroquine 20 mg/kg IV initially, followed by 10 mg/kg PO at 6, 18, and 24 h	Primaquine 1 mg/kg PO q24h × 2d, repeat q7d × 3–5 treatments	*Plasmodium*	8
	Raptors	25 mg/kg IM q24h		*Plasmodium, Leucocytozoon*	47
	Galliformes, Passeriformes	2000 mg/L drinking water × 14d (juice covers bitter taste of drug)			27
Doxycycline	Humboldt penguins *(Spheniscus humboldti)*	20 mg/kg PO q12h × 10d			7
	Sphenisciformes	50 mg or 100 mg (based on penguin weight) PO q24h × 2–3 weeks		*Plasmodium*	178
Mefloquine	Strigiformes	30 mg/kg PO at times 0, 12, 24, and 48 h			114
	Falconiformes	30 mg/kg PO at times 0, 12, 24, 48, and 72 h, then weekly			29
	Strigiformes (eastern screech owl [*Otus asio*])	30 mg/kg PO q12h × 1d, then q24h × 1–2d		*Plasmodium* ± *Hemoproteus*	179
	Raptors	30 mg/kg PO q12h × 1d, then q24h × 2d, then q7d × up to 6 months		Long-term administration	7
	Raptors	50 mg/kg PO q24h × 7d			27
Primaquine				Dosage based on amount of active base, not total tablet weight	7
	Most species	0.3–1.0 mg/kg PO q24h × 3–10d	10–25 mg/kg PO loading dose, then 5–15 mg/kg at 6, 18, and 24 h	*Plasmodium* and other hemoparasites *(Atoxoplasma, Sarcocystis)*	7,16

TABLE 9.7

Select Hemoparasite Treatment Protocols—cont'd

Drug	Species	Dosage	Combination Therapy	Comments	References
	Broiler chicks *(Gallus gallus domesticus)*	0.50 mg/kg PO q24h × 5d		The lowest mortality rate (4.76%) for broiler chicks experimentally infected with *Plasmodium gallinaceum*	170
	Falconiformes	0.75 mg/kg PO q24h × 5d		*Hemoproteus tinnunculi*	180
	Sphenisciformes	26.3 mg tablet PO q24h × 10d	Chloroquine 10 mg/kg PO loading dose, then 5 mg/kg PO q24h × 9d	*Plasmodium*	178
Pyrimethamine	Raptors	0.5 mg/kg PO q12h × 14–28d		*Leucocytozoon*	8
	Psittaciformes, Passeriformes	0.5 mg/kg PO q12h × 30d		*Leucocytozoon (Atoxoplasma, Sarcocystis, Toxoplasma)*	16
Quinacrine	Canaries (*Serinus canaria* f. *domestica*)	0.24 mg/kg PO q12h		*Plasmodium* (mepacrine)	16
	Most species	5–10 mg/kg PO q24h × 7–10d		*Plasmodium*	8
	Psittaciformes, Passeriformes	5–10 mg/kg PO q24h × 10d		*Plasmodium (Atoxoplasma)*	16
	Most species	7.5 mg/kg PO q24h × 7–10d		*Atoxoplasma*	18
Tafenoquine	Chickens *(Gallus gallus domesticus)*	30 mg/kg IM q24h × 5d		Unable to clear the blood-stage *Plasmodium* parasites but did reduce mortality in chickens	171
		30 mg/kg IM q24h × 5d	Artesunate 1 mg/kg q24h × 5d	Completely cleared experimental *P. gallinaceum* blood stages and sped up gametocyte clearance	171

d, Day(s); *h*, hour(s); *IM*, intramuscular; *IV*, intravenous; *PO*, per os.

mefloquine, and halofantrine, as well as aminoquinoline derivatives like chloroquine, amodiaquine, primaquine, lumefantrine, piperaquine, and tafenoquine.[125,175,177]

The use of primaquine and chloroquine together has been a popular treatment choice for avian patients.[89,165] This regimen is based on the combined effect of chloroquine on blood-stage parasites and of primaquine on tissue stages of *Plasmodium*. Grilo et al. have summarized a variety of protocols used in captive penguin colonies, including a regimen recommended by the Association of Zoos and Aquariums Penguin Taxon Advisory Group consisting of primaquine (1.25 mg/kg PO q24h) and chloroquine (10 mg/kg PO q24h) over 10 to 14 days, then continuation with chloroquine (5 mg/kg q12h PO) over 3 days.[89] Some institutions stop therapy at this point and others continue chloroquine (5 mg/kg PO q24h) concurrently with primaquine.[89] A treatment regimen described in two snowy owls consisted of chloroquine (10 mg/kg loading dose, then 5 mg/kg PO q24h for three doses, then 5 mg/kg PO q7d for four doses) and primaquine (1 mg/kg PO q7d for five doses).[157] Blood smears evaluated at 3 and 4 weeks after initiation of treatment were negative for all hemoparasites.[157] This was followed by an additional round of chloroquine (10 mg/kg PO q24h for 10 doses) and primaquine (1 mg/kg PO q24h for 10 doses) to ensure parasitemia had been eliminated.[157] Tafenoquine is active against both liver and blood stages of malaria.[125] Tafenoquine (30 mg/kg IM for 5 days) was highly effective against gametocytes and completely blocked the transmission of *P. gallinaceum* to the mosquito vector.[171] A 5-day low-dose treatment of tafenoquine (3–10 mg/kg) was unable to clear blood-stage parasites, but did reduce the mortality rate.[171] It should also be noted that higher doses of tafenoquine (60 or 100 mg/kg IM once) caused death in chickens.[171]

Mefloquine can be used for treatment or prophylaxis (see Prophylaxis) of malaria.[125] Treatment of an eastern screech owl *(Otus asio)* with mefloquine (30 mg/kg PO) repeated after 12, 24, and 48 hours proved successful in eliminating *P. subpraecox* from blood smears and resolving clinical signs consistent with malaria.[179]

Antifolate compounds. Antifolate combination drugs include dihydrofolate reductase inhibitors, such as pyrimethamine, proguanil, and chlorproguanil. The inhibitory effect that these drugs have on folic acid synthesis increases when used in combination with sulfa drugs.[125,169]

Artemisinin compounds. Artemisinin and its derivatives, dihydroartemisinin, arteether, artemether, and artesunate, are the current first-line antimalarials for activity against the blood stage and gametocytes of *Plasmodium*.[125,171] Artemisinin compounds are exceptionally fast-acting against asexual, intraerythrocytic *Plasmodium*, effecting up to 10,000-fold reductions in parasite burden every 48 hours.[125,181] A disadvantage of artemisinin compounds is their very short in vivo half-lives,

approximately 1 hour in humans.[181] Therefore artemisinins are most successful in nonsevere malaria when used in combination with a second drug, such as mefloquine or sulfadoxine-pyrimethamine.[125,127,181] A 5-day combination of tafenoquine (30 mg/kg IM q24h) and low-dose artesunate (1 mg/kg q24h) completely cleared experimental *P. gallinaceum* blood stages and sped up gametocyte clearance from chickens, suggesting synergistic action between the two drugs.[171] Recent studies demonstrated that a 7-day regimen of artesunate (10 mg/kg) cleared the blood-stage parasites and gametocytes and also blocked transmission of the parasite to mosquitoes.[182,183]

Atovaquone. Atovaquone has a unique mode of action that involves selective action against mitochondrial electron transport in the parasite. The mitochondrial toxicity of atovaquone is enhanced by proguanil hydrochloride (chloroguanide).[125] The combination of atovaquone and proguanil (Malarone, GlaxoSmithKline) is a fixed-dose combination containing 250 mg of atovaquone and 100 mg of proguanil in a single tablet.[125,162] Malarone clears blood-borne forms of *Plasmodium*.[125,162,168,169] Malarone was highly efficient for clearing blood stages of *P. relictum* in experimentally infected greenfinches *(Carduelis chloris)*.[168]

Antimicrobials. Tetracyclines, clindamycin, azithromycin, and fluoroquinolones have demonstrated variable degrees of schizontocidal activity.[125–129] Antibacterial protein synthesis inhibitors, like doxycycline and azithromycin, exert antimalarial activity by inhibiting function of the apicoplast.[129,184] This action is necessarily slow, presumably due to the ability of parasites to survive most of their life cycle without functional apicoplasts.[184] The slow-acting, sometimes weak activity of these antimicrobials against *Plasmodium* means that they are recommended for treatment only in combination with a fast-acting schizonticide like quinine, or as a single agent for prophylaxis (see Prophylaxis).[125,185] A mohua *(Mohoua ochrocephala)* with avian malaria survived for a year following treatment with chloroquine and doxycycline.[186]

To overcome the challenges caused by the spread of antimalarial drug resistance (see Prophylaxis), new antimicrobials that are being explored include eravacycline, tigecycline, mirincamycin, fusidic acid, thiostrepton, and nocathiacin.[127,187]

Ionophores. In a 2015 study, the monovalent ionophores, salinomycin, monensin, and nigericin, efficiently killed both asexual forms and gametocytes of *P. falciparum*.[188]

Prophylaxis. The effectiveness of treatment for avian malaria usually decreases dramatically by the time the first clinical signs appear.[89,156] Therefore continuous or seasonal prophylactic treatment is widely used at some institutions with susceptible collections, like penguins, to prevent the development of and/or reduce the severity of clinical signs.[89] Protocols often rely on daily to weekly administration of prophylactic antimalarial drugs, such as primaquine, pyrimethamine, or proguanil (Table 9.8).[6,89,169] Doxycycline is also considered a highly effective antimalarial prophylaxis against *P. falciparum*, the cause of human malaria,[126,187] particularly in multidrug resistance zones.[128,187] When drug prophylaxis is employed, it is usually done on an annual basis since this practice can prevent some individuals from developing natural immunity to *Plasmodium* spp.[89]

Oral supplementation with folic acid can be administered when prophylaxis includes an antifolate compound.[89] Caution must also be taken when a folic acid inhibitor is used in laying females during the reproductive season.[89] Discontinuation of pyrimethamine treatment has been recommended 10 days before breeding/laying season.[89]

ECTOPARASITICIDES

A variety of drugs can be used to control or destroy ectoparasites in birds (Table 9.9). Broad-spectrum ectoparasiticides include MLs, organophosphates and carbamates (OP/C), phenylprazoles, pyrethroids, isoxazolines, and spinosyns.[4,191–193] Although MLs and isoxazolines can also be administered systemically, most ectoparasiticides are administered topically as sprays, mists or fogs, and dusts.[4,191,192] On most large-scale production farms in the United States, pesticides are sprayed from underneath as birds sit within suspended wire or "battery cages."[192,194] These pesticides are most effective when they are applied where mites are concentrated to feathers in the vent region.[192,194] The pesticides are sprayed at high pressure because it can be difficult to penetrate the plumage in the vent region.[192] It can also be difficult to achieve acaricidal levels in all the hard-to-reach areas that mites may hide.[4,192] Topical application of ectoparasiticides tends to be a bit less aggressive for individual birds. Powders or dusts can be applied with a shaker can or hand duster; however, the dust can be difficult to apply and is easily inhaled. Another technique involves placing the bird in a canvas bag, with the head sticking out. The bag (and bird) can then be gently manipulated to spread the dust into the plumage and around the vent. Following application of any insecticide to a cage or room, the environment should be vacated and thoroughly cleaned.[39] Nests and perches should also be replaced.[39]

Organophosphates/Carbamates

Organophosphates (OPs) are applied to housing or individual birds to treat infestations of ectoparasites, like ticks, mites, and lice (Table 9.9).[52] OP are potent acetylcholinesterase inhibitors that cause acetylcholine to accumulate at the autonomic ganglia, neuromuscular junction, and central nervous system, which in turn leads to parasite paralysis and death.[6,52] OP can be absorbed through the skin as well as through mucous membranes of the eyes, respiratory tract, and digestive system.[52] Following absorption, OP is extensively metabolized and distributed widely to tissues.[52] OP can persist on the surface for reasonable periods even though residues within animal tissues are relatively short-lived.[6] Phoxim, malathion, and dichlorvos are examples of OP. The closely related carbamates (C), such as carbaryl and propoxur, behave similarly.[52]

Although veterinary OP/C products are considered relatively safe in birds, their potential impact on the environment and nontarget species like honey bees is extremely serious.[203] Therefore the use of many OP/C insecticides has narrowed considerably in recent years. Carbaryl and many other OP/C are no longer approved for use in poultry. If a product like carbaryl is administered to a companion bird, it should be used indoors, with masks and gloves and the handlers should clean their shoes before going outside.

Pyrethrins

Pyrethrins are a group of insecticides derived from the pyrethrum flower *(Chrysanthemum cinerariaefolium)*. Synthetic forms

TABLE 9.8

Avian Malaria Prophylaxis Protocols (See Box 9.10)

Drug	Species	Dosage	Combination Therapy	Comments	References
Chloroquine	Most species	10 mg/kg PO q7d	Primaquine 1 mg/kg PO q7d	Preventive regimen for birds recovering from *Plasmodium* infection, once bird is stable	7,8
	Penguins	15 mg PO q24h	Primaquine 1–4 mg/kg PO q24h to SIW (26.3 mg most common dose)	Primaquine was most commonly dosed SIW	178
Doxycycline	Penguins	100 mg PO q24h		One institution reported administering the medication for 14 days and then resting the birds for 7 days	178
Mefloquine	African penguins *(Spheniscus demersus)*	30 mg/kg PO SIW		Regurgitation has been noticed in birds treated for several consecutive days	89
Primaquine	African and Humboldt penguins *(Spheniscus demersus, Spheniscus humboldti)*	0.75 mg/kg PO SIW or twice in a week		During vector season or year round depending on location	189
	African and Humboldt penguins *(Spheniscus demersus, Spheniscus humboldti)*	1.25 mg/kg PO SID		During vector season or year round depending on location	189,190
	Penguins	1.25 mg/kg PO q24h during vector season		During vector season or year round depending on location	190
	Penguins	1–4 mg/kg PO SIW		26.3 mg was the most common dose reported	178
Sulfonamides	Penguins	Sulfadiazine 125 mg PO q48–72h	Pyrimethamine 4 mg PO q48–72h	Regimen most commonly dosed q48h, alternatively q72h Supplement folic acid 0.4 mg PO	178

h, Hour(s); *PO*, per os; *q*, every; *SIW*, once weekly.

based on this naturally occurring compound are called pyrethroids.[52] Permethrin is the primary pyrethroid used in poultry.[52] Additional pyrethroids include deltamethrin, cypermethrin, and fenvalerate.[52] These drugs act by altering the activity of the sodium ion channels of nerves, leading to insect paralysis and ultimately death. All pyrethroids are lipophilic, which helps them to serve as contact insecticides.[52] Pyrethroids also have a repellent effect, persisting on plumage and skin but not in tissue.[6] Therefore these ectoparasiticides are of particular value against parasites that feed on the skin surface, like lice, some mites, fleas, and nuisance flies (Table 9.9).[52]

Pyrethrins and pyrethroids are among the least toxic of the insecticides and are not as well absorbed through skin as other insecticidal compounds.[52] The death of two zebra finches *(Taeniopygia guttata)* was attributed to a pyrethrin-based spray (0.16 mg/100 mL water).[199] Spray was applied twice to a flock of approximately 350 birds in situ, with a 2-week interval between treatments. Examinations carried out a few weeks later demonstrated incomplete mite eradication, and four additional treatments at biweekly intervals were performed by individually spraying each bird that lived outside a nest box.[199] Hatchlings were sprayed within the nest.[199] Pyrethroids are extremely toxic to fish and aquatic invertebrates,[6] which should be a consideration if birds are housed in mixed aquatic zoo exhibits.

Phenylpyrazoles

Phenylpyrazole insecticides are a class of broad-spectrum compounds that act by interfering with activation of insect-specific GABA receptors leading to neuronal hyperexcitation of the insect nervous system and death.[6,204,213] Fipronil (Frontline, Boehringer Ingelheim, Duluth, GA) is a phenylpyrazole that is used for the treatment and control of tick infestations as well as some mites (Table 9.9).[6]

Fipronil is not approved for use in any food animal species because it is highly lipophilic and concentrates within eggs.[6,60,197,204] It is important to use this product with caution in any novel avian species because metabolites of fipronil have shown higher toxicity to birds than the parent compound.[25] This toxic response appears to be quite variable and has appeared in different avian families.[25,213,214]

Macrocyclic Lactones

MLs, such as IVM, doramectin, abamectin, and selamectin, are important for ectoparasite control in birds,[1] showing very high potency against various mites (Table 9.9).[4] IVM is also of

TABLE 9.9

Ectoparasiticides for Use in Birds

	Pyrethrins Permethrins Pyrethroids	Organophosphates/ Carbamates	Macrocyclic Lactones	Isoxazolines	Phenylprazole	Spinosyn
Examples	Bifenthrin Cypermethrin Deltamethrin Permethrin	Carbaryl (C) Propoxur (C) Dichlorvos (OP) Phoxim (OP) Tetrachlorvinphos (OP)	Doramectin (DOR) Eprinomectin (EPR) Ivermectin (IVM) Milbemycin oxime Moxidectin (MOX) Selamectin (SEL)	Afoxolaner Fluralaner Sarolaner	Fipronil	Spinosad
Clinical applications	Mites,[a] lice, fleas, ticks, nuisance flies[52,60]	Broad-spectrum activity against insects, ticks, arachnids, surface mites, fleas, flies, and lice[3,195]	Broad-spectrum anthelmintic and insecticidal activity, including mites, particularly burrowing mites (i.e., *Knemidokoptes*)[3,196]	Mites, lice[197]	Broad-spectrum insecticidal activity, including mites, lice, ticks, flies[6,39]	Mites
Administration	Two topical applications are necessary at an interval of 10d; more applications[b] may be required Lightly dust plumage, spray until runoff, or dip with dilution[7] Permethrin-impregnated plastic strips[c] are marketed for ectoparasite control in caged or cage-free commercial and backyard poultry flocks[198]	Two topical applications at an interval of 10d Lightly dust plumage and/or paint, spray, and/or dust perches, nest box, and/or premises[7] Remove treated litter after 24 hours[7,18]	Two systemic or topical doses 10–14d apart[d] IVM or DOR can be diluted 1:10 with propylene glycol or sesame oil, respectively[39] Apply topical IVM to the skin in the axillary and inguinal regions, or on the bare skin dorsolateral to the thoracic inlet (at the site of jugular vein blood collection)[199,200]		Apply two to three squirts using a pad to skin at base of neck, tail base, axillary and/or inguinal area. Avoid plumage. Do not soak bird[8,25,16,201] Use spray formulation and not spot-on preparation[7]	Topical spray (1.94 or 3.88 g/L) [202] Residual activity was maintained throughout the course of a 28d post-spray study period, with a peak in product efficacy seen 14d after spraying.[202]
Contraindications, precautions	Topical application can be stressful for birds[199] Hang dichlorvos strips out of reach of the birds[39]	Topical application can be stressful for birds and a potential health risk for humans exposed to pesticides and residues[4] Veterinary products are considered relatively safe in birds; however, carbaryl and many other OP/C are no longer approved for use in poultry because of their potential impact on the environment and nontarget species[203]	Due to high residue levels found in eggs, IVM should be avoided in laying hens until establishment of reliable withdrawal periods[5] Propylene glycol-based IVM can cause severe respiratory tract irritation if aspirated during oral administration[201]	—	Not approved for use in any food animal species. High residue levels within eggs[204] Alcohol may create dry, brittle feathers[8,16] Beware of inhalation or dermal absorption of the alcohol base Do not exceed 7.5 mg/kg[8]	—

		If a product like carbaryl is administered to a companion bird, it should be used indoors, with masks and gloves and the handlers should clean their shoes before going outside.				
Potential adverse effects or toxicity	The death of two zebra finches *(Taeniopygia guttata)* in a flock of ~350 birds was attributed to spraying[199]	—	No adverse effects have been reported in chickens *(Gallus gallus domesticus)*[5]; however, there are sporadic, reports of IVM toxicosis in birds, including finches (presumably due to propylene glycol)[15] No evidence of IVM toxicity was seen in budgerigars *(Melopsittacus undulatus)* treated with 5–10 times the recommended dose[200] No adverse effects in Gouldian finches *(Erythrura gouldiae)* given SEL up to 92 mg/kg[205] Risk of IVM toxicity may be higher if the drug is injected, particularly via the IM route[16]	No adverse effects have been described in poultry treated with flural aner[191,197,206,207] In fact, a 3-fold safety margin has been demonstrated with fluralaner in chickens[191,206]	One fipronil metabolite has higher toxicity to birds than the parent compound; therefore this product should be used with caution because it shows variable toxicity in different avian families[25] Use with caution in raptors, pigeons, passerine birds[7] Reported toxic effects include death, tremors, seizure activity, anemia, leukopenia, impaired immune function, reduced growth and reproductive success[7,208–210]	Application of spinosad in vivo had no effect on hen body weight or egg production parameters (number and weight)[202]
Dosage in most species			IVM 0.2–0.4 mg/kg PO, topical, SC, IM once; repeat in 10–14d prn (see Potential adverse effects or toxicity, earlier)[8] See Table 9.1 for additional doses		7.5 mg/kg topically once, repeat in 30d as needed in most birds, including raptors, pigeons, passerine birds[8,18,25,38]	
Galliformes	Cypermethrin 60–120 mg per chicken[30]		EPR, MOX, or IVM 5.0 mg/kg PO (chicks, *Dermanyssus*)[4] SEL 20 mg/kg topical (helmeted guineafowl [*Numida meleagris*])[35]	Fluralaner 0.5 mg/kg PO given twice at 7d intervals administered in the drinking water or by gavage[e] was shown to be safe and to have a 0d egg withdrawal period following treatment[4,191,197,206] Afoxolaner 2.5 mg/kg PO once to pheasants, chachalacas[211]		
Passeriformes	—	—	—	—	3 mg/kg topically once[16]	—

Continued

TABLE 9.9

Ectoparasiticides for Use in Birds—cont'd

	Pyrethrins Permethrins Pyrethroids	Organophosphates/ Carbamates	Macrocyclic Lactones	Isoxazolines	Phenylprazole	Spinosyn
Psittaciformes	—	—	MOX 1 mg/bird topically once, repeat q10d × 2 treatments prn (budgerigars [*Melopsittacus undulatus*], *Knemidokoptes*)[7] SEL 23 mg/kg topically, repeat in 3–4 weeks (budgerigars, *Knemidokoptes*)[7,45]	—	3 mg/kg topically once[16]	—
Raptors	—	—	SEL 23 mg/kg topically, repeat in 4 weeks (golden eagle [*Aquila chrysaetos*], *Micnemidocoptes*)[212]	—	—	—

[a]Pyrethrin-based spray (0.16 mg/100 mL water) was applied twice to zebra finches *(Taeniopygia guttata)* in situ, with a 2-week interval between treatments. However, feather examinations carried out a few weeks later demonstrated incomplete mite eradication, and four additional treatments at biweekly intervals were performed by individually spraying each bird that lived outside a nest box. Hatchlings were sprayed within the nest.[199]

[b]To eradicate feather mites from a flock of zebra finches *(T. guttata)*, multiple treatments[a] using a pyrethrin spray were effective. Topical IVM was later found to be more efficacious, better tolerated by birds, and less labor-intensive.[199]

[c]Resistance is a growing problem. In a recent study, permethrin strips had no effect on poultry mite populations.[198]

[d]A "drop on" liquid ivermectin solution containing 0.1% is commercially available in Australia (1000 μg/mL) (Avimec, Vetafarm Pty Ltd, Australia) and 0.01% (100 μg/mL) w/v ivermectin solution were evaluated in budgerigars *(Melopsittacus undulatus)* with *Knemidokoptes*. One drop (0.05 mL) was effective for birds <30 g and two drops (0.1 mL) for birds 30 to 100 g.[200]

[e]Fluralaner (10 mg/mL) had no effect on water consumption at up to 5× the recommended dose.[206,207]

C, Carbamates; *d*, day(s); *DOR*, doramectin; *EPR*, eprinomectin; *IM*, intramuscular; *IVM*, ivermectin; *MOX*, moxidectin; *OP*, organophosphates; *PO*, per os; *prn*, "*pro re nata*" or as needed; *SC*, subcutaneous; *SEL*, selamectin.

proven benefit in pediculosis (see Macrocyclic Lactones for precautions with respect to the use of MLs in poultry).[3]

There are sporadic, anecdotal reports of acute, potentially fatal IVM toxicosis in birds, particularly small passerine birds like finches.[18] This effect is believed to be an acute anaphylactic reaction to the propylene glycol in IVM,[39] although there was no evidence of toxicity in budgerigars treated with five to ten times the recommended dose.[200] Some clinicians avoid injecting IVM into birds altogether, particularly small birds, and instead use doramectin, which contains sesame oil.[39,196]

Isoxazolines

Isoxazolines are a class of insecticides that paralyze and kill parasites by selectively targeting noncompetitive GABA receptors, binding to chloride channels within insect nerve and muscle cells.[6] Isoxazoline compounds include fluralaner, sarolaner, lotilaner, and afoxolaner (Table 9.9).[6] Fluralaner in drinking water has been used to control mites and lice infestations in the chicken.[197,211] Afoxolaner has been successfully used for chewing lice *(Goniodes pavonis)* infestations in pheasants (*Chrysolophus* spp., *Lophura* spp., *Phasianus colchicus*, *Syrmaticus reevesii*), West Mexican chachalacas *(Ortalis poliocephala)*, and peacocks *(Pavo cristatus)*.[211,215]

This class of drugs has been used in poultry without a negative impact on health or production and no adverse effects were observed in pheasants, chachalacas, or peacocks.[211,215] Nevertheless, products containing isoxazolines have been associated with adverse neurologic reactions, including muscle tremors, ataxia, and seizures in some dogs and cats.[216]

Spinosyns

Spinosyns quickly kill insects using a novel mode of action that alters the function of nicotinic and GABA-gated ion channels, causing rapid excitation of the insect nervous system, involuntary muscle contractions, tremors, paralysis, and death.[217] Spinosad is a fermentation product derived from the spinosyn-producing soil actinomycete *Saccharopolyspora spinosa* that has demonstrated acaricidal activity against *Dermanyssus gallinae* (see Therapy for Specific Ectoparasite Concerns) (Table 9.9).[192,202,217] *D. gallinae* showed high mortality by 2.5 hours posttreatment.[192]

Therapy for Specific Ectoparasite Concerns

Dermanyssus gallinae

Dermanyssus gallinae, the poultry red mite (PRM) or roost mite, is a worldwide pest of chickens and other gallinaceous birds.[37,218] The PRM is also seen in passerine birds and sporadically in other exotic birds. This blood-sucking ectoparasite visits its host only during the night.[25,37] During the day, mites hide in the environment of their host, within nest material as well as cracks and crevices in the immediate surroundings.[37,218] Although *D. gallinae* favors poultry and other birds, this mite will feed on blood from other animals, including humans.[218] PRMs usually feed every 2 to 4 days. They are very resistant to desiccation and can survive up to 8 months without feeding.[4] Seasonal activity of mites is mainly temperature-driven.[218] The optimum temperature for egg production is 25°C to 30°C (77°F–86°F) and its best survival rate is observed at a relative humidity of 70% to 90%.[218] Temperatures below −20°C (−4°F) and above 45°C (113°F) are considered lethal.[218]

Treatment of PRM requires treating the environment as well as the birds. The premises, including the nesting material and all equipment associated with birds, should be sprayed with an insecticide, sometimes multiple times.[39] Regular cleaning of housing and maintenance of good hygiene practices is also essential.[218] Cracks and crevices should also be sealed.

Treatment of birds can be administered topically or systemically. Multiple treatments are often required. MLs, such as IVM, eprinomectin, and moxidectin, are an excellent systemic treatment choice for *D. gallinae* in individual birds.[1,4,39,219] In a study evaluating the efficacy of these three MLs administrated orally to chicks at 5.0 mg/kg, all three drugs significantly increased the mortality of *D. gallinae*.[4] Among the three MLs, eprinomectin exhibited the highest performance against PRMs.[4] Fluralaner in drinking water has also been used to control PRM infestations in the chicken and this product is currently approved for use in poultry.[4,197,202,206]

Historically, chemical control of mites in large poultry flocks has been largely dependent on the use of topical acaricides, including OP/C, amidines, pyrethroids, isoxazolines, and spinosyns,[4,191–193,197,202,206] however, legislation frequently (and appropriately) constrains the products that can now be used to prevent the exposure of eggs, poultry, and humans to drug residues.[218] Spinosad spray is currently approved for use in poultry, and a single application of spinosad has demonstrated acaricidal activity against *D. gallinae* when applied at either 1.94 or 3.88 g/L.[202] Residual toxicity to *D. gallinae* was maintained for 28 days post–spinosad spray with a peak in product efficacy 14 days post-spray.[202]

In individual companion birds, for which extra-label drug use is legal, a pyrethroid/permethrin dust, carbaryl powder or spray (5 g–85% carbaryl powder/L water) can be applied.[39] Fipronil is also effective against PRM and well tolerated in many avian patients (see Organophosphates/Carbamates for precautions with respect to use of carbaryls; see Phenylpyrazoles for precautions with regard to fipronil).[39]

Ornithonyssus sylviarum

The northern fowl mite (*Ornithonyssus sylviarum*) is one of the most common and most damaging ectoparasites of poultry.[194] Over 70 species of wild and exotic birds can also serve as hosts of this blood-sucking mite.[192,194] Box 9.9 and Figure 9.1 provide an overview of the life cycle of this parasite. Heavy infestations can be associated with reduced egg production, blackened feathers due to the accumulation of dried blood, as well as scabbed, cracked skin around the vent.[195]

This mite spends its entire life on the bird, and therefore only the bird, and not the enclosure, is treated for *Ornithonyssus*.[195] Permethrin is the primary pyrethroid used to treat infestations in poultry.[52] An ML, like IVM, is also a good choice in many avian patients.[60] Northern fowl mites are also highly sensitive to fluralaner and moderately sensitive to phoxim and propoxur.[192]

Knemidokoptes mites

Knemidokoptes spp., also known as the scaly leg and face mite, is most commonly reported in passerine birds, such as canaries and finches, and psittacine birds, like budgerigars. This common burrowing mite spends its entire life cycle on the

BOX 9.9 LIFE CYCLE OF THE NORTHERN FOWL MITE *(ORNITHONYSSUS SYLVIARUM)*[192]

- Eggs hatch into sessile, nonfeeding larvae that last about a day.
- This is followed by an active, blood-feeding protonymph stage and a nonfeeding deutonymph stage, before molting into active, blood-feeding adults.
- The entire life cycle is completed in approximately 5–12 days.
- Mites are able to survive off-host for at least 1–3 weeks, perhaps months, particularly at high temperatures.



FIGURE 9.1 Life stages of the northern fowl mite *(Ornithonyssus sylviarum)*: egg *(e)*, egg with developing larva *(e-l)*, larvae *(l)*, protonymph *(p)*, and adult female *(a)*. Eggs mature to adults in as few as 5 days. Adults can persist in the environment for weeks and perhaps months. (From Murillo AC, Mullens BA: A review of the biology, ecology, and control of the northern fowl mite, *Ornithonyssus sylviarum* (Acari: Macronyssidae), *Vet Parasitol* 246:30–37, 2017.[194])

bird and is transmitted from bird to bird via prolonged, direct contact.[195] Lesions appear as hyperkeratotic, honeycomb encrustations on featherless skin.[195] Older birds are most commonly affected.[195] Diagnosis is based on the typical pitted appearance of the legs and face of the bird and through microscopic visualization of the mite after a tape prep or scrape of the affected area.

Treatment in many avian species is with IVM (0.2 mg/kg PO, SC, or topically) at 1- to 2-week intervals, for often three to four treatments. In poultry that cannot be treated with IVM, individual birds can be treated by directly applying an oil-based product, such as mineral oil or petroleum jelly, to the entire affected area once daily for at least 2 weeks.[195] After a few days, the dead scales will be softened and can be gently rubbed or scraped from the bird.[195] For larger poultry groups, dusts and sprays (described earlier) can be directly applied.[195]

Sternostoma tracheacolum

Sternostoma tracheacolum is found worldwide in both captive and wild birds.[220] This species can be found throughout the avian respiratory tract, from the trachea to the air sacs.[220] Due to the difficulty of detecting mites, infestation of *S. tracheacolum* is often diagnosed at necropsy after the death of some avian hosts has occurred.[220]

Individual birds can be treated with IVM or doramectin.[39] There is also an intriguing, albeit dated, case report in which a flock of canaries *(Serinus canaria)* with *S. tracheacolum* were exposed to 5% malathion powder pumped into the cage to create a fairly dense fog for 5 minutes.[221] This treatment was repeated weekly for 4 weeks and it was successful in controlling the mite. In another dated study, Gouldian finches *(Erythrura gouldiae)* infested with *S. tracheacolum* were fed millet coated with olive oil (1 mL olive oil to 50 g millet) and thoroughly mixed with Sevin dust (0.04 g carbaryl to 50 g grain) once a week for 3 weeks.[221] Although birds were euthanized at the end of this 3-week study, no deaths occurred while finches were fed treated seed.[221] And although eradication was not achieved, adequate control was obtained. Within 1 to 2 weeks of treatment, the clinical status of birds was noticeably improved and mature birds again began breeding.[221]

Chewing lice

Avian chewing lice belong to suborders *Amblycera* and *Ischnocera*.[222] These small, dorsoventrally flattened insects feed on fragments of feathers and other epidermal products.[197,222] This behavior degrades plumage quality, creating small holes on feathers reducing thermoregulatory capacity, and increasing feather breakage.[222] Over 4000 species of bird lice have been identified,[222] including the slender pigeon louse *(Columbicola columbae)*, the poultry shaft louse *(Menopon gallinae)*, and the chicken body louse *(Menacanthus stramineus)*. Lice that feed on birds typically spend their entire life on the host.[195]

Pyrethrin sprays or permethrin dust are probably the most common options available for use.[60] Fipronil spray is another option.[60] Lice eggs are resistant to insecticides, and topical treatments must be repeated for optimal results.[195] Systemic drug options include IVM and afoxolaner.[3,196,211]

Hippoboscid flies

Hippoboscidae (flat flies), such as *Ornithomyia avicularia*, are commonly found on birds of prey and may cause significant blood loss in very young birds.[29,162] Flat flies may be removed manually or birds can be dusted with pyrethrin (0.5%–2%), malathion (5%), or carbaryl (0.5%).[29] There are also anecdotal reports of fipronil spray (0.29%) used successfully in pigeons and raptors to remove hippoboscids and reduce black flies and mosquitoes.[25]

Myiasis

Myiasis or fly strike is the infestation of healthy or necrotic tissue by fly larvae.[223] Traumatic myiasis has been observed in a variety of wild and companion birds. Treatment requires manual removal of maggots from affected tissue and management of the wound to promote healing.[223] Birds recovering from myiasis should also be kept indoors and protected from flies to prevent reinfestation of the healing wound.[223]

Systemic IVM is frequently used for management of fly strike. The successful use of an IVM sustained-release varnish,

with or without concurrent injectable IVM, has also been described for severely affected patients that would have been stressed by multiple handling events.[224] There are also anecdotal reports on extra-label use of the neonicotinoid, nitenpyram, in a variety of avian patients. Nitenpyram use has been described for fly larvae as well as sticktight fleas *(Echidnophaga gallinacea)* in poultry. Neonicotinoids work by interfering with neural transmission in the central nervous system.[213] They bind to the nicotinic acetylcholine receptors in the postsynaptic neuron, acting as false neurotransmitters that cause continuous activation of the receptor and neurotoxicity in the parasite.[213] Neonicotinoids have greater affinity for, and thus bind more strongly to, insect receptors.[213] Nitenpyram is administered either by mouth or as a topical formulation that can be created by crushing and suspending the tablet in water. Neonicotinoids have demonstrated outstanding potency and they are generally considered to have low toxicity in mammals, birds, and fish.[225] However, more recently, this class of drugs has been shown to have direct negative impacts on wild birds in North America and Europe.[226]

Ticks

Ticks that will feed on birds include *Hyalomma* spp., *Ixodes* spp., *Haemaphysalis* spp., *Amblyomma* spp., and *Argas* spp.[227–231] Ixodid ticks are most commonly found on birds in Europe, particularly passerine birds.[228,229] Most ticks on North American birds are neotropical species with host ranges that involve Central and South America.[231] Birds are most frequently infested by tick larvae and nymphs and much less commonly by adult forms.[230,232] During the nonparasitic phase of their life cycle, ticks reside on the soil surface and in the lower parts of plants, where they have the greatest chance of finding a host.[230] Therefore ground-foraging birds tend to be more heavily infested with ticks,[230,233] particularly orders Galliformes (Phasianidae) and Passeriformes (Alaudidae and Corvidae).[230,233] Although in a survey of 46 gyrfalcon *(Falco rusticolus)* carcasses from Iceland, 20% of first-year birds and second-year birds harbored the tick *Ixodes caledonicus*.[234]

The fowl tick, *Argas persicus*, is a parasite of poultry and wild birds in tropical and subtropical regions around the world.[235] Fowl ticks are rare pests of commercial poultry, but they are likely to be seen in crowded, backyard poultry houses in warm, dry climates.[236,237] Nymphs and adults feed at night for 15 to 30 minutes at a time,[237] and fowl ticks are an important cause of anemia.[236] Additional signs can include a marked reduction in egg production, weight loss, lethargy, and even tick paralysis (see Tick Paralysis).[236,237]

All fowl tick stages may be found hiding in cracks and crevices during the day.[237] After the environment is thoroughly cleaned, all surfaces, including walls, ceilings, cracks, and crevices, should be treated thoroughly.[237] Products that have been applied to the premises include carbaryl, coumaphos, malathion, permethrin, stirofos, or a mixture of stirofos and dichlorvos.[237] A study also revealed that abamectin spray (0.5 mL/L) was sufficient to eradicate *A. persicus* population.[238] After cleaning and treating, cracks and crevices should also be sealed.[237]

For chemical treatment of the bird, MLs, such as IVM or selamectin, are effective for one-host ticks, like the fowl tick.[6,239] Additional topical treatments have included pyrethrin/permethrin and carbaryl.[239] Peracetic acid (0.5%) dips were also a highly effective treatment for *A. persicus*, resulting in 100% mortality of ticks after 2 minutes.[235] Adverse effects were not observed in chickens with this technique.[235] By contrast, respiratory signs, such as temporary coughing and sneezing, as well as ocular inflammation were observed in chickens dipped in deltamethrin.[235] Fipronil, afoxolaner, and fluralaner are also used for tick infestations in small animals and should prove effective in birds based on their proven acaricidal activity.[6]

Tick paralysis

Tick paralysis is a neurotoxin-mediated flaccid paralysis syndrome that has been described in wild birds and gallinaceous birds that has been associated with heavy infestations of *Ixodes* spp. as well as at least 10 species of argasid ticks.[239–241] Birds exhibiting signs of tick paralysis should be treated as a medical emergency, and tick removal should be performed as soon as possible to halt exposure to salivary toxins.[65,240] Removal is best accomplished using fine forceps applied close to the skin with gentle, steady, upward and outward traction, taking care to avoid leaving mouth parts embedded in the wound.[242] Care is otherwise supportive but may require intubation for respiratory support in severe cases. Rapid resolution of clinical signs after tick removal should be observed.[239,241] In a survey of 23 wild birds, most birds (74%) recovered with supportive care within 3 to 7 days.[239–241] After removal of more than 350 adult and larval ticks, a nestling bald eagle *(Haliaeetus leucocephalus)* recovered from paresis within 36 hours and was released after 3 weeks.[239]

DRUG RESISTANCE

Resistance is the inevitable result of natural selection that occurs in response to continuous exposure to antiparasitic drugs.[125,243,244] Selection pressures enhance the fitness of parasites and promote the development of heritable traits that allow the organism to survive and/or multiply despite the administration of a drug in doses equal to or higher than those usually recommended, but within the limits of tolerance of the host.[80,90,243] Ever-rising levels of resistance have been documented in virtually all antiparasitic drug classes.[243] Although veterinary medicine often describes antiparasitic resistance as an emerging problem, resistance was first reported in the 1950s and by the 1970s, resistance had become widespread in poultry production.[2,77,78] Beginning in the 1950s and continuing through the 1980s, the approach to drug resistance was the introduction of a new class of antiparasitic agents each decade.[243] These highly effective and relatively inexpensive drugs led to parasite control strategies that were based on the frequent use of antiparasitics, which in turn fed a cycle of ever-growing drug resistance.[243] Today, judicious use of antiparasitic agents (Box 9.10) and alternative management methods (see Alternative Control Methods) should be encouraged whenever possible.

Surveillance

The fecal egg count reduction test (FECRT) is a useful and inexpensive method for field diagnosis of resistance.[2,51,74,90] This test is based on counting worm eggs in the feces of animal groups.[246] The mean egg count of a group that has been treated with the antiparasitic is compared with a group

BOX 9.10 RESPONSIBLE USE OF ANTIPARASITIC DRUGS IN BIRDS

- Strive to keep resistant parasites out of the flock by quarantining and testing all new additions.[243]
- Avoid the use of antiparasitic drugs as preventative treatment in healthy, adult birds since this promotes the development of resistance.[91,245]
- Administer the proper dose, optimally based on body weight obtained before treatment. Underdosing exposes parasites to sublethal doses of drug, increasing selection for resistance.[80]
- When managing flocks, use a combination of antiparasitics rather than rotating between drugs.[243] Combination drug therapy can increase drug efficacy and decrease the development of resistance. For example, the combination of nitroimidazoles with quinacrine has been shown to have an additive effect in vitro and in vivo against *Giardia*.[6,80,243]
- When managing flocks, do not employ a drug rotation program. Rotation does virtually nothing to slow the development of resistance, and given the rising incidence of resistance among antiparasitic drugs, it is much more likely to expose the flock to a drug that will not be effective.[243]
- Utilize environmental control measures, such as strict attention to sanitation and hygiene to prevent reinfection.[77,79,246]
  - For instance, coccidia oocysts require 3–5 days to sporulate and become infective, so frequent cleaning is the best preventative measure.[25] Also, some parasites, like cestodes and some nematodes, require intermediate hosts, so prevent birds from foraging where such organisms are found.[25]
  - Take measures to prevent wet litter, which aids the sporulation of oocysts. Adequate ventilation and leak-free watering systems serve to reduce excessive moisture.[78]
- Select nondrug approaches to parasitism whenever possible (see Alternative Control Methods).[243]

of equivalent untreated animals to calculate the egg count reduction due to treatment.[246] The FECRT is labor and cost-intensive and relatively insensitive, since usually only a single dose level is tested.[2] Therefore resistance can only be detected at relatively high levels.[2] Another approach is to use susceptibility assays of field isolates to test multiple antiparasitic agents and concentrations.[2,197] Such testing can provide more detailed information about the level of resistance since it is based on a dose response.[2]

Resistance and Drug Classes

Acaricides

Poultry mite resistance to OP/C as well as pyrethrins/pyrethroids is widespread.[4,192,194]

Nitroimidazoles

Resistance to metronidazole and other nitroimidazole drugs has been reported since 1962 for *T. vaginalis*, but it was not documented until the 1990s for *T. gallinae*.[80,147] Nitroimidazoles are routinely administered to racing pigeons at subtherapeutic doses as a preventive treatment. This prolonged exposure promotes the development of *T. gallinae* isolates that have proven resistant to ronidazole, carnidazole, and metronidazole.[86,91,146] Homing pigeons from around the world are considered a reservoir for nitroimidazole-resistant *T. gallinae*.[86,245] Interestingly, variations in sensitivity to nitroimidazoles have been detected within different isolates obtained from the very same bird.[91,147]

Macrocyclic lactones

Resistance to MLs is commonplace in nematode parasites of small ruminants and is of increasing concern in other animals, including birds.[1] In most animals, resistance first arises to avermectins like IVM and subsequently to moxidectin.[1] In ostrich treated for *Libyostrongylus* spp., an efficacy of 60% was found for IVM (0.2 mg/kg), while ABZ and moxidectin were both 100% effective.[51]

Triazines

The first cases of toltrazuril resistance in coccidia were reported in 1990.[90] In a recent study evaluating the effectiveness of toltrazuril against *Eimeria* spp. in critically endangered black grouse *(Tetrao tetrix)*, FECRT indicated that the coccidia infecting black grouse had natural resistance to toltrazuril.[90]

Anticoccidial drugs

Resistance to ionophores develops slowly because this drug class does not completely suppress parasite development, thereby allowing some immunity to develop in the host.[78] However, long-term, prophylactic use of ionophores in the poultry industry has moderated their effectiveness through selection for resistant strains of coccidia.[75,122] There is also evidence of growing drug resistance of *Eimeria* spp. field strains to sulfonamides and amprolium.[25,81] Other (synthetic chemical) products can be used only for a short time because of rapidly emerging drug resistance.[122]

Antimalarial drugs

Resistance of *Plasmodium* to antimalarial drugs is a major concern. Resistance to chloroquine first emerged in the 1960s, and in fact, most of the currently used antimalarial drugs suffer from increasing drug resistance.[125,129,169,171,184] Most antimalarial drugs, including chloroquine and mefloquine, are included on the World Health Organization's Model List of Essential Medicines.[177] Although it is no longer used as a frontline treatment for malaria, even quinine is included on this list because it may be used in combination with doxycycline for severe human cases where artemisinins are not available.[177]

ALTERNATIVE CONTROL METHODS

Alternatives to traditional antiparasitic drugs have been explored in poultry.[192] These techniques have been employed alone or in combination.[218,247]

Temperature

In production settings, a promising control method applied in parts of Europe combines chemical treatments with heating the hen house to temperatures above 45°C (113°F) between production cycles.[218]

Essential Oils

The repellent and/or toxic effects of plant essential oils (EOs) have been evaluated in various studies.[194,218,247–249] Of 10 EOs tested in vitro against *D. gallinae*, thyme and lavender were the most effective acaricides and the strongest repellents.[249] The

combination of *Eucalyptus globulus* EO and *Beauveria bassiana* (see Fungi) was another effective acaricide for *D. gallinae*.[247] In another study evaluating the effects of EOs on *O. sylviarum*, cade applied at 0.21 mg/cm^2 and thyme applied at 0.21 mg/cm^2 were the most consistent and fast-acting of the EOs in terms of toxicity.[248] One limitation to the use of EOs is that the acaricidal effect is rapid, within 2 hours for northern fowl mites, but short-lived.[247,248]

Plant Products

Herbs or plant extracts can also be used as natural acaricides.[218] In one study, garlic was as effective as metronidazole in inhibiting *T. gallinae* trophozoite growth both in vitro and in vivo.[85,194] Garlic (200 mg/kg PO) for 4 days was considered safe and effective for prophylaxis and therapy in pigeons.[85] *Note:* Garlic is often considered potentially toxic to companion birds.[250] More work is needed and caution should be employed before utilizing this alternative control method.

The botanical insecticide, azadirachtin, is a secondary metabolite present in neem seeds.[194,251] Azadirachtin appears to have direct pathological effects on the arthropod cuticle, acting on contact and as a fumigant.[251] In one study, direct application of a 0.06% solution of azadirachtin to infested vent region feathers and skin using a calibrated hand pump sprayer significantly reduced *O. sylviarum* numbers for 24 days after treatment.[251]

"Self-Treatment"

"Self-treatment" is a useful strategy for birds housed in larger, enriched cages or cage-free systems.[192] This practice takes advantage of the chicken's innate need to dustbathe (Figure 9.2).[192] Although sand alone is too coarse to impact mites, very finely textured materials, like diatomaceous earth (DE), can suppress ectoparasites by compromising the thin layer of cuticular wax that is essential to prevent desiccation and death of small terrestrial arthropods.[192,194,252,253] Mite populations can be kept at low levels when DE-filled dust boxes are offered to a flock.[194] Additional dust box products that have been used to reduce ectoparasite populations include kaolin clay and sulfur.[253]

Sulfur is one of the oldest pesticides used for control of *O. sylviarum*.[194,198,251,253] Sulfur appears to act as a biocide through reduction into hydrogen sulfide, which has a fumigating action.[251] Interestingly, the use of sulfur dust boxes eliminated mites from all hens, including individuals that did not use dust boxes.[253] Residual sulfur also controlled mites until the end of the experiment (up to 9 weeks), even after the dust box was removed.[253] Slow-release "dust bags" can also be used to disperse sulfur.[198] Insecticide-filled bags have long been used for fly control in cattle.[198] Animals bump or rub against the bags, which then apply insecticide.[194] On poultry farms, dust bags can be hung near flock resources, such as nest boxes or food dishes.[194,198] Empirical evaluation has determined bags clipped to the inside front of poultry cages were less effective than bags hung from cage tops.[198]

Predatory Mites

The use of predatory mites is a relatively new option for control of *D. gallinae*.[37,218,254] Predatory mites are natural enemies of PRM that are normally found in the environment and even in poultry houses.[37,254,255] In some regions of the world, predatory mites are mass-reared for agricultural use, and biological control of pests is common practice in greenhouse cultivation of vegetables, fruits, and ornamental plants.[218] The use of predatory mites in animal husbandry is still in its infancy; however, predatory mites have been commercially available for PRM control in the poultry industry since 2010.[37] Predatory mites that have been evaluated for biocontrol of *D. gallinae* include *Hypoaspis aculeifer*, *Androlaelaps casalis*, and *Stratiolaelaps scimitus* (formerly *H. miles*).[218,254,255] There are also anecdotal reports of using *S. scimitus* to control *D. gallinae* and other blood-feeding mites on canaries, pigeons, and reptiles.[37] The usefulness of predatory mites could explain why free-ranging birds reuse old nesting sites or old nest material.[254]

Fungi

Entomopathogenic fungi are capable of infecting and killing insect and mite species.[218,251] Fungal spores germinate on and then penetrate the host cuticle, spreading through the insect's



FIGURE 9.2 "Self-treatment" in chickens takes advantage of the normal fowl need to dustbathe. Chickens are offered a dust box containing sand and an inert dust, such as diatomaceous earth or kaolin clay, or a low-toxicity pesticide, like sulfur. (Photo courtesy Guttorm Flatabø Attribution 2.0 Generic [CC BY 2.0] via Flickr Creative Commons.)

body.[218] After the fungus has killed the mite, it can grow out of the mite cadaver and produce more spores, infesting other PRM.[218] A few entomopathogenic fungi are commercially available.[256] Organisms such as *B. bassiana* and *Metarhizium anisopliae* have shown promise for control of *D. gallinae* and perhaps *O. sylviarum*, although the results have been mixed.[192,194,247,251,256] The acaricidal effect of *B. bassiana* is delayed, but long-lived[247]; however, *B. bassiana* tends to perform poorly at temperatures exceeding 30°C (86°F), which makes this strategy particularly challenging for ectoparasite control in the warm-blooded bird.[251]

Future Considerations

Future areas of development that show promise for alternative control of parasites include the use of a vaccine against PRM or volatile organic compounds, such as aggregation pheromone, sex pheromone, and alarm pheromone, to promote attraction behavior in *D. gallinae*.[218,257] The manipulations of photoperiod and odors are additional control methods that are being investigated.[218,258]

CONCLUSION

A wide array of parasites infect birds. These infections and infestations are important causes of morbidity and mortality in birds worldwide. Antiparasitic drugs are a group of medications used in the management and treatment of parasites. Antiparasitic agents include several classes of drugs that can be broadly grouped as anthelmintics, antiprotozoals, and ectoparasiticides. The control of parasitic helminths relies primarily on the use of a few chemical drug classes that exhibit a broad spectrum of biological activity, such as BZDs and MLs, with diverse structures and mechanisms of action.[52] Antiprotozoal activity is observed in a wide and complex range of chemical compounds that often display a narrow spectrum of activity, such as nitroimidazoles, triazines, and ionophores.[6] Control of the ectoparasites found in birds is largely based on the use of chemicals that are used as insecticides or acaricides. Drug resistance is a growing problem in all antiparasitic drug groups.

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CHAPTER 10

Antiviral Drugs

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INTRODUCTION

While there are many useful treatments for bacterial, fungal, and parasitic infections, antiviral therapies for veterinary medicine are in short supply despite the numerous viral infections common in animals. Three main reasons for the lack of antiviral drugs for veterinary purposes include high cost of development, narrow spectrum of activity, and absence of rapid diagnostic tests that would allow prompt antiviral use in acute infections.[1] Probably the most challenging aspect of antiviral drug development is that virus replication is intimately linked to host cell metabolic processes. Hence, many drugs that inhibit virus replication may also damage the host. Appropriate targets for antiviral drugs must be teased out by performing detailed studies of virus replication and the interactions between host and viral proteins. However, even when a unique facet of viral replication is discovered, a targeted drug may have unforeseen negative effects on the host. Compared to antibiotics, antivirals have a higher risk for side effects and toxicity due to the viral replication cycle utilizing the host cell biochemical machinery. Nephrotoxicity is the main adverse reaction of antiviral drugs in humans and animals.[2] The concept of a "broad-spectrum antiviral" (other than immunomodulators or cytokines) has been difficult to realize, because agents with a broader spectrum of action have normally been associated with increased host cell toxicity. The goal in antiviral development is identifying select targets and developing a drug with few side effects.[3] Other challenges include dosing considerations and the ability to achieve therapeutic doses at appropriate tissue sites. Despite these issues, antiviral use in veterinary medicine is growing, along with increasing antiviral research.

Most of the antiviral drugs currently used in veterinary medicine were originally developed against human viral infections and their clinical use in veterinary medicine is not widespread. Antivirals approved for use in humans, and available for use in veterinary medicine, include drugs that target retroviruses and herpesviruses. Examples include acyclovir (ACV), idoxuridine, famciclovir, and trifluridine for use against feline herpesviruses. Azidothymidine (AZT), used in the treatment of human immunodeficiency virus (HIV), has been evaluated and found effective against feline retroviruses.[1]

Currently, the only licensed veterinary antiviral drugs are feline interferon and polyprenyl immunostimulant. These are both immunomodulating drugs rather than drugs that target a specific biochemical process in virus replication. Feline interferon is approved for use in treating feline retroviruses and canine parvovirus. Feline interferon has also been used off label for feline calicivirus. Unfortunately, treatment protocols and cost prevent its routine use.[1] Polyprenyl immunostimulant has also been approved in the United States for treatment of feline herpesvirus. Other studies have looked at off license use of ACV and famciclovir for feline herpes, oseltamivir and immune plasma for canine parvovirus,[1] and lysine for feline herpesvirus.[1]

In avian medicine, there are a number of viral infections commonly seen, including polyomavirus, psittacine herpesvirus, avian bornavirus, West Nile virus, avian influenza, and Marek disease. As there are no approved antivirals, practitioners have utilized an assortment of antivirals off label in birds with these viral diseases. To date, most research in avian antiviral medicine has occurred in poultry, although amantadine and neuraminidase (NA) inhibitors are prohibited to use in poultry at this time.[4]

Classes of Antiviral Drugs

Many of the commonly used antivirals are nucleoside analogs. Nucleoside analogs are a large and important class of drugs including drugs for cancer, rheumatological disease, and antivirals. This chapter will focus on the antiviral drugs commonly used in human viral disease that have been or have the potential to be utilized in avian medicine. Nucleoside analogs are used in the treatment of hepatitis B and C virus (HBV, HCV), cytomegalovirus (CMV), herpes simplex virus (HSV), and HIV. Nucleoside analogs resemble naturally occurring nucleosides and act by termination of a replicating DNA chain. The nucleoside analogs include those drugs with activity against both DNA- and RNA-dependent polymerases and nucleoside reverse transcriptase inhibitors (NRTIs). While most of the antiviral agents approved for use in humans interfere with the synthesis or regulation of viral nucleic acids and act to block elongation of newly synthetized DNA or RNA chains, an exception is the NA inhibitor oseltamivir that is widely used to treat human influenza. This drug very specifically inhibits a key influenza virus enzyme.[5]

Inhibitors of DNA Replication

ACV, vidarabine, valacyclovir, entecavir, famciclovir, cidofovir (CDV), penciclovir, and ganciclovir are nucleoside analogs

that block nucleic acid synthesis by inhibiting elongation of a growing DNA chain.

Acyclovir

Acyclovir (acycloguanosine) is an acyclic analog of the purine nucleoside deoxyguanosine (Figure 10.1). ACV is used to treat herpesvirus infections.[4] The drug is exquisitely targeted to herpesvirus-infected cells because to be active, it must first be phosphorylated by the herpesvirus thymidine kinase. The phosphorylated drug is then inserted into growing DNA chains by the viral DNA polymerase. Due to its lack of a 3′-hydroxyl group, DNA synthesis stops at the point at which the drug is inserted. (Note that in uninfected cells, the drug is not phosphorylated and thus does not inhibit cellular DNA replication.) As a therapeutic, it is mainly used to treat herpesvirus infections: HSV types 1 and 2 and varicella zoster virus.[6] In veterinary medicine, it has been primarily used against feline herpesvirus-1 (FHV-1) infections.[5-7]

The disposition of ACV has been investigated in several species. The drug was well distributed into all the tissues, including the brain, in mice and rats. Marked species differences were observed in the gastrointestinal absorption and transformation of ACV. Oral dosing produced better absorption in dogs and mice than in rats and rhesus monkeys.[8] Topical ACV treatment was shown to be effective against FHV-1 conjunctivitis and keratitis in cats.[7] Studies in dogs report oral absorption of ACV of 80% to 90%, but it becomes saturated at high doses.[8]

Chickens (*Gallus gallus domesticus*) experimentally infected with Marek disease virus that received ACV daily at 10 mg/kg intramuscularly (IM) for 5 days, beginning day 3 post challenge, developed no clinical signs of disease. When given IM daily for 9 days, beginning 14 days post challenge, mortality was reduced.[9]

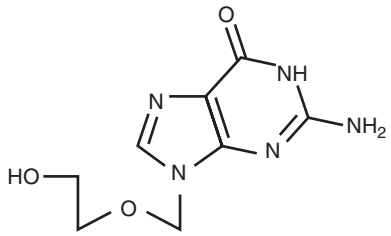


FIGURE 10.1 Chemical composition of acyclovir.

A study utilizing ACV at 120 mg/kg every 12 hours was performed in tragopans (*Tragopan* spp.) to determine the absorption, distribution, and linearity of ACV after administration of a single dose. Oral treatment with an ACV oral suspension (40 mg/mL; Alpharma USPD, Baltimore, MD, USA) at doses of 40, 80, or 120 mg/kg was administered by crop gavage. ACV at 120 mg/kg every 12 hours maintained plasma ACV concentrations above the accepted effective ACV plasma level (1.0 mg/mL) for 12 hours following administration, suggesting that ACV at 120 mg/kg every 12 hours achieved effective plasma concentrations for prevention and treatment of herpesviral infections in tragopans.[10]

The pharmacokinetics of intravenous (IV) and IM single-dose administration of ACV were determined in Quaker parakeets (*Myiopsitta monachus;* Tables 10.1 and 10.2).[11] Peak plasma ACV concentrations occurred 15 minutes following IM injection, and plasma concentrations of ACV were undetectable 4 to 6 hours post IV and 6 to 8 hours post IM injection. In the same study, oral (capsules) and intravenous (sodium salt) formulations of ACV were given by gavage at 80 mg/kg. Peak concentrations with the sodium salt formulation developed slower and were lower than with the capsules. In a second study, ACV was administered IM at 40 mg/kg every 8 hours for 7 days and then gavage-fed at a dose of 80 mg/kg every 8 hours for 4 days to evaluate excessive drug accumulation or adverse side effects. Excessive drug accumulation was not detected in either study, and the birds maintained normal appetite and weight. A third study evaluated plasma concentrations following the addition of ACV in the feed (400 mg in 2 quarts of parrot seed) and water (1 mg/mL). Plasma concentrations of ACV ranged from 2.07 ± 1.09 μg/mL to 3.93 ± 1.13 μg/mL,[11] which is below the accepted effective ACV plasma level (1.0 mg/mL).

Based on the aforementioned PK study, the efficacy of ACV against experimentally induced herpesvirus infection (Pacheco disease) was evaluated in Quaker parakeets.[12] Thirty-two of 40 birds were challenged with 0.1 mL of a suspension of herpesvirus IM. Treatment with ACV was started 24 hours later and continued for 7 days. The birds were divided into five groups with eight birds each. In group 1 (control), eight birds were given the virus IM and were not

TABLE 10.1

Pharmacokinetic Values for Intravenous, Intramuscular, and Oral Doses of Acyclovir Administered in Quaker Parrots *(Myiopsitta monachus)*

Value	20 mg/kg IV	40 mg/kg IM	80 mg/kg Oral Capsules	80 mg/kg IV Sodium Salt (Given Orally)
$T_{½}$ (h)	0.65	0.71	2.18	9.49
Mat (h)		0.11	2.25	13.05
VOD (mL/kg)	627.65			
AUC (μg/mL/h)	29.70	60.86	363.92	2600.10
Cl (mL/kg/min)	11.22			
PC (μg/mL)			34.65	12.49
Bioavailability (%)		90.1	77.4	146.4

AUC, Area under the curve; *Cl*, total body clearance; *IM*, intramuscular; *IV*, intravenous; *MAT*, mean absorption time; *PC*, peak concentration; *T½*, biological half-life; *VOD*, volume of distribution.

TABLE 10.2

Mean Plasma Concentration (μg/mL) Following a Single Intravenous, Intramuscular, or Oral Dose of Acyclovir in Quaker Parrots *(Myiopsitta monachus)*

Sample Time (H)	20 mg/kg IV	40 mg/kg IM	80 mg/kg Oral Capsule	80 mg/kg IV Sodium Salt (Given Orally)
0	0	0	0	0
0.25	29.28 ± 7.57	40.19 ± 5.77	16.00 ± 6.10	12.8 ± 1.76
0.5	15.44 ± 1.25	33.95 ± 4.57	7.66 ± 3.04	10.49 ± 5.62
0.75	17.15 ± 3.90	28.05 ± 1.03	7.24 ± 5.59	11.27 ± 15.39
1	9.42 ± 2.03	20.77 ± 6.07	5.71 ± 7.97	4.38 ± 0.17
2	5.51 ± 1.55	8.96 ± 0.74	2.55 ± 1.78	7.88 ± 2.65
4	0.41 ± 0.23	1.22 ± 0.64	3.82 ± 3.00	5.66 ± 5.02
6	0	0.31 ± 0.22	3.19 ± 3.40	4.85 ± 3.45
8	0	0.02 ± 0.04	2.58 ± 0.73	3.61 ± 0.18

IM, Intramuscular; *IV*, intravenous.

treated. In group 2 (infected, gavage fed), eight birds were given a suspension of ACV 80 mg/kg every 8 hours via gavage. In group 3 (infected, low dose, IM), eight birds were given injectable sodium salt of ACV IM at 40 mg/kg q8h. In group 4 (infected, high dose, IM), eight birds were given injectable sodium salt of ACV IM at 250 mg/kg q8h. In group 5 (contact control), eight birds were not given the virus. Four of the uninfected birds were given a suspension of ACV 80 mg/kg every 8 hours via gavage and four birds were untreated. To determine if natural virus transmission would occur between challenge-exposed and nonchallenged-exposed birds, one treated and one untreated bird were placed in cages with birds in groups 1 through 4. Clinical signs observed throughout the study included lethargy, anorexia, regurgitation, polyuria, yellow urates, and seizures. Gross findings in infected birds included hepatomegaly, splenomegaly, and yellow discoloration of organs and coelomic surfaces. Birds given IM ACV had areas of hemorrhage in the pectoral muscles with the most severe lesions seen in group 4. Group 1 (control) had the highest mortality, with six out of eight birds dying; group 2 had one bird die; group 3 had three birds die; group 4 had four birds die; and no birds died in group 5. There was a significant difference in mortality between groups 1 and 2 ($P = .023$), but no difference in mortality among groups 1, 3, and 4. Birds in group 1 died within a mean period of 6 days after virus inoculation. Clinical signs and death occurred in groups 2 and 3 only after ACV treatment was discontinued. Postmortem examination was performed on all birds that died, with histologic evidence of herpesvirus infection found in all birds that died. Lesions were most severe in the positive control group.[12] In addition, herpesvirus could be recovered from all infected birds through inoculation of chick embryo cell culture with pooled tissue suspensions.

Acyclovir Derivatives or Prodrugs

Vidarabine (9-D-arabinofuranosyl adenine) was one of the first antiviral agents licensed for systemic treatment of herpes viral infections in humans.[5] It is an adenosine analog that is converted by cellular enzymes to its active intracellular derivative, vidarabine triphosphate. Vidarabine is used topically in feline herpes keratitis. In vitro studies have demonstrated activity against feline and equine rhinopneumonitis.[6] A vidarabine 3% ophthalmic ointment has been effective and well tolerated in cats for feline keratoconjuctivits.[5] Valacyclovir is a prodrug for ACV. It has enhanced bioavailability over ACV resulting in faster absorption after oral administration. In cats, administration of valacyclovir has been associated with adverse side effects including renal and hepatic necrosis, myelosuppression, and nephrotoxicity. Penciclovir [9-(4-hydroxy-3-hydroxymethylbut-1-yl)] is a guanosine analog that resembles ACV in structure, mechanism of action, and antiviral activity, but penciclovir-triphosphate accumulates in virus-infected cells in much higher concentrations and for a longer half-life (10–20 times longer) than ACV.[5] Unfortunately, it is less potent than ACV and has poor bioavailability (<5%). Due to the low bioavailability of penciclovir, famciclovir was developed. Famciclovir (the diacetyl ester of 6-deoxypenciclovir) is the oral prodrug of ACV with improved bioavailability. Following oral administration, famciclovir gets rapidly converted to its active metabolite penciclovir by di-deacetylation and oxidation. In cats, famciclovir must be administered in high doses orally to get adequate plasma concentrations due to their lack of the hepatic aldehyde oxidase enzyme. Despite the high oral doses, the drug is well tolerated in cats and has been successful in treatment of FHV-1 conjunctivitis.[7] Ganciclovir is another purine nucleoside analog that resembles ACV and is widely used to treat CMV infections in human medicine. In vitro, it has been effective against FHV-1 in cats. Unfortunately, there is lack of data on its efficacy and safety in animals.[5] No anecdotal or clinical studies could be found in these ACV derivatives or prodrugs in birds at this time.

Cidofovir

CDV [(S)-1-(3-hydroxy-2-phosphonylmethoxypropyl) cytosine, HPMPC] is an acyclic nucleoside analog. It is approved for clinical use in the treatment of CMV retinitis in human AIDS patients. CDV has broad-spectrum activity against DNA viruses, including herpesvirus, adenovirus, papillomavirus, polyomavirus, and poxvirus. In vitro, CDV has shown activity against orthopox (vaccinia, variola [smallpox]), cowpox, monkeypox, camelpox, molluscipox (molluscum contagiosum),

and parapox (orf) viruses. In vivo, CDV activity has been shown in different models of pox infection when administered intraperitoneally, intranasally (aerosolized), and topically. In immunocompromised human patients, CDV has been effective in the treatment of recalcitrant molluscum contagiosum virus and orf virus. CDV remains a reference compound against poxviruses and holds potential for the therapy and short-term prophylaxis of not only orthopox but also parapox and molluscipoxvirus infections.[13]

An adult great-horned owl (*Bubo virginianus*; GHOW) was treated successfully with topical CDV 1%, interferon (IFN) alpha 2B ophthalmic solutions, and oral L-lysine for a recurrent corneal ulceration of the right eye caused by a novel alphaherpesvirus.[14] This novel herpesvirus, referred to as Strigid herpesvirus 1 (StrHV1), was identified utilizing polymerase chain reaction (PCR) and gene sequencing.

Nucleoside Reverse Transcriptase Inhibitors

NRTIs are DNA chain terminators that are potent inhibitors of retroviral reverse transcription; they block the synthesis of viral DNA in the infected cell. These drugs are recognized by viral reverse transcriptase and are incorporated into growing DNA chains. Once incorporated, the DNA chains cannot be elongated, thus terminating the DNA chain. These drugs are used in retroviral infections.

Zidovudine

Azidothymidine (AZT) (Figure 10.2), used in the treatment of HIV, acts by inhibiting HIV reverse transcription. Originally, AZT was developed to target reverse transcription of avian retroviruses that may cause cancer in poultry and was later successfully applied to HIV as well.[15]

AZT has been evaluated and found effective in feline retroviruses. The long-term effectiveness of AZT was evaluated in feline immunodeficiency virus (FIV)–seropositive cats with clinical symptoms compared with conventional symptomatic therapy. The long-term oral treatment of AZT at a dosage of 5 mg/kg body weight three times a day led to a total recovery from clinical symptoms in six of nine FIV-seropositive cats. A transient decrease of red blood cell (RBC) count, packed cell volume (PCV), and hemoglobin was noted in treated cats until the 4th week of treatment; the hemogram was within the normal range after 3 months. Hyperglycemia was noted in one cat and Heinz bodies were noted in two treated cats.[16]

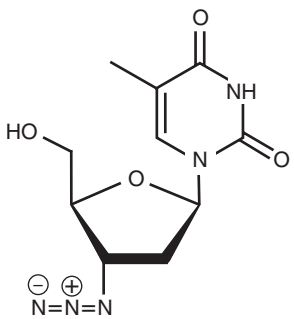


FIGURE 10.2 Chemical composition of zidovudine.

The inhibitory effect of AZT on the replication of avian leukosis virus (ALV) was assessed *in vitro*.[17] AZT was shown to be a potent inhibitor of viral DNA synthesis in infected cells. It was determined that variations in the concentrations of drug were needed to inhibit virus replication in different cell types, with higher concentrations of drug needed to inhibit virus replication in chicken embryo fibroblasts than in the quail cell line QT6. Several parameters of AZT effects on quail cells were evaluated. The effect of AZT to decrease virus replication in a continuous quail line was examined. The amount of AZT required to inhibit colony formation 70% was approximately 40 μM. The amount required to inhibit colony formation 95% was 80 μM. In a separate assay, the effect of AZT on virus spread was tested. Viral spread was inhibited 70% in the presence of 1 μM AZT, 85% at 3 μM AZT, and 95% at 10 μM AZT. No virus was detected at 30, 80, or 200 μM AZT concentrations. The anticipated target of AZT is the viral DNA polymerase reverse transcriptase. To test this hypothesis, they examined viral DNA synthesis in infected cells. At 1 μM, there was no inhibition, and at 30 μM, 80 μM, and 200 μM, there was a 65%, 90%, and 100%, respectively.[17] Based on data in other species and the inhibitory effects seen in vitro, it would stand to reason that AZT may be beneficial clinically in the treatment of avian retroviral infections.

Abacavir

Studies in chickens revealed that phosphorylated abacavir (ABC-1) and phosphorylated derivatives of didanosine reduced Newcastle disease virus (NDV)–induced tissue oxidative stress and inhibited the expression of the fusion protein and was protective in NDV-infected chickens.[18] Suresh et al. assessed the protective role of phosphorylated ABC-1 in vivo in NDV-infected 2-day-old chickens. Chickens were divided into three groups of six birds: group 1 (control), group 2 (NDV infected), and group 3 (NDV infected + ABC-1). Groups 2 and 3 were infected with 200 μL of an infectious dose of NDV IM. Group 1 received 200 μL of phosphate-buffered saline (PBS) IM. Group 3 was treated with phosphorylated ABC-1 orally at 2 mg/kg dissolved in water given daily for 10 days beginning day 4 post infection. The NVD-induced oxidative damage and protective role of ABC-1 were evaluated in vital organs (liver, lung). Results confirmed that phosphorylated ABC-1 protects chickens from NDV-induced pathogenesis and protein oxidation/nitration, and has potent antiviral activity. A similar study by the same author revealed similar findings using a phosphorylated didanosine (DDI-10) in NDV virus in 2-day-old chicks.[19]

NUCLEOSIDE ANALOGS

Ribavirin

Ribavirin (1-β-D-ribofuranosyl-1,2,4-triazole-3-carboxamide; Figure 10.3) is a triazole nucleoside analog that inhibits replication of both DNA and RNA viruses. Several mechanisms of action have been proposed for ribavirin. These include reduction of intracellular guanosine-5′-triphosphate (GTP) pools, which can inhibit translation; interference with viral genome or mRNA capping; inhibition of viral RNA genome synthesis; and increased mutagenesis of RNA viruses.

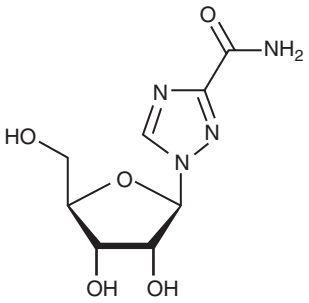


FIGURE 10.3 Chemical composition of ribavirin.

Ribavirin is active against a wide range of viruses including adenoviruses, arenaviruses, bunyaviruses, herpesviruses, orthomyxoviruses, paramyxoviruses, picornaviruses, poxviruses, retroviruses, rhabdoviruses and rotaviruses, and influenza virus. In combination with IFN, ribavirin has activity against HCV.[20]

Ribavirin in cats has resulted in drug-induced thrombocytopenia, depression of red and white blood cells, increased alanine aminotransferase activity, icterus, and weight loss.[21] These side effects have not been observed in humans, dogs, or birds. Ribavirin activity has been demonstrated against bovine viral diarrhea virus and bovine herpesvirus 1.[22] Ribavirin has shown activity against mammalian Borna disease virus 1 (BoDV-1), which is a close relative of the avian bornaviruses, both in mammalian cell lines and experimentally in infected gerbils.[23] Musser et al. reported an antiviral effect in duck embryo fibroblast (DEF) cultures infected with parrot bornavirus-4 (PABV-4).[24]

Ribavirin inhibited avian bornavirus activity in a polymerase reconstitution assay and reduced viral load in avian cell lines infected with two different parrot bornaviruses. It was demonstrated that 1 $\mu g/mL^{-1}$ of ribavirin reduced polymerase activity to 10%, while higher doses reduced it further, indicating that direct interference with viral polymerase activity is the major mode of ribavirin activity against avian bornavirus. The same researchers also evaluated the effect of ribavirin on cell culture. Quail cell cultures persistently infected with PaBV-1 or PaBV-4 were grown in medium containing 1 or 5 $\mu g/mL^{-1}$ of ribavirin passaged twice a week. Both doses of ribavirin substantially decreased the number of infected cells after 5 days of treatment. They also evaluated 10U mL^{-1} of chicken IFN-α (ChIFN-α) alone and combined with 1 $\mu g\ mL^{-1}$ of ribavirin and found that both reduced the number of bornaviral-infected cells, with the combined ribavirin and ChIFN-α having the most pronounced cell reduction. In all models, once the treatment stopped, the proportion of virus-infected cells rapidly retuned. These results suggest that ribavirin and recombinant IFN-α in a combined treatment in vivo may be more effective than ribavirin alone.[25] In a study conducted at the Schubot Avian Health Center at Texas A&M, treatment of three Grey parrots (*Psittacus erithacus*, GP) naturally infected with avian bornavirus infection with ribavirin at 15 mg/kg per os (po) daily for 1 week, 18 mg/kg po daily for 4 weeks, and 18 mg/kg po twice a day for 8 weeks resulted in no change in viral shedding in the feces based on biweekly PCR testing.[26]

Favipiravir (T-705)

Favipiravir (T-705) (Toyama Chemical Company, Ltd. Tokyo, Japan) is an antiviral similar to ribavirin. It is a novel antiviral compound that selectively and potently inhibits the RNA-dependent RNA polymerase (RdRP) of influenza and many other RNA viruses. Favipiravir is active against a broad range of influenza viruses, including influenzas (H1N1), (H5N1), and the recently emerged (H7N9) avian virus. It also inhibits influenza strains resistant to current antiviral drugs and shows a synergistic effect in combination with oseltamivir, thereby expanding influenza treatment options.[27] Favipiravir is also active against many arenaviruses, bunyaviruses, flaviviruses, alphaviruses, picornaviruses, and noroviruses.[28]

Favipiravir was found to be a potent inhibitor of both BoDV-1 and PaBV-4 in vitro. T-705 at 200 or 400 mM given for 28 days suppressed BoDV-1 to undetectable levels with no significant increase in the amount of BoDV-1 RNA by 30 days post treatment. In the same study, quail cells persistently infected with PaBV-4 cells were treated with 20 mM ribavirin or 50 or 400 mM of T-705 for 28 days and avian bornavirus replication was evaluated via immunofluorescence assay (IFA) and real-time (RT) PCR. T-705 decreased the amount of PaBV-4 RNA more effectively than ribavirin, with the 400 mM dose being most effective. There was also no increase in the number of PaBV-4 RNAs at 14 days post treatment cessation. No cytotoxic effects were seen with T-705 at the test doses. Further studies are needed to determine if T-705 could be used to treat persistent bornavirus infection in various animals in vivo, but these results suggest that T-705 may be effective against a broad spectrum of bornaviruses. T-705 is an orally administered drug approved for clinical use in humans, so if studies confirmed effectiveness in vivo, T-705 could be readily used to treat persistent avian bornavirus infection in birds.[29]

INFLUENZA ANTIVIRALS

There have been two types of antivirals developed against influenza; the first were the M2 ion channel blockers (amantadine and rimantadine). The second are the NA inhibitors. The efficacy of the M2 ion channel blockers is limited to influenza A. Currently, most influenza A strains have developed resistance to the M2 channel blockers. The NA inhibitors have efficacy for both influenza A and B viruses, and although there is some documented resistance, they are still effective drugs.

M2 Protein Target Inhibitors

The M2 protein is a protein on the surface of influenza A. It is absent in influenza B. The M2 proton channel is required for virus replication and maturation. The M2 protein is an integral membrane protein that functions as an ion channel. It enables hydrogen ions to enter the virion. When the internal component of the virus achieves a low pH, a dissociation or uncoating occurs. M2 protein inhibitors block the acid-mediated dissociation of the ribonucleoprotein complex in the early replication cycle and initiate pH-induced changes in hemagglutinin during intracellular transport in late replication cycle. M2 inhibitors are effective against influenza A viruses and include amantadine and rimantadine. Both amantadine and rimantadine are active only against susceptible influenza A viruses. Rimantadine is 4- to 10-fold more active than amantadine. Virtually all H3N2 strains of influenza circulating worldwide are resistant to these drugs.

Amantadine hydrochloride and rimantadine hydrochloride are antivirals that inhibit virus replication during the early

stage of infection by blocking the ion channel formed by the M2 protein.[9] Historically, they were effective prophylactic agents in the control of influenza A virus infections in humans. They have no action against influenza B and C viruses. Previous studies in domestic fowl revealed reduced mortality and reduction of disease in treated birds. Currently, amantadine is prohibited for treatment of influenza due to the development of resistance.[30] In a study conducted at the Schubot Avian Health Center at Texas A&M, three GP naturally infected with avian bornavirus infection with amantadine were treated with 1 mg/kg po daily for 8 weeks. There was no reduction in viral shedding in the feces based on PCR (P. Gray, personal communication).

In addition to its antiviral effects, amantadine is also used in humans to reduce the severity of symptoms in Parkinson disease by antagonizing the N-methyl-D-aspartate (NMDA) receptor. Amantadine has many effects on the brain including the release of dopamine and norepinephrine. Amantadine causes NMDA receptors to occupy closed conformations; this interaction with the NMDA receptor makes it effective at inhibiting NMDA responses during prolonged depolarizations that accompany neurological insults, which may occur in ABV-affected birds or in animals experiencing chronic pain. There have been studies looking at the analgesic effects of amantadine in dogs that suggest amantadine may be a useful adjunct therapy for the clinical management of dogs with osteoarthritic pain.[31] There is anecdotal evidence that amantadine hydrochloride (due to its inhibition of NMDA response) may contribute to the reduction of clinical signs in birds with proventricular dilatation disease (PDD) when combined with celecoxib.[32]

Neuraminidase Inhibitors

A key step in the replication cycle of any virus is the ability to exit a cell and infect new cells. NAs (sialidases) cleave glycosidic linkages of neuraminic acids. Influenza A and influenza B viruses require the enzyme NA to efficiently move from an infected cell to other cells. If this activity is inhibited, newly formed flu virus particles remain associated with producer cells and form large clumps, preventing new host cells from being infected. The NA inhibitors oseltamivir and zanamivir are considered our first line of defense against potentially fatal variants of influenza A pandemic strains that may affect humans and susceptible animals and birds worldwide.

Few studies have been performed on the effectiveness of NA inhibitors in birds. The anti-influenza efficacy of the potent NA inhibitor oseltamivir was evaluated in 16 SPF 3-week-old chickens and 24 3-week-old ducks. The birds were divided into four groups: group 1—8 infected, untreated chicken control group, group 2—8 infected, treated chicks, group 3—12 infected, untreated duck control group, and group 4—12 infected, treated ducks. Oseltamivir was mixed 1:1 with PBS and administered orally to groups 2 and 4. Oseltamivir treatment (0.5 mg/kg given twice a day for 5 days) was began 4 hours prior to inoculation. Groups 1 and 3 received sterile PBS on the same schedule. The chickens were sacrificed on day 5 post infection and the ducks on days 3 and 6 post infection for virus isolation and titration. In group 1, virus replication was detected in all eight trachea and cecal tonsil samples. Group 2 birds had significant reduction of viral replication from both the trachea and the cecal tonsils at day 5 post infection with three of eight samples positive for viral replication. Group 3 showed the highest frequency of virus detection in the trachea, with viral replication detected in seven of eight tracheas at 3 days post infection and in seven of eight cecal tonsil samples at 6 days post infection. Group 4 had completely reduced viral replication from both the trachea and cecal tonsils, with no virus detected at 3 and 6 days post infection. In this study, viral replication of low pathogenic avian influenza virus (AIV) was significantly reduced in the chicken model and completely reduced in the duck model indicating that antiviral treatment may be a valuable addition to the prevention of avian influenza in zoological and other avian species collections.[33]

One of the issues with influenza viruses is their ability to mutate and develop resistance to antiviral drugs. This resistance affects all species that might become infected. A study investigated if and to what extent NA inhibitor–resistant mutants exist in the wild population of influenza A viruses that inhabit wild birds.[34] Two sets of databases were examined, evaluating more than 6000 birds, 379 swine, and 122 environmental isolates. Of the sequences from the avian dataset, 132 (2.4%) carried at least one, or in two cases, even two or three NA inhibitor resistance mutations. Swine and environmental isolates from the same data set had 18 (4.75%) mutants and 1 (0.82%) mutant, respectively, with at least one mutation. The Ottenby sequences carried at least one mutation in 15 cases (6.52%). In summary, antiviral resistance–related mutations already exist in populations of avian influenza A viruses isolated from their natural hosts, that is, mallard duck, other waterfowl as well as domestic poultry, and domestic swine.[35]

Arildone

Arildone is an antiviral drug of the 4-[6-(2-chloro-4-methoxy) phenoxyl]hexyl-3,5-heptanedione class; it is a novel aryl diketone which is active against both DNA and RNA viruses.[34] It is primarily used as a broad-spectrum antiviral agent because it is relatively a less toxic drug and inhibits viral replication at lower concentration. Arildone inhibits replication of enterovirus, in particular poliovirus, via interaction with the viral capsid and hence blocking viral uncoating. It has primarily been studied in poliovirus. It also inhibits HSV type 2 protein synthesis and viral DNA replication by an unknown mechanism of action. Due to its poor solubility in water, it is administered as a solution in dimethyl sulfoxide (DMSO). In studies on mice intracerebrally infected with a high dose of polio type 2, arildone was able to block the virion uncoating property and prevent paralysis and death.[36] No studies have been performed on birds.

OTHER ANTIVIRAL APPROACHES

Although research on antivirals continues and the number of antivirals continues to grow, treating viral disease, especially chronic viral disease, remains challenging. Immunotherapy has been utilized to modulate the immune system, and medications such as antifungals, antiparasitic drugs, and antibiotics have been found to have antiviral properties and have been repurposed. In addition, many herbs and plants are known to have antiviral properties.

Immunotherapy

Modification of immune responses is another possible intervention for ongoing viral infections. The most suitable for immunotherapy are chronic viral infections.[37] There are several forms of immunotherapy that are in use, including monoclonal antibodies (mAbs), recombinant antibodies (rAbs), and IFNs.

mAbs, which are antibodies specific for one antigen or one epitope, have been shown to inhibit rabies virus, in a dose-dependent way, by impairing transcription of the genome or neutralizing newly translated proteins. mAbs have been shown to be effective against noncapsid proteins of poliovirus, rotaviral diarrhea, classical swine fever, bluetongue, Hendra, and Nipah viral infections.[2,38]

rAbs are another therapeutic strategy for infectious viral diseases. They are synthetic antibodies that can be created using antibody genes made in the laboratory, eliminating animals from the process. An example is avian antibody against VP2 of infectious bursal disease virus protecting against viral infection of chickens.[37] Advantages of rAbs include non-animal technology, a speedier more efficient process, and high reproducibility. Because rAbs are derived from human or synthetic genes, they do not trigger the intense immunogenic reactions in patients that animal-derived antibodies do, making them ideal for clinical use.[39,40]

IFNs are glycoproteins that possess strong antiviral activity and represent one of the first lines of host defense against invading pathogens. They represent an early host defense prior to the immune response onset. These proteins are classified into three groups, Type I, II, and III IFNs, based on the structure of their receptors on the cell surface. Due to their immune modulating capability, they have become therapeutic options to control chronic viral infections in humans. In combination with other drugs, Type I IFNs are utilized in suppressing HCV and HBV infections, while Type III IFN has generated encouraging results as a treatment for HCV infection.[41]

A study evaluating IFN treatment in GP with psittacine beak and feather disease (PBFD) revealed that IFN-γ of poultry origin has a potential use in the treatment of circovirus infection in young GP. The study evaluated 1 million IU feline interferon in 12 GP infected with PBFD with profound leucopenia and found that feline interferon was not effective. In a second study, 10 GP infected with PBFD with a severe leucopenia were treated with 1 million IU of poultry IFN-γ. Seven of 10 birds treated with poultry IFN-γ were alive at week 30 and had elevations in their total leucocyte count. Nine months post diagnosis, the birds were alive and exhibited no signs of circovirus infection.[42]

Another study demonstrated that pretreatment with ChIFN-α prior to infection with the pandemic H1N1 and H5N9 AIVs significantly reduced viral replication in both chicken and turkey origin lung cells.[43] Similar conclusions were recently described following oral administration of rChIFN-α and H9N2 AIV infection.[44] In this study, 7-day-old and 33-day-old white leghorn SPF chickens were divided into 5 groups of 20 birds each: group 1—preventative group (treated then infected), group 2—the therapeutic group (infected then treated), group 3—virus challenged (untreated), group 4—ChIFN-α treated (uninfected), and group 5—blank control group (uninfected and untreated). Group 1, 2, and 4 birds were given 0.5104 U of ChIFN-α orally to the 7-day-old chicks and a 2-fold dose given to the 33-day-old chicks. Results revealed that the body weight of chickens was not influenced by ChIFN-α, and ChIFN-α significantly reduced the viral load in the trachea of infected chicks. The study indicated that ChIFN-α could be used as a preventive and therapeutic agent against AIV. They found that the therapeutic effect was better than the preventive effect on 7-day-old chicks, which was opposite to 33-day-old chicks.[44]

Previous studies have shown that ChIFN-α administered to chickens by oral ingestion or intravenous injection can inhibit avian viruses including H9N2 AIV, NDV, infectious bursal disease virus, infectious bronchitis virus, Rous sarcoma virus, and Marek disease virus.[45,46]

Based on these studies in birds and in the successful use of IFNs in human medicine, further studies in IFN treatment of avian viral diseases are warranted.

Drug Repurposing

Drug repurposing (or drug repositioning) is the method of assigning a new medical indication for an existing drug. The repositioned drug might be currently on the market for other disease process.[2]

Examples of repurposed drugs include antibiotics and anthelmintics.

Antibiotics

Antiviral potential of antibiotics has been studied on a number of drugs. Rifampin, a widely used derivative of rifampicin, is a semisynthetic rifamycin. Rifamycins can cross mammalian tissue and cell membranes.[6] Among the antibacterial agents of these derivatives, some act by inhibiting the bacterial DNA-dependent RNA polymerase. Rifamycin derivatives were also found to act against type II DNA topoisomerases. Rifampicin, rifapentine, and rifabutin are semisynthetic and water-soluble derivatives used in therapies against different gram-positive and gram-negative bacterial organisms including methicillin-resistant *Staphylococcus aureus* (MRSA), mycobacteria (*Mycobacterium bovis* or *Mycobacterium tuberculosis*), and leprosy. They are also able to prevent viral infections (e.g., influenza, retrovirus).[2,47] In addition, rifampin can inhibit poxvirus replication in vitro. In vitro analysis for selective inhibition of RNA-dependent DNA polymerase (reverse transcriptase) on several rifamycins revealed that certain derivatives inhibited production of Rous sarcoma virus in vitro and prevented focus formation by RNA tumor viruses.[48] Further studies are needed to see if rifampin antiviral activity will be effective in vivo in avian species with poxvirus or retroviral infections.

Minocycline has been investigated for its antiviral action. It is a synthetic second-generation tetracycline derivative that readily crosses the blood-brain barrier. Minocycline has antibacterial, immunomodulatory, and antiinflammatory action and is used for the treatment of gram-negative and gram-positive bacterial infections, chlamydial infections, acne, rheumatoid arthritis, and urinary tract infections in humans.[2] There has been reported antiviral activity of minocycline against HIV, Japanese encephalitis virus, Dengue virus, and West Nile virus. Minocycline diminishes viral RNA synthesis, intracellular viral protein synthesis, and thus viral production.[48] Minocycline reduced the severity of encephalitis, suppressed viral load in the brain, and decreased the expression of CNS inflammatory markers in five simian immunodeficiency virus (SIV)–infected pigtailed macaques that were treated with 4 mg/kg per day of minocycline beginning at

early asymptomatic infection (21 days after inoculation with SIV).[49] Further studies are needed to determine if minocycline could be an effective treatment for West Nile virus or retroviral infections in birds. It is also possible that minocycline could reduce inflammation and clinical signs in avian bornaviral–infected birds.

Fluoroquinolones have shown antiviral activity. Quinolones inhibit prokaryotic type II topoisomerases, namely DNA gyrase and, in a few cases, topoisomerase IV, through direct binding to the bacterial chromosome. Fluoroquinolones were shown to be effective against vaccinia virus and papovaviruses, and with modification (introduction of an aryl group at the piperazine moiety), activity is shifted from antibacterial to antiviral, with a specific action against HIV.[50]

Teicoplanin is an antibiotic used in the prophylaxis and treatment of serious infections caused by gram-positive bacteria, including MRSA and *Enterococcus faecalis*.[2] It is a semisynthetic glycopeptide antibiotic with a spectrum of activity similar to vancomycin. Its mechanism of action is to inhibit bacterial cell wall synthesis. Teicoplanin has shown significant inhibitory activity against Ebola virus, HIV, HCV, flavivirus, coronaviruses, influenza virus, and respiratory syncytial virus (RSV).[51]

Anthelmintics

Ivermectin and abamectin are anthelmintic drugs that have shown antiviral activity against Chikungunya virus and yellow fever virus thru specifically targeting the viral helicase.[51] Antimalarial drugs chloroquine and amodiaquine have displayed activity against West Nile virus, Dengue virus, and Ebola virus by interfering with viral entry and replication.[51] Estrogen receptor inhibitors used for cancer treatment and inhibitors of dopamine receptors used as antipsychotic medications demonstrated antiviral activity against Middle East respiratory syndrome (MERS) and severe acute respiratory syndrome (SARS).[52]

Benzimidazoles

Benzimidazole and its derivatives are best known in veterinary medicine for their activity against intestinal parasites (nematodes and trematodes), but they have a wide variety of biological activities. These include analgesic, antibacterial, antifungal, anticancer, anticonvulsant, and antiviral activities.[52] A number of benzimidazoles are commercially available including telmisartan for hypertension, omeprazole utilized for gastric ulcers, and albendazole which is an antiparasitic drug.

The antiviral activity of benzimidazole nucleosides was first reported in 1954. They developed 5,6-dichloro-1-(β-D-ribofuranosyl) benzimidazole (DRB), which had various biological activities including antiviral activity against RNA and DNA viruses. DRB inhibits cellular RNA polymerase II and viral and cellular RNA synthesis.[53] Biologically active compounds such as vitamin B_{12}, albendazole, mebendazole, and thiabendazole contain a benzimidazole nucleus in their structure.[54] Although no studies have been done in birds, there are studies looking at their use in human viral diseases, including poxvirus, HSV-1, HIV, HBV, HCV, human cytomegalovirus (HCMV), and RSV.[55] Future studies evaluating their effectiveness in avian paramyxovirus, poxvirus, or herpes infections are needed.

Herbal/plant Remedies

Medicinal herbs and plants have been evaluated for antiviral activity and some have shown antiviral activity in vitro and in vivo for many viruses including NDV. Alkaloids, saponins, tannins, and flavonoids in plants have been shown to have some antiviral properties. *Cucumis metuliferus* and *Anthocleista nobilis* have shown some antiviral activity in vivo and *Mangrove halophytes*, *Nigella sativa*, *Polysavone*, *Melissa officinalis*, *Momordica cochinchinensis*, *Echinacea purpurea*, *Withania somnifera*, and *Asparagus racemosus* have shown potential to improve immunity in infected birds.[56]

Baicalin (BA; formulated as 7-D-glucuronic acid-5,6-dihydroxyflavone) was purified from the plant *Scutellaria baicalensis Georgi*. It is used as a traditional Chinese herbal medicine. BA is known for its antiviral properties but solubility is poor. To increase solubility, absorption, and pharmacological activity, a BA phospholipid complex (BAPC) was developed and studied in ducklings infected with duck hepatitis A virus type 1 (DHAV-1). Results showed BAPC inhibited DHAV-1 adsorption, replication, and release. Furthermore, it played antioxidative and immunoenhancing roles in the treatment.[57]

BA's antiviral effects were evaluated against avian leukosis virus subgroup J (ALV-J) in duck fibroblast cells. The cells were treated with 5 or 20 μg/mL of BA and DMSO at different time points: prior to, during, and post viral adsorption. Adding BA pretreatment resulted in much lower viral inhibition. It was found that BA could inhibit viral mRNA, protein levels, and overall virus infection in a dose- and time-dependent manner using a variety of assays. The 20 μg/mL was the most effective dose, with higher doses (of 40, 80, and 100 μg/mL) cytotoxicity occurred.[58]

A flavone/polysaccharide-containing prescription was developed and tested for efficacy against duck viral hepatitis (DVH) and investigated for its hepatoprotective effects. Flavones were derived from *Hypericum japonicum* (HJF) and *Salvia plebeia*, and polysaccharides were derived from *Radix Rehmanniae Recens* (RRRP). This prescription combination of *H. japonicum*, *Radix Rehmanniae Recens*, and *S. plebeia* (HRS) was based on theories in traditional Chinese veterinary medicine. In vitro and in vivo experiments were conducted using the three single ingredients compared to the combined HRS prescription to determine their anti–duck hepatitis A viral (anti-DHAV) activity. The results showed that all experimental conditions displayed anti-DHAV activity, but the HRS prescription was most effective.[59]

Antiviral Drug Therapies in the COVID-19 Pandemic

In 2020, severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), a novel coronavirus that was first detected in China in December 2019, led to a global outbreak that became known as the COVID-19 pandemic. Due to the worldwide spread and severity of the disease, several pharmaceutical companies have worked on developing new antiviral drugs as well as repurposing already available antiviral drugs to reduce morbidity and spread of the virus. Drugs targeting SARS-CoV-2 replication include PF-07321332, also known as nirmatrelvir. PF-07321332 is an antiviral drug that inhibits the main protease (Mpro) of coronaviruses that is essential for viral replication and is highly conserved. Nirmatrelvir binds to Mpro forming a reversible bond with the catalytic cysteine residue, Cys145, thereby inhibiting viral replication. Nirmatrelvir has

demonstrated antiviral activity against all known variants of SARS-CoV-2 but has the potential to treat a variety of coronavirus infections, possibly also in other species.[60] Ritonavir (RTV), an oral antiviral medication that was initially developed to treat HIV infections, is a potent CYP3A (cytochrome P450 isoform) inhibitor that aids to slow down the metabolism of nirmatrelvir, thereby increasing its bioavailability. As such, the combined use of these drugs, which are packaged together under the trade name Paxlovid, has been advocated for effective treatment of COVID-19.[60]

Remdesivir is a nucleoside analog that acts as a competitive inhibitor of viral RdRP. It is a prodrug given intravenously and subsequently transformed in the body into an active molecule known as GS-441524. Initially, remdesivir had been proposed for the treatment of Ebola. However, further research showed that remdesivir not only had antiviral activity against Ebola virus but also against MERS-CoV, SARS-CoV, and other coronaviruses, hence making it a possible candidate drug for treatment of COVID-19.[61]

Aforementioned developments in antiviral drug therapies demonstrate that the medical and scientific communities are capable of acting quickly to find effective antiviral strategies in case of a global viral outbreak. Nevertheless, while close monitoring of the developments in the human field may provide useful starting points toward identifying antiviral drugs that may be useful for implementation in viral infections in birds and animals, there are also notable global concerns that the use of antiviral agents in veterinary medicine, particularly in food-producing animals, may lead to the development of resistance to these medications in human pathogens. Hence, several countries, including the European Union, have restricted the use of antiviral drugs in animals.

Conclusion

The most effective strategy against viral disease includes preventing and reducing exposure when possible and vaccination and antiviral drugs (see Section 4: Infectious Disease Prevention). Most antivirals selectively target only one or a few types of virus but broader-spectrum antivirals are being developed.[59] These drugs will cover multiple viruses and genotypes reducing the potential for resistance. In some cases, the combination therapy of vaccines and antivirals may increase survival. The combined use of antiviral agents to target both viral components and cellular machineries may allow for decreased dosing of the individual drugs and improve antiviral efficacy, reduce viral resistance, and minimize toxicity. Synergistic activity between favipiravir and oseltamivir against influenza virus has been documented.[27] The goals for optimal antiviral use in veterinary medicine include effectiveness of treatment, safety, cost effectiveness, and ease of delivery. The needs of aviculture and flock medicine will be different than that of an owner with only one or two pet birds.

Advances in the field of molecular biology and DNA technology, in particular computational approaches, along with the repurposing of drugs and evaluation of the antiviral effects of plants will contribute to the development of a new generation of antivirals including broader-spectrum antivirals, which will be important in the control of many animal diseases, including those encountered in the avian patient.[62] The COVID-19 pandemic revealed how quickly the medical and science communities can react to severe viral threats. In an effort to keep these drugs readily available and effective for human use, current antiviral use in general is frowned upon in veterinary medicine, and its use is banned in poultry. However, this could change when being confronted with a viral outbreak that poses a severe threat to livestock, poultry, or possibly could lead to extinction of an endangered species.

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CHAPTER 11

Interaction of Host, Agent, Environment, and Disease

David N. Phalen

INTRODUCTION

Infectious diseases have been and continue to be major causes of morbidity and mortality in pet and aviary birds. Developing strategies to reduce the impact of infectious diseases requires an understanding of the characteristics of the infectious agent that allow it to cause disease, host factors that may make them susceptible to infection and disease, and environmental factors that predispose to pathogen exposure and host susceptibility. Reducing the impact of infectious diseases also requires an understanding of human behavior. Many effective strategies have been developed that can break infection cycles for specific pathogens in pet and aviary birds. However, these strategies are of no value unless the pet bird owner or aviculturist is willing and able to implement them and often they are not. In these situations, nontraditional or novel means of control are required if control is to be achieved. However, it is a reality that in many bird collections, control of infectious diseases cannot be achieved.

PRIMARY VERSUS OPPORTUNISTIC PATHOGENS

Pathogens can be divided into primary and opportunistic pathogens. Primary pathogens are not typically present in the bird's environment but when introduced to a collection are capable of causing infection and disease. Opportunistic pathogens can be divided into two groups: environmental pathogens and enzootic pathogens. Opportunistic environmental pathogens are ubiquitous and when present in low numbers rarely cause disease on their own. Opportunistic environmental pathogens generally are not passed from one bird to the next. They become pathogens when environmental conditions are optimum for their growth in the environment, resulting in birds being exposed to concentrations of the pathogen that exceed the bird's immune system's capacity to prevent infection. In addition to environmental factors, host factors can predispose birds to infection and disease caused by environmental opportunistic pathogens. Birds whose immune system is compromised by a concurrent infection with an immunosuppressive organism, for example, psittacine beak and feather disease virus (PBFDV), or *Chlamydia psittaci*, are more likely to become infected with environmental opportunistic pathogens. Similarly, birds experiencing stress are more susceptible to opportunistic pathogens. Lastly, naïve species, those that have not coevolved with an opportunistic pathogen, are also at risk of infection and disease by them if these species are added to collections or brought into care in environments that differ significantly from their natural environment.

Examples of environmental opportunistic pathogens are *Pseudomonas aeruginosa*, *Aspergillus* spp. that infect the respiratory systems of birds, and *Candida* spp. that infect the digestive tract of birds. *P. aeruginosa* is a moisture-loving bacterium. It can grow to heavy densities on persistently wet surfaces like hand-rearing implements, bird baths, polyvinylchloride pipes that are used in automatic watering systems and for sprinklers, and sprouted seeds. *Aspergillus* spp. are ubiquitous, but environmental growth is enhanced by warm damp environments. Therefore, their airborne spores reach a higher density in tropical parts of the world where there is a high relative humidity and in warm damp aviaries with poor ventilation. *Candida* spp. cause problems in nestling birds that are improperly managed, finches kept in aviaries where hygiene is poor and where moist foods are left out for many hours, in dense colonies of finches where more timid species are under the constant threat of bullying, birds infected with immunosuppressive circoviruses or *C. psittaci*, and birds whose gut flora has been altered as the result of antibacterial treatments.

Opportunistic enzootic pathogens are pathogens that cause lifelong subclinical infections but only cause disease in birds under conditions of extreme stress. There are not many

documented examples of these in birds, but Psittacid adenovirus 2 is an example of an opportunistic enzootic pathogen, and it is possible that the Gouldian finch adenovirus behaves in the same way.[1] Research has shown that many adenoviruses infect many species of birds persistently and subclinically, but that virus replication increases with resultant disease in some instances, for example, when stressors such as high temperature or co-infection with *Aspergillus* spp. occur.

OPPORTUNISTIC PATHOGENS AND THEIR CONTROL

The key to control opportunistic environmental pathogens is reducing the concentration of the pathogens in the environment and reducing opportunities for them to amplify. How this is done will depend in part on the pathogen. For all environmental pathogens, however, regular cleaning, in particular removing fecal material and uneaten food, is the first step to controlling them. In addition to cleaning, an effective way of reducing contact with feces and spoiled food is to house birds in suspended wire cages and individual pet birds in cages with wire grids at the bottom. The use of medium-sized pebbles or small rocks as substrates in the bottom of bird flights has also worked for some because these allow food to slip between the pebbles and out of reach of the birds in the flight.

Reduction of fecal/oral transmission of environmental pathogens requires the use of appropriate feeding practices and attention to the cleanliness of food and water dispensers. All food and water dispensers should be covered to prevent fecal contamination. Similarly, perches should be arranged in such a way that they are not over food and water dispensers. Many birds will foul their drinking water by putting food in it. When this happens, water containers will need to be changed out more than once a day. It is very common for aviculturists to house finches *(Haemorhous mexicanus)*, canaries *(Serinus canaria)*, budgerigars *(Melopsittacus undulatus)*, and cockatiels *(Nymphicus hollandicus)* in flights and to feed them on tables or wooden shelves where the birds walk through the food and defecate on the food that they are eating. This type of feeding makes control of opportunistic environmental pathogens impossible and should be discouraged in all instances. Another common practice, particularly in finch collections, is to feed soft foods. However, if the uneaten food is not removed from the aviary, it may overgrow with opportunistic pathogens. There are multiple ways to clean and disinfect food and water dispensers using soapy water and disinfectants, but in the author's experience, the most effective and easiest way of preventing pathogen buildup in food and water containers is to run them through a dishwasher. Dishwashers will sterilize the containers, while washing and treating with disinfectants generally does not.

One solution to having to clean water dishes every day is to use an automatic watering system. These are typically fed from a reservoir. In some instances, the reservoir is a rainwater tank that fills from the runoff from a roof. These systems are very effective at providing fresh drinking water but are predisposed to an overgrowth of *P. aeruginosa*. Chlorinating the water in the reservoir or flushing the system regularly with dilute bleach will control *P. aeruginosa* overgrowth. Sprinkler systems used to cool aviaries will also need either a chlorinated water supply or regular flushing with dilute bleach to prevent *P. aeruginosa* overgrowth.

Creating sufficient ventilation in buildings in which birds are housed is essential for multiple reasons. It provides fresh air, can assist with cooling during hot times of the year, and dilutes airborne pathogens. Most importantly, air movement through an aviary reduces the relative humidity and allows the aviary to dry out after cleaning. A damp aviary will support the growth of *Aspergillus* spp. and a lack of ventilation will allow the spore density to rise to dangerous concentrations. Even small enclosures such as brooders and hospital cages can create environments that favor aspergillosis. Often, brooders and hospital cages are designed more to maintain temperature within specific ranges than to facilitate ventilation. Chicks or sick birds fed liquid diets produce moist droppings, as do frugivorous species. If the brooder or hospital cage is not properly ventilated, *Aspergillus* spp. will grow in the areas housing the fan and heating elements, areas that are rarely cleaned.

Also, no matter how good the ventilation is, some environments are not suitable for certain species of birds. Classic examples of birds that cannot be housed in warm and humid environments include the gyrfalcon *(Falco rusticolus)*, penguins *(Sphenisciformes)*, and ostriches *(Struthio camelus)*. Gyrfalcons and penguins must be housed in climate-controlled buildings. Similarly, ostriches do not do well in warm humid coastal environments, and it is not possible to raise emu *(Dromaius novaehollandiae)* chicks in tightly sealed dairy barns with hay used for bedding. Rehabilitation of seabirds poses a number of difficult challenges of which one is infection with *Aspergillus* spp. Even when all housing recommendations are met, and meticulous care is provided, aspergillosis remains a common problem. In these situations, prophylactic systemic antifungal treatment is required to prevent aspergillosis (see Chapter 8).

PRIMARY PATHOGENS AND THEIR CONTROL: BIOSECURITY

Primary pathogens can impact the individual pet bird, small "flocks" of pet birds, and large avicultural collections and are of concern for the avian veterinarian because of the risk of transmission in the hospital. Control or mitigation of the impact of primary pathogens requires a detailed knowledge of the epizootiology of the pathogen, allowing the identification of specific measures that can be implemented to prevent pathogen transmission.

EPIZOOTIOLOGY

The key to mitigating the impact of any infectious disease-causing agent is understanding its epizootiology. The first step to understanding the epizootiology of a primary pathogen is determining how the pathogen is spread. This means that the route of shedding must be determined, including if it can be passed in the egg (vertical transmission), its ability to survive or multiply in the environment, whether it requires or utilizes an intermediate host, by which route it infects the next host, and how many organisms are required to induce infection. Next, it is necessary to know the incubation period between exposure and shedding and what the consequences are of infection (Figure 11.1). Understanding the epizootiology of an infectious disease in one species is complex, let alone when it comes to infectious disease circulation and spread across multiple types of birds as the pattern and

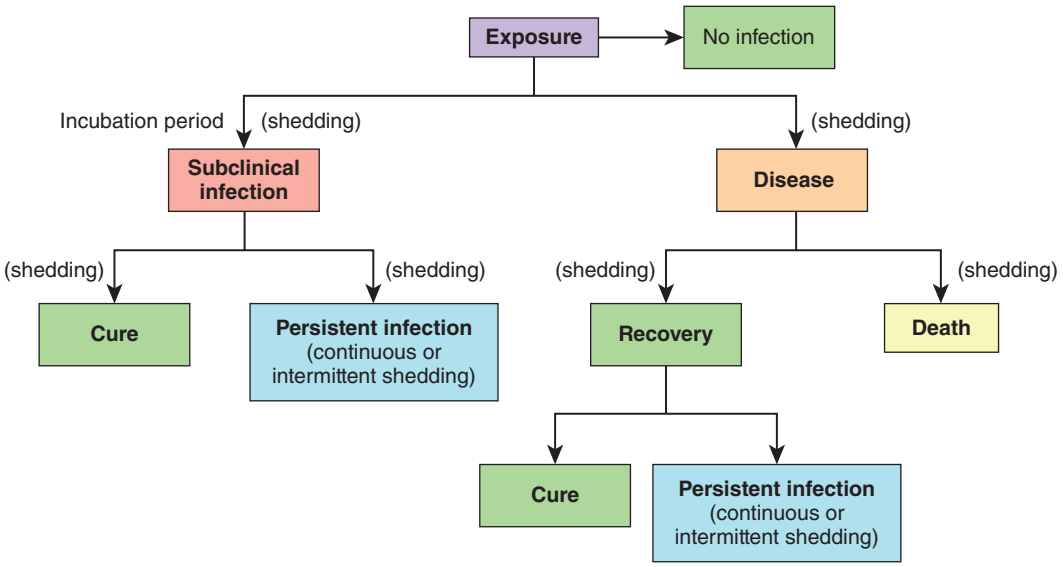


FIGURE 11.1 Possible outcomes resulting from exposure to an infectious disease. Which outcome occurs in the individual bird will depend on the number of organisms to which the bird is exposed, virulence of the pathogen, strain of the pathogen, immune status of the bird at the time of exposure, the species of the bird, and, in some instances, the age of the bird. Knowing when an infected bird will shed the pathogen and for how long will determine what kind of testing and control measures will prevent an infected bird from infecting others, or if control for the infectious agent is even possible.

outcome of infection can vary tremendously. Additionally, the outcome of infections can also be impacted by coinfections with other pathogens. As examples, coinfection with PBFDV can result in avian polyomavirus infection causing death in an adult *Eclectus* parrot *(Eclectus roratus)*, whereas infection in otherwise healthy adult birds is typically subclinical.[2] Similarly, adult lovebirds (*Agapornis* spp.) are more likely to remain persistently infected with and shed avian polyomavirus if concurrently infected with PBFDV. Similarly, they are also more likely to shed *Encephalitozoon hellem* if infected with PBFDV.[3]

In parrots and other species of captively managed birds, the infectious agents that have been the most difficult to control have been those that cause subclinical and lifelong infections in selected individual birds or certain species of birds but are pathogenic in others. Although less common, infectious diseases that spillover from wildlife can cause disease in birds kept in outdoor aviaries. These may result from direct transmission, (e.g., salmonellosis or yersiniosis from mice and rats); vector-borne transmission, (e.g., West Nile virus and poxvirus); or indirect transmission, (e.g., *Sarcocystis falcatula* of the Virginia opossum *[Didelphis virginiana]* that is highly pathogenic to Old World psittacine species).

DISEASE CONTROL TOOLBOX

There are many tools that the veterinarian has to mitigate the impact of infectious disease. Which ones are used will depend on the species of the bird, whether the bird is going into a pet bird home either as an individual companion bird or part of the owner's "flock," or whether it is going into a breeder's collection. Some recommended control measures for specific pathogens are shown in Table 11.1.

GUIDING CLIENTS ON SELECTING THE SOURCE OF THE BIRD

Anyone purchasing a bird should buy it from a breeder that has been recommended by others who have purchased birds from them previously. Also, they should visit the breeder's aviary to make sure that they are happy with the breeder's management. Veterinarians may be able to recommend breeders to their clients and can help the client who wishes to purchase a bird make up a list of questions to ask the breeder and have a checklist of points to consider before purchasing a bird (Box 11.1).

QUARANTINE

Quarantine has proved to be a powerful tool for preventing disease transmission in aviary and pet birds in many settings. Nevertheless, it has significant limitations.[4] The value of quarantine is based on the premise that if birds exposed to infectious diseases are held in quarantine for a sufficiently long period of time, they will either develop manifestations of this infection and not be allowed into the collection or will cease shedding the infectious agent and can be safely released into the aviary. This is true for some infectious diseases, and for these, specific quarantine periods can be developed to prevent their introduction to a bird collection. An example of this is the avian polyomavirus. This virus is typically shed for 3 months following infection.[2] However, quarantine will not prevent birds that are persistently and subclinically infected from introducing a pathogen into an aviary, so additional biosecurity measures are required in all instances in addition to quarantine. Examples of important pathogens that cannot be excluded from an aviary by quarantine alone include avian bornaviruses, *C. psittaci*, and

TABLE 11.1

Control Options for Selected Primary Infectious Diseases of Importance to Pet and Aviary Birds and Their Likelihood of Success

	Quarantine/ Closed Aviary	Physical Examination and Nonspecific Testing	Testing	Treatment	Artificial Incubation of Eggs	Vaccination	Vermin/Vector Control
Adenoviruses[1] (pigeons, psittacine birds, possibly others?)	No	No	Test available, unknown specificity	No	Possible, but unknown	No	NA
Avian polyomavirus[3] (psittacine birds)	Yes	No	Yes	No	Possible, but unknown[a]	No	NA
Avian bornavirus[5] (psittacine birds)	No	No	Likely with repeat testing	No	Viral RNA found in eggs[b]	In development	NA
Psittacine beak and feather disease virus (PBFDV; psittacine birds)	No	Some birds show signs, many do not	Quality test but infected birds not always positive	No	Viral DNA found in eggs[b]	In development	NA
Pigeon circovirus[6]	No	No	Test available, unknown specificity	No	NA[c]	No	NA
Pigeon paramyxovirus	No	No	No	No	NA	Yes	NA
Poxviruses (canaries *(Serinus canaria),* pigeons, poultry and other bird species)	No	Signs are characteristic	No	No	NA	Yes	Yes—canaries
Psittacid herpesvirus 1[7]	Yes	Most do not show signs, some will have mucosal papillomas	Yes	No	Possible, but known	No	No
Columbid herpesvirus	No	No	Test available, sensitivity unknown	No	NA	No	No
Chlamydia psittaci	No	Nonspecific signs and hematological findings, not present in all birds	Yes	Yes	Possible, but unknown	No	No
Mycobacteria spp.	No	Some but not most birds, nonspecific signs	Tests not widely available and not sensitive	No	Possible, but unknown	No	No
Macrorhabdus ornithogaster[8]	No	Some but not most birds, nonspecific signs	Testing is specific, but not sufficiently sensitive	No	Yes	No	No
Sarcocystis falcatula[11]	No	No	No	No	NA	No	Yes
Helminths	Yes	No	Yes	Yes	Yes	No	Yes

[a]One paper suggests vertical transmission, but it is possible that had the eggshells been removed after hatch, that infection may not have occurred.
[b]Viral RNA (bornavirus) and viral DNA (PBFDV) found in a subset of eggs from positive hens. It is uncertain whether this would have resulted in an infection in the chicks.
[c]*NA,* Not applicable. Artificial incubation is rarely used in racing pigeon *(Columba livia domestica)* lofts, so this option was not included.

BOX 11.1 QUESTIONS THAT COULD BE ASKED OF A BIRD BREEDER PRIOR TO PURCHASING A BIRD FROM THEM[a]

- How long have you been breeding birds?
- What species do you breed?
- Would it be possible to do a walkthrough of the collection and see the nursery?
- Do you work with a veterinarian?
- What kind of preventative medicine program do you have?
- Do you quarantine incoming birds? If so, can you describe the procedure that is followed?
- If a bird dies in the collection, are postmortem examinations routinely done?
- Would you be able and willing to give me the names of some of your clients that could serve as a reference for you?
- Was the bird artificially incubated, or incubated naturally?
- Is the bird hand-raised or parent-raised?
- Is the bird fully weaned?[b]
- Are the parents related? (Especially relevant with color mutations)
- What are the parents fed?
- What breeding records do you keep, and can I inspect records relevant to my purchase?
- If a young bird, do you have a growth chart for the bird, and can I see it?
- Has there been any effort to socialize the bird? If so, how?
- What is the bird being fed?
- Is there a guarantee so that a bird can be returned if a problem is found during the first Vet Check? What procedure should be followed in this case?

[a]This list may not apply to all situations. A discussion between a client and their avian veterinarian prior to meeting with the aviculturist may help to refine this list or identify additional pertinent questions to ask.
[b]In many countries it is illegal to sell unweaned birds. Even in countries where the sale of unweaned birds is legal, buying an unweaned bird is generally not recommended for inexperienced owners or handfeeders.

PBFDV. Detecting subclinically infected birds with these pathogens requires that birds be tested for them.

EXAMINATION AND NONSPECIFIC TESTING

Ideally, all newly acquired birds would be examined by a veterinarian as soon as possible after acquisition and before they are in contact with any other birds in the owner's home or aviary. A thorough history and physical examination coupled with a complete white blood cell count, serum biochemistries, wet preparations, Gram stains, and quick stains of crop swabs and feces or cloacal swabs will identify many unwell birds and birds with manifestations of infectious diseases.

TESTING FOR SPECIFIC PATHOGENS

There are an increasing number of infectious agents that can be detected in subclinically infected psittacine birds with targeted tests. The main purpose of testing a newly acquired individual pet bird is to make sure that it is healthy and not carrying an infectious agent that will subsequently make it, other birds, or the owner ill. Depending on the bird's signalment, history, and disease prevalence in your region or country, it may be appropriate to test new birds for one of more of the following: PBFDV (circovirus), polyomavirus, avian bornavirus, Psittacid herpesvirus 1, *C. psittaci*, or *Macrorhabdus ornithogaster*. Testing birds going into a home or an aviary where there are multiple other birds often requires more extensive testing (Table 11.1). Which tests are appropriate from new birds entering a multiple bird aviary will depend on the species of birds in the collection, why these birds are kept, and the disease status of the collection, with final selection being done in consultation with the bird owner or aviculturist.

Testing is not a panacea and there are limitations to every type of test. For example, tests that detect an organism may yield false-negative results if the bird is recently infected and not yet shedding the organism.[2,5] Recent antimicrobial treatment can also interfere with polymerase chain reaction (PCR) assays to detect *C. psittaci*. Similarly, if a bird is only intermittently viremic, or bacteremic, or intermittently sheds the organism, infected birds can be missed. In such instances, repeated testing may be indicated. Alternatively, in some settings, holding a bird in quarantine for 2 weeks prior to testing may allow an infection that occurred in the bird near the time of purchase to progress to a point where the pathogen can be detected.[4]

Lastly, we cannot test for those infectious diseases that we do not know are there. Newer techniques such as next-generation sequencing and other molecular technologies are finding new potential pathogens all the time. Many of these are viruses, but it is often uncertain if these actually are pathogenic or not. In some instances, such microorganisms have been found to be widespread in collections, probably for many years, without evidence that they were causing disease. Two examples of viruses that appear to be widespread in avian collections but are not or are rarely associated with disease are Psittacid adenovirus 2 and the avihepadnaviruses.[1,6] In addition, commercially available testing is often not available for many rarely identified infectious diseases and newly discovered infectious diseases.[1,6]

DISEASE PREVENTION THROUGH TREATMENT

There are few instances where "prophylactic" treatment of birds is indicated. However, this is commonly done by aviculturists and is encouraged by some vendors who sell water-soluble medications. Treatment should be evidence based, whenever possible. This means that a pathogen should be identified in an animal before it is treated, and if the pathogen is a bacterium, then the treatment should be selected on the results of a culture and sensitivity, whenever possible (see Chapters 6 and 7). The only time that treatment might be appropriately used as part of a disease control measure is when attempting to prevent the introduction of parasites into collections. In the prepatent stage of development, nematodes may be present and not be shedding eggs, which may lead to these infestations being easily missed on a fecal examination. Similarly, a bird may be infected with tapeworms,

without eggs being detected in a fecal examination. If deworming is to be done as a "prophylactic" treatment, then follow-up fecal examinations are recommended, as resistance to antiparasiticides can develop (see Chapter 9).

MANAGEMENT PRACTICES THAT CAN REDUCE THE IMPACT OF INFECTIOUS DISEASES IN AVIARIES

The Closed Aviary Concept

The closed aviary concept is a management concept centered on the management of traffic flow of birds, people, and fomites into and within an aviary once the collection has been established (Figure 11.2). Using this concept, management factors can be used to minimize the risks of infectious disease to bird collections. The criteria for movement into, out of, and between segments of the closed aviary are established through careful veterinary consultation and are aimed at the primary goals of the aviculturist. These criteria may range immensely from an absolute rule of no new birds to be introduced to the facility (nor birds returning to the facility once they have left the premises), breeding stock replaced only with offspring from the current breeding stock or specific quarantine timelines and testing criteria prior to (re)introduction of (new) birds to the facility, to mere observational assessment and entry of new birds to the facility with no specific testing procedures required.[6] Under ideal circumstances, specific primary pathogens would also have been eliminated from the aviary as a result of routine testing, treatment, or management practices, resulting in a specific pathogen-free flock.[4] Aside from control of bird movements, human traffic in and out of the closed aviary would also be controlled. Under ideal circumstances, visitors would not be allowed into the aviary at all, and employees would not have contact with birds when off the property.

Unfortunately, even though the modular concept of the closed aviary and its management can be modified, adjusted, and fine-tuned to meet the needs of the collection and aviculturist, acceptance to standardization of traffic flow still remains inconsistent in aviculture. Standards may be set too strictly, an infusion of new birds will generally be necessary to maintain genetic diversity, procedures may carry unacceptable costs, testing procedures selected may not carry an adequate sensitivity, or even a true understanding of the concept may not be fully present.

Species Composition

A general rule of thumb is that the more species held in an aviary, the more likely it is for infectious disease transmission to occur. Minimizing species diversity in an aviculture collection has additional advantages besides disease prevention, including allowing the aviculturist to become an expert in the species that they are raising, typically resulting in increased breeding success and an improved quality of life for their birds.

There are certain species that are known to be commonly and subclinically infected with important primary pathogens. Based on the author's experience, aviculturists who breed high-value birds often enjoy breeding other species like budgerigars, cockatiels, and lovebirds, even bringing chicks from

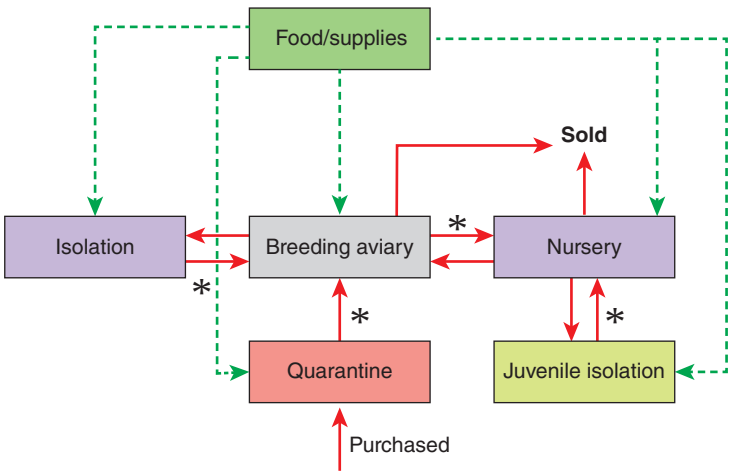


FIGURE 11.2 The closed aviary concept utilizes controlled traffic flow to reduce variables, including but not limited to infectious agents into or within a flock. The modular units of quarantine, the breeding aviary, nursey, isolation, and nursery isolation are depicted, as well as the separate source of food and supplies. Because of its modular concept, there can be multiple quarantines, breeding areas, nurseries, etc. Depending on their purpose, not all collections have to have a breeding aviary or nursery, as not all are focused on breeding. Independent traffic flow of food and supplies to these areas is shown (*green arrows*), not through multiple components. Potential movements of birds are shown with *red arrows*. Areas of key interest for management of pathogen movement are marked with *stars*, and these areas are where management rules, procedures, or protocols will have their greatest merit in strengthening the impact of the use of the concept. Another unique aspect of the closed aviary concept is that the rules or protocols of controlled bird movement are specifically tailored to serve the mission and purpose of the facility, and what is known about the bird species involved as well as the pathogens of interest that are to be managed.

these species into their nursery. This is a recipe for disaster and has resulted in many disease outbreaks resulting from introduction of avian polyomavirus and PBFDV. If aviculturists or institutions want to keep many different species, then housing different species in separated areas in the facility may reduce the risk of disease transmission.

It is a widespread practice for aviculturists to house small species of parrots such as Bourke's parrots (*Neopsephotus bourkii*) and scarlet-chested parrots (*Neophema splendida*) in planted aviaries together with canaries and other finches, buttonquail (*Turnix* spp.), and Japanese quail (*Coturnix japonica*). However, how diseases spread back and forth between these species is largely unknown, thus hindering the design and development of preventive and treatment protocols for such aviaries. Zoos and other institutions that display birds for the public also tend to have mixed collections of birds housed together. In many instances, these are birds from similar environments and the same part of the world, but in others, species from many different geographic origins are housed together. Again, in these instances, disease control will be challenging or even impossible to achieve.

Aviary Design and Workflow

Aviary design can also be used to reduce disease transmission within a bird collection. Birds housed outdoors in separate cages are less likely to transmit diseases because of the dilution effect of wind and the sterilizing effects of the sun. In contrast, birds housed indoors in adjacent flights with a shared wire wall are much more likely to transmit disease.

Keeping birds in outdoor aviaries can also have a number of drawbacks. These aviaries provide an increased chance of contact between captive birds and wild birds. This often requires trade-offs in aviary design between aviaries where birds are exposed to sunshine and rain but have an increased exposure to wild birds, on the one hand, and aviaries that are fully covered by a roof and partially enclosed to keep wild birds from entering and/or defecating into the aviary, on the other. The author has observed many instances where aviary birds were housed in suspended wire cages and wild birds were seen to be feeding on food that fell through the bottom of the cages. This food will also attract mice, rats, and flies. In North America, it can also attract Virginia opossums, thereby increasing the risk for infection with *Sarcocystis*. In large fenced-in aviaries in North America, having a dog free-ranging within the aviary can help to reduce Virginia opossum numbers and also problems with raccoons (*Procyon* spp.). Similarly, allowing chickens (Gallus gallus domesticus), ducks (Anatidae), or guinea fowl (Numididae) to forage under suspended wire cages will reduce the amount of food available to vermin. Vermin control in any aviary, particularly in outdoor aviaries, is essential. It is the author's impression that *Salmonella* outbreaks are more likely to occur in aviaries where mice or rats are in high density. The presence of mice and rats can also be disruptive to breeding birds at night.

Designating specific areas of an aviary to specific purposes, and controlling movement between these designated areas, may reduce the chance of disease movement within a bird collection.[4] Large aviaries can be divided into different functional areas such as the food preparation area, nursery, replacement stock holding facility, main breeding facilities, cleanup area, and quarantine. Ideally, all food preparation would be done before moving into other parts of the aviary, and dirty food and water bowls would be disinfected at a site separate from the food preparation area. Similarly, chicks would only be transferred from the breeding facility into the nursery and never return into the nursery or other parts of the facility once they left, with care for these chicks ideally being done by people who do not work with the breeding stock. Birds in quarantine would either be cared for after care of all other birds is finalized or done by designated staff who have no contact with other birds in the facility. These practices can generally only be applied in full in large collections of birds with multiple staff. However, with some degree of innovation, modifications of these practices can be applied even in small collections. For instance, people caring for the major collection could also care for nestlings if they practice adequate hygiene (e.g., wash hands) and change their clothes or put on coveralls and shoe covers when going into the nursery (and do the same when leaving the nursery again).

Incubator Hatching Eggs

Pulling eggs from adult birds and incubator hatching may result in the interruption of disease transmission from adults to chicks (see also Section 9: Embryology, Neonatology, and Pediatrics). This can be assumed to be the case for parasitic diseases, most bacterial diseases, and with *M. ornithogaster*.[10] How effective it will be with viral diseases is less certain and may depend on whether the eggs are cleaned, if chicks are separated from the shells and put in a clean environment as soon as they hatch, and if viruses are vertically transmitted through the egg.

Hygiene and Disinfection

The same principles regarding hygiene and disinfection apply for primary pathogens as they do for opportunistic pathogens. Diluting pathogens by cleaning reduces the risk that a bird will be exposed to an infectious dose. Disinfection (see Chapter 12) of food preparation and food storage areas, handfeeding utensils, food and water bowls, breeding boxes between breeding seasons, and the nursery after breeding will decrease the chance of the transmission between birds in a bird collection. Hand washing or sanitization and daily washing of work clothes should also be included in a disease prevention program. Similarly, cleaning and disinfecting cages and other surfaces in a veterinary hospital needs to be done multiple times a day to prevent the spread of contagious diseases.

Disease Monitoring

An important part of disease control in a collection is keeping records of the disease problems the collection has experienced. Aviculturists often think that they know what is causing disease in their birds, but they may be wrong. It is therefore highly recommended that necropsies be done on a representative number of birds that die in a collection and on all birds that die unexpectedly. How many birds and which birds that die that should be necropsied would be based on discussions between an aviculturist and their veterinarian. It is these patterns of morbidity and mortality noted in an established flock that can guide to changes in the details of closed aviary management concepts, as well as population management recommendations overall.

MITIGATING THE IMPACT OF DISEASE IN SELECTED CIRCUMSTANCES

Minimizing Infectious Disease Problems in the Pigeon Loft

A protocol for mitigating the impact of disease in the pigeon loft and protecting pigeons *(Columba livia domestica)* from common infectious diseases can be found at https://www.auspigeonco.com.au/rdquoone-loft-racerdquo-protocol.html. This protocol was developed by Dr. Colin Walker, an experienced pigeon veterinarian and a racing pigeon enthusiast. Recommendations include a combination of tools. Dr. Walker recommends a brief quarantine period that allows the owner to examine their new birds for signs of illness and treat for common parasitic diseases, including *Trichomonas gallinae*, coccidia, roundworms, and tapeworms. Birds exhibiting signs of disease are sent back to the breeder. During the quarantine period, pigeons can also be tested for pigeon circovirus infection (assays are available from multiple commercial laboratories) and adenovirus infection (https://www.cd-diatest.com/cdia-sup-tm-sup-pigeon-adenovirus-antigen-test-kit_p542.html). Based on research in Europe and the author's experience, pigeon circovirus is enzootic in Europe and Australia,[6] so testing for it may not be useful as most fledglings will be infected. Adenovirus testing may also not be beneficial as adenovirus infections are likely to be widespread and, at least in the author's experience, rarely cause disease. Columbid herpesvirus is also widespread in feral and captive-raised pigeons and testing for it has little merit, especially as it only appears to cause disease in immunosuppressed birds. Pigeon pox and pigeon paramyxovirus are controlled by immunization.

These recommendations may benefit some pigeon breeders, but it is impossible for racing pigeon lofts to be maintained as closed colonies. Encounters with feral pigeons and pigeons from other lofts occur when birds are released to exercise. During races, pigeons are held the night before the race in proximity with hundreds of other birds and may share water sources, creating opportunities for disease transmission between birds. Also, to maintain the best breeding stock, pigeon breeders often purchase new birds on a regular basis. Excess birds from their loft are sold to others, often through a pigeon auction where there will be many pigeons from multiple aviaries held in relatively close quarters. The ability of viral diseases of racing pigeons to rapidly disseminate across large geographic areas has been well demonstrated in Australia where both pigeon paramyxovirus and then subsequently rotavirus spread across the entire continent from a single point source in 1 year. Therefore, no matter what biosecurity efforts undertaken by racing pigeon fanciers, disease will always be a potential problem.

Preventing Primary Disease Outbreaks in a Rescue Center

Rescue centers for unwanted pet and aviary birds have been established all over the world. Controlling diseases caused by primary pathogens in these settings is challenging, given that there is a constant flow of birds in and out of these facilities; birds are generally a mix of species and they come from many different sources. In these high-risk environments, disease control requires careful examination and nonspecific testing of every bird coming into the facility, testing for specific diseases, and judicious use of quarantine until the disease status of newly acquired birds can be determined. Often, it is necessary to specifically test birds for PBFDV, *C. psittaci*, Psittacid herpesvirus 1, and avian bornavirus. Birds infected with *C. psittaci* can be treated and retested. Birds infected with Psittacid herpesvirus 1 or avian bornavirus can be housed together in a separate location or sent to a home where they are a sole pet or are part of a flock that contains other positive birds. PBFDV–positive birds must be housed away from other negative birds, or, if showing signs of disease, euthanasia may be elected as the best option.

Biosecurity in the Veterinary Hospital

Bird clinics and hospitals can provide an ideal place for disease transfer between patients. Many patients seen by avian veterinarians are either sick with an infectious disease or are subclinically infected with one. Additionally, many veterinarians board healthy birds in their hospitals that could be exposed either directly or indirectly to sick patients, and it is common for wild birds to be seen at veterinary hospitals, creating additional opportunities of disease transmission between wild and client-owned birds.

To minimize the risk of disease transfer in the hospital, a designated isolation ward needs to be available for all birds suspected of having an infectious disease, and policies need to be in place to prevent movement of an infectious disease on clothing, food bowls, or waste out of the isolation ward and into other areas of the hospital. Appropriate ventilation, in both the isolation area and the rest of the hospital, reduces the risk for disease transfer. Wild birds should not be housed in the same ward as client-owned animals, and boarding birds should not be housed in the same ward as hospitalized patients. Many veterinarians require that boarding birds be recently tested for known pathogens such as PBFDV and *C. psittaci* before they can be boarded. Cleaning and disinfection of all aspects of the avian hospital is also a required routine as birds tend to be messy and generate particulate matter that can readily contaminate the hospital. Cage design can also reduce the risk of disease transmission. All cages should have solid walls separating patients from each other and be made of impervious substances that are readily cleaned. Some sophisticated housing units with negative airflow have been used by avian veterinarians and could be considered for use in a specialist avian hospital. Constant attention to hygiene and disinfection is required in treatment areas of the hospital. Swabbing the hospital twice a year and submitting the swabs for molecular testing for, minimally, the beak and feather disease virus will document how successful cleaning and disinfection efforts have been.

Antimicrobial-resistant bacteria including *Escherichia coli*, *Staphylococcus aureus*, *Enterococcus faecium*, and *Enterococcus faecalis* are major sources of nosocomial infections in humans. Once established in a hospital, they can be very difficult to control. Antimicrobial-resistant bacteria can also establish themselves in veterinary hospitals (see Chapter 6). Therefore, all bacterial infections suspected of being acquired in hospitalized patients should be cultured, the organism identified,

and its susceptibility to antimicrobials determined (see Chapters 5 and 7). Repeated detection of the same organism with the same antimicrobial resistance pattern is a call for alarm and should trigger steps to identify and eliminate its source.

The Role of Education in Biosecurity

Talks on biosecurity and diseases of cage and aviary birds are popular with local bird clubs and at regional and national bird club meetings. I recall giving one on biosecurity many years ago, after which I overheard one of the attendees saying, "Man, I wondered when he was going to stop banging on about biosecurity. After all, I quarantine all new birds coming into my aviary, especially if they are sick." This was a bit humbling at the time, but it taught me some lessons. First, no matter how hard veterinarians push clients to improve their biosecurity, they will only do what they consider to be feasible, and this will largely be based on their past experiences and the tendency of all people to be optimists. Secondly, I have learned that comprehensive disease control issues are complex and that they may be difficult to explain. Therefore, focusing on practices that are the most easy and cost-effective to implement is likely to get the best uptake by the audience. Lastly, aviculturists come and go and there are always new ones just starting out. Therefore, continuous education about biosecurity is required, so that those just starting to build their collections do not make the same mistakes as those who proceeded them.

When Traditional Biosecurity Measures Are Not an Option

The infectious disease prevention methods outlined in this chapter cannot be implemented in some bird collections. This is particularly the case for breeders of racing pigeons (discussed earlier) and breeders of various species of birds that are routinely taken to bird shows. Aviculturists breeding show birds must take their birds to shows, where they are exposed to other birds, and return them back to the aviary multiple times during the year, creating opportunities for disease transmission. Most also buy and sell birds regularly. The use of quarantine and, in most instances, any form of testing is rarely practiced in these collections. Additionally, these birds are typically bred in relatively close quarters, and it is difficult to keep these breeding facilities clean, allowing disease spread between adult birds and disease spread to and between nestlings. All these issues are further complicated by a lack of uptake of even basic biosecurity measures by some aviculturists. Because of these challenges, viral, fungal, bacterial, and parasitic pathogens are enzootic in many collections of show birds and small hobbyist collections. Under these conditions, efforts to control diseases are largely done through flock treatment. Treatment typically has only limited short-term and no long-term effect because many of the drugs used are not effective against the pathogen being treated, medication is administered improperly or for an improper duration, and many pathogens are resistant to antimicrobials to which the aviculturists have access. Moreover, there are only limited control options for most viral diseases (see Chapters 10 and 13).

In the end, the only way pigeon breeders and other aviculturists are going to control infectious diseases that are enzootic in these species is to breed for resistance to the common and most significant ones. Based on conversations that the author has had with budgerigar breeders and his own experience, breeding for disease resistance is possible in some circumstances. In Australia, Europe, and possibly other countries, the most common cause of feather dysplasia of the flight feathers in nestling and fledgling budgerigars is PBFDV. Based on anecdotal information that I have received, certain color types appear to be much more likely to develop this disease than others, suggesting a genetic predisposition for the development of disease and genetic factors that might protect from disease. Similarly, if flocks of budgerigars are screened for *M. ornithogaster*, some birds will be found to have extensive colonization of the stomach, while others will have low-level infections or not be infected at all. Given aviculturists' talent for manipulating the genetics of their birds to achieve desired conformation and color mutations, selecting for disease resistance seems like a goal for which they could strive and that perhaps the veterinary profession could encourage.

CONCLUSION

Infectious diseases continue to cause morbidity and mortality in companion and aviary birds. Based on the knowledge of the epizootiology of these infectious diseases, control measures are available for many. These control measures have the greatest use when applied to the individual pet bird. Application in the avicultural setting is more difficult and requires tailored approaches that will depend on the resources and objectives of the aviculturist. Even with this knowledge, the use of biosecurity measures even at the most rudimentary levels is often not an option for many aviculturists and hobbyists. In the future, aviculturists and veterinarians, working together, can work to develop strategically designed novel approaches to disease control that may be more widely practiced.

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CHAPTER 12

Hygiene and Disinfection

David N. Phalen

The combination of cleaning and disinfection is a two-step process that is a critical element of disease control for both opportunistic and primary pathogens in the veterinary hospital, aviary, and home environment. Cleaning (sanitation) refers to reduction of the amount of contamination in an environment, but unlike disinfection, which refers to destroying (-cide) or irreversibly inactivating (-static) pathogenic bacteria and viruses, it does not eliminate all infectious agents. Cleaning must always be done first to remove all organic debris, as disinfection is only possible after cleaning has been done. Overall, this process is not rocket science but is often done poorly, and if done poorly is ineffective. Few people enjoy cleaning. Therefore, in veterinary hospitals, pet stores, and small as well as large bird collections, it is important not just to provide protocols for cleaning and disinfection but also to teach staff about the importance of good hygiene, regularly monitor the facility to make sure that protocols are being followed, and to provide positive feedback when they are.

WHAT SURFACES CAN YOU CLEAN AND DISINFECT?

Only surfaces that are impervious to water and completely free of organic material can be disinfected. Therefore, it is possible to disinfect sealed countertops and fronts, stainless steel surfaces, wire cages, many floors, walls that are tiled or made of other impervious surfaces, and plastic or metal chairs. It is not possible to disinfect cloth-covered chairs, unsealed wood, soil, or living plants. Disinfecting rocks or rough concrete surfaces can also be challenging. Wood and concrete surfaces can be painted over with water-resistant paints and then these surfaces can be properly disinfected. This means that most surfaces in a veterinary hospital can be disinfected. It also means that in most aviaries only the kitchen and the nursery can be adequately cleaned and disinfected, and in peoples' homes, only the kitchen can be disinfected. Most cages designed for pet birds can be disinfected, but to do so, they need to be largely disassembled, scrubbed, and hosed down to be thoroughly cleaned. Wooden perches cannot be disinfected and should be replaced regularly.

CLEANING AND DISINFECTION

Cleaning is generally done in two steps. The first step is to remove as much of the loose organic material as possible. To do this, floors are typically swept, dry mopped, or vacuumed. If brooms or dry mops are used, then they also need to be regularly cleaned and disinfected. In the veterinary setting, cleaning and disinfecting brooms and mops is recommended every day. Only brooms that have rubber or plastic bristles should be used. These can be cleaned in the sink and then soaked in disinfectant, or they can be run through a dishwasher. Dry mops should be washed in a washing machine and dried every day. Vacuum cleaners are also efficient for removing loose material. They should have a high-efficiency particulate absorbing (HEPA) filter to prevent aerosolization of pathogens. The head of the vacuum cleaner should be cleaned and disinfected every day that it is used.

The second step in cleaning is to remove organic matter using a detergent solution. Cages, countertops and fronts, and examination tables are generally cleaned with disposable paper towels. To reduce waste, they can also be cleaned with cloth towels of the appropriate size that are used once and laundered. Sponges should never be used as they cannot be disinfected. Given the high levels of waste generated by birds, including powder down in some species of birds, cleaning bird cages and the ward that they are in is difficult. Solid surfaces are relatively easy to clean, but the bars on the front of a typical dog or cat cage or the bars on a traditional bird cage are not. In addition to the individual cage, banks of cages need to have their tops, sides, backs, and underneath surfaces cleaned, as well as the walls behind and to the sides of them and the floor underneath them. Filters in air handling units also need to be replaced regularly.

Cleaning feeding dishes, water bowls, perches, and other cage furniture is best done by removing organic material by cleaning in a sink with detergent followed by additional cleaning and sanitizing them by running them through a dishwasher. Dishwashers reach a high level of sanitation, one that should be acceptable under most circumstances. In the United States, dishwashers should meet the National Sanitation Foundation/ANSI Standard 184. Commercial dishwashers used in restaurants are effective at sanitizing their contents and have the added advantage of having shorter cycles. Sinks used for cleaning dishes can rapidly become contaminated with organic material and need to be aggressively cleaned and disinfected after use. Dishrags and towels should not be used to clean dishes unless they are washed after every use. Plastic scrub brushes can be used but should be run through the

dishwasher between uses. Single-use plastic or metal scrubbing pads can be used, as long as they are discarded after a single use. Soaking water and food bowls and feeding tubes in a disinfectant may also disinfect them but is generally not effective as soaking solutions are often not changed regularly, and the disinfectants in them may be inactivated by detergents and remnant organic material. Ideally, the cleanup area would be separated from the food preparation area.

Towels are used extensively in avian practices to restrain birds, for cleaning surfaces, and occasionally as bedding. Disinfecting towels and other items made of cloth can be challenging. Washing towels or other fabrics in a washing machine is the most commonly used way to clean and disinfect them. To make this method most effective, they should be washed using hot water and washed with a powdered detergent containing activated oxygen bleach.[1] Washing machines with steam cycles may more effectively disinfect laundry than those that do not have this function. Even with these precautions, heavily soiled fabrics may contaminate other fabrics in the same load and may not be fully disinfected. To minimize this chance, heavily soiled fabrics should not be washed with other fabrics, should be washed in small loads, and should be washed twice. Alternatively, heavily soiled towels and other fabrics can be discarded. Washing machines may not be able to inactivate nonenveloped viruses; therefore, towels used to handle birds with virus infections such as the beak and feather disease virus should also be discarded or soaked in bleach after washing and washed again.

Metal and rubber feeding tubes are particularly difficult to clean and disinfect as food can dry inside them, and feeding tubes are slender enough that they fall through the cutlery basket in a dishwasher. To effectively disinfect them, they need to be flushed thoroughly with water containing detergent. They can then be sanitized using the dishwasher. Plastic mesh bags can be used to hold the feeding tubes in the cutlery basket. Not all rubber feeding tubes will tolerate the dishwasher, requiring other means of disinfection. Also, feeding tubes are often in high demand. Therefore, after cleaning, flushing the tubes with alcohol or another disinfectant and allowing them to dry may be a more practical means of disinfection.

Stethoscopes and focused light sources should be wiped with a paper towel soaked with disinfectant between uses. Computer keyboards and handheld electronic devices should also be cleaned and disinfected by wiping with a detergent-soaked paper towel followed by a paper towel soaked in disinfectant. Otoscope cones and oral specula should be washed with detergent after use and then disinfected.

Floors are cleaned using mops and a detergent solution. Mop water gets dirty quickly and needs to be changed regularly; otherwise, the mop serves to move pathogens around rather than reduce their density.[2] Studies suggest that there is little benefit to using disinfectants in the mop water and that soapy water is sufficient to reduce populations of surface bacteria on floors.[3] Flooring is difficult to keep clean especially in heavily used areas. It is recommended that consultation and treatment rooms be mopped with soapy water during breaks in the day (treatment area) or between blocks of consultation periods (consultation rooms). Most mop heads are made of cloth and therefore need to be cleaned and disinfected after every use. The most effective way of doing this is to run them through a washing machine and dryer. Alternatively they can be soaked in freshly prepared diluted bleach.

Microfiber multiple-use and single-use mop heads have been developed and appear to be highly effective at removing dirt and reducing bacterial loads. Current data suggest that clumping of the microfibers occurs in the reusable mop heads after cleaning and may interfere with their function.[4] Therefore, it is recommended that only the single-use microfiber mop heads be used.

Footbaths containing disinfectants are commonly used at the entrance to quarantine rooms or facilities. As most disinfectants are partially or completely inactivated by organic material and footbaths become contaminated quickly, they also quickly lose their effectiveness, even if the disinfectant solution is changed regularly. Using foot covers or changing into boots at the entrance of the quarantine room may be a more effective means of preventing pathogen movement in and out of a quarantine facility.

An emerging disinfectant method that can be applied locally to high touch surfaces and to entire rooms is the use of UVC light–emitting devices (e.g., https://xenex.com/solutions/). These devices rapidly disinfect surfaces and can replace some manual disinfectant protocols or be used to supplement them.[5] They come at a considerable cost and may not be financially viable for a small veterinary hospital but may be cost-effective in larger practices.

Cleaning, disinfection, and sterilization of surgical instruments and endoscopes is outside the remit of this chapter. For details and guidelines on sterilization and disinfection of instruments, see https://www.cdc.gov/infection-control/hcp/disinfection-and-sterilization/index.html.

SELECTION OF DISINFECTANTS

There are many disinfectants, and each has a range of efficacies against different pathogens. They also differ in the amount of time that they have to be in contact with an infectious agent to inactivate it, their corrosiveness, their ability to irritate skin, how long they remain effective after dilution, and the impact of organic material on their effectiveness. Because of the number of pathogens that might be encountered in an avian practice, particularly practices that see wild birds, only disinfectants that are effective against gram-positive and gram-negative bacteria, mycobacteria, enveloped and nonenveloped viruses, fungi, and bacterial spores will be considered here. It is assumed, but not proven, that these disinfectants will also effectively kill rickettsiae, mycoplasmas, and *Chlamydia psittaci* elementary and reticulate bodies. Commonly used disinfectants will not inactivate cryptosporidia oocysts, but it is thought that thorough cleaning is sufficient to reduce the number of cryptosporidia oocysts to below the infectious level.[6] Comprehensive reviews on the efficacy and use of disinfectants have been developed by the Centers for Disease Control and Prevention (CDC; Guideline for Disinfection and Sterilization in Healthcare Facilities; (https://www.epa.gov/pesticide-registration/antimicrobial-pesticide-registration).

SPECIFIC DISINFECTANTS

Four categories of disinfectants with viricidal, bactericidal, fungicidal, and sporicidal activities are discussed in the following (Table 12.1). It is difficult to compare the efficacies of

TABLE 12.1

Overview of Common Disinfectants, Their Mode of Action, and Activity Against Specific Pathogens

						Halogens		Oxidizing Agents Peroxygens			
Type of Disinfectant	**Acids**	**Alcohols**	**Aldehydes Glutaraldehyde Formaldehyde**	**Alkalis**	**Biguanides-Chlorhexidine**	**Chlorines**	**Iodophor Solutions**	**Accelerated Hydrogen Peroxide**	**Potassium Peroxymonosulfate**	**Phenolic Compounds**	**Quaternary Ammonium Compounds**
Examples	Hydrochloric acid Acetic acid Citric acid	Ethyl alcohol Isopropyl alcohol	Formula H	Sodium hydroxide Ammonium hydroxide Sodium carbonate	Hibitane Nolvasan Savlon	Sodium hypochlorite Bleach	Betadine Povidone iodine	Rescue	Virkon S	Lysol Synphenol	Benzalkonium chloride F-10 (+biguanide)
Mode of action	Disrupt nucleic acids, precipitate proteins, change pH	Cell lysis, precipitate proteins, denature lipids	Denature proteins and alkylate nucleic acids	Alter cell membrane permeability	Alter cell membrane permeability	Denature proteins	Denature proteins, disrupt nucleic acids	Denature proteins and lipids	Denature proteins, disrupt cell membrane, inactivate enzymes	Alter cell membrane permeability, denature proteins	Denature proteins, disrupt cell membrane, inactivate enzymes
Dilutions	0.2%–4%, dependent on type	70%–90%	According to datasheet	0.4%–10%, dependent on type	0.5%–2%	2%–10%	10%–100%	3%–6%	0.5%–2%	According to datasheet	0.5%–1%
Contact time	10–30 minutes	20 minutes	20 minutes	10–30 minutes	10 minutes	10 minutes	1–5 minutes	10–30 minutes	10 minutes	10 minutes	5–10 minutes
Common applications	Water delivery systems, surfaces	Instruments, skin	Endoscopy equipment, empty cages, furnishings	Formerly used for disinfection of equipment, animal conveyances, surfaces, and water-resistant clothing	Instruments, cages, hard surfaces, preoperative scrubs	Hard surfaces	Wound aseptic, preoperative scrub	Endoscopy equipment, aerial disinfection	Hard surfaces, floors, equipment, footbaths, aerial disinfection, water delivery systems	Hard surfaces, floors, footbaths, laundry rinse	Hard surfaces, instruments, and rubber equipment

Continued

TABLE 12.1
Overview of Common Disinfectants, Their Mode of Action, and Activity Against Specific Pathogens—cont'd

						Halogens		Oxidizing Agents Peroxygens			
Type of Disinfectant	**Acids**	**Alcohols**	**Aldehydes Glutaraldehyde Formaldehyde**	**Alkalis**	**Biguanides-Chlorhexidine**	**Chlorines**	**Iodophor Solutions**	**Accelerated Hydrogen Peroxide**	**Potassium Peroxymonosulfate**	**Phenolic Compounds**	**Quaternary Ammonium Compounds**
SPECTRUM OF ACTIVITY											
Enveloped viruses (avian bornavirus, avian influenza virus, herpesvirus, paramyxovirus, poxvirus)	+	+	++	+	±	++	++	++	++	+	±
Large, nonenveloped viruses (aviadenoviruses, reovirus)	–	±	+	±	–	++	+	+	++	–	±
Small, nonenveloped viruses (circovirus, polyomavirus)	–	–	+	±	–	++	±	+	+	–	–
Gram-positive bacteria (*Staphylococcus aureus, Streptococcus* spp., *Listeria* spp.)	+	++	++	+	++	++	+	+	+	++	++
Gram-negative bacteria (*E. coli, Pasteurella* spp., *Salmonella* spp.)	+	++	++	+	+	+	+	+	+	+	+

Acid-fast bacteria (*Mycobacterium* spp.)	–	±	+	+	–	+	+	±	±	+	±
Chlamydiae (*Chlamydia psittaci*)	±	±	+	±	+	±	+	+	+	±	+
Mycoplasmata (*Mycoplasma* spp.)	+	++	++	++	++	++	++	++	++	++	+
Pseudomonas aeruginosa	+	++	++	+	±	++	+	+	+	++	±
Bacterial spores (*Clostridium* spp.)	±	–	+	±	–	+	+	+	+	–	–
Fungi (*Aspergillus* spp., *Candida* spp.)	±	±	+	+	±	+	±	±	±	+	±
Protozoan oocysts (*Cryptosporidium* spp., *Eimeria* spp.)	–	–	–	+	–	–	–	±	±	+	–
Protozoan cysts (*Giardia*)	±	+	–	N/A	±	±[a]	±[a]	+	+	+	±
Helminth ova (*Ascaridia* spp.)	+	–	+	+	–	±	±	±	±	+	–

[a]Significant effect of water temperature (higher efficacy at lower temperatures).
++, Highly effective; +, effective; ±, limited or partial activity; –, no activity; *N/A*, data not available.

each of these formulations, as not all have been compared to each other, and testing of efficacy has occurred under varying conditions and with different organisms. Nevertheless, it is likely that all of these, if used properly, will be effective at disinfecting cleaned surfaces. At the same time, none of these products will perform as expected if the proper concentration is not used for the pathogen of concern and the proper contact times are not used. In general, for resistant organisms, such as nonenveloped viruses and bacterial spores, required contact times are often long (up to 30 minutes in some instances) and concentrations of the disinfectant need to be increased (up to 10-fold) over concentrations used for other organisms.[7] Therefore, routine disinfection protocols will need to be modified in cases where difficult-to-inactivate pathogens are intermittently or regularly encountered.

Accelerated Hydrogen Peroxide

Accelerated hydrogen peroxide products patented disinfectants (Rescue: Virox Technologies Inc., Oakville, ON, Canada) contain multiple ingredients that provide them with a number of advantages including relatively fast killing times, the ability to wet a surface evenly, efficacy in the presence of organic material and resistance to inactivation by hard water; they are noncorrosive and are environmentally friendly.[8] They are effective against nonenveloped viruses and therefore should be effective against avian polyomavirus and the beak and feather disease virus. They can significantly reduce the concentrations of environmental clostridial spores but are less effective than bleach.[9]

Quarternary Ammonium-Biguanide Combinations

These products, for which F-10SC (Health and Hygiene, Loughborough, South Africa) and Trigene II (MediChem International, Sevenoaks, UK) are examples, are made up of a combination of disinfectants that work in synergy with each other. They have the advantage of not being irritating, of working solutions being stable for long periods (1 year), and having activity in the presence of limited amounts of organic material. They kill bacteria with limited contact time (<1 min) but may require longer times (up to 15 min) to kill nonenveloped viruses and longer periods may be required to inactivate clostridial spores. Different dilutions have different levels of efficacy against different pathogens. Higher concentrations are recommended to inactivate fungi and the highest concentrations are recommended for inactivation of nonenveloped viruses.

Potassium Peroxymonosulfate

Potassium peroxymonosulfate is an oxidizing agent. It is sold under the trade names Trifectant (Evsco Pharmaceutical, Fort Worth, TX, USA), Virkon S (Laxness Corporation, Pittsburgh, PA, USA), and others. One of its advantages is that its efficacy is maintained to some extent in the presence of organic matter. Disadvantages are that it must be made fresh every 7 days, it is mildly corrosive, the powder prior to mixing with water is an irritant, and it may not be effective against spores from dermatophytes.

Sodium Hypochlorite (Household Bleach)

Bleach is an inexpensive board-spectrum disinfectant. It is unaffected by water hardness and is effective against biofilm-producing organisms. It is particularly useful for disinfecting mop heads, brooms, perches, food bowls, and water bowls that can be soaked in diluted bleach. It has a quick kill time for many pathogens, and of the disinfectants considered in this section, it is likely to be the most effective against clostridial spores. It is recommended that for routine disinfection, 20 mL of a 5% bleach concentrate is added to 1 L of water. The main disadvantage of bleach is that it is readily inactivated by organic debris requiring surfaces be cleaned thoroughly before use. Additionally, it is corrosive, tends to bead up and does not provide even surface contact, and should be made up fresh at least daily. There are also concerns that toxic gases, including chloroform, are released during the cleaning process and long-term exposure of patients and staff may represent a health hazard.[10]

Polyvinylchloride (PVC) pipes are commonly used by aviculturists as water conduits in automatic water delivery systems for drinkers and for misters used to cool birds in hot environments. Water reservoirs for these systems and the PVC pipes can become overgrown with *Pseudomonas* spp., particularly if there are gaps in their use.[11] Regular disinfection of water reservoirs and disinfection and flushing of PVC pipes is recommended with diluted bleach. Protocols for disinfection can be found at https://www.health.nsw.gov.au/environment/water/Documents/rainwater_tanks.pdf.

WHAT ABOUT ALCOHOL?

Ethanol and isopropyl alcohol in concentrations of 60% to 90% (volume/volume) are used as disinfectants in most veterinary practices. Alcohol is bactericidal and fungicidal and is effective against enveloped viruses. It is also highly effective at inactivating mycobacteria. It inactivates other bacteria and some viruses almost immediately on contact. Because of its quick kill capacity for bacteria, it has been used to disinfect stethoscopes, thermometers, and rubber stoppers on multiple-dose injection bottles. Its major disadvantage is that it is not effective against some nonenveloped viruses and bacterial spores. Also, because it evaporates quickly, it has limited contact time on surfaces.

Alcohol-based hand sanitizers (60%–80% alcohol) are recommended for use after examining patients or touching potentially contaminated surfaces if hands are not soiled. They have the advantages that they are convenient, dry quickly, and reduce bacterial counts on hands better than handwashing with soap and water. They are only effective if hands are not soiled; soiled hands should be washed. Liquid soap from a dispenser, and not bar soap, should be used for handwashing. The World Health Organization Guidelines on Hand Hygiene in Health Care can be accessed at https://apps.who.int/iris/bitstream/handle/10665/44102/9789241597906_eng.pdf;sequence=1.

TOXICITY OF DISINFECTANTS

All disinfectants are toxins, so every effort should be made to minimize bird exposure of residual disinfectant solutions to them.[12] Cages should never be disinfected while the bird is in the cage. Cage furniture, water and food bowls, and surgical instruments should be rinsed well after disinfecting. If

disinfectants are used on surfaces, they should be allowed to air dry until they can no longer be smelled. In general, however, unless a bird is in direct contact with a concentrated disinfectant, is exposed to concentrated vapors from a disinfectant, ingests the disinfectant, or has long-term exposure to a disinfectant, the risk of toxicity is minimal to none.

CONCLUSION

Cleaning and disinfection are key pillars to infectious disease control in the veterinary hospital, avicultural facilities, and homes. Cleaning and disinfection protocols should be developed following well-established guidelines discussed and referenced here. Failure to follow these guidelines will minimally result in wasted effort or, in a worst-case scenario, outbreaks of preventable diseases.

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CHAPTER 13

Vaccination Strategies

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Infectious bacterial and viral diseases are a constant threat to poultry, exotic pet birds, and wild birds. Infectious bacterial diseases include chlamydiosis, salmonellosis, clostridial infection, and mycoplasmosis. Viruses that affect companion birds and avicultural collections include avian polyomavirus, psittacine herpesvirus, psittacine circovirus, avian bornavirus, West Nile virus (WNV), avian influenza (AI), poxvirus, and paramyxovirus.

VACCINE DEVELOPMENT

In human and domestic animal medicine (dogs, cats, and livestock, including poultry), vaccines have been a life-saving tool. However, in the nondomestic bird populations, commercial production and widespread use of vaccines have been underdeveloped and underutilized.[1] Development and successful promotion of vaccines for use in nondomestic birds have proven to be more difficult than for puppies and kittens. The cost of development, licensing, and marketing for the companion bird market has made vaccines expensive and therefore more difficult to market to the pet bird owner, who often only has one or two birds. This has resulted in pharmaceutical companies being unwilling to invest in new vaccine production for companion birds. In general, the developmental costs associated with minor species' vaccines outweigh the profit margin. Additionally, there are no government mandated vaccines for birds (such as rabies), and many bird owners do not schedule yearly health visits with veterinarians where vaccinations are administered. Although the poultry industry has multiple effective and inexpensive vaccines, delivery of vaccines to small flock owners by veterinarians is uncommon. This in part is because commercial poultry vaccines are typically only available in bulk, thereby making it difficult for the backyard poultry owner to invest in vaccinating a couple of birds. In countries where pigeon racing is popular, vaccination of these birds has become routine for several diseases (and is often mandated in the case of paramyxovirus).

Almost all vaccines are subject to rules and licensing within the country and/or regions of manufacture and use. Companies producing vaccines in the United States must now have their facilities licensed by the US Department of Agriculture (USDA) except for companies located and selling vaccines within California. Procedures for licensing a vaccine in the United States are detailed in a book called the *Code of Federal Regulations #9* (9 CFR).[2] Some vaccines do not have licensing procedures outlined in the 9 CFR (e.g., new products). In these cases, a company must submit to the USDA a proposal detailing the steps that they wish to take to obtain a license for this vaccine. The USDA will then either approve (with or without comments) or disapprove the proposal. If no vaccines are available for a particular disease and a critical need exists, then a company has the option of pursuing a conditional license for that vaccine. Essentially this means that the company only has to prove vaccine safety before releasing the vaccine to the consumer. Eventually, however, the company will be required to submit the appropriate efficacy data to the USDA, CVB (Center of Veterinary Biologics), to acquire a full license for the vaccine. In addition, the vaccine must be labeled as conditional. The only exception to this rule are autogenous vaccines that can be produced by veterinarians, poultry companies, and so on for use against a specific strain or organism. Rules for producing an autogenous vaccine can also be found in the 9 CFR.

After an organism is in the company's possession, the culture is screened for contaminants and a plan is detailed for development of the product. This plan will anticipate whether the isolate needs to be further processed (i.e., attenuated, decontaminated, concentrated, etc.) before a master seed stock is produced. In addition, the most appropriate route(s) of application will be determined. Vaccines are subjected to numerous *in vitro* tests, which include sterility, purity, pleuropneumonia-like organism (PPLO) broth, complement fixation for avian leukosis (COFAL), *Salmonella*, formaldehyde residue (inactivated vaccines), and moisture (lyophilized vaccines). In addition, companies must prove that the vaccine will be safe and efficacious. Efficacy is established by determining the minimum dose that is required to protect a set percentage of birds as specified in the 9 CFR (usually 90%). Once this dose is determined and a "buffer" amount is added, all vaccine produced must have at least this amount of organism per dose to be sold. The buffer amount, which is 1.2 log above the minimum protective dose (MPD) for most vaccines, is added to compensate for any loss of potency that might occur during storage of the vaccine up to the expiration date. However, a vaccine can have a much higher amount of organism per dose when the vaccine is sold; it just cannot be lower.

VACCINE TYPES

Historically, live attenuated or inactivated (killed) forms of microbial pathogens (bacteria or viruses) have been

successfully utilized in both human and veterinary medicine to induce an immunological response that protects the host against subsequent infections.[1] Although effective, both technologies have their limitations (Table 13.1).[3–6] Outbreaks have occurred due to incomplete inactivation or inactivation limiting the immune response. Attenuated vaccines must be able to provide immunity without causing disease, but there is risk of an attenuated vaccine reverting to full virulence.

Based on the pathogen, inactivated and live attenuated vaccine formulations contain a number of proteins that elicit a response. However, immunity is typically dependent on only a few selected proteins, whereas most of the proteins are not immunologically relevant. With some organisms, the immunologically relevant proteins are different from the pathologically important proteins, and separating these can significantly reduce the virulence of live vaccines. However, these immunologically relevant proteins may also induce allergenic reactions. Due to these inherent issues, interest in new vaccine technology to optimize vaccine functionality has grown (Box 13.1).

TABLE 13.1

Comparison of Vaccine Types

Vaccine	Advantages	Disadvantages	Delivery System
Inactivated (killed) vaccines	Long lasting, uniform immunity, fewer systemic reactions, no risk of spread of antigen to other birds, good maternal antibody response	Labor-intensive, reaction to adjuvant, only stimulates humoral immunity, often need live primer, risk of contamination of needles during delivery	IM (leg, breast, or wing) or SC (neck, tail, head)
Live (attenuated) vaccines	Often easier to apply, can be stored easier and fewer local reactions, stimulates cell-mediated, humoral, and local immunity, more options for delivery	May produce shorter immune response compared to killed, may cause adverse reaction in respiratory system, need care in handling of vaccines to keep live organisms	IM, SC, *in ovo*, ocular or nasal, wing web, spray, gel, water
Recombinant/ vectored vaccines	Protection both against the vector (e.g., herpesvirus of turkeys) and the vectored virus (Newcastle disease virus), no risk of reversion to pathogenicity, DIVA compatible, and no interference with maternal immunity	Previous generations of vaccines have limited technology to have multiple antigens, although third-generation recombinant vaccines can now accommodate immunogens from 2+ different pathogens (multivalent vaccines)[3-5] Need for individual vaccination and interference by maternally derived antibodies	SC
Subunit vaccines	No risk of reversion to pathogenicity. Relatively shelf-stable and safe to administer in young and immunocompromised birds	Cost prohibitive, often requires boosters (multiple doses) for long-term immunity	SC
DNA vaccines	DIVA compatible, relatively inexpensive and can be mass applicated, safe and stable vaccine with persistence of immunogenicity toward a specific antigen of interest[6]	Limited to protein immunogens and relatively poor immunogenicity[6]	IM, oral, *in ovo*, eye drop, gene gun, intranasal
Peptide vaccines	Safe and cost-effective for manufacturing	Limited data and use in commercial poultry production (none licensed in the United States)	None available at the moment

DIVA, Discriminates between infected and vaccinated animals; *IM,* intramuscular; *SC,* subcutaneous.

BOX 13.1 GUIDELINES ON THE QUALITIES OF AN IDEAL VACCINE (ROYAL SOCIETY 2002 REPORT ON INFECTIOUS DISEASE OF LIVESTOCK)

Guidelines on the Qualities of an Ideal Vaccine

- Broad-spectrum protection against all virus isolates in the host species, preventing disease and shedding and transmission
- Stimulated immunity to induce effective and long-lasting immune response
- Inexpensive to produce and ease of administration
- Long shelf-life and heat stable
- Live attenuated vaccines do not revert to virulence
- Provides strong levels of maternal immunity
- Discriminates between infected and vaccinated animals (DIVA)

New vaccine technology includes subunit vaccines, split product, isolated protein, peptide, live vector, and nucleic acid (DNA) vaccines. These methods of vaccine production are more economical, safer, and protective and allows for the cloning of viral antigens within bacteria and yeasts producing large quantities of pure antigen.

SUBUNIT AND SPLIT-PRODUCT VACCINES

A subunit vaccine is developed utilizing a fragment of a pathogen, often a surface protein that is used to stimulate an immune response against the pathogen from which it is derived. The simplest form of a subunit vaccine is one that the infectious agent has been disassembled or broken up to its component parts. Although subunit vaccines from natural infectious agents are effective, the cost of production can be prohibitive. It is more economical to identify and produce the immunogenic protein in mass quantity, allowing to produce a safe, effective, and relatively reasonably priced vaccine. Once a sufficiently pure, immunogenic protein is identified, large quantities can be produced. With the development of DNA technology, foreign proteins can be produced, providing an economical and relatively limitless source of protein from an infectious agent for vaccinations.[7] Split virus vaccines are produced by using detergent to disrupt the viral organism.[7]

PEPTIDE VACCINES

Peptide vaccines are developed by identifying and sequencing important immunogenic sites on infectious agents that can be mimicked using short chains of amino acids. Peptides used in these vaccines are 20 to 30 amino acid sequences that are synthesized to form an immunogenic molecule representing the specific epitope of an antigen. These peptides are sufficient for stimulating the immune response while eliminating allergenic and/or reactogenic responses. Production of peptide vaccines is considered safe and cost-effective when compared to conventional vaccines. Peptide vaccines under development include vaccines for human immunodeficiency virus, swine fever, influenza, anthrax, and human papilloma virus. Peptide vaccines may also be effective as therapeutic anticancer vaccines. Only a few peptide-based vaccines have been licensed to date, but studies have shown they can elicit immune response in a wide variety of veterinary viruses, including rabies virus, bovine rotavirus, bovine enterovirus, canine parvovirus, equine herpesvirus, feline leukemia virus (FELV), and bovine leukemia virus.[7,8]

LIVE VECTOR VACCINES/RECOMBINANT VACCINES

Live attenuated vaccines offer some advantages over conventional inactivated and subunit vaccines. They stimulate a more comprehensive immune response, are often easier to administer, provide longer lasting immunity, are easier to store, and typically do not cause muscle or skin reactions. These qualities have led researchers to investigate ways of delivering subunit or peptide vaccines using live vectors.

Viral vectors have focused on relatively large DNA viruses such as poxviruses and herpesviruses. These viruses have a large genome that facilitates insertion of new genes. They express high levels of the recombinant antigen, and in some cases, vectored vaccines appear able to induce immunity even when high levels of maternal antibody are present. Avipoxviruses have been utilized in development of veterinary vaccines, in particular, fowlpox and canarypox.[7] Veterinary herpesviruses and adenoviruses are also being developed as viral vectors. Turkey herpesvirus has been particularly successful as a vector for bivalent and trivalent vaccines against Marek disease (MD), Newcastle disease (NCD), infectious laryngotracheitis (ILT), and infectious bursal disease (IBD).[7,9] Third-generation recombinant vectored vaccines now have the capacity to be trivalent, including an NCD base against IBD and MD (Ultifend ND IBD), as well as a herpesvirus of turkey (HVT) base with IBD and NCD, or IBD and ILT (Vaxxitek). Using the CRISP technology, there are event multivalent vaccines that have simultaneous protection against ILT, AIV, IBD, and MD.[10]

Bacterial and protozoal vectors are also being studied. Most of the work in the study of bacterial vectors has focused on *Salmonella*. Variable vaccination results have been reported, but successful induction of cell-mediated, systemic, and local antibody responses following oral immunization has been achieved, leading to successful production of attenuated vaccines against *Salmonella* in poultry. Other bacterial vectors are being researched.

NUCLEIC ACID (DNA) VACCINES

Nucleic acid vaccines are a relatively new technology. These vaccines utilize DNA-encoded specific antigens inserted into a plasmid or the direct injection of messenger RNA. Nucleic acid vaccines can be produced cost-effectively and can elicit an effective T-cell and antibody response. In addition, there is no risk of infection, and the vaccines can bypass passive immunity. Currently, DNA vaccines are licensed for use against infectious hematopoietic necrosis in Canadian Atlantic salmon, for the control of WNV in horses, salmon pancreas disease infection, and canine melanoma. Recently, a commercial DNA vaccine against H5N1 in chickens has been conditionally approved in the United States.[7,9]

Results of the use of DNA vaccines in poultry have been encouraging, with vaccines being developed and providing full protection against several bacterial, protozoal, and viral diseases including avian influenza virus (AIV) in chickens and quail, IBD virus (IBDv), duck Tembusu virus (DTMUV), and Newcastle disease virus (NDV) in chickens. Host age, the frequency and number of vaccines given, and the vehicle, adjuvant, and delivery system affect the efficacy of these vaccines.

MODIFIED LIVE MARKER VACCINES: DIFFERENTIATING INFECTED FROM VACCINATED ANIMALS

Incorporation of new or unusual antigens into vaccines can be a valuable tool in disease control and eradication programs by allowing the user to discern the infected from vaccinated. These marker or DIVA (differentiating infected from vaccinated animal) vaccines can be recombinant deletion mutants of wild-type pathogens or subunit/peptide vaccines. In birds, these vaccines have specifically been used for AIVs.[11]

VACCINATIONS IN POULTRY

There are many effective vaccines utilized in commercial poultry, but only a few of these are used in backyard flocks. Vaccination protocols are available for viral, bacterial, and parasitic diseases in poultry.[12] The majority of poultry vaccines are inactivated (killed), live, or live recombinant. Delivery routes include injection, oral (in water), spray, ocular, wing web, or in the egg (*in ovo*). Several factors determine delivery route, including species, age, disease, vaccine type, cost, and labor involved in delivery.[12] The focus in this chapter will be on vaccination protocols for backyard chickens and nonpoultry avian species.

VACCINATIONS IN BACKYARD POULTRY

With the many diseases and vaccinations for commercial poultry, it can be confusing when recommending vaccines to backyard flocks. Many of the vaccines available and recommended for commercial poultry are contraindicated in backyard poultry. An example is a *Salmonella* vaccine that could result in a positive reactor for a pullorum-typhoid test that is required for health certificates used in interstate travel or qualifications for a local or regional poultry show. In addition, some live vaccines have side effects that small flock owners may not be comfortable seeing in their pets. These may include agents such as a live, chicken embryo origin ILT vaccine that may revert to virulence and cause disease.

Ideally, vaccine selection should be determined by the prevalence (or geographical distribution) of a particular disease, the risk for the bird becoming infected and developing disease, the value of the bird to the owner (their bond), and the type/location of the immune response needed. Although there are many diseases that can affect backyard poultry, in most cases only a few do. Contacting your state, national or regional veterinary diagnostic laboratory or state, national or regional poultry extension veterinarian will assist you in identifying the diseases present in your area. If a particular disease is not prevalent in the area, then vaccination for that disease may not be necessary. However, if a client shows birds at exhibitions, then the risk of certain diseases may be increased, and vaccination is warranted. In general, vaccine recommendations for backyard flocks include vaccination against MD, and in cases where historical outbreaks are present, fowlpox vaccine and NCD vaccine are recommended.[13]

MAREK DISEASE

The MD vaccine is an example of a successful control strategy in veterinary medicine. MD is caused by an alphaherpesvirus that is ubiquitous in the environment; therefore, chicks can become infected readily after hatching. Prevention requires either *in ovo* vaccination on the 18th day of incubation or a subcutaneous (SC) vaccination when the chicks are 1 day old. However, the MD vaccines available for commercial poultry (serotype I ± II MD) are not readily accessible for backyard flocks due to storage of the cell-associated vaccines in liquid nitrogen. Nevertheless, individual owners may request vaccinated chicks that have received it directly from the hatchery, similarly to commercial poultry.

For backyard breeders, the lyophilized HVT vaccine can be used to vaccinate for MD. The vaccine is easily purchased, reconstituted, and can be given SC to 1-day-old chicks. The vaccine is effective in preventing disease and tumor formation; however, it does not prevent viral infection or shedding. The vaccine is extraordinarily safe, even if all 500 doses were given to one chick. Although the MD vaccine is given at hatch or prior in commercial poultry, the vaccine may be given to adults if there is no history of MD. Time of MD exposure in backyard flocks is often unknown; therefore, adults may still be naive to the virus. However, it is always preferred to vaccinate as early in life as possible. For flocks with a known history of MD infection, no birds are added without vaccination and quarantine for 2 weeks prior to addition. Measures should be in place to decontaminate an area for newly hatched and vaccinated chicks, so that they have time to develop an immune response. Immunity should develop in 2 weeks. Regular cleaning and disinfection programs should be implemented and an all-in/all-out policy needs to be in place whenever bringing in new birds.[13]

AVIAN POX

Avipoxvirus infections have been reported in more than 200 species of birds, including chickens (see also Chapter 15). Several vaccines have been used to control avipoxvirus infections. There may be cross protection, but, in most cases, it is strain specific. When quail, chickens, and turkeys were vaccinated with pigeon and fowlpox virus and challenged with quail poxvirus, they developed severe pox lesions.[14] When quail and chickens were vaccinated with quail poxvirus and challenged with pigeon and fowlpox virus, they were not protected against disease either. Similar results occurred in chickens that were vaccinated with psittacine pox and challenged with quail pox. However, when quail, psittacine, and fowlpox virus were combined in a multivalent vaccine and given to chickens, the birds were protected against challenge with all three respective viruses.[14]

For backyard poultry, the wing web method (Figure 13.1) of vaccine is utilized for poxvirus, which is administered using a double-pronged stab stick into the center of the web, avoiding muscle and vasculature. Turkeys are vaccinated in the thigh after parting the feathers, as a wing web vaccine could cause transference of the vaccine and subsequent lesions on the head due to turkeys sleeping with their heads under their wings. Vaccinated birds should be examined 7 to 10 days post vaccine for an area of swelling or a scab (vaccine "take") to determine that the vaccine was effective (Figure 13.2). However, wing web reactions do not always correlate with immunity. Birds can be vaccinated when chicks are one day old or, alternatively, at 4 weeks of age followed by a booster after 1 month. Pox is one of the very few vaccines that can be administered in the face of challenge to slow the spread. High-risk flocks should be vaccinated annually. A combined pigeon pox and fowlpox vaccine may be utilized for maximum coverage.[15]

NEWCASTLE DISEASE

NCD is caused by avian avulavirus, further subtyped as avian paramyxovirus serotype 1 (APMV-1). NDV can affect all species of birds. The severity of the disease depends on the species affected and virus strain. Severe disease and outbreaks are the result from velogenic Newcastle disease (vND), formerly termed as exotic NCD. Clinical disease is often systemic and can affect the neurologic, gastrointestinal, respiratory, and



FIGURE 13.1 Wing web vaccination for fowlpox in a chicken (*Gallus gallus domesticus*). For this purpose, the chicken is held and one of the wings is spread to locate the vaccination site (i.e., the propatagium or wing web). Following thorough mixing of the vaccine, a double prong sewing machine needle (supplied alongside the vaccine) is dipped into the vaccine and then used to pierce through the wing web while avoiding blood vessels. and musculature.



FIGURE 13.2 Following wing web vaccination for poxviruses, the vaccination site needs to be examined for "take" 6 to 10 days post vaccination. Successful vaccination can be recognized by presence of a swelling and scab formation, as seen in this pigeon (*Columba livia domestica*). Lack of such a response to the vaccine may indicate improper vaccination, administration of an ineffective vaccine (e.g., incorrect storage), or presence of pre-existing antibodies.

reproductive systems. Physical examination and necropsy findings of birds with vND include diphtheritic lesions in the oral cavity and necrotic, hemorrhagic lesions of the gastrointestinal tract including the esophagus, proventriculus, small and large intestines, and cecal tonsils.

Prevention of disease has involved strict biosecurity measures along with vaccination. This has included implementation of quarantine stations for imported birds, controlling movement of birds and eggs in areas of concern, and the administration of vaccines. Effective control and containment of vND outbreaks is achieved with routine vaccination, rapid diagnostic assays, and, if disease occurs, culling of affected flocks.[16]

NDV vaccines are available for chickens, turkeys, and pigeons. The vaccines are used to elicit an antibody response and do not provide complete immunity. Thus, vaccinated birds require a greater vNDV exposure to become ill.[16]

The most widely used vaccines are the live lentogenic vaccines, B1 and La Sota strains.[17] These viruses belong to genotype 11 and are antigenically and genetically similar. The differences between the vaccines are the tropism and ability to replicate in naive chickens (Table 13.2). The La Sota strain is the most antigenic and results in higher antibody levels and is most widely used in countries where virulent NDV is endemic. The B1 strain is the most attenuated and is often used in cases with low exposure or in very young chicks. The VG/GA (Villegas-Glisson/University of Georgia) strain is typically sold as an enterotropic vaccine.[17] All of these are relatively inexpensive yet effective.

Methods of delivery include adding it to the water, ocular or nasal drops, or coarse spray.[16] Coarse spray vaccines result in more significant reactions, but their antibody response is stronger.[18] In contrast to the immune response induced by an inactivated vaccine, the mucosal immunity induced in birds vaccinated through these routes with the live vaccine decreases the viral shedding by infected birds. However, while live vaccines induce mucosal and humoral immunity and can be administered utilizing mass vaccination methods, they may also cause clinical respiratory disease, drop in egg production, and are easily inactivated if not stored at the appropriate temperature (commonly 4°C).[16]

Although mass vaccination methods are less labor-intensive, uneven vaccine application could lead to fewer than 85% of the flock being immunized, thereby reducing herd immunity. Another cause of decreased efficacy of vaccination

TABLE 13.2

Characteristics of Newcastle Disease Virus Vaccines Available for Poultry

Vaccine	Type	Method of Delivery	Tropism	Use
La Sota strain	Live lentogenic	In water, ocular, nasal, spray	Respiratory Increased antigenic stimulation/ higher antibody levels	Regions—endemic velogenic newcastle disease virus (vNDV)
B1 strain	Live lentogenic	In water, ocular, nasal, spray	Respiratory Most attenuated/fewer side effects	Regions—low exposure/young chicks
VG/GA strain	Live lentogenic	In water, ocular, nasal, spray	Enterotropic	Regions—where intestinal shedding for DIVA is not a problem (sold internationally for 20+ years, available in the United States since 2017)

DIVA, Discriminates between infected and vaccinated animals; *VG/GA,* Villegas-Glisson/University of Georgia; *vNDV,* velogenic newcastle disease virus.

is the presence of maternal antibodies that can neutralize the vaccine virus. The frequency of revaccination depends on the risk of exposure and virulence of the field virus challenge.[16]

Bivalent NDV vectored vaccines have been developed to induce solid protective immunity against NDV and other foreign antigens.[17] The Meleagrid alphaherpesvirus 1, commonly called HVT, is one of the most widely used vectors in production of recombinant vaccines. Two studies showed the protective capability of HVT vector–based vaccines to protect chickens from NDV and MD.[19,20] Currently, two of these bivalent recombinant HVT vaccines are commercially available and used internationally.[16] They produce long-term immunity and can be administered *in ovo* or SC at any time post hatch. The disadvantage is that the buildup of protective immunity requires 4 weeks and is hindered by the presence of maternal antibodies. Moreover, the vaccine needs to be kept in liquid nitrogen and administered within an hour of being thawed.

INFLUENZA

AI is an infection of birds with AI type A viruses. AI occurs naturally among wild aquatic birds worldwide and can infect domestic poultry and other bird species. Waterfowl are often asymptomatic carriers, but AI can be very contagious among birds including chickens, ducks, and turkeys. In domestic poultry, many AI viruses are of low pathogenicity (LPAI), causing subclinical infections, respiratory disease, or drops in egg production, but a few AI viruses are highly pathogenic (HPAI); these can cause severe systemic disease with high mortality (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).

Although rare, AI can infect humans. Most human cases have originated from infection with Eurasian H5 HPAI virus (A/Goose/Guangdong lineage) or, most recently, H7N9 LPAI virus (Chinese lineage). Most human infections have been from direct contact with infected poultry. Flu-like signs or respiratory infection is the most frequent presentation in human AI infections. Due to the impact on the poultry industry, the zoonotic potential and the potential threat to other bird species, research on effective vaccines is ongoing.

AIVs are a threat to the poultry industry worldwide. In the United States and EU countries, historically, slaughter of infected birds was the accepted policy. Currently, over 95% of the AI poultry vaccines are inactivated, adjuvanted products and require approval from the state and federal government for administration. A recent study found that formalin-inactivated vaccines induced similar antibody titers and protection against challenge as beta-propiolactone (BPL)–inactivated vaccines. BPL is extremely caustic to humans and is rarely used as an inactivate for commercial vaccines. This study also found that vegetable and mineral oil–based adjuvants induced the highest antibody tiers with 100% seroconversion by 3 weeks post vaccination.[21]

Advances in molecular biology have allowed a number of different viral vector vaccines that express the AI virus hemagglutinin (*HA*) gene to be developed and licensed for AI control. Vector vaccines can stimulate both humoral and cellular immune responses, and when antigenically matched to the target AI strain, they are effective at preventing clinical disease and reducing viral shedding. These vaccines can provide early protection (1-day-old chicks) and are cost-effective. Because they only express the *HA* gene, they can potentially be used to differentiate vaccinated from vaccinated and infected birds (DIVA strategy). Viral vectored vaccines work well with a prime boost strategy, where the vectored vaccine is given first, followed by a killed adjuvanted vaccine booster given 2 to 3 weeks later.[22]

Currently, only inactivated whole AI virus vaccines, typically H1 and H3, are licensed in the USFA for turkey breeders. The use of any licensed AI vaccine requires approval of the state veterinarian. In addition, use of H5 and H7 AI vaccines in the United States requires declaration of an emergency and approval by the Secretary of Agriculture.

VACCINATION IN PIGEONS

The vaccination protocol recommended for racing and show pigeons includes *Salmonella typhimurium*, pigeon paramyxovirus type 1 (PPMV-1), and pigeon poxvirus.[23]

Salmonella typhimurium

S. typhimurium is one of the major health issues in pigeons worldwide. Other strains of *Salmonella* have also been recovered, some with potential to infect humans and that are quite resistant to antibiotics. Historically, *Salmonella* vaccines in pigeons have not been very protective against disease but they have reduced polyuria and fecal shedding.[24]

Recently, a killed adjuvant *Salmonella* vaccine (Nu-Sal Vaccine KM-1), developed by Arko Laboratories Ltd, was licensed by the USDA for use in birds older than 6 weeks. A recent study evaluated the effects of this vaccine against *S. typhimurium* in 15 pigeons (*Columba livia domestica*), which were vaccinated twice through SC injection at 3- to 4-week intervals and challenged 10 days following the booster vaccination. None of the vaccinated birds developed clinical signs, had any shedding of the organism, or had any lesions of *Salmonella* on necropsy. In contrast, control birds became sick, shed the organism, and had *Salmonella*-induced lesions observed on necropsy, demonstrating the effectiveness of the vaccine in preventing disease.[25]

PIGEON PARAMYXOVIRUS TYPE 1

Pigeon paramyxovirus (PPMV) is another major health issue in pigeons worldwide. PPMV-1 is a serious disease that can kill up to 100% of pigeons in some lofts. Some of the signs of PPMV-1 infection include lethargy, vomiting or regurgitation, green diarrhea, circling, head flicking, labored breathing, and ocular discharge and may result in chronic enteritis. Sick birds can die within 3 days. The infection spreads easily between birds and there is no specific treatment. Several vaccines have been studied for prevention of PPMV-1.[26]

Young pigeons were protected for up to 1 year following a SC vaccination with an inactivated aqueous NDV La Sota vaccine. Squabs were vaccinated at different ages: 1 to 8 days of age, 10 to 16 days of age, and at 21 days of age. Maternal antibodies did not affect response.[27] Occasionally, the vaccine caused the development of a granuloma or abscess at the injection site.[28] A bivalent inactivated oil emulsion vaccine was found to be safe and protective in pigeons.[29]

A pigeon (PPMV-1) isolate and the La Sota and Ulster NCD strains were used to prepare BPL-inactivated vaccines.

Pigeons and chickens were vaccinated SC and evaluated for antibody response. The pigeons were challenged 10 weeks post vaccine with a virulent PPMV-1 and were protected from disease and death, but not from infection from the challenge virus. Viral shedding was greatly reduced 6 days following challenge.[30]

One study assessed PPMV-1 and *S. typhimurium* vaccine together. A combined inactivated montanide ISA-206 oil-adjuvanted vaccine of local isolates of PPMV-1 and *S. typhimurium* was utilized. The vaccination challenge assay with the virulent strain of PPMV-1 showed 100% protection in the vaccinated group, while the virulent *S. typhimurium* organism showed 90% protection in vaccinated birds. In the unvaccinated control group, 10% and 20% did not become infected after challenge with both virulent PPMV-1 and *S. typhimurium*, respectively, indicating this vaccine could be recommended as safe, potent, and useful for protection against PPMV-1 and *S. typhimurium* under field conditions.[31]

A recent licensed killed adjuvant PMV vaccine (KM-PMV from Arko Laboratories Ltd) has shown efficacy. This vaccine is given SC twice at 3-week intervals and is licensed for birds greater than 6 weeks of age.[25]

PIGEON POXVIRUS

Poxvirus in pigeons has similar clinical signs of disease and morbidity as fowlpox. The vaccination protocol is similar to backyard chickens.[13]

PIGEON HERPESVIRUS 1

Pigeon herpesvirus 1 causes respiratory disease and encephalitis in doves and pigeons. This disease can also affect raptors that consume infected birds. A comparison of a killed or attenuated vaccine was performed in pigeons with and without the addition of cyclophosphamide to determine if either vaccine could prevent infection, the carrier state, or control shedding. Both vaccines gave similar results, being able to reduce primary viral excretion and clinical signs after challenge. However, neither vaccine was able to prevent reappearance of carriers.[32]

VACCINATIONS IN PSITTACINE BIRDS

Several pathogens are known to cause specific diseases in Psittaciformes, including paramyxovirus, polyomavirus, parrot bornavirus (PaBV), psittacid herpesvirus, psittacine beak and feather disease (PBFD) virus, and *Chlamydia psittaci*. All the listed viruses will give rise to serious, often lethal disease in young birds. With no effective treatment available other than alleviating clinical signs, vaccination strategies may be attempted to prevent disease transmission and development of clinical signs.

PARAMYXOVIRUS

Paramyxovirus type 3 (PMV-3) is found to cause illness and high mortality in small psittacines, most notably *Neophema* spp., and passerines (finches). Gastrointestinal and neurological signs may develop. An inactivated, adjuvanted PMV-3 experimental vaccine has been effective in protecting parakeets from developing disease.[33]

POLYOMAVIRUS

Avian polyomavirus (APV) primarily affects young birds. Clinical signs include a nestling with crop stasis, diarrhea, cutaneous hemorrhage, abdominal distention, and feather abnormalities. APV can cause severe disease and acute mortality in fledgling budgerigars (*Melopsittacus undulatus*), nestling macaws (*Ara* spp), and finches. A commercial inactivated vaccine is available and has been used to vaccinate prior to shipping, in pet stores and in rescue and sanctuary settings. The vaccine is administered IM (intramuscularly) or SC and has had few reported reactions.

Both inactivated and attenuated APV vaccines can safely immunize psittacines.[34,35] In a study reviewing the effects of two vaccines (adjuvant and nonadjuvant), in over 200 parrots, both vaccines induced a virus neutralizing antibody response in previously seronegative birds. Seroconversion occurred in 93% of birds 2 weeks post boosting. There was no significant difference in seroconversion between the two vaccines and only three Grey parrots (*Psittacus erithacus*) had mild local reactions (small scab formation at the injection site).[34]

Avian polyomavirus, in contrast to mammalian polyomavirus, encodes an additional capsid protein (CP) VP4 and its variant VP4D. Both proteins induce apoptosis. Mutation of this initiation codon prevents virus replication. A study assessing a replication competent deletion mutant where either VP4 or VP4D was deleted elicited neutralizing antibodies against APV after intraperitoneal (IP) and intramuscular (IM) infection of the chicken, although only the wild-type virus revealed VP4-specific antibodies. Based on the biological properties of these deletion mutants, they could be candidates for APV marker vaccines. In addition, an APV DIVA vaccine for APV may be possible.[34]

PSITTACINE BORNAVIRUS

Proventricular dilation disease (PDD) or avian ganglioneuritis (AGN) is caused by psittacine bornavirus (PaBV). PaBV is a neurotropic virus that can cause gastrointestinal and/or neurological signs of disease in birds (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease and Chapter 14, Avian Bornavirus). Currently, there is no cure, and treatment only reduces clinical signs of disease. A vaccine, if available, would be instrumental in prevention of disease.[36] Because avian bornavirus disease is T-cell mediated, theoretically, a vaccination could exacerbate disease.[36] Nonetheless, there have been several vaccination studies performed. Two reports utilized viral vector vaccines against PaBVs using a prime boost strategy.[37,38]

One reported vaccination strategy utilized recombinant NDV and modified vaccinia virus Ankara (MVA) that were engineered to express the phosphoprotein and nucleoprotein genes of two avian bornaviruses, parrot bornavirus 4 (PaBV-4) and canary bornavirus 2 (CnBV-2). The vaccine induced a bornavirus-specific humoral immune response in cockatiels (*Nymphicus hollandicus*) and canaries (*Serinus canaria*). Vaccinated birds were challenged with a homologous bornavirus. However, while the immunized birds had significantly reduced shedding of bornavirus RNA and reduced viral loads in tissue, they still developed clinical signs of AGN.[37]

A second study revealed that prime boost vaccination of cockatiels with NDV and MVA vectors expressing the

nucleoprotein and phosphoprotein genes of PaBV-4 blocked bornavirus replication following parenteral challenge infection with PaBV-2. In this study, two of six vaccinated birds developed mild PDD-associated lesions, while the unvaccinated birds were not protected from infection or disease.[38]

A vaccination developed utilizing a killed PaBV with recombinant PaBV-4 nucleoprotein N in alum was protective against disease in cockatiels challenged with a virulent bornavirus isolate (PaBV-2), but birds still developed gross and histologic lesions of PDD. There was no evidence that vaccination prior to or post challenge made infection more severe.[39]

These results suggest that some vaccinations can protect against disease and do not exacerbate infection, but do not reduce viral persistence or shedding. However, none of the studied avian bornavirus vaccines are currently available for commercial use.

PSITTACINE BEAK AND FEATHER DISEASE VIRUS

Psittacine beak and feather disease (PBFD) is primarily a disease of young parrots (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). The etiological agent is a DNA circovirus (PBFD virus [PBFDV]). Clinical signs of disease include abnormal feathers (pinched and hemorrhagic feather shafts), feather loss, necrosis of the beak, and a generalized loss of feather dander. Viral replication begins in the lymphoid organs, spreads to the liver and thymus, with ultimate invasion of the epithelium, which leads to the beak and feather malformations. The disease affects the immune system resulting in immunosuppression, so birds may present with secondary bacterial infections. An acute stage occurs in recently fledged birds, especially Grey parrots, in which birds die of liver failure presenting with severe leukopenia and anemia. Birds that recover from the acute disease may become chronic carriers (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).

There have been several experimental vaccines assessed, but none of these vaccines are currently available for commercial use.[40,41] A recombinant PBFD-CP vaccine was studied in long-billed corellas (*Cacatua tenuirostris*). The vaccine was given IM at approximately 50 days of age. Vaccinated birds were boosted on day 11 and challenged 16 days post booster. The vaccinated birds had a transient viremia but showed no clinical signs of disease or persistent infection post challenge. The control birds developed antibodies against PBFDV, were PCR-positive 41 days post challenge, and developed transient feather lesions. A quantitative PCR revealed decreased viral replication in the vaccinated birds as compared to the control birds.[42]

A second PBFD-CP study in nestling and adult galahs (*Eolophus roseicapillus*) that were vaccinated with an inactivated double oil emulsion vaccine had similar antibody response to those induced with a primary oil emulsion vaccine. The birds were given two IM injections 14 days apart. A post-vaccination challenge demonstrated the nestlings to be protected by both vaccines whereas the unvaccinated controls developed acute PBFD within 4 weeks of challenge.[43]

In another study using 30- to 45-day-old Grey parrots, umbrella cockatoos (*Cacatua alba*), and sulphur-crested cockatoos (*Cacatua sulphurea*), chicks from unvaccinated hens and chicks from vaccinated hens were exposed to PBFDV. Chicks from the unvaccinated hens developed disease, whereas chicks from the vaccinated hens remained free of disease. In the same study, an adult pair of umbrella cockatoos, Moluccan cockatoos (*Cacatua moluccensis*), Grey parrots, and a yellow-headed Amazon parrot (*Amazona ochrocephala oratrix*) were vaccinated IM or SC with a vaccine containing 13-propiolactone (BPL)–inactivated PBFDV. All birds seroconverted. In addition, 30- to 45-day-old chicks were vaccinated with the same inoculum and seroconverted. Following challenge with purified PBFDV, the chicks from vaccinated hens were protected whereas chicks from unvaccinated hens developed disease.[44]

Although no vaccine for PBFDV is currently available, multiple experimental vaccines have been assessed, including a recombinant vaccine, a subunit vaccine, and a live attenuated vaccine. A recombinant vaccine expressing the PBFDV CP, the major antigenic determinant, expressed in a plant, yeast, or bacterial system could be particularly useful and is promising based on recent research.[40,41]

PSITTACID HERPESVIRUS

Herpesviruses are DNA viruses that cause acute and latent infections (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Psittacine herpesvirus is an alphaherpesvirus that is the etiological agent of Pacheco disease and internal papillomatosis in parrots. Pacheco disease manifests as acute hepatitis with a high mortality in New World parrots. Survivors of Pacheco disease may develop internal papillomatosis. Quaker parrots (*Myiopsitta monachus*) vaccinated twice with killed Pacheco vaccine were protected when challenged.[45] A formaldehyde heat-inactivated experimental vaccine prepared from polyethylene glycol purified virus was given to a collection of psittacines. There were no adverse effects and the birds seroconverted.[46] Several oil-adjuvanted, inactivated vaccines have been developed against Pacheco disease. These vaccines were protective but did not prevent shedding of latent infections nor did they protect against all forms of avian herpesvirus. Oil emulsion vaccines have been developed but have led to serious injection site reactions (swelling, muscle necrosis, and formation of granulomas).[47]

An inactivated emulsified vaccine against Pacheco disease in budgerigars demonstrated that an effective vaccine could be produced utilizing concentrated virus. The virus was propagated in chicken kidney cells, sonicated, and concentrated 100× by centrifugation to produce a multiple emulsion vaccine. Young adult budgerigars were vaccinated once IM and challenged 4 weeks post inoculation. The vaccine was protective against challenge, but there was minimal serologic response post vaccine.[48]

An autogenous vaccine was developed to control a Pacheco disease virus outbreak in a zoological setting. This autogenous vaccine, formalin inactivated with an aluminum hydroxide adjuvant, was utilized IM 2 weeks and 6 weeks post outbreak. There were no reported negative reactions to the vaccine, and morbidity and mortality ceased. Vaccinated birds possessed

neutralizing antibodies 9 months following the second vaccination. Two out of four birds remained antibody-positive 25 months post vaccination.[49] Currently, there is no psittacine herpesvirus vaccine commercially available.

CHLAMYDIA PSITTACI

Chlamydia psittaci is a gram-negative intracellular obligate pathogen that causes psittacosis in parrots, ornithosis in other bird species, and chlamydiosis or parrot fever in humans (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Clinical signs in birds range from asymptomatic carriers to severe disease and may include ocular or nasal discharge, anorexia, dyspnea, polyuria, biliverdinuria, and diarrhea. Because many birds can be asymptomatic carriers, an effective vaccine may significantly reduce zoonotic risk.[50]

Turkeys (*Meleagris gallapavo*) were protected from infection with *Chlamydia psittaci* with a plasmid DNA vaccine against the major outer membrane protein.[51] Based on these data, a plasma DNA vaccine expressing the major outer membrane protein of *Chlamydia psittaci* genotype A was evaluated for protective immunity against *C. psittaci* challenge in budgerigars. Prior to the experimental infection, all birds were negative on both PCR and culture; however, all birds revealed a low pre-existing serum antibody titer, indicating previous infection. All birds were vaccinated twice (3 weeks apart) with either the placebo or the DNA vaccine. An aerosol challenge occurred 2 weeks after the booster vaccine. Placebo-vaccinated birds showed severe clinical signs and lesions and excreted high amounts of *C. psittaci* as compared to the DNA-vaccinated group.[50] Currently, there is no vaccine available.

VACCINATIONS IN OTHER SPECIES

Several infectious diseases can pose a threat to the well-being of captive birds. Particularly, birds that are housed in outdoor enclosures are at increased risk for exposure to infectious diseases that are transmitted by wild birds. Such diseases include AIV, poxvirus, paramyxovirus, WNV, equine encephalitis, and picornavirus. Current research related to vaccination strategies to prevent these infectious diseases in companion and zoo birds is discussed in the following sections.

AVIAN INFLUENZA VIRUS

Due to the spread of the H5N1 HPAI virus, preventive vaccination strategies have been implemented in various zoologic institutions to prevent the spread of the disease in their collection (Table 13.3).

In the early 2000s, following the spread of HPAI across Europe, various inactivated influenza vaccines have been successfully administered in various zoological institutions.

In a 2003 HPAI (H7N7) outbreak in the Netherlands, the European commission directive allowed vaccination of valuable susceptible zoo species. Birds were vaccinated IM with an inactivated H7N1 vaccine twice at 6-week intervals. Pre- and post-vaccination titers were measured to establish efficacy. Following booster vaccination, 81.5% of the vaccinated birds

TABLE 13.3
Overview of Selected Studies on Influenza Vaccines in Zoo Birds

Vaccine	Number of Birds	Protocol	Species With Minimal or No Seroconversion	Species With Adequate Seroconversion	Reference
H5N1-inactivated vaccine	540	Two IM vaccinations with 6-week interval; evaluation of serological response 4–6 weeks post second vaccination.	Pelicans, ducks, geese, herons, cranes, guinea fowl, cockatiels, lovebirds, barbets (poor response)	Ibis, rheas, Congo peafowl, flamingos, kookaburras, black-winged stilts, Amazon parrots. Overall 84% of birds seroconverted with 76% achieving titers ≥32	Bertelsen et al.[53]
H5N2-inactivated vaccine	118	Two IM vaccinations with 4-week intervals. Serological testing 4- and 6–8 weeks post vaccination using hemagglutination inhibition test.	Pelecaniformes, Strigiformes (no response)	Galliformes and Anseriformes (100% seroconversion)	Oh et al.[55]
H5N9 commercial–inactivated vaccine and H5N3-inactivated recombinant vaccine	48	Two IM vaccinations with 4-week interval 4 weeks apart, and booster 1.5 years later. Long-term serological effects (>7 years) were studied.	N/A	Flamingos (high titers remained present for over 7 years)	Fernandez-Bellon et al.[56]
H7N1-inactivated vaccine	211	Two IM or SC vaccinations at 6-week intervals. Pre- and post-vaccination antibody titers determined using hemagglutination inhibition test.	Struthioniformes	Anseriformes, Galliformes, and Phoenicopteriformes (81.5% of birds developed titers >40)	Philippa et al.[52]

IM, Intramuscular; *SC*, subcutaneous.

developed a titer greater than 40. Anseriformes, Galliformes, and Phoenicopteriformes showed the largest response. Antibody response decreased with increasing body weight in birds over 1.5 kg.[52]

In 2006, an inactivated H5N9 vaccine was administered to 540 birds from three Danish zoos. The birds were vaccinated twice at 6-week intervals. Serological response was evaluated 4 to 6 weeks following the second vaccination. Seroconversion was documented in 84% of the birds and 76% developed a titer of 32 or more. Species variation in response was noted: pelicans (*Pelecanus* spp.), ducks, geese, penguins, (Sphenisciformes), herons (Ardeidae), cranes (Gruiformes), guineafowl (Numididae), cockatiels, lovebirds (*Agapornis* spp.), and barbets had a poor response, whereas high titers and seroconversion rates were seen in ibis (Threskiornithidae), rheas (*Rhea* spp.), Congo peafowl (*Afropavo congensis*), flamingos (Phoenicopteridae), kookaburras (*Dacelo* spp.), black-winged stilts (*Himantopus himantopus*), and Amazon parrots (*Amazona* spp.).[53]

In 2009, a H5N2 vaccine was administered to approximately 25,000 birds among 135 French zoological institutions.[54] A follow-up serological evaluation using 1686 blood samples collected from 126 species representing 15 different avian orders demonstrated that 71% of the birds developed titers of 32 or more (mean titer 558 following the second vaccination with a subsequent decrease in titers). As a result, the percentage of birds having a titer of 32 or more (the level considered protective in poultry) dropped to 42% at day 180 and to only 26% at day 330. While no deleterious effects were seen in any of the vaccinated birds, the aforementioned results do demonstrate the necessity for adjustments to the protocol (e.g., booster vaccination every 6 months).

During an HPAI outbreak in Asia, the Singapore Zoological Gardens evaluated an inactivated H5N2 vaccine in their high-risk avian species (Galliformes and Anseriformes) along with birds on open exhibit. They used the Nobilis Influenza H5N2 vaccine and vaccinated birds IM with a booster at 4 weeks. A variation in response was noted between species and orders. Some Galliformes and Anseriformes had 100% seroconversion while others had only partial conversion. Some species like the pelicans (order Pelecaniformes) and owls (order Strigiformes) failed to respond to vaccination at all.[55]

A study at the Barcelona Zoo evaluated the long-term efficacy of H5 vaccination in flamingos (order Phoenicopteriformes). H5-antibody titers were maintained at high levels for over 7 years post vaccination against the H5N9 and H5N3 vaccine strains and the H5N3 and H5N1 reference strains.[56]

AVIAN POX

As discussed earlier under Vaccinations in Backyard Poultry, homologous, live virus vaccines that provide long-lasting immunity are available for use in fowlpox. However, nonpoultry species can have severe reactions to pox vaccines, therefore warranting caution when using pox vaccines in species other than poultry. A fowlpox vaccine was found to be protective and safe in zebra finches (*Taeniopygia guttata*) and a live pigeon pox vaccine has been used in hunting falcons.[57,58] A commercial fowlpox vaccine was protective in healthy ostrich chicks (*Struthio camelus*) during an outbreak of poxvirus.[59] Attenuated strains of poxviruses (fowlpox and canarypox) have been utilized as vaccine vectors to deliver heterologous vaccines. These vaccines stimulate both cellular and humoral immunity and are thermostable. Unfortunately, despite a structured vaccination program and strict biosecurity, canarypox virus remains to regularly affect captive houbara bustards (*Chlamydotis undulata*).[60]

PARAMYXOVIRUS/NEWCASTLE DISEASE

Paramyxoviruses can be infectious to all bird species and are found in both wild and captive populations. In the United States, sampling for APMV-1 may occur during periods of morbidity or mortality involving susceptible families of wild birds, such as pigeons, doves, cormorants (*Phalacrocorax* spp.), or pelicans. A study quantifying the prevalence and geographic distribution of APMV-1 in apparently healthy wild birds in 2013 found antibody prevalence was highest in birds of the order Suliformes (44.9%), Pelecaniformes (24.4%), Anseriformes (22.7%), and Columbiformes (11.7%), with a high occurrence of virulent viruses in Columbiformes (100% of virulent viruses isolated). However, viral shedding occurred rarely and in very low numbers.[61]

As paramyxoviruses may also affect captive populations, vaccination may be indicated, especially for birds living in zoos and bird sanctuaries that have a higher risk of being exposed to the virus through contact with wild birds. Vaccination is typically given in methods available to be delivered in small and/or individual doses. In some ostriches vaccinated with live NDV vaccine via eye drop method (Figure 13.3), an antibody response was induced in both the vaccinated birds and the unvaccinated control housed with them, indicating transmission of the virus post vaccination.[62] In contrast, ostriches vaccinated with the La Sota strain NDV vaccine via injection responded in a dose-dependent manner, with no transmission occurring between vaccinated and controls. Ostriches vaccinated with a series of 3 to 4 vaccines developed an



FIGURE 13.3 Application of a vaccine by eye drop method, as shown in this chicken (*Gallus gallus domesticus*), is a very effective route for administering live vaccines in poultry, as the vaccine passes to the Harderian gland just behind the eye, which is a key organ in the development of the immune response. Correct dilution of the vaccine is, however, critical to ensure correct dosing. If eye droppers are being used, they should be calibrated beforehand. Alternatively, it is possible to use the tip of a 1-mL syringe to administer the drop, but this is less accurate and will result in more wastage of vaccine.

adequate antibody response. The results of this study indicated that it is highly unlikely for ostriches vaccinated according to recommended vaccine schedules to transmit the virus to nonvaccinated birds.[63]

WEST NILE VIRUS

WNV infection is an arthropod-borne virus in the genus *Flavivirus* (family Flaviviridae; see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). The virus was introduced in the United States in 1999. Since its introduction, WNV has spread across the continent causing neurological disease in humans, horses, and birds. It has been reported in more than 320 bird species. Many bird species have been affected including corvids, passerines, psittacines, and raptors. Affected endangered or threatened species include the whooping crane (*Grus americana*), the sandhill crane (*Antigone canadensis*), the California condor (*Gymnogyps californianus*), and the Florida scrub jay (*Aphelocoma coerulescens*). Psittacines appear to be somewhat resistant, but the disease has been reported in parakeets, conures, rosellas, cockatoos, caiques, lorikeets, and a king parrot. Clinical signs in birds include depression, anorexia, weight loss, head tremors, ataxia, blindness, seizures, and death. Juvenile birds are the most affected. WNV outbreaks have been reported in Europe since the 1950s. These outbreaks were small and remained focal. In contrast to the United States, bird mortality is rare in Europe.[64]

Recently, a comprehensive review of the current progress of WNV vaccine in birds has been published.[65] Although birds are clearly at risk with this disease, and several studies assessing vaccination efficacy in birds have been performed, no avian WNV vaccine is commercially available. Hence, susceptible species in endemic areas are vaccinated utilizing the equine vaccines.

In the United States, until recently, three commercially available vaccines for use in horses.[66] The first licensed vaccine (K-WN) (West Nile-Innovator; Fort Dodge, Fort Dodge, IA) is a formalin-inactivated whole virus adjuvant and has been available since 2001. In 2004, a recombinant vaccine (CP_WN) was licensed and consists of an adjuvant and canarypox virus vector with insertion and expression of the membrane (prM) and envelope (E) proteins of *WNV* genes (Recombitek equine WNV vaccine; Merial Limited, Athens, GA). The latest equine vaccine, licensed in 2006, is a single dose, attenuated WNV, live flavivirus chimera vaccine (WN-FV; PreveNile; Intervet, De Soto, KS), which is marketed without an adjuvant. The equine vaccine that has been used most extensively in birds is the formalin-inactivated, adjuvanted whole virus vaccine called West Nile-Innovator marketed by Zoetis. As the Recombitek vaccine from Merial is no longer available, currently, the West Nile-Innovator vaccine is also the only vaccine that is commercially available.

Efficacy of West Nile Virus Vaccines in Psittacine Birds

Twelve captive adult thick-billed parrots (*Rhynchopsitta pachyrhyncha*) were given a series of three initial Innovator vaccines at 3-week intervals with an annual booster over 6 years. Most adult birds (67%) seroconverted after 5 years of annual vaccination. In addition, the serological responses of 11 chicks that were given the initial series of Innovator vaccines were assessed. None of the chicks seroconverted following the initial three vaccine series. Two of four chicks seroconverted at the 1-year booster and three of four chicks seroconverted at the 2-year booster.[67]

Efficacy of West Nile Virus Vaccines in Passerine Birds

Three WNV equine vaccines were evaluated in California scrub jays (*Aphelocoma californica*): Innovator, Recombitek, and an experimental DNA plasmid vaccine called pCBWN. Vaccination lowered peak viremia compared to positive controls, but the viremia was sufficient to infect susceptible mosquito vectors. Recombitek vaccination caused five of six vaccinated birds to develop lesions at the injection site. The Innovator vaccine and the experimental pCBWN vaccine stimulated antibody formation with no side effects.[68] A fowlpox vectored vaccine with inserts of ANV prM and E proteins followed by a canarypox virus vector vaccine is protective in geese.[69]

Experimental WNV DNA vaccines have been evaluated in American crows (*Corvus brachyrhynchos*). Crows were vaccinated IM with a DNA vaccine and a DNA vaccine with adjuvant-developed antibodies. A challenge at 10 weeks post initial vaccine resulted in decreased mortality but no sterile immunity. An oral microencapsulated DNA vaccine failed to produce antibodies and did not result in reduced mortality when challenged.[70]

An experimental DNA plasmid WNV vaccine was evaluated in American robins (*Turdus migratorius*), a key WNV amplification host. A single dose of vaccine injected IM resulted in a >400-fold decrease in viremia. Although a small sample size, results suggest that vaccinated robins develop viremia that is unlikely to be infectious to biting *Culex* mosquitoes.[71]

Efficacy of West Nile Virus Vaccines in Birds of Prey

The immunogenicity of both Recombitek and West Nile-Innovator has been evaluated in gyrfalcons (*Falco rusticolus*) and other falcons.[72] Increased protection was achieved with Recombitek as compared to Innovator, but Recombitek caused moderate injection site reactions. The Recombitek 2 dose regimen resulted in only partial protection. Improved results occurred following a third booster.[72] In red-tailed hawks (*Buteo jamaicensis*), however, results were less promising as both the birds that were vaccinated once ($n = 5$) with 0.2 mL of the Innovator vaccine in the pectoral muscle, as well as those receiving a booster vaccination 3 weeks following the first injection ($n = 5$), showed no detectable antibodies to WNV upon serological evaluation 3 weeks post vaccination.[73]

With the further spread of WNV across the continent, and just prior to its arrival to the Western United States, a prospective vaccination program was conducted for both the captive and wild endangered California condors. Due to the negative results with the Innovator vaccine in red-tailed hawks, the researchers chose to use a DNA vaccine. The vaccine DNA (pVWN) used in this study was modified by replacing the ampicillin resistance gene in the published pCBWN construct with a kanamycin resistance gene derived from the pVAX plasmid. The DNA vaccine was safe and induced protective immunity in adults, nestlings, and newly hatched condor chicks (see also *Current Therapy in Avian*

Medicine and Surgery, Vol 1, Chapter 2, Infectious Disease and Chapter 23, Conservation of Avian Species).[74]

In 2011, an experimental WNV DNA plasmid vaccine in an aluminum phosphate adjuvant was tested in red-tailed hawks. All birds received two IM injections 3 weeks apart. Birds were challenged 4 weeks following the second injection. Three vaccinated birds seroconverted after the second booster; all other birds seroconverted following the live virus challenge. Vaccinated birds had reduced viremia and a shorter and reduced shedding period compared to the controls.[75]

Efficacy of West Nile Virus Vaccines in Waterfowl and Poultry

In Attwater's prairie chickens (*Tympanuchus cupido attwateri*) vaccinated at least twice IM with the Innovator vaccine showed insufficient response to the vaccination protocol.[76] However, a WNV vaccine developed for humans (WN-80E) was found to be highly immunogenic for the nene (*Branta sandvicensis*), an endangered Hawaiian goose susceptible to WNV. Six captive nonbreeding birds were vaccinated with two doses 4 weeks apart. Two birds were injected with "mock" controls. The vaccine was highly immunogenic and did not produce adverse effects.[77]

A recombinant avirulent NDV La Sota strain expressing WNV premembrane/envelope proteins was assessed in chickens, ducks, and geese. A significant level of neutralizing antibodies was induced. Moreover, it also elicited significant levels of WNV-specific immunoglobulin (Ig) G in the birds following intramuscular, oral, or intranasal immunization.[78] Similar findings were obtained in another study assessing the efficacy of a WNV enveloped recombinant (rE) protein vaccine in red-legged partridges (*Alectoris rufa*). In this study, the birds were vaccinated three times at 2-week intervals. Vaccinated birds developed anti-WNV antibodies prior to challenge and survived challenge, whereas a third of the unvaccinated birds died. Unvaccinated birds were furthermore found to develop viremia and WNV RNA was detected in their feathers and swabs. In contrast, only 14% of the vaccinated birds developed viremia and no WNV RNA was detected in their feathers or swabs.[79]

In an effort to establish an avian model for testing WNV vaccines, avian antibody–based reagents were assessed for cross-reactivity with Japanese quail (*Coturnix japonica*) T-cell markers CD4 and CD8. The most reactive were found to be the anti-duck CD8 antibody, clone Du-CD8-1, and the anti-chicken/turkey CD4 antibody, clone CT4. These reagents were utilized to assess vaccine performance and T-cell populations in quail. The WNV vaccines developed were based on a nonreplicating recombinant adenovirus platform. Japanese quails were selected for testing the vaccines, as they provide an avian model that represents the population diversity of wild birds. Both WNV-specific antibodies and the number of T cells in vaccinated birds were increased compared to unvaccinated controls indicating that the recombinant vaccines were effective in increasing both humoral and cellular immune responses in quail.[80]

Efficacy of West Nile Virus Vaccines in Other Birds

The West Nile-Innovator vaccine was evaluated in sandhill cranes. The cranes were vaccinated with 0.5 mL IM with a booster at 21 days and 28 days. They were challenged with live WNV following the third booster. None of the vaccinated or controls birds died from the experimental infection, but 2 days post challenge, the vaccinated cranes did have significantly less viremia than the control. Seven days post challenge the vaccinated cranes had less cloacal shedding and 14 days post challenge the vaccinated cranes had significantly higher titers than the controls. At necropsy, unvaccinated challenged cranes had lesions in the brain and brainstem that were not seen in the vaccinated challenged or vaccinated unchallenged birds.[81]

Greater flamingo (*Phoenicopterus roseus*) chicks have also been vaccinated using Innovator vaccine IM. The birds were given 2 vaccines 3 weeks apart with a booster given 280 days post initial vaccination series. Antibody titers were assessed before and after the initial injection and 3 weeks post booster. No adverse reactions were reported, and serum antibodies were detected in 60% of the samples post booster.[82]

Similar findings were obtained in a study investigating the effect in both American (*Phoenicopterus ruber*) and Chilean flamingos (*Phoenicopterus chilensis*).[61] Another study, however, showed lack of detectable antibodies to WNV 3 weeks post vaccination in Chilean flamingos ($n = 16$), which had been vaccinated either once ($n = 8$) or twice (with 3-week interval; $n = 8$) with 0.2 mL of the Innovator vaccine in the pectoral muscle.[73]

The serologic response to WNV vaccines has also been evaluated in penguin species. For the Innovator vaccine, a vaccination protocol consisting of two IM injections with 3-week interval resulted in significant changes in antibody titer over time in black-footed penguins (*Spheniscus demersus*), but not in little blue penguins (*Eudyptula minor*), indicating that efficacy cannot be extrapolated easily to other species.[76] However, another study that evaluated effects of WNV vaccination in four species of penguins showed more promising results. In this study, Humboldt (*Spheniscus humboldti*), Magellanic (*Spheniscus magellanicus*), gentoo (*Pygoscelis papua*), and rockhopper (*Eudyptes chrysocome*) penguins were inoculated with either the Innovator killed virus vaccine or an experimental plasmid-mediated DNA WNV vaccine. Both vaccines induced seroconversion in all four species without adverse reactions. Moreover, the study revealed that in all four species, the Innovator vaccine resulted in a faster and greater seroconversion and a longer duration of antibody response than the DNA vaccine.[83]

EQUINE ENCEPHALITIS VIRUSES

Eastern equine encephalomyelitis (EEE), Western equine encephalomyelitis (WEE), and Venezuelan equine encephalitis (VEE) are caused by members of the genus *Alphavirus* of the family Togaviridae. Disease is arthropod borne and can occur in many species of birds, including pigeons, pheasants, and ratites such as emus (*Dromaius novaehollandiae*). Birds and rodents are the primary reservoirs, but unvaccinated horses are particularly susceptible and often serve as sentinels. Equine encephalitis viruses are zoonotic, infecting humans, and are a public health concern.

Several equine and human vaccines have been evaluated in avian species.[84,85] In 2001, a study evaluated the humoral immune response in emus vaccinated with either one of two commercially available equine polyvalents or an experimental monovalent EEE virus (EEEV) or WEE virus (WEEV) vaccine. All four vaccines induced detectable neutralizing antibody titers and led to full protection of the vaccinated birds when exposed to an otherwise lethal dose of EEEV. In contrast, unvaccinated challenged birds developed viremia and shed virus in feces, oral secretions, and regurgitated material. An unvaccinated pen-mate became infected in the absence of mosquito vectors, presumably because of direct virus transmission between birds. Results indicate that the commercially available polyvalent equine EEE vaccines are protective in emus.[86]

The immune response of whooping cranes and sandhill cranes to a formalin-inactivated EEE vaccine was evaluated. SC and intravenous vaccination failed to induce detectable antibodies in the sandhill cranes, but both species developed neutralizing antibodies to the IM vaccine. Additional boosters elicited detectable antibody and/or increased antibody titers in the whooping cranes, although cranes with naturally preexisting titers exhibited a rapid increase and sustained antibody titers. Even though EEEV vaccine induced neutralizing antibodies with no adverse reactions, its protective efficacy was unclear as no challenge occurred.[87]

An inactivated human EEE vaccine was evaluated in whooping cranes. All whooping cranes received an IM injection of 0.5 mL of inactivated EEE vaccine (PE 6 WRAIR strain) followed by a second injection 6 months later. Neutralizing antibodies were present after IM injection. All newly hatched whooping cranes received 0.5 mL of vaccine with a second injection 30 days later. All whooping cranes received a 1.0 mL IM booster annually. In the years following the vaccination program, there were no adverse reactions. The absence of virus-related deaths and adverse vaccination reactions following the vaccination protocol was evidence that the EEE immunization program was both safe and efficacious.[88] A second study was done years later evaluating the antibody titers measured in whooping cranes vaccinated with the human EEE vaccine for 3 years with those measured using the Equine EEE vaccine Encephaloid M (EEE and WEE vaccine, Fort Dodge Laboratories) for 2 years. Titers observed with the human vaccine were considered protective due to the absence of morbidity or mortality seen in whooping cranes in years since 1984 when EEE was detected in mosquitoes and sentinel birds. The equine vaccine produced titers equal to or higher than those produced by the human vaccine, which would suggest adequate protection.[89]

DUCK VIRAL HEPATITIS (PICORNAVIRUS)

Duck viral hepatitis (DVH) is an acute, highly contagious viral disease typically affecting ducklings less than 6 weeks of age. Duck hepatitis A viruses type 1 (DHAV-1) and type 3 (DHAV-3) are picornaviruses. Affected ducklings become lethargic, lose balance, paddle spasmodically, and often die. Currently, live attenuated virus vaccines of chicken embryo origin for DHAV-1 are either administered to 1-day-old ducklings to induce a protective immune response or to breeder ducks to provide maternal antibodies to their ducklings.[90,91] There is no vaccine for DHAV-3 and no licensed commercial vaccine to address both viruses.

A duck embryo epithelial (DEE) cell line was evaluated as a candidate for DHAV-1 vaccine development. Results showed that the propagated DHAV-1 vaccine strain SDE in DEE cell line was able to protect ducklings against DHAV-1 challenge. Taken together, findings suggest that the DEE cell line can serve as a valuable tool for DHAV-1 propagation and vaccine development.[92]

Immunization of ducklings with a yeast-derived recombinant VP1 (major CP) that was administered SC three times at 2-week intervals induced a significant immune response indicating this could be a candidate for a subunit vaccine against DHV-1.[93]

A live attenuated vaccine with the viral isolate AP-04203P100 given to 1-day-old ducklings via IM injection was protective from viral challenge. In addition, the vaccine candidate exhibited complete protection from circulating pathogenic DHAV-3 isolates.[94]

Ducks that received a single dose of rC-KCE-2VP1 (bacterial recombinant containing viral protein 1) showed potent humoral and cellular immune responses and were completely protected against challenges of both pathogenic DHAV-1 and DHAV-3 strains. Protection was rapid and occurred as early as 3 days post vaccination and viral replication was fully blocked as early as 1 week post vaccination.[95]

COVID-19

In 2019, a novel coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), was identified in Wuhan, China, which caused a contagious disease that became known as COVID-19. The first known case of COVID-19 was identified in Wuhan, China, in December 2019.[96] Since then, the disease rapidly spread worldwide, resulting in the COVID-19 pandemic. Currently, the disease predominantly affects humans. In addition, COVID-19 has been documented in domesticated dogs, cats, hamsters, ferrets, and mink and is also known to infect zoo species including lions, tigers, snow leopards, a fishing cat, a binturong, a coatimundi, otters, hyenas, hippopotamuses, and manatees. Wildlife reports include white-tailed and mule deer, a giant anteater, and a black-tailed marmoset.[96] To date, the disease has not been reported in any avian species.

The virus is thought to be a spillover infection of natural animal origin. According to the World Health Organization, the virus most likely descended from a coronavirus that infects wild bats, to likely spread to humans through an intermediate wildlife host. Although COVID-19 and its variants have not caused illness in avian species, the zoonotic component and rapid mutation rate could allow for a strain that crosses over to birds.

During the COVID-19 pandemic, several COVID-19 vaccines were developed and approved in a relatively short period of time, including vaccines by Pfizer–BioNTech and Moderna, which utilized RNA to stimulate an immune response,[97] and the Oxford–AstraZeneca COVID-19 vaccine, Convidecia, and the Janssen COVID-19 vaccine, which comprised of adenovirus vector vaccines.[98] These developments

have demonstrated that vaccine production can be accomplished in a relatively short time when the need arises, with mRNA vaccines being especially effective in generating a rapid and appropriate immune response.

VACCINE STRATEGIES IN NONPOULTRY CAPTIVE BIRDS

Deciding when and if to vaccinate a bird for a certain disease can be a challenging question for veterinarians, especially because so many vaccines are off label. Factors to consider include the following:

- Risk of the disease to the individual bird and/or a population of birds.
- Is the bird endangered or threatened?
- Is the vaccine safe and effective in the species your considering vaccinating? Is there a known protocol for vaccination? (Table 13.2)
- What is the cost of the vaccine?
- How do you assess efficacy?
  - Measurement of antibody response?
- Do the risks of disease outweigh the risk of vaccination?

After considering these questions, the attending veterinarian should keep detailed records of the vaccination including, but not limited to, vaccination dose (e.g., full/half-dose), manufacturer, protocol used, age(s) of the birds when vaccinated, signed, and dated with a disclaimer that an off-label vaccine has been used. It is best to have signed informed consent by the client in case adverse reactions may occur.

SUMMARY

Historically, vaccines have been underutilized in nonpoultry avian medicine. The numerous published studies in vaccine strategies that have been successful and protective demonstrate that it is possible to develop safe and effective vaccines. However, a few of these vaccines have made it to commercial development (Table 13.4). Nevertheless, with recent advancement in vaccine technology and increased knowledge of avian immunology, the potential to develop safe, effective, and cost-effective vaccines is available. As more and more of our exotic avian species become threatened or endangered, it is the role of the avian veterinarian to push for the development, production, and commercial sale of safe and effective avian vaccines.

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TABLE 13.4

Overview of Selected Vaccines Available for Use in Birds

Vaccine	Disease	Species	Vaccine Protocol	References
Psittimune APV (Creative Science): inactivated (killed) virus vaccine	Polyoma (polyomavirus)	Parrots	Birds >200 g give 0.5 mL SQ. Birds <200 g give 0.25 mL SQ. First dose at 35 days, repeat in 2–3 weeks. Annual booster.	Ritchie et al.[34]
Psittimune PDV: (Ceva Biomune): inactivated virus, oil emulsion vaccine	Pacheco disease (herpesvirus)	Parrots	Vaccinate before exposure or disease outbreak.	No longer commercially available
Innovator (Zoetis): formalin-inactivated virus vaccine	West Nile virus	Raptors/corvids	1.0 mL IM (>300 g), 0.5 mL (<300 g) IM initially followed by 2–3 boosters 1 month apart then annually before mosquito season.	Angenvoort et al.[72] Wheeler et al.[68] *Merck Manual of Veterinary Medicine:* Viral Diseases of Pet Birds[99]
Innovator (Zoetis): formalin-inactivated virus vaccine	West Nile virus	Flamingos	1.0 mL IM initially followed by 2 boosters 1 month apart and then annually before mosquito season.	Okeson et al.[76] Baitchman et al.[82]
Innovator (Zoetis): formalin-inactivated virus vaccine	West Nile virus	Penguins	1.0 mL IM initially followed by 2 boosters 1 month apart and then annually before mosquito season.	Davis et al.[83] Wallace R.[100]
Equine EEE vaccine Encephaloid M (Fort Dodge Laboratories)	EEE, WEE	Emus (*Dromaius novaehollandiae*)	Emu: give full equine dose IM at 6 weeks, 10 weeks, 5 months, and 6 months, and annually thereafter before and after breeding season and if viral disease occurs within 16 km or before transfer to a known endemic area.	Tengelsen et al.[86]
Equine EEE vaccine Encephaloid M (Fort Dodge Laboratories)	EEE, WEE	Pheasants/cranes	Give 0.5 ml at 30 days of age; 1 mL IM booster in 30 days and then annually.	Clark et al.[87]

Continued

TABLE 13.4

Overview of Selected Vaccines Available for Use in Birds—cont'd

Vaccine	Disease	Species	Vaccine Protocol	References
Inactivated monovalent avian influenza virus vaccines, including either H5 or H7 strains; bivalent, including H5 and H7 strains (EU)	Avian influenza	Multiple species	Emergency, preventive, or prophylactic, and routine vaccination strategies may be used. Note: live recombinant vaccines (fowlpox H5) are efficacious only in 1-day-old chickens.	https://ec.europa.eu/food/animals/animal-diseases/control-measures/avian-influenza_en https://www.aazv.org/page/107/Avian-Influenza-Information-and-Updates.htm
Inactivated Newcastle disease vaccine and live canarypox vaccine	Newcastle disease	Kori bustards (*Ardeotis kori*)	Annual vaccination in Middle East but typically not in zoos in the United States for bustards. Vaccinations best given during health assessment at 1–2 months before breeding season.	Le Loc'h et al.[62]
KM-PMV (ARKO Labs, Jewell, IA): killed virus vaccine, serotype 1	Pigeon paramyxovirus type 1	Pigeons	Two vaccinations 4 weeks apart SC at the base of the neck or in the loose inguinal region.	Arko Lab: http://www.arkolabs.com/pb/wp_6de3bd73/wp_6de3bd73.html
Nu-Sal Vaccine KM-1 (ARKO Labs, Jewell, IA)	*Salmonella typhimurium*	Pigeons	Two vaccinations 3–4 weeks apart. Vaccinations should be completed 2–3 weeks prior to racing or showing. Semi-annual boosters.	Arko Lab: http://www.arkolabs.com/pb/wp_6de3bd73/wp_6de3bd73.html
Fowl pox vaccine for chickens, turkeys, pheasants, ostriches Live pigeon pox vaccine for poultry and pigeons	Poxvirus	Pigeons/poultry/ratites	Pigeon or fowlpox vaccine—healthy chickens aged 8 weeks or older, but at least 4 weeks prior to start of lay.	Winterfield and Reed[14] Perelman et al.[60]
Lyophilized herpesvirus of turkey (HVT) vaccine	Marek disease	Backyard poultry	Give SC at day 1 of age.	*Merck Manual of Veterinary Medicine:* Marek Disease in Poultry

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CHAPTER 14

Avian Bornavirus

Michael Lierz

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INTRODUCTION

Avian bornavirus was first discovered in 2008 in psittacine birds suffering from proventricular dilatation disease (PDD). This strongly suggested that the cause of this devastating disease, threatening parrot populations in captivity and species conservation projects, was found. In 2012, the postulates of Henle and Koch were fulfilled,[1] and the pathogen was proven as the cause, by reproducing clinical and pathological signs in cockatiels (*Nymphicus hollandicus*) after virus application and reisolation of the same virus afterward. Prevalence studies have demonstrated the worldwide distribution of bornaviruses, both in captive and free-ranging bird populations. Different virus strains are found in various bird orders, in particular, Psittaciformes, Passeriformes, and Anseriformes. The main clinical importance remains in psittacine birds where the virus causes various neurological disorders in addition to the "classic" gastrointestinal signs (PDD). Within just 14 years of its discovery, many forward steps have been made toward understanding the transmission of the virus as well as its pathogenesis. Management strategies with which to control the virus in populations and first steps toward a vaccination protocol have also been made. This chapter provides a short update on recent discoveries in this field and is intended to serve as an addition to the information provided in the first volume of this book. Additionally, some key features are repeated, especially focusing on diagnosis and clinical management in the individual bird.

NOMENCLATURE

The complete taxonomy of the family Bornaviridae is being constantly updated due to the discovery of novel strains, particularly in birds and reptiles. The latest update was made in 2019.[2] Most importantly two new genera, *Carbovirus* and *Cultervirus*, were added to the family. The genus name *Bornavirus* was substituted by *Orthobornavirus*, leading to new virus species names. This was necessary as by adding the new genus, confusion between "Bornaviridae" and "*Bornavirus*" when talking about "bornavirus" or "bornaviral" is to be avoided. Some virus strains, for example, parrot bornavirus 6 (PaBV-6) and PaBV-8, are still not classified due to the lack of genetic information. Table 14.1 provides the current taxonomy of the family Bornaviridae.

Avian Orthobornavirus

The different species of the genus *Orthobornavirus* in birds seem to be specific to the different bird orders—that is, waterbird *Orthobornavirus* is generally detected only in Anseriformes, passeriform orthobornaviruses in Passeriformes, and psittaciform orthobornaviruses in Psittaciformes. Cross-order detection of those viruses is rare and only anecdotally reported. In Japan, PaBV-2- and PaBV-4-RNA was detected in samples of free-ranging birds, other than psittacine birds *(Anas, Grus, Larus, Calidris, Haliaeetus, Emberiza)*, but clinical signs were not investigated.[3] In addition, PaBV-RNA was demonstrated from the brain of a Himalayan monal *(Lophophorus impejanus)* with central nervous system (CNS) signs, flocked together with infected psittacines.[4] Aquatic bird bornavirus 1 (ABBV-1) was described in 9 of 439 investigated free-ranging gulls, with only one bird showing an encephalitis,[5] and once in a captive emu *(Dromaius novaehollandiae)*, demonstrating anorexia, maldigestion, weight loss, and various subtle neurological deficits.[6] Despite these reports, crossing birds orders, and particularly the development of clinical signs following cross-infection, seem to be a very rare occurrence, hence indicating the risk of infection with *Orthobornavirus* from other bird orders to be low. Nevertheless, close monitoring and reporting of cross-order infections and clinical cases should provide further insight as to whether this risk indeed is negligible.

TABLE 14.1
Taxonomy of the Family Bornaviridae

Genus	Species	Virus	Abbreviation
FAMILY BORNAVIRIDAE			
Carbovirus	Queensland carbovirus	Jungle carpet python virus	JCPV
	Southwest carbovirus	Southwest carpet python virus	SWCPV
Cultervirus	Sharpbelly cultervirus	Wǔhàn sharpbelly bornavirus	WhSBV
Orthobornavirus	Elapid orthobornavirus	Loveridge's garter snake virus 1	LGSV-1
	Mammalian orthobornavirus 1	Borna disease virus 1	BoDV-1
		Borna disease virus 2	BoDV-2
	Mammalian orthobornavirus 2	Variegated squirrel bornavirus 1	VSBV-1
	Passeriform orthobornavirus 1	Canary bornavirus 1	CnBV-1
		Canary bornavirus 2	CnBV-2
		Canary bornavirus 3	CnBV-3
	Passeriform orthobornavirus 2	Estrildid finch bornavirus 1	EsBV-1
	Psittaciform orthobornavirus 1	Parrot bornavirus 1	PaBV-1
		Parrot bornavirus 2	PaBV-2
		Parrot bornavirus 3	PaBV-3
		Parrot bornavirus 4	PaBV-4
		Parrot bornavirus 7	PaBV-7
	Psittaciform orthobornavirus 2	Parrot bornavirus 5	PaBV-5
	Waterbird orthobornavirus 1	Aquatic bird bornavirus 1	ABBV-1
		Aquatic bird bornavirus 2	ABBV-2

OCCURRENCE OF AVIAN ORTHOBORNAVIRUS

Avian orthobornaviruses (ABVs) are detected worldwide. Infections have been found in the United States,[7,8] Australia, Europe,[9–12] Brazil,[13–16] Japan,[17,18] South Africa,[19] Thailand,[20] and Canada.[21,22] In Denmark, ABBV-1 was found in free-ranging geese, which is the same strain occurring in waterfowl in North America.[23] Canary bornavirus 1 (CnBV-1), first discovered in Germany, was also found in free-ranging and captive passerine birds in Brazil, indicating worldwide distribution of *Orthobornavirus* among Passeriformes.[24] Most studies, however, have focused on the prevalence of orthobornaviruses in Psittaciformes. In psittacine birds the most prevalent virus types remain PaBV-4 followed by PaBV-2. Other types, such as PaBV-5, are detected in different countries and only reported in single case reports.[20] So far, there is no evidence that certain subtypes are restricted to certain countries or populations. Studies in captive psittacine collections indicate a prevalence of approximately 4.3% to 32.2% in clinically healthy birds.[9,10,25] The prevalence of asymptomatic infections among captive-maintained birds makes worldwide viral distribution through international trade easily possible. It can realistically be assumed that nearly all larger breeding flocks of psittacines contain infected individuals, except for those flocks where diligent efforts to clear the presence of the pathogen are being employed. In contrast, studies in free-ranging psittacine populations demonstrate a very low prevalence.[15,26] The reason for the difference between captive and free-ranging flocks is not yet clear. Interestingly, in neozoan free-ranging psittacine populations established in Europe, PaBV could not be found.[27] It is hypothesized that through the release of a few birds, which were negative, a PaBV-free population may have been established. Given that this study investigated free-ranging *Psittacula* species, which also demonstrated a very low prevalence in captivity in Europe (~2%), this would be a plausible explanation. Alternatively, the routes of transmission might explain the found differences, as birds in captivity have closer contact and bird exchange between groups occurs more often in captivity than in the wild. Asymptomatically infected birds might not seroconvert and shed virus intermittently, as compared to clinically affected birds with high titers and high viral RNA shedding. This latter group usually does not occur in the sample bag when investigating free-ranging populations as they are chronically ill and die without being found. The occurrence of new strains in some free-ranging populations, which are not detected by current laboratory methods, can also not be excluded. Last but not least, the bird species investigated in the wild might play a role. However, so far there is no evidence that PaBV strains are specific to some species and all studies involving different parrot species on different continents resume a similar prevalence.

AVIAN ORTHOBORNAVIRUS TRANSMISSION

ABV-RNA is regularly detected in feces and urine as well as in cloacal and crop swabs. A fecal-oral route of transmission has therefore been postulated.[28–31] Reports also demonstrated that ABV spreads in a flock after introduction of infected birds,[32] indicating horizontal transmission. However, a fecal-oral route of transmission would not explain the variable, and often rather low prevalence in psittacine flocks, the very low prevalence in free-ranging populations, and the observation that, in single pairs of parrots, only one of the partners is found to test positive for PaBV. Multiple studies demonstrate that uninfected

sentinel birds kept in close contact to experimentally infected animals did not get infected.[1,29,33–35] Experimental infection is usually accomplished through intramuscular, subcutaneous, intravenous, or intracerebral injections or a combination of these routes,[1,29,33,36] which do not represent natural routes of transmission. Recent infection studies clearly showed that oral or nasal inoculation of PaBV does not result in valid infections in various bird species, including cockatiels, canaries (*Serinus canaria* f.d.), and grey parrots (*Psittacus erithacus*).[37,38] Heckmann et al.[39] were able to demonstrate that infection through wounds is possible, but ability to transmit infection seems to differ according to the type of wound. After experimental inoculation of PaBV-4 on lesions in the oral cavity, infection did not occur. In contrast, inoculation of a similar virus suspension on footpad lesions leads to a successful infection. PaBV-RNA was found in sciatic nerves, the footpad tissue, skin, and intestine in that study. Histopathology demonstrated mononuclear infiltrations in the skin and footpad tissue typical for PaBV infection and immunohistochemistry showed positive reactions in spinal ganglia and spinal cord, and, to a lesser amount, in the skin, footpad tissues, and ischiatic nerves.[39] The reason why lesions in the oral cavity did not act as a portal of entrance, compared to footpad lesions, remains unclear. It was speculated that the severe bleeding of oral cavity lesions during the experimental infection might have washed out inoculated virus. Footpad lesions, in contrast, did not bleed as much and were covered by a bandage after inoculation, preventing early removal of the virus. Therefore, it seems fair to conclude that wounds in general represent a valid port of entrance. The results of the footpad infection group suggested a spread of the virus in the host through retrograde axonal transport, like rabies. This assumption is supported by another study where after intramuscular inoculation, PaBV-2 was first detected in macrophages and lymphocytes, followed by invasion of the brachial plexus and spread centripetally through the spinal cord toward the brain.[40] This was followed by a centrifugal spread toward the ganglia in the periphery, including the gastrointestinal system. Only after this, at a late time point following infection, the virus was no longer restricted to the nervous system.[40] This viral movement explains the different findings noted previously, where in some cases PaBV was restricted to the nervous system, whereas in other cases the virus was found in a multitude of other tissues. The speed of viral spread in the body, as well as the phase which is reached are likely influenced by different host factors including immunocompetency, which might explain the observed high variation of clinical signs or latency. As said, the literature[39,40] strongly suggests that PaBV uses the nervous system as a port of entry and spread within the host, and that the CNS is the first target. Following PaBV-2 injection into the pectoral muscle, the virus was observed in the spinal cord after 20 days postinfection (dpi) and after 25 dpi in the brain, indicating rapid spread of the virus into the nervous system tissues. Until day 40 postinfection, the virus was only detected in the central core of the brain to spread to gray and white matter of the CNS at 60 dpi, where it was accompanied by encephalitis.[41] Depending on the port of entry, the brain is reached at a variable time frame, hence in part explaining the different incubation times prior to the onset of clinical signs. Moreover, the study showed that, if the brain is affected severely first, severe clinical signs develop rapidly, often in birds with good body condition. However, if the spread of the virus within the nervous system takes longer and is not as severe, clinical CNS signs might not be as overt and easily missed, leading to further spread of the infection through the body. As it takes longer for the virus to reach the peripheral nervous tissues, this would also explain why the development of gastrointestinal signs takes much longer to develop.

In addition to horizontal transmission, vertical transmission is also possible. Knowledge about the circumstances of vertical transmission is highly important as it would allow for breeding with rare birds, even if they are PaBV infected. PaBV antigen has been demonstrated in ovarian follicular cells of diseased birds[42] and PaBV-RNA has been identified in eggs and embryos.[43,44] It has been shown that CnBV-RNA was present in canary embryos of CnBV-positive parents. Only female birds with a persistent CnBV infection and high levels of virus shedding laid eggs with CnBV-positive embryos. Intermittent CnBV-RNA–shedding females and females of the contact group laid only CnBV-RNA–negative eggs.[45] However, all of these studies failed to isolate live virus, which leaves some doubt regarding vertical transmission. Hand-raised Amazon chicks originating from PaBV-positive parents were tested negative for PaBV-RNA from crop and cloacal swabs.[46] Wüst et al.[47] investigated vertical transmission of PaBV-4 and PaBV-2 in a laboratory setup using cockatiels as a model. Susceptibility of the inoculates for infectivity of embryonic cockatiel cells was first examined. Embryonic cockatiel brain cell cultures (CEB) and embryonic cockatiel fibroblast cell culture (CEF) from 10-day-old cockatiel embryos were compared with viral growth patterns in cells from CEC-32 cell lines that were typically used before to grow ABVs. In CEC-32 cell culture, PaBV-2 showed slower growth compared to PaBV-4. CEB cells were successfully infected with PaBV-2 and PaBV-4 without differences in growth speed. Fertile cockatiel eggs were inoculated with PaBV-4 by yolk sac route at days 3 to 5 of incubation. The organs of each embryo were examined at 13 to 14 days postinfection (day 17 of incubation) by different laboratory methods. In 28.1% of the inoculated embryos PaBV-4-RNA and in 81.3% PaBV-4 antigen were detected in the brain. Nevertheless, similar to the studies previously described, virus isolation failed. In a different component of this study, inoculated embryonated cockatiel eggs inoculated with PaBV-2 or PaBV-4 via yolk sac or chorioallantois membrane were inoculated at days 5 to 7 of incubation. Compared to the first part of the study where organs were divided between different types of examinations, this time the complete organ was subjected to one laboratory method to increase sensitivity. Now, in 80% to 100% of the embryos PaBV-RNA and in 90% to 100% PaBV antigen were detected only in the brain, independently of the PaBV strain. The infected cells demonstrated a clear pattern through all the embryos but could not be identified using different brain cell markers (i.e., anti–neuronal nuclear protein [NeuN] antibody [neuron marker], anti–glial fibrillary acidic protein [GFAP] antibody [astrocyte marker], and anti–S-100 [glia cell and neuron marker]. While virus isolation again was unsuccessful, these studies do demonstrate a specific distribution pattern and spread of PaBV-4 and PaBV-2 with restriction to the brain when injected into fertile eggs. Hence, the potential for vertical transmission remained in place. However, an abortive infection can also not be ruled out at this point.[47]

Successful vertical transmission of PaBV was proven in another study for the first time.[48] Vertical transmission of the virus was compared in eggs originating from birds that were experimentally infected with PaBV as adults (group 1), and eggs originating from birds infected at the first days of life and raised until sexual maturity with persistent infection (group 2) Ninety-two eggs (49 from group 1 and 43 from group 2) were laid and incubated until day 17, following which the embryos were examined by immunohistology, PaBV-specific real-time polymerase chain reaction (PCR), and virus isolation. Results indicated 45.2% of the embryos to be positive by PCR in group 1 versus 74.9% of the embryos in group 2. Reisolation of PaBV was unsuccessful from embryos in group 1, but in group 2, reisolation of PaBV was possible from 42.9% of the embryos.[48] Based on these findings, it appears that vertical transmission may only occur under specific circumstances. Specifically, the time point of infection of the birds and the degree of development of the immune system are hypothesized to play a role (see Pathogenesis). In summary, results of this study indicate that vertical transmission might play a major role in transmission and the development of asymptomatically infected birds, which in turn may serve to maintain and spread infections in and between flocks. Controlling vertical transmission could therefore be a key factor in controlling viral spread and clearing the virus from flocks.

PATHOGENESIS

There remains considerable need for further understanding of the factors that influence the development from infection to clinical disease. It is known that several birds may be positive in a flock, but only a few develop clinical signs.[10] Clinically healthy but infected individuals are often seen, which could play a major role in the epidemiology of the disease.[9,15] It appears clear that bornavirus infections cause an immune-mediated disease and that clinical signs are in line with signs of inflammation, in particular involving the nervous system tissues. These inflammatory changes are triggered as an immune response, rather than through direct cell damage. Transmission of T lymphocytes from borna disease virus (BDV)-diseased rats to infected but immunoincompetent, healthy rats induced clinical signs, clearly indicating a T-cell–mediated disease, at least in rats.[49] In rats, a neural invasion of CD8 T lymphocytes seems to cause the cellular damage rather than the virus itself.[49–53] It is fair to speculate that a similar pathogenesis can be presumed for ABV. A delayed-type hypersensitivity effect, as suggested by Payne et al.,[54] could fit with the hypothesis of clinical disease resulting from a T-cell–mediated response.

Hameed et al.[55] showed that in cockatiels, a treatment with cyclosporin A (which reduces the immune T-cell response) was successful in preventing clinical signs. Gartner et al.[56] evaluated the age of the bird at the time of infection as a factor for developing clinical disease. For this purpose, the authors compared differences in latency, development, and severity of clinical signs, as well as shedding of virus and seroconversion between newly hatched cockatiels (1–6 days old; with a non–fully competent immune system, $n = 11$) and adult birds ($n = 11$) following intravenous inoculation with a PaBV-4 isolate. All 22 birds seroconverted and demonstrated PaBV-RNA shedding during the investigation period of 233 days. However, the juvenile group seroconverted and began shedding virus RNA earlier and more homogeneously than the adult group. In contrast, only one juvenile bird died following a bacterial septicemia, whereas 9 of 11 birds from the adult group developed clinical signs, of which 5 eventually died or were euthanized. Postmortem examination of the birds revealed eight birds of the adult group to demonstrate a dilatation of the proventriculus versus none of the juvenile group. In all birds, PaBV-RNA was detected in various tissues, followed by successful virus reisolation. Clinical outcome of a PaBV infection thus seems to be influenced by the age of the host at the time of infection, with infection at a younger age, when the immune system is not fully developed, seemingly predisposing to the development of asymptomatically infected birds. This is of particular importance, as eggs originating from birds infected as chicks allowed vertical transmission,[48] hence increasing the likelihood of production of positive offspring and potential spread of the infection across the flock. However, it remains unclear whether PaBV-RNA–positive embryos, originating from parents infected as adults, remain positive during their development, thereby developing into asymptomatically infected carriers as well. The development of carriers is a key factor to understand the epidemiology of the disease and is essential to manage the virus in psittacine populations, especially in species conservation programs where the exclusion of positive birds is very difficult due to the low number of birds available.

Viral factors may also play a role in the pathogenesis of a PaBV infection. So far, it has not been determined which factors are of importance. Different ABV strains act differently within the same host, by terms of viral replication and pathogenicity. It was experimentally shown that PaBV-2 was more pathogenic to cockatiels than PaBV-4 and that viral RNA shedding occurred significantly earlier in PaBV-4–infected birds compared to those with PaBV-2. However, seroconversion occurred significantly earlier in the PaBV-2 group.[57] In the PaBV-2–infected group, reisolation of the virus from birds that died earlier was only successful after several passages compared to the birds that died late, in which reisolation was already possible after the first passage. This indicates a lower viral load in birds dying quickly after infection, hence demonstrating that viral load (and viral replication) seemingly does not correlate with the severity and progress of the disease. This underlines the hypothesis that the virus induces disease through mechanisms independent from the viral load (e.g., earlier activation of the immune system), or even that earlier activation of the immune system may prevent quick virus replication, but nevertheless cause more severe disease. Hence, immunocompetency at the time of infection and other immunological factors, or factors influencing the strength or weakness of the immune system, are likely to be key factors in the clinical outcome of infections.

CLINICAL SIGNS OF AVIAN ORTHOBORNAVIRUS INFECTION

In general, it seems that all orthobornaviruses found in birds have the potential to cause clinical illness. Infection with subsequent clinical signs has been reported in Passeriformes,

Anseriformes, and Psittaciformes. Recently, ABBV-1–associated clinical signs, including esophageal dilation and encephalitis, were reported in free-ranging Canada geese *(Branta canadensis)*.[21] Seeing the high prevalence of ABBV in free-ranging waterfowl, occurrence of clinical signs nevertheless seems to be a rare event, with most infections seemingly staying subclinical. However, it needs to be considered that in free-ranging birds, sick birds are often not found. Currently, psittaciform orthobornaviruses appear to be of large clinical significance to Psittaciformes as compared to the other groups. PaBV-2 and PaBV-4 have been clearly demonstrated as the causative agent of PDD,[1,57] a syndrome that is known for decades. PDD is therefore considered the main clinical sign of a clinically apparent PaBV infection. PDD is characterized by a nonpurulent, lymphoplasmacytic infiltration in neural ganglia of the peripheral nerves, particularly the autonomic nervous system of the gastrointestinal tract (esophagus, crop, proventriculus, and ventriculus).[58] As a consequence, peristalsis becomes impaired, and food accumulates in the proventriculus and crop.[59] Clinically, this results in maldigestion, passage of undigested seed in feces, and emaciation of the bird despite normal food intake. Birds can show ruffled feathers and often show reduced activity. As the virus may also cause an inflammatory response in the nervous system (i.e., lymphoplasmacytic ganglioneuritis), neurological disorders have been seen regularly by this infection and may occur more commonly than previously thought.[58] Clinical signs resulting from central nervous involvement may include head shaking or incoordination.

One study detected neurologic lesions in 82% (23/28) of birds naturally infected with PaBV-4. In addition, epicardial ganglioneuritis has been found regularly in naturally and experimentally infected psittacines,[60] which may explain sudden death in some PaBV-infected birds. A recent study demonstrated that PaBV infection was more prevalent in birds presenting with neurological disorders in the absence of PDD-associated clinical signs than in healthy birds presenting for routine examinations. Importantly, they had much higher antibody titers and demonstrated higher levels of viral shedding as compared to birds from the healthy control group.[61] Based on these results, the authors concluded that PaBV infection and associated disease should be included in the differential diagnosis of birds presenting with neurologic signs.[61] It has also been suggested that some feather-damaging behaviors may, at least in part, be signaled by PaBV infection. Birds with feather-damaging behaviors showed a higher prevalence of PaBV infection than control birds, with some having higher antibody titers and viral shedding. However, comparison to healthy controls was not as significant as compared to birds demonstrating neurological disorders.[61] Therefore, feather-damaging behavior (which often is a multifactorial condition) cannot be clearly linked to the presence of PaBV infection.[61] Further long-term studies would be needed to evaluate whether such a correlation, even in individual cases, does exist. Different virus strains seem to be associated with different disease patterns, with some predominantly affecting the CNS tracts leading to neurological signs without presence of gastrointestinal signs whereas others predominantly affect the enteric nervous system, causing gastrointestinal signs, in the absence of central neurologic disease.[57] Due to the consistent involvement of inflammatory changes surrounding the nervous tissues, several experts have suggested to rename the disease to avian ganglioneuritis

DIAGNOSIS OF AVIAN ORTHOBORNAVIRUS INFECTION AND PARROT BORNAVIRUS–INDUCED CLINICAL DISEASE

It is very important to differentiate between the diagnosis of the virus infection and the diagnosis of clinical signs induced by PaBV. As mentioned earlier, PaBV infection does not automatically lead to clinical disease, and clinically healthy carriers are regularly seen. These infected but asymptomatic birds seem to play a major role in virus transmission and distribution. On the other hand, they might become clinically ill due to other reasons while remaining positive for PaBV when tested. Therefore, it is important for the practitioner to be clear in regard to the goal(s) of the testing of the bird, i.e., to determine whether a bird is infected with the virus (e.g., for a prepurchase examination, transfer or release of birds, pairing of birds, establishment of flocks that are free from PaBV, etc.) or to investigate the underlying cause for clinical disease to determine a therapeutic plan or prognosis. Diagnosing the presence or absence of ABV infection in an individual as well as in a flock is explained in more detail in the first volume of this book. Here, a short summary is provided.

Diagnosis of Avian Orthobornavirus Infection

Veterinarians are regularly requested to investigate whether a bird is infected with ABV or not. This request is particularly common as part of a prepurchase examination to exclude entry of infected birds into a flock in an effort to establish and maintain a flock free from the virus, to transfer birds to other places (e.g., breeding, vacation care), or release birds back into the wild. In these situations, it is important to find out if a bird is infected and carries the virus, thereby posing a risk to other birds. For this purpose, it is important to keep in mind that it has lifelong consequences for the bird once it is diagnosed to be infected, as orthobornaviruses appear to cause lifelong infections in most birds. Infected birds may shed the virus for years without developing clinical signs, as was discovered following long-term monitoring of shedding by PaBV-infected birds. Nevertheless, the possibility that some birds might clear the infection cannot be ruled out. Some cockatiels that tested positive for PaBV-RNA in cloacal swabs at least once, showed discontinual of viral shedding and lack of signs of PaBV infection following necropsy.[62] Possible explanations for these findings are that birds were either able to clear the infection or that the cloacal swab tested positive due to passing PaBV-RNA ingested from other positive birds in the flock, while the bird with the positive test had actually never been infected. Usually birds shedding the virus do this for a very long time and most likely lifelong.

Like in other infectious diseases, diagnosis of ABV infection is focused on the demonstration of the pathogen in samples of the birds (direct proof) or the detection of specific antibodies against the pathogen (indirect proof). In veterinary practice, direct demonstration of the pathogen is most frequently accomplished by PCR. It must be underlined that different PCR protocols detect different avian orthobornaviruses, especially from different avian taxonomic groups. If focusing on the detection of ABV from bird groups other than psittacines (e.g., waterfowl, songbirds), specific PCR

protocols need to be used.[45,22] For psittacine birds, commercial laboratories commonly use real-time PCR protocols to detect PaBV-2 and/or PaBV-4 (which represent the most common strains in psittacines). However, this might lead to a false negative diagnosis if facing other PaBV genotypes or strains of the same genotype.[57] Therefore, it is vital to ask the laboratory which PCR protocol is used and which strains can be detected with the test, as this will help to identify whether the test will be useful to help answer the question that initiated the testing in the first place.

Best samples for ABV-RNA detection by PCR are crop and cloacal swabs (merged in one sample) in the live bird, or nerve tissue (e.g., brain, retina) or adrenal gland tissue from a deceased bird. Feces is not recommended, as it carries certain disadvantages for use in PCR, such as presence of inhibitors leading to false-negative results. It has also been shown that blood is not very sensitive as sample material, as blood samples from PaBV-positive birds regularly test negative for the virus.[63] Feather calamus has been suggested to provide adequate samples for testing, but experimental studies have demonstrated feathers to not be first choice sample material. Feathers also contain a higher risk of being contaminated by other birds or the environment leading to false-positive results. Samples for PCR must be stored cooled or in transport media (RNAlater, Qiagen, Hilden, Germany) and sent immediately to the laboratory. False-negative results might occur simply due to poor transport conditions. Repeated freezing and thawing of samples can also lead to false-negative results. Apart from false-negative results, birds might not shed viral RNA at the time of sampling. Hence, a negative PCR result does not mean that the bird is not infected. Repeated sampling, and especially serologic evaluation, is therefore important.

For a complete investigation, screening for anti-PaBV antibodies is indispensable. Some birds may shed viral RNA intermittently but will demonstrate specific anti-PaBV antibodies. This means without serological investigations for anti-PaBV antibodies such birds may be falsely classified as noninfected when tested only with PCR. On the other hand, some birds are detected as shedders without seroconversion.[10,64] This clearly demonstrates that only the combined investigation using PCR and serology will be able to provide a complete picture and allow for interpretation of clinical signs and infection status of a bird.

There are different assays available to detect anti–ABV-specific antibodies, including indirect enzyme-linked immunosorbent assay (ELISA) tests,[65] and Western blot[9] tests, which use specific recombinant proteins as antigens. When using a single protein as antigen, it is required that the protein demonstrates cross-reaction between different ABV genotypes to ensure a positive reaction when antibodies against any ABV subtypes should be detected. This is not ensured yet. Therefore, the sensitivity of tests focusing on one protein only needs to be questioned until otherwise proven. Comparison of these serological tests with tests based on infected cell cultures (providing several antigens) points toward a higher sensitivity for the latter method, especially when low antibody titers are present. However, comparison of the tests also showed that results between the methods were congruent in birds with high antibody titers as commonly found in clinically affected birds. Based on these findings, it may be concluded that results from ELISA or Western blot focusing on single proteins might be adequate for diagnosing clinically ill birds in daily veterinary practice, but not for managing flocks where the aim is to also detect clinically healthy carriers that might have low antibody titers. As a result, tests based on ABV-infected cell cultures (e.g., indirect immunofluorescence test [IFT]),[66] which present a wide range of different ABV antigens, are recommended if a higher sensitivity is needed, even though these tests are more difficult to perform.

Diagnosis of Parrot Bornavirus–Induced Clinical Disease

Diagnosis of PaBV-induced clinical disease is not easy, particularly in the living patient. As birds are commonly asymptomatically infected, detection of the presence of PaBV should not automatically be presumed as the cause of the clinical signs observed in a patient. Instead, other causes for the clinical signs must be ruled out in addition to the detection of a PaBV infection by PCR and serology (Figure 14.1). Typical clinical signs that are induced by PaBV are maldigestion, which may present by shedding of undigested seeds in feces, as well as signs indicative of neurologic disease, such as ataxia, convulsions, or tremors. Experimental trials have shown that some birds only demonstrate gastrointestinal signs (referred to as PDD), whereas others only show neurological signs, and others show involvement of both organ systems. Whether and which organ system is involved seems to be related to the individual bird, the age at which the bird got infected, and the PaBV strain involved. In birds with gastrointestinal signs, radiographic imaging should be considered to evaluate the size of the proventriculus. If the proventricular silhouette is found to be enlarged with a thin-walled proventricular lining (as can be demonstrated during contrast radiography or fluoroscopy), PDD becomes more likely as several other diseases (including infection with *Macrorhabdus ornithogaster* or *Candida* spp.) more commonly lead to a thickening of the proventricular wall. A fecal cytologic examination from the bird with Giemsa or Diff-Quick will easily demonstrate *M. ornithogaster* should these organisms be present, whereas a crop swab stained by methylene blue can help to rule out the presence of *Candida* spp. Contrast radiographic or fluoroscopic imaging will also provide evidence for foreign bodies, which should be listed in the differential diagnosis for PDD. As other systemic diseases or infections might lead to gastrointestinal signs, a blood chemistry and hematological profile should be performed. If abnormal, the data obtained might point toward other disease processes than PaBV-associated disease. Hematological and blood chemistry values are seemingly of little significance to PaBV-induced clinical disease. After experimental infection, grey parrots *(Psittacus erithacus)* showed only minor hematological alterations.[67] Blood chemistry values of aspartate aminotransferase, creatine kinase, total protein, glucose, and uric acid did alter compared to values of the same individuals prior to infection, but not in a predictable degree. An increase in γ-globulin fraction was noted 10 weeks postinfection, but by week 34, postinfection values decreased again to levels like those noted before infection. During the trial, three birds died of typical PDD signs. However, none of these birds demonstrated alterations in their blood panel prior to death. This means that significant alterations in the blood profile could point toward other diseases as the underlying cause for the clinical signs.

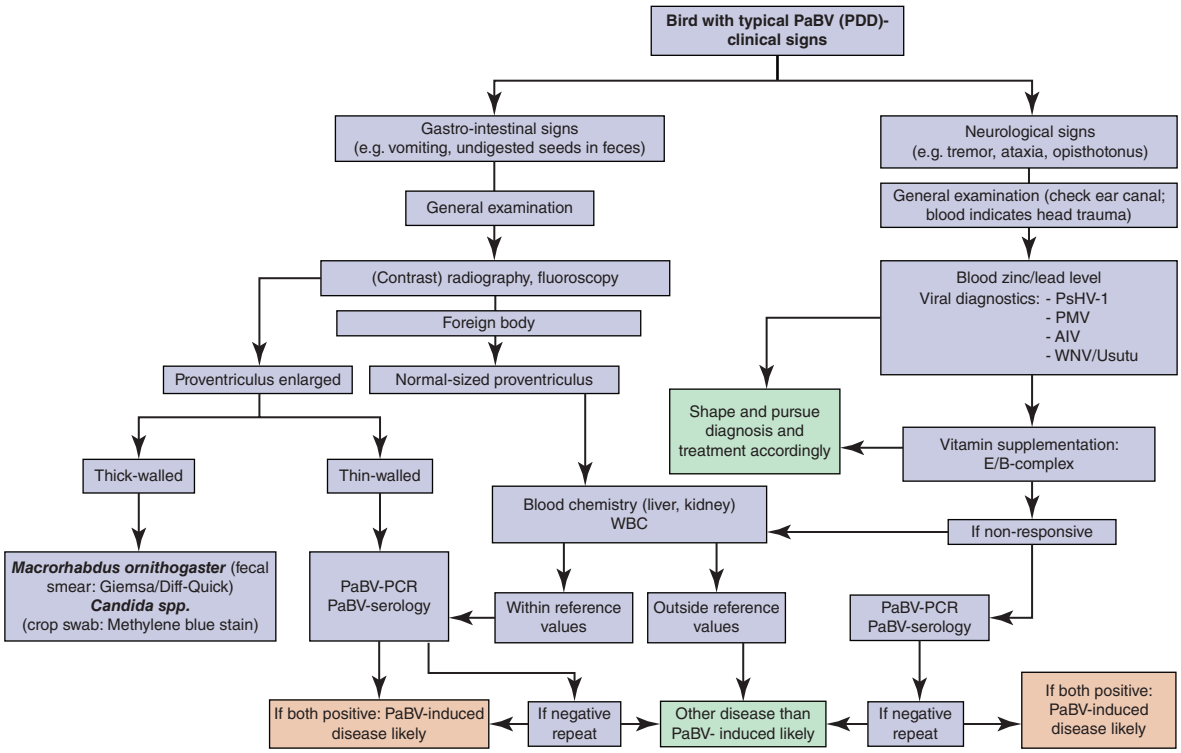


FIGURE 14.1 Diagnostic process schematic for a bird presenting with compatible clinical signs attributable to bornavirus infection, but which could also include a differential diagnosis of other disease processes. Because avian bornavirus infections are common in birds without causing clinically apparent disease, a bird can be showing clinical signs due to another disease process or combination of conditions while additionally being positive for bornavirus. Ruling out and identifying comorbidities that could contribute to the presenting clinical signs is important, in addition to diagnostic investigation for bornavirus infection. *AIV*, Avian influenza virus; *PaBV*, parrot bornavirus; *PCR*, polymerase chain reaction; *PDD*, proventricular dilatation disease; *PsHV-1*, Psittacine Herpesvirus 1; *PMV*, Paramyxovirus; *WBC*, white blood cell; *WNV*, West Nile virus.

If neurologic signs are present, heavy metal intoxication (zinc, lead), vitamin deficiencies, and infections with paramyxoviruses, orthomyxoviruses, herpesviruses, and flaviviruses should be on the list of differential diagnostic considerations, hence necessitating these to be ruled out. This can be done by toxicologic and viral testing and by supplementing the bird with vitamins, which should lead to a fast recovery if a deficiency was present. Similarly, trauma should be ruled out as a cause. Radiographic imaging may be helpful to identify the presence of anatomical alterations (e.g., aspergillosis) or detect the presence of a dilated proventriculus suggestive for PDD. To exclude metabolic causes, a complete blood biochemistry and hematologic profile can be performed.

Crop and cloacal swabs should be taken for PaBV PCR, whereas serological samples can be collected to detect anti–PaBV-specific antibodies. It cannot be emphasized enough how important it is to combine both PCR and serology to maximize the chances of detecting a PaBV infection. Field observations as well as infection trials demonstrate that birds developing clinical disease from a PaBV infection do shed viral RNA and will often demonstrate high antibody titers. Especially titers seem to be an excellent predictor for association of the clinical signs with a PaBV infection. However, it needs to be mentioned that there is no exact "limit" to the antibody level confirming the diagnosis, as interpretation of the serological results highly depends on the test that is used. Therefore, the laboratory should set its standards regarding what is considered a low, moderate, or high titer. In experimental settings, birds with clinical PaBV infection will often demonstrate seroconversion, even in the rare event of a quick death, prior to shedding viral RNA. If PaBV-RNA is detected together with high antibody titers and presence of typical clinical signs in the absence of evidence for the most important other differential diagnoses, it becomes very likely that clinical signs are PaBV related.

Very few cases will demonstrate typical PaBV-associated clinical signs with negative PaBV PCR and serology while all other differential diagnoses have effectively been excluded. In such cases, it should be kept in mind that due to the high variability of PaBV, primers might fail to detect a specific PaBV genome or that antibodies are not detected due to the selection of a serologic assay using only a single antigen. Therefore, negative PCR results especially should be interpreted in the context of the type of PCR that has been used, and balanced against the clinical presentation and serological test results as well as the possibility of occurrence of novel genomic variants that are not readily detected. In such cases, further testing should be considered and discussed with the laboratory. This may include retesting of birds after 2 weeks, as seroconversion may have taken place, thereby allowing for

detection of seropositive birds that were initially negative on serology.

Owners with clinically healthy birds sometimes inquire about testing for PaBV infections to obtain a prognosis regarding the lifespan of their bird(s) or the potential onset of clinical disease. Such a prognosis cannot be given, as the factors influencing the development of clinical disease have not been fully elucidated. Birds with low viral shedding or low antibody titers might demonstrate an increase in viral shedding or rise in antibody titers and develop clinical disease some weeks later. On the other hand, several birds that demonstrated high titers and/or high viral shedding have remained clinically healthy for several years. This last group might represent carriers, which may have been infected vertically or early in life and might remain infected their entire without ever developing clinical disease.

Before PaBV was proven as the cause of PDD, antiganglioside antibodies were proposed to cause the nonpurulent inflammation typical for PDD as part of an autoimmune disease. At present time, the detection of antiganglioside antibodies is considered by some as a PDD test, used to gain inductive strength in diagnosing clinical disease. However, this would imply that the presence of those antibodies leads to the development of PDD, hence suggesting that these should always present in birds with PDD. Unfortunately, thus far there is no proven evidence for this, even though this theory might have its merit. Early comparative studies have shown a poor correlation between antiganglioside antibody levels and PDD-associated clinical signs, as well as between PaBV infection and the occurrence of antiganglioside antibodies.[68] Further doubt is raised by studies immunizing quaker parrots (*Myiopsitta monachus*) with gangliosides (producing antiganglioside antibodies), following which no PDD-like lesions were induced.[69] Hence, confirming infection through detection of the causative agent seems more straightforward. However, if a test were to be developed that would be able to confirm the presence of PDD resulting from PaBV infection or the use of antiganglioside test were confirmed for support or confirmation of diagnosis, this might be helpful in daily clinical practice. To evaluate the merit of such novel tests, studies with experimentally infected birds are necessary, as this will allow for correlation with the time point following infection and the occurrence of clinical disease.

To summarize, a test for PaBV infection (neither PCR nor serology) *is not* a test to detect PDD. Typically, PDD can only be diagnosed by histopathology or by demonstrating PaBV infection in the presence of the typical clinical signs and following exclusion of other causes for the clinical signs. In such cases, PaBV PCR and serology are indispensable and can strengthen the diagnosis by considering the amount of shed PaBV-RNA and antibody titer.

Diagnosis of Parrot Bornavirus Infection to Identify Positive Birds

Independent from clinical disease, there is a strong request to test individual birds for PaBV infection. This is very useful as it allows for control of the virus and sanitizing flocks. Isolating PaBV-positive birds will certainly lead to reduced transmission, and clearing flocks will lead to elimination of the virus, hence reducing the number of clinical cases. As such, tests to rule out PaBV infection are highly recommended in prepurchase examinations, when translocating birds to other collections, when releasing birds back into the wild, or when adding new individuals to pathogen-free flocks. In these circumstances, in which the tested birds are usually clinically healthy, combined testing using PaBV PCR and serology is warranted.

Birds with positive test results in both PaBV PCR and serology should be considered as clearly positive. Those being positive by PCR only should be retested, to confirm the result and exclude contamination of the sample. It is advised to retest 4 to 6 weeks later, as this helps to evaluate whether seroconversion has occurred in the meantime. During this time, birds should be separated from other birds. In case of seroconversion or repeatedly positive PCR results, birds should be considered positive. Birds positive by serology only are likely to be infected, as PaBV seems to cause lifelong infections.[29,62] However, despite this being likely, lifelong infectivity has not been proven and with existing evidence and reports regarding birds that have been able to clear infection, conclusions should be drawn with some caution.[65] The height of antibody titers may be helpful to determine whether retesting is needed. Birds with a high PaBV antibody titer usually do not vary in their results; hence there is no immediate need to retest them. However, retesting is warranted for birds with low titers that are negative in additional tests, as evidence for persistent infection is lacking in these. In such cases, false-positive serological results from cross-reaction with antibodies directed against other antigen cannot fully be excluded albeit being unlikely. Separating these birds from other birds and retesting them after 4 to 6 weeks using PCR and serology is therefore recommended. If titers are maintained or increase and/or if viral RNA is detected, these birds should be considered positive. Decreasing titers or seronegativity, together with negative PCR, warrants retesting after 4 to 6 weeks. Only if repeated testing demonstrates the bird to remain seronegative and there is repeated failure of virus demonstration by PCR, the birds can be considered ABV negative. Figure 14.2 provides an overview of suggested interpretation of test results to categorize tested birds.

The main goal in controlling PaBV is to clear flocks from the virus. This should be feasible, since the virus is not easily transmitted horizontally between two birds (see Avian Orthobornavirus Transmission). Efficacy of this approach has been demonstrated in recent years, even in large breeding flocks involving several thousands of birds. Clearance of PaBV from flocks will also result in less infected birds being sold to the market, thereby decreasing the spread of the infection and resulting in less pet birds being affected by clinical disease. Hence, viral control is the best measure to prevent disease. Managers of large flocks cleared from PaBV report no further PDD cases, as compared to discovering several cases per year before the flock achieving a specific pathogen-free status. However, clearance of PaBV from a flock can be a long, expensive, and sometimes frustrating task and will only be successful with strict compliance of the owner. Requests to investigate a flock are common, but when owners or managers learn that, at least initially, it is likely that a considerable number of birds will test PaBV positive, conflict arises, in particular when this implies the need to separate or exclude expensive and successfully breeding birds from the collection. Therefore, owners should be counseled as to exactly what is involved and expected at the onset of the effort to establish a PaBV-free collection.

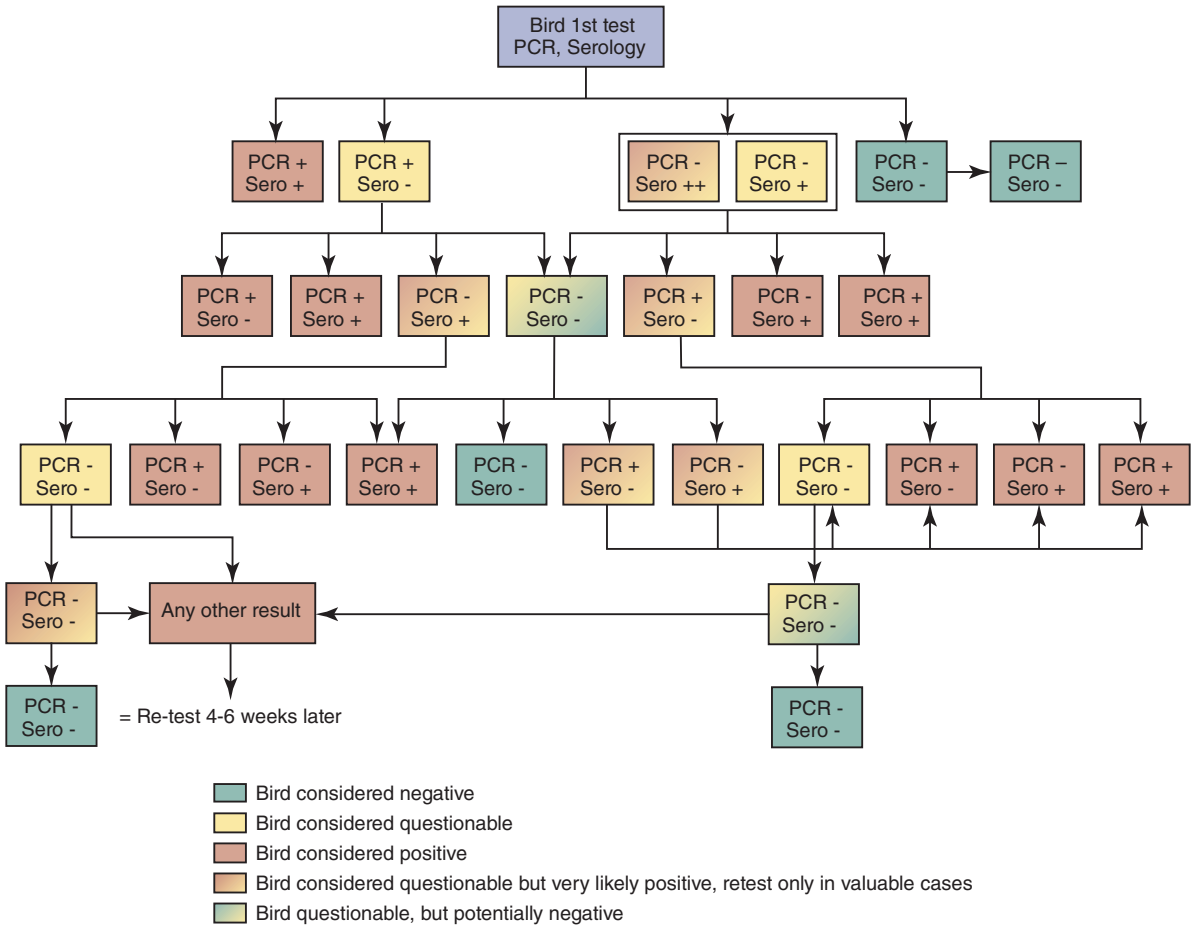


FIGURE 14.2 Suggested interpretation of laboratory test results to categorize birds as positive or negative for avian bornavirus (ABV). Polymerase chain reaction (*PCR*) should be made from a pooled cloacal and crop swabs of each bird, serology with a multiprotein enzyme-linked immunosorbent assay (ELISA) or preferably immunofluorescence test based on ABV persistently infected cell culture. Each *box* represents an individual bird, and so all the potential test results can be seen by following a box through the complete diagnosing process. Each *arrow* represents a retest 4 to 6 weeks apart with combined PCR and serology. Usually, most of the birds tested are assigned to a final result after two (second row) or three tests (third row). Only in very rare cases, more tests are necessary, or the birds should be considered positive. First of all, the individual bird is tested and then assigned a positive, negative, or questionable status. *Red* means that those birds should be taken as positive. *Yellow* indicates that there is a questionable interpretive status and that from those birds all possibilities may arise; hence, a retest is necessary. *Green* birds are negative, but it is advised to have at least two negative tests of one individual bird to ensure negativity. In the circumstance that a bird is considered positive, no retest is necessary. Often in clinical circumstances, a negative bird is considered as such in a single test. In the case where the bird is planned to be sold or integrated into a known negative flock, the test should be repeated, and only with a second negative result is the bird considered negative. In case a bird has a high antibody titer with a negative PCR, it should be considered positive. However, in the case of a bird that is deemed very rare or valuable species or individual, the test may be repeated. These birds are depicted as *red/yellow*. Questionable birds are retested and usually will be assigned positive or potentially negative status. As described previously, only in very rare events birds being positive in one of the tests again may still be considered questionable and tested again. The vast majority of these previously positive cases, when retested, will remain positive. Potentially negative birds with a prior questionable status are tested a third time and are only considered negative if they are found to be negative again, i.e. after having two consecutive negative tests. The odds that from a *red/yellow box* a result other than a positive can occur are very low, according to the experience of this author, but can theoretically happen. −, Negative test result, +, positive test result, ++, high anti-ABV antibody titer. (From Lierz M. Avian bornaviruses and proventricular dilation disease. In: Speer B. L., editor. Current Therapy in Avian Medicine and Surgery. St. Louis, MO: Elsevier, Inc.; 2016. pp. 28–46.)

All birds of the flock need to be tested by PCR and serology. After the first round of tests, three groups will be established according to the diagnostic criteria discussed previously (Figure 14.2). These three groups, of which the first contains the positive birds, the second the questionable birds, and the third the negative birds, need to be kept epidemiologically separated. The assignment of birds to one of these groups should be purely based on the laboratory results, not on the owner's preference or pairing status of the birds. If an owner prefers to not split a pair when one partner is positive and the other negative, then both birds should be placed in the positive group. Owners might also decide to relinquish the positive birds, which can be a difficult task and might only be accomplished by rehoming them to private owners to be kept as pets. Euthanasia of positive birds is sometimes also requested, but this should only be considered in clinically affected birds. Ethically, the euthanasia of PaBV-positive but clinically healthy birds is hard to justify and not recommended. In breeding programs with several partner institutions, it should be considered to have one of the institutes establish a separate breeding flock with positive birds (which are kept strictly isolated from the negative birds), as it is feasible to produce negative offspring from this flock.

The second phase involves retesting of all birds from the questionable and negative groups after 4 to 6 weeks (retesting always implies a combined PCR and serologic test performed simultaneously). Based on the new results, some birds from the questionable group will now have to be regarded as positive for PaBV and will thus be transferred to this group, whereas others are potentially considered negative and will remain in the group of questionable birds, while separated from the others that still have a questionable status (Figure 14.3). In the negative group, some birds might retest positive, necessitating a transfer of these birds to either the positive or the questionable group, whereas others remain negative and can remain in the negative group. Once the negative group reveals negative results for both PCR and serology in all birds from this group on two consecutive test moments performed 4 to 6 weeks apart, the negative group can be considered as clear(ed) from infection. Once birds from the questionable group are tested negative on PCR and serology on two consecutive occasions, without having had contact with birds that had positive results in the meantime, these birds can be transferred to the negative group. Over time, the questionable group will therefore dissolve, as birds will be transferred to either the negative or positive group. Especially in a flock setting, sample contamination or passing of PaBV-RNA originating from other positive birds in the flock is possible. Special care should thus be taken to prevent this, as this may necessitate additional retesting. After all, whenever one or more birds from the questionable or negative groups show up as being positive in one or both tests, the risk remains that virus was transferred from this bird to another individual from the group, therefore warranting retesting of all birds from this group until no further positives are identified on two consecutive tests.

If positive birds are rehomed as pets or positive flocks from different origins are created, it might be worthwhile to investigate which PaBV genotype infected the bird(s), as infection of PaBV-4–positive birds with PaBV-2 has been reported to trigger clinical disease.[36] As stress might trigger the development of clinical disease in PaBV-infected birds, clinical disease may ensue following relocation of positive birds to the new home. This warrants new owners to carefully monitor these birds following arrival so that onset of disease is caught as early as possible.

Further details on flock management, especially in managing positive breeding birds, can be found in the first volume of this book.

THERAPY

To date, there is no proven treatment to control ABV infection in birds. In studies using bornavirus-infected cell lines, ribavirin was able to reduce the viral load and bornavirus activity.[70,71] If the treatment was combined with IFN-α, the observed effect was stronger.[70] Clinically, ribavirin treatment was found ineffective on its own,[72] but the proposed combination of drugs has yet to be evaluated in infected birds. Favipiravir (T-705) showed an effect on orthobornaviruses in vitro,[73] but *in vivo* studies evaluating its effect are also still lacking.

Currently, treatment options focus on the reduction of clinical signs of disease, rather than antiviral treatments. One strategy is to use immunosuppressive drugs to reduce the ABV immune-mediated lesions, while adjusting the dosing regimen based on observed clinical signs during follow-up visits. The other strategy focuses on reducing the inflammatory lesions associated with the disease.

Immunosuppression is accomplished with cyclosporine A, which induces a selective T-cell suppression and has been described as promoting a favorable response in single case reports.[74] Cyclosporine was used at 10 mg/kg per os (PO) BID in combination with a COX-2 inhibitor in birds where the COX-2 inhibitor alone did not show any effectiveness. In those cases where combined immunosuppressive and COX-2 inhibition were used, reduction of clinical signs has been observed in single cases.[62] Nevertheless, (placebo-) controlled studies in infected and clinically ill birds are required to definitively demonstrate its benefits. Moreover, as immunosuppression can potentially increase the susceptibility to other infections, the risks and benefits of such treatments need to be carefully weighed.

To reduce the perigangliar and perineural inflammation, the use of anti-inflammatory drugs has been advocated. Meloxicam is widely used with variable success in dosages ranging from 0.5 to 1 mg/kg intramuscularly (IM) or PO BID or SID. In one study, meloxicam was administered in a dose of 0.5 mg/kg PO BID to cockatiels challenged experimentally with PaBV-4. Surprisingly, challenged birds that received the drug showed more severe clinical signs and higher incidence of death compared to birds that were only challenged or only received the drug.[75] However, based on more recent insights, the dose that was given could have been too low to exert an effect. Alternatively, the frequency of handling of the birds from these groups was different, which may have negatively influenced the results, as stress is suggested to influence the onset of clinical disease. A recent study investigated the effect of meloxicam (1.0 mg/kg PO q24h) and celecoxib (10 mg/kg PO q24h) on the clinical outcome of PaBV-2 experimentally infected cockatiels. Treatment with either drug was started

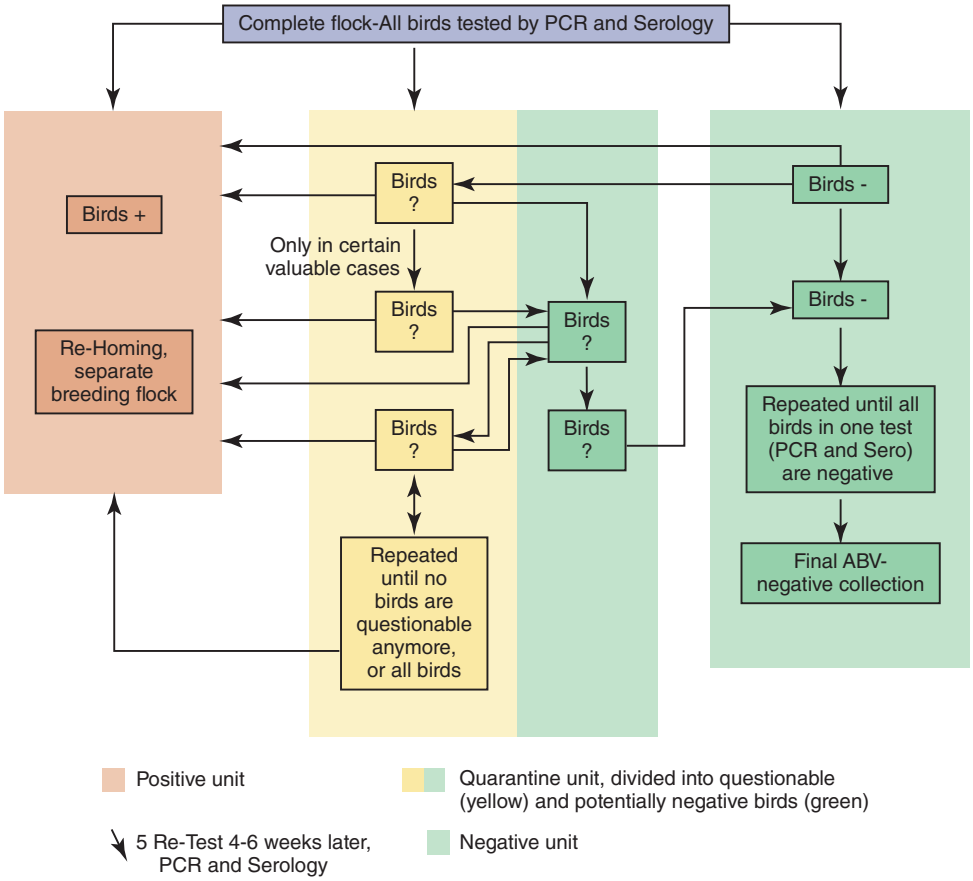


FIGURE 14.3 Suggested diagnostic plan to clear a flock from avian bornavirus (ABV) infection. The *large box* represents the epidemiologic units (populations), which should be clearly separated and treated as independent populations. The *smaller boxes* represent the group of birds that demonstrated the same results (positive, questionable, negative). Each *arrow* represents a retest 4 to 6 weeks later of all birds in that group at the same time. It is important that all birds within these groups are sampled on the same day and that polymerase chain reaction (*PCR*) and serology are performed always performed simultaneously on an individual bird. First, the complete flock is tested, and three major groups are established (positive, questionable, and negative). A retest 4 to 6 weeks later places additional birds from the negative group into the positive and questionable groups and from the questionable group to the positive group and establishes a potentially negative group within the quarantine section. The negative group is only ultimately considered ABV negative if all birds in that group are tested at the same time and are found to be negative in two consecutive tests. Questionable birds within the questionable groups are only placed in the negative group after two negative testing rounds while housed separately within the isolation area from the others after their first negative test result. Birds that are retested as questionable (very rare; see Figure 14.2) should be considered positive for the purpose of clearing a flock of ABV; however, in very rare or valuable species, the risk might be taken to keep them in quarantine in the questionable group. However, should those birds test questionable again, they should definitely be considered positive. The isolated population is tested until all the birds are assigned to one of the other groups (positive or negative) or the owner decides that the remaining birds should not be retested, and at that time, all remaining birds in isolation will be assigned to the positive group. Once a bird is assigned to the positive group, it will remain there. Reprinted from: Lierz M. Avian bornaviruses and proventricular dilation disease. In: Speer B. L., editor. Current Therapy in Avian Medicine and Surgery. St. Louis, MO: Elsevier, Inc.; 2016. pp. 28–46.

23 days postinfection; however, no differences were detected between the two treatment groups or the untreated infected control group in the clinical outcome or histopathological lesions.[76] As a result, the beneficial effects of anti-inflammatory drugs have not been definitively proven.

At present, the use of the selective COX-2 inhibitor celecoxib remains the treatment of choice for birds with PaBV-induced clinical signs. In the past, dosages of 10 mg/kg orally SID for 6 to 12 weeks were recommended.[77,78] Based on anecdotal reports from varying practitioners, the current

recommended dosage is 13 to 30 mg/kg PO BID for several weeks. If severe CNS signs occur, the use of higher dosages (up to 30–40 mg/kg PO BID) has been suggested.[79] Before the discovery of ABV tepoxalin, a combined COX-1, COX-2, and 5-lipoxygenase (LOX) inhibitor was experimentally used to treat PDD-related signs.[78] A treatment effect was seen only by administering 40 mg/kg and placing the birds on a rice-based hypoallergenic diet. In the same dose on a seed diet, no treatment effects were seen,[80] hence making its efficacy in PDD cases questionable.[80] The use of robenacoxib, another COX-2 inhibitor, has also been reported in the treatment of birds with PaBV-related clinical signs at dosages from 2 to 10 mg/kg IM weekly for 4 weeks and then monthly.[62,79] In many cases, treatment will be pursued for several months, whereby cessation of treatment is regularly suggested to lead to recurrence of clinical signs, necessitating treatment to be resumed either sooner or later. Bird owners should therefore be informed that these treatments do not cure the bird, but rather serve to help increase their lifespan.

A novel, yet interesting, approach includes the use of gabapentin, a gamma-aminobutyric acid agonist, which is commonly used to relieve neuropathic pain. In the author's experience, the drug has had excellent effects in birds with chronic pain or chronic, neurologic lameness, especially in cases where other drugs for pain management such as nonsteroidal anti-inflammatory drugs (NSAIDs) have not been successful. For birds with PaBV-related CNS signs, including seizures and ataxia, dosages of 10 to 25 mg/kg PO BID have been recommended,[62] whereas dosages of up to 50 mg/kg PO have been recommended in cases that demonstrate self-mutilation.[79]

VACCINATION

Efforts to produce an effective vaccine against orthobornavirus infections have been made, with the primary aim to protect birds from clinical disease. Some vaccination trials were performed with recombinant live viruses. Newcastle disease virus (NDV) and modified vaccinia virus Ankara (MVA) were produced to express PaBV-4 phosphoprotein (P) and nucleoprotein (N), respectively.[81] Cockatiels were subsequently vaccinated with the mixture of NDV-PaBV-4/P and NDV-PaBV-4-N strain and boosted with MVA-PaBV-4/P and MVA-PaBV-4/N virus at day 21 or at day 14 and 29. The obtained results demonstrated that the NDV-virus replicated in the cockatiels and caused a self-limiting infection. The first anti-PaBV antibodies were detected between 28 to 42 days postvaccination, and titers did not increase further after the second vaccine booster. Challenge with a PaBV-4 isolate demonstrated both a reduced shedding of PaBV-RNA and a reduced viral load in investigated tissues in the vaccinated group compared to the challenged, nonvaccinated control group.[81] However, birds of the vaccinated groups still developed clinical signs of PDD. In another group of six cockatiels, which were vaccinated using the same protocol but challenged with a heterologous PaBV-2 at a lower infection titer, only one bird developed a PDD-typical inflammatory response, whereas viral load in different tissues could only be demonstrated in two birds.[82] It remains unclear why partial protection was seen upon heterologous challenge with PaBV-2 but not following challenge with homologous PaBV-4 virus. However, as the minimal infectious doses of PaBV and the route of transmission at the time of this study were unknown, interpretation of the results remains difficult as both factors may have had a huge influence on the results of the study. A vaccine study with similar study design was performed in canaries, using NDV and MVA viruses expressing CnBV-2 phosphoprotein or nucleoprotein and challenging the birds with a CnBV-2 strain postvaccination. Here, viral challenge was repeated after 12 weeks, as the first challenge did not lead to infection. Subsequent findings demonstrated a significant reduction in shedding of CnBV-2-RNA, but virus distribution was demonstrated in all tissues of the birds. All canaries seroconverted and titers stayed high, indicating persistent infection.[81] A different study using different recombinant viruses carrying PaBV showed that cockatiels that were challenged with PaBV-2 following vaccination and two boosters with NDV-PaBV-4 or Orf virus constructs (ORFV-PaBV-4) were not protected and became permanently infected with PaBV-2. Cockatiels vaccinated and boostered twice with MVA-PaBV-4, in contrast, showed protection when challenged with PaBV-2, with only one bird developing a latent infection of PaBV-2.[83] Challenge with PaBV-4 virus was not investigated in this study. In another study, inactivated PaBV-2 was used as a vaccine. Even after two booster vaccinations with this vaccine, birds did not seroconvert. However, they did seroconvert following an additional booster with a recombinant PaBV-4 nucleoprotein, with 7 out of 9 cockatiels producing PaBV-specific antibodies. A second group was vaccinated twice with the recombinant PaBV-4 nucleoprotein. All birds were subsequently challenged with a PaBV-2 isolate. In all groups, PaBV-RNA shedding as well as a persistent infection in various tissues were observed. Similarly, pathological lesions typical for PDD were observed in some of vaccinated birds from either group, but to a much lesser extent than in the controls. Vaccination trials have also been performed with birds already infected with PaBV. While vaccination of these birds did not induce clinical signs during the follow-up period, it also did not reduce the virus load in tissues, hence indicating that the used vaccination protocols are unsuccessful in clearing the infection.[55]

To date, a reliable vaccination protocol has yet to be established. Recombinant vaccines appear to offer the best protection against heterologous challenge. However, many questions still need to be answered, such as protection against homologous challenge or against other PaBV types. It also remains unclear how long this partial protection lasts, or whether latent infection in vaccinated birds will develop into clinical disease at a later stage. Vaccination of already infected birds did not result in clearance of the infection in any of the cited studies. Although in the future, vaccination protocols might offer (partial) protection against clinical disease, uncertainties remain whether a sterile immunity against PaBV can be reached. If carrier birds remain after vaccination, vaccination strategies cannot be recommended as the mainstay of PaBV control. In addition, flock management strategies, which include the use of serology paired with PCR to detect carrier birds, may be complicated by vaccine-induced seroconversion. Especially in the light of the novel knowledge about transmission routes, the best option to control the virus globally is to sanitize flocks from the infection. Vaccination, inducing titers, but leaving carriers, may interfere with this important strategy.

TAKE-HOME MESSAGE AND OUTLOOK

In recent years, large steps have been taken to further our understanding of ABV infection in birds, including its transmission and factors influencing the clinical manifestations of disease. It is becoming increasingly obvious that understanding the details of viral transmission offers a large window of opportunities to help control the virus within flocks, which will subsequently lead to a reduction of clinical cases seen in practice. As the virus is not readily transmitted horizontally between birds, separation and repeated testing of birds from a flock provides good opportunities to establish and maintain orthobornavirus-free collections. Transmission and time point of infection may also be key to understanding which circumstances lead to development of clinical disease. While further clarification is needed, there is strong evidence to indicate that birds infected later on in life (through horizontal transmission) are more prone to develop clinical disease, whereas birds infected early on in life (through vertical transmission) seemingly develop into carriers. These findings also disprove the theory that infected yet clinically healthy birds are genetically resistant to develop clinical disease and should therefore be selected for breeding. In the absence of proof of heritability of clinical resistance against infection, and in light of the results regarding vertical transmission, infected yet clinically healthy birds are more likely to produce carriers due to vertical transmission. Thus preventing infection is still the best prophylaxis from clinical disease. This is important, as there is still no proven effective therapy. In future, novel vaccination strategies might lead to effective immunity and will then represent another option to control infections. Until then, if no effective vaccination strategies are in place, flock management and retesting birds remains the best way of controlling the virus (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Carrier birds need to be identified and separated. Similarly, breeding circumstances need to be critically evaluated in relation to the potential for vertical transmission as this may allow to produce negative offspring from positive parents. This seems to be especially important for species conservation projects where the number of breeding birds is limited. In addition, there should be a close monitoring of viral occurrence and tracking of bird movements and releases into the wild. Although prevalence of ABVs in the wild is seemingly low, there is a risk of introducing novel strains into a naïve population, which can have a severe negative impact on species conservation and should therefore be avoided at all costs.

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CHAPTER 15

Poxvirus

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Avian poxvirus is a group of large, relatively stable, double-stranded DNA virus strains that include fowlpox, pigeon pox, canarypox, and other poxviruses of birds. All of the strains are in the genus *Avipoxvirus*, subfamily Chordopoxvirinae. Strains are somewhat species-specific but can occasionally affect other bird species. One strain may not cross-protect with another. Poxviruses are "old" viruses recognized more than a century ago and are known to affect more than 232 species of birds worldwide.[1] However, new isolates/species/strains are frequently reported (Table 15.1). Poxviruses can be particularly devastating to endangered species.[2] Vaccines are available for some of the strains and/or avian species affected.

AVIPOXVIRUS FAMILY

Fowlpox virus is the type strain for the avian poxvirus family. Basic morphology for all strains is a large brick-shaped virus. Historically, 10 separate species have been recognized using restriction fragment length polymorphism (RFLP) analysis of DNA. These include fowlpox virus, canarypox virus, juncopox virus, mynah pox virus, psittacine pox virus, sparrowpox virus, starlingpox virus, pigeon pox virus, turkeypox virus, and quailpox virus (International Committee on Taxonomy of Viruses). Both fowlpox virus and canarypox virus have been completely sequenced.[8,9] The avian poxvirus family recently has been phylogenetically separated into different clades.[2,10,11] The relationships among species are determined using the P4b core protein–coding region and the DNA polymerase gene.[2,12,13] Fowlpox virus, canarypox virus, and poxviruses from psittacine birds form the three major clades, with most of the avipoxvirus species showing relatedness to one or the others. Poxvirus is not a public health concern, with only one mammalian isolation of fowlpox virus being reported, found in a rhinoceros.[14]

EPIDEMIOLOGY AND TRANSMISSION

Clinical infection is most frequently observed in warm regions. Fowlpox infection is transmitted horizontally through mechanical transmission of the virus through injury or breach of the skin. The primary vectors are insects, specifically mosquitos. Other insects including chicken mites *(Dermanyssus gallinae)* and 11 different species of Diptera have also been reported to be mechanical vectors.[15] There is no evidence that the virus replicates within the vector.[16] Aerosolized dried scabs and dander can infect via the respiratory route. Viral movement within a flock is aided by cannibalism of lesions on other birds or via aerosol generated by feathers and dried scabs containing the virus.

The virus, once entering the epithelium, replicates within the cytoplasm and varies greatly in duration. Electron microscopic evidence suggests that pH-independent fusion with the plasma membrane is the primary route of virus entry into the cell. The virion protein and lipid are lost, followed by exposure of the genome to DNase, allowing almost immediate synthesis of virus-specific transcripts, which are approximately 50% homologous to the host genome upon entry into the cell. Poxvirus replication is independent of the host nucleus within the viroplasm. Manufactured viruses are then disseminated by either migrating to the cell surface and exiting via microvilli, transported by the Golgi apparatus, and released via vacuoles or independently, or attached with the proteinaceous inclusion body.[16] Upon the formation of virus copies, massive migration of inflammatory cells into the cell cytoplasm results in formation of vesicles and/or diphtheritic membranes on mucous membrane surfaces. Once the diphtheritic membrane is removed, a hemorrhagic surface remains, later forming a scar.[15]

CLINICAL SIGNS AND GROSS EXAMINATION FINDINGS

Infection of the skin (dry pox) is the most common clinical presentation, and it has a variable mortality dependent both upon the strain of poxvirus and the species of bird infected. Morbidity rate is highly variable (10%–95%). For example, the dry form of fowlpox infection in gallinaceous birds such as chickens *(Gallus gallus domesticus)* and turkeys *(Meleagris gallopavo)* is usually associated with a low mortality (1%–5% without secondary infections) and turkeys is usually associated with a low mortality (1%–5% without secondary infections); however, wet pox infection in the same birds can result in up to 50% to 60% mortality in unvaccinated flocks.[17] Infection tends to be more severe in psittacine birds, and in passerines such as canaries *(Serinus canaria)*, with canarypox infection approaching 100% mortality. Infection of mucous membranes of the mouth, esophagus, and trachea (wet pox) can have a high mortality due to asphyxiation. Canary pox infections, and co-infections with disease agents such

TABLE 15.1

Identified Bird Host Spectrum of Avian Poxvirus

Order	Family	English Name	Latin Name	Location
Apodiformes	Apodidae	Chimney swift	*Chaetura pelagica*	USA (Pennsylvania)
Accipitriformes	Accipitridae	Goshawk	*Accipiter gentilis*	Austria
		Common buzzard	*Buteo buteo*	France
				Germany
Anseriformes	Anatidae	Canada goose	*Branta canadensis*	Australia
		Black swan	*Cygnus atratus*	Canada
		Mallard	*Anas platyrhynchos*	China
Charadriiformes	Scolopacidae	Dunlin	*Pelidna alpina*	Great Britain
	Laridae	Common gull	*Larus canus*	Denmark
	Alcidae	Common murre	*Uria aalge*	USA (California)
Ciconiiformes	Ciconiidae	White stork	*Ciconia ciconia*	Switzerland
		Black stork	*Ciconia nigra*	
Columbiformes	Columbidae	Rock pigeon	*Columba* sp.	Germany
		Pigeon/feral pigeon	*Columba livia*	The Netherlands
Coraciiformes	Upuidae	Red-billed wood hoopoe	*Phoeniculus purpureus*	France
Falconiformes	Falconidae	Peregrine falcon	*Falco peregrinus*	Arabian Gulf
		Gyrfalcon	*Falco rusticolus*	Germany
		Common kestrel	*Falco tinnunculus*	United Arab Emirates
Galliformes	Meleagridae	Common turkey	*Meleagris gallopavo*	France
				Germany
				India
				Malaysia
				USA (Florida, Georgia, Illinois)
	Numididae	Guineafowl	*Numida* sp.	Germany
	Phasianidae	Common quail	*Coturnix coturnix*	China
				Italy
				Mexico
Gruiformes	Gruidae	Common crane	*Grus grus*	Germany
		Sandhill crane	*Grus canadensis*	USA (Florida)
	Otididae	Great bustard	*Otis tarda*	Germany
				Romania
	Rallidae	Eurasian coot	*Fulica atra*	India
Passeriformes	Carduelidae	European goldfinch	*Carduelis carduelis*	Germany
				Great Britain
	Corvidae	Common raven	*Corvus corax*	Denmark
		Blue jay	*Cyanocitta cristata*	USA (Pennsylvania)
	Estrildidae	Java sparrow	*Lonchura oryzivora*	Germany
	Fringillidae	Apapane	*Himatione sanguinea*	USA (Hawaii)
	Icteridae	Grackle	*Quiscalus* sp.	USA (Texas)
	Mimidae	Mockingbird	*Mimus polyglottus*	Spain
	Monarchidae	Magpie-lark	*Grallina cyanoleuca*	Australia[3]
	Muscicapidae	Red-flanked bluetail	*Tarsiger cyanurus*	China[4]
	Passeridae	American tree sparrow	*Spizelloides arborea*	Brazil
		House sparrow	*Passer domesticus*	Canada
				Germany
				Norway
				USA (Washington, California)
	Pipridae	Golden-headed manakin	*Ceratopipra erythrocephala*	Trinidad
	Turdidae	Common blackbird	*Turdus merula*	Italy
Pelecaniformes	Pelicanidae	White-tailed tropicbird	*Phaethon lepturus*	USA (Bermuda)
Piciformes	Picidae	Common flicker	*Colaptes auratus*	USA (Illinois)
				Canada
Podicipediformes	Podiipedidae	Great crested grebe	*Podiceps cristatus*	Switzerland
Procellariiformes	Procellariidae	Manx shearwater	*Procellaria puffinus*	Australia
		Pacific shearwater	*Ardenna* sp.	Great Britain[5]
Psittaciformes	Psittacidae	Rosy-faced lovebird	*Agapornis roseicollis*	Germany
		Blue and yellow macaw	*Ara ararauna*	Japan
		Green parakeet	*Psittacara holochlorus*	USA (Florida, Illinois, Indiana,
		Red-winged parrot	*Aprosmictus erythropterus*	Philadelphia)
		Budgerigar	*Melopsittacus undulatus*	

Continued

TABLE 15.1
Identified Bird Host Spectrum of Avian Poxvirus—cont'd

Order	Family	English Name	Latin Name	Location
Sphenisciformes	Spheniscidae	Humboldt penguin	*Spheniscus humboldti*	Brazil[6]
		Magellanic penguin[a]	*Spheniscus magellanicus*	Poland
Strigiformes	Strigidae	Barred owl	*Strix varia*	USA (Florida)
Struthioniformes	Rheidae	Greater rhea	*Rhea americana*	Israel
				South Africa
				Australia
	Struthionidae	Ostrich	*Struthio camelus*	Spain

[a]Denotes new species/strains isolated in the last decade. References listed with location where the new species/strain identification occurred. Adapted from Bolte AL, Meurer J, Kaleta EF: Avian host spectrum of avipoxviruses, *Avian Pathol* 28(5):415–432, 1999. doi: https://doi.org/10.1080/03079459994434.[7]



FIGURE 15.1 Canarypox virus infection in a canary *(Serinus canaria)*. In canaries, systemic poxvirus infection has been associated with mortality rates approaching 100%.



FIGURE 15.2 Dry poxvirus lesion on the leg of a chicken *(Gallus gallus domesticus)*. Cutaneous, wartlike lesions most commonly develop on the nonfeathered parts of the skin.



FIGURE 15.3 Wet pox lesions in the oral cavity of a chicken *(Gallus gallus domesticus)*.

as psittacine pox and Psittacid alphaherpesvirus 1 (PsHV-1; see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease), can result in systemic poxvirus infection[18] (Figure 15.1). Intravenously infected chickens have lesions in the epithelial cells of the kidney and thymus.[19] Lesions can persist for 4 weeks or more. In birds of prey, storks (Ciconiidae), cranes (Gruidae), and other wild birds, infection of the legs, feet, and surrounding the eye is often fatal due to both starvation and predation. Chronic infection in layers and turkeys can result in weight loss and a drop in production. The incubation period ranges between 4 (canaries with canarypox) and 10 (turkeys with turkeypox) days.[15]

The most common gross lesions with dry pox are pustules and papules on the epidermis and underlying feather follicles with white to yellow exudates and epithelial hyperplasia of the nonfeathered skin[20] (Figure 15.2). Initially the lesions consist of small white focal nodules that then begin to coalesce and become purulent. Papules start forming after day 5 to 6, which turn into vesicles. After 1 to 2 weeks, lesions generally become hemorrhagic eventually leading to erosions when the scabs fall off. In the wet form, yellow to opaque white diphtheritic lesions form in the oral mucosa, esophagus, and trachea and coalescence of lesions can block the airway (Figure 15.3). The lesion may extend into the sinus, pharynx, and larynx (leading to asphyxiation) and the esophagus (leading to dysphagia). Removal of lesions leads to bleeding erosions.[21] It is possible to find both the dry and wet forms in the same bird. The fowlpox



FIGURE 15.4 Poxvirus affecting the right eye in a blue-fronted Amazon parrot *(Amazona aestiva)*.

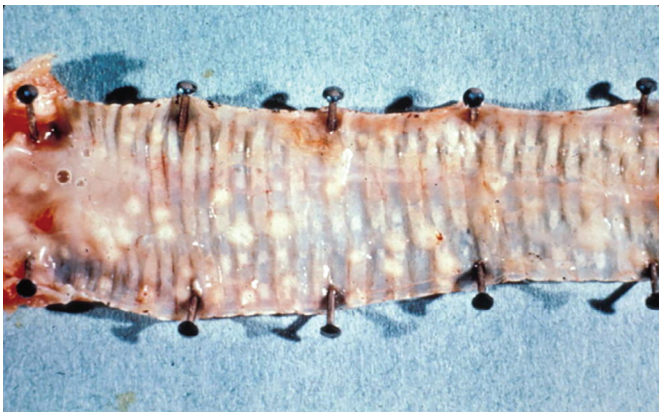


FIGURE 15.6 Wet pox pustules in the trachea of a chicken *(Gallus gallus domesticus)*.

genome carries a gene similar to epidermal growth factor, which may be responsible for cellular hyperplasia.[15] For canaries with canarypox[22] and blue-fronted Amazon parrots *(Amazona aestiva)* with psittacine pox, nodules on the eyelids can lead to loss of the eye and blindness (Figure 15.4). With systemic pox, firm white nodules can be observed in the viscera.[23]

DIAGNOSIS

Gross lesions alone are sometimes enough for diagnosis, although histopathologic examination is beneficial in making a definitive diagnosis. Severe hyperplasia of the epithelium with ballooning degeneration is the classic microscopic lesion in all forms of avian pox. With turkeypox, inclusion bodies may be uncommon and not readily observed. Inclusion bodies can become so large that they cause cellular degeneration. Viral inclusions recognized on histopathology are large eosinophilic intracytoplasmic inclusion bodies (Bollinger bodies) containing small viral particles (Borrell bodies; Figure 15.5). The inclusion bodies can be observed 72 to 96 hours after infection of the epithelial cells, and exit of the virus particles from cells is through budding.[24,25] Lesions in the tracheal and laryngeal mucosa are characterized by proliferative hypertrophy and ballooning degeneration of epithelial cells containing intracytoplasmic inclusion bodies. The lesions resemble papillomas and can range from partial to full obstruction of the tracheal lumen (Figure 15.6). There is hyperplasia and hypertrophy of goblet cells.[26]

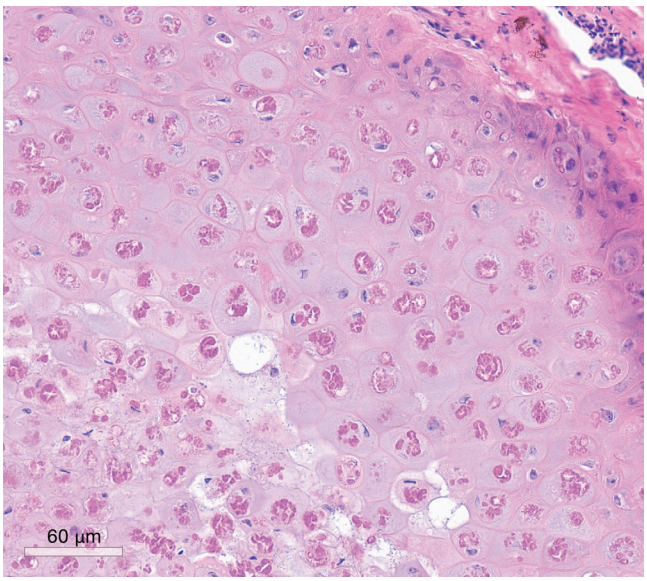


FIGURE 15.5 Large eosinohili intracytoplasmic pox inclusion bodies in the epithelium of a chicken.

Field strains of fowlpox virus often contain nearly intact provirus copies of reticuloendotheliosis virus (REV).[27] This can be used to help distinguish between vaccine strains and field strains. Traditionally, isolation of poxviruses has been in 10- to 12-day-old embryonated chicken eggs via chorioallantoic membrane (CAM) injection. Visualization of "pocks" on the CAM 7 days later is indicative of pox infection.[15] CAM injection does not identify strains of poxviruses. Cell cultures of avian origin can also be used, but most mammalian cell cultures are refractive to infection. Fowlpox is a very stable virus and can last in dried scabs and skin cells for years. Fowlpox viruses can also be propagated in cell cultures of avian origin including chicken embryo fibroblasts and kidney cells. A permanent cell line, QT35 of Japanese quail *(Coturnix japonica)* origin, and a chicken liver cell line LMH, can also support the growth of some avian poxviruses after adaptation.[15]

For practical purposes, diagnosis is usually based on clinical presentation, with histopathology and observation of inclusion bodies being confirmatory.[28] Cytology of lesions in wild turkeys has been reported to have a 90% specificity and sensitivity when compared to polymerase chain reaction (PCR).[29] Although cell-mediated immunity is the most important, antibodies are elicited and can be measured by ELISA (enzyme-linked immunosorbent assay) and used to monitor vaccine immunity.[30] Neither of these will identify the strain of the virus. Historically, strain identification required cross-protection assays, which are labor intensive and cost prohibitive. Immunoblotting can differentiate some strains but not all due to the number of shared antigens.[31] The most economical choice is strain identification by RFLP and restriction endonuclease analysis. On RFLP, different strains of pox can be like the standard fowlpox virus. However, strains can be further separated with restriction endonuclease analysis. Fowlpox virus strains are highly related but distinct from canary, mynah, quail, and Hawaiian poxvirus strains. It has been suggested

that the number of ankyrin repeat genes is associated with the host range.[15] Mixed infections of fowlpox and infectious laryngotracheitis virus are common in commercial layers and can be distinguished by PCR. P4b is a highly conserved protein and has been used to distinguish strains by PCR.[32] PCR is mainly used to distinguish between vaccine and field strains by looking for REV provirus in field strains versus LTRs (long terminal repeats) in vaccine strains.[28]

Differentials for fowlpox infection depend largely on the type of manifestation. For cutaneous pox, gross lesions may include rule-outs such as skin scabs from trauma, avian ringworm, ectoparasites such as lice and mites, bacterial dermatitis, and papillomavirus infections. Observations of skin scabs and scrapings through microscopy or histopathology can be utilized for diagnosis. If infections are at a flock level, cutaneous lesions may also appear similar to pantothenic acid deficiency, biotin deficiency, and fungal infections such as T-2 (trichothecene) toxicity. Feed analysis may be warranted if there is a wide-spread problem within a flock, especially in young chicks. Differentials for wet pox include trichomoniasis (canker/frounce; see Chapter 22), oral candidiasis and other fungal infections such as aspergillosis (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease), hypovitaminosis A, capillariasis, and most commonly in chickens, infectious laryngotracheitis (ILT) virus infection.[33] Especially in commercial chickens, histopathologic diagnosis of fowlpox and ILT is a simple and quick procedure, and these diseases must be distinguished in order to employ proper management strategies, including vaccination.

TREATMENT

There is no treatment for pox infections other than supportive care. Isolation of birds to prevent pecking/cannibalism is recommended. Supportive care in pet birds may include fluids, oxygen supplementation, parenteral vitamin A, ointments for eye infections, assisted feedings, and antibiotics to treat secondary infections.[35]

Skin lesions can be kept clean daily with mild detergents, diluted iodine, or soaps being careful to avoid the eyes. In the wet form, oral iodine in the water can be used at a flock level to dehydrate the proliferative epithelial lesions in order to avoid asphyxiation or anorexia, depending on location. For individual or small numbers of anorexic birds, such as backyard chickens, a two times dilution of powdered dry milk will help with hydration and protein.

Birds should not be returned to the flock until completely healed, which may take up to several weeks. Severely infected birds with poor quality of life should be removed and humanely euthanized.

PREVENTION AND CONTROL

Vaccination is the primary method of control in those species where the vaccine is well tolerated. There is limited attenuation of the two primary pox vaccines, Fowlpox and Pigeonpox. Vaccination in the wing web or the drumstick (turkeys) places the virus in areas that are usually not infected, thus reducing any pathogenic effects of the vaccine. However, some exotic species can have significant reactions to the vaccine viruses and caution is recommended. Both Fowlpox and Pigeonpox vaccines are usually freeze-dried, and often used in combination to achieve more cross-protection. Combining vaccines can lead to over dilution of each vaccine; therefore, each vial needs to be reconstituted using only half of the diluent before mixing the two together. Care needs to be taken to avoid contamination of parts of the bird other than the site of vaccination. Vaccination of laying hens is not recommended. In endemic areas, vaccination may need to be done yearly. Fowlpox vaccine cannot be used for pigeons *(Columba livia)* and Pigeonpox vaccine can cause severe reactions if not used properly. All canaries raised in large numbers should be vaccinated with Canarypox vaccine every 6 to 12 months and prior to lay. Quailpox and turkeypox vaccines were historically available commercially. Autogenous vaccines may be needed for strains other than fowlpox, pigeon pox, and canarypox viruses. Several recombinant vaccines using fowlpox as a vector are available for commercial poultry. However, these are not recommended for small flocks or exotic birds (see also Chapter 13).

Currently available commercial vaccines do not 100% protect against poxvirus infections particularly if the field strain is not contained in the vaccine. Flocks with high density and multiple ages are at greatest risk of ongoing infections despite vaccination.

In commercial poultry, vaccination in the face of an outbreak is utilized to reduce mortality in cases where the flock is naïve to infection or vaccine failure is suspected. Due to the viral immunity being predominantly cell mediated, the infection spreads slower in large flocks, and thus timely vaccination may help protect a susceptible population. In wet pox infections, low-dose oral iodine may be used in waterlines to dehydrate diphtheritic lesions to increase the chance of survival and to prevent secondary bacterial infections.[34]

Pest control for mosquitos and other insect vectors is crucial. An integrated pest control program and good biosecurity practices to reduce other mechanical vectors (e.g., crews/personnel used for vaccination, transfers, etc.) from spreading the virus are recommended (see Chapter 11). In addition, controlling cannibalism with proper beak trimming and reducing environmental stimuli (e.g., high intensity lights, high stocking density, poor sanitation) along with effective cleaning and disinfection using common household disinfectants (e.g., 10% bleach and water) is warranted to prevent future infections (see Chapter 12).

SUMMARY

Avian pox is a disease caused by many divergent strains. The predominant groups, or clades, are fowlpox, canarypox, and poxviruses from Psittaciformes. All the avian poxviruses cause similar lesions in cutaneous and mucosal tissues. Lesions confined to the skin are the "dry" form and lesions in the mucosa and/or internal organs are the "wet" form. Mosquitos and other insects are the primary mechanical vectors from flock to flock and pecking within a flock. There is no treatment, and vaccination is recommended for poultry in endemic areas and canaries and pigeons in large flocks.

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CHAPTER 16

West Nile Virus and Other Flaviviruses

Dominik Fischer • *Ute Ziegler*

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The family Flaviviridae consists of four genera—*Flavivirus*, *Pestivirus*, *Hepacivirus*, and *Pegivirus*—with more than 70 small, enveloped viruses, composed of spherical virions with a diameter of 40 to 60 nm and positive-sense RNA genomes of approximately 9.0 to 13 kb.[1] While the genera *Pestivirus*, *Hepacivirus*, and *Pegivirus* are usually restricted to mammalian hosts, several members of the genus *Flavivirus* may act as pathogens in birds and other vertebrates.[1]

GENUS *FLAVIVIRUS*

The genus *Flavivirus* includes more than 50 arthropod-borne viruses (arboviruses), with distinct groups infecting mosquitoes or ticks.[1,2] These viruses are amplified and transmitted via their arthropod vectors following consumption of a vertebrate host's blood and are usually maintained in an enzootic cycle between vertebrates and arthropods (dual-host viruses; Figure 16.1). Viruses are transmitted horizontally by mosquito or tick bites to vertebrates. Viral transmission takes place when an infected arthropod takes a blood meal from a naïve animal host and injects the virus during this process.[2] Inside the vertebrate host, the virus starts its amplification process, replicating itself at large quantities within the bloodstream (viremia) to enable transmission to further hosts via the biologic arthropod vector. The transmission of flaviviruses is generally influenced by environmental factors, as these influence the population dynamics of the biological vectors, the extrinsic incubation period (time it takes for the virus to develop inside the vector from the point of ingestion via the blood meal to the point when the arthropod becomes infectious), and the population densities of the amplifying hosts. In this regard, warmer weather conditions are usually advantageous for host and vectors as seen in Europe in the unusually hot summer of 2018.[3] The extremely long period of high temperatures over months may have provided conditions for the incursion and establishment of flavivirus infections, such as West Nile virus (WNV) and the Usutu virus (USUV), into new areas and countries. The availability of artificial light during nighttime has been demonstrated to additionally extend the infectious-to-vector period of avian reservoir hosts of WNV in urban regions and contributes to prolonged maintenance of transmissible viral titers.[4,5]

Depending on the virus, some incidental host species are "dead-end hosts" as the viral load does not reach sufficient levels to allow the virus to be transmitted back to the arthropod; however, many avian species are regarded as competent amplifying hosts and various flaviviruses are important epornitic (capable of causing epizootics in birds) pathogens in free-ranging and companion birds as well as in domestic poultry (Table 16.1).[2,3] Around 40 flavivirus species are known

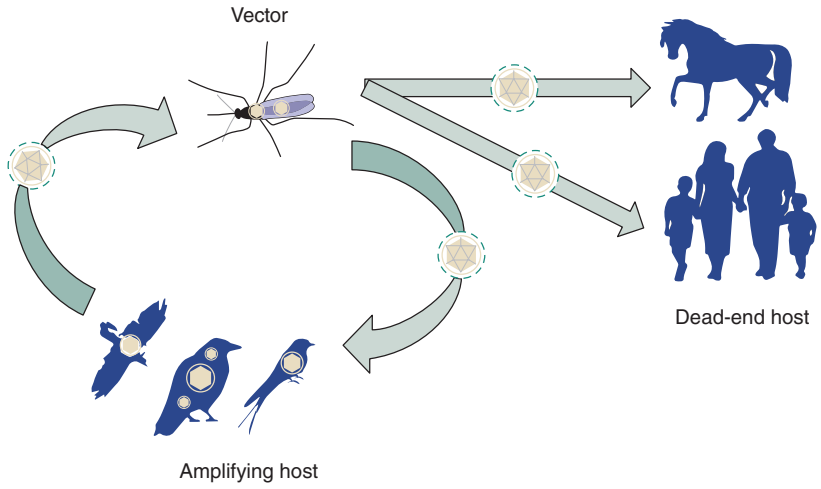


FIGURE 16.1 Transmission cycle of flaviviruses using mosquito-borne West Nile virus as an example.

TABLE 16.1

Flaviviruses Isolated From Avian Species

Virus Type	Virus Name	Abbreviation	Avian Hosts
mbfv	Alfuy virus	ALFV	Cuculiformes (Pheasant coucal *[Centropus phasianinus]*)
mbfv, nvc	Bagaza virus (together with ITV summarized as avian meningoencephalomyelitis virus [AMEV])	BAGV	Phasianidae (partridges, pheasants)
mbfv, nvc	Baiyangdian virus (subtype of TMUV)	BYDV	Anseriformes
mbfv, jevc	Cacipacore virus	CPCV	
mbfv, nvc	Duck egg drop syndrome virus (subtype of TMUV)	DEDSV	Anseriformes
mbfv, nvc	Ilheus virus	ILHV	
mbfv, nvc	Israel turkey meningoencephalitis virus (together with BAGV summarized as AMEV)	ITV	Turkeys *(Meleagris gallopavo)*
mbfv, jevc	Japan encephalitis virus	JEV	Ardeidae (herons, egrets)
tbfv	Kadam virus	KADV	Sea birds: Procellariiformes (albatrosses, petrels), Suliformes (gannets and cormorants), Charadriiformes (gulls, skuas, terns, auks and skimmers), Sphenisciformes (penguins)
mbfv, jevc	Koutango virus	KOUV	
mbfv, jevc	Kunjin virus	KUNV	Passeriformes (Green oriole *[Oriolus flavocinctus]*)
tbfv	Louping ill virus	LIV	Tetraonini (grouse)
mbfv, nvc	Malaysian duck Tembusu virus, Perak virus	DTMUV	Peking ducks *(vAnas platyrhynchos domesticus)*
tbfv	Meaban virus	MEAV	Sea birds: Procellariiformes (albatrosses, petrels), Suliformes (gannets and cormorants), Charadriiformes (gulls, skuas, terns, auks and skimmers), Sphenisciformes (penguins)
mbfv, jevc	Murray Valley encephalitis virus	MVEV	Different species, including Anseriformes, chicken *(Gallus gallus domesticus)*
mbfv, nvc	Ntaya virus	NTAV	Chickens *(Gallus gallus domesticus)*
mbfv, nvc	Rocio virus (subtype of ILHV)	ROCV	Wild birds, Passeriformes, chickens *(Gallus gallus domesticus)*
mbfv, jevc	Saint Louis encephalitis virus	SLEV	Different species
tbfv	Saumarez Reef virus	SREV	Sea birds: Procellariiformes (albatrosses, petrels), Suliformes (gannets and cormorants), Charadriiformes (gulls, skuas, terns, auks and skimmers), Sphenisciformes (penguins)
mbfv, nvc	Sitiawan virus (subtype of TMUV)	SV/STWV	Chickens *(Gallus gallus domesticus)*
mbfv, nvc	Tembusu virus	TMUV	Anseriformes
tbfv	Tick-borne encephalitis virus	TBEV	
tbfv	Tyuleniy virus	TYUV	Anseriformes
mbfv, jevc	Usutu virus	USUV	Different species
mbfv, jevc	West Nile virus	WNV	Different species
mbfv, jevc	Yaoundé virus	YAOV	

jevc, Japan encephalitis complex; *mbfv*, mosquito-borne flavivirus; *nvc*, Ntaya complex; *tbfv*, tick-borne flavivirus.

to be able to infect humans (zoonotic potential) and nonavian vertebrates. Recent prognoses predict an increase and spread of zoonotic flaviviruses into wildlife avian hosts parallel to an increase of potential biological vectors associated to climate change and increase in global temperatures.[6] This already corresponds to the increase and the expansion of geographic distribution of emerging flaviviruses in the last two decades, as seen for Tembusu virus (TMUV) in China, USUV and Bagaza virus (BAGV) in Europe, and WNV in the Americas and Eurasia.[2,4] New flaviviruses are discovered regularly, and accordingly the taxonomy of this genus changes frequently.[1] Hence, the impact and the pathological role of many "new" flaviviruses in avian species are currently unknown. Likewise, the role of birds as vectors and as wildlife reservoirs of these recently discovered flaviviruses is not elucidated, and thus the relevance of birds during the spread of various zoonotic flaviviruses is poorly understood. In the absence of specific studies, there is a need to extrapolate and transfer knowledge from well-known and extensively studied flaviviruses such as WNV and USUV to other flaviviruses, even though mistakes are possible following this strategy.

WEST NILE VIRUS AND USUTU VIRUS

WNV is regarded as the most widespread flavivirus with reports from all continents except Antarctica, and with increasing numbers of bird infections in European countries (particularly in South Europe), in the Americas, and in Australasia. Besides WNV, USUV is regarded as most important avian pathogen in the genus *Flavivirus* in Europe.[7–9]

WNV and USUV belong to the Japanese encephalitis virus (JEV) serogroup and share cross-neutralization antibodies with other encephalitis viruses of this group. Both viruses are named after the African place of origin—WNV after the former West Nile District, today's Uganda, and USUV after the Usutu River in Swaziland/Kingdom of Eswatini.[10] These viruses have been spread over long distances to other countries and continents via migration of infected birds and accidental transport of infected arthropod vectors.[11] Additionally, overwintering mechanisms such as hibernation of infected mosquitoes or of chronically infected amphibians have been demonstrated as important factors for the establishment of WNV infections in temperate zones besides a transovarial infection of mosquitoes.[12]

WNV was first isolated in 1937 from the blood of a woman suffering from a febrile illness in Uganda.[12] In avian species, it was isolated in 1953 from the blood of two rock pigeons *(Columba livia)* and one hooded crow *(Corvus cornix)* in Egypt. In Europe, neutralizing antibodies against WNV were first detected in humans from Albania in 1959; however, no viral RNA was detected in these patients.[13] The first WNV outbreak in Europe associated with clinical symptoms and death in humans and horses occurred in the Camargue region in Southern France in 1962/1963.[13] Since the 1990s, epidemics have occurred in several European countries, such as Romania, Russia, France, Italy, and Hungary.[14] Between 1997 and 2000, WNV has been associated with two outbreaks in white storks *(Ciconia ciconia)* and domestic geese *(Anser anser domesticus)* in Israel. In 1999, a closely related WNV isolate was introduced into the United States, with the first WNV outbreak in the city of New York and surrounding areas.[12] Over the next years, WNV spread over the whole American continent, causing neurological disease among humans and horses and epornitics with high mortality (see https://www.cdc.gov/westnile/index.html). Likewise, WNV has spread further throughout Europe during the last two decades, starting from the Mediterranean countries northward.[8,13] Especially in the hot summer of 2018, a large number of WNV cases were observed by the European Centre for Disease Prevention and Control (ECDC) in southeast, south, and central Europe, with a total of 2,083 autochthonous human WNV cases in the European Union (EU) member states and EU neighboring countries (see https://www.ecdc.europa.eu/en/west-nile-virus-infection). This represented a 7.2-fold increase compared to 2017 and was linked to an unusually early start of the WNV transmission season. In 2019, a larger epizootic outbreak occurred in Germany with 76 confirmed cases in free-ranging and zoo birds, 36 confirmed cases in horses as well as 5 clinical cases in humans.[15] There are currently nine lineages known for WNV worldwide, with lineages 1 (clade 1a, clade 1b or Kunjin virus, and clade 1c) being reported in Europe, the United States, Africa, and the Middle East. Lineages 2, 3, and 8 are also reported from Europe.[7] Several mosquito species, mainly ornithophilic species of the *Culex* genus, are involved in WNV transmission. *Culex pipiens* is considered to be the main vector in Europe. Moreover, other genera such as *Aedes*, *Anopheles*, *Culiseta*, *Mansonia*, and *Ochlerotatus* may be involved as biological vectors,[16] and recently, WNV replication has been demonstrated even in tick species. The number of avian species with confirmed WNV infection exceeds 300 and includes Accipitridae (eagles, hawks, Old World vultures), Cathartidae (Cathartid vultures and condors), Falconiformes (falcons), Strigiformes (owls), Psittaciformes, Passeriformes, Corvidae (corvids), Anserinae (geese), Phoenicopteridae (flamingos), Rheidae (rheas), Ciconiidae (storks), and many other birds as summarized previously.[10,17,18] In North America, corvids, great horned owls *(Bubo virginianus)*, American kestrels *(Falco sparverius)*, red-tailed hawks *(Buteo jamaicensis)*, Cooper's hawks *(Accipiter cooperi)*, bald eagles *(Haliaeetus leucocephalus)*, American white pelicans *(Pelecanus erythrorhynchos)*, and eared grebes *(Podiceps nigricollis)* have been affected in large numbers, with the latter two in course of large mortality events.[18] In Europe, most clinical cases have been reported in the Eurasian goshawk *(Accipiter gentilis)*, the sparrow hawk *(Accipiter nisus)*, great gray owls *(Strix nebulosa)*, and snowy owls *(Bubo scandiaca)*, even though various WNV-positive songbirds and different zoo birds were found dead regularly as well.[3,10,15]

USUV was first detected in a *Culex naevei* mosquito (originally described as *C. univittatus*) near the Usutu River, Natal, South Africa, in 1959 (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).[16] A retrospective study in the Tuscany region (Italy) demonstrated the first evidence in Europe dating back to 1996. From Italy, USUV spread to several other European countries.[9,16] The most important outbreak of USUV occurred in 2001 in and around Vienna, Austria, with significant wild bird mortality, primarily in common blackbirds *(Turdus merula)*,[9] but also in different captive birds such as great gray owls. Thereafter, USUV has been demonstrated to circulate in different European countries, including Austria, Italy, Hungary, Switzerland, and Spain (only mosquitoes), leading to the first human infections outside Africa in two immunocompromised patients in Italy in 2009.[16] USUV introduction in Germany in 2011 caused a massive reduction of the local blackbird populations. Today, USUV isolates can be phylogenetically differentiated into eight different lineages: Africa 1 to 3 and Europe 1 to 5, with introduction events from Africa into Europe still ongoing.[4,7]

More than 93 bird species have been listed as susceptible to USUV.[4,10,16] This list contains the following avian orders: Passeriformes, Strigiformes, Accipitriformes, Anseriformes, Caprimulgiformes, Ciconiiformes, Charadriiformes, Columbiformes, Coraciiformes, Falconiformes, Galliformes, Pelecaniformes, Phoenicopteriformes, Piciformes, Psittaciformes, and Rheiformes.[10,16] In the Strigiformes order, highly susceptible species include great gray owl, snowy owl, and northern hawk-owl *(Surnia ulula)*. In the passerine order, highly susceptible species include Eurasian blackbird, song thrush *(Turdus philomelos)*, barn swallow *(Hirundo rustica)*, fieldfare *(Turdus pilaris)*, common starling *(Sturnus vulgaris)*, house sparrow *(Passer domesticus)*, Eurasian bullfinch *(Pyrrhula pyrrhula)*, lesser whitethroat *(Sylvia curruca)*, European pied flycatcher *(Ficedula hypoleuca)*, gray-headed bullfinch *(Pyrrhula erythaca)*, great tit *(Parus major)*, blue tit *(Cyanistes caeruleus)*, domestic canary bird *(Serinus canaria forma domestica)*, golden-breasted starling *(Cosmopsarus regius)*, magpie *(Pica pica)*, carrion crow *(Corvus corone)*, and western jackdaw *(Coloeus monedula)*.

OTHER FLAVIVIRUSES ISOLATED FROM BIRDS

Sea birds may be subclinical hosts of the tick-borne flaviviruses Meaban virus (MEAV), Saumarez Reef virus (SREV), Kadam virus (KADV), and Tyuleniy virus (TYUV) and may contribute to the distribution of infected ticks.

The TMUV, the duck egg drop syndrome virus (DEDSV), and the Baiyangdian virus (BYDV) have been associated with egg drop syndrome, retarded growth, inappetence, and mortality in waterfowl in Asia.[2]

Neurological signs and pathological lesions have been associated with the Israel turkey meningoencephalitis virus (ITV) in turkeys, the BAGV in partridges and pheasants, the Sitiawan virus (SV/STWV) in chickens *(Gallus gallus domesticus)*, the Malaysian duck Tembusu virus (DTMUV, Perak virus) in Peking ducks *(Anas platyrhynchos domesticus)*, the louping ill virus (LIV) in red grouses *(Lagopus lagopus)* as well as with the Ntaya virus (NTAV) in avian species of various bird orders.[2,4] NTAV has been reported to cause additional clinical signs in birds including splenomegaly and hemorrhages in the central nervous system (CNS), liver, lung, and ovaries.

Other flaviviruses have been isolated from bird species with or without clinical manifestation. This applies to the Saint Louis encephalitis virus (SLEV), the Kunjin virus (KUNV), the Yaoundé virus (YAOV), the Koutango virus (KOUV), the Murray Valley encephalitis virus (MVEV), the Alfuy virus (ALFV), the JEV, the Cacipacore virus (CPCV), the Ilheus virus (ILHV), the Rocio virus (ROCV), and the tick-borne encephalitis virus (TBEV).[2,4]

CLINICAL SIGNS IN BIRDS

Clinical signs associated with flaviviruses vary from subclinical infections and mild viral signs to severe neurological and hemorrhagic diseases depending on the virulence of the virus strain and the susceptibility of the host species.[10,17,18] Many birds are subclinically infected by flaviviruses, and therefore the *intra vitam* detection of viral diseases may be challenging. However, severe and fatal clinical diseases are reported in susceptible avian species varying from acute to chronic courses.[19] Especially in immunocompromised and highly susceptible individuals, death may occur without previous clinical signs in the course of flavivirus infection. Acute courses and sudden death are frequently observed in naïve bird populations, as seen during the WNV outbreak in 1999 in New York City.[19,20] There 17 of 27 birds were observed clinically with neurological signs several hours to maximum 6 days prior to death.[19]

Likewise, USUV has been reported with high mortality, especially in blackbirds and other members of the genus *Turdus* (Figure 16.2).[9]

Unspecific signs of flavivirus infection include general signs of illness such as depression, apathy, weakness, reduced stamina, drooping wings, inability to fly, dysphagia, regurgitation, anorexia, weight loss, disorientation, bad posture, and recumbency.[9,18,19,21,22] Especially in USUV-infected wild birds, nutritional status has been reported to be poor, whereas the nutritional status of captive birds was more variable.[9] In large falcons, the shedding of greenish urine/urates has been reported following artificial infection with WNV and associated damage to inner organs.[10,23] In commercial poultry, infections may cause a drop in performance such as reduced egg numbers and quality (egg drop), decreased spermatogenesis and spermatocyte swelling, retarded growth, and weight loss, as demonstrated in ducks and geese associated with TMUV, DEDSV, and BYDV infections and in chickens associated with SV/STWV.[2,4] Additionally, diarrhea has been observed in partridges and pheasants following BAGV infection.[2]

Feather disorders including ruffled feathers, pinching of blood feathers, discoloration, abnormal development or loss of feathers have also been reported as clinical signs of flavivirus infections, especially in birds that survived the acute stage of the WNV- or USUV-associated disease (Figure 16.3; see also Chapter 32).[9,10] This corresponds to a persistence of WNV in feather pulps as demonstrated in gyr-saker *(Falco rusticolus x Falco cherrug)* hybrid falcons.[4]

Most common signs of flavivirus infection are neurological signs such as head tremors, circling, stumbling, staggered movements, ataxia, torticollis, and convulsions (Figure 16.4).[9,10,24] They have been reported in association with ITV in turkeys *(Meleagris gallopavo)*, BAGV in partridges and pheasants, SV/STWV in chickens, DTMUV in Peking ducks, LIV in grouses, as well as with NTAV, USUV, and WNV in various avian species.[4,9] Flamingos and gulls (*Larus* spp.) have been reported with difficulties to hold their heads and wobbly gait, and cormorants swimming in circles in the course of a WNV infection.[19] Leg stretching and impaired mobility were caused in 4- to 6-week-old chickens due to SV/STWV infections.



FIGURE 16.2 Usutu virus is known to cause mortality among songbirds and owls. Mortality is particularly high in common blackbirds *(Turdus merula)*, house sparrows *(Passer domesticus)*, and great gray owls *(Strix nebulosa)*. Prior to death, birds may show general signs of illness, including depression, apathy, weakness, anorexia, drooping of the wings, disorientation (A), and inability to fly (B). (Courtesy Sylvia Urbaniak, Germany)

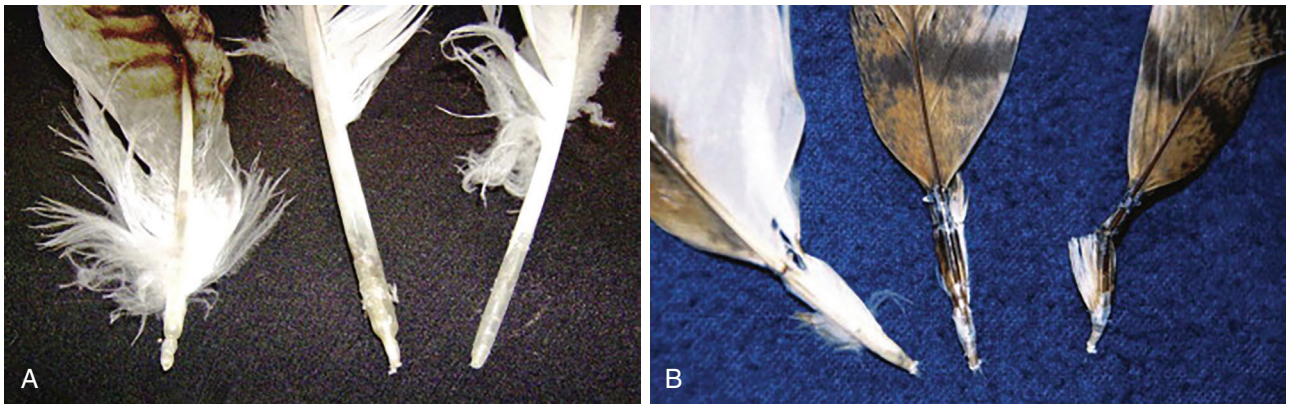


FIGURE 16.3 Feathers with abnormal morphology that molted prematurely from West Nile virus–infected raptors. (A) Red-tailed hawk *(Buteo jamaicensis)*; (B) Barn owl *(Tyto alba)*. (From Miller R, Fowler M, editors: *Fowler's Zoo and Wild Animal Medicine*, Vol 7, St. Louis, 2011, Elsevier.)



FIGURE 16.4 European goshawks *(Accipiter gentilis)* with central neurological signs caused by natural West Nile virus infection. (*Left:* courtesy Georgios Vergatos, Germany; *Right:* courtesy Thomas Schön, Wildvogelhilfe Saalekreis, Germany.)

Additionally, WNV has been associated with anisocoria, nystagmus, difficulties to accommodate, and impaired vision or blindness due to ocular lesions.[9,10,18,19,23,24] These lesions varied from anterior uveitis and fundic disorders such as fibrin adhesions to chorioretinal lesions and retinal atrophy. Vision disorders have also been diagnosed in pheasants and partridges infected with BAGV.[4] Half-closed eyes and nystagmus have been associated with WNV in blackbirds.[9]

CLINICAL SIGNS IN HUMANS AND OTHER VERTEBRATES

Mammals, amphibians (e.g., *Rana ridibunda*), and reptiles (e.g., *Varana salvidorii* and *Alligator mississippiensis*) have been infected by WNV.[25] Infected mammals include more than 30 species of livestock and companion animals such as horses, hamsters, cats, dogs, camelids, and sheep, as well as zoo and wildlife animals such as rodents, rabbits, polar bears, marine mammals, wolves, and skunks.[10,25] These nonavian hosts are usually considered to be dead-end hosts of WNV due to the lower level of viremia. Clinical signs are reported in horses and humans, even though the majority of WNV infections are asymptomatic. In horses, fever is rarely seen, and neurologic signs may develop in 8% of the infected individuals. Neurologic signs may persist throughout life, and up to 44% of the horses with central nervous signs may die in the course of a WNV infection.[10]

USUV-specific antibodies have been detected in the serum of humans and many mammalian species including horses, dogs, wild boar, wild ruminants, bats, squirrels, and rodents, which most likely act as dead-end hosts for USUV with less severe

pathogenicity.[26] Moreover, viable USUV has been isolated from the brain of bats (*Pipistrellus* sp.), as well as from rodent and shrew species. The expanding range of wild animals susceptible to USUV highlights its potential as an important emerging pathogen.

Around one-fifth of the infected humans may develop flu-like symptoms (e.g., fever, malaise, headache, rash, myalgia, sore throat, chills, gastrointestinal, and ocular disorders such as conjunctival congestion, ocular pain due to uveitis, chorioretinitis, vitritis, or optic neuritis) in association to an infection with WNV and several other zoonotic flaviviruses.[10,17] A severe and potentially fatal neuroinvasive disease may occur in less than 1% of WNV-infected people, but in larger numbers after JEV infection. In comparison, USUV was considered to have a low zoonotic potential because the number of human infections is comparably low. In Europe, the first USUV disease with clinical symptoms similar to WNV was detected in 2009 in Italy in two immunocompromised elderly patients.[27] Recently, neuroinvasive disease of USUV was also reported in Croatia (6 cases) and France (1 case), summarizing to date a total number of 25 human cases of USUV infection.[27]

GROSS PATHOLOGICAL FINDINGS

Gross pathological findings of flavivirus infections are limited. Depending on the course of the disease, the body condition may vary from good to thin or emaciated.[28] Swelling, mottling, and necrosis of spleen and liver are most common findings due to infections with WNV, USUV, NTAV, BYDV, and BAGV (Figure 16.5).[2,4] Additionally, USUV may cause seromucous (catarrhal) enteritis and BAGV ovary atrophy and oophoritis with hemorrhages and necrosis.[4,10]

Myocardial hemorrhages, myocarditis (with multiple 2–3 mm sized pale foci), and lesions such as degeneration, mineralization, necrosis, and fibrosis in several organs may be associated to WNV infection (Figure 16.6).[19,21,24,29] In this regard, tubular epithelial and glomerular cell degeneration and necrosis may develop besides congestion, swellings, and pinpoint white foci in the kidneys.[19] White streaks may be seen in the ventricular walls and interventricular septum, producing a mottled to striped appearance. Bleedings may occur in the mucosa or lumen of the jejunum and the duodenum, possibly transforming to necrotic or diphtheric lesions due to secondary bacterial infection.[20] Minor bleedings may be seen in mucosa of the upper gastrointestinal tract. Intraosseous calvarial hemorrhages

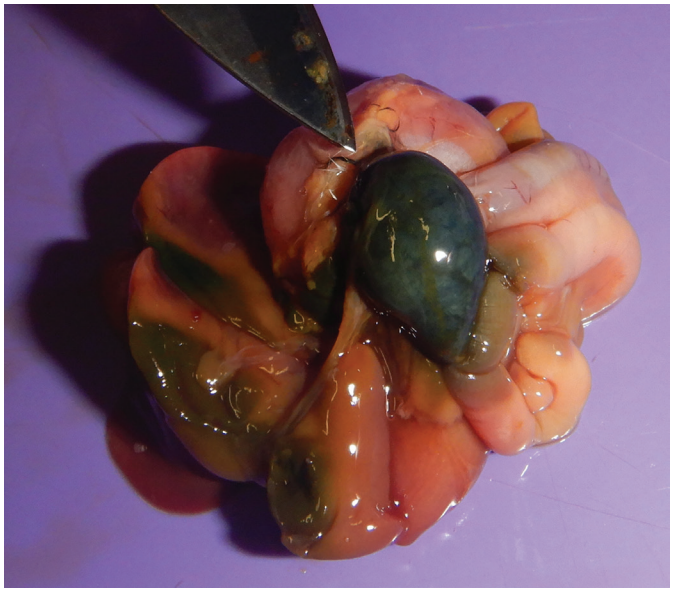


FIGURE 16.5 The most common postmortem finding in songbirds infected with Usutu virus infection and disease is an enlarged spleen, as can be seen in this common hill mynah *(Gracula religiosa)*. Other findings may include a seromucoid (catarrhal) enteritis and swelling, mottling, and necrosis of the liver. (Courtesy Dr. Helena Vaidlová, Veterinary Clinic Mada, Kralupy nad Vltavou, Czech Republic.)

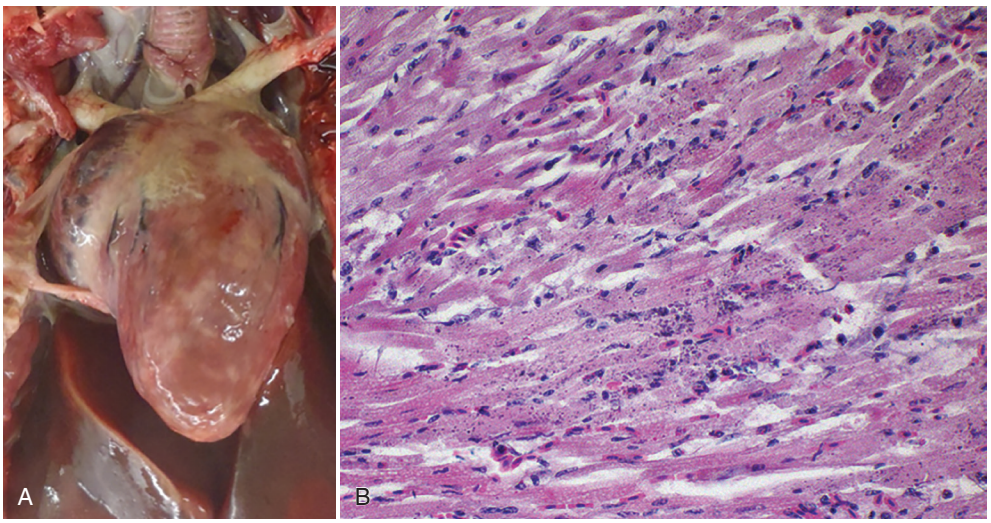


FIGURE 16.6 Macroscopic (A) and histologic (B; Hematoxylin and eosin stain, 400×.) pictures of a severe, acute, necrotizing myocarditis and myocardial necrosis in a gyr-saker falcon *(Falco rusticolus × Falco cherrug)* infected with West Nile virus lineage 1 strain NY'99. (Courtesy Dr. Christine Fast, Friedrich-Loeffler Insitut (FLI), Federal Research Institute for Animal Health, Institute of Novel and Emerging Infectious Diseases, Greifswald, Germany.)

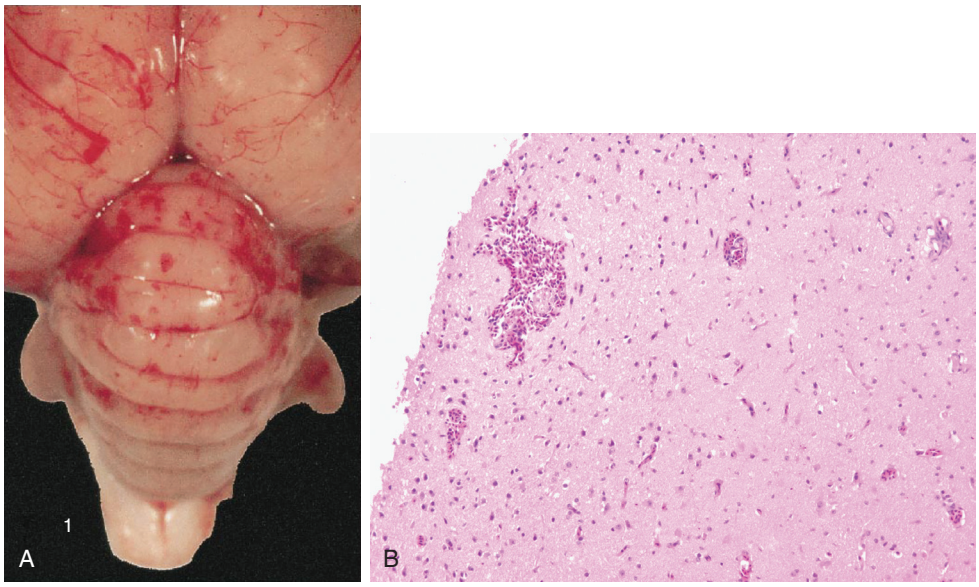


FIGURE 16.7 (A) Brain of a Chilean flamingo *(Phoenicopterus chilensis)* with multifocal hemorrhage in the cerebellum and both cerebral hemispheres due to West Nile virus (WNV) infection. (B) Cerebrum of a juvenile, female scarlet-breasted lorikeet *(Trichoglossus forsteni)* with military necrosis, multifocal hemorrhage, hyperemia, and intravascular fibrin thrombi. Perivascular, mononuclear cuffing is spurious to absent. PCR was positive for WNV-2. Hematoxylin and eosin stain, 100×. (A, From Steele KE, Linn MJ, Schoepp RJ, et al: Pathology of fatal West Nile virus infections in native and exotic birds during the 1999 outbreak in New York City, New York, *Vet Pathol* 37(3):208–224, 2000. doi: https://doi.org/10.1354/vp.37-3-208. B, Courtesy Dr. Helene Pendl, Pendl Lab, Zug, Switzerland.)

(especially in the supraoccipital region of the skull) and acute meningeal hemorrhages have been reported in WNV-infected corvids, flamingos, and other birds (Figure 16.7).[19]

In bald eagles, severe cerebral pan-necrosis with hydrocephalus *ex vacuo* is described.[24] Other findings of WNV infections may include subdural hematomas and cerebral congestion as reported in mallards *(Anas platyrhynchos)*. Similar hemorrhages in the CNS, liver, lung, and ovaries have been associated to NTAV infections in several bird species.[4]

HISTOPATHOLOGICAL FINDINGS IN BIRDS

Various degrees of inflammatory lymphoid and histiocytic infiltrates and multifocal acute hemorrhage and necrosis in the spleen, liver, kidney, brain, eyes, and heart have been described besides intestinal lesions in lieu of USUV, WNV, and BAGV infections.[4,19]

Myocardial lesions may include mild to severe, lymphoplasmacytic, and histiocytic to necrotizing myocarditis with multifocal to coalescing dense inflammatory infiltrates adjacent to fairly unaffected tissue (Figure 16.6).[21]

Acute nonsuppurative encephalitis with multifocal areas of neuronophagia and microgliosis has been associated with USUV, BAGV, ITV, and TMUV infection. Similarly, mild to severe acute nonsuppurative (lymphoplasmacytic and histiocytic) encephalitis and meningoencephalitis with mononuclear infiltrates, glial nodules, and perivascular cuffing have been described in the course of WNV infections (Figures 16.7 and 16.8), primarily affecting the cerebral periventricular parenchyma and mesencephalon.[19,26,27] Additionally, WNV and BAGV may cause nonsuppurative nephritis, hepatitis, and pneumonia. TMUV has been reported to cause oophoritis and vacuolar degeneration of the testes in ducks.[4]

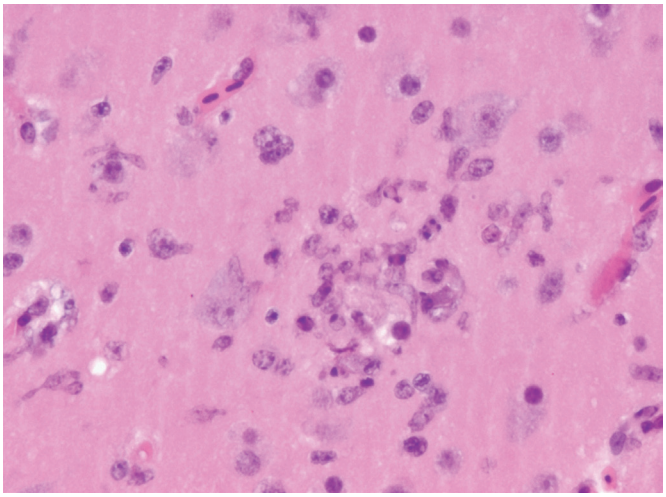


FIGURE 16.8 Cerebrum of the juvenile, female scarlet-breasted lorikeet *(Trichoglossus forsteni)* from Figure 16.7, demonstrating miliary necrosis with glia nodule formation as a result of WNV-2 infection. Hematoxylin and eosin stain, 400×. (Courtesy Dr. Helene Pendl, Pendl Lab, Zug, Switzerland.)

Ocular lesions of WNV may consist of lymphoplasmacytic and histiocytic uveitis, iridocyclitis, endophthalmitis, optic neuritis, and pectenitis, with inflammatory cells marginating in and around (cuffing) the dilated vessels of the pectin.[29]

Rarely, inflammatory lesions in adrenals and pancreas have been documented for WNV and TMUV.[4]

FURTHER DIAGNOSTICS

For specific flavivirus diagnosis, serum and blood clot samples (preferably paired, acute, and convalescent samples), oral and cloacal swabs, and feather pulp may be taken in live birds.[10] Additionally, a complete blood count and blood chemistry evaluation may help to assess the current general health status and organ function of the patient.

For the detection of antibodies against WNV and some other flaviviruses, plaque reduction neutralization tests (PRNT), virus neutralization tests (VNT), and indirect enzyme-linked immunosorbent assays (ELISAs; blocking ELISAs using monoclonal antibodies) are available.[17,30] As there is the potential for cross-reactivity with other members of one serogroup (e.g., the JEV antigen complex) within the family of Flaviviridae, blood should be collected using serum tubes without anticoagulant. Then viral genome may be detected from blood cells of the coagulum using polymerase chain reaction (PCR) parallel to the assessment of antibodies.[17] This increases the likelihood of (correct) pathogen identification.[30]

Virus isolation may be performed from oral and cloacal swabs, serum or liver, kidney, heart, spleen, and brain samples. Several mammalian cell lines as well as mosquito cells have been described for this method.[31–33]

The demonstration of viral antigen or genomic sequences is more often used and may be performed by using infected tissues (especially brain, liver, heart, kidney, and spleen), blood, cerebrospinal fluid, or other body fluids.[22,24] Real-time reverse transcriptase-polymerase chain reaction (RT-PCR) is a fast and sensitive test for virus RNA detection; however, in situ hybridization, NASBA (nucleic acid sequence-based amplification) assays and VecTest antigen-capture assays are also used regularly.[17] Immunohistochemistry may be performed on tissue samples from these organs as well, offering nearly equivalent sensitivity compared to virus isolation. In raptors, brain (cerebrum and cerebellum), heart, kidney, eye, and spleen have been demonstrated to be more often positive for WNV compared to pancreas, proventriculus duodenum, and other organs (Figure 16.9).[24,29]

THERAPY IN BIRDS

Supportive and symptomatic treatment is a key factor of flaviviral therapy, as there are no confirmed effective antiviral treatments available. This includes intravenous fluid supplementation (e.g., lactated Ringer solution), nutritional support (e.g., forced or tube feeding), and padding and sling support for ataxic and/or recumbent individuals. Medication usually includes antimicrobial (antibiosis and antimycosis to cover secondary infections), antiinflammatory and analgesic treatment (e.g., nonsteroidal antiinflammatory drugs [NSAIDs] such as meloxicam or celecoxib), vitamin supplementation (especially vitamins B and E), and thermal support.[10] Ivermectin has been anecdotally reported to have the ability to suppress virus replication and therefore may be added to the treatment for WNV infections in raptors.[34] This treatment may also be applicable to other flavivirus infections based on recent confirmation of its effect in USUV infected cell cultures. Likewise, robenacoxib has been suggested to promote favorable results in geese with CNS signs from WNV infection, with a return to clinical normalcy achieved in some birds. However, given the lack of evidence-based studies on the efficacy of these drugs, no definitive recommendations can be made on their use in the treatment of avian flaviviral infections. In any case, the infected birds should be isolated from the flock and strict hygiene and vector control must be implemented to prevent further spreading of the pathogen to other animals and humans. All involved staff must furthermore be informed about the potential zoonotic risk of flaviviral diseases, and personal protective cloth should be provided besides disinfectants.[10]

DISINFECTION AFTER FLAVIVIRUS CONTACT

Disinfection and virus inactivation of flaviviruses may be archived by low pH, ultraviolet light, gamma irradiation, and/or high temperature.[10] To inactivate WNV, the thermal inactivation point (TIP) has been set to 40°C and temperatures above 56°C for over 30 minutes have been tested sufficient.

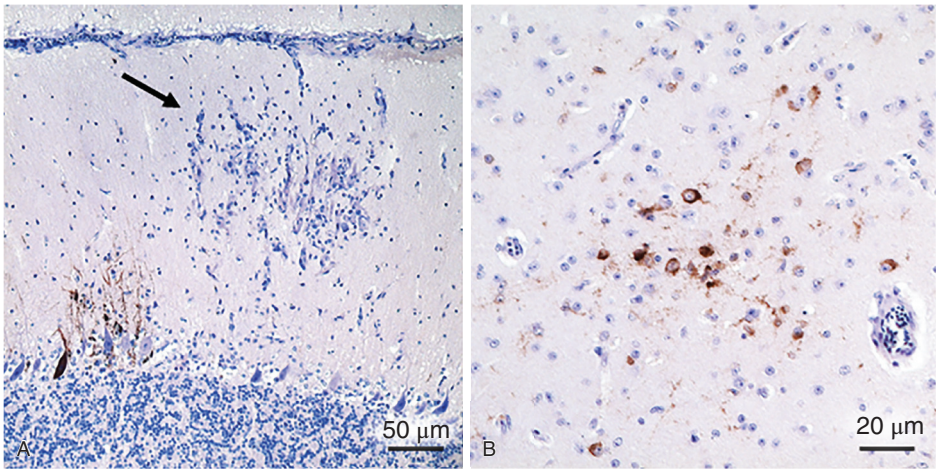


FIGURE 16.9 Immunohistochemical detection of West Nile virus (WNV) antigen in the brain of two Eurasian goshawks *(Accipiter gentilis)*. (A) Cerebellum showing focal viral antigen in Purkinje cells as well as in neurons and glia cells of the molecular layer in close proximity to a subacute brain lesion *(arrow)*, bar 50 µm. (B) Cerebrum with focal accumulation of viral antigen in few neurons and glia cells of gray matter, bar 20 µm; in-house polyclonal antibody specific for WNV (pab OM8). (Courtesy Dr. Christine Fast, Friedrich-Loeffler-Institut (FLI), Federal Research Institute for Animal Health, Institute of Novel and Emerging Infectious Diseases, Greifswald, Germany.)

WNV has been reported to be stable in alkaline environment (pH 7–9), but it may be inactivated in acid environment (pH 1–3). Common detergents, organic and lipid solvents, 70% ethanol, phenol iodophors, 1% iodine, 2% to 3% hydrogen peroxide, 2% glutaraldehyde, 3% to 8% formaldehyde, 1% sodium hypochlorite, and 500 to 5000 ppm available chlorine are reported to offer a sufficient disinfection.[10]

MONITORING AND SURVEILLANCE

A robust disease surveillance protocol should be implemented to monitor flaviviral diseases in free-ranging and captive bird populations. This may provide an early warning system for veterinarians, aviculturists, and authorities. In Europe, Australia, and the United States, different surveillance and monitoring regimes are implemented for flaviviruses in the avifauna and every veterinarian is encouraged to contribute and support these initiatives.[30,35] Especially in larger collections, dead birds should be sent for necropsy and screened for flaviviruses.[10] Examples of monitoring sites include the following: ECDC (https://www.ecdc.europa.eu/en/west-nile-fever/surveillance-and-disease-data/disease-data-ecdc); CDC (https://www.cdc.gov/west-nile-virus/php/resources/surveillance.html); California West Nile Virus Monitoring (https://westnile.ca.gov); and the Government of Canada (https://www.canada.ca/en/public-health/services/diseases/west-nile-virus/surveillance-west-nile-virus.html).

Besides monitoring programs as listed above, free-ranging birds found dead near aviaries should also be screened. A list of selected laboratories that offer PCR or serologic testing can be found in Table 16.2.

GENERAL MEASURES FOR FLAVIVIRUS PREVENTION

To reduce flavivirus transmission, vector control measures are crucial.[10,36] For mosquito control this may include monitoring of water-management systems, draining of pools, and removal of stagnant water sites, as these may serve as breeding habitat for mosquitoes. Adding fish to ponds may also

TABLE 16.2

List of Laboratories That Offer Testing for West Nile Virus in Birds

WOAH (World Organisation for Animal Health)/OIE Reference Laboratory:

Dr. E.N. Ostlund Diagnostic Virology Laboratory, National Veterinary Services Laboratories, PO Box 844, Ames, IA 50010, USA; Tel: +1 515-663-7551; Fax: +1 515-663-7348; Email: eileen.n.ostlund@aphis.usda.gov

Australia[a]:

The State Veterinary Diagnostic Laboratory, Woodbridge Road, Menangle, NSW 2568; Tel: +61 1800 675 623; Email: laboratory.service@dpi.nsw.gov.au

Australian Centre for Disease Preparedness (CSIRO), 5 Portarlington Road, East Geelong, Victoria 3220; Tel: +61 3 5227 5000; Website: https://www.csiro.au/en/about

Belgium:

CODA-CERVA, Sciensano, Rue Juliette Wytsman 14, 1050 Brussels; Tel: +32 2 642 51 11; Email: info@sciensano.be

France:

Anses, UMR 1161 Virologie, 94700 Maisons-Alfort, Paris; Website: https://www.anses.fr/en

Germany:

Friedrich-Loeffler-Institut, Institut für neue und neuartige Tierseuchenerreger, Südufer 10, D-17493 Greifswald; Tel: +49383517-1163/-1287; Fax: +49 38351 7-1226; Website: https://www.fli.de/en/institutes/institute-of-novel-and-emerging-infectious-diseases-innt/reference-laboratories/nrl-for-wnv/

Italy:

Istituto Zooprofilattico Sperimentale dell'Abruzzo e del Molise (Istituto G. Caporale) Teramo, Via Campo Boario, 64100 Teramo; Tel: +390861 332440, 28; Fax: +390861 332251; Website: https://www.izs.it/IZS/Centres_of_excellence/International_Centres/WOAH_Reference_Laboratory_for_West_Nile_Fever

The Netherlands:

Dr. van Haeringen Laboratorium B.V., Agro Business Park 100, 6708 PW Wageningen; Tel: (+31) 0317 416 402; Email: info@vhlgenetics.com

USA:

California Animal Health & Food Safety Lab System, College of Veterinary Medicine, University of Davis, 620 W. Health Sciences Dr., Davis, CA 95616; Tel: +1 530 752-9700; Fax: +1 53-752-6253; Email: daviscahfs@ucdavis.edu; Website: https://cahfs.vetmed.ucdavis.edu

Cornell Animal Health Diagnostic Center, College of Veterinary Medicine, Cornell University, 240 Farrier Rd, Ithaca, NY 14853; Tel: +1 607 253-3900; Fax: +1 607 253-3943; Email: diagcenter@cornell.edu; Website: www.vet.cornell.edu/animal-health-diagnostic-center

Georgia Infectious Diseases Lab, College of Veterinary Medicine, University of Georgia, 110 Riverbend Rd, Riverbend North (Room 150), Athens, GA 30602; Tel: +1 706 542-5812; Fax: +1 7-6 583-0843; Email: idl@uga.edu; Website: https://vet.uga.edu/diagnostic-service-labs/infectious-diseases-lab/

Veterinary Molecular Diagnostics, Inc., 5989 Meijer Dr. Suite 5, Milford, OH 45150; Tel: +1 513-576-1808; Fax: +1 513-576-6177; Website: www.vmdlabs.com

[a]In Australia, certain laboratories have a multiplexed MVEV/Kunjin virus PCR available, which has been used on bird tissues. However, it is questionable whether the PCR would detect exotic strains of WNV.

PCR, Polymerase chain reaction; *WNV*, West Nile virus; *MVEV*, Murray Valley encephalitis virus.

help to reduce the number of mosquito larvae.[10] The use of chemical insecticides, detergents, and biological agents from agriculture (e.g., *Bacillus thuringiensis*) may be effective for controlling mosquito plagues, but they should be used carefully, as they may be harmful to the environment and other organisms.[10]

The use of light- or pheromone-based live traps and mosquito netting to mosquito-proof aviaries is recommended.[10] Application of repellents might be beneficial, but most repellents may have strong side effects in birds and therefore should be used with caution. Independently, quarantine and disease screening of newly introduced birds are strongly recommended prior to including the animals in the collection.[10]

SPECIFIC PROPHYLAXIS—VACCINATION

Various flavivirus vaccines have been developed for the protection of humans against JEV and horses against WNV.[17,37] Even though these vaccines are not licensed for use in birds, several WNV vaccines have been used in various domestic and nondomestic/wild bird species with variable results, as recently thoroughly reviewed by Jiménez de Oya et al. (see Chapter 13).[37] Specific vaccines for use in birds have been developed against ITV in Israel and against TMUV in China.[4]

INACTIVATED VACCINES/KILLED VACCINES

The application of formalin-inactivated WNV lineage 1 vaccines (e.g., Duvaxyn WNV, Fort Dodge Animal Health, Hedge End, Southhampton, UK; Equilis West Nile, Intervet International BV, Boxmeer, Netherlands; West Nile-Innovator, Zoetis Inc., Florham Park, NJ, USA and Equip WNV, Zoetis Belgium SA, Louvain-la-Neuve, Belgium) induced antibody response against WNV in various avian species, even though a protection has not been assessed by virus challenge in many studies (see Chapter 13). In falcons, vaccination results were evaluated by virus challenge, recommending a three-injection scheme of the killed vaccine to achieve protection from lethal infection and reduced levels of viremia and virus shedding (Figure 16.10).[37] An inactivated TMUV vaccine (HB strain, Ringpu, Tianjin, China) is available to protect ducks against TMUV.[4]

RECOMBINANT VACCINES

Recombinant vaccines (e.g., PreveNile, Intervet; RecombiTEK, Boehringer Ingelheim Animal Health USA Inc., Duluth, GA, USA; Proteq West Nile, Boehringer Ingelheim GmbH & Co. KG, Ingelheim am Rhein, Germany), using live attenuated strains of different viruses as genetic backbone, have been evaluated in various bird species (see Chapter 13).[4,35] The recombinant vaccine RecombiTEK expressing the membrane precursor and envelop protein of WNV has been used successfully in falcons and led to decreased levels of viremia and virus shedding and reduced mortality using a three-injection scheme. Only minor side effects (local inflammation and low-grade necrotic lesions) at the injection site have been noted.[33]

Vaccination with recombinant vaccines induced specific antibody response in Japanese quail *(Coturnix japonica)* but not in western scrub-jays *(Aphelocoma californica)*.[4] Anecdotally, a three-injection scheme (interval 4 weeks) has been used successfully in snowy owls in Slovenia; however, seroconversion has not been

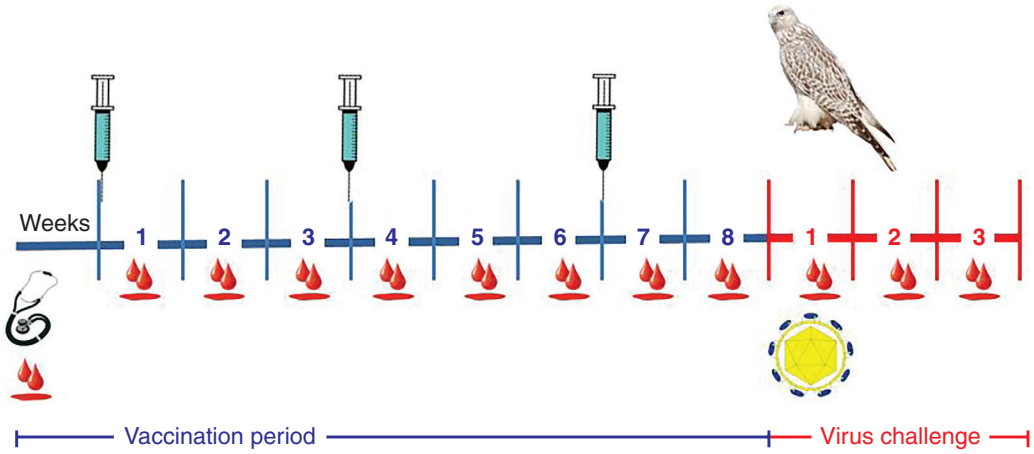


FIGURE 16.10 Example of a vaccination scheme for protection against West Nile virus (WNV) in large falcons using the killed vaccine Duvaxyn WNV, Fort Dodge (Fort Dodge Animal Health Limited, Hedge End, Southampton, UK; in the European Union [EU] now Equip WNV, Zoetis Belgium SA, Louvain-la-Neuve, Belgium; in the United States, West Nile-Innovator, Zoetis Inc., Florham Park, NJ, USA) and a canarypox recombinant live virus vector vaccine RecombiTEK-Equine rWNV vaccine, Boehringer Ingelheim Animal Health USA Inc., Duluth, GA, USA (now approved in the EU as Proteq West Nile, Boehringer Ingelheim Pharma GmbH & Co. KG, Ingelheim am Rhein, Germany). Best results were obtained for both vaccines using a three-dose vaccination scheme with an interval of 3 weeks between the intramuscular injections, resulting in alleviation of clinical signs, absence of fatalities, and reduction of virus shedding and viremia. (From Angenvoort J, Fischer D, Fast C et al: Limited efficacy of West Nile virus vaccines in large falcons (*Falco* spp.), *Vet Res* 45:41, 2014).

evaluated and no virus challenge was performed in these birds. In Israel, ITV is controlled by vaccination using a live attenuated virus vaccine (Biovac Biological Laboratories, Akiva, and Phibro, Beth Shemesh, Israel). To protect against TMUV, the live duck Tembesu virus vaccine (Zhengye, Jilin, China) has been used in ducklings and layer ducks.[4] Moreover, multivalent vaccines using recombinant avian viruses as vectors for the membrane precursor and the envelop protein of TMUV resulted in full protection of ducks during virus challenge tests.[4]

CHIMERIC VACCINES

Three chimeric vaccine candidates (ChimeriVax-WN, chimeric WN/DEN4, and WN/DEN4-3'D30) were used in fish crows *(Corvus ossifragus)* and geese, but failed to prevent clinical signs, viremia, or mortality after WNV challenge.[4,37]

SUBUNIT VACCINES

Subunit vaccines based on the truncated envelope protein of WNV have been used in domestic goose, Hawaiian goose *(Branta sandvicensis)*, and red-legged partridge *(Alectoris rufa)* and protected the partridges against WNV challenge.[4]

DNA VACCINES

DNA vaccines encoding the truncated envelope protein of WNV lineages 1 and 2 induced partial protection against live WNV challenge in falcons when *in vivo* electroporation was applied parallel to vaccination.[38] In fish crows, a DNA vaccine expressing the membrane precursor and envelop protein of WNV prevented mortality and reduced viremia levels after experimental WNV infection.[39] However, antibodies were absent in island scrub-jays *(Aphelocoma insularis)*, and western scrub-jays were not protected during WNV challenge by using the same vaccine.[4] In American crows *(Corvus brachyrhynchos)*, American robins *(Turdus migratorius)*, and red-tailed hawks, the intramuscular administration of DNA vaccines reduced viremia levels effectively, even though antibody titers were low after vaccination.[4] Likewise, in penguins, immune response to plasmid DNA vaccine has been inferior compared to a killed vaccine.[4] In contrast, stimulation of antibody production has been demonstrated in Californian condors *(Gymnogyps californianus)* 4 weeks after vaccination.[4] A DNA vaccine expressing the membrane precursor and envelope or the core genes of TMUV reduced clinical signs and prevented death in ducks following TMUV challenge.[4]

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CHAPTER 17

Campylobacteriosis

Yuko Sato

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INTRODUCTION

Campylobacteriosis in poultry has historically been focused on its zoonotic significance for human public health. *Campylobacter* spp. are generally regarded as a commensal bacteria in birds, and their infection rarely results in clinical disease or significant pathological lesions. This chapter focuses on four *Campylobacter* species isolated from birds and their significance to human or poultry health. These species are *Campylobacter jejuni*, *Campylobacter coli*, *Campylobacter lari*, and *Campylobacter hepaticus*. All of these are associated with foodborne illness in humans except for *C. hepaticus*, a recent etiological agent of spotty liver disease historically known as avian vibrionic hepatitis, where the pathogenesis of the disease in poultry is poorly understood due to its recent identification and characterization.

ETIOLOGY

Campylobacter spp. are motile, non–spore-forming, spiral-shaped, gram-negative rod bacteria. The genus *Campylobacter* contains at least 30 species and subspecies, with genus *Campylobacter* being associated with a wide variety of diseases in humans and animals. Of significance to poultry and human health are the thermophilic *Campylobacter* spp., *C. jejuni*, *C. coli*, and *C. lari*. These species grow optimally at 37°C to 42°C on artificial media and are slow-growing, fastidious organisms that require a microaerobic atmosphere.[1] Other species including *Campylobacter upsaliensis* and *Campylobacter hyointestinalis* have been isolated from poultry, but at a much lower rate with no significance to poultry health.[2]

Many species of poultry, especially gallinaceous birds, frequently carry *C. jejuni* or *C. coli* as a commensal organism. Despite it producing little to no pathology in birds, colonization of *Campylobacter* spp. in commercial chickens and turkeys results in it being among the leading cause of bacterial pathogens in food safety and public health. *C. jejuni* is most frequently associated with human campylobacteriosis, followed by *C. coli*, and—rarely—*C. lari* or others.[3]

In the 1950s, a condition known as avian vibrionic hepatitis associated with *C. jejuni* and *C. coli* was been described in laying hens and ostriches in the United States. The disease was recognized in many countries including Australia, New Zealand, Canada, Egypt, Germany, Austria, Italy, Hungary, Bulgaria, Switzerland, the United Kingdom, Ukraine, and the Netherlands.[4] However, an experimental study suggested that *Campylobacter* alone would not be sufficient to cause mortality or lesions associated with vibrionic hepatitis, but that, rather, a predisposing factor in chickens might be required for the development of the disease.[2,5] A novel *Campylobacter* spp. was isolated from infected birds in England (2015) and subsequently in Australia (2016). In 2016, this novel species was characterized and named *C. hepaticus*. Currently, the disease it causes has been redefined as spotty liver disease (SLD), and it has been identified from the United States and from other parts of the world.[6,7]

EPIDEMIOLOGY AND PATHOGENESIS

Generally, *C. jejuni* and other commensal *Campylobacter* species colonize the avian gut in their ceca around 2 to 3 weeks of age. The bacterium is introduced horizontally by fecal-oral route and is very well adapted to the avian species. As few as 35 colony-forming units (cfu) can be sufficient for colonization in the chicken gut.[4] Within 24 hours postingestion, *C. jejuni* reaches the cecum and multiplies. Although the main sites of colonization include the ceca and colon, other organs such as the small intestines and liver have also been colonized.[8] A unique feature of *Campylobacter* colonization is the fact that it resides predominantly in the mucus layer of the intestinal crypts without direct adhesion or invasion of the epithelial cells.[1,4] This explains why there may be no overt clinical signs associated with poultry infected with the bacterium.[3]

Although circumstantial evidence suggests that vertical transmission of *C. jejuni* from breeder flocks to progeny may occur, the current understanding is that *Campylobacter* is a horizontally transmitted bacteria, as isolation from newly hatched poultry has not been reported under field conditions. This most likely is due to the presence of maternally derived antibodies in young chicks conferring protection against colonization of the bacteria in their gut.[2,3,9] However, vertical transmission has been hypothesized because of isolation of *Campylobacter* spp. from the outer and inner shell surfaces of infected breeders, from reproductive tracts of hens, and from semen of infected broiler breeder roosters.[3,10–12] Recent molecular diagnostics also detected *Campylobacter* DNA from embryos and newly hatched chicks.[13,14] Contrasting studies indicate that transmission of *Campylobacter* through fecal contamination of eggshells post-lay is unlikely due to its limited capability for invading the eggshell and poor survivability on

eggshell surface, in air sacs and albumen.[15] Most studies indicate that the bacteria only survive a couple of hours (up to 6 hours) in any part of the egg laid at body temperature of the bird.[1,9,10,16] In addition, many studies report the finding of different *Campylobacter* strain genotypes in broiler flocks versus their parent flocks. Based on these findings, it was concluded that transmission of *Campylobacter* spp. through the egg is a rare, though not impossible, event and does not play a role in transmission in poultry farms. Introduction of *Campylobacter* to newly hatched flocks remains a gap in our knowledge of the epidemiology of this bacteria.[3,9]

The prevalence of *Campylobacter* spp. in commercial poultry is relatively high but may vary according to region, seasons, and production types, ranging from 2% to 100%. Seasonal variations occur with a peak in summer and autumn months, perhaps due to an increase in fly population and fly-mediated transmission.[3,11,17,18] This correlates with human campylobacteriosis that peaks in summer months, especially in temperate climates.[2] In addition to flies, epidemiologic studies also indicate that other mammals including rodents and the presence of other livestock, including cattle, sheep, pigs, pets, and other birds, have been indicated as potential vectors and associated with risk factors for poultry with *Campylobacter* infection.[3,19]

In terms of the novel *C. hepaticus*, much is still to be discovered. *C. hepaticus* DNA has been detected in a variety of environmental samples including wild bird feces, flies, rat feces (and ceca), soil, water, dust, and mites from farms positive with SLD.[7] However, the organism is quite fastidious compared to other *Campylobacter* spp. and no viable isolate was recovered from these samples. In addition, there were instances where *C. hepaticus* was detected but disease was not detected for several weeks later. This study suggests that perhaps presence of the organism is not enough to induce disease, and predisposing/concurrent factors may be required to either increase abundance of the organism or by increasing susceptibility of the host to the organism.[5] In terms of commercial chickens *(Gallus gallus domesticus)*, *C. hepaticus* is more prevalent in organic and free-range flocks versus conventional production, with perhaps predilection toward floor-raised birds due to the fecal-oral transmission of the organism.[6,7] Due to the majority of egg production being in cages in the United States, the disease was not identified until recently in 2018.[6,7] However, disease has also been observed in caged layers and broiler breeders, although to a much lesser extent.[2]

CLINICAL SIGNS

Campylobacter infections in poultry, with the exception of *C. hepaticus*, typically produce no clinical signs of disease under natural conditions. There is one report that ostriches *(Struthio camelus)* can have clinical illness that resulted in lesions in the liver and intestines.[20] Some studies reported that experimental infection of young chickens with high doses of *Campylobacter* can induce clinical diseases including watery/mucoid/bloody diarrhea, weight loss, or mortality as early as within 72 hours.[2,3]

Clinical signs with SLD are much more dramatic, especially with birds coming into peak lay. SLD can have upwards of a 10% increase in mortality and a 10% to 25% drop in egg production, which can occur simultaneously but without any effects on external and internal egg quality. Hens can also appear normal with no apparent clinical signs.[6,7] A few reports furthermore mention loose stools.[2]

GROSS AND HISTOPATHOLOGIC LESIONS

The lack of clinical signs correlates to the lack of gross lesions in most cases of natural *Campylobacter* infections. In experimentally infected chicks, lesions are often mild, nonspecific, and confined to the gastrointestinal tract. There may be accumulation of fluid, gas, or excess mucus, distention of the gut including ceca with watery/foamy contents. Blood and mucus in the lumen of the small intestine and petechial hemorrhages in the gizzard mucosa of chicks can be seen occasionally.[2,3]

Microscopic lesions are often absent in natural infections but have been described in experimental infections. Lesions are typically observed in the ileum and ceca, with studies that report mild to moderate responses including increased inflammatory cells (predominantly lymphocytes and heterophils), mucosal damage with thickening, shortening, and fusion of the villi in the ileum, as well as edema of the lamina propria and submucosa.[21] In some cases, the bacteria can be seen attached to the brush borders on enterocytes, within intestinal epithelial cells, and inside or outside of the cells of lamina propria with minimal damage and a lack of necrosis.[2]

Gross lesions with SLD are typically characterized by 1- to 2-mm, white to gray or cream, random military foci on the surface of the liver (Figure 17.1). In most cases, these military foci are observed in all liver lobes, but in some cases, only one or two lobes are involved. Liver foci may also occasionally coalesce, especially in coinfections with cecal worms and mild intestinal lesions characterized by watery/catarrhal enteritis.[6,7] Livers may be swollen or slightly enlarged, and the liver lesions may be accompanied by catarrhal enteritis on rare occasions.[2]

Histopathologic lesions of SLD are characterized by multifocal to coalescing hepatocellular necrosis characterized by discrete margins and necrotic centers mixed with degenerating heterophils in acute to subacute stages (Figure 17.2). Lesions do not have a predilection or affinity to any specific cell type or structure and appear to be random. Earlier studies with avian vibrionic hepatitis showed that histopathologic examination of these livers did not reveal any intralesional bacteria, which is true in cases of *C. hepaticus* infections. Coupled with this observation and the random necrosis, studies speculate that the liver necrosis is a result of a hepatotoxin produced at a remote location, although this has not yet been discovered.[7]

DIAGNOSIS

Thermophilic *Campylobacter* spp. grow optimally at 37°C to 42°C (98.6°F–107.6°F) on artificial media and are slow-growing, fastidious organisms that require a microaerobic atmosphere (5% O_2, 10% CO_2, 85% N_2). Because target organs are typically intestines and intestinal contents where many background organisms may be present, the use of selective media containing a cocktail of antibiotics to inhibit growth of others is typically used. In addition, an enrichment step in liquid media followed by plating on agar plates is usually preferred to direct plating for the isolation of *Campylobacter* from

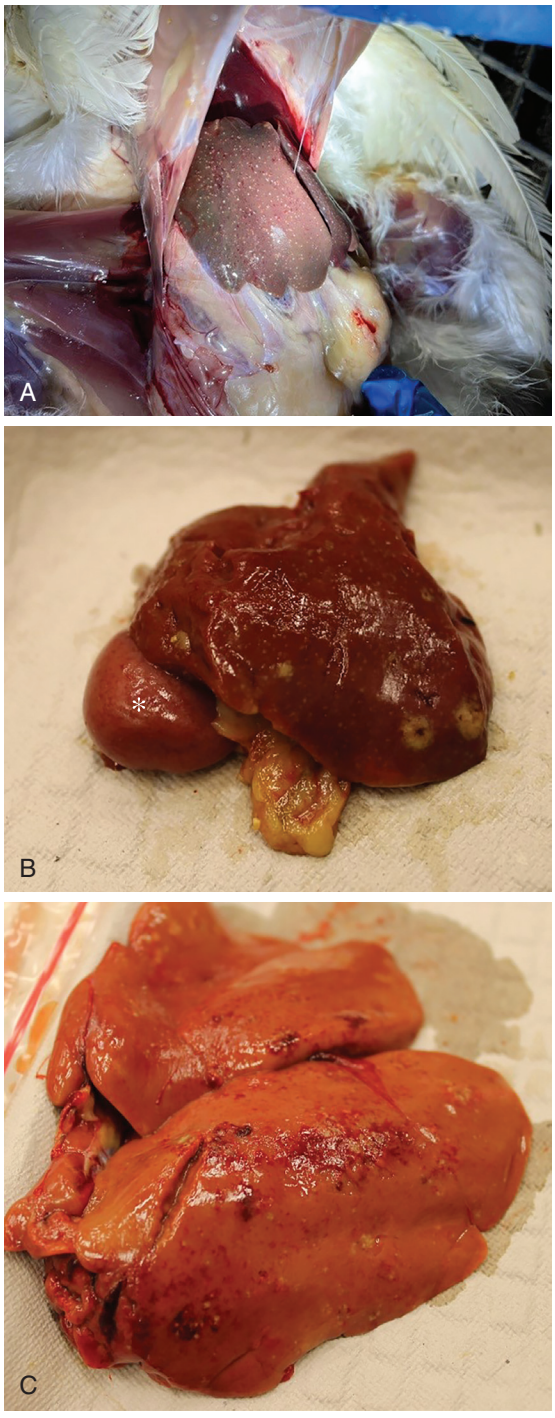


FIGURE 17.1 Gross lesion of typical livers with spotty liver disease characterized by (A) numerous necrotic foci throughout the surface of the liver. (B) Necrotic lesions can coalesce in some cases. Note splenomegaly (*) in this case. (C) The liver's edges are rounded, and it is slightly pale. (Courtesy Yuko Sato, Iowa State University.)

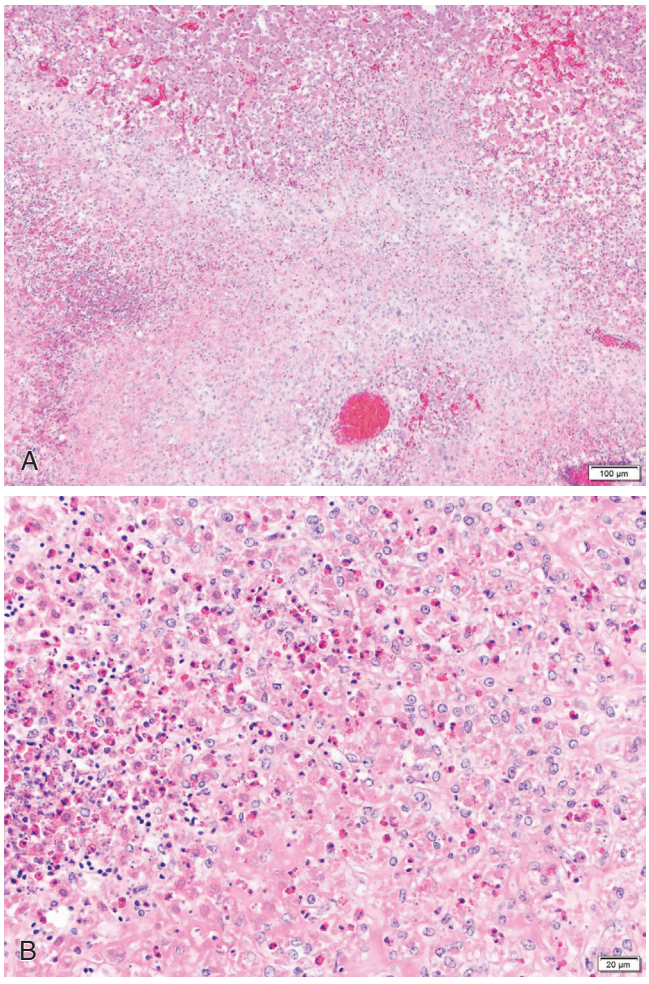


FIGURE 17.2 Photomicrograph of liver from Figure 17.1B. Histopathologic lesions of livers affected by *Campylobacter hepaticus*. (A) Large lesions can coalesce, with this lesion surrounding a portal vein (4× magnification, hematoxylin and eosin [H&E]). (B) Higher magnification of necrotic foci showing diffuse hepatocellular necrosis and marked heterophilic infiltrates surrounding areas of necrosis. (40× magnification, H&E).

processed foods in which bacteria are usually in relatively low numbers. Colonies are visible on solid media after 48 hours of incubation, but it may take up to between 72 and 96 hours to observe some slow-growing strains.[2] Moisture tends to affect appearance of the colonies: colonies may appear gray, flat, irregular, and thinly spreading in moist plates versus round, convex, and glistening colonies when plates are dry. Classical bacteriology identification of *Campylobacter* is based on morphology, typical cellular shapes (spiral or curved rods; Figure 17.3) using fuchsin stain, and characteristic rapid darting motility observed under a phase-contrast microscope.[2,22] Currently, many diagnostic laboratories utilize matrix-assisted laser desorption ionization time-of-flight (MALDI-TOF) mass spectrometry for species-level identification of *Campylobacter* similarly to other bacteria. Species-level polymerase chain reaction (PCR) targeting housekeeping or species-specific genes, including *23S rRNA*, *glyA*, *ceuE*, *lpxA*, *hipO*, *mapA*, and *ask*, have been developed and used. However, several studies report its inferiority in sensitivity and specificity compared to real-time multiplex PCR; hence, these molecular assays have been increasing in popularity.[23]

In terms of *C. hepaticus*, isolation has been best from bile samples despite the lesions being present in the liver. This organism is even harder to grow compared to other *Campylobacter* spp. and does best in incubation in Preston broth for up

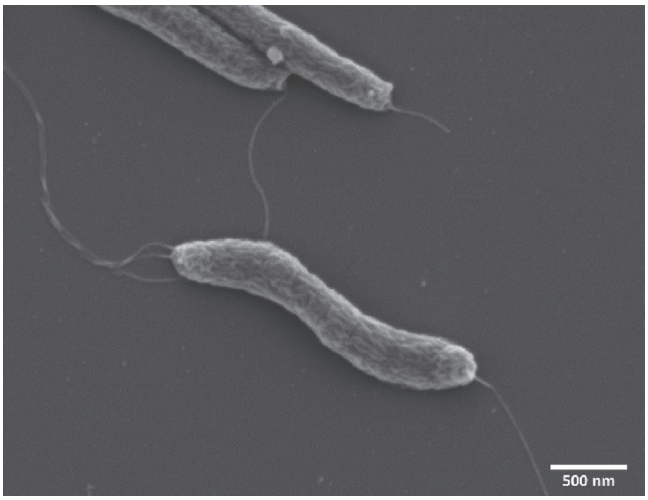


FIGURE 17.3 *Campylobacter* spp. can be diagnosed using phase-contrast microscopy, where they can be recognized as spiral or curve-shaped rods, with a single flagellum at each end. (Courtesy LAGier, Pratt and Threadgill. ASM MicrobeLibrary.org.)

to 7 days at 35°C (95°F), followed by plating into *Brucella* agar with 5% horse blood for up to 7 days prior to subculturing. Similar to other *Campylobacter* spp., biochemical reactions as well as MALDI-TOF methods have also been used for identification. 16S rRNA gene sequencing methods and PCR identification using targets such as glycerol kinase gene specific to *C. hepaticus* have been used for higher sensitivity in identification.[5,6]

TREATMENT, PREVENTION, AND CONTROL

Anecdotal evidence states that the use of broad-spectrum antibiotics such as tetracyclines in addition to management strategies has been helpful to reduce clinical signs and mortality associated with *C. hepaticus* infections. However, these antibiotics are not labeled for controlling these infections. Thus, judicious use is recommended, only when secondary pathogens contribute to significant and/or elevated mortality. Historically, erythromycin and fluoroquinolones have been used to control avian vibrionic hepatitis, but these are not approved for use in commercial poultry. There have also been issues with high incidences of antibiotic-resistant *Campylobacter* strains, some of which are multidrug resistant.[1,2,21]

Because thermophilic *Campylobacter* is considered a commensal and, in most instances, birds do not exhibit clinical disease, nor do they have associated mortality, the focus has been predominantly on control rather than treatment. However, because colonization in poultry intestines is very optimized and the organism is ubiquitous in a farm environment, prevention of a flock from turning up *Campylobacter* positive is an extremely difficult task.[24] In general, preharvest control methods focus on three principles: reduction of environmental exposure, increase in host resistance, and using alternatives to antimicrobials. Single strategies are not successful when applied alone for preharvest and postharvest control[1–3,18]; hence, it is recommended to instill multiple measures in combination for higher efficacy. However, this may prove to be challenging due to economic and practical reasons.

The most fundamental method is implementation of strict biosecurity and good hygiene methods to prevent *Campylobacter* from entering poultry houses. Focus and attention to potential routes of infection (see Epidemiology and Pathogenesis) appear to be successful. One study looked at tidiness and sanitation in the facilities including frequency of changing footbaths and the cleanliness of the anterooms correlated with prevalence of *Campylobacter* in flocks. Another study looked at rodent control associated with lower risk (odds ratio [OR] = 0.15) of colonization.[25] A Danish study looked at the use of fly screens around barns and reducing prevalence of *Campylobacter*-positive flocks from 41.4% to 10.3% while also preventing peak incidence during summer months.[26] Similar studies looking at flies as mechanical vectors of the bacteria saw a similar approximately 30% decrease in positive flocks.[19,26] One study decreased the prevalence of *Campylobacter* in broiler houses from 80% to less than 40% where strict personnel hygiene and disinfection protocols were utilized.[27] In contrast, production practices such as thinning (early removal of a subset of birds to allow more space for the rest of the flock), commonly used in Europe, reported an increase in risk of *Campylobacter* contamination due to higher human traffic, use of equipment, and contaminated vehicles.[3,28] However, challenges exist with biosecurity measures in commercial settings due to it being cost prohibitive, hard to maintain compliance, and highly variable in effectiveness, dependent on the production systems used.[2,3,9]

Reduction of colonization through administration of feed additives and water acidification has also been explored.[29] Water additives include short- and medium-chain fatty acids, plant-derived additives, egg yolk powder, chlorination, monocaprin, and organic acids. The idea behind its use is that these acids might aid in lowering pH of the gut, making it unfavorable for colonization by *Campylobacter* spp., as these organisms do not tolerate pH levels below 2.5 or 3 for more than an hour in in vitro experiments.[2] Organic acids such as lactic acid, formic acid, acetic acid, and propionic acid, during grow-out and feed withdrawal prior to harvest were reported to reduce recovery and colonization of *Campylobacter* (62.3% vs 85.1%) from the crop in one study.[30] Another study showed a combination of 2% formic acid and 0.1% potassium sorbate, resulting in total prevention of colonization of broilers *C. jejuni*.[31] Other studies showed log reductions of *Campylobacter* in broiler ceca.[32] However, conflicting studies showed that addition of organic acids in experimentally infected birds continued to shed and be colonized with *Campylobacter*.[33] Feed additives including prebiotics (such as lactose, fructooligosaccharides, mannose-oligosaccharide), immune response stimulators (such as selenium and beta-glucan), activated charcoal, and other organic acids have been used to acidify the feed, similar to their use in water. Many of these products have limited success when used alone and are often used in combination with water and other management strategies. It is interesting to note that *Campylobacter* spp. associated with protozoa such as *Acanthamoeba polyphaga* allows increased tolerance to these products including chlorine and acids.[2,9]

Litter treatments have been assessed for their efficacy in controlling *Campylobacter* colonization. There is some success in decreasing colonization (from 90% to 10% in treatment groups) and cecal load (up to 5 $\log_{10}$ reductions) in laboratory trials using aluminum sulfate and sodium bisulfate in broiler barns.[34] However, the effect on commercial farms was found to be negligible unless low pH in the litter was maintained throughout the entire grow-finish period.[2,9] In addition, the organism thrives better in wet environments, so maintaining low moisture levels will also help. A study by Sahin et al. showed that paw quality (due to wet litter and poorly ventilated barns, resulting in ammonia burns) had a positive association (OR = 21.67, 1.36-fold increase in odds ratio of having positive cfu for each 10% increase in paw lesions) with increased risk of *Campylobacter* colonization. This correlates with the fact that higher litter temperature and lower ammonia levels in the litter have negative correlations with *Campylobacter* colonization within broiler flocks (Sahin et al., unpublished data).

Another method to control *Campylobacter* colonization implicates the use of antibiotic alternatives for competitive exclusion of *Campylobacter* in the gut. The potential use of bacteriophages has been evaluated in several studies.[32,35] Some studies had immediate effects of log reductions of cfu in poultry ceca after oral administration, but there seem to be conflicting studies that show limited success depending on strains and combination of bacteriophages.[36] In addition, a few studies indicated that the impact of the bacteriophages deteriorated after a few days and stabilized to potentially insignificant differences between the controls.[37] Overall, there seem to be several challenges with the use of bacteriophages, including the narrow spectrum of efficacy, rapid development of resistance, and the low efficiency of phage production in *Campylobacter*.[2,23]

AMP (antimicrobial peptides) such as bacteriocins have had some promise as a means for controlling *Campylobacter* colonization in chickens. Most successes described are those produced from commensal bacteria isolated from the chicken gut. Several AMPs purified from *Paenibacillus polymyxa*, *Lactobacillus salivarius*, and *Enterococcus* spp. have shown high efficacy in reducing *C. jejuni* infections and even total prevention of detectable levels,[38] although on-farm control has not been evaluated due to the cost. Purified AMPs such as defensins and cathelicidins also have promise for broad-spectrum antimicrobial activity, but it is not a cost-effective option for commercial use and also has potential issues in relation to meeting regulatory approval.[2,9]

Although there is well-established evidence that protective host responses do occur with *C. jejuni* infection,[4,39] hence supporting development of immunization strategies, currently, there are no commercial vaccines available for controlling *Campylobacter* infections in poultry. Some of the challenges include antigenic/genetic diversity of different strains, lack of understanding of protective immunity in poultry, and failure to induce strong and persistent local, mucosal immune responses. Two types of vaccines have been described in the literature, including killed whole-cell, live-attenuated, flagellin-based subunit vaccines and glycoconjugate vaccines using the conserved bacterial N-glycan. Both vaccines showed up to 10-log reduction in *C. jejuni* colonization and induced specific antibodies.[40] Coupled with the short life span of broilers (6–7 weeks) and the high cost of development and administration of a vaccine for food safety purposes, the use of vaccination in commercial poultry farms needs further investigation.[2,9]

Finally, host genetic resistance research has been ongoing to look at different lines of chickens to look at susceptibility to infection. Recent studies report that resistance to infection is linked with the inhibition of small, GTPase-mediated signal transduction as well as the tumor necrosis factor receptor superfamily genes.[41] In the future where antibiotic use is severely limited, perhaps there will be a time where selective breeding of broilers with resistance to *Campylobacter* infection will occur.[2,9]

NONPOULTRY AVIAN HOSTS

As in poultry, healthy, nonpoultry avian hosts have been implicated in carrying *Campylobacter* spp. However, there is a knowledge gap about the epidemiology of *Campylobacter* spp. in wild bird populations. In studies looking at prevalence of *Campylobacter* spp. in wild birds, important factors that influence recovery include feeding habit, forage area, and proximity to livestock operations.[19] In addition, sparrows (*Passer* spp.) at poultry or swine farms in the summer were more likely to carry the organism than those caught at cattle farms, speculating on the fact that there is lower cross-contamination of foraging areas due to the fact that cattle were out grazing in the pasture and thus minimizing contact with sparrows closer to farm buildings.[19] Although the prevalence rate of wild birds carrying *Campylobacter* spp. is much lower (7.7%–12.9%) than of domestic poultry, there have been several instances where human outbreaks have been linked to wild birds, thus suggesting that wild birds are potential vectors and reservoirs of infection for poultry and for humans.[42]

A study in the United Kingdom surveilled wild animals, including birds, that exhibited clinical signs of diarrhea and other gastrointestinal disease for presence of *Campylobacter*. There was a positive association with mixed concurrent infections with other bacterial infections, with the highest prevalence in Gruiformes, Ciconiiformes, and Anseriformes.[43] Although disease from infection is rare, *Campylobacter* spp. has been isolated as a coinfection or opportunistic infection from the small intestines of clinically ill birds, especially Psittaciformes and Passeriformes exhibiting clinical signs and pathologic lesions of hepatitis, lethargy, loss of appetite, weight loss, and yellow diarrhea with potential mortality.[44–46]

PUBLIC HEALTH SIGNIFICANCE

Campylobacter can exist in very high numbers in the intestines and feces of poultry and other food-producing animals. Foodborne infections with *Campylobacter* are estimated to affect over 800,000 people annually in the United States and infect 1% of the population in the European Union.[47,48] Infection by *Campylobacter* results in severe gastroenterocolitis in humans. During 2012, over 70,000 cases of human campylobacteriosis were reported in the United Kingdom, of which 90% of the cases were due to *C. jejuni*. While these cases were often genetically linked to production animals (including poultry), small proportions (2.1%–3.5% annually) were also linked to wild bird isolates, further complicating the epidemiology of the disease.[47]

The Centers for Disease Control and Prevention constantly ranks *Campylobacter* spp. as a major problem in food safety and continues to see increased rates of infection reported annually. With regards to poultry, several interventions with improved preharvest strategies and postharvest strategies, such as better storage and handling of poultry meat, will reduce the risk of campylobacteriosis in humans.[1]

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CHAPTER 18

Avian Chlamydiosis—Beyond *Chlamydia psittaci*

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INTRODUCTION

Chlamydiaceae are a group of obligate intracellular, gram-negative bacteria that can infect a wide variety of birds and mammals, with some having zoonotic potential. This family comprises two genera. The first genus, named *Chlamydia*, includes the species *C. trachomatis*, *C. pneumoniae*, *C. pecorum*,[1] *C. abortus*, *C. felis*, *C. caviae*, *C. muridarum*, *C. suis*, *C. psittaci*, *C. avium*, *C. gallinacea*,[1,2] *C. poikilothermis*,[3] *C. buteonis*,[4] and *C. serpentis*,[5] as well as the candidate species, *Candidatus* (*Ca.*) *C. ibidis*,[6] *Ca. C. sanzinia*,[7] *Ca. C. corallus*,[3] and *Ca. C. testudines*.[5] The other genus, recently described as *Chlamydiifrater (Cf.)*,[8] includes two new species that have only been detected so far in flamingos, namely *Cf. phoenicopteri* and *Cf. volucris*.

Until very recently, *C. psittaci* was considered to be the main *Chlamydia* species infecting birds. However, recent research has identified new *Chlamydia* species in domestic bird populations, captive birds, and wild avian species, albeit frequently without clinical signs in the host animals. These discoveries have led to the identification of novel species that appear to be globally distributed. Notable among these are *C. gallinacea*, which appears to be endemic in poultry species, and *C. avium*, frequently detected in pigeons (Columbiformes) and psittacine birds (Psittaciformes). Additionally, other emerging species, such as *C. buteonis*, initially misclassified as *C. psittaci* M56 and as *C. abortus*, were delineated using advanced molecular techniques. These breakthroughs were made possible by using polymerase chain reaction (PCR) methods targeting the Chlamydiaceae family and the development of whole-genome sequencing techniques applicable to this obligate intracellular bacterium.

Because of their wide host range, Chlamydiaceae species have a significant impact on human and animal health worldwide.[9,10] The discovery of new species within this family from avian hosts highlights how much we still have to learn about the biology and pathogenicity of the Chlamydiaceae as a whole. The continued identification of novel chlamydial organisms provides evidence that some may be pathogenic to birds, humans, or other animals, thereby posing new zoonotic risks.[11] The intensification of farming and the widespread popularity of companion animals also present ongoing opportunities for emerging zoonoses.[9,11]

In this chapter, we will provide an overview of current knowledge regarding *C. avium* in pigeons and psittacine birds, *C. gallinacea* in poultry, as well as novel *Chlamydia* species circulating in avian wildlife. Furthermore, we report on an outbreak of *C. psittaci* in Sphenisciformes as little documentation about *Chlamydia* in this bird order is available.

CHLAMYDIA AVIUM IN PIGEONS AND PSITTACINE BIRDS

A retrospective analysis of chlamydial strains isolated from asymptomatic feral pigeons *(Columba livia)* in Italy and France, as well as from clinically affected psittacine birds in Germany, led to the identification and description of a novel chlamydial species that was designated *C. avium* in 2014.[12] This new agent was clearly distinguished from *C. psittaci* through phylogenetic analysis based on 16S rRNA and whole-genome analysis, and it was found to be phylogenetically closest to another novel chlamydial species, *C. gallinacea*, which was isolated from poultry birds.[12] Within the Chlamydiaceae family, *C. avium* and *C. gallinacea* represent a distinct clade alongside *C. psittaci*, *C. abortus*, *C. felis*, and *C. caviae* (Figure 18.1).

The Agent

C. avium has primarily been detected in pigeons and psittacine birds, although its presence in other birds is assumed and has anecdotally been shown. There is evidence for the existence of host-specific genotypes,[12,13] although data on this topic remain limited. Currently, there are two complete *C. avium* genome assemblies available: one from the type strain 10DC88^T, isolated from a parrot in Germany, and the other from the strain PV 4360/2, which was isolated from a feral pigeon in Italy. When compared, these genomes of about 1 Mbp differ by 3696 single nucleotide polymorphisms (SNPs), resulting in a nucleotide identity of 99.64%.[14]

C. avium can be grown in cell culture or embryonated chicken eggs. Electron micrographs of the designated type strain 10DC88^T in infected cells (Figure 18.2) show multiple inclusions of variable size (2–10 μm)[12] inside infected cells. These inclusions typically consist of small, electron-dense elementary bodies (EBs) measuring 240 to 330 nm in diameter, alongside larger, more translucent reticulate bodies (RBs) with a diameter ranging from 450 to 1100 nm. These observations

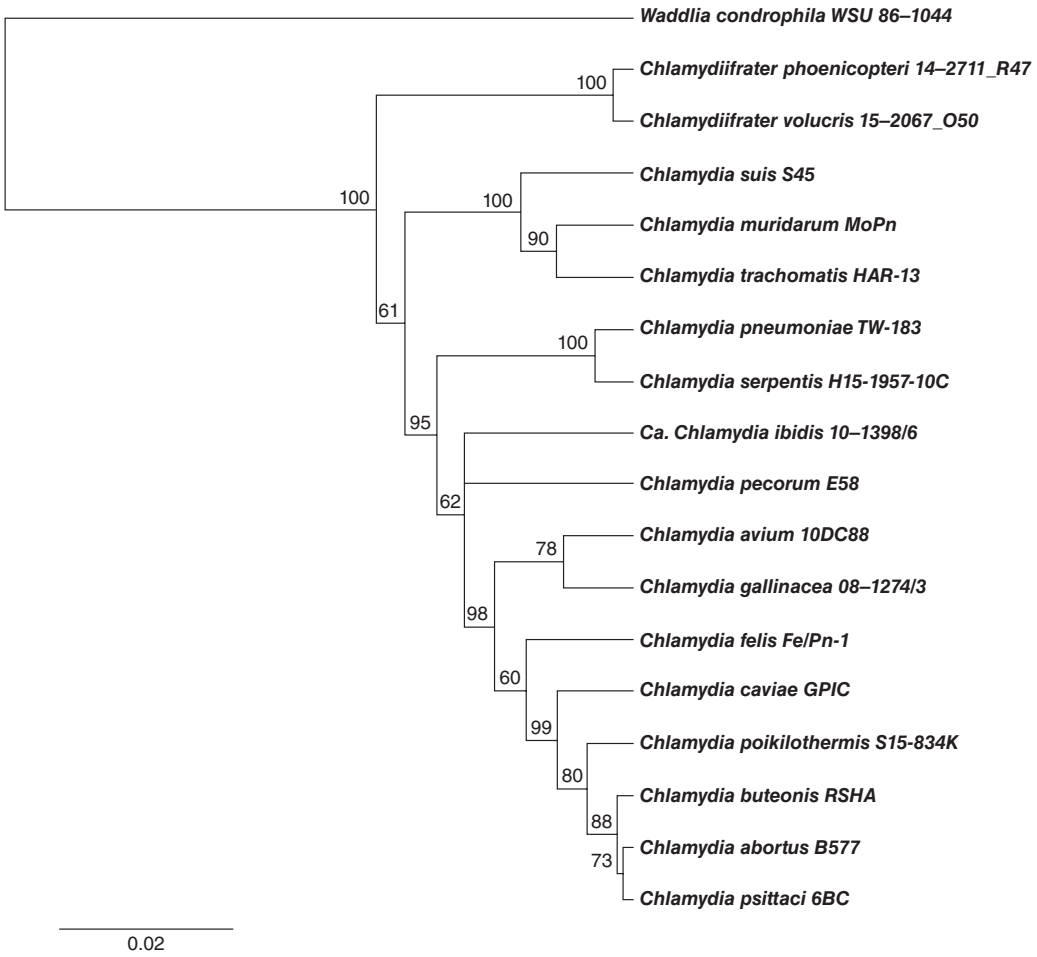


FIGURE 18.1 Phylogeny of the family Chlamydiaceae including the type strains of all currently valid members and those of *Waddlia condrophila* as an out-group. The phylogenetic tree was reconstructed based on a ClustalW alignment of 16S RNA gene sequences with the Geneious Tree Builder using the Jukes-Cantor model and unweighted pair group method with arithmic mean algorithm. Bootstrap values are based on 1000 replicates. The scale bar indicates percentage of sequence divergence.

indicate that *C. avium* has a binary life cycle, which is typical for all Chlamydiaceae. Unlike some fast-growing *C. psittaci* strains that take 36 to 48 hours to complete a developmental cycle, *C. avium* strains take longer, around 60 to 72 hours.[12]

From the limited epidemiological data available, it appears that *C. avium* is widely disseminated, particularly among pigeons. Investigations of feral pigeon populations in different European cities have identified the agent in a range of 0.6% to 28.4% of total samples and in 3.1% to 85.2% of Chlamydiaceae-positive samples (Table 18.1).[15–18] In domestic or racing pigeon flocks, *C. avium* was detected in 5.9% to 7.4% of total samples and in 17.4% to 41.6% of Chlamydiaceae-positive samples (Table 18.1).[16] In most studies, *C. psittaci* appeared as the predominant chlamydial agent in pigeons, but there were instances where *C. avium* outcompeted *C. psittaci* in feral pigeon populations in two Dutch cities.[15] Co-infections with both *C. psittaci* and *C. avium* in individual birds appear also to be common.[15,17] In psittacine birds, assessing the prevalence and impact of *C. avium* is challenging, as reported findings are often individual cases associated with chlamydiosis outbreaks. There have been limited systematic epidemiological investigations conducted so far.[19] Due to limited reports and information, the worldwide distribution of *C. avium* remains unknown.

Detection and isolation of *C. avium* have mainly been reported in European countries, including France,[16,21] Belgium,[19] Germany,[18] Italy,[14] Switzerland,[17] The Netherlands,[15] and Poland.[22] There have also been reports of *C. avium* detection in an asymptomatic feral pigeon in Thailand[23] and in two asymptomatic psittacine birds in Argentina.[24]

Transmission

The transmission pathway of *C. avium* is not specifically documented, but it is likely to be similar to that of *C. psittaci*, involving direct bird-to-bird transmission or airborne transmission through inhalation of aerosolized dried fecal particles or ocular and nasal secretions containing the bacteria.[25] Chlamydial shedding is supposed to be intermittent and may depend on the virulence of strains and the host's immune status. For *C. psittaci*, stressors such as nutritional deficiencies, overcrowding, chilling, breeding, transport, and handling have been identified as important factors influencing both increased shedding

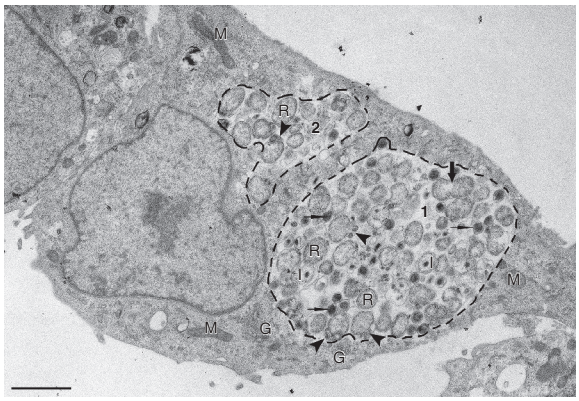


FIGURE 18.2 Electron microscopic image of Buffalo green monkey (BGM) cell culture infected with *Chlamydia avium* strain 10DC88, bar = 1.5 μm. Two inclusions are indicated by *dashed lines*. They contain reticulate bodies *(R)*, elementary bodies *(thin arrows)*, and intermediate bodies *(I)*. Binary fission and budding events are marked with a *thick arrow* and *arrowheads*, respectively. Mitochondria *(M)* and stacks of Golgi membranes *(G)* are closely associated with the inclusion's membrane. (Courtesy Elsevier Ltd. From Sachse K, Laroucau K, Riege K, et al: Evidence for the existence of two new members of the family Chlamydiaceae and proposal of *Chlamydia avium* sp. nov. and *Chlamydia gallinacea* sp. nov., *Syst Appl Microbiol* 37(2):79–88, 2014. http://dx.doi.org/10.1016/j.syapm.2013.12.004.)

and susceptibility to infection,[25] and these factors are assumed to apply to *C. avium* as well. Other potential transmission pathways, such as transmission through feeding, blood-sucking ectoparasites, or vertical transmission have not been documented but cannot be ruled out. So far, only individual cases of *C. avium* infection inside groups of captive psittacine birds have been investigated, suggesting a limited spread of the bacterium in aviaries.[13] Susceptibility to *C. avium* infection appears to vary significantly between different bird species, as well as within the same avian species.

The zoonotic potential of *C. avium* is unknown. The fact that it has never been isolated from or detected in human samples could merely be a consequence of limited diagnostic approaches targeting only traditional chlamydial agents rather than indicate a lack of bird-to-human transmission. Therefore, a risk of transmission to humans in close contact to parrots and pigeons such as pet bird owners and breeders, staff in zoos and pet shops, pigeon fanciers, and veterinarians cannot be excluded.

Clinical Signs

The pathogenic role of *C. avium* has not been investigated through experimental infections to fulfill Koch's postulates. Current knowledge is primarily based on limited epidemiological data from natural infections. It appears that the majority of pigeons harboring *C. avium* do not develop clinical signs. In contrast, in parrots and parakeets, the agent can be associated with a range of clinical symptoms, including respiratory disease, enteritis, anemia, emaciation, and sudden death without prior symptoms.[12,21] At necropsy, pathological findings similar to acute *C. psittaci* infections were noticeable, including hepatosplenomegaly, thickened air sacs, congested lungs, and the accumulation of fibrinous exudate in respiratory organs and the liver. Histological examination showed lymphohistiocytic, moderate necrotizing hepatitis and moderate necrotizing splenitis with hyperplasia.[13] Co-infection with other *Chlamydia* spp. or pathogens could exacerbate the course of the infection with *C. avium* in birds.

Diagnosis

The choice of sample types for analysis depends on the clinical signs observed in the birds. In case of acute illness, samples should include inflammatory or fibrinous exudate in or around affected organs, ocular and nasal exudates, as well as tissue samples from the spleen, liver, and lungs. For cases with diarrhea, samples from colon contents and feces can also be included. In live birds, it is recommended to analyze pharyngeal, nasal, and cloacal swabs. Due to intermittent shedding, especially in asymptomatic carriers, it is advisable to perform repeated sampling on several consecutive days.

Nucleic acid amplification tests are recommended for fast, sensitive, and accurate diagnosis. A *C. avium*–specific PCR method, based on the *enoA* gene, is available.[26] For the detection and identification of *C. avium* in suspected birds,[27] it is advisable to follow a hierarchical approach. This one begins with an initial Chlamydiaceae-specific PCR screening.[28,29] Positive cases identified though this screening should then undergo both a *C. avium*–specific[26] and a *C. psittaci*–specific PCR assay.[30] In previous studies, *C. avium* may have been overlooked or misidentified with *C. psittaci* due to the lack of appropriate diagnostic tools.

Proper handling and storage of samples is essential for successful culture of *C. avium*. Samples should be stored in a special preservation medium consisting of sucrose/phosphate/glutamate (SPG) with a cocktail of antibiotics/antifungals (streptomycin, vancomycin, gentamicin, and amphotericin)

TABLE 18.1

Epidemiological Surveys on *Chlamydia* sp. Dissemination in Pigeons: Number of Samples Tested Positive for *Chlamydia avium*

	Country	*C. avium* Positive/Total (%)	*C. avium* Positive/ Chlamydiaceae Positive (%)	Reference
Feral pigeon	France	10/708 (1.4%)	10/125 (8%)	16
	Germany	4/630 (0.6%)	3/128 (3.1%)	18
	The Netherlands	23/81 (28.4%)	23/27 (85.2%)	15
	Switzerland	3/323 (0.9%)	3/64 (4.7%)	17
Domestic pigeon	Germany	4/54 (7.4%)	4/23 (17.4%)	20
	Switzerland	2/34 (5.9%)	2/5 (40.0%)	17
	Germany	n.d.	10/24 (41.6%)	C. Schnee, personal communication

and stored at −80°C if they cannot be processed immediately.[27] For the isolation and cultivation of *C. avium*, both embryonated chicken eggs and Buffalo green monkey (BGM) cells have proven to be suitable methods. Detection of *C. avium* can be accomplished through direct or indirect immunofluorescence using antibodies targeting the family-specific lipopolysaccharide (LPS) or using relevant staining techniques (Gimenez, Giemsa, Ziehl-Neelsen, or Macchiavello stain).

Treatment and Prevention

No vaccine is currently available for *Chlamydia* infections in birds. Sanitary measures must be implemented, in particular, the separation of sick animals and shedders, quarantine procedures for newly acquired birds, the separation of birds from multiple sources, and the application of strict hygienic practices (disinfection; see Chapter 11).[31]

Antibiotic treatment is only indicated by the onset of symptoms and laboratory confirmation of chlamydial infection. The primary choice of antibiotics for treatment includes tetracyclines, but quinolones, macrolides, and even rifamycins can also be administered.[32,33] Recently, psittacine birds with respiratory troubles and confirmed *C. avium* infection have been successfully treated with doxycycline administered at 20 mg/kg orally every 24 hours for a duration of 6 weeks. This treatment was completed with hepatoprotectors, vitamins, and tube feeding.[13] In racing and fancy pigeons, a shortened treatment period of 25 days with doxycycline at 40 mg/kg in drinking water has proven effective against *C. psittaci*/*C. avium* co-infections. However, achieving complete elimination of infection may be challenging, as reported in other animal and human chlamydial infections, due to the potential for persistence and resistance.[32] Therefore, laboratory testing after treatment is advisable, with at least two posttreatment analyses, typically 7 and 14 days after therapy completion.

While the zoonotic potential of *C. avium* has not been established, it is advisable to implement preventive measures similar to those used in case of *C. psittaci* infection. Wearing personal protective equipment when handling infected animals or samples, as well as during cleaning activities, is crucial for reducing the risk of aerosol exposure of people. This precaution is essential to minimize the possibility of inhaling infectious particles in confined environments.

CHLAMYDIA GALLINACEA IN POULTRY BIRDS

While an investigation was underway in France in 2009 at a poultry slaughterhouse in response to reports of unusual pneumonia cases among its workers, atypical *Chlamydia*, different from *C. psittaci*, was primarily isolated from chickens, the predominant avian species slaughtered at this facility.[34] This led to the identification and description of a novel chlamydial species that was designated *Chlamydia gallinacea* in 2014.[12] These atypical bacteria were clearly differentiated from *C. psittaci* through phylogenetic analysis based on 16S rRNA and whole-genome analysis, and they found to be phylogenetically closest to another novel chlamydial species, *C. avium*. Although *C. gallinacea* was suspected to be the cause of the pneumonia cases, this was never confirmed. Furthermore, the fact that ducks carrying *C. psittaci* also passed through this slaughterhouse suggests a potential *C. psittaci* infection in these individuals.

The Agent

C. gallinacea has primarily been detected in poultry, although its presence in other birds is assumed and has only been shown anecdotally.[35] Genome analysis has revealed that *C. gallinacea* constitutes a separate species, as it exhibits an average nucleotide identity of less than 81% when compared to its closest relative, *C. avium*.[34] Several complete genome assemblies are available for different *C. gallinacea* strains. Strain 08-1274/3^T,[12,36–38] isolated from a chicken in France, is considered the type strain.[12] Multilocus sequence typing (MLST) conducted on two complete genomes and 23 Chinese samples from nine farms has revealed 15 sequence types (STs) and two main clades. All Chinese STs clustered within one clade, further subdivided into 14 lineages, highlighting the genetic diversity of *C. gallinacea*.[39] Currently, it is unknown if these genetic differences have functional implications for host adaption or virulence.

C. gallinacea has been successfully isolated in cell culture and in embryonated eggs through yolk sac inoculation.[12,38,40] When cultivated in cell culture, it takes 60 to 72 hours to complete one developmental cycle, and serial passaging is more challenging compared to *C. avium* or *C. psittaci* strains.[12] In infected cells, single and multiple inclusions of variable size can be observed, containing small EBs and larger RBs. Within RBs, different stages of division through binary fission have been observed, indicating a binary life cycle typical for *Chlamydiaeceae*.[12]

C. gallinacea is mainly detected in poultry but has been found in other species such as pigeons, woodcocks (*Scolopax* spp.), a parrot, ultramarine grosbeak (*Cyanoloxia brissonii*), and cattle.[2,38,41–44] Its presence is widespread, with reports from various regions, including Asia,[38] Australia,[44] Europe,[15,22,45–47] North America,[48] and South America.[49] Most studies have focused on chickens, where *C. gallinacea* is highly prevalent (Table 18.2). Interestingly, it is more frequently detected in layers than in broilers, possibly due to differences in age and breeding systems.[49] Among layers, those within the age of 40 to 60 weeks appeared to have the highest risk of infection. This may be influenced by factors such as the timing of introduction, the duration of *C. gallinacea* infections, and the acquisition of immunity.[15]

A higher risk of *C. gallinacea* infection has also been reported in backyard chickens or chickens in open-sided poultry houses compared to commercial breeding farms with stricter biosecurity measures.[49] Furthermore, an association with certain bedding materials has been found.[50] These associations suggest that the environment plays a crucial role in the introduction and survival of *C. gallinacea*. This hypothesis is further supported by partial *ompA* gene sequence data, which have shown that individual farms, regardless of the flock, harbor their own unique *C. gallinacea* strain.[51]

Co-infections with both *C. gallinacea* and *C. psittaci* have been documented in chickens, but they seem to be sporadic.[46,52] In one case, co-infected chickens were traced back to a farm that also bred ducks.[52] It is likely that the ducks were the source of the *C. psittaci* infection through environmental contamination. While *C. psittaci* is considered to be the main chlamydial species in ducks and *C. gallinacea* in chickens,[45] there is a report of a high prevalence of *C. psittaci* and the absence of *C. gallinacea* in commercial Belgium chicken farms (18 out of 19).[53] A recent study has shown that experimental *C. gallinacea* infection in chickens does not provide protection against a subsequent experimental infection with *C. psittaci*.[54] Future research will help clarify the roles and possible interactions of *C. gallinacea* and *C. psittaci* in different poultry species.

TABLE 18.2

Chlamydia gallinacea Prevalence Data in Poultry Species

Host	Country	*C. gallinacea*–Positive Farms (%)	*C. gallinacea*–Positive Samples (%)	*C. gallinacea*–Positive Samples/Chlamydiaceae-Positive Samples (%)	Reference
Chicken	Australia	2/6 (33%)	36/247 (15%)	—	44
	China	—	4/71 (6%)	4/4 (100%)	47
	China	—	359/1791 (20%)	359/442 (81%)	38
	Croatia	—	2/4 (50%)	2/2 (100%)	47
	France	—	38/70 (54%)	38/43 (88%)	47
	France	—	190/1315 (14%)	190/248 (77%)	45
	France	—	18/45 (40%)	18/22 (82%)	50
	Greece	—	22/65 (40%)	22/32 (69%)	47
	Italy	9/16 (56%)	24/160 (15%)	24/24 (100%)	46
	Mexico	—	29/668 (4%)	29/42 (69%)	49
	Poland	19/113 (17%)	—	—	22
	Slovenia	—	29/73 (40%)	29/47 (62%)	47
	The Netherlands	71/151 (47%)	245/755[a] (32%)	245/247 (99%)	51
	United States	—	64/472[a] (14%)	64/64 (100%)	48
Duck	China	—	15/179 (8%)	15/43 (35%)	38
	France	—	0/570 (0%)	0/75 (0%)	45
	France	—	17/60 (28%)	17/36 (47%)	50
	Poland	0/23 (0%)	—	—	22
Goose	China	—	3/115 (3%)	3/11 (27%)	38
	Poland	0/18 (0%)	—	—	22
Guinea fowl	France	—	20/315 (6%)	20/29 (69%)	45
	United States	—	2/6[a] (33%)	2/2 (100%)	48
Turkey	France	—	9/30 (30%)	9/11 (82%)	45
	Mexico	0/1 (0%)	0/35 (0%)	—	49
	Poland	0/28 (0%)	—	—	22
	Switzerland	—	7/1008 (1%)	7/7 (100%)	17
Chicken, guinea fowl	France	—	29/75 (39%)	29/36 (81%)	45
Pheasant, quail, duck	Mexico	—	56/176 (32%)	56/61 (92%)	49
Various[b]	United States	—	0/53 (0%–7%)	—	48

[a]Pooled samples.
[b]Turkey, goose, duck, quail, pheasant, peafowl, and pigeon.

Transmission

C. gallinacea is shed in high quantities in fecal droppings, and environmental contamination appears to play an important role in transmission. The main route of transmission is the fecal-oral route, and unlike *C. psittaci*, airborne transmission through dried fecal material does not seem to occur.[55] Vertical transmission has been proposed based on the detection of *C. gallinacea* in the albumen and yolk of embryonated eggs. However, an alternative explanation could involve environmental contamination through penetration of the eggshell.[55] Cases of mechanical transmission through biting flies, lice, or mites have not been reported.

Currently, there is no conclusive evidence supporting the potential for zoonotic transmission of *C. gallinacea*. Initial concerns about transmission to humans arose when the first strains of *C. gallinacea* were isolated, notably in a French slaughterhouse where workers displayed signs of pneumonia.[40] However, this has never been confirmed, and it is more likely that contamination with *C. psittaci* through duck exposure was the probable source. In a recent prevalence study involving 69 Dutch poultry farmers and workers, testing failed to detect any *Chlamydia* DNA in human samples.[50] Additionally, during an outbreak of human psittacosis, in which eight women were exposed to chickens infected with both *C. gallinacea* and *C. psittaci* during plucking, no *C. gallinacea* DNA was found in their samples.[52] More recently, an investigation among Italian commercial or backyard poultry farmers identified 12 positive *C. gallinacea* cases out of 16 farmers in farms where the bacteria was also detected.[56] Notably, there were no associated clinical cases, indicating a low potential for *C. gallinacea* to induce disease in exposed individuals.

Clinical Signs

In chickens and turkeys, *C. gallinacea* infections are mainly asymptomatic, although reduced weight gain has been observed in chicken broilers.[37,44,49,56] Experimental data on the pathogenesis of *C. gallinacea* in poultry show that *C. gallinacea* mainly resides in the gut,[57] although it has been detected in blood, lung, heart, liver, trachea, kidney, pancreas, and spleen samples.[38,51,55] In contrast, experimental infections with *C. psittaci* in poultry birds can lead to severe clinical outcomes, although chickens are relatively resistant to *C. psittaci* infections.[58,59] The clinical signs and pathogenesis of *C. psittaci* infections depend on both the avian host and the *C. psittaci* strain.[60]

Diagnosis

A cloacal swab is the preferred sample for detecting *C. gallinacea* in poultry birds, as cloacal shedding of the bacterium is often high.[43,57] Additionally, *C. gallinacea* can also be detected in oropharyngeal swabs and, in lower amounts, in samples of lung, heart, kidney, liver, pancreas, trachea, spleen, and whole blood.[38]

PCR-based detection is considered the gold standard for *Chlamydia* diagnostics.[27,61] It is advised to first run a broad Chlamydiaceae PCR that targets the 23S rRNA gene.[28] Other 23S rRNA–based assays have also been described.[29,38] For the specific detection of *C. gallinacea*, PCR or sequence-based approaches can be used.[43,50]

Isolation can be performed using various cell culture systems, including Human Epidermoid carcinoma-2 (HEP-2) cells, BGM cells, and embryonated eggs via yolk sac inoculation.[12,37,38,40] Proper handling and storage of samples are crucial for successful isolation. In cases where immediate processing is not possible, samples should be stored at −80°C in a preservation medium consisting of SPG containing a cocktail of antibiotics and antifungals (streptomycin, vancomycin, gentamicin, and amphotericin).[27] For identification in cell culture or pathology, standard staining techniques, immunofluorescence, or immunohistochemical protocols for other *Chlamydiae* can be applied.[27] These last techniques mainly rely on the cross-reaction of antibodies targeting the LPS of chlamydial organisms.

Indirect detection of *C. gallinacea* can be challenging and is not recommended, as specific serological assays are not readily available. Generic serological assays for *Chlamydia* can be used, based on cross-reactive chlamydial antigens. For example, Stokes et al. reported the use of the ImmunoComb solid-phase ELISA (enzyme-linked immunosorbent assay) (Biogal, Israel) with serum samples of *C. gallinacea*–positive chickens.[44] However, it is essential to interpret results with caution, as cross-reactivity with other *Chlamydia* species and even gram-negative bacteria can occur when using generic serological assays. The development of peptide-based assays offers a promising solution to address issues related to cross-reactivity but requires further validation.[62,63]

Treatment and Prevention

The clinical relevance of *C. gallinacea* is questionable, and a zoonotic potential has not been established. Infections in poultry appear to be mostly asymptomatic. It is advised to only consider treatment when clinical symptoms are noticed, *C. gallinacea* infection is confirmed, and other possible infectious causes are excluded. There is no reason to believe that *C. gallinacea* responds differently to treatment than other Chlamydiaceae. Therefore treatment guidelines for *C. psittaci* can be applied.[31]

While there is no evidence of a zoonotic potential, it is advisable to use a flow cabinet when isolating or propagating the bacterium in cell or egg culture as high loads of the bacterium can be achieved during isolation.

Environmental contamination is thought to play an important role in transmission, so general cleaning and disinfection measures should be implemented. *Chlamydia* is susceptible to heat and most disinfectants, including 1:1000 dilutions of quaternary ammonium compounds, 70% ethanol, and a 1:32 preparation of household bleach. The bacterium is resistant to both acid and alkali.[31]

CHLAMYDIA BUTEONIS IN FREE-RANGING BIRDS OF PREY

The Agent

Chlamydiaceae DNA has been consistently detected from raptors for years.[64–71] A unique chlamydial isolate was identified from a cluster of four clinical cases of juvenile red-tailed hawks (RTHA, *Buteo jamaicensis*) in California, and partial sequence analysis of the 23S rRNA and the *ompA* genes indicated that this strain was closely related to, but distinct from, the *C. psittaci* M56 genotype, which had previously been detected in snowshoe hares and muskrats. At that time, it was designated as "*C. psittaci* strain G."[72] Interestingly, *C. psittaci* M56 was recently identified by PCR in samples from five raptors in Switzerland that were also tested with *C. buteonis* primers.[73] The same authors re-evaluated cases from falcons in the United Arab Emirates that had originally been identified as intermediate to *C. psittaci* and *C. abortus* and found they were all positive using *C. buteonis* primers.[74] Additional studies are necessary to evaluate whether avian chlamydiosis identified from raptors is attributed to a single or multiple species.

The prevalence of Chlamydiaceae DNA detection in apparently healthy, free-ranging wild *Buteo* species in California was found to be 1.4% (4/291).[71] Prevalence of 1.6% for *C. psittaci* DNA was identified among raptors in wildlife rehabilitation centers in Oregon.[75] A spatial cluster of seropositive *Buteo* raptors and raptors testing positive for *C. buteonis* was detected between Sacramento and San Francisco in California.[76] An isolate was cultured from a hatch-year red-shouldered hawk (RSHA, *Buteo lineatus*) from California and designated as the reference strain of the new *Chlamydia buteonis* species.[4] Chlamydial DNA was isolated from a juvenile Swainson's hawk *(Buteo swainsoni)* and an adult RTHA, and the full genetic sequences aligned 100% with each other and 99.97% with the recently described C. *buteonis*.[4] A retrospective study of all raptors admitted to the University of California, Davis Veterinary Medical Teaching Hospital from 2000 to 2019 identified chlamydial DNA in 1.7% of all raptors and 5.1% of all RTHAs presented (M.G. Hawkins, unpublished data, November 8, 2023). Sixty-one raptors were identified as confirmed or probable clinical cases: 57/61 cases were RTHAs; 2/61 were Swainson's hawks; 1/61 a rough-legged hawk *(Buteo lagopus)*; and 1/61 a ferruginous hawk *(Buteo regalis)*.

Transmission, Signalment, and Clinical Signs

To date it is unclear how *C. buteonis* is transmitted, but this organism is expected to be similar in transmission to *C. psittaci* and *C. abortus*.[4,25] The zoonotic potential of *C. buteonis* has not yet been evaluated. Human cases have been linked to other chlamydial species in free-living raptors.[77,78] There was suspected *C. psittaci* genotype B zoonotic transmission between a long-eared owl (Asio otus) and volunteers at a rehabilitation center.[78] Chlamydial organisms can be transmitted horizontally to other birds via ingestion or inhalation of ocular and nasal discharges, feces, urine, and fomites, increasing the potential risk of an outbreak at a rehabilitation center.[25,79,80] Enhanced surveillance and control measures should be in place in wildlife rehabilitation centers in order to minimize the risk of both zoonoses and of reintroduction of infection back into wildlife populations.

To date, *Buteo* raptor species seem to be the most affected of the raptors; however, a number of falcons have tested positive with *C. buteonis* PCR primers,[74] so much more testing is necessary to determine the global host range. Positive birds were significantly more likely to present during winter months (December–February) in California, and hatch-year

RTHAs were more frequently diagnosed (M.G. Hawkins, personal communication). Clinical signs observed were similar to those described in *C. psittaci*–infected psittacine birds and included lethargy, dehydration, emaciation, weakness, conjunctivitis, diarrhea, and biliverdinuria. Respiratory signs have been uncommon.

Diagnosis

Diagnosis of chlamydial infections is challenging due to the variability of nonspecific clinical signs, thus multimodal diagnostic testing is always recommended for confirmatory diagnosis.[31]

Culture requires specialized handling in the United States due to the zoonotic potential of *C. psittaci* and very few laboratories offer this testing. The most commonly available antemortem molecular testing modality available today is PCR for *C. buteonis*. This organism also cross-reacts with *C. psittaci* PCR primers, so, if suspected, it should be tested for specifically. For screening purposes, conjunctival-choanal-cloacal combination swabs are commonly obtained. Samples are collected from commonly infected tissues including the liver, lung, air sac, gastrointestinal tract, kidney, and spleen at postmortem examination.[31,81,82] Separate swabs from the conjunctiva and choana can support infection whereas cloacal swabs may detect a chlamydial organism passing through the gastrointestinal tract. Two non-*Buteo* raptors, one great horned owl (*Bubo virginianus*) and one prairie falcon (*Falco mexicanus*) tested positive via PCR for *C. buteonis*[76] and falcons originally identified as having chlamydial infections with an organism between *C. psittaci* and *C. abortus* were retested and found to be infected with *C. buteonis*.[74] Additional work is needed to further evaluate whether the organism affects additional raptorial species. Amplicons from swab samples from raptors did not identify as *C. psittaci* and were reported as "atypical" positives based on the amplitude of the detected amplicon.[71,82] If there is no commercially available specific PCR assay for *C. buteonis*, it is advised to run a broad *Chlamydia* PCR for raptor samples, then perform further subtyping. A commonly used *Chlamydia* PCR detects all Chlamydiaceae and targets the 23S rRNA gene.[28]

Paired acute- and convalescent-phase serum samples, obtained at least 2 weeks apart, are ideal and should be tested in the same laboratory at the same time.[31] Elementary body agglutination (EBA) detects chlamydial IgM antibodies, while the indirect fluorescent antibody test (IFA) detects IgY antibodies at the genus level for any *Chlamydia* spp.[31] The *C. psittaci* EBA serologic assay available in the United States is specific and does not detect host antibody response to *C. buteonis*.[71] An EBA assay specific for *C. buteonis* was utilized in a recent study;[76] the test is now commercially available in the United States.

Ancillary testing includes imaging, a complete blood count (CBC), and a biochemistry panel. The most consistent radiographic finding was splenomegaly (M.G. Hawkins, personal communication; Figure 18.3). Anemia with a moderate-to-severe leukocytosis were the most reported CBC abnormalities, characterized by heterophilia, bands, monocytosis, and eosinophilia—extraordinarily like those found in parrots with *C. psittaci*. Hyperfibrinogenemia was also commonly identified (M.G. Hawkins, personal communication; Table 18.3). The most significant changes on plasma biochemistry panels were elevations in creatine kinase (CK), aspartate aminotransferase (AST), uric acid, globulins, and hypoalbuminemia (Table 18.4).



FIGURE 18.3 Lateral radiograph of a red-tailed hawk (*Buteo jamaicensis*) with avian chlamydiosis. Hepatosplenomegaly and cranial thoracic air sac lines are evident. Splenomegaly is the most common radiological finding in raptors with *Chlamydia buteonis*.

Histopathological evaluation of postmortem tissues from *Buteo* hawks found lesions like those of *C. psittaci* in psittacines in the liver, spleen, air sac, heart, lung, and kidney (M.G. Hawkins, personal communication). Chlamydial immunofluorescent IHC testing has been recently reported to be nonspecific and therefore not useful for diagnosis.[31] However, many cases have tested positive by IHC and have been correlated with tissue PCR and in situ hybridization, suggesting that pathologists familiar with chlamydial lesions can use IHC staining in the diagnosis of this disease (M.G. Hawkins, personal communication).

Treatment and Prevention

To our knowledge, treatment of *C. buteonis* or other chlamydial organism infections in wild raptors has not yet been reported. As modes of transmission are also not known yet for this organism, it is challenging to predict which preventative protocols may be successful in minimizing this infection. The severity of chlamydiosis in *Buteo* raptors raises the question of whether this disease could also threaten raptors at a population level. From November, 2021, through March, 2022, increased admissions and mortalities of free-ranging red-tailed hawks into rehabilitation centers throughout the state of California were reported to the California Department of Fish and Wildlife. Fifty of 76 bird carcasses tested were positive for chlamydiosis on IHC and tissue PCR during this outbreak. Of the four species that have shown clinical signs and have tested positive, the RTHA, rough-legged hawk, and ferruginous hawk populations are considered stable, whereas the Swainson's hawk is listed as a threatened species in California due to loss of nesting grounds. Additionally, non-endangered species may be a reservoir for the disease with the potential for spillover into less stable populations, such as the California condor *(Gymnogyps californianus)*, a federally listed endangered species in the United States. More work is needed to determine the host range of this chlamydial species to gain better understanding of the potential threat it poses to wildlife populations.

TABLE 18.3

Median ± SE Complete Blood Count Values of 28 Red-Tailed Hawks *(Buteo jamaicensis)*, 1 Ferruginous Hawk *(Buteo regalis)*, and 1 Swainson's Hawk *(Buteo swainsoni)* With *Chlamydia buteonis*

Parameter	Median ± SEM	Range	Reference Interval (Mean ± SD)[83]
Hematocrit (%)	28.5 ± 9.2	5–42	37.2 ± 4.6 (PCV)
Leukocytes ($\times 10^3$ cells/μL)	34.55 ± 23.13	7.40–99.75	19.28 ± 7.51
Heterophils ($\times 10^3$ cells/μL)	18.43 ± 18.43	2.70–78.80	10.81 ± 4.82
Bands ($\times 10^3$ cells/μL)	3.1 ± 5.05	0–16.53	
Lymphocytes ($\times 10^3$ cells/μL)	1.0 ± 2.01	0–10.00	5.16 ± 2.98
Monocytes ($\times 10^3$ cells/μL)	2.74 ± 2.81	0.21–11.27	1.47 ± 1.24
Eosinophils ($\times 10^3$ cells/μL)	0.81 ± 1.64	0–8.64	1.96 ± 1.69
Basophils ($\times 10^3$ cells/μL)	0.20 ± 0.45	0–1.68	0.06 ± 0.19
Total protein (g/dL)	4.65 ± 1.86	0.70–7.5	3.41 ± 0.65
Fibrinogen	400 ± 163	<100–700	

SEM, Standard error of the mean; *SD*, standard deviation; *PCV*, packed cell volume.

TABLE 18.4

Mean ± SD Plasma Biochemistry Values of 23 Red-Tailed Hawks *(Buteo jamaicensis)*, 1 Swainson's Hawk *(Buteo regalis)*, and 1 Ferruginous Hawk *(Buteo swainsoni)* With *Chlamydia buteonis*

Parameter	Mean ± SD	Range	Reference Interval[84]
Total protein (g/dL)	4.3 ± 2.06	0.4–8.4	2.8–4.8
Albumin (g/dL)	0.90 ± 0.39	0.2–1.5	
Globulin (g/dL)	3.4 ± 1.67	0.2–7.2	
Glucose (mg/dL)	280 ± 102	56–628	297–461
Uric acid (mg/dL)	14.4 ± 8.6	6–38.8	1.66–15.2
AST (IU/L)	488 ± 3035	199–15,650	160–495
CK (IU/L)	3386 ± 33,452	563–171,450	744–3230

AST, aspartate aminotransferase; *CK*, creatine kinase; *SD*, standard deviation.

CHLAMYDIA ABORTUS

Avian strains of *C. abortus*, originally detected during a 2014–2015 study in Poland, have recently been identified alongside the well-established ruminant strains primarily associated with ruminant abortions.[22] These avian strains share a close genetic resemblance to ruminant *C. abortus* strains but possess distinctive traits, notably a plasmid similar to those found in *C. psittaci* strains.

These avian *C. abortus* strains have been detected in cloacal swabs collected from various healthy wild bird species in Poland, including swans, mallard ducks (*Anas platyrhynchos*), Eurasian teals (*Anas crecca*), Eurasian coots (*Fulica atra*), magpies (*Pica pica*), and hooded crows (*Corvus cornix*).[22] They have also been found in crows in Italy[85] and cormorants (*Phalacrocorax* spp.) in the Galapagos islands. PCR tools have been developed to identify all *C. abortus* strains, encompassing both avian and classical ruminant strains. This discovery challenges the initial classification of some strains as *C. psittaci*.[86] For example, strains such as 84/2334, isolated from an Amazon parrot (*Amazona* spp.) in Germany, and Prk/Daruma, isolated from a parakeet in Japan, exhibit notable differences from classical[87] *C. psittaci* strains. These strains have been recently reclassified as avian *C. abortus* strains.

CANDIDATUS CHLAMYDIA IBIDIS

While investigations were being conducted in the wild fauna due to the significant presence of *C. psittaci* in duck farms in France, ibises observed in the areas visited by these ducks were sampled to assess their potential role in transmission and contamination of the ducks. This is how atypical strains, distinct from *C. psittaci*, were isolated alongside the typical strains of *C. psittaci* commonly found in ducks.[6] This study led to the isolation and description of strains later identified as *Ca. C. ibidis*. Notably, the study also indicated the potential for the risk to be reciprocal, suggesting that farmed animals could present a contamination threat to wild fauna as well.

Ca. C. ibidis was isolated from cloacal swabs from healthy feral African sacred ibises *(Threskiornis aethiopicus)* in western France.[6] More recently, 20.2% of fecal samples from crested ibis *(Nipponia nippon)*, an endangered species with a very limited population, were found positive for Chlamydiaceae DNA and were related to *Ca. C. ibidis*, with the co-existence of *C. psittaci* in one positive sample. In this study, the samples positive for *Ca. C. ibidis* displayed substantial genetic diversity, with only 77% identity for the *ompA* gene sequence when compared to those of the French 10-1398/6 *Ca. C. ibidis* strain.[4]

OTHER CHLAMYDIACEAE IN WILD BIRDS

The Agents

In wildlife, the first chlamydial infections were recorded in the 1930s, when juvenile fulmars (*Fulmarus* spp.) were hunted for food. Human infections from preparing and cooking the fulmars prompted a ban on their hunting for human consumption in the United States until 1954.[88] To date, most of the attention has been on wild birds acting as reservoirs for *C. psittaci*, with little consideration given for disease manifestation in wildlife species for this or other new chlamydial species.[89] However, there is evidence for the zoonotic and/or cross-species transmission potential of bacteria within the order Chlamydiales, with an increasing number of reports suggesting contact with wildlife and/or livestock is a risk factor for these infections.[9,11]

As such, Chlamydiaceae species in wildlife could have higher impact on human and animal health worldwide than currently recognized.[10,11] The discovery and description of novel species in avian hosts highlights how much we still must learn about the biology and pathogenicity of the Chlamydiaceae as a whole. Throughout the continued identification of novel chlamydial organisms there is growing evidence that some of these novel organisms may be pathogenic to birds, humans, or other animals, thereby posing new zoonotic risks.[11] The intensification of farming and the widespread popularity of companion animals also present ongoing opportunities for emerging zoonoses.[10,11]

Thus far, chlamydial infections have been identified in over 70 species of wild birds.[89] In addition to the more traditional hosts (most notably Columbiformes), birds from the Corvidae and Accipitridae families seem to be emerging as chlamydial hosts.[22,90–92] Other species in which Chlamydiaceae have been isolated include Anatidae (ducks and geese[22,34,93–96]), Rallidae (rails[95]), Alcidae (auks[97]), Laridae (gulls[97,98]), Sulidae (gannets and boobies[34]), Procellariidae (fulmars[99]), Gruiformes (cranes[94]), Threskiornithidae (ibises[100]), Phoenicopteriformes (flamingos[8]), Sphenisciformes (penguins[98]), Strigiformes (owls[90–92]), Apodidae (swifts[101]), and various species of Passeriformes (songbirds, e.g., Paridae [tits], Turdidae [thrushes[96,102–105]]). Aside from the traditional members of the genus *Chlamydia*, including *C. psittaci*,[78] *C. trachomatis*,[94] and avian strains of *C. abortus*,[106] newly identified Chlamydiales are also regularly discovered in wild birds. These novel species likely represent the "tip of the iceberg" in terms of diversity of *Chlamydiae* (and their hosts) present in wild birds.[97]

Prevalence estimates also greatly vary between studies, even if focusing on the same host lineages (see Stokes et al., 2021 for an overview[89]). For example, in a study conducted in Oregon, 3 out of 82 choanal and cloacal swabs from raptors (1.6%) from wild bird rehabilitation centers in Oregon were positive for chlamydial DNA using PCR,[75] suggesting that the carriage rate of *C. psittaci* in this region is low. However, in a Belgian wildlife facility, 26% (11/42) of pharyngeal swabs were culture positive in sampled wild birds, suggesting prevalence can vary greatly according to the region involved. Additionally, molecular characterization of the isolates from these birds revealed *C. psittaci* genotypes A, B, D, and E/B, reflecting multiple distinct infections and highlighting the endemic nature of *C. psittaci* in avian wildlife.[78] However, as the reported prevalence estimates in free-ranging birds are based on detection of Chlamydiaceae DNA in cloacal and fecal swab samples,[18,64,71,97] these rarely provide a definitive diagnosis of infection.[103]

Transmission, Clinical Signs, and Zoonotic Potential

There are few reports to date documenting transmission and clinical disease associated with chlamydial organisms in wild birds.[41,65,67,68,72,78,94,103,107] Similarly, little data is available on the influence of chlamydial infections on the reproductive success of wild birds, although one study in crimson rosellas *(Platycercus elegans)* found that breeding birds were less likely to be infected than nonbreeding birds,[108] suggesting that chlamydial infections may reduce the likelihood of breeding in this and potentially other species of wild birds as well. Some preliminary evidence also exists that in some wild bird populations (e.g., white-winged doves, *Zenaida asiatica*), young birds are more susceptible to *C. psittaci* infection than adult birds,[108,109] yet further studies are needed to confirm these findings.

Many *Chlamydia*-positive birds are believed to harbor chlamydial organisms without showing clinical signs. Nevertheless, there are some reports of wild birds showing clinical signs associated with chlamydiosis (e.g., emaciation, diarrhea, and, less commonly, respiratory signs), as well as reports on suspected epizootics in white-winged doves and various gull species in the United States,[109,110] which suggest that chlamydial disease can affect wild bird populations.[111] A postmortem study investigating tissues from wild bird mortalities in Great Britain that showed histopathologic lesions characteristic for chlamydial infection such as hepatitis were evaluated using *C. psittaci* PCR and ArrayTube microarray assays. Twenty-one of the 40 birds tested positive: 8 dunnocks *(Prunella modularis)*, 7 great tits *(Parus major)*, 3 blue tits *(Cyanistes caeruleus)*, 2 collared doves (*Streptopelia decaocto*, order Columbiformes), and 1 robin *(Erithacus rubecula)*.[103] Of the 21 *C. psittaci*–positive birds, 10 had histological lesions consistent with chlamydiosis and colocalizing *Chlamydia* spp. antigens on immunohistochemistry.[103] *C. psittaci* genotype A was identified in all positive passerines and in a further three dunnocks diagnosed with chlamydiosis from a previous study. Two collared doves had genotype E.

Zoonotic transmission of chlamydial infections is mostly thought to result from contact with infected captive birds, but there are also cases where infection is suspected to be contracted through direct or indirect contact with wild birds. While in most cases this seemed to involve feral pigeons,[112–115] other species have been implemented in human-transmitted psittacosis as well.[116,117] Close contact with or handling of wild birds is a common source of zoonotic infection, but in some cases, infections have been linked to cleaning of bird feeders or mowing of lawns[117–119] without a grass catcher. Suspected zoonotic transmission between a long-eared owl and volunteers at a rehabilitation center highlighted that wildlife rehabilitation workers were unaware that signs of chlamydiosis were similar to cold and flu symptoms.[78] These findings demonstrate that chlamydial infections in wild birds may be problematic for contact individuals, including rehabilitation center workers, wildlife biologists, banders, veterinarians, and falconers.

Diagnosis

As the majority of wild birds are not diagnosed antemortem; instead, diagnosis is often made from postmortem tissues using qPCR or in situ hybridization. Care must be taken in the design of PCR primers for the phylum *Chlamydiae* such that insect infestation is not mistaken for an infection by known chlamydial pathogens.[75] Immunohistochemistry can be utilized as a tool to diagnosis but in the United States, it is not considered highly diagnostic because of indiscriminate staining.[31] Culture can be used to identify chlamydial species from fresh tissues, if a willing laboratory can be found to perform the culture. Tissue qPCR has confirmed infection in a number of wild bird species.[69,94,103,107] If heart blood can be collected, the blood can be centrifuged and serology performed for IgM/IgY antibodies. Seroprevalence in wild birds has been identified from 1% to 63%.[70,120–122] While both IgM and

IgY chlamydial antibodies are differentiated serologically, the presence of one or both does not confirm active infection.

Treatment and Prevention

There are no reports to our knowledge of successful treatment of a wild bird documented with chlamydiosis. As the pathogenesis and zoonotic potential of the disease is mostly undefined in wild birds, methods of prevention should follow those outlined for *C. psittaci*.

Chlamydial infections of wildlife may pose a threat to conservation.[9] Perhaps the most well-known example of this is the devastating effect *C. pecorum* and *C. pneumoniae* have had on koala *(Phascolarctos cinereus)* populations in Australia[123] As *C. psittaci* can cause severe disease in highly susceptible hosts (e.g., immunocompromised individuals) or when exposed to additional stressors (e.g., human-induced habitat destruction) or concurrent infections (e.g., beak and feather disease virus [BFDV]), chlamydial infections may potentially result in population declines in wild birds.[89] However, the pathogenicity can differ greatly across species, whereby the strain may cause severe disease in one species but no clinical signs in another.[89] This poses additional concerns as a particular host may introduce a novel chlamydial strain in a naïve population or alternative host, thereby potentially inducing severe disease and species decline. Detailed studies are thus required to assess whether the new chlamydial species have the capacity to cause serious disease and/or zoonosis in wild birds and to be transmitted to humans or other animals.

AN OUTBREAK OF *CHLAMYDIA PSITTACI* IN SPHENISCIFORMES

The Agent

C. psittaci is a well-documented agent of disease in captive avian populations, but few reports of clinical disease associated with chlamydial species have been described in Sphenisciformes. Positive *Chlamydia* antibody titers have been documented in several wild species in Argentina, the Galapagos, and Antarctica.[98,124–126] In addition, a large survey of Antarctic seabirds and penguins showed that the prevalence of *C. psittaci* is low but another Chlamydiaceae-like bacterium is widespread.[98] These studies suggest that *Chlamydia* spp. may be wide-ranging in penguin species but are not known to commonly cause disease. The one confirmed outbreak of *C. psittaci* has been described in Magellanic penguins (*Spheniscus magellanicus*) and was successfully treated in a captive care facility in California.[127]

Transmission

The source of infection for the outbreak in Magellanic penguins was never discovered. The outbreak occurred in an outdoor flock with frequent trespasses by gulls and pigeons, which are documented carriers[128] and may have served as a source of exposure. Alternatively, wild caught birds or birds from other zoological institutions within the colony may have been latently infected and served as the source of infection. Banked serum samples from the effected colony were submitted for serology and showed positive antibody titers from samples taken up to 9 years earlier. Possible predisposing factors in this colony included overpopulation, as the colony was approximately 10 birds more populated than historical levels. The outbreak also occurred during breeding season, which may have added additional stress leading to emergence of latent infections.

An aggressive quarantine protocol was established to protect other species of birds in the collection and the caretakers. Zoonotic spread likely occurred in one human caretaker during this time, but diagnostics were not performed to confirm infection.

Clinical Signs

Initial clinical signs emerged in 14% of the colony and were nonspecific including acute lethargy, inappetence or anorexia, and green urates. As clinical signs were vague, aspergillosis or malaria, which had previously been diagnosed in the colony, were top differentials and birds were prophylactically treated as such. Three of these birds died before a diagnosis of avian chlamydiosis was made. During the outbreak and treatment period, four birds presented with seizures, of which two died. Fifty birds exhibited severe keratoconjunctivitis with blepharospasm (Figure 18.4A), serous to purulent conjunctival exudate, chemosis, and varying degrees of corneal edema while undergoing treatment. An additional five of these birds presented with dermatitis of the legs and dorsal feet in varying degrees of severity (Figure 18.4B,C). The dermatitis developed within days after completion of treatment with doxycycline. The keratoconjunctivitis and dermatitis of the legs and feet were attributed to light sensitivity secondary to doxycycline administration.

Diagnosis

A variety of diagnostic modalities were employed in the outbreak investigation. Clinical signs were nonspecific initially and *C. psittaci* was not high on the differential list when signs first appeared. Histopathology and immunohistochemical staining of mortalities were the first diagnostics to suggest *C. psittaci* infection and lesions observed were consistent with avian chlamydiosis. PCR of tissues confirmed the diagnosis. PCR of choanal-cloacal/fecal swabs and environmental swabs was most useful in determining presence of the bacteria and shedding in live birds. PCR of environmental and choanal-cloacal/fecal swabs was also used as a monitoring and follow-up tool. Fecal samples collected randomly remained positive for 2 weeks following the start of treatment after which they tested negative.

Blood work, conducted on nine individuals, showed nonspecific abnormalities including leukocytosis, heterophilia with toxic change, and hyperproteinemia. A moderate increase in AST and uric acid was observed in a subset of these birds. Rising or persistently elevated titers to *C. psittaci* were not observed in the colony in monthly serologic tests over a 3-month period.

Treatment and Prevention

Treatment with doxycycline at 100 mg/bird (17–33 mg/kg depending on the weight of the bird) PO q24h for a minimum of 30 days was effective in treating the disease. Initially, a dose increase to 200 mg/bird was attempted but a significant number of birds exhibited side effects and the dose was lowered again to 100 mg/bird. Loss of appetite was the most common side effect and often occurred within 5 days of starting oral



FIGURE 18.4 Clinical signs exhibited in Magellanic penguins (*Spheniscus magellanicus*) with avian chlamydiosis included keratoconjunctivitis with (A) blepharospasm and (B, C) dermatitis of the legs and dorsal feet in varying degrees of severity.

treatment. In those cases, oral medications were assist-fed or long-acting doxycycline injections 50 to 75 mg/kg IM q7d were given in alternating sites in combination with subcutaneous fluids. Some birds received combinations of injectable and oral doxycycline. Fluconazole was also given (100 mg PO q24h) as prophylaxis for fungal infections and was continued for 30 days after completion of doxycycline. Avian malarial prophylaxis with primaquine was maintained for the first 2 weeks of the treatment period.

Light sensitivity was a side effect of treatment with doxycycline and birds with dermatitis and ocular lesions received nonsteroidal antiinflammatory drugs (NSAIDs), either ketoprofen at 2 mg/kg IM PRN or carprofen 2 mg/kg PO q12h and zinc oxide topically. Topical ocular treatments included saline flush, followed by ophthalmic lubricant. Dermatitis resolved over an average of 3 to 4 weeks after beginning treatment. Anorexia and weight loss were also side effects of the treatment and in cases with pronounced weight loss, assist feeding oral doxycycline in fish was used to maintain caloric consumption. Behavioral side effects occurred as well, with birds spending time in unusual places or hiding more in dens. Abnormal swimming patterns occurred in a quarter of the birds. These were characterized by birds swimming in small circles with heads pointed to the sky and eyes held tightly shut. Behavioral side effects resolved with the completion of treatment.

Death due to *C. psittaci* only occurred prior to diagnosis and targeted treatment. Additional deaths occurred and were attributed to a variety of cofactors or complications from treatment. Other diagnoses included visceral gout, myocardial degeneration and insufficiency, and severe esophagitis leading to esophageal perforation and sepsis. Caution is recommended in pilling doxycycline and this medication should be given in a whole fish or gavage fed. The majority of birds recovered fully.

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CHAPTER 19

Erysipelas in Wild Birds

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INTRODUCTION

Erysipelas is the infectious disease caused by the opportunistic pathogen *Erysipelothrix rhusiopathiae* in a wide range of mammals, birds, some reptiles, and fish.[1,2] Erysipelas is a zoonotic disease and usually causes mild dermatitis in humans, but in rare cases can cause systemic disease and endocarditis.[3,4] Confusingly, in humans, the term erysipelas refers to skin infection due to *Streptococcus* spp.,[5] while cutaneous disease caused by *E. rhusiopathiae* in humans is known as erysipeloid.[6] In birds, erysipelas can be an acute, fulminating infection or, more rarely, a chronic infection of the reproductive tract causing infertility in male birds and reduced egg production in females.[1] However, many avian infections of *E. rhusiopathiae* are subclinical. It is possible that erysipelas in wild birds is underrecognized and underdiagnosed.[7] The disease has been reported as individual cases or in isolated clusters of cases in over 60 species of wild birds, including little blue penguins *(Eudyptula minor)*,[8] laughing kookaburras *(Dacelo novaeguineae)*,[9] rainbow lorikeets *(Trichoglossus moluccanus)*,[10] eclectus parrots *(Eclectus roratus)*,[10] and the endangered Hawaiian crow (*Corvus hawaiiensis*; Table 19.1).[11]

Mass mortality events associated with *E. rhusiopathiae* have been reported in some wild bird populations.[7] For example, an erysipelas outbreak in the Great Salt Lake in Utah, USA, killed more than 10,000 eared grebes *(Podiceps nigricollis)*. Similar events at the same location have also affected California gulls *(Larus californicus)*.[7] An estimated 600 brown pelicans died in another outbreak of erysipelas in California.[7] A mass mortality event due to *E. rhusiopathiae* killed little swifts *(Apus affinis)*, nesting in South Africa.[12] Erysipelas has also been recorded in Southern Ocean albatrosses and is suspected to have caused outbreaks of mortality in these birds in the mid-1980s.[13] The prevalence of *E. rhusiopathiae* in dead seabirds on New Zealand offshore islands used for intensive conservation varied from 3.4% to 11.4% and was most commonly found in sooty shearwaters (*Puffinus griseus*; 5.9%), mottled petrels (*Pterodroma inexpectata*; 2.7%), and Cook's petrels (*Pterodroma cookii*; 13.5%). These burrowing seabirds are possible reservoirs of *E. rhusiopathiae* and may be a source of spillover to other species managed on the islands.[14]

ETIOLOGY: *ERYSIPELOTHRIX RHUSIOPATHIAE*

E. rhusiopathiae is a gram-positive, rod-shaped bacillus, ubiquitous in nature, and both a commensal and pathogen in diverse species of animals, wild or domestic, from mammals and birds to reptiles and fish.[2] Transmission of *E. rhusiopathiae* is thought to be through the gastrointestinal tract by the intake of contaminated food or water or through breaks in the mucous membrane or skin.[1] Asymptomatic carriage of the organism in animals with subsequent dissemination into the environment and other animals has been suggested as an important mechanism for maintenance of *E. rhusiopathiae* in nature.[15] The organism may persist in aquatic and soil environments, meat products, and decomposing animal remains. The bacteria may also be spread through insect vectors, ticks, mites, and flies; however, the role of these vectors in the epidemiology of *E. rhusiopathiae* remains unclear. There is no evidence to support vertical or egg transmission of the bacterium. Control of infections is challenging because of the organism's ubiquitous nature in the environment and its ability to survive asymptomatically within reservoir hosts.

The genus *Erysipelothrix* consists of five species, and the differentiation of *Erysipelothrix* species was historically based on their serovar, which is determined by the cell wall peptidoglycan antigens. Serovars 1a, 1b, 2, 4 to 6, 8, 9, 11, 12, 15 to 17, 19, 21, 23, and N were characteristic of the only pathogenic species, *E. rhusiopathiae*.[16] Disease in pigs, poultry, and humans is mostly associated with *E. rhusiopathiae* serovars 1a, 1b, and 2. Serotypes 1b, 5, 6, 8, 14, and 21 have been isolated from *E. rhusiopathiae* isolates from captive native Australian birds.[17] Although serotyping is important as a diagnostic technique in diagnosing *E. rhusiopathiae* infection, the ability of horizontal exchange of capsule-specific genes in many bacterial species has reduced its use as a technique to detect evolutionary relatedness among isolates.[18,19] Recent genomic studies revealed that there is, as yet, no indication of geographic or host specificity of the *E. rhusiopathiae* isolates, suggesting that the mammals and birds are infected by phylogenetically similar strains either through a common reservoir or indirect transmission between animal orders.[20]

TABLE 19.1

A summary of published reports of the clinical and pathologic features of wild bird mortality due to *Erysipelothrix rhusiopathiae*

Avian Species	Clinical Signs	Gross Lesions	Means of Diagnosis	References
Little swifts *(Apus affinis)*	High mortality	Moderate splenomegaly	Bacterial culture from liver, spleen, and heart blood	12
Bald eagle *(Haliaeetus leucocephalus)*	Sudden death	Little subcutaneous fat and a slight reduction of the pectoral musculature, diffusely pale heart with light streaks. The atrioventricular valve leaflets were thickened with numerous vegetations (proliferative outgrowths) on their surfaces. Irregular cream-colored 1- to 2-mm foci were observed on the surface and within the parenchyma of the liver	Bacterial culture from liver	36
Hawaiian crow *(Corvus hawaiiensis)*	Found dead	Partially scavenged and loss of tissues, moderate atrophy of the left pectoralis, and the absence of abdominal and subcutaneous fat	Culture of the kidney	11
Indian yellow-nosed albatross *(Thalassarche carteri)*	Chick mortality, apparently healthy well-fed chicks died within a few minutes after convulsions	Not recorded	Necropsy, bacterial culture, histopathology	13
Little blue penguins *(Eudyptula minor)*	Anorexic for 1 day, found acutely recumbent and moribund the next day	Pulmonary hemorrhage, edema of the lungs and intestine and with clotted blood in bronchi, and mucosal petechiation of the proximal third of the small intestine with clotted blood in the lumen	Bacterial culture from liver, lungs, and intestine. Histopathology	8
Laughing kookaburras *(Dacelo novaeguineae)*	Weakness and weight loss over 2 weeks	Moderately atrophied pectoral muscle, edematous red lungs	Bacterial cultures of lung, liver, heart, and cloacal swabs. Pulsed-field gel electrophoresis (PFGE)	9
Kakapo *(Strigops habroptilus)*	Lower level of activity than commonly seen posttranslocation. Mortality within 1–4 days post translocation	Congested, slightly swollen and mottled liver and a congested and swollen spleen, variable degree of enteritis. All birds were in good body condition with good fat reserves and food still present in the crop	Clinical findings, gross necropsy changes, cytology, histopathology and bacterial culture	33
Takahe *(Porphyrio hochstetteri)*	Found dead	Not recorded	Bone marrow culture from the frozen carcasses and then PCR	35
Rainbow lorikeets *(Trichoglossus moluccanus)* and eclectus parrots *(Eclectus roratus)*	Died with no premonitory signs	Moderately atrophied adipose tissues, moderate to severe dehydration, severe hepatomegaly, a large area of dark red discoloration in the left ventricle, mild accumulations of yellowish transparent fluid in the coelomic and pericardial cavities, mild splenomegaly, and mild bilateral thyroid enlargement	Histopathology, Gram staining, bacterial culture and sequencing, immunohistochemistry, and real-time PCR	10
North Island brown kiwi *(Apteryx mantelli)*	Moderately swollen right hock joint for 1–2 weeks	Moderate body condition. Loosely adhered clumps of gelatinous dull yellow material in the reddened and thickened synovium of the hock joint. Multiple areas of dull yellowing, softening, and pitting of the articular cartilage from both the right distal tibiotarsus and proximal tarsometatarsus	Bacterial culture	Unpublished

PCR, Polymerase chain reaction.

Clinical Signs

With the exception of lethargy, there have been no specific clinical signs associated with *E. rhusiopathiae* infection in wild birds. Death of one or more birds in a flock is generally the first clinical sign in acute septicemia.[7] In chickens, the main clinical signs include general weakness, depression, diarrhea, and sudden death.[1]

Pathogenesis

The pathogenesis of erysipelas in wild birds or in animals in general is not well known, and while the organisms can be found in large numbers in endothelial cells and macrophages, there is often little host inflammation or cellular damage present. Capsule antigens, neuraminidase, surface adhesion molecules, hemolysins, and hyaluronidase are considered important in the pathogenicity of the organism. Neuraminidase releases sialic acid residues from glycoproteins, glycolipids, and oligosaccharides expressed on host cells leading to vascular damage and hyaline thrombus formation.[16] It has been found that the capsule is associated with resistance to phagocytosis and intracellular survival of the bacteria.[21] However, experimental studies have shown that hyaluronidase is not associated with the virulence of *E. rhusiopathiae*.[22]

Diagnosis of Erysipelas

Gross lesions of erysipelas include signs of septicemia, such as hepatomegaly and splenomegaly (Figure 19.1), similar to other infectious diseases in birds including avian chlamydiosis, fowl cholera, *Escherichia coli* infections, and salmonellosis. Most field cases of erysipelas have been diagnosed with necropsy examination including culture and identification of *E. rhusiopathiae*. Demonstration of the presence of clumps and segregated gram-positive, beaded, slender, and pleomorphic rods in smears prepared from liver, spleen, heart blood, or bone marrow provide rapid tentative diagnosis (Figure 19.2). The organism is more readily isolated from birds that died of the disease rather than ill birds that are killed for sampling. Sampling from multiple organs, including bone marrow, is necessary in order to detect the organism from carrier birds. Packer's medium, which contains sodium azide-crystal violet, and the tryptose phosphate broth medium with 5% horse serum plus kanamycin, neomycin, vancomycin, and novobiocin are two inhibitory culture media routinely used to isolate *E. rhusiopathiae*.[1]

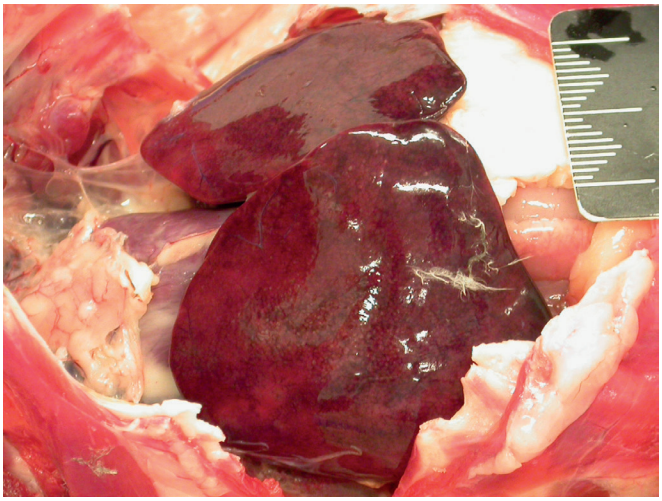


FIGURE 19.1 A postmortem image of the swollen liver of a young kakapo *(Strigops habroptilus)* that died suddenly in good condition due to erysipelas.

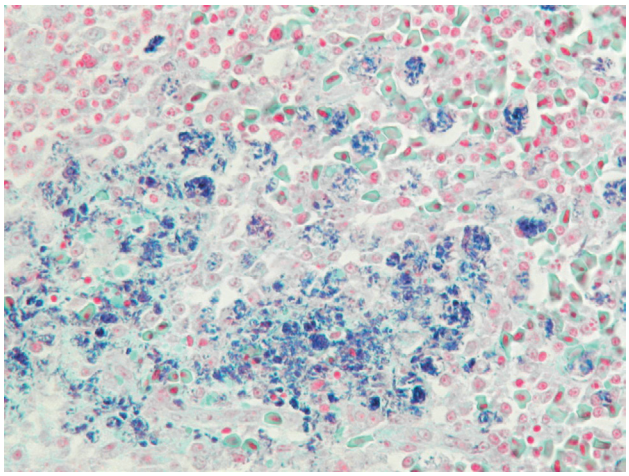


FIGURE 19.2 Gram-stained section of the spleen of a young kakapo *(Strigops habroptilus)* that died due to erysipelas (400× magnification). The aggregation of *Erysipelothrix rhusiopathiae* organisms is evident as blue staining material within macrophages and endothelial cells.

Morphological and biochemical identification of *Erysipelothrix* species colonies is based on Gram stain, cultural morphology, motility, hemolytic characteristics, and biochemical properties. Colonies are clear, circular, and very small, with a diameter of 0.1 to 0.5 mm after 24-hour incubation at 37°C or 0.5 to 1.5 mm after 48 hours. They may be either smooth colonies with bluish, transparent, and convex appearance or rough colonies that are larger and have a flat rough surface with irregular edges. Microscopically, smooth colonies contain small, slightly curved, slender rods with rounded ends, while rough colonies have long filaments rods up to 60 mm or more, in chains. Alterations in pH and incubation temperature can result in these morphological differences. *Erysipelothrix* species are relatively inactive biochemically and negative for catalase, oxidase, methyl red, indole, esculin, and nitrate. The majority of *Erysipelothrix* species produce hydrogen sulfide (H_2S) as demonstrated in triple sugar iron (TSI) agar. Acid is produced from glucose, fructose, galactose, and lactose but not from maltose, xylose, and mannitol. The ability of *E. tonsillarum* to ferment sucrose differentiates it from *E. rhusiopathiae*.[2]

Polymerase chain reaction (PCR) has been used to diagnose *E. rhusiopathiae* from tissue samples of swine, poultry, and other animal species. A PCR technique has also been used to diagnose *E. rhusiopathiae* using the DNA extracted from formalin-fixed, paraffin-embedded liver tissues[23] and for the rapid diagnosis of *E. rhusiopathiae* in chicken blood.[24] Restriction fragment length polymorphism analysis[25] and pulsed-field gel electrophoresis[26] are two types of molecular methods alternative to PCR and DNA sequencing that have been used to diagnose and characterize *E. rhusiopathiae* isolates. In addition, the use of MALDI-TOF-MS (matrix-assisted laser desorption/ionization with time-of-flight mass spectrometry) for diagnosis of *E. rhusiopathiae* in tissue samples, blood, and tissue

fluids has been demonstrated recently.[27–29] For the confirmation of diagnosis in a postmortem investigation any of these techniques may be sufficient. However, for the investigation of the mortality clusters of erysipelas in wild birds, culture of *E. rhusiopathiae* from affected individuals with subsequent confirmation by PCR and whole-genome sequencing is recommended. The best way for us to understand the epidemiology of erysipelas is by phylogenetic comparison of field strains with published sequences.

Treatment and Control

Treatment for erysipelas in wild birds has not been formally reported due to the acute nature of the disease and most of the affected birds were found dead. However, *E. rhusiopathiae* is susceptible for many antibiotics such as penicillin, cephalosporin, clindamycin, and the fluoroquinolones.[1,7] At the time of a cluster of mortality in kakapo *(Strigops habroptilus)*[30] the remaining birds were captured, placed in temporary pens, and treated with amoxicillin and clavulanate at 125 mg/kg PO bid for 5 days. There were no further deaths once this treatment commenced, but it is uncertain if that was a result of treatment (B. Gartrell, personal communication, 2021). During an outbreak in domestic poultry, it has been recommended to treat and vaccinate all the birds in a flock.[1,30] Treatment of choice is a rapid-acting form of penicillin given intramuscularly. Penicillin can be administered via drinking water for 4 to 5 days to control an infection when a presumptive diagnosis has been made.[1] Prompt removal of carcasses and incineration has been recommended to control an infection during an outbreak.[1] In addition, adequate sanitation, stress reduction, and health monitoring are important in controlling and eradicating the diseases in captive birds.

Prevention of erysipelas in intensively managed wild populations can be achieved through vaccination[30,31] and by reducing management actions during harsh seasonal periods, for example, not translocating susceptible birds in winter.[30,31] However, for most wild bird populations, control of erysipelas is not a feasible option.

Epidemiology of Erysipelas

The prevalence of erysipelas among poultry or wild birds is not well understood. Two studies have reported the prevalence of antibodies to *E. rhusiopathiae* in domestic poultry as 5.5% (200 samples) and 39.8% (545 samples).[32,33] Stephenson and Berman (1978) estimated that 30% to 50% of healthy swine harbor *E. rhusiopathiae* in their tonsils making them the most important reservoir for other domestic livestock. Infected pigs can transmit the organism indirectly by contaminating soil, bedding, and water.[34] However, the reservoirs and host species in wild ecosystems are far less well studied; *Erysipelothrix* spp. has been isolated from the cutaneous slime of both fresh and saltwater fish and can persist long periods in marine locations.[2] In addition, these bacteria may persist on insects, mollusks, and crustaceans.[2,16] Rodents can be infected and have been suggested as a possible source of infection for other animals. It is believed that more than 30 species of wild birds can harbor the organism and act as reservoirs in natural ecosystems. The source of infection is often not identified in many erysipelas outbreaks. Stress or various other factors that weaken the host defense mechanisms may lead to clinical disease in *E. rhusiopathiae* carriers. In wild birds, extreme weather conditions during winter are suspected to increase susceptibility to infection with *E. rhusiopathiae*.[7]

Effects of Erysipelas on the Conservation of Wild Birds

Erysipelas is a sporadic cause of mortality in a range of species, but clusters of mortality have been reported in a number of seabirds and erysipelas may be of special concern for these birds. *E. rhusiopathiae* has been identified as a potential cause of chick mortality causing population decline in yellow-nosed albatross *(Thalassarche carteri)* in Amsterdam Island. The presence of this bacterium has also been detected in oropharyngeal swabs of Amsterdam albatross *(Diomedea amsterdamensis)* and sooty albatross (*Phoebetria fusca*) and in cloacal swabs of Northern rockhopper penguin *(Eudyptes moseleyi)* through PCR.[35]

E. rhusiopathiae has killed endangered New Zealand fauna on offshore islands used for conservation purposes. The species affected have included kakapo, takahe *(Porphyrio hochstetteri)*, and kiwi (*Apteryx* spp.). An outbreak of erysipelas affected the success of translocation of group of juvenile kakapo from Codfish Island to Chalky Island killing 3 of the 19 birds translocated,[30] and subsequent studies have shown that most kakapo living on offshore islands have antibodies to *E. rhusiopathiae*.[31] A vaccination program is in place for kakapo and takahe using a commercial killed bacterin for turkeys,[31] but this was controversial among conservation managers who were concerned about the possible side effects of vaccination. Initially, the kakapo team vaccinated all remaining birds,[30] but management has subsequently decided that only juvenile birds will be vaccinated, based on antibody studies that show older birds are exposed naturally.[31] Unfortunately, there has been a subsequent death due to erysipelas of an old male kakapo who was not vaccinated in the preceding 2 years (B. Gartrell, personal communication, 2021).

The formalin-inactivated, aluminum hydroxide-adsorbed *E. rhusiopathiae* bacterins are the most used vaccine types for the immunization of turkeys *(Meleagris gallapavo)* against erysipelas and have been used to good effect in takahe and kakapo in New Zealand.[31] These bacterins may contain either whole or lysed bacterial cells. Two vaccine doses have been recommended for turkeys at 4-week intervals. The first dose can be administered at 16 to 20 weeks of age. Live erysipelas vaccines have been used for swine over 50 years and now becoming available for turkeys as well.[1]

Our knowledge on the effect of this disease on the conservation of other free-living wild bird populations is limited due to the opportunistic nature of the pathogen and the problems associated with detecting the disease in those populations. It is believed that some environmental factors predispose avian species to *E. rhusiopathiae*. However, it is not fully understood and outbreaks are related to rainy, cold weather in many species.[1,30] Feeding on contaminated feed, soil, or decaying matter has been considered as the source of infection for some outbreaks in wild birds.[8,12] Immunity to this pathogen in wild birds is not studied, and it has been suggested that young and stressed birds are more likely to be infected than older or less stressed individuals in domestic species.[7] There is a need for epidemiological studies of this disease that go beyond identifying erysipelas as a cause of mortality. In the southern hemisphere at least, this research should be prioritized for endangered seabird species.

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CHAPTER 20

Mycoplasmosis

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Mycoplasma spp. are bacteria that can affect a wide variety of avian species. Most documentation of *Mycoplasma* infections in birds have been reported in poultry, psittacines, and raptorial species due to their economic and/or human-animal bond connection. While the infection is usually subclinical in most birds, it can be exacerbated with concurrent infections. The lack of cell wall and fragile nature of this bacteria necessitates the need for close bird-to-bird contact. This organism is susceptible to most disinfectants as the bacteria does not survive long in the environment. Moreover, *Mycoplasma* is not affected by those antibiotics, which disrupt cell wall development since it lacks a cell wall. As a pathogen, due to its poor survival in the environment, one of its strategies is to be transmitted vertically. Hence, infection is spread from hen to chick besides direct horizontal infections.

MYCOPLASMA IN POULTRY

There are four species of *Mycoplasma (M.)* that are pathogenic to poultry: *M. gallisepticum*, *M. synoviae*, *M. meleagridis*, and *M. iowae*. These species generally affect the respiratory system (Figure 20.1), but other bodily systems can also be affected.[1–3]

M. gallisepticum infection (also known as chronic respiratory disease [CRD] or infectious sinusitis in turkeys [*Meleagris gallopavo*]) causes respiratory illness primarily in chickens (Gallus gallus domesticus) and turkeys. Infection has also been reported in other poultry species including pheasants, partridges, quail, peafowl, guinea fowl, ducks, and geese.[1] Clinical signs in affected birds include coughing, sneezing, rales, snicks, and discharges from the eyes and nares. Affected turkeys have a swollen infraorbital sinus (Figures 20.2 and 20.3). Signs can be more severe if there is concurrent respiratory disease, poor management conditions, and/or stress placed on the birds.[1–3] Mortality can be increased in severe cases. Necropsy of affected birds often reveals thickened air sacs with occasional caseated debris, pneumonia (Figure 20.4), and salpingitis.[1–3]

Mycoplasma synoviae infection (also known as infectious synovitis) often occurs subclinically in poultry. However, the infection can become systemic and result in lameness due to inflammation of the synovial membranes. Often, there is caseated exudate within the joint, especially the hock joint, and tendon sheath. Chickens are most often affected. Mortality is minimal unless there are other diseases present and/or if management conditions are poor.

While *M. gallisepticum* and *M. synoviae* are the primary pathogenic species involved in chickens and turkeys, the two remaining pathogenic species, *M. meleagridis* and *M. iowae*, affect turkeys. These two *Mycoplasma* species tend to have a greater impact on the reproductive system. *M. meleagridis* infection is primarily associated with poor hatchability, often occurring late in the incubation period. Moreover, airsacculitis can be observed in newly hatched turkey poults, as early as day 1 of the hatch. Affected poults may show severe respiratory signs and/or sinusitis. In addition, skeletal abnormalities have been reported, often with osteomyelitis in the cervical vertebrae. This can lead to deformation and a crooked neck in some poults. Other birds may have leg deformities as a result of chondrodystrophy. There may also be a unilateral or bilateral varus deformity.[1,2]

M. iowae causes a venereal disease in turkeys. Affected adults rarely display any clinical signs but will have reduced reproductive performance due to poor hatchability and late embryonic mortality.[1,2]

Since *Mycoplasma* spp. are vertically transmitted, disease monitoring in the commercial poultry industry often includes



FIGURE 20.1 *Mycoplasma gallisepticum* conjunctivitis and infraorbital sinusitis in a chicken *(Gallus gallus domesticus)*. (Photo courtesy Robert E. Porter, Jr.)



FIGURE 20.2 Turkey *(Meleagris gallopavo)* with infraorbital sinusitis from *Mycoplasma gallisepticum*. (Photo courtesy Robert E. Porter, Jr.)



FIGURE 20.3 Open infraorbital sinus in a turkey *(Meleagris gallopavo)* demonstrating characteristic seromucoid exudate from a *Mycoplasma gallisepticum* infection. (Photo courtesy Robert E. Porter, Jr.)

testing whereby infected breeders are removed to maintain *Mycoplasma*-free flocks.[3] However, among backyard poultry and small poultry flocks, *Mycoplasma* spp. may be highly prevalent. On-farm biosecurity, preventing other concurrent respiratory diseases, maintaining good management practices, and reducing stress can help to minimize the impacts of *Mycoplasma* in these flocks.[3]

MYCOPLASMA IN CAPTIVE BIRDS

Mycoplasma spp. may also play a role as a causative agent in respiratory disease in captive birds. In companion birds, cases of mycoplasmosis in psittacines and finches are described, especially when there is close contact between these bird species and backyard poultry.[4] Therefore biosecurity



FIGURE 20.4 Pneumonic lung from a turkey *(Meleagris gallopavo)* with *Mycoplasma gallisepticum* isolated. (Photo courtesy Robert E. Porter, Jr.)

and hygienic measures should be kept in mind when keeping these bird species within one household. In pigeons (*Columba livia*), *Mycoplasma* spp. were long suspected to be the cause of severe respiratory disease. However, *M. columbinum*, *M. columborale*, and *M. columbinasale* are also found in healthy individuals. Hence, these organisms seem to be part of the commensal bacterial flora in pigeons.[5,6] In cases of mycoplasmosis, clinical signs are not pathognomonic since infection is often associated with other bacterial pathogens. Generalized respiratory signs such as rhinitis, tracheitis, swollen infraorbital sinuses, airsacculitis, and bronchopneumonia, as well as serous conjunctivitis and blepharitis, may be seen (Figures 20.1). The latter may lead to starvation due to blindness especially in smaller bird species like finches.[7]

In birds of prey, *Mycoplasma* spp. were first thought to be obligatory pathogens as they were isolated from different individuals showing respiratory signs due to tracheitis, airsacculitis, and pneumonia.[8,9] Subsequently, Poveda et al. (1994)[10] described novel *Mycoplasma* spp. isolated from different birds of prey species: *M. buteonis* isolated from a buzzard *(Buteo buteo)*, *M. falconis* isolated from a saker falcon *(Falco cherrug)*, and *M. gypis* isolated from a griffon vulture *(Gyps fulvus)*. However, as Morishita et al.[11] and Lierz et al.[12] isolated these species regularly from the respiratory tract of clinically healthy individuals, these species were no longer interpreted as obligate pathogens but to be more likely part of the physiological bacterial flora of these bird species. However, as some *Mycoplasma* spp. were found to be associated with pathological alterations, their role as a pathogen needs to be further investigated (e.g., *M. corogypsi* found in a bumble foot lesion of a black vulture [*Coragyps atratus*][13] or *M. vulturii* from dead found white-backed vultures [*Gyps bengalensis*][14]).

MYCOPLASMA IN WILD BIRDS

The knowledge on the occurrence of *Mycoplasma* spp. in different wild birds is limited. In some bird species *Mycoplasma* spp. are regularly found in healthy individuals, hence, considered apathogenic. However, as *Mycoplasma* spp. are absent in healthy individuals of some other wild bird species, they might play a role as a respiratory pathogen in these bird species.[15]

There are two main aspects, which are important regarding the occurrence of mycoplasmas in wild birds. On the one hand, wild birds may play a role as potential reservoir or vector for mycoplasmas infecting poultry and humans. On the other hand, mycoplasmas may represent pathogens for wild birds, potentially leading to population declines.

Wild birds are direct and indirect vectors for multiple pathogens, which are transferred to farm animals and humans. Migratory birds can spread pathogens over long distances.[16] In particular, *M. gallisepticum* can be transferred by wild birds between poultry farms.[17] *M. gallisepticum* has been found in various wild bird species, including wild turkeys,[18] thin-billed murre *(Uria aalge)*,[19] mallards *(Anas platyrhynchos)*, grey harrier *(Ardea cinerea)*, wood pigeon *(Columba palumbus)*,[20] peregrine falcon *(Falco peregrinus)*,[21] and various passerines (Passeriformes).[22] Meanwhile, passerine birds and corvids, which are regularly found nearby poultry farms, are classified as transient carriers of *M. gallisepticum*.[23] In addition, *M. gallisepticum* was found to be responsible for severe disease outbreaks in house finches (*Haemorhous mexicanus*) in North America, firstly reported in 1994.[24,25] Infected birds often suffer from conjunctivitis and may subsequently die due to starvation, predation, or trauma.[7]

Systematic studies investigating the occurrence and role of mycoplasmas in wild birds are rare.[26,27] Due to the ease of collection, many studies include collection of samples from injured or diseased birds. However, the significance of such studies needs to be discussed carefully, as preexisting health conditions may increase the possibility of disease or accidents occurring in individual birds.[28] Wild birds being kept in rehabilitation centers are stressed too, so that an immunosuppressive effect also needs to be considered.[29] Furthermore, these injured and diseased wild birds are preselected, therefore not reflecting a true representation of the free-ranging population and its microbial flora. In a study investigating the occurrence of mycoplasmas in corvids, two groups of birds were sampled: (1) individuals originating from hunting bags (group 1) and (2) juveniles or injured birds that were presented in a veterinary clinic (group 1). In sum, 7% of the healthy, adult birds from group 1 versus 32% of the birds presented in the veterinary clinic were positive for *M. sturni*.[30] These findings support the assumption of bias introduced by the chosen selection method and warrant cautious interpretation of study results. Moreover, the pathogenic relevance of *Mycoplasma* spp. isolated from diseased wild birds remains uncertain. To be able to interpret their relevance, studies on the prevalence of *Mycoplasma* spp. in healthy individuals from free-ranging populations are therefore highly needed.

Summarizing several studies focusing on mycoplasmas in wild birds, there are clear differences in the occurrence of mycoplasmas in the physiological flora in the respiratory tract. While *M. buteonis*, *M. gypis*, and *M. falconis* are regularly found in the respiratory tract of birds of prey, there were no mycoplasmas found in clinically healthy tits and nightingales.[31] To date, 28 avian *Mycoplasma* spp. have been described (Table 20.1), and new species are isolated on a regular basis.

TABLE 20.1

Twenty-eight Validly Described Avian *Mycoplasma* species (Published in and/or Approved by the *International Journal of Systematic and Evolutionary Microbiology*)

Mycoplasma Species	Host Species, Other Affected Species	Clinical Signs
M. anatis	Ducks[a]	Sinusitis, airsacculitis, pneumonia, central nervous signs in adult ducks; decreased clutch size and hatching rate, weak chicks; also found in clinically healthy wild duck species[32]
M. anseris	Geese[a]	Inflammation of the cloaca and the phallus[33]; also found in clinically healthy geese[34]
M. anserisalpingitidis	Geese[a]	Inflammation of the phallus[35]
M. buteonis	Accipitrids,[a] falcons	Found in clinically healthy birds of prey[36]; also found in birds of prey showing respiratory signs[11,37]
M. ciconiae	White storks (*Ciconia ciconia*)[a]	Found in clinically healthy nestlings[38]
M. cloacale	Galliforms,[a] turkeys (*Meleagris gallopavo*), waterfowl, songbirds	Found in clinically healthy chicken[39]; also found in clinically healthy turkeys, ducks, geese, and song birds[34,40,41]; inflammation of the cloaca and the phallus in geese[33]
M. columbinasale	Pigeons (*Columba livia*)[a]	Found in clinically healthy pigeons[39]; also found in pigeons showing respiratory signs[42]
M. columbinum	Pigeons[a]	Found in clinically healthy pigeons[39]; also found in pigeons showing respiratory signs and conjunctivitis[43]
M. columborale	Pigeons,[a] crows, peregrine falcon	Found in clinically healthy pigeons[39]; also found in pigeons showing respiratory signs and conjunctivitis[44]; also found in crows and a peregrine falcon showing pneumonia[21,45]
M. corogypsi	Falcons,[a] vulture	Found in clinically healthy falcons[46]; also found in a vulture showing pododermatitis, polyarthritis and tendovaginitis[47]
M. falconis	Falcons[a]	Found in clinically healthy falcons[11,12]; also found in falcons showing sinusitis[21]
M. gallinaceum	Galliforms,[a] turkeys, ducks, peregrine falcon, hoopoe	Found in clinically healthy chicken[39]; also found in chicken showing tracheitis and sinusitis; also found in clinically healthy wild turkeys[48]; also found in ducks and a peregrine falcon showing respiratory signs[49]; also found in a hoopoe showing central nervous signs[21]

TABLE 20.1

Twenty-eight Validly Described Avian *Mycoplasma* species (Published in and/or Approved by the *International Journal of Systematic and Evolutionary Microbiology*)—cont'd

Mycoplasma Species	Host Species, Other Affected Species	Clinical Signs
M. gallinarum	Galliforms,[a] turkeys, waterfowl, crows, peregrine falcon	Found in clinically healthy chicken, turkeys, ducks, and geese[39]; also found in crows and a peregrine falcon showing respiratory signs[21]
M. gallisepticum	Galliforms,[a] turkeys, passerines, crows, peregrine falcon	Chronical respiratory signs, decreased laying and hatching rate, synovitis and arthritis in chicken[50]; found in clinically healthy wild turkeys[18], also found in turkeys showing respiratory signs, sinusitis and conjunctivitis; respiratory signs, sinusitis, conjunctivitis and central nervous signs in passerines, crows, peregrine falcons[37,51]
M. gallopavonis	Turkeys,[a] crows	Found in clinically healthy wild turkeys[52]; also found in turkeys showing airsacculitis[53]; also found in crows showing respiratory disease[45]
M. glycophilum	Galliforms[a]	Found in clinically healthy chicken; also found in chicken showing decrease in laying, increased embryonal mortality; also found in pheasants showing respiratory signs[39,54,55]
M. gypis	Accipitrids,[a] falcons, vultures	Found in clinically healthy birds of prey; also found in vultures showing respiratory signs[12,21]
M. hafezii	Falcons[a]	Found in clinically healthy falcons[56]
M. imitans	Water fowl[a]	Found in ducks and geese showing respiratory disease[57]
M. iners	Galliforms,[a] pigeons, peregrine falcon	Found in clinically healthy chicken[39]; also found in pheasants, partridge, pigeons, and a peregrine falcon showing respiratory signs[37,58]
M. iowae	Turkeys,[a] chicken	Increased embryonic death, dwarfism, chondrodystrophy, edemas, feather disorders and airsacculitis in poults; increased embryonic death, skeletal deformations, and malformations of chicks[59,60]
M. lipofaciens	Galliforms,[a] turkeys, ducks, geese, Eurasian goshawk (*Accipiter gentilis*)	Found in clinically healthy chicken, turkeys, and ducks[39]; also found in an infertile egg of a Eurasian goshawk (increased embryonic mortality in chicken and turkey after experimental infection)[61,62]
M. meleagridis	Turkeys[a]	Found in clinically healthy turkeys and turkeys showing airsacculitis; increased embryonic death, skeletal deformations, malformations, and immunosuppression in poults[63,64]
M. nasistruthionis[b]	Ostriches (*Struthio camelus*)	Respiratory signs in ostriches[65]
M. neophronis	Vultures[a]	Found in dead found vultures without specific alterations[66]
M. pullorum	Galliforms,[a] pigeons	Found in clinically healthy chicken and pigeons; also found in pheasants and partridges showing respiratory signs[39,58]
M. seminis[b]	Hierofalcons	Found in the respiratory tract and semen of clinically healthy hierofalcons[67]
M. sphenisci[b]	African or Jackass penguin (*Spheniscus demersus*)	Found in jackass penguins showing respiratory signs[68]
M. struthionis[b]	Ostriches	Found in ostriches showing respiratory signs[65]
M. sturni	Songbirds,[a] cliff swallows (*Petrochelidon pyrrhonota*)	Found in clinically healthy songbirds (crows, fieldfares, and starlings)[30]; also found in other songbird species and cliff swallows showing rhinitis, sinusitis, and conjunctivitis[51,69,70]
M. synoviae	Galliforms,[a] turkeys	Synovitis, tendovaginitis, airsacculitis in broilers, egg-pole shell defects or without clinical signs in laying hens[71]; synovitis and tendovaginitis in turkeys[72]
M. tullyi	Humboldt penguin (*Spheniscus humboldti*)	Found in clinically healthy Humboldt penguins[73]
M. vulturii[b]	White-backed vulture (*Gyps africanus*)	Found in a white-backed vulture dying from intoxication (showing mild inflammation in its respiratory tract)[14]

[a]Host species.
[b]Some species are also described in the literature (published in other journals and not approved by *IJSEM* yet).

Diagnostics

The gold standard for diagnosing the presence of *Mycoplasma* spp. is cultivation. Due to the small genome size and associated limited biosynthetic potential, *Mycoplasma* spp. are strongly dependent on their metabolism. Therefore cultivation media need to provide all components for growth. The composition of the fluid and solid media, as well as the general procedure for the cultivation of avian mycoplasmas, is well described by Hayflick[74] and Bradbury.[75] The main advantage of cultivation is the possibility of differentiating between multiple *Mycoplasma* species within one sample. After performing several single-colony subcultures, each isolate can be further investigated by different diagnostic methods (e.g., molecular biological, antigenetic, or biochemical tests). Due to different growth rates, some slowly growing *Mycoplasma* species may easily get overgrown in mixed infections. For example, in samples collected from chickens, pathogenic species such as *M. gallisepticum* and

M. synoviae may become overgrown by commensal species like *M. gallinarum*.[76] This impedes the possibility of detecting these pathogenic *Mycoplasma* species via culture. Other advantages include the possible isolation of novel, undescribed *Mycoplasma* spp. and the ability to perform antibiotic sensitivity tests, which is only possible following the culture of the mycoplasmal organisms.[77] Newer diagnostic tools allow the differentiation of *Mycoplasma* spp. within a few hours. The MALDI-TOF MS (matrix-assisted laser desorption/ionization with time-of-flight mass spectrometry) is currently established for the common avian *Mycoplasma* species. However, this diagnostic depends on the successful culturing process as colony material is needed for testing. Furthermore, colonies need to be pure cultures as this may result in inaccurate species-level identification. Nevertheless, MALDI-TOF MS is a promising tool for reliable species identification of avian *Mycoplasma* species.[78,79]

Besides a broad range of biochemical tests focusing on different metabolic characteristics of *Mycoplasma* spp., antigenetic test methods can also aid in the differentiation of *Mycoplasma* spp. Such tests include the immunobinding assay, growth inhibition test, and immunofluorescence test. However, cross-reactivity may occur between some *Mycoplasma* spp.[80] Therefore monoclonal antibody tests such as the enzyme-linked immunosorbent assay (ELISA) test are regularly used, as these provide a higher specificity.[81]

Currently, molecular biological methods are also becoming increasingly relevant as test methods, as these tests provide a reliable differentiation of *Mycoplasma* spp. based on multiple gene segments. The 16S rRNA gene is used as *Mycoplasma* genus-specific polymerase chain reaction (PCR) protocol,[82] while sequencing the 16S-23S intergenetic transcribed spacer region (ISR) allows distinction between each *Mycoplasma* spp.[83] Moreover, gene segments such as RNA polymerase beta subunit (rpoB) and RpoB protein sequences may be used, as well as whole-genome sequencing.[84] There are multiple PCR protocols detecting specific *Mycoplasma* species (e.g., *M. gallisepticum*–specific PCR[85]). For the detection of *Mycoplasma* spp. in companion birds, this may be the first-line diagnostic tool. Relying on the bird species affected, the *Mycoplasma* species should be differentiated in case of a positive result by the sequencing of the amplicon and the subsequent comparison with the help of reference databases.[86] Diagnostic tools like amplified fragment length polymorphism (AFLP) or restriction fragment length polymorphism (RFLP) are based on specific restriction enzymes digesting DNA samples in fragments, producing a specific restriction fragment pattern through gel electrophoresis. Each of these methods can aid in the typing of different strains of *Mycoplasma* spp.

Serological tests are the most common diagnostic tool used in poultry due to their efficiency and manageable costs for larger production flocks.[87] With these diagnostic tests, antibodies against a specific *Mycoplasma* spp. can be detected. Hemagglutination inhibition test, antibody-ELISA, and serum agglutination tests are commercially available for the detection of antibodies against pathogenic *Mycoplasma* spp. such as *M. gallisepticum*, *M. synoviae*, *M. meleagridis*, and *M. iowae*. Serum agglutination provides rapid results, but due to the possibility of cross-reactions (leading to false-positive results), positive results need to be confirmed with additional tests such as species-specific PCR testing, respectively.[88]

In wild birds, the detection of *Mycoplasma* spp. should be interpreted with caution, even if these birds are showing clinical signs suggestive of mycoplasmosis and *Mycoplasma* spp. are isolated upon performing further diagnostics. Unidentified *Mycoplasma* spp. could represent commensal bacteria found widely in association with mucous membranes. Therefore differentiation of *Mycoplasma* spp. is considered essential for understanding their role as a pathogen in a single bird species.

Treatment and Control

Since *Mycoplasma* spp. can both be spread horizontally and transmitted vertically from infected hen to chick, the primary method for the control in poultry is to start a new flock with *Mycoplasma*-free birds. This implies obtaining hatching eggs and/or chicks from a *Mycoplasma*-free breeder hen to obtain *Mycoplasma*-free progeny. Poultry should be obtained from a hatchery and/or breeder flock that participates in a program such as the National Poultry Improvement Plan (NPIP) in the United States or similar international program that routinely monitors for the presence of *Mycoplasma* sp. in poultry flocks through serological testing and culture.

If a poultry flock is found to be positive for the pathogenic species of *Mycoplasma*, including *M. gallisepticum*, *M. synoviae*, and *M. meleagridis* or *M. iowae*, there are three options available to move forward: depopulation, vaccination, or no action (expectative). In a commercial poultry operation, depopulation of the infected flock is the option of choice, especially in all-in/all-out production management systems. If clinical signs are nonexistent to mild, birds may be sent to the slaughterhouse and/or further processed instead of treated through the administration of medication. In commercial poultry flocks that keep birds of multiple ages on the premises (e.g., commercial laying flocks), vaccination of replacement pullets prior to introducing them to the *Mycoplasma*-infected layer flock is acceptable in certain US states. For poultry, vaccination can be accomplished via spray or eyedrop method during the growth phase and prior to placing them in the layer houses. However, one should establish first whether there are state, regional, provincial, or national laws that prohibit vaccine usage.

While eradication and/or vaccination are exercised in commercial flocks, in backyard poultry, hatching eggs, chicks, and adult birds of different ages may be obtained from a variety of non-NPIP (United States Department of Agriculture) sources. Many owners prefer not to take any action and will simply allow the birds to live with the infection in their backyard flocks. Under these circumstances, it is important that flock owners maintain proper management and ensure proper environmental conditions. This includes the reduction of the amount of dust and ammonia in the enclosure through adequate ventilation and the routine removal of moist and caked litter from the floors as these can all lead to irritation of the respiratory tract hence increasing the risk of the birds developing a clinical *Mycoplasma* infection. If a backyard flock owner wishes to maintain a specific genetic line (e.g., in case of exhibition poultry), vaccination practices such as those employed in the commercial industry may need to be utilized.

Since mycoplasmas have no cell walls, they do not survive in the environment for extended periods of time. Hence, routine cleaning and disinfection should help to eliminate the bacterium from the farm premises. If possible, the building should

be kept isolated and devoid of birds for a minimum of 2 weeks following disinfection before the location is repopulated.

Treatment of *Mycoplasma* infections can be attempted but will generally not result in clearance of the infection. Relapses are common. If treatment is attempted, this usually comprises the use of tylosin or tetracycline, delivered via drinking water. As with any medication usage in poultry, caution is warranted as withdrawal times need to be adhered, especially if the bird and/or eggs are to be used for human consumption (http://www.usfarad.org/poultry.html).

For *Mycoplasma* spp. that cause venereal disease, specifically *M. meleagridis* and *M. iowae* in turkeys, medications such as lincomycin or spectinomycin, delivered via the drinking water, can be used to control the infection. However, long-term use of antibiotics can lead to increased antimicrobial resistance (see also Chapters 6 and 7). Dipping of hatching eggs in an antibiotic solution has been used in the commercial turkey industry, but this method does not seem practical in backyard flock situations unless the flock is a strain or variety from a specific genetic breeding line that the backyard flock owner wishes to continue to perpetuate (e.g., in case of exhibition and ornamental poultry).

For the treatment of individual companion birds or small flocks with respiratory disease (e.g., pet birds or small flocks), systemic antibiotic treatment (direct per os or through drinking water) using tetracyclines or gyrase inhibitors may be considered. In addition, antibiotic-containing ophthalmic preparations (ointment or drops) can be administered topically in case of conjunctivitis. Treatment of wild birds in rehabilitation centers is not advised, as treatment will not clear the infection and only pose a risk for these birds to become permanent shedders of the bacteria when released back into the wild.

Public Health and Zoonotic Potential of *Mycoplasma* spp.

Avian mycoplasmas are generally not known to cause disease in humans. However, as there is one report of the accidental airborne transmission of *M. lipofaciens* (strain ML64) causing mild respiratory signs (rhinitis), transmission of avian mycoplasmas to humans needs to be considered in some *Mycoplasma* species.[89]

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CHAPTER 21

Cryptococcosis

Anne E. McDonald

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INTRODUCTION

Fungal diseases are anything but dull. Fungi are innovative, tenacious, and motivated. They are masters of genetically driven change. Fortunately, comparatively very few are animal pathogens, but the genus *Cryptococcus* is one of those.[1] It is an ancient, highly adaptive, invasive fungus whose genome dates back millions of years. It is a formidable pathogen in the arena of infectious disease. It is the Harry Potter of contagious disease, complete with a multifunctional cloak or capsule (Figure 21.1). It is an adaptive organism and when caught in a foreign environment, such as an animal host, fights with all its adaptability to survive (McDonald AE, personal observations, 2010-2020).[2–6,7] Luckily, infections in mammals and birds are rare,[7,8] except in humans in association with HIV and AIDS with *Cryptococcus neoformans*.[9]

There are over 50 species of *Cryptococcus*,[5] but only a few are considered animal pathogens.[5] The initial named species was *C. neoformans*, a pathogen, which in time became known as *C. neoformans neoformans*. Over 10 million years ago, the first split occurred, yielding a separate pathogenic species *C. gattii*.[5] This set the foundation for cryptococcal classifications as we know it today.[5,10,11] Collectively, they are known as the *C. neoformans-C. gattii* species complex and are responsible for almost all mammalian and avian cryptococcal infections.[7–10]

Both complexes have gained attention in recent medical history and, in doing so, exemplified a major difference in their pathogenicity.[9,12] These pathogenic traits have also ended any medical complacency regarding *C. neoformans-C. gattii* species complex in causing disease.[10]

Globally, the *C. neoformans* complex species has been strongly associated with host immunodeficiency, emerging as a global epidemic in HIV/AIDS patients in the 1980s.[13] Prior to the HIV/AIDS pandemic, there were fewer than 200 human cases of cryptococcosis in the literature.[9] In 2009, Park et al. estimated the global burden of the disease (HIV/AIDS/cryptococcosis) to be close to 950,000 cases, with approximately 625,000 deaths annually.[9] However, with the development of antiretroviral therapeutics for AIDS patients, antifungal treatment became highly successful, enabling successful cryptococcal treatment, extending and saving many lives.[9] While it was initially thought that a negative HIV status would exclude one from *C. neoformans* cryptococcosis, in New York City, interestingly many children have antibodies to *C. neoformans*.[9] However, it is now recognized that other factors causing immune suppression, such as cancer chemotherapy, steroid medication, and some comorbidities, can activate dormant *C. neoformans* causing secondary infection.[9] Quite differently, *C. gattii* acts as a primary pathogen, apparently not requiring immune compromise to invade an animal host and primarily affecting immunocompetent individuals.[5,7] In 1999, cryptococcosis caused by *C. gattii* appeared unexpectedly, causing a novel epidemic on Vancouver Island, British Columbia, Canada. The Vancouver Island outbreak qualified as a true epidemic among humans.[14,15]

By 2000, cryptococcosis was well established as an emerging disease on Vancouver Island, and by 2004 was considered endemic. The locations of disease were mapped, and the organism's environmental range was found to encompass the east coast of Vancouver Island, the southern Gulf Islands, Washington, and Oregon states.[5,14,16] It is now recognized that *C. gattii* had been present silently in the Parksville area silently of Vancouver Island, prior to the onset of the epidemic.[15]

This pathogen caused illness and mortality in humans and animals, including wild and exotic birds.[15] Although the disease incidence has declined in recent years, *C. gattii* has remained endemic to the Pacific Northwest geographic niche.[5,7,15,17] The author's avian practice has treated nine psittacine cases (2000–2020) from this endemic area. Only two are documented

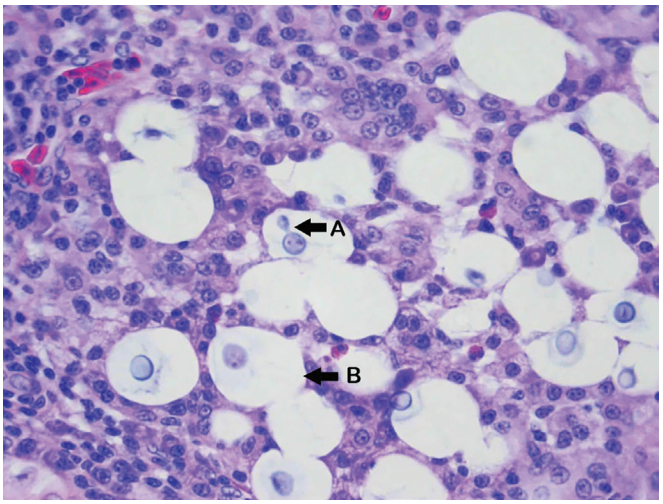


FIGURE 21.1 Cryptococcal infection in brain tissue. *Arrow A* identifies the narrow-based budding yeast, and *arrow B* identifies the polysaccharide capsule; depending on environmental conditions, capsular size can be highly variable.

survivors, who are both asymptomatic but require maintenance with oral medications (McDonald AE, personal observations, 2010-2020).

Human literature points out the sentinel value of nonhuman clinical cases.[7,11,15,18] The fact that these organisms can produce epizootics had led to extensive surveillance of both the pathogen's environmental distribution and its biological behavior.[7,12,14,15,19]

CLASSIFICATION

Pathogenic cryptococcal classification has been revised extensively to reflect morphology, biological characteristics involving serotyping (A, B, C, and D), and genotyping *C. neoformans neoformans* (VN1V), *C. neoformans* var. *grubii* (VN1, VN11), the AD hybrid (VN111), and *C. gattii* (VG1, VG11, 111, and 1V) (McDonald AE, personal observations, 2010-2020).[5]

More recently, the high molecular discriminatory power of the amplified fragment length polymorphism genotyping method has been used, defining the current nomenclature. The *C. neoformans* species complex is currently composed of four species, whereas the *C. gattii* species complex is composed of five molecular types, which are argued to be five distinct species.[4]

However, revision is continual.[10] As of 2018, cryptococcal nomenclature was revised again, suggesting that nomenclature goes back to *C. neoformans* species complex and *C. gattii* species complex, and the Latin names for individual species of *C. gattii* be removed.[10]

C. neoformans species complex:

1. *neoformans neoformans* or VNIV
2. *neoformans* var. *grubii* VNI
3. *neoformans* var. *grubii* VNII
4. *AD hybrid* var. *neoformans/gattii* VNIII

C. gattii species complex:

1. AFLP4NGI (*C. gattii* sensu stricto)
2. AFLP7NGII *(C. deuterogattii)*
3. AFLP5NGIII *(C. bacillisporus)*
4. AFLP7NGIV *(C. tetragattii)*
5. AFLP410NGIV *(C. decagattii)*

Genotypes VG1 and VG11 are prevalent worldwide, whereas VGIII and VGIV are less common. *C. gattii* V11a, recognized as highly virulent, was the most common animal isolate involved in the Vancouver Island outbreak.[15] It had devastating consequences on the inadvertently affected psittacine birds (McDonald AE, personal observations, 2010-2020).

LIFE CYCLE

Cryptococcus spp. are free-living environmental microbes, with a life cycle that does not appear to require an animal host.[4,9,14,20] They are diphasic environmental yeasts; the most basic form is the asexual phase that utilizes budding for reproduction. However, with the appropriate environmental factors (substrate, warmth, and dryness), hyphae grow to form blasidioform arms that may produce basidiospores: these undergo meiosis to form haploid spores. These spores are believed to be the infective organisms or propagules. Hyphae growth signifies development of the sexual phase or teleomorph, which is characteristic of all biphasic fungi. Sexual reproduction appears to have the potential to boost cryptococcal spore numbers to aid in the survival of the organism.[10] The sexual form is responsible for pathogenic animal host involvement. Inhaled yeast can also cause illness.[7,11]

C. neoformans mycelia grow commonly in bird excreta, especially when desiccated, using its high creatinine content as a nitrogen source.[21] The mycelial growth of *C. gattii* is strongly tree-associated, growing in decaying tree hollows and much less commonly in bird excreta.[3,9,15,16,20] This difference is the most defining ecological characteristic that distinguishes *C. neoformans* species complex from *C. gattii* species complex.

DISTRIBUTION

Pathogenic *Cryptococcus* spp. have been recognized on every continent.[2,6,8,15] Until recently, Antarctica was exempt. However, studies on Antarctic volcanic interior ash have identified viable *C. laurentii* organisms, which have recently become recognized as an emerging human pathogen associated with a rising number of meningitis cases.[22,23] There is one documented case report in a glossy starling *(Lamprotornis chalybaeus)*, with dermal pathology related to *C. laurentii*.[24] The organism isolated in this case was not particularly virulent. The bird in that case exhibited patchy feather loss around the back and beak area, and grayish crusts were noted, which were firmly adhered to the underlying skin. *C. laurentii* has been isolated from Canada goose droppings in New York state,[25] from feral pigeon *(Columba livia)* droppings in Sweden,[26] from crop and fecal samples from pigeons in Grand Canary Island,[27] and from captive pet bird droppings in pet shops in Brazil.[28]

In the literature, one of the problems recognized in determining geographical distribution of pathogenic *Cryptococcus* species has been the lack of reported differentiation between pathogenic and nonpathogenic species and between the pathogenic species. Historically, this resulted from the lack of ready access to sophisticated, rapid, and affordable diagnostics. The importance of pathogenic species identification globally was not appreciated until the AIDS virus/*Cryptococcus* relationship was realized.[9]

Several researchers emphasize the importance of further clinical studies to define differences between *C. gattii* and *C. neoformans*, to define potential differences in disease syndromes, treatments, outcomes, and potentially the management of potential outbreaks of cyrptococcosis.[4,29]

C. neoformans and *C. gattii* have both been found in the excreta of many wild and captive avian species, including birds kept indoors and outdoors, in private homes, churches, pet stores, zoos, and private collections.[15,30,31] The avian species documented to be affected are numerous and include canaries *(Serinus canaria)*, lovebirds *(Agapornis roseicollis)*, raptors, and kiwis (*Apteryx* sp.).[10,15,30,29–32]

Taxonomic orders in which *Cryptococcus* infections have been reported include Anseriformes, Accipitriformes, Apterygiformes, Charadriiformes, Columbiformes, Passeriformes, Psittaciformes, Galliformes, Strigiformes, and Spheniscifor mes.[2,7,30,31,32] *Cryptococcus* spp. have also been found in the environment in association with vegetation (trees, fruit, and vegetables), avian droppings and skin, commercial bird seed, soil, fresh and saltwater.[7,8,14,15,30,20,29]

Surprisingly, the presence of cryptococcal organisms environmentally, even in avian droppings, does not necessarily

correspond to the prevalence or incidence of avian or mammalian disease, and when noted, such a correlation seems to be more the case with *C. gattii* than with *C. neoformans*.[20,22,33] Environmental presence in excreta as opposed to cloacal samples may reflect environmental contamination, especially if the dropping sample is dry.[9] *C. gattii* tends to be more fastidious and found in specific biogeoclimatic niches as compared to the more ubiquitous, *C. neoformans*.[12] Cryptococcal environmental survivability can be long. A culture of *C. gattii* was documented to be viable after 20 years.[34]

Historically, *C. neoformans* was said to favor temperate climatic zones, while *C. gattii* favored (sub)tropical zones. This simplistic climatic classification system is no longer considered valid for predicting the distribution of pathogens and likelihood of pathogenicity.[5,10,11,15] Recently, the concept of ecological niche modeling has emerged.[14,15,35] Using this model, climatologists and epidemiologists work together to predict public health impact and identify areas with similar ecologies at risk for pathogen spread. The model considers ecosystems, meteorology, and the presence of environmental pathogens and has been utilized successfully for *C. gattii* to predict the presence of the organism, including human and animal involvement.[12,15,16] Hopefully, the information from these models can eventually be used to advance disease prevention and treatment.[36,37]

TRANSMISSION

Spores and yeast are carried by air currents, especially when soil is disrupted.[14,29] *C. gattii* has been found in high traffic areas, vehicle wheel wells, and footwear and is associated with forestry activities such as wood chipping.[4,14,15,18] Spore counts are higher in summer months and drop precipitously after rainfall.[14,15]

The source and method of arrival of *C. gattii* to Vancouver Island are still unknown. Possibilities proposed include soil contamination by flooding contaminated sea water, tourism, importation of wood or trees, dispersal via infected bird droppings, and water currents.[10,14,15] *C. gattii* is capable of long-term survival in freshwater lakes and streams and in salt water.[10,14,29] It is a lethal marine mammal pathogen.[7,38]

C. neoformans is strongly associated with pigeons and, less so, with chickens *(Gallus gallus)*, where it causes intestinal colonization without systemic illness.[9,39,29] The digestive tract of these species provides a nutritionally rich environment for yeast replication and physical distribution.[9,22,] The thick exterior capsule of the *Cryptococcus* organisms is highly protective, enabling successful travel via fomites and within the digestive system. Singh et al. have cited evidence for rodents as reservoirs and passenger hosts, akin to pigeons and chickens for *Cryptococcus* spp.[10]

PATHOGENICITY

Cryptococcus is a virulent organism that has evolved very refined self-protecting survival mechanisms. The rare ability of *Cryptococcus* to replicate at warmer temperatures is the basic factor permitting successful animal invasion.[34,37] Cryptococci will grow at 37°C (98.6°F) (human body temperature) and even up to 40°C (104°F) but prefer temperatures less than 40°C (104°F).[7,21] These temperatures, which are lower than typical avian core body temperatures, are consistent with the more peripheral temperatures of the beak and upper respiratory system. This probably explains the predilection of these sites for cryptococcal growth and possibly eventual tissue invasion (Figures 21.2–21.4).[11,30,23]

Bielska has presented an extensive review of the role of cryptococcal pathogenic characteristics, including the ability to grow at mammalian temperatures, up to 37°C (98.6°F).[7] Three important virulence factors are the production of the polysaccharide capsule, melanin synthesis that combats oxidative stress damage by the host, and cell invasion.[7,11] The capsule swells in response to contact with host serum, initiating excessive polysaccharide production.[7] This biofilm has immune-modulating effects on host cells.[7] The capsular factors can upregulate and downregulate immune modulation.[7] As the organisms age within a lesion, they undergo a phenotypic switching related to increased capsule size, increased resistance to phagocytosis, and increased drug resistance.[11] Cryptococcal cell wall–associated virulence includes the presence of capsular channels, which allow expulsion of internally produced toxic products in "bags," called extracellular vesicles (EVs), into host cells, as well as the transportation of melanin to coat the cell membrane for protection against ultraviolet irradiation and reactive oxygen species (ROS).[7]

The effects of cellular invasion include the yeast's ability to survive in the bloodstream but also to survive phagocytosis.[7] The engulfed yeast can reduce the host systemic inflammatory response within the host macrophage allowing its multiplication.[7] The macrophage then delivers the yeast to other body sites where vomocytosis can, without harm to either the host or engulfed organism, deliver the yeast to other tissues or back into the blood.[7] This aids in deep dissemination of the fungus.[7] The virulence of the cryptococcal strain is based in part on fertility or the number of propagules (infectious particles) produced. Probability of infection is believed to be related to the number of inhaled spores, called the inoculum.[6,11,34,35]

The period of incubation (IP) is the period between contact with the organism and the development of disease. In humans, the IP ranged from 2 to 11 months, and in animals, it was as short as 1 month and up to 12 to 13 months after exposure. The period between phagocytosis and the production of disease is the period of latency, which occurs in birds, and may explain, in part, why recurrence of infection occurs after treatment.[5,10] The period of latency has been documented at up to 110 months in a human case and 8 years in a citron-crested cockatoo *(Cacatua sulphurea citrinocristata)*.[5,16]

CLINICAL SIGNS

There is comparatively a low number of clinical avian cryptococcosis cases reported in the literature. [5,11,14,30,21,31,38] These reports and the clinical cases treated at the Night Owl Bird Hospital have been used for the following discussion (McDonald AE, personal observations, 2010-2020). Every clinical case has the potential of providing valuable information.

Cryptococcus spp. have been associated with three distinct clinical syndromes, which are characterized by dermal, peripheral, and deep involvement.[9] Dermal involvement is rare in birds and usually results from traumatic injury. Lesions may be granulomatous and, with immune competency, local and oral treatment, carry a fair but guarded prognosis.[11]

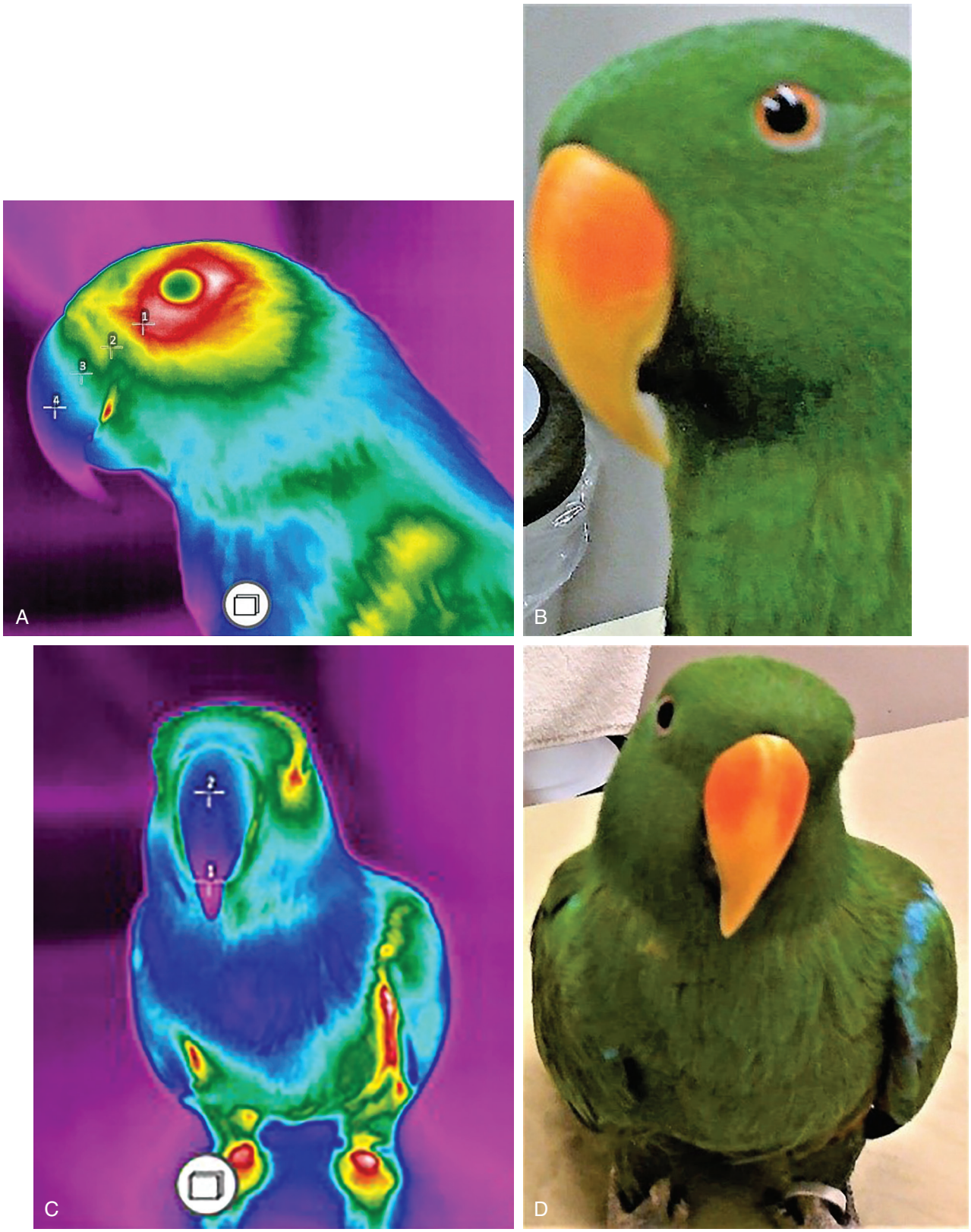


FIGURE 21.2 Lateral thermographic (A) and plain (B), and frontal thermographic (C) and plain (D) images of a fully feathered male grand Eclectus parrot *(Eclectus roratus roratus)*. Using the thermographic legend shown in Figure 21.4, and comparing these thermographic images to those of the female grand Eclectus parrot with feather destructive behavior in Figure 21.3, the difference in temperature of the beak and head as opposed to core body temperature is demonstrated, thereby helping to visually explain why the beak and head comprise the most common sites of cryptococcal lesions in avian species, and possibly also any further tissue invasion.

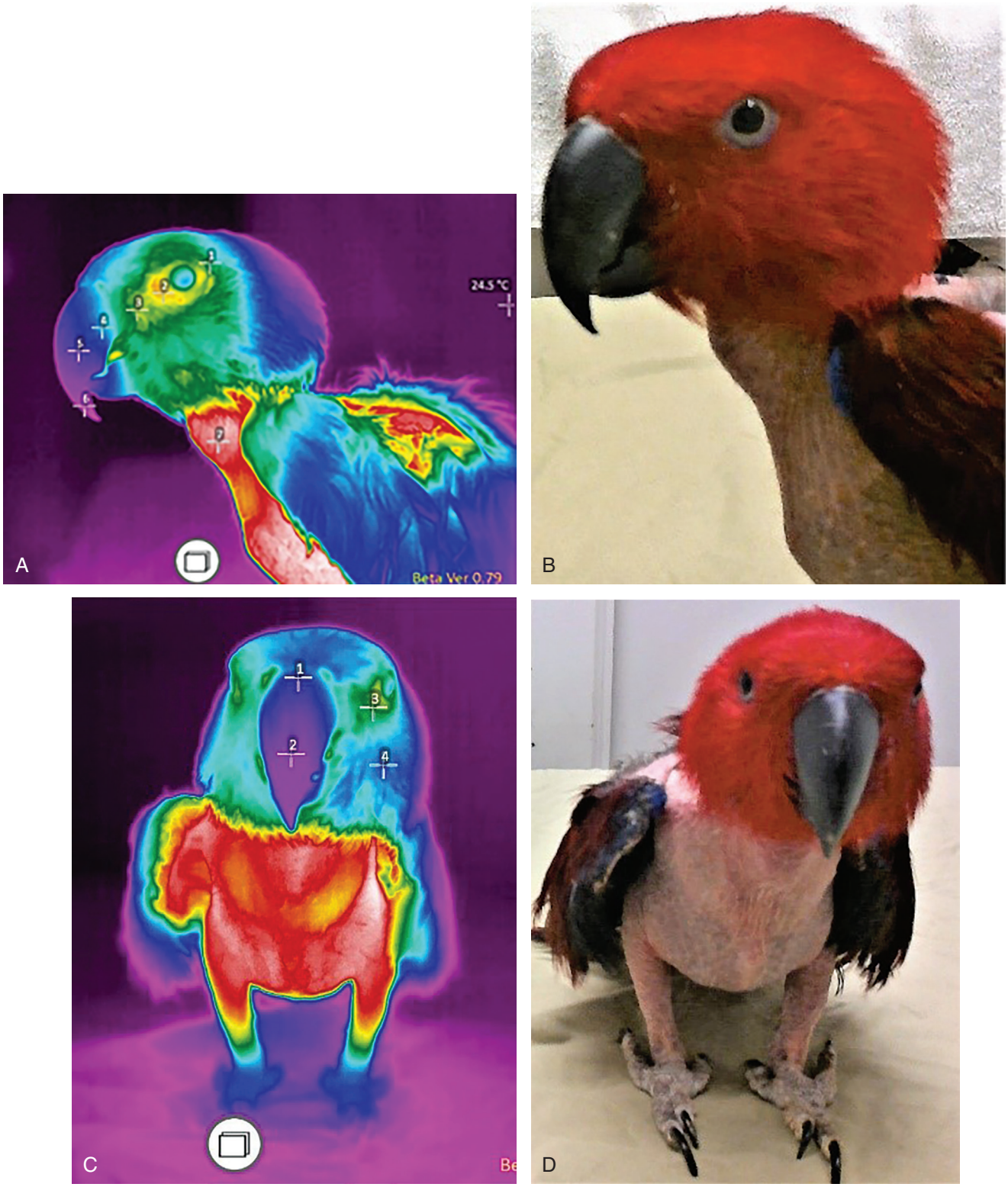
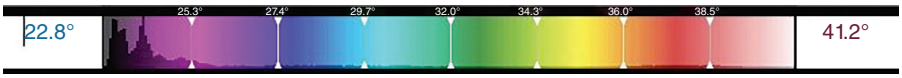


FIGURE 21.3 Lateral thermographic (A) and plain (B), and frontal thermographic (C) and plain (D) images of a female grand Eclectus parrot *(Eclectus roratus roratus)* with severe feather destructive behavior. These images can be compared against the thermographic images of the fully feathered male grand Eclectus parrot in Figure 21.2 to help visualize differences in core body temperature to those of the beak and head, thereby explain the predilection sites for cryptococcal lesions in avian species.



Site	Temp	Color
1	32.8°C	
2	36.8°C	
3	34.6°C	
4	30.4°C	
5	27.9°C	
6	26.7°C	
7	39.6°C	

FIGURE 21.4 Temperatures and gradient by color, used to interpret thermographic images.

This is different from secondary extension to the skin from a deeper-seated lesion, which does not carry a good prognosis (Figure 21.5).

Peripheral involvement refers to lesions located in areas of lower temperature such as the beak and head (nares, nasal sinuses, and perhaps choana). These infections start with nasal tissue colonization and invasion, with their lower, non-core body temperatures favoring fungal growth.[7,35,40] The developing lesions are commonly granulomatous and usually progress slowly but may disseminate in time. Initial surgical treatment and continued local and oral medication may help to control and possibly resolve the infection, but relapses are known to occur (McDonald AE, personal observations, 2010-2020).[5,30,41]

Particularly in the choana, the lesions may appear more as a solid tissue mass; often these lesions are referred to as a cryptococcomas.[40] Such cryptococcomas can foster exuberant mucous production, leading to extreme sinus distension over a few weeks' time, which despite treatment can markedly interfere with life quality (McDonald AE, personal observations, 2010-2020).[30] Both *C. gattii* and *C. neoformans* can cause the development of cryptococcomas. They are inflammatory, tumor-like masses representing areas of fungal invasion and multiplication.[40] They can occur in many tissues.[30,20,40,41] They can be solitary or diffuse and of variable size. In humans, surgical removal is the treatment of choice for single masses.[5] Surgical debulking is thought to decrease antigen load and facilitate recovery (Figure 21.5).[5]

Singular, choanal polyps, when noted in birds, carry a good prognosis with excision (McDonald AE, personal observations, 2010-2020).[30] Local head lesions may present with ocular and/or retrobulbar involvement and/or blindness (Figure 21.6). In avian species, ocular lesions may result from a peripheral lesion, but they can also result from disseminated disease. Invasion and spread to adjacent tissues may lead to brain involvement.[14,19]

C. neoformans is more commonly associated with central nervous system (CNS) signs.[9] In psittacine birds, *C. gattii*–caused CNS lesions may present as a flock outbreak with birds displaying ataxia or difficulty flying, which can rapidly progress





FIGURE 21.5 *Cryptococcus neoformans* right-sided sinusitis in a long-billed corella *(Cacatua tenuirostris)*. Note the rostral-ventral distention of the infraorbital sinus region and dried exudate about the right nare (A). Successful treatment included sinusotomy and debulking of the cryptococcoma (B), followed by a total of 4 months of systemic itraconazole treatment. (Photos courtesy Bob Doneley.)

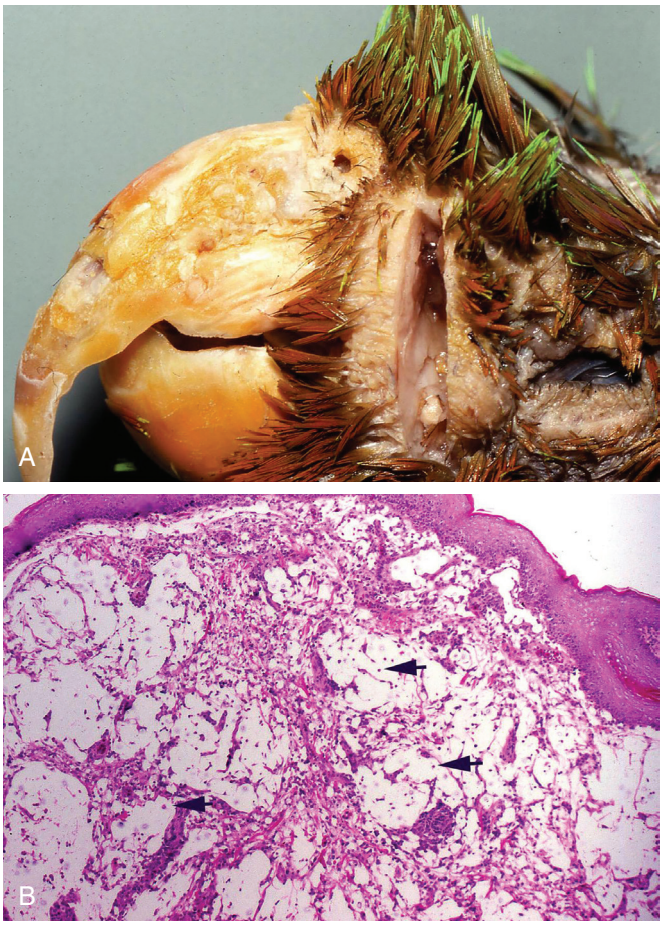


FIGURE 21.6 Deep head lesions due to cryptococcal infection that have erupted through the sinuses (A). Histopathology revealed invasion of *Cryptococcus neoformans (arrows)* into the brain (B). (Photos courtesy R. Schmidt.)

to seizure activity and death.[38] Neurological involvement carries a poor prognosis.[9,31]

Anatomically, involvement of the trachea and syrinx can be seen. Initially, the patient may sneeze but otherwise appear healthy. However, over the course of several days to weeks and without treatment the patient's condition can quickly deteriorate, with clinical signs such as exercise intolerance and inspiratory dyspnea being noticeable. Tracheal lesions can start with a mucoid tracheitis, following which cryptococcoma development may occur, which will require surgical removal or debulking. The infection can reoccur at other tracheal sites. Pulmonary involvement may also take place and dissemination can occur from there.

Infection involving the respiratory system should be treated aggressively as a systematic disease, in which intravenous (IV) antifungal medications, nebulization/topical treatment, and long-term oral medication for at least a year should be considered.[11] Ensuring exposure to excellent air quality is important.

Patients with systemic dissemination may present in critical condition. Cryptococcemia is reported to produce shock proteins and cryptococcal capsular proteins can provoke severe oxidative stress.[40] Deeper infections start with lung invasion, following which all body tissues become vulnerable.[1,38] These deeper syndromes tend to progress quickly and aggressively and may lead to lung lesions with necrosis, encephalitis, and meningitis with associated CNS signs, coelomic involvement with focal organ involvement, osteomyelitis, and occasionally myxedematous peritonitis.[11,14,19,38] Stimulated cryptococci increase their mucoid capsular layer, which physically impedes effective treatment.[7,11,40] The organism can be found in lesions of the brain, sinuses, lungs, coelom, and even vertebrae.[14,38] In peritonitis, its bulk may cause respiratory distress (McDonald AE, personal observations, 2010-2020).[36]

DIAGNOSIS

Diagnosis usually begins with clinical suspicion based on the history of patient exposure to an endemic area (originated from or visited), woodland exposure via hiking, or exposure to accumulated bird droppings (e.g., city squares). This, in addition to the clinical signs, may lead to a suspicion of cryptococcal involvement.[14,15]

The initial diagnostic tool is often cytology "done in-house," on nasal/choanal swabs, secretions, tissues, or tracheal wash. Effective stains for identifying the characteristic capsule include India ink, Wright-Giemsa and modified Wright stain (Figure 21.1).[10,11,34] Recognition on cytology of the characteristic capsule is considered a sensitive point-of-care diagnostic test for pathologic *Cryptococcus* species.[10,11,34] It is easy, fast, and inexpensive. However, organism size and number, capsular development, and budding characteristics can be variable (Figure 21.7).[7,9–11]

The gold standard for distinguishing *C. gattii* from *C. neoformans* is by fungal culture and isolation, utilizing different growth characteristics on CBG or Pal media.[8,10,34] The use of molecular diagnostics is revolutionizing fungal species identification, and although, at present time, these tests largely remain

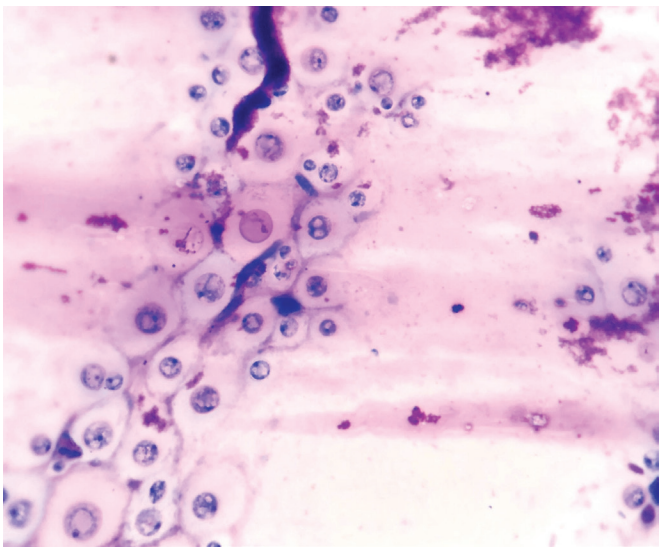


FIGURE 21.7 Modified Wright stain showing the characteristic myxedematous capsule of *Cryptococcus,* and its narrow-based budding.

a research tool, they may be available in some regions for use as a diagnostic tool.[5,9] *Cryptococcus* spp. grow rapidly, appearing in 48 hours on blood agar and Sabouraud dextrose media.[11] This rapid growth enables drug sensitivity testing, which can be used to guide ongoing treatment plans, particularly for those patients that survive the initial 2 weeks of treatment and will need long-term medication.[5,11] Drug sensitivities and minimum inhibitory concentration (MIC) values can vary with the cryptococcal species involved and their geographic origin.[5] It is recognized that in vitro drug sensitivities may not translate into in vivo efficacy.[5] Only a few laboratories offer fungal drug sensitivity testing. For those interested in having such test performed, it would be advised to contact university-associated diagnostic labs (e.g., Texas A&M, Cornell University), the CDC, medical school pathology laboratories, or regional agriculture laboratories.

Other diagnostic options involve cryptococcal antigen serology, or CrAg testing. The CrAg indicates the amount of cryptococcal capsular antigen that has gained access to patient circulation (antigenemia).[11] It does not necessarily indicate severity of infection. The capsular antigen is highly specific to *Cryptococcus* spp. but does not differentiate *C. neoformans* from *C. gattii* or quantitate live versus dead organisms.[11] Any titer elevation can be significant, as circulating antigen implies tissue invasion.[11] The CrAg antigen tests commonly used are the latex agglutination (LAT) and the enzyme-linked immunoassay (EIA). Both provide comparable results. False negatives and positives are rare but can occur.[11] Either test requires approximately 500 μL of serum, limiting its usefulness in small or anemic patients. CrAg testing is available through commercial and university veterinary laboratories.

ADDITIONAL DIAGNOSTIC TOOLS: IMAGING AND ENDOSCOPY

Noninvasive diagnostic imaging tools are in general less challenging for the compromised patient. Advanced imagining techniques (magnetic resonance imaging [MRI], computed tomography [CT]) are preferential for detection of small cryptococcomas of the lung and CNS. CT usage is much more readily available and clinically adapted (see Chapter 2 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 14, Advances in Diagnostic Imaging).

Digital radiography aids in patient evaluation for general health status including cryptococcal lesions, particularly of the beak, sinuses, trachea, and coelomic cavities (Figure 21.8). Fluoroscopy or a swivel head generator can also be used for radiographic body screening in patients whose condition is considered too critical for anesthesia and standard radiographic positioning (see Chapter 2). Attention to patient positioning, imaging modalities chosen, magnification, and adjustment of contrast can make a great difference in identifying the presence of these lesions (see Chapter 2).

In a female Eclectus parrot *(Eclectus roratus)* that had resided on Vancouver Island for several years prior to presentation for lethargy, poor appetite, and marked inspiratory distress at the author's practice, radiography suggested presence of a tracheal lesion (Figure 21.8). Based on the presence of a seropositive CrAg result, a tracheal cryptococcoma was suspected, which was subsequently removed via endoscopy and confirmed cytologically (Figure 21.7). Sixteen

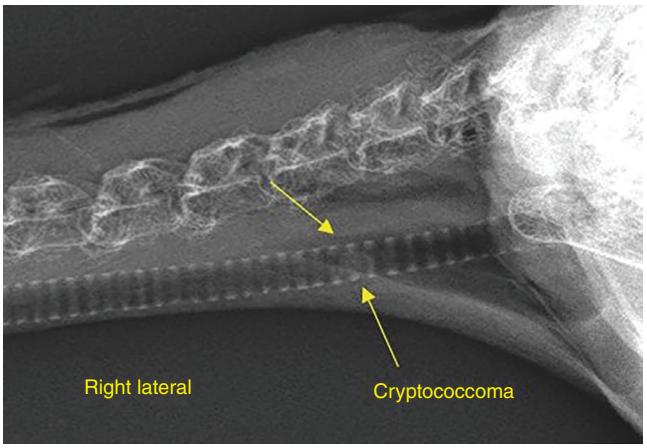


FIGURE 21.8 Lateral radiographic image of a female Eclectus parrot *(Eclectus roratus)* with a proximal obstructive tracheal lesion due to cryptococcoma development. This lesion was successfully debrided; however, clinical signs reoccurred in this patient.

months after this initial cryptococcoma was removed, clinical signs recurred due to the development of another lesion deeper in the trachea. A second endoscopic surgery was more difficult, as the base was congruent with the tracheal wall, allowing only a debulking of approximately 60% of its overall size. Due to comorbidities, the patient was eventually euthanized 6 weeks later, with postmortem histopathology of the affected tracheal wall confirming recurrence of the cryptococcoma (Figure 21.9).

Ultrasound and CT imaging can be particularly valuable for identification of comorbidities of the cardiovascular system and liver and suspect cryptococcal masses within the respiratory tract and coelomic cavities (Figures 21.10–21.12). Gross coelomic distension can result from exuberant capsular material.[36] In cryptococcal peritonitis, the echogenicity of the myxedematous gel is that of water; however, on centesis, its consistency makes aspiration and drainage potentially more difficult (McDonald AE, personal observations, 2010-2020).

Endoscopy plays an important role, especially in tracheal lesions (see Chapter 30). Air sac cannulation anesthesia facilitates examination, diagnostic sampling, and treatment. Tracheal cryptococcoma removal or biopsy (debulking) may require basket type biopsy forceps to successfully grasp the lesion (see Chapter 30 for further information on endoscopic removal of masses). The mass may be firmly attached to the tracheal wall, making removal challenging and time consuming, or it may require tracheal surgical resection and anastomosis (see Chapter 29 for afurther information on tracheal resection and anastomosis).

An air sac tube can also be used for limited laparoscopy and/or therapeutics (see Chapters 29 and 34), although great care should be taken to avoid any further cryptococcal contamination of the respiratory or coelomic cavities. If the air sac tube insertion wound does not heal quickly following tube removal, it may indicate a compromised patient and perhaps a more guarded prognosis. While the tube is in place, it is recommended to perform maintenance cleaning every 2 to 4 hours using a Calgi-sized swab moistened with a tissue-compatible (ophthalmic) dilution of IV amphotericin B or voriconazole (McDonald AE, personal observations, 2010-2020).

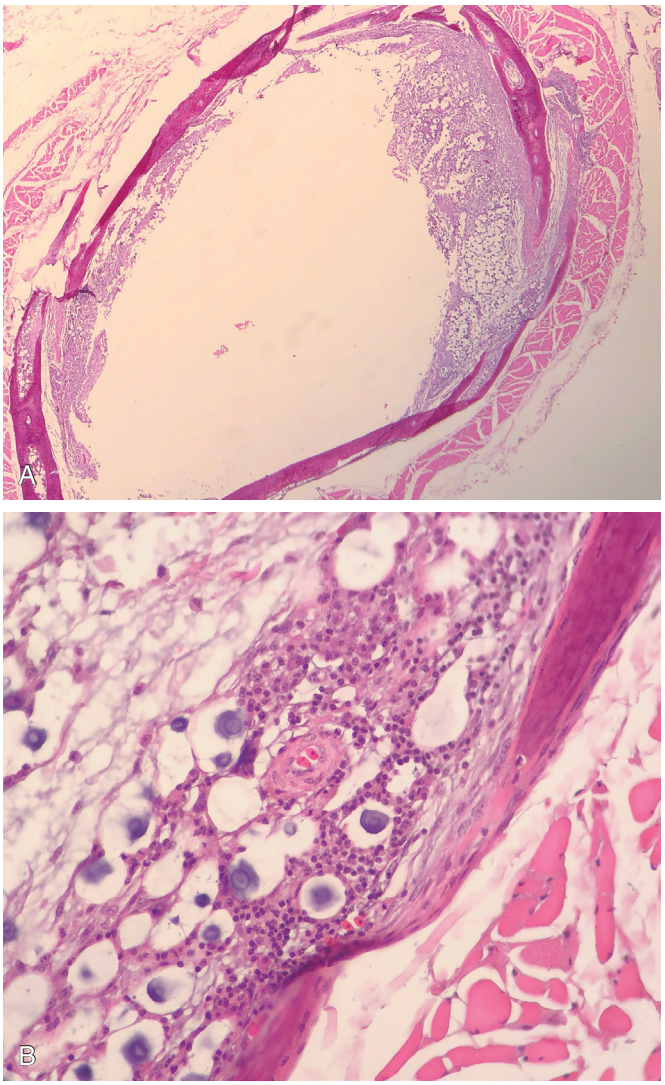


FIGURE 21.9 (A) Postmortem section of affected tracheal ring of the Eclectus parrot *(Eclectus roratus)* from Figure 21.8 with recurrence of clinical disease 16 months after endoscopic removal of the initial tracheal lesion. (B) Magnified view of tracheal ring with cryptococcal invasion.

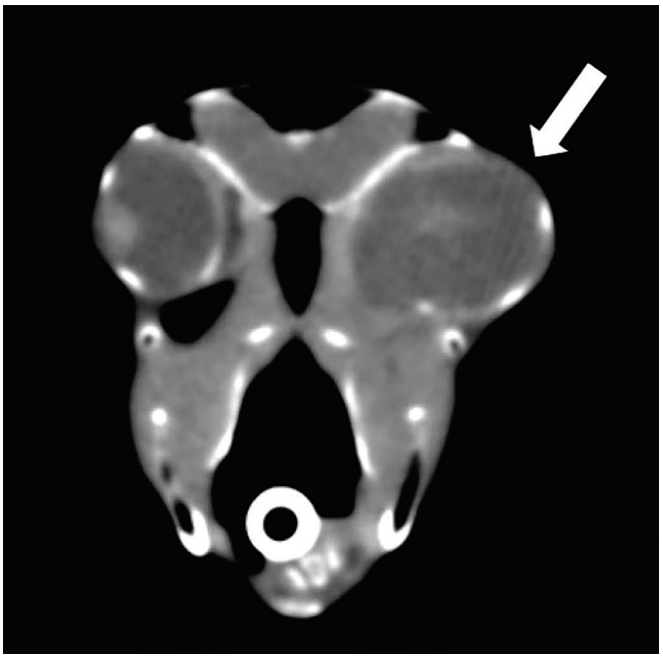


FIGURE 21.10 Computed tomography image of a Grey parrot *(Psittacus erithacus)* with multicentric cryptococcosis. In this image, exophthalmos and buphthalmos of the left eye are apparent, as well as loss of normal intraocular architecture *(arrow)*. Cytologic examination of fluid aspirated from the eye showed extracellular yeast, 4–15 μm in diameter, with narrow-based budding and a thick, nonstaining capsule, consistent with *Cryptococcus*. (Photo courtesy Raina Schunk.[41])

TREATMENT

Initial treatment is divided into general patient support and targeted antifungal therapy, which is largely drug oriented (see Chapter 8). Supportive care includes the use of antiinflammatory medication (NSAIDS, colchicine), antioxidant therapy (silymarin, glutathione), analgesics (butorphanol, tramadol, NSAIDs, gabapentin), anxiolytics, and antiseizure medication (midazolam, gabapentin).

Ideally, perhaps through the Center for Disease Control, historical local geographic information exists as to which species of *Cryptococcus* prevails, its antifungal drug sensitivities, and its MIC values. Unfortunately, drug sensitivities and MIC values vary geographically.[5,11,30] Regardless, fluconazole and voriconazole are generally good first choices.[5] In addition, new antifungal drugs are being developed, one of which is the drug T2307, which in future may provide novel, effective therapeutic options for treatment of cryptococcosis.[6,42]

Induction of targeted therapy for systemic infections often starts with IV amphotericin B combined with fluids, especially in cases involving the CNS (see Chapter 8). After induction, ongoing therapy combined with a long-term oral antifungal (fluconazole or voriconazole) (McDonald AE, personal observations, 2010-2020). Itraconazole and terbinafine may not be effectively absorbed orally to reach adequate plasma levels to treat infection. In humans, IV amphotericin B is given every 24 hours for a variable period depending on the response to treatment. Midazolam/butorphanol sedation has made IV administration much safer and easier in birds (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 19, Anesthesia). Both fluconazole and voriconazole are well tolerated by avian patients for continued monotherapy (McDonald AE, personal observations, 2010-2020). Fluconazole pediatric suspension as Diflucan (Pfizer, Kirkland, Quebec, Canada) is palatable and can be given once a day; it is also more economical than voriconazole. It is less likely to produce significant side effects than itraconazole and has the additional advantage of reaching higher concentrations in the eye and CNS.[30]

All three drugs are available in IV formulations and cross the blood-brain barrier. They are usually readily available from human hospital–associated pharmacies. The IV form of amphotericin B and voriconazole can also be used for

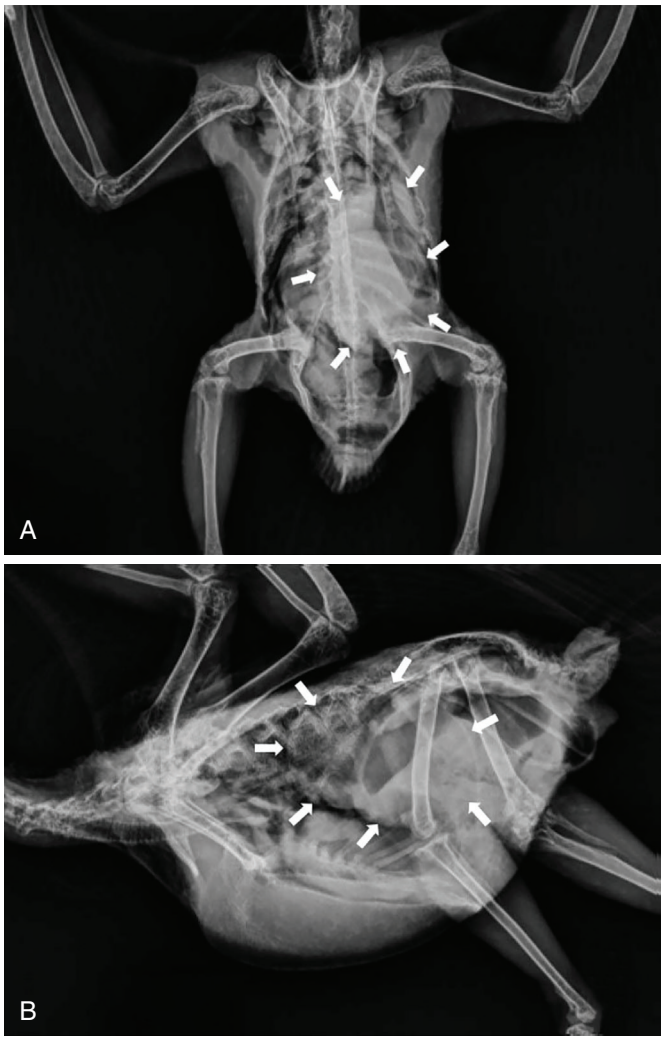


FIGURE 21.11 Ventrodorsal (A) and right lateral (B) radiographs of the Grey parrot *(Psittacus erithacus)* from Figure 21.10. A large mass is visible in the left coelom *(arrows)*. (Photo courtesy Raina Schunk.[41])

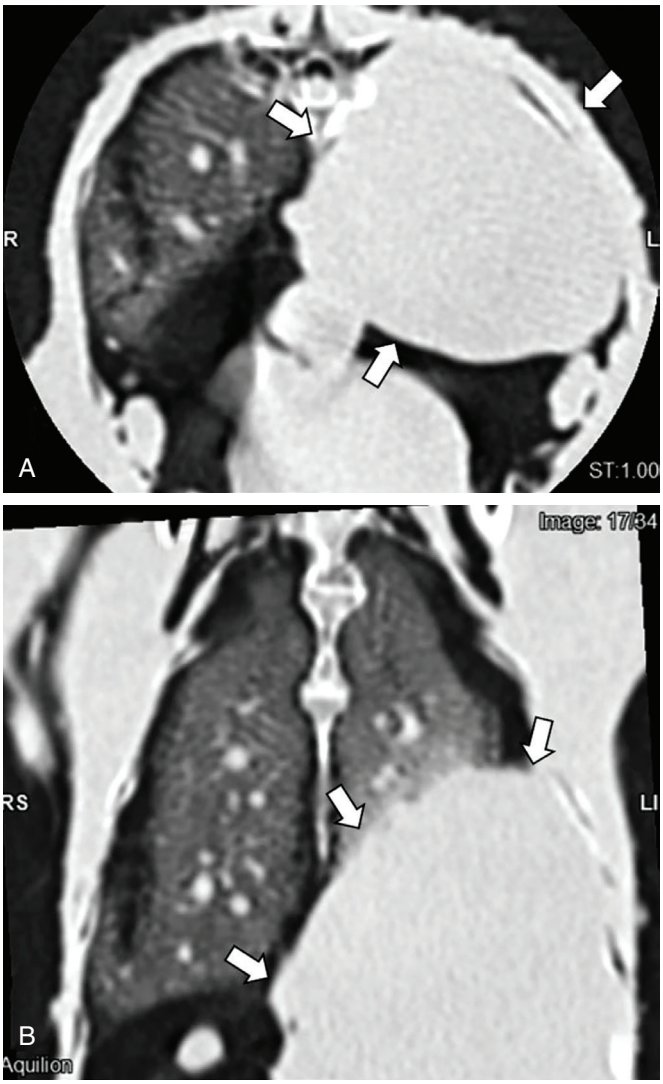


FIGURE 21.12 Computed tomographic image of the Grey parrot *(Psittacus erithacus)* from Figure 21.10, obtained after administration of intravenous iohexol contrast. A large left-sided intracoelomic mass is demonstrated in the axial plane (A) and coronal plane (B) *(arrows)*. (Photo courtesy Raina Schunk. From Schunk R, Sitinas N, Quesenberry K, Grodio J: Multicentric cryptococcosis in a Congo African Grey parrot *(Psittacus erithacus erithacus), J Avian Med Surg* 31(4):373–381, 2017.[41])

nebulization and can be compounded into ophthalmic or nasal drops or used topically (intratracheal lesion spray). Drug toxicity from topically used, tissue-compatible (ophthalmic) dilutions is unlikely (see Chapter 8). If multiple modalities or multiple antifungal drugs are used together, great care needs to be taken to ensure patient safety from adverse drug interactions.

Nebulization with IV voriconazole produces drug levels in the lungs but does not achieve therapeutic blood levels (pigeons).[43] Nebulization with antifungals has been utilized for lower respiratory involvement with questionable efficacy.[43] The concerns and potential for variability relate to the length of nebulization time and the resultant uneven distribution of particles deposited within the respiratory system with nebulization. Aerosol exposure periods of 0.5 or 1 hour did not result in a readily observable distribution of 1.0-μm fluorescent microspheres to the cranial thoracic, caudal thoracic, or abdominal air sac membranes. The 2- and 4-hour exposure periods resulted in readily observable deposition of the 1.0-μm fluorescent microspheres in the cranial thoracic, caudal thoracic, or abdominal air sac membranes, with the 4-hour exposure period resulting in the greatest number of particles on the membrane surfaces.[43]

Another factor to consider is the mucinous gel found in upper airway cryptococcosis.[30] Acetylcysteine is a powerful mucolytic that also reduces host oxidative stress (see Chapter 28).[44] As such, the author hypothesizes that nebulization of the upper airways with acetylcysteine, directly before nebulizing with an IV antifungal, may enhance treatment efficacy (McDonald AE, personal observations, 2010-2020).

To minimize the potential for renal damage, concurrent use of IV amphotericin B with nephrotoxic drugs (NSAIDs or

aminoglycosides) should be avoided. In addition, adequate fluid therapy should be provided, even if IV amphotericin B is used as a monotherapy (see Chapter 8). Other potential adverse drug effects to consider are adrenal suppression from ketoconazole and itraconazole hepatotoxicity in Grey parrots (*Psittacus erithacus*; see Chapter 8).

If treatment of a comorbidity such as bacterial conjunctivitis or cloacitis is indicated, selection of an immune-modulating antibacterial or an antibiotic that also has anticryptococcal activity should be considered. Several "natural" substances have been studied in vitro as antifungal drug enhancers. These include cinnamic and benzoic acids, which have been tested as effective synergistic adjunct treatment for fluconazole, itraconazole, and amphotericin against *C. neoformans*.[3] Similarly, the avian microbiome is reported to have an inhibitory effect on survival of *Cryptococcus* in the gut and lungs.[30] As such, fecal translocations from healthy birds could potentially assist in patient immune support. Currently there is no vaccine available for prevention of cryptococcosis[42].

The longest still living case of *C. gattii* in the author's experience is a male Eclectus parrot, who developed a choanal cryptococcoma 19 years ago, with infection acquired and identified at age 3. The mass self-amputated within 24 hours of the initial presentation. The patient has maintained a positive CrAg titer since but appears normal (McDonald AE, personal observations, 2010-2020). Initially, the patient was medicated with a succession of antifungals, but over the last 11 years, the patient has received voriconazole (monotherapy) orally every 12 hours and has an excellent quality of life (McDonald AE, personal observations, 2010-2020).

MONITORING RESPONSE TO TREATMENT

In veterinary medicine, the CrAg has been of value in monitoring patient response to therapy. Repeat testing should be delayed for 8 weeks after initiation of treatment to avoid confusion from rising titers from degenerating organisms, rather than treatment failure.[11] If the titer is positive, treatment should be maintained, as even low-level titers indicate active infection.[11] Titers typically decline with successful treatment, although the drop in titer generally lags the clinical improvement.[10]

Therapy should be continued for at least 2 months, but possibly longer (years) post–negative titer results. At this time, the CrAg should be remonitored every 2 months, following which the intervals can gradually be increased to every 6 to 12 months. A negative titer is not a guarantee that relapse or reinfection will not occur.[11] Nasal tissue may remain colonized despite a negative antigen titer and a lack of clinical signs. In such cases, culture, polymerase chain reaction (PCR), or cytology may be helpful in determining if the organism has been effectively eliminated.[11]

For identified imaging abnormalities, such as skull, sinus, visceral, tracheal, or pulmonary lesions, long-term monitoring is advisable at appropriate intervals for each pathological area and modality. Initially, radiographic imaging is recommended to be repeated every 1 to 2 weeks where relevant while gradually extending that to monthly or greater intervals.[5,11,30] Periodic biochemical monitoring for hepatic function or cellular damage is also advisable with long-term drug administration.

CLIENT COMMUNICATION

The client needs to be informed in a timely manner of the public health implications of cryptococcosis, pointing out that it is an environmental organism with potential for serious human illness. Emphasizing that their bird has acted as a sentinel and that their environment rather than their bird is a disease risk to them is important.[11] Also, encourage owners to visit their family doctor to discuss the human illness with them.[11]

Educating the owner on the latency period, clinical presentations, life cycle of the organism, and disease prevention methods, as well as the low incidence of disease in people and birds, can help manage their expectations and engage them in their potential role of monitoring and day-to-day care.[11] Clients also need to be prepared both financially and emotionally for the initial and long-term care that are required to manage these cases, as well as be aware of the prognosis (which is often "uncertain" at best) and possibility for relapses or reinfection. Life quality as always needs to be considered. Birds that survive the initial critical period of 2 weeks are candidates for long-term care, which is often the basic tenet of long-term survival.[9,11]

PREVENTION OF DISEASE RECURRENCE IN THE HOST

There are at least two main factors involved in recurrence of disease. One is reinfection when exposed to the infective organisms, whereas the other is reactivation of latent, preexisting (dormant or latent) organisms within the host, which can occur several years later due to exposure to environmental stressors.[8,9,30,40]

Minimizing exposure to sources of spore production and maintaining good air quality is a key to reduction of the potential for reinfection. Poor air quality negatively impacts respiratory tissues, causing inflammation, and increases the susceptibility to fungal invasion. The use of air purifiers is recommended as these will help improve air quality, desiccate the spores, and negatively impact their viability. Owners and caretakers should furthermore be advised to avoid bringing in tree branches, leaves, and old cut wood to use as perches, and so on, particularly if sourced from endemic areas for the organism. In outdoor aviaries, wire enclosures should be kept free of organic debris, whereas access to wild birds, rodents, and especially their droppings should be avoided. Additionally, it is important to avoid landscaping with trees, particularly mature trees, or exposure to soil from outdoor construction, gardening, and pressure washing enclosures.[29]

Prevention of recurrence also depends on avoiding host stress. Avoid any female reproductive activity, including egg production, to curb the associated physiologic and behavioral changes. Similarly, nesting and incubation should be avoided as these promote inactivity, with the bird spending weeks to months in an enclosed dark place (nest box).

Exposure to airborne particles from sources such as books, carpeting, and unwashed toys should also be minimized as cryptococcal organisms have been isolated from regular house dust. The use of an inline vacuum system or a vacuum with filtration will help to trap spores and avoid their further

dissemination. Daily cage cleaning is essential to avoid any build-up of avian droppings in cages, especially in conjunction with fruit or vegetable debris. Peach juice, tomatoes, and other fruits and vegetables will support and possibly enhance cryptococcal mycelial growth.[5,10,14,15] Chlorhexidine, 70% ethyl alcohol, and quaternary ammonium disinfectants are effective against *Cryptococcus* and their implementation in routine hygiene and disinfection protocols should therefore be recommended.[33,45]

CONCLUSIONS

Cryptococcus spp. are fungal pathogens that are far more widely distributed geographically than historically thought. The organism has developed the means to manipulate the potential host's defense mechanisms such as the inflammatory response and utilizes host phagocytosis to further its pathogenesis. It can adapt to adverse conditions in the host, and remain and survive in a latent form for years. While it rarely causes disease in birds, it can lead to severe clinical disease in individual or outbreaks in larger groups if given the right environmental circumstances. *Cryptococcus* spp. can alter its genome for better self-protection and propagation and amazingly can harness other organisms such as amoeba to alter their behavior to evoke environmental changes that benefit the fungus. It needs no animal host to survive. Due to its unique characteristics and ability to induce long-term infections that are difficult to treat, researchers and medical professionals have been forced to continue to search for key determinants of its pathogenicity, which will hopefully aid in identifying novel therapeutic approaches to manage and hopefully in future cure clinically affected individuals[42].

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CHAPTER 22

Trichomoniasis

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Trichomoniasis is a parasitic disease affecting a range of avian species. The disease, also known as canker in pigeons or frounce in raptors, is the clinical form of an infection of the upper digestive tract of birds by protozoa of the *Trichomonas* genus. It causes significant economic losses for aviculturists and commercial pigeon producers and is responsible for several spillover and mortality events resulting in population declines in avian wildlife.

ETIOLOGY

Trichomoniasis in birds is caused by infection with single-celled parabasalids belonging to the genus *Trichomonas*. The trophozoites of these protozoa are small, measuring 6 to 20 μm in length and 3 to 8 μm wide, and have four anterior flagella, a fifth recurrent one that forms an undulating membrane, and a distinct nucleus (Figure 22.1). Trichomonads have a direct life cycle and reproduce by binary fusion; they are present in two life stages—motile trophozoites and nonmotile pseudocyst stages that are only encountered under suboptimal growth conditions in vitro.[1]

It is currently still unclear if the etiology of trichomoniasis in birds concerns one species of *Trichomonas*, specifically *Trichomonas gallinae*, or several distinct *Trichomonas* species. Some studies suggest that the genetic differences among isolates, together with morphology and host species predilection, are sufficient to consider them taxonomically distinct species such as *Trichomonas stableri* and *Trichomonas gypaetinii*.[2–4] Others consider that the partial genetic characterization of isolates with evidence for hybrid lineages, make it premature to conclude the disease is caused by multiple *Trichomonas* species, and classify the different genotypes as *T. gallinae* types, alphanumerically based on the combination of the internal transcribed spacer (ITS) ribotype sequences (A, B, …) and subtypes based on the Fe-hydrogenase (FeHyd) gene sequences (1, 2, …).[5] Regardless, all studies do corroborate that genotypes are evolving in different metapopulations and that genotypes are often associated with specific host species (Figure 22.2). Classically, *T. gallinae* has been associated with the rock pigeon *(Columba livia)*. A recent phylogenetic study showed that all *Trichomonas* genotypes are highly likely to have originated from columbid host species and are evolving along at least four lineages: a specific lineage for mesocarpal fruit doves, an old-world lineage (which includes *T. gallinae* ribotypes A and B, *T. tenax*, and *T. brixi*), a new-world lineage (which includes *T. stableri*, *T. gypaetinii*, and *T. vaginalis*), and an Australasian lineage.[6] For simplicity, *Trichomonas* species or strains causing trichomoniasis in birds are, from this point forward, referred to as *Trichomonas* spp.

EPIDEMIOLOGY

While the ancestral parabasalid from which avian *Trichomonas* spp. arose was possibly a symbiont of the lower gastrointestinal tract of Columbiformes,[6] these species have found a niche in the oral cavity, ingluvies, and esophagus of a range of avian species. They do not survive passage through the lower gastrointestinal tract and therefore rely on direct or indirect oral transmission (Figure 22.3). Direct transmission is particularly favored in Columbiformes because pigeons and doves feed their young with the desquamated lining of their ingluvies known as "crop milk." This feeding method enables highly effective transmission of *Trichomonas* and explains the high prevalence of infection reported in these species.

Both in the wild and in an avicultural setting, infected birds have been confirmed to transmit *Trichomonas* spp. through billing, courtship feeding, or via contaminated food or drinking

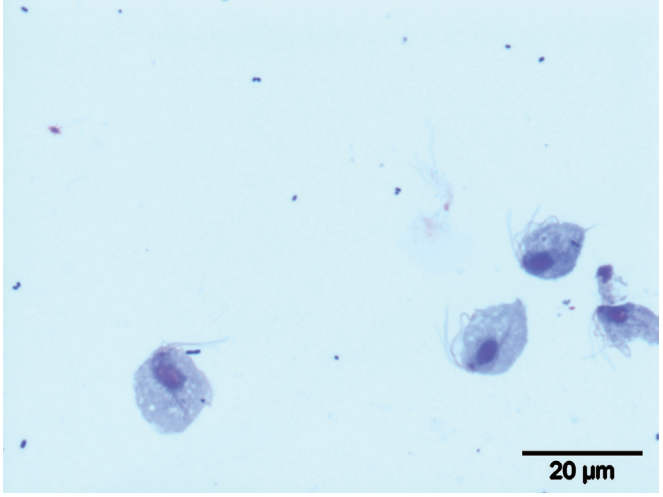


FIGURE 22.1 Avian trichomonads (trophozoites) in cytology, Hemacolor (Merck, Darmstadt, Germany). (Photo courtesy Marja Kik, Dutch Wildlife Health Centre.)

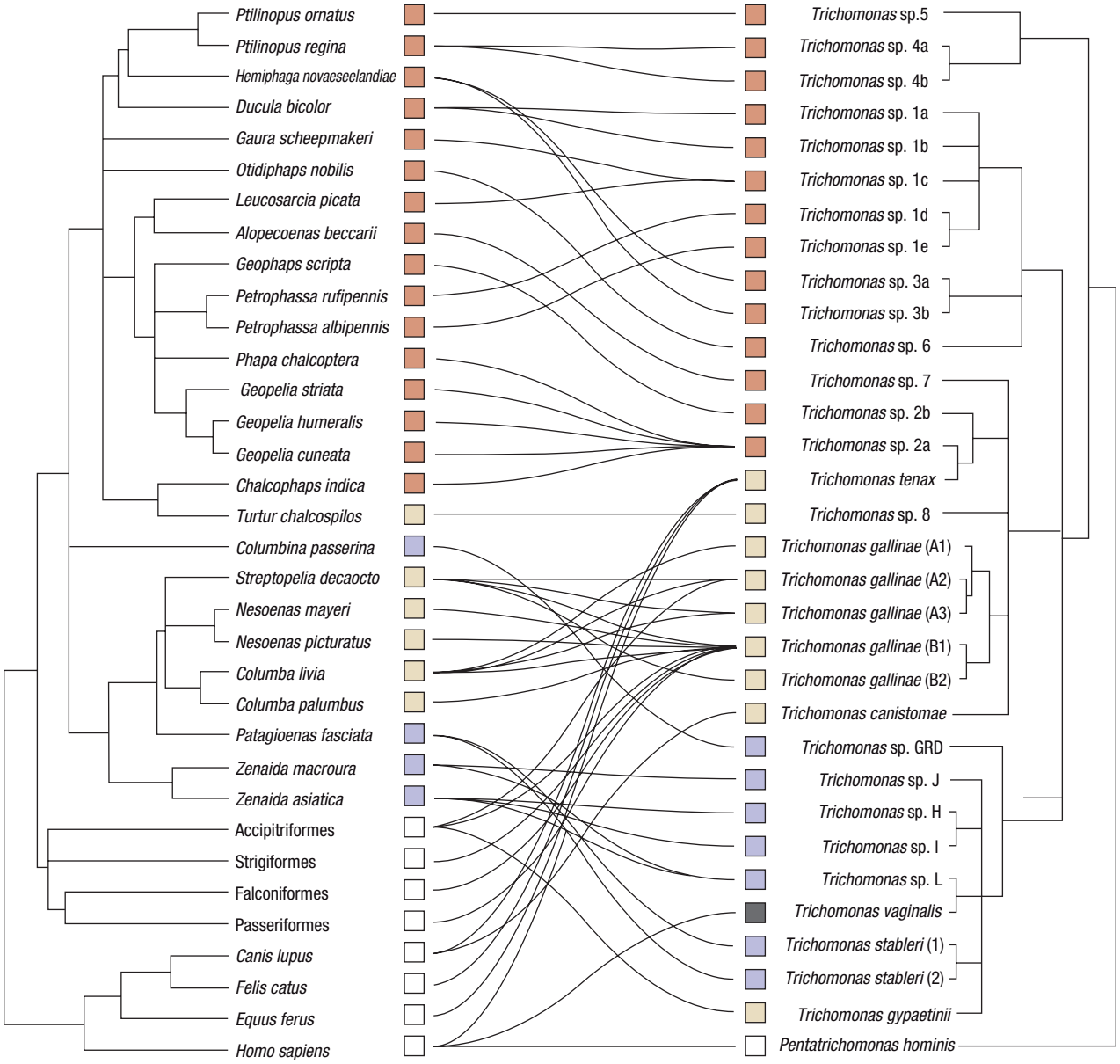


FIGURE 22.2 Cophylogram of all known *Trichomonas* genotypes and their associated host taxa. *Bold branches* and *text* indicate columbid or *Trichomonas* species. Cladogram topologies are based on multigene and nicotinamide-adenine-dinucleotide (NADH) dehydrogenase subunit 2 gene phylogenies (host) and ITS1/5.8S/ITS2 rRNA gene consensus phylogenies *(Trichomonas)*. Associations are indicated by *lines*. *Square boxes* accompanying columbid and *Trichomonas* taxa indicate geographic distribution (*orange,* Australasia; *yellow,* Eurasia; *blue,* Americas; *black,* cosmopolitan). *Circles* indicate geographic origin (>50% support) for a host or *Trichomonas* clade. (Reprinted with permission from Peters A, Das S, Raidal SR: Diverse *Trichomonas* lineages in Australasian pigeons and doves support a columbid origin for the genus *Trichomonas*, *Mol Phylogenet Evol* 143:106674, 2020.)[6]

water. While the direct transmission of trophozoites in infected saliva or regurgitated food material may be the most common route of conspecific infection, direct infection via predation, carrion feeding or cannibalism on infected carcasses, and indirect infection via contaminated water sources or birdseed, all represent possible transmission routes within and between species (Figure 22.3).[7–10]

Columbiformes act as a reservoir host, and transmission to other species occurs as a result of spillover events. In some populations it is likely that the *Trichomonas* spp. persist, with infected birds transmitting the organism within and between members of that population. These populations, in which *Trichomonas* spp. are endemic, act as a primary source of infection for birds of prey (raptors and owls) that either hunt,

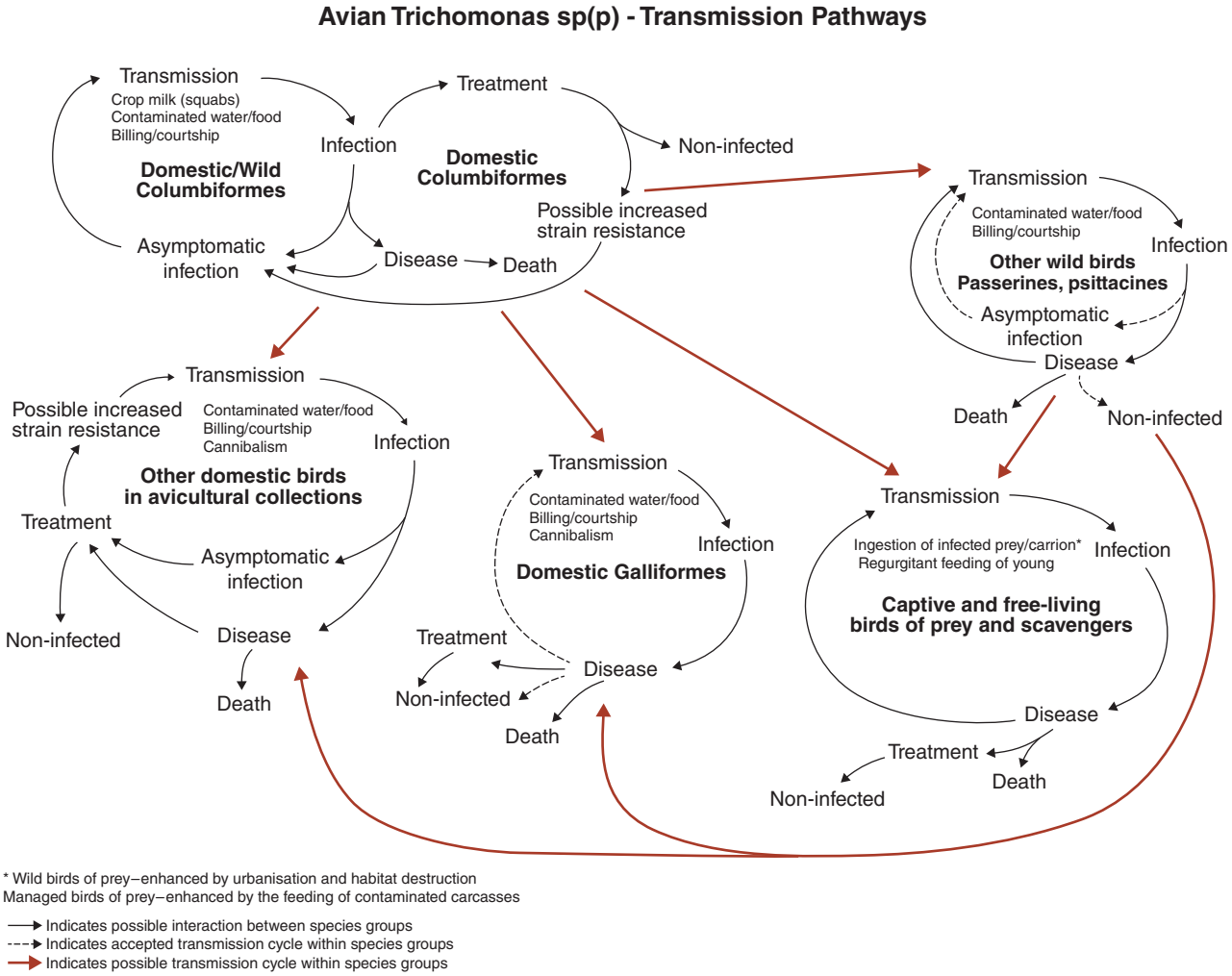


FIGURE 22.3 Avian *Trichomonas* spp. transmission pathways, outlining the most commonly encountered species groups and the possible and accepted transmission pathways between and within these groups.

feed on, or are fed carrion from doves and pigeons. Trophic transmission of *T. gallinae* has been documented in raptors that feed heavily on infected prey such as mourning doves *(Zenaida macroura)* and rock pigeons. Raptor nestlings may then become horizontally infected with trichomonads from infected parents or from being fed infected prey (by their parents).

Spillover is also reported in Passeriformes that share feeding stations or water sources with Columbiformes. Such spillover is presumably enhanced by unnaturally high stocking densities. Recently, a specific *T. gallinae* A1 subtype has caused mortality in finches in the United Kingdom and continental Europe. In these outbreaks, there appears to be a prominent seasonal pattern, which starts in July and peaks in late summer or early autumn.[11–13] To date, no studies have examined long-term climatic data and its association with *Trichomonas* infections, but dry weather and warmer temperatures have been identified as risk factors for the emergence of trichomoniasis in finches and doves.[14,15] During times of drought or stress, when activity at water sources becomes more concentrated and water becomes contaminated, infection rates in species such as the band-tailed pigeon *(Patagioenas fasciata)* have risen, causing mass mortality events.[16] *Trichomonas* spp. survival outside the host is temperature dependent. *Trichomonas vaginalis* survives exposure at temperatures near the freezing point for 96 to 144 hours and at 24°C to 25°C (75.2°F–77°F) for 120 to 154 hours. Exposure to −72°C (−97.6°F) for 1 minute destroys the organisms. The thermal death time for temperatures above 43°C (109.4°F) has been recorded. At 50°C (122°F) it is 4 minutes. The lowest temperature consistent with multiplication is 25°C (77°F); the upper limit 41°C (105.8°F).[4] The minimum time required for cell division is 5 to 8 hours and was observed over a broad range from 34°C to 39°C (93.2°F–102.2°F). Because of this, transmission via an indirect route is far less likely to occur during seasons or in climates where temperature precludes the survival of the parasite in water sources or in carcasses being scavenged. It stands to reason that niche sharing with Columbiformes, geographical distribution, and stressors during major life events (e.g., breeding, migration, molting) increase transmission opportunities and may contribute to the successful transmission of the *Trichomonas* parasite.

PATHOGENESIS

Infection with *Trichomonas* spp. results in various disease syndromes that depend on the host-pathogen interaction. Many columbid species are asymptomatic carriers of *Trichomonas* spp. and these birds are often infected by the parents with the endemic strain of the parasite as day-old chicks. Several studies indicate a wide spectrum of virulence in *Trichomonas* spp. Birds can succumb to infection with a single, highly virulent trichomonad isolate within 14 days postinfection, whereas they fail to seroconvert when exposed to 1×10^6 organisms of avirulent strains. Clinically normal pigeons can harbor both avirulent and virulent strains, although naive doves and pigeons challenged with mixed strains will succumb to infection.[17] With highly virulent strains, mortality may be as high as 50% before sufficient protective immunity develops and can become as high as 80% to 90% in young squabs that have not yet fully developed a functional immune system.[18] Analysis of clones of strains of variable virulence shows that there is significant variation in the cell membrane proteins expressed; in strains with the highest virulence, proteins related to cell adhesion were more abundant, whereas the strains with the lowest virulence displayed large numbers of proteins associated with carbohydrate metabolism.[19]

The variability in virulence is likely related to a multitude of factors, but recent work has identified that genetically different *T. gallinae* isolates caused a diverse magnitude of a cytopathogenic effect on permanent chicken liver cell and permanent quail fibroblast monolayers. The damage to these monolayers in vitro is thought to be the result of cysteine peptidases produced by the parasite.[20] This pathogenesis is well documented in the closely related *T. vaginalis*, the causative agent of a human sexually transmitted infection. Cysteine peptidases have been associated with nutrient acquisition, hemolysis, and evasion of the host immune response; they have been implicated in the virulence, the cytotoxicity, the adherence to host cells, and the detachment of host cells. *T. vaginalis* cysteine peptidases found extracellularly (as has been shown to occur in *T. gallinae*) are involved in the degradation of the mucin layer covering epithelial cells and in the subsequent apoptosis of epithelial cells, indicating an important role in the virulence of this parasite.[20]

CLINICAL SIGNS

Clinical signs associated with trichomoniasis vary across species. In Columbiformes, lesions and clinical signs largely depend on the virulence of the infecting strain and the age of the pigeon. Most endemic strains are of low virulence, and the majority of pigeons infected with *Trichomonas* spp. do not show macroscopic lesions. Early gross lesions in the oral cavity consist of irregular yellow plaques and exudate on the mucosa of the soft palate. Small, yellowish, circumscribed areas can increase in number and become progressively larger, finally developing into very large, caseous masses that may invade the roof of the mouth and even extend to the sinuses (Figure 22.4A). Early lesions in the esophagus and crop may be visible via endoscopy and may be palpable if they have progressed to thick necrotic masses. Squabs may be failing to thrive, show poor growth rates and weakness, and, in addition to previously mentioned lesions, show yellow plaques in the umbilical region (Figure 22.4B). Infected nests may be wet when compared with an uninfected nest—the result of the parents developing polydipsia and consequently regurgitating more watery feed to their young.

Raptors suffering from trichomoniasis develop similar signs to those mentioned previously but may also present with flicking food and regurgitating because of the inability to swallow. Birds of prey appear to be particularly susceptible to *Trichomonas* sinusitis with invasion of the choanal cleft and sinuses leading to open-mouth breathing, nasal discharge, decreased flying performance, and respiratory distress. There have also been reports of vultures presenting with clinical signs including disorientation and depression, as well as emaciation due to dysphagia.[4]

In passerines infected with *Trichomonas*, diseased birds will often be fluffed up and perching in a huddled position with signs including gagging, regurgitation, stretching of the neck, and greenish to yellow nasal and oral discharge (Figure 22.4C). In advanced cases, respiratory distress, emaciation, weakness, and death may be seen. There is evidence of asymptomatic infection in some passerine species.[21] Similar signs are reported in psittacine birds, with budgerigars *(Melopsittacus undulates)* being the most commonly infected species. Diseased birds often present with thick, mucus-covered feathers over the top of

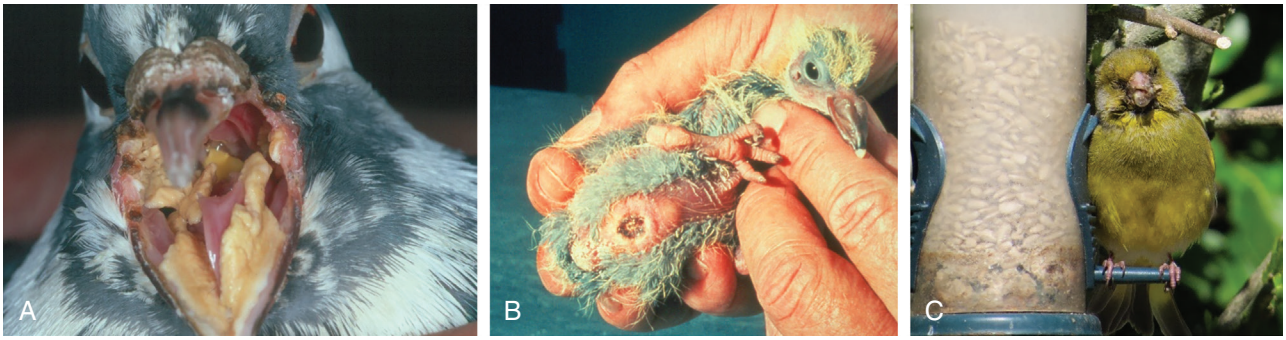


FIGURE 22.4 Clinical signs of trichomoniasis. (A) Characteristic caseous plaques in the oral cavity and glottis of a stray homing pigeon *(Columbia livia)* caused by *Trichomonas* spp. (B) A pigeon squab showing an umbilical lesion caused by *Trichomonas* spp. (C) Fluffed-up European greenfinch *(Chloris chloris)* showing greenish-yellow mucous discharge and bubbles, huddled on a backyard seed feeder. (A, B, Photos courtesy Nico Schoemaker/Yvonne van Zeeland. C, Photo courtesy Yne ten Wolde.)

their head from vomiting/regurgitation, bubbles, or viscous clear mucous from the lateral aspects of the beak with white or yellow plaques occasionally visible within the oral cavity. In budgerigars, there often is palpable thickening of the crop mucosa present on examination. If the budgerigar was infected as a young bird, it may show poor growth and will often have green diarrhea and show signs of ataxia and emaciation depending on the stage of disease progression.

In summary, in severe infections, all species may develop large caseous granulomas in the upper gastrointestinal tract, choanae, or extending into the periorbital sinuses, leading to partial or complete obstruction of the upper gastrointestinal tract and respiratory distress, and causing severe halitosis, emaciation, and starvation.

PATHOLOGY

Gross Lesions

The preferred site for *Trichomonas* spp. is the upper digestive tract including the mouth, pharynx, esophagus, and crop (Figure 22.5). The flagellate is also able to enter the respiratory sinuses and invade the neck, nasopharynx, brain, and infraorbital sinus and its periorbital diverticula (Figure 22.5B). Systemic trichomoniasis involving the liver, lung, heart, pancreas, air sacs, and pericardium, as well as focal trichomoniasis of the umbilicus, has been documented. It is likely that infection with and severity of these atypical lesions are attributed to co-infections or secondary infections with other etiologies such as bacteria or viruses. Degenerative lesions have also been documented in the kidney and genitalia of pigeons infected with a virulent strain.[22] In one incident, pigeons had trichomoniasis with bacterial co-infections in the trachea and lung, presenting as necrotizing and hemorrhagic tracheitis with an obstructive, tracheal plug and fibrinopurulent pneumonia, but a lack of gross and microscopic lesions in the upper digestive tract.[23] The severity of pathologic lesions of *T. gallinae* strains with moderate virulence in the upper digestive tract ranges from a mild catarrhal inflammation of the oral mucosa, throat, and crop to caseous necrosis of the proximal esophagus, crop, and thoracic esophagus that may progress to obstruct and occlude the esophageal lumen, often resulting in weight loss or starvation. Birds infected with avirulent strains will often develop no visible signs in gross pathology.

Experimental infection of pigeons with the virulent *T. gallinae* Eiberg strain created necrosis of the epithelial cells and submucosa of the oropharyngeal regions by day 5 postinfection.[22] Purulent exudates containing mainly heterophils were noticed in the oropharynx, crop, and lungs after 1 week. The authors also noted that this virulent strain also resulted in diphtheritic membranes and fibrinous lesions in internal organs such as the liver, lungs, and peritoneum, resulting in high mortality.[22] Liver lesions are uncommon but typically superficial and can extend into the parenchyma as solid, white to yellow circular or spherical masses.[18]

There are significant differences in the pathology seen in columbid populations in which a *Trichomonas* strain is endemic and species that are naive to the parasite. The pathology present in the naive species can be so severe that it results in mortality, while columbids that appear to be infected by the same strain are seemingly healthy.[13] In greenfinches *(Chloris chloris)*, goldfinches (*Carduelis* spp.), house finches *(Haemorhous mexicanus)*, and other passerines, some birds have been found to have no macroscopic lesions after being infected and dying with virulent *T. gallinae*.[24,25]

Trichomonas infections may readily be confused with some other pathological conditions that result in similar plaques in the oral cavity. Conditions resulting in similar lesions include infections with avian poxviruses, fungi (*Candida* sp., *Aspergillus* sp.), and nematodes (*Capillaria* sp.), salmonellosis, mycobacteriosis, nonspecific bacterial or fungal pharyngoesophagitis, oral neoplasia (squamous cell carcinoma), and vitamin A deficiency (especially in young passerines). Systemic spread of trichomonads, involving multiple organ systems should be differentiated from pigeon herpesvirus, avian paramyxovirus, or fowl adenovirus, while navel *Trichomonas* should be differentiated from bacterial infection.

Histopathology

Histopathological changes associated with *T. gallinae* in the upper digestive tract include hyperplastic mucosa with necrotic pseudomembranes and multifocal, superficial erosions (Figure 22.5E) to deep, ulcerative necrosis of the epithelium. Macrophages and plasma cells predominate the inflammatory response, but in acute phases, when tissue invasion by the organisms occurs, there may be a marked heterophilic response. Numerous pale eosinophilic, ovoid to piriform trichomonads with a round basophilic nucleus may be present in secretions and on the mucosal surface of the oropharynx (Figure 22.5F). Ulceration of the mucosa with a severe transmural heterophilic inflammation is well established by the fourth day of experimental infection with a virulent strain.[22] Caseous granulomas may also be present in the lung, liver, in the intestinal mesentery, and around the heart. A mixed population of inflammatory cells predominated by heterophils surrounds necrotic areas with occasional epithelioid macrophages and giant cells. In granulomatous lesions, trichomonads appear to be most numerous at the periphery. Passerines such as greenfinches that have died from trichomoniasis may have no macroscopic lesions but will have microscopic lesions similar to those described previously, resulting in obstruction or hemorrhagic diathesis.[24]

In tissues, hematoxylin and eosin (H&E) and periodic acid-Schiff (PAS) stains were proven to be of limited use for identification of the flagellates, especially in organs that contained only a few protozoal cells.[26] Although the technique is complex and interpretation may be unreliable, Bodian silver stain may potentially be used to facilitate detection of the organism, as this stains the organism purple to black.[23]

DIAGNOSIS

History, signalment, clinical signs, and salient gross lesions are highly suggestive of trichomoniasis and can be confirmed by microscopic observation of organisms in direct wet smears from the mouth or crop. Finding characteristic motile trophozoites, moving briskly in a jerky, spastic motion in fresh wet preparations, is diagnostic. Samples include swabs obtained from the crop, oral cavity, or conjunctiva. Organisms are most readily observed when examined immediately following collection, ensuring the stage diaphragm is down. Examination at 100× and 400× will facilitate the best opportunity for visualization.

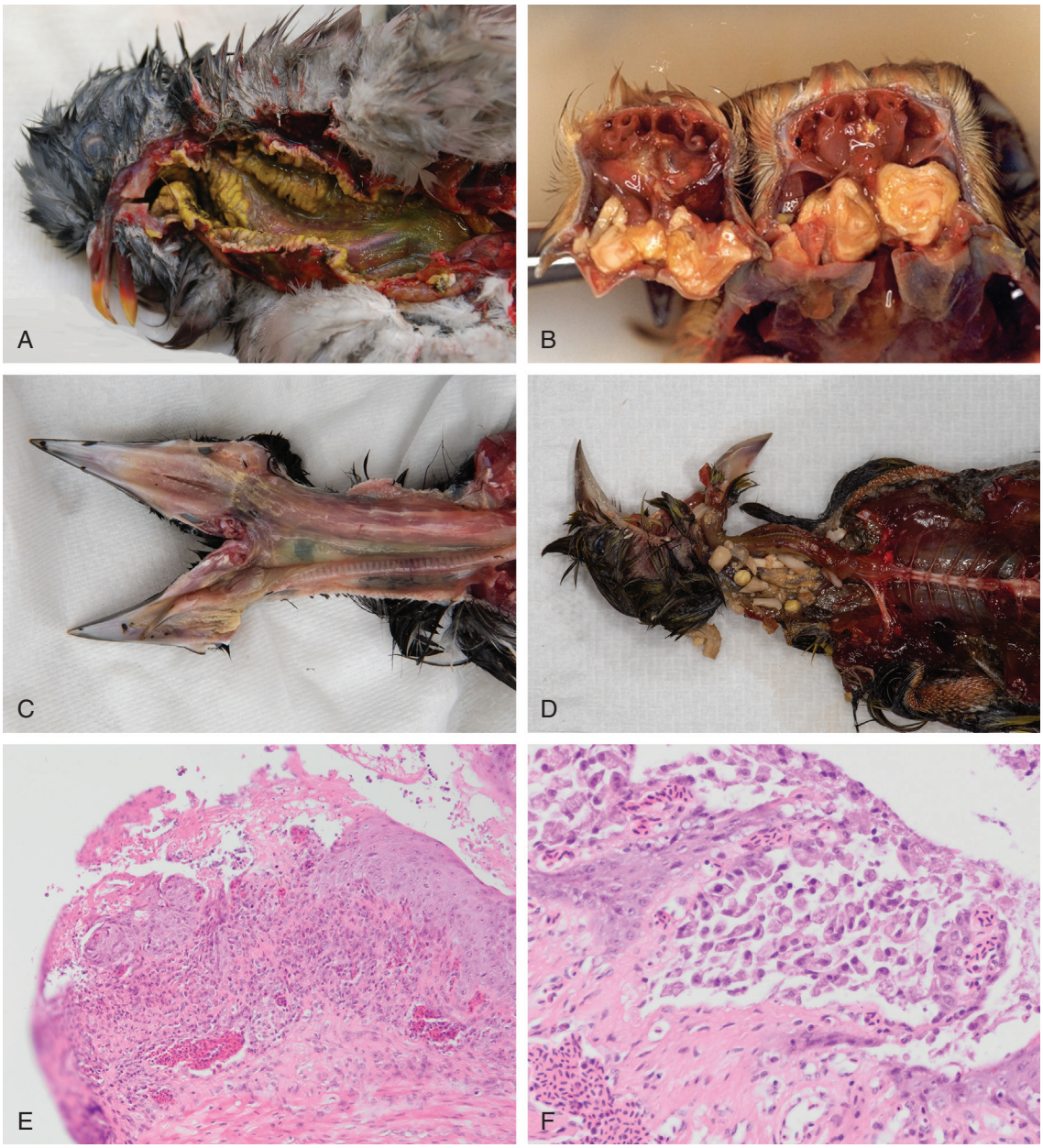


FIGURE 22.5 Trichomoniasis gross pathology and histology. (A) Severe necrotic lesions in the upper digestive tract of a wild woodpigeon *(Columba palumbus)*. (B) A transverse section at the base of the beak of a female saker falcon *(Falco cherrug)* showing large bilateral caseous plugs obstructing the periocular sinuses. (C) Mild necrotic lesions in the upper digestive tract of a magpie *(Pica pica)*. (D) Necrotic lesions and stuck seeds in the crop of a European greenfinch *(Chloris chloris)*. (E) Ulceration of the crop of a greenfinch due to *Trichomonas* infection, hematoxylin and eosin stain, 20×. (F) Trichomonads (trophozoites) invading the crop of a greenfinch, hematoxylin and eosin stain, 40×. (A, Photo courtesy Dutch Wildlife Health Centre, DWHC. B, Photo courtesy Jaime Samour. C, D, Photos courtesy DWHC. E, F, Photos courtesy Marja Kik, DWHC.)

However, the sensitivity to detect trichomonads in wet mount preparations is significantly lower than culture techniques, especially if the number of parasites in the host is marginal. When suspected cases are negative for motile trophozoites, or during research and disease investigations, a variety of molecular and microbiological techniques can be used to further characterize the pathogenic organisms.

Samples collected for culture should be inoculated into an appropriate culture media and stored at room temperature; common commercial media include InPouch TF (Biomed Diagnostics, White City, OR, USA) or Diamond's commercial media (Thermo Fisher Scientific, Scoresby, Australia). Culture of *Trichomonas* spp. is carried out using trypticase-yeast extract-maltose and various other *Trichomonas*-specific media, with or

without the addition of antibiotics. Optimal growth conditions include a water bath temperature of 35.5°C, but lower temperatures prolong sample viability. Culturing *Trichomonas* facilitates further molecular analysis and DNA extraction with a higher concentration of organisms and higher-quality parasite DNA.

Polymerase chain reaction (PCR) to detect trichomonads is common. The most readily available reactions include the ITS1/5.8S rRNA/ITS2 region used for species confirmation in a diagnostic setting, the *FeHyd* gene used for strain variability in research settings and nested PCR for the small subunit *(SSU)* rRNA gene. Rapid detection kits, developed for the identification of positive *T. vaginalis* samples, have been used in the diagnosis of *T. gallinae*. The OSOM Trichomonas Rapid Test (SEKISUI Diagnostics, Kent, UK) has been demonstrated as being sensitive and specific for *Trichomonas* spp. However, while suitable for the rapid detection of trichomonads, the test is not able to distinguish between *Trichomonas* spp.[27]

Immunohistochemistry using a rabbit polyclonal antibody that targets the outer membrane of *Trichomonas* genus with antigen-specific antibody has been described in literature. Although this stain is not specific for *T. gallinae*, staining correlated with areas with trichomonad regions observed on H&E.[23] In situ hybridization was found to be more sensitive than histochemical staining (such as PAS and H&E), especially in tissues with small numbers of the parasite. The protozoa were clearly visible as dark blue cells, which allowed the demonstration of cells in different organs and locations within the affected tissue, helping to correlate the histological changes with the presence of the protozoon.[26]

TREATMENT

Various nitroimidazoles, including metronidazole (10–30 mg/kg PO BID for 10 days), dimetridazole (666 mg/L drinking water for 7–12 days), ronidazole (1 g/L drinking water for 6 days), and carnidazole (20–30 mg/kg tablet PO once) are the standard treatment for trichomoniasis in birds (see Chapter 9). Historically, these therapeutic options have been used effectively against flagellate infections, but several have subsequently been banned (see Table 24.1). Metronidazole is the drug of choice for the treatment of *T. gallinae*, but in some countries the law prohibits its use. Advanced clinical cases may require surgical debridement of caseous lesions. Surgical excision using a curette to remove granulomas from the choanae, or oral cavity, should be carried out with extreme care, as these lesions are prone to hemorrhage. An ingluviotomy approach can be performed to allow for surgical debulking of lesions in the esophagus or crop. Following opening of the crop, the affected mucosa can be exteriorized and isolated to facilitate the debulking. Once resected, the crop wall can be closed using simple continuous sutures before the skin is closed. While the success rate is not documented, treatment of advanced disease states, which may lead to complete occlusion of the esophageal lumen, carries a poor prognosis.

Treatment in young pigeons is usually limited to those showing clinical disease in the nest. These birds can be treated using carnidazole tablets by mouth. In severe cases where granulomatous lesions preclude the administration of oral tablets, a dimetridazole or ronidazole "paste" can be prepared using the in-water medication that can be applied topically onto the oral cavity lesions with a cotton tip. In infected squabs, it is common practice to also treat the nest-mate and the parents using oral carnidazole tablets.

There is an increasing body of evidence, as well as anecdotal reports, indicating nitroimidazole resistance, with resistant isolates of *T. gallinae* having been reported in Belgium, Spain, the United States, and Iran (see Chapter 9).[8] To prevent subclinical disease-causing decreased racing performance, nitroimidazole drugs are routinely administered to racing pigeons for 3 to 4 days each month during the racing season. This prolonged exposure to potentially subtherapeutic doses of nitroimidazole creates a suitable environment for resistant strains to develop; a phenomenon that is well documented in related trichomonads, *T. vaginalis*.[28] This resistance is now documented in budgerigars and racing pigeons, where clonal cultures of *T. gallinae* obtained from each species had significantly different minimal lethal concentrations (MLCs) against four 5-nitroimidazoles (Table 22.1).[29]

Development of resistance is a concern; not only because the nitroimidazoles provide the mainstay for current therapy but also because of the husbandry practices of racing pigeons that are most likely to carry resistant strains of *Trichomonas*. These birds are driven long distances and released during races, and also released from their loft twice daily during training. If they do not return to their loft, they will sometimes join groups of feral pigeons in city parks or areas of high avian density, where food is readily available. Here, the potential for spread of resistant strains to wild birds increases. *T. gallinae* isolates, with similar nitroimidazole resistance profiles detected in racing pigeons and wild pigeons in Belgium, provide support for the exchange of resistant strains.[30]

In wild birds, treatment is much more problematic and not recommended.[31,32] Studies have shown variable success with in-water medications with possible suboptimal dosing, which increases the risk for the development of resistance.[33,34] Variable intake of food and water may have detrimental effects on nontarget bird species that share the feeding stations. This is especially true for dimetronidazole, where intake of high doses is also potentially toxic to birds.[35] In practice, treatment is only feasible when individual wild birds are presented for veterinary assessment.

Despite nitroimidazole-resistant strains of *T. gallinae* becoming more frequently reported, there is only a small body of research investigating alternative treatment modalities.[36] There has been recent work evaluating various essential oils and natural remedies in vitro and in vivo. There is evidence that essential oil of *Artemisia sieberi* or *Pelargonium roseum*, wild rue *(Peganum harmala)* alkaloid extract, and aqueous extract of garlic *(Allium sativum)* may act as potent natural antitrichomonal agents in vitro and in vivo in pigeons.[37–40] Essential oils of East Indian lemongrass *(Cymbopogon flexuosus)* were tested in quails.[41] Based on in vitro results, other possibly promising extracts and pure compounds include some from the curry leaf tree *(Murraya koenigii)*, pitanga *(Eugenia uniflora)*, pepper fruit *(Dennettia tripetala)*, the fool's curry leaf tree *(Clausena lansium)*, ginger *(Zingiber officinale)*, lavender *(Lavandula angustifolia)*, and other Lamiaceae.[36,42] Pure compounds of Lamiaceae plants with good antitrichomonal activity and limited cytotoxicity in vitro, included linalyl acetate, thymol, 4-terpinenol, γ-terpinene, *p*-cymene, or D-fenchone.[42] Further work is required to establish whether safe and therapeutic doses of treatments based on

TABLE 22.1

Minimal Lethal Concentrations of Metronidazole, Dimetridazole, Ornidazole, and Ronidazole for the Six Different Cloned Xenic *Trichomonas* Cultures Investigated in the Present Study

Cloned Cultures	Minimal Lethal Concentrations (MLC) of the Test Compounds (μg/mL)				Classification After Phylogenetic Analyses
	Metronidazole	Dimetridazole	Ornidazole	Ronidazole	
Trichomonas gallinae/budgerigar/Austria/5895-C1/06	2.0 ± 0.3	2.5 ± 0.3	6.7 ± 1.7	6.7 ± 1.7	*T. gallinae*-like
T. gallinae/budgerigar/Austria/15935-C3/06	2.7 ± 0.3	3.0 ± 0.7	2.0 ± 0.6	2.7 ± 0.3	*T. gallinae*-like
Trichomonas spp./racing pigeon/Austria/231-C1/07	8.8 ± 1.3	5.0 ± 0.0	16.7 ± 1.7	5.0 ± 0.0	*Trichomonas* sp.
Trichomonas spp./racing pigeon/Austria/231-C3/07	28.3 ± 1.7	33.3 ± 3.3	**76.7 ± 3.3**	**65.0 ± 2.9**	*T. tenax*–like
Trichomonas spp./racing pigeon/Austria/7895-C2/06	25.0 ± 2.9	36.7 ± 3.3	**73.3 ± 6.7**	**61.7 ± 1.7**	*T. tenax*-like
T. gallinae/racing pigeon/Austria/8855-C6/06	**103.3 ± 3.3**	**83.3 ± 6.7**	**103.3 ± 3.3**	**83.3 ± 6.7**	*T. gallinae*-like

Values are given as mean ± Standard error of mean of three independent experiments performed in duplicate. Values (in columns) having no common superscript are significantly different at $p \leq 0.05$; values in bold indicate resistance (= MLC ≥ 50 μg/mL) of the isolate to the respective drug.

Modified from Zimre-Grabensteiner E, Arshad N, Amin A, Hess M: Genetically different clonal isolates of *Trichomonas gallinae*, obtained from the same bird, can vary in their drug susceptibility, an in vitro evidence, *Parasitol Int* 60(2):213–215, 2011. https://doi.org/10.1016/j.parint.2011.02.003.

natural products from such plants can be consistently effective against trichomonads.

PREVENTION

The prevention of clinical disease outbreaks should be considered as an essential aspect of treatment. Identification and elimination or treatment of carrier birds in avicultural collections, alongside regular cleaning and disinfection of drinkers and drinking water, will help to prevent disease spread. The provision of a dry environment and preventing moisture buildup in the aviary will assist in preventing transmission. This may be achieved by using nipple drinkers, covered water containers, or by natural or artificial ventilation and heat. Wild birds should have no access to water or unfinished seed (especially spoiled or moist seed where *Trichomonas* can survive for up to 24 hours) from the avicultural collection. To prevent infection in raptors or birds of prey, the feeding of frozen or parasite-free pigeon carcasses, or seeking alternative food sources, is the most effective course of prevention. Feeding farmed quail, chickens, or rodents as a less susceptible protein alternative will help to prevent the introduction of disease. Where there is a history of disease, or in collections that are highly susceptible to infection, routine health checks and prophylactic treatment with in-water ronidazole during breeding, weaning, and at other susceptible times of the year are recommended.

Management of trichomoniasis in avicultural collections depends on reducing stress in birds and removing severely affected birds from the flock. In Columbiformes, establishment of carrier states and humoral immunity has been shown to have a protective effect postexposure in adults. There is no vaccine available for this parasite; however, future studies looking into immunity may be of help due to potential protective effects from humoral immunity. Murine model studies looking at *T. vaginalis* vaccines for humans have been explored. However, despite some success in controlling symptoms or duration, none of them were able to achieve clearance of the parasite. Since the organism is an extracellular parasite, cell-mediated immunity will likely be less effective than a humoral response in triggering immunoglobulin-dependent elimination of the parasite,[43] hence rendering the development of an effective and successful vaccine challenging. For a related parabasalid, *Trichomonas foetus*, there is a commercial vaccine (TrichGuard; Boehringer Ingelheim Animal Health USA, Duluth, GA, USA). However, while routine vaccination strategies in asymptomatic bulls help to reduce shedding, it does not protect the animals from infections with the organism.[44]

To prevent disease outbreaks in wild birds, limiting the unnatural congregation and mixing of species at feeding stations or communal watering points will help to limit transmission of disease. However, such congregations may not always be preventable, for example, during times of drought, which will result in birds congregating at the few remaining water sources. Under these conditions, providing supplementary watering points with clean water may help to maintain sanitary conditions and limit transmission between and within species. Similarly, if feed must be supplied, it should be done in moderation (equivalent to daily consumption) and any feeder cleaned and disinfected daily. If different types of feed are offered, these should be supplied in different locations to avoid unnatural mixing of species.[32]

CONSERVATION AND ONE HEALTH CONSIDERATIONS

Trichomoniasis can be a leading cause of death in wild avian species, whether columbids[45,46] or finches.[23,24] It can contribute to the decline of a species, as likely seems to be the case for the threatened turtle dove *(Streptopelia turtur)* and the

band-tailed pigeon.[47,48] The parasite may even drive population declines in naive populations of novel host species. Numbers of greenfinches are declining in North and Western Europe since *T. gallinae* was first detected in the species in 2005, and this trend shows no indication of leveling off yet.[32,49] Models have shown that endangered populations can be driven to extinction by repeated exposure to a pathogen from the reservoir host species,[50] and such a scenario is not to be excluded for species exposed to trichomonads.

Besides contributing to or causing species decline, trichomoniasis can also be a substantial threat to the recovery of species, as exemplified with the endangered pink pigeon *(Columba mayeri)* in Mauritius. In this species, *T. gallinae* was demonstrated to be the most significant pathogen hampering species' recovery by reducing both reproductive success (being a prime cause of squab mortality) and adult survival (the more persistent the infection, the shorter the adult bird survival).[46,51,52] This led to initiation of (preventive) treatment during the initial recovery phase to ensure pink pigeon squab survival.[51] Treatments have also been used to ensure the local survival of raptor nestlings, such as golden eagle *(Aquila chrysaetos)* nestlings.[53]

The trichomoniasis context is constantly evolving, partly through anthropogenic influence. The effects of human perturbation of host habitat and food sources on *Trichomonas* transmission and trichomoniasis occurrence have been documented to some extent. The practice of supplementary feeding of game birds, which promotes the congregation of columbids, has been shown to increase the risk of *Trichomonas* infection and the occurrence of trichomoniasis outbreaks in these species.[31,54] The year-round feeding of garden birds with multiple types of seed, which supports aggregation and bird species mixing, has most likely altered *Trichomonas* transmission dynamics and promoted cross-species disease transmission events from columbids to finches.[32] The advent of a more urban environment and the loss of native habitat and prey have caused some raptor populations to substantially increase the proportion of synanthropic columbids in their diet, and with this the probability of *Trichomonas* infection.[53,55,56] The translocation of columbids from aviaries into the wild for restocking purposes has introduced novel *Trichomonas* strains into habitats, such as occurred in a raptor recovery program.[57]

Should *Trichomonas* infecting birds, humans, and mammals be shown to be more of a continuum as perceived by some authors,[6,58] then disease emergence risk and the development of nitroimidazole resistance require closer collaboration among human, animal, and ecosystem health scientists. This would, among others, require better insight into the scale of nitroimidazole treatments in (racing) pigeons and surveillance for nitroimidazole-resistant strains.

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CHAPTER 23

Diplomonad Protozoal Diseases: Giardiasis, Spironucleosis

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PROTISTS

Protists are a diverse group of unicellular eukaryotes that are taxonomically considered separate from animals, plants, and fungi. Protists include protozoa, unicellular algae, and slime molds. The protist supergroup Excavata contains species that are morphologically asymmetrical and have a feeding groove that is "excavated" on one side. Most excavates are flagellated protozoa that are either free-living or parasitic and feed on organic matter such as organic debris, other microorganisms, or host tissues.[1]

FORNICATES

Excavates in the clade Fornicata possess one or two karyomastigonts per cell. A karyomastigont system consists of a single nucleus with four basal bodies (kinetosomes). Each basal body is typically associated with a single flagellum, and therefore there are generally four flagella per karyomastigont and eight per cell.[1,2] All fornicates are anaerobic or microaerophilic.[1] Fornicates lack classical mitochondria but possess mitochondria-related organelles (MROs), which are organelles that are believed to have evolved from mitochondria in certain eukaryotes that live in low-oxygen environments.[1–4] The Fornicata clade consists of four lineages: free-living *Carpediemonas* and *Dysnectes*, the retortamonads (almost exclusively parasitic), and the diplomonads.[1,2,5]

DIPLOMONADS

Diplomonads are mostly parasitic, but some species are free-living.[1,2,5] Most diplomonads ("two units") are diplozoic, meaning their cells contain two apparently symmetrical karyomastigonts.[1–3] Based on morphologic features such as the presence or absence of cytostomes (cell "mouth" supported by microtubules), diplomonads have been divided into the monophyletic groups Giardiinae and Hexamitinae.[1,2]

Giardiinae Diplomonads

Giardiinae diplomonads consist of the genera *Giardia*, *Brugerolleia*, and *Octomitus*.[1] These diplomonads lack cytostomes, and their recurrent flagella pass through the cytoplasm as naked axonemes.[1,2] Among the Giardiinae, the ventral adhesive disc is unique to *Giardia*.[2] *Giardia* has been widely reported in birds, but to date there are no confirmed reports of infection with *Brugerolleia* or *Octomitus* in avian species.

Giardia

Giardia trophozoites are pear-shaped with a convex dorsal and a concave ventral side (Figure 23.1).[3] Trophozoites range from 10 to 20 μm in length and 5 to 15 μm in width.[3,6] The *Giardia* cytoskeleton contains several important microtubule structures including the ventral disc, three pairs of dorsal flagella, one pair of ventral flagella, the funis, and the median bodies.[3,6] The morphological appearance of the median bodies has historically been used to assist with the taxonomic classification of different *Giardia* species.[6,7] Cysts are oval to egg-shaped and range from 10 to 14 μm in length and 8 to 10 μm in width.[2,3,6,8] Cysts have a thick wall that contains a double membrane and contain 2 to 4 nuclei.[2,3,6] Cysts from almost all *Giardia* species appear similar by light microscopy.[9]

A total of eight *Giardia* species are currently recognized: *G. duodenalis* (syn. *G. intestinalis*, *G. lamblia*), *G. psittaci*, *G. ardeae*, *G. muris*, *G. agilis*, *G. microti*, *G. peramelis*, and *G. cricetidarum*.[3,10] The major hosts for *G. duodenalis* are mammals, and *G. duodenalis* is the only *Giardia* species known to infect humans.[10,11] All other *Giardia* species are found in animal species including birds, amphibians, and mammals.[10] *G. duodenalis*, *G. psittaci*, and *G. ardeae* are the only *Giardia* species currently known to infect birds.[10–12] There are no confirmed reports to date of zoonotic transmission from birds to humans for any *Giardia* species.[10,13]

Giardia duodenalis. *G. duodenalis* is a species complex that consists of eight genotypes with specific host preferences, categorized as assemblages A through H (Table 23.1).[3,11] Assemblages A and B are the most dominant assemblages in humans.[2,10,14] Assemblages A and B are also found in other vertebrate species and therefore have zoonotic potential.[2,3,10] Assemblages A and B have been reported in point prevalence studies of fecal shedding in wild birds including the mallard duck *(Anas platyrhynchos)*, European quail

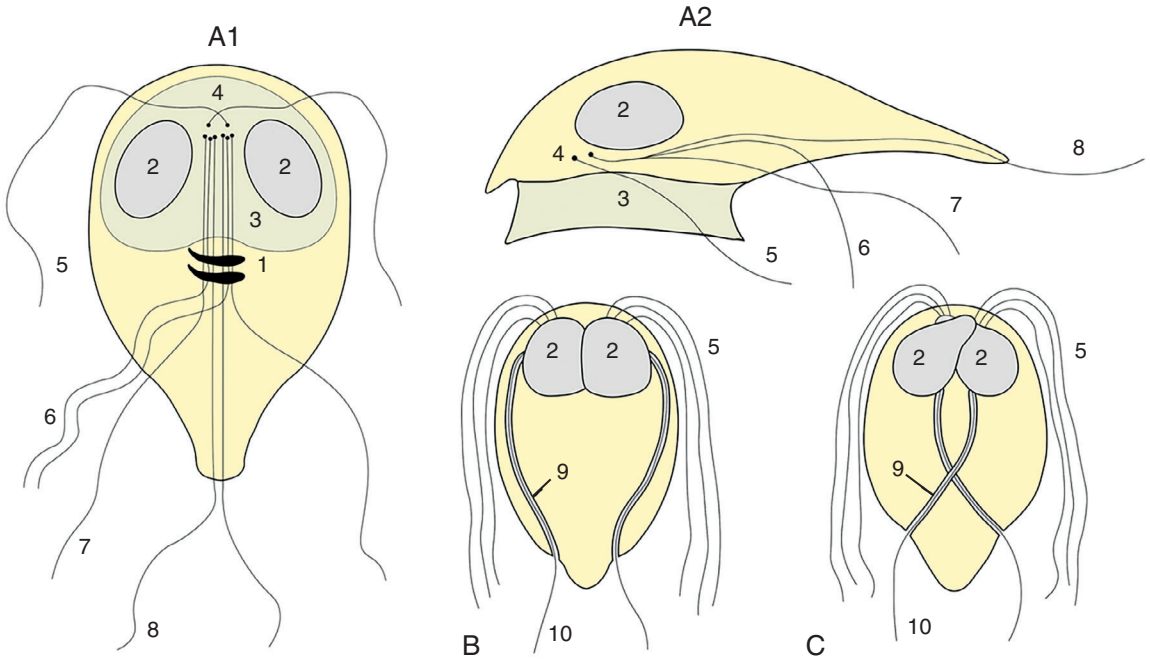


FIGURE 23.1 Morphological differences between *Giardia* (A1, A2), *Hexamita* (B), and *Spironucleus* (C). *1*, Median bodies. In *G. duodenalis* and *G. ardeae*, the median bodies appear as a "clawhammer" shape. In *G. psittaci*, the median bodies are rod-shaped and pointed but similar in appearance to *G. duodenalis* and *G. ardeae* by light microscopy. *2*, Nucleus. The nuclei in *Hexamita* are generally round, while the nuclei in *Spironucleus* are somewhat spiral-shaped. *3*, Ventral disc. The marginal groove (not illustrated here) around the ventral disc is incomplete in *G. psittaci* but complete in *G. duodenalis*. *4*, Basal bodies. *5*, Anterior flagellum. *6*, Ventral flagellum. *7*, Posterior flagellum. *8*, Caudal flagellum. *9*, Cytostome (cell "mouth"). *Giardia* species lack cytostomes. *10*, Posterior (or recurrent) flagellum. The posterior flagella and associated cytostomes travel laterally caudal to the nuclei in *Hexamita* and medially caudal to the nuclei in *Spironucleus*. (Illustration by Lily Baine © 2020, courtesy Lauren Powers.)

(Coturnix coturnix), European turtle dove *(Streptopelia turtur)*, European nightjar *(Caprimulgus europaeus)*, Eurasian magpie *(Pica pica)*, and common buzzard *(Buteo buteo)* in Spain[15,16]; a gadwall *(Anser strepera)* from a lake catchment in Hungary[14]; and gulls (*Larus* spp.) from Dennis, MA, USA, and from Appledore Island in Maine.[17] Assemblages A and B have also been reported in surveys of domestic birds including ducks and chickens *(Gallus gallus domesticus)* living in closed enclaves in the Ivory Coast[18]; domestic graylag geese *(Anser anser)* and domestic ducks from farms and a lake catchment in Hungary[14]; and birds kept in zoological collections including a toco toucan *(Ramphastos toco)* in Brazil.[19] Assemblages D, F, and H have been reported in surveys of wild aquatic and nonaquatic birds, including the mallard duck and the Eurasian jay *(Garrulus glandarius)* in Spain[12,15] and a herring gull *(Larus argentatus)* from Dennis, MA.[17]

Assemblages A and B have been identified in psittacine birds kept as pets or in zoological collections in fecal shedding surveys, including a blue-and-gold macaw *(Ara ararauna)*, scarlet macaw *(Ara macao)*, blue-fronted Amazon parrot *(Amazona aestiva)*, and white-bellied caique *(Pionites leucogaster)* in Italy.[11] In this report, 5.3% of fecal samples from psittacine birds were positive for *G. duodenalis* by polymerase chain reaction (PCR).[11] *G. duodenalis* was also identified in sick wild-caught sulfur-crested cockatoos *(Cacatua galerita)* from Victoria, Australia, in a report published prior to the establishment of *Giardia* assemblages.[20] In this report, axenically cultured *G. duodenalis* trophozoites from cockatoos established infection in experimentally infected mice *(Mus musculus)*, affirming the concern for zoonotic potential.[20]

Giardia psittaci. The first report of *Giardia* in a psittacine bird was published in 1962 and described postmortem findings in a 2-year-old budgerigar *(Melopsittacus undulatus)* with poor body condition and chronic diarrhea.[21] In 1981, based on morphologic findings by light microscopy of *Giardia* isolated from sick budgerigars, it was suggested that budgerigar *Giardia* be classified as *G. duodenalis* race *psittaci*.[13] Trophozoites possess rod-shaped, pointed median bodies that are similar in light microscopic appearance to the clawhammer-shaped median bodies of *G. duodenalis*.[13,22] However, in 1987 it was proposed that the *Giardia* isolated from budgerigars be classified as its own species, *G. psittaci*, based on scanning electron microscopy morphologic findings including the lack of a complete marginal groove around the ventral disc.[22] More recently, partial molecular sequencing of *G. psittaci* from budgerigars[23,24] and lineolated parakeets *(Bolborhynchus lineola)*[23] supports the current taxonomic classification of *G. psittaci* as its own species. *G. psittaci* was also isolated and partially sequenced from 12 asymptomatic adult psittacine

TABLE 23.1

Giardia duodenalis Assemblages Referenced in This Text

Assemblage	Species Affected	Human Reports	Reports in Avian Species and Geographic Area
A	Mammals, birds[10]	Human reports are common.[10] Zoonotic potential	Four species of psittacine birds (blue-and-gold macaw [*Ara ararauna*], scarlet macaw [*Ara macao*], blue-fronted Amazon parrot [*Amazona aestiva*], white-bellied caique [*Pionites leucogaster*]) kept as pets or in zoos in Italy[11] Gadwall (*Mareca strepera*) from a lake catchment in Hungary[14] Mallard ducks (*Anas platyrhynchos*), common buzzard (*Buteo buteo*), and a European nightjar (*Caprimulgus europaeus*) from a wildlife center in Spain[15] Gulls (*Larus* spp.) from Cape Cod, MA, USA[17] Domestic duck (*A. platyrhynchos;* mixed A+B assemblage) and chickens (*Gallus gallus domesticus*) in the Ivory Coast[18] Toco toucan (*Rhamphastos toco*) kept in a zoo in Brazil[19]
B	Mammals, birds[10]	Human reports are common.[10] Zoonotic potential	Domestic graylag geese (*Anser anser*) and domestic ducks from farms in Hungary[14] Mallard ducks (*A. platyrhynchos*), European turtle dove (*Streptopelia turtur*), Eurasian magpie (*Pica pica*), and European quail (*Coturnix coturnix*) from a wildlife center in Spain[15] Aquatic birds (mixed population of 17 different avian species) from the Salburua wetlands in Álava, Northern Spain[16] Gulls (*Larus* spp.) from Dennis, Massachusetts, and Appledore Island, Maine[17] Domestic duck (*A. platyrhynchos;* mixed A+B assemblage) and chickens (*Gallus gallus domesticus*) in the Ivory Coast[18]
C	Mammals (e.g., dogs, cattle)[10]	Human reports in China and Slovakia[10]	None reported
D	Mammals (e.g., dogs, cattle, chinchillas), birds[10]	Single human report in German travellers[10]	Eurasian jay (*Garrulus glandarius*) from a wildlife center in Spain[15]
E	Mammals (e.g., hoofed animals, cats)[10]	Several reports[10]	None reported
F	Mammals (e.g., cats, pigs), birds[10]	Single report in Ethiopia[10]	Mallard duck (*A. platyrhynchos*) from a wildlife center in Spain[15]
G	Rodents[10]	None reported[10]	None reported
H	Pinnipeds (e.g., gray seal), birds	None reported[10]	Herring gull (*Larus argentatus*) from Dennis, Massachusetts[17]

birds from 6 genera after fecal sampling during a bird exhibition in Brazil.[12] In this report, 93/359 (25.9%) of psittacine birds were positive for *G. psittaci* by nested PCR including 2/18 (11.1%) cockatiels *(Nymphicus hollandicus)*.[12]

To date, *G. psittaci* has only been reported in psittacine birds.[12,13,21,22,24,25] Attempts at the transmission of budgerigar *G. psittaci* to canaries *(Serinus canaria domestica)* and mice were not successful in one report.[13] In a separate study, inoculation of budgerigar *G. psittaci* failed to produce infection in chicks, ducklings, Mongolian gerbils *(Meriones unguiculatus)*, or neonatal mice.[9]

Giardia ardeae. *G. ardeae* also has clawhammer-shaped median bodies, but the long axis of the clawhammer shape is parallel to the caudal flagellar axonemes rather than perpendicular as seen with *G. duodenalis*.[9,26] Ultrastructural imaging and partial molecular sequencing have been performed for *G. ardeae*, which support *G. ardeae* as a unique species.[7] *G. ardeae* has been reported in aquatic birds including the aquatic great blue heron *(Ardea herodias)*,[26] gray heron *(Ardea cinerea)*,[27] and straw-necked ibis *(Threskiornis spinicollis)*.[7,28] Inoculation of great blue heron *G. ardeae* failed to establish infection in chicks, budgerigars, Mongolian gerbils, and neonatal mice in one report.[9]

Giardiasis

Giardia is a common cause of gastroenteritis (giardiasis) in humans, livestock, and companion animals worldwide.[3,10,24,29–31] Infection occurs almost exclusively through the ingestion of cysts.[2,3,10,14,24,29] Cysts are immediately infectious when excreted in feces and are remarkably stable in the environment.[2,3,32] Excreted cysts can contaminate drinking water sources and recreational bodies of water.[10,14,29,32] After ingestion, excystation occurs after exposure to gastric acids and peptidases.[2,3] Excyzoites then divide into trophozoites that colonize the small intestine.[2,3] After disrupting the local microbiome and mucus layer barriers, trophozoites adhere to the intestinal epithelial microvilli using the ventral adhesive disc.[2,3,22,30]

Infections with *G. duodenalis* in humans and animals can be asymptomatic or result in acute illness or chronic disease associated with malabsorption. Symptoms in humans with acute illness include abdominal pain, watery diarrhea, vomiting, and weight loss.[2,3,10,30] Pathologic findings are variable but can include blunting of intestinal brush border microvilli, villous flattening, hyperplasia of the crypts of Lieberkühn, and intraepithelial lymphocyte accumulation. Trophozoites are often visible within the crypts and may extend to the villi. There is no intracellular form, and mucosal and submucosal invasion are not typical of infection.[2]

Giardiasis in birds. As stated earlier, *Giardia* has been widely reported in wild and managed populations of birds worldwide, including domestic birds and species kept as pets such as psittacine birds. Most reports of *G. duodenalis*, *G. psittaci*, and *G. ardeae* are point prevalence studies of shedding in apparently asymptomatic wild and managed bird populations, particularly in aquatic birds.[33] However, there are also reports of morbidity and mortality associated with *Giardia* in birds, such as in managed breeding populations of straw-necked ibis infected with *G. ardeae*.[7,28]

Giardiasis in psittacine birds. Giardiasis has been reported in psittacine birds. Several wild-caught sulfur-crested cockatoos from Victoria, Australia, succumbed to infection with *G. duodenalis* despite treatment with ronidazole in the drinking water.[20] Giardiasis caused by *G. psittaci* was reported in a pet 4-year-old male gray-hooded parakeet *(Psilopsiagon aymara)* that died despite treatment with ronidazole orally and in the drinking water.[25]

Numerous sources state that *Giardia* is common in budgerigars and cockatiels.[6,34–38] Although there are many confirmed reports of *G. psittaci* and giardiasis in budgerigars (see section on Giardiasis in Budgerigars), only one published report documented *Giardia* shedding in cockatiels by electron microscopy or molecular sequencing.[12] In fact, other *Giardia* prevalence studies that included cockatiels did not identify *Giardia* in this species.[11,19,32,38,39] It has been proposed that a flagellated protozoan commonly observed in fecal samples from cockatiels is not *Giardia*,[40,41] but rather possibly *Spironucleus*.[41] While cockatiels appear susceptible to infection with *Giardia*,[12] *Giardia* and giardiasis may not be as common in cockatiels as widely believed.

Clinical signs of giardiasis in birds include lethargy, inappetence, weight loss, ruffled feathers, regurgitation, diarrhea, and death, especially in young birds.[6,20,23,25,42,43] Many anecdotal sources state that *Giardia* infection may be associated with rough plumage and self-harm to feathers and skin in pet birds,[6,8,10,34,36–38,43–46] particularly in cockatiels, perhaps associated with a micronutrient malabsorption syndrome. However, evidence is lacking for a definitive causal association.

At necropsy there may be few visible gross changes in birds infected with *Giardia*, although thickening of the gastric intermediate zone and hyperemia and distension of the small intestine have been reported.[12,25,35,42] Microscopic changes include intestinal villous atrophy and mononuclear inflammatory cell infiltration into the lamina propria.[21,35,39,47] *Giardia* trophozoites can be found within the intestinal crypts, often in large numbers, and may extend the entire length of the villi.[12,21,35,39,42] Secondary bacterial and yeast enteritis may be present.[12,47]

Giardiasis in budgerigars. The first report of *Giardia* in the budgerigar was published in 1962 and described postmortem findings in a 2-year-old budgerigar with poor body condition and chronic diarrhea.[42] Since then, a number of other reports of *Giardia* in budgerigars have been published.[9,13,21,23,25,31,38,39,47–55] Prevalence rates of fecal *Giardia* shedding in asymptomatic adult budgerigars ranged from 36.5%[55] to 90.3% in several studies.[31,52,53] Mortality rates of 20% to 50% in affected commercial budgerigar flocks have been described, particularly during hot summer months.[49,50]

While budgerigars are often inapparently infected,[12,23,25,37,47–49] acute and chronic forms of giardiasis have been described in this species.[23,25,47,49] The acute form is more common in nestling, juvenile, and young adult birds. Clinical signs in budgerigars are similar to those in other avian species and include lethargy, ruffled feathers, dehydration, regurgitation, inappetence or polyphagia, weight loss ("going light"), emaciation, diarrhea, and death.[6,7,10,13,25,47,49,50,53] Poor growth and high mortality can be seen in nestlings.[6,12,53] Inappetence, weight loss, lethargy, and diarrhea can also be seen with chronic infections in budgerigars.[47] Comorbidities, including concurrent infection with *Macrorhabdus ornithogaster* or circovirus, can exacerbate the disease and worsen the prognosis.[12,25,37]

Diagnosis. *Giardia* is most commonly identified through the direct observation of trophozoites or cysts in fecal samples (Table 23.2; Figure 23.2A).[6,12,19,30,37,42,45,47] Shedding can be intermittent, and therefore repeat sampling of feces over several days is recommended when possible.[12,37,44,45,47,48] Trophozoites can also be identified in small intestinal contents sampled surgically (e.g., duodenal aspirates) or at necropsy.[6,8,22,35,42] Aspirates or scrapings of small intestinal mucosa should be collected during the gross necropsy since trophozoites are often washed away after tissue processing.[8,35] Microscopic inspection of feces or intestinal contents should be performed as soon as possible, ideally within 10 minutes of collection,

TABLE 23.2

Diagnostic Methods for the Detection of *Giardia* in Birds in Feces or Intestinal Contents

Test	Sample Type	Trophozoite Detection	Cyst Detection
Wet mount (e.g., saline, LRS), no stain	Very fresh (<10 minutes after leaving host)	Yes, motile	Yes, but easily mistaken for yeasts
Wet mount (e.g., saline, LRS) with iodine stain (e.g., Lugol's)	Fresh	Yes, nonmotile	Yes
Flotation concentration (e.g., saturated zinc sulfate, sucrose, sodium nitrate)	Fresh	No	Yes
Biologic stains (e.g., carbol fuchsin, trichrome, modified Wright-Giemsa)	Dried or fixed (e.g., 10% neutral buffered formalin)	Yes	Yes
Antigen detection techniques and molecular diagnostics (e.g., ELISA, IFA, DFA, ICT, PCR)	Fresh, dried, or fixed	Yes (antigenic or genetic material)	

DFA, Direct fluorescent antibody; *ELISA,* enzyme-linked immunosorbent assay; *ICT,* immunochromatographic dipstick test; *IFA,* immunofluorescence assay; *LRS,* lactated Ringer's solution; *PCR,* polymerase chain reaction.

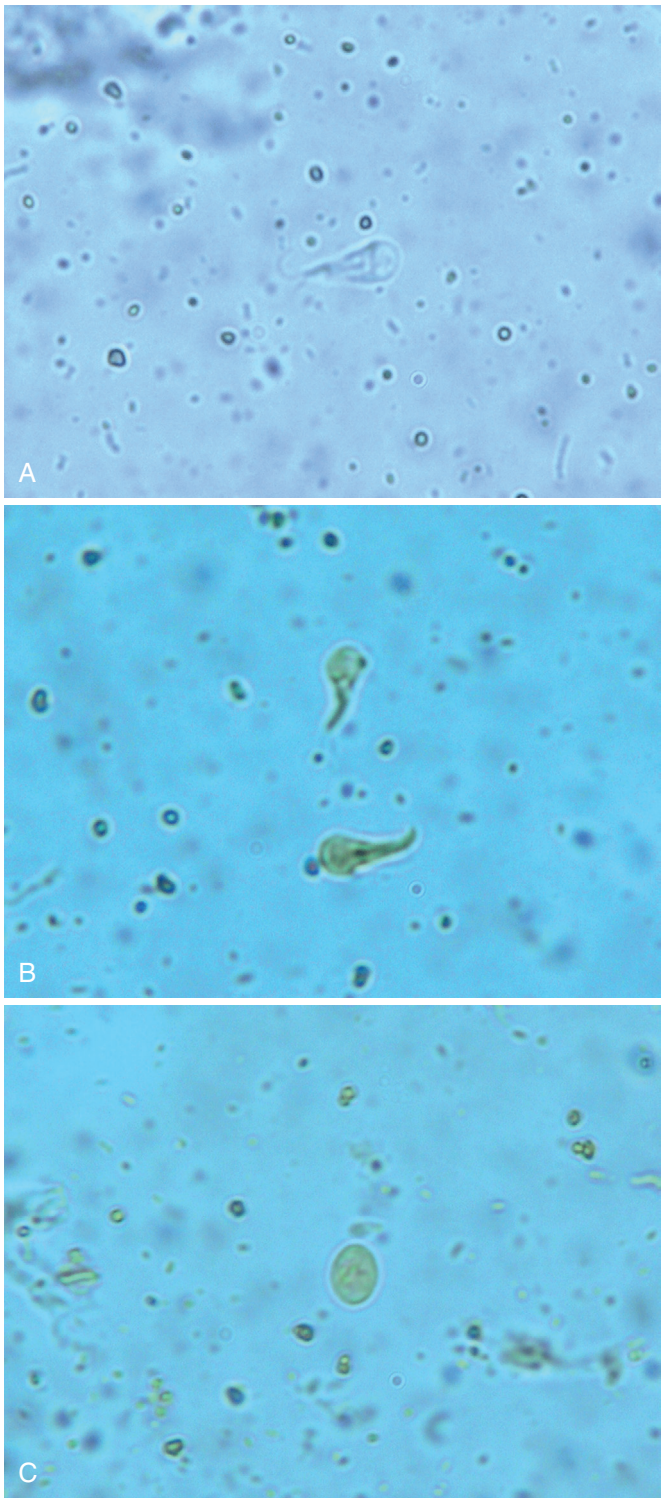


FIGURE 23.2 Saline wet mount appearance of *Giardia psittaci* in feces from an asymptomatic adult budgerigar (*Melopsittacus undulatus*) (magnification ×400). *G. psittaci* trophozoites without stain (A) and with the addition of a small amount of Lugol's iodine (B). *G. psittaci* cyst with the addition of a small amount of Lugol's iodine to the saline solution (C). (Courtesy Lauren Powers.)

BOX 23.1 PRACTICAL TIP

Preparation of the wet mount for direct microscopic observation of *Giardia* and *Spironucleus* trophozoites and cysts.

- Collect fecal samples over a period of several days to increase the likelihood of identifying cysts.
- To evaluate for motile trophozoites, examine samples (feces, intestinal contents) within 10 minutes of leaving the host.
- Use a balanced electrolyte solution such as normal physiologic saline or lactated Ringer's solution instead of tap water.
- Mix the sample thoroughly into solution—no chunks!
- The wet mount should be thin enough so that paper print can be read through it.
- Lower the microscope condenser to its lowest position in order to enhance contrast.
- If no motile trophozoites are observed, consider adding a small amount of Lugol's iodine to aid in the detection of trophozoites and cysts.

since trophozoites cease movement soon after leaving the host (Box 23.1).[6,8,45,56] A balanced electrolyte solution such as normal physiologic saline or lactated Ringer's solution should be used instead of tap water for preparing the wet mount.[6,45] The sample should be mixed well with the solution and should be thin enough so that paper print can be read through the sample. The microscope condenser should be dropped to its lowest level to enhance contrast. Viable *Giardia* trophozoites swim in a "falling leaf" pattern.[6,8,45] A small amount of iodine stain such as Lugol's iodine or D'Antoni's aqueous iodine solution can be added to the wet mount to aid in the detection of cysts and nonmotile trophozoites (Figure 23.2B,C).[12,44] Cysts can also be observed after concentration with saturated zinc sulfate, sucrose, or sodium nitrate solutions.[6,8,30] However, the delicate trophozoites will be destroyed if placed in saturated salt or sugar solutions.[6,8,44,45] Biologic stains such as carbol fuchsin (found in some acid-fast staining kits),[57] trichrome, and modified Wright-Giemsa can be used to aid in the detection of trophozoites and cysts in dried or fixed samples (Table 23.2).[6,8,30,47] *Giardia* cysts are similar in size and shape to yeasts and can be misidentified by light microscopy.[8,30]

Antigen detection techniques including enzyme-linked immunosorbent assay (ELISA),[58] immunofluorescence assay (IFA),[58] direct fluorescent antibody (DFA), and immunochromatographic dipstick tests (ICT) have been developed for the detection of *Giardia* in humans and domestic animals (Table 23.2).[30]

In one report, two commercially available antigen capture reagent tests (Color-Vue Giardia Kit [ELISA test, since discontinued; Seradyn Inc., Indianapolis, IN, USA] and Merifluor Cryptosporidium/Giardia [DFA test; Meridian Diagnostics, Cincinnati, OH, USA]) correctly identified birds that were positive for *Giardia* by fecal microscopic observation.[58] A commercially available ICT for the detection of *G. duodenalis* in veterinary patients including birds (Primagnost Giardia H+K; Albrecht GmbH, Aulendorf, Germany) and a commercially available ELISA (ProSpecT Giardia Microplate Assay; ThermoFisher Scientific, Lenexa, KS, USA) were both positive in two budgerigars and a gray-hooded parakeet with confirmed *Giardia* infection in a separate report.[25] PCR assays and molecular sequencing techniques can also be used for diagnostic and research purposes.[25,30]

Treatment, prevention, and control. Giardiasis in humans is generally treated with nitroimidazole drugs including metronidazole and tinidazole, acridine derivatives such as quinacrine, or monoamine oxidase inhibitors such as furazolidone.[30] In veterinary medicine, the nitroimidazoles metronidazole and dimetridazole are commonly used and are the most widely reported drugs used in birds (Table 23.3; see also Chapter 9).[8,20,51,52,56] Metronidazole can be effective when administered orally to birds.[8,37] Although drinking water dosages have been published for metronidazole, the drug has poor palatability and birds may be reluctant to drink medicated water.[8,37,43,47,51,52] Dosing of metronidazole in the drinking water may not be effective or safe in birds. Dimetridazole has been reported to be used in birds by gavage or in the drinking water. Dimetridazole has been shown to be effective in budgerigars when delivered in the drinking water,[49,50,52] although reportedly this drug may be toxic in finches and other birds.[42,46] Dimetridazole dosages of 0.1% in the drinking water were well-tolerated in adult cockatiels but resulted in high morbidity in nestling chicks in one report.[46] At 0.5% drinking water, all age groups of cockatiel nestlings showed severe morbidity. The authors of this report advised that dimetridazole not be used in the drinking water for cockatiels during the breeding season.[46] Effective treatment with ronidazole has been reported in budgerigars when administered orally once to twice daily or in the drinking water.[26,37] Tinidazole delivered in the drinking water was effective in budgerigars and lineolated parakeets in a separate report.[23] Drinking water dosages for any medication may need to be reduced during hotter summer months due to the potential for increased water intake.[49] Of the nitroimidazole drugs described here, only metronidazole is approved for use in the United States by the Food and Drug Administration (FDA).

In dogs, benzimidazole drugs such as oxfendazole, albendazole, and fenbendazole have been shown to eliminate cyst excretion and improve clinical signs.[8,56] Fenbendazole may be an effective treatment in birds.[8] However, benzimidazole drugs may potentially cause life-threatening radiomimetic bone marrow suppression,[30] which is uncommon but has been reported in birds.[35] Other less-widely studied and utilized treatments for avian giardiasis include nitazoxanide (a thiazolide), azithromycin (a macrolide), and paromomycin (an aminoglycoside).[23,30] Paromomycin was effectively used to treat budgerigars and lineolated parakeets with giardiasis in one report.[23] Relapses and reinfections are common with *Giardia*, suggesting that acquired immunity may be short-lived.[6,12,30,47] Published treatment durations may be inadequate to prevent reinfection from contaminated environments.[30]

Prevention of *Giardia* infection in birds is best accomplished through sound husbandry, hygiene, and biosecurity practices.[12,45,47] Physiologic stressors such as overcrowding, competition for food and water resources, and temperature extremes should be avoided.[6,43] Screening of at-risk birds prior to entry into breeding collections should be considered.[12,47] Food and water containers should be placed in elevated positions in the enclosures and cleaned regularly.[47] Trophozoites are sensitive to most disinfectants, but cysts can remain infectious for many months in water and soil.[12] Cysts may survive in chlorinated water, although quaternary ammonium compounds may be effective disinfectants.[30,45]

Hexamita *and* Spironucleus

The Hexamitinae diplomonads *Hexamita* and *Spironucleus* have very similar morphologic characteristics by light microscopy. Older reports that lacked ultrastructural details by electron microscopy placed some *Spironucleus* species that affect birds and fish into the genus *Hexamita*, or even *Octomitus*.[1,5,63,64] Ultrastructurally, *Hexamita* and *Spironucleus* both possess cytostomal canals (flagellar pockets) that pass through the whole cell and open posteriorly, whereas *Octomitus* (a Giardiinae diplomonad) lacks cytostomal canals.[1,2,64] *Hexamita* and *Spironucleus* differ from each other in the shape of their nuclei and in the position of their basal bodies (Figure 23.1).[1] *Hexamita* species have round nuclei, whereas the nuclei of *Spironucleus* species are spiral-shaped and subapical in location.[2,64] Despite the differences in nuclear structure and other distinctive features, molecular sequencing has placed *Spironucleus* phylogenies into three different clades that differ as much from each other as they do from *Hexamita* species.[2,5] The third clade of *Spironucleus* consists of *S. muris* from the mouse and *S. meleagridis* from turkeys *(Meleagris gallopavo)*.[65] A cyst form is present for many, but not all, *Spironucleus* species.[2,64]

Spironucleus species are primarily intestinal organisms and are parasitic in a wide range of vertebrates from fish to birds and mammals including primates.[59] *Spironucleus* species are significant pathogens to economically important Atlantic salmon *(Salmo salar)*, rainbow trout *(Oncorhynchus mykiss)*, cichlids, and other fish.[2] To the author's knowledge, the only Hexamitinae diplomonads known to infect birds are *S. meleagridis* and *S. columbae* and none has documented zoonotic potential.

Spironucleus meleagridis *S. meleagridis* was initially classified as *Hexamita meleagridis* but was later reassigned to the genus *Spironucleus* based on ultrastructural morphologic findings as determined by scanning and transmission electron microscopy.[60,64] Trophozoites range from 5.5 to 9.0 μm in length and 1.0 to 6.0 μm in width, and cysts range from 4.5 to 7.0 μm in length and 3.5 to 5.5 μm in width.[64]

S. meleagridis is the cause of avian infectious catarrhal enteritis, or avian spironucleosis, in turkeys and other birds.[64–68] The terms "hexamitiasis" and "hexamitosis" are still occasionally used to describe this disease.[64,65] Spironucleosis has had considerable economic impact on the commercial turkey industry in the United States since the late 1930s, although the incidence of the disease in turkeys has declined dramatically in recent decades due to changes in commercial production management.[42,65,66,67,69] However, spironucleosis remains a disease of considerable economic importance in farm-reared game birds such as chukar partridges *(Alectoris chukar)*,[66] red-legged partridges *(Alectoris rufa)*,[70,71] ring-necked pheasants *(Phasianus colchicus)*,[64,70,71] quail (*Coturnix* spp.), and guinea fowl *(Numida meleagris)*. Mortality rates of up to 80% have been observed in some chukar partridge broods.[66] *Spironucleus* has also been identified in non-galliform avian species including ducks, sandhill cranes *(Grus canadensis pratensis)*,[72] demoiselle cranes *(Anthropoides virgo)*,[73] ostriches (*Struthio camelus*),[74] rheas *(Rhea americana)*, and a Gouldian finch *(Erythrura gouldiae)*,[39] among others.[6,61]

Both cysts and trophozoites may be capable of infecting the host through direct ingestion or ingestion of contaminated

TABLE 23.3

Some Published Drug Dosages for the Treatment of Giardiasis and Spironucleosis in Psittacine Birds and Pigeons

Drug and Dosage	Species	Protozoan	Efficacy and Safety	References
METRONIDAZOLE				
25–30 mg/kg PO q24h × 10 days	Various	*Giardia*	Some birds may require 25 mg/kg PO q12h up to 30 days	8
25 mg/kg PO q12h × 5 days; 50 mg/kg PO q24h × 5 days	Various	*Giardia*		37
50 mg/L DW × 5 days	Budgerigar (*Melopsittacus undulatus*)	*Giardia*	Not considered effective in a group of four birds	52
200 mg/L DW × 5 days	Budgerigar	*Giardia*	Not considered effective in a group of four birds	52
800 mg/L DW × 5 days	Budgerigar	*Giardia*	No further shedding at day 5	47
1000 mg/L DW × 7 days	Budgerigar	*Giardia*	3/6 birds died and all lost weight, no trophozoites found on necropsy of surviving birds	51
15–50 mg/kg IM single dose	Various	*Giardia*	Reportedly safe in budgerigars, cockatiels, and lovebirds but relapses occurred	56
1.5 mg per bird q8h × 5–7 days	Australian king parrot (*Alisterus scapularis*)	*Spironucleus*	Followed by 250 mg/L DW × 10–21 days. Considered effective	59
DIMETRIDAZOLE				
50 mg/kg PO, three doses total, q12h	Budgerigar	*Giardia*	Virtually cleared all birds of infection. Administered via gavage	52
200 mg/L DW × 5 days	Budgerigar	*Giardia*	Partially effective in a group of four birds	52
200 mg/L DW × 5 days	Budgerigar	*Giardia*	Treatment considered effective. Dosage should be reduced to 100 mg/L on hot summer days. Treatment can be repeated	13, 49
2000 mg/L DW × 10 days	Budgerigar	*Giardia*	Cleared shedding and no further mortality	53
4000 mg/L DW × 10 days	Budgerigar	*Giardia*	1/6 birds died and all lost weight, no trophozoites found on necropsy of surviving birds	51
3000 mg/L DW × 5 days	Australian king parrot	*Spironucleus*	Followed by 5 mL of 3000 mg/L via crop lavage. Most birds failed to respond to treatment	60
RONIDAZOLE				
6–10 mg/kg PO q24h × 6–10 days	Various	*Giardia*		36
100–200 mg/L DW × 7 days	Various	*Giardia*		36
2000 mg/L DW × 10 days	Budgerigar, gray-hooded parakeet (*Psilopsiagon aymara*)	*Giardia*	Effective in two budgerigars but an adult gray-hooded parakeet died on day 3 of treatment	25
4000 mg/L DW	Sulfur-crested cockatoo (*Cacatua galerita*)	*Giardia*	Not considered effective, treated birds died	20
1111 mg/L DW	Pigeon, (*Columba livia*), cockatiel (*Nymphicus hollandicus*)	*Spironucleus*	Considered effective following a single oral dose of carnidazole	61
DW for 7–10 days	Pigeon	*Spironucleus*	Treatment considered effective. Drinking water dosage not reported	62
TINIDAZOLE				
800 mg/L DW × 7–14 days	Budgerigar, lineolated parakeet (*Bolborhynchus lineola*)	*Giardia*	Treatment considered effective	23
CARNIDAZOLE				
10 mg single PO dose	Pigeon, cockatiel	*Spironucleus*	Considered effective followed by ronidazole in drinking water	61
PAROMOMYCIN				
100 mg/kg PO q12h × 7 days	Budgerigar, lineolated parakeet	*Giardia*	Treatment considered effective	23
FURALTADONE				
1778 mg/L DW	Pigeon	*Spironucleus*	Considered effective following single oral dose of metronidazole 50 mg	61

DW, Drinking water.

drinking water or food sources.[45,64] Cysts contained within fecal material were capable of infecting turkey poults for up to 6 weeks after storage at room temperature in one report.[60] Cysts in the mucus in the cloaca of carrier hens could potentially adhere to eggshell surfaces as eggs are laid and potentially result in infection of newly hatched chicks.[64]

Although *Spironucleus* can be found in apparently healthy birds,[71] the protozoan can also be associated with significant clinical disease, particularly in young birds.[6,59,75] In turkeys, the incubation period is short at 4 to 7 days.[8] Mortality begins to occur from 1 to 21 days after the onset of clinical signs in an affected flock.[8] Clinical signs of avian spironucleosis in galliform birds include lethargy, poor feathering, reduced appetite, weight loss, diarrhea, and death.[8,65,67,71,76] Diarrhea is often watery and yellow in affected turkeys and pheasants.[65,76] Concurrent infection with other intestinal pathogens may increase the pathogenicity of *S. meleagridis*.[32,73,68]

Gross necropsy findings may include small intestinal distension with watery luminal contents.[64] Microscopically, large numbers of trophozoites can be observed within the lumen of the duodenum and jejunum and within the intestinal crypts (Figure 23.3).[59,65] Cysts are almost exclusively seen within the luminal mucus and are rarely observed within the intestinal crypts. Varying degrees of intestinal congestion, goblet cell hyperplasia, mononuclear inflammatory cell infiltration within the lamina propria, and necrosis are observed.[39,59,65]

Spironucleosis in psittacine birds Spironucleosis has been documented in several psittacine species including juvenile wild Australian king parrots *(Alisterus scapularis)*,[59] an emaciated splendid grass (scarlet-chested) parakeet *(Neophema splendida)*,[61] an emaciated wild sulfur-crested cockatoo,[75] and cockatiels.[41,54,61,77] The organism was identified as *S. meleagridis* in several of these reports by molecular analysis.[77] Partial genomic sequencing of a flagellated protozoan isolated from cockatiel feces demonstrated a 98% molecular identity with *S. meleagridis* isolated from turkeys in one report.[77] Prevalence of infection in asymptomatic birds was found to be 70% and 86% from two unrelated cockatiel breeding facilities using a diagnostic PCR assay on fecal samples in this same study.[77]

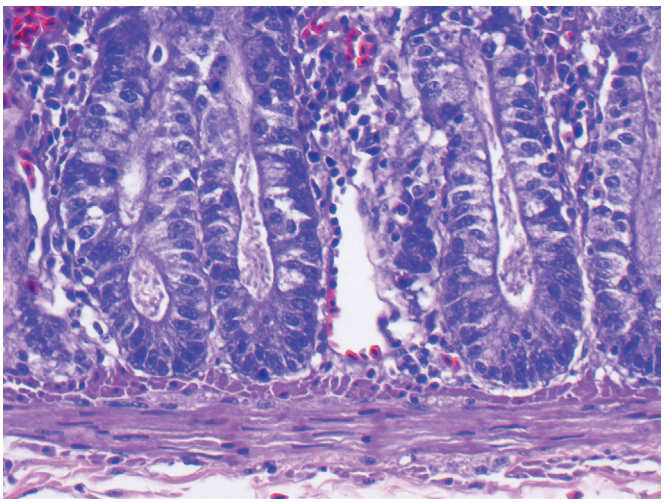


FIGURE 23.3 *Spironucleus meleagridis* trophozoites visible within hyperplastic intestinal crypts in a cockatiel *(Nymphicus hollandicus)*. (Courtesy Michael Garner.)

Clinical signs of spironucleosis in psittacine birds are similar to those described in galliform birds, including listlessness, anorexia, weight loss, emaciation, diarrhea (Figure 23.4), and death.[59,61] In one report, five 3- to 4-month-old cockatiels in one aviary and juvenile birds from four separate aviaries with spironucleosis were described as having lethargy, changes in the appearance of feces, and emaciation.[61] In one retrospective report, 8/83 (9.6%) juvenile cockatiels submitted for necropsy demonstrated a flagellated protozoal enteropathy consistent with spironucleosis.[54] Necropsy findings in psittacine birds are similar to those for galliform birds.

Spironucleus columbae In 1923, a report of catarrhal enteritis in young pigeons *(Columba livia)* in Germany associated with a flagellated protozoal parasite was published and the protozoan was classified as *Octomitus columbae* nov. spec.[64] Trophozoites measured 5 to 9 μm in length and 2.5 to 3.5 μm in width, similar in size to *S. meleagridis*.[64] Since then, the protozoan has been reclassified as *S. columbae* and reported in other pigeon collections.[44,78–80] Clinical signs in pigeons include reduced or selective appetite, slow crop emptying, weight loss, emaciation, reduced flight performance, and death.[61,79] In one report, clinical signs were highest in birds 7 to 10 weeks of age and in loft imports.[61] The morbidity rate was 15% to 25%, and the mortality rate was about 5% in this report.[61] Immunosuppression caused by circovirus co-infection may affect susceptibility to disease.[78] Postmortem findings included a characteristic dilatation of the duodenum.[66] Attempts at infecting turkey poults with *S. columbae* were unsuccessful in one report.[80] However, to the author's knowledge, no ultrastructural imaging by electron microscopy or molecular sequencing has been performed to date for *S. columbae* to confirm that it should continue to be taxonomically classified as a separate species from *S. meleagridis*.

Diagnosis Techniques for the detection of *Spironucleus* are similar to those for *Giardia*, although commercial antigen detection techniques are not currently available. Motile trophozoites and cysts can be observed by wet mount observation of fresh feces or intestinal contents.[8,44,45,59,65] *Spironucleus* trophozoites can easily be confused with those of *Giardia* by light microscopy.[42] However, viable *Giardia* trophozoites move in a "falling leaf" pattern, whereas viable *Spironucleus* trophozoites move in a rapid, linear, darting motion.[44,45,59,65] As with *Giardia*, *Spironucleus* trophozoites are motile for only a very short time after exiting the host, and so diagnostic samples should be evaluated soon after collection.[44,59] *Spironucleus* cysts are similar in appearance to those of *Giardia* but somewhat smaller.[71] Biologic stains described for detecting *Giardia* such as modified Wright-Giemsa can also be used to detect *Spironucleus* trophozoites and cysts in dried or fixed samples. As with *Giardia*, PCR can be used for diagnostic and research purposes. In one report, a diagnostic PCR assay was developed and used to evaluate the fecal shedding of *S. meleagridis* in cockatiels.[77]



FIGURE 23.4 Chronic diarrhea in an adult cockatiel *(Nymphicus hollandicus)* with spironucleosis. (Courtesy Lauren Powers.)

Treatment, prevention, and control Reported treatment options for the treatment of avian spironucleosis are similar to those reported for giardiasis.[43] Nitroimidazole drugs including metronidazole, dimetridazole, ronidazole, and carnidazole have been used to treat spironucleosis in turkeys, farm-reared game birds, pigeons, and psittacine birds (see also Chapter 9).[45,64,59,61,79] However, treatment failures and disease recurrences are common, and severely emaciated birds may not recover.[44,45,59] In one report, shedding of *Spironucleus* was reduced in Australian king parrots after treatment with dimetridazole, but only two mildly affected birds from one aviary clinically recovered.[59] Reports suggest that pheasants with spironucleosis respond better to a combination of dimetridazole and oxytetracycline than to dimetridazole alone.[76] However, the addition of neomycin and oxytetracycline to the drinking water was not considered effective for chukar partridges.[66] Treatment with a single oral dose of carnidazole followed by ronidazole in the drinking water for 7 days was considered successful in pigeons and cockatiels in another report.[61] Furaltadone in the drinking water following a single oral dose of metronidazole was considered successful in pigeons.[61] Effective control and prevention measures have not yet been standardized for *S. meleagridis* in farm-reared game birds, psittacine birds, and pigeons.[8,64] In the absence of evidence-based information, control measures recommended for the management of *Giardia* in birds appear to be reasonable options for the control and prevention of *Spironucleus*.

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CHAPTER 24

Histomoniasis

Yuko Sato

INTRODUCTION

Histomoniasis (blackhead disease) is a protozoal disease that affects mainly the liver and ceca of turkeys *(Meleagris gallopavo)* and other gallinaceous birds, including chickens *(Gallus gallus domesticus)*, peafowl (*Pavo* spp.), and quail (*Coturnix* spp.). This disease causes high mortality in turkeys and game birds. In chickens, mortality usually is 10% to 20%, although the morbidity is very high, as they could serve as asymptomatic reservoirs of disease.[1] Currently, there are no efficacious drugs for treatment or prevention that are approved for use in poultry in the United States, Europe, and many other countries. There are no vaccines for this disease, and management practices are key in reducing outbreaks. Severe outbreaks may potentially lead to loss of entire flocks or euthanasia to relieve bird suffering.[2]

ETIOLOGY

The causative agent of blackhead disease is the protozoal parasite *Histomonas meleagridis*. *H. meleagridis* is a member of the family Dientamoebidae, order Trichomonadida.[3] It is a pleomorphic, single flagellated protozoa in the cecal lumen, approximately 3 to 16 μm in diameter, and can also exist in an amoeboid form (tissue form), 6 to 20 μm in size, in the liver and other tissues during infection. Recently, a cyst-like stage was discovered, perhaps explaining the resistance of the organism in the environment.[2] Histomoniasis was first described in turkeys in Rhode Island by Cushman in 1893 and has since been reported across the country and soon in many other countries.[4] The organism itself is not resistant to environmental conditions, and variables such as temperature extremes, drying, and lack of oxygen will destroy the organism outside of its host.[2] However, control of the intermediate host is key to controlling the disease. Bacterial flora in the gut is shown to be important in development of the disease, although little is affected by the course of the disease with the administration of antibiotics.[5]

There are two known methods for transmission: (1) ingestion of embryonated ova or adult worms of the intermediate host, *Heterakis gallinarum*, also known as cecal worms, containing the histomonads, and (2) direct transmission of histomonads through "cloacal drinking."[1] Although the most significant disease in turkeys, blackhead has been reported to infect all gallinaceous birds and others such as ostriches *(Struthio camelus)*, rheas *(Rhea americana)*, and a variety of game birds, including pheasants *(Phaesianus colchicus)*, quail, grouse (*Tetraonini* spp.), ducks (*Anas* spp.), guinea fowl (*Numida* spp.), and chukar partridges *(Alectoris chukar)*. Blackhead is sometimes reported from zoo birds such as peacocks.[6] Species such as ducks and ostriches show a high resistance to disease and may become asymptomatic carriers.[3]

EPIDEMIOLOGY AND PATHOGENESIS

Histomoniasis is typically a seasonal disease that occurs mostly in late spring and summer (e.g., April through June in the United States). The intermediate host, the cecal worm, is the most common vector of the histomonads, and according to a study by Hauck and Hafez, the coinfection rate is 50.8% and could be up to 60% to 91% in chickens.[7] The hosts of *H. gallinarum*, the cecal worm, include birds that are maintained in soil or litter, which include the chicken, turkey, duck, goose, grouse, guinea fowl, partridge, pheasant, and quail. Birds become infected with blackhead by ingestion of the embryonated eggs of *H. gallinarum*, and chickens are the best host and the best reservoir for infection. Thus biosecurity is a critical control method when it comes to housing turkeys and other susceptible species from chickens. Since worm eggs may remain infective up to 3 years in soil and remain positive for *H. meleagridis* after 150 weeks, care must be taken when overlapping housing environments with chickens for the susceptible population. Other *Heterakis* worms, such as *Heterakis bonasae* found in bobwhite quail *(Colinus virginianus)* and ruffed grouse *(Bonasa umbellus)*, do not have the ability to act as a vector for *H.* gallinarum.[8] However, further investigations on other vectors including *Heterakis isolonche*, which infects ducks and other gallinaceous birds, as well as *Heterakis dispar*, which infects ducks and geese, have not been performed.[7]

Additional studies show that earthworms can harbor cecal worm larvae for long periods of time and transmit blackhead disease. There have been reports of outbreaks of histomoniasis following heavy rains in commercial turkeys, which could be due to a surge in earthworm population indoors, which are subsequently ingested by turkeys. Other vectors such as houseflies, grasshoppers, and sow bugs have been reported to cause low rates of mechanical transmission of cecal worms.[2]

Direct transmission by "cloacal drinking" has now been adopted as the main method of inoculation and infection due to its high reproducibility and reliability.[7] The histomonads

are taken inside the cloaca by retrograde movement caused by reverse peristalsis following muscular contractions of the cloacal lip during normal cecal function. This method is important in explaining the rapid and explosive spread of blackhead throughout turkey flocks in the absence of intermediate hosts and vectors. By the same token, contaminated litter or paper has been shown to spread blackhead within a floor pen.[2] In contrast to its major contribution to transmission in turkeys, studies have shown that infection does not spread by direct contact in chickens. Possible theories include the difference in behavior and the water composition in chicken droppings versus turkey droppings.[9]

The relationship of histomonads with bacteria and other organisms to create pathology has been debated. Some studies show that histomonads are avirulent in gnotobiotic poults and only fully virulent when certain bacteria such as *Escherichia coli*, *Clostridium perfringens*, and *Bacillus subtilis* are present.[10] However, most lesions from turkeys infected by *H. meleagridis* are sterile, and treatment with therapeutic levels of antibiotics has had little to no effect on disease lesions in chickens.[11] Although cecal coccidiosis in chickens can potentially affect the severity of gross lesions, there seems to be no significant effect of concurrent infection on pathogenicity or mortality in chickens. In contrast, *Eimeria adenoides* infection in turkeys appears to interfere with blackhead infection and cause lessened severity of cecal lesions.[5]

Pathogenesis begins when histomonads penetrate through the cecal wall, multiply, and enter the bloodstream to eventually parasitize the liver. Clinical signs can be visible in birds from 7 to 12 days and death may occur 11 to 14 days postinfection. The incubation period varies with the size of the infective dose and the route of infection. Indirect infections from *H. gallinarum* eggs require longer than those from direct transmission. Experimentally, lesions in turkeys develop about 3 days earlier with cloacal inoculation compared with infection via *H. gallinarum* eggs.[2,9]

CLINICAL SIGNS

Clinical signs in birds depend greatly between species of gallinaceous birds, ranging from high mortality and severe hepatitis/typhlitis in turkeys, while in chickens, the clinical signs are milder and pathology is mostly restricted to ceca.[1]

Clinical signs in turkeys include:

- Anorexia and emaciation
- Bowing of the head and wings
- Depression and drowsiness
- Yellowish green (sulfur-colored) droppings
- Huddling together
- Ruffled feathers
- Death

By 6 to 12 days postinfection, turkeys will start to become emaciated.[3] The yellow-green droppings are observed because of liver necrosis and the excreta contains bile pigments. Birds exhibiting this clinical sign often succumb to death. Clinical signs in chickens can range anywhere from asymptomatic, mild depression, and loss in production to mortality. Mortality may be low (10%–20%), but morbidity is high.[9] Disease is more commonly seen in young pullets or layers and broiler breeders in early stages of egg production.[2] Droppings may be bloody in chickens and resemble cecal coccidiosis.[1]

GROSS AND HISTOPATHOLOGIC LESIONS

Gross lesions in turkeys and game birds are characterized by severe caseous typhlitis with a fibrinonecrotic core (cecal cores) and necrotic foci in the liver (Figures 24.1 and 24.2). Characteristic bull's-eye liver lesions and cecal lesions, including caseous cores, serosal inflammation, and occasional ulceration and hemorrhage, are almost pathognomonic with matching history and signalment. The ulcerative cecal lesions may perforate and lead to secondary peritonitis. Cecal contents often have a foul-smelling odor. The liver is often enlarged with pale tan to yellow, crater-like lesions with depressed centers and well-demarcated edges.[12] Severity of the lesion depends on multiple factors but may be up to 4 cm in diameter and can involve parts or the entire organ.[3] Atypical lesions have been reported in the kidney, spleen, pancreas, and lungs of turkeys, presumably due to the histomonads being

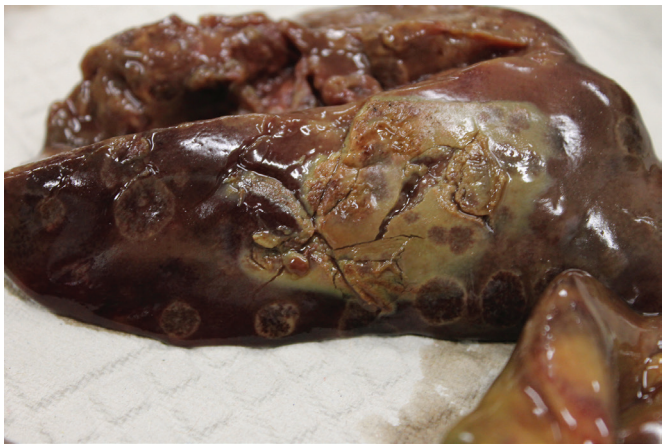


FIGURE 24.1 Gross liver lesions from a turkey *(Meleagris gallopavo)* with histomoniasis. Note the prominent, multifocal circular necrotic lesions on the liver surface.



FIGURE 24.2 Caseous typhlitis from the turkey *(Meleagris gallopavo)* pictured in Figure 24.1. Note sloughing of the mucosal epithelium into a thick caseous mass in the lumen of the ceca.

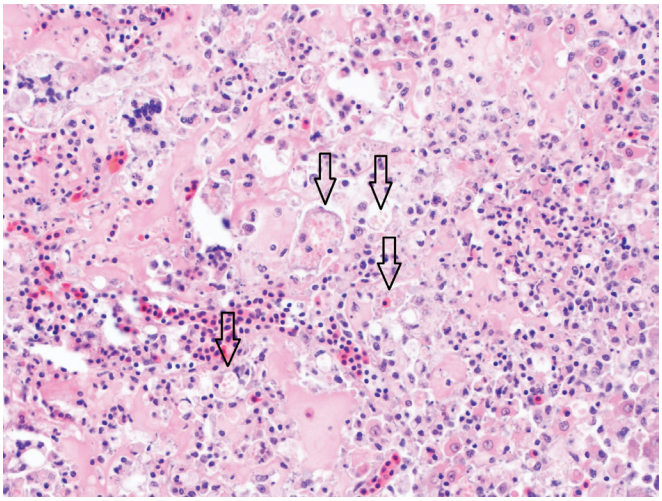


FIGURE 24.3 Histopathologic lesions from the turkey *(Meleagris gallopavo)* liver pictured in Fig. 24.1 with coagulative necrosis, hemorrhage, and inflammatory cell infiltrates. Note numerous clusters of histomonads *(arrows)* within necrotic centers and digestive chambers of histiocytes that are taking up weak eosinophilic to brown staining. (40× magnification, hematoxylin and eosin stain.)

carried from infected tissues in the ceca to the liver and other tissues via the bloodstream. Histomonads can also reach the bursa of Fabricius without entering the bloodstream.[9,13] In chickens, lesions are often restricted to the ceca and liver lesions may be present but occur less frequently.[3]

Microscopic lesions correlate with gross lesions and result in multifocal necrosis and granulomatous inflammation in the liver, ceca, and other tissues. Necrosis is typically coagulative and extensive, with loss of normal tissue architecture, hemorrhage, and presence of inflammatory cells. Invasion of the cecal wall results in hyperemia and heterophilic infiltration in early stages. By 5 to 6 days postinfection, numerous histomonads are visible as weakly brown to pale eosinophilic staining, ovoid, intracellular organisms in clusters approximately 10 μm (3–16 μm) in diameter within necrotic centers and lacunae in the mucosa (Figure 24.3).

Live organisms may be observed with phase-contrast microscopy. Organisms may be seen as clusters in the liver, ceca, and other sections stained with hematoxylin and eosin (H&E), with some aid in visualization by periodic acid-Schiff (PAS) and Grocott methenamine silver (GMS) stain to distinguish from molds and other fungal infections.[9]

DIAGNOSIS

Blackhead in turkeys and game birds is easily diagnosed by salient gross lesions with or without histopathologic lesions. Coupled with potential clinical history of sulfur-colored urofeces, having (1) dilated ceca with a necrotic core characterized by sloughed mucosa, blood, and other debris, and (2) necrotic liver lesions (target or bull's-eye lesion) is almost pathognomonic. Histomonads can also be isolated in vitro from the ceca and liver in suspected cases using Dwyer medium.

Polymerase chain reaction (PCR) methods have recently been used using primers based on published sequence of *H. meleagridis* small subunit ribosomal RNA gene using the ITS (internal transcribed spacer) region, the 18S RNA gene, and a conserved region of the β-tubulin gene.[14] Many of the other described assays comprise mostly conventional gel-based assays except for a qPCR for *H. meleagridis*. While qPCR is generally more sensitive and much less time-consuming compared to conventional PCR, this previously developed qPCR for *H. meleagridis* was found to be less sensitive.[15] Analysis by Clarke et al. revealed that the PCR specific for the ITS region had higher sensitivity using pooled ceca and liver samples versus cloacal swabs and PCR of other organs.[14]

TREATMENT, PREVENTION, AND CONTROL

Blackhead disease has been historically controlled by efficacious drugs and chemicals. The first set of compounds used were arsenicals such as tryparsamide, atoxyl, nitarsone, and roxarsone. The mode of action of these drugs involves interference with DNA repair processes and cellular energy metabolisms in the parasite. Early in vitro studies by Tyzzer in 1923 and others have repeatedly shown curative effects.[16] The benefit of this class of chemicals is the ability for it to be used for both prophylaxis and treatment, depending on the dosage. However, arsenicals have never been registered in the European Union (EU), and all arsenicals have been either banned or withdrawn by the Food and Drug Administration (FDA) in the United States since 2016.[12,17]

Nitroimidazoles such as dimetridazole and ipronidazole were used for a long time in the turkey industry for both prophylaxis and treatment (see Chapter 9). In addition, the medications were useful for increased weight gain and improved feed efficiency. However, due to concerns with food safety and illegal drug residues following the use of nitroimidazoles in food animals, the drugs were banned or voluntarily withdrawn.

The last available prophylactic product against blackhead was nitarsone (Histostat-50, Zoetis, Parsippany, NJ) but FDA approval was withdrawn in January 2016, which now creates a situation where all effective therapeutic and prophylactic medication are banned in the United States and several other countries such as Japan, Canada, Australia, and New Zealand.[10,17–19] A list of common treatment and prophylactic compounds for histomoniasis is listed in Table 24.1, some of which are historical archives and no longer available on the market.

With the loss of efficacious chemotherapeutics, three different approaches have been used to control histomoniasis: antibiotic therapy, phytogenics, and vaccine development.[18] Coupled with the movement toward antibiotic-free poultry production, phytobotanicals and alternative products have gained a lot of traction in recent years. There are numerous reports indicating in vitro efficacy to *H. meleagridis* using plant products including but not limited to plant extracts, prebiotics, probiotics, organic acids, essential oils, enzymes, and volatile fatty acids. Some of the more common compounds used in studies include oils such as cinnamaldehydes and terpene, ethanol and water extracts such as flavonoids and fatty acids, yeast products, alkaloids, and plant materials. Several challenges exist, including contradictory results, a lack of well-defined infection models in live animal trials, and the safety margins expressed in minimal lethal concentrations.

TABLE 24.1

List of Most Common Treatment, Prophylaxis, and Control Options for Blackhead Disease and Current Status of Availability

Type	Subtype	Product	Trade Name (Manufacturer)	Current Status (Organization and Year)
Treatment, prophylaxis	Nitroimidazole	Dimetridazole Ipronidazole	Emtryl (Solvay) Ipropran (Hoffman-La Roche)	Banned (FDA 1987, EU 1995–2001) Banned/withdrawn (FDA 1989)
Treatment, prophylaxis	Nitrofurans	Furazolidone Salfuride	Furox (Smith-Kline) Nifursol (Solvay)	Banned (FDA 1991) Banned EU 2002–2003)
Prophylaxis	Arsenicals	Carbarsone Nitarsone Roxarsone	Carb-O-Sep (Alpharma) Histostat-50 (Zoetis) 3-Nitro (Zoetis)	Not marketed Withdrawn (FDA 2016) Banned (EU 1999), withdrawn (FDA 2013)
Control	Phytogenics	Plant extracts	FitoTril (Ravet Sp. Zoo.)	Poland, on market
Control	Phytogenics	Essential oils	Protophyt (Phytosynthese)	France, on market
Control	Phytogenics	Combination	Natustat (Alltech)	United States, on market
Control	Phytogenics	Essential oils/acids	DeviSTAT (Devenish)	United States, on market
Control	Phytogenics	Oregano oil	Orego-Stim (Anpario)	United States, on market
Control	Antibiotics	Hygromycin	Hygromix 2.4 (Elanco)	Withdrawn (FDA 2014)
Control	Antibiotics	Paromomycin	Parofor (Huvefarma)	Italy, Bulgaria, on market
Control	Antibiotics	Tiamulin	Multiple companies	Extra-label, on market

EU, European Union; *FDA*, US Food and Drug Administration.

Modified from Clark S, Kimminau E: Critical review: future control of blackhead disease (histomoniasis) in poultry, *Avian Dis* 61(3):281–288, 2017. doi: 10.1637/11593-012517-ReviewR; Liebhart D, Ganas P, Sulejmanovic T, Hess M: Histomonosis in poultry: previous and current strategies for prevention and therapy, *Avian Pathol* 46(1):1–18, 2017. doi: 10.1080/03079457.2016.1229458.

Nonetheless, in vivo studies often failed to achieve efficacy, if not have contraindications in comparison to success in in vitro studies, further highlighting the complexity of the disease. A few products available commercially have been tested for efficacy and potential to prevent histomoniasis and subsequently published; however, none of the phytobotanicals tested so far seem to provide robust protection either in vivo or in field studies.[17,18,20]

Other alternative treatment strategies include the potential of anticoccidials as it pertains to controlling protozoal infections. However, anticoccidials had no effect in vivo, although studies in chickens with concurrent coccidiosis with *Eimeria tenella* showed reduction of liver lesions in broilers. In the same study, several other antibiotics including penicillin, chlortetracycline, tylosin, and sarafloxacin were tested with no effect on histomoniasis.[11] Prophylaxis with paromomycin, an aminoglycoside antibiotic, was reported to have effects in reducing mortality and severity of lesions in turkeys. The efficacy appears to be dose dependent and treatment must be applied prior to infection, rendering it ineffective as a treatment strategy for clinical disease. Field and experimental trials with tiamulin, a pleuromutilin class of antibiotic, have had mixed results when administered via drinking water for 5 days at 25 mg/kg body weight. Along with the risk for adverse reactions ("knockdown syndrome") due to the antibiotic potentiating the ionophores in the feed, when administered together combined with feed, it appears that its use as a blackhead treatment is limited.[9,17,18]

Vaccination is not a realistic option for prevention, as birds do not reliably become resistant to reinfection after suffering exposure. However, chickens and turkeys do produce antibodies against *H. meleagridis* antigens prepared from infected livers and ceca after natural or experimental infections.[6] In turkeys, these antibodies persist at least for up to 16 weeks postinfection. Some literature states partial immunity, which may or may not work for protection against reinfection. Until recently, vaccination against blackhead disease was, in most cases, contraindicated. Recent research showed complete protection of turkeys using vaccination with attenuated clonal culture containing clonal histomonads. In commercial settings, this may not be currently viable, although future research into vaccination using this method appears to be promising.[2,12,18]

Prophylaxis is therefore based mainly on biosecurity measures. One of the basic concepts is separation of species, especially chickens and turkeys. Hence, a free-range location used for chickens should not be used for turkeys. It is also advisable not to mix young poults and older turkeys. Deworming against intermediate hosts *(Heterakis)* is also recommended in a high worm burden environment, which could be checked with a simple fecal examination. Prevention of contamination of feed and especially water by infected droppings and proper litter management may also reduce the transmission of the parasite via cloacal drinking.[1,7,12,17]

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CHAPTER 25

Cochlosomosis

Peter Sandmeier

CLASSIFICATION

The genus *Cochlosoma* includes intestinal flagellates in birds and mammals. *Cochlosoma anatis*, which causes disease in poultry and waterfowl, is the only species that has been described within this genus.[1] More distantly defined *Cochlosoma* spp. have been reported as pathogens in various nondomestic avian species.

Morphologically, the presence of an adhesive disk suggests a possible relationship to *Giardia*, whereas the presence of the parabasal apparatus and the axostyle suggests a relationship to trichomonads. For many years, the genus *Cochlosoma* was described as belonging to the family Cochlosomidae, distinct from the families Trichomonadidae and Hexamitidae.[2] More recently based on the 16S rRNA gene sequence the genus *Cochlosoma* has been placed in the family Trichomonadidae within the order Trichomonadida together with the genera *Trichomonas*, *Trichomonoides*, *Tetratrichomonas*, and *Pentatrichomonas*.[3]

MORPHOLOGY

C. anatis is an oval-shaped flagellate that measures between 6 to 12 μm long and 4 to 7 μm wide. It tapers from a broad, rounded anterior end to a narrow, tapered posterior end. In light microscopy it shows a characteristic ventral sucking disk, which opens to the left side.[4] In stained smears, a centrally located nucleus and an axostyle, which curves dorsally along the curvature of the body, are clearly visible.[5] *Cochlosoma* spp. are readily distinguished from other flagellates by their jerking spiral motion. The six flagella produce a whipping movement causing a jerking motion and rotation around the long axis (Figure 25.1).[6]

On transmission electron microscopy, *C. anatis* has a kinetosomal complex. The kinetosomes (or basal bodies) 1 to 4 of the anterior flagella are located below the adhesive disk. The kinetosome of the recurrent flagellum is oriented perpendicular to the kinetosomes 1 to 4. The sixth flagellum kinetosome is located outside the complex on the dorsal side of the body. The parabasal apparatus is situated anteriorly beneath the kinetosomal complex and includes the parabasal fiber as well as the parabasal body or Golgi complex. The nucleus is located at the broad anterior part of the cell and is surrounded by a double membrane with pores. *C. anatis* has no mitochondria. The cytoplasm contains various food vacuoles, cisterns of the rough endoplasmic reticulum, free ribosomes, and glycogen particles (Figure 25.2).[7] In hematoxylin and eosin-stained histological sections, little attachment of the ventral suction disk to epithelial cells is observed. Although the exact role of the ventral disk in the pathogenicity of *C. anatis* infections is unknown, its morphology suggests that it serves for the attachment to the intestinal mucosa (Figure 25.3).[6]

LIFE CYCLE

Like all trichomonads, *C. anatis* reproduces asexually by cell division (binary fission). Electron microscopy images have documented longitudinal binary fission of the trophozoite stage and pseudocyst formation. A pseudocyst is a cyst with a flexible membrane covering the trophozoites. This pseudocyst stage is presumed to be a mechanism for transmission of *C. anatis*.[8]

COCHLOSOMOSIS IN COMMERCIAL POULTRY AND WATERFOWL

Transmission

Transmission studies in turkeys (*Meleagris gallopavo*) have confirmed the fecal-oral route of transmission. In turkeys orally infected with 1×10^7 trophozoites, the development of the parasite occurred throughout the digestive tract, and they were detectable within 5 to 6 days.[9]

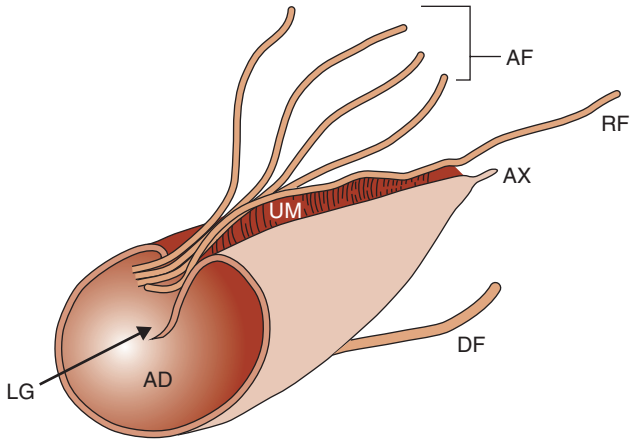


FIGURE 25.1 Schematic drawing of *Cochlosoma anatis* morphology features. *AD,* Adhesive disk; *AF,* anterior flagella; *AX,* axostyle; *DF,* dorsal flagellum; *LG,* lateral groove; *RF,* recurrent flagellum; *UM,* undulating membrane.

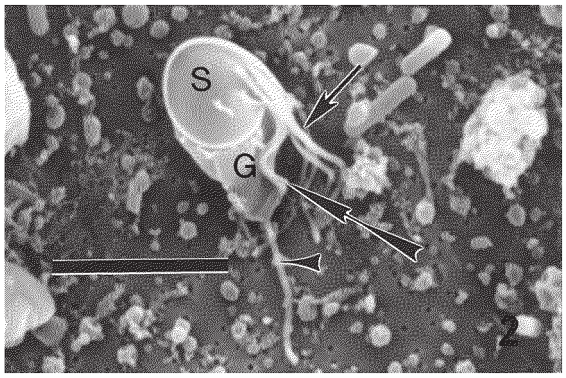


FIGURE 25.2 Scanning electron micrograph of the ventral surface of a *Cochlosoma anatis* trophozoite. Note the ventral sucking disk *(S)*, the prominent grove *(G)*, anterior flagella *(arrow)*, undulating membrane *(double arrowhead)*, and recurrent flagellum *(arrowhead)*. (Magnification 6660×; bar = 5 μm.) (Reprinted with permission from Lindsay DS, Larsen CT, Zajac AM, Pierson FW: Experimental *Cochlosoma anatis* infections in poultry, *Vet Parasitol* 81(1):21–27, 1999. doi:10.1016/s0304-4017(98)00227-1.)

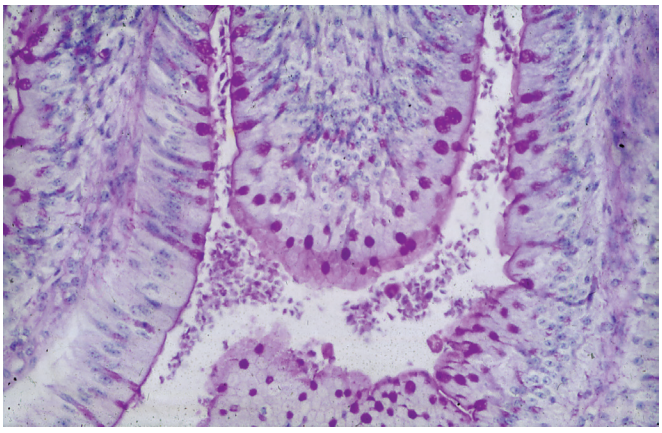


FIGURE 25.3 *Cochlosoma* spp. within the intestinal tract of a Gouldian finch (periodic acid-Schiff stain, 25×). (Reprinted with permission from Gerry M. Dorrestein.)

Trophozoites were readily destroyed by immersion in water. Trophozoites exposed to tap water or distilled water for 45 minutes at 37°C were nonmotile and appeared dead upon microscopy. None of the turkeys inoculated with trophozoites exposed to water for 5 minutes had demonstrable infections upon necropsy. This indicates that contamination of litter or feed is likely the primary means of transmission to other turkeys.[9] Only one of 12 turkeys became infected when placed on litter within 2 hours after the experimentally infected birds had been removed, whereas 7 of 8 turkeys became infected when placed in boxes together with experimentally infected turkeys that were excreting trophozoites. This suggests a lack of long-term survival of trophozoites in the environment.[9]

Cross-species transmission showed that four of six bobwhite quails (*Colinus virginianus*) and one of eight chickens (*Gallus gallus domesticus*) orally inoculated with *C. anatis* trophozoites from turkeys had demonstrable infections. Trophozoites were observed only in the ilea of quail and the ceca of positive chickens. Trophozoites collected from chickens and quails remained infectious to turkeys.[9] Over a 3-year period, 181/1996 tested houseflies collected from turkey farms were shown to be positive for *C. anatis* using a polymerase chain reaction (PCR) detection. Positive houseflies were collected inside and outside turkey facilities, proving that they potentially move the protozoan parasite to and from adjacent poultry facilities.[10]

Incubation Period

Experimentally inoculated 1-day-old Muscovy ducklings (*Cairina moschata*) were shedding *C. anatis* trophozoites by 7 days postinoculation.[11] Postmortem examination of inoculated turkeys showed that by 4 days postinoculation some turkeys were positive for *C. anatis* on intestinal scrapings and by 6 days postinoculation all were positive.[9]

Hosts

Naturally occurring *C. anatis* infections have been described in turkeys,[12,13] ducks,[11] and geese.[14] Experimental infections have been produced in chickens, turkeys, bobwhite quails, and ducks.[9,11,15]

Site of Infection

With the exception of infections in turkeys *Cochlosoma* spp. are found mainly in the large intestine, cecum, colorectum, and cloaca. *C. anatis* was first described in the cecum of the domestic duck (*Anas platyrhynchos*).[1] Similarly, in another study *C. anatis* was found in the large intestine below the junction of the cecum and in the cloaca.[6] In geese (*Anser anser domesticus*), *C. anatis* was also found primarily in the large intestine but also in the cecum.[7,14] In both natural and experimental infections in turkeys, *C. anatis* has been described in large numbers in the small intestine.[9,12]

Clinical Signs and Coinfections in Waterfowl and Poultry

Clinical signs described in affected turkeys included intense thirst, diarrhea, depression, ruffled plumage, loss of appetite, and death in chicks between 2 and 12 weeks of age.[5,12] In addition to infection with *C. anatis*, co-infections with *Trichomonas* spp., *Hexamita* spp. and other pathogens have been described. Stunted diarrheic turkey poults, 1 to 7 weeks of age, demonstrated a variety of intestinal parasites including *C. anatis* and concurrent co-infections with *Cryptosporidium* spp., *Hexamita* spp., *Coccidia* spp., *Chilomastix* spp., and *Trichomonas* spp.[13]

Co-infections with bacterial and viral pathogens are also described. In ducklings, a runting syndrome associated with *C. anatis* has been described.[11] In addition, small numbers of *Trichomonas* spp., *Hexamita* spp., and *Campylobacter jejuni* were isolated from the runted ducklings. In an experimental study, a combination of *C. anatis* and turkey coronavirus was proven more pathogenic than either agent alone.[16] In another study, a pure *C. anatis* infection in specific pathogen-free turkeys produced no clinical signs and only a mild decrease in production.[16] These results suggest that, although *C. anatis* can cause a decrease in productivity, clinical signs of disease should only be expected in waterfowl or poultry suffering from co-infections with other protozoa or bacteria or in juvenile birds.

Prophylaxis/Treatment in Commercial Poultry

Feeding young turkey poults inoculated with *C. anatis* with nitarsone or diclazuril did little to ameliorate production losses.

Although feeding nitarsone at US Food and Drug Administration (FDA)–approved levels (170.3 g/ton) decreases the severity of the infection and the number of positive poults, the production losses are not fully prevented. Feeding diclazuril (0.91 g/ton) had no significant efficacy in minimizing the infection or in decreasing the number of positive poults.[4]

COCHLOSOMOSIS IN FINCHES AND WAXBILLS

Classification

Cochlosoma spp. described in finches and waxbills were identified by microscopic identification of motile *Cochlosoma* trophozoites.[17,18] It is not known if these represent a new and different species or the only described species, *C. anatis* (Table 25.1).

Transmission

Although no information is available on the transmission of *Cochlosoma* spp. in finches and waxbills, it can be assumed that asymptomatic adult birds will transmit the parasite via fecal contamination to the nonimmunocompetent nestlings.

Hosts

Cochlosoma spp. infections have been reported in a variety of finches, mainly red-headed parrot finches *(Erythrura cyaneovirens)*, Bengalese or society finches *(Lonchura striata domestica)*, and Gouldian finches *(Erythrura gouldiae)*, but also in blue-faced parrot finches *(Erythrura trichroa)*, zebra finches *(Taeniopygia guttata)*, nutmeg mannikins *(Lonchura punctulata)*, double-barred finches *(Taeniopygia bichenovii)*,[17,19] and various waxbill species.[18]

Site of Infection

In Bengalese or society finches, serial sectioning of the digestive tract revealed presence of the flagellates within the colorectum and cloaca.[17]

Clinical Signs in Waxbills and Finches

In waxbills and finches, adult birds appear to be asymptomatic carriers. Clinical signs can be expected in young birds from 10 days to 6 weeks of age.[20] These signs include dehydration, diarrhea, whole seeds in the droppings, yellow staining of nestlings, and death.[21] Clinical signs classically occur in chicks hatched by asymptomatically infected Bengalese or society finches used as foster parents.[21]

Diagnosis

Diagnosis is made by demonstrating the jerking motion of the motile flagellates in saline smears from fresh warm feces.[21]

Prophylaxis and Treatment

Prophylaxis in finch and waxbill breeding facilities is best performed by screening fresh fecal smears of all newly introduced birds and of all breeding birds at the start of the breeding season.[21]

Cochlosoma spp. can be treated with the same medication as other flagellates such as *Trichomonas* spp. Ronidazole at 50 mg/L of drinking water and 50 mg/kg of soft food for 7 days has proven to be effective.[21] Dosages up to 400 mg/L of drinking water have been described but the lower dosages have been shown to be equally effective.[17] Metronidazole has also been proven to be effective at 30 mg/kg PO once only or 40 to 80 mg/L drinking water for 3 days.[17] Finally, dimetridazole at 100 mg/L drinking water for 5 days will also be effective against infections[21] with dosages up to 250 mg/L drinking water having been described. Be aware that overdosage can result in reversible central nervous system signs.[21] If bacterial co-infections are suspected, these drugs can be used in combination with antibiotics.

Differential Diagnosis

Other diseases causing similar signs in nestling birds include infections with *Isospora* spp., *E. coli* and other bacteria, infections with *Macrorhabdus ornithogaster*, and toxic enteritis.[21]

TABLE 25.1

Quick Reference: Cochlosomosis in Waxbills and Finches

Age Group	Young Birds 1–6 Weeks of Age
Clinical signs in juveniles	Dehydration, diarrhea, yellow staining of nestlings, death
Clinical signs in adult birds	Asymptomatic
Transmission	Asymptomatic adult birds to nonimmunocompetent nestlings
Diagnosis	Motile flagellates in saline smear of fresh warm feces
Prophylaxis	Screening of fresh fecal smears during quarantine and before breeding season
Treatment	Ronidazole, metronidazole, dimetridazole

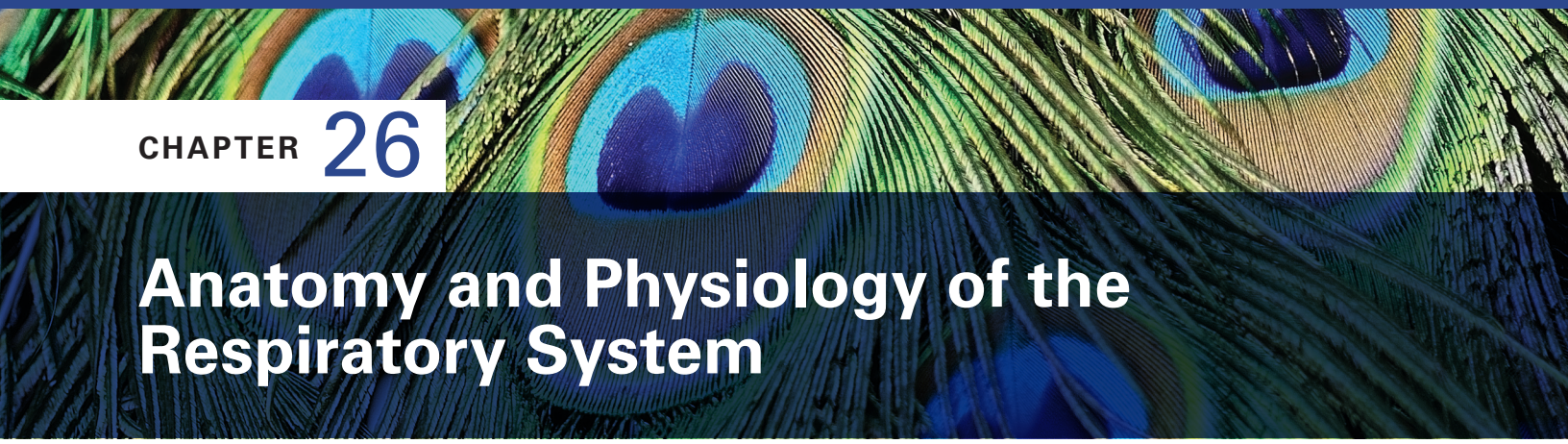
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CHAPTER 26

Anatomy and Physiology of the Respiratory System

Susan E. Orosz

INTRODUCTION

The primary function of the respiratory system is to provide oxygen to the tissues and remove carbon dioxide. In addition, the respiratory system is involved with thermoregulation by evaporative losses and for vocal communication. Moreover, the lungs are the site for the conversion of angiotensin I to angiotensin II for the maintenance of blood pressure.

The anatomy of the respiratory system has several unique anatomic features. These features most likely result from evolutionary consequences that allowed for flight. In birds, ventilation uses one-way parabronchial valving and air sacs, which segregate ventilation from gas exchange.[1] The separation of these functions provided a more efficient system for gas exchange compared to mammals in which gas exchange and ventilation both occur at the level of the alveoli.

Interestingly, when the two functions are separated, it allows the lungs to be smaller. As such, the lungs of birds have a reduced volume, which ranges from 1% to 3% compared to 7% for mammals. However, the lungs and air sacs combined have a total volume of 15%, which is much higher than mammals. As the fixed lungs do not change in volume during the respiratory cycle, the paired air sacs act as a bellow to move air through the parenchyma in a unidirectional manner. This allows for a greater surface area for gas exchange between the air capillaries and blood, when compared with the movable alveoli of mammals.

Another important difference between mammals and birds is the thoracic cage and its dynamics. Birds do not have a diaphragm that separates the thoracic cage from the abdominal cavity as in mammals. As a result, the pressure within the thorax of birds is essentially atmospheric without the possibility to create differences in pressure, as is the case in mammals, which have subatmospheric pressures in their thoracic cage, thereby allowing air to rush into the lungs.

The specialized respiratory system of birds is extremely efficient when the patient is healthy. However, when ill, respiratory signs are often pronounced, and respiratory distress is a common reason for birds to be presented to the veterinarian. The first step in assessing birds with respiratory signs is to determine whether the signs are localized to the upper or lower respiratory tract or whether both components are affected (see Chapter 27). Moreover, a basic understanding of the anatomy and physiology of the avian respiratory system is required to successfully manage the patient. This chapter aims to provide an overview of the anatomical and physiological characteristics of the avian respiratory tract that are relevant for the veterinary clinician dealing with birds with respiratory distress.

UPPER RESPIRATORY SYSTEM

Nasal and Sinusal Cavities

The cere is the area around the most dorsal surface of the maxillary rhamphotheca or upper bill. It may be feathered or nonfeathered, depending on the species of bird involved. In addition, ceres in each species have their own characteristic size and shape. In adult male budgerigars *(Melopsittacus undulatus)* of the wild phenotype, the cere is usually blue (Figure 26.1); in adult females it is usually brownish pink. Changes in the cere's normal color pattern are suggestive of gonad tumors in this species.

Air moves into the nasal cavity through the nares or nostrils, which are located dorsally within the area of the cere at the base of the beak (Figure 26.2). In several species, the nares are covered by feathers making them hard to visualize. Examples of birds with feathered nares include Eclectus parrots *(Eclectus roratus)*, some species of cockatoos (e.g., *Callocephalon fimbriatum*, *Cacatua ducorpsii*, *C. goffiniana*, *C. haematuropygia*, *C. leadbeateri*, *C. pastinator*, *C. sanguinea*, *C. tenuirostris*, *Eolophus roseicapilla*, *Probosciger aterrimus*) and macaws (e.g., *Anodorhynchus* spp., *Ara ambiguus*, *Ara militaris*, *Ara rubrogenys*), crows (*Corvus* spp.), and grouse (Tetraonini). Some may be perforated as in Gruiformes and Cathartiformes; closed as in Pelecaniformes; tubular in shape as in Procellariiformes; or located at the tip of the bill as in kiwis (*Apteryx* spp.).[2–4] In

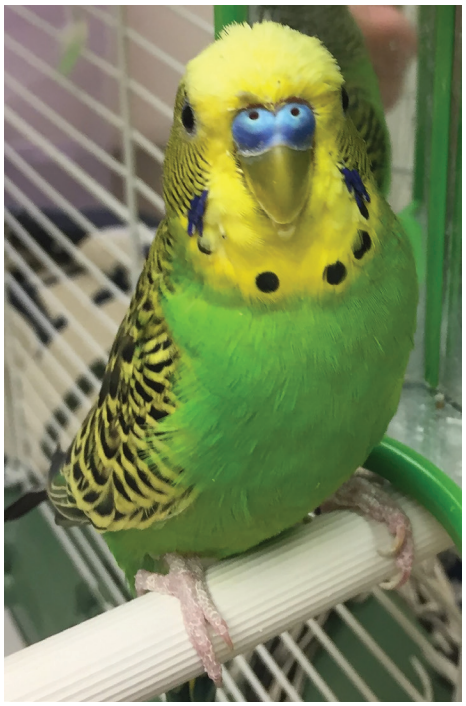


FIGURE 26.1 Budgerigar *(Melopsittacus undulatus)* with a blue cere that signifies a male with the wild phenotype green coloration. (Photo courtesy Susan Orosz.)



FIGURE 26.2 Orange-winged Amazon parrot *(Amazona amazonica)* head. Note the location of the nostrils at the base of the beak, at the cere, with the presence of opercula inside. (Photo courtesy Susan Orosz.)

many parrot species, including grey parrots *(Psittacus erithacus)*, Amazon parrots (*Amazona* spp.), and Galliformes, there is a rounded, keratinized structure called the operculum, which is found in the rostral-most extent of the nasal cavity. It acts as a baffle to deflect and prevent inhalation of foreign bodies.[3] The openings of the nares may be shaped abnormally as a result of a chronic upper respiratory infection. Therefore,

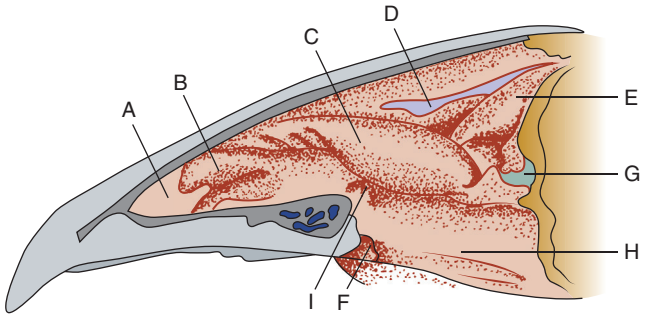


FIGURE 26.3 Anatomical structure of the nasal cavity through a vertical sectional view of a chicken's *(Gallus gallus domesticus)* skull. *(A)* Nostril, *(B)* concha nasalis rostralis, *(C)* concha nasalis media, *(D)* continuation of the nasal meatus, *(E)* concha nasalis caudalis, *(F)* vomer, *(G)* opening into sinus cavity, *(H)* choanal cleft, *(I)* opening of ductus nasolacrimalis. (Reprinted from Kang H, Yan M, Yu Q, Yang Q: Characteristics of nasal-associated lymphoid tissue (NALT) and nasal absorption capacity in chicken, *PLoS One* 8(12):e84097, 2013.)

clinicians should be familiar with the normal shape and size of the nares of the species that is presented. Changes from normal, as well as possible occlusions obstructing airway passage, should be noted during the physical examination.

The nasal cavity in most species is divided by a nasal septum. Within the lateral walls of the nasal cavity are highly vascularized nasal conchae. Most birds have three scroll-like cartilaginous nasal conchae, that is, the rostral, middle, and caudal nasal conchae (Figure 26.3). The middle nasal concha is the largest of the three.[3]

The rostral portion of the nasal cavity (Figure 26.4) can have a vestibule that is lined by a squamous epithelium. This epithelium continues over the rostral nasal conchae. Distally, the epithelium changes to a mucociliary epithelium over the middle nasal conchae. The caudal nasal conchae, which is absent in grey parrots,[2,4] have an olfactory epithelium for sensing smell. In all aquatic birds, there is a mucosal fold that acts as a valve to deflect the water from the caudal conchae where olfaction is detected. This sense of smell is highly developed in a number of species, including albatrosses (Diomedeidae), which can discern bacon up to 10 miles away on the ocean surface. Some species with increased scrolling of the caudal nasal conchae include vultures (Cathartidae, Aegypiinae, Gypaetinae,), crows and ravens (Corvidae), hummingbirds (Trochilidae), and kiwis. The increasing amounts of scrolling of this concha in those species are hypothesized to be linked to a greater sense of smell, most likely as a result of the increased surface area. It is thought that this may be important for location of food.

The nasal gland, or formerly the salt gland, often has two lobes—a medial and lateral—with a duct that opens into its own ostium at the vestibular region of the rostral nasal cavity. In marine birds, the gland concentrates salt from the salt water ingested and secretes it, helping to maintain the osmolality of the blood. It serves a similar function in other species of birds that have access to a limited water supply including some of those in deserts, such as the ostrich *(Struthio camelus)*.[3]

A clinically important anatomic feature is the relationship of the middle nasal conchae to the openings of the infraorbital sinus, which is the only true paranasal sinus of birds. The infraorbital sinus in various publications has been referred to



FIGURE 26.4 Photographs of the transverse sections of the nasal cavity at the level of the rostral vestibular region of a Muscovy duck *(Cairina moschata)* (A), Egyptian goose *(Alopochen aegyptiaca)* (B), laughing dove *(Spilopelia senegalensis)* (C), and Japanese quail *(Coturnix japonica)* (D) showing the rostral *(RC)* and middle *(MC)* nasal conchae and the nasal septum (*asterisk*). (From Madkour F: Anatomical descriptions of the nasal cavity of the aquatic and non-aquatic birds, Int J Vet Sci 2(2):101–110, 2019. https://journals.ekb.eg/article_44862.html.)

as the "subocular sac," "orbital sinus," "maxillary sinus," and several other terms. As the infraorbital sinus is located within the antorbital fossa, the term "antorbital sinus" has also been proposed.[5] For the purposes of uniformity, this sinus is referred to as the infraorbital sinus in this book, as this is the most common name used in the veterinary literature. The infraorbital sinus opens dorsally into both the middle and caudal nasal conchae (Figures 26.5 and 26.6). The caudal nasal concha drains only into the nasal cavity by its dorsal opening into the infraorbital sinus. As a result, the only passageway for drainage of mucopurulent material in the infraorbital sinus is through the dorsal opening of the caudal nasal concha, or over the middle nasal concha, into the nasal cavity. This is important from a clinical perspective as drainage of materials from infections of the infraorbital sinus is limited due to these dorsal openings.

The infraorbital sinus acts to pneumatize the head and neck and is suggested to lighten the head for flight. It is located ventromedial to the orbit and has numerous diverticula. In psittacine birds, the diverticula are well developed, whereas they are absent in other species such as cormorants (*Phalacrocorax* spp.). In most birds, a rostral diverticulum of the infraorbital sinus extends into the maxillary rostrum or bill, which is unpaired (Figure 26.7), but in macaws there appear to be two unpaired rostral diverticula with a transverse canal. In Amazon parrots and macaws, at least there are eight paired diverticula that are distal to the rostral one. These include paired preorbital diverticula, which lie on either side of the skull rostral to the orbit; paired postorbital diverticula, which may be subdivided to surround the opening of the ear; and paired mandibular diverticula, which extend into the mandibular rostrum.[6,7] In addition to its communication with the nasal conchae dorsally, the infraorbital sinus also communicates with the

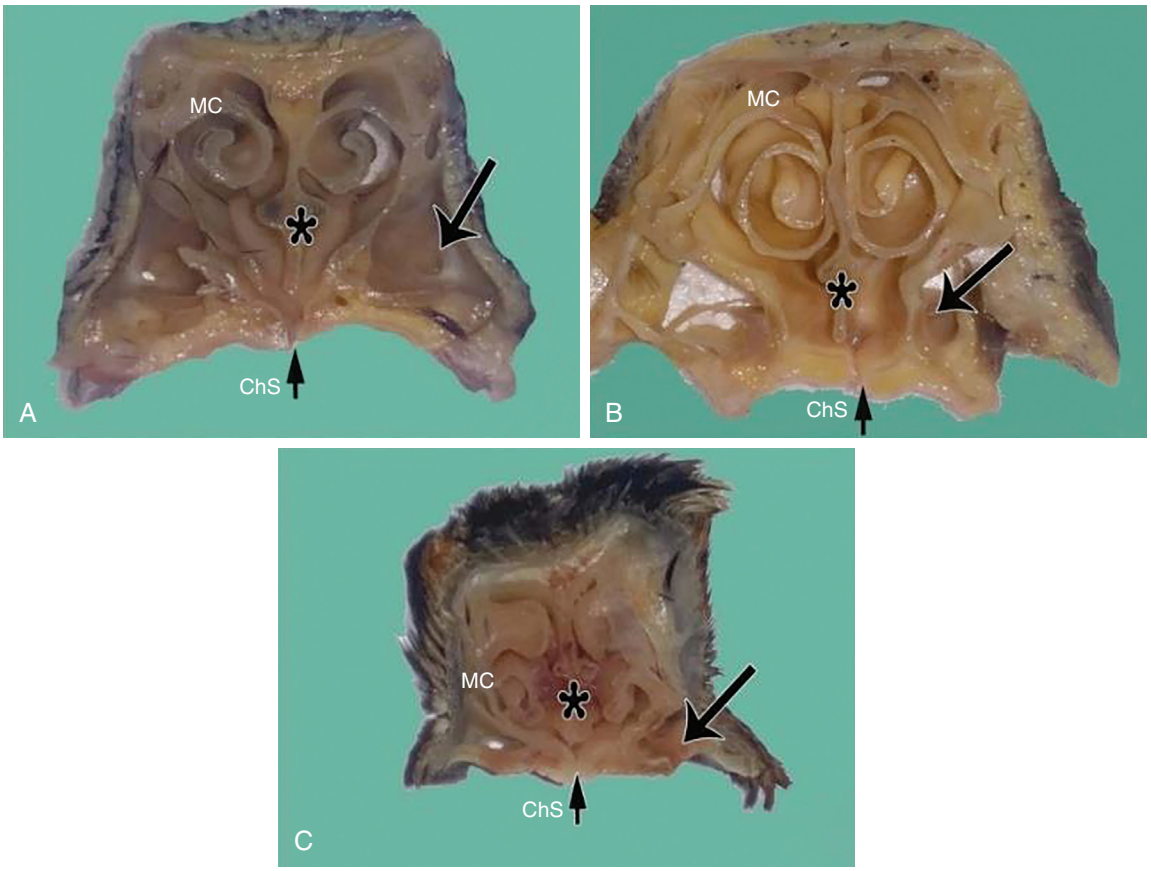


FIGURE 26.5 Photographs of the transverse sections of the nasal cavity at the level of the middle respiratory region of a Muscovy duck *(Cairina moschata)* (A), Egyptian goose *(Alopochen aegyptiaca)* (B), and Japanese quail *(Coturnix japonica)* (C) showing the middle *(MC)* nasal concha, nasal septum *(asterisk),* choanal slit *(ChS),* and infraorbital sinus *(arrow).* (From Madkour F: Anatomical descriptions of the nasal cavity of the aquatic and non-aquatic birds, *Int J Vet Sci* 2(2):101–110, 2019. https://journals.ekb.eg/article_44862.html.)

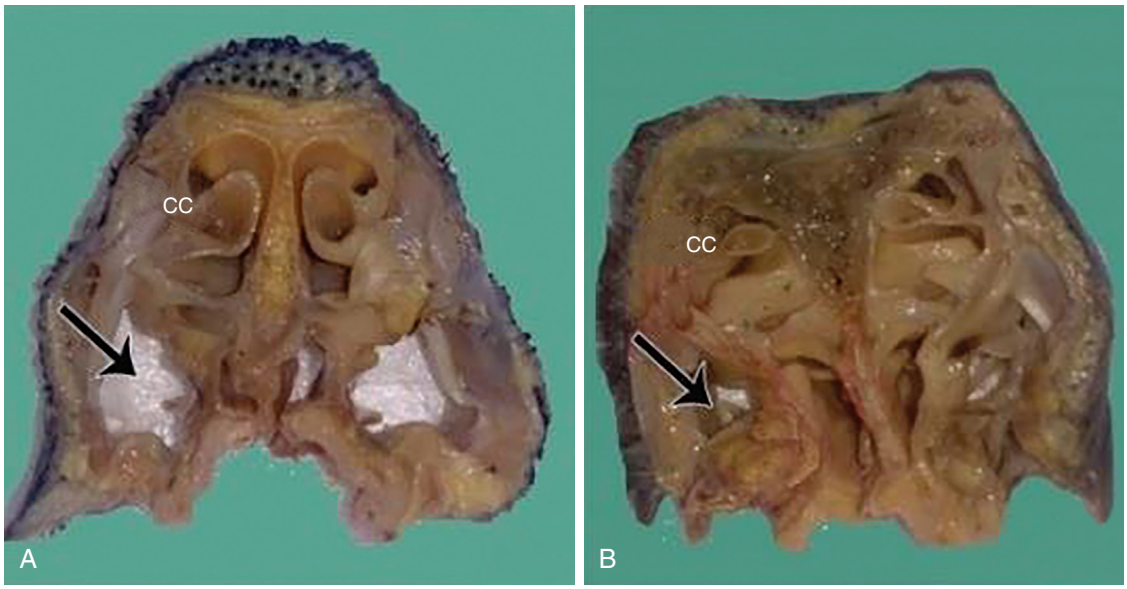


FIGURE 26.6 Photographs of the transverse sections of the nasal cavity at the level of the caudal olfactory region of a Muscovy duck *(Cairina moschata)* (A), Egyptian goose *(Alopochen aegyptiaca)* (B), laughing dove *(Spilopelia senegalensis)*

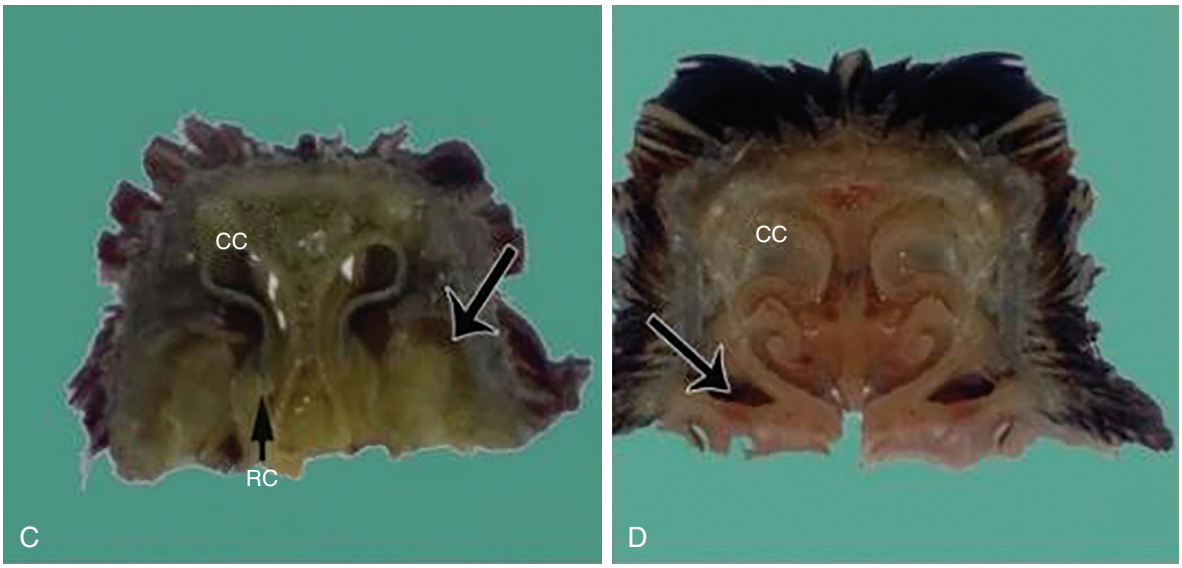


FIGURE 26.6, cont'd (C), and Japanese quail *(Coturnix japonica)* (D) showing the rostral *(RC)*, and caudal *(CC)* nasal conchae and infraorbital sinus *(arrow)*. (From Madkour F: Anatomical descriptions of the nasal cavity of the aquatic and non-aquatic birds, *Int J Vet Sci* 2(2):101–110, 2019. https://journals.ekb.eg/article_44862.html.)

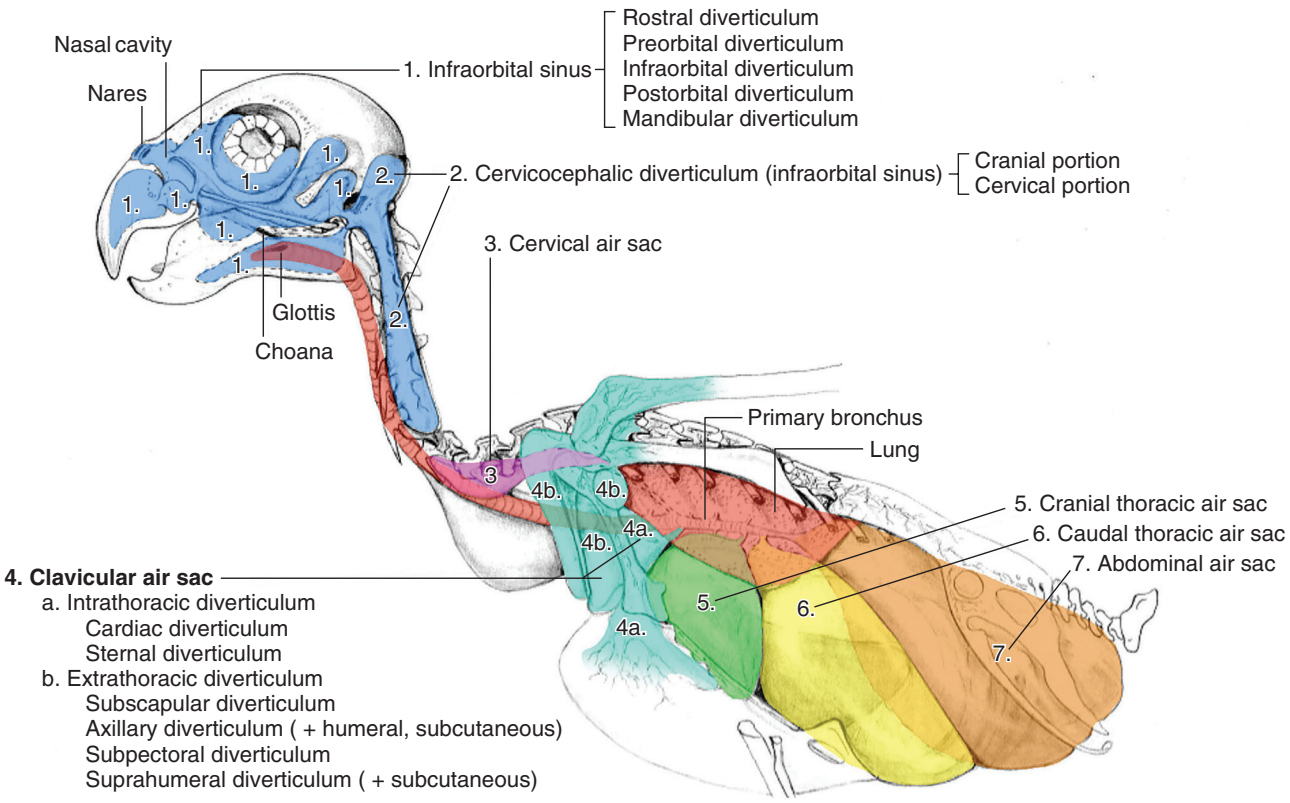


FIGURE 26.7 Respiratory anatomy of the Amazon parrot (*Amazona* spp.). (Modified and reprinted with permission from McKibben J, Harrison G: Clinical anatomy, with emphasis on the Amazon parrot. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986, W.B. Saunders, pp 31–66.)

cervicocephalic diverticulum at its caudal-most extent. This diverticulum is the largest of the diverticula in psittacines and may extend to the thoracic inlet. While it has been classified as an air sac due to its large size, it is not connected to the air sacs that are associated with the lungs.[4,6]

The infraorbital sinus extends out from the skull in various locations so that its lateral walls underlie soft tissues and skin (Figure 26.7). In case of an infection of inflammatory response, this can result in swellings that are noticeable on the exterior surface of the head, as is commonly seen in Psittaciformes and Galliformes. Several bones of the skull, including the quadrate bone, articular bone, and bones of the braincase, are pneumatized through its connection with the tympanic cavity.[5] The infundibular cleft is a slit-like opening just caudal to the choanal slit. This cleft is the opening of the right and left Eustachian or pharyngotympanic tubes that connect to the middle ear cavity. The opening of this tube at the cleft is without folds to quickly equalize pressures during a dive or when flying at higher altitudes and then descending.[3] Knowledge of the relationship of the infraorbital sinus and its diverticula in relation to the bones of the skull is important during examination of the upper respiratory system, during irrigation/flushing of the infraorbital sinus, and for surgical (drainage) procedures.

The Larynx, Trachea, and Syrinx

While anatomists often describe the upper respiratory tract extending from the nares to the choanal slit, others include the larynx through the trachea. The larynx is composed of four partially ossified cartilages with an overlying mucosa or laryngeal mound (Figure 26.8A). The median cricoid cartilage articulates dorsally with a small median comma-shaped procricoid cartilage. This procricoid cartilage has a gliding contact with the paired arytenoid cartilages. The glottis is the opening between these two arytenoid cartilages. They are abducted by a dilator muscle and adducted by a constrictor muscle, regulating the size of the opening (Figure 26.8B,C). The larynx is not involved in the production of sound in birds. However, it may alter the sound produced, as has been observed in parrots.[8–10]

The mucosa covering the laryngeal cartilages is a pseudostratified epithelium with unicellular mucus glands. There are cilia associated with the areas where the ridges project.[3] This entrance to the trachea is not covered by an epiglottis as it is in mammals, making it easier to intubate a bird compared with a mammal. In addition, birds do not have a thyroid cartilage. In some species of birds, including but not limited to pelicans (*Pelecanus* spp.), hornbills (Bucerotidae), kiwis, penguins (Spheniscidae), and some ducks,[4,10] there is a median crest *(crista ventralis)* that comes off the cricoid cartilage ventrally, thereby reducing the opening of the glottis in that location. This should be taken into consideration when intubating these species, and care should be taken to examine the opening in others as they may have one like it or another complication.

The *rima glottidis* and trachea are wider in diameter on a per weight basis than they are in mammals, being up to 1.3 times the radius of that of a mammal of similar size. This principle provides another reason why it is easier to intubate birds than mammals. This increased diameter directly reflects one physiologic adaptation of birds: the use of their beaks for the manipulation of objects. The increased length of the trachea (2.7 times that of a mammal of similar size) is part of an adaptation in birds whereby the increased length of their necks allows the use of their beak to manipulate objects. However, the increase in length also causes an increased air resistance. A wider *rima glottidis*, as well as a wider trachea, is necessary to reduce this resistance. This results in an increased dead space, which is four times that of a similarly sized mammal.[3] To compensate for the wider and longer trachea and its corresponding increase in dead space, the respiratory rate of birds is less than that of mammals of similar size with a larger tidal volume. These anatomic and physiologic differences are important when selecting endotracheal tubes and ventilating birds for anesthetic procedures.

The tracheal rings of birds are complete (Figure 26.9) and shaped like a signet ring. The shape is fairly constant across species.[3] The cartilages that make up the tracheal rings overlap to form a more rigid trachea compared with mammals. These adaptations are important to reduce the possibility of tracheal kinking when moving the neck. The risks of overinflation of a cuffed endotracheal tube are potentially greater in birds because of the less expansive nature of the trachea and

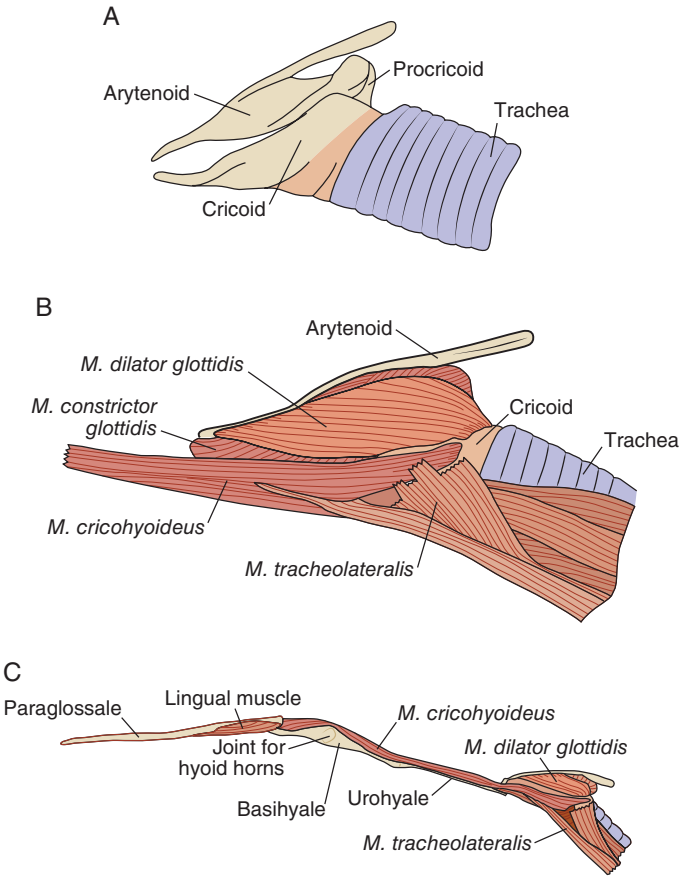


FIGURE 26.8 Lateral view of the larynx and upper end of the trachea in a snowy egret *(Egretta thula)*: (A) skeletal elements, (B) extrinsic and superficial intrinsic laryngeal muscles, and (C) the laryngeal apparatus with its muscular connections to the hyoid apparatus. (Reprinted from Homberger DG: The avian tongue and larynx: multiple functions in nutrition and vocalisation. In Adams NJ, Slotow RH, editors: *Proceedings of the 22nd International Ornithology Congress*, Durban, Johannesburg, 1999, BirdLife South Africa, pp 94–113.)

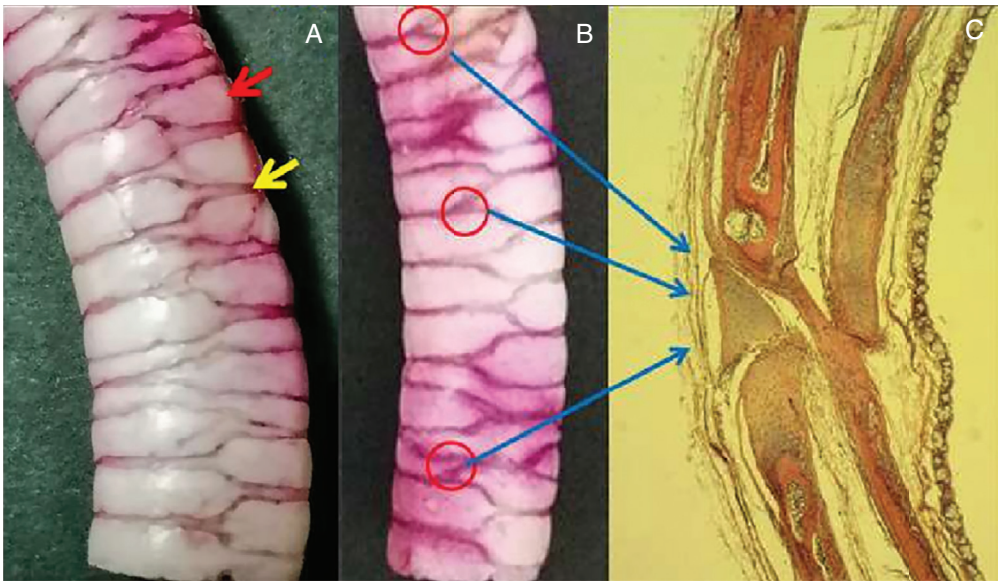


FIGURE 26.9 (A) and (B) Dorsal view of the trachea of a female peacock *(Pavo cristatus)* demonstrating the shape of the tracheal cartilages and the way they overlap; *red arrow*, wide part of tracheal ring; *yellow arrow*, narrow part of tracheal ring. (C) Histological section showing the connection and overlap area between tracheal rings. (Reprinted from AL-Araji HM, Mohammed HH: Comparative anatomical study of the trachea in adult male and female indigenous peafowl (*Pavo cristatus*), *Ann For Res* 66(1):1149–1157, 2023.)

can lead to mucosal edema, bleeding, necrosis, scarring, stricture formation, and respiratory compromise. Thus, whenever possible, uncuffed endotracheal tubes should be used, or the cuff should be left deflated. In addition, when intubating a bird, the tube should not be inserted too deep as in several species (e.g., domestic fowl, macaws, cockatoos) up to 45% decreases in the distal tracheal diameter have been reported, which can significantly increase the risk for trauma and stricture formation following deep endotracheal intubation.[3,11]

The location of the trachea varies with the species. In passerines (Passeriformes) and chickens *(Gallus gallus domesticus)*, the trachea initially sits in the midline but moves to the right side of the neck before returning to the midline at the thoracic inlet. In spoonbills (*Platalea* spp.) and curassows (Cracinae), the trachea forms coils under the skin and pectoral muscles. In swans (*Cygnus* spp.) and cranes (Gruidae), these coils are excavated into the sternum. In penguins, the trachea bifurcates at the cranial neck into right and left halves that are separated by a cartilaginous septum that is continuous with the tracheal rings. There is a tracheal sac that is an inflatable diverticulum in the ruddy duck *(Oxyura jamaicensis)* and the emu *(Dromaius novaehollandiae)*. In many male Anseriformes, the trachea expands at its distal end into a tracheal bulb (Figure 26.10).[3]

The trachea often bifurcates into a right and left bronchus immediately after entering the thoracic inlet. The syrinx, which is the voice box of birds, is located at this bifurcation. The syrinx is a complex structure and is unique to birds. While it is highly variable among species, it is composed of several cartilages, which may be (partially) ossified, and of syringeal muscles and membranes. The derivation of the cartilages—from the trachea and/or bronchi—determines its

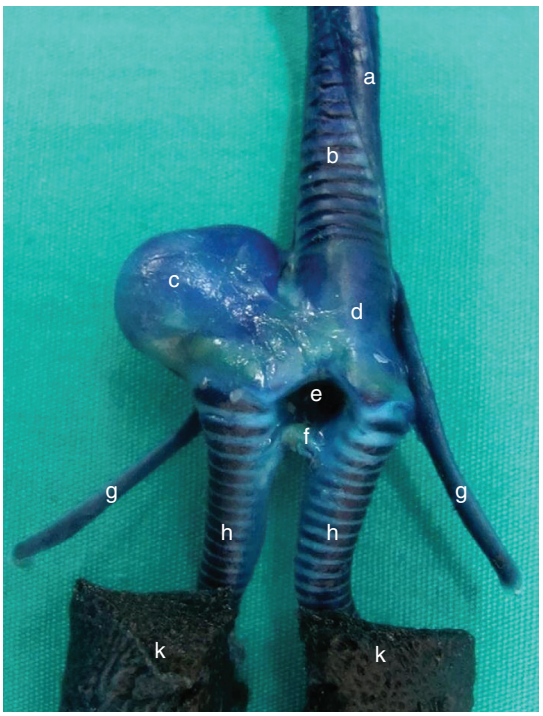


FIGURE 26.10 Dorsal view of the syrinx in the male mallard (*Anas platyrhynchos*): tracheolateral muscle (*a*), trachea (*b*), syringeal bulla (*c*), tympanum (*d*), interbronchial foramen (*e*), interbronchial ligament (*f*), sternotracheal muscle (*g*), right and left primary bronchi (*h*), and lungs (*k*). (Reprinted from Yilmaz B, Yilmaz R, Arican I, Yildiz H: Anatomical structure of the syrinx in the mallard *(Anas platyrhynchos)*, *Harran Univ Vet Fak Derg* 1(2):111–116, 2012.)

classification (tracheal, tracheobronchial, or bronchial), although this may be difficult to determine. New-world vultures (Cathartidae) lack a syrinx. The location of the syrinx can vary widely across bird groups. In most species, including all songbirds, the syrinx is classified as tracheobronchial as the syrinx covers both the lower end of the trachea and the upper parts of the bronchi, suggesting that both elements are involved in the syrinx, with partially ossified cartilages. In other, nonpasserine birds, notably owls (Strigiformes), cuckoos (Cuculidae), and nightjars (Caprimulgidae), the syrinx may be restricted to the bronchi, or to the trachea, as can be observed in a very small number of bird groups that are collectively known as tracheophonae. This group of tracheophonae comprises a subset of the suboscine Passeriformes that includes ovenbirds (Furnariidae), woodcreepers (Dendrocolaptidae), ground antbirds (Formicariidae), typical antbirds (Thamnophilidae), tapaculos (Rhinocryptidae), and gnateaters (Conopophagidae).

The syringeal muscles are important for sound production as they modulate the sound shape by changing the tension of the membranes and the bronchial openings. The muscles to the syrinx are supplied by the 12th (hypoglossal) cranial nerve (CN XII). Most syringeal muscles are external to the tracheal bifurcation (Figure 26.11).[3] However, depending on the species, birds may also have internal syringeal muscles that are located within the lumen of the trachea and/or bronchi. Passerines, for example, can have up to five pairs of internal muscles of the syrinx. Psittacines have two pairs of short syringeal muscles, that is, a superficial and deep pair. In addition, parrots lack a pessulus that is an upright bar of cartilage in the midline between the dividing bronchi (Figure 26.12).[4,12,13] The narrowed internal diameter of the syrinx is a common site for tracheal granulomas and foreign bodies.

The syrinx functions to produce voice. How that happens is not well understood and may vary with the species examined. It is thought that the tympaniform membranes vibrate to produce sound. Another alternative is that the membranes form two slots that produce two streams of turbulent air like in a whistle. It is thought that the vibration of the membranes results from the changes in the pressure of the clavicular air sac, the syrinx, and its lumen. At the onset of expiration, an elevation in pressure in the clavicular air sac closes the syrinx momentarily as the tympanic membranes are forced into the lumen. Tension is applied to the membranes, which results in vibration as air flows past these partially tightened membranes, thereby producing sound.[3] While it is generally believed that sound is produced only during expiration, some of the warblers may be able to use mini breaths to trill and produce pulsed sounds that are unbroken for long periods of song. Breathing and vocalization are coordinated through nuclei in the midbrain of the brainstem.[3]

LOWER RESPIRATORY SYSTEM

Large Airways

The main stem or primary bronchus represents the continuation of the syrinx. Each perforates the septal surface of the lung and continues into the lung parenchyma as the intrapulmonary primary bronchus. That portion of the bronchus is long as it continues to the caudal extremity of the lung (Figure 26.13). Its diameter is variable among species but is widest at its entrance into the lung and then tapers progressively. The epithelium of the primary bronchus is pseudostratified with goblet cells and there are areas with ridges carrying cilia. There are four groups of secondary bronchi that arise from the primary bronchus. Their anatomy will be described with the description of the air sacs and lung parenchyma.[3]

Lung—Pulmonary Air Sacs

The lungs are paired and attached firmly to the ribs and dorsal body wall. They extend from the thoracic inlet at the first rib to the cranial border of the ilium (Figure 26.7). In storks (Ciconiidae) and geese (*Anser* spp., *Branta* spp.), the lungs extend caudally to the hip joint.[3,13] Each lung is often quadrilateral in shape depending on the species, rather than being lobed as in mammals. The lungs appear spongy on radiographs and are best visualized using a lateral view. In this view, the lungs have a honeycomb appearance. The lungs on their dorsolateral margins or costal surfaces are deeply seated against the vertebral ribs. The hilus is where the primary bronchus and the pulmonary artery and veins enter each of the paired lungs. The dorsomedial surface is in contact with the vertebrae and is termed the vertebral surface. Interestingly, while the weight of the lung in relation to total body weight is similar to mammals, its volume per gram of body weight is about 25% smaller.[3]

There are four groups of secondary bronchi that branch off the primary bronchus and they are named according to the region that they supply (Figure 26.14). The mediodorsal and medioventral secondary bronchi connect to form a functional unit. The medioventral secondary bronchi connect with the mediodorsal secondary bronchi in rows of parallel tubes from the primary bronchus. These and their branches form the thick medial portion and the cranial part of the lungs. Some of these bronchi terminate at the cranial pulmonary air sacs and form the basis of the paleopulmonic respiratory system. The cranial pulmonary air sacs are expandable sacs that are connected to the lungs by these bronchial arrangements and include the cervical, clavicular, and cranial thoracic air sacs.

The parabronchi of the lateroventral secondary bronchi connect with the laterodorsal secondary bronchi, and they in turn are connected to the last few mediodorsal secondary bronchi. The laterodorsal and lateroventral secondary bronchi and their terminations form the thin lateral region of the lungs. The lateroventral bronchi are connected to the caudal air sacs of the lungs. These caudal air sacs are the caudal thoracic and abdominal air sacs. This latter arrangement represents the neopulmonic respiratory system. The paleopulmonic and neopulmonic pulmonary air sac systems are best defined physiologically rather than anatomically.[3]

Pulmonary air sacs, in general, are similar histologically to a peritoneum, with squamous to columnar epithelium, a basement membrane, and an underlying connective tissue support. Their interrelationship with the coelomic cavities is discussed in depth in *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 8, Pleura, Pericardium, and Peritoneum: The Coelomic Cavities of Birds and Their Relationship to the Lung–Air Sac System. Only in the area immediately surrounding an opening of a parabronchus does the tissue change to ciliated columnar epithelium. Pulmonary air sacs have limited blood vessels and do not provide for gas exchange.[2]

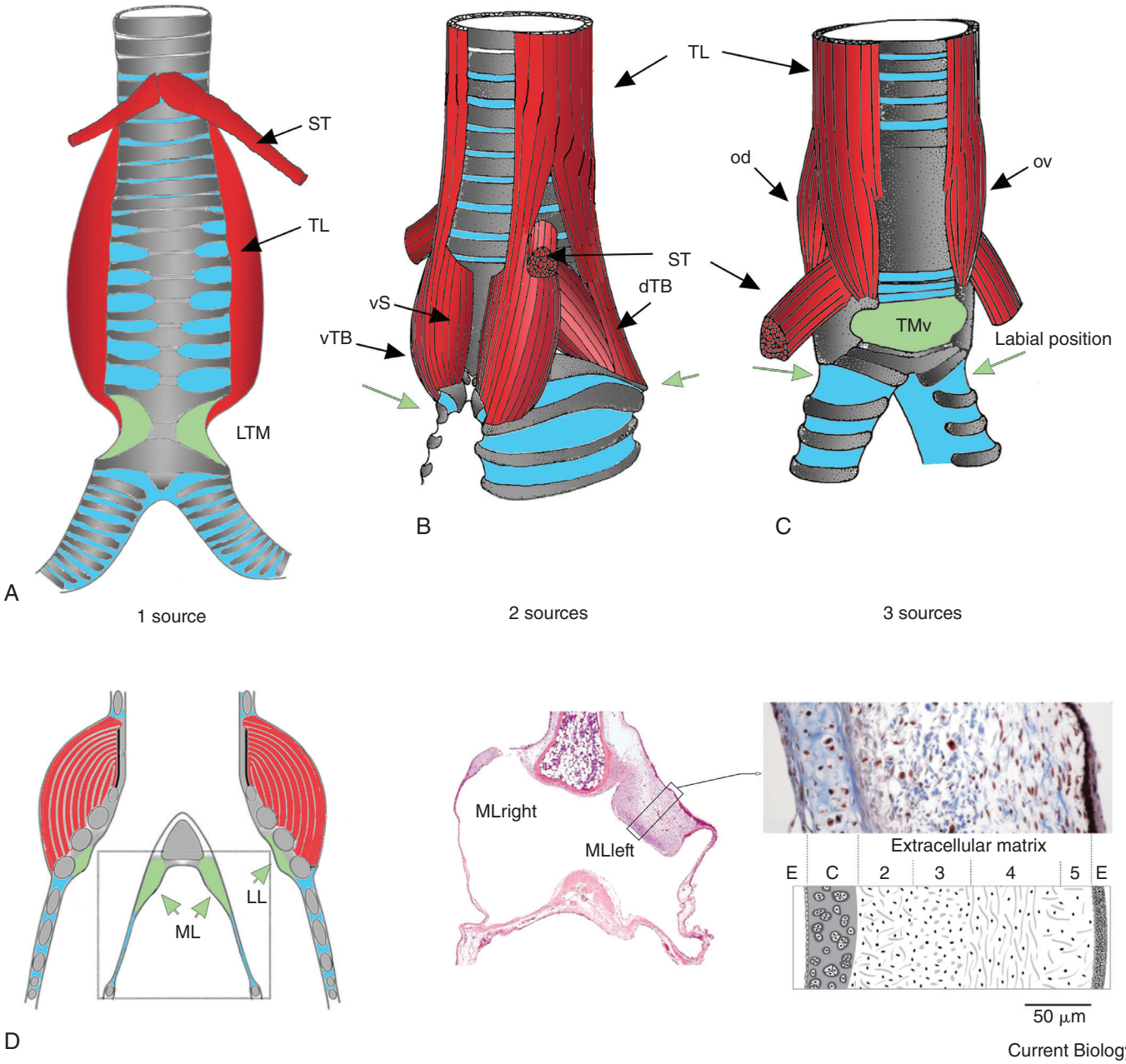


FIGURE 26.11 Schematic illustration showing the overall morphology of the cartilage framework *(grey)* and musculature *(red)* of the syringeal area for a dove (a), oscine (b), and tracheophone suboscine songbird (c). Depending on the location of the sound-generating structures (membranes or labia) along the tracheobronchial juncture, the syrinx contains one, two or, in the case of tracheophone suboscine birds, three sound sources (green membranes and green arrows point at the location of labia in the lumina of the bronchi). *LTM*, lateral tympaniform membrane; *Od, m. obliquus dorsalis; Ov, m. obliquus ventralis; ST, m. sternotrachealis; dTB, m. tracheobronchialis dorsalis; TL, m. tracheolateralis; TMv*, ventral tracheal membrane; *vS, m. syringealis ventralis; vTB, m. tracheobronchialis ventralis*, the dorsal counterpart of which is not visible in this view.The frontal transsectional image (d) illustrates the location of the two pairs of sound generating labia in an oscine songbird (schematically, *LL*, lateral labium; *ML*, medial labium). (Reprinted from: Goller F: The syrinx, *Curr Biol* 32(20):R1095–R1100, 2022. https://doi.org/10.1016/j.cub.2022.08.034.)

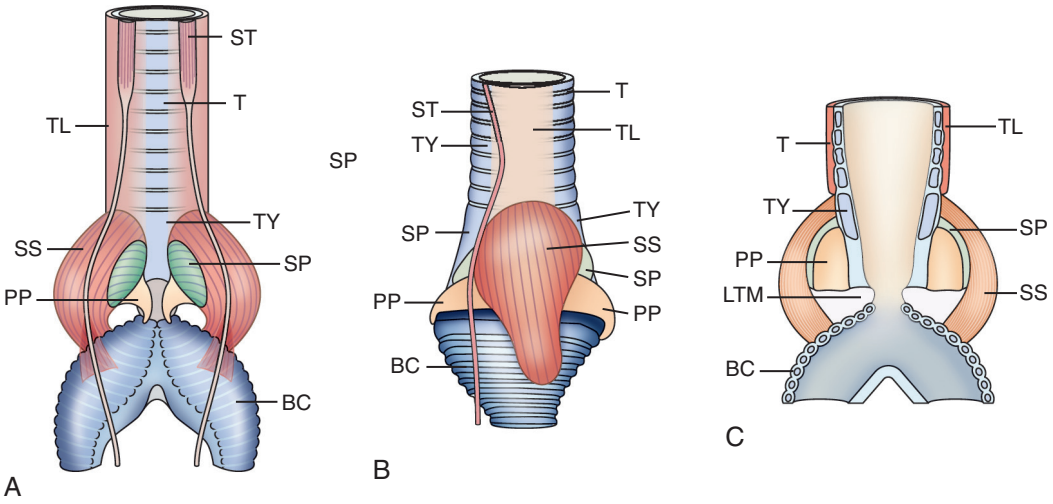


FIGURE 26.12 Diagram of the cockatiel *(Nymphicus hollandicus)* syrinx. (A) External ventral view; (B) external left-side view; (C) horizontal section. *BC*, Bronchial cartilage; *LTM*, lateral tympaniform membrane; *PP*, paired protrusions; *SP*, m. syringealis profundus; *SS*, m. syringealis superficialis; *ST*, m. sternotrachealis; *T*, trachea; *TL*, m. tracheolateralis; *TY*, tympanum (composed of four tracheosyringeal cartilages). (Modified and reprinted from Larsen ON, Goller F: Direct observation of syringeal muscle function in songbirds and a parrot, *J Exp Biol* 205:25–35, 2022 with permission from the Company of Biologists Limited; reprinted from Beaufrère H, Summa N, Le K: Chapter 3: Respiratory system. In Mitchell MA, Tully TN, editors: *Current therapy in exotic pet practice*, ed 1, 2015, St. Louis, Elsevier Inc.)

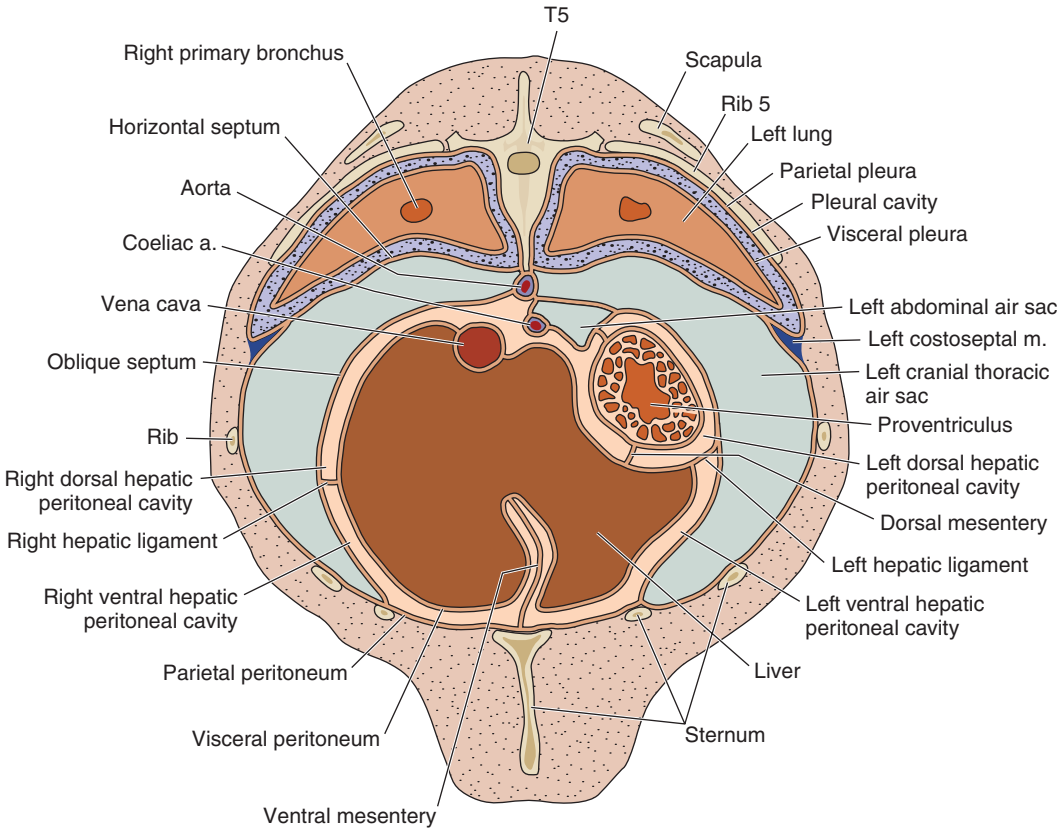


FIGURE 26.13 Transverse section through the fifth thoracic vertebra just caudal to the heart in a young domestic fowl *(Gallus domesticus)*, cranial view. (Redrawn from Canipana [1875]. Reprinted from King AS, McLelland J: Respiratory system. In *Birds: their structure and function*, 2nd ed., Eastbourne, UK, 1984, Ballière Tindall.)

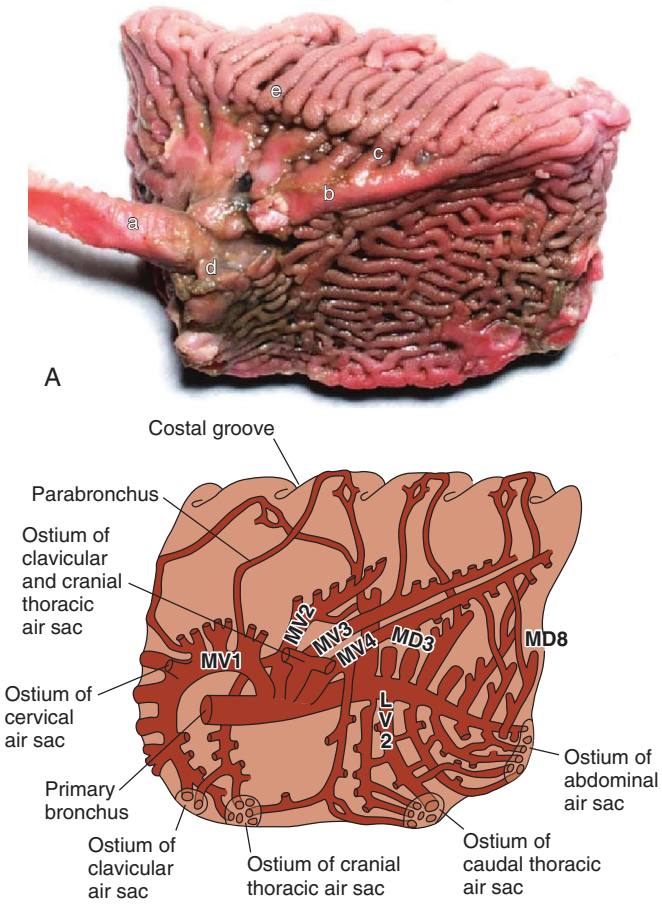


FIGURE 26.14 (A) Ventromedial view of the plastic cast of the left lung of the Japanese quail *(Coturnix japonica)*. *a*, Extrapulmonary part of the primary bronchi, *b*, intrapulmonary part of the primary bronchi, *c*, medioventral secondary bronchi, *d*, mediodorsal secondary bronchi, *e*, parabronchus. (B) Line drawing (modified from King, 1966) showing the arrangement of the airways in the avian lung. The first secondary bronchi to arise from the primary bronchus are the four medioventral secondaries *(MV1–MV4)*. Additionally, eight mediodorsal secondary bronchi (e.g., *MD3* and *MD8*) arise from the dorsal aspect of the caudal half of the primary bronchus, and eight lateroventral secondaries (e.g., *LV2*) arise from its ventral aspect. The air sacs connect to the lung at six sites, known as ostia. Four of these occur on the costoseptal border of the lung; the fifth, for the cervical sac, is in the craniomedial region of the lung. The sixth is somewhat cranial to the center of the septal surface, and consists of two large direct connections, one to the clavicular and one to the cranial thoracic sac. (A, Reprinted from Cevik-Demirkan A, Kürtül I, Haziroglu RM: Gross morphological features of the lung and air sac in the Japanese quail, *J Vet Med Sci* 68(9):909–913, 2006. B, Modified from King AS: Structural and functional aspects of the avian lung and air sacs. In Felts WSL, Harrison RJ, editors: International review of general and experimental zoology, vol 2, New York, London, 1966, Academic Press.)

In the embryo, there are six pairs of air sacs but these are reduced in the adult. Commonly, there are paired cervical air sacs, fused clavicular, paired cranial thoracic, paired caudal thoracic, and paired abdominal air sacs (Figure 26.15). In passerines, the cranial thoracic air sacs fuse to the clavicular air sac. In chickens, the cervical air sacs fuse, and in turkeys *(Meleagris gallopavo)*, the caudal thoracic air sacs are absent while the cervical air sacs fuse to smaller clavicular paired air sacs.[3]

The cervical air sacs often have diverticuli on each side of the vertebral column, one inside the neural canal and one outside, stretching from the transverse foramina to the axis. The clavicular air sac takes origin from four primordial sacs, that is, the paired medial and paired lateral sacs. The medial pair most often fuses to form a single large sac that surrounds the heart, great vessels, and nerves. In addition, the esophagus, trachea, and syrinx are suspended in folds of the clavicular sac or between it and the cervical sac. The extrathoracic diverticula arise from the lateral sacs and extend between the bones and muscles of the thoracic girdle and the shoulder joint. The paired cranial thoracic air sacs are large in chickens and small in parrots. In contrast, the paired caudal thoracic air sacs are small in chickens but large in parrots. Both do not have diverticula.[3] The abdominal air sac is paired in all species examined. There are three paired perirenal diverticula that separate the kidney from the pelvis and the synsacrum, to then invade the vertebrae and the pelvic girdle. There can be femoral diverticula that travel into the medullary cavity of this bone as well as into some of the muscles to the pelvic limb. In general, all of these air sacs are often compressed and form potential spaces.[3]

Birds do not have a diaphragm and therefore rely on changes in pressure between their air sacs relative to the atmospheric pressure to move air through the nonexpandable lungs. The pressure changes within the air sacs are the result of volume changes in the thoracoabdominal or coelomic cavity (Figure 26.16). On inspiration, the ribs move cranioventrally, thereby increasing the thoracoabdominal space. This results in a lowering of the pressure in the air sacs.[3] On expiration, the ribs move caudodorsally, thereby reducing the space in the thoracoabdominal space and increasing the pressure in the air sacs. Expiration is an active process requiring skeletal muscle contraction. For this reason, it is extremely important when holding a bird that the sternum can move freely, as otherwise the bird may suffocate. Alternatively, to ventilate an apneic bird, the sternum can be compressed and then allowed to recoil into its resting position.

Gas exchange occurs in the walls of the parabronchi, the atria, and more importantly in the air capillaries, which are the avian equivalent of alveoli (Figure 26.17). These air capillaries branch and anastomose freely. Their size varies among species but in general they are smaller than the alveoli in mammals. However, unlike in mammals, the size does not vary during the respiratory cycle, which allows a higher number of air capillaries per volume compared to mammals. Additionally, the small diameter of air capillaries suggests that the pressure gradient for the diffusion of oxygen is greater in birds than in mammals.[3] Due to their small size, there is an increase in the number of air capillaries per lung, and hence an increase in the total surface area that is available for gas exchange in birds compared with that in mammalian lungs.

The blood-gas barrier of birds is like that in mammals in that it consists of an endothelial capillary cell, a common basal lamina, and an air capillary epithelium of squamous cells (Figure 26.18). The difference is that the blood-gas barrier is much thinner in birds than in mammals due to the

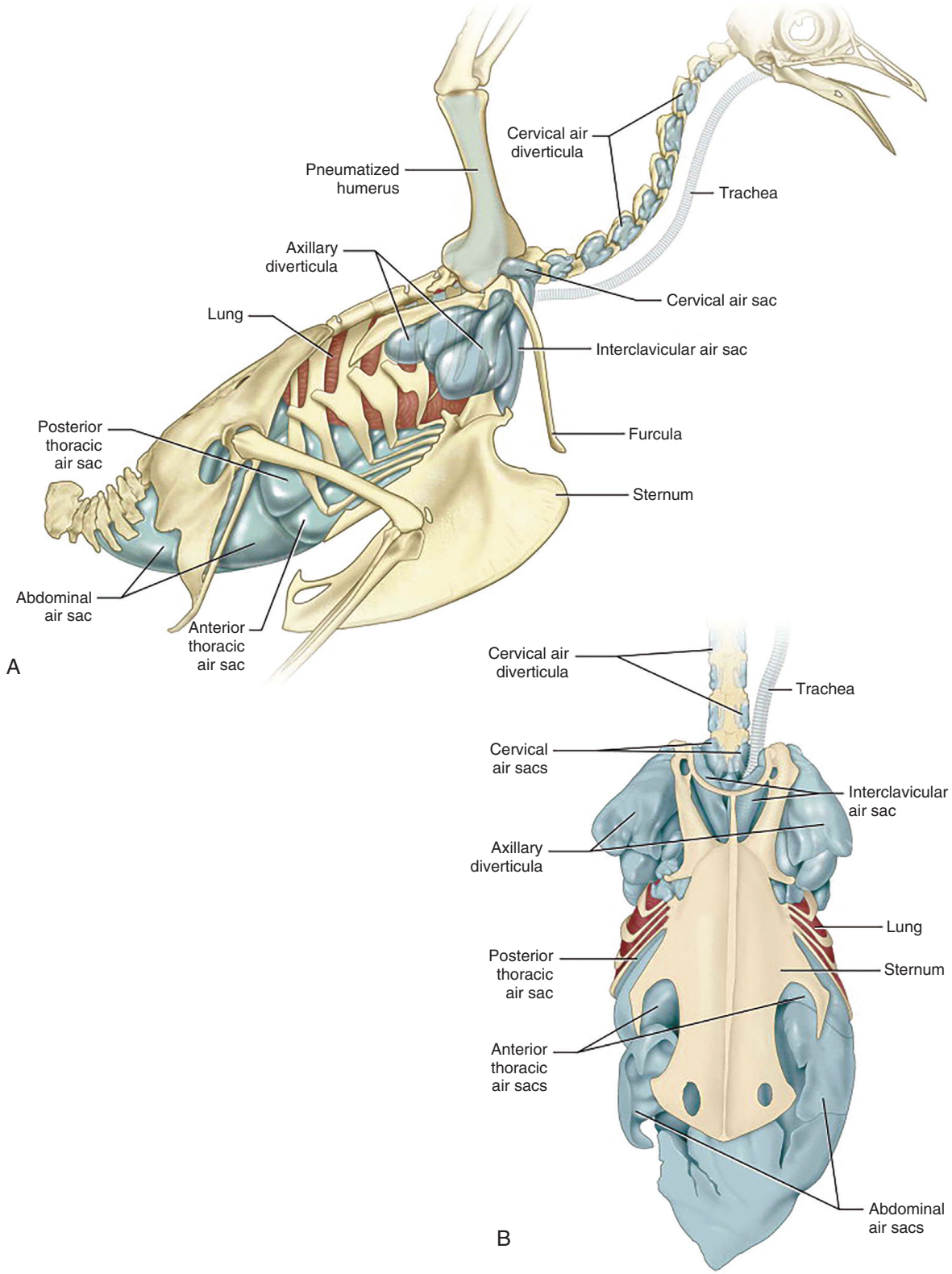


FIGURE 26.15 Diagram of the air sac system of the pigeon *(Columbia livia domestica)* in (A) left lateral and (B) ventral views. There are six primordial pairs of air sacs. The first pair, the cervical air sacs, is fused across the midline. The second and third pairs are fused to each other and across the midline, forming a single median sac, the interclavicular air sac. The cranial thoracic, caudal thoracic, and abdominal air sacs are paired in the pigeon, like in most birds. (Reprinted from De Iuliis G, Pulerà D: Chapter 9: the pigeon. In De Iuliis G, Pulerà D, editors: *The dissection of Vertebrates*, ed 2, 2011, Academic Press, pp. 287–310. https://doi.org/10.1016/B978-0-12-375060-0.00009-7.)

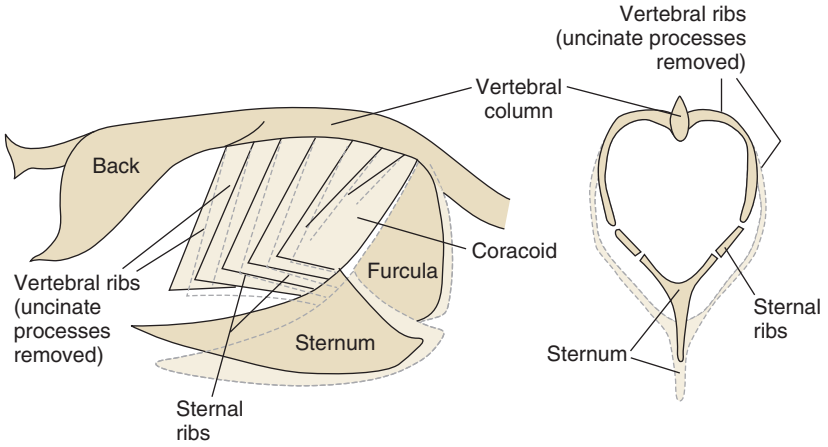


FIGURE 26.16 The movements of the thoracic cage during breathing: (A) cranial view; (B) lateral view. (*Solid lines*, position at the end of expiration; *dotted lines*, position at the end of inspiration.) The inspiratory muscles move the ribs craniolaterally during inspiration, pushing the sternum ventrally and cranially. These inspiratory movements increase the dorsoventral, transverse, and craniocaudal diameters of the thoracic cage. (Reprinted from Powell FL: Chapter 20: respiration. In Scanes CG, Dridi S, editors: *Sturkie's Avian Physiology*, ed 7, 2022, Academic Press, pp 445–484. https://doi.org/10.1016/B978-0-12-819770-7.00033-5.)

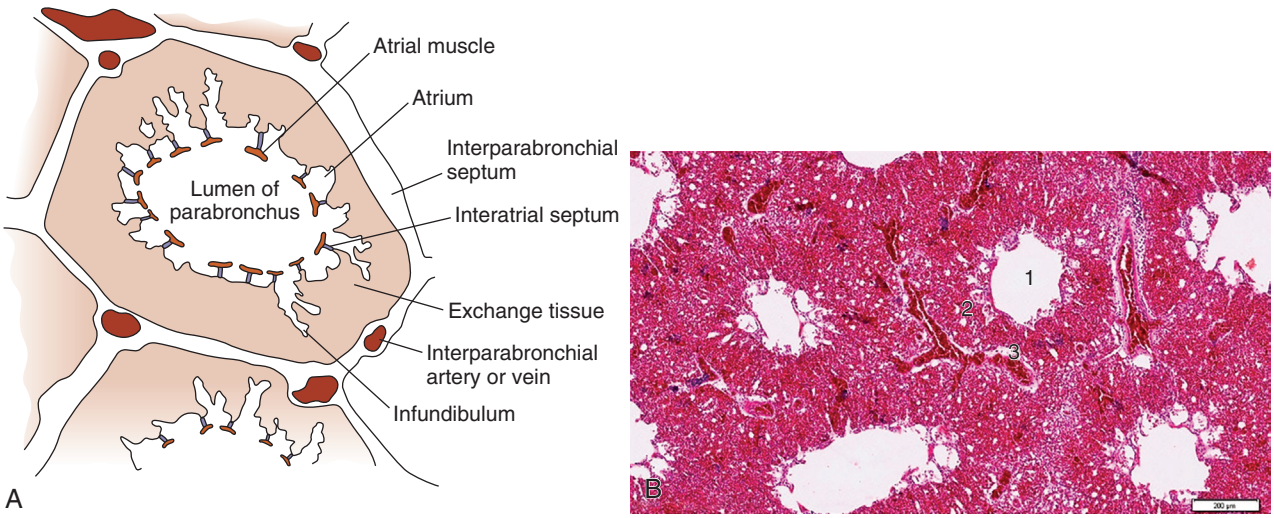


FIGURE 26.17 A drawing (A) and photomicrograph (B) of a histological section showing parabronchi of the domestic fowl *(Gallus domesticus)* in transverse section. The photograph (B) further depicts the tertiary bronchi and parabronchial structures of the domestic fowl. 1 represents the lumen, 2 exchange tissue, and 3 an interparabronchial artery or vein. (Figure A adapted from King AS, McLelland J: Respiratory system. In Birds: their structure and function, 2nd ed, Eastbourne, UK, 1984, Baillière Tindall; Figure B (reprinted from https://histology-of-birds.com/galleries.php?id=20&v=3. Courtesy Ghent University, Belgium.)

reduced thickness of the air capillary. The resistance of the barrier to oxygen diffusion is less with this reduced thickness. There are cells in the lining of the air capillary that have osmophilic bodies that produce surfactant. It is thought that the surfactant produced in birds restricts the diffusion of fluid from the blood capillary across the blood-air barrier.[3] This would promote normal function for oxygen and carbon dioxide exchange. However, due to this reduced barrier thickness, when there is increased pressure on the blood capillary side, for example, due to high blood pressure in the lungs (right-sided heart failure) and/or changes in the lungs from chronic or acute infections, there is increased leakage with faster crashing of the system compared to mammals.

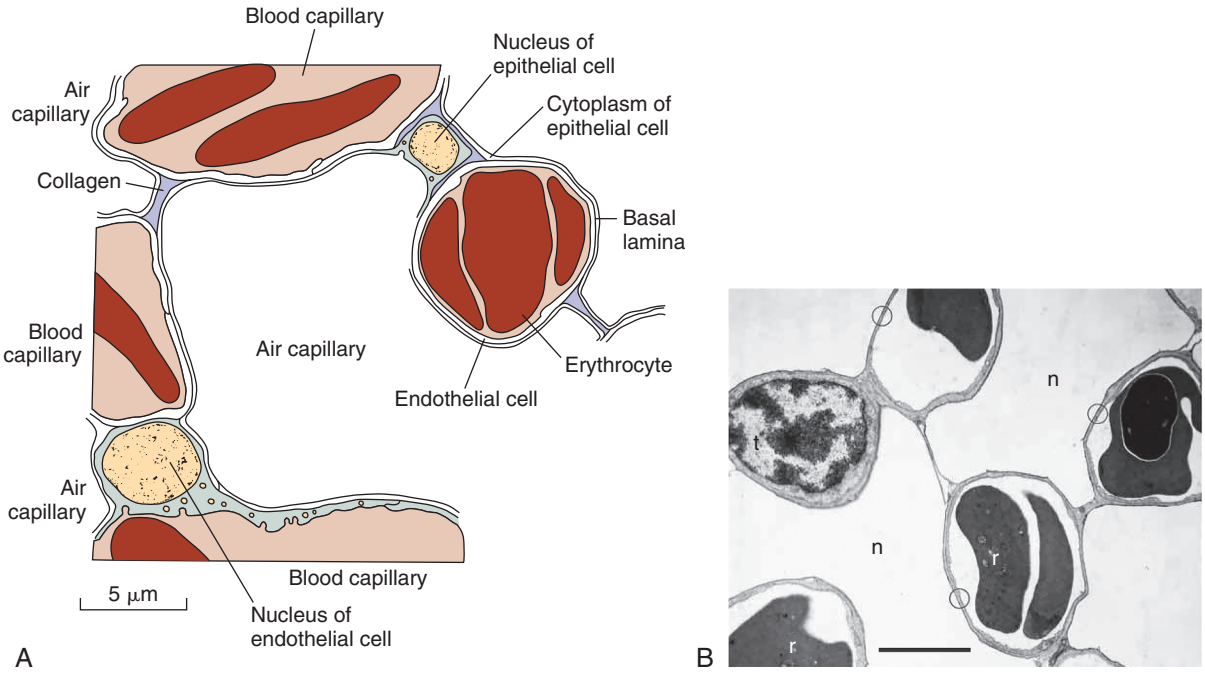


FIGURE 26.18 A drawing (A) of a photomicrograph of the exchange tissue of the lung of the domestic fowl and a transmission electron microscopy (TEM) photomicrograph (B) showing the structure of the lung of a Japanese quail (Coturnix japonica). Air capillaries (n) are alternately situated between erythocyte-containing capillaries (r) with a very thin blood-gas barrier in between. Magnification 3500×. (Reprinted from Rombout PJA, Dormans JAMA, van Bree L, Marra M. Structural and biochemical effects in lungs of Japanese quail following a 1-week exposure to ozone, *Environ Res* 54(1):39–51, 1991. https://doi.org/10.1016/S0013-9351(05)80193-8.)

The epithelial cells lining the lungs act as fixed macrophages and transfer engulfed material to interstitial macrophages. Air that enters the caudal air sacs is filtered less than that of the cranial air sacs, making the caudal air sacs more susceptible to disease. This is due to the anatomic arrangement of origin of the lateroventral secondary bronchi, which arise from the ventral wall of the primary bronchus.[3] If infectious or other particles are able to travel down the primary bronchus, as they reach its terminal end, these particles are more likely to fall ventrally into lateroventral secondary bronchi. The secondary bronchi from the paleopulmonic system, on the other hand, arise dorsally from the primary bronchus making them less susceptible. This can be observed radiographically as opacities in the most caudal (i.e., caudal thoracic and abdominal) pulmonary air sacs.

Because of the unidirectional continuous flow of air of the paleopulmonic system through the series of parabronchial tubes, birds are more efficient than mammals in capturing oxygen (O_2) and removing carbon dioxide (CO_2). The nonoxygenated blood enters at an approximately 90° angle from the parabronchus. This cross-current gas exchange enhances the ability to oxygenate the blood. There is abundant smooth muscle in the walls of the interparabronchial arteries, as well as smooth muscle in the walls of the parabronchi and the atria that allows altering of the air flow into this system by regulating their diameters.[3] These muscles are innervated by nerves of both the sympathetic and parasympathetic nervous systems and would most likely respond to bronchodilators (see Chapter 28).

Respiratory Cycle

In birds, respiration does not rely on changes in the volume of the thorax that create negative pressures as in mammalian lungs. Instead, pressure changes in the air sacs move the air through a nonexpanding lung. These pressure changes result from volume changes in the thoracic and abdominal space, and require muscular exertion during both inspiration and expiration.[3,14]

On inspiration, the ribs and sternum are drawn cranially and the more caudal portion of the thoracoabdominal wall is drawn ventrally.[3,14] This action can be compared to that of a bellows. The junction of the coracoid and sternum corresponds to the fixed end of the bellows, whereas the distal end of the sternum corresponds to the more movable end. When the thoracoabdominal space is expanded, the air rushes into primarily the caudal sacs (caudal thoracic and abdominal) as a result of the reduced pressure and inspiratory aerodynamic valving (Figure 26.19). On expiration, the ribs and sternum move caudodorsally, closing the "bellows" and reducing the thoracoabdominal space. Consequently, the pressure in the caudal air sacs is increased, moving the air by expiratory aerodynamic valving into the paleopulmonic system. In those birds with both neopulmonic and paleopulmonic systems, air enters and exits the respiratory system in a two-breath cycle.[14] This two-breath cycle allows a continuous flow of air through the parabronchi to the gas exchange surfaces (Box 26.1), whereas the less efficient mammalian breathing cycle allows gas exchange to occur only at the end of inspiration.

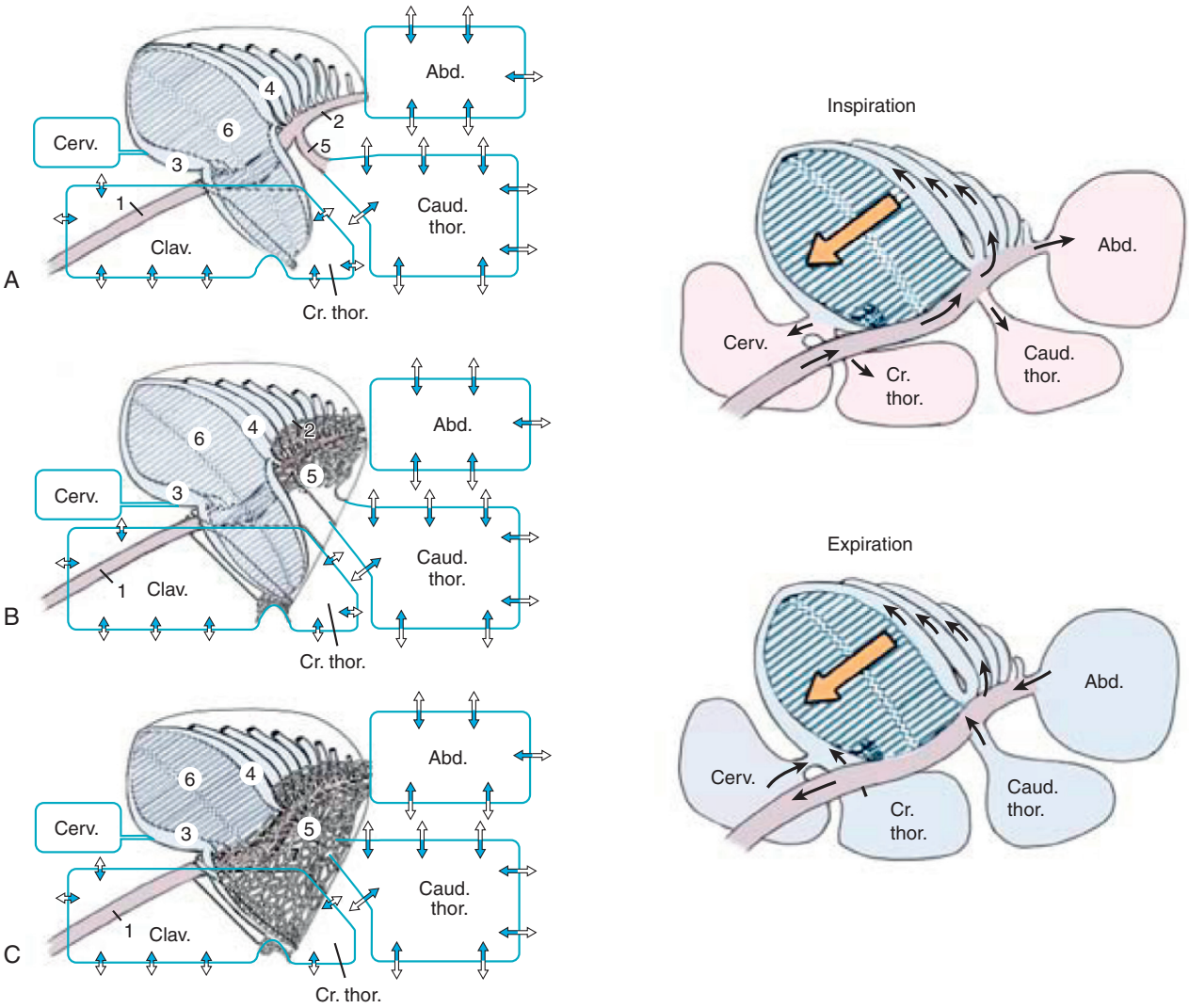


FIGURE 26.19 (A) and (B) *Left panels*, Successive stages in the division of the lung into a paleopulmo and neopulmo. (A) Lungs composed only of the paleopulmo such as observed in Sphenisciformes; (B) moderate development of the neopulmo forming the caudal part of the lungs such as observed in Anseriformes; (C) highly developed neopulmo occupying the entire lateroventral aspect of the lungs, such as occurring in Passeriformes. *1*, Trachea; *2*, primary bronchus; *3*, medioventral secondary bronchi; *4*, mediodorsal secondary bronchi; *5*, lateroventral secondary bronchi; *6*, paleopulmo; *7*, neopulmo. The length of the *white arrows* in A, B, and C indicates the estimated dilatation of the air sacs in inspiration; the length of the *shaded blue arrows* indicates the estimated compression in expiration. *Right panels*, The orange arrows indicate the flow of air across the gas exchange tissues of the lungs, in inspiration and in expiration. The *black arrows* indicate the flow of air within the trachea and to and from air sacs, in inspiration and expiration. *Abd*, Abdominal air sac; *Caud thor*, Caudal thoracic; *Cerv*, Cervical air sac; *Clav*, Clavicular air sac; *Cr. Thor*, Cranial thoracic. (Modified and reprinted with permission from McLelland J: Anatomy of the lungs and air sacs. In King A, McLelland J, editors: *Form and function in birds*, Vol 3, London, UK, 1985, Academic Press, pp 221–280; reprinted from Beaufrère H, Summa N, Le K: Chapter 3: respiratory system. In Mitchell MA, Tully TN, editors: *Current therapy in exotic pet practice*, ed 1, 2015, St. Louis, Elsevier.)

Defense Mechanisms of the Lower Respiratory Tract

One of the first lines of defense of the lower respiratory tract is the mucociliary escalator.[14] This anatomic and physiologic escalator uses mucus secreted from the goblet cells to trap foreign material, which is then swept away by cilia for removal from the respiratory tract. This defense mechanism can be overwhelmed by infection. When this happens, the mucociliary escalator cannot move infectious material and products of inflammation out of the lung quickly enough to keep the parabronchi clear.[14] Nebulization reduces the increased viscosity of the mucus associated with infection and

BOX 26.1 RESPIRATORY CYCLE IN BIRDS

First inspiration—Air flows into the trachea through the primary bronchi, and most of the air goes directly into the abdominal air sac and/or into the caudal thoracic air sac via the lateroventral secondary bronchi.

First expiration—Air travels back through the paleopulmonic parabronchi and is then shunted via aerodynamic valving into the mediodorsal secondary bronchi; the air moves through the parabronchi of this system and gas exchange occurs.

Second inspiration—Air is pulled from the mediodorsal secondary bronchi through the medioventral secondary bronchi and into the cranial pulmonary air sacs.

Second expiration—Air moves out of the cranial air sacs, into the opening of the medioventral secondary bronchi, and out of the trachea.

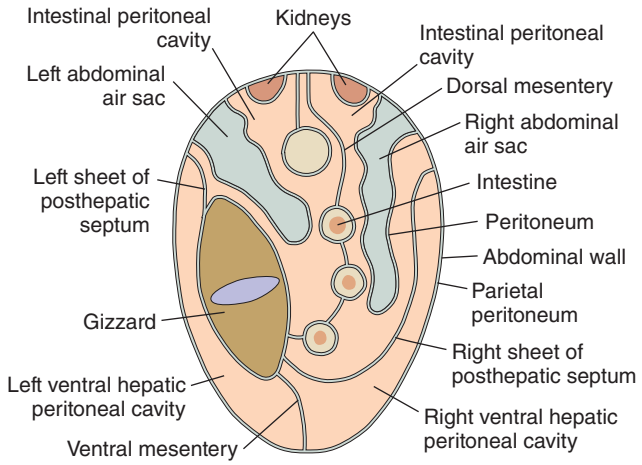


FIGURE 26.20 Diagrammatic transverse section through the peritoneal cavities of the bird, caudal view. The diagram shows three of the five peritoneal cavities, that is, the intestinal peritoneal cavity and the left and right ventral hepatic peritoneal cavities. The left and right dorsal hepatic peritoneal cavities, which are much smaller than the other three, are not shown. Note that the left and right ventral hepatic peritoneal cavities enclose the intestinal peritoneal cavity so that an incision anywhere in the abdominal wall at the level of this transverse section can only enter either the left or the right ventral hepatic peritoneal cavity; at a more caudal level of the body, immediately cranioventral to the vent, a ventral incision will enter the intestinal peritoneal cavity. (From McLelland J: Pericardium, pleura et peritoneum. In Baumel JJ, King AS, Lucas AM, Breazile JE, Evans RE, editors: Nomina anatomica avium, London and New York, 1979, Academic Press; with kind permission of the publisher. Reprinted from King AS, McLelland J: Coelomic cavities. In: *Birds: their structure and function*, 2nd ed., Eastbourne, UK, 1984, Baillière Tindall.)

helps the escalator work more efficiently.[14] In birds with vitamin A deficiency, cells undergo a change to a squamous type without mucus or cilia. This results in a breakdown of this fundamental system to remove foreign material.

A second line of defense involves the epithelial cells themselves. These cells of the lung parenchyma act as fixed macrophages,[14] engulfing foreign material and transferring it to interstitial macrophages for destruction. Because macrophages are involved early in defense, granulomatous reactions to infection are common in birds.

When defense mechanisms of the lower respiratory tract are overwhelmed, birds often exhibit a number of signs, including voice change (particularly when the syrinx is involved), increased abdominal/coelomic exertion (when the parabronchi are plugged with infection), tail bobbing (associated with increased respiratory effort), and clicks and wheezes (caused by air moving through a lumen that is reduced in size).[6] If lower respiratory tract infections obstruct the trachea, the abdominal or caudal thoracic air sac can be cannulated for oxygenation of the respiratory system (see Chapters 29 and 34). Oxygen can be administered into the clavicular air sac as well.

Extrapulmonary Factors

Because birds have air sacs that are connected to the lungs, extrapulmonary anatomic and physiologic factors can affect their ability to breathe normally. By understanding the anatomy of surrounding structures in relation to the respiratory tract, the clinician can make educated decisions regarding their patient. For example, structures in the neck that impinge on the trachea can cause difficulty breathing for the avian patient. An understanding of regional anatomy in the head, neck, and body cavity is also of particular importance when approaching the respiratory tract.

One important difference that must be understood clinically is the anatomy of the coelomic cavities of mammals compared to birds. The coelomic cavities of birds are similar between species; in general, there are eight coelomic cavities in the adult bird (*Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 8, Pleura, Pericardium, and Peritoneum: The Coelomic Cavities of Birds and Their Relationship To The Lung–Air Sac System). The mammalian coelomic cavities include the right and left pleural cavities, the intestinal peritoneal cavity, and the pericardial cavity. The four additional coelomic cavities of birds include the left and right ventral hepatic peritoneal cavities and the left and right dorsal hepatic cavities (Figure 26.13). The kidneys, adrenals, gonads and their ducts are found within the intestinal peritoneal cavity (Figure 26.20).

This anatomic arrangement is important to gain understanding of how and where an infection or neoplasia can spread or affect the function of other organ systems, in particular the function of the air sacs. For example, enlargement of the liver will reduce the size of the caudal thoracic and the abdominal air sacs, making it more difficult to move air through the parabronchi for oxygen exchange. Similarly, tumors or infections from the ovary will often seed into the intestinal peritoneal cavity; while being retained within it, this cavity will expand, thus reducing the ability of the abdominal air sac to expand and leading to respiratory distress (see also Chapter 28).

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CHAPTER 27

Diagnostic Workup of Respiratory Problems

Nico J. Schoemaker • Yvonne R. A. van Zeeland

When evaluating the respiratory system of a patient, several steps need to be taken. First, an extended history should be collected focusing on issues that may affect the respiratory system. Subsequently, the patient should be evaluated and inspected from a distance with a special focus and emphasis on respiratory sounds and movements. Finally, a hands-on physical examination should be performed during which the separate parts of the respiratory system are examined, starting with the nares, and finishing with the coelom.

HISTORY

When taking the history of a patient with respiratory problems, questions need to be asked on the time of onset, duration, and clinical course of the disease, as well as any potential treatments that may have been initiated prior to the visit (including their effects). Additionally, information is warranted on presence of nasal discharge, respiratory sounds, and labored respiration (dyspnea). Other pertinent questions relate to the bird's living environment (including diet) as certain factors can predispose to respiratory issues. Occurrence of similar problems in other birds within the household can point toward infectious, toxic, and/or managemental causes. Gender and reproductive status are important to note as egg binding and yolk peritonitis are frequent causes of respiratory distress in hens.

In relation to *nasal discharge*, important features to evaluate include the type of discharge (i.e., watery/serous, mucus, exudate/purulent, blood, etc.), sidedness (i.e., unilateral or bilateral), pattern (i.e. continuous, intermittent, recurrent) and circumstances under which the discharge is produced (e.g., following exposure to certain fumes, smoke or aerosoles). Clear, serous discharge is often seen in the acute stages of chlamydiosis or viral infections, in case of allergies, or upon inhalation of dust or irritating agents. Chronic problems are usually associated with discharge that is more mucoid in nature. Once (secondary) bacterial infections are involved, the exudate often becomes purulent. Unilateral discharge often pinpoints a localized problem (e.g., aspergilloma or foreign body), while viral, fungal or bacterial infections more commonly lead to bilateral nasal discharge.

Respiratory sounds provide information on the location of the problem in the upper airways, as characteristic sounds may be heard if exudate or other material is present in the airways. A nasal stridor produces a typical "sniffing sound" and disappears as soon as the bird starts breathing with an open beak. As such it is important to inquire whether open beak breathing has been observed and whether the sound changes or disappears upon opening of the beak. Additionally, sneezing can be observed in birds with rhinitis or sinusitis. It is important to inquire about the frequency, duration, and moments at which this occurs. If a tracheal, syringeal, or laryngeal obstruction is present, a characteristic "sawing", "air racing", or "g"-sound can often be heard. Additional sounds that may be heard in birds with respiratory problems include the characteristic clicking sounds in case of an infectious tracheitis (e.g., tracheal mites, *Syngamus trachea*) or high-pitched squeaks as may be heard in budgerigars *(Melopsittacus undulatus)* in case of goiter. In addition to the presence of respiratory sounds, it is important to take note of changes (e.g., hoarseness) or loss of voice, as this is commonly heard in parrots with tracheal or syringeal obstructions.

Dyspnea (labored breathing) is characterized by forced respiratory movements, whereby auxiliary respiratory muscles are activated. The most frequently seen sign of labored breathing in birds is "tail bobbing" during which the tail is flexed ventrally in an effort to expel air out of the air sacs into the lungs. As such, tail bobbing can be considered as the avian counterpart of abdominal breathing in mammals. Other signs pointing toward labored breathing include inflation and deflation of the sinuses and cervicocephalic diverticulum, and open-beak breathing.

During the history taking it is vital to determine whether the dyspnea is acute or chronic. If the dyspnea is acute, inquiries should be made to inquire about events immediately preceding the dyspnea as this may reveal an underlying cause for the dyspnea (e.g., polytetrafluoroethylene [PTFE] toxicosis following cooking with recently purchased Teflon-coated pans; foreign body aspiration in a cockatiel *[Nymphicus hollandicus]* associated with consumption of millet seed; hypersensitivity/allergic pneumonitis in a macaw [*Ara* spp.] developing following exposure to the feather dust and dander of a grey parrot *[Psittacus erithacus]* or cockatoo [Cacatuidae]). In the early stages of dyspnea, dyspnea will mostly be observed following exercise or stress (while taking longer to recover), whereas it will often be present continuously in more advanced stages of disease.

Retrieving information on the *living environment* of the patient is essential to provide clues regarding potential causes and predisposing factors to respiratory disease. Details that are pertinent to collect include information on (recent) exposure or (in) direct contact with other birds (e.g., wild birds, new additions to

the household), outdoor versus indoor housing (including exact location), exposure to potentially irritating or toxic (aerosolized) substances (e.g., incense, hair spray, perfume, smoke, dust, fumes, etc.; see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 18, Advancements in diagnosis and management of toxicologic problems), and the climate conditions under which the bird is kept. Especially low environmental humidity can predispose to dry mucous membranes and associated irritation of the respiratory tract. Inquiries should also be made regarding the type of diet that is fed (including any supplements provided and consumed amounts), as vitamin A deficiency results in metaplastic changes of the mucous membranes along the respiratory tract, hence predisposing to infection (see also Chapter 28). When other birds are kept in the household, it is important to note whether these are also suffering from disease. Similarly, if diseases with zoonotic potential are expected, inquiries can be made in relation to illness of (other) members of the household as this may help narrow down the list of differentials and direct the focus of the further diagnostic work-up towards specific pathogens (e.g. *Chlamydia* spp., see Chapter 18).

OBSERVATIONAL ASSESSMENT FROM A DISTANCE

Prior to handling the bird, an observational assessment is made from a distance. Ample time should be taken to inspect the patient, starting when the client enters the examination room with the patient. In a new environment or when stressed, birds will often try to hide signs of disease but are commonly unable to maintain this during the examination. Hence, the bird should be allowed to acclimatize to the examination room prior to commencing the hands-on examination while preventing exposure to extraneous stimuli that can lead to stress (e.g., sudden or unfamiliar noises; see also Chapter 46).

While observing the bird from a distance, the rate, rhythm, and depth of respiration should be assessed. In a normal, calm bird, respirations should hardly be noticeable. With space-occupying masses in the coelom, the respiration will often be rapid and shallow, while obstructions in the upper respiratory tract (particularly the trachea/syrinx) will generally lead to a deeper respiration, with relatively limited increase in respiration rate. Some avian species (e.g., Pionus parrots, [*Pionus* spp.], Amazon parrots [*Amazona* spp.]) may pant when stressed. This is a normal physiologic response that should not be misinterpreted as dyspnea due to a pathological process.

Attention should be paid to potential respiratory sounds, change of voice (in psittacines), and presence of accessory respiratory movements (i.e., open-beak breathing, tail bobbing, inflation of the sinuses during the respiratory cycle, vent lip motions coindicing with the respiratory cycle) indicative of the severity of the dyspnea, as discussed earlier. Birds with dyspnea often show a characteristic tail bob with every breath, which may be accompanied by open-beak breathing in case of more severe respiratory problems. Respiratory problems associated with excess fluid production may cause gurgling sounds that are audible during inspiration and expiration. Be aware that some parrots produce noises that may be mistaken for abnormal sounds by the inexperienced owner or veterinary surgeon unfamiliar with the species. For example, grey parrots may show a characteristic growling, whereas cockatoos and Pionus parrots will hiss or produce a sniffling sound when anxious or stressed, respectively. In parrots, in particular, it is important to realize that these species are capable of mimicking human sounds (e.g., coughing, sneezing), which an owner may inadvertently interpret as respiratory disease.

The head and neck should be carefully inspected for any obvious abnormalities that can be present such as ocular or nasal discharge; occlusion; lesions or deformities of the nares, beak, and/or cere; periorbital swelling (in case of sinusitis); hyperinflation of the cervicocephalic diverticulum; and/or subcutaneous emphysema. Finally, the posture and movements of the bird are worthwhile to assess. For example, a wide-legged stance or a hunched stance with a ventrally deflected, kinked tail may be indicative of a space-occupying mass in the coelom (e.g., in budgerigars with gonadal or renal neoplasia), whereas swaying movements or stretching of the neck versus yawning or excessive rubbing or scratching of the head can indicate problems in the trachea versus nasal cavity and sinuses, respectively. In birds with respiratory distress or those suffering from hyperthermia, the wings may also be drooped and/or held away from the body.

HANDS-ON PHYSICAL EXAMINATION

If the bird is well enough to withstand the stress of being handled, a physical examination should be performed. Prior to handling, the bird can temporarily be placed in an incubator or oxygen cage to preoxygenate the bird. Additionally, the use of sedatives (e.g., midazolam) should be considered as this may help to reduce stress, hence reducing the risks of handling (see Chapter 46). Care should be taken to minimize handling times while carefully monitoring the bird throughout and (temporarily) interrupting the physical examination to allow the bird to recover in the oxygen cage, if necessary.

During the physical examination, all components of respiration should be evaluated. An initial assessment of the respiration is done from a distance and should be performed prior to handling of the bird (see Observational Assessment from a Distance). This will help to ensure that baseline values are obtained by eliminating effects from human interference as the stress from handling and restraint (especially in birds that are not used to handling) will inevitably result in significant alterations of the bird's respiration. Nevertheless, the bird's respiration should also be evaluated during and following handling, as changes in the respiration, including the time to return to baseline, will provide additional clues on the severity of the problem.

Evaluation of the respiration rate may be challenging as reference ranges are not routinely available for all species, and respiration rates may be significantly altered in a novel, unfamiliar environment. For some species, frequencies are reported (i.e., 3–5 breaths per minute [BPM] in the ostrich *[Struthio camelus]*; 13 BPM in turkeys *[Meleagris gallopavo]*; 25–30 BPM in pigeons *[Columba livia domestica]*, and 40–50 BPM in cockatiels).[1] Studies that have been performed over half a century ago came up with a formula that would allow calculation of the normal respiration rate in rest based on the weight of the bird. Within this formula [$f = 17.2\ (kg^{-0.31})$][2,3] f represents the respiration rate. When using this formula for the species listed earlier, the frequencies are pretty much in accordance with the reported frequencies (i.e., cockatiels [100 g] = 35 BPM, pigeons [500 g] = 21 BPM, and ostrich [70 kg] = 4 BPM).

Although the aforementioned formula theoretically allows normal frequencies to be calculated for all species, some

authors report that recording respiration rates has limited value as it can differ so widely.[4] However, this is not only true for birds but also for mammals. As respiration rate by itself rarely allows for accurate assessment of compromised respiratory function, rhythm, depth, and type of respiration should also be assessed. These parameters are less likely to change significantly from baseline values in birds without respiratory distress. The rhythm should always be regular; irregular or agonal breathing often indicates a life-threatening emergency and ensuing death. In birds, depth of respiration is often difficult to assess, unless it has increased significantly. Deep respirations are characterized by an increased excursion of the sternum and tail bobbing. Some authors hypothesize that increased excursion of the sternum could be considered the equivalent of "costal breathing" in mammals, while tail bobbing would be the equivalent of "abdominal breathing" in mammals.

Respiration may be uneventful at the time of presentation in the clinic, but once the bird is handled, respiratory parameters may change drastically. The bird may become so dyspneic during handling that it should immediately be released and returned in its cage. After releasing the bird, it is important to evaluate how much time it needed for the respiration to return to baseline values prior to handling. The longer it takes for these values to normalize, the more clinically significant the changes to the respiratory system are.

Once the respiration is evaluated at rest, the bird is handled to allow for a closer inspection of relevant body parts. Important parameters to assess include the bird's overall body condition (by assessing pectoral musculature and fat deposits), body weight, and hydration status (e.g. by testing skin tenting of the lower eyelid), as these provide general insight into the bird's overall health status and chronicity of the problem. Nest, the head of the bird is closely inspected. Particular attention is paid to the dorsal and cranial aspects of the head and the periorbital region, which are all carefully inspected for presence of subcutaneous masses or swellings, and rhythmic inflation of the sinuses (concurrent with each respiration). The nares and cere should be inspected for changes in size, shape, and symmetry; presence of lesions or other deformities; presence of nasal discharge, which may be visible as fluid or dried exudate in or around the nare(s), or result in staining of the surrounding feathers. Gentle pressure can be applied to the periorbital region and ceres to check whether exudate can be expelled from the nares or lacrimal ducts. The oropharynx should be inspected from up close, whereby special attention should be paid to the choanal slit and glottis. The choanal slit is evaluated for blunting of the choanal papilla (as can be observed with vitamin A deficiency, infections, and other diseases) and presence of swellings, exudate, plaques, or other lesions. The laryngeal opening or glottis is also evaluated for presence of swellings, lesions, or discharge that may block the entrance, as can be seen, for example, in case of infectious laryngotracheitis (ILT) in chickens *(Gallus gallus domesticus)* and other Galliformes. Dependent on the temperament and species, a paperclip, mouth gag, or speculum may need to be used to enhance visualization of these structures. Alternatively, tape or gauze loops can be used to open the beaks and facilitate examination. The mucous membranes should be assessed for color to determine if pallor (associated with anemia) or cyanosis (indicative of severe hypoxia) are present. In chickens and other Galliformes, anemia and cyanosis are often noticeable upon inspection of the combs or wattles. Severe forms of anemia can also lead to pale discoloration of the iris.

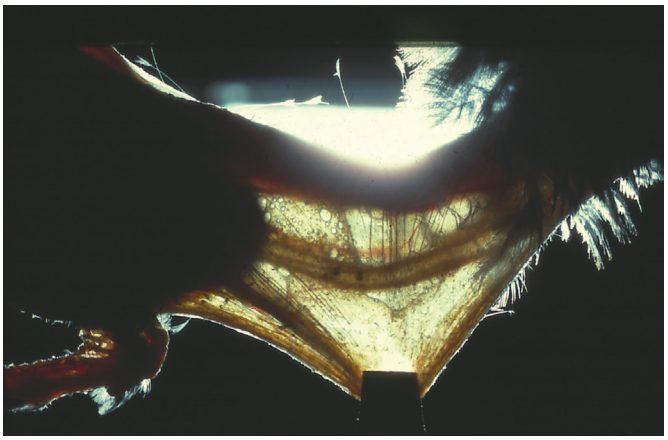


FIGURE 27.1 In small birds, transillumination using a bright light source may be useful to detect foreign bodies or tracheal mites, as can be seen in this Gouldian finch *(Chloebia gouldiae)*. (Photo courtesy Gerry Dorrestein.)

The physical examination continues with an evaluation of the neck and trachea. The neck should be closely inspected and palpated for abnormalities, such as hyperinflation of the cervicocephalic diverticulum. Palpation of the trachea may reveal anatomic alterations indicative of trauma, which are commonly accompanied by subcutaneous emphysema (see also Chapter 34). In smaller passerines and psittacines, transillumination of the trachea can be helpful to identify intraluminal abnormalities, such as tracheal mites (Figure 27.1) or foreign bodies (e.g., millet seed that got stuck in the trachea of a cockatiel

The lungs and air sacs can be auscultated over the dorsum and flanks, respectively. Ideally, a good-quality pediatric or infant stethoscope should be used. Although the lungs in birds do not change in size during respiration, auscultation may still provide valuable information as crackles may be heard in case of lung alterations and a lack of sounds may indicate a large lesion in the lung. Friction or wheezes may be heard in case of an airsacculitis. Heart auscultation, which is performed over the sternum, is warranted in patients suspected of lung edema or ascites due to cardiovascular disease. (see Current Therapy in Avian Medicine and Surgery, Vol 1, Chapter 6, Cardiology).

The respiratory examination is concluded with a palpation of the coelomic region. The distance from the sternum to the pubic bones and interpubic distance are evaluated to determine whether there is coelomic distension leading to compression of the air sacs. Ascites, eggs, or masses can often be discerned following careful exterior palpation. In larger species (e.g., macaws, chickens), a cloacal palpation can also be performed, while in smaller birds, detailed inspection of the internal organs may be possible following wetting of the feathers (e.g., using alcohol) and subsequent transillumination of the region using a bright light source.

While other aspects of the physical examination may bear relevance to the underlying condition and management of the patient (e.g. assessment of fractures in case of traumatic events), their further evaluation will depend greatly on the patient's condition and how well it coped with the examination (see also Chapter 34).

BOX 27.1 CLINICAL SIGNS ASSOCIATED WITH INVOLVEMENT OF THE SPECIFIC PARTS OF THE RESPIRATORY TRACT

Part of the Upper Respiratory Tract Involved in the Disease Process	Clinical Signs Associated With Involvement of the Specific Parts of the Respiratory Tract
Nares and nasal cavity	◆ Sneezing ◆ Nasal discharge ◆ Staining or dried discharge in the feathers surrounding the nares ◆ Nasal plugs, occlusion of the nares ◆ Abnormally shaped nares ◆ Swollen cere ◆ Scratching or rubbing of the beak (with or without concurrent feather loss) ◆ Head shaking ◆ Yawning ◆ Discharge or mucus in the choanal slit ◆ Open-beak breathing ◆ Epiphora, conjunctivitis ◆ Longitudinal groove(s) in the beak (chronic rhinitis) ◆ Often seen in combination with sinusitis
Sinuses	◆ Periorbital swelling (may be soft, firm, or fluctuating) ◆ Protrusion of the eye (exophthalmos) ◆ Sunken eyes (chronic sinusitis, particularly in macaws) ◆ Scratching or rubbing of the head ◆ Periorbital feather loss ◆ Often seen in combination with rhinitis
Trachea and/or syrinx	◆ Loss of vocalization ◆ Change of pitch or voice ◆ Abnormal breathing sounds (stridor), particularly on inspiration ◆ Head shaking ◆ Coughing ◆ Gurgling ◆ Dyspnea, breathing with an open beak and tail bobbing ◆ Breathing with an extended neck
Lungs and/or air sacs	◆ Severely altered respiration pattern or frequency, e.g., increased sternal movements ◆ Accessory respiratory movements, most notably tail bobbing ◆ Exercise intolerance; slow recovery following handling or activity ◆ Respiratory sounds including wheezes, friction, or crackles upon auscultation ◆ Weakness, collapse ◆ Often accompanied by other clinical signs indicative of general malaise such as weight loss, failure to gain weight (in raptors), fluffed up appearance, loss of appetite
Air sac compression due to space-occupying lesions in the coelomic cavities	◆ Rapid, shallow respiration pattern ◆ Widened leg stance ◆ Increased sternal-pubic and/or interpubic distance, or bulging of the coelom ◆ Palpable mass (e.g., egg) in the abdominal region ◆ Hunched posture with kinking of the tail ◆ Other clinical signs such as tenesmus, unilateral paresis/paralysis can be present

Modified from van Zeeland YRA: Upper respiratory tract disease. In Chitty J, Monks D, editors: *BSAVA Manual of avian practice: a foundation manual*, Quedgeley, UK, 2018, BSAVA British Small Animal Veterinary Association, pp. 299–316.

DIAGNOSTIC WORKUP

Dependent on history and the physical exam findings, it may already be possible to specify the anatomical location involved in the respiratory distress (Box 27.1):

- Nasal discharge, sneezing and/or staining, or dried discharge in the feathers around the nares suggest involvement of the nasal cavity.
- Periorbital swellings or sunken eyes (especially in macaws) point toward sinus involvement.
- In case of tracheal or syringeal disease, signs often include coughing, abnormal breathing sounds (stridor), and change in pitch or voice (particularly in psittacine birds).
- Frictions, crackles, or wheezes heard upon auscultation are suggestive of involvement of the lungs or air sacs in the disease process.
- A widened leg stance, increased interpubic or sternopubic distance, or palpable mass is indicative of space-occupying lesions in the coelomic cavity.

Differential diagnoses for each of the aforementioned locations can be found in Tables 27.1 to 27.4.

Dependent on the suspected location of the problem, additional workup may include one or more of the following diagnostic tests:

- Nasal or sinus flushing, including collection of samples for cytology, bacteriology, mycology, parasitology (wet mount),

TABLE 27.1

Differential Diagnosis for Nasal/Sinus Disease (Distorted Nares, Nasal Discharge, Sinus Swelling)

Differential Diagnosis	Comments
BACTERIAL INFECTIONS	
Gram-negative bacteria (e.g., *E. coli*, *Haemophilus*, *Klebsiella*, *Pasteurella*, *Pseudomonas*), gram-positive bacteria (e.g., *Mycobacterium*, *Streptococcus*, *Staphylococcus*)	Gram-negative infections most common; *Haemophilus* in chickens *(Gallus gallus domesticus)* (see Chapter 6)
Spirochetes	Spirochetes in cockatiels *(Nymphicus hollandicus)* of 3–10 weeks of age (lockjaw syndrome; see Chapters 31 and 41)
Chlamydia psittaci	All bird species susceptible; consider in all birds that have recently been in contact with other birds; often systemic illness (see Chapter 18)
Mycoplasma spp.	Common in smaller Psittaciformes (budgerigars *[Melopsittacus undulatus]*, cockatiels *[Nymphicus hollandicus]*), Passeriformes, Galliformes (see Chapter 20)
FUNGAL INFECTIONS	
Aspergillus spp., *Candida albicans*, *Cryptococcus* spp., *Mucor* spp.	Common in birds with malnutrition or stress; formation granulomas that block the trachea or syrinx; may also affect other parts of the respiratory system (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease) *Cryptococcus* infrequently causes disease in birds (see Chapter 21)
VIRAL INFECTIONS	
Avian influenza, herpesvirus, poxvirus, reovirus	Poxvirus common in Columbiformes, Galliformes, Passeriformes (canaries [Serinus canaria]), birds of prey (see Chapter 15)
PARASITIC	
Cryptosporidium spp.	Particularly in Galliformes, birds of prey (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease)
Trichomonas gallinae	Columbiformes, Passeriformes, smaller psittacines, birds of prey (see Chapter 22)
Aprocta cylindrical	Nematode, identified in European robins *(Erithacus rubecula)*, a variety of African and Asia passerines, and a parrot
Knemidocoptes (scaly mite)	Particularly in budgerigars *(Melopsittacus undulatus)*
NUTRITIONAL	
Hypovitaminosis A, resulting in squamous metaplasia and noninfectious granulomas	Particularly in Psittaciformes fed an all-seed diet and/or eating selectively
Rhinolithiasis	Often resulting from nutritional imbalances, particularly hypovitaminosis A, leading to squamous metaplasia of the epithelium (see Chapter 28)
ALLERGIC	
Inhalant allergens (e.g., smoke, perfume, feather dander)	Macaws *(Ara spp.)* may be more susceptible (see Chapter 28)
TOXIC	
Cigarette or tobacco smoke, aerosol sprays, ammonia	History, demonstrating a relationship between exposure and time of onset of clinical signs, is suggestive (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 18, Advancements in Diagnosis and Management of Toxicologic Problems)
NEOPLASTIC	
Lymphoma, fibrosarcoma, (adeno)carcinoma, melanoma, papilloma, squamous cell carcinoma, etc.	Older birds (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 3, Neoplastic Diseases in Avian Species)
ENVIRONMENTAL	
Low humidity, dust, feather dander of other birds (notably cockatoos, [Cacatuidae], grey parrots [*Psittacus erithacus*])	Birds housed under suboptimal conditions (see Chapter 28)
FOREIGN BODY	
For example, millet seed	May result in secondary rhinitis
TRAUMA	
Resulting in (subcutaneous) hemorrhage, emphysema, distortion, and/or fractures	History is suggestive (see Chapter 34)

TABLE 27.1

Differential Diagnosis for Nasal/Sinus Disease (Distorted Nares, Nasal Discharge, Sinus Swelling)—cont'd

Differential Diagnosis	Comments
COAGULOPATHY	
Bleeding disorders resulting in epistaxis (e.g., conure bleeding syndrome)	Conure bleeding syndrome (only in conures, including -but not limited to - the blue-crowned conure *[Thectocercus acuticaudatu]*, nanday conure *[Aratinga nenday]*, sun conure *[Aratinga solstitialis]*, peach-fronted conure *[Eupsittula aurea]*, orange-fronted conure *[Eupsittula canicularis]*, and Patagonian conure *[Cyanoliseus patagonus]*)
HORMONAL, ENDOCRINE	
Brown cere hypertrophy	Hormone-related, female budgerigars *(Melopsittacus undulatus)*
CONGENITAL DEFORMITIES	
Choanal atresia	Young birds (particularly grey parrots *[Psittacus erithacus]*; see Chapters 31 and 41)

Modified from van Zeeland YRA: Upper respiratory tract disease. In Chitty J, Monks D, editors: *BSAVA Manual of avian practice: a foundation manual*, Quedgeley, UK, 2018, BSAVA British Small Animal Veterinary Association, pp 299–316.

TABLE 27.2

Differential Diagnosis for Tracheal Disease (Voice Change, Stridor, Coughing)

Differential Diagnosis	Comments
BACTERIAL INFECTIONS	
Gram-negative (e.g., *Pseudomonas*)	Common in cockatoos (Cacatuidae); resulting in diphtheritic plaques and granulomas (see also Chapter 6)
Chlamydia psittaci	All bird species susceptible; consider in all birds that have recently been in contact with other birds; often systemic illness (see Chapter 18 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease)
FUNGAL INFECTIONS	
Aspergillus spp.	Common in birds with malnutrition or stress; formation granulomas that block the trachea or syrinx; may also affect other parts of the respiratory system (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease)
VIRAL INFECTIONS	
Adenovirus, avian influenza virus, cytomegalovirus, herpesvirus (e.g., Amazon tracheitis), poxvirus, polyomavirus, paramyxovirus	Species commonly affected: Cytomegalovirus: finches (Fringillidae) Herpesvirus: finches (Fringillidae), Amazon parrots *(Amazona spp.)*—rare (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease) Poxvirus: Columbiformes, Galliformes, Passeriformes (canaries *[Serinus canaria]*), birds of prey (see Chapter 15) Paramyxovirus: birds of prey, chickens (Newcastle disease) (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 15, Management and Medicine of Backyard Poultry)
PARASITIC	
Cryptosporidium spp. Gape worms: *Syngamus trachea*, *Cyathostoma* spp. Tracheal mites: *Sternostoma tracheacolum*, *Cytodites* spp. *Trichomonas gallinae, Tetratrichomonas anatis*	Species commonly affected: Cryptosporidiosis: Galliformes, birds of prey (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease) Gape worms: larger Passeriformes (e.g., crows *[Corvus spp.]*, starlings [Sturnidae]), chickens *(Gallus gallus domesticus)* Tracheal mites: smaller Passeriformes (e.g., canaries *[Serinus canaria]*, finches [Fringillidae]) Trichomoniasis: Columbiformes, Passeriformes, birds of prey, smaller Psittaciformes (see Chapter 22) Tetratrichomoniasis: Anseriformes
NUTRITIONAL	
Hypovitaminosis A, resulting in squamous metaplasia and noninfectious granulomas	Particularly in Psittaciformes fed an all-seed diet and/or eating selectively

Continued

TABLE 27.2

Differential Diagnosis for Tracheal Disease (Voice Change, Stridor, Coughing)—cont'd

Differential Diagnosis	Comments
ALLERGIC	
Inhalant allergens (e.g., smoke, perfume)	Macaws [*Ara* spp.] may be more susceptible? (see Chapter 28)
TOXIC	
Airborne toxins, e.g., cigarette or tobacco smoke, perfume, aerosol sprays	History, demonstrating a relationship between exposure and time of onset of clinical signs, is suggestive (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 18, Advancements in Diagnosis and Management of Toxicologic Problems)
ENVIRONMENTAL	
Dust, dander of other birds (particularly cockatoos, [Cacatuidae], grey parrots *[Psittacus erithacus]*)	Birds housed under suboptimal conditions (see Chapter 28)
NEOPLASTIC	
Carcinoma, adenocarcinoma, papilloma, squamous cell carcinoma, tracheal osteochondroma, etc.	Older birds (see see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 3, Neoplastic Diseases in Avian Species)
FOREIGN BODY	
For example, millet seed	Common in cockatiels *(Nymphicus hollandicus)*
TRAUMA	
Postintubation stricture, resulting in tracheitis and stenosis	History is suggestive; macaws (*Ara* spp.) may be predisposed (see Chapter 28)
IATROGENIC	
Aspiration of food, medication	May result in acute asphyxia and death
EXTRAMURAL MASSES OR COMPRESSION	
Goiter; granulomas; neoplasia	Goiter common in budgerigars *(Melopsittacus undulatus)* as a result of all-seed diet resulting in iodine deficiency (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 10, Clinical Endocrinology of the Protein Hormones)
Fractures of the coracoid bone (excessive callus formation)	

Modified from van Zeeland YRA: Upper respiratory tract disease. In Chitty J, Monks D, editors: *BSAVA Manual of avian practice: a foundation manual*, Quedgeley, UK, 2018, BSAVA British Small Animal Veterinary Association, pp 299–316.

TABLE 27.3

Differential Diagnosis for Disease Affecting the Lungs and/or Air Sacs

Differential Diagnosis	Comments
BACTERIAL INFECTIONS	
Gram-negative (e.g., *Pseudomonas, Salmonella*), gram-positive (e.g., *Nocardia*)	May occur secondary to inflammatory disease (see Chapter 6)
Chlamydia psittaci	All bird species susceptible; consider in all birds that have recently been in contact with other birds; often systemic illness (see Chapter 18 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease)
Mycoplasma spp.	Common in Galliformes, Passeriformes; often difficult to diagnose (see Chapter 20)
Mycobacterium spp.	Commonly *M. avium-M. intracellulare, M. genavense*; causes granulomatous or miliary pneumonia, systemic disease; occasionally involvement of the air sacs (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease)
FUNGAL INFECTIONS	
Aspergillus spp.	Common in birds with malnutrition or stress; granulomas in the lungs and/or air sacs (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease)
VIRAL INFECTIONS	
Avian influenza virus, infectious bronchitis, herpesvirus (e.g., Amazon tracheitis, parakeet herpesvirus), poxvirus, polyomavirus, paramyxovirus I, reovirus	Species commonly affected: Infectious bronchitis—chickens (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease and Chapter 15, Management and Medicine of Backyard Poultry) Poxvirus: Columbiformes, Galliformes, Passeriformes (canaries) *[Serinus canaria]*) (see Chapter 15) Polyomavirus: nestling psittacines (cockatoos [Cacatuidae], macaws [*Ara* spp.])

TABLE 27.3

Differential Diagnosis for Disease Affecting the Lungs and/or Air Sacs—cont'd

Differential Diagnosis	Comments
PARASITIC	
Sarcocystis spp. *Leukocytozoon* spp. *Toxoplasma gondii* *Sternostoma tracheocolum* Nematodes, e.g., *Serratospiculum, Syngamus/Cyathostoma* Trematodes (*Circumvitellatrema* spp.), Pentastomids	Species commonly affected: *Sarcocystis falcatula*: definitive host—Virginia opossum; *(Didelphis virginiana)*; cowbirds (*Molothrus* spp.) and grackles (*Quiscalus* spp.)—intermediate hosts; parrots are inadvertent hosts (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease) *Sternostoma*: finches (Fringillidae), canaries *(Serinus canaria)* *Syngamus/Cyathostoma:* seen particularly in birds (Galliformes, softbills) kept in soil- or turf-based aviaries *Serratospiculum:* common in birds of prey from the Middle East *Circumvitellatrema* spp. have been described in barbets (Capitonidae, Lybiidae, Megalaimidae, Semnornithidae), mynahs (*Acridotheres* spp.), motmots (Momotidae); trematode uses terrestrial snails as part of its life cycle
NUTRITIONAL	
Hypovitaminosis A, resulting in squamous metaplasia of the bronchi	Particularly in Psittaciformes fed an all-seed diet and/or eating selectively; predisposes to secondary bacterial and fungal infections
Pulmonary mineralization	Excess dietary calcium or severe renal disease
ALLERGIC	
Inhalant allergens (e.g., smoke, perfume) Macaw pulmonary hypersensitivity	History, demonstrating a relationship between exposure and time of onset of clinical signs, is suggestive (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 18, Advancements in Diagnosis and Management of Toxicologic Problems). Macaw pulmonary hypersensitivity likely due to exposure to dander from grey parrots *(Psittacus erithacus)* or cockatoos (Cacatuidae) (see Chapter 28)
TOXIC	
Polytetrafluoroethylene (PTFE, Teflon) Other airborne toxins, e.g., cigarette or tobacco smoke, perfume, aerosol sprays	Diagnosis based on history (cooking with new pans or other cooking utensils with nonstick coating); very common; often leads to death unless very small amounts are inhaled (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 18, Advancements in Diagnosis and Management of Toxicologic Problems).
ENVIRONMENTAL	
Pneumoconiosis, e.g., due to exposure to airborne pollutants	Birds housed under suboptimal conditions
Chronic pulmonary interstitial fibrosis	Particularly common in (older) Amazon parrots, (*Amazona* spp.), possibly associated with right-sided heart failure (see Chapter 28 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 6, Cardiology)
NEOPLASTIC	
Primary: Bronchial (adeno)carcinoma, air sac cystadenocarcinoma, fibrosarcoma, undifferentiated pulmonary tumors, etc.	Older birds
Secondary: metastases	Tumor metastases in the lungs include metastases from (adeno)carcinoma, fibrosarcoma, hemangiosarcoma, liposarcoma, lymphosarcoma, melanoma, mesothelioma, and osteosarcoma (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Neoplastic Diseases in Avian Species)
FOREIGN BODY	
Embolic pneumonia	Pulmonary emboli from fat, bone marrow, and egg yolk have been recognized.
Inhalation of foreign material	Potentially due to trauma
TRAUMA	
Air sac rupture, hemorrhage	History is suggestive (see Chapter 34)
IATROGENIC	
Aspiration of food, medication	Aspiration of hand-feeding formula, particularly in hand-fed psittacine chicks; may result in acute asphyxia and death (see Chapter 41)
CARDIOVASCULAR	
Lung edema	Due to left-sided cardiac insufficiency, pulmonary hypertension; particularly in older birds (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 6, Cardiology)
IMMUNE-MEDIATED	
Amyloidosis	In response to many chronic conditions
IDIOPATHIC	
Endogenous lipid pneumonia	Unknown cause; may represent storage disease or acquired disorder due to reduced airway clearance

Continued

TABLE 27.3

Differential Diagnosis for Disease Affecting the Lungs and/or Air Sacs—cont'd

Differential Diagnosis	Comments
Pulmonary proteinosis	Characterized by eosinophilic, amorphous, or crystalline material within the lumen of tertiary bronchi and air capillaries; cause and significance are unknown
Osseous metaplasia	Characterized by small spicules of bone in the pulmonary parenchyma, particularly in older birds; cause and significance are unknown
Melanosis	Pigment deposition in the lungs of larger birds, particularly cockatoos (Cacatuidae); should be considered normal

Modified from van Zeeland YRA: Upper respiratory tract disease. In Chitty J, Monks D, editors: *BSAVA Manual of avian practice: a foundation manual*, Quedgeley, UK, 2018, BSAVA British Small Animal Veterinary Association, pp 299–316.

TABLE 27.4

Differential Diagnosis for Extrarespiratory Disease Resulting From Coelomic Distension

Differential Diagnosis	Comments
ASCITES	
(Modified) transudate	Cardiac disease, liver cirrhosis, hypoproteinemia/hypoalbuminemia (e.g., due to renal disease, hepatopathy, protein-losing enteropathy), neoplastic (e.g., mesothelioma) (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 3, Neoplastic Diseases in Avian Species, Chapter 6, Cardiology, and Chapter 8, Pleura, Pericardium, and Peritoneum: The Coelomic Cavities of Birds and Their Relationship to the Lung–Air Sac System)
Exudate	Egg yolk coelomitis to peritonitis, e.g., due to intestinal perforation
Blood	Trauma (e.g., liver rupture), coagulopathy
ORGANOMEGALY	
Proventricular dilatation disease	Common in psittacine birds; avian ganglioneuritis, associated with bornavirus infection (see Chapter 14 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease)
Intestinal impaction, ileus	Mechanical obstruction (e.g., neoplasia, foreign body); paralytic ileus (e.g., heavy metal toxicity; see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 18, Advancements in Diagnosis and Management of Toxicologic Problems)
Hepatomegaly	
Renal	
Gonadal	Fatty liver; viral or bacterial hepatitis (e.g., *Chlamydia* spp.); aflatoxicosis; neoplastic (e.g., lymphoma) (see Chapter 18 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease, Chapter 3, Neoplastic Diseases in Avian Species, Chapter 18, Advancements in Diagnosis and Management of Toxicologic Problems)
	Neoplasia (e.g., renal carcinoma; see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 3, Neoplastic Diseases in Avian Species), cysts; hydronephrosis
	Cysts; testicular or ovarian neoplasia Cysts; testicular or ovarian neoplasia (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 3, Neoplastic Diseases in Avian Species)
FAT ACCUMULATION	
Obesity; nutritional	Birds with a sedentary lifestyle, being fed ad libitum, often seed-based diets. Particularly common in Amazon parrots (*Amazona* spp.), galahs *(Eolophus roseicapillus)*, budgerigars *(Melopsittacus undulatus)*
EGG	
Egg binding	Often related to calcium deficiency, but other options (e.g., oviductal torsion, salpingitis, neoplastic processes, geriatric birds, primiparity, obesity) also possible
TRAUMATIC	
Ventral abdominal herniation	Common in female birds, related to chronic reproductive problems (e.g., excessive egg laying; see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 12, Reproduction))

Modified from van Zeeland YRA: Upper respiratory tract disease. In Chitty J, Monks D, editors: *BSAVA Manual of avian practice: a foundation manual*, Quedgeley, 2018, BSAVA British Small Animal Veterinary Association, pp. 299–316.

and/or testing for specific pathogens (see Section 5: Updates on Infectious Diseases and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease);
- Choanal or pharyngeal swabs for further testing (as listed for nasal flushes);
- Tracheal swab or lavage for further testing (as listed for nasal flushes);
- Diagnostic endoscopy of the nasal cavity, trachea, and/or coelomic cavities, with subsequent sampling for culture and sensitivity testing, cytology, and/or histopathology (see Chapter 30);
- Hematology and biochemistry (see *Current Therapy in Avian Medicine and Surgery*, Vol 1., Chapter 13, Advances in Clinical Pathology and Diagnostic Medicine);
- Blood gas analysis;

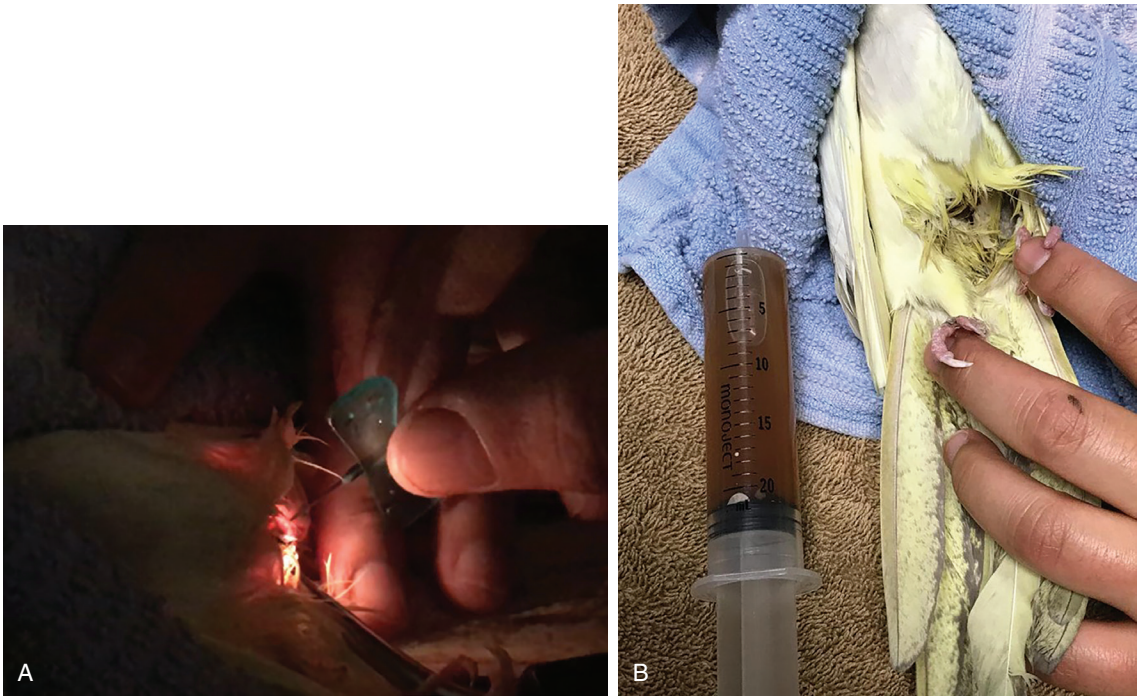


FIGURE 27.2 Coelomocentesis in a female cockatiel *(Nymphicus hollandicus)* with marked ascites. (A) There are three people involved in the procedure, which is performed in a darkened room: one person restrains the bird and provides flow-by oxygen, another transilluminates (light source is at the right lower aspect) and places the butterfly needle for aspiration, and a third aspirates fluid. (B) A total of 22 mL of transudative fluid was removed from this bird through a single puncture. Once stabilized, a primary diagnosis was able to be pursued and was confirmed to be ovarian carcinoma at surgery.(Photos courtesy Brian Speer.)

- Diagnostic imaging of the lungs and air sacs by means of radiography, standing fluoroscopy, and/or computed tomography (CT) (see Chapter 2 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 14, Advances in Diagnostic Imaging);
- Evaluation of the coelom by means of transillumination in small bird species (Figure 27.2), ultrasonography, radiography, and/or CT imaging (see Chapter 2 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 14, Advances in Diagnostic Imaging);
- Blind or ultrasound-guided coelomocentesis or fine needle aspirates for biochemical analysis, microbiology, and/or cytology (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 13, Advances in Clinical Pathology and Diagnostic Medicine);
- Fecal examination for presence of nematode eggs (*Syngamus trachea*, *Capillaria* spp.).

Boxes 27.2 to 27.4 provide flow diagrams to help guide the workup of nasal discharge, tracheal disease, and respiratory distress.

Nasal and Sinus Flushing

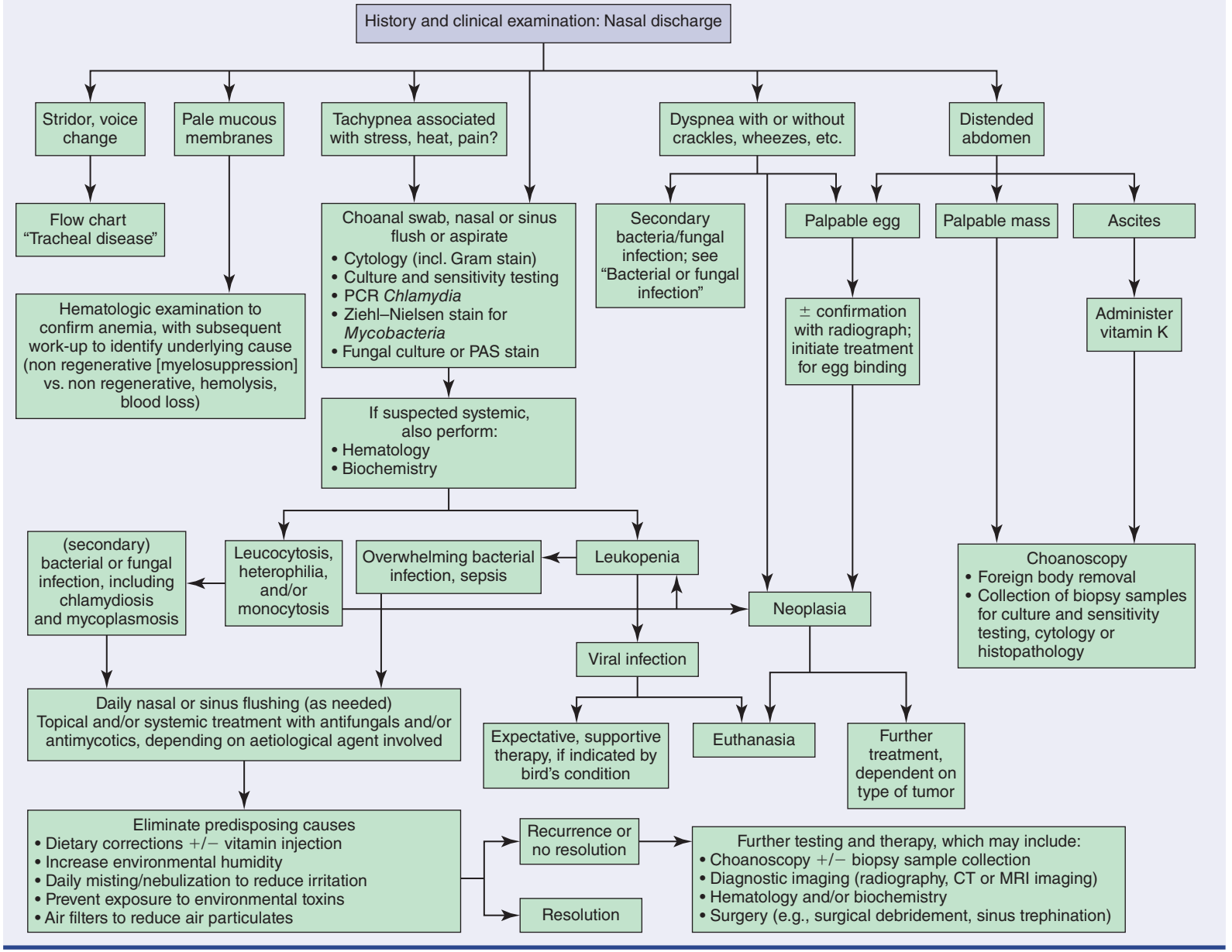
A nasal flush can be obtained with relative ease by placing a syringe filled with sterile, lukewarm saline in one of the nares, tilting the patient upside down, and then forcefully flushing the saline into the nasal passage while the other naris is simultaneously held closed with a finger (Figure 27.3). Commonly used volumes per naris include 3 to 5 mL for smaller birds (e.g., budgerigar, cockatiel), 6 to 12 mL for middle-sized birds (e.g., grey parrot, Amazon parrot), and 12 to 20 mL for larger-sized birds (e.g., large cockatoos, macaws). Flushing of the nares should be performed carefully while closely monitoring the patient as excessive and/or rapid flushing may cause the patient to aspirate fluids and suffocate. Following a nasal flush, the infused saline should be expelled via the choana and can be collected for, for example, cytology or microbiology.

Aside from obtaining samples, nasal flushing is also helpful from a therapeutic standpoint, helping to clear exudate from the nasal cavity and sinuses. However, the complex anatomy of the sinuses makes it difficult to successfully flush out all the material and remove the nidus of an infection, hence increasing the likelihood of relapse or reoccurrence of the infection. Infraorbital aspiration and flushing may therefore provide better results.

Collection of Swabs from the Choanal Slit

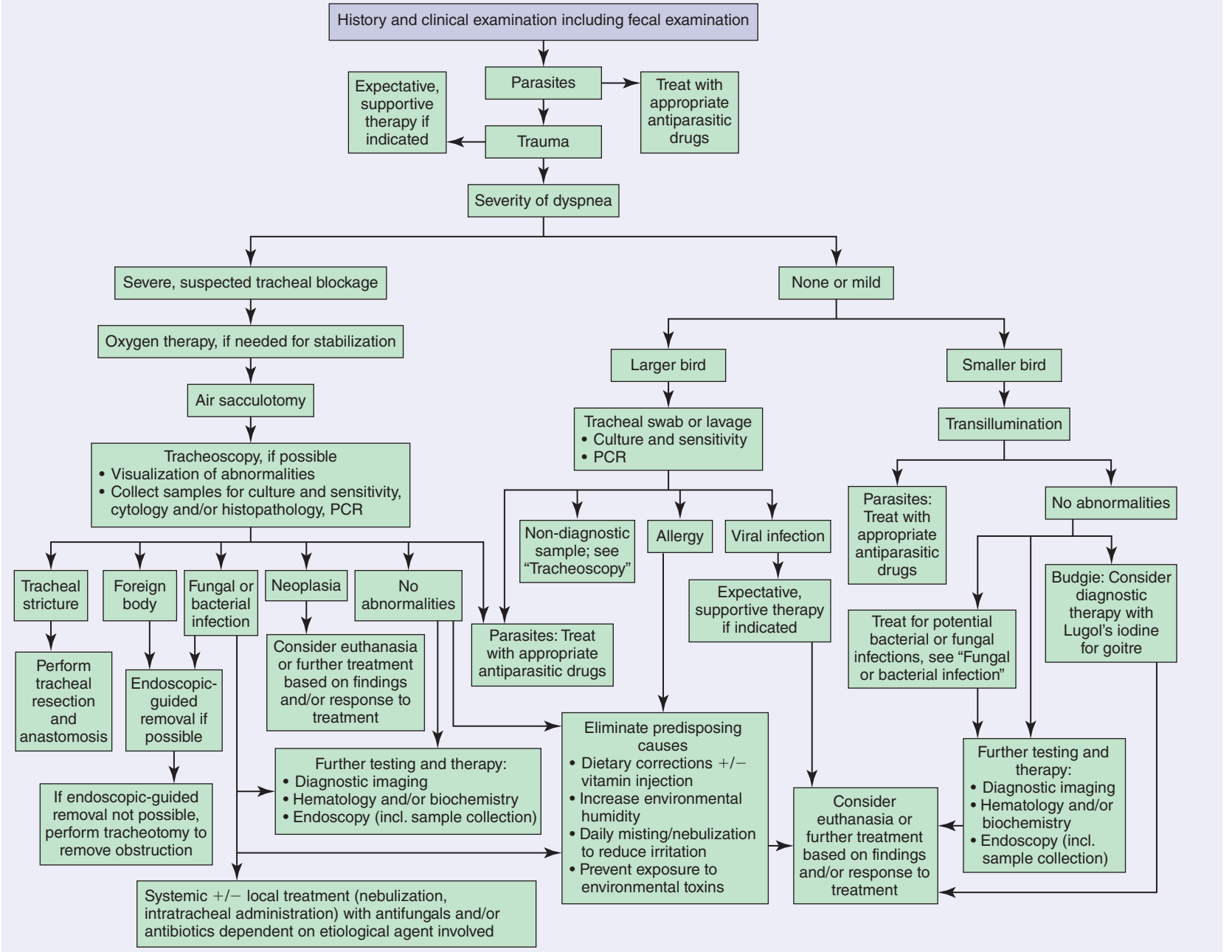
Swabs may be collected from the choanal slit to be submitted for cytology, microbiology, or testing for specific pathogens, such as *Chlamydia psittaci* (see Chapter 18 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). If fungal disease is suspected, samples may be submitted for periodic acid-Schiff staining or fungal culture; Ziehl-Nielsen staining or polymerase chain reaction (PCR) testing may be useful in case of suspected mycobacteriosis (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Results should always be interpreted with care since samples collected from the nares or nasal cavity may easily be contaminated. The normal flora of the nasal cavity in many bird species primarily consists of

BOX 27.2 FLOW DIAGRAM OF NASAL DISCHARGE



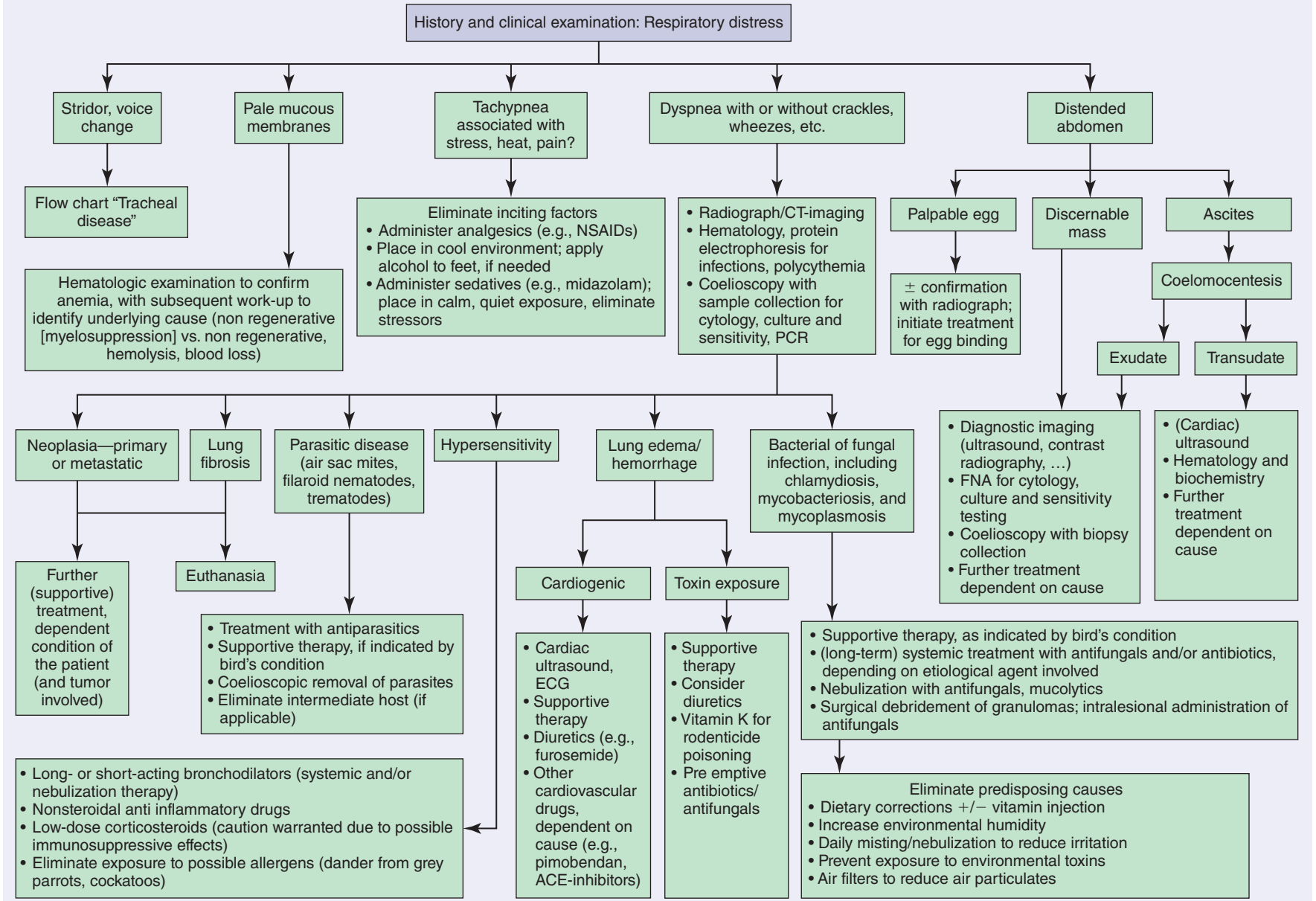
Modified from van Zeeland YRA: Upper respiratory tract disease. In Chitty J, Monks D, editors: *BSAVA Manual of avian practice: a foundation manual*, Quedgeley, 2018, BSAVA British Small Animal Veterinary Association, pp. 299–316.

BOX 27.3 FLOW DIAGRAM OF TRACHEAL DISEASE



Modified from van Zeeland YRA: Upper respiratory tract disease. In Chitty J, Monks D, editors: *BSAVA Manual of avian practice: a foundation manual*, Quedgeley, 2018, BSAVA British Small Animal Veterinary Association, pp. 299–316.

BOX 27.4 FLOW DIAGRAM OF RESPIRATORY DISTRESS



Modified from van Zeeland YRA: Upper respiratory tract disease. In Chitty J, Monks D, editors: *BSAVA Manual of avian practice: a foundation manual*, Quedgeley, 2018, BSAVA British Small Animal Veterinary Association, pp. 299–316.



FIGURE 27.3 Nasal flush in a blue-fronted Amazon parrot *(Amazona aestiva)*. For this procedure, the bird is tilted with the head and beak held low to prevent water from running into the trachea. After placing a syringe filled with sterile, lukewarm saline in one of the nares, the saline is forcefully flushed into the nasal passage, while the other naris simultaneously can be held closed with a finger. Often, material leaks out of the choanal slit, which may subsequently be collected for further workup (e.g., cytology, culture and sensitivity testing). (Photo courtesy Yvonne van Zeeland/Nico Schoemaker.)

gram-positive rods and cocci, while yeasts and/or gram-negative rods are less common as normal flora.[5–8] False-negative results may also occur, especially if *Mycoplasma* spp. are involved, which are notoriously difficult to isolate using routine testing (see Chapter 20).

Sinus Aspirate

Sinus aspirates may be collected from the ventral and rostral parts of the infraorbital sinus using a 22- to 25-gauge (G) needle and syringe (Figure 27.4; see also Chapter 29). It is preferable to sedate or anesthetize the patient for this procedure as sudden movement by the patient may lead to puncture of the eye or other vital structures.

The rostral area may be approached by inserting the needle at the commissure of the beak and directing it perpendicular to the skin below the jugal arch to a point between the eye and naris, whereas the ventral area may be approached from two directions:

- ventral to the jugal arch and just ventral to the eye, while holding the needle perpendicular to the skin;
- just caudal to the commissure of the beak while directing the needle toward the point ventral to the eye and jugal arch.

If aspiration fails to produce any material, sterile fluid may be infused into the sinus using one of the abovementioned locations. The collected fluid can be submitted for further microbial and cytological analysis. Should a surgical approach be indicated (see Chapter 29), biopsies can be collected from the sinus epithelium and submitted for histopathology or culture and sensitivity testing.

Tracheal Swabs or Lavage

In case of suspected tracheitis, tracheal swabs or lavage can be performed to collect material for cytology or microbial culture. Tracheal lavage is preferably performed under general anesthesia, using a soft, sterile tube to infuse sterile saline (0.5–10 mL/kg) into the trachea, which is subsequently aspirated using a sterile syringe that is attached to the tube. Alternatively, 18- to 22-G Teflon catheters may be transdermally inserted into the trachea.

Hematology and Biochemistry

In patients with respiratory distress, a complete blood count (CBC) and protein electrophoresis can be helpful to determine whether an infection is present. Although results rarely point toward a specific disease, a leukocytosis, heterophilia, or monocytosis may indicate presence of a (bacterial or fungal) infection or inflammation, whereas leukopenia is highly suggestive of presence of an overwhelming viral or bacterial infection. The hematocrit or packed cell volume (PCV) can be helpful to evaluate the presence of anemia (which may result

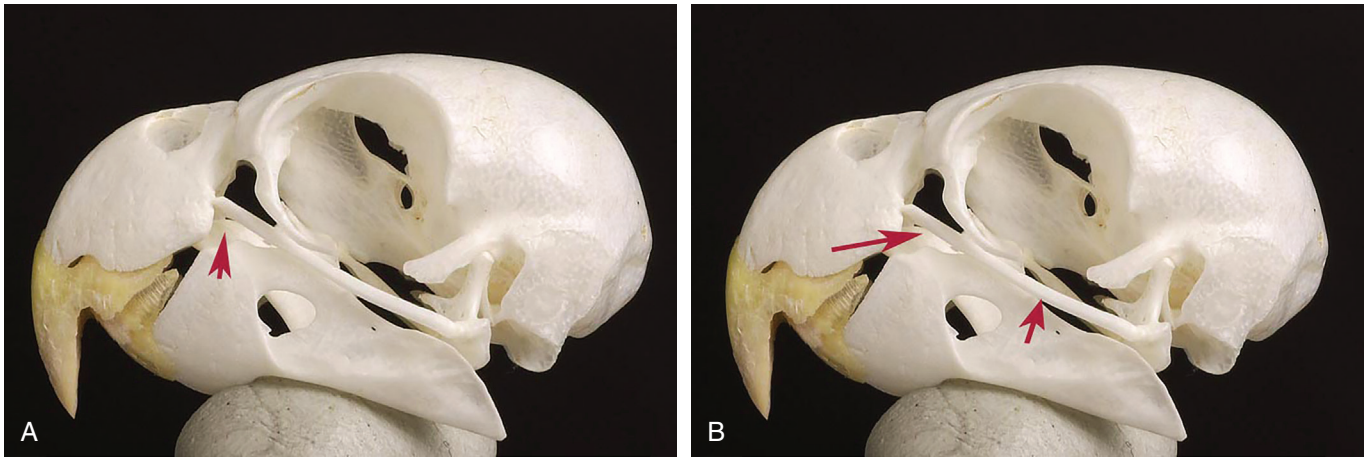


FIGURE 27.4 (A) Photograph of a peach-faced lovebird *(Agapornis roseicollis)* skull indicating the location to perform a sinus aspirate of the rostral area *(arrow)*. (B) Photograph of a lovebird skull indicating the location to perform a sinus aspirate of the ventral area using a rostral *(left arrow)* or ventral *(right arrow)* approach. (Photo courtesy Yvonne van Zeeland/Nico Schoemaker.)

TABLE 27.5

Venous Blood Gases in a Variety of Avian Species and Arterial Blood Gases in Amazon Parrots

Results	Budgerigars *(Melopsittacus undulatus)* $n = 20$[11,12]	Pigeons *(Columba livia domestica)* 37°C; $n = 60$[13]	Pigeons *(Columba livia domestica)* 42.1°C; $n = 60$[13]	American or Carribean Flamingo *(Phoenicopterus ruber)*; $n = 9$[14]	Falcons (*Falco rusticolus*, cross breds); $n = 70$[15]	Mourning Dove *(Zenaida macroura)*; $n = 24$[16]	Boat-Tailed Grackle *(Quiscalus major)*; $n = 17$[16]	House Sparrow *(Passer domesticus)*; $n = 18$[16]	Mottled Duck *(Anas fulvigula)*; $n = 59–62$[17]	Blue-Fronted Amazon Parrot *(Amazona aestiva)*; $n = 35$[18]	Quaker Parrots *(Myiopsitta monachus)*; $n = 35$[19]	Grey Parrots *(Psittacus erithacus)*; $n = 47$[20]
pH	7.33–7.49	7.48–7.60	7.41–7.51	7.17–7.47	7.4–7.6	7.29–7.56	7.42–7.58	7.30–7.52	7.38 ± 0.05	7.34–7.55	7.41–7.45	7.14–7.49
PCO_2 (mm Hg)	30.6–43.2	24.5–30.8	30.4–39.0	23–39	27–49.9	21–45.9	23.1–36.9	31.8–49.7	36.3 ± 0.6	14.6–29.8	27.3–29.9	19.9–40.6
PO_2 (mm Hg)	85–99	58.2–71.2	83.4–100.3	30–50		22–63	39–56	27–49	45.6 ± 0.6	85–113	39.5–42.4	28–48
HCO_3 (mmol/L)	21–26	20–25	21–26	8–21	26–29	26–29	16–31	16.9–30.1	20.6 ± 0.2	9.5–21	17.8–20.7	9.7–22.3

in tachypnea) or polycythemia, which can develop in response to chronic hypoxia, for example, due to lung fibrosis.

Blood Gas Analysis

Blood gas analysis of arterial blood samples provides superior information on the efficacy of gas exchange compared to venous blood samples.[9] However, obtaining arterial blood samples from dyspneic birds generally poses a greater risk for the patient as the handling is profoundly longer, and the risk of complications (e.g., hematoma formation, infection, and thrombosis) is high. Venous blood samples, on the other hand, can be obtained relatively quick, safe, and easy, with minimal stress for the bird. In mammals, venous blood samples are also used in case of acute exacerbation of chronic pulmonary disease. Blood gas analysis of these samples has been shown to correlate well with blood gas analysis of arterial samples and can therefore be used to reliably predict the arterial blood gas values.[10]

In recent years, multiple studies have been performed on healthy individuals from different bird species. Except for Amazon parrots, in which results were obtained from arterial samples, venous blood samples were used. Table 27.5 summarizes the data on blood gas analysis in the various bird species.

Aside from the type of blood sample collected, circumstances under which the samples are obtained also influence the results. When blood is collected using sedation or anesthesia, results are commonly influenced, for example, as a result of supplemental oxygen being provided during, for example, isoflurane anesthesia, or as a result from hypoventilation that can occur as a result of the drugs used for sedation/anesthesia (Table 27.6).[22]

Despite the increasing number of studies in healthy birds, data on blood gases in birds with respiratory diseases are still limited[21] and warrant further studies to enable evaluation of their true merit.

Conventional Radiography versus Computed Tomography

In the first volume of *Current Therapy in Avian Medicine and Surgery*, an extensive chapter (Chapter 14) was devoted to Advances in Diagnostic Imaging.[23] With the ongoing advancements and increased availability of CT imaging, this modality seems to be gaining popularity in clinical practice. It has been demonstrated many years ago that CT is superior to conventional radiography for evaluation of the respiratory tract (Figure 27.5).[24] Preliminary results from a more

TABLE 27.6

Venous Blood Gases (First and Third Quartiles) From 95 Passeriformes After Sedation With Intranasal Midazolam (~5 mg/kg), or Without Sedation[21]

Results	Without Midazolam	Following Sedation With Midazolam
pH	7.54–7.63	7.52–7.61
PCO_2 (mm Hg)	23.0–29.2	26.1–33.9
PO_2 (mm Hg)	34–40	31–38
HCO_3 (mmol/L)	21.4–27	25–30.4

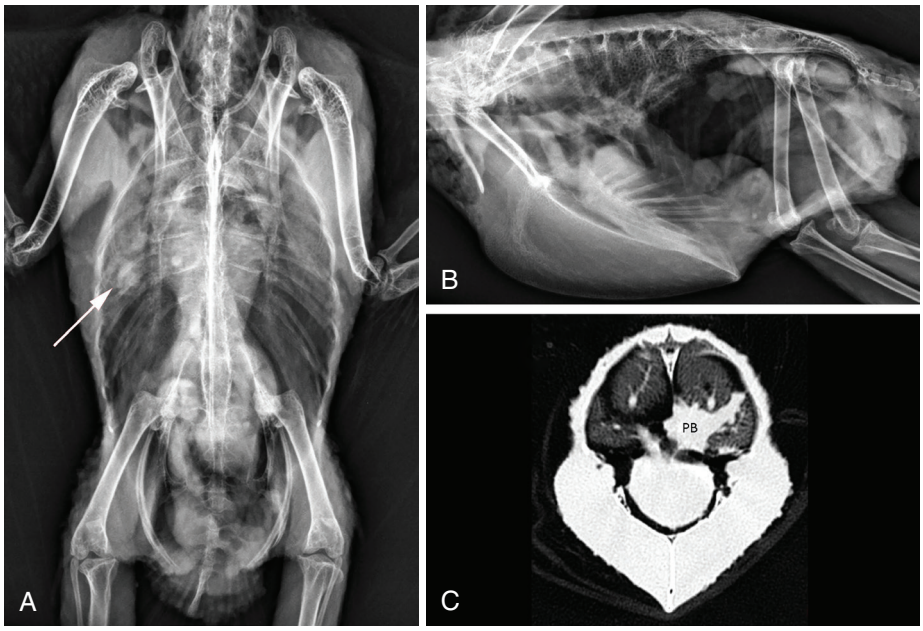


FIGURE 27.5 Comparison of radiographs (A and B) and CT images (C) of a 5-year-old female grey parrot *(Psittacus erithacus)* with severe dyspnea due to the blockage of the right primary bronchus (PB). The arrow in ventrodorsal radiograph (A) indicates the location that is suggestive for the lesion that is clearly seen on the CT image (C). On the lateral view of the radiograph (B), however, no such lesion can be clearly distinguished. *CT,* Computed tomography.

recent study comparing the interpretation of plane radiographs and CT images with postmortem results also showed that avian and radiology specialists who were asked to evaluate both the radiographs and CT could identify all lesions that were present on postmortem examination through CT while missing 50% of the pathologies on radiography, particularly those situated in the lungs. Additionally, 50% of the lesions that were identified upon radiography turned out to be absent on pathology, hence indicating that conventional radiography poses a high risk for misdiagnoses, including both false-negative and false-positive results (M. Huynh, personal communication).

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CHAPTER 28

Diseases of the Respiratory Tract

Hugues Beaufrère

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NASAL AND SINUS DISEASES

Rhinitis and sinusitis are common diseases in birds. They frequently extend to the periorbital region, and have the potential to also spread to other structures of the head owing to the extensive pneumatization of the head. Sinus exudates do not drain well in birds because of the dorsal location of the sinus opening into the nasal cavity (see Chapter 26) and the thick nature of many inflammatory exudates.

Infectious rhinitis and sinusitis may be caused by a variety of agents and frequently manifest as nasal discharge, periorbital swellings, redness, and facial feather loss (Figure 28.1). Gram-negative bacteria, *Chlamydia psittaci*, and *Mycoplasma* spp. are frequently cited as common etiologic agents in various species of birds. Uncomplicated chlamydiosis in cockatiels *(Nymphicus hollandicus)* and pigeons *(Columba livia domesticus)* frequently presents solely as mild rhinitis and conjunctivitis. Poultry suffer from a high rate of sinusitis for which *Mycoplasma gallisepticum*, *Avibacterium paragallinarum* (infectious coryza of chickens, formerly *Haemophilus*), and *Bordetella avium* (turkey coryza) are common etiologic agents.[1] *Mycoplasma gallisepticum* is also a significant pathogen causing significant morbidity in house finches *(Haemorhous mexicanus)* in North America and typically presents as conjunctivitis and sinusitis.[2] Mycoplasma infection is also associated with upper respiratory disease in psittacine birds.[3] In Falconiformes and Accipitriformes, *Mycoplasma* spp. appear to be commensal organisms.[4,5] *Mycobacterium tuberculosis* and *M. marinum* have been associated with upper respiratory granulomas in psittacine birds.[6–10] Spiral bacteria (spirochetes) have been implicated in upper respiratory disease, pruritus, and infection of the choanal slit and oral cavity in (juvenile) cockatiels (see also "lockjaw syndrome" in Chapter 31 and 41).[11] Chronic sinusitis in macaws may lead to the so-called sunken-eye syndrome.[7,12] Diagnosis is typically achieved through computed tomography (CT) scan of the head, endoscopy of the nares, choana, and/or culture of choanal swabs or sinus samples. Projection radiographs are not sensitive for these disorders except in severe cases and in larger species of birds (see Chapter 27).

Medical treatment for rhinitis and sinusitis depends on the isolated bacterial or fungal pathogen and the amount of necrotic and caseous debris present in the upper airways. Aerosol therapy may help humidify the nasal passage and facilitate local delivery of antimicrobials. Flushing the nasal cavity with saline with or without antimicrobials and/or mucolytics may help dislodge nasal debris. The head is typically held down to prevent aspiration of instilled liquid. Nasal flushing may be performed once to twice daily for several days/weeks. A nasal flush formula has been described combining amphotericin B, trypsin, and neomycin.[13] More drastic debridement is usually needed in case of rhinoliths (see later). In terms of general management and prevention, increasing the environmental humidity is frequently beneficial in psittacine species considering their natural tropical range. Improving air quality with high-efficiency particulate air (HEPA) filter may also help decrease potential airborne toxins and allergens.

Chronic granulomatous rhinitis (or rhinolith) is frequently encountered in psittacine birds on a seed-only diet and is particularly common in cockatiels, grey parrots *(Psittacus erithacus)*, and Amazon parrots (*Amazona* spp.). It seems to be promoted by nutritional deficiencies, most notably those in vitamin A. Concretions of desquamated epithelium, debris, necrotic, and inflammatory materials occupy the nasal cavity and induce progressive tissue destruction, which can be extensive. Opportunistic organisms frequently involved in the disease process include a variety of gram-negative bacteria (*Escherichia coli*, *Enterobacter* spp., *Pseudomonas* aeruginosa, *Klebsiella* spp.) and fungi (*Aspergillus* spp., *Candida* spp., *Cryptococcus* sp., Zygomycetes).[7,12] Nasal plugs consecutive to chronic rhinitis also occur in falcons.[14] It is recommended to use surgical loupes with a light source to facilitate thorough debridement of the nasal cavities and preserve the nasal operculum (only in parrots). Debridement should be done with caution as the nasal structures are fragile and are prone to bleeding. A small dental curette or an equivalent instrument with a narrow extremity should be used to remove the concretion starting at the edges to detach it from the nasal wall. The granuloma is either removed *in toto* or in fragments. Once the bulk of the granuloma is removed (Figure 28.2), the nasal cavity is thoroughly debrided with a mixture of curettage and flushing with physiological saline. (Note: The bird should be intubated to prevent aspiration during nasal irrigation; see also Chapter 29.) The nasal deficit left by the granuloma is often extensive and permanent. Granulomatous materials should be submitted for bacterial and fungal culture, and topical treatment should be pursued. Since the integrity and function of the nasal structures are compromised, this procedure would unfortunately have to be repeated periodically to clean nasal debris that constantly accumulates. A rigid endoscope may be used to explore the nasal cavities after debridement.

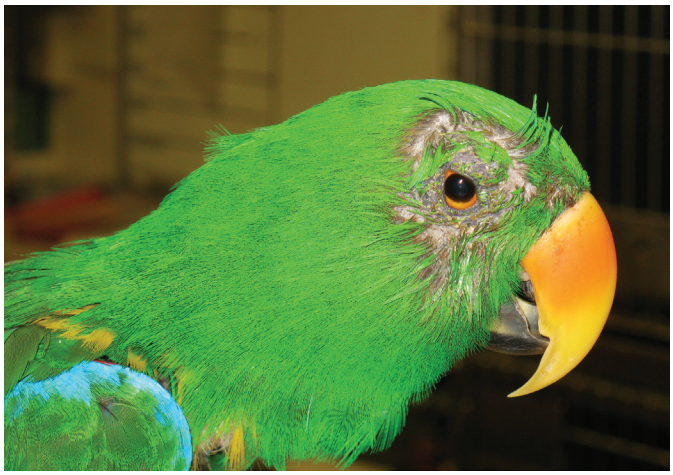


FIGURE 28.1 Periocular feather loss in an Eclectus parrot *(Eclectus roratus)* diagnosed with infraorbital sinusitis.



FIGURE 28.2 Rhinoliths removed from the nasal cavities of a grey parrot *(Psittacus erithacus)*.

For chronic sinusitis or granulomatous sinusitis, a sinusotomy or sinus trephination may be attempted. The infraorbital sinus is generally easily approached from just below the eye and jugal arch (Figure 28.3).[15] Sinus trephination is rarely performed but may allow access to hard-to-reach sinus diverticula such as in the maxillary or frontal bone (see also Chapter 29). An example of such indication can be found in a case report of a Swainson's toucan *(Ramphastos ambiguus swainsonii)* with rhinosinusitis.[16]

Viruses with specific nasal and sinus tropism are uncommon, but turkey rhinotracheitis is caused by a pneumovirus. Poxvirus infection may cause localized skin diseases around the nostrils (see Chapter 15). *Cryptococcus neoformans* was identified as the cause of a chronic rhinosinusitis in a Major Mitchell's cockatoo *(Cacatua leadbeateri)* (see Chapter 21).[17] Parasitic diseases of the nasal and sinus system are less common. Knemidocoptic mites may cause hyperkeratosis of the cere with subsequent obstruction of the nares in budgerigars *(Melopsittacus undulatus)* (Figure 28.4). Trichomoniasis may extend to the choanal area and sinus in raptors and pigeons (see Chapter 22). *Cryptosporidium baileyi* was the cause of upper respiratory infections in falcons (*Falco* spp.) and scops owls *(Otus scops)* (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).[18–20] Cryptosporidia can be diagnosed through polymerase chain reaction (PCR) or conjunctival cytology. Treatment of respiratory cryptosporidiosis may be attempted using paromomycin or azithromycin, but response appears variable (see Chapter 9).[18,19,21] Various helminth parasites may be found in the nasal and sinus tissues in wild water birds, in particular Anseriformes, including trematodes of the Cyclocoelidae family, *Trichobilharzia* spp. schistosomes, and *Theromyzon* spp. nasal leeches.[22–24]

Plant or seed foreign bodies can be lodged in the nasal cavity or the choanal opening, and chicks may aspirate formula through their choanae. Respiratory irritants may also promote nasal inflammation and sneezing. Choanal atresia has been reported in grey parrots and an umbrella cockatoo *(Cacatua alba)* and is characterized by the incomplete development of communication from the nasal passageways to the choanal slit; however, the detailed anatomic nature of the

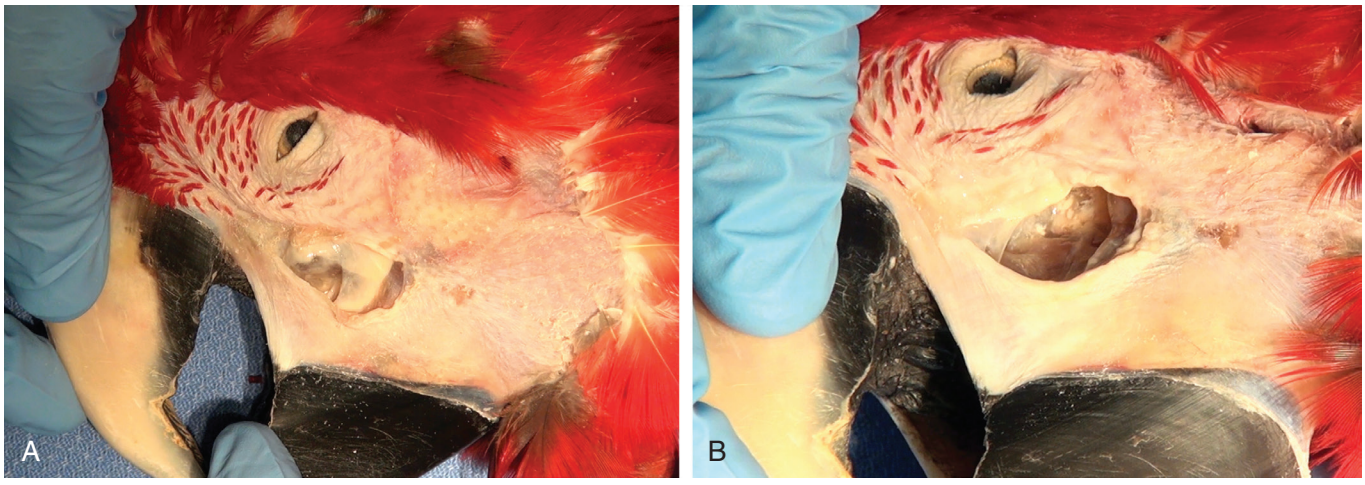


FIGURE 28.3 Sinusotomy in a scarlet macaw cadaver (*Ara macao*). A) The upper beak is flexed (protraction) and the lower beak is extended, which opens the infraorbital space and optimizes exposure as is seen in close up image B. (Courtesy Brian Speer.)



FIGURE 28.4 Budgerigar *(Melopsittacus undulatus)* with periocular knemidocoptosis that has progressed to the ceres and is starting to partially block the nares.

problem has yet to be clarified (see Section 9: Embryology, Neonatology, and Pediatrics).[7,25] It is typically repaired surgically by creating an artificial choanal opening (see Chapter 29). A pin is used to perforate a communication through each nostril to the choanae. The pin is inserted perpendicularly to the frontal bone into the nostril. A fenestrated (e.g., red rubber catheter) tube is passed through one nostril to the choanae and passed through the contralateral nostril. The tube ends are then tied on the back of the head and the tube is left in place for 4 to 6 weeks until tissue heals around it, which creates permanent communication. Significant bleeding should be expected with this surgery.

Hyperinflation/rupture of the cervicocephalic diverticulum of the infraorbital sinus is a relatively common occurrence and manifests by one or multiple large subcutaneous air pockets. This condition has been described in multiple avian species.[7,26–28] In severe cases, it may significantly restrict neck motions (Figure 28.5) as well as compromise the welfare of the bird. The pathophysiology is poorly understood, and the location of air leakage in case of a rupture has not always been properly documented. This condition may be caused by different diseases of the cervicocephalic diverticulum such as trauma, infection, congenital abnormality, and disruption of normal connection with the infraorbital sinus. The condition can either be treated through repeated aspiration of air from the cervicocephalic diverticulum, creating a stoma by marsupialization to the skin, or the placement of a stent. Another approach consists of connecting the inflated area with other parts of the head-neck and cervicocephalic diverticulum to the interclavicular air sac, deep between the clavicles to shunt air into the lower respiratory system (see Chapter 29). Finally, a technique of "pleurodesis" or "ablation" of the diverticulum may be attempted by injecting a sclerosing agent in the inflated tissue (such as doxycycline[28] or tetradecyl sodium sulfate). The neck is massaged to distribute the solution in the entire area, and a light bandage is placed to maintain pressure between the walls of the diverticulum.

Choanal and sinus squamous cell carcinoma have been reported at least in a black-footed penguin *(Spheniscus demersus)* and an Eclectus parrot *(Eclectus roratus)*, but many other cases have likely been diagnosed by veterinarians without being formally published (Figure 28.6).[29,30] Other nasal and sinus neoplasms reported in psittacine birds include lymphosarcoma, carcinoma, fibrosarcoma, and melanoma.[12]

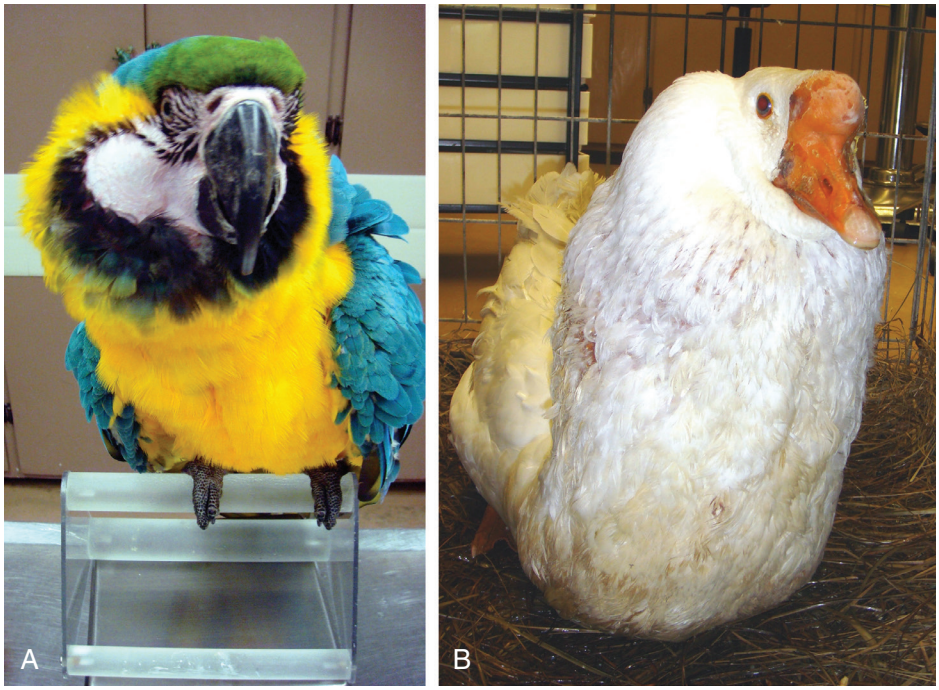


FIGURE 28.5 Cervicocephalic diverticulum rupture in a blue-and-gold macaw (*Ara ararauna*; A) and a domestic goose (*Anser anser domesticus*; B).



FIGURE 28.6 Squamous cell carcinoma in a blue-fronted Amazon parrot *(Amazona aestiva)* almost completely blocking the glottis.

TRACHEAL DISEASES

Diseases of the larynx are uncommon. Reported diseases include glottal internal papillomatosis (mainly in green-winged macaws [*Ara chloropterus*]) and laryngeal trauma from intubation (damage to the *crista ventralis*).[31–33] However, other causes (e.g., neoplasia and bacterial, mycotic and parasitic infections) should be considered in the differential diagnosis as well. (see also Chapter 27).

Postintubation tracheal stenosis usually occurs approximately 1–2 weeks after an intubation event and has been reported in parrots, raptors, and many other species.[34–38] Tracheal stenosis secondary to severe tracheitis or tracheal trauma has also been reported.[39–41] The diagnosis is readily made by tracheoscopy (Figure 28.7) but may also be made at CT imaging or even radiography in some cases. These cases are typically treated with a partial tracheal resection and anastomosis (see Chapter 29). Generallym, between five to ten tracheal rings are removed, but up to 12 to 15 rings have been removed in extreme cases.[35–38,40–42] It is essential to remove all of the tracheal tissue affected by fibroproliferation as this may inevitably lead to recurrence. Even when minimally reactive sutures are used (e.g., polydioxanone), granulomas and exudates around the sutures are not uncommon, and it is recommended to monitor the trachea periodically by tracheoscopy during the following months/years. Voice changes may also be encountered due to neurologic and muscular trauma of syringeal muscles and modification of tracheal airflow. Stenting of the trachea has been described using a custom-made nitinol stent, which was deployed in the trachea of an Eclectus parrot using tracheoscopy and fluoroscopy (see Chapter 29).[43]

Tracheal foreign bodies are more common in small psittacine birds, and particularly in cockatiels inhaled millet seed is a common cause for obstructions.[26,44–46] A tracheotomy may be necessary to remove the foreign body (see Chapter 29). Prior to the removal of a tracheal foreign body, a tracheoscopy or—in smaller species—transillumination of the trachea may be required to precisely locate the foreign body, mark the area, and insert a needle into the trachea distal to the obstruction to prevent distal migration (see Chapter 27). In small birds for which tracheotomy is challenging, an alternative technique has been proposed whereby a needle is introduced just distal to the seed with the bevel up and a large quantity of air is forced up the trachea to dislodge the seed.

When tracheal obstruction is suspected, an air sac tube may be placed either in the caudal thoracic or the abdominal air sac (see Chapter 29). The tubes frequently get obstructed, so it is advisable to fenestrate its extremity and monitor patency of the tube at least daily. During the placement of the

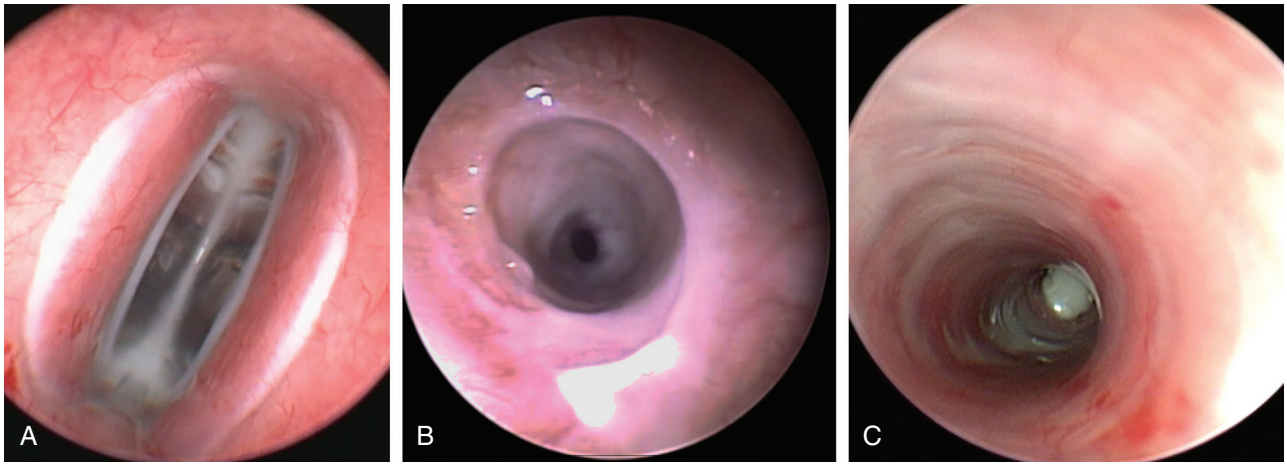


FIGURE 28.7 Tracheoscopy in birds showing the normal syrinx in an Eclectus parrot (*Eclectus roratus*; A), a fibrogranulomatous lesion associated with tracheal stenosis in a blue-and-gold macaw (*Ara ararauna*; B), and a syringeal aspergilloma in an Eclectus parrot (C).

air sac tube, it is best to perform a brief endoscopic examination through the tube to both ensure correct placement regarding the caudal ostia and perform an examination of the air sac, lung, and coelomic organs. The tube is sutured to the skin using a retention disk (Figure 28.8), tape, or directly onto the tube using a Chinese finger trap suture. A filtering system should be placed on the tip of the tube to avoid inhalation of dust and feather debris. Several materials may be used for this purpose such as found in surgical or facial masks. The tube is commonly kept for a maximum of 5 to 7 days, following which occlusion frequently occurs, and the tube should be replaced. A new tube should be placed in the contralateral air sac, if needed. When the air sac tube is removed, it is recommended to culture its tip and to perform a brief endoscopic examination of the area. Air sac tubes induce considerable inflammatory reaction, and it is not uncommon to diagnose focal aspergillosis. If such lesions are observed, a topical instillation of amphotericin B (1 mg/kg) is recommended. The stoma should be left patent until it heals by secondary intention. Topical ointment may be used to prevent or treat local infections of the site.

Various bacterial agents have been implicated in tracheitis, which may lead to tracheal stenosis either by fibrous tissue or by granuloma formation. The trachea was the principal replication site for experimental *C. psittaci* infection in the turkey (Meleagris gallopavo domesticus).[47] *Bordetella avium* primary infects the trachea in turkeys.[1] *Mycoplasma* spp. are routinely recovered from the trachea of normal birds of prey.[4,5] *Enterococcus faecalis* is associated with chronic tracheitis in canaries *(Serinus canaria)*.[48,49] A granulomatous tracheitis caused by *Mycobacterium genavense* was diagnosed in an Amazon parrot.[50]

Viral diseases of the trachea cause diphtheritic lesions and include mainly herpesvirus infections such as the infectious laryngotracheitis virus in recently imported chickens (and also pheasants and guinea fowl), the Amazon tracheitis virus in recently imported Amazon parrots and a few other psittacine species, cytomegalovirus in Australian finches, psittacid herpesvirus 3 infection in Eclectus parrots and Bourke's parakeets (*Neopsephotus bourkii*; see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease), and poxviruses which cause typical diphtheric lesions (wet form of poxviruses) that are typically encountered in passerine birds, various parakeets, lovebirds, and mynahs (see Chapter 15).[1,7,12,31,51–54] Vaccination with chicken attenuated infectious laryngotracheitis virus may cause disease in other galliform species.

Fungal infections of the trachea are seen notably with tracheal and syringeal aspergillosis (Figure 28.7; see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Species more commonly diagnosed with a tracheal location of aspergillosis include Amazon parrots, grey parrots, macaws, and falcons. Hypovitaminosis A is frequently cited as a predisposing factor for the formation of tracheal and syringeal aspergilloma.

Several parasites are known to infest the trachea and include nematodes such as *Syngamus* spp. (mainly *S. trachea*) and *Cyathostoma* spp. (mainly *C. bronchialis*) and acarids such as *Sternostoma tracheacolum*.[1,7,55] Tracheal worms infest a wide range of species, but clinical diseases are most frequently encountered in captive birds of prey, poultry, and gamebirds. The life cycle of *S. trachea* is direct, and earthworms may act as paratenic hosts, which facilitates reexposure in a closed environment with access to the ground (larvae may remain viable for up to 3 years in earthworms). *C. bronchialis* infests mainly Anseriformes and birds of prey and requires earthworms as intermediate hosts.[56] *S. tracheacolum* (tracheal, air sac mites) infests primarily canaries and Gouldian finches *(Chloebia gouldiae)*. The mite has a direct life cycle and is passed from parents to chicks.[12,56] It is usually treated with oral or percutaneous ivermectin (see Chapter 9).

Inhaled toxins and smoke inhalation injuries may cause severe necrotizing tracheitis (see Chapter 26). Hypovitaminosis A not only predisposes to respiratory diseases by altering mucosal defenses but may also promote epithelial changes, notably in the syrinx. Tracheal neoplasia is uncommon, but tracheal osteochondroma has been reported in parrots.[12] Extra-respiratory diseases resulting in tracheal compression are also numerous and are dealt with in the corresponding section.

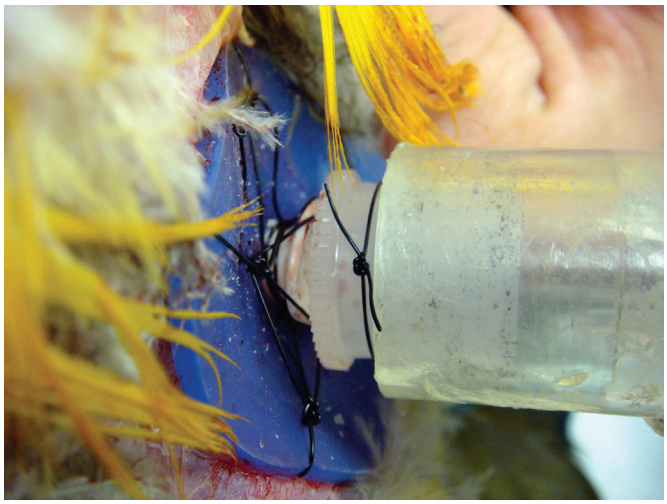


FIGURE 28.8 Air sac cannula placed in a blue-and-gold macaw *(Ara ararauna)*, secured using a retention disk, and connected to an anesthetic machine.

LUNG/AIR SAC SYSTEM

Infectious Diseases

Bacteria

Lower respiratory diseases are common in birds, and infectious causes tend to predominate. Bacterial pneumonia and airsacculitis have been reported with a variety of agents, but gram-negative bacteria seem to predominate. Bacteria commonly reported to cause lower respiratory infections in birds include *Pasteurella multocida*, *Ornithobacterium rhinotracheale* (mainly chickens), *E. coli*, *Klebsiella* spp., *Salmonella* spp., *C. psittaci*, *Riemerella anatipestifer* (mainly waterfowl), *Pseudomonas aeruginosa*, *Staphylococcus* spp., *Streptococcus* spp., *Mycoplasma* spp., and *Mycobacterium* spp.[1,7,14] The importance and pathogenicity of bacterial isolates also depend on the species of bird involved. *C. psittaci* often causes diffuse airsacculitis, whereas *Mycobacterium* spp. generally results in large granulomatous lesions.[1,57–59]

Chlamydiosis is an important disease of birds in general and of Psittaciformes in particular due to its zoonotic potential.

Clinical signs are variable but commonly involve the respiratory system and mild to severe airsacculitis is a frequent necropsy finding. Diagnosis is typically confirmed by PCR from a conjunctivo-choano-cloacal swab or tissue biopsies (see Chapter 27). A strong inflammatory leukogram and hyperglobulinemia are frequently seen on the complete blood count (CBC) and protein electrophoresis, and liver parameters may be elevated as well. The treatment of chlamydiosis relies on using antibiotics with intracellular distribution such as tetracyclines and macrolides.

For further information on chlamydiosis, the reader is referred to Chapter 18 and to *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease.

Viruses

Viral diseases of the lungs are less common than other infectious causes but include orthoavulavirus 1 (formerly paramyxovirus I), avian influenza, infectious bronchitis (chickens), avian polyomavirus (in nestling cockatoos), and canarypox.[1,12,31]

Fungi

Aspergillosis is the predominant respiratory fungal infection in birds. Avian species reported to be more susceptible to respiratory aspergillosis include northern raptorial species (snowy owls, gyrfalcons *[Falco rusticolus]* and hybrids, rough-legged hawks *[Buteo lagopus]*), juvenile red-tailed hawks *(Buteo jamaicensis)*, golden eagles *(Aquila chrysaetos)*, Eurasian goshawks *(Astur gentilis)*, penguins, flamingos, seabirds (e.g., pelicans, auks, gannets, gulls, petrels, loons, etc.), certain parrot species, waterfowl, storks, and mynahs. Large epizootics in wild birds have been reported when gregarious species are exposed simultaneously to a large amount of spores.[60] Wild bird chicks of susceptible species may have a high mortality rate that seems to be associated with nest materials and dynamics.[60] *Aspergillus fumigatus* is the main species isolated, but other *Aspergillus* species can be cultured (e.g., *flavus*, *niger*, *nidulans*, etc.; see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Aspergillosis is not zoonotic and is not contagious, but is an opportunistic environmental pathogen. Any environmental strain may be potentially pathogenic. The pathogenesis seems to rather involve either exposure to a high spore load resulting in acute pulmonary aspergillosis or some degree of immunosuppression leading to chronic pulmonary—air sac aspergillosis. Tracheal and syringeal aspergillosis constitutes a third form of the disease (Figure 28.7). Risk factors that have been associated with an increased susceptibility include lead toxicosis (waterfowls, raptors), smoke inhalation injury, captivity (especially seabirds), stress, steroids, tetracyclines (likely due to immunomodulatory effect of long-term use), neoplasia, circovirus infection, hypovitaminosis A, and others.[60–64] In the authors' experience, an air sac cannula kept for several days frequently leads to focal aspergillosis lesions. Since the air sac system pneumatizes most of the body, aspergillosis lesions may extend to any internal organ by local invasion or distantly by hematogenous dissemination as *Aspergillus* spp. demonstrate angiotropism in tissues. In addition, *Aspergillus* spp. produce a wide variety of mycotoxins, some with immunosuppressive effects such as the gliotoxin, which may participate in the disease progression and pathogenesis.[65] Various experimental avian models have been developed to further elucidate the pathogenesis and progression of the disease.[66–70]

Aspergillosis can be suspected on diagnostic imaging such as radiographs (Figure 28.9 and Figure 27.5) or CT scan (much more sensitive) (Figure 27.5). A strong inflammatory leukogram and globulinemia are also frequently, but not

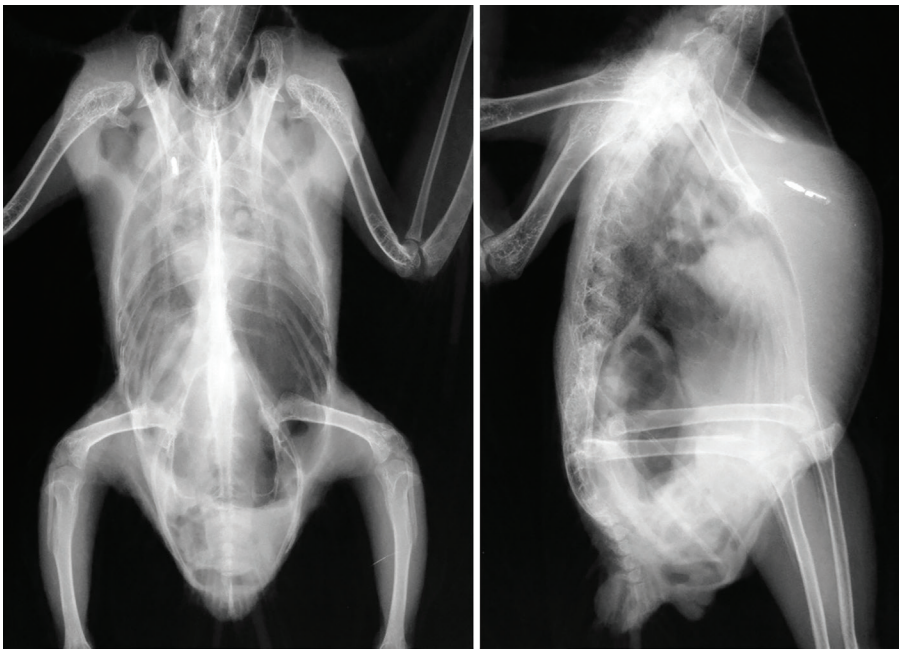


FIGURE 28.9 Whole body radiographs of a grey parrot *(Psittacus erithacus)* with respiratory aspergillosis. On the ventrodorsal view *(left)*, the right part of the air sac system presents some soft tissue opacities, and on the lateral view *(right)*, the air sacs are considerably thickened by aspergillosis lesions, which form characteristic "caverns."



FIGURE 28.10 Close-up view of a focal lesion of aspergillosis on an air sac membrane in a snowy owl *(Bubo scandiacus)*. The air sac is displaying mild thickening and hypervascularization and the small fungal plaque is supplied by a small vessel.

always, present on the CBC and protein electrophoresis. Host systemic response may be significantly influenced not only by the severity of infection but by the location of Aspergillus infection as well. The diagnosis can be confirmed by histopathology, cytology, and/or fungal culture from endoscopic biopsies of lesions (Figure 28.10).[71] Galactomannan assay and protein electrophoresis are not particularly useful in the diagnosis of aspergillosis in the individual bird owing to their low diagnostic accuracy.[72,73] *Aspergillus* serology is also of limited usefulness.[71] Culture or PCR on swabs and lesions without demonstrable histologic lesions may be difficult to interpret due to the ubiquity of the fungus in the environment.[71] Newer fungal biomarkers have also been proposed, but data on diagnostic accuracy on clinical cases are lacking for most of these tests, and they are also mainly restricted to the research laboratory.[71]

The treatment of aspergillosis is complicated, long, and multifaceted. Usually a combination of nebulized, local, and systemic antifungals is used, but antifungal monotherapy has resulted in good outcomes in some cases (see Chapter 8 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).[74] The prognosis should be determined based on radiographic, CT, and/or endoscopic evaluation. The treatment is lengthy with typically 3 to 8 months of oral antifungal medications required. Ideally, the treatment should be based on fungal culture and sensitivity as resistance to azoles such as itraconazole, clotrimazole, and voriconazole has been described,[76–79] but laboratories offering fungal sensitivity are few and minimum inhibitory concentrations of antifungals for fungal isolates may not be readily available.

Available antifungals include the polyenes (e.g., amphotericin B, nystatin, not absorbed orally), which bind ergosterol (fungal wall constituent) and permeabilize fungal cell membrane; the azoles (e.g., ketoconazole, fluconazole, itraconazole, voriconazole, clotrimazole), which inhibit a P450-dependent enzyme of lanosterol 14-α demethylation thereby decreasing ergosterol synthesis; the allylamines (e.g., terbinafine), which inhibit squalene epoxidase, another enzyme involved in ergosterol synthesis; and echinocandins (e.g., caspofungin), which interfere with glucan synthesis (see Chapter 8).[80,81] Pharmacokinetic and pharmacodynamics studies have been performed on a few species for itraconazole, fluconazole, voriconazole, and terbinafine.[74,81–91] Polyenes are not absorbed orally. Using a combination of antifungals with different mechanisms of action, and different routes of delivery, somewhat dictated by disease location can be beneficial (see Chapter 8). Drug treatment choices may have a synergistic effect and may reduce fungal resistance. Voriconazole is the newest antifungal used in birds and seems particularly effective. It has been shown to be effective in treating experimental aspergillosis in pigeons and spontaneous aspergillosis in falcons and parrots.[75,82,85] For more information on antifungals, the reader is invited to consult Chapter 8 as well as *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease.

The author typically favors an aggressive approach in moderate to advanced cases using a combination of antifungals with potential synergistic effects consisting of 1 to 3 days of intravenous (IV) amphotericin B or voriconazole, combined with nebulized amphotericin B and oral voriconazole, occasionally combined with terbinafine. Endosurgical debridement has also been employed for larger granulomas in birds,[93] followed by endoscopic-guided local instillation across fungal plaques or intragranuloma injections of antifungals, depending on clinical details and location of lesion(s). Intratracheal administration of (IV solutions of) amphotericin B or voriconazole may also be performed. The use of multiple routes and methods of delivery, as well as the use of antifungals with different mechanisms of action, may enhance effectiveness and is relatively well tolerated by most patients. Also, the slow onset of some triazoles may make early combination therapy advisable. Various results have been obtained in humans where concentration-dependent synergism or antagonism was observed between antifungals.[94] Synergistic action between triazoles and amphotericin B or triazoles and terbinafine has been observed on human *Aspergillus* isolates in vitro.[95] However, no in vivo research has been done on antifungal synergisms and antagonism in birds, so combination therapy is largely empirical.

Parasites

Several lower respiratory parasitic diseases are of significance in birds. *Sarcocystis falcatula* is a sporozoan parasite, which has an indirect life cycle with the Virginia opossum as the final host and cockroaches as potential mechanical vectors (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). It is associated with an acute pulmonary form in Old World species such as cockatoos rather than the classic muscular form.[96,97] Pulmonary lesions are also common with disseminated visceral coccidiosis due to *Eimeria* spp. in cranes.[98] *Toxoplasma gondii* is a protozoan parasite

whose final host is the cat, which can affect a wide range of avian species. In birds, pulmonary lesions are common with toxoplasmosis.

Atoxoplasmosis is caused by the coccidian parasite *Isospora serini* and affects diverse passerine birds from various families (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Canaries and finches are commonly reported to have the disease. Pulmonary lesions are reported, but hepatic and splenic lesions predominate. The treatment of atoxoplasmosis can be challenging, and treatment response is usually highly variable. Drugs that have been used to treat systemic coccidiosis in passerines include mainly toltrazuril and sulfachloropyridazine (see Chapter 9 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).[99,100]

The air sac mites, *S. tracheacolum*, live in the air sacs and lungs but are most often pathogenic in the upper airways (see earlier). Various filarioid nematodes have been identified in the lower respiratory system of birds including the genera *Paronchocerca*, *Splendidofilaria*, *Chandlerella*, *Serratospiculum*, and *Diplotriaena*.[101,102] Most infections do not lead to clinical disease. *Serratospiculum seurati* is a common parasite of air sacs in saker *(Falco cherrug)* and other falcons in the Middle East. Beetle species are the intermediate hosts. Other species of *Serratospiculum* have been reported in birds of prey from various geographical locations. *Serratospiculum* and *Diplotriaena* do not produce microfilariae but rather eggs that develop in insect hosts. These parasites may produce airsacculitis and promote lower respiratory infections. Several genera of air sac flukes (trematodes) have been reported to parasitize wild and captive zoo birds and are associated with mollusks intermediate hosts.[24,103] Treatment of respiratory parasites mainly consists of administering parenteral antiparasitic medications (see Chapter 9). Intramuscular ivermectin is most commonly used for helminthiasis (e.g., falcon serratospiculosis, tracheal worms).[21] Fenbendazole is also effective against tracheal worms. Endoscopic removal of tracheal worms may be indicated if tracheal obstruction is an issue. Endoscopic removal of nematodes from air sacs may also be performed but is rarely necessary if an appropriate ivermectin dose is used. Praziquantel has been used with variable results in treating air sac flukes.[103] Preventative measures should also be implemented by reducing access to intermediate host such as limiting access to the ground and having appropriate aviary design.[21]

Noninfectious Diseases

Birds are very sensitive to smoke inhalation toxicosis and airborne toxins because of their large and efficient pulmonary exchange surface (see Chapter 26). Toxic fumes frequently include PTFE (polytetrafluoroethylene [Teflon], Fluon, Hostaflon, and Polyflon) from overheated cookware, cigarette smoke, household aerosols (house perfumes, candles, disinfectants), carbon monoxide, and ammonia (chickens). Signs are most often acute, especially in PTFE inhalation and other toxic fumes, and severe pulmonary hemorrhage and congestion typically ensue (Figure 28.11). Respiratory signs may develop as long as 3 days after toxin exposure. Oxygen therapy is the mainstay of treatment, and many of these patients may benefit from preemptive treatment with antibiotics and antifungals.

Several other noninfectious diseases of clinical significance have been reported in captive birds. Aspiration pneumonia may be encountered in baby birds (chronic poor doer) and hospitalized birds being gavage fed. The "conure bleeding syndrome" frequently manifests as bleeding in the respiratory tract and hemoptysis. Hemoptysis and pulmonary hemorrhage also occur in birds of prey with secondary rodenticide poisoning.[104] Advanced atherosclerosis and cardiac diseases may cause respiratory signs. Chronic pulmonary interstitial fibrosis has been reported in Amazon parrots, is frequently seen in the Netherlands, and has been associated with right heart failure in some birds.[105,106]

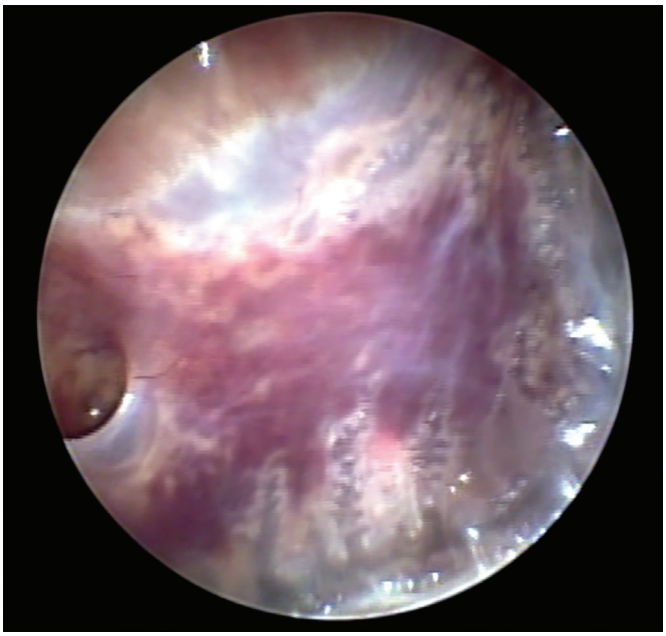


FIGURE 28.11 Pulmonary congestion diagnosed in a grey parrot *(Psittacus erithacus)* with smoke inhalation injury. The tip of the endoscope is in the left caudal thoracic air sac.

A chronic obstructive respiratory disease is also seen in blue-and-gold macaws (*Ara ararauna*) (and in several green-winged macaws by the author) housed with feather powder–producing species such as cockatoos, cockatiels, and grey parrots.[12,107] This syndrome is characterized by polycythemia, pulmonary congestion, atrial smooth muscle hypertrophy, and atrial loss. Severe, intermittent inspiratory dyspnea with open-mouth breathing is often seen. Lung biopsies can be obtained endoscopically from the thoracic air sacs or through a surgical intercostal approach (if lesions are not amenable to endoscopic biopsies) to confirm the diagnosis and assess its severity (see Chapter 29). The condition is typically treated with short- or long-term bronchodilators, nonsteroidal antiinflammatory drugs, and strict environmental management to reduce further antigenic exposures (see General Therapeutic Principles). The author has frequently used nebulized terbutaline or albuterol for acute treatment in combination with oxygen therapy and oral theophylline/salbutamol for chronic treatment. Increasing the relative humidity of ambient air seems to be beneficial for tropical bird species. Short-term low-dose corticosteroids may also help to decrease pulmonary inflammation initially (prednisone 0.1–0.2 mg/kg PO q12–24h), though caution is warranted when using these drugs due to their immunosuppressive effects. It seems that advanced disease, when chronic

histopathologic changes in the lung parenchyma are too severe, does not continue to respond to bronchodilators. Consequently, and preventatively, macaws should not be kept in an area with dander-producing parrot species.

Pneumoconiosis (synonyms: anthracosis, silicosis) results from the accumulation of foreign materials (dust) and dust-laden macrophages in the lungs and has been reported in various species.[22] Pulmonary fat embolism has been reported in an osprey *(Pandion haliaetus)* and yolk embolism in pet psittacine birds.[22,108] Air sac cannulation for several days often causes moderate to marked air sac inflammatory reaction. Subcutaneous emphysema around the body may be caused by an air sac trauma such as resulting from a surgical procedure (e.g., coeliotomy, air sacculotomy), endoscopy, abdominal wall herniation, or fracture of a pneumatized bone.

Neoplasms described in the lower respiratory tract include pulmonary adenocarcinoma, air sac adenocarcinoma, and metastasis of malignant tumors. Pulmonary/bronchogenic carcinomas have been reported in multiple bird species and often manifest as pelvic limb ataxia/paresis due to local invasion of the tumor into the spine.[109–113] These tumors frequently invade the humeral air sac diverticulum. Air sac cystadenocarcinoma has mainly been reported in cockatoos, but a case in a grey parrot has also been documented. These tumors usually arise from the proximal humeral and axillary air sac diverticulum[114–117] Undifferentiated neoplasms of the lungs and air sacs have been recognized in cockatiels and seem to be most often diagnosed in the thoracic inlet occasionally compressing the trachea (Figure 28.12).[22,31] Marek disease may cause pulmonary tumors in chickens.[1] Treatment is limited for respiratory neoplasia, but surgery, chemotherapy, and/or radiation therapy may be attempted if diagnosed early on (see Chapter 29 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 3, Neoplastic Diseases in Avian Species).

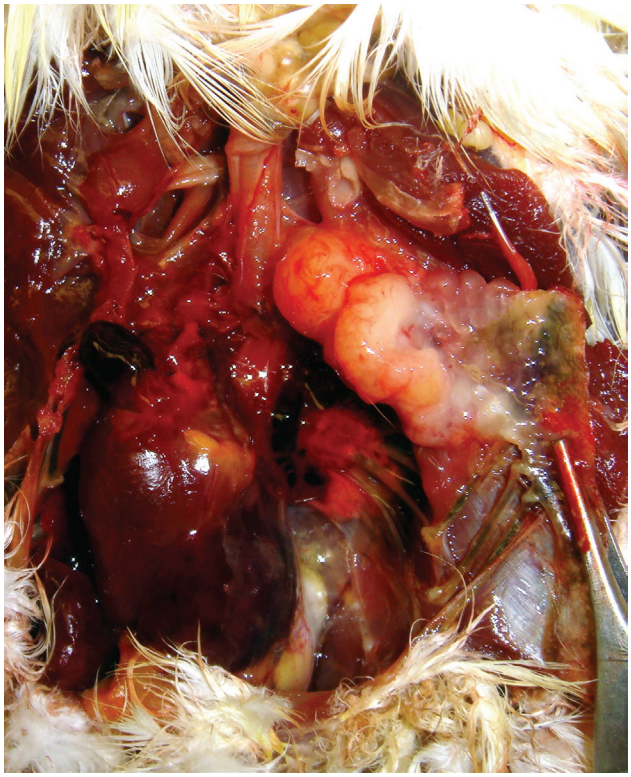


FIGURE 28.12 Soft tissue sarcoma in the interclavicular air sac of a cockatiel *(Nymphicus hollandicus)* that presented with open-mouth breathing and regurgitation.

EXTRA-RESPIRATORY DISEASES: AIRFLOW OCCLUSION AND AIR SAC COMPRESSION

Due to the extensive nature of the air sac system in birds in their abdominal and thoracic regions, any extra-respiratory disease resulting in reduction of air sac volume or occlusion of major airways may result in respiratory clinical signs.

In the thoracic inlet, masses of nonrespiratory origin may occlude the trachea, syrinx, and interclavicular air sac leading to inspiratory dyspnea, aphonia, or voice changes. These masses include thyroid masses (goiter, especially in budgerigars, thyroid neoplasia), bony callus from a coracoid fracture, aneurysms, large granulomas, and neoplasia such as undifferentiated sarcoma, hemangiosarcoma, thymoma, and other tumors.[118–121] Cardiomegaly does not typically induce coughing in birds as it does in mammals.

A significant reduction in air sac space may occur with fluid accumulation in the hepatic and intestinal peritoneal cavities of birds such as ascites resulting from cardiac, hepatic, or neoplastic diseases and egg yolk peritonitis. Severe respiratory complications may arise if the fluid/yolk gains access to the inside of the respiratory system. Coelomic fluid should be drained by centesis until dyspnea has improved. Additionally, diuretics can be used to promote forced diuresis (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 6, Cardiology).

Space-occupying masses are also associated with impairment of abdominal ventilation and are encountered with large tumors (e.g., in budgerigars), granulomas, eggs, and organomegaly. Obstruction of air sac ostia is rare but may interfere with ventilation. An ostrich *(Struthio camelus)* was reported with intestinal entrapment in a right pulmonary ostium.[122] Finally, obesity with intraperitoneal fat accumulation may impair normal ventilation and induce dyspnea, especially after exercise.

GENERAL THERAPEUTIC PRINCIPLES

Principles of Oxygen Therapy

The goal of oxygen supplementation is to increase the oxygen concentration of inspired air (fraction of inspired oxygen [FiO_2]), to improve blood oxygenation, and to increase tissue delivery of oxygen. General hypoxia may be due to anoxic hypoxia (low FiO_2, hypoventilation, diffusion impairment, ventilation/perfusion mismatching), anemic hypoxia (anemia, methemoglobinemia, carbon monoxide toxicosis), stagnant hypoxia (low blood flow due to cardiac failure or hemorrhage), and histiocytic hypoxia (cyanide poisoning).[123] Common indications of oxygen therapy include severe anemia, hemodynamic compromise, and hypoxemia (decreased blood oxygen concentration) due to pulmonary and obstructive airway diseases.[124]

Oxygen therapy is typically implemented using an oxygen cage in birds because of its low stress and noninvasiveness.

The authors strongly encourage veterinarians to measure the FiO_2 that can be achieved in their oxygen cages using an oxygen sensor to verify their performance. Some incubators have doors within doors, which allow manipulation of the animal without massive loss of oxygen from the cage. An FiO_2 of 25% to 45% may be reached using flow-by oxygen and 35% to 60% using facemasks.[123,124] It is ideal to monitor the SpO_2 or PaO_2 during oxygen supplementation, but this is rarely practical in birds.

Response to therapy should also be used for monitoring and titrating the oxygen flow. The response to oxygen therapy may be poor depending on the disease process(es) present. For instance, a poor to fair response may be seen in diseases producing a low ventilation/perfusion mismatch (perfusion predominates with decreased supply of oxygen to the exchange surface) such as pulmonary edema, pneumonia, asthma, pulmonary neoplasia, pulmonary fibrosis, and atelectasis. In extreme cases of these diseases (severe pneumonia), oxygen therapy may not be efficacious because blood essentially makes no contact with ventilated lungs. Response is more commonly good in diseases with a high ventilation/perfusion mismatch (low perfusion in normally ventilated lungs) such as pulmonary thromboembolism.[123] Improving cardiac function and fluid resuscitation are best to correct stagnant hypoxemia. While the degree of response to oxygen therapy may be variable, regardless of the underlying conditions, any patient with acute respiratory distress and signs of hypoxia (cyanosis, dyspnea, tachypnea, open-mouth breathing) may benefit from supplemental oxygen. Oxygen therapy seems extremely beneficial to birds, which have higher metabolic demand for oxygen and may benefit more from increased FiO_2 due to their more efficient gas exchange surface. Sedation with midazolam in dyspneic birds may also reduce anxiety thereby resulting in better ventilation.

The most common complication associated with oxygen therapy is oxygen toxicosis because oxygen is a potent oxidizing agent. The degree of toxicity is related to the level of oxygen and duration of exposure. The lung is the most vulnerable organ to oxygen radicals. General guidelines recommend not to supplement oxygen at 60% to 100% for more than 24 to 48 hours in mammals.[123,124] Depletion of endogenous antioxidant levels may also promote oxygen toxicosis at lower oxygen percentages. Oxygen toxicosis has been described in birds. A study demonstrated oxygen toxicity in canaries and budgerigars at 68% to 100% after 3 to 8 days.[125] In another experiment, budgerigars were found to have pulmonary lesions 3 hours after exposure to 100% oxygen.[126]

Principles of Aerosol Therapy

The goals of aerosol therapy are to deliver airborne therapeutics directly to diseased respiratory tissues and to moisten airways in order to increase clearance of exudates and necrotic debris.[7] Local therapeutic delivery may be indicated when vascularization is decreased in diseased tissues (e.g., inflammatory and infected exudates in airways) or in low-perfused respiratory areas such as the air sacs (see Chapter 5). Nebulization allows one to reach high local concentration of therapeutics, maximizing its efficacy while minimizing systemic absorption, reducing potential for toxicosis, and reducing drug biotransformation. Humidification of the mucociliary escalator may also improve its efficiency.

Depending on particle sizes and airway diameters, aerosolized therapeutics may only reach the upper respiratory system, the larger airways, or lungs and air sacs. Birds' airways have extremely small diameters with parabronchi diameters ranging from 0.5 mm (e.g., hummingbirds) to 2 mm (e.g., chicken, penguins) and air capillaries ranging from 3 μm (e.g., passerines) to 10 μm (e.g., swans, coots; see also Chapter 26).[127] In comparison, mammalian alveoli have an approximate diameter of 30 to 150 μm (the smallest is in mice).[128] Consequently, birds' airways require smaller nebulized particle sizes and particle diameters of 0.5 to 3 μm are generally recommended.[7,129] During endoscopy, the author has seen amphotericin coating caudal air sacs of birds that have been nebulized, suggesting that nebulized drugs reach the caudal thoracic and abdominal air sacs also in diseased birds.

Aerosol therapy includes humidification, vaporization, and nebulization. The first two methods produce particles that are too large to reach the lower respiratory system but may be adequate for upper respiratory or tracheal diseases. The latter produces particles small enough to be deposited in smaller pulmonary airways. Nebulization can be achieved with a variety of devices using different technology such as air-jet nebulizers and ultrasonic nebulizers (including vibrating mesh technology).[130] Air-jet nebulizers are noisier and heavier but are less expensive and have a greater ability to nebulize viscous liquid than ultrasonic nebulizers (Figure 28.13). Pressurized metered-dose inhaler aerosols such as classically available for albuterol are a type of air-jet nebulizer and deliver 1- to 2-μm particles.[131] Drugs used for nebulization should ideally be hydrosoluble, of low viscosity, and display adequate nebulization characteristics. Drugs that are commonly nebulized include 0.9% saline, antibacterials, antifungals (when from a hydrosoluble formulation), antiparasitics, bronchodilators, and mucolytic agents (Table 28.1). Nebulized drugs may induce bronchospasm and patients should be assessed before and after administration (e.g., *N*-acetylcysteine [NAC]).

Nebulization typically lasts 15 to 30 minutes and is performed once to twice daily. However, a study on nebulized

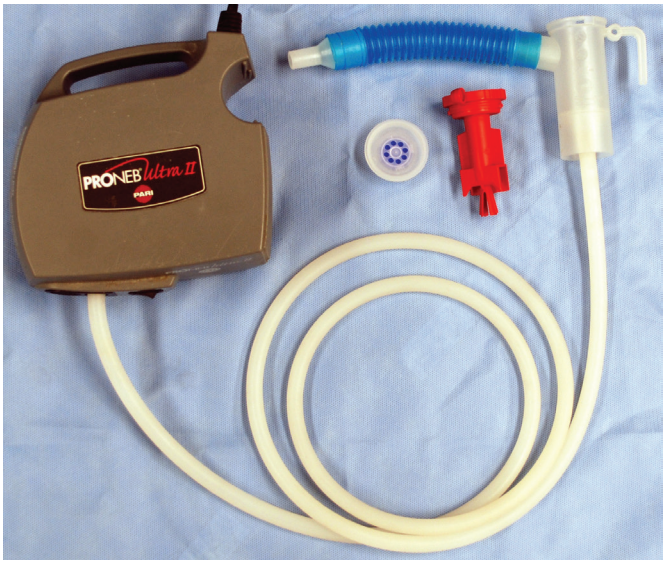


FIGURE 28.13 Air-jet nebulizer commonly used in avian medicine.

TABLE 28.1

Drugs Commonly Used for Nebulization in Birds

Drug	Dose
ANTIBACTERIALS	
Amikacin	5–6 mg/mL sterile water/saline
Gentamicin	3–6 mg/mL sterile water/saline
Cefotaxime	10 mg/mL saline
Doxycycline	13 mg/mL saline
Tobramycin	1 mg/mL saline
Enrofloxacin	10 mg/mL saline
Piperacillin	10 mg/mL saline
ANTIFUNGALS	
Amphotericin B	0.1–5 mg/mL sterile water
Enilconazole	10 mg/mL sterile water/saline
Clotrimazole	10 mg/mL in propylene glycol
Terbinafine	1 mg/mL sterile water
Voriconazole	10 mg/mL saline
BRONCHODILATORS	
Aminophylline	3 mg/mL sterile water/saline
Terbutaline	0.01 mg/kg in 5–10 mL saline
MUCOLYTIC	
N-acetylcysteine	22 mg/mL sterile water

fluorescein-labeled propylene glycol and saline in pigeons found that only mild staining of the air sacs was observed at 30 minutes, with much higher intensities achieved at 4 hours.[132] These findings suggest that nebulization may have to be performed for longer than previously thought. In birds, the respiratory surface and functional efficiency of their lungs may lead to greater systemic absorption of the nebulized drugs than in other species. This may be advantageous in certain circumstances to obtain drug plasma therapeutic levels without having to restrain the bird. For instance, the pharmacokinetics of the following nebulized drugs have been performed in birds: terbinafine, voriconazole, oxytetracycline, and ceftriaxone.[81,133–136] However, plasma concentrations achieved are usually low and of short duration.

Principles of Respiratory Pharmacology

Other than antimicrobials and antiinflammatories, the pharmacologic options for treatment of respiratory tract pathologies include expectorants, mucolytics, decongestants, bronchodilators, cough suppressors, and respiratory stimulants.[137,138] Most of these drugs have been studied in mammals, and their effects may be variable in birds due to different pharmacokinetic properties, different mechanisms of respiratory diseases, and different physiology.

The main mucolytic used in veterinary medicine is NAC. It breaks the disulfide bonds within mucus molecules, which decreases the viscosity. NAC is mainly used for upper respiratory and tracheal diseases. Another mucolytic drug is bromhexine. Expectorants include potassium iodide and guaifenesin. They increase mucus production, which aids in airway secretion clearance. The use and efficacy of expectorants and mucolytics is debatable. Moreover, NAC is well known to induce bronchospasm and is recommended to be combined with a bronchodilator, if used.[137]

Decongestants include sympathomimetic drugs such as ephedrine and phenylephrine, which constrict dilated blood vessels within the nasal cavity, thereby reducing swelling and congestion of the upper respiratory mucus membranes and allowing air to pass more freely. H1-antihistamines such as diphenhydramine or hydroxyzine, which have a synergistic effect and can help to relieve symptoms that occur in response to allergens, such as sneezing and nasal discharge, may also be considered in birds with sinusitis and rhinitis.

Birds have bronchial and parabronchial smooth muscles, so bronchodilators may be beneficial in decreasing resistance to airflow. However, bronchodilators should be used appropriately as they can increase the ventilation-perfusion mismatch in some diseases, which results in worsening of hypoxia. Different classes of bronchodilators with different mechanisms of action are available. Parasympatholytic drugs such as atropine and glycopyrrolate induce bronchodilation by competition with acetylcholine on muscarinic receptors in the airways. Beta-2-sympathomimetic (=beta adrenergic) drugs such as albuterol (=salbutamol) and terbutaline are the most effective bronchodilators and induce bronchodilation by agonist effects on beta-2 adrenergic receptors in the airways. Side effects are related to residual beta-1 effects on the cardiovascular system potentially leading to tachycardia and arrhythmias. Methylxanthines such as aminophylline and theophylline are phosphodiesterase inhibitors that induce bronchodilation and have local antiinflammatory effects. Adverse effects seen with methylxanthines include nausea, cardiac arrhythmias, and neurological signs.

Doxapram is a centrally acting respiratory stimulant, but its use is questionable in most respiratory diseases.

Antibacterials, antiparasitics, and antifungals are used when such microbial infection is diagnosed at doses like other systems. For more detailed information, the reader is referred to Section 3: Therapeutic Intervention and Appendix 1.

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CHAPTER 29

Surgery of the Respiratory Tract

David Sanchez-Migallon Guzman • Brian L. Speer • M. Scott Echols

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SURGICAL PRINCIPLES, INSTRUMENTATION, AND MATERIALS

Magnification, focused light, and microsurgical instrumentation are important components of many surgical procedures of the respiratory system, including sinusotomy and tracheal procedures. Magnification makes hand tremors more apparent to the surgeon and necessitates slow, deliberate motions. It can result in a slower surgical pace, requiring appropriate anesthesia protocols and patient monitoring for the duration of the procedure.[1] Focused light is sometimes essential to seeing anatomic details in dark recesses common within body cavities. The basic microsurgical pack should contain a long jeweler's forceps that has a fine delicate tip but flat handles, round handled microsurgical forceps, Castroviejo needle holder that is at least 5.25 inches (13.3 cm) with fine jaws and rounded handles, and microsurgical scissors that are spring loaded and at least 7 inches (17.8 cm) or similar size and have rounded handles.[1] Sterile cotton-tipped applicators should be available for focal tissue manipulation and point hemostasis.[1] Hemostatic clips are also very useful and come in a variety of sizes. Right-angled appliers are available for special applications in deep cavities.[1] Heiss blunt retractors, Alm retractors, or ring retractors are suitable for use in varied species and procedures (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 21, Surgery).

Halsted's principles of surgery should be followed when performing surgery in any species or anatomic location. These principles include (1) gentle handling of tissue, (2) meticulous hemostasis, (3) preservation of blood supply, (4) strict aseptic technique, (5) minimum tension on tissues, (6) accurate tissue apposition, and (7) obliteration of dead space (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 21, Surgery).

PATIENT PREPARATION

Birds have a very thin epidermis that is diffusely supplied by capillaries in the dermis, unlike what is seen in mammals. There is little subcutaneous tissue, and the dermis is generally firmly attached to the underlying muscle fascia, although there is more free space and relative mobility in the neck of most birds as opposed to other areas of the body.[1] Most blood vessels are comparatively less protected by surrounding tissues, leaving more potential for hemorrhage as compared to mammals.[1] This concern is particularly of merit when performing surgery in the cervical regions of the upper respiratory tract, where blood loss can lead to associated complications into the distensible spaces beneath cervical skin or even to blood clot occlusions of the trachea. Body contour feathers are easily plucked in the direction of their growth, once the patient is anesthetized.[1] Some bird species may have particularly thin skin, predisposing them to iatrogenic tears while feathers are being plucked, including many dove species. The skin is prepared with routine cleansing with chlorhexidine alternating with sterile saline, or similar disinfection protocols. Draping the patient quickly with the use of clear, transparent plastic drapes helps to retain heat better than using cloth drapes and also allows respiration to be monitored during the procedure.[2] Supplemental heat (e.g., warm water bottles, circulating warm water blankets, heat lamps, and forced warm air) should be provided to the patient to maintain normothermia and prevent heat loss.[2]

SURGERY OF THE UPPER RESPIRATORY SYSTEM

Rhinotomy and Rhinolith Removal

Indications

Removal or biopsy of masses present in the nares, due to the presence of rhinolithiasis, foreign bodies, infections, or neoplasms. Malnutrition (e.g., hypovitaminosis A) can lead to progressive development of rhinoliths, and secondary infections can often occur.

Surgical technique

Rhinoliths may often be gently removed using saline to soak and soften them beforehand, if needed. Topical local anesthetics can be applied, following which a stainless steel aural curette can be used to gently remove the material.[3,4] Some rhinoliths can be very firm, dry, and somewhat impenetrable to topical softening attempts (Figure 29.1). Some larger rhinoliths may need to be sectioned or otherwise reduced in situ to facilitate removal. Significant distortion of the naris and the operculum can occur, and computed tomography (CT) may be indicated for some patients prior to the contemplated procedure (see Chapter 2 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 14, Advances in Diagnostic Imaging). Fistulas into the maxillary and adjacent diverticulae of the infraorbital



FIGURE 29.1 (A) Large rhinolith in the left nare of a grey parrot *(Psittacus erithacus)*. The size and firmness of this rhinolith presented mechanical challenges for the application of topical moistening agents to offer much immediate benefit. The patient has been sedated with midazolam and butorphanol, in preparation for removal using a stainless steel aural curette. (B) The left nare of the same grey parrot, following complete removal of the rhinolith. Focal areas of bleeding are noted where a local block with lidocaine was injected through the caudal aspect of the nare, prior to its removal. Mild bleeding is present, which was easily controlled with topical pressure using cotton-tipped applicators. Aerobic bacterial cultures from deep in this cavity produced a pure isolation of *Pseudomonas aeruginosa*. (C) The rhinolith removed. The keratinaceous material on the right was the most superficial portion initially removed, and the material on the left was the remaining portion removed. (D) A different grey parrot that had a similar sized rhinolith removed from its right nares 6 months earlier. The nasal opening remains distorted but has reduced by approximately 50% of its original distorted diameter. Some degree of permanent disfigurement is common following the removal of large rhinoliths as demonstrated in these figures.

sinuses can occur, as well as osteomyelitis. General anesthesia may be required for more involved procedures. In some cases, an incision may be required to increase access to the nasal cavity, in which case radiosurgery or laser should be available to achieve adequate hemostasis. Samples should be collected for cytology and culture to guide the treatment plan (see Chapter 27). Postoperative treatment might require nasal flushing, topical and/or systemic antimicrobial treatment, as well as pain management (see Chapter 28).

Potential complications

Hemorrhage, fistula into the infraorbital sinus, and permanent distortion of the nares.

Choanal Atresia Repair

Indications

Confirmed choanal atresia or obstruction between the nasal cavities and the nasopharynx. Choanal atresia typically is a congenital problem and has been described in the grey parrot (Figure 29.2)[7] and umbrella cockatoo *(Cacatua alba)*.[5] The detailed anatomic deformities associated with atresia are incompletely described in birds but may represent either or both bony and soft tissue developmental abnormalities.[6]

Surgical technique

A technique for surgical correction has been described in the grey parrot (Figure 29.2).[7] By hand drilling a 7/64-1/8 Steinmann pin from each naris, through the nasal conchae and into the rostral choana, a functional communicating channel is created. Following this, a tomcat urinary catheter is placed, into which a single 8-French red rubber catheter is nested to enable passage of the softer urinary catheter through the nares and out the choana. The procedure is replicated on the opposite side, with the cut end of the urinary catheter similarly inserted and pulled through, ultimately leaving the central loop of the catheter bridging the cere. The ends of the urinary catheter are then tied behind the head. The catheter is left in place for 4 to 6 weeks to allow the formation of permanent communication between the nasal cavity and rostral choanal slit (see Chapter 41). During this period, the catheter may be replaced with larger sized tubing to help increase the size of the hole gradually. Nasal flushing should be performed for 7 to 10 days after catheter removal to prevent the buildup of mucus and development of occlusion of the newly created communicating pathway.

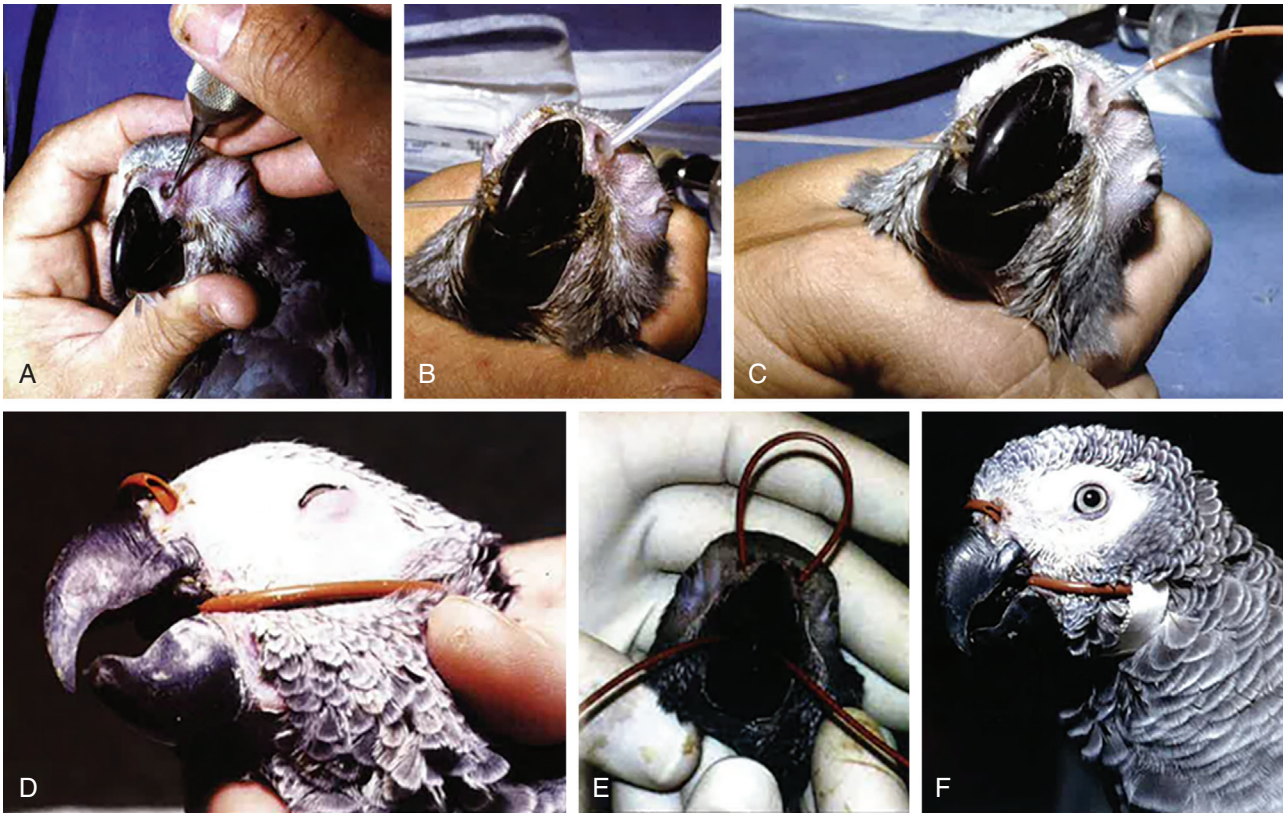


FIGURE 29.2 (A) Placement of Steinman pin through the nares and out of the choana in a grey parrot *(Psittacus erithacus)*. (B) A tomcat urinary catheter, which is firmer than the rubber urinary catheter, is then inserted through the opening created with the Steinman pin. (C) The red rubber urinary catheter is nested into the end of the tomcat catheter, and the entire structure is pulled out of the choana, functionally leaving the red rubber catheter in the newly created passageway. (D) The same procedure is duplicated on the opposite side, allowing the red rubber catheter to be looped over the cere, with both ends equally exiting the choana. To prevent further mucus accumulation in the nares and conchae, note the fenestrated opening that has been cut into the exiting tube from both nares. (E) A rostral to caudal view of the urinary catheter placement, showing the looped placement over the cere. (F) The ends of the catheter are tied behind the head and left in place for 4 to 6 weeks. Following catheter removal, nasal flushes should be regularly performed to minimize the further accumulation of mucus and debris and occlusion of the newly created pathway. (Photos courtesy Don J Harris.)

Potential complications

Damage to the palatine bone, hemorrhage, reocclusion of the drainage pathway, and (secondary) infection.

Sinusotomy

Indications

Known or suspected infectious or neoplastic disorders in the infraorbital sinus or associated regions.[8] This sinus has been referred to as the "infraorbital sinus," "subocular sac," "orbital sinus," "maxillary sinus," and several other terms. As the infraorbital sinus is located in the antorbital fossa, the term "antorbital sinus" has also been proposed.[9] For the purposes of uniformity, this sinus is referred to as the infraorbital sinus in this document. In addition to their identification, surgical access can enable complete or partial excision of lesions within the sinus and in the regions adjacent to the sinus. Infraorbital sinusotomy can also provide access to the maxillary process of the palatine, jugal, pterygoid, and quadrate bones.[5] In some instances, an infraorbital sinusotomy may be used to facilitate access to the palatine bone when attempting to reduce palatine luxation (see Chapter 31).

Surgical technique

To approach the body of the infraorbital sinus, in most psittacine species, retract the maxillary beak (closing) while the mandible is extended (opening or lowered; Figure 29.3). This functionally enlarges the sinus space and enables greater access within. In the grey parrot, due to the larger relative size of the ethmomandibularis muscle, the upper beak should be elevated (protracted) to facilitate best exposure to the infraorbital sinus. Incise into the infraorbital sinus space ventral to the jugal arch. The infraorbital sinus space can be visually inspected, and magnification loupes with a focal light source, operating microscopy or endoscopy can be used in selected cases for deeper visualization, if indicated. The jugal arch may be incised and reflected to improve exposure, if needed. Reanastomosis of the jugal arch can be accomplished using a monofilament suture placed through predrilled holes in the bone. Every attempt to preserve the encountered muscles should be made. Closure of the surgical site is accomplished in two separate layers: the lateral wall of the infraorbital sinus and the overlying skin.

Different surgical methods have been described in nonpsittacine species to address sinus-associated problems (Figure 29.4). Infection, extending from the nasal passageways and resulting in mucus accumulation and necrosis of the ventral air pouch, was described in a marabou stork *(Leptoptilos crumenifer)*.[10] Surgical removal of the necrotic lower portions of the pouch, placement of a Penrose drain to facilitate further drainage, and targeted antibacterial treatments for the

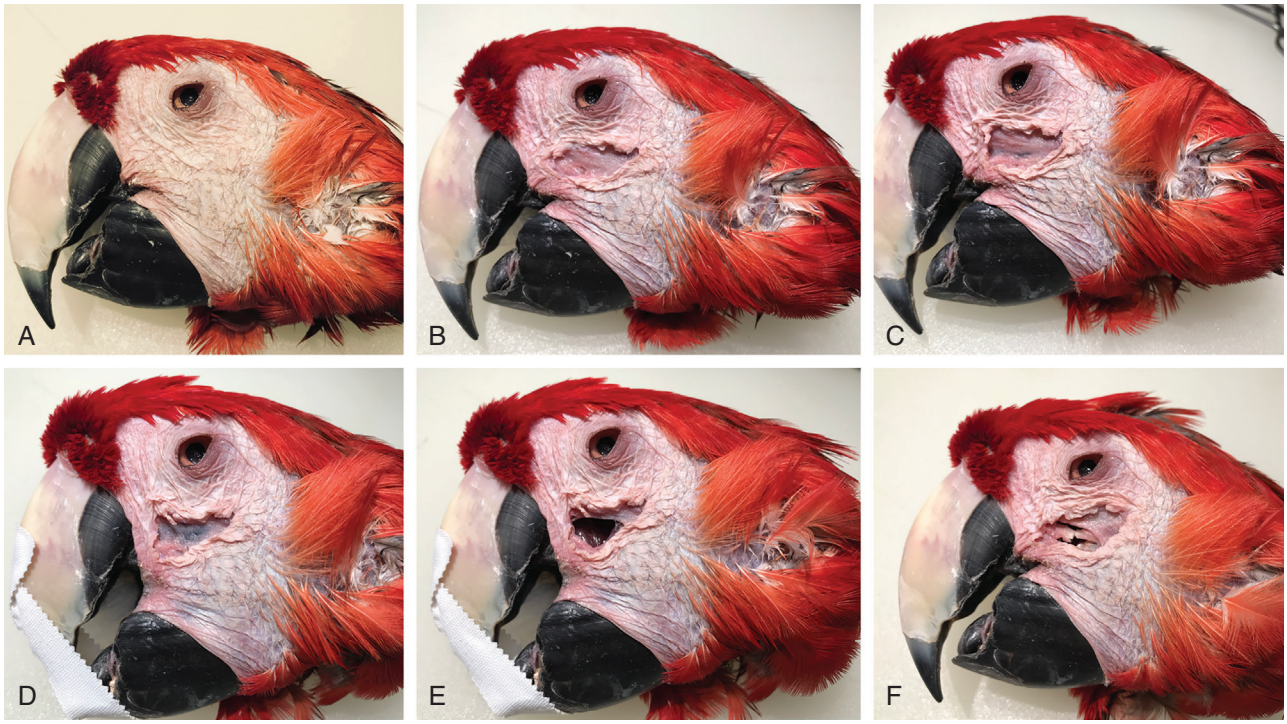


FIGURE 29.3 (A) Cadaveric head of a scarlet macaw *(Ara macao)*. Prior to making a skin incision, the jugal arch is palpated, and the incision may be made longitudinally to the jugal, ventral to the bone, or on some occasions, dorsally. (B) Skin incision has been made, and the immediate subcutaneous space is exposed. (C) Using magnified sharp-sharp dissection, the semitransparent outer wall of the infraorbital sinus is exposed. (D) By retracting the maxillary beak and placing its rostral tip inside the mandibular beak, the functional access into the infraorbital sinus is greatly increased ventral to the jugal. (E) Sinusotomy into the infraorbital sinus space, exposing the ethmomandibularis in the rostral portion of the sinus space. (F) Transection of the jugal arch, which can then be reflected or retracted to facilitate increased exposure, particularly for access to the dorsal and deep sinus spaces.

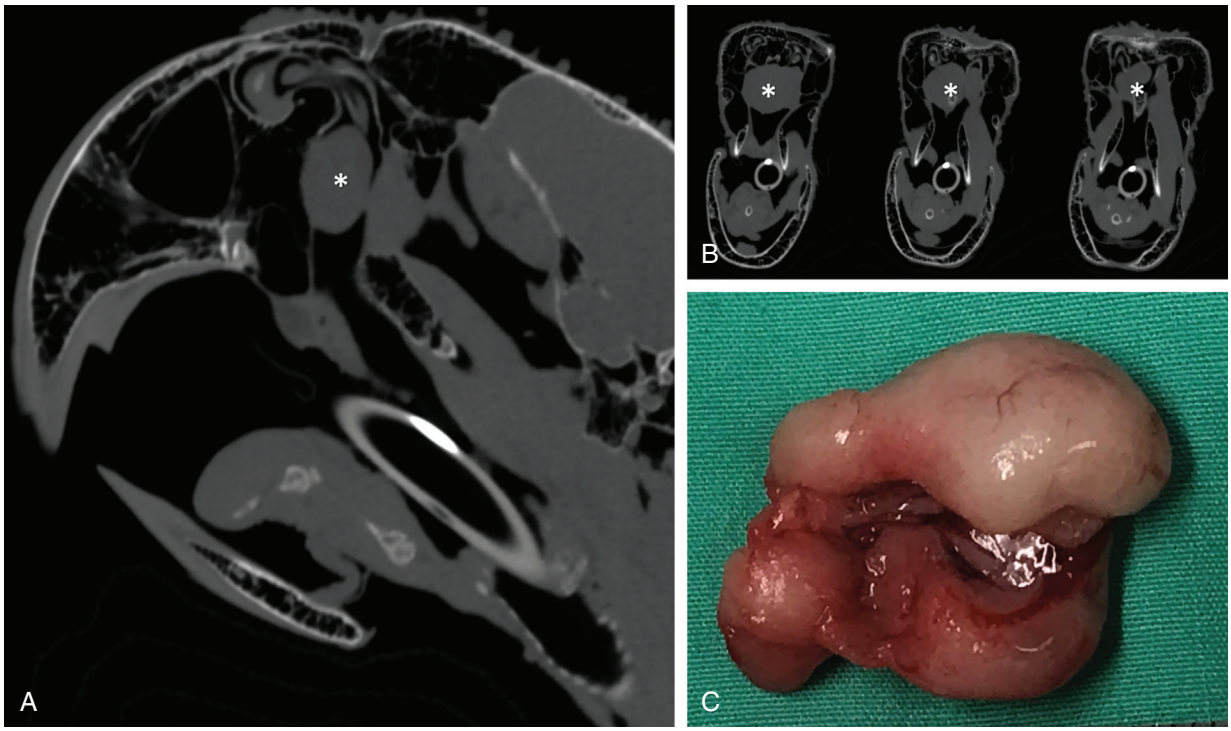


FIGURE 29.4 (A) Sagittal computed tomographic view of the adult blue-and-gold macaw *(Ara ararauna)* with a large, well-defined mass in the central and caudal nasal passageways bilaterally at their communication with the rostral choanal region, medial to the infraorbital sinus. Choanal endoscopic evaluation of this area confirmed the presence of this mass-effect; however, diagnostic full-thickness endo biopsies were unable to be obtained *(asterisk)*. (B) Transverse views of the same computed tomographic study at the level of the mass *(asterisks)*. (C) The right jugal was transected and reflected to facilitate entrance to this region medial to the infraorbital sinus via sinusotomy, and the fibromatous polyp, measuring 2.5 × 2 cm in size, was successfully removed.

Aeromonas and *Proteus* isolates lead to successful resolution of the problem 2 months postoperatively.

Potential complications

Subcutaneous air leakage from the sinus incision or jugal bone, (secondary) infection, nerve damage, hemorrhage.

Sinus Trephination

Indications

Known or suspected presence of neoplasms, granulomas, or specific infections localized in the sinus cavities beneath the cortical bone of the skull.[8] The more common areas where trephination may be performed include the dorsal-rostral aspect of the frontal bone, the area caudal to the nasal-frontal hinge, and the maxillary bone (Figure 29.5).[11]

Surgical technique

Approach to the sinus beneath the frontal bone cortex. After making a skin incision, holes are drilled into the frontal bone using Michele trephine or pneumatic bone burrs, at about one-half to two-fifths the distance from the rostral most plane of the eye to the naris.[3,11] Cortical bone is removed until the cancellous bone above the supraorbital sinus is visualized. Drilling continues into the sinus while widening the hole to an appropriate diameter. Cancellous bone is removed as needed to facilitate exposure and access to the



FIGURE 29.5 Lateral radiographic image of the skull of an anesthetized blue-and-gold macaw *(Ara ararauna)*. The location of the frontal bone site for trephination is depicted (^) and the rostral (nasal or maxillary) diverticulum of the infraorbital sinus is visualized and its location for trephination *(asterisk)*. (Courtesy Brian Speer.)

desired location. When trephining into the frontal bone, care should be taken to avoid injury to the eyes or their sockets. Following the collection of samples for diagnostic investigation and the remainder of the surgical procedure, the sinus may be irrigated.

Approach to the rostral (nasal) diverticulum of the infraorbital sinus. Trephination of the maxillary bone allows surgical access to the nasal (rostral) diverticulum of the infraorbital sinus. Using an appropriate diameter trephine or dental drill, a hole is made into the lateral maxillary beak, rostral to the nares, at approximately half the distance ventrally from the culmen. The specific location of where to drill is best determined by imaging and careful determination of surgical landmarks.[12] Once the sinus cavity is penetrated with the trephine, the core of bone is removed. This may be moistened with sterile isotonic fluids, saved and replaced at closure, submitted for histologic or other diagnostic evaluations, or discarded. As with the infraorbital sinusotomy, the exposed sinus space can be visually inspected, and magnification loupes with a focal light source, operating microscopy or endoscopy can be used in selected cases for deeper visualization, if indicated (Figure 29.6). Following collection of diagnostic samples, the open trephination often is covered with light nonporous tape, although in some circumstances, it may be closed with acrylics or resins, with or without the removed bony core being replaced. In parrot species, the speed of closure and healing of these trephination sites occur within a few weeks, while in toucan species full closure may take longer.

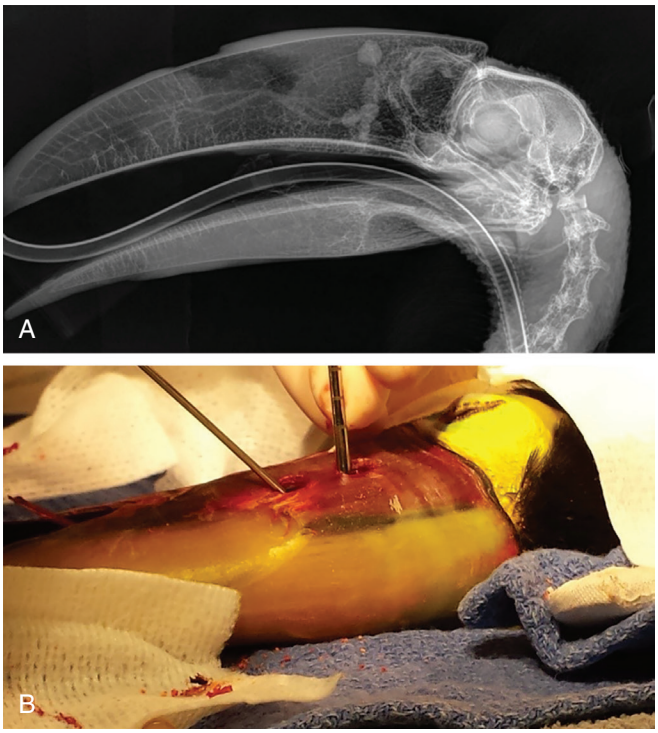


FIGURE 29.6 (A) Lateral radiographic image of a Swainson's toucan *(Ramphastos swainsonii)* with granulomas present within the rostral (nasal, or maxillary) diverticulum of the infraorbital sinus within the maxillary beak. (B) Use of two trephination sites into the sinus for inspection and removal of granulomas identified by projection radiographic and CT imaging studies. The rigid endoscope is being inserted in the more proximal site, and biopsy/grasping forceps are being introduced into the second more rostral site.

Potential complications

Iatrogenic trauma (including hemorrhage) during the trephination process, and (secondary) infection of the surgical sites.

Cervicocephalic Airsacculotomy

Indications

Traumatic injuries to the neck including the air sac system, infectious and neoplastic disease conditions (see Chapter 28).[13,14]

Surgical technique

A skin and air sac incision is made in the non-feathered (apteria) regions, parallel with the longitudinal axis of the neck. The exposed air sac space can be visually inspected, and magnification loupes with a focal light source, operating microscopy or endoscopy can be used in selected cases for deeper visualization, if indicated (Figure 29.7). Following collection of diagnostic samples, and/or debridement and flushing, the air sac incision is apposed with appropriate size monofilament suture (5-0 to 7-0 in many companion bird species). Skin is closed routinely in a second layer.

Complications

Hemorrhage into the air sac or open spaces,[13] epistaxis and subcutaneous emphysema,[15,16] and iatrogenic trauma to adjacent structures (nerves, trachea, esophagus).

Cervicocephalic-Clavicular Air Sac Shunt

Indications

Chronic hyperinflation of the cervicocephalic air sacs in psittacine species that is resulting in appreciable compromise to the patient welfare.[17]

Surgical technique

On the affected side, a superficial longitudinal incision is made laterally in the region of the thoracic inlet. Where the cervical portion is intact, a purse-string suture is made in the air sac. If the air sac is ruptured, a nonabsorbable purse-string suture can be placed between the inner lining medially and the lateral subcutaneous tissue. A shunt is prepared by modifying a small (1.0–3.0 mm) tracheal or other sterilized tube by removing the cuff and placing the side-port openings near one end. Through a stab incision, the tube is placed into the air sac about halfway up the neck. The purse-string is tied and then sutured around the tube. The crop is gently dissected ventrolaterally from its fascia attachment to the coracoid and clavicle until the clavicular air sac lumen is entered. The tracheal tube is then shortened, and side-ports added distally before it is inserted into the clavicular air sac. After anchoring the suture to the clavicle, the crop and muscular fascia is closed around the tube in a purse-string fashion. Sutures to the coli longus muscle further anchor the tube in place. The skin is closed routinely, and a small stab incision is made laterally to allow some postoperative air leakage until the clavicular air sac completely seals around the shunt.

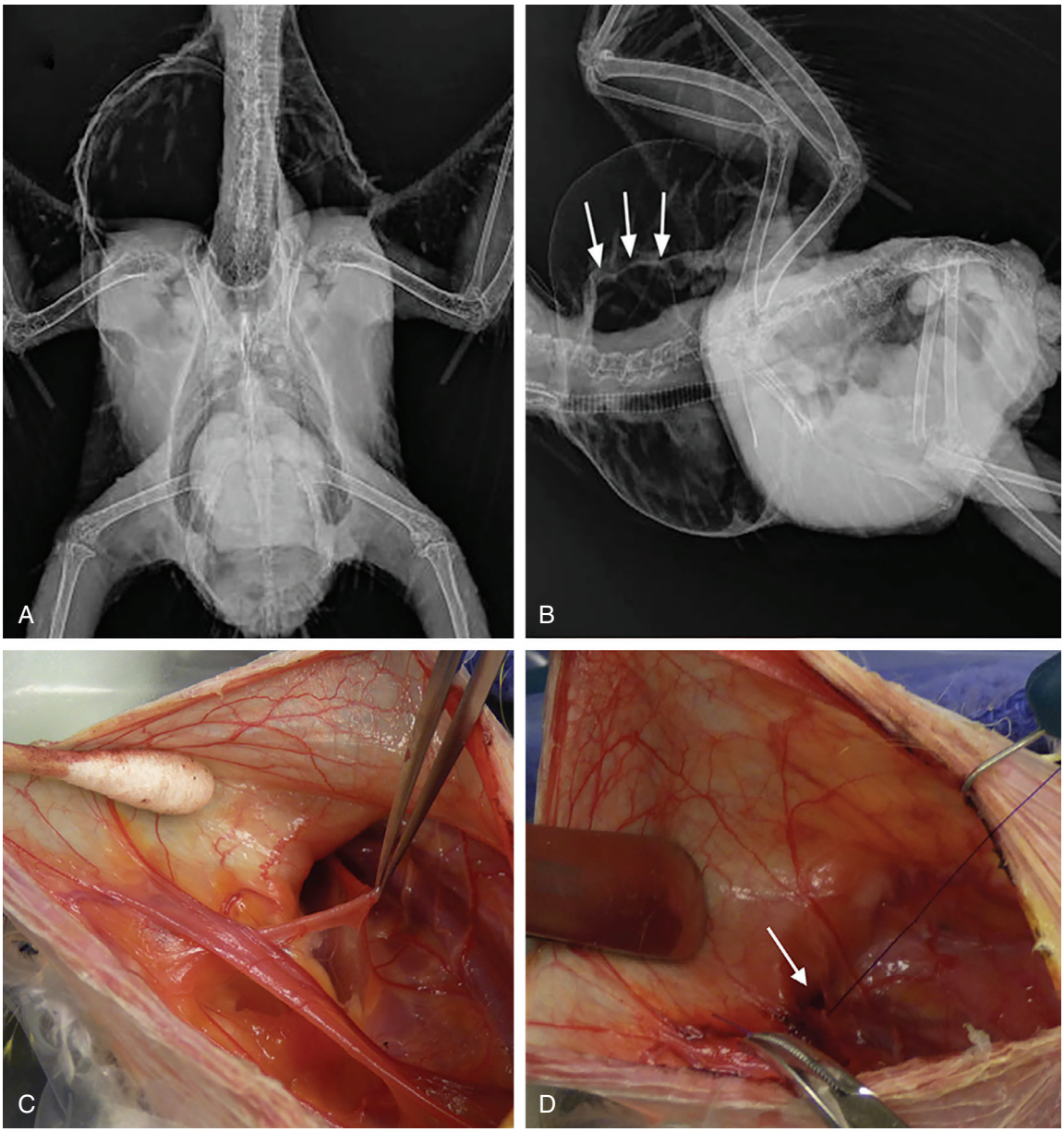


FIGURE 29.7 Closure of a large cervicocephalic air sac rupture in a double yellow-headed Amazon parrot *(Amazona ochrocephala oratrix)*. The airsac deficit was identified with a combination of projection radiographic (A and B) and CT imaging studies. Note the linear radiopaque structure that was most consistent with a thickened air sac rupture site *(arrows)*. A monofilament absorbable suture was used to close the air sac membrane (C and D) (remaining area to be closed identified by *white arrow*, D), and skin was closed separately. (Courtesy David Eshar.)

Potential complications

Occlusion of the tube, shunt failure, and infection.

Air Sac Cannulation

Indications

Placement of an alternative airway to facilitate ventilation includes the presence of obstructive tracheal disease or the need to have free access to the upper airways of the bird surgically. Common indications for air sac cannulas include obstructive tracheal granulomas (*Aspergillus* spp.), tracheal foreign bodies, and tracheal injuries that require surgical repair. Less commonly, intratracheal hemorrhage, neoplasms, redundant tracheal membranes or strictures, or even mucus tracheal plugs may be seen, necessitating placement of this manner of an alternative airway.[18,19]

Surgical technique

Preoxygenation may help facilitate the procedure, and midazolam often can have great value as an anxiolytic and conscious sedative. Combined, these may reduce the time required for induction and hence the ability to safely place the air sac cannula. General anesthesia often is required, and

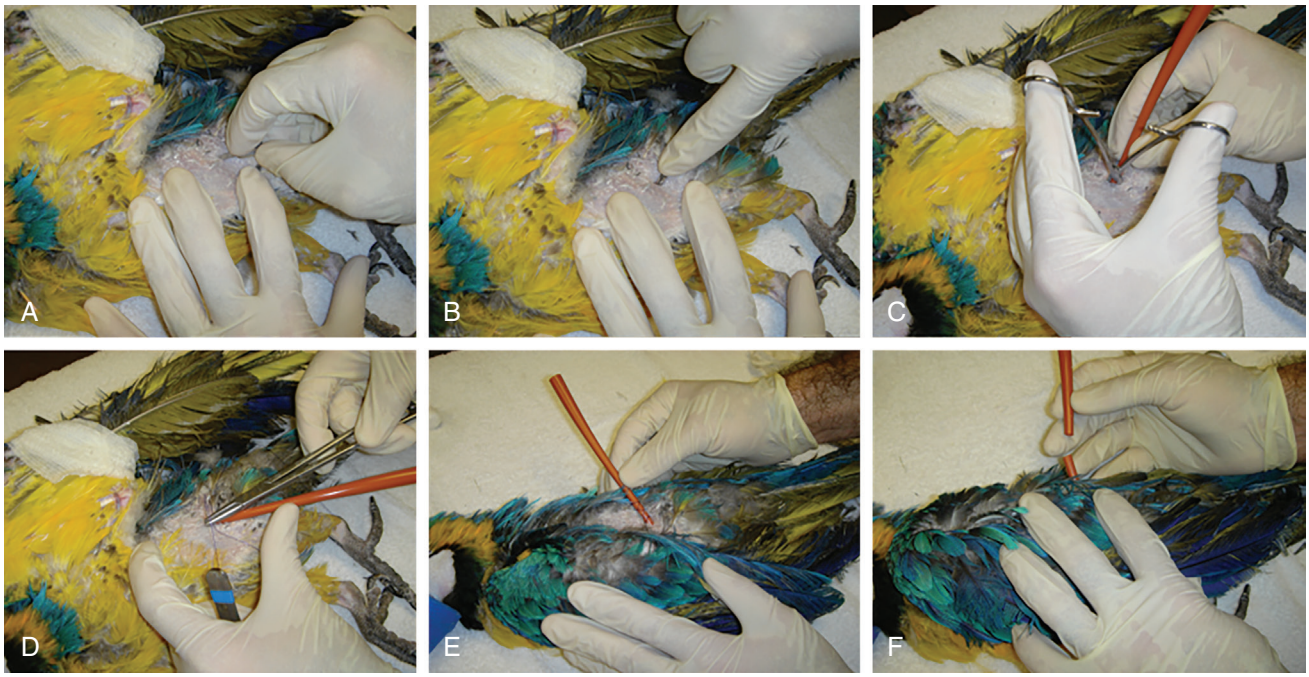


FIGURE 29.8 For air sac tube placement, the bird is positioned in right lateral recumbency. The tube is placed in the left lateral flank. The skin is prepared with a surgical scrub and a small incision is made caudal to the last rib or between the last two ribs (A). Mosquito forceps are used to bluntly penetrate the muscle wall and enter the air sac (B). The end of the fenestrated tube is inserted into the air sac between the opened jaws of the mosquito forceps (C). A finger-trap suture is used to and suture to the skin and abdominal muscles (D). Finally, the tube is cut at the appropriate length (E and F). (Courtesy David Sanchez-Migallon Guzman.)

speed is important, especially in patients with (obstructive) tracheal disease. The caudal thoracic and abdominal air sacs are the most common sites for tube placement. To perform the procedure, the patient should be positioned in lateral recumbency (Figure 29.8). Landmarks for access to the caudal thoracic air sac include the caudal margin of the last (eighth) rib, the cranial thigh musculature, and the dorsal trunk musculature. The caudal thoracic and the abdominal air sacs can both be accessed with the leg extended cranially, whereby the caudal thigh muscles define the cranial margin, and the pubic bones and the dorsal trunk musculature define the caudal and dorsal margins, respectively. Alternatively, with the leg extended caudally, the tube can be placed into the caudal thoracic air sac in the left flank area between the last two ribs or caudal to the last rib. The end of an endotracheal tube can be used as an air sac cannula; alternately, a red rubber tube can be cut to an appropriate diameter; or commercially manufactured air sac tubes can be used. The inner diameter of the tube placed should be equal to or exceed the (estimated) inner diameter of the trachea of the patient. After making a small incision in the skin, bluntly dissect through the muscle of the lateral body wall to the air sac. Apply gentle pressure to "pop" hemostats through the air sac membrane into the air sac. Use caution and keep the index finger close to the tip of the instrument to avoid penetrating viscera. Insert the air sac tube craniomedially through the open hemostats, but not too deeply. Test for air flow with cotton strands, cottonwool, down feather, or a microscope slide. Suture the tube in place using a tape tab placed at the tube at its skin interface, or a finger-trap suture. Test again for patency and stability of the tube once the sutures are in place. It is uncommon to require cervical restraint collars or Elizabethan collars to block the bird from accessing the tube. Oxygen or anesthetic systems can be attached directly to the air sac tube as long as these attachments do not add weight and discomfort for the bird. These alternative airways are left in place typically for periods up to 5 days. Patency needs to be regularly checked. To determine if the trachea is functional, the air sac breathing tube may be sealed with a piece of (paper) tape or clamped off with a hemostat while observing the patient's breathing character. If no change in respiration occurs, and the patient breathes normally, then the air sac tube can be removed. The entry site of the cannula may be closed with suture or be allowed to heal by second intention.

Potential complications

Obstruction of the tube, discomfort due to placement location of the tube, visceral organ trauma, hemorrhage or perforation, and infection.

Tracheotomy

Indications

Foreign bodies or masses in the trachea might require tracheotomy.[20,21] Tracheotomies are performed when less invasive approaches, like endoscopy or suction,[22,23] have failed to resolve the problem. Tracheal surgery requires prior air sac intubation to maintain anesthesia.

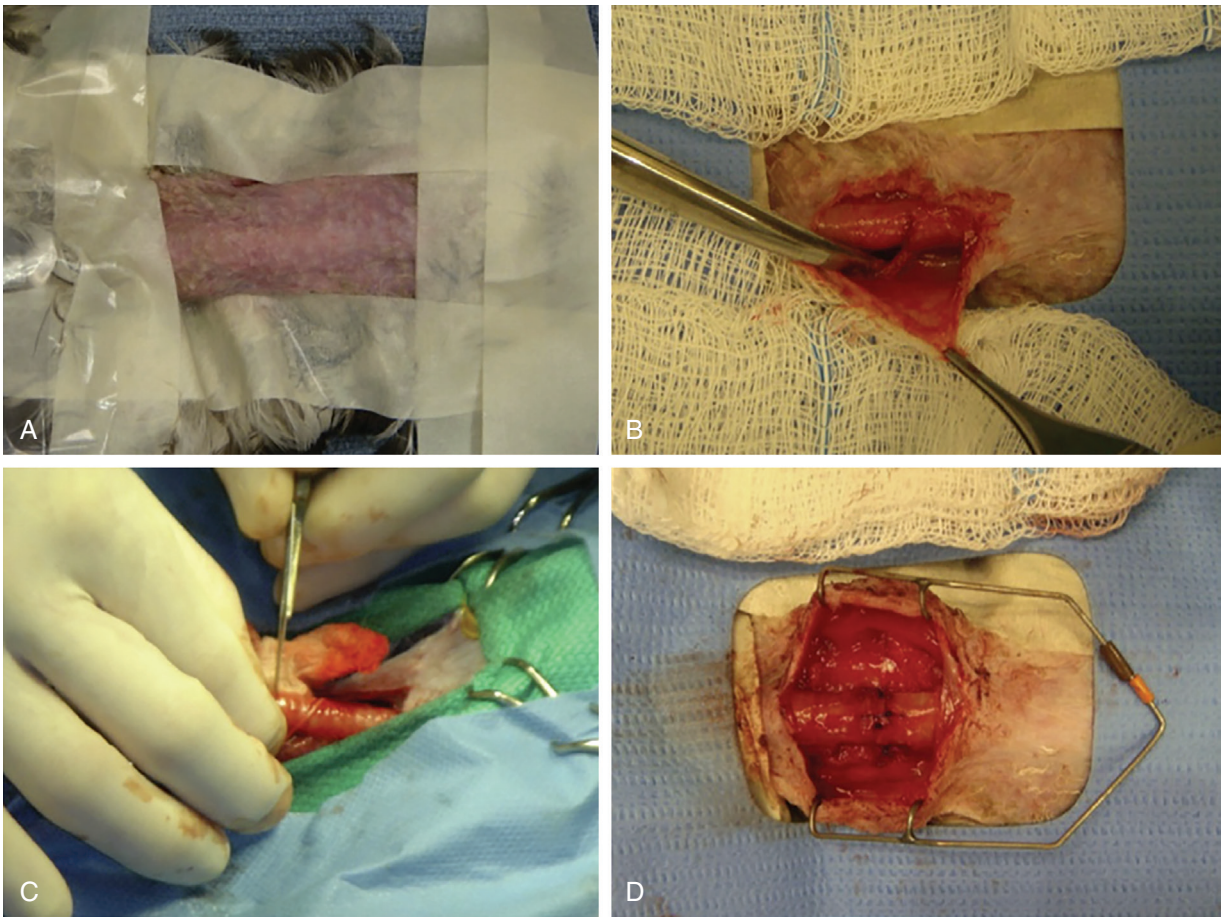


FIGURE 29.9 Tracheotomy in a red-tailed hawk *(Buteo jamaicensis)*. (A) The feathers have been removed over the skin in the ventral cervical region with the bird positioned in dorsal recumbency. (B) The subcutaneous tissues are dissected to reveal the paired sternothyroideus muscles. The recurrent nerves are identified and avoided. (C) A transverse incision over the ventral aspect of the trachea is performed in a different bird with a No. 11 blade. (D) Simple interrupted sutures are placed incorporating one tracheal ring on each end of the incision using an absorbable monofilament suture. (Courtesy David Sanchez-Migallon Guzman.)

Surgical technique

The bird is positioned in dorsal recumbency (Figure 29.9). The skin is incised along the ventral midline of the neck or just lateral to the crop. The subcutaneous tissues are dissected to reveal the paired tracheal muscles that are associated with the trachea in birds (i.e., *Mm. sternohyoideus*, *tracheolateralis*, *sternotrachealis*, and *cleidotrachealis*). Note that the location of these muscles is variable depending on the species. The recurrent nerves are identified along the ventral lateral to lateral (depending on species) aspects of the trachea and care should be taken to avoid these through the use of careful dissection techniques, if possible. If the location of the lesion cannot be determined externally, a red rubber catheter can be passed through the trachea to a point of resistance in the case of stenosis, or a rigid endoscope can be inserted to identify the affected area. Transillumination or measurements taken from projection radiography or CT imaging may also help localize the lesion prior to surgical incision (see Chapters 2 and 27 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 14, Advances in Diagnostic Imaging). A transverse incision with a No. 11 scalpel blade or beaver miniblade over the surgical site of approximately 50% of the tracheal circumference is performed on the ventral tracheal surface. Next, stay sutures are placed around the tracheal rings adjacent to the tracheotomy site to manipulate the trachea. Foreign material may be grasped and removed, gently debrided, suctioned, or pushed cranially to exit through the glottis. A hypodermic needle may be placed transversely caudal to the obstruction to help prevent it from being inadvertently moved caudally during extraction efforts. A rigid endoscope may facilitate visualization and confirmation of complete removal of the target material or resection of the affected area. Simple interrupted sutures are preplaced to incorporate one to two tracheal rings on each end of the incision using small, 4-0 to 8-0 monofilament absorbable (e.g., polydioxanone) suture. The subcutaneous and skin closure is routine.[4]

Postoperative care

Antibiotics, antifungals, opioids, and antiinflammatories are administered as needed. Removal of the air sac cannula after

temporary occlusion and careful observation for respiratory distress following air sac cannula removal are recommended. Cage rest can be considered to prevent trauma of the surgical site.

Complications

Intraluminal granuloma formation at the suture site has been described as common.[4] Hemorrhage early after surgery and stenosis late after surgery have been reported in a blue crane *(Grus paradisea)* that underwent tracheotomy to remove a rock.[20] Other possible complications include infection of the surgical site, aspiration, and pneumonia.

Prognosis

For uncomplicated procedures, the prognosis is good. If further damage to the trachea occurs or diseased tissue is not removed, tracheal stenosis may develop, and resection and anastomosis might be required.

Tracheal Resection and Anastomosis

Indications

Trauma, infection, inflammation, or neoplasia may result in tracheal stenosis requiring resection of the affected segment of the trachea. There are several case reports in the literature involving a wide range of species.[20,24–34] In most cases, the trachea is of sufficient length that several tracheal rings can be removed without the creation of undue tension when anastomosed.

Surgical technique

The patient is positioned typically in dorsal recumbency, and the surgical site is approached as described earlier for the tracheotomy (Figure 29.10). Ideally, the recurrent nerves and their associated vascular bundle are carefully dissected off the trachea and not severed. Suture can be used to retract these nerves and vessels away from the surgical site during the remainder of the procedure. Cranial and caudal to the affected segment,

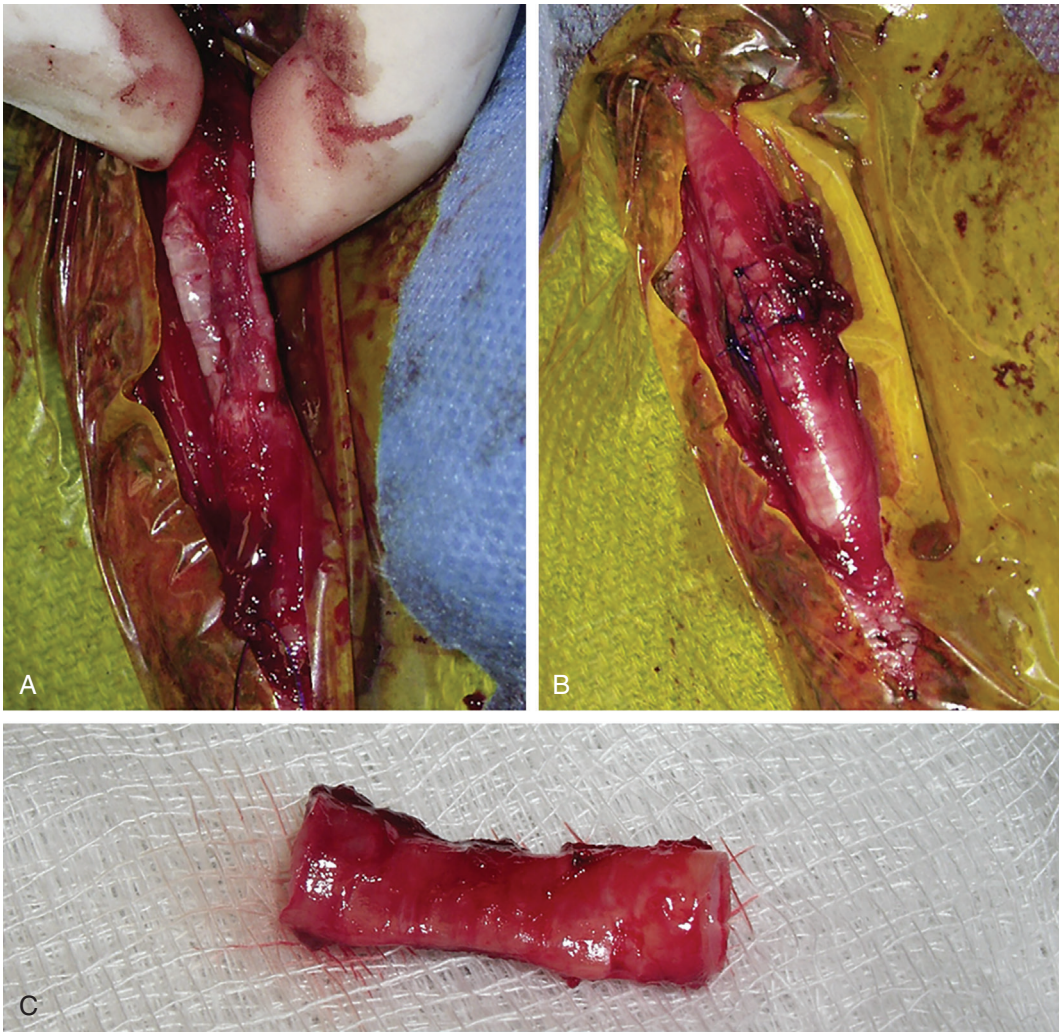


FIGURE 29.10 Tracheal resection and anastomosis in a mallard duck *(Anas platyrhynchos)*. (A) The collapsed tracheal segment is exposed following incision of the skin and muscles over the ventral aspect of the trachea with the bird positioned in dorsal recumbency. (B) The collapsed segment has been resected and the ends of the trachea anastomosed with simple interrupted and tension relief sutures. (C) Segment of the trachea resected. (Courtesy David Sanchez-Migallon Guzman.)

a tracheal ring is bisected circumferentially with a No. 11 or beaver scalpel blade. Alternatively, the intratracheal ring tissue is cut one tracheal ring cranial and caudal to the affected segment. The tracheal ends are approximated by preplacing simple interrupted sutures of 4-0 to 8-0 monofilament absorbable (e.g., polydioxanone) on a tapered needle. Typically, a minimum of four sutures are used for the anastomosis. The sutures are tightened individually with extraluminal knots to oppose the tracheal ends. If there is a need to remove a larger portion of the trachea, it may be noted that the distal end is smaller than the proximal portion with some species, and it is impossible to oppose the tissues. In these circumstances, the proximal end is usually placed such that the distal end fits into it. The subcutaneous and skin closure is routine.

Postoperative care

Antibiotics, antifungals, opioids, and antiinflammatories as needed. Removal of the air sac cannulation after temporary occlusion, careful observation for respiratory distress following air sac cannula removal, and cage rest are commonly recommended. Endoscopic examination of the trachea may aid for confirmation of effective healing and an absence of stricture or granuloma formation.

Complications

Injury to the recurrent nerves resulting from surgical manipulation or radical resection. The recurrent nerve does not innervate the larynx in birds but instead innervates the esophagus, crop, tracheal, and syringeal muscles; thus damage to the recurrent nerves will not cause laryngeal paralysis but might instead cause a change in voice. The larynx in birds is innervated by the glossopharyngeal nerve. Postsurgical tracheal stenosis might develop if there is no good apposition of the anastomosis site, if reactive suture material or traumatic suture technique is used, and/or if high tension is applied to the anastomosed site. A mismatch of the diameter of the tracheal ends is more likely to occur in some species (e.g., macaws, cockatoos) when a large segment is resected in the most cranial third of the trachea, since there is a significant decrease in diameter from cranial to caudal in this area.

Prognosis

The prognosis for complete resolution in uncomplicated cases is good. In some cases, problems may reoccur. Reassessment of birds undergoing tracheal anastomosis for tracheal stenosis by endoscopy and radiography at 1, 2, and 6 months after surgery has been suggested.[28]

Tracheostomy

Indications

Glottal and periglottal lesions can result in upper airway obstruction requiring a tracheostomy.

Surgical technique

The patient preparation and surgical approach are as described for the tracheotomy. The surgical site is 1 to 2 cm ventral to the mandible, which helps to ensure that the stoma is not obstructed when the bird flexes the neck (Figure 29.11). A transverse incision between the tracheal rings of approximately one-third the circumference of the trachea is made with a No. 11 or beaver blade. A second parallel incision is made in the trachea so that final stoma length would be approximately three times the width. The parallel incisions are connected incising the tracheal rings, resulting in a rectangular-shaped stoma. The placement of a (custom-made) stainless steel stent in the stoma may help prevent it from closing prematurely, although this has not yet been described. The skin is sutured to the stoma with 5-0 to 7-0 monofilament absorbable suture material with cutting or reverse cutting needle in a simple continuous pattern.[35]



FIGURE 29.11 Postoperative tracheostomy in a blue-and-gold macaw *(Ara ararauna)*. Subglottal obstruction, which was nonresponsive to medical and surgical treatments, leads to the decision to create a more permanent stoma. Complications included progressive mucus accumulation and the formation of granulation tissue at the stoma site. (Courtesy Angela Lennox.)

Postoperative care

Antibiotics, antifungals, opioids, and antiinflammatories as needed. Daily cleaning of the surgical site with saline to remove secretions.

Complications

Dehiscence of the surgical site, subcutaneous emphysema, (secondary) infections, granuloma formation around the stoma with reduction in diameter, and mucus accumulation in the lower airway tract.[35,36]

Prognosis

Poor long-term prognosis. Of the three cases reported in the literature, survival times were 5 weeks in a hooded vulture *(Necrosyrtes monachus)* versus 3 and 4 months in two psittacine birds, respectively.[35,36] Euthanasia or spontaneous death has

occurred largely due to a loss of effective airflow through the stoma and recurrence of obstructive airway–associated clinical signs.

Tracheal Stenting

Indications

In extensive tracheal stenosis, when other approaches like balloon dilation or tracheal resection fail or are not considered possible because of the length of the affected trachea.[37]

Surgical technique

Endoscopic examination and/or CT imaging is performed to confirm the cranial and caudal extent of the tracheal narrowing and stricture. The stent is introduced through the glottis and advanced under fluoroscopic guidance. In the case of the eclectus parrot *(Eclectus roratus)* described in the literature, a custom-made nitinol wire stent measuring 4 × 36 mm (Vet Stent; Infiniti Medical, Menlo Park, CA, USA) was used.[37] The undeployed stent is positioned expanding cranially and caudally over the affected area, and then the stent is deployed and expanded (Figure 29.12).

Postoperative care

Antibiotics, antifungals, opioids, and antiinflammatories as needed. Nebulization with a combination of saline, acetylcysteine, and dexamethasone was needed in the eclectus parrot described in the literature. Multiple attempts to wean the patient off nebulization therapy and to switch to a corticosteroid-free combination were unsuccessful.[37]

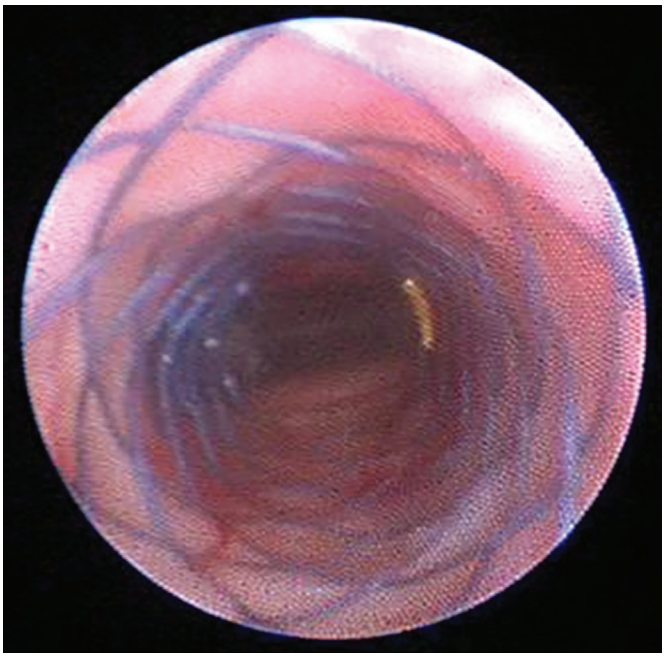


FIGURE 29.12 Endoscopic view following deployment of a nitinol wire stent in an eclectus parrot *(Eclectus roratus)* with tracheal stenosis. The undeployed stent was positioned over the region of stenosis and then expanded. The syringeal membranes are visible centrally, distal to the deployed stent. (Courtesy of Stephen Divers, University of Georgia)

Complications

In the eclectus parrot, tracheitis and tracheomalacia were reported as complications resulting in euthanasia almost 2 years after the procedure.[37] Other potential complications of stent placement are obstructive granulation tissue, stenosis at the ends of the stent (epithelium growing over stent wires), migration of the stent, mucous plugging, infection, and stent fracture.

Prognosis

In the single case reported with an internal tracheal stent, the bird survived almost 2 years following surgery. After stent placement, intermittent episodes of mild to moderate dyspnea continued, which responded to medical management. Given the possible complications, short- and long-term reassessments of birds undergoing stent placement for tracheal stenosis by endoscopy and/or radiography are recommended.

Tracheal Prosthesis

Indications

Tracheal collapse.[38]

Surgical technique

In the literature, the procedure has been described in an ostrich *(Struthio camelus)*. In this bird,[38] six tracheal rings had been transected by a previous laceration, leaving a 1.5-cm section of the trachea devoid of medial/lateral support, resulting in tracheal collapse on inspiration. Following a standard midline approach to the trachea, a 2.5-cm diameter, 0.75-cm-wide polypropylene ring devised from a 12-mL syringe case was sutured to the tracheal cartilages and submucosa as a splint-ring prosthesis, without perforating the tracheal lumen, with 2-0 polydioxanone or other appropriate monofilament suture. The subcutaneous tissues and skin were closed routinely.

Postoperative care

Antibiotics, antifungals, opioids, and antiinflammatories as needed.

Complications

Short-term complications described in the ostrich included swelling of the surgical site 3 days after the surgery.[38] No long-term complications were reported up to 6 months after surgery. Other complications that have been described in dogs include laryngeal paralysis and recurrence of laryngeal collapse. However, these are less likely to occur in birds due to their anatomy and the likelihood of a traumatic event underlying the tracheal pathology necessitating such approach.

Prognosis

In the single case reported with an external tracheal prosthesis, the bird survived for at least 6 months.

Syringotomy

Indications

Localized infections (e.g., bacterial, fungal), foreign bodies, when noninvasive methods like endoscopic removal or suction are not possible.

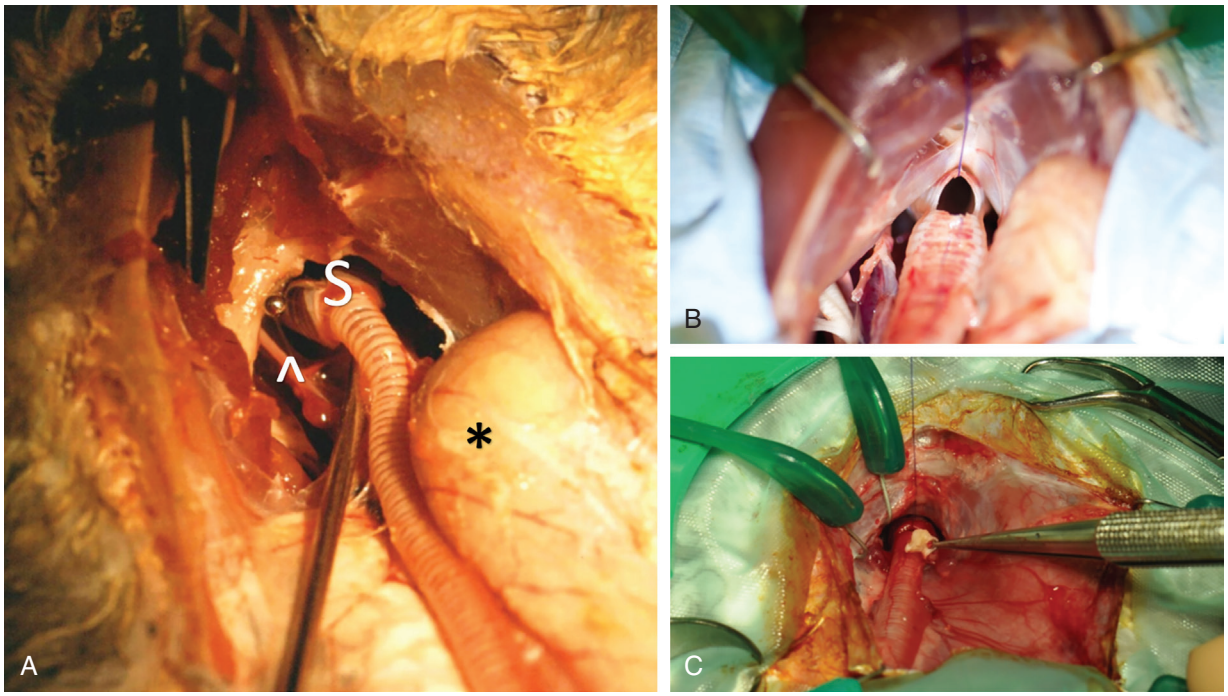


FIGURE 29.13 Ventral approach to the proximal trachea and syrinx. (A) The ingluvies *(asterisk)* is visible and has been reflected to the right. The paired sternotrachealis muscles have been transected, and the left internal carotid artery is visible (^) with its associated thyroid gland. The syrinx *(S)* has been elevated, exposing the trachea and its attachments to it. (B) The syrinx has been elevated with a stay suture, and a 180-degree incision three to five tracheal rings distal to the syrinx has been made, exposing the inner aspects of the syrinx. (C) Syringotomy being used for excision of a single mycotic plug at the syrinx in an Amazon parrot (*Amazona* spp.). (A, C, Courtesy Neil Forbes.)

Surgical technique

The patient is positioned in dorsal recumbency. Incise the skin along the ventral midline of the thoracic inlet and elevate the skin from the crop to the level of the clavicle (Figure 29.13). The crop is dissected free from its clavicular attachments and reflected to the left side. Fat that may be present must be removed using careful blunt tissue dissection. The clavicular air sac is incised, and the trachea is exposed as it enters the region of the thoracic inlet. If additional access is required, the superficial pectoral muscles are elevated, which will allow an osteotomy of the clavicle to be performed on one side of its symphysis. The paired sternotrachealis muscles are visualized traversing obliquely along the trachea and attaching caudolaterally. Before transecting the muscles bilaterally, which will allow cranial retraction of the syrinx, the large vessel between these muscles should be coagulated. To manipulate the trachea and syrinx atraumatically, a blunt hook can be placed around the syrinx. Alternatively, stay sutures can be placed around tracheal rings craniad to the obstruction. In most species, it is impossible to completely exteriorize the syrinx. A syringotomy is performed three to five rings cranial to the bifurcation on the ventral surface of the tracheal syringeal cartilages using the method as described for the tracheotomy. The tracheal incision is closed as described earlier for the tracheotomy, using 2-0 polydioxanone or other appropriate monofilament suture. If a clavicular osteotomy is performed, the two ends of the clavicle are opposed at closure. The skin is closed routinely.

A lateral approach to the syrinx, which includes performing a thoracotomy, has also been described when other options are not available but is rarely performed.[4] For this procedure, the bird is placed in right lateral recumbency with the wings elevated in an upward position. A skin incision is made over the second and third ribs at the dorsolateral margin of the pectoral muscles, reflecting ventrally the pectoral muscles. A portion of each of the second and third ribs is transected and removed, exposing the cranial portion of the lung. This portion of the lung is gently dissected and reflected from its attachments using a moistened cotton-tipped applicator. The syrinx is visualized dissecting between the major vasculature (jugular vein, pulmonary artery, and branches of the subclavian artery) and incised using microsurgical scissors at its junction with the left primary bronchus. The foreign material is removed. The syringeal incision is allowed to heal by secondary intention, and following placing the lung back in its normal position, the pectoral muscle is pulled over the defect in the rib cage, to then close the skin in a routine manner.

Complications

For the ventral approach to the syrinx, unintentional tears of the trachea that are too large for repair, tracheal stricture. Hemorrhage.

Prognosis

In localized lesions, without the abovementioned intraoperative and immediate postoperative complications, the access to the syrinx through the trachea has the potential to be curative and carry good long-term prognosis. No information is

available on cases in which a lateral approach of the syrinx via thoracotomy has been performed.

SURGERY OF THE LOWER RESPIRATORY TRACT

Pneumonotomy/Bronchiotomy

Indications

Surgical access to bronchi with obstructive lesions, localized infections, and neoplastic processes of the lung for collection of diagnostic samples.[3,39,40]

Surgical technique

A direct approach to the lesion is best planned though advanced imaging (Figure 29.14; see also Chapter 2 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 14, Advances in Diagnostic Imaging). In most cases, the bird is placed in lateral recumbency with the wings extended upward. An intercostal approach is made by reflecting skin and incising through the intercostal musculature. If needed, a rib may be transected or even portions of the rib can be removed. If a biopsy is needed, a sample is collected with biopsy forceps. If approach to a bronchus is needed, the bronchi are exposed and incised using microsurgical scissors. Using a small curette or catheter, the mucopurulent to granulomatous material can be removed from the bronchus to successfully restore airflow (Figure 29.14). Hemorrhage can be controlled with pressure, moistened cotton-tipped applicators, or hemostatic clips as needed. Vascular clips are placed on the lung, as needed, to minimize air leakage and hemorrhage. Placement of oxidized regenerated cellulose (Surgical; Johnson & Johnson Medical, Arlington, TX) may be utilized to help reduce continued bleeding.

Complications

Air leakage with subcutaneous emphysema, hemorrhage, bronchial tears.[40]

Prognosis

Pneumonotomy and bronchiotomy patients appear to function normally postoperatively.[3,39,40]

Pneumonectomy

Indications

Infection or neoplasia may require partial pneumonectomy.

Surgical technique

The lungs may be approached through the caudal thoracic air sac or the dorsal intercostal spaces following retraction or

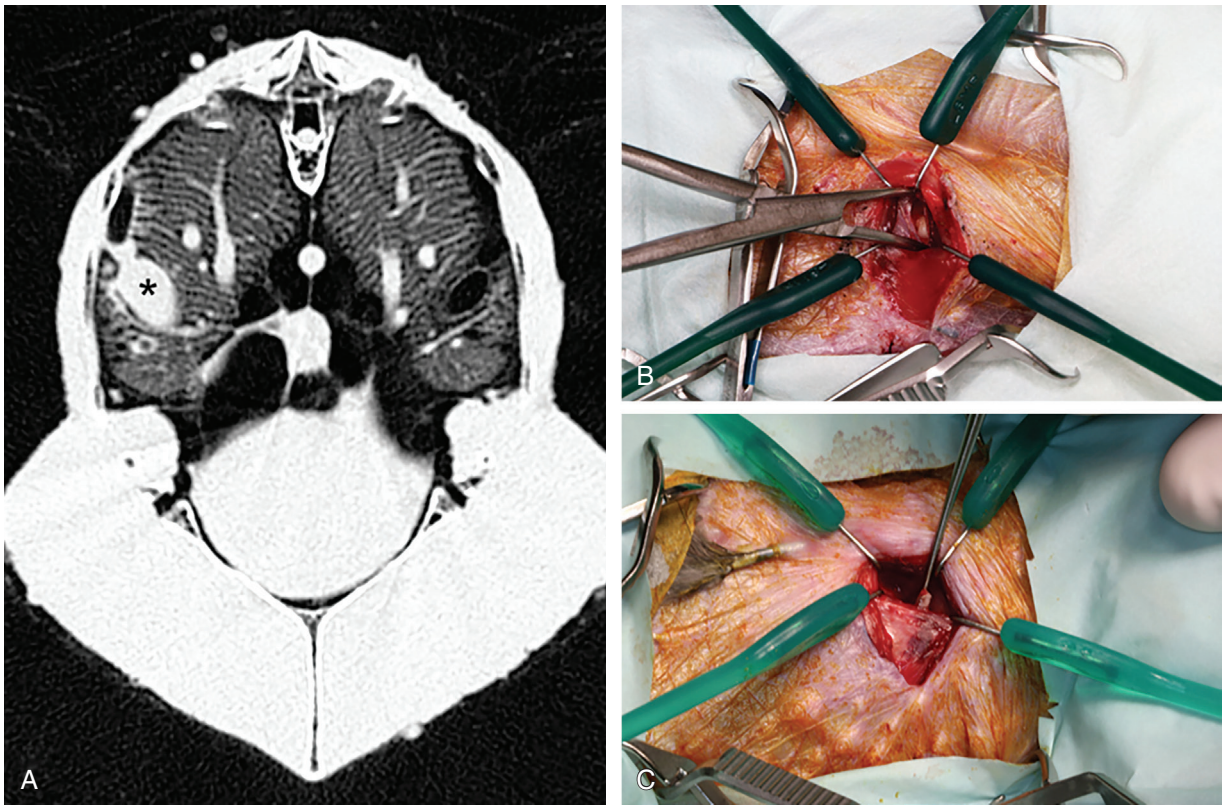


FIGURE 29.14 (A) Transverse plane of a computed tomography study in a parrot showing an obstructed primary bronchus *(asterisk)*. (B) Direct lateral approach to the lung and bronchus, ventral to the *top* and cranial to the *right* of the image. The skin has been incised over the previously localized lesion, and intercostal musculature incised and retracted. Hemostats are used to retract the pleural membrane, exposing the caseous lesion within the bronchus. (C) Removal of the bronchial plug with a curette, following surgical exposure via lateral approach to the lung. (Courtesy Nico Schoemaker/Yvonne van Zeeland.)

removal of the ribs. The lung is elevated from the ribs and other structures using a spatula or moistened cotton-tipped applicator. Hemostatic clips are placed on the lung tissue to isolate the portion to be removed. The portion distal to the clips is then removed. The skin is closed routinely.[41]

Complications

Air leakage with subcutaneous emphysema, hemorrhage.

Prognosis

No information is available on pneumonectomy reported cases, and the procedure is rarely performed.

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CHAPTER 30

Diagnostic Endoscopy, Endoscope-Assisted Therapy, and Endosurgery of the Avian Respiratory System

Stephen J. Divers

INTRODUCTION

There are around 10,000 species of birds that vary tremendously in their anatomic, physiologic, and ecologic adaptations, and it is impossible to cover all species and possible situations. Consequently, focus has been concentrated on describing endoscopic principles of diagnosis and therapy with multiple-taxa examples, while prioritizing those species most encountered in clinical practice.

Endoscopy offers several advantages over other diagnostic modalities. Unlike serologic testing for aspergillosis, which takes several days to obtain results, endoscopic diagnosis is immediate, and the collection of biopsies for culture and sensitivity testing provides greater confidence in appropriate drug selection and permits minimum inhibitory concentration (MIC)-calculated drug doses. Indeed, general reliance on broad-spectrum antimicrobials implies a low level of skill and ability on the part of the clinician. Unlike cross-sectional imaging, including computed tomography (CT), endoscopic examination also facilitates biopsy for a definitive (histologic and microbiologic) diagnosis and topical or intralesional therapy to be administered. It is for these reasons why endoscopic evaluation of the respiratory system is the accepted standard of care for domesticated mammals and humans, and birds are no exception. Indeed, by the time a bird at rest shows clinical evidence of respiratory disease, severe compromise has often occurred, and the case should be considered emergent. Diagnostic investigations should proceed quickly following stabilization.

Endoscopy is a surgical procedure and requires general anesthesia. Therefore, although a brief tracheoscopic examination may only require 30 seconds of gas anesthesia, any contraindication for anesthesia is generally a contraindication for endoscopy. That said, birds, even with pronounced respiratory compromise, are often more stable when anesthetized with 100% oxygen than when handled in room air.

ENDOSCOPY EQUIPMENT

A full list of equipment can be found in Table 30.1.

Light Sources

The telescope is connected via a fiber-optic light guide cable to a light source. While halogen light sources are inexpensive but still effective for birds under 2 kg, xenon and LED light sources provide better-quality light and an intensity that can illuminate the body cavities of larger species. The LED light source of the mobile Karl Storz Tele Pack Vet X LED (Karl Storz Veterinary Endoscopy [KSVEA], Goleta, CA, USA) has an extended life expectancy of up to 30,000 hours.

Cameras

An endovideo camera connected to the eyepiece of the telescope is an integral part of the endoscopy system and greatly facilitates the surgeon's performance (Figure 30.1). Cameras, available in both European PAL and American NTSC formats, can vary dramatically in cost from budget single-chip cameras to three-chip digital high-definition (HD) and 4K models. The traditional video system provided a resolution of 720 × 480 and therefore inevitably led to some cropping of the image for display on a traditional 4:3 aspect monitor. The advent of HD systems has increased the video size to 1920 × 1080 pixels. In addition, the new 16:9 aspect ratio provides a 33% wider field of view while progressive (as opposed to interlaced) scan produces improved resolution, color clarity, and refresh rates. Additional camera advances including 4k high resolution (3840 × 2160 pixels), three-dimensional (3D), light filters, fluorescence imaging, and chromoendoscopy are already available, although such equipment may currently be cost-prohibitive for anyone outside of a large academic or referral hospital. The fact remains that *any* camera will greatly improve performance compared to using the eyepiece directly and will also facilitate photodocumentation. Operating room setup is important, and the monitor should be positioned directly in front of the endoscopist, with instruments within easy reach.

Photodocumentation

The ability to record still images and video of procedures is becoming increasingly important for maintaining accurate medical records, as well as for generating pictures for brochures, presentations, or scientific publications. Most camera systems generate S-Video or digital outputs that can be connected to a myriad of nonprofessional, commercial recording devices (e.g., laptops) that can ultimately save material in various formats to digital media. Professional-grade devices, although more expensive, provide dedicated functions that are important in medicine. For example, most professional endoscopy recording devices will support still images (tiff, bmp, jpeg) and video (MPEG2) with audio voice-over (WAV). Modern systems can also provide network and web-based

TABLE 30.1

Diagnostic and Endosurgical Endoscopy Equipment

Equipment Description	Primary Indications
VISUALIZATION AND DOCUMENTATION	
Endovideo camera and monitor Light source and light guide cable Digital capture device	Required for all endoscopy procedures
RIGID TELESCOPES AND ENDOSCOPES	
1-mm × 20-cm semirigid miniscope, 0 degrees	Stomatoscopy, rhinoscopy, tracheoscopy
1.9-mm × 18.5-cm telescope, 30 degrees oblique, with integrated 3.2-mm operating sheath	Stomatoscopy, rhinoscopy, tracheoscopy, and coelioscopy (<300 g)
2.7-mm × 18-cm telescope, 30 degrees oblique, with separate 3.5-mm protection and 14.5 Fr operating sheaths or 2.7-mm × 18-cm telescope, 30 degrees oblique, with integrated operating sheath (11.5 Fr)	Stomatoscopy, rhinoscopy, tracheoscopy, and coelioscopy (100 g–1 kg)
5-mm × 29-cm telescope, 0 degrees, with 6-mm × 6.5-cm Ternamian EndoTIP cannula	Coelioscopy (over 15 kg)
5-mm × 29-cm telescope, 30 degrees oblique, with 6-mm × 6.5-cm Ternamian EndoTIP cannula	Coelioscopy (over 15 kg)
5-mm × 8.5-cm otoendoscope, 0 degrees, with integrated operating sheath	Stomatoscopy (>1 kg)
FLEXIBLE INSTRUMENTS FOR USE WITH RIGID TELESCOPES AND OPERATING SHEATHS	
3 Fr biopsy forceps 3 Fr grasping forceps	For use with 1.9-mm telescope and integrated sheath
5 Fr biopsy forceps 5 Fr single-action scissors 1.5 mm remote injection needle 5 Fr grasping/retrieval forceps 5 Fr wire basket retrieval 5 Fr polypectomy snare	For use with 2.7-mm telescope and 4.8-mm operating sheath or the 11.5 Fr telescope with integrated sheath
FLEXIBLE ENDOSCOPES	
Fiberscope, 2.5-mm × 100-cm working length, 1.2-mm instrument channel, two-way tip deflection	Transglottal respiratory evaluations (>500 g)
Video bronchoscope, 5.9-mm × 61-cm working length, 2.2-mm instrument channel, four-way tip deflection, suction, and irrigation	Transglottal respiratory evaluations (>5 kg)
INSTRUMENTS FOR USE WITH FLEXIBLE ENDOSCOPES	
Grasping forceps, 3 Fr × 120 cm Biopsy forceps, 3 Fr × 120 cm	For 2.5-mm fiberscope
Biopsy forceps, round cupped jaws, diameter 5 Fr, working length 120 cm Grasping forceps, alligator jaws, 5 Fr × 120 cm	For 5.9-mm video bronchoscope
ENDOSURGICAL INSTRUMENT HANDLES	
Two plastic handles without rachets One plastic handle with Mahnes-style rachet One plastic handle with hemostat-style rachet	These ClickLine handles are interchangeable and can be used with most instruments
CANNULAE, TROCARS, AND ENDOSURGICAL INSTRUMENTS	
3.9-mm graphite and plastic cannula and trocar, with silicone leaflet valve, and luer-lock insufflation port	Accommodates 2.7-mm telescope and 3.5-mm protection sheath
2.5-mm × 4-cm, graphite and plastic threaded cannula and trocar, with silicone leaflet valve, and luer-lock insufflation port (for 2-mm instruments, typically two required) 2-mm Reddick-Olsen dissecting forceps 2-mm Metzenbaum scissors 2-mm Babcock forceps	For endosurgery (<300 g) Instruments are all 20 cm in length
3.5-mm graphite and plastic threaded cannula and trocar, with silicone leaflet valve, and luer-lock insufflation port (for 3-mm instruments, typically two required) 3-mm fenestrated grasping forceps 3-mm Reddick-Olsen dissecting and grasping forceps	For endosurgery (>200 g) Instruments are all 20 cm in length

Continued

TABLE 30.1

Diagnostic and Endosurgical Endoscopy Equipment—cont'd

Equipment Description	Primary Indications
3-mm short curved Kelly dissecting and grasping forceps	
3-mm atraumatic dissecting and grasping forceps	
3-mm Babcock forceps	
3-mm Blakesley dissecting and biopsy forceps	
3-mm scissors with serrated curved double-action jaws	
3-mm microhook scissors, single-action jaws	
3-mm Mahnes bipolar coagulation forceps	
3-mm irrigation and suction cannula	
3-mm palpation probe with centimeter markings	
3-mm distendable palpation probe	
3-mm ultramicro needle holder	
3-mm knot tier for extracorporeal suturing	
6-mm × 5.5-mm Ternamian EndoTIP cannula, with silicone leaflet valve and insufflation stopcock (for 5-mm instruments, typically two required)	For endosurgery (15–150 kg) Instruments can be 36 cm or 43 cm in length
5-mm biopsy forceps	
5-mm Blakesley dissecting forceps	
5-mm curved Kelly dissecting and grasping forceps	
5-mm Babcock grasping forceps	
5-mm curved Metzenbaum scissors	
5-mm suction and irrigation cannula	
5-mm needle holders	
5-mm hook scissors	
5-mm bipolar forceps	
HEMOSTASIS EQUIPMENT	
Autocon III Vet electrosurgery and vessel sealing (or similar)	Enables many endoscopic instruments to be used as monopolar devices and facilitates bipolar coagulation using bipolar forceps. A wide variety of 2-, 3-, and 5-mm instruments are available.
Monopolar lead to connect to plastic instrument handles	
Bipolar lead to connect to 3-mm Mahnes bipolar coagulation forceps	
LigaSure Force-Triad energy platform with foot pedal	
5-mm × 37-cm LigaSure laparoscopic instrument	Only available in 5- or 10-mm instrument sizes but a useful instrument that combines sealing and cutting. The instruments are disposable human items but most veterinarians clean and reuse.

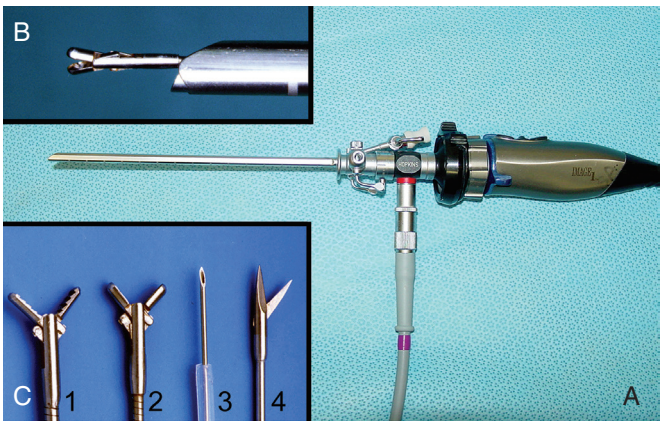


FIGURE 30.1 A 2.7-mm telescope system. (A) A 2.7-mm telescope housed within a 4.8-mm operating sheath, connected to a light cable and an endovideo camera. The newer 11.5 Fr telescope with integrated sheath has the same 2.7 mm optics. (B) A 5 Fr biopsy forceps inserted down the instrument channel, emerging directly in front of the telescope. (C) A variety of 5 Fr instruments can be used through the operating channel, including retrieval forceps *(1)*, biopsy forceps *(2)*, a remote injection/aspiration needle *(3)*, and single-action scissors *(4)*. (Courtesy Dr. Stephen Divers, University of Georgia)

storage and file sharing, including industry-standard DICOM (Digital Imaging and Communications in Medicine) support.

Mobile Units

The Tele Pack Vet X LED system combines an LED light source, endovideo camera, 38 cm (15″) color LED, with digital image and video capture onto SD memory cards or USB drives into a portable unit that is as well suited for field work, as in the hospital environment (Figure 30.2). This unit has also become popular in practice as it supports both rigid and flexible endoscopy systems, and a newer HD version of the Tele Pak has recently become available. An even more mobile option is the endogo HD camera (Cook Medical, Bloomington, IN, USA) and battery-powered light, which provides real pocket mobility albeit with reductions in light intensity and quality.

Flexible Endoscopy

There are two main classifications of flexible endoscope: the fiberscope (which transmits the image via a bundle of fiber-optic fibers) and the video endoscope (which transmits the image electronically from a charge-coupled device [CCD] stationed at the distal tip). Fiberscopes are less expensive but suffer from pixelation and poorer image quality compared to video endoscopes. Video endoscopes are considerably more



FIGURE 30.2 Tele Pack Vet X LED. This small, portable unit includes a camera, light source, 15″ monitor, and insufflation pump, is capable of recording still images and video, and is compatible with both rigid and flexible endoscopes. (Courtesy Dr. Stephen Divers, University of Georgia)

expensive but offer superior image quality. Furthermore, until relatively recently, flexible and rigid endoscopy light sources and cameras were generally incompatible, which necessitated separate rigid and flexible tower systems. This undoubtedly forced practitioners to make a choice for one or the other. Fortunately, the advent of modern cameras and light sources now enables practitioners to invest in just one endoscopy unit that can be used with both rigid and flexible endoscopes. Consequently, the ability to offer both flexible and rigid services has substantially increased in practice.

Flexible endoscopes vary from 14 mm to less than 1 mm in diameter, and those greater than 2 mm often offer tip deflection and possess a working instrument channel. Current commercial video endoscopes are limited to around 2.5 mm with a 1.2-mm instrument channel (although technology now exists to produce a <1-mm video camera). Therefore, smaller fiberscopes (0.5–2.5 mm and considerably cheaper) still have a role in avian bronchoscopy, although they often have just one- or two-way tip deflection and smaller working channels.

Telescopes and Sheaths

The 2.7-mm telescope system, first championed by Taylor for use in avian species, still remains the workhorse in avian practice because of its versatility and ability to be expanded as caseloads dictate.[1] The 2.7-mm Hopkins telescope can be used with a 3.5-mm protection sheath or with a 4.8-mm operating sheath that possesses ports and an operating channel for the introduction of 1.7-mm (5 Fr) instruments (Figure 30.1). More recently, the same 2.7 mm telescope became available with an integrated sheath resulting in an overall reduction in diameter to 11.5 Fr, while maintaining the same 5 Fr instrument channel. A smaller 9.5 Fr telescope with an integrated sheath can accommodate the introduction of 3 Fr instruments. A 1.0-mm semirigid (fiber-optic) miniature straight endoscope can be useful for smaller species (Figure 30.3), while 4-, 5-, and even 10-mm telescopes can be used with large to giant species.

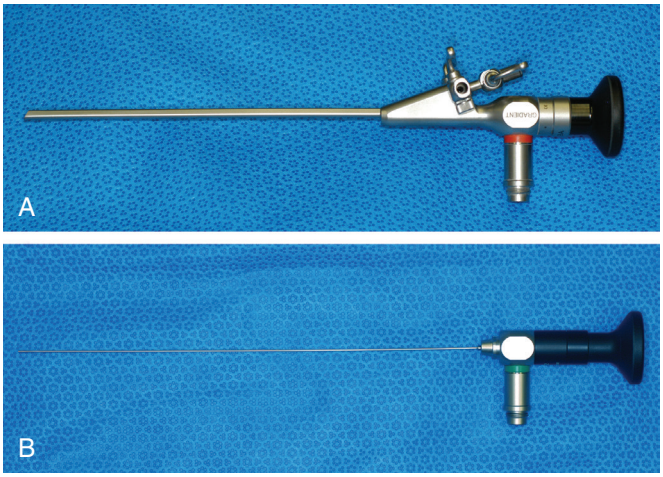


FIGURE 30.3 (A) A 9.5 Fr telescope with an integrated operating sheath. (B) A 1.0-mm semirigid miniature straight endoscope. (Courtesy Dr. Stephen Divers, University of Georgia)

Flexible Instrumentation

Flexible instruments can be used through the instrument channel of the 2.7-mm/11.5Fr and 1.9-mm/9.5 Fr systems in a single-entry endoscopic procedure. The most frequently used instruments include biopsy forceps, retrieval forceps, scissors, and remote injection/aspiration needle (Figure 30.1C). The 3 and 5 Fr (1.0- and 1.7-mm) grasping forceps are useful for manipulating tissues, debridement, and retrieving foreign objects or parasites. The fine aspiration/injection needle can be used for the aspiration of fluid from cystic structures where biopsy may be contraindicated due to risks of leakage. The needle can also be used for irrigation and endoscopic drug administration. The flexible biopsy forceps are used to harvest tissue samples for histopathology and microbiology in patients as small as 30 g. The small sample size usually permits the collection of several biopsies for multiple laboratory tests and serial biopsies to monitor disease progression over time to assess response to treatment. To take a sample, the biopsy forceps are inserted down the operating channel and into the field of view (Figure 30.4). It is much easier to advance and manipulate the telescope-instrument as a single device than to try and keep the telescope still and independently move the biopsy forceps back and forth. With the biopsy forceps held open, the telescope-instrument is advanced to the tissue of interest and when tissue enters the biopsy cup, the handle is released. Little pressure on the handle is required to close the jaws. Excessive clamping force will damage the forceps and does nothing to improve hemostasis. Some structures may be protected by a more fibrous membrane or capsule, while chronic airsacculitis can result in grossly thickened air sac walls. The fixed blade of the scissors is inserted at a shallow angle through such membranes, and the sheath-telescope-scissors are advanced as a single unit, cutting the membrane as they proceed. The scissors can then be replaced by the biopsy forceps to take a sample through the capsular incision or of the air sac wall. Other instruments that can be invaluable in specific situations include flexible suction catheters (e.g., 3 or 5 Fr red rubber catheters), radiosurgical needle electrodes (for pinpoint ablation or coagulation), and a dedicated CO_2 laser guide fits the 5 Fr instrument channel.

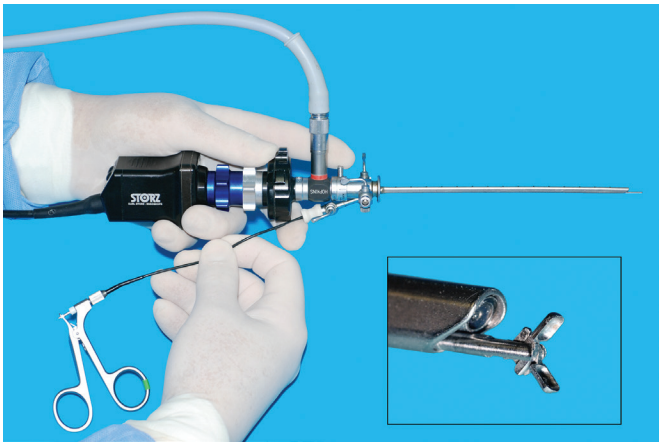


FIGURE 30.4 A 2.7-mm telescope within a 4.8-mm operating sheath, with an endovideo camera and light guide cable attached. Biopsy forceps (1.7 mm) passed through the working channel emerging in front of the terminal lens *(insert)*. (Courtesy Educational Resources, University of Georgia)

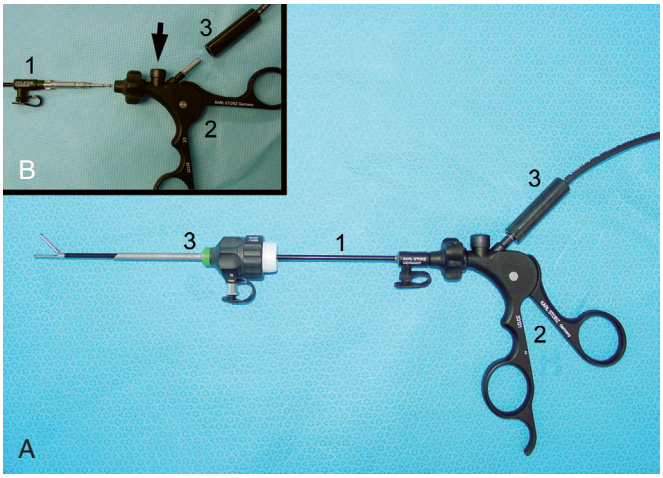


FIGURE 30.5 A 3-mm laparoscopy equipment. (A) A 3-mm instrument *(1)* attached to a standard ClickLine handle *(2)*. The instrument, attached to a radiosurgery unit via a connector on the handle *(3)*, has been inserted through a 3.5-mm graphite/plastic cannula *(4)*. (B) Instrument *(1)* and handle *(2)* can be quickly exchanged by pressing the release button (*arrow*). The radiosurgical connection is also shown *(3)*. (Courtesy Dr. Stephen Divers, University of Georgia)

Multiple-Entry Endosurgery

Rigid instruments, triangulated with, but physically separate from, the telescope are used to perform more complex endosurgical procedures within coelomic cavities and lower respiratory tract. In order to operate within the coelomic cavities, in addition to the telescope, it is necessary to introduce additional, independent instruments (Figure 30.5).[2] In order to maintain CO_2 insufflation during endosurgery in mammals and reptiles, it is necessary to use cannulae. These cannulae possess valves to prevent CO_2 leakage when instruments and telescopes are inserted or removed from the animal. CO_2 insufflation is not required (and contraindicated within the respiratory tract) in birds, as the air sacs remain inflated during anesthesia (especially if positive pressure ventilation is used). Nevertheless, cannulae are still valuable for facilitating faster transfer of instruments in and out of the coelomic cavities and preventing anesthesia compromise through the surgical site(s). As the air sac system does not collapse, it is also practical to insert telescope and rigid instruments through the same incision without cannulae, although triangulation becomes more challenging and active scavenging of anesthetic gas from the surgical site is required.

These instruments, and the cannulae through which they pass, are grouped into various size classes that are often color coded. For example, 2-mm instruments are used in conjunction with 2.5-mm cannulae, 3-mm instruments with 3.5-mm cannulae, and 5-mm instruments with 6-mm cannulae. The 2.7-mm telescope housed within a 3.5-mm protection sheath will pass through a 3.9-mm cannula, while the unsheathed 2.7-mm, integrated 1.9-mm, or 3 mm telescopes will pass through a 3.5-mm cannula.[2]

Cannulae

Cannulae are made of surgical steel or graphite/plastic construction and have internal leaflet valves (and optional insufflation stopcocks for gas delivery) (Figure 30.6). The metal cannulae (3.9 mm and greater) are more robust and heavier and therefore are reserved for large birds over 10 kg. The plastic-graphite models are lightweight and ideally suited to most smaller birds (i.e., 2.5- and 3.5-mm cannulae for 2- and 3-mm instruments, respectively). The threaded cannula (e.g., Ternamian EndoTIP) has an external screw thread to enable gradual advancement by rotation.[3] These threaded cannulae are available in 3.5, 3.9, and 6 mm sizes and do not require a trocar or much in the way of axial penetrative force for insertion.

Rigid instruments and handles

At least one, often two, and rarely three instruments are inserted through cannulae, and into the endoscopic field of view. Instruments must be triangulated to bring the instrument tips together (convergence) to enable surgery within the coelomic cavities. There is a huge assortment of 5-mm instruments available, initially developed for humans but equally applicable for animals over 10 kg. However, it was not until the development of human pediatric laparoscopy that smaller 2- and 3-mm instruments became available, and multiple-entry endosurgery became feasible in most birds.[4]

Currently, 2-mm instruments are limited to Babcock forceps, Reddick-Olsen forceps, and scissors. A greater selection of 3-mm instruments is available including a variety of dissection forceps, grasping forceps, scissors, biopsy forceps, palpation, and irrigation probes (Figure 30.7). All these instruments have a standard attachment that enables them to be used interchangeably with a selection of ClickLine handles (Figure 30.8). Handles are of plastic or metal construction and can possess a radiosurgical connection that enables scissors or forceps to be used as monopolar devices. An optional hemostat or Mahnes (disengageable) rachet mechanism is available to maintain a firm hold of tissue even if the endoscopist releases their grip on the handle.

Surgical devices and hemostasis

To effectively and safely perform endosurgery, accurate hemostasis becomes increasingly critical as the mere act of clamping

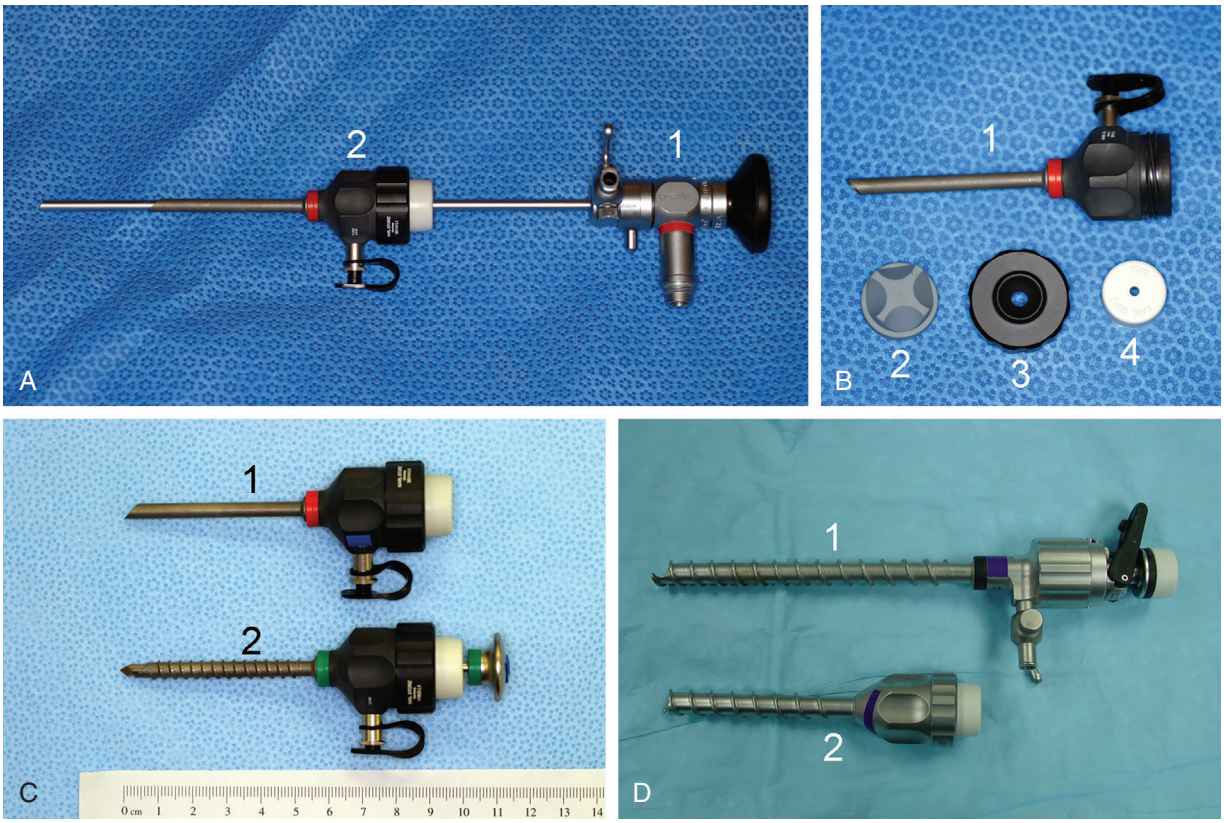


FIGURE 30.6 Cannulae and trocars. (A) A 2.7-mm telescope within a 3.5-mm protection sheath *(1)* inserted through a 3.9-mm × 10-cm graphite/plastic cannula with an insufflation side port *(2)*. (B) A 3.9-mm × 10-cm graphite/plastic cannula disassembled to illustrate the graphite cannula *(1)*, leaflet valve *(2)*, screw cap *(3)*, and instrument seal *(4)*. (C) A 3.9-mm × 10-cm graphite/plastic cannula with an insufflation side port *(1)*, and a 3.5-mm × 10-cm threaded cannula with an insufflation side port and a trocar inserted *(2)*. The 3.9-mm cannula can accommodate the 2.7-mm telescope housed in a 3.5-mm protection sheath while the 3.5-mm cannula can accommodate 3-mm instruments, and thanks to the threaded design, it resists dislodgement in small exotic species. (D) Ternamian EndoTIP cannulae; a 6-mm × 15-cm with an insufflation side port and a multifunctional valve *(1)*, and a 6-mm × 10.5-cm cannula with a silicone leaflet valve *(2)*. These metal cannulae are far heavier and best restricted to animals over 10 kg. (Courtesy Dr. Stephen Divers, University of Georgia)

a bleeding vessel or dabbing with gauze may be impossible. Surgical devices including electrocautery, radiosurgery and laser have become available and facilitated the ability to incise and debride internally while controlling hemorrhage. It is important to appreciate that such devices represent an ignition risk within the high-oxygen environment of the air sac system, and considerable care is required to ensure that nothing flammable is present (e.g., alcohol associated with surgical site preparation).

Diode lasers (e.g., AccuVet Lumenis Inc., Norwood, MA, USA) are by design able to pass through flexible fiber-optic probes that can be inserted through instrument channels or cannulae.[5,6] A variety of diode laser probes are available; however, 400 to 600 μm conical or flat tips at 2 to 10 W are generally most useful (SurgiMedics Inc., The Woodlands, TX, USA). Until recently CO_2 lasers (AccuVet Lumenis Inc.) could not be used via endoscopic instrument channels because of the inflexible nature of the ceramic delivery probes. The development of a long semirigid probe (AccuVet Lumenis Inc.), however, has enabled the use of the CO_2 laser via the 1.7-mm instrument channel of the 4.8-mm operating sheath, but, although functional, it is rather cumbersome. Probably the most versatile, especially when using ≤3-mm instrumentation systems, is electrocautery/radiosurgery because a wide variety of tools are available for use with foot pedal–activated units (e.g., Autocon III 400, Karl Storz Veterinary Endoscopy America Inc.). The most useful of these include various needle electrodes, bipolar forceps, and retractable polypectomy snares. The degree of power required during endosurgery varies with operating conditions and the instrument being used, but due to the microsurgical nature of most endosurgical procedures, lower settings are generally required compared to open surgery. Considerable growth in endoscopic electrocautery/radiosurgery has led to the availability of many monopolar and bipolar devices. One particularly favored device among human and veterinary endosurgeons is the Ligasure Force-Triad (Covidien, Boulder, CO, USA). This system detects the thickness (by measuring electrical impedance) of tissue to be coagulated and automatically

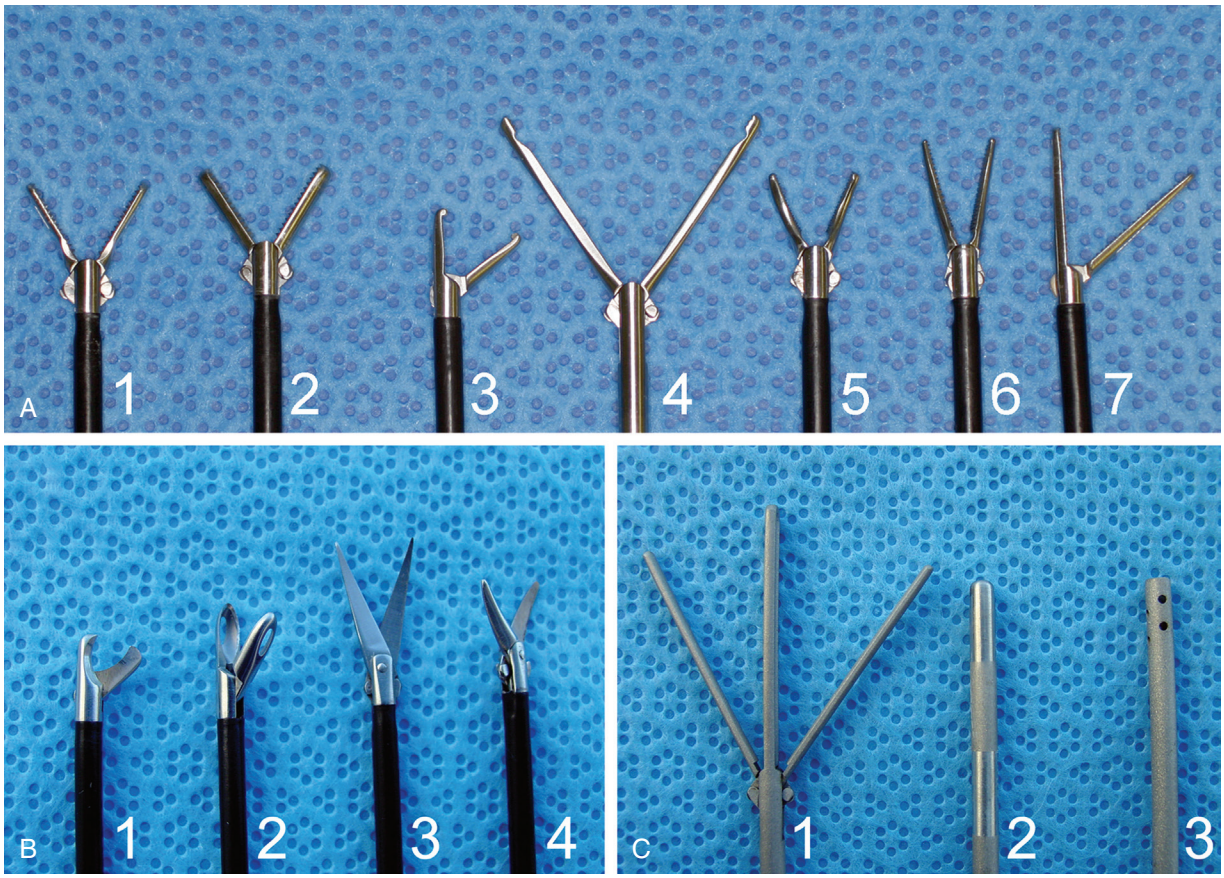


FIGURE 30.7 Images of 3-mm instruments. (A) Forceps—fenestrated atraumatic grasping forceps *(1)*, Reddick-Olsen dissecting forceps *(2)*, small Babcock forceps *(3)*, large Babcock forceps *(4)*, short, curved Kelly dissecting and grasping forceps *(5)*, long curved Kelly dissecting and grasping forceps *(6)*, atraumatic dissecting and grasping forceps with single-action jaws *(7)*. (B) Scissors and biopsy instruments—microhook scissors with single-action jaws *(1)*, Blakesley dissecting and biopsy forceps *(2)*, scissors with long sharp curved double-action jaws *(3)*, scissors with serrated curved double-action jaws *(4)*. (C) Probes—distendable palpation probe *(1)*, palpation probe with centimeter markings *(2)*, irrigation and suction cannula *(3)*. (Courtesy Dr. Stephen Divers, University of Georgia)

defines the amount of energy required and the delivery time. An acoustic signal informs the surgeon when vessel obliteration is complete, and transection is possible. Unfortunately, endoscopic handpieces below 5 mm are not available, limiting its usefulness to 5-mm systems and the largest of birds.

Another useful item from human and domestic animal laparoscopy is the endoscopic vascular clip applicator. These devices contain a cartridge of clips that can be rapidly employed without having to remove from the patient and reload. Again, the smallest available size is 5 mm, which restricts their use to large birds.

DIAGNOSTIC ENDOSCOPY PROCEDURES

Diagnostic endoscopy focuses on the appreciation of normal and abnormal structures within the respiratory tract using magnification and focused light. Furthermore, when abnormalities are identified, specific samples can be collected under direct visual control for laboratory analyses, which typically provide a definitive diagnosis. General anesthesia is required, and close monitoring is essential as these are often critical patients.

One of the greatest benefits of endoscopy is that when an abnormal structure or pathologic lesion is observed, biopsy specimens can be taken under direct visual control. Biopsy specimens can be harvested from virtually any soft structure, taking care to avoid perforating cardiovascular, intestinal, or fluid-containing structures. It is important to try and examine as much of the target structure as possible to determine whether pathology is focal, multifocal, or diffuse. In cases of diffuse lung or air sac disease, biopsy specimens taken from the most convenient sites are preferred. In cases of focal or multifocal disease, single or multiple discrete lesions may be visible and biopsy specimens should ideally be harvested from the edge of the lesion (to acquire both normal and abnormal tissue in the same biopsy sample) or, size permitting, multiple biopsies should be collected for both microbiologic and histologic analysis. Focal disease deep within an organ showing no surface changes, although rare, may be missed on endoscopic examination. The advent of endoscopes with terminal ultrasonography probes is already available in human

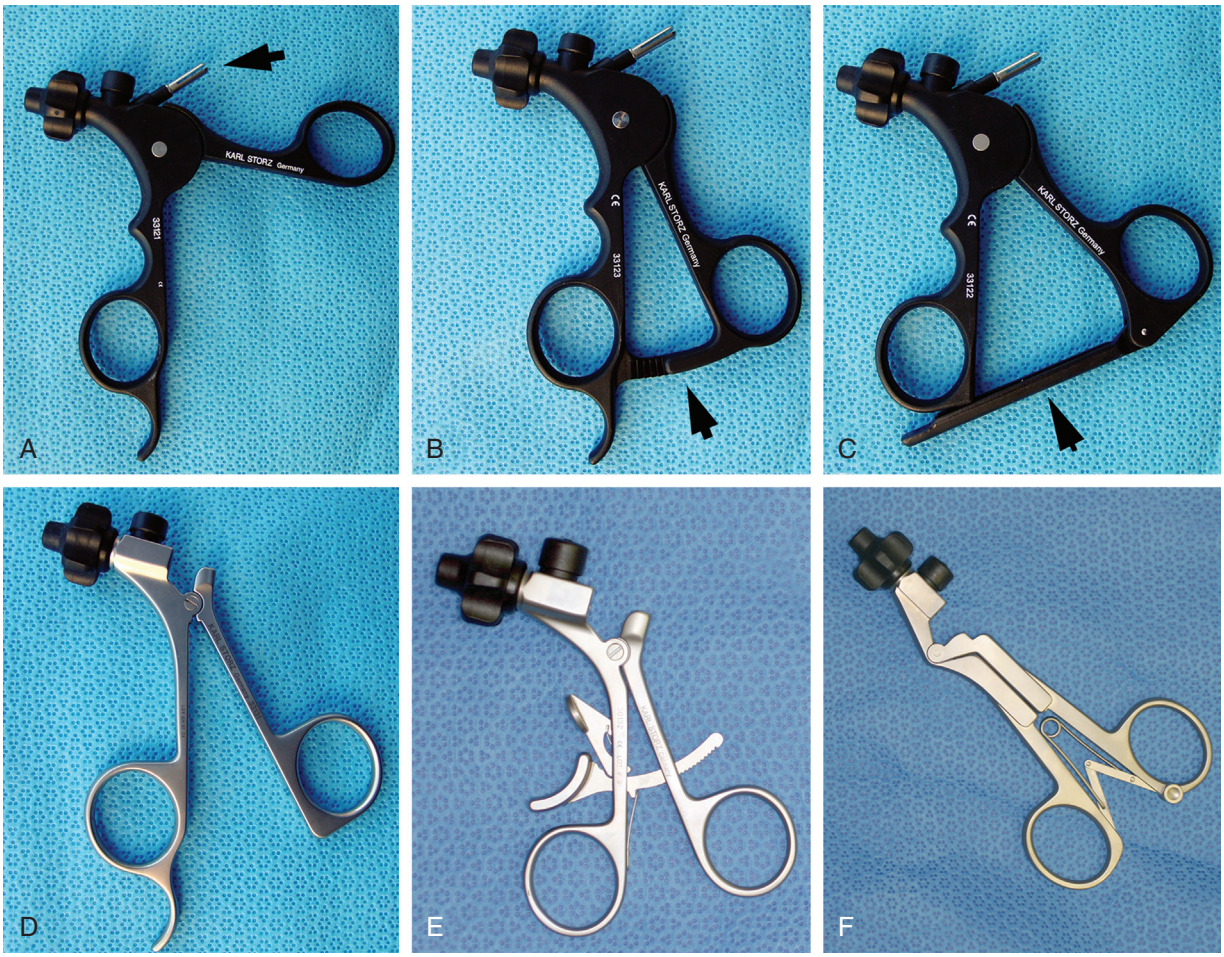


FIGURE 30.8 Endoscopy instrument handles. (A) Plastic handle (without rachet) with radiosurgery connector *(arrow)*. (B) Plastic handle with hemostat-style rachet *(arrow)*. (C) Plastic handle with Mahnes-style rachet *(arrow)*. (D) Metal handle without rachet or radiosurgery connection. (E) Metal handle with disengageable rachet but no radiosurgical connection. (F) Metal Y-handle with spring action. (Courtesy Dr. Stephen Divers, University of Georgia)

medicine. It is important to correlate histopathologic and microbiologic biopsy results with clinicopathologic data when organ disease is present. It is often surprising how biopsy results provide a definitive diagnosis even in the face of unremarkable or conflicting imaging and clinicopathologic data.[7]

When a potentially cystic lesion or abscess is present, it is safer to first attempt fine-needle aspiration rather than risk leakage into the air sacs or coelomic cavities. When confronted by a thickly encapsulated lesion, incising the capsule before inserting the biopsy forceps is often more rewarding. Postsampling hemorrhage tends to be minor and inconsequential, thanks to the extrinsic coagulation pathway of birds.

Rhinoscopy and Sinuscopy

Following insertion of the telescope through the external naris, careful negotiation around the operculum (if present) allows advancement into the nasal cavities. The medial nasal concha are visible dorsad and the caudal nasal concha are more caudal. Pigmentation is variable. It is often possible to visualize the opening to the choanae but not always possible to advance the rigid endoscope down to their level. However, it is not possible to navigate from the nasal conchae into the infraorbital sinus because of the acute turns required to enter the different diverticula of the sinus. However, a focal surgical approach to an infraorbital sinus does permit entry of the telescope and provides far greater visualization than would be possible externally. The endoscope can also be introduced into the oral cavity and the 30-degree deflection aids in examining the dorsal choanae. Again, it is often practical to enter the choanae and direct the endoscope craniodorsad to examine the nasal meatus and conchae from this caudoventral perspective. Biopsies are readily collected for histopathology and microbiology and common pathologies include choanal atresia/dysplasia, rhinitis, and sinusitis (Figure 30.9).

Laryngotracheoscopy

A brief examination (<30 seconds) of the larynx and trachea is possible following mask induction and is useful for a rapid assessment of possible obstruction. However, where more prolonged surgery time is anticipated, placement of a caudal thoracic or—less preferable—abdominal air sac tube to maintain anesthesia is



FIGURE 30.9 Endoscopic views of (A) choanal atresia in a grey parrot *(Psittacus erithacus)*. (B) Nasal meatus and concha as seen from an intraoral view through the choana in an Amazon parrot (*Amazona* spp.). (C) Caseous laryngitis in a green-cheeked conure *(Pyrrhura molinae)*. (Courtesy Dr. Stephen Divers, University of Georgia)

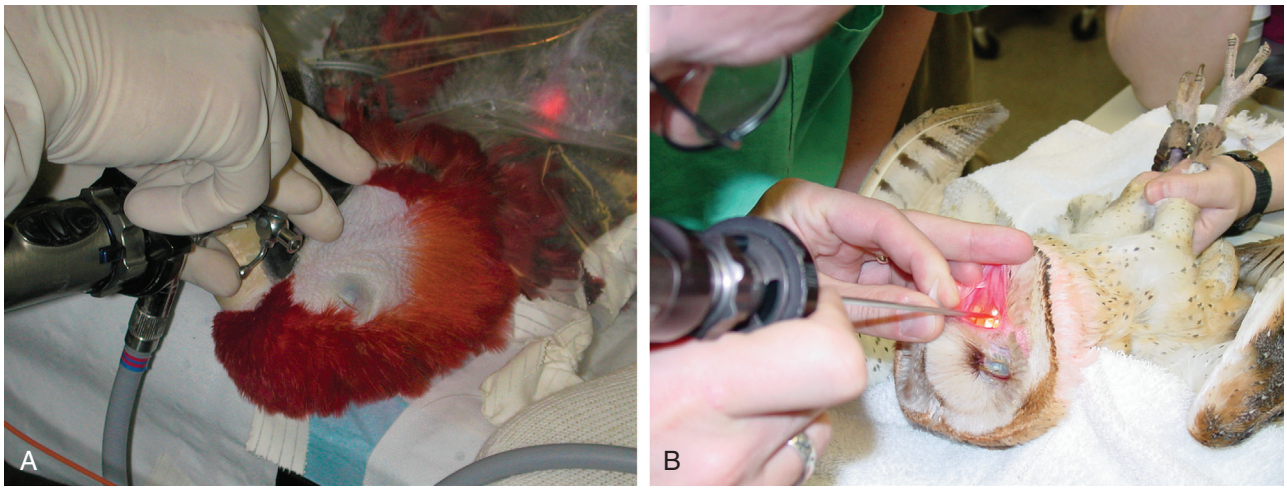


FIGURE 30.10 Tracheoscopy in large birds, such as this scarlet macaw (*Ara macao*) can be performed using the 2.7-mm telescope within the operating sheath (A). However, in smaller birds, such as this barn owl (*Tyto alba*), the naked 2.7-mm or smaller endoscopes must be used (B). (Courtesy Dr. Stephen Divers, University of Georgia)

essential (see Chapter 29). The bird can be positioned in dorsal or ventral recumbency. Examination of the larynx is typically straightforward, and entry through the glottis should be performed with care to avoid damaging the laryngeal cartilages and their associated mucus membranes. Additional caution is required when performing a biopsy of caseous laryngitis (Figure 30.9).

To avoid damage to the tracheal mucosa, the 30-degree telescope should be advanced with the lumen in the lower third of the monitor image while keeping the neck as straight as possible. This is often achieved by the endoscopist holding the head and maintaining mild tension (against an assistant holding the pelvic limbs) to keep the head and neck maximally extended (Figure 30.10). In most birds under 1 kg, it is possible to advance the 2.7-mm telescope down to the tracheal bifurcation and, if present at that location, the syrinx (Figure 30.11). By rotating the telescope around its longitudinal axis, the 30-degree deflection often permits examination of each primary bronchus. In larger birds, small flexible endoscopes can navigate the primary bronchi, pass the parabronchi, and even enter the abdominal air sacs. This is particularly useful in Sphenisciformes and Anseriformes where the relatively reduced volume of the air sacs can make surgical access more challenging. Tracheal modifications of note include anterior bifurcation (e.g., penguins); short loop or coiling (e.g., male magpie geese, both sexes of guinea fowl); long loops and extensive coiling (whistling and whooping swans, cranes); sac-like diverticula (female emus and male ruddy ducks); and bulbous expansions (many male ducks).[8]

As the telescope is advanced, the integrity of the mucosa and any exudates or abnormal membranes or foreign bodies can be appreciated. In large psittacines and raptors, the 2.7-mm telescope can be used with an operating sheath to facilitate instrument entry, whereas in smaller birds it is often necessary to advance instruments alongside the unsheathed telescope (or use a smaller 11.5 Fr or 9.5 Fr integrated endoscope). Foreign body material can be removed with grasping forceps or suctioned, while abnormal tissues or granulomas can be biopsied and submitted for histopathology and microbiology. Samples should also be submitted for immediate cytology, which may direct intraoperative drug therapy.

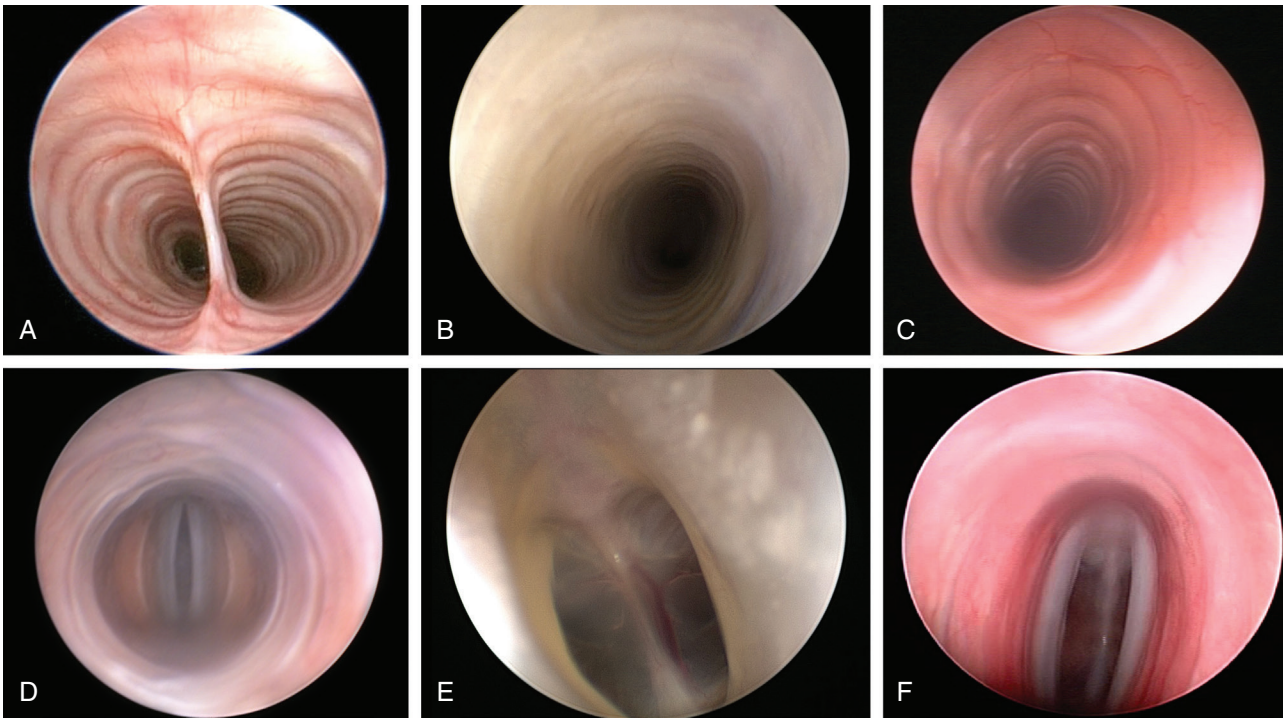


FIGURE 30.11 Tracheoscopy. (A) Bifurcated trachea in a penguin (Spheniscidae). (B) Midtrachea in a pigeon (Columbidae). (C) Midtrachea in a cockatoo (Cacatuidae). (D) Syrinx in a hawk (Accipitridae). (E) Syrinx in a pigeon (Columbidae). (F) Syrinx in cockatoo (Cacatuidae). (Courtesy Dr. Stephen Divers, University of Georgia)

Pulmonoscopy (Lung and Air Sacs)

There are three possible approaches to the lower respiratory system: left, right, and interclavicular. Physical examination and diagnostic imaging (radiography, CT) should be conducted to identify the most appropriate approach. Cross-sectional imaging is particularly valuable when considering entry into the lower respiratory system of a less familiar species or those with relatively smaller air sacs (Sphenisciformes and Anseriformes). Significant ascites represents a contraindication for either lateral approach, as the procedure creates a connection between the hepatic/intestinal peritoneal cavities and the caudal air sacs.

Left and right caudal thoracic air sac approaches

The most common endoscopic approach by convention is the left approach to the air sac system, and therefore unless imaging indicates a predominantly right-sided problem, this preference can prevail. The following description pertains to psittacines, passerines, and raptors, while modifications of this approach are used for aquatic birds. The bird is positioned in right lateral recumbency with wings secured dorsad over the bird's back with the use of self-adhesive tape. In large birds (e.g., macaws) it is advisable to place a towel or foam separator between the wings to reduce ventilatory compromise. The left pelvic limb is pulled craniad and secured to the neck, again with the use of a self-adhesive bandage, to expose the left flank (Figure 30.12). The entry site is located immediately behind the last rib and just ventral to the flexor cruris medialis muscle as it courses from the caudal stifle to the ischium. Few feathers typically need to be plucked before aseptic preparation of the area. Following a 2- to 4-mm skin incision, straight hemostats, directed in a slight craniodorsal direction, are used to bluntly dissect between the thin subcutaneous tissues and enter the caudal thoracic air sac. The hemostats are withdrawn and replaced by the telescope, and the correct position within the caudal thoracic air sac is confirmed by the identification of left lung (straight ahead), left cranial thoracic air sac (left), left abdominal air sac (right), caudal edge of left liver lobe and proventriculus (ventral), and ribs and intercostal muscles (dorsal) (Figure 30.13). The primary ostium of the caudal thoracic air sac has a craniodorsal location, and it is often possible to advance the telescope to examine the lung and parabronchi.

Lung tissue is most accessible from within the caudal thoracic air sacs (Figure 30.13). The air sac and pleural membranes must first be incised with scissors. It is generally easier to rotate the scissors within the instrument channel, such that the fixed blade is dorsal. The scissors-telescope unit is advanced to the target site, and the point of the fixed blade is inserted shallowly through the pleural membranes by advancing the scissors-telescope unit in a ventromedial direction. This creates an opening through the pleura through which biopsy forceps can be inserted to collect lung tissue. While there is typically more hemorrhage from lung biopsies, the pleural membrane acts as a barrier and facilitates clot formation. Common pathologies include hemorrhage, exudates, allergic pneumonitis, and aspergillosis (Figure 30.14).

Exploration of adjacent air sacs is accomplished by pressing the leading edge of the telescope against the air sac membranes and advancing the telescope in a sweeping motion until the air sac membranes are breached. Normal membranes are transparent, and tissues seen through the adjacent air sac can be visualized and avoided. Great care is required when breaking

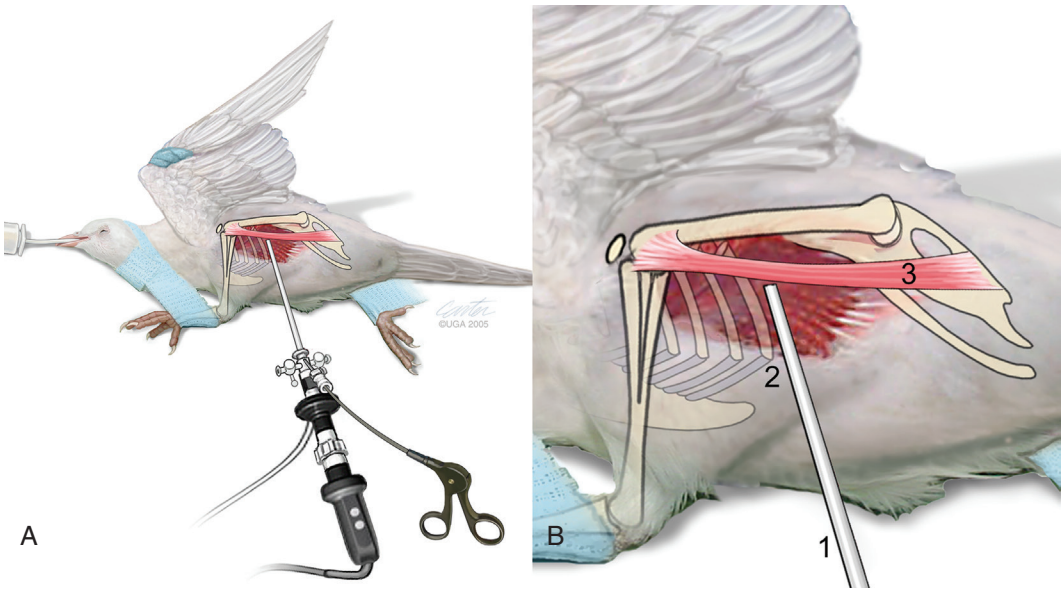


FIGURE 30.12 (A) Bird positioning and entry for left-sided endoscopic examination. (B) Close-up of the telescope *(1)* point of entry behind the last rib *(2)* and just ventral to the flexor cruris medialis muscle *(3)*. (Courtesy Kip Carter, Education Resources, University of Georgia)

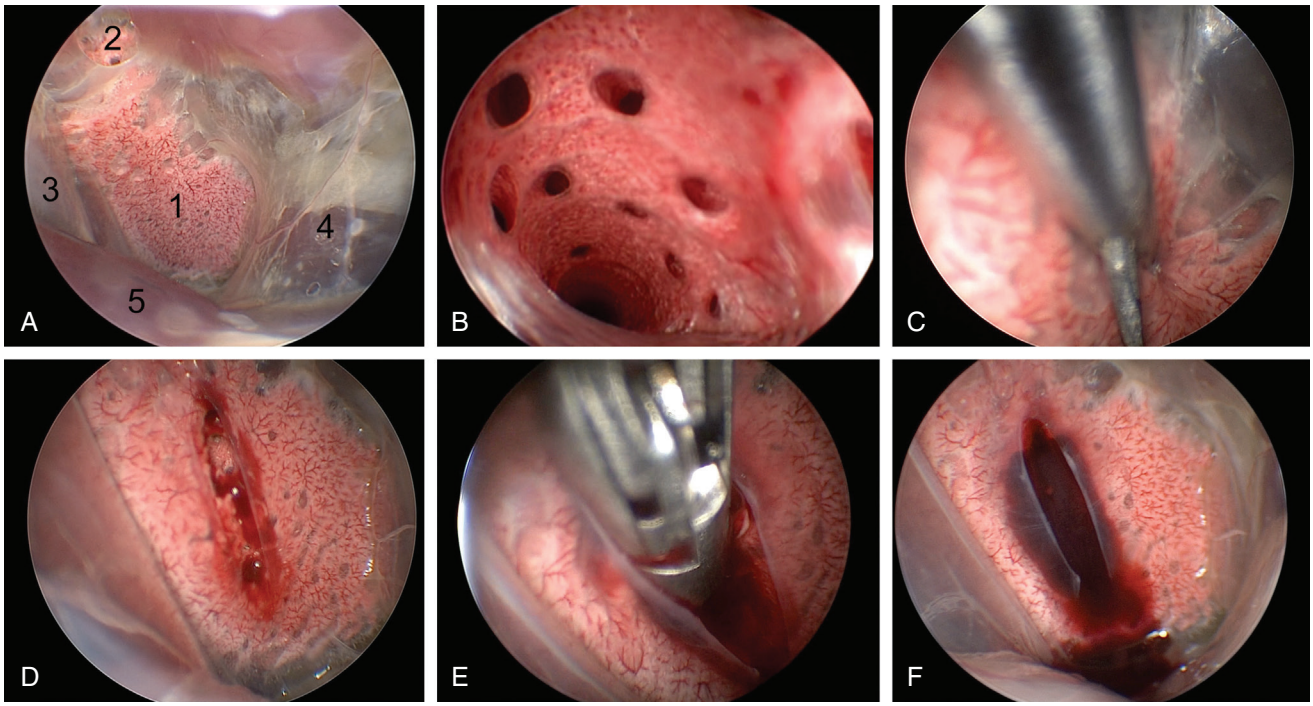


FIGURE 30.13 Endoscopic evaluation and biopsy of the left lung from the left caudal thoracic air sac in a pigeon (Columbidae). (A) Endoscopic view of the ventrolateral lung surface *(1)*, primary ostium of the caudal thoracic air sac *(2)*, cranial thoracic air sac *(3)*, abdominal air sac *(4)*, and ventral liver *(5)*. (B) A view through the primary ostium revealing the lung tissue and parabronchi. (C) Incising the air sac and pleural membranes using scissors to gain access to the lung. (D) Air sac and pleural incision. (E) Advancing biopsy forceps through the incision to take a lung biopsy. (F) Postbiopsy hemorrhage is largely contained by the pleural and air sac membranes and quickly leads to clot formation. (Courtesy Dr. Stephen Divers, University of Georgia)

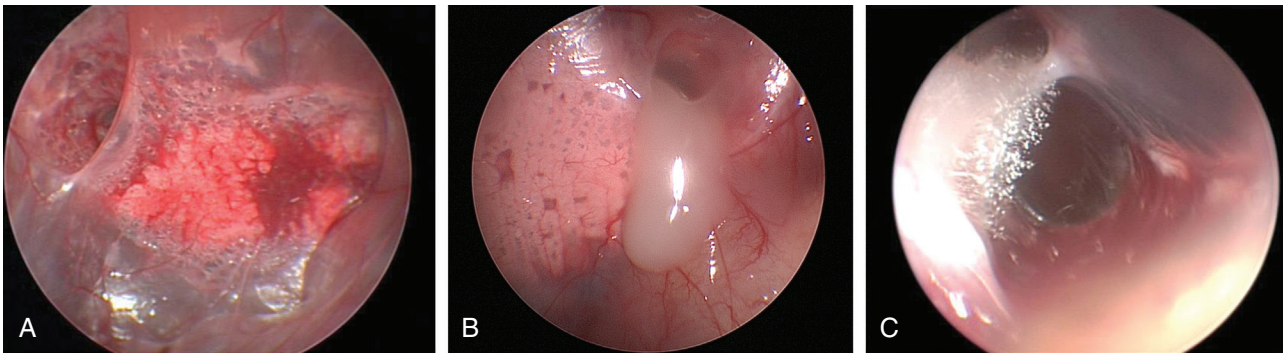


FIGURE 30.14 Endoscopic views of lung pathology. (A) Hemorrhage can often be associated with trauma (e.g., repetitive coughing, dyspnea) or intoxication (e.g., Teflon, anticoagulants). (B) Exudate emanating from the primary ostium of the right caudal thoracic air sac in an owl (Tytonidae). (C) Filamentous hyphae and terminal vesicles consistent with *Aspergillus* within the primary ostium of the caudal thoracic air sac of a macaw (*Ara* spp.). (Courtesy Dr. Stephen Divers, University of Georgia)

through thickened opaque air sacs because vision is impaired, and trauma can occur if the telescope is blindly advanced. In such cases it is safer to incise the air sac or collect full-thickness air sac biopsies before advancing the telescope into the adjacent air sac. Common pathologies include airsacculitis, hemorrhage, exudates, and granulomas (Figure 30.15).

There is no need to repair the small air sac or pleural perforations because they typically heal within 10 days. However, large rents in the air sac will result in permanent connections, which, although insignificant for captive birds, would potentially harm the athletic performance of falconry or free-ranging individuals. Postoperative subcutaneous emphysema may be seen in some birds when only skin closure is performed. Therefore, either a single absorbable (poliglecaprone 25) suture that incorporates both muscle and skin or a two-layer closure is recommended.

The same approach described earlier is used for the placement of an air sac tube. Instead of a protection sheath, a sterile endotracheal tube is placed over the telescope before being advanced into the left caudal thoracic air sac. Once the correct position is confirmed, the endotracheal tube is slid off the telescope into the air sac and secured with suture.

Most right-handed surgeons find the right approach more challenging, but it is primarily indicated if imaging identifies pathology within the right lung or air sacs. The right approach is essentially the same as previously described.

Clavicular air sac approach

The clavicular approach is seldom indicated, except for cases involving the extramural syrinx, clavicular air sac, great vessels, and craniodorsal surface of the heart. This approach requires considerable care due to the proximity of the carotid/brachiocephalic/subclavian arteries, jugular veins, and major nerves. The bird is positioned in dorsal recumbency, and a small towel or gauze is placed under the midscapular region to allow the neck to be placed dorsal to the coelomic inlet. A 2-cm dorsoventral skin incision is made in the coelomic inlet (Figure 30.16). Careful dissection and lateral displacement of the crop (if present) provide access to the coelomic inlet. The clavicular air sac is identified and perforated by grasping and retracting on of the air sac membrane. Perforation by pushing is not recommended due to the likelihood of damaging vital structures. The telescope is advanced, and the distal trachea and external surface of the syrinx are usually obvious. Careful navigation around the syrinx provides visualization of the brachiocephalic and subclavian arteries. Given the proximity to the heart, there are usually rapid movements of all structures in time with cardiac activity. If abnormal structures are identified, then biopsy forceps may be used with caution. Great care is required but this technique has been used to definitively diagnose neoplastic lesions within the cranial coelom (Figure 30.17).

THERAPEUTIC ENDOSCOPY AND ENDOSURGERY

During the last 10 to 15 years, there has been significant development in the evolution of endoscopy from solely diagnostic to include more therapeutic and endosurgical options.[4,9,10] Single-entry endosurgery (e.g., syringeal granuloma removal) is relatively straightforward, but progression to multiple-port endosurgery and the additional demands of triangulation certainly represents a significant increase in procedural complexity and difficulty (Figure 30.18). However, the ability to access remote areas of the coelom and perform procedures that would otherwise be markedly more invasive or simply impossible are major advantages. Advanced training is typically required and regularly offered within the United States and elsewhere.

Tracheal Stenosis and Strictures

Tracheal stenosis and strictures are not uncommon and can be easily identified endoscopically, although determining their etiology is more difficult (Figure 30.19). Endoscopic biopsy from these stenotic lesions typically reveals fibrosis. Despite the relative ease by which these strictures can be debrided (using scissors, retrieval, and biopsy forceps) and airway patency restored, although clinical signs improve dramatically, recurrence can be frustratingly common. In those cases where the lesion(s) extend over just a few tracheal rings, tracheal resection and anastomosis offer the

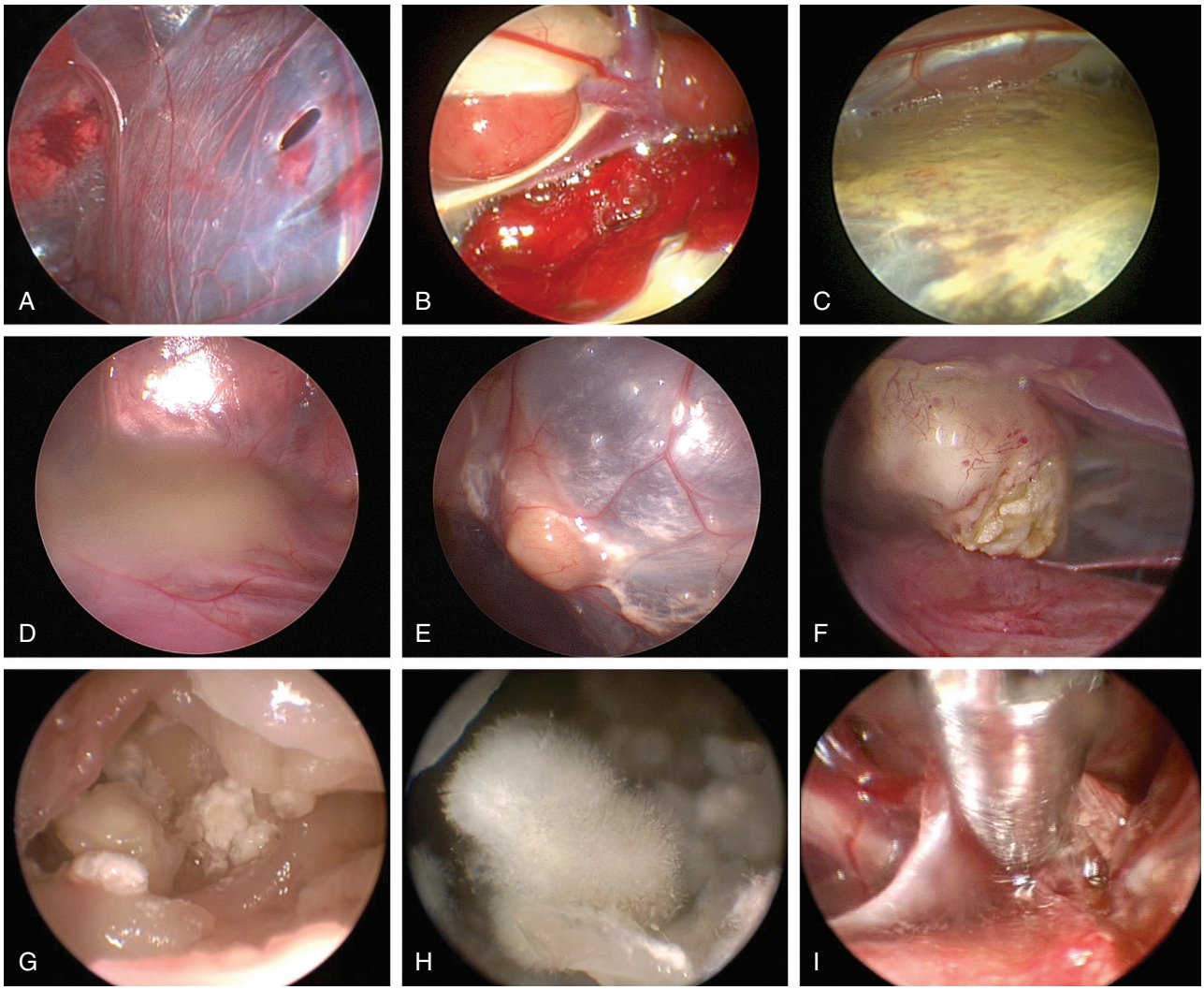


FIGURE 30.15 Endoscopic views of air sac pathology. (A) Inflamed air sacs often appear thickened (less transparent) with more obvious blood vessels. (B) Hemorrhage within the abdominal air sac of a traumatized wild bird. (C) The caudal thoracic air sac of an eclectus parrot (*Electus roratus*) 2 days after air sac tube placement. (D) Liquid exudate on the ventromedial surface of the caudal thoracic air sac in an Amazon parrot (*Amazona* spp.). (E) Solid granuloma attached to the left caudal thoracic and abdominal air sacs in a gyrfalcon *(Falco rusticolus)*. (F) Following biopsy of an air sac granuloma, the avascular nature of the lesion is obvious and helps to explain the poor response to medical therapy. (G) and (H) Left abdominal air sac in a yellow-crowned Amazon parrot (*Amazona ochrocephala*) demonstrating extensive mycosis with filamentous hyphae and terminal vesicles consistent with *Aspergillus*. (I) Biopsy of a grossly thickened air sac. (Courtesy Dr. Stephen Divers, University of Georgia)

greatest chances of success (see Chapter 29). For more extensive cases, the fluoroscopic deployment of a tracheal stent and endoscopic evaluation have proven effective in a parrot for nearly 2 years (Figure 30.20).[11]

Aspirated Foreign Bodies and Syringeal Granulomas

Aspirated foreign bodies often become lodged at the level of the syrinx, which is also the most common site for *Aspergillus* granulomas. Typically, endosurgical debridement using biopsy forceps continues at the time of initial endoscopic diagnosis and biopsy (Figure 30.21). On rare occasions, the entire granuloma may come away with the first biopsy attempt, but more typically, repeated biopsy attempts are required to achieve patency. However, large pieces should not be pulled through the instrument channel or past the endoscope; instead, the telescope should be removed at the same time to prevent breaking up large pieces. Any sign of bleeding indicates the need for greater caution, particularly to avoid damage to the syringeal cartilages and membranes. If the foreign body or granuloma cannot be removed, then pushing the structure into the primary bronchus and into the abdominal air sac for subsequent (endo)surgical removal is preferable to continued blockage of the upper airway (see Chapter 29).

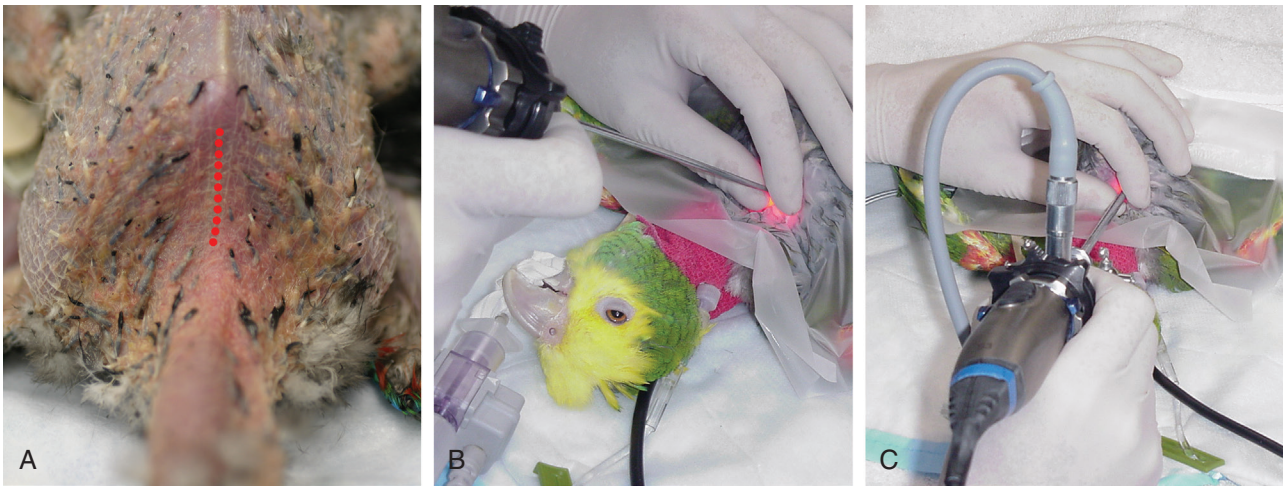


FIGURE 30.16 (A) In preparation for clavicular air sac endoscopy, this feather-picking eclectus parrot (*Eclectus roratus*) has been positioned in dorsal recumbency, with the proposed skin incision indicated. (B) and (C) Following the entry into the clavicular air sac, the unsheathed 2.7-mm telescope is used to carefully navigate in this yellow-headed Amazon parrot *(Amazona oratrix)*. Following the creation of a path, the operating sheath is used to facilitate instrument use. (Courtesy Dr. Stephen Divers, University of Georgia)

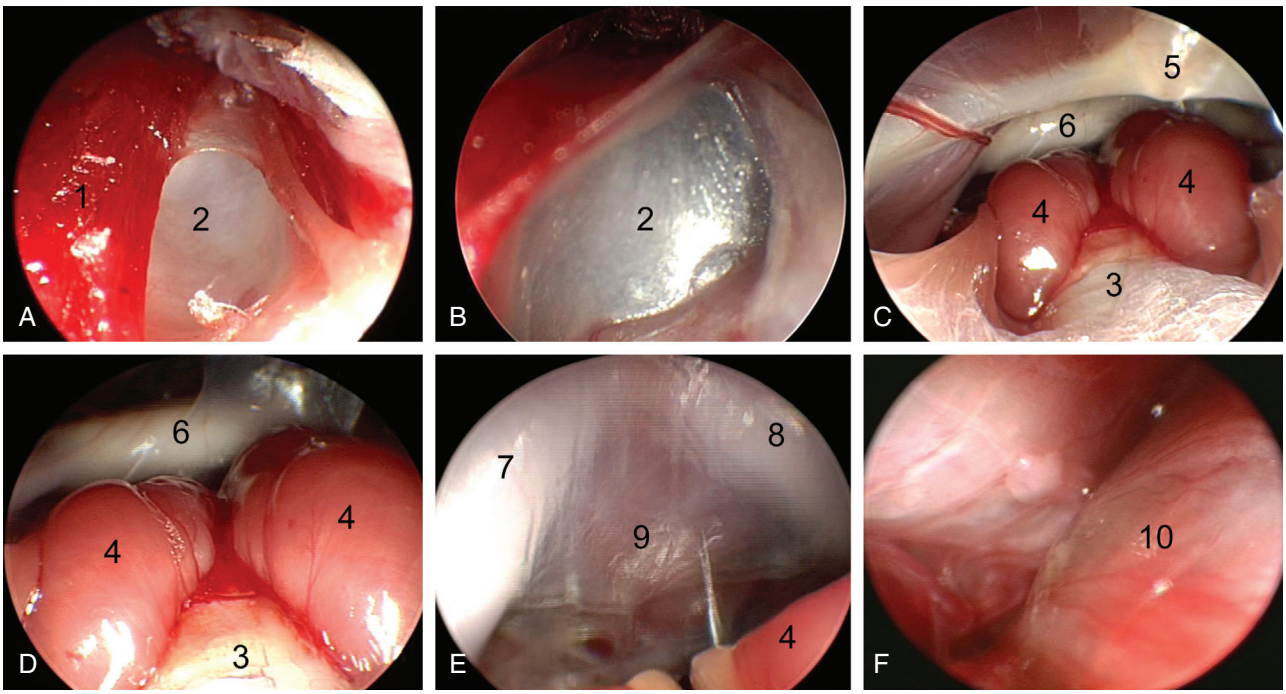


FIGURE 30.17 Clavicular air sac endoscopy in a yellow-headed Amazon parrot (*Amazona oratrix*). (A) and (B). Following the lateral displacement of the crop and subcuticular tissues *(1)*, the clavicular air sac *(2)* is identified and perforated by grasping and retracting, not pushing. (C, D, E, F) Upon entry, the extramural, ventral surface of the syrinx is visible *(3)*, along with the syringeal muscles *(4)*, medial surface of the keel *(5)*, and left subclavian artery *(6)*, left *(7)* and right *(8)* brachiocephalic trunks, heart *(9)*, and clavicular hemangiosarcoma *(10)*. (Courtesy Dr. Stephen Divers, University of Georgia)

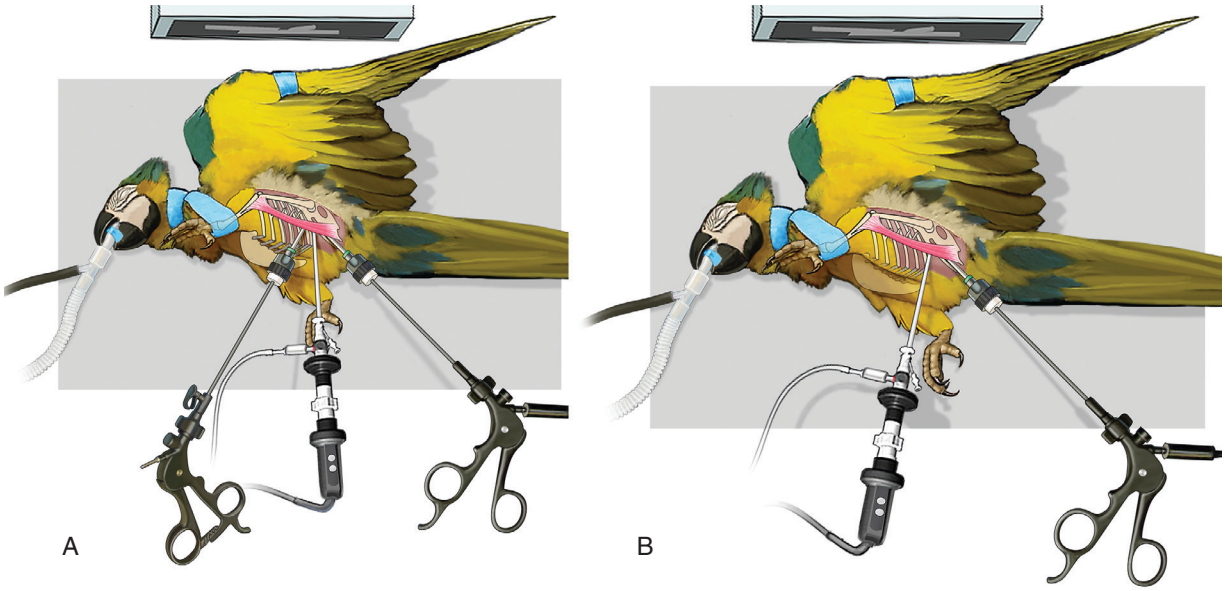


FIGURE 30.18 Diagrammatic representations of (A) three-port and (B) two-port endosurgery in an avian patient. In some cases, two instruments can be inserted through the same coeliotomy approach, and cannulae are not required. (Courtesy Kip Carter, Education Resources, University of Georgia)

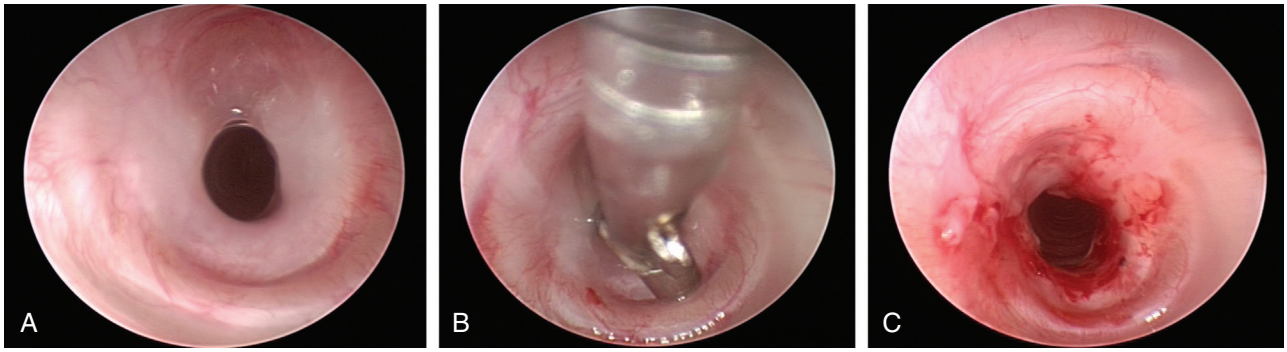


FIGURE 30.19 (A) Endoscopic view of tracheal stenosis in a grey parrot *(Psittacus erithacus)*. (B) Endoscopic debridement using biopsy forceps. (C) Postoperative view. In this case, stenosis did not recur, but recurrence is not uncommon. (Courtesy Dr. Stephen Divers, University of Georgia)

Withdrawing infected material up the trachea has not resulted in multifocal disease spread.

Following debridement of a granuloma, the area is sprayed with either aminoglycoside (bacterial abscess) or an intravenous formulation of an antifungal (mycotic granuloma) delivered via the remote injection needle. It is often beneficial to dilute these drugs to create a large enough volume, often 1 to 3 mL, to flood the area. Neither amphotericin B nor aminoglycosides are well absorbed across the respiratory epithelium and therefore double the recommended systemic dose appears well tolerated within the respiratory tract.

The air sac tube must be maintained postoperatively as there is often pronounced tracheal swelling; however, it is helpful to place a filter over the tube to reduce contamination of the air sacs. Even so, it is important to remove these air sac tubes within 2 to 3 days, and to that end repeat tracheoscopy should be performed 24 to 48 hours after initial surgery. If there is adequate patency then the air sac tube should be removed; if not, further debridement is undertaken. Tracheoscopy is again repeated in 10 to 14 days to determine recovery and whether therapy can be discontinued. Syringeal granulomas that are endosurgically debrided can be completely resolved within 7 to 14 days, thereby avoiding protracted antifungals for weeks or even months.

Lung and Air Sac Aspergillosis, Masses, and Granulomas

Air sac granulomas have been successfully removed in several parrots using a combination of endoscopic debridement and diode laser ablation.[5] Because of the avascular nature of granulomas, debridement with a 5 Fr biopsy forceps is possible without additional hemostasis (Figure 30.22). Lasers and radiosurgery have been used to ablate and sterilize infected areas. When dealing with small masses and granulomas,

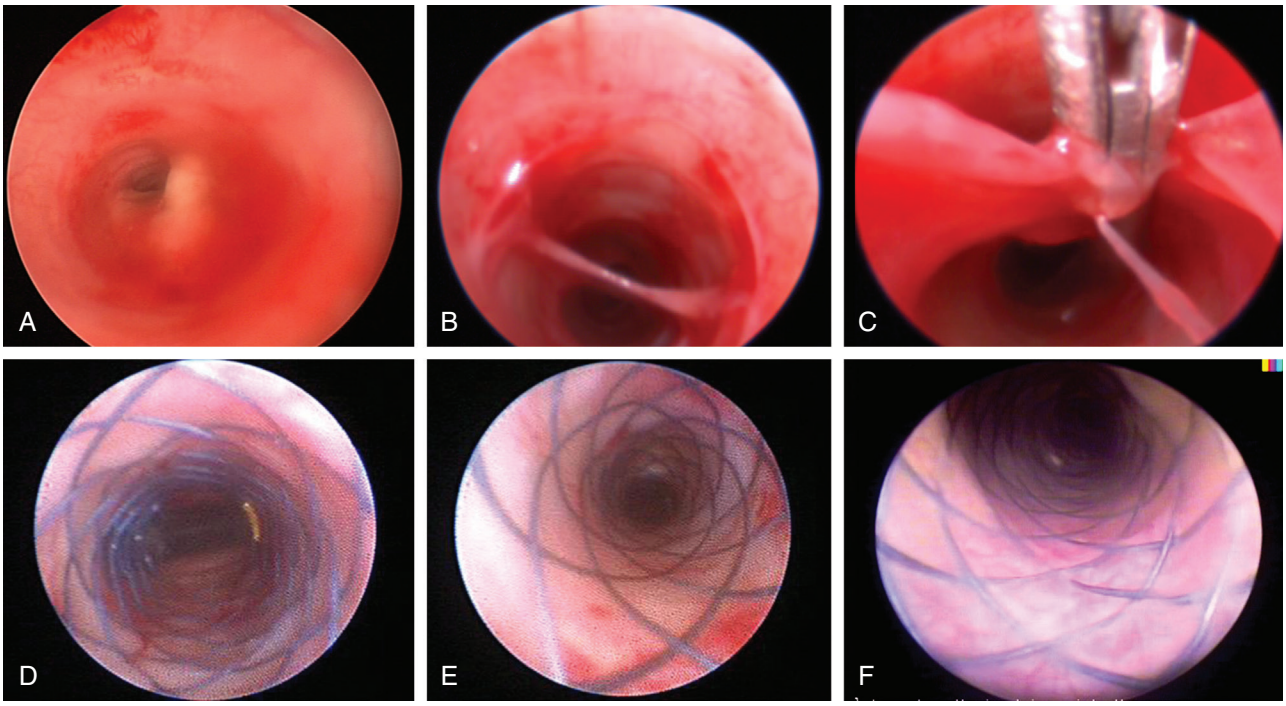


FIGURE 30.20 (A) and (B) Extensive tracheal stenosis and strictures in an eclectus parrot *(Eclectus roratus)*. (C) Debridement using biopsy and retrieval forceps resolved the dyspnea temporarily but the membranes recurred. (D–F) Endoscopic evaluations of a fluoroscopically delivered custom-made wire stent that resolved clinical signs for 2 years. Note how the mucosa has grown over the wire. (Courtesy Dr. Stephen Divers, University of Georgia)

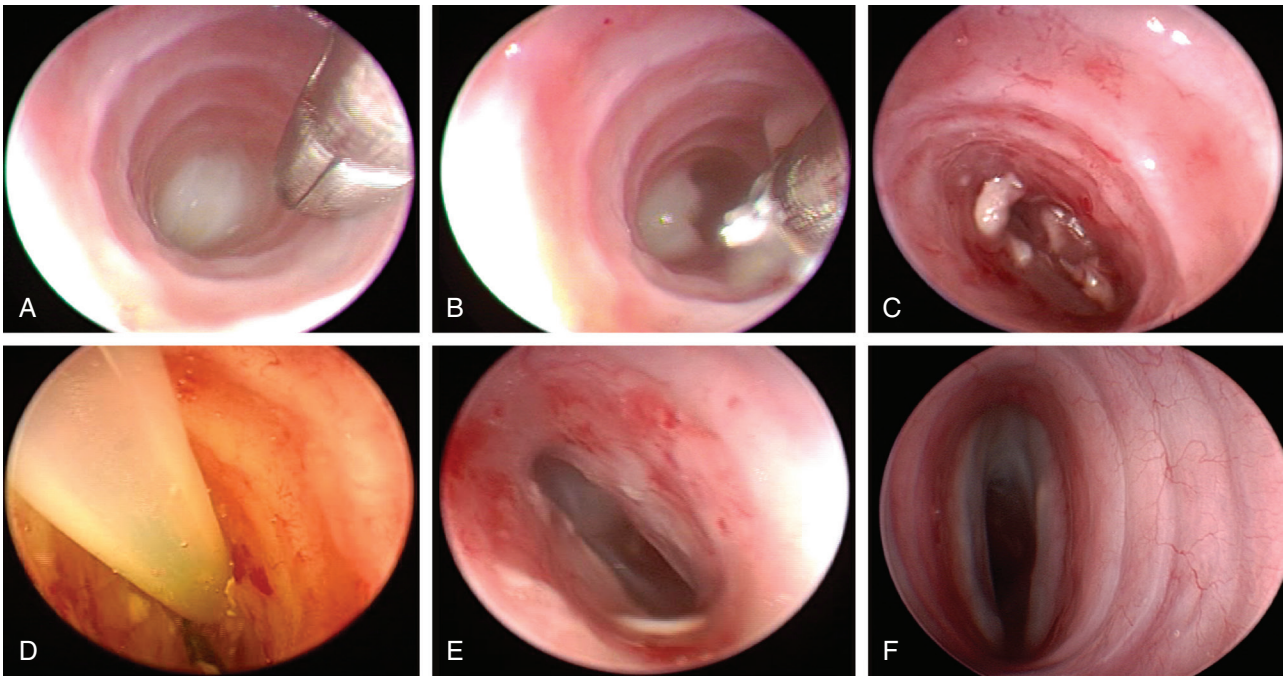


FIGURE 30.21 (A) Syringeal granuloma in a Moluccan cockatoo (*Cacatua moluccensis*) anesthetized using an air sac tube. (B) Endosurgical biopsy and debridement. (C) Postoperative view of the syrinx. (D) Flooding the syrinx with amphotericin B as intraoperative cytology confirmed mycosis. (E) Re-examination of the syrinx 2 days later. There is still some exudate and inflammation, but the syrinx is patent and so the air sac tube was removed. (F) Re-examination of the syrinx 14 days later demonstrated complete resolution, and all medications were discontinued. (Courtesy Dr. Stephen Divers, University of Georgia)

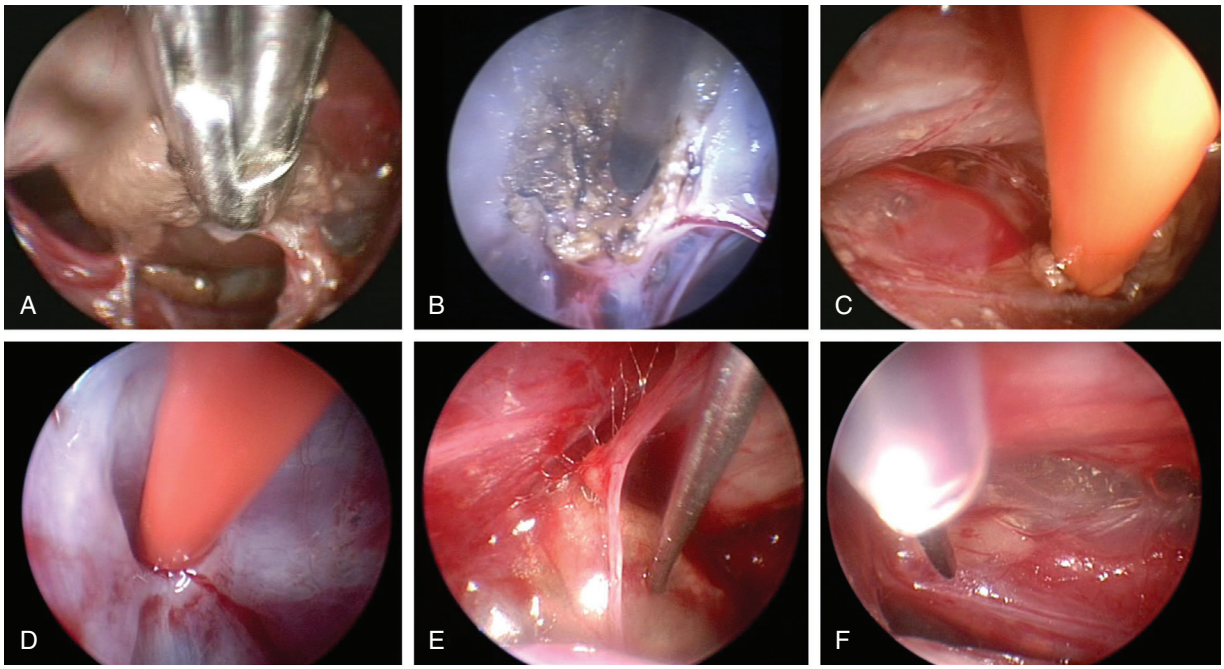


FIGURE 30.22 Single-entry endoscopic therapeutic techniques within the air sac system. (A) Debridement and removal of caseous debris. (B) Diode laser ablation of a debrided granuloma. (C) Liquid exudate aspiration from an air sac using a 5 Fr red rubber catheter. (D) Aspiration of exudate from the primary ostium using a 5 Fr red rubber catheter. (E) Intralesional injection of antifungal medication into a granuloma. (F) Spraying a debrided surgical site with an injectable antimicrobial solution. (Courtesy Dr. Stephen Divers, University of Georgia)

resection is often best accomplished with the biopsy forceps and the telescope, and hemostasis is rarely a concern. However, it is often more practical to remove forceps and the telescope together than to risk fracturing material by attempting to pull it through the instrument channel of the sheath.

When dealing with more widespread air sac mycosis, debridement should continue until most of the material has been removed; however, surgery may have to be prematurely curtailed due to patient risks and anesthesia-related concerns. Diligence is required to avoid contaminating adjacent air sacs or the coelomic surgical access with infected material. Before closure, the area is sprayed with an intravenous formulation of an antimicrobial medication as described earlier in Laryngotracheoscopy. In the case of drugs like amphotericin B, which are poorly absorbed across the respiratory epithelium, up to double the daily systemic dose can often be used, but the volume should be diluted (often 1–4 mL) to allow dispersal throughout the infected air sac. Following 1 to 2 weeks of parenteral antifungal therapy, repeat endoscopy will usually demonstrate consolidation of the active vegetative lesions into a more organized granuloma. Intralesional injections of antifungal medication into these granulomas can result in further size reduction but are seldom curative, ultimately necessitating endosurgical removal.

Double- and triple-entry techniques have been used to endoscopically sample, debulk, and remove coelomic cavity neoplasms, abscesses, and granulomas from the respiratory tract (Figure 30.23). The use of 3-mm biopsy and debriding forceps facilitates faster tissue removal when compared to using 5 Fr biopsy forceps. A combination of instruments is often beneficial for incision, blunt dissection, and removal of solid material. Suction probes are useful when fluids are encountered after opening a granuloma. Generally, a surgical assistant holds the telescope so that the primary surgeon can focus on endosurgery, and this becomes essential when two instruments are being used simultaneously (Figure 30.24). Care is required to remove as much gross material as possible, including from around the surgical site. While systems exist in human and domesticated laparoscopy to resect and remove large masses without causing contamination, this is far more difficult in the confines of the coelomic cavities of birds. Therefore, the endoscopist must take precautions to remove as much material as possible and be diligent in trying not to seed infection along the instrument tracts. In cases of bacterial or fungal granulomas, it is often beneficial to treat locally (by intralesional injections/spray) following endosurgical debridement.

CONCLUSIONS

Endoscopy, endoscope-assisted therapy, and endosurgery offer significant advances over other diagnostic and treatment options. No other techniques can provide the same visual acuity, ability to collect biopsies, and minimally invasive approach to therapeutic and surgical interventions. The value of endoscopy is entrenched within human and domesticated animal pneumonology and is well-documented in avian respiratory medicine.

ACKNOWLEDGEMENTS

The author is grateful to Karl Storz Veterinary Endoscopy for their continued support in both endoscopic research and avian procedural development.

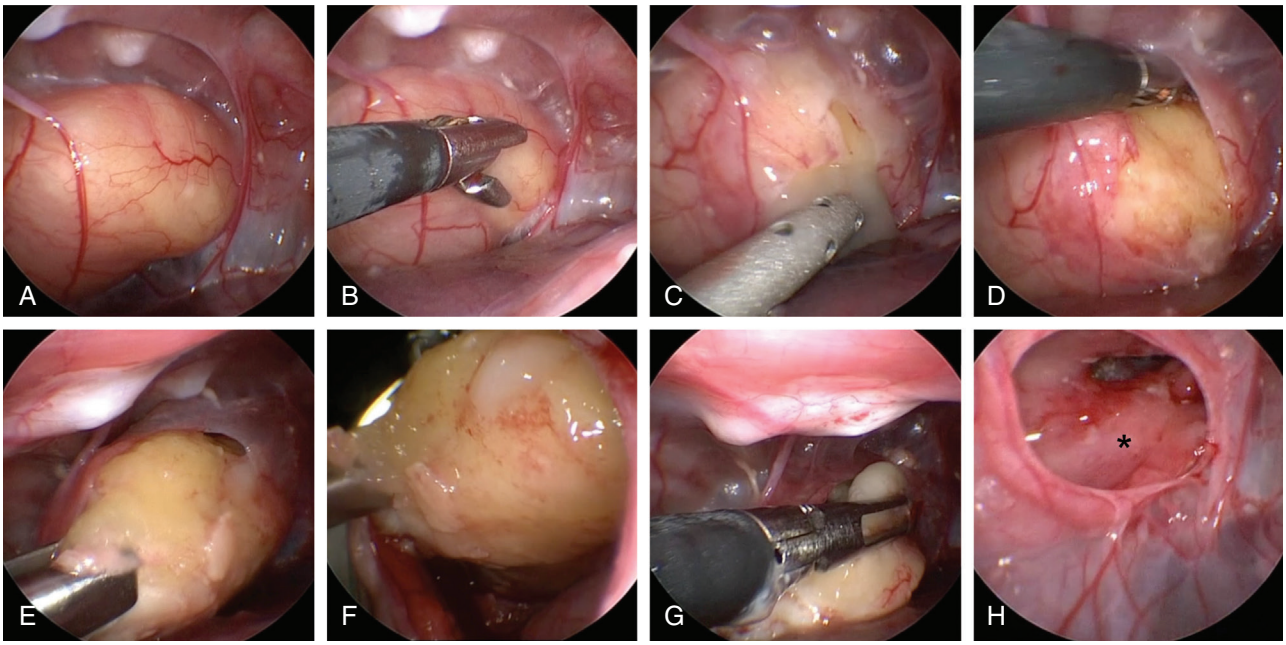


FIGURE 30.23 Endosurgery in a gyrfalcon *(Falco rusticolus)* using a 2.7-mm telescope and 3-mm instruments. (A) *Aspergillus* granuloma is attached to the heart and seen from within the cranial thoracic air sac. (B) Initial breach of the thick fibrous capsule using biopsy/debriding forceps. (C) Aspiration of liquid exudate using a fenestrated suction probe. (D) Blunt dissection around the caseous material to separate it from surrounding tissues. (E) and (F) Grasping and removal of a large piece of the granuloma using Babcock forceps. (G) Removing the remainder of the material using atraumatic forceps. (H) View of the debrided granuloma and closely associated heart (*), prior to spraying the area with amphotericin B. (Courtesy Drs. Stephen Divers and Izidora Sladakovic, University of Georgia)



FIGURE 30.24 Endosurgery in the same gyrfalcon *(Falco rusticolus)* utilizing a primary surgeon and a surgical assistant. (Courtesy Drs. Stephen Divers and Izidora Sladakovic, University of Georgia)

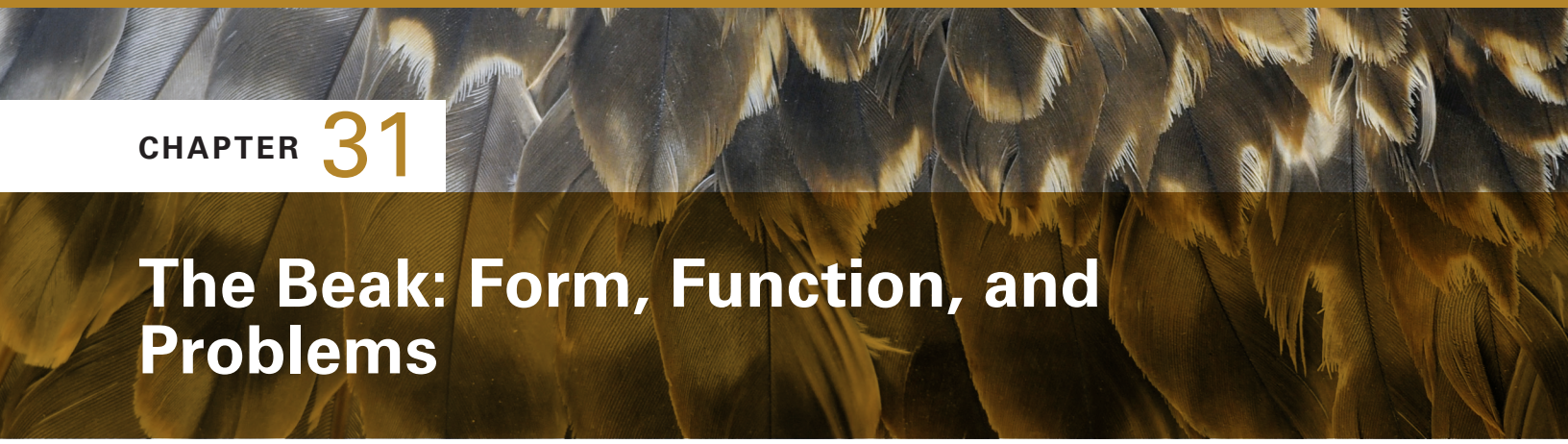
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CHAPTER 31

The Beak: Form, Function, and Problems

Brian L. Speer • Roberto S. Fecchio • Casey M. Holliday

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The anatomy and function of the heads and beaks of birds are complex, but there are some shared common features on top of many unique adaptations and niche functions specific to species groups. The beak of birds serves multiple functions related to eating and procuring food, deglutition, grooming, killing prey, fighting, courtship, thermoregulation, locomotion, nest construction, and the feeding of young. Several disease conditions can result in direct or secondary damage to the bony and soft tissue structures of the beak. These injuries can lead to secondary problems such as beak deviation, abnormal keratin growth and wear, and opportunistic infections. A sound understanding of the species-specific anatomy, accuracy of the diagnosis established, and clarity of the medical and surgical options that may be employed are essential when contemplating treatment.[1–6]

ANATOMY AND FUNCTION OF THE BEAK

Topographical Anatomical Terminology

The bill, or beak, of birds, is known anatomically as the rostrum. It includes the bones of the maxillary and mandibular beak, dermal and other soft tissues, and horny sheaths. The upper component of the bill is the maxillary rostrum *(rostrum maxillare)* and the lower component is the mandibular rostrum *(rostrum mandibulare)*.[7,8] The rostral parts of the upper and lower beaks of birds are covered by a hard, epidermal structure derived from keratin. Synonyms for this keratin covering are the horny bill and rhamphotheca. Synonyms for the upper and lower horny sheaths are the maxillary rhamphotheca (or rhinotheca) and the mandibular rhamphotheca (or gnathotheca), respectively.[8] The mid-dorsal profile of the maxillary rhamphotheca is the culmen. The maxillary rhamphotheca has a cutting edge or tomium on the left and right side (the maxillary tomia), which matches the opposing mandibular tomium. The rostral tomia of the maxillary and mandibular rhamphotheca do not line up in all birds depending on their diet and feeding behaviors. There is an inner occlusal rhinothecal ledge that facilitates force delivery from the rostral gnathothecal tomium against food items, particularly prominent in most Psittaciformes.[8] The midventral profile of the whole length of the mandibular rhamphotheca is termed the gonys. The rhamphotheca is a constantly growing and dynamic structure. The inter-ramal region, or inter-ramal space, is the zone of soft tissue that begins at the caudal end of the gonys and extends caudally between the mandibular rami as far as the end of the mandible. The caudal portion of the inter-ramal space is the gular region, which forms the floor of the caudal part of the oropharynx.[8] The entire opening of the mouth is called the oral opening or gape. The rictus is the caudal part of the oral opening, beginning at the caudal end of the tomia and ending caudally at the angle of the mouth.[8] These terms and their synonyms are displayed in Table 31.1 and Figure 31.1.

Osteology and Joints

The bony upper beak is formed by paired and fused premaxillary and nasal bones. In most birds, the upper beak functions as a rigid triangular block, which can be elevated or depressed.[7,9] The upper beak hinges in many bird species at the flexible junction of the upper beak with the neurocranium at the craniofacial hinge, or nasofrontal hinge. In parrots, this joint is in part a synovial joint, yet with a bridge of chondroid bone near the midline and is also known as the ginglymus. Movement of the upper beak requires accompanying movement of four pairs of bones: the jugal arch, the pterygoid, the palatine, and the quadrate bones. Movement of the bony upper beak is also made possible by the presence of flexible elastic zones in the facial bones known as streptognathism. In prokinetic birds, a short elastic zone called the craniofacial elastic zone occurs at the junction between the braincase and

TABLE 31.1

Terms and Common Synonyms Used to Describe Beak Topographical Anatomy, Function, and Types of Prosthetics

Anatomic Term	Synonyms
Rostrum	Bill or beak
Maxillary rostrum	Upper bill or beak
Mandibular rostrum	Lower bill or beak
Rhamphotheca	Keratin sheath of the beaks, horny bill
Rhinotheca	Maxillary rhamphotheca Keratin sheath of the upper bill
Gnathotheca	Mandibular rhamphotheca Keratin sheath of the lower bill
Tomium (singular), tomia (plural)	Rostral and lateral cutting edges of the rhamphotheca
Maxillary tomium, tomia	Rostral and lateral cutting edges of the maxillary rhamphotheca
Mandibular tomium, tomia	Rostral and lateral cutting edges of the mandibular rhamphotheca
MIDLINE BEAK PROFILE TERMS	
Culmen	Mid-dorsal profile of the maxillary rhamphotheca
Gonys	Midventral profile of the mandibular rhamphotheca
OTHER TOPOGRAPHIC TERMS	
Gape	The entire opening of the mouth
Rictus	Caudal part of the oral opening, beginning at the caudal end of the tomia and ending caudally at the angle of the mouth
Inter-ramal region or space	Zone of soft tissue beginning at the caudal end of the gonys, extending between the mandibular rami as far as the caudal end of the mandible
Gular region	Caudal portion of the inter-ramal space
Naris (singular), nares (plural)	Nostril, nostrils
FUNCTIONAL TERMS	
Protraction of the maxillary beak	Elevation of the maxillary rostrum
Retraction of the maxillary beak	Lowering of the maxillary rostrum
Protraction of the mandible	Movement of the mandible rostrally
Retraction of the mandible	Movement of the mandible caudally
Cranial kinesis	The relative movement between the upper beak, bones of the palate, and the braincase
Prokinesis	The upper beak is elevated about a clear craniofacial hinge joint, usually through movements of the palate powered by jaw muscles
Rhynchokinesis	The upper or lower beaks can flex, either through deformation about craniofacial joints or within the bones themselves (i.e., streptognathism)
Amphikinesis	The upper beak elevates or depresses through a series of internal rhynchokinetic movements of the craniofacial bones
Coupled kinesis	Movements of the upper and lower beaks are somewhat dependent on one another, often through the powering activities of jaw musculature and the linkage system imparted by the palatal bones
PROSTHETIC TERMS	
Autologous	Tissue derived from the same individual
Homologous, allogenic	Tissue originating from another individual of the same species
Heterologous, xenogenic	Tissue originating from an individual of a different species
Synthetic	Made with inorganic materials
Endoprosthesis	Prosthesis is fully implanted and surrounded by body tissue
Exoprosthesis	Prosthesis is worn outside of the body

the nasal and premaxillary bones. This joint varies in its degree of flexibility among avian taxa and can be sutured, synovial, or bridged by chondroid bone.[7,9–11] The palate is formed by a combination of the palatal processes of the premaxillae and maxillae, the palatine bones, the vomers, the jugal arches, and the pterygoid and quadrate bones.[7,9,10] Unlike the palate of mammals, the palate of birds does not form a complete shelf between the nasal and oral cavities. The internal nares lie between the vomers and the palatine bones, rostrally. The pterygoid articulates rostrally with the rostral portion of the palatine bone and caudally with the quadrate bone in a synovial hinge joint.[7,9] The jugal arch articulates rostrally with the maxilla and caudally, it forms a ball-and-socket joint with the quadrate bone.[7,9] The joint formed between the quadrate and the braincase is a double articulation. Rotation of the quadrate bones rostrally pushes on the pterygoid and jugal arches rostrally, resulting in an upward push being applied to the palatine bone and the palatovomeral complex. This in turn causes it to slide rostrally about the palatobasal joint formed with the central bony ridge of the ventral braincase (os

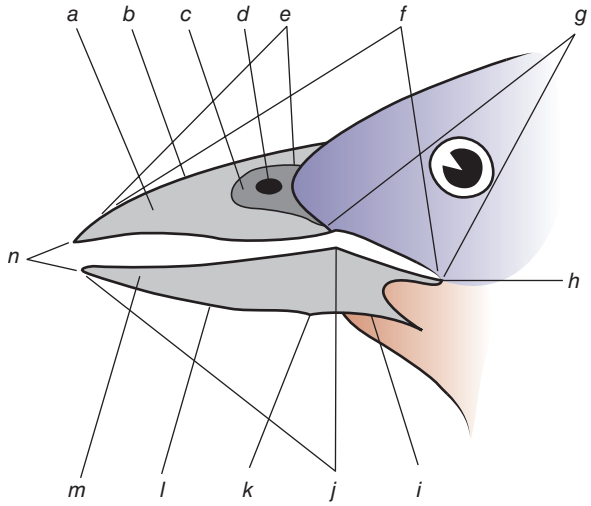


FIGURE 31.1 Parts of a bird's bill. *a*, side of upper mandible; *b*, culmen; *c*, nasal fossa; *d*, nostril; *e*, tomia of the upper mandible; *f*, gape, or whole commissural line; *g*, rictus; *h*, commissural point or angle of the mouth; *i*, ramus of under jaw; *j*, tomia of under mandible; *k*, angle of gonys; *l*, gonys; *m*, side of under mandible; *n*, tips of mandible.

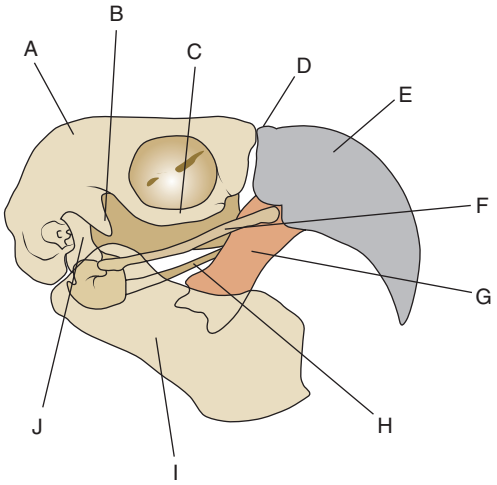


FIGURE 31.2 Illustration of an articulated scarlet macaw skull *(Ara macao)*. *A*, neurocranium (braincase); *B*, postorbital process; *C*, suborbital arch; *D*, craniofacial hinge (nasofrontal hinge, ginglymus); *E*, premaxilla; *F*, jugal arch; *G*, palatine; *H*, pterygoid; *I*, mandible; *J*, quadrate. (From Abramson J, Speer BL, Thomsen JB, editors: *The large macaws*, Fort Bragg, CA, 1995, Raintree Publications.)

mesethmoidale; parasphenoid) and forcing the maxillary rostrum to swing upward at the craniofacial hinge (protraction). This articulation is a diarthrosis with fibrous tissue. Rotation of the quadrate bones caudally causes the maxillary rostrum to swing downwards (retraction). These bony structures are displayed in Figure 31.2.

The jugal arch consists of three bones fused together; from rostral to caudal, they are the jugal process of the maxillary bone, the jugal bone, and the quadratojugal bone.[7,9] There is no true zygomatic arch or bone in birds. The quadrate bones are immensely mobile and function as the key elements in most of the movement of the upper and lower beaks in the prokinetic beak: articulating with the mandibles, the braincase, the jugal arch, and the pterygoid bone.[7,9] The quadrate bone forms the primary link between the mandibles and the neurocranium.[7,9,10] All of these bony elements are coupled mechanically; hence, force applied at any point in the system, either internally by muscle contraction or via external factors, will be transmitted throughout the system and cause deflection of the system in one or the other direction.[7,9] Convention is to consider the quadrate bone as the starting point and to describe all movements as initiated from it.[7] Articular and linkage ligaments unify the function of the mandibles with the braincase, the palate, and the maxillary rostrum.[7,9] Linkage ligaments function to join the upper and lower beaks together mechanically, resulting in coupled kinesis, in which the movements of the upper and lower beaks are somewhat dependent on one another, but not entirely so. The two rami of the mandible are joined in the rostral midline at the rostrum (symphysis) mandibulae.[7] Some birds have intramandibular mobility (e.g., nightjars, pelicans).[12] Intraramal synovial joints and streptognathy in pelicans enable the swallowing of large prey. A similar joint has been described in loons, grebes, albatrosses, penguins, herons, storks, New World vultures, great frigate birds *(Fregata minor)*, and the brown booby *(Sula leucogaster)*.[13] All bones of the skull are pneumatized by air diverticula coming from the tympanic cavity (braincase) or the infraorbital sinus (all other bones), although some species of aquatic birds including penguins, grebes, cormorants, mergansers, and alcids have reduced or absent pneumatization.[14]

Osteology of the juvenile psittacine skull

Juvenile macaw skull osteology can be distinguished from adults by specific unique characteristics. In the juvenile skull, suture lines can be found in the premaxilla, maxilla, nares, prefrontal, frontal, temporal, and parietal regions, as well as along the jugal arch. These bones are in a very active phase of growth and remodeling and, as such, can be guided in their direction of growth easier than in their adult counterparts. Their softer, partially ossified nature also can directly influence the choices of surgical repairs needed for injuries and deformities in these immature birds. The bony upper beak consists of several bones including the premaxilla, maxilla, and nasal bone, which eventually fuse together. The orbital ring is incomplete in all macaws when they hatch. In *Ara* species, the preorbital process continues to grow and joins the postorbital process by the time the bird is 11 months old. This does not occur in the genera *Anodorhynchus* or *Cyanopsitta*.[15] In certain species, such as the blue-and-gold macaw *(Ara ararauna)*, the cranial bones mature more rapidly than in other macaw species. The opening of the nares is quickly reduced to the adult size, and the maxilla retains a midline suture for several months. Other species, such as the green-winged macaw *(Ara chloroptera)*, have clearly visible suture lines for several months and have a significantly larger naris at the same age.[15] The skulls of an adult and juvenile macaw are displayed in Figure 31.3.

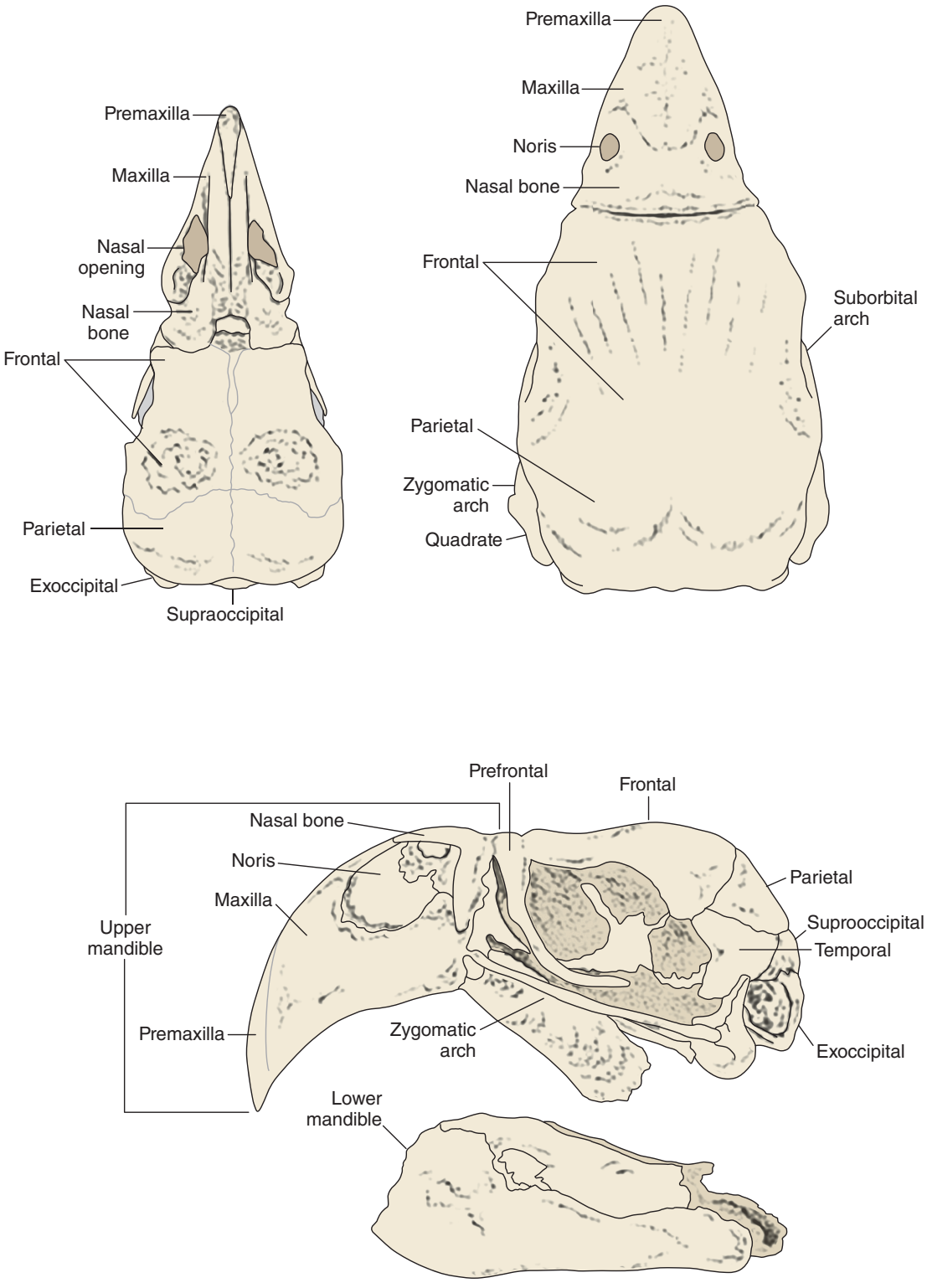


FIGURE 31.3 Illustration comparing osteology of juvenile (lateral and dorsal views) and adult macaw skulls (dorsal view). (From Abramson J, Speer BL, Thomsen JB, editors: *The large macaws*, Fort Bragg, CA, 1995, Raintree Publications.)

The Prokinetic Muscles of the Beaks

In most birds, seven pairs of muscles act on the upper and lower beaks. Parrots have two unique pairs of muscles and lack one of the original seven pairs.[7,10] These muscles are displayed in Figures 31.4 and 31.5; their origins, insertions, and actions are displayed in Table 31.2. Parrots have neither a *m. pseudotemporalis profundus* nor its attachment, the orbital process of the quadrate, while these are present in other bird lineages. Two muscles open the beaks: *m. protractor pterygoidei et quadrati* by raising the upper beak through the linkage system of the palate, and *m. depressor mandibulae* by lowering the lower beak. Two muscles simultaneously close both the upper and lower beaks, i.e., *m. pterygoideus* and *m. pseudotemporalis profundus*. The pterygoideus muscles in parrots also function in the movement of the lower mandible forward (protraction) and in lowering the upper beak during chewing. Three muscles raise or elevate the mandibles: *m. adductor mandibulae externus*, *m. pseudotemporalis superficialis*, and *m. adductor mandibulae caudalis* (*m. adductor mandibulae posterior* in reptiles).[7,16] The two unique pairs of muscles that are present in parrots are *m. ethmomandibularis*, a derived belly of *m. pterygoideus*, and *m. pseudomasseter*, a derived belly of *m. adductor mandibulae externus* (Figure 31.4).[17] *M. ethmomandibularis* closes and protracts the lower beak, and the *m. pseudomasseter* raises the mandible. These muscles can act without eliciting a prokinetic behavior and exist in species without prokinesis. Rostral rotation of the quadrate bone at its articulation functions to raise the upper beak, and this action is accomplished by the contraction of *mm. protractor pterygoidei et quadrati*, *pseudotemporalis superficialis*, *adductor mandibulae caudalis*, and *adductor mandibulae externus*. Extension of the jaw (quadrate-mandibular) joint (depressing the lower beak) is accomplished by contraction of *m. depressor mandibulae*. Closing of the jaw joint (elevating the lower beak) is accomplished by contraction of *mm. pseudotemporalis profundus*, *adductor mandibulae externus*, *ethmomandibularis*, and *pterygoideus*. The superficial prokinetic muscles of the Grey parrot (*Psittacus erithacus*) are displayed in Figure 31.5. The occlusal force applied by these four muscles is greater on objects caudally near the base of the beak and decreases gradually as objects are prehended and manipulated more rostrally, closer to the tip of the beak.

Innervation

In birds, generally the nerve endings of the upper bill are innervated by the maxillary and ophthalmic divisions of the trigeminal nerve (5th cranial nerve; CNV) and those of the lower bill by the mandibular division.[20] The ophthalmic nerve supplies the palate, the edge of the upper beak, and the bill tip organ.[20] The maxillary nerve is purely afferent and supplies receptor endings in the lateral tomia of the upper bill. The mandibular nerve is both somatic afferent and efferent and passes the length of the mandibular canal, providing branches to the skin associated with the lower beak, including the mandibular bill tip organ.[20] The mandibular division of the trigeminal nerve provides the primary motor innervations for the jaw muscles, except for the *m. depressor mandibulae*, which is supplied by the facial nerve (CNVII).[7]

Vascular Anatomy

The common carotid artery is the primary source of blood to the structures of the beak and oral cavity in birds. This artery divides into internal carotid and vertebral arteries, which ascend the neck and form numerous anastomoses. Areas supplied by the common carotid artery include the lower beak and its muscles, the palate, oropharynx, upper beak, larynx, nasal cavity, and orbit.[21] The vascular anatomy of the head of a domestic duck (*Anas platyrhynchos*) is shown in Figure 31.6.

Integumentary Anatomy of the Bill

Keratin is produced by the underlying dermis of the beak with varying contributions from the cere. Both keratin surfaces are made up of a covering type (very thin) and a pressure-bearing type (very thick) of keratin. These surfaces are worn continuously through use and mechanical abrasion. The degree of wear may vary due to foraging styles and differences between individuals, seasonal variations in food types, and within populations.[22,23] Figure 31.7 shows the variation of keratin type and thickness in three selected species (hawk-headed parrot *[Deroptyus accipitrinus]*, Harris' hawk *[Parabuteo unicinctus]*, and hyacinth macaw *[Anodorhynchus hyacinthinus]*). The base of the culmen in some birds is enlarged to form ornamental excrescences.[23] Examples of these are seen in coots (*Fulica* spp.) and hornbills (Bucerotidae). Puffins (*Fratercula* spp.) molt their decorative rhinotheca after completion of the breeding season.

The horny bill resembles skin, consisting of epidermis, dermis, and bone. The dermis is closely attached to the periosteum. The epidermis is modified in two ways: the stratum corneum is very thick, and the cells of the stratum corneum contain free calcium phosphate and orientated crystals of hydroxyapatite. These two modifications are in addition to the abundant keratin and keratin-bound phospholipids and calcium, which characterize the cells of the stratum corneum of ordinary skin. The combination of these modifications provides the horny bill with its typical hardness.[24] Keratin arises from the Malpighian cells of the bird's epidermis, growing from plates at the base of each mandible. There is a vascular layer between the rhamphotheca and the deeper layers of the dermis, which is attached directly to the periosteum of the bones of the beak. Covering-type keratin is located on the outer and lateral surfaces of the upper and lower beak. As implied, covering-type keratin is quite thin. Pressure-bearing-type keratin is much thicker and denser, located on the undersurface of the upper beak (occlusal ledge of the rhinotheca), the piercing/shearing point of the upper beak (rostral rhinothecal tomium), the chisel edge of the lower beak (rostral mandibular tomium), and at the lateral tomia of the rhamphothecae (Figure 31.7). In the monk parakeet *(Myiopsitta monachus)*, a keratin monomer of approximately 15,500 daltons (Da) molecular weight is typical for the harder, pressure-bearing surfaces, and a larger monomer of 25,000 Da is more common with the softer, covering-type keratin structures.[22] Comparatively, solubilized keratinized epithelium in the monk parakeet had a molecular weight of 46,000 to 66,000 Da.

Keratin normally migrates rostrally along the surface of the beak and laterally from its vascular bed. The keratin replacement time of the rhamphotheca is closely linked to the natural use of the beak. In large parrots, complete replacement of the rhinotheca occurs in approximately 6 months, whereas in Ramphastidae there is a growth rate of approximately 0.25 cm per year.[2,4] Normally the gnathotheca is replaced two to three times faster than the rhinotheca.[2] The

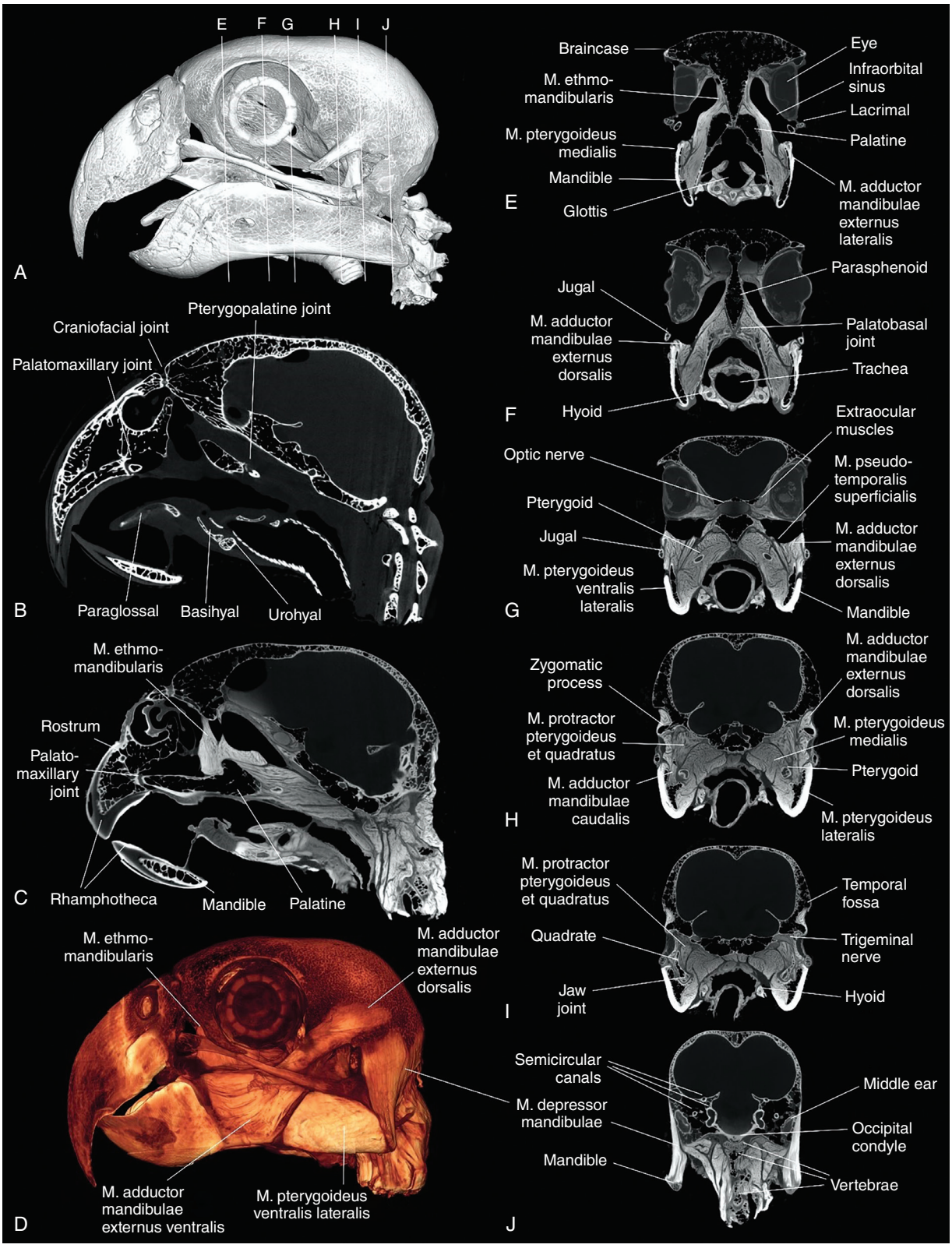


FIGURE 31.4 Micro-computed tomography (micro-CT) scan and iodine contrast–enhanced micro-CT scan of a Grey parrot (*Psittacus erithacus*); University of Missouri AV092 showing major osteological and myological features of the feeding apparatus. Iodine contrast renders muscles, nerves, and other soft tissues visible in radiography[18,19] (A) Left lateral view of the skull with locations of slices used in (E–J). (B) Midsagittal section through the skull showing joints involved in cranial kinesis. (C) Slightly parasagittal section through contrast data showing jaw muscles. (D) Left lateral view of volume rendering of iodine contrast CT data of the head. (E–J) Axial sections of iodine contrast micro-CT of the Grey parrot illustrating joints, muscles, and other key cranial features. (Photo courtesy Casey Holliday.)

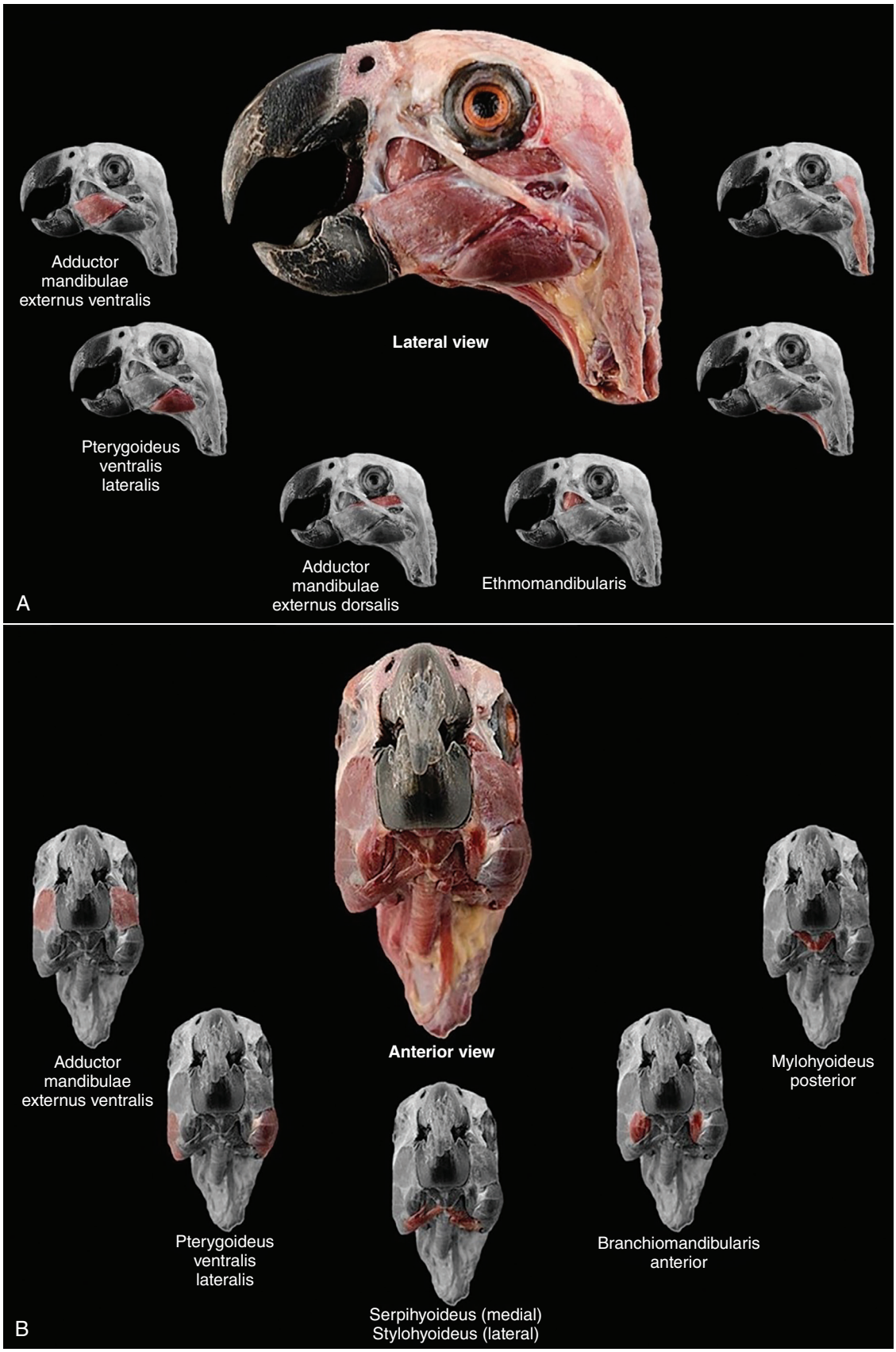


FIGURE 31.5 Dissection and display of the superficial prokinetic muscles of the Grey parrot *(Psittacus erithacus)*. (A) Lateral view. (B) Frontal view. (Dissection - Shawn Miller, Photography - Mark Nielsen Copyright Nielsen and Miller)

TABLE 31.2

Prokinetic Muscles, Skeletal Attachments, and Action

Muscle	Origin	Insertion	Action
M. adductor mandibulae externus	Temporal fossa and otic process of quadrate	Dorsal and lateral surfaces of the mandibles	Raises the lower beak
M. adductor mandibulae caudalis (m. adductor mandibulae posterior in reptiles*)*	Rostral surface of body of quadrate	Medial surface of the mandibles	Raises the lower beak
M. pseudotemporalis superficialis	Posterior part of interorbital septum, posterior surface of orbital wall	Medial surface of the mandibles	Raises the lower beak
M. pseudotemporalis profundus[a]	Aponeurosis and the fleshy fibers at the top of the orbital process of the quadrate	Medial surface of the mandible	Elevates and retracts the mandible
M. pterygoideus	Palatine and pterygoid bones, braincase	Medial or lateral surface of the mandibles	Closes upper and lower beaks simultaneously Moves lower beak forward and lowers upper beak during chewing
M. protractor pterygoidei et quadrati	Posterior part of interorbital septum, ventrolateral surface of basicranium	Rostromedial surface of quadrate, pterygoid	Propels the palate and raises the maxillary beak (protraction)
M. ethmomandibularis[b]	Rostrodorsal part of interorbital septum	Medial surface of the mandibles	Raises and protracts the mandible
M. pseudomasseter[b]	Suborbital arch	Lateral surface of mandibles	Raises the mandible
M. depressor mandibulae	Caudolateral surface of exoccipital	Caudal aspect of mandibles	Lowers the mandible

[a]Absent in parrots.
[b]Parrot-specific muscles.

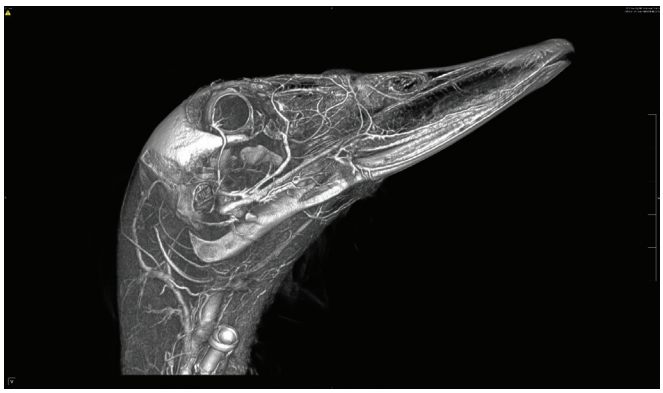


FIGURE 31.6 Vascular anatomy of the head of a Swedish blue duck *(Anas platyrhynchos)*. The cadaver image obtained was enhanced with BriteVu contrast agent (Scarlet Imaging LLC, Murray, UT, USA). (Photo courtesy M. Scott Echols.)

beaks of birds are usually short and thick or long and thin. The beak of the toucans is an exception: it is both long and thick. Its length is about one-third of the total length of the animal, but its weight represents approximately 5% of the total live weight.[4,25] The toucan beak possesses extraordinary biomechanical resistance, aided by its layers of keratin and a trabecular fibrous network of bone and calcium-rich proteins. The thickness of the keratin layer in toucans ranges from 0.5 to 0.75 mm and is arranged in the form of hexagonal plate overlays. Its density is approximately 0.1 g/cm^3, which allows the toucan to fly by keeping the center of mass in the plane of the wings.[25,26]

In waterfowl, the bill is hard only at the tip of both the upper and lower bill. These specific adaptations form the maxillary and mandibular nails, respectively.[24] The nail and its associated dermis form a complex sensory organ. The whole complex is known as the bill tip organ and plays an important role in the sensory discrimination between food and other particles. The horn of the nail is perforated by canals that open through minute pores on the occlusal surface of the nail. Each of these canals is filled by a dermal core, corresponding to a highly specialized dermal papilla containing a profuse array of mechanoreceptor nerve endings. These dermal papillae project slightly through the pores on the occlusal surface of the nail, their projecting tips being capped by a thin covering of keratinized epithelium. Since the tips of the dermal papillae reach the occlusal surface of the nail, they are directly accessible to tactile stimuli during feeding. A bill tip organ has been described in the Senegal parrot *(Poicephalus senegalus)*, and likely exists in many other parrot species.[27] In the Senegal parrot, seven pairs of pits within the rhamphotheca can be seen along the tomial edges of the rhinotheca, with a single pit at the bill tip. These pits, as described in waterfowl, probably contain clusters of mechanoreceptors. Irregular grooves are present in the maxillary or mandibular bones of the Senegal parrot, leading down toward the bill tip, suggesting the course of the blood vessels or nerve fibers supplying mechanoreceptors embedded in the keratin sheath. These tactile pits are visible in Figure 31.8. Papillae have been identified three-dimensionally (3D) and two-dimensionally (2D) within these pits recently, demonstrating the clear presence of neurosensory structures within them.[28]



FIGURE 31.7 Sagittal sections of the beak/head of three birds: (A) hawk-headed parrot *(Deroptyus accipitrinus)*; (B) Harris' hawk *(Parabuteo unicinctus)*; (C) hyacinth macaw *(Anodorhynchus hyacinthinus)*. Note the variations in the thickness of keratin over the culmen and the occlusal regions and tomia of the rhinotheca. (Photos A and B courtesy Nigel Harcourt-Brown; photo C courtesy Brian Speer.)

Many parrots also have grooves in the hard keratin (occlusal ledge) of the oral aspect of the rhinotheca, which likely aid in grip, grinding, and cracking of food items (Figure 31.7).[24,27,28] A similar arrangement of tactile pits in the maxilla is found in the mandible of the Senegal parrot, but in smaller numbers.[27]

Mechanoreceptors and innervation of the beak

The principal type of mechanoreceptor in the bill tip organ is the Herbst corpuscle.[24] These corpuscles have an onion-like structure with a central axonal ending surrounded by numerous slender concentric cellular lamellae. These corpuscles are highly sensitive to vibration and are capable of following very fast frequencies up to 1000 Hz. The number and location of Herbst corpuscles that are present in the bill are related to the way the bill is used as a tactile exploratory organ during feeding.[24,27,29]

In grain-eating birds, including many parrot species, which cut or crack open seeds, the tip and lateral ridges of the bill are also well supplied with Herbst corpuscles. These corpuscles are the most widely distributed mechanoreceptors in the avian body, occurring wherever there is a mechanical disturbance that may require tactile detection. These can be found at the base of feather follicles, along the bones of the wing and pelvic limb, in muscles, tendons and joint capsules, near large blood vessels, and even in association with the alimentary canal and especially the cloaca. Grandry corpuscles are distributed in the dermis of the bill that is associated with the soft horn in waterfowl, particularly near the sides and tip.[30] These corpuscles consist of one or two granular cells associated with



FIGURE 31.8 The white tactile pits for mechanoreceptors are visible in this young blue-and-gold macaw *(Ara ararauna)* through the surface of the rostral gnathotheca *(A)*.

numerous axonal endings and closely resemble mammalian Merkel cells. These are rapidly adapting, velocity-sensitive mechanoreceptors. Grandry corpuscles are present in the dermis of the bill of grain-eating passerine species as well. Merkel cell receptors share a similarity with the intraepidermal Merkel cell receptors of mammals, but these are in the dermis of birds rather than the epidermis.[30] Merkel cells have been found predominantly in the beak and tongue of various nonaquatic birds. It is speculated that Merkel cells function as secondary sensory cells like hair cells in the inner ear. Merkel cells may occur as single cells as well as in groups and may even be organized as corpuscles with stacked arrangement like Grandry corpuscles of aquatic birds.[30]

Biomechanics of the Beak

The occlusal biomechanics and force delivery of the beak of many bird species have been explored in different ways.[14,26,31,32] In the order Psittaciformes, biomechanics have been detailed in the monk parakeet,[22] the Grey parrot,[33] and Pesquet's parrot *(Psittrichas fulgidus)*.[34] During feeding or locomotion, when the beaks are occluded, three forces act on the beak. The first, applied by *m. pterygoideus*, is transferred in the caudorostral direction of the maxillary beak. A second force is applied perpendicular to the longitudinal axis of the beak during occlusion. This force has been described in toucans and reaches values close to 270 N (~27 kg) in beaks weighing about 20 g. The third force is angular to the nasofrontal hinge joint. The combination of these three forces maintains the structural stability of the maxillary beak. The occlusal force applied by *m. pterygoideus* is greater to objects at the base of the beak and decreases gradually as the objects are pressed more rostrally close to the tip of the beak.[31]

Biomechanics and Function of the Beak

Cranial kinesis

Cranial kinesis of the beak is the term used to describe significant movement of skull bones relative to each other in addition to movement at the joints between the upper and lower beaks. Generally, cranial kinesis refers to relative movement between the upper beak and the braincase (neurocranium). There are three basic forms of cranial kinesis in birds: prokinesis, rhynchokinesis, and amphikinesis (Figure 31.9). The more widespread form is prokinesis, where the upper beak itself is inflexible and moves at the point where it articulates with the braincase at the craniofacial hinge joint, which is particularly evident in the order Psittaciformes, though it is found to some degree in several taxonomic orders. Rhynchokinesis describes the ability possessed by some birds to flex their upper beak. Unlike prokinetic birds, rhynchokinetic birds do not have a single craniofacial bending zone that includes the nasal bones and the nasal processes of the premaxillae. Instead, they have one bending axis passing through the dorsal ends of the lateral bars, and one or more others further rostral on the dorsal bar.[9,35] Flexion of the upper beak some way along its length is seen, either upward, in which case the upper beak and lower beak diverge, resembling a yawn, or downward, in which case the tips of the beaks remain together while a gap opens up between them at their midpoint. There are five distinctly described forms of rhynchokinesis: proximal, central, distal, double, and extensive.[35] Rhynchokinesis is known in Struthioniformes, Rheiformes, Apterygiformes, Ciconiiformes, Gruiformes, Charadriiformes, Columbiformes, Apodiformes, and Passeriformes. In amphikinesis, the entire upper beak is raised, and the tip of the beak is bent up in addition during protraction, and in retraction the tip bends down with respect to the rest of the upper beak. Amphikinesis is seen in Rallidae.[35]

Thermoregulation

Thermal regulation of excess body heat from the oropharyngeal cavity in some avian species such as pelicans (*Pelecanus* spp.) is accomplished through evaporative heat loss by thermal tachypnea and gular flutter. Thermal tachypnea involves open-mouth breathing or thermal panting, which is associated with an increased respiratory rate, resulting in an increased rate of evaporative cooling effect. The gular flutter is characterized by a rapid flutter of the gular region caused by flexure and relaxation of the hyoid apparatus.[36] In addition to evaporative cooling mechanisms for heat exchange, toucans and hornbills are capable of heat exchange from their bill, using it as a controllable vascular radiator.[3]

DEFORMITIES AND DISORDERS OF THE BEAK AND SKULL

Beak deformities in birds are either congenital or acquired. Comparatively, there are few congenital deformities described (present at or before the time of hatch), and the vast majority of problems are acquired, and manifest with progressive developmental changes as continued keratin growth occurs and force vector delivery is altered. Acquired beak deformities can result from primary mechanical injuries to bone, dermis, innervation, or the prokinetic musculature itself. Abnormal keratin growth can also result from disease conditions. Traumatic

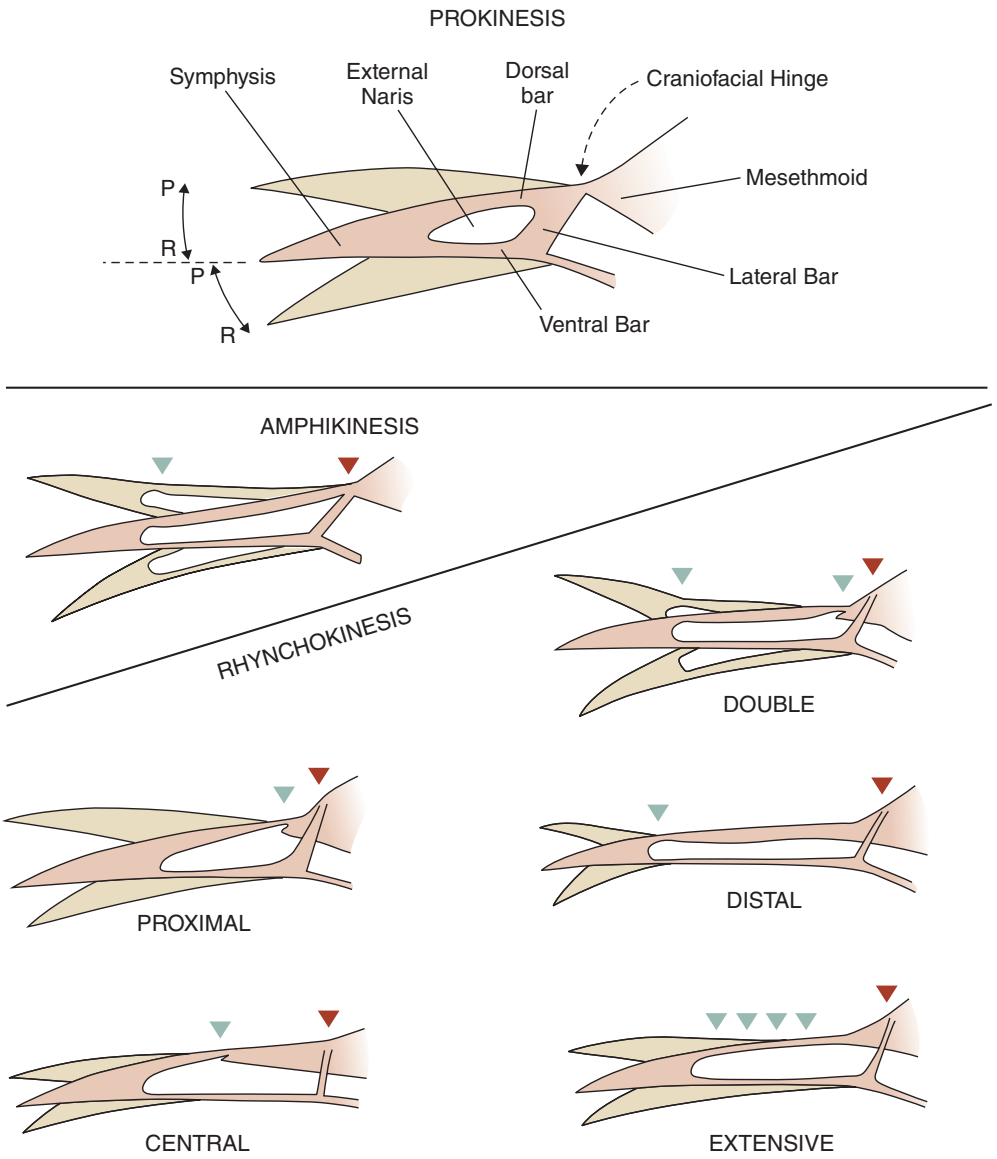


FIGURE 31.9 The forms of avian kinesis: prokinesis (A), amphikinesis (B), and rhynchokinesis (C), of which five subforms (proximal, distal, central, double and extensive rhynchokinesis) can be distinguished. *Stippled* figures show upper jaw in closed position. *P,* Protraction; *R,* retraction; *solid pointer* indicates craniofacial hinge; *open pointers* indicate additional bending axes on dorsal bar.

injuries, infectious organisms (parasitic, mycotic, viral, bacterial), nutritional, metabolic, and neoplastic causes are all well represented in the literature. These etiologies may have primary or secondary roles in beak deformities seen in birds, with secondary rhamphothecal overgrowth. Beak malalignment often results in abnormal keratin wear, requiring periodic corrective rhamphothecal reshaping procedures. More severe cases of congenital or acquired beak deformities may result in difficulty prehending food, and thus impaired welfare and quality of life. As such, it is appropriate to describe the deformities carefully and hypothesize or diagnose their causality with an understanding that the primary cause may be elusive at times. Diagnosis should be sought to the level of understanding required to facilitate therapeutic intervention and strategize the most anatomically sound way to trim and shape rhamphothecal surfaces.

Nutritional Disease

Nutritional deficiencies and imbalances can result in pathology of the beak and skull. Malnutrition may result in softening and flaking of the rhamphotheca. Nutritional metabolic bone disease can cause abnormal bone growth in juvenile birds and can potentially result in developmental beak deformities when the bones of the skull are involved. These types of deformities may also be acquired secondary to metabolic bone disease at later ages (Figure 31.10). Secondary (opportunistic) bacterial and mycotic infections are common and can set the stage for the development of beak deformities.[5,37]



FIGURE 31.10 Chronic rhamphothecal deformities in a 23-year-old Grey parrot *(Psittacus erithacus)*, with right lateral rhinothecal overgrowth and left lateral rostral gnathothecal tomial overgrowth. Not visible, but also present is considerable overgrowth of the rhinothecal occlusal ledge, where the rostral gnathothecal tomium should normally occlude. Note the lingual and inward folding of the left mandible and gnathotheca, likely the cause of the abnormal mandibular angulation and resulting malocclusion and tomial overgrowth. Metabolic bone disease and weaker mandibular structural integrity and/or trauma were hypothesized as underlying causes of this deformity when the bird was younger, and the rhamphothecal overgrowth was first noted at 6 years of age. (Photo courtesy Brian Speer.)



FIGURE 31.11 Squamous cell carcinoma in a 35-year-old yellow-fronted Amazon parrot *(Amazona ocrocephala)* of unknown gender, resulting in a crusting, wart-like growth of the rictus on the right side of the beak. Squamous cell carcinomas are well known to invade the adjacent tissues, as can be observed in this bird as well, where oral inspection revealed the neoplasia to have invaded the hard palate. (Photo courtesy Yvonne van Zeeland and Nico Schoemaker.)



FIGURE 31.12 Squamous cell carcinoma involving the mandibular beak in a budgerigar *(Melopsittacus undulatus)*. Note loss of the rostral gnathothecal tomium, erosion of the keratin of the gonys, and overgrowth of the rostral rhinothecal tomium. (Photo courtesy Lauren Powers DVM, DABVP (Avian Practice); with permission.)

Neoplasia

Many neoplasms form visible masses of the beak or oral cavity (Figure 31.11), while others are not directly visible. Such neoplasms may eventually become clinically apparent by the distortion of rhamphothecal growth,[37] presence of tissue necrosis or hemorrhage,[37] dysphagia and/or difficulty prehending food, or dyspnea.[5,37] Definitive diagnosis generally requires cytology or histopathology.[37]

Squamous cell carcinoma

Squamous cell carcinoma has been described in a number of avian species, including a timneh parrot *(Psittacus timneh)*,[38] a Buffon's macaw *(Ara ambigua)*,[39] great hornbill *(Buceros bicornis)*,[40,41] a common stork *(Ciconia ciconia)*,[42] and an African penguin *(Spheniscus demersus)*.[43] Squamous cell carcinoma is often locally invasive, with a high rate of local recurrence but a low potential for distant metastasis (Figure 31.12).[37] Secondary bacterial and mycotic infections are common.[37] Aggressive surgical resection,[39–41] chemotherapy (intralesional cisplatin),[40] radiation therapy (cobalt 60),[39] photodynamic therapy,[40] and topical and oral antiangiogenic therapy[41] have been described with mixed success in birds. Therapeutic options for squamous cell carcinoma are reviewed in *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 3, Neoplastic Diseases in Avian Species. In some of these cases, particularly those where amputation or aggressive resection is a part of the treatment, the use of surgical beak repair and/or prosthetics may have value in improving quality of life (see also Chapter 56).[41]

Melanoma

Melanoma is generally considered a malignant neoplasm in birds.[37,44] Malignant melanoma of the beak or surrounding tissues has been described in the Grey parrot,[45] thick-billed

parrot *(Rhynchopsitta pachyrhyncha)*,[46] pigeon *(Columba livia)*,[47] mandarin duck *(Aix galericulata)*,[48] and macaroni penguin *(Eudyptes chrysolophus)*.[44] Not all melanocytic tumors in birds are grossly pigmented, and therefore all suspicious masses should be closely evaluated and sampled for diagnostic testing.[44,48] Reported treatments include surgical excision,[45,47] radiation therapy,[44,46] and oral antiangiogenic therapy.[46] Distant metastasis to the lungs, liver, kidney, spleen, bone marrow, and other organs is common.[37,44–48] As with squamous cell carcinoma, the use of surgical beak repair and/or prosthetics may have particular value in improving quality of life, when amputation or aggressive resection is required.

Fibroma and fibrosarcoma

Fibrosarcoma is generally considered to be the most common primary beak tumor in birds, although fibromas may also occur (Figure 31.13).[37,49] Fibrosarcoma is most common in budgerigars *(Melopsittacus undulatus)* and cockatiels *(Nymphicus hollandicus)*,[37] although it has also been reported to be associated with a pathologic fracture and distal avulsion of the maxillary beak in a Grey parrot.[49] Fibrosarcomas tend to be locally aggressive with a low risk of distant metastasis, although metastasis has been documented and local recurrence is common after surgical resection.[37] Local invasion into the nares, nasal turbinates, and bone can result in deformation of the beak. Histopathology is generally considered diagnostic.[37,49] Chemotherapy (intralesional cisplatin) and radiation therapy were used in the treatment of a facial fibrosarcoma in a blue-and-gold macaw.[50]

Infectious Diseases

Viral diseases

Several viral diseases are associated with damage to the structures of the beak and oral cavity. Psittacine beak and feather disease (PBFD) is caused by a nonenveloped virus in the genus *Circovirus*, family *Circoviridae*. In the chronic form, beak lesions and feather loss are often observed (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Diseases). Reported beak and oral lesions include rhamphothecal elongation, transverse or longitudinal beak fractures, oral ulceration, and palatine necrosis.[37,51] The rhinotheca may separate from the underlying dermis, potentially resulting in exposure of bone (Figure 31.14). Diagnosis can be made through the use of serology, polymerase chain reaction (PCR), and histopathology and demonstration of basophilic intranuclear and intracytoplasmic inclusion bodies.[37,51] Although birds can survive with supportive care for long periods after developing lesions, immunosuppression, and life-threatening opportunistic infections are common.[37,51]

Avian pox is caused by viruses of the genus *Avipoxvirus* in the family *Poxviridae*. Poxvirus infections are common in avian species but are only occasionally diagnosed in psittacine birds.[36] There are two major forms of clinical disease—a dry (cutaneous) or a wet (diphtheroid) form, although a septicemic form can also occur (see Chapter 15).[37] In the dry form, papules, pustules, nodules, crust, and eschar form on nonfeathered areas of the skin, frequently around the eyes, beak commissures, and cere.[37] Beak and keratin deformities can on occasion result from some of these lesions. In the wet form, plaque and diphtheritic membranes form over oral mucus membranes and respiratory mucosa. Diagnosis is generally made through biopsy and histopathology, with demonstration of intracytoplasmic eosinophilic inclusion bodies (Bollinger bodies; see Figure 15.5), although PCR can also be used to demonstrate the presence of virus.[36] Mortality rates are higher with the wet forms of the disease.[37] Cutaneous lesions will usually spontaneously resolve, and treatment is primarily supportive.



FIGURE 31.13 Budgerigar *(Melopsittacus undulatus)* with a slow-growing soft tissue mass at the base of the beak. The bird initially presented with a scaly, eroding beak, which the primary care veterinarian suspected to be related to a scaly mite infestation. Following treatment with ivermectin and a beak trim, growth of the flesh-colored mass was noted, resulting in significant distortion and lateral deviation of the beak. Biopsy revealed the mass to be a fibroma. (Photo courtesy Yvonne van Zeeland and Nico Schoemaker.)



FIGURE 31.14 Rhamphothecal overgrowth and deformity in a sulfur-crested cockatoo *(Cacatua galerita)*, secondary to degeneration and necrosis of the dermal layers of the maxillary beak. Note the scissors deformity and marked overgrowth of the rostral gnathothecal tomium. Secondary and opportunistic bacterial and fungal infections of these exposed tissues are not uncommon. (Photo courtesy Bob Doneley.)

Avian polyomavirus (genus *Polyomavirus*, family *Polyomaviridae*) is often fatal in finches. However, some surviving birds, particularly Lady Gouldian finches *(Erythrura gouldiae)*, may develop elongation to tubular deformation of the rostral beak tips and inflammation and necrosis of the beak germinal epithelium.[37] A novel picornavirus and poecivirus are associated with rhamphothecal deformities in birds of prey and passerine species in northwest North America, respectively.[52,53,54]

Viral diseases that are largely representative of systemic processes with manifestations including rhamphothecal deformities carry overall a poor prognosis. However, poxviral infections may be ultimately self-limiting, and restorative or corrective beak procedures may offer value for some affected individuals where the biomechanics of beak function is impaired.

Bacterial diseases

Primary bacterial infections of the beak are unusual in birds, whereas secondary bacterial infections are somewhat more common.[37,55] Secondary bacterial infections often involve opportunistic Gram-negative bacteria as well as Gram-positive organisms. The diagnosis of bacterial infection is generally made through culture, although additional diagnostics such as Gram stain and acid-fast stain cytology can be used to further characterize observed bacteria.[37] Isolation of bacteria from fragmented slabs of keratin of the beak alone does not necessarily confirm infection. Treatment of bacterial infections should be based on results of culture and sensitivity testing, may include both topical and systemic modalities, and should also include treatment of predisposing and underlying diseases as well as supportive care as indicated.

Infection with *Bordetella avium* in juvenile cockatiels can result in a severe sinusitis and rhinitis with involvement of surrounding jaw musculature and bone.[56,57] Some affected birds have reduced ability to open their beaks. Isolated strains of *B. avium* from cockatiels have been found to possess a dermatonecrotic toxin gene (DNT)[57–59] and to produce osteotoxin.[56,59,60] Diagnosis of *B. avium* infection and resultant lockjaw may be made with clinical signs, physical examination findings, aerobic isolation of the organism, or PCR identification of the same. While *B. avium* appears susceptible to most antimicrobial agents,[52] antibacterial treatment should be based on the results of culture and sensitivity testing. Once muscular stiffness sets in, the prognosis for recovery is guarded to poor, although treatment can still be attempted and should include nonsteroidal antiinflammatory drugs (NSAIDs), pain management, and physical therapy in addition to targeted antibacterial treatments. Successful treatments where these approaches have been used in combination are reported.[61]

Mycotic diseases

Primary mycotic infections of the beak are uncommon in birds, but secondary or opportunistic infections have been reported and are likely more frequently encountered.[37,62] Sinusitis caused by *Cryptococcus neoformans* var. *gattii* in a 7-year-old male Major Mitchell's cockatoo *(Cacatua leadbeateri)* was associated with destruction of the rhinotheca.[62] Infection with *Penicillium cyclopium* resulted in rhinothecal necrosis and destruction in a blue-and-gold macaw.[63] Topical itraconazole had been ineffective prior to the diagnosis.[63] *Aspergillus* sp. was cultured from an adult yellow-naped Amazon parrot *(Amazona auropalliata)* with sinusitis, radiographic osteolysis of facial bones, and distorted growth of the distal rhinotheca.[64] Fungal mats were observed by histopathology on debrided beak tissues. The bird was treated with itraconazole administered *per os*.[64] During and following treatment of these infections, strategic rhamphothecal shaping procedures often will be required to augment and normalize force vector delivery and return normal keratin wear.

Parasitic diseases

Several ectoparasites can result in damage to the beak and surrounding tissues. Infection with *Knemidocoptes pilae* (scaly face mite; scaly leg mite) is somewhat common in small passerine birds and psittacine birds such as budgerigars, kākārikis *(Cyanoramphus novaezelandiae)*, and *Neophema* and *Polytelis* genera parrots.[37] The entire life cycle of the mite is spent on the host.[37] Mites are often found along the beak margins but can be found elsewhere on the body, such as the legs, wing tips, and vent.[37] The skin and rhamphotheca can develop a hyperkeratotic and pitted or honeycombed appearance (Figure 31.15).[37,65,66] Advanced lesions can result in severe distortion of the rhamphotheca.[37,67] Diagnosis is made through direct visualization and microscopic examination of skin or deep rhamphothecal scrapings (Figure 31.16).[37,66,67]

Avermectins such as ivermectin and moxidectin are commonly used to treat infections and are often administered at 7- to 10-day intervals until clinical resolution. Ivermectin can be administered orally or applied topically to lesions.[65,67] Weekly topical application of 0.05 mL of a 1 mg/mL solution was effective in eradicating mites within 2 weeks in budgerigars.[67] Systemic treatment is recommended for birds with severe or generalized infections.[67] As mentioned with mycotic infections, during and following treatment of infections, strategic rhamphothecal shaping procedures often will be required to augment and normalize force vector delivery and return normal keratin wear.



FIGURE 31.15 Knemidocoptic mange in an adult female Pacific parrotlet *(Forpus coelestis)*, resulting in rhamphothecal deformity and overgrowth, particularly of the rhinotheca. (Photo courtesy Lauren Powers DVM, DABVP (Avian Practice); with permission.)

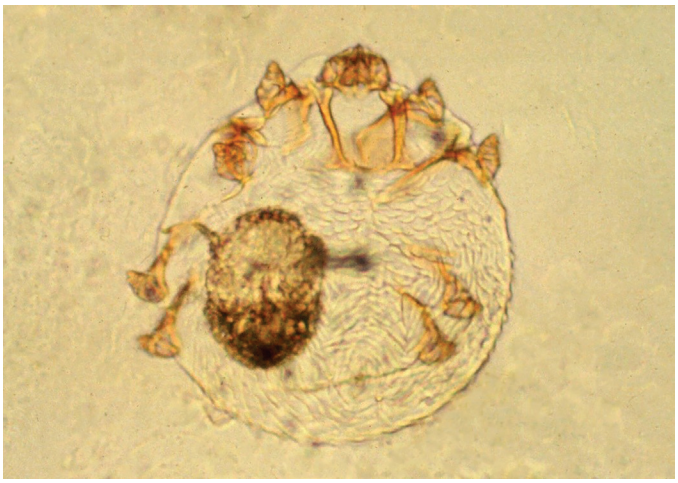


FIGURE 31.16 *Knemidocoptes pilae* (female), potassium hydroxide stain, 4×. *Knemidocoptes* are small, round burrowing sarcoptiform mites that can be found in various avian species. Mites usually invade the lightly or nonfeathered portions of the legs and skin, causing feather loss, scaliness, hyperkeratosis, and distortion of the beak, legs, and claws. Larvae undergo two nymphal stages before reaching maturity, which will take between 14 and 21 days to complete. Diagnosis can be made through deep skin scrapings. (Photo courtesy Yvonne van Zeeland and Nico Schoemaker.)



FIGURE 31.17 Marked gnathothecal overgrowth, resulting in actual penetration of the right lateral gnathothecal rostral tomium into the cere of an adult cockatiel *(Nymphicus hollandicus)*. There also is a paired overgrowth of the rhinothecal rostral and left lateral tomia. By keeping serial digital photos of a patient such as this, the strategy for, and assessment of effectiveness of a rhamphothecal shaping procedure toward the longer-term goal of functional correction can be made. (Photo courtesy Brian Speer.)

Trauma

Beak trauma is very common in companion and aviary birds as well as in wild birds and includes bite wounds from other birds, predators, crushing, and impact injuries.[37] Most wildlife cases are related to window collisions, gunshot wounds, bite wounds, and vehicular trauma.[6] Birds should be stabilized and fully evaluated for the extent of the injuries. An overall assessment of the patient's stability and systemic health status is essential prior to focusing specifically on the beak and musculoskeletal injuries of these patients (see Section 8, The Trauma Patient). Trauma involving the bony structures of the beak is common, and poorly healed fractures can result in beak deviations and uneven rhamphothecal wear.[68] Patients should receive appropriate supportive care and analgesic therapy.[5,6,68] The clinical assessment of the beak and skull includes a visual evaluation of the external beak, external palpation of the mandible and jugal arches, and range of motion assessment of the maxillary and mandibular beaks. Diagnostic imaging procedures are often, but not always, indicated.[5,6] Specific traumatic injuries and corrective surgical procedures are discussed in the following.

RELEVANT DIAGNOSTIC IMAGING OPTIONS

Photographic Imaging

Imaging is a useful and important diagnostic modality for evaluating disorders of the beak and oral cavity. It is particularly useful to document lesions with a high-quality digital camera, ideally one with excellent macro and lighting features. Standardization of patient position while obtaining these images will help improve methods of comparison when they are evaluated serially. These types of digitally recorded images can facilitate direct comparison of patient status and progression over time, allow quantitative measurements of lesions or deformities, and provide photodocumentation of current patient status as is described in medical records. With the growing use of digital medical record–keeping systems in veterinary health care, these images are easily embedded in most medical records, allowing rapid access and easier sharing with specialists for further consultation if indicated.[5] An example of a specific deformity where practical application of this means of serial photographic imaging could have value is shown in Figure 31.17.

Projection Radiographic Imaging

Radiographic imaging is a standard and key aspect of the practice of avian medicine by allowing for noninvasive visualization of anatomic features in both health and disease states.[5,69] Due to the often relatively small body size of many companion bird species, variations in technique and image quality obtained, and the significant overlap of the multiple structures of the head, diagnostic acuity may be more limited as compared to what can be obtained with other imaging studies or plain images of other anatomic regions (Figure 31.18). Regardless, in the correct clinical situation, these plain radiographic images can offer a significant benefit as a component of a diagnostic investigation.[5,69] Particular problems and diagnostic challenges, which may be aided with plain radiographic imagery, may include

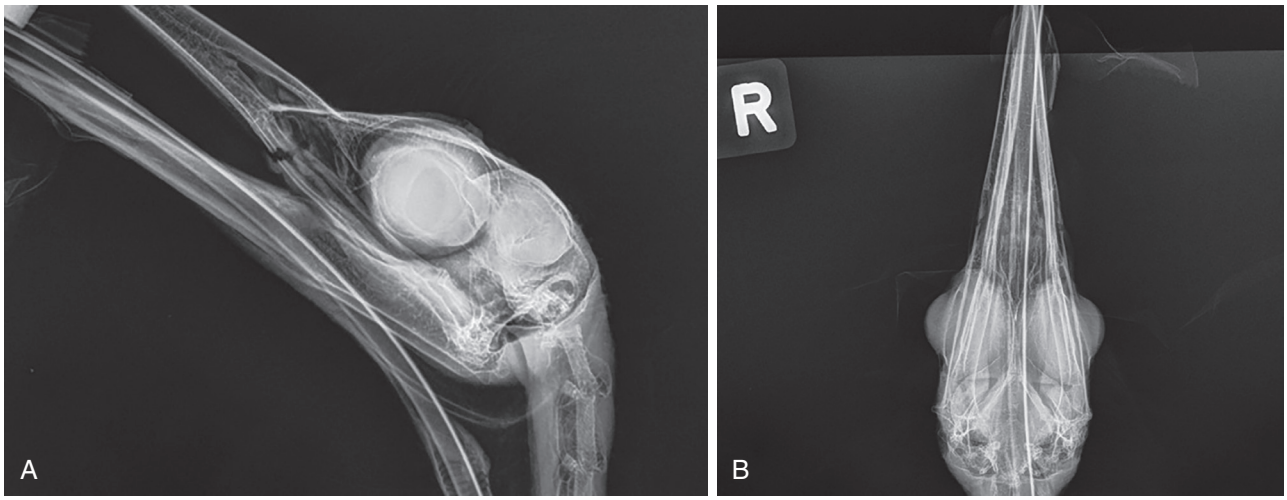


FIGURE 31.18 Projection radiographic images of a black-crowned night heron *(Nycticorax nycticorax)* that was found injured in its enclosure. Although fractures to the jugal and palatine bones can be identified in these lateral (A) and ventrodorsal (B) images, superimposition makes detailed evaluation of other bony structures more challenging without the use of CT imaging. (Photos courtesy Brian Speer.)

malocclusions with keratin overgrowth, certain skull fractures, and some sinus problems.[5,69]

Computed Tomography

Computed tomography (CT) has become an increasingly valuable tool in avian species, with applications that are useful for planning, guiding, and monitoring therapy (see Chapter 2, and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 14, Advances in Diagnostic Imaging).[69–71] The main advantage of CT imaging compared with standard projection digital radiography is that there is no interference from superimposition. Two-dimensional and 3D renderings obtained through CT imaging enable better visualization of various tissues and structures, including those that would otherwise have remained undetected with conventional radiographs (see Chapter 2). In general, high-definition, high-resolution CT imaging has greater diagnostic acuity than projection digital radiography. In areas with significant bone superimposition, CT imaging has been found especially useful. An example of some of the merits of CT imaging is provided in Figure 31.19. CT has a large range of relevant and practical applications, particularly for the head, and is considered superior to radiographs for evaluating the nasal cavity, conchae, and sinuses, allowing detailed studies of the various anatomic structures.[69] Contrast media may be used to increase the radiodensity of tissues, organs, and/or vascular systems, which helps depict lesions or abnormalities. Soft tissue structures with abnormal vascularization caused by disease (e.g., neoplasia, inflammation) will show increased uptake of contrast medium, differentiating it from normal tissue.[71]

Other Imaging Modalities

Other imaging techniques may be useful in specific conditions of the beak and head, such as magnetic resonance imaging (MRI), nuclear imaging (including positron emission tomography [PET]), and scintigraphy (see Chapter 2, and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 14, Advances in Diagnostic Imaging). Because of its characteristics, MRI is primarily used to image and diagnose soft tissue structures. The primary disadvantages associated with MRI may include prolonged imaging procedure times for some studies and special spatial resolution challenges. With the increased availability of higher resolution systems, the use and practical implication of MRI in avian medicine is likely to become more prevalent in veterinary practice overall and avian practice.[71]

RHAMPHOTHECAL TRIMMING AND SHAPING STRATEGIES

Normal and Abnormal Keratin Wear

Despite its hardness, the horny tissue of the bill is normally lost by wear and is continuously being replaced.[8] In birds that are maintained in companion or captive settings and the diets being fed as well as their means of acquisition are not consistent with those of their wild counterparts' behavior, the bill is sometimes not subjected to its ethologically normal intended use and resulting wear. This may be the result of the consumption of different food items, a lack of the normal enrichments that would provide such wear or other factors. A frequent consequence of the lack of normal wear is the perceived or potential need for trimming of these beaks, which is often focused on the flaky layers of covering-type keratin as a cosmetic procedure.[5] Many of these grooming procedures are unnecessary, and strategic enrichment of the birds' environment and self-maintenance behaviors may be most appropriate (see Chapter 47). An example of normal keratin accumulation in the absence of malocclusion that suggests a lack of normal beak use and rhamphothecal surface wear is provided in Figure 31.20. Where there is appreciable malocclusion or rhamphothecal overgrowth present, a keratin-shaping strategy may be indicated, based on functional diagnosis of its cause. In addition to a lack of normal use and keratin wear, overgrowth can often be seen secondary to uneven rhamphothecal wear resulting from other disease processes.[5,52,68,72]



FIGURE 31.19 Selected 3D-constructed CT images of a griffon vulture *(Gyps fulvus)* with multiple skull deformities. (A) Photo of the bird. (B) Left lateral 3D reconstruction. (C) Right lateral 3D reconstruction. Interpretive findings from the CT imaging study: the mandible is skewed to the right, and maxillary beak to the left; there is a left jugal fracture, with the nasal-most component missing and no apparent articulation with the left premaxilla; the palatine bone has an incomplete fracture at the symphysis, with depression of the left side apparent. The vomer appears intact, and the left and right pterygoids appear intact. The left lacrimal bone is ventrally displaced and appears to be pressing on the scleral ossicles of the left eye, causing ventral displacement of the globe. Most significantly, however, are the changes to the left temporal bone. There is a displaced fracture of the temporal bone's postorbital process over the articular region for the quadrate and including the articular points for the quadrate. The dorsal aspect of the quadrate appears to be fractured as well. These fragments are displaced rostrally, leading to what appears to be rostral extension of the quadrate, whereas it is merely due to the displacement of that ventral quadrate fragment. (Photos courtesy Louisville Zoological Gardens.)

In principle, any disease process that causes uneven growth or abrasion (wear) of the rhamphotheca can result in deformation and overgrowth of the beak keratin. Chronic liver disease appears to be associated with excessive rhinothecal growth and elongation in some psittacine species, but the explanation for this association is unknown.[5]

The Beak Trim

Chickens

Beak trimming (amputation of the distal aspect of the upper bill) has been used in the poultry industry to provide a mechanical prevention of feather pecking and cannibalism, and while several countries have banned the use of this technique,



FIGURE 31.20 Covering-type keratin buildup in a red-lored Amazon parrot *(Amazona autumnalis)*. Although a keratin-grinding procedure may help remove these exfoliating layers, longer-term maintenance would be most effectively accomplished with strategic enrichment of more normal beak use and wear. (Photo courtesy Laura Wade.)



FIGURE 31.21 This leghorn shows a healed maxillary beak following a beak trimming procedure that was performed 8 days earlier. (Photo courtesy Pat Wakenell.)

its practice still continues in many countries today.[73–75] The practice of beak trimming in poultry is typically done very early in life and, although still intrusive, is less so than when performed in adults.[73] When carried out correctly, the upper beak, and in some methods, the lower beak, is trimmed or amputated not more than one-third of the distance from the tip to the entrance to the nostrils (Figure 31.21).

Trimming the maxillary beak, specifically using the hot blade method, results in altered prehension between the upper and lower bills, which mechanically prohibits these undesired behaviors. Normal food prehension, feather care, and normal occlusion and keratin wear are also altered,[76] as well as the mechanoreceptors and magnetoreceptors of the beak.[77]

Feeding efficiency (number of pecks per gram of pellets ingested) is also reduced by beak trimming (hot blade method). In adult poultry, this type of beak trimming has been observed to result in a temporary fall in the intake of food and a reduction in body weight that lasts at least 6 weeks. These effects are greater if half of the beak is removed rather than one-third. Humanely and ethically, the profuse sensory innervation of the beak and the loss of capability of normal behaviors to be performed should be considered before advocating this procedure, particularly in adults.[78] The only time beak trimming is used after 1 day of age in layer hens is for prevention of severe behavioral persecution/pecking injuries leading to death, and it has been done with a hot blade. As opposed to the traditional hot blade method for beak trimming, the more current use of infrared technology done at 1 day of age has become a preferred method. Infrared beak treatment methods have shown better weight gains in chicks and other species of poultry. It is not as painful as the hot blade method, and birds will perform their normal behaviors and will remain active after having their beaks treated. The beak will fall off around 2 weeks of age, and the birds exhibit normal eating, drinking, and eating behaviors after it falls off as well.[74,76]

Birds of prey

Coping is a medieval term used to describe the trimming and reshaping of talons and beaks. Overgrown beak keratin is often encountered in captive raptors (Figure 31.22), due to insufficient keratin wear associated with the use of soft, inadequate perching surfaces and lack of a natural diet. Injuries to rhamphothecal surfaces may also require reshaping and alignment of tomial edges. In some cases, rhamphothecal overgrowth may be seen because of malnutrition or other medical conditions.[79]

Psittacine species

Abnormal keratin buildup is frequently removed from captive parrots and the procedure(s) are often also referred to as beak trim procedures, although they do not imply the removal of bone as occurs in poultry. Beak trimming is often performed on parrots by veterinarians in private practice for cosmetic purposes, to remove excessive keratin that could lead to abnormal force vector delivery, or to shape overgrown keratin that is clearly associated with malocclusion. With the latter and more chronic example, jaw muscle tension, form, and function can become altered, as can the shape of the bones of the upper and lower beaks themselves. Keratin-trimming procedures aimed at blunting the beaks to prevent feather-damaging behaviors or biting behaviors are largely viewed as unethical and should be discouraged. The joints and linkage ligaments associated with prokinesis can also become secondarily strained or altered as a result. These deviations in force vector delivery in turn lead to continued development of malocclusion and continued abnormal keratin growth as a result. As keratin grows abnormally, force vector delivery of the bills becomes progressively altered, leading to augmented malocclusion. Beak-trimming procedures for many chronic rhamphothecal overgrowths should best reflect a working level of diagnosis and strategy, rather than merely be accepted as a routine procedure. As such, when trimming the beaks of these birds, it is logical and prudent to describe the deformities carefully, hypothesize the causality of the deformities with



FIGURE 31.22 This captive-bred, juvenile (6–8 months old) female gyrfalcon *(Falco rusticolus)* shows a vertical splitting crack on the caudal edge of the left lateral rhinothecal tomium. This type of injury is often due to a lack of normal abrasion and wear, caused by the absence of hard surfaces (e.g., rocks) where food is offered and consumed. Normally, falcons tend to brush the beak repeatedly after eating on the edge of these hard substrates to clean it after eating, providing the abrasive wear needed to file the beak and maintain its shape. If these are not corrected on time it could cause a more severe fracture. A coping procedure to remove this keratin and balance the overall tomial occlusion is appropriate. Since the rhamphothecal surfaces are comparatively soft, coping may be accomplished with the use of a straight-edged human nail cutter as opposed to a concave edge, to cut the tomial tooth into shape and reduce the crack or split reduced by using a modified nail file. These types of cracks cannot be corrected in one session. Repeat procedures, weekly, are recommended until the crack is resolved. (Photo courtesy Jaime Samour.)

an understanding that the primary cause may be at times elusive, and strategize the most anatomically sound way to trim. In addition, once keratin overgrowth has been shaped more correctly, a targeted enrichment strategy may help to reduce the frequency of necessary trimming procedures, if not ultimately eliminate them through a careful restoration of normal form and function.[5,80]

Procedural concepts

Low-stress handling and restraint methods are an essential component of these procedures, regardless of the species (see Chapter 46). The potential for escalating escape and avoidance behaviors, learned fear, increased aggression, generalized fear, or even apathy is very real. These undesired consequences may not only adversely affect the outcome of the procedure itself directly but also the longer-term health and welfare of the bird, as well as the continued strength of the doctor-client relationship. Where appropriate, skilled handling, strategic shaping of the restraint experience, conscious sedation, or even general anesthesia may be indicated (see Chapter 46 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 19, Anesthesia). Effectiveness at reducing the degree of learned fear is best judged by assessment of the behavior of the bird during future visits and procedures and comparing with what was observed and recorded previously in past assessments in the medical record.[80]

The use of a hobby drill tool (e.g., Dremel) or other grinding tools is popular and effective for many keratin-trimming procedures. These tools offer procedural speed and access to the multiple keratin surfaces that may require attention. The optimal use of this tool requires the selection of the proper size and shaped bits, manual skills in handling and using the tool, and appreciation of the potential for heat that can be generated with excessive pressure or contact time. Not all keratin deformities require the use of a grinding tool, and a variety of other hand-held trimming tools can be used, depending on the patient species, size, behavior, and the specifics and complexity of the deformities being addressed.

Keratin structures that should be evaluated and shaped in concert with each other include the rostral and lateral rhinothecal tomia, the rostral and lateral gnathothecal tomia, the culmen, and the inner occlusal ledge of the rhinotheca, which functions as the occlusal site for the rostral gnathothecal tomium. An example of the description of specific rhamphothecal deformities in a macaw is provided in Figure 31.23. With careful assessment, a more focused and effective reshaping procedure can be developed for an individual bird's set of deformities. Where dermal or bony involvement is planned or is known to have occurred, pain management should be incorporated into the procedural plan. In some select cases, preprocedural radiography (Figure 31.24) can help delineate the extent of trimming that may be most appropriate, and general anesthesia can be helpful for some more involved beak corrective trimming procedures.

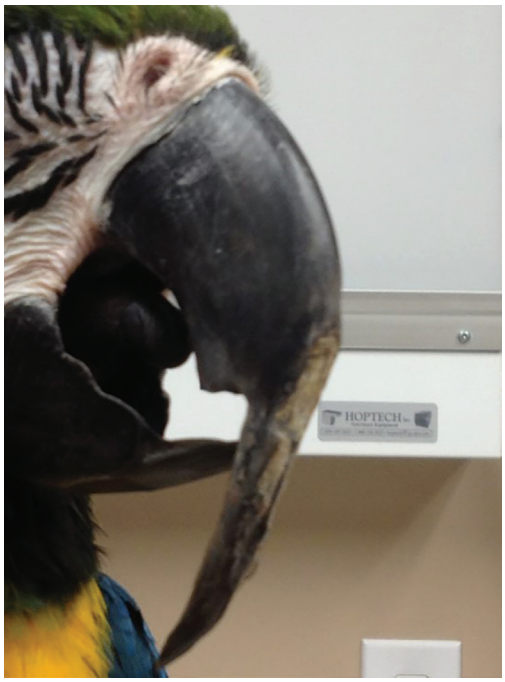


FIGURE 31.23 Rhamphothecal overgrowth in a chronically obese 1754-g blue-and-gold macaw *(Ara ararauna)*. There is overgrowth of the rostral rhinothecal tomium with abnormally soft and malodorous keratin at the distal culmen, rostral overgrowth and distortion, and overgrowth of the rhinothecal occlusal ledge, overgrowth of both the left and right lateral rhinothecal tomia, and overgrowth of the rostral gnathothecal tomium. (Photo courtesy Brian Speer.)



FIGURE 31.24 (A) Marked chronic rhamphothecal overgrowth in a green-winged macaw (*Ara chloroptera*). Note the scissoring deformity of the gnathothecal rostral and lateral tomia on the bird's left and rhinothecal rostral and lateral tomia on the bird's right. This deformity had been present for more than 15 years, and the apical aspects of the rhinothecal overgrowth had been periodically trimmed. The overgrowth of the occlusal ledge of rhinotheca is not visible in this first image. (B) Lateral projection survey skull radiograph of the bird depicted in (A). Measurements of the upper and lower bill allow for strategic calculation of what keratin overgrowth should be trimmed to optimize occlusion and force vector delivery under general anesthesia. (C) Following removal of the right lateral and rostral tomial overgrowth, the tomial surfaces are ground down to produce a symmetrical base for occlusion. (D) Preparation for cutting and shaping the gnathotheca. Note the black indelible ink marker at the point where the mandibular diaphysis was measured to be located. Overgrowth and distortion of the rhinothecal occlusal ledge are also visible from this angle. This was also ground and shaped appropriately.



FIGURE 31.24, cont'd (E) The same patient immediately following the procedure. Note the apparent improved kinetic function and ability to chew hard foods. Longer-term follow-up still is indicated, with a balanced and enriched behavior change strategy focused on increased chewing activities. (Photos courtesy Brian Speer, DVM, DABVP (Avian Practice), DECZM (Avian), Oakley CA; with permission.)

Following completion of the procedure, a targeted enrichment strategy to result in the functional increased use of these structures in more normal alignment should be contemplated and incorporated as a part of the whole procedure and its follow-up with a goal to reduce the need for and frequency of future procedures, if not eliminate their need completely (see Chapter 47).

ORTHOGNATHIC CORRECTION PRINCIPLES

Orthognathic procedures are those designed to correct deformities of the jaw and associated malocclusion in mammals. Applied to deformities of birds, these principles are applicable to both the upper and lower bills. The principle for correction of beak deviations is similar to that used in mammals and is based on the use of forces of small but constant intensity, applied in the opposite direction of the deviation. The repetitive compression force on the same side during normal kinesis causes the orientation of the cells of the dermal keratin-producing layers to migrate to the opposing side that is undergoing stress, correcting some of the malocclusions being addressed. As a result of these changes, the prokinetic musculature is utilized in more of a balanced manner and the resultant return of effective force delivery across keratin tomial contact points, more normal beak occlusion, and balanced prokinetic muscle usage in principle set the stage for effective resolution of many, but not all, problems. Most orthognathic procedures are used in young birds with deformities including lateral beak deviations (scissors beak) and mandibular prognathism, as there is more rapid growth and a greater potential for rapid remodeling. Those species with higher rates of rhamphothecal growth rates, such as birds of prey and psittacine birds, are also better orthognathic candidates.[4,80,81] Dental acrylics and resin-based composites can be used for orthognathic procedures and beak repair. Chemically activated monomeric compounds are efficient adhesive systems for use on the rhamphothecal surfaces of birds. This efficiency is attributed to the micromechanical retentivity obtained, due to the low-molecular-weight adhesives and their ability to penetrate between the layers of keratin and then polymerize afterward, producing a stronger and more efficient seal. Combinations of different primers, acrylics, and resins can be utilized for added strength and versatility. Table 31.3 displays some of the common adhesive systems that may be used, examples of common brand names, and their applications in beak repair and rhamphorthotics. Because some of these products, particularly the methyl methacrylates, produce an exothermic reaction during the polymerization process that may be damaging to living tissues, the specific physiochemical properties of the product should be understood prior to its use. Resin-based composites generally reach lower temperatures than acrylics during polymerization.[5,6,82]

Light-curing composites are composed of light-sensitive synthetic materials that can be molded and used to fill large defects or create beak orthoses or prosthetics. The use of a curing light may speed composite polymerization but, depending on the product, may not be mandatory as some are self-curing. Methyl methacrylate can be used as a light but strong binding adhesive material in the construction of external fixators (see Box 31.1 for a summary of the procedure).

TABLE 31.3

Materials Available for Beak Repair, Orthotics, or Prosthetics[82–89]

Material Class	Material Subdivision (When Applicable)	Applications	Trade Names (Not Comprehensive)	Notes for Use
Cyanoacrylics—soft tissue medical adhesives		Beak repair, soft tissue repair	Tissue-glue, Vetbond, Dermabond	
Methacrylate resins—also known as acrylic or acrylic resins	Methyl methacrylates	Beak prosthesis/orthosis	Ortho-Jet, Technovit, Vipi Flash	Very exothermic, strong fumes; come as a powder and liquid, which must be mixed
	Ethyl methacrylates		Kooliner	Less exothermic; comes as a powder and liquid, which must be mixed
	Other methacrylates		Tuff-Temp Plus	Dual cure; comes in a dual-syringe system with a mixing tip
Proprietary material	Urethane dimethacrylate	Prosthesis/orthosis	Triad TranSheet	Light cure, comes as a formable sheet; must be used with a bonding agent or cement
Provisional composite resins and restoratives	Bis-acrylic composite; BIS-GMA	Prosthesis/orthosis	Protemp Plus, 3M ESPE, Maxi-Temp, TempSpan, Build-It	Self-curing or dual curing; typically come in a dual-syringe system with a mixing tip; much less exothermic
	Flowable composite	Prosthesis/orthosis in conjunction with other materials	Silkflow, Natural Elegance Flowable	Light cure
	Packable composite	Prosthesis/orthosis in conjunction with other materials	Filtek P60, Filtek Z250, Filtek Z350	Light cure
Stainless steel orthopedic wire		Prosthesis/orthosis in conjunction with other materials	Many brands	Used for additional stabilization in association with other techniques
Fiberglass		Prosthesis/orthosis	Many brands	Used for additional stabilization in association with other techniques
Resin cements		Prosthesis/orthosis in conjunction with other materials	3M RelyX, Enforce, Panvia	Dual- and light cure; come as two syringes, which are mixed
Bonding adhesives		Prosthesis/orthosis in conjunction with other materials	Prime + Bond 2.1, Optibond, Scotchbond, Enforce	May need to be used with an etch, or may be self-etching; light cure

BOX 31.1 STEPS FOR APPLYING ADHESIVES TO BEAK KERATIN

1. Clean and remove debris from the surfaces.
2. Remove organic tissues and oils from the surface of the beak, exposing the spaces between the layers of keratin for polymer application. 70% Ether and alcohol are helpful. Take caution to prevent contact of these solutions or their fumes with the eyes or upper respiratory epithelium.
3. Prepare the surface to be restored with chemically activated monomeric adhesives, providing for optimal mechanical microretention.
4. Final construction is accomplished with polymeric material capable of polymerizing on the monomer adhesive previously used.

Once polymerization is complete, both resin-based composites and acrylics can be reshaped and/or polished with a drill or Dremel tool, although some can be extremely hard and difficult to reshape. A review of bone cements and their potential use in mandibular endoprosthesis in humans concluded that plain polymethyl methacrylate (PMMA) is the default choice for the initial development phase of this procedure. Modified PMMA cements, composite resin cements, osteoinductive calcium phosphate compounds, and cementless fixation are options that offer some advantages over PMMA cements, and, therefore, further research should be conducted to study their suitability and use in birds.[90]

SPECIFIC PROBLEMS AND PROCEDURES

Lateral Beak Deviation (Scissors Beak)

Lateral beak deformity is most often seen in macaws[1,68,91] but can also be seen in other psittacine species as well as doves, raptors, and domestic chickens, among others.[1] The causes of many pediatric developmental beak abnormalities are incompletely understood but are likely complex, variable, and multifactorial.[1,5,84] Lateral beak deformity is characterized by a lateral bowing of the maxillary beak, including the rhinotheca and often including the underlying maxillary beak bony

structures,[1,27,81,92] which generally occurs when there is a disrupted or altered growth of the bony structures of the maxillary beak such as the rictus or the premaxilla, nasal, and frontal bones.[1] Proposed underlying causes of lateral beak deviations include genetic abnormalities, incorrect egg incubation techniques, malnutrition, mycotoxicosis, improper hand-feeding techniques, and trauma.[5] As force vectors are repeatedly applied during regular beak use and with the normal rates of continued rhamphothecal growth, this deformity often will become progressively worse, ultimately generating a "scissors effect".[66,78] Uneven and imbalanced abrasion results in lateral rhinothecal and gnathothecal tomial elongation or shortening, leading to more pronounced abnormal occlusion.[5,80,91] Severe deformity may ultimately result in difficulty prehending food.

The details of treatment vary with the severity of the problem and the age of the bird.[5] In young birds with mild and early deviations, application of digital pressure regularly to the appropriate side of the beak for several minutes (two to three times daily) may lead to effective realignment. Progressive beak malalignment and abnormal rhamphothecal wear often require periodic corrective keratin-reshaping procedures in adult birds.[67,79] In some circumstances, this may be an effective management strategy, if the frequency of these procedures and stress sustained by the bird are not excessive or noted to carry adverse behavioral consequences. Corrective surgical procedures may be indicated for more severely affected cases or for those noted in young birds to reduce or eliminate the need for a lifetime of corrective rhamphothecal shaping procedures and optimize the welfare of the bird.[1,5,80,91]

Surgical corrective techniques for lateral beak deviation

Surgical correction is most successful in young birds when the beak tissues are more fully ossified, but still developing and more pliable than in adult birds.[1,5,80] There are two main types of orthognathic repair procedures. The first involves building a ramp orthotic device along the lateral wall of the beak to redirect bite forces to a more normal plane. This technique is employed more often in immature birds. The second involves applying lateral redirectional forces to the maxillary beak through transfrontal pinning and the use of tension wires or bands.[1,80]

For the beak ramp orthosis, an extension (inclined plane) of the lateral mandibular beak wall is created using structural adhesive resins such as liquid acrylic, epoxy putty, or light-cured dental resin composites.[80,81,84,85] The device is applied to the mandibular beak on the same side to which the maxillary beak is deviating and redirects bite forces to a more natural plane (Figure 31.25). Although ramp orthoses are most commonly applied to the mandibular beak, a maxillary beak ramp orthosis was successful in correcting a lateral beak deformity in a 2-year-old striped owl *(Rhinoptynx clamator)*.[81] The height of the orthosis must be tall enough to prevent the opposite beak from extending over it.[80] Stainless steel mesh, nylon mesh, cerclage wire, or a combination of materials can be used to support, build, and reinforce the orthosis.[1,81] Anesthesia may not be necessary if rapidly setting materials are used, such as dental composites, and if construction of the device does not involve perforation of deeper tissues such as bone, and if the patient is amenable to being held still during application.[1] Lightly notching the rhamphotheca can improve adhesion of the ramp construct to the beak keratin. Extending the orthosis to wrap around the beak to the opposite side can also strengthen the device.[80] The orthotic device should be designed to functionally create a deflecting lateral pressure from it that is relayed to the lateral maxillary beak. The amount of resin or orthotic material applied should be sufficient to cause occlusion of the key rhamphothecal tomial normal position, without preventing the bird from feeding. Normal beak movements and feeding, in this light, facilitate the effectiveness of the orthotic device. If the lateral rhamphothecal tomia are elongated due to uneven wear, the keratin should be reshaped to a more normal appearance to permit improved apposition. Potential complications include malalignment at the mandible-quadrate interface and fracture of the mandible, particularly in immature birds. Some orthotic devices may loosen and fall off, necessitating repeat application.[4,79] Correction using a ramp orthotic typically requires 1 to 3 weeks.



FIGURE 31.25 Ramp orthosis applied to a young scarlet macaw *(Ara macao)* to correct a left lateral maxillary beak deviation. The height of the ramp must be sufficient to prevent the bird from elevating the maxillary beak up and over the point of the orthosis. The device is removed when the alignment appears to have been corrected, or the ramp no longer contacts the lateral rhinothecal wall. (With permission from Abramson J, Speer BL, Thomsen JB, editors: *The large macaws*, Fort Bragg, CA, 1995, Raintree Publications.)

Tension band techniques are considered by some to be more effective for severe beak deviations in adult birds but can be effective also in subadult birds.[1,4,91] In the first technique, a transfrontal pin is utilized to facilitate lateral tension delivery to the distal maxillary beak (Figure 31.26). An intramedullary (IM) pin or Kirschner wire is carefully drilled transversely through the frontal bone caudal to the nasofrontal hinge and caudoventral to the nares.[1] Threaded pins are preferable to nonthreaded pins. The exposed end of the pin on the side of the beak opposite the direction of the deviation is left long and used to construct a connecting rod for tension band placement. The pin can either be left straight and short or bent at a 90-degree angle and extended rostrally parallel to the maxillary beak with the end in close apposition to the tip



FIGURE 31.26 (A) Right lateral beak deformity in a young blue-and-gold macaw *(Ara ararauna)*. Note that the maxillary beak is mildly skewed to the bird's right, exposing the left lateral aspect of the gnathothecal tomium, which in turn will lead to more progressive rhamphothecal overgrowth and deformity. (B) Ventral view of the rhinothecal surfaces of the same bird. Note the distortion of the rhinothecal occlusal ledge as the result of imbalanced occlusion of the rostral gnathothecal tomium. (C) A transfrontal pin has been placed with the threaded end of the pin seated in the right frontal bone and a 90-degree angle employed to facilitate direct lateral force being applied by a rubber band to the distal tip of the maxillary beak. Note that malocclusion appears to have been corrected. Prior to anesthetic recovery, the deformity of the rhinothecal occlusal ledge was reshaped. (D) A different blue-and-gold macaw 3 days after a similar procedure. The tension band has been loosened, and occlusion appears to have been corrected. Note that the transfrontal pin procedure was slightly different, in that the pin was completely driven through the right frontal bone and then curled, as opposed to the threaded pin used in (C). One potential disadvantage of this latter method is that the pin can shift laterally, resulting in variability in the degree of tension delivered to the distal maxillary beak. This pin was left in place for another 24 hours to verify that functional occlusion had been corrected, prior to cutting the pin where it exits on the left frontal side and removing it. (Photos courtesy Brian Speer.)

of the maxillary beak.[1,4,80] An elastic band is then looped around the tip of the maxillary beak at one end and through a loop created in the distal end of the pin at the other, applying gentle lateral tension to the beak tip. There are no biomechanical studies that have determined the amount of force to be applied in this procedure, but the use of small forces for long periods is generally preferred. The exit points of the pins or screws implanted in the frontal bone should be cleaned daily with chlorhexidine solution (0.12%). Effective correction can often be accomplished in a matter of a few days to 2 weeks in most birds.[1,4,80] Potential complications from this procedure include infection and iatrogenic damage to the frontal bone and sinus, nasofrontal hinge, and maxillary beak structures, particularly in young birds.[80,92]

In another tension band technique, an orthopedic screw (wire or pin may on occasion be used alternatively) is placed transversely into the frontal bone caudal to the nasofrontal hinge and caudoventral to the nares, opposite to the direction of the deviation, similarly to the placement of the transfrontal pin described earlier (Figure 31.27). Another screw (or wire/pin) is then placed into the rostral portion of the beak, seated in the distal maxillary or premaxillary bone. In some cases, the screw can be placed laterally into the rhinothecal occlusal keratin ledge, and not be directly seated into bone itself.[93] An orthodontic elastic (rubber or band) is then added, extending from the frontal bone pin/screw and stretched to the rostral pin, providing tensile force that is delivered throughout the lateral side and length of the beak.[1,4,26] There are no biomechanical studies that have determined the amount of force to be applied in this procedure, but the use of small forces for long periods is generally preferred, as with the transfrontal tension band procedure described earlier. The exit points of the pins or screws implanted in the frontal bone should be cleaned daily with chlorhexidine gluconate solution (0.12%). Complete correction of the lateral beak deformity may take up to 10 months.[1] Similar to the transfrontal technique, potential complications from this procedure include infection and iatrogenic damage to the frontal bone and sinus, nasofrontal hinge, and distal maxillary beak structures, particularly in young birds.[1,4]



FIGURE 31.27 In this golden conure *(Guaruba guaruba)*, orthopedic screws are being used to provide the anchor for elastics in the correction of a right lateral maxillary beak deviation. (Photo courtesy Roberto Fecchio.)

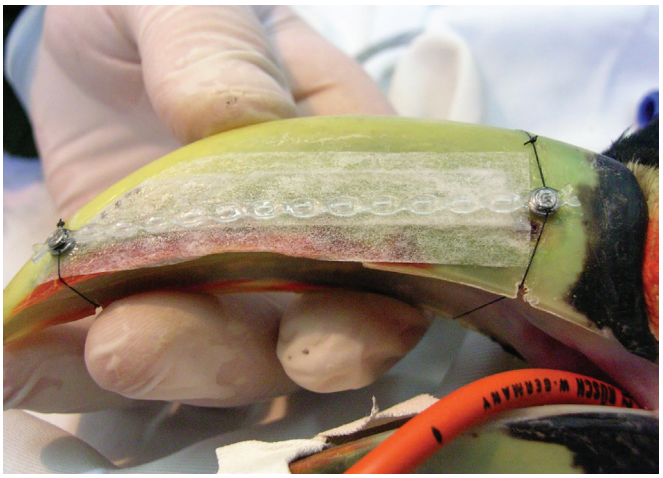


FIGURE 31.28 Application of orthodontic buttons for elastic fixation and correction of right lateral maxillary beak deviation in green-billed toucan *(Ramphastos dicolorus)*. The buttons provide functional anchors for the elastics applied, which then can allow for a much greater timeline for pressure to be applied to achieve the desired correction. The different techniques for correction of lateral maxillary beak deformities should be expected to vary depending on species and patient-specific details. In a longer-billed species such as this toucan, this approach may be a more viable option over a transfrontal pin or ramp orthosis. (Photo courtesy Roberto Fecchio.)

In bird species with slower beak keratin growth rates such as toucans, variations of these techniques should be used in a minimally invasive manner with less deep attachments to bone so that the corrective device can be maintained for years. This also can allow less secondary damage to the underlying structures and easier replacement of the device in the future. In many cases, the use of orthodontic buttons (or brackets) attached helps to anchor elastics, as in the case of toucans (Figure 31.28).[4]

Mandibular Prognathism

In brachygnathism, the maxillary beak is longer than the mandible (overshot). In prognathism, the mandible is longer than the maxilla (undershot).[94] The appearance of these two general deformities can be the result of true anatomic deformity in the actual length of the bills, or as the result of hyper-retraction of the maxillary beak in the circumstance of mandibular prognathism. Mandibular prognathism is most often seen in cockatoos but can occur in other avian species (Figure 31.29).[91] The distal maxillary beak rests caudal to the rostral margin of the mandibular beak, within the oral cavity ("underbite").[91] Mandibular prognathism can be a result of excessive contraction of the muscles that lower the maxillary beak (retraction), an abnormally curved or otherwise malformed maxillary beak, or excessively elongated mandibular beak structures.



FIGURE 31.29 Mandibular prognathism in a young hand-feeding cockatiel *(Nymphicus hollandicus)*. This deformity was successfully corrected by regular manipulation of the upper beak during each handfeeding, without the need for the application of orthotics. (Photo courtesy Brian Speer.)

Surgical corrective techniques for mandibular prognathism

As with lateral beak deviation, repair is most successful in young birds when the bony structures are still growing and pliable. Manipulation of the beaks can have early corrective merit in early cases or particularly younger birds. There are two main methods of repair; one that employs the construction of an extension along the tip of the maxillary beak and another that involves applying rostral redirection forces to the maxillary beak through tension wires or bands. Both methods apply tension to the *mm. pterygoideus* and *pseudotemporalis profundus*, which are often contracted in affected birds, resulting in continued abnormal lowering (retraction) of the upper beak.[1,6,26,92,93] The treatment choice varies with the severity of the condition and the age of the bird. In young, hand-feeding chicks, regular manipulation and extension of the maxillary beak may help mild cases be returned to normalcy. There are two general types of surgical repair for mandibular prognathism: application of an orthotic device and utilization of tension band techniques.

Surgical procedure. A variety of acrylic or dental composite products can be used for this procedure. A functional cap, extending from the cere distally, and encompassing the pressure-bearing keratin at the occlusal ledge of the upper mandible, is progressively constructed. The prosthesis is structured to duplicate and exaggerate the relative dimensions of a normal upper mandible so that the bird can function normally when it is applied. The lower bill must not be able to extend out and beyond this prosthesis, and the tomium of the gnathotheca should be capable of applying normal force at the area of the occlusal ledge of the rhinotheca (Figure 31.30).

Careful placement of a small sterile pin dorsally to ventrally directed through the tip of the premaxilla[5] or a small pin/screw into the lateral aspect of the rhinothecal occlusal ledge may help to stabilize the device.[94] This process functionally extends the upper beak, preventing placement of the maxillary beak into the lower beak, enabling normal occlusion and a normalization of the range of motion of the hyperflexed and hyperextended prokinetic joints. Pins placed through the bone of growing birds may result in trauma to the softer, pliable tissues and caution is warranted with their use. A method using dental composites has been described, which does not utilize anesthesia and may offer advantages in some cases.[95] A surgical procedure, utilizing a modification of the transfrontal pinning procedure for lateral beak deformity, has also been described in adult cockatoos with chronic mandibular prognathism.[96]

For the tension band technique, transverse placement of a pin through the frontal bone is similar to that described earlier for correction of lateral beak deviation. A second small pin or small threaded screw is placed in the bone of the maxillary beak along the midculmen at the point where inward rotation of the maxillary beak is most severe.[1] Structural adhesive resins can then be applied to support the second pin.[1] An elastic band is placed from either exposed end of the frontal bone pin to the premaxilla pin. The band can be removed after normal alignment is restored, and the pins removed once this correction is assured.[1] Potential complications with this procedure are related to the risks of trauma to bony and vascular tissues.

Beak Fractures

Fractures of the beak are often the result of trauma, although pathological fractures are also noted.[4–6,97,98] Initial treatment for these injuries consists of controlling local hemorrhage, pain management, and general patient stabilization.[5,6] Once the patient is stable, the wound should be debrided in order to remove dirt and cellular debris, and it should be kept clean until the specific treatment of the injuries is performed.[4,6,97,98] Those individual birds that have compromised ability to prehend and eat food items should be supported as indicated to maintain their caloric balance. Although some traumatic fractures and avulsions significantly alter the bird's appearance, many psittacine birds with functional amputations of their upper and (on occasion) lower beaks can learn to eat on their own, with time.[99] Thus, despite the belief of some, euthanasia is often not an absolute indication. Moreover, for those birds that learn to feed adequately on their own, and that do not have substantial malocclusion, further efforts at developing prostheses are not indicated.[26,99]

Imaging procedures including projection radiography and CT of the skull can be particularly helpful in planning a surgical repair. When there is outright loss of bone or vascular compromise, the prognosis for return to normal form and function is guarded. An attack by a cat resulted in slight lateral deviation of the mandibular beak and reluctance to open the mouth in an adult double yellow–headed Amazon parrot *(Amazona oratrix)*.[100] A mandibular fracture and disruption to the joint space between the mandible and quadrate bone were identified by CT. An esophagostomy tube was placed and the injury was allowed to heal without surgical intervention, although the ability to open the beaks remained



FIGURE 31.30 (A) Mandibular prognathism in a juvenile Moluccan cockatoo *(Cacatua moluccensis)*. This condition was successfully corrected using a maxillary beak orthosis. (B) Ducorps' cockatoo *(Cacatua ducorpsii)* approximately 10 days following application of a maxillary beak acrylic orthosis. Note the exposed proximal rhinothecal keratin, which was originally covered by the orthotic device because of the relatively rapid keratin growth rate. (C) The same Ducorps' cockatoo at day 20, when the orthosis loosened and fell off, and prior to removal of accumulated laterally exfoliating keratin. In this case, dental pins or screws were not employed to aid in stabilization of the orthosis. (Photos courtesy Brian Speer.)

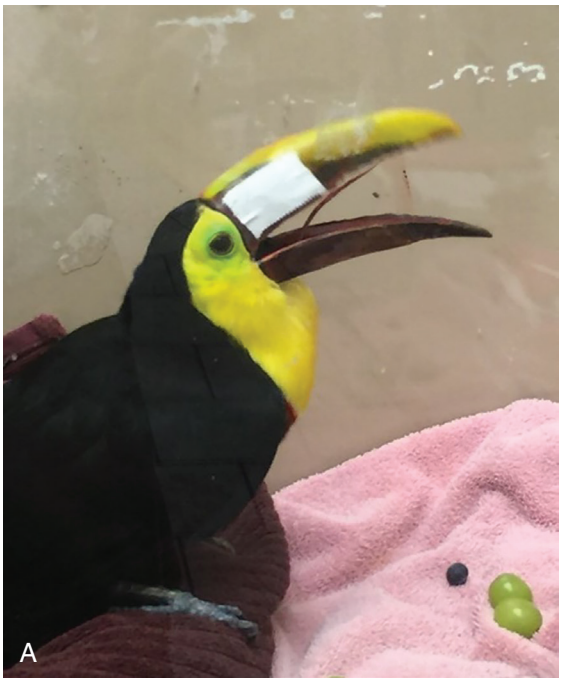
compromised.[100] A 22-year-old hybrid macaw with a rigid nasofrontal hinge and laterally deviated jugal bone from a dog bite was diagnosed by CT and was successfully treated with surgical removal of the nasal-frontal-braincase and jugal-orbital arch.[70] Alternatively, surgical procedures using orthopedic or dental techniques and devices such as stainless steel plates,[101] IM pins,[102] osteogenic distractors,[103,104] dental resin composites,[105] and elastic bands,[1,4] among others, have been described and used to surgically repair beak fractures in avian patients.

Rhamphothecal cracks, fissures, and puncture wounds

Rhamphothecal cracks and fissures are most often the result of mild trauma, although there can be metabolic, nutritional, behavioral, or other factors that can contribute to their development. Treatment typically includes corrective rhamphothecal grinding to remove the crack and restore normal occlusion and alignment or local antisepsis and recoating with resins to avoid opportunistic infections. Cracks in the rostral gnathothecal

tomium, where there is no dermal or bony involvement, may be effectively addressed by grinding the surfaces to eliminate or shorten them. This often is done serially, with quantitative measurements of the length of the crack before and after each procedure. Afterward, many birds can benefit from strategic training aimed at altering their chewing/force delivery behaviors and resultant tomial wear.

Fissures and puncture wounds that penetrate through dermis and into bone structures of the beak can be covered with cellulose-based adhesive and cyanoacrylate-based adhesives (Figure 31.31), but the latter have irritative volatile substances that should not come in contact with the ocular and nasal mucous membranes. As indicated, pain management should be considered. When secondary bacterial infection is present,



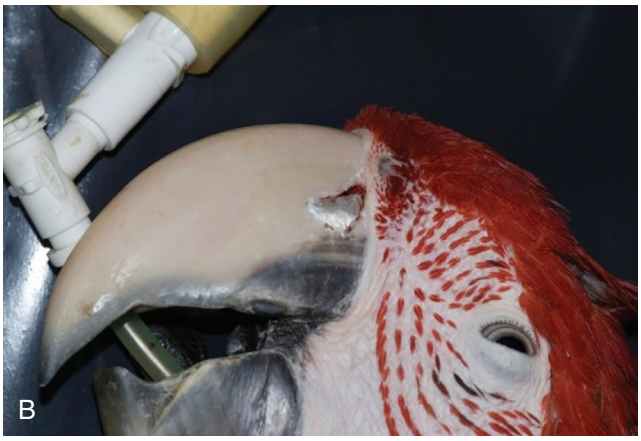




FIGURE 31.31 (A) This Swainson's toucan *(Ramphastos ambiguus swainsonii)* is recovering from sinus trephination and sinoscopy to remove granulomas that were within the nasal diverticulum of the infraorbital sinus of the maxillary beak. Both surgical and traumatic perforations through the rhamphotheca and into the bony structures and airspaces beneath them can be managed as open wounds, patched as in this case with adhesive bandaging material, or a variety of acrylic and dental composite materials. (B) Penetrating injury to the left proximal maxillary beak of a green-winged macaw *(Ara chloroptera)*. An injury like this must be investigated, as there will be variable degrees of bone, dermal, and keratin injury. With the bird anesthetized, the wound can be aseptically cleaned, and general wound care principles include replacement of compressed bony substructure, repositioning germinal layer, providing support for these tissues, maintaining a moist environment, removing fibrin clots, and preventing infection. (C) Following careful cleaning and replacement of the depressed tissues, this wound was covered with dental composites to protect and stabilize while healing. (A, Photo courtesy Brian Speer. B, C, photos courtesy Nigel Harcourt-Brown.)

systemic antibacterials and/or antifungals should be administered.[91] Some puncture wounds can effectively be treated topically without the need for covering them, or with light covering with adhesive bandages.

Mandibular symphyseal diastasis and fracture

Separation (diastasis) and/or fracture of the mandibular symphysis is a common injury in companion birds and is most often a result of an impact injury to the beak. The two mandibles are joined in the rostral midline at the *rostrum (symphysis) mandibulae* but this connection can easily become separated with trauma. Separation of this joint would be properly termed a diastasis, although fractures of the adjacent mandibular bone can also be seen, producing a similar net result (Figure 31.32). These injuries often have a guarded to poor prognosis for successful repair due in part to the thin nature of the vascular dermis and bone in this region.[72,106] If a repair is to be successful, it must be attempted soon after the injury occurs.

Successful repair techniques have been described in a Grey parrot, and have been performed in smaller psittacines (Figure 31.33), and, by the author, in chickens, and a mallard duck. In the Grey parrot case report in an acute injury scenario, interfragmentary wiring was placed through predrilled pilot holes, a 22-gauge wire was placed to provide fragmentary alignment, and the twisted ends of the wire were incorporated into a self-curing, provisional bis-acryl composite splint. Particular care was taken to minimize the amount of composite contacting the gnathothecal surfaces of the injury and the exposed dermal tissues beneath.[100] In smaller birds, a bridging construct created from structural adhesive resin may be adequate (Figure 31.33).[67] In larger birds, reinforcement with transmandibular placement of stainless steel cerclage wire or pins is often necessary (Figure 31.32).[72] The pins can be externally secured to each other using elastic bands,[1] or with loops of polypropylene or stainless steel suture or wire.[5] Pins and wire can then be covered with adhesive resin for added support and protection, with care taken to avoid direct contact with the resins to the rhamphothecal surfaces of the injury.

General beak fracture surgical repair

Significant fractures that may result in compromised healing and beak function should be stabilized and repaired. Basic



FIGURE 31.32 (A) Mandibular symphyseal diastasis fracture in an adult 8-year-old Grey parrot *(Psittacus erithacus)*. (B) Interfragmentary wire placement through predrilled holes, allowing intraoral wire passage and circumferential interfragmentary compression across both mandibles. (C) Following application of dental composite, which incorporates the wire twists but has minimal composite directly contacting the beak surface. (D) 18 Days postoperative photograph of the patient. The interfragmentary wires were removed at day 47 postoperatively, and at 21 months postoperatively, functional healing and normal rhamphothecal growth and wear were affirmed. (Photos courtesy Christoph Mans.)

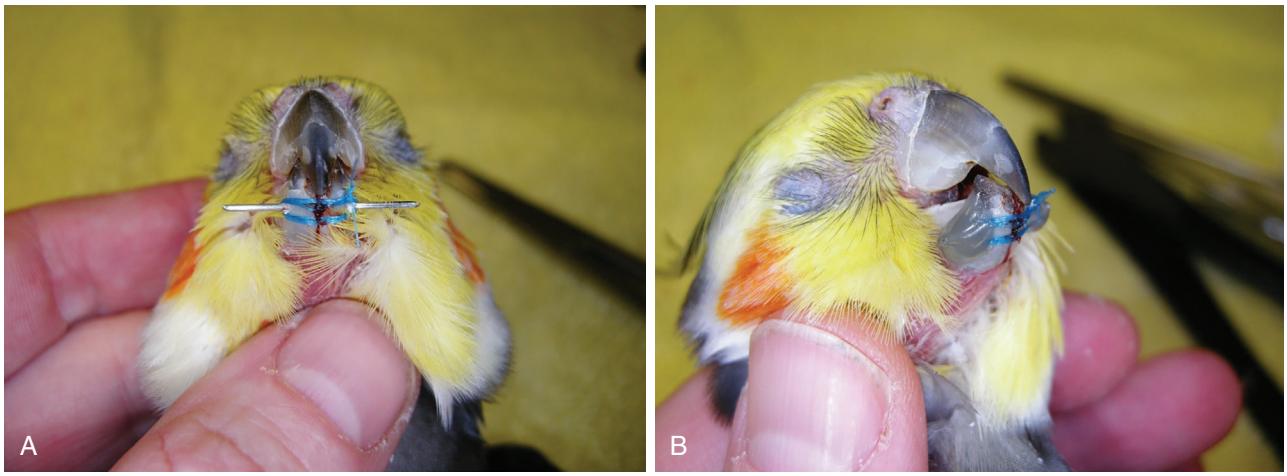


FIGURE 31.33 Repair of a mandibular symphyseal diastasis/fracture in an adult male cockatiel *(Nymphicus hollandicus)*. A successful repair was accomplished with the use of a single transmandibular pin (25 G hypodermic needle), around which polypropylene suture was looped (A), and then covered with liquid acrylic (Technovit Liquid; Jorgensen Laboratories, Loveland, CO, USA) (B). Note that the acrylic was not applied to the fracture line. (Photo courtesy Lauren Powers DVM, DABVP (Avian Practice); with permission.)

immediate functional goals with repair methods are to restore the repaired beak to immediate capability of function while healing occurs.[2] Fracture repair details are dependent on the location and extent of the injury. Fractures closer to the neurocranial articulations may be more complicated due to the biomechanical forces that will be present during the postoperative period.[4] Fractures with loss of bone fragments tend to present greater difficulty of repair and, consequently, reserved prognosis. In young birds, the chances of bone growth and remodeling in the fracture site are higher. To initially stabilize the fracture fragments, it is necessary to apply cerclage with atraumatic material (fine wire, nylon) that functions to prevent excessive torque and mobility of the fragments (similar to surgical correction of symphysis fracture or luxation). High-speed dental drills may be utilized to assist the perforation of the beak and its bony structures for the passage of the cerclage wires or nylon (Figure 31.34). The perforations should be made perpendicularly to the fracture lines when the fragments are lightly reopposed. Historically, bone plates and screws have not been utilized because the bone of the beak is so thin and has virtually no soft tissue coverage, although there are case reports of successful procedures.[101] Additionally, there are plates specifically designed for maxillofacial surgical procedures that may be more amenable to use in birds than the traditionally adapted ones used in the past. Plates may be used in large birds with fractures of the caudal mandibular beak where the bone is thicker and covered by muscle and normal skin. The thin bone of particularly the maxillary beak in most birds does not provide adequate screw purchase and bony integration. Another potential application for the use of plates is by placement over the rhamphothecal surface of some fractures. Used in this manner, the plate acts more like an external fixator because the device is outside the body tissues while the screws placed in the bone maintain effective reduction of the fracture and provide stability. Necrosis of the tissues (dermis) underneath the plate is possible, but if the fracture quickly heals these collaterally damaged tissues may still be capable of healing.[107] IM pins and orthopedic wires are also difficult to apply to the avian beak because there is no distinct medullary cavity within the flat bones of the beak, and the paucity of soft tissue over the bone would result in wires being exposed. Mandibular

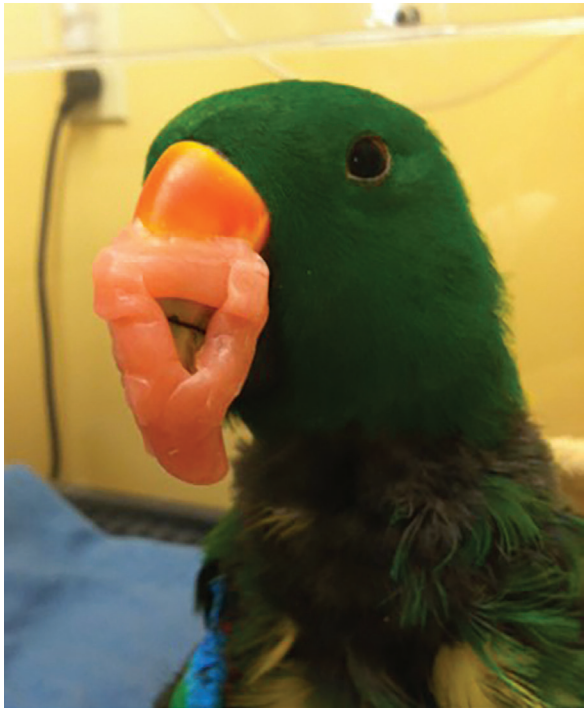


FIGURE 31.34 A transverse, distal third fracture of the maxillary beak in an adult male Eclectus parrot *(Eclectus roratus)*. Stabilization and protection of the fracture site in this case was accomplished with the use of a framework of light-cured acrylics while keeping direct contact with the fracture line minimized. (Photo courtesy Laura Wade.)

fractures, however, may facilitate the use of intermedullary pin placement and even the use of IM tie-in fixation strategies (Figure 31.35).

Interfragmentary wires or silk alone are inadequate to counter the forces acting on the fracture; however, they will hold fragments in apposition.[107] After this initial aspect of fixation, the strength of the interfragmentary wires or silk should be augmented to facilitate a rapid return of functional use of the injured beak. Such augmentation to the interfragmentary stabilization is obtained with the aid of adhesive materials and resins. Small dental pins or posts can be utilized to improve attachment of resins to the nonfractured sites of the rhamphotheca. It is essential that the surface of the beak is clean and dry prior to the placement of adhesive and resin material. As with the method described for mandibular symphyseal diastasis and fracture repair, particular care should be



FIGURE 31.35 Bilateral mandibular fracture repair in a Mallard duck *(Anas platyrynchos)*. (A) Bilateral distal third fractures of both mandibular rami are evident in this 4-week-old injury. (B) Necrotic debris was cleaned and removed, providing fresh edges of the distal and proximal mandibular fragments, and the wound was then flushed and aseptically prepared. (C) Small intermedullary pins were placed normograde into the distal fragments and out of the rostral aspect of the mandibles, then retrograded to ultimately be seated at the angle of the mandible, providing linear stability to the fractures. The ventrally bent rostral end of the pin is evident *(a)*, the location of a small threaded pin that was placed into the distal angle of the ramus is pointed out *(b)*, intravenous tubing filled with methyl methacrylate is placed over the ends of the distal intramedullary pin and the proximal threaded pin *(c)*, and a small transverse bar of methacrylate filled tubing is placed laterally between the two rami for additional stability.

Continued



FIGURE 31.35, cont'd (D) and (E) Four weeks postoperatively following removal of the fixator at 3.5 weeks, there is good functional healing of both rami.

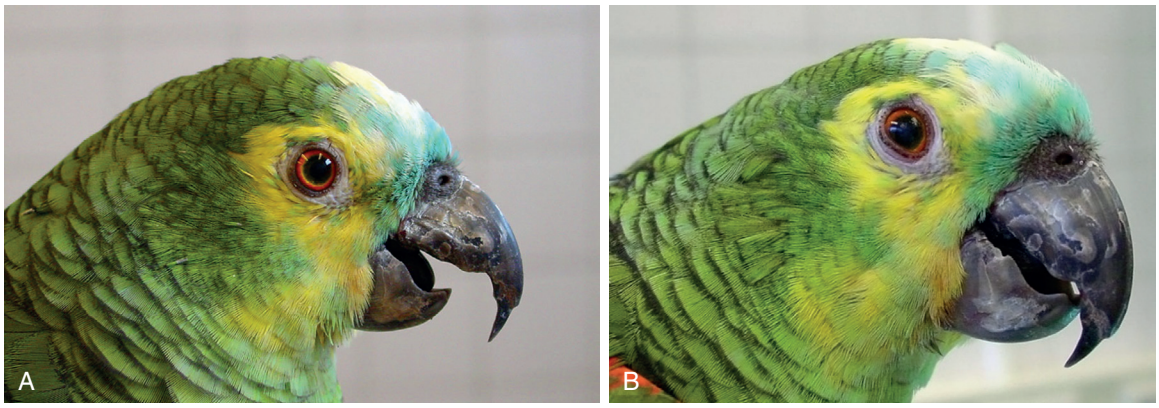


FIGURE 31.36 Palatine luxation in a blue fronted Amazon (*Amazona aestiva*). Note the hyperextension (protraction) of the maxillary beak (A). There was a complete inability to retract the beak as a result. To reduce the luxation, an intramedullary pin or Kirschner wire is driven transversely through the preorbital diverticulum of the infraorbital sinus dorsal to the jugal arch (or similar access is accomplished by a surgical infraorbital sinusotomy. Downward pressure is applied on the palatine bone while gently protracting (elevating) the maxillary beak. This enables the palatine to slide back over the mesethmoidal ridge, allowing the maxillary beak to return to its normal position (B). Photo courtesy Yvonne van Zeeland and Nico Schoemaker, with permission. Copyright Veterinary Information Network®, Inc.; Photo by Dr. Chaowaphan Yinharnmingmongkol. Republished with permission.

taken to minimize the amount of composite contacting the rhamphothecal surfaces of the injury.[2,4,87] Several resins can be used for fracture repair, but methacrylate-based resins must be cooled (irrigated or aerated) during their polymerization, as they have an exothermic phase and can cause burns and necrosis of the beak. In addition, volatile gases from acrylic resin can irritate the eyes and respiratory tracts of birds as well as the operators.[94]

Palatine bone luxation

Traumatic luxation of the palatine bone has been described in several psittacine birds, most commonly macaws.[1,80,108–110] Luxation occurs when the palatine bone is moved rostrally and then slips dorsally beyond its normal end-point on the mesethmoidal bony ridge at the ventral base of the neurocranium.[5,80] Luxation results in pathologic hyperextension and inability to close the maxillary beak (Figures 31.36 and

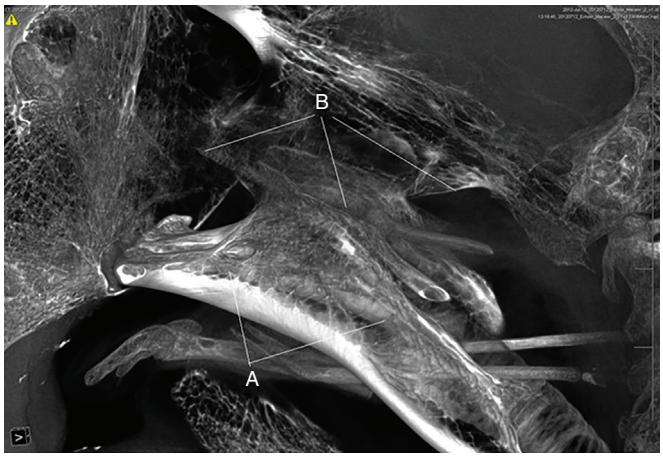


FIGURE 31.37 Micro-computed tomography (micro-CT) image of normal anatomy in a green-winged macaw *(Ara chloropterus)*. Rostral is to the left, and caudal is to the right. The mesethmoidal ridge at the base of the neurocranium (B) is visible, where it allows the central articulation with the palatine (A). When the palatine is moved too far cranially by hyperextension (protraction) of the maxillary beak, its caudal edge can catch at the cranial aspect of the mesethmoidal ridge *(far left line)*, enabling a functional lock, unless it is pulled slightly more craniad, then pushed downwards, allowing it to be replaced on the mesethmoidal ridge. (Image courtesy Scott Echols.)

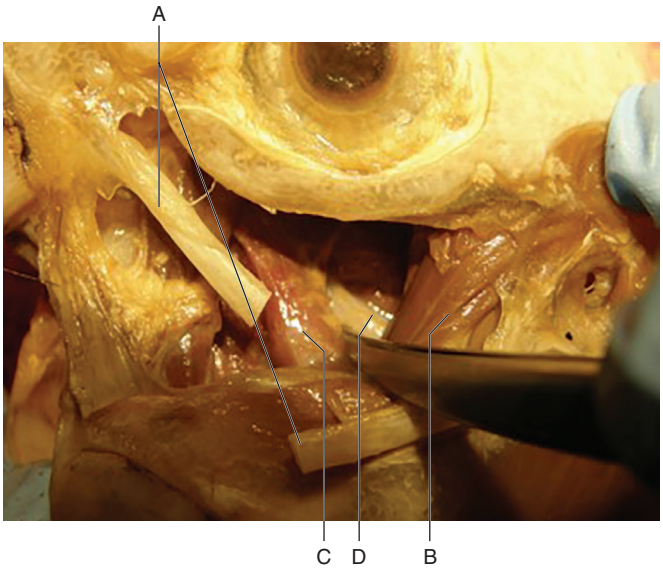


FIGURE 31.38 Cadaver dissection showing surgical access to the right jugal and pterygoid bones in a blue-and-gold macaw *(Ara aurana)*. Rostral is to the right. The infraorbital sinus has been accessed, and the jugal (A) has been cut with its rostral segment ventrally retracted. *M. adductor mandibulae externus ventralis* (B) is being cranioventrally retracted, and *M. ethmomandibularis* (C) is exposed at the tip of the scissors. The diaphysis of the pterygoid is exposed and visible deep to these two prokinetic muscles (D).

31.37).[5,80] The diagnosis is made based on history, examination findings, and radiographic findings.[1,80,108–110]

Birds may be able to spontaneously reduce the luxation on their own.[100] If the luxation does not quickly self-correct, surgical intervention is warranted.[1,80,108–110] To reduce the luxation, an IM pin or Kirschner wire is driven transversely through the preorbital diverticulum of the infraorbital sinus dorsal to the jugal arch. Care should be taken to not damage the ethmomandibularis muscle, which originates high on the rostral part of the interorbital septum and inserts onto the dorsomedial aspect of the mandible, serving to elevate (protract) the mandible (see Figures 31.4 and 31.5 and Table 31.2). Downward pressure is applied to the pin and the dorsal margins of the palatine bone, or directly should an infraorbital sinusotomy be utilized, while simultaneously extending the maxillary beak, enabling the palatine bone to slide back over the mesethmoidal ridge and allowing the maxillary beak to return to its normal position.[1,80,109,110] The pin is then removed from the sinus. Alternatively, a sinusotomy can be performed, and direct placement of pressure on the palatine bone with a variety of blunt instrument options can be accomplished in this open manner.[80] Additional stabilization can be provided by passing a suture around the suborbital arch (in those species that possess one) and jugal arch through a lateral approach (see Chapter 29).[1,80,110]

Fractures of the jugal, pterygoid bones

These specific injuries may be identified when evaluating injuries to the head and skull. When indicated, surgical repair is possible. The surgical approach to these bones can be made through the infraorbital sinus. The body of the infraorbital sinus is a large and spacious triangular cavity under the skin in the lateral region of the maxillary beak, and rostroventral to the eye.[111] The margins of the largest aspects of the infraorbital sinus are largely defined by the jaw muscles.

Surgical procedure (Anodorhynchus and Ara species). The skin ventral to the jugal arch is incised, exposing the lateral wall of the sinus. Retract the maxillary beak (closing), while extending the lower beak (opening). This functionally enlarges the sinus space and enables greater access within. Incise into the infraorbital sinus space ventral to the jugal arch (see Chapter 29). The jugal can be cut and reflected to increase deeper exposure into the infraorbital sinus (Figure 31.38). Fractures of the jugal arch can be identified and repaired with the use of hexalite shuttle pins and bone cement, or transcortical suture or wire. The infraorbital sinus space can be visually inspected, and magnification loupes with a focal light source, operating microscopy, or endoscopy can be used in selected cases for deeper visualization if indicated. Identify and caudoventrally retract *m. pseudomasseter* and other bellies of *m. adductor mandibulae externus*, *m. pseudotemporalis superficialis*, and rostrally retract the *m. ethmomandibularis*. The diaphysis of the pterygoid will be visualized deep and caudally. The pterygoids are pneumatized, enabling the potential for fracture repair methods using transcortical suture, wire, or hexalite shuttle pins and bone cement.[14] Closure of the surgical site is accomplished in two layers: the lateral wall of the infraorbital sinus and the skin.[80]

Beak avulsion

Traumatic beak avulsion is somewhat common in psittacine birds, toucans, and waterfowl. In psittacine birds, the injury is often a result of a bite from another psittacine bird. Avulsion injuries may be seen in the maxillary or the mandibular beaks

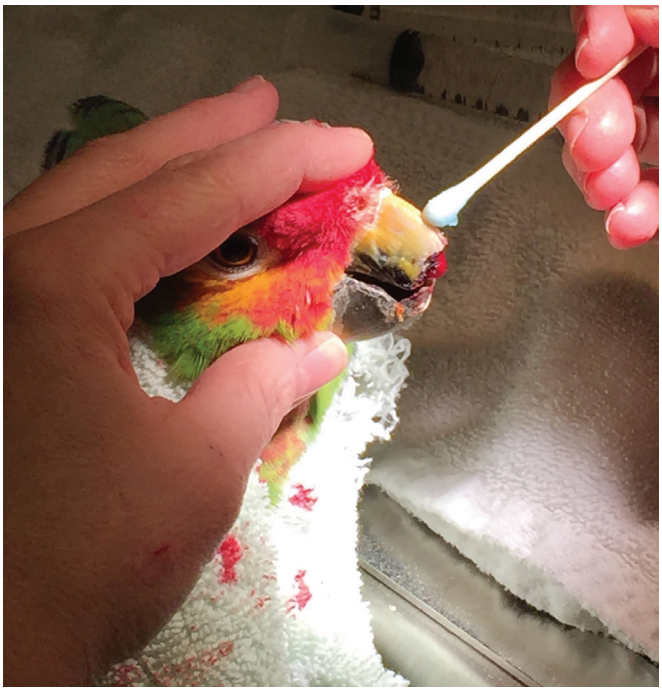


FIGURE 31.39 Traumatic avulsion of the distal maxillary beak in a red-lored Amazon parrot *(Amazona autumnalis)*. The fracture is at the level of the occlusal ledge of the rhinotheca, lending potential risk for permanent malocclusion. Rhinothecal keratin, dermis, and premaxillary bone are all missing. If the amputated portion of the beak is still available, it could be used as a part of an autologous prosthesis, to facilitate enough proximal beak growth to allow occlusion of the rostral gnathothecal tomium with an occlusal ledge and effective ability to chew and crack food items. Regardless, many parrot species can learn to eat on their own with these types of deformities. (Photo courtesy Laura Wade.)

(Figure 31.39). Surgical amputation of beak tissues may be indicated for aggressive local diseases such as neoplasia and some localized infections.[41] Ducks and geese are occasionally subjected to bites to the beak from snapping turtles or other predators. Most parrots can adapt and survive traumatic beak avulsion with appropriate wound care, supportive care, and analgesic therapy and do not require surgical intervention.[38,39,68,107] Some birds may require assisted feeding or placement of an esophagostomy tube during the initial recovery period. In one case series, 9 out of 10 psittacine birds with amputation of the beak survived, and all surviving birds were able to self-feed within 20 days, some much sooner.[99] Psittacine birds that lose most or all of the maxillary beak often lose the ability to crack seeds and nuts and other hard food items, and must be fed an altered, soft diet. Overgrowth of the rostral gnathothecal tomium occurs with amputation of the maxillary beak, typically when the injury is proximal to the occlusal ledge of the rhinotheca. Affected birds may require periodic gnathothecal restorative procedures due to inadequate keratin wear from the opposing beak.

Beak prosthetics

Avulsion of the maxillary beak in waterfowl can result in difficulty prehending food and exposure of the tongue to further desiccation and injury. These injuries of the maxillary beak can also result in difficulty with self-feeding as in other species. Loss of portions of the mandibular beak can also result in difficulty self-feeding and the performance of other maintenance behaviors.[26,97,99,101,112,113] These birds may benefit from surgical placement of a beak prosthesis. Unlike an orthosis, which functions to extend or add to an anatomic part to correct alignment or provide support, a prosthesis is an artificial replacement of a part of the body (Figure 31.40). An exoprosthesis is worn outside of the body, and endoprostheses are fully implanted and surrounded by body tissue. With most beak restorative prosthetics, the bulk of the prosthetic material is worn outside of the body, but there are often implants required to secure these in place and maintain stability. The cosmetic appearance of the bird should never be the only factor in deciding whether or not to place a beak prosthesis, particularly in psittacine birds with maxillary beak avulsion, where there is a high percentage that are capable of learning how to eat on their own in time.[92] Moreover, the strength of the jaw musculature of most psittacine birds, combined with their varied coupled kinetic directional forces applied, can more easily lead to failure of maxillary beak prosthetics. There are several descriptions of prosthetic beak placement in birds.[5,6,97] All prostheses implanted into bone will eventually fail due to cycling, resulting in bone resorption and/or implant failure. All prostheses applied to the rhamphothecal surfaces of the beak will eventually be lost due to the natural turnover of the keratin surface. If there is bone resorption, subsequent attempts to apply a prosthesis will generally fail more quickly because there is less bone stock. At the present time, there is no known permanent beak prosthesis applicable to birds.[107] Regardless, for a variable degree of time, prosthetic beak applications can return form and function to many birds and may have relevant application in specific cases.

Autologous prostheses are those that are derived from the same individual when there has been recovery of the severed beak remnant (Figure 31.41). Homologous or allogeneic prostheses originate from another individual of the same species (cadaver). Heterologous or xenogenic prostheses are derived from the cadaver of an individual of a different species. Synthetic prostheses are made with inorganic material of a variety of types (Table 31.3). 3D computer-generated prosthetics may be designed, constructed, and utilized. These can then be secured with the use of IM pins, screws, Kirschner wires, or cerclage wire secured to the trabecular bony structures of the remaining beak. For many years it was considered that the ideal beak prosthetic is one that remains fixed to its abutment for the entire remaining life of the bird. More current thought has changed, with the realization that a goal of creating a permanent, life-long prosthetic is not practical or feasible. Instead, efforts to preserve the potential for periodic maintenance and reattachment of the prostheses are more prudent. There are no studies on the long-term growth of beaks with prostheses, including studies of the rate of exchange of keratin layers. Temporary prostheses may be used if needed until effective wound healing occurs with some injuries.[42] The best results for temporary, shorter-duration prosthesis are achieved using devices applied to the surface keratin, as they do not affect the remaining tissues and allow for easier replacement and maintenance.[26,112,113] Prosthetic beaks must be monitored closely for complications such as failure and infection, and repaired and managed accordingly (Figure 31.42).[112,113]

For the use of autogenous, homologous, and heterologous prostheses (Figure 31.43), the severed beak segment should

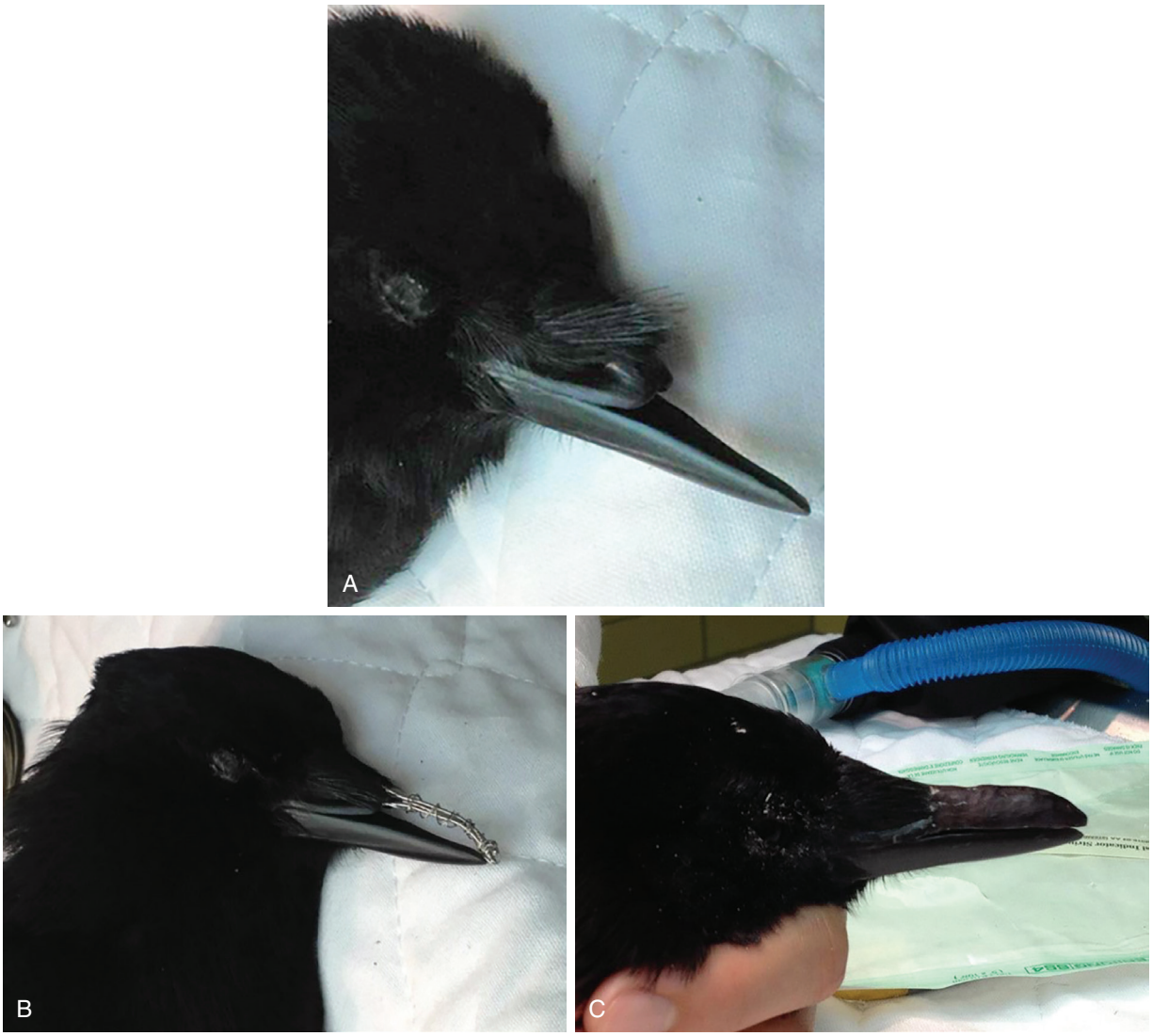


FIGURE 31.40 Construction of a synthetic prosthetic maxillary beak in an American crow *(Corvus brachyrhynchos).* (A) The maxillary beak has been amputated distal to the nares bilaterally. (B) Threaded pins are placed in the nasal, lateral, and maxillary processes of the premaxilla and maxilla, and fine wire is used to stabilize the structure. (C) Immediate postoperative image of the constructed prosthesis. This prosthetic device stayed in place for 12 months. (Photos courtesy Greg Burkett.)

be collected and stored aseptically. Potential significant infectious agents should be ruled out.[112] An aseptic medium capable of maintaining the biological properties of the beak should be used until the date of the surgical implantation procedure. Ninety-eight percent glycerin is a more common and economically accessible method for storage at room temperature; however, beak fragments may also be sterilized using gas sterilization or autoclave techniques. Glycerin-stored tissues should be kept submerged for at least 30 days before use and must be hydrated with a physiological solution prior to use. It is feasible to maintain a bank of stored glycerin-preserved beaks, for potential future use.[112]

An impression of the beak fragment to be used for construction of a synthetic prosthetic can be made in alginate or silicone, and a model can then be generated using gypsum or resin. Similar impressions may be obtained from the avulsed stump of the severed beak in the patient, particularly when synthetic prostheses are to be employed. These models then can be manipulated and tested for optimal fit and function prior to the actual surgical procedure.[4,26] The overall procedural processes for utilizing impression-generated models are the same as those used for making metal dental crowns in mammals (Figure 31.44). The models generated can be used for fabrication of acrylic, resin, or metallic prosthetics. The use of metal prostheses may be indicated in species that can support their relatively greater weights. Waterfowl and psittacine species are common candidates in this regard for potential metallic prosthetic use. Metal alloys utilized are the same metal alloys used in mammalian dental crowns: nickel-chromium, chromium-cobalt, or copper-aluminum.

The use of 3D computer technology to produce prosthetic beaks involves four processes: (1) obtaining the 3D digital data file; (2) digital modeling of the prosthesis; (3) 3D prosthesis printing; and (4) surgical planning and fixation of the prosthetic. The ability to produce patient-specific parts directly from scan data is an obvious and cost-effective benefit,



FIGURE 31.41 Photo of a severed beak tip, including keratin as well as bony structures, which could be aseptically prepared, stored, and utilized as an autologous, homologous, or heterologous prosthesis. (Photo courtesy Laura Wade.)



FIGURE 31.42 Synthetic prosthetic mandible in a Rhode Island red rooster *(Gallus gallus)*. Note that the length of the maxillary beak has been shortened as well, and the mandibular prosthesis is adjusted to match it, allowing a return of normal prehension for the bird. (Photo courtesy Greg Burkett.)

as compared with more traditional conventional manufacturing techniques. These tailored parts are made possible through software that converts the scan data, utilizing CT, MRI, laser scanning, and photogrammetry. CT is considered the imaging method of choice for bone imaging for most beak prosthetic construction. MRI uses an analog method to obtain a 3D digital dataset as well. Laser scanning surveys the surface of an object and captures data represented as a collection of points (a point cloud), which is then used to generate a 3D surface. This enables parts that would be very difficult to precisely measure and 3D models to be digitized and accurately reproduced. Photogrammetry is the science of making measurements from photographs, using digital cameras or smartphones. Several photographs are required around the bird at a 360-degree angle to obtain an accurate digital model. The conversion of the photographs into a 3D digital surface is obtained through free or paid software. Unlike CT or MRI scanning, laser scanning and photogrammetry do not show the internal features of the object being scanned. After obtaining the digital 3D model, it is possible to perform the virtual surgical planning. Many software programs allow the virtual construction of real-life scale portions or the entire missing beak for the purpose of making a prosthesis. There are currently low-cost 3D printers for home use, which can be utilized for this purpose. Also, the market offers low-cost polymers for 3D printing. The most commonly used materials are polylactic acid (PLA) and acrylonitrile butadiene styrene (ABS), but the medical industry also has the ability to employ 3D printing in titanium. Based on CT or MRI data, surgeons can devise a custom surgical guide for the surgical procedure (Figure 31.45). This surgical guide is then 3D printed and used for reference before and during the surgical procedure. A fitted surgical guide can be used to indicate where the surgeon should drill or cut, and then it can be removed when the actual prosthesis is to be applied.

Surgical fixation of the prostheses in a manner that will last as long as possible can be a challenge. There is no ideal model for fixation of all prosthetics to beaks, and the details of fixation are ideally specifically chosen for the patient at hand. A functional understanding of the biomechanics of the skull and the forces that act during feeding and mastication is essential for the preparation and positioning of prostheses. Several factors contribute to the common problems encountered, namely the prosthetic loosening or falling off in a comparatively short duration of time. These include the innate characteristics of the bony structures involved (thin and porous), the absence of specific biomechanical studies of force transfer in most beaks, the lack of bone orthopedic materials that are specifically applicable to beaks, a lack of biocompatibility of the surgical materials with the bone and keratin surface, and the experience and skill set of the surgeon. Attachment of the prosthesis is accomplished using orthopedic techniques, with intradentin pins, cerclage wire/silk, and screws covered with an adhesive system (resins or composites). Fixators that transversely cross the beak and are secured laterally may be preferable. In some birds, the use of intravenous catheters can allow placement of nylon as an aid in fixation. Prostheses secured with implants such as screws or threaded pins will generally last longer. Orthopedic pins or screws can be used to help form a base structure for fixation of the prosthesis. These implants (screws, threaded pins) may be predisposed to infection and will in time result in bone reabsorption and loosen at their attachments.[4,107] The potential for growth of microorganisms at the prosthesis and beak interface is not known. As a result, periodic evaluations are rational. The maintenance time recommended for evaluation of the prosthesis can vary from every 6 months to more than a year. Since a detailed evaluation of the prosthesis and its abutments often necessitates general anesthesia and removal of the prosthesis for inspection, the interval between these inspections should be kept as far apart as possible.

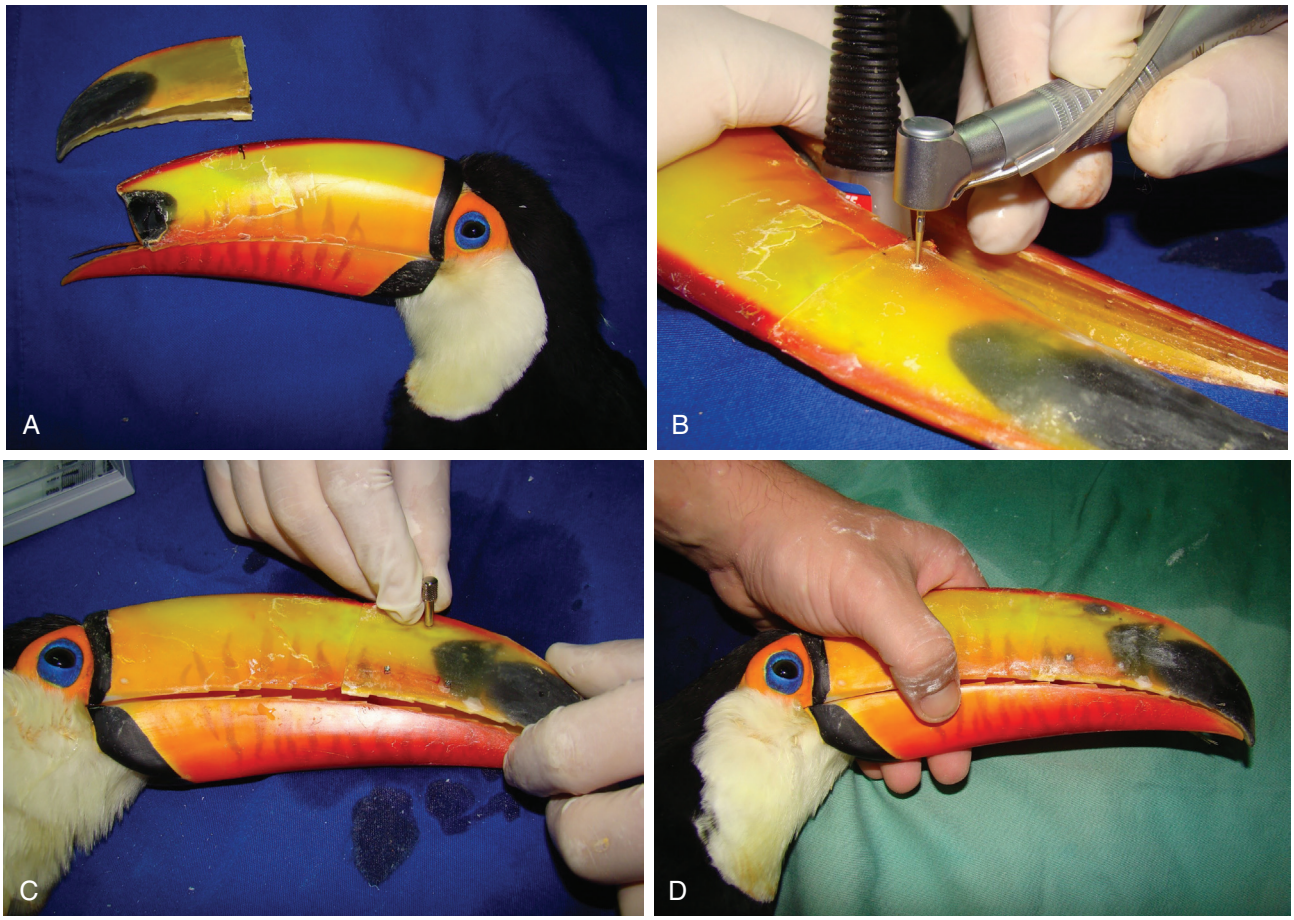


FIGURE 31.43 Construction and fixation of a homologous maxillary beak prosthesis in a toco toucan *(Ramphastos toco)*. (A) Cadaver distal maxillary beak fragment of the same species, and patient with visible injury. (B) The prosthesis is fitted into position, and pilot holes are drilled. (C) Placement of dental screws. (D) Completed prosthetic fixation, after covering screws with acrylic.

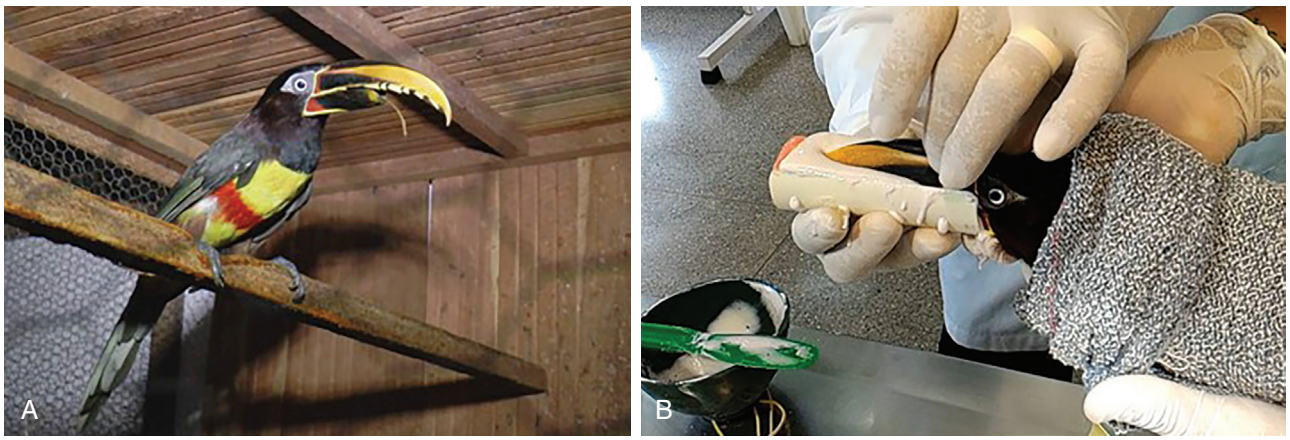
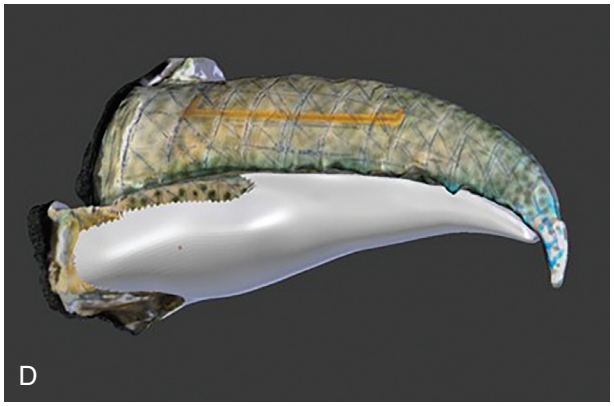
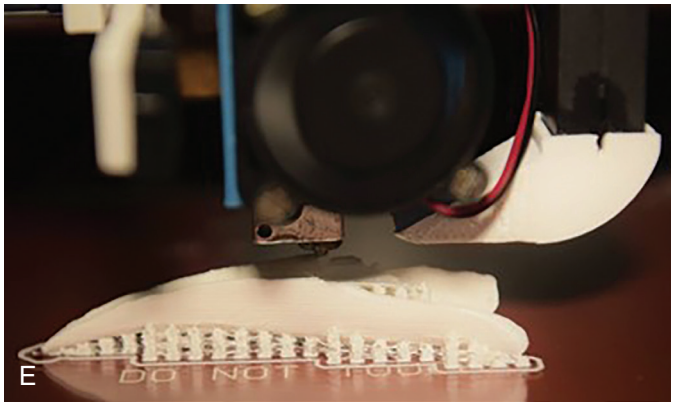


FIGURE 31.44 Mandibular synthetic prosthesis application in a chestnut-eared aracari *(Pteroglossus castanotis)*. (A) The distal half of the mandibular beak is fractured and missing. (B) Obtaining an impression of the surfaces of the maxillary beak and the mandibular beak with alginate.

Continued







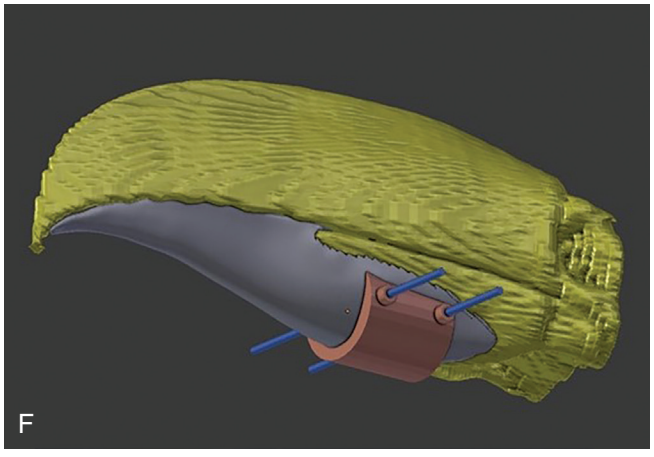




FIGURE 31.44, cont'd (C) The impression obtained. (D) Laser scan modeling of the impression and design of the prosthetic. (E) Three-dimensional (3D) printing of the designed prosthetic. (F) 3D generated surgical guide for placement of fixators. (G) Postoperative image, following fixation of the prosthesis.



FIGURE 31.45 Application of a titanium 3D printed maxillary beak prosthesis in a blue-and-gold macaw *(Ara arauana)*. (A) Maxillary beak amputated stump. (B) Patient following application of prosthetic.

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CHAPTER 32

Feathers and Conditions of the Plumage

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ANATOMY

The outer surface of a bird is covered with feathers that form a lightweight, durable, and flexible protective barrier against mechanical impact, solar and terrestrial radiation, and water. This is important because avian skin is generally less substantial and therefore lighter but less protective than mammalian skin. Feathers are responsible for the aerodynamic shape of the bird's body as well as forming the aerofoiled shape of the wings and tail of flying birds. Feathers are colored to provide crypsis, and their form and color are a key feature of the bird's breeding strategies. Because of associated nerve supply, feathers are important tactile organs. There are two books that cover the anatomy of feathers and skin in a comprehensive and useful manner; neither are still in print but are extremely important texts.[1,2] Two in-print books describe feather anatomy[3,4] and feather color and molt.[4]

The feather is a complicated epidermal structure: it is a tough, keratinized, cellular derivative of the epidermis and is formed in a follicle that penetrates deep into the dermis. Once formed, the feather is full of airspaces as well as pigment granules and oils. The keratin of feathers is mostly β-keratin, which is only found in birds and reptiles.[5,6] Birds and reptiles also have α-keratin, but this is a softer protein structure. Feather keratins contain large amounts of the amino acids, but despite the differences in feathers between species, their compositions are very similar.[7,8] In reptiles and birds, hard-cornified β-keratins form filamentous structures made of polypeptide chains folded into β-sheets. Two β-sheets are stacked on top of each other, forming a dimer, which twists to form a helical structure.[9] The filamentous structure of reptilian and avian hard keratins is quite different from that of mammals. The β-keratin dimers are formed into sheets that are held together with a carbohydrate matrix, making a very strong but flexible structure that does not require any other supporting structure compared to, for example, horns in mammals, which are composed of a softer α-keratin laid down over bone.

FEATHER TYPES

While there are many types of feathers, they all have some similar key features (Figure 32.1). Contour feathers form most of the external plumage. The various groups of contour feathers are of great importance to ornithologists. These groups of feathers and their colors are used to describe differences in species, subspecies, and hybrids, as well as the bird's sex and age. Because of their importance, the feather groups are all named. It is often possible to predict hunting strategies (and make taxonomic judgments) from feather shape in the absence of the live bird.

Contour feathers can be divided into three main groups: primary, secondary, and—in some birds—tertiary remiges (wing feather—*remex*, Latin "rower"; Figure 32.2); rectrices (tail feather—*rectrix*, Latin "one who directs"); and body feathers. Contour feathers that cover the base of the remiges and rectrices are called tectrices or coverts (covert—*tectrix*, Latin "cover"). Contour feathers have relatively flat, close-knit (pennaceous) vanes, although the basal part of the vanes can be variably plumaceous (Figure 32.3). Each feather has a long central stalk *(scapus)*. The initial part of the stalk, seated in a tightly fitting follicle, is the calamus. The tip of the calamus is embedded in the follicle and has an opening, the proximal umbilicus that is attached to the follicle's dermal papilla. Feather follicles have sensory nerve endings that appear to be stimulated by movement of the rachis, which is the remainder part of the stalk that is more prominent on the ventral aspect of the feather. On the ventral aspect of the rachis a groove runs the whole length from the (distal) tip of the feather to the junction between the calamus and the rachis, where there is a small hole, the distal umbilicus that leads inside the calamus. The umbilici and associated groove were formed around the blood supply to the pulp core of the growing feather. The area around the distal umbilicus may have a small tuft of barbs or even a complete plumaceous feather-like structure, the afterfeather (*hypopenna*; Figure 32.4). The rachis forms the support for a series of parallel stiff branches arranged at about 45 degrees to the rachis; these are the barbs (Figure 32.5). Contour feather barbs consist of a ramus that has further branches or barbules on either side called proximal and distal barbules. The rami are spaced along the rachis to allow distal barbules from one ramus to hold onto the adjacent proximal barbules with hamuli or hooklets. In a clean, normal, healthy feather, this system forms a strong interlocked sheet, the vane (Figure 32.6). The vanes of the wing feathers are asymmetric, the leading edge being narrower. This helps to produce an aerodynamic shape (Figure 32.7).

Remiges (wing feathers) are grouped into primary and secondary feathers. The carpus is the point of division: carpus to tip of the hand is occupied by the primaries, carpus to the elbow are

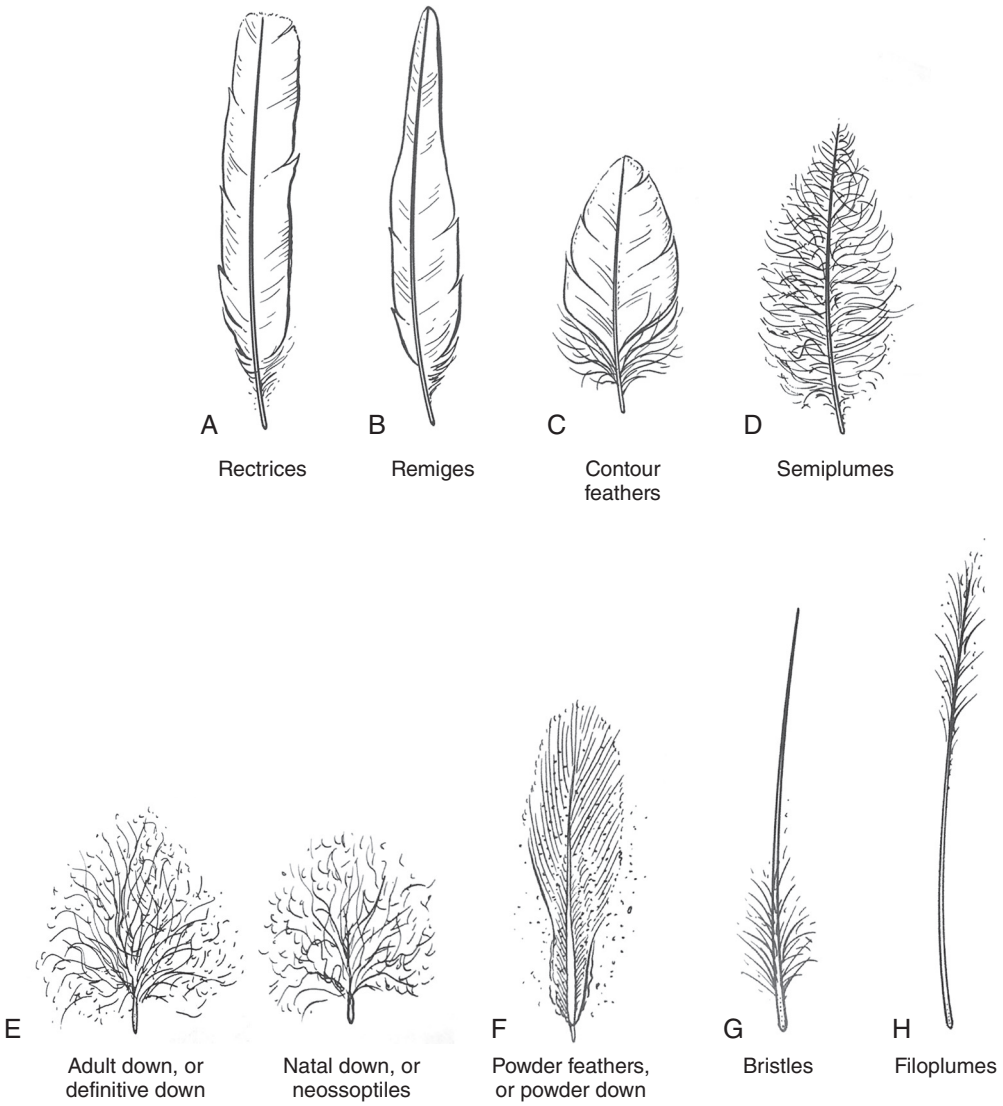


FIGURE 32.1 The main feather types that can be distinguished in birds include (A) tail feathers or rectrices, which have a symmetrically shaped vane; (B) the primary flight or wing feathers or remiges that have an asymmetrically shaped vane; (C) contour or covert feathers; (D) semiplumes; (E) down feathers; (F)powder down feathers, which are a specialized type of down found in few groups of birds; (G) bristle feathers; and (H) filoplumes.

secondaries. Some birds, such as vultures, have tertiary remiges that arise from the humerus. The primary feathers are closely and firmly attached to the periosteum of the bones of the hand (metacarpals plus major and minor digits). The secondary feathers are more loosely attached to the ulna, although in some flighted bird species (e.g., turkey vultures [*Cathartes* spp.], magnificent frigatebird [*Fregata magnificens*], pink-backed pelican [*Pelecanus rufescens*], rock pigeon [*Columba livia*]), there are bony prominences (quill knobs) on the ulna where the follicles arise.[11–13] In many groups of birds (i.e., Anseriformes, Caprimulgiformes, Charadriiformes, Ciconiiformes, Falconiformes, Gaviiformes, Gruiformes, Pelecaniformes, Phoenicopteriformes, Podicipediformes, Procellariiformes, Psittaciformes, Sphenisciformes, Strigiformes, Suliformes) there is a space *(diastema)* between the fourth and sixth secondary feathers on the wing that seemingly corresponds to a missing fifth feather (*diastataxy*; Figure 32.8), while this gap is absent in most Galliformes, Passeriformes, Coliiformes, Coraciiformes, Cuculiformes, Piciformes, and Trogoniformes *(eutaxy)*. As the remiges are attached to the dorsal aspect of the wing bones, it is impossible for the bird to move the flight feathers on the wing without moving the bones of the wing. The feather follicles are linked together subcutaneously by a complicated set of elastic ligaments and various aponeuroses, which ensures that the remiges are correctly spaced and oriented when the wing is flexed, extended, and in use.

Conversely the rectrices (tail feathers) are very mobile either by the bird moving its caudal vertebrae or by a set of intrinsic muscles that allow the tail to be spread as a fan. The rectrices steer the bird during flight and can also be used for communication/display. The caudal vertebrae end in a special bone, the pygostyle, which supports the feathers, and there is a set of complicated muscles that are used to support and move the tail feathers. In the peacock *(Pavo cristatus)*, the massive and

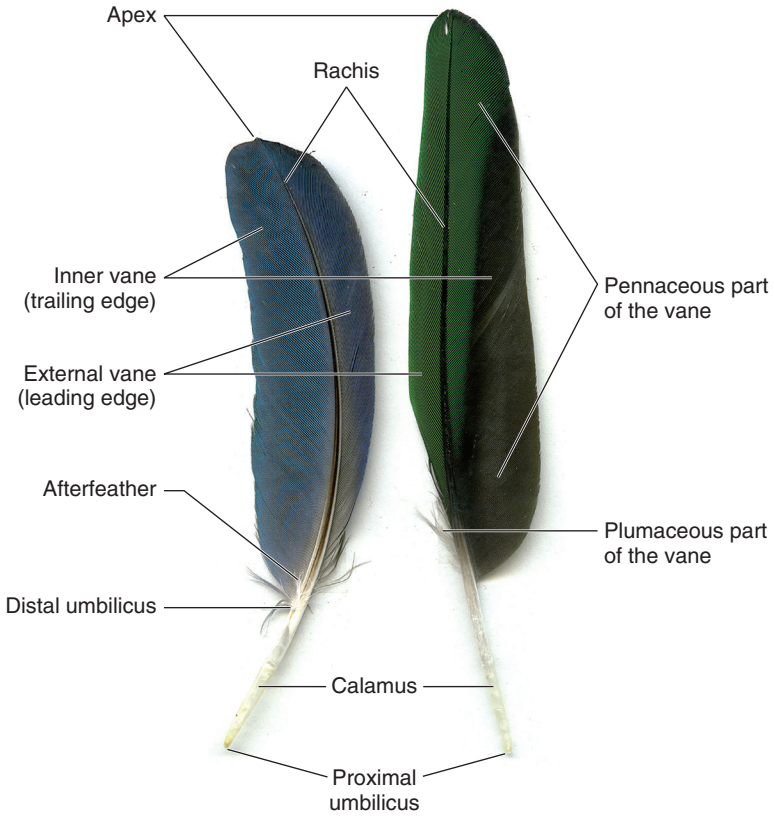


FIGURE 32.2 Remiges from bronze-winged parrot, *Pionus chalcopterus* (ventral secondary feather, blue), and scaly-headed parrot, *Pionus maximiliani* (dorsal primary feather, green). Irrespective of the feather's position on the body, the part of the feather that faces the body is the ventral surface; the dorsal surface faces outwards. In most birds, the exposed parts of the feather are colored whereas the unexposed parts are not. The primary feather in this illustration shows green barbs on the leading edge up to where it is covered by the overlapping adjacent (green) feather when the wing is extended. The ventral aspect of this feather is colored on the trailing edge where it is no longer covered by the adjacent feather, this can be seen where the edge of the feather is curled on the trailing edge. Secondary feathers are colored more extensively. (Photo courtesy Nigel Harcourt-Brown.)

gaudy tail fan is made from dorsal tectrices (coverts), while the rectrices are brown, relatively short, and support the fan.[14]

Down feathers have a short rachis and long, soft barbs (Figure 32.9). They are often the first feathers on a newly hatched chick. The down feather either occupies the same follicle as the contour feather, in which case it breaks away from the tip of the contour feather as the bird fledges, but mostly down feathers arise from a separate follicle. In adult birds, down feathers are found under the contour feathers, where they form an insulating undercoat. Their distribution varies with families, for instance covering the whole body in ducks (Anatidae) and penguins (Sphenisciformes), showing an uneven distribution in gulls (Laridae) and owls (Strigiformes), or being completely absent in hummingbirds (Trochilidae) and passerines (Passeriformes).[15]

Semiplumes are characterized by a wholly fluffy (plumaceous) vane and a long rachis, which is always longer than the longest barb (Figure 32.10). They are found along the margins of the contour feather tracts or sometimes singly in the feathered areas. They are insulative and in some aquatic species aid buoyancy. The spectrum of changes between down, semiplumes, and contour feathers makes it impossible to classify intermediate forms.

Powder down feathers are found in pigeons (Columbiformes), parrots (Psittaciformes), as well as birds from several other orders (Figure 32.11).[16–19] While they can have the basic structure of contour feathers or semiplumes, they usually resemble down feathers and are modified to grow slowly. As part of their normal maturation, these feathers shed particles of keratin as a fine waxy dust that can coat the whole bird, including its beak; this is very noticeable in white cockatoos *(Cacatua alba)*. Powder down feathers are usually found in dense patches, as in cockatoos on the body under the wing, or are scattered over the body as, for example, is seen in *Pionus* spp. The feathers are white to gray and are replaced more regularly than normal feathers. They also grow slowly so that there is a continued supply of shedding particles. Most birds produce some powder from normal contour and down feathers, but this is minimal compared to powder down feathers. The distribution of powder down feathers was previously used as a taxonomic classification feature, but this was not found to be reliable.



FIGURE 32.3 Ventral view of a contour feather from a Eurasian griffon vulture *(Gyps fulvus)* showing the well-developed plumaceous portion; the hypopenna is also well developed (compare with Figures 32.1 and 32.4). (Photo courtesy Nigel Harcourt-Brown.)

Hypopennae, also known as afterfeathers, are usually small and attached to the ventral aspect of the contour feather (Figure 32.4). They are not usually associated with remiges and rectrices. In emus *(Dromaius novaehollandiae)* and cassowaries (*Casuarius* spp.), the hypopenna is often as large as the main feather forming a double body feather.[20]

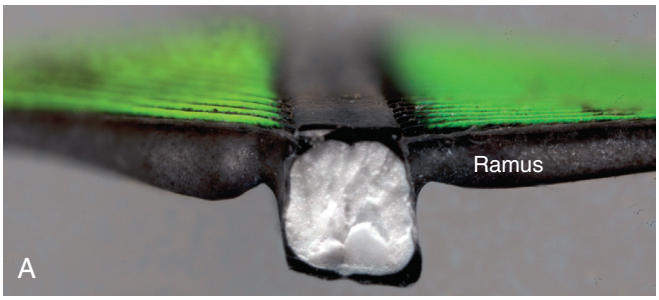
Bristle feathers *(seta)* are the short spiky feathers that only have barbs at the base, if any are present. They are found variably around the eye and loral region, also as eyelashes (Figure 32.12). They are protective and have lots of sensory nerve ends attached to their follicles to aid this function.[17,21–23]

Filoplumes are present in nearly all birds (excluding ratites), although they are not usually apparent (unless the bird is plucked; Figure 32.13). When the filoplume first grows, the long rachis is often surrounded by barbs, but these usually break off, leaving a long spike with a tassel of barbs at its tip. Filoplumes are usually associated with a contour feather and are assumed to serve as sensory detectors as they are closely associated with sensory nerve ends and Herbst corpuscles. It is thought that these feathers probably assess the strains and movement of the contour feathers, which is considered vital for flight.[24] Each flight feather has a number (up to 10) of closely associated filoplumes. A few birds (e.g., bulbuls, Pycnonotidae) have long modified filoplumes in their neck regions that extend beyond the contour feathers, which are thought to serve a decorative purpose only.[25]

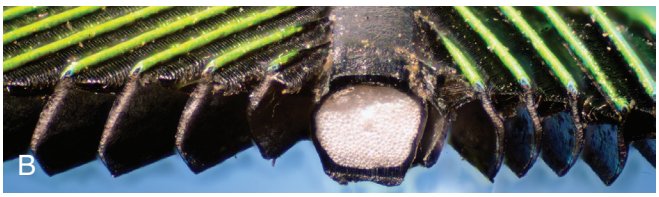
Feathers are important tactile organs. Not only the filoplumes and bristle feathers serve a sensory function, but the feather follicle wall is supplied with numerous sensory nerve endings as well.



FIGURE 32.4 (A) Ventral view of a contour feather from a red grouse *(Lagopus lagopus scotica)* showing a well-developed hypopenna. The hypopenna has a central hyporachis supporting a plumaceous hypovexillum. (B) Lateral view of the same feather showing the relationship between the contour feather and its hypopenna. (Photo courtesy Nigel Harcourt-Brown.)



Feather barbs on the trailing edge | Rachis | Feather barbs on the leading edge



Feather barbs on the trailing edge | Rachis | Feather barbs on the leading edge

FIGURE 32.5 (A) Secondary remex from a scaly-headed parrot *(Pionus maximiliani)*, viewed toward the apex. The rachis has a hard melanin-rich cortex (mainly formed by β-keratin). The medulla is white and full of pith that adds strength to the feather with little extra weight. The pith is a firm spongy tissue formed from the keratinized walls of what were large polygonal epithelial cells. Because of the air inside these cells the rachis is opaque in contrast to the calamus. The barb consists of a ramus which has proximal and distal barbules along its length. (B) Primary remex from the same species, viewed from the apex. The barbs on the external vane *(vexillum externum)* are closer together and at a narrower angle to the rachis than those on the internal vane *(vexillum internum)*. This arrangement makes the leading edge stronger at the expense of slightly extra weight. The distal barbules overlie the proximal barbules. In these and the next illustrations from parrots, the colors of the remiges and rectrices are produced by the dorsal and ventral parts of the barbs. The ramus is usually not colored. The center is pith and the outer keratin layer, being melanin rich, is usually black to brown. Contour feathers in parrots usually have the rachis and the whole barb colored by pigment (psittacofulvin).[10] (Photo courtesy Nigel Harcourt-Brown.)



Pionus chalcopterus



Pionus maximiliani

FIGURE 32.6 (A) The barbs are attached to the rachis and are arranged regularly; they are more prominent on the ventral surface of the feather than the dorsal, as is the rachis. The barbs maintain their position by hooklets on each distal barbule which overlap and latch onto the flange of the proximal barbule (i.e., the barbule closest to the calamus). All these features form the smoothest, strongest surface to each feather. (B) The barbules can become separated while flying or foraging and the birds use their beak while preening to reunite the barbules. Feathers that have grown with adequate nutrition and are covered with normal sebum return to their perfect form very easily. (Photo courtesy Nigel Harcourt-Brown.)

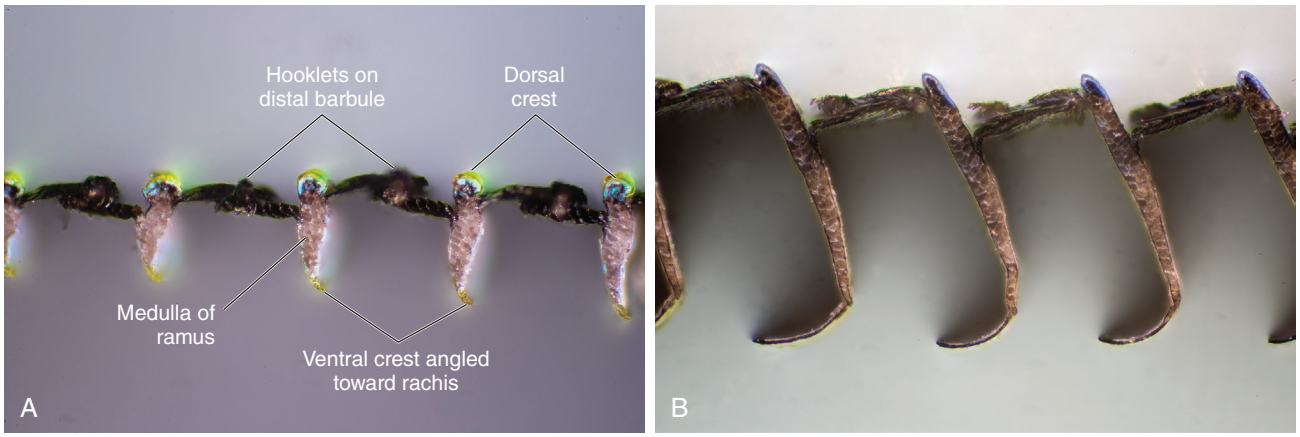


FIGURE 32.7 These illustrations are sections through (A) a green secondary remex and (B) a blue primary remex. In all the remiges and rectrices the shape of the barbs differs, seemingly to share the load on the flying wing and keep an aerodynamic shape. The hooklets on the barbules can be seen to be inserted into the flange of the proximal barbules. The secondary remex barbs are short with color produced by the dorsal and ventral extremities. Like the rachis, the barbs are much less prominent dorsally, keeping the surface as smooth as possible. The primary remex has a much deeper ramus and the ventral crest is bent over. In some birds this is extreme e.g., mute swan *(Cygnus olor)* where it is known as the tegmen. In the illustrated part of this primary remix the dorsal crest is colored; the ventral crest is not. (Photo courtesy Nigel Harcourt-Brown.)

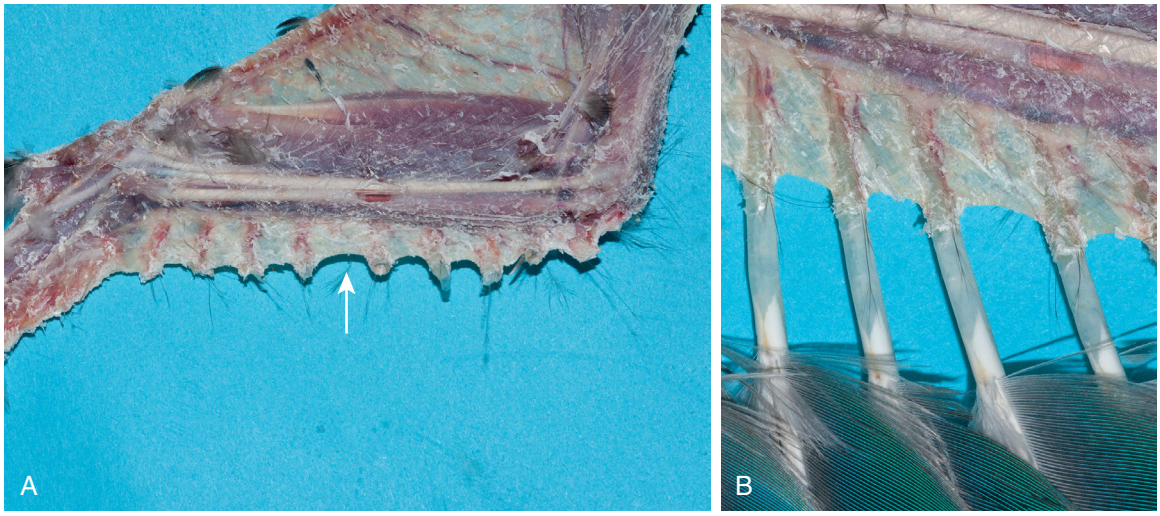


FIGURE 32.8 (A) Two features in the plucked wing of this bronze-winged parrot *(Pionus chalcopterus)* are the filoplumes associated with many of the empty feather follicles and the obvious diastataxic gap between the fourth and fifth secondary feathers (as indicated by the *arrow*). (B) The secondary feathers of this bronze-winged parrot show filoplumes emerging from very close to the feather follicle of each secondary feather. It is likely that some of the filoplumes were removed when the coverts were plucked. (Photo courtesy Nigel Harcourt-Brown.)

Pterylosis

Pterylosis is the arrangement of contour feathers within tracts *(pterylae)* over the body of the bird. Down feathers also occur with the contour feathers in these tracts. Areas between the pterylae are regarded as featherless tracts *(apteria)* and are either featherless or contain only down feathers and occasionally a semiplume. These apteria can at times be misinterpreted as regions of feather loss, or even skin lacerations, by inexperienced owners and veterinarians. Within the class of Aves, several patterns of pterylosis can be distinguished (Box 32.1).[26] In most birds, except those that do not have apteria (i.e., certain Struthioniformes, Sphenisciformes), there are usually eight pterylae involving about half the body surface (Figure 32.14), though the size of these can vary across species.[28–30] The contour feathers are shaped and arranged so that the bird has a continuous covering of feathers that would

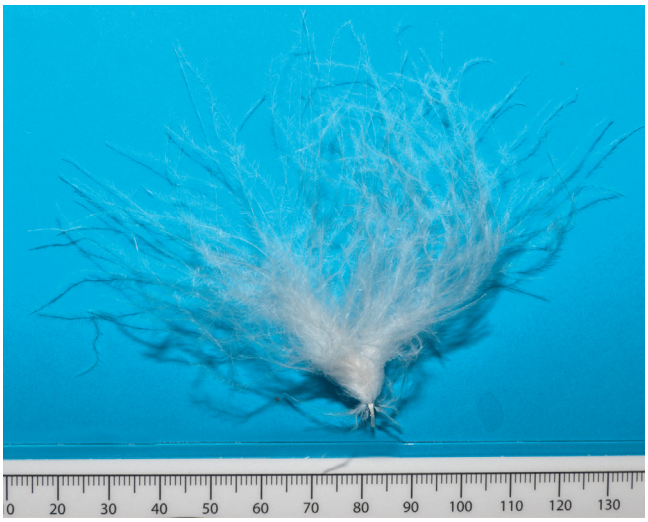


FIGURE 32.9 A down feathers has completely fluffy (plumaceous) barbs with its rachis when present shorter than its longest barbs. These feathers can be very large as from this Rüppell's griffon vulture *(Gyps rueppelli)*. Scale in millimeters. (Photo courtesy Nigel Harcourt-Brown.)



FIGURE 32.10 This ventral view of a semiplume from a Rüppell's griffon vulture *(Gyps rueppelli)* shows a relatively long rachis supporting fluffy pennaceous barbs. (Photo courtesy Nigel Harcourt-Brown.)



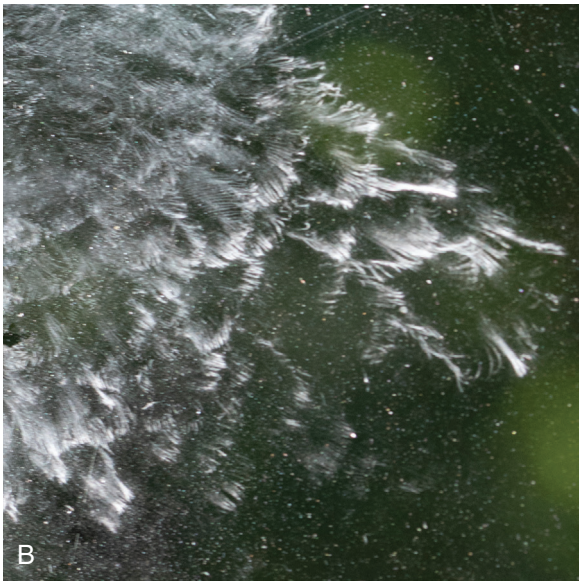


FIGURE 32.11 Powder down is shed from special feathers and covers the whole bird. (A) A window collision by a wood pigeon *(Columba palustris)* gives an obvious outline of the bird. (B) In close-up, the feathers are well defined. The powder has sebum mixed with it so the imprint lasts for some time. (Photo courtesy Nigel Harcourt-Brown.)

need to be parted to reveal the apteria. The area of the body under the wing and the adjacent wall of the abdomen often have reduced numbers of feathers. The skin of the apterium is thinner than the skin of the pteryla.

Molt

Full-grown feathers are inert structures and one of their roles is to absorb damage from external sources. They are not being continuously renewed, unlike beaks and claws, but become worn by life as well as affected by pathogens, such as ectoparasites, bacteria, or fungi. Sometimes they are lost traumatically and must be replaced as soon as possible. The parts of feathers that contain melanin are more resistant to wear, as well as damage by some bacteria. This might be because the area that contains the melanin is thicker. Melanin also protects against ultraviolet (UV) damage.

The plumage is made almost completely of dried protein, and there is a significant energetic cost to replacing it. Most passerines' feathers are 18% to 39% of total lean dry body mass (or approximately 6% of normal body mass) and the energetic cost has been calculated to be around 12% to 36% of resting energy output.[31] Small birds, for example, barn



FIGURE 32.12 Bristle feathers can be seen on the eyelids and periorbital region of this newly fledged cinereous vulture *(Aegypius monachus)*. The eyelid feather is just a stiff rachis; the periorbital feathers have small barbs at their base. There are some (5) natal down feathers still attached to the juvenile feathers on the top of the bird's head *(arrows)*. They will wear off in the next 10 days. (Photo courtesy Nigel Harcourt-Brown.)

swallows *(Hirundo rustica)*, have 1500 feathers but large birds have more, for example, mute swans *(Cygnus olor)* have 25,000. Small birds usually change all their plumage annually and do this outside the breeding season and at a time when they are not migrating—both high–energy cost activities.[32,33] The process of molt in birds requires the investment of substantial amounts of energy and nutrients in replacement feather production. Flight performance may be reduced with attendant risk to the individual, but it may affect the next generation too. To demonstrate this, researchers cut off the last two primaries on both wings of some pied flycatchers (*Ficedula hypoleuca*, a long-distance migrant) to reduce flight performance.[34] Just before fledging of the young, manipulated males and females, as well as the fledglings, had a lower body condition score than the group of control birds. Next year, less of the flight-impaired males returned (the females were unaffected), and the number of nestlings that returned was significantly lower for the group with flight-impaired parents. The reproductive performance in the groups of returning nestlings and old females was unaffected, as was the feather quality of the returning birds.

Birds have developed strategies to minimize the energy costs while molting. Molting birds tend to reduce their level of activity and in some instances, this can completely offset the energy costs of the molt. This reduction in activity should be considered in captive falcons and other birds as it could increase the potential for bumblefoot.[35] Molt tends to occur during a period when there is adequate food and minimal energy output. It is unusual for a bird to molt during the breeding period or migration. Molting large feathers is usually done in regulated stages.[33] Fledgling birds do not leave the safety and constraint of their nest, or try to fly, until the feathers (and bones) have almost completely matured. Growing feathers are much heavier and will cause deformation of the wing if used before they have finished growing.[36,37]

Large birds tend to change only a proportion of their plumage annually. In the wild, and captivity, large vultures and eagles take around 4 years to change all their remiges and rectrices[38,39]: clinicians must therefore try to protect and preserve them during examinations and medical/surgical procedures.

Anatidae (ducks and swans) have a different molting strategy.[40–44] At the end of the breeding season, they molt their body feathers, and the breeding plumage is replaced by

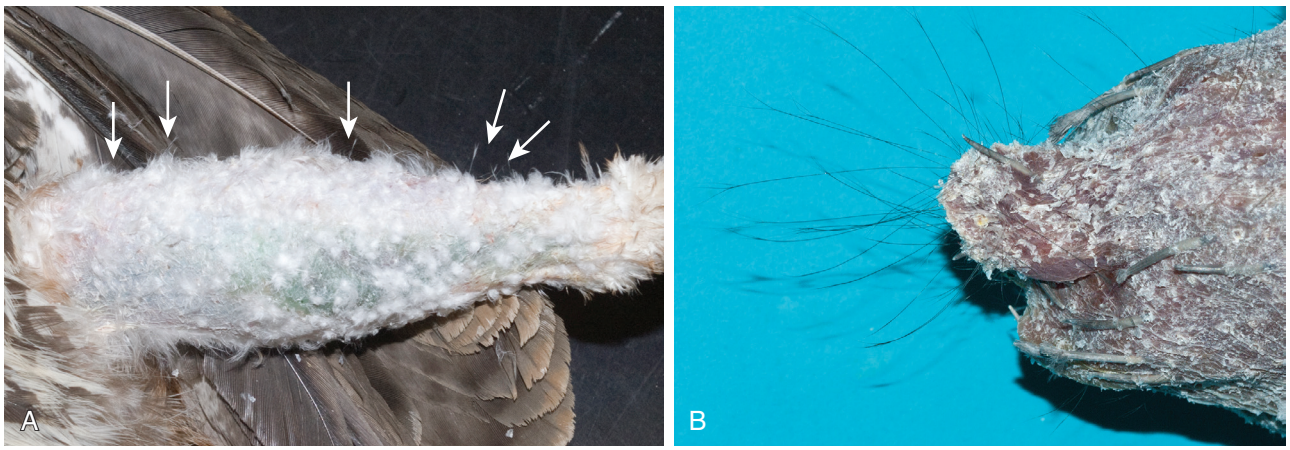


FIGURE 32.13 (A) A Harris hawk *(Parabuteo unicinctus)* had a fracture of the proximal third of the tibiotarsus. Surgical preparation requires feather removal. Plucking all the contour feathers leaves the down and also several filoplumes *(arrows)*. (B) This bronze-winged parrot *(Pionus chalcopterus)* has had all its contour feathers removed. There are many filoplumes left that were associated with the remiges. Birds use their tail to steer during flight but *Pionus* spp. use their tails to signal to each other so presumably feedback from the sensory filoplumes is useful. Note: *Pionus* spp., like *Amazona* spp., have no uropygial (preen) gland. (Photo courtesy Nigel Harcourt-Brown.)

BOX 32.1 PATTERNS OF PTERYLOSIS IN BIRDS[26]

Pattern	Characteristics	Examples
Struthioniform	◆ No apteria in the emu or cassowary; apteria are present in the ostrich and rhea, despite some references claiming otherwise[27] ◆ Uropygial gland is absent ◆ Plumage is soft and lax, intermediate between down and contour feathers	Ostrich, rhea, emu, cassowary
Sphenisciform	◆ No apteria in the adult bird ◆ Uropygial gland is present ◆ Dense plumage	Penguins
Columbiform	◆ Upper and lower cervical tracts are only separate near the shoulder ◆ Deeply forked upper cervical tract, without diverging branches ◆ Broad dorsal tract that is separate from the cervical tract and is without any apterium ◆ Broad, distinct humeral tracts ◆ Small femoral tracts that are mostly separate from the dorsal tract ◆ Broad sternal tracts that are directly continuous with branches of the (quire deeply forked) lower cervical tract and have no side branch ◆ Broad ventral tracts that are directly continuous with the sternal tracts ◆ Narrow ventral apterium that is broadest at the most caudal end	Loons, grebes
Anseriform	◆ Upper and lower cervical tracts are only separate near the shoulder ◆ Forked cervical tracts with somewhat diverging branches ◆ Dorsal tract that is directly continuous with upper cervical, thereby enclosing a variably elongated apterium ◆ Broad, strong humeral tracts that are sometimes connected with the dorsal tract ◆ Large femoral tracts that are united with the dorsal tract ◆ Sternal tracts that usually have a prominent side branch and are directly continuous with the lower cervical tracts ◆ Broad ventral tracts that are directly continuous with sternal tracts ◆ Narrow ventral apterium that is not notably broader toward the caudal end	Petrels, albatrosses, pelicans and other totipalmate birds, auks, geese, ducks, swans
Falconiform	◆ Well-marked, usually narrow upper cervical tract that is strongly forked between the shoulders with diverging branches, and only slightly or not at all connected with the dorsal tract ◆ Variable dorsal tract: can be broad or narrow, forked, or solid ◆ Strong but not very broad humeral tracts ◆ Absent or weak femoral tracts; usually full feathering of the tibia, sometimes with a strongly marked patch that runs along the femur, crossing the head of the tibia ◆ Considerably forked lower cervical tract ◆ Strongly marked sternal tracts ◆ Narrow ventral tracts that are widely separated over the abdominal area, wider on the chest area, and more or less fused with the sternal tracts; tracts may sometimes unite at the furcula only *(Asio)*, touch at the edge of the sternum *(Strix)*, or unite across the full sternal length *(Pandion)*	Vultures, hawks, owls, and other birds of prey, psittacine birds
Pelargiform	◆ Deeply divided upper cervical tract in the neck area, no diverging branches ◆ Solid or deeply forked (rear) dorsal tract that is variably connected with the cervical tract ◆ Well-marked humeral tracts ◆ Long, narrow, not very strong femoral tracts ◆ Lower cervical tract like upper, but with a less deep division and branches that tend to diverge ◆ Comparatively broad sternal tracts that are either continuous with or separate from the lower cervical tract ◆ Broad ventral tracts that are continuous with the sternal tracts, though sometimes separated from those for some distance, while joining near the furcula	Rails, cranes, storks, herons, bustards, flamingos
Charadriiform	◆ Sharply defined upper cervical tract that is not very deeply forked, and with diverging branches ◆ Variably forked dorsal tract (toward rear) that is slightly—if at all—connected with the forks of the upper cervical tract ◆ Narrow humeral tracts ◆ Fairly strong femoral tract that is usually narrow and distinct ◆ Deeply forked lower cervical tract that is continuous with the sternal tracts ◆ Moderately sized yet strong sternal tracts ◆ Narrow or moderately wide ventral tracts that are well separated over the abdominal area and join the sternal tracts across the chest, usually near the furcula	Gulls, terns, plovers, snipes, sandpipers, other waders

BOX 32.1 PATTERNS OF PTERYLOSIS IN BIRDS—cont'd

Pattern	Characteristics	Examples
Galliform	◆ All tracts broad, but usually well-defined ◆ Variably forked upper cervical that can be merged with the dorsal tract ◆ Broad dorsal tract broad, often with a mid-dorsal apterium, that is occasionally connected with the femoral tracts ◆ Broad, strong humeral tracts ◆ Large femoral tracts that sometimes unite with the dorsal tracts ◆ Deeply forked lower cervical tract of which the branches are continuous with the sternal tracts ◆ Strong sternal tracts, which are widest anteriorly, with or without connection to the ventral tracts near the furculum ◆ Narrow ventral tracts that are most narrow anteriorly, unite posteriorly (just cranial to the cloaca), and sparse distancing in between the tracts over the abdominal region	Gallinaceous birds, curassows, tinamous
Columbiform	◆ Wide upper cervical tract that is strongly forked between the shoulders, and often united with the lower cervical tract up to the level of the shoulders ◆ Broad and diffuse dorsal tract that is fairly separable from the cervical tract, but inseparable from the femoral tracts ◆ Broad and strong humeral tracts ◆ Large femoral tracts that are not clearly distinct from the dorsal tract ◆ Slightly or no forked lower cervical tract that is continuous with the sternal tracts ◆ Broad sternal tracts that are completely united with the ventral tracts ◆ Broad ventral tracts that are nonunited at the posterior end and have sparse distancing	Pigeons, sand grouse
Passeriform	◆ Narrow but well-defined upper cervical tract that is continuous with the dorsal tract ◆ More or less widened dorsal tract (often enclosing a prominent apterium) that is not clearly distinguished from the cervical, and generally narrowed near the tail area; frequently the tract is distinctly divided into two parts, i.e., anterior part which is usually forked, and the posterior part which is less commonly forked ◆ Moderate, frequently narrow humeral tracts ◆ Usually weak and small femoral tracts; sometimes these can be absent, while in other species they are more clearly defined ◆ Variably forked lower cervical tract ◆ Usually strong and well-marked sternal tracts ◆ Ventral tracts are rather narrow, widely separated over the abdominal area, and not reaching the cloaca; they are usually not separated from the sternal tracts, but sometimes they can be partly distinct	Cuculiformes, Coraciiformes, Passeriformes

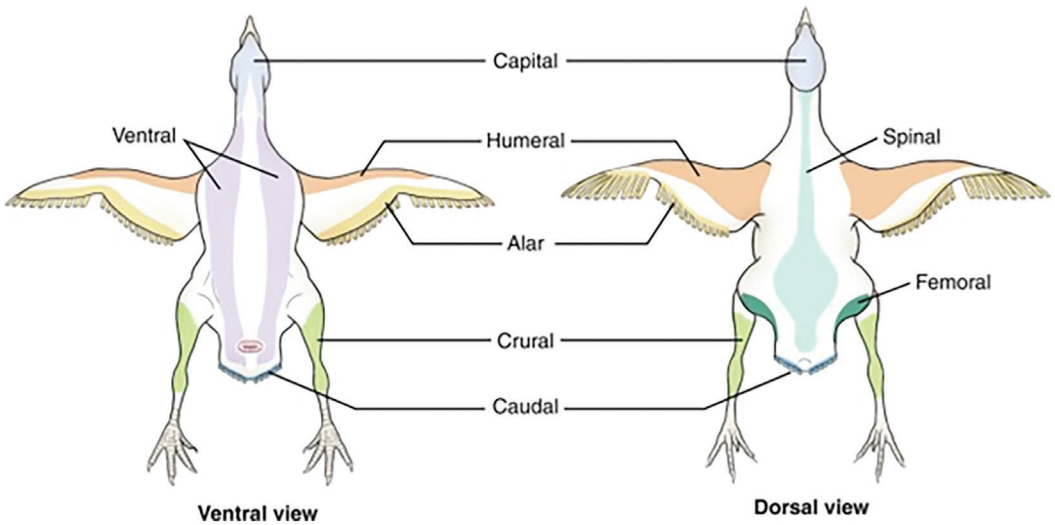


FIGURE 32.14 Unlike hair in (most) mammals, feathers do not cover the entire skin surface of birds. Instead, feathers are arranged in symmetrical tracts (pterylae) and are separated by areas of bare skin (apteria). Each species has its own characteristic arrangement of feather tracts. Represented are the feather tracts that can be recognized in the chicken including the (single) capital, and spinal tract, and the paired ventral, humeral, alar, femoral, crural, and caudal feather tracts.

a subdued (eclipse) plumage. Next, they lose all their flight feathers at once and are unable to fly while these feathers grow back; during this time, they use the water as a refuge. Once they can fly (after 3–4 weeks in smaller ducks, and 6–8 weeks for swans), the males molt their body feathers and grow their normal bright plumage in time for courtship.

Age and Feathers

Young birds are usually covered in down feathers that change to contour feathers as the *pullus* grows. Because the growing feathers are relatively heavy in many birds, the remiges and rectrices grow last—once the skeleton has formed sufficiently to cope with the weight of a heavy growing feather (Figure 32.15). Most birds have a juvenile plumage, which is characterized by feathers that are often smaller than those of the adult and have subdued colors—both of which are features having a lower energy cost. Smaller feathers grow to maturity more quickly, allowing the fledgling to be able to fly sooner. Juvenile psittacids tend to start to lose their juvenile feathers from 6 weeks of age. This is obvious in budgerigars *(Melopsittacus undulatus)* where the bars on the forehead are replaced with solid color feathers. *Pionus* parrots start to lose their head feathers at this time too and have replaced all the contour feathers on their head, neck, and shoulder region by spring the next year, while molting the rest of their juvenile feathers over the following 4 months.[34]

Feather Growth and Molt

In the embryo, each feather germ arises as an outpushing of the epidermis which encloses a core of the dermis. As it develops, the base of this structure invaginates to form a follicle. Once the follicle has grown, the dividing epidermis that lines the follicle remains active at the base of the follicle, forming a collar which becomes a germinal region for the cells that produce the feather. This area gives rise to the intermediate cells that form the rachis, barbs, and barbules by growth and then keratinization. It is a complicated process and while the mature feather can be seen arising from a cylinder of epidermis that eventually dries out and falls away the growing part of the feather is supplied through the umbilici with cells and blood vessels until the feather is fully formed (Figure 32.16).[45]



FIGURE 32.15 In the developing bird, the feather groups tend to grow at the same time. In many birds the wing feathers start growing after the leg bones have reached sufficient development to support the extra weight. This is particularly obvious in precocial birds who run around from a few days old. This wing of a 35-day-old bronze-winged parrot *(Pionus chalcopterus)* shows the growing wing feathers erupting from their sheath and uncurling. At this time, the weight of both wings is a significant proportion of the bird's total body mass. Strain on the skeleton is avoided by behavior as well as development.[37] The primary and secondary remiges in the illustrated species are light blue on the ventral aspect and dark blue dorsal. The coverts are bronze. (Photo courtesy Nigel Harcourt-Brown.)

The mature calamus fits closely within the follicle. The inner layer of the follicle, the epidermis, is continuous with the proximal umbilicus, and it surrounds the dermal papilla which projects into the calamus, holding it place. When the bird is stimulated into molt, the mesenchymal pulp that has been lying dormant over the dermal papilla is activated and starts to divide to produce the feather pulp. This activity pushes the old feather out of the follicle. The trigger for this process has been studied in many species, with significantly differing results.[32,46–50] Molts can occur either before or after breeding, and in many passerines they occur before or after migration. Juvenile plumage must be changed to adult plumage and in some species an adult winter plumage must change to breeding/summer plumage. All these molts can be complete or partial, so it is hardly surprising that the triggers and control can be so variable. It is generally accepted that thyroxine stimulates feather growth and estrogens inhibit it, but there seem to be a lot of different triggers to start the process that can vary tremendously between species. Once molt is underway, local factors come into play to control the sequence. In most of the birds molting their remiges, the molt starts at the carpus and works along the sequence of feathers to both the wing tip and the elbow. The next feather in the sequence is not lost until the adjacent feather is reasonably well grown. The process tends to be a mirror image between both wings. A new flight feather needs support from mature feathers on either side to prevent the relatively weak growing point at the feather's base from being damaged when the bird flies. This is important to realize as it can influence wing trim strategies that may be utilized with companion birds. (See also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 22, Advancements in Management of the Welfare of Avian Species.)

Traumatic loss of a feather initiates the growth of a replacement feather only if the dermal papilla is broken when the whole feather is removed.

FEATHER COLOR

Birds have tetrachromatic (or even pentachromatic) vision and their feathers reflect this. Only the trichromatic plumage characteristics of birds are obvious to human observers (Figure 32.17).[51] For instance, in one study, the application of UV-blocking sunscreen to the throat region of male bluethroats *(Luscinia svecica)* caused no obvious change in color to the human observers, but sexual and territorial displays went relatively unnoticed by other birds.[52] It diminished their hierarchical status and decreased their ability to select and hold a territory and mate.

Feathers distinguish between adult and immature birds by coloration and pattern, the coloration changing by molting and growing new feathers. Some birds molt twice yearly, and the breeding plumage is different to the off-season plumage.

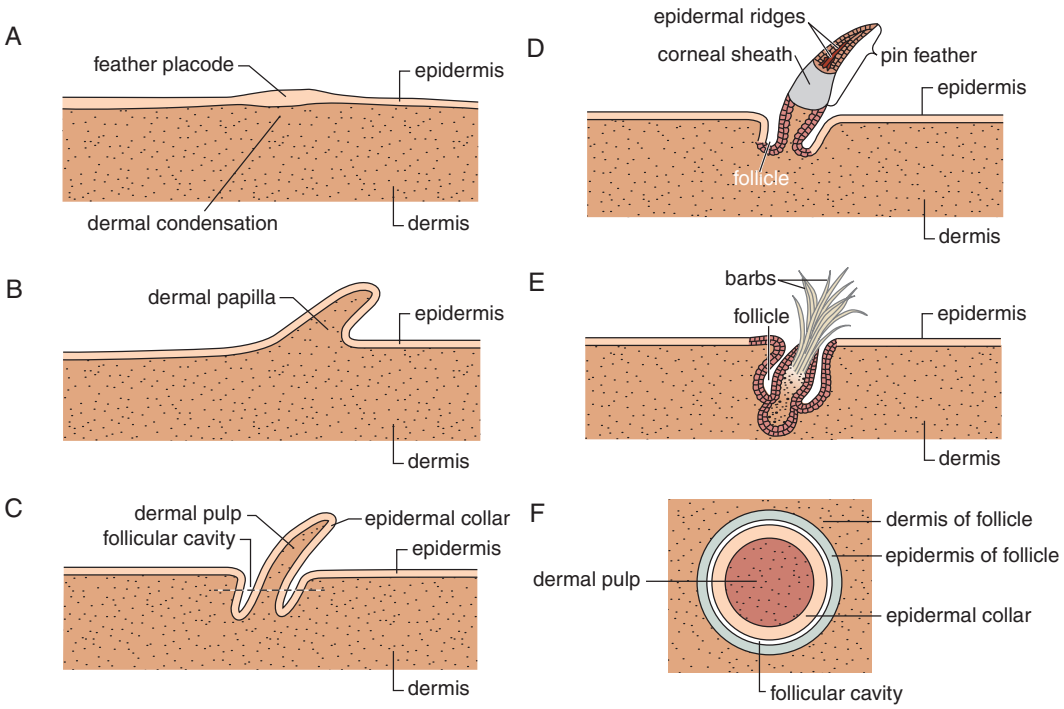


FIGURE 32.16 Schematic of the development of a feather follicle: (A) Formation of the dermal papilla which begins with the development of the epidermal feather placode and the dermal condensation. (B) Development of a feather papilla (or elongated feather bud) via the proliferation of dermal cells. (C) Formation of the feather follicle by the invagination of a cylinder of epidermal tissue (epidermal ridge) around the base of the feather papilla. (D) Keratinocyte production forces older cells up and out, eventually forming the entire feather that erupts from its protective sheath (E). (F) Cross-section of the feather follicle as indicated by the *dashed line* in (C). The follicle consists of a series of tissue layers (from peripheral to central), including the dermis of the follicle, the epidermis of the follicle (outer epidermal layer), the follicle cavity or lumen (the space between epidermal layers), the follicle (epidermal), collar (or inner epidermal layer), and the dermal pulp (tissue at the center of the follicle). The proliferation of feather keratinocytes and most of the growth of the feather occurs in the follicle or epidermal collar. (Adapted from Prum RO: Development and evolutionary origin of feathers, *J Exp Zool* 285:291–306, 1999.)[17]

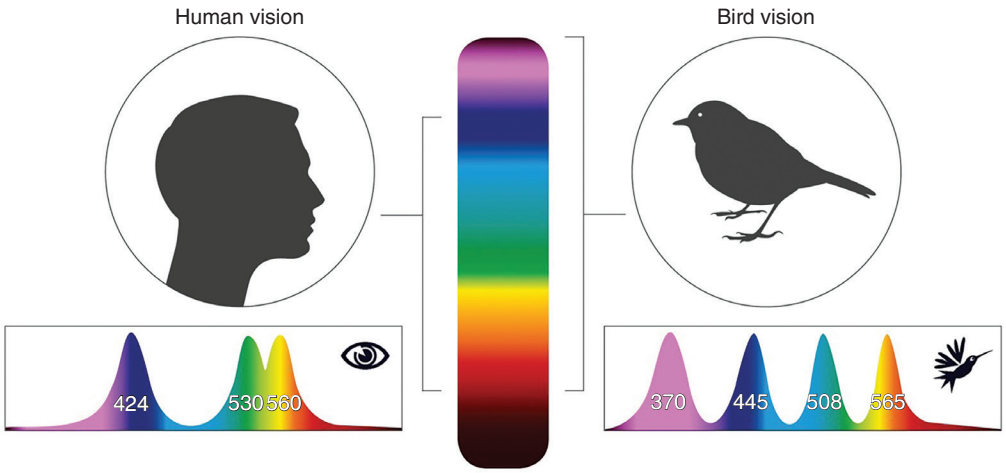


FIGURE 32.17 Comparison of human and avian vision. The human retina has three types of cones (red, green, and blue) resulting in trichromatic vision. Birds, on the other hand, have four or five types of photoreceptors, resulting in tetrachromatic or pentachromatic vision, which allows birds to detect wavelengths near the ultraviolet (UV) electromagnetic spectrum (400 nm). The ability to detect UV light is thought to be important for many biological processes including finding mates, finding materials to build nests, removing nest parasites, finding food, and (intra- and interspecies) communication.

This is very noticeable in, for example, whydahs and weavers (*Euplectes* spp.)[53–55] and black-headed gulls *(Larus ridibundus)*.[56] Some birds only molt once a year and their coloration changes after winter as the feather tips, which are lighter in color, wear down to reveal a more strident breeding coloration, as can be seen in, for example, brambling *(Fringilla montifringilla)*[54,57] and snow bunting *(Plectrophenax nivalis)*.[58] It also seems that the quality of the sebum can change the coloration of the feathers.

Coloration of the plumage is achieved in several ways. There are pigmentary colors *(biochromes)* and structural colors *(schemochromes)*; some colors are produced by a combination of these two methods.

Pigmentary Colors

Melanin is a tyrosine-derived pigment. It is bound to a tough protein component that forms the basis of a granule. These granules have a definite size, shape, and color that varies between species.[59] Work done on melanin granules in fowl showed they were larger than those of mice. Melanins are the most common pigments in feathers; they have been shown in fossil feathers.[60–62] On their own, melanin granules produce subdued colors but combined with other pigments or structural modifications they create many colors and vivid hues. Four types of melanin can be distinguished:

- eumelanin, which is the commonest and produces black or dark brown;
- phaeomelanin, which is lighter brown, reddish brown, or yellow;
- a trichosiderin-like pigment, which produces red, brown, and buff feathers and can be separated into purple I and II;
- erythromelanin, which produces chestnut red colors.

The color density is related directly to the granule density. For example, black and gray are formed by different concentrations of the same pigment.

Carotenoids are bright red, orange, or yellow pigments that are dissolved in fats (lipochromes) and occur in the feathers as colloidal globules. Birds, like other animals, cannot synthesize these pigments and must obtain them directly or indirectly from plants.[63–65] Carotenoids are found in two groups: carotenes and xanthophylls. Birds take up more xanthophylls than carotenes. The pigments are absorbed and carried into the body, where they are deposited either unchanged or, more often, altered, probably into oxidative intermediates. These new pigments often have distinct and sometimes unique properties. While dull yellows are caused by small amounts of phaeomelanin, bright yellows are carotenoid in origin. The principal yellow pigment is the xanthophylls—lutein. The principal red pigments are astaxanthin, rhodoxanthin, and canthaxanthin; these are transformation products of yellow carotenoids.

Carotenoids in body fat, egg yolk, and in the uropygial gland are diffused in lipoid droplets. In feathers the pigment is transferred into the cells of the growing feather and builds up until the "correct" color is formed. As the cells start to keratinize the fat solvent moves out of the cell and the pigment remains trapped in the keratin. Sometimes the deposit is formed as scales or layers. In some feathers, the pigments become hugely concentrated and the feather swells, for example, a red (astaxanthin) wax on the tip of the rachis of the secondary remiges of waxwings *(Bombycilla garrulus)*.[66]

Parrots have a novel set of yellow to red pigments (psittacofulvins) that are lipid soluble but differ from carotenoids by solubility and absorption spectra; some are fluorescent.[10] The pigments appear to be derived from carotenoids but have not been isolated from blood at the time of maximum feather production indicating that the pigment is made in the feather-producing cells.

Melanins and carotenoids are commonly seen in the same feather.[64,67] This combination forms various colors, for instance, olive green can be produced by a juxtaposition of tiny spots of yellow and black, as is seen in the greenfinch *(Carduela chloris)* and great tit *(Parus major)*. Generally, the exposed portions of the feathers are more pigmented than the covered portions. Melanins tend to occur in all types of feathers. Carotenoids, on the other hand, tend to occur in parts of feathers that are exposed, such as the contour feathers and the tips of semiplumes. They do not occur in feathers that are associated with flight and are also absent from the small interlocking parts of the feathers. Melanins can be found in all these structures.

Porphyrins have been found in red or brown feathers in a few families.[68] They characteristically fluoresce red under UV light and are a copper-containing pigment based on a porphyrin ring. In various embryos, coproporphyrin I is present in increasing quantities as hemoglobin increases. Porphyrins are abundant in owls and bustards (Otididae) as coproporphyrin 3[69] and have been found to be a positive indicator of how well the young bird was reared—body condition and quality of rearing territory. In adult bustards, they were found to allow females to select the "best" mate. These porphyrins occur in the down feathers but degrade quickly in UV light.

Some members of the turaco family (Musophagidae) have two porphyrins that color their plumage.[68,70] There is a dark red pigment, turacin, that is present in the remiges and a green pigment, turacoverdin, that occurs in most of the contour feathers. The amount of turacoverdin present in each green turaco species corresponds to their habitat: those that live in the greenest forest have the most turacoverdin in their plumage. These two pigments are based on copper-containing porphyrins. Some birds have up to 20 mg of copper in their plumage and based on analysis of their diet they need to eat 20 kg of fruit to obtain this. Some Galliformes (*Ithaginis* spp., *Rollolus* spp.), Anseriformes (*Somateria* spp., *Nettapus* spp.), and a jacana (*Jacana spinosa*) have feathers that contain a similar green pigment.[71] The yellow color in penguins is sphenisicin.[64,72]

Adherent Colors

There is a reddish secretion from the preen gland of some pelicans, which colors the plumage. The preen gland secretion in pied hornbills (*Anthracoceros* spp.) is bright yellow, which colors the beak. Some birds become a rusty brown shade from amorphous iron oxide in muddy soil, for example, the bearded vulture *(Gypaetus barbatus)*, common crane *(Grus grus)*, and some ducks.[18]

Structural Colors

Structural color production in feathers has been the subject of a huge range of research leading to what often appears to be confusing and even conflicting results. There are basically three classes of structures that produce color in feathers[73–76]:

1. Unspecialized, unpigmented keratin produced appears white because of incoherent scattering of light by the

keratin surface of the feathers and by air-filled, irregularly shaped, and randomly distributed vacuoles within the feather. This can be very efficient—a white ptarmigan *(Lagopus mutus)* can produce a reflectance that approaches 80% of that of fallen snow combined with the blue tinge of fresh snow (by Rayleigh scattering). Pure keratin fluoresces a light blue color which is usually masked by the presence of melanin granules.

2. Feather barbules in many birds have arrays of melanin granules which either alone or in combination with air spaces in the barbules can produce iridescent structural colors. The iridescence changes hue with the angle of observation. Hummingbirds are good examples of this type of structural color production.
3. Specialized, spongy, medullary layer in the feather barbs. This includes noniridescent blue, violet, green, as well as UV. In combination with carotenoids or similar pigments, structural barb colors are an essential component of almost all green bird plumages. Although the brightly colored feathers of *Amazona* spp. contain a range of biochromes, psittacofulvins, in both barbs and barbules, specific feather areas contain spongy nanostructured barb cells that reflect blue or blue-green wavelengths.[6] These blue-green spongy structures (or cloudy cells[1]) are partly enveloped by a blue-absorbing, yellow-coloring pigment which acts as a filter and gives a green color to the barb. Melanin granules act as reflectors to enhance the green color production (Figures 32.7, 32.18, and 32.19).

Structural colors are produced by light scattering from interfaces between structures of different refractive indices. This can occur as incoherent or coherent scattering.

Incoherent scattering is caused by structures where the space between them is greater than the wavelength of the light. This causes random scattering of light. The most cited models are Rayleigh and Tyndall scattering. Mie scattering is a better model but is far more complicated and requires a computer and complex program.[77,78]

Coherent scattering of light produces summation of light waves as they pass through the tissue. The light can shift in phase. Scattered waves that are approximately out of phase will cancel each other out while others that are in phase, or close to being so, will reinforce each other and produce color. The phase relationships among scattered waves are determined by the size and spatial distribution of the scatterers.

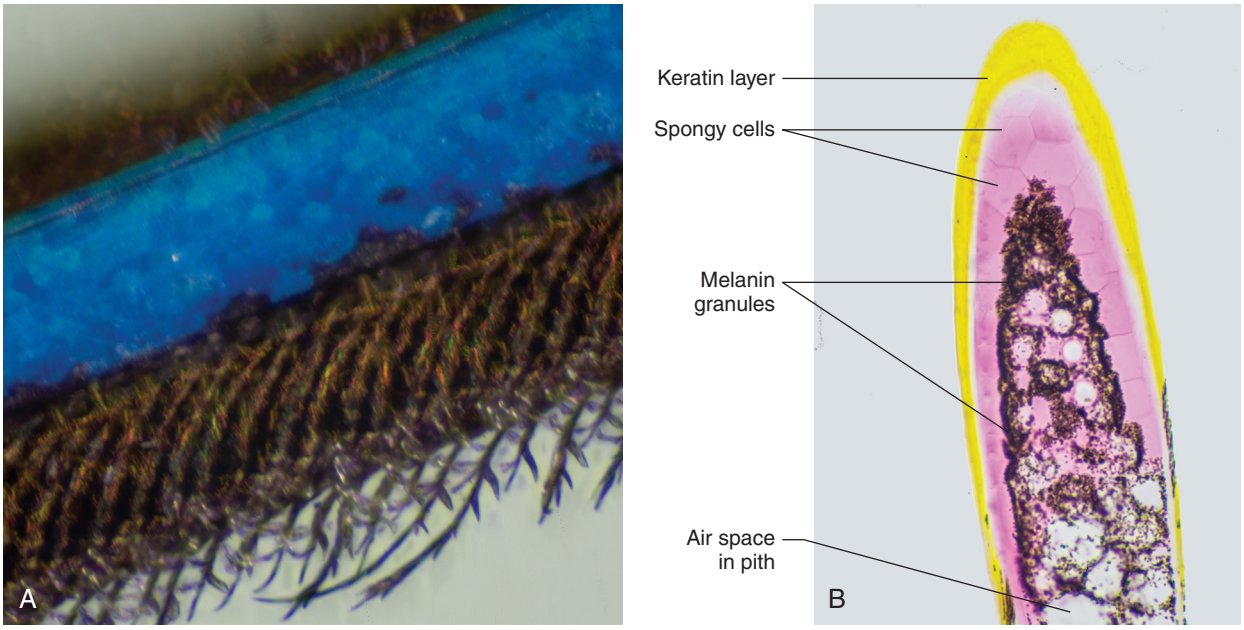


FIGURE 32.18 (A) Using blue feathers from a bronze-winged parrot *(Pionus chalcopterus)* and (B) green from a scaly-headed parrot *(Pionus maximiliani)*, it is easy to demonstrate the layer of spongy cells and show their importance in color production. A view of the dorsal aspect of a single blue barb shows the distal surface of the ramus and the distal barbules and their hooklets. The spongy cells can be seen to form a mosaic of slightly disparate shapes with differing depths of blue color. Areas where the cells are absent are dark because of the melanin showing from below the spongy cell layer. (B) A section of the dorsal aspect of the barb from a green feather, prepared in the usual manner for histology and stained with hematoxylin and eosin (H&E). Although the spongy cells have been shown to contain a fine spongy structure,[8] this is not shown on the H&E section; the cells are a homogeneous pink (eosin) although the cell walls can still be seen. In a green feather the spongy cells lie on top of a layer of melanin granules. This combination along with the overlying psittacofulvin pigment layer gives rise to a reflected green color. The space between the pigment layer and spongy cell layer may be artifactual but possibly the pigment in this area is more loosely held in sebum and has been removed by the xylene used in processing. In brown to black parts of the feather, the variation in melanin particle density gives rise to the depth of color. (Photo courtesy Nigel Harcourt-Brown.)

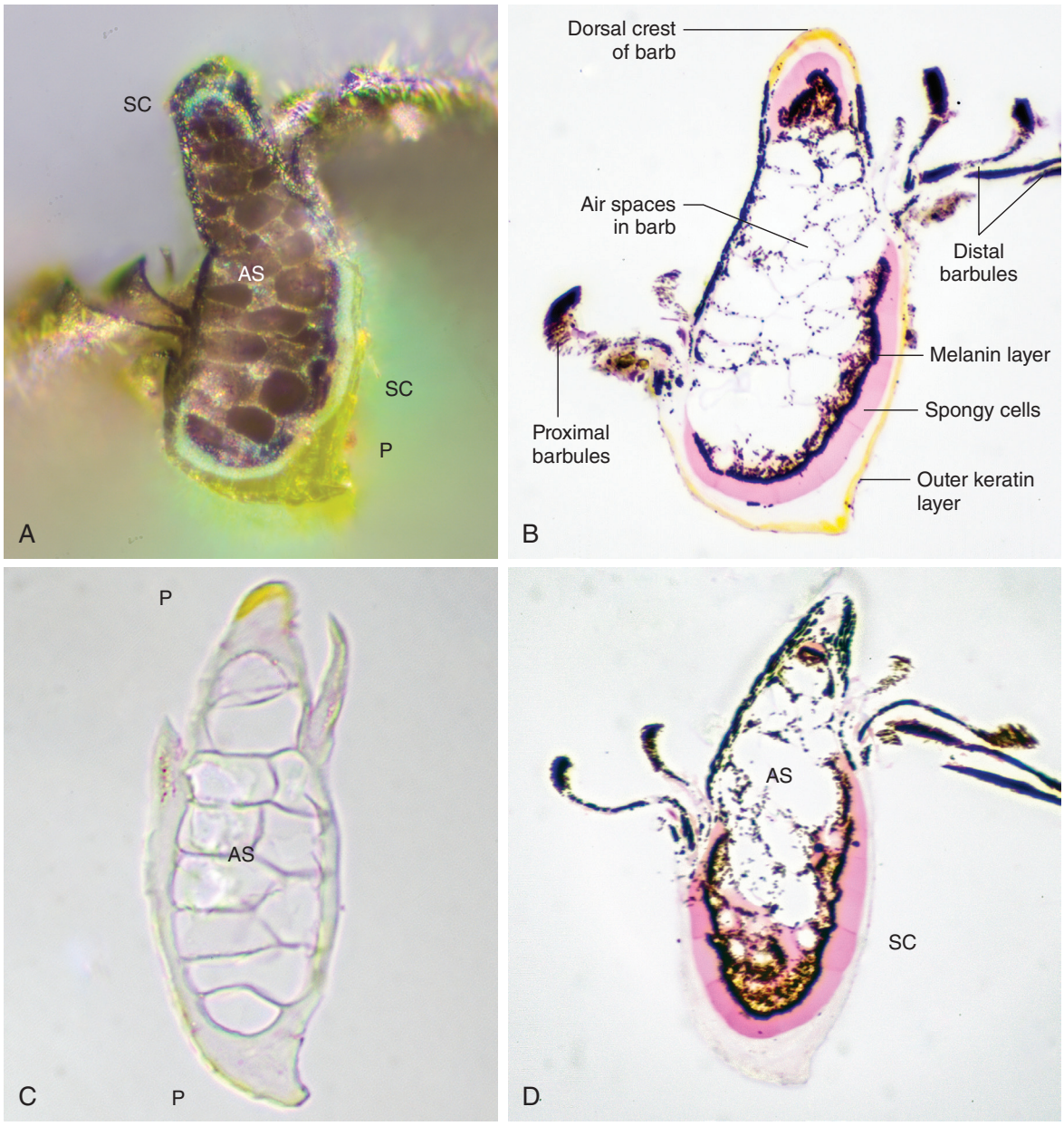


FIGURE 32.19 (A) Barb from the trailing edge *(Vexillum internum)* of a secondary remex of a scaly-headed parrot *(Pionus maximiliani)*; dark dorsal and green ventral crests. The barb has been cut and cross-illuminated to show the yellow psittacofulvin and melanin; because the section is cross-lit, the spongy layer shows some of the blue effect. The dorsal crest on this barb is very dark due to melanin granules mixed in with the carotenoid. (B) Section from a similar feather, except both dorsal and ventral crests are green. This barb has been cut and processed as a histological section stained with hematoxylin and eosin (H&E). In this section the area missing yellow pigment is obvious, compare with (A). (C) A feather from an area of old injury on a slender-billed conure *(Enicognathus leptorhynchus)*, cut as a histological section and stained with H&E. Trauma has left a scar and also affected the feather's color. This feather would normally be green but is completely yellow when viewed in normal light. Melanin granules have not been laid down and the spongy cells are also affected. (D) Barb from the trailing edge from a secondary remex from a bronze-winged parrot *(Pionus chalcopterus)*; dark dorsal and light blue ventral crests. There is an obvious lack of the yellow psittacofulvin pigment. The dorsal crest has a layer of melanin granules and no spongy cell layer; the loose distribution of the melanin granules gives a light black color. *AS,* air space, *SC,* spongy cells, *P,* psittacofulvin. (Photo courtesy Nigel Harcourt-Brown.)

The scatterers would need to be arranged regularly. If they are arranged randomly, there will be no wavelength reinforcement and the light will be incoherently scattered.

In biological tissues, coherent scattering is usually caused by structures of one refractive index immersed in a medium of different refractive index. Thin film reflectors are the best-known model. A thin film system produces the iridescent color of the feather barbules of hummingbirds[79] and the quetzals (*Pharomachrus* spp.).[77]

In any structurally colored tissue somewhere there must be an interface between the schemochrome and the supporting anatomy of the tissue. This supporting structure would normally interfere with color production. Therefore most tissues have a barrier, usually melanin granules, but occasionally a layer of carotenoid, which absorbs incident light that has passed through the entire color-producing system.

White, unpigmented feathers

White is a structural color that is a result of incoherent scattering in unpigmented feathers by the keratin surface of the feathers and by air-filled, irregularly shaped, and randomly distributed vacuoles within the feather. In the rock ptarmigan *(Lagopus mutus)* a feather from the winter plumage can reflect 50% of all wavelengths of visible light, compared to 15% to 18% by a white chicken's feather. In the rock ptarmigan, feathers overlap, giving a depth of color reflectance that approaches the 80% reflectance of the fallen snow in which they forage. The smaller wavelengths of light are slightly better reflected, giving the genuine blue tinge that characterizes fresh snow. Pure keratin fluoresces a light blue color. This is usually masked by the presence of melanin granules.[80,81]

Blue feathers

Gas-filled vacuoles in spongy cells[6] (also known as cloudy cells) within the rami in the cortex or medulla cause the distribution of light and it is reflected as a blue color (Figures 32.18 and 32.19). Light of long wavelengths is absorbed after passing through this layer by a layer of melanin. When dry, the cells reflect blue or violet light; when impregnated with liquid with the same refractive index as keratin (1.54) they become transparent. Xylene, cresol, Canada balsam, and oil of cedar wood (immersion oil) all have a refractive index of close to 1.54.

Structural colors of feather barbules

Schemochromes are usually produced by scattering from arrays of melanin granules, or melanin granules and air arranged through the keratin of the feather. These structures are best seen using transmission electron microscopy. The greater the number of scattering layers or depth of arrays, the more vivid the color.

Structural colors of feather barbs

The medulla of the barbs is filled with specialized light-scattering boxy or spongy cells. These cells scatter the light. The cells are made of a matrix of keratin rods interspersed with air vacuoles of varying shapes. Backscattering of incoherent light is prevented by underlying feather structures or by a layer of eumelanin (black to brown) or in a few species phaeomelanin (reddish brown).

Structural absorption by feather barbules

Feathers of some birds of paradise have highly modified contour feather barbules arranged in vertically tilted arrays, which increase multiple scattering of light and thus structural absorption. This causes extreme directional reflectance, giving an intense black feather when viewed from in front of the displaying bird.

Genetics of Bird Color

Many species of birds are maintained in captivity by the breeding of successive generations. In time, various color anomalies occur, which in the wild would be removed by predators or diluted in a large gene pool. However, many bird breeders are keen to propagate color mutations. Pigeons and poultry were among the first. Canaries *(Serinus canaria)*, budgerigars, cockatiels *(Nymphicus hollandicus)*, lovebirds (*Agapornis* spp.) and other parrots, and Gouldian *(Chloebia gouldiae)* and zebra finches *(Taeniopygia guttata)* have also been bred for color variations for a long time. In fact, any captive bird that is bred regularly has been "improved" by bird keepers in shape and plumage color. The mode of inheritance differs between species. Some colors are a direct Mendelian inheritance. Others are far more complicated. For those interested in learning more on breeding for color in the various bird species, a multitude of books are available.[82–84]

FEATHER ABNORMALITIES

Introduction

The integument is the largest organ system in the body, and the most obvious. Clients will commonly notice changes in their birds' plumage before they notice other health problems. Feather abnormalities can include changes in structure or changes in color. These abnormalities may be specific to the integument and plumage but can also often reflect more generalized health issues.

Changes in Feather Structure

Changes in feather structure (feather dystrophies) include any abnormalities of the feathers caused by genetic influences, trauma, disease, or malnutrition. These can include breaks in the feather vane, weakness in the rachis, retained sheaths, abnormal colors, feather cysts, clubbed feathers, and other abnormalities.

Genetic Influences on Feather Structure

During domestication of various bird species, a tremendous variety in hereditary features has arisen, with several of these genetically modifying the structure, distribution, and arrangement of feathers. Certain breeds have been specifically bred for those characteristics and need to adhere to specific descriptions detailing the ideal standard (e.g., American Standard of Perfection, British Poultry Standards, Deutscher Rassegeflügel-Standard, etc.). Selective breeding for these characteristics may significantly reduce the genetic diversity of the population. In addition, some mutations can have direct or indirect negative effects on the bird's health and/or reproductive capacity[85]:

- structural alterations of the vane or feather length (e.g., silky-feathering in domestic chickens, lace-feathering in

domestic pigeons) can affect the bird's flying ability or even lead to flightlessness;
- vane alterations (e.g., frizzle-feathering in domestic chickens) can result in a reduction or loss of the feathers' close-knit texture, thereby impairing thermoregulation and/or affecting the bird's ability to cope with weather conditions such as heat, cold, and rain;
- feather crests and/or vortices (e.g., in chickens, pigeons, and canaries) can impede the bird's sight;
- leg-feathering (e.g., muffed legs in domestic pigeons) and hypertrophies of single feather tracts or of the entire plumage can hinder mating, egg incubation, and rearing of the chicks;
- partial feather loss (e.g., loss of neck hackles in naked neck chickens), pleiotropic effects of a feather-altering trait (e.g., reduction in wattle size in chickens), or alterations in feather structure can negatively impact intraspecific communication due to loss of optical signaling functions;
- due to pleiotropic effects of single genes or close linkages with hereditary defects, modifications to the feathers and plumage can be associated with (semi)lethal effects or lowered vitality in homozygous individuals, and—to a lesser extent—in heterozygous individuals (e.g., feather crests in domestic ducks and canaries, ear tufts in domestic chickens);
- expression of specific phenotypes (e.g., long tail feathers in Onagadori roosters) may require specific environmental factors and housing conditions.

Feather duster or chrysanthemum syndrome in budgerigars

Feather duster syndrome is seen occasionally in juvenile budgerigars that exhibit continued growth of flight, tail, and contour feathers, with the feathers continuously growing to become long, soft, and curled, giving the bird the appearance of a "feather duster" (Figure 32.20).[85,86] The contour feathers often grow four to six times longer than the remiges and rectrices, whereas the remiges and rectrices are twice as long as in birds with a normal plumage. The elongated feathers hamper the vision of the birds, rendering them unable to fly, and the ongoing feather growth drains energy and nutrients from the body. Affected birds barely make an audible noise and many have microphthalmia. Most birds die before reaching adulthood, generally within the first few weeks of life, although some may live for several years. This has been suggested to be the result of a therapy-resistant protein deficiency related to the hypertrophied development of the feathers.[87,88] Feather duster syndrome is believed to be a lethal recessive genetic disorder with a strong correlation between affected birds and buff feathering.[89]



FIGURE 32.20 Feather duster disease or chrysanthemum disease refers to a condition seen occasionally in juvenile budgerigars *(Melopsittacus undulatus)*. Affected birds exhibit continued growth of flight, tail, and contour feathers, with the feathers continuously growing to become long, soft, and curled. (Photo courtesy Bob Doneley.)

Straw feathers in canaries

Straw feather disease is an unusual, lethal disorder, where the feathers fail to emerge from the feather sheath and the barbs and barbules fail to develop. This gives the feathers a straw-like appearance. It may occur in first-molt or adult birds.[90,91] The disease is believed to be hereditary in canaries, although viral causes have also been postulated. It has also anecdotally been reported in other passerine species, including zebra finches.[92]

Porcupine feathers

Porcupine feathers have been reported in Japanese quail *(Coturnix japonica)* and in homer and fantail pigeons. The condition resembles "straw feather disease" in canaries. The shafts of the feathers of affected birds appear normal, but the barbs fail to uncoil, giving the feathers the appearance of porcupine quills. Gross examination under light microscope reveals that both distal and proximal barbules are present and appear normal. Affected feathers are predominantly noticeable on the wings (i.e., the primary and secondary remiges) and on the back. As the birds grow older, the barbs partially uncoil so that the trait can be clearly observed only on the wings.[93]

Baldness in cockatiels, canaries, and zebra finches

A bald patch is often present behind the crest feathers on the crown of the head in lutino cockatiels (Figure 32.21). As this alopecia is not seen in most other color mutations of cockatiels, it is believed to have a sex-linked genetic origin. Baldness, characterized by loss of feathers at the back of the head, is also recognized in canaries and zebra finches. Certain canary breeds, such as the Giboso Espagnol or the Gibber Italicus, not only have a partially featherless head but also have a reduced number of feathers in the ventral pteryla and nearly featherless tibiotarsi and thighs.[94,95] In canaries, aside from hereditary factors, endocrine or hormone deficiencies have also been proposed. Since the baldness tends to be more common in males than in females, a gender-linked predisposition has been suggested. In finches, baldness on the head can be seen in both genders and is believed to also be caused by feather picking by a dominant individual from the group. In this case, the bird believed to be the picking bird should be separated from the group to see if this results in improvement. Supplementation with L-thyroxine (0.02–0.04 mg/kg q24h PO) or testosterone has also been attempted to diagnose or rule out endocrine disease. For genetically related baldness, no treatment is necessary (or available).[90]



FIGURE 32.21 Alopecia on the crown of the head of lutino cockatiels *(Nymphicus hollandicus)* is quite common and should be considered normal for this color mutation.

Featherlessness in other bird species

A hereditary decrease in the number of feathers, resulting in partial or complete featherlessness, is noted in various domesticated species of birds, including chickens, pigeons, and waterfowl.[85] Scaleless chickens, for example, are almost completely devoid of feathers, scales, foot pads, and spurs.[96] The condition renders them useful as research models for examining the role of epidermal-dermal tissue interactions but also makes them susceptible to temperature changes, and because of an impaired sense of balance they cannot copulate normally. Partial featherlessness in the neck region is characteristic of certain breeds, such as the Romanian Naked Neck Tumbler, Canard Cou-nu, and Transylvanian Naked Neck chicken. As the trait in the Transylvanian Naked Neck chicken is autosomal inherited and incompletely dominant, heterozygous individuals are characterized by an isolated tuft of contour feathers on the ventral side of the neck above the crop, while this tuft is absent or reduced to very few pinfeathers or small contour feathers in homozygous individuals.[97] While the reduced feather coverage results in increased heat loss from the body, hence allowing the bird to cope better with higher temperatures, it also exposes the featherless skin to potentially harmful UV radiation and hinders intraspecific communication due to missing neck hackles that are important in threat postures.

Feather cysts in canaries

Feather cysts appear as in-grown feathers, forming hard, yellow nodules on the skin of type canary breeds (i.e., canaries that are bred for their shape and/or stance) such as the Norwich, Border, and Gloucester strains (Figure 32.22). Affected canary strains have been genetically selected to produce an extra down-type feather (double buff) that may predispose them to cyst formation. It has been hypothesized that the feather cyst formation results from an altered expression of molecules that regulate feather morphogenesis and feather growth. For example, Widelitz et al. proposed that an abnormally high level of β-catenin in the skin leads to enlarged feathers with abnormal shapes.[98] The mode of inheritance of feather cyst formation is not clear.[89,90] In canaries, the cysts are believed to be benign neoplasms of the feather follicle (folliculomas or basal cell tumors). Several dermal papillae form in each follicle, resulting in a tangle of feathers that fail to erupt from the skin.

The dorsal thoracic area is a frequent site of multiple cysts, but they can occur anywhere on the body and wings. Cysts often occur in several sites along a feather tract and appear as hard, yellow nodules containing the trapped curling feathers (Figure 32.23). Badly affected birds have irregularly directed feathers all over their bodies. Actively growing feather cysts will have vascular walls and contain blood and gelatinous material. Mature cysts will contain drier keratinous material, and the cyst wall may be more expansive, thickened, and reduced in vascularity.

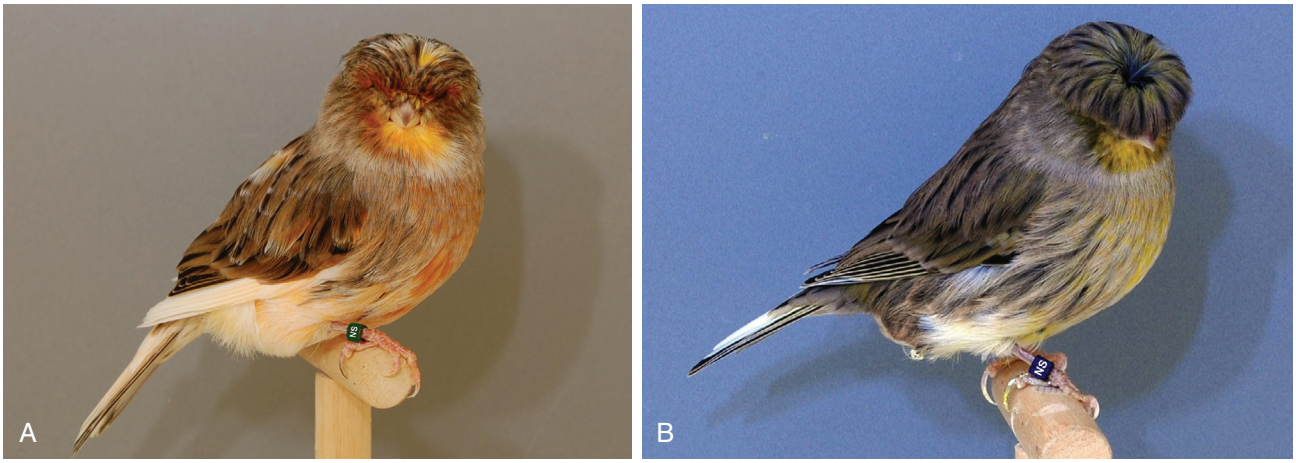


FIGURE 32.22 Examples of soft-feathered canary breeds: (A) Norwich. (B) Gloster. These canary breeds are prone to developing feather cysts. (A, from Wikipedia: https://commons.wikimedia.org/wiki/File:Norwich_gescheckt.jpg; B, from Wikipedia: https://commons.wikimedia.org/wiki/File:Gloster_corona_melanin.jpg.)



FIGURE 32.23 Close-up of a feather cyst on the left chest area in a Norwich canary *(Serinus canaria)*. Feather cysts can develop anywhere along a feather tract and typically present as a hard, yellow nodule containing feather keratin. (Photo courtesy Yvonne van Zeeland.)

Diagnosis is usually based on the clinical signs and appearance of the mass. Cytology may reveal erythrocytes, erythrophagocytosis, mixed-cell inflammation with a marked amount of background debris, multinucleated giant cells, and/or the presence of feather fragments. Treatment of individual cysts can be conservative by simply lancing and expressing the contents of the cyst. Recurrence is common, however. Surgical excision of the affected follicle(s), on the other hand, is generally curative. When multiple cysts are present, surgical excision of the affected pteryla(e) may be required. Cysts on the wings may not be amenable to surgery without partial wing amputation, making repetitive conservative therapy warranted.

Silkiness and lace-feathering

In silky-feathered chicken and lace-feathered pigeon breeds (Figures 32.24A and 32.25A), the contour feathers have no interlocking barbules and therefore do not form closed pennaceous vanes. Silky feathers obtain their characteristic hairy structure from the loss of hooklets on the distal barbules combined with an increased plumaceous portion of the vane. This typical change in feather structure can also be induced by thyroidectomy[99,100]; however, no differences have been found in thyroxine secretion rates of silky-feathered chickens compared to that of normal individuals.[101] In lace-feathered pigeons *(Columbia livia* f. *domestica)* and ring-neck doves *(Streptopelia roseogrisea* f. *risorii)*, the lace feathering is inherited by an autosomal, incompletely dominant gene,[102,103] hence resulting in heterozygous and homozygous individuals differing in the degree of lace-feathering. In the heterozygous birds, the feathers are plumulose, but in the homozygous individuals, the feathers appear tattered, rough, and brittle.

Frizzled feathers

Frizzled feathers have a peculiar upward-turning shape, which gives the appearance of the plumage having been brushed against the normal feather orientation. The condition results from an altered shaft morphology of the contour feathers, which results in an upward curving. Other structural abnormalities, such as thickening of the barbs and barbules and modifications of the hooklets, can also be present. Rectrices and remiges are less often affected but commonly have a ragged appearance. Secondaries also frequently lack inner vanes. Although this type of feathering has been described in various domesticated bird species, it has solely been established as a breed characteristic in domestic chickens. In these frizzled chickens (Figure 32.24B), several organ and physiologic changes (e.g., enlargement of the heart, spleen, and intestinal tract, lack of subcutaneous fat depositions, delayed sexual maturity, and decreased fertility) have been associated with a continuous, elevated loss of body heat that results from the abnormal plumage.[100]

Curled feathers

In certain breeds of domestic pigeons, such as the Frillback pigeon (Figure 32.25B), feather modifications like those seen in the frizzled chickens can be seen. These birds typically have wavy feathers as a result of uneven growth during feather development,[104] which leads to curling of the individual feathers. The curling is most pronounced on the wing coverts, where the distal parts of the contour feathers roll up like corkscrew spirals, but is less visible in the remiges, rectrices, and contour feathers of the neck and back.

Spiraled feathers

Elongated, spiraled feathers are found in the Sebastopol (Figure 32.26) and Lockengänse breeds of the domestic goose *(Anser anser* f. *domestica)*. This spiraling is caused by changed growth processes and disturbed keratinization during feather development, which lead to a lack of closed vanes in affected feathers. As the shafts of these feathers are soft and fragile, they are often fissured or cleaved and droop.[105]

Henny feathering

The so-called "henny-feathering trait," as can be seen in domestic chicken breeds such as the Sebright and Campine (Figure 32.24C), has been associated with a cutaneous hormonal defect that results in conversion of testosterone into estrogen in the male. As a result, the secondary male sex characteristics of the plumage are not formed, leading to affected roosters having a feather structure like hens.[106,107]

Fat quills

In some pigeon breeds (e.g., Nuremberg swallow, Thuringian swallow; Figure 32.25C), so-called "fat quills" are developed, which are modified powder downs that remain enclosed within a persistent feather sheath.[108] Transitional stages between fat quills and normal pulviplumae also occur. The "fat" in the quills originates from a fatty degeneration of the inter-radiogenic (non-feather-building) epithelium of the developing feather and has been hypothesized to assist in conditioning and waterproofing of the feathers, like preening oil produced by the uropygial gland.

Ear tufts

Ear tufts, which can be seen in, for example, the Araucana chicken as peduncular, feather-covered skin appendages positioned near the ear opening (Figure 32.24D), are inherited as an autosomal, incompletely dominant trait with variable expression. In homozygous individuals, the ear tuft gene leads to dead-in-shell embryos, whereas elevated fetal and postnatal mortality is observed in heterozygous specimens. As ear tuftedness also



FIGURE 32.24 Examples of fancy chicken *(Gallus gallus)* breeds with characteristic plumage. (A) Silkie chicken. (B) Frizzled chicken. (C) Silver Sebright bantam chicken, male, expressing the henny-feathering phenotype. (D) Araucana. (E) Crevecoeur. (F) Huiyang Bearded chicken. (G) Silky bantam chicken. (H) Onagadori chicken. (A, Courtesy Bob Doneley. B, from Wikipedia: https://commons.wikimedia.org/wiki/File:Pekin_poule.png. C, from Wikipedia: https://commons.wikimedia.org/wiki/File:Coq_Sebright_Argent%C3%A9.JPG. D, from Wikipedia: https://commons.wikimedia.org/wiki/File:Kogut_rasy_araukana.jpg. E, from Wikipedia: https://commons.wikimedia.org/wiki/File:Crevecoeur_chicken.JPG. F, from Guo Y, Gu X, Sheng Z, et al: A complex structural variation on chromosome 27 leads to the ectopic expression of HOXB8 and the muffs and beard phenotype in chickens, *PLoS Genet* 12(6):e1006071, 2016. G, from Wikipedia: https://commons.wikimedia.org/wiki/File:Silky_bantam.jpg. H, photo courtesy Diandra Dills, Wikimedia Commons: https://commons.wikimedia.org/wiki/File:Gold_Onagadori.jpg.)

affects the size and shape of the ear opening and the length of the ear canal,[109] hearing ability of these individuals may be impaired though this has not yet been confirmed.

Feather crests

Feather crests are seen in many of the domestic bird species, including chickens, ducks, and geese. Among chicken breeds, two phenotypes of feather crests can be distinguished, that is, the helmet-shaped, forward-inclining feather crest (e.g., in the Appenzeller chicken) and the full crest (e.g., in the Polish chicken). The full crest is inherited as an autosomal, incompletely dominant trait, with homozygous crested chickens lacking filoplumes in their crest compared to the heterozygous individuals.[110] Gender differences also exist, with contour

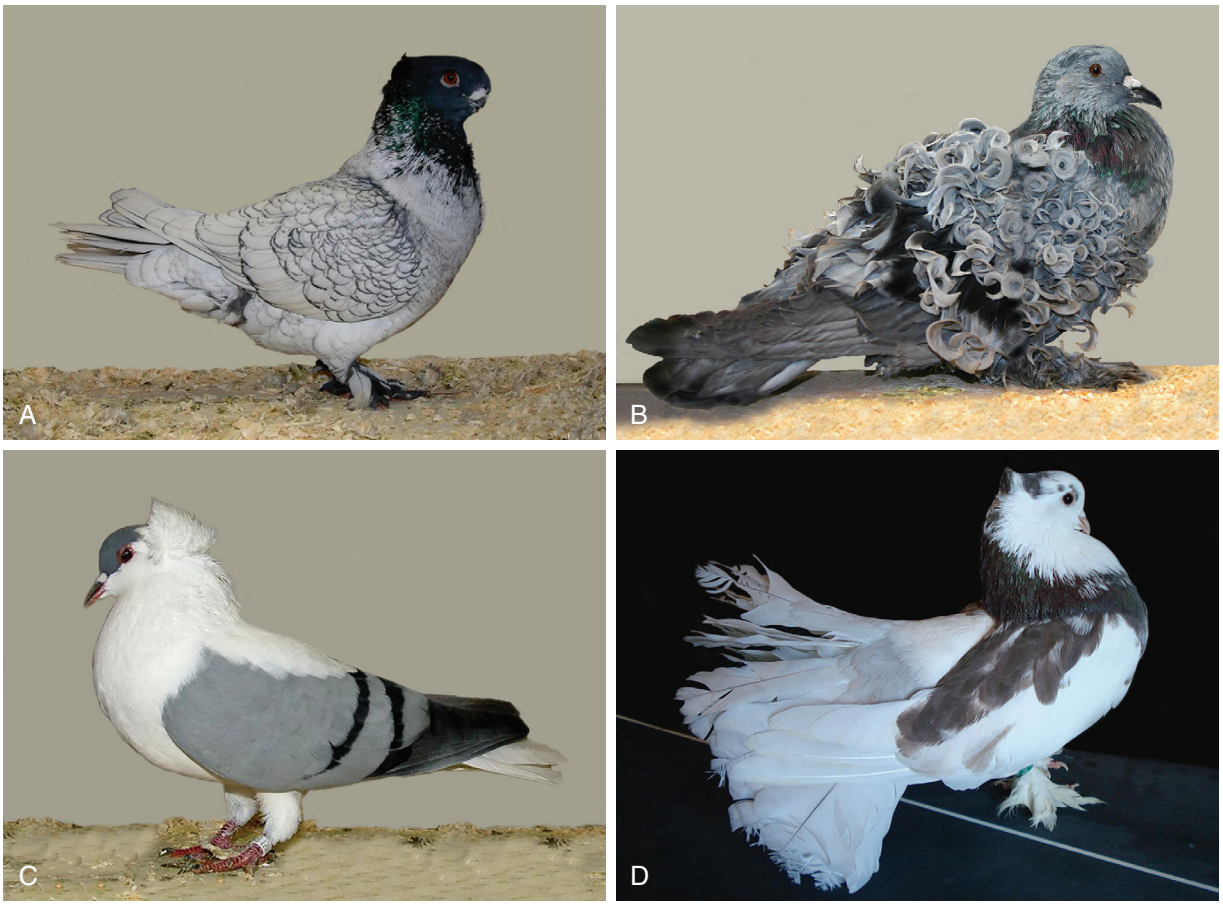


FIGURE 32.25 Examples of fancy domestic pigeon *(Columba livia)* breeds with characteristic plumage. (A) Oriental Frill pigeon. (B) Frillback pigeon. (C) Thuringian swallow. (D) Fantail pigeon. (A, from Wikipedia: https://commons.wikimedia.org/wiki/File:Oriental_frill (blue_laced_blondinette).jpg. B, from Wikipedia: https://commons.wikimedia.org/wiki/File: Frillback_pigeon.jpg. C, from Wikipedia: https://commons.wikimedia.org/wiki/Category: Thuringian_Swallow. D, from Wikipedia: https://commons.wikimedia.org/wiki/File:Indian_ fantail(red_splash).jpg.)



FIGURE 32.26 Sebastopol domestic goose *(Anser anser)*. This breed is unique in that it has long, curly feathers which is the result of disturbed keratinization of the feathers due to a genetic mutation. (Photo courtesy Davee, Wikimedia Commons.)

feathers being long and pointed in the male and rounded in the female. The scalp area in which the crest is vertically inserted is thickened and has a conspicuous hypodermal fatty layer. Crested chicken breeds also commonly have anomalies of the skull, including protuberant and vaulted frontal bones, and deformities of the nasal, intermaxillary, and other cranial skeletal bones.[100,110] These anomalies are caused by dysmorphogenesis of the brain, and in particular a bulging of the cerebral hemispheres. Furthermore, certain cerebral areas (e.g., the optic tract, diencephalon, telencephalon, parts of the hyperstriatum, and neostriatum) are significantly enlarged in crested chickens compared to noncrested individuals.[111] While the formation of skull protuberances is not causally connected to the formation of a feather crest, it is closely linked to the crest locus.[110] In some crested breeds of chicken, such as the Polish crested, Silkie, and Crevecoeur chickens (Figure 32.24E), the formation of a crest has been linked to cerebral herniation. This herniation, which results from a defective skull formation during incubation that allows the telencephalon to bulge out through a hole in the skull (Figure 32.27), is inherited as an autosomal recessive trait and can lead to a variety of neurological signs (e.g., ataxia,

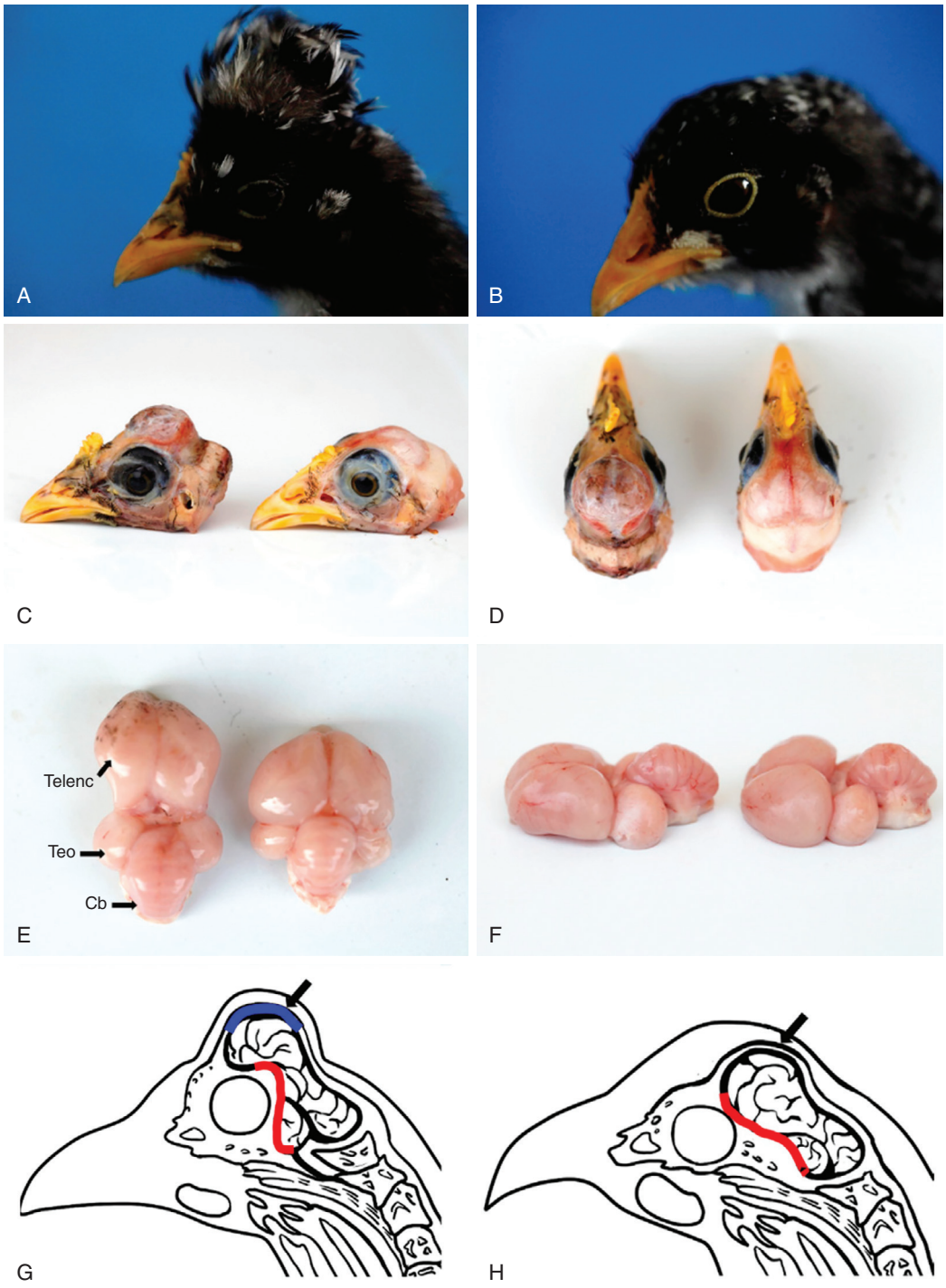


FIGURE 32.27 Anatomy and morphology of cerebral hernia-type and wild-type chickens. (A, B) The appearance of chickens (A) with cerebral hernia (cerebral hernia type) and (B) without cerebral hernia (wild type). (C, D) The differences of frontal bone between cerebral hernia type and wild type are shown in (C) the lateral view and (D) the dorsal view, respectively (*left*, cerebral hernia type; *right*, wild type). (E, F) The external morphology of the brain in cerebral hernia-type and wild-type chickens from (E) the dorsal and (F) the lateral view, respectively (*left*, cerebral hernia type; *right*, wild type). (G) The diagram reveals abnormal development of the sphenoid bone and membranous frontal bone in the cerebral hernia-type chickens (misplaced sphenoid bone is denoted in *red*, and the membranous frontal bone is denoted in *blue* region and with a *black arrow*). (H) The diagram shows the morphology of the sphenoid bone and frontal bone in wild-type chickens (sphenoid bone is denoted in *red*, and frontal bone is indicated by a *black arrow*). *Cb*, Cerebellum; *Telenc*, telencephalon; *TeO*, optic tectum. (Reprinted from Tao Y, Zhou X, Zheng X, Li S, Mou C: Deciphering the forebrain disorder in a chicken model of cerebral hernia, *Genes* 11(9):1008, 2020.)

torticollis) due to the bulging brain being susceptible to trauma and damage.[112,113]

Similar to chickens, crested ducks have a crest consisting of proportionally enlarged contour feathers and downs which arise from a thickened hypodermis of the occiput. In crested ducks, the underlying skull bones are rarely closed but rather show numerous perforations in the junctions of the frontal, occipital, and parietal bones *(Sutura supraoccipitoparietalis*, *Sutura interparietalis*, and *Sutura frontoparietalis)* and in the caudolateral region of the parietal bones *(Ossa parietalia)*. Originally, it was hypothesized that fat and connective tissue from the crest could protrude into the skull through these perforations, leading to compression of the cerebellum and other brain structures with subsequent onset of neurological signs such as ataxia and blindness.[110–111] However, it was later discovered that the intracranial fat bodies represent intracranial lipomas that can develop in both crested and plain-headed individuals that have at least one parent descending from a crested specimen.[114,115] In addition to these problems, increased mortality among offspring with craniocerebral malformations or malformations associated with duplication of body parts has been reported in crested ducks.[116] A phenotype similar to that in crested ducks (i.e., feather crests arising from hypodermal thickenings of the occiput, and perforated skull roof) can be observed in crested (tufted) breeds of geese (e.g., Tufted Roman, Emporda).[117]

Feather vortices

The feather crests as can be observed in some pigeon breeds (e.g., Bokhara Trumpeter, Komorner Tumbler, Oriental Frill), budgerigars (crested budgerigars), and passerines (e.g., Gloster, crested, and German crest canaries, crested varieties of the zebra finch and Bengalese finch, *Lonchura striata*) are termed feather vortices (Figure 32.22 and Figure 32.25C).[85] These vortices differ from the crests in chickens and ducks in several aspects:

1. They are formed by proportionally enlarged yet normal contour feathers.
2. Their location is variable across species, that is, above the beak in, for example, the beak-crested German trumpeter, on the forehead in, for example, the Harzburger Trommeltaube, on the back of the head in, for example, the Oriental Frill, or central on the top of the head with feathers radiating from the center in passerines. In addition, feather vortices can occur in other body regions such as the lateral neck in, for example, the Jacobin pigeon, or on the back, breast, and flanks in "frilled" type canaries or Bengalese finches.
3. Their appearance can be variable, for example, tufted, semicircular, or circular, which are the three forms that are recognized in budgerigars.[118]

In budgerigars, it has been suggested that vortices are inherited by an incompletely dominant autosomal gene, leading to one (in heterozygous birds) or two centers of vortex formation (in homozygous individuals).[119] Like the crests in ducks, the vortex trait in budgerigars has been associated with increased fetal mortality resulting from internal hydrocephalus and cerebral hemorrhage in both homozygous and heterozygous individuals. In addition, a relative high proportion of the (live) birds display abnormal behavior, ataxia, and tremors.[119]

In canaries, zebra finches, and Bengalese finches, the vortices are inherited by an autosomal incompletely dominant gene which leads to mortality of the homozygous chicks just before or shortly after hatching.[120,121] Aside from increased mortality and neurologic signs, vortices can also lead to impaired vision when they envelop the top and sides of the head (e.g., in Jacobin pigeons). This requires breeders to clip the vortex feathers to allow the pigeons to successfully raise their chicks.

Muffs and beards

Muffs and beards are feather modifications that can be seen in chickens and always occur in conjunction with a reduced wattle size.[122] Muffs are formed by elongated contour feathers that project horizontally from both sides of the face, while the beard consists of elongated contour feathers that project downward under the chin (Figure 32.24F).

Ptilopody and vulture hocks

Several chicken and pigeon breeds are characterized by ptilopody or "feet feathering," which is the result from a genetic mutation that leads to a transformation of the scales on the tarsus, feet, and toes into feathers.[107] Feet feathering can be expressed in various degrees. For example, "groused" feet are covered with small contour feathers, whereas "slippered" legs have enlarged feathers on the feet, and "muffed" legs develop primary-like feathers on the feet and toes. "Vulture hocks," which are characterized by elongated, stiff, and caudally projected contour feathers covering the tibia, can be found, for example, in the Booted Bantam and Sultan chicken breeds (Figure 32.24G) and Old Holland Pouter and Saxon Fairy Swallow pigeon breeds.

Longtail

Some Japanese chicken breeds, for example, the Onagadori breed (Figure 32.24H), are characterized by extremely long tail feathers, which can grow up to 13 m long with a growth rate of approximately 1 m per year.[123] While the trait is genetically determined, it is also influenced by environmental factors, for example, restricted housing.[124]

Hypoplastic tail feathers. In the so-called Ingie breed of chicken, rectrices and tail coverts are underdeveloped, showing a slender rachis as well as abnormal anterior and posterior barbules with a reduced number of hooklets.[107]

Increased number of tail feathers. Certain pigeon breeds (e.g., Danzig Highfliers, Oriental Rollers, Fantails; Figure 32.25D) have an increased number of tail feathers compared to the normal 12 that are present in the rock pigeon.[125] In Fantails, which can have >30 rectrices, the rectrices are kept spread out and are carried erect, forming an expanded fan. As such, they cannot assume a normal mating posture and copulate successfully unless the lateral rectrices are trimmed.

Physical Influences on Feather Structure

Physical conditions can also temporarily affect the appearance of the feathers. These feathers will eventually molt out and be replaced with normal feathers.

Stress lines

Stress lines are translucent lines across the vane of a feather, oriented perpendicular to the shaft (Figure 32.28). They represent segmental dysplasia that occurred in the developing

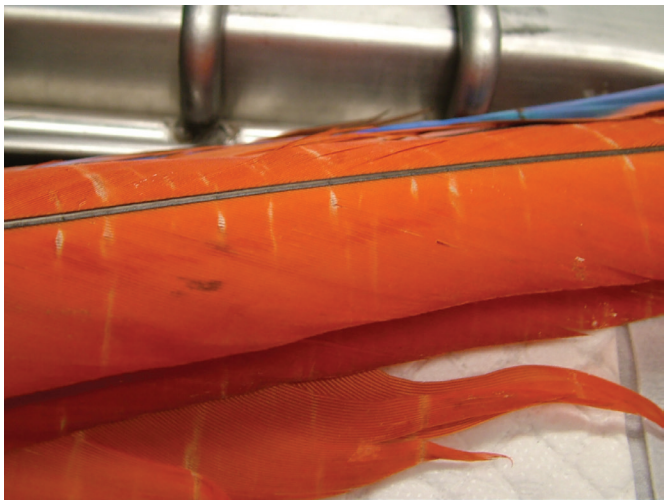


FIGURE 32.28 Stress lines, as seen in this scarlet macaw *(Ara macao)*, are translucent lines across the vane of a feather, oriented perpendicular to the shaft. They represent segmental dysplasia that occurred in the developing barb and barbules due to a brief period of dysfunction in the epidermal collar from which the feather arises. (Photo courtesy Bob Doneley.)



FIGURE 32.29 Damage or trauma to the feathers can be the result of inappropriate housing, damage by other birds, animal attacks, or through trauma such as heavy falls or impacts. This sulphur-crested cockatoo *(Cacatua galerita)* was housed in an inappropriately sized cage for an extended period. (Photo courtesy Bob Doneley.)

barb and barbules due to a brief period of dysfunction in the epidermal collar from which the feather arises. This dysfunction is generally the result of the release of corticosteroid hormones as a result of a stressful event (e.g., transport, restraint, nutritional deficiencies, food deprivation, or environmental stressors) that occurred while the feather was growing. The barbules do not form, resulting in a line of weakness at the same level. Stress lines may also develop following exogenous corticosteroid administration during feather growth. They are commonly noted in young birds and may be associated with illness or a change in diet or disruption of their feeding schedule when they are being weaned (see Chapter 41). Stress marks are easily identified upon inspection of the plumage or spreading the wing and/or tail feathers and holding them against the light. Their clinical relevance depends largely on the number of stress marks that are present and the bird's overall condition. A few stress marks in an otherwise healthy bird may have mere aesthetic impact. A larger number of stress bars generally warrants further investigation into the underlying cause, but this is often difficult as the marks are the result of a problem that occurred during feather development in the past that may not be present any longer. The underlying cause has to be identified & eliminated; no further treatment for the feather abnormalitiy is needed as it will now be able to correct itself at the next moult.[89,90]

Damage or trauma to the feathers

Damaged feathers are relatively common in companion, aviary, and wild birds (Figure 32.29). They can be the result of inappropriate housing (e.g., too small an enclosure so that the bird brushes its feathers against the side constantly), damage inflicted by other birds (particularly over-amorous mates), predator attacks (e.g., birds of prey, dogs, or cats), restraint, or through trauma such as a heavy fall or collision impact. Often the primary feathers on the wings, the alular and carpal covers, and/or tail feathers will be abraded, broken, or removed. Malnutrition may predispose the bird to such damage as nutritional deficiencies may weaken the feathers, rendering them susceptible to damage. Elimination of the predisposing factors may help to prevent problems in future, but a molt is necessary to shed the damaged feathers. If an early return to function is required (e.g., for the release of a wild bird), a falconry procedure (imping) can graft replacement feathers onto the stump of damaged feathers (Box 32.2).[90]

Damage to newly erupted (blood) feathers may be more problematic as this can result in severe hemorrhage, especially if the bird has an underlying coagulopathy (e.g., hepatic lipidosis). These newly emerged feathers, known as "blood feathers" (because of the blood vessels in the calamus and rachis; Figure 32.30), are enclosed in a soft keratin sheath which is easily damaged. Such damage is often associated with improper wing trims exposing the blood feathers to damage. If the damage is mild (e.g., a longitudinal split in the keratin sheath), hemorrhage may be controlled by manual pressure or applying a paste of flour and water to the split. Chemical or thermal cauterization should not be used. If the bleeding is severe or cannot be controlled, removal of the feather may be required. This is achieved by administering appropriate analgesia, with or without sedation or anesthesia, followed by grasping the feather at its base and pulling it from its follicle, along the line of feather growth, and then applying manual pressure to the follicle until the bleeding is stopped.[90]

BOX 32.2 CLINICAL TECHNIQUE: IMPING PROCEDURE

1. Anesthetize the bird (e.g., using isoflurane) to reduce stress and prevent the bird from destroying the freshly repaired feather through movement and struggle. Analgesia is not required; this feather repair technique is painless because noninnervated and nonvascularized parts of the avian integument are manipulated.
2. Place the anesthetized bird on a towel or paper cloth, and provide thermal support with conductive (e.g., heating pad) and/or convective (e.g., warm air) equipment.
3. Assess the damaged primary feathers (Figure 32.2.1) and retract the covert feathers away from the broken flight feather with masking tape; this will expose the base of the affected shaft (calamus; Figure 32.2.2).





4. Compare the length and shape of the possible replacement feathers (Figure 32.2.3) with the damaged one. If the length and shape of the replacement feather are acceptable, align it in proper position relative to the adjacent feathers (Figure 32.2.4). Make sure to maintain bilateral symmetry with the opposite side of the tail or wing when replacing a feather.





5. After determining the correct length of the replacement feather, mark the cutting line with a pen on both feathers (Figure 32.2.5). If more than one feather needs to be imped, replace the innermost feather first for optimum results.



6. Trim the damaged feather approximately 15 to 25 mm distal to the skin with a nail cutter (Figures 32.2.6 to 32.2.8).





BOX 32.2 CLINICAL TECHNIQUE: IMPING PROCEDURE—cont'd



7. Trim the new feather along the marked cutting line, leaving a clean, cylindrical shaft. Do not use scissors to trim the feather because this will cause the shaft to split.
8. Clean out the feather shafts with a blunt object (e.g., small drill bit, paper clip).
9. Shave a bamboo stick until it fits tightly within the feather shafts of both feathers. Confirm that the shaft of the donor feather has perfect alignment with the feather shaft of the recipient site.
10. Place a piece of paper or cardboard under the imping site to prevent glue exposure on adjacent feathers (Figure 32.2.9).



11. Prepare the fast-setting 5-minute epoxy glue (a mix of equal parts of epoxy resin and hardener), then lightly coat the implant with the glue and insert approximately 2.5 cm into the shaft of the new feather. Aspirate the remainder of the prepared glue with a 1-mL tuberculin syringe and inject into the old feather shaft. Insert the implant into the recipient feather shaft and connect both feather fragments (Figures 32.2.10 and 32.2.11).





12. Rotate the new feather for proper positioning, close the wing/tail in the natural anatomical position, and hold all the feathers in place with hair clips until the epoxy cures (Figures 32.2.12 and 32.2.13).





13. After the epoxy glue has cured, remove the paper or cardboard and recover the patient from anesthesia.

Adapted from Lierz M, Fischer D: Clinical technique: imping in birds, *J Exot Pet Med*, 20(2):131–137, 2011.[126]



FIGURE 32.30 Damaged blood feather in a Senegal parrot *(Poicephalus senegalus)*. In this case, excessively short-clipped primary remiges appeared to have signaled feather shaft automutilation by the bird as can be seen in the adjacent remiges. When these damaged plumes are molted and replaced, a single pin feather may extend out and beyond the others, rendering it more susceptible to mechanical trauma. These injured pin, or blood, feathers should be considered to be painful for affected birds. (Photo courtesy Bob Doneley.)

Disease Influences on Feather Structure

Noninfectious diseases

Malnutrition. Malnutrition remains a common problem in birds kept in captivity. The feeding of a predominantly seed-based diet, which is commonly high in fats and carbohydrates and low in protein, essential amino acids (particularly lysine and methionine), minerals (calcium, phosphorus, sodium, manganese, zinc, iron, iodine, selenium), and vitamins (most notably A, D, K, B_{12}, riboflavin, pantothenic acid, choline, and niacin),[127–130] can have a direct effect on feather quality, or an indirect effect through changes to liver function, skin infections and inflammation (related to hypovitaminosis A), and other systemic diseases. The normal molting process may be delayed or interrupted in birds, thereby giving rise to development of depigmented or discolored, poor-quality feathers that break easily and have a dull, ragged-looking appearance.[127,131] Changes in feather color will be discussed later. Changes in keratin, associated with essential amino acid deficiencies, may cause changes in feather structure and quality as well as cause dry, flaky skin, particularly of the face, feet, and legs. Feather structure that was adversely affected by malnutrition may be representative of an event in the past during its development and does not necessarily have to always imply ongoing malnutrition.

Although the previously mentioned abnormalities may not necessarily and specifically point to ongoing malnutrition, an underlying nutritional component should be suspected in any abnormally feathered captively maintained bird that is primarily fed seeds. Treatment in these cases should aim at correction of the malnutrition by conversion to a balanced (pelleted) diet, combined with a multivitamin injection (5000–20,000 IU/kg IM, once; based on vitamin A content) and/or oral supplements.

Lack of bathing opportunities. In the wild, most birds bathe regularly to ensure their feathers are maintained in optimal condition for flight, thermoregulation, waterproofing, camouflage, and communication. Bathing also stimulates preening, which helps to remove dirt, dander, and ectoparasites. Bathing may take place in standing water (standing-water bathing), in the rain (rain bathing), or the bird may rub its feathers against wet foliage (foliage bathing).[132–134] Alternatively, birds in environments where water is scarce may resort to dustbathing.

In captivity, restricted bathing (opportunities) can have a detrimental effect on the bird's skin and plumage. For example, in the zebra finch, restricting bathing opportunities was found to reduce plumage condition through increased lipids and dirt in the short term, while long-term bathing deprivation led to increased corticosterone,[135] which could affect the molting process and lead to immunocompromise.

It is therefore recommended to bathe birds regularly (i.e., at least weekly, but preferably daily) to keep the plumage and skin in optimal condition. More frequent bathing may be required in the winter months, especially in homes with central heating where relative humidity is low. Bathing opportunities can be provided in multiple ways:

- Wading in a birdbath, bowl or pan of water, or sink with the water running gently.
- Misting, whereby water is sprayed in such a way that the water falls gently onto the bird (note that the bird should not be sprayed directly in the face as this can be uncomfortable and frightening).
- Showering, which is preferably done on a perch with a nonslippery surface for grip that is placed off to the side (not directly under the spray).
- Rolling in wet leafy greens (e.g., lettuce leaves), which can be placed in the bottom of the cage of small birds.

Preference for one option over the other may differ according to species and per individual. When bathing a bird, care should be taken to use lukewarm to slightly cool water and to avoid drenching the bird's feathers as this could lead to significant heat loss. While some birds like a blow-dry after their bath, it is not necessary nor recommended as this may lead to drying of the skin and poses a risk of skin burns if using too high temperatures. Similarly, the use of (bird-friendly) shampoo is rarely required, unless the bird is severely soiled or greasy. For birds that are fearful of bathing, systematic desensitization and counterconditioning may need to be employed (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). If using a birdbath, pan, or bowl of water, this should be removed from the enclosure when bathing is complete, and any wet cage bedding should be removed to prevent mold growth.

Feather cysts. In species other than canaries (e.g., parrots, chickens), feather cysts most often represent an acquired condition, resulting from an infection, trauma, or other conditions that interfere with the normal feather growth (Figure 32.31). These cysts usually occur as solitary masses that develop due to curling of a new, growing feather within its follicle. While they can develop anywhere on the body, they are most found on the wings, tail, or neck. Baker described three types of cysts in budgerigars: variably sized spherical cysts containing



FIGURE 32.31 A feather cyst, as seen in a sun conure *(Aratinga solstitialis)*, may be an acquired condition or the result of an infection, trauma, or other conditions that interfere with the normal feather growth. (Photo courtesy Bob Doneley.)



FIGURE 32.33 An inability to preen, for any reason, can result in retained feather sheaths, especially in powder down feathers over the thighs, as can be seen in this cockatiel *(Nymphicus hollandicus)*. (Photo courtesy Bob Doneley.)



FIGURE 32.32 Multiple feather cysts in a budgerigar *(Melopsittacus undulatus)*, likely to be a hereditary condition. (Photo courtesy Bob Doneley.)

caseous material and the remains of one or more feathers, encased in a fibrous capsule; similar cysts but with the surrounding skin inflamed and thickened; and long, narrow cysts on the wing, often found side by side, with each cyst corresponding to the follicle of one primary feather (Figure 32.32).[89] These cysts contain caseous material like the other types but often have a malformed feather protruding from the tip of the cyst. Diagnosis and treatment are like that described for the genetic feather cysts in canaries.

Delayed molt ("stuck in molt"). Delayed molt is frequently caused by a combination of malnutrition, abnormal diurnal rhythm (especially for companion birds kept indoors and exposed to extended hours of artificial lighting), concurrent illness, and/or hormonal disorders (e.g., hypothyroidism).[90] Excessive egg laying in cockatiels and budgerigars may cause abnormal or delayed molting due to endocrine effects on feather growth. Affected birds have untidy plumage, bald spots, and damaged, brittle feathers. The ends of the feathers often become frayed and lose their reflective coloration. Other (nondermatological) signs may include decreased vocalization in male canaries, excessive or decreased egg production, and general lethargy. A thorough history and physical examination will reveal whether and which predisposing factors are involved. Treatment is primarily aimed at eliminating and correcting these conditions. Normal diurnal rhythms should be re-established by ensuring the bird gets at least 12 to 14 hours of "sleep time" in a darkened, quiet environment. Simply covering a cage in a busy family area is insufficient to meet the sleep requirements for most birds, as most of these covers do not block all light or sound. Nutritional problems must be corrected. Egg production should be controlled through nutritional, environmental, social, and possibly reproductive hormonal manipulation.

Inability to preen. Inability to preen can result in retained feather sheaths and is common in birds that have an Elizabethan collar in place to stop self-mutilation or are unable to preen due to spinal or beak problems. In cockatoos (*Cacatua* spp.), Grey parrots (*Psittacus erithacus*), and cockatiels, retained sheaths are especially seen in the powder down feathers over the thighs, which can become very elongated and friable, thereby breaking easily (Figure 32.33).

Chronic liver disease. Chronic liver disease can cause feather changes (Figure 32.34). The hyperbiliverdinemia associated with liver disease will cause keratinocytes to excrete biliverdin, resulting in yellowing of feathers, particularly in lutino cockatiels.[136] Birds with chronic hepatopathies may show feather structural changes including loss of barb and barbule interlocking, giving the feathers



FIGURE 32.34 Chronic liver disease can cause feather and beak changes, especially in older birds. It needs to be distinguished from psittacine beak and feather disease and possibly hypothyroidism. This galah *(Eolophus roseicapilla)* shows darkening of the plumage and loss of the barbule structure. Without diagnostic testing, the cause cannot be elucidated.



FIGURE 32.35 Polyfolliculosis/polyfolliculitis, as can be seen in this peach-faced lovebird *(Agapornis roseicapilla)*, is a chronic pruritic condition that can cause multiple short feathers to erupt from one follicle. There is some discussion as to whether this condition is the cause of the bird's intense pruritus or is the result of damage done by the bird in response to the pruritus. (Photo courtesy Bob Doneley.)

more of a hair-like appearance. Feathering in birds with chronic liver disease is often dull and the plumage unkempt. Delayed molt or failure to molt is not uncommon with chronic disease of many forms, not specific to liver disease. There may be a degree of pruritus leading to feather damage and loss, particularly over the torso. Nails and beaks are often overgrown, possibly due to derangements in carbohydrate and protein metabolism.

Diagnostic tests that can be performed to confirm a suspicion of liver disease can include serum/plasma biochemistry (including aspartate aminotransferase, creatine kinase, γ-glutamyl transferase, glutamate dehydrogenase, lactate dehydrogenase, alkaline phosphatase, and bile acids), imaging (e.g., radiography, ultrasound), endoscopy, and/or collection of (ultrasound-guided or endoscopic) fine needle aspirates or liver biopsies (see Chapter 2 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 13, Advances in Clinical Pathology and Diagnostic Medicine and Chapter 14, Advances in Diagnostic Imaging). Treatment should aim at supporting the patient, treating the specific condition, and creating an environment that allows the liver to heal and regenerate.[137,138]

Polyfolliculosis/polyfolliculitis. Polyfolliculosis or polyfolliculitis refers to a chronic pruritic condition that can cause multiple short feathers to erupt from one follicle (Figure 32.35). It is most encountered in lovebirds and budgerigars.[139] While the etiology is still currently unclear, some veterinarians have hypothesized a viral or genetic origin. There is some discussion as to whether this condition is the cause of the bird's intense pruritus or is the result of damage done by the bird in response to the pruritus. Affected birds are presented for pruritus predominantly over the neck, back, and tail. Diagnosis can be made based on the clinical appearance of the feather follicles, which typically reveal several feathers with short, stout quills and retained sheaths emerging from a single, enlarged follicle. Histologically, there is the appearance of multiple short feather shafts growing from the same follicle, with a thin layer of epidermis separating the shafts. Chronic inflammation occurs beneath the pulp cap and the feather sheath is thickened. Surgical excision or electrocautery of affected follicles or feather tracts may be of benefit. Nonsteroidal antiinflammatory drugs such as meloxicam, as well as other analgesics, may also be helpful to alleviate associated pain.[136] Collars may be considered in birds that mutilate their skin.

Hypothyroidism. Hypothyroidism has been documented in poultry with immune-mediated thyroiditis demonstrating changes in feather quality and color. Black, brown, and yellow feathers become red, longer, and more pointed and have fewer pennaceous barbules than normal. These changes are reversible with thyroid supplementation.[140]

An obese scarlet macaw *(Ara macao)* with confirmed hypothyroidism had nonpruritic feather loss, mild nonregenerative anemia, mild leukocytosis, heterophilia, hypercholesterolemia, and sparse feathers, and had not molted in over a year. It responded well to thyroxine therapy.[141] Hypothyroidism has also been suspected in eclectus parrots *(Eclectus roratus)*, galahs *(Eolophus roseicapilla)*, lovebirds, budgerigars, and cockatiels with plumage changes including elongation of powder down feathers (in cockatoos and cockatiels), narrowing and elongation of outer primary feathers, and generalized feather color changes (Figures 32.36and 32.37).[142] Galahs develop a pink-red discoloration of the gray plumage and the normally pink feathers deepen in color intensity. Lutino cockatiels with suspected hypothyroidism may show deepening color intensity and a loss of barbs and barbules in the feathers, giving the



FIGURE 32.36 Hypothyroidism is suspected in galahs *(Eolophus roseicapilla)*, budgerigars *(Melopsittacus undulatus)*, and cockatiels *(Nymphicus hollandicus)* with plumage changes including elongation of powder down feathers, narrowing and elongation of outer primary feathers, and generalized feather color changes. This cockatiel shows the darkened, often "greasy," appearance of the feathers and slight beak overgrowth. (Photo courtesy Bob Doneley.)

bird a "greasy" deep yellow color. Hypothyroidism in parrots nevertheless tends to be much overdiagnosed (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 10, Clinical Endocrinology of the Protein Hormones). Diagnosis is difficult because normal resting thyroxine (T4) levels in birds (5–15 ng/mL) are much lower than in mammals and are often below the detectable limits of many laboratory techniques and equipment. Additionally, Triiodothyronine (T3; 0.5–4 ng/mL) may be as important as T4 in overall thyroid function, therefore measuring only T4 may not present an accurate picture of the health of the thyroid gland. The half-life of both hormones is shorter in birds than in mammals, resulting in a significant diurnal variation in a normal bird. There is also a seasonal variation, with higher baseline levels in spring. Diagnosis of hypothyroidism therefore requires demonstration of a failure to respond to thyroid-stimulating hormone (TSH). As avian TSH is not commercially available, mammalian TSH must be used. After obtaining a baseline T4, the bird is given 1 IU/kg of TSH and T4 is determined 4 to 6 hours later. A positive response in a normal bird will result in a 2.5-fold or greater increase in the resting T4 level.[143]

Supporting evidence for a diagnosis of hypothyroidism includes elevated cholesterol, triglycerides, liver enzymes, and a mild nonregenerative anemia. It has been suggested that, in

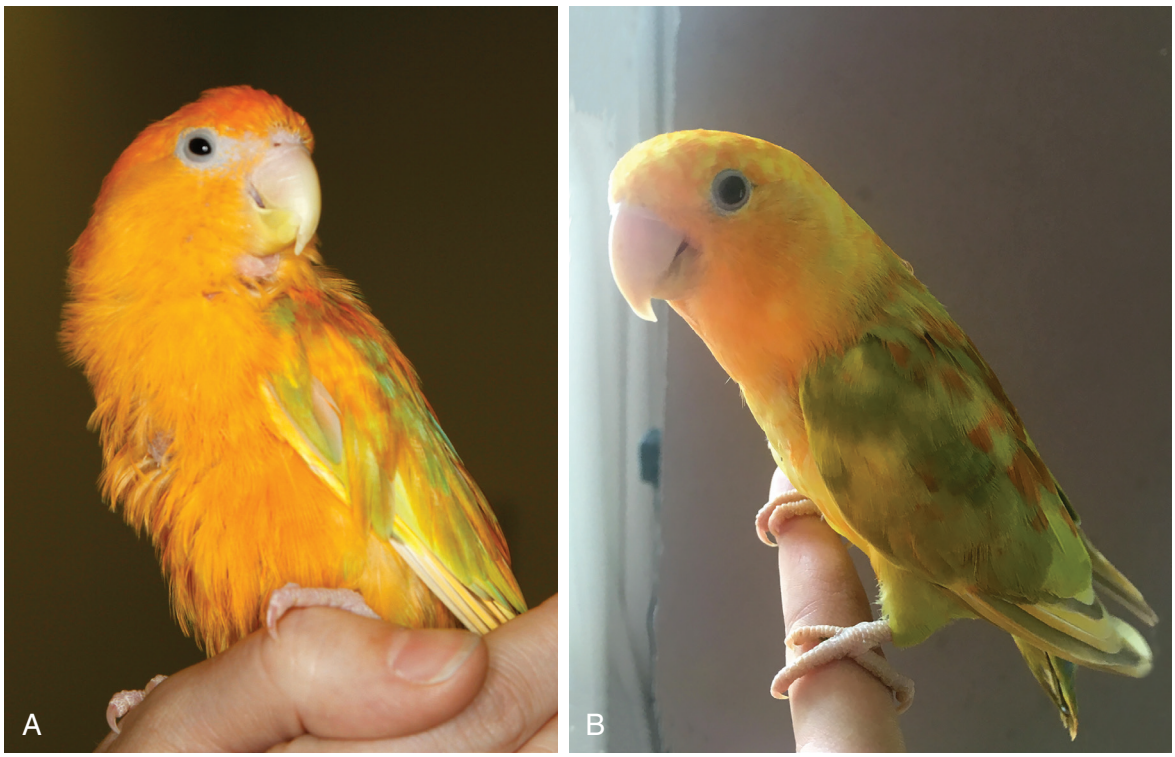


FIGURE 32.37 Peach-faced lovebird *(Agapornis roseicollis)* with suspected hypothyroidism. (A) The bird was presented with obesity, decreased activity, and progressive color changes of the feathers. (B) Following supplementation with L-thyroxine the bird lost weight, became more active, and the plumage gradually returned to normal coloration over a course of several months, as can be seen in these two serial images. (Photo courtesy Yvonne van Zeeland/Nico Schoemaker.)

larger birds, endoscopic biopsy of the thyroid gland may lead to a diagnosis of hypothyroidism.[142]

Despite the lack of laboratory confirmation, some obese birds that demonstrate a lack of weight loss following a rigid diet, accompanied by poor-quality feathers and infrequent molts, respond favorably to thyroxine therapy. It must be noted that no pharmacologic data exist to confirm the proper administration of thyroid hormone replacement in birds and that response to thyroid hormone supplementation does not confirm the diagnosis (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 10, Clinical Endocrinology of the Protein Hormones).

Toxic effects of drugs. Certain drugs can cause weakened, twisted feathers if administered during molting. The best-known example of this is fenbendazole. Silver sulfadiazine, used as a topical antibacterial cream, and colloidal silver have been reported to cause agyria (e.g., silver poisoning) after excessively prolonged application or administration. It causes a bluish discoloration of the skin, feathers, and oral mucosa.[143]

Pinching off syndrome

Pinching off syndrome (POS) refers to a condition in which all remiges and rectrices become malformed and are lost during the nestling stage, thereby rendering the bird unable to fly.[144] Contour feathers can sometimes be more or less malformed as well. The syndrome was first reported in 1975 in a juvenile white-tailed sea eagle *(Haliaeetus albicilla)*,[145] but has since been described in several species of birds of prey in both Europe and North America.[146–150] In birds with POS that were temporarily held captive, feather abnormalities worsened, with new flight feathers becoming progressively shorter with each new feather generation. Though various (viral, parasitic, genetic, endocrine) causes have been postulated, researchers have thus far failed to determine the underlying etiology for this condition.

Infectious diseases

Psittacine beak and feather disease. Psittacine beak and feather disease (PBFD) is caused by a circovirus (PBFDV), a nonenveloped single-strand DNA virus measuring 14 to 17 nm. The virus has a minimum incubation period of 21 to 25 days, but incubation periods could be up to several years. It is shed in feces, crop secretions, and feather dust. Vertical transmission is suspected but not confirmed. Following infection, the virus is disseminated hematogenously, residing in the feather follicle epithelium, the thymus, and the bursa. In cockatoos, it also resides in the growth areas of the beak (Figure 32.38). The result is feather dystrophy, immunosuppression, and, in cockatoos, overgrowth and underrunning of the rhamphotheca. All parrots are susceptible, but New World parrots and cockatiels appear to be rarely affected. PBFD is more common in juveniles than in adults, but naive adults are susceptible. There are two forms of PBFD: acute and chronic (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).

Acute PBFD is seen in juveniles (especially Grey parrots) around weaning age. Following infection, they become lethargic, fluffed, and anorexic. Hematology may show pancytopenia and nonregenerative anemia. Affected birds often die (within a few days) with severe hepatic necrosis before feather abnormalities develop. However, feather lesions have been noted in fledgling birds at 28 to 32 days old.[151]



FIGURE 32.38 Psittacine beak and feather disease (PBFD) in two wild-caught sulphur-crested cockatoos *(Cacatua galerita galerita)* showing the classical presentation of overgrown beaks and lost/dystrophic feathers. (Photo courtesy Bob Doneley.)

Chronic PBFD causes progressive replacement of normal feathers with dystrophic feathers, which can present as retained sheaths and blood supply, clubbed appearance, stress lines, constrictions, and abnormal shapes. The degree and location of the feather loss may depend on the state of molt that the bird was in when it was initially infected. Typically, lesions develop first in the powder down feathers, followed by the contour feathers, primaries, secondaries, tail, and then the crest. *Poicephalus* species and lorikeets may only lose tail feathers and primary flight feathers (Figure 32.39). These feathers may then regrow. Neophemas (*Neophema*



FIGURE 32.39 Psittacine beak and feather disease (PBFD) in a juvenile wild-caught rainbow lorikeet *(Trichoglossus moluccanus)* showing the loss of primary flight feathers on the wings. (Photo courtesy Bob Doneley.)

spp.) may develop untidy plumage and will easily lose feathers when handled. Many parrots develop feather color changes, for example, blue feathers become white, green feathers become yellow. Beak lesions, which include palatine necrosis, ulceration, elongation, and fracturing of the beak, are seen only in cockatoos. Immunosuppression is common in all species.

A presumptive diagnosis can usually be made based on the characteristic clinical signs, with further testing confirming the suspicion. Testing for evidence of infection with PBFDV can be done via polymerase chain reaction (PCR), serology, or histopathology (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease.

Since no treatment exists for PBFD, affected birds may be euthanized once the disease progresses. Some birds (especially cockatoos) may live for 10 to 30 years despite showing clinical signs, but most infected birds die within 2 years due to developing secondary diseases related to immunosuppression. Lorikeets and lories appear to mount an effective immune response and recover to look clinically normal. However, many of these birds become unapparent carriers.[90,152]

In Australia, where the disease is endemic, experimental inactivated vaccines are available, but these do not prevent infection (see Chapter 13 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).[153] Prevention therefore primarily aims at reducing the spread of the disease by maintaining closed flocks, quarantine measures, and (repeated) testing. Proper hygiene and disinfection protocols are also recommended to reduce the risk of transmission (see Chapters 11 and 12).

Circovirus infection in other birds. While primarily recorded in psittacine birds, circovirus infections have been suspected or confirmed in several nonpsittacine birds (Table 32.1).[154] Similar to circovirus infection in psittacine birds, disease is

TABLE 32.1

Circovirus Infections in Nonpsittacine Birds

Taxonomic Order	Species	Type of Circovirus	Clinical Features	Reference
Anseriformes	Pekin *(Anas platyrhynchos domestica)* and mulard ducks (sterile hybrid between a Muscovy duck, *Cairina moschata*, and Pekin duck)	Duck circovirus (DuCV)	Growth retardation, poor body condition, feather dystrophy (hemorrhage of the shaft), particularly in the dorsal region, increased mortality in young ducklings related to immunosuppression	Atterman et al., 2003[155]; Soike et al., 2004[156]; Hong et al., 2018[157]
	Goose *(Anser anser)*	Goose circovirus (GoCV)	Runting syndrome associated with a feathering disorder and increased mortality in young geese	Soike et al., 1999[158]; Ball et al., 2004[159]; Chen et al., 2006[160]
	Mute swan *(Cygnus olor)*	Swan circovirus (SwCV)	Wild swans, found dead. No further clinical presentation available	Halami et al., 2008[161]
Charadriiformes	Southern black-backed gull *(Larus dominicanus)*, herring gull *(Larus argentatus)*, black-headed gull *(Larus ridibundus)*	Gull circovirus (GuCV)	(Juvenile) birds that were found dead, with secondary infections (aspergillosis) suggestive of immunosuppression	Twentyman et al., 1999[162]; Smyth et al., 2006[163]
Columbiformes	Racing pigeon *(Columba livia)*	Columbid circovirus (CoCV)	Immunosuppression with associated increases in morbidity and mortality in young pigeons. Feather abnormalities are relatively rare but may include thickening of the feather pulp, quill congestion, pinching off, vane discoloration, feather deformation, feather growth retardation, and (symmetrical) loss of feathers	Woods et al., 1993[164]; Woods et al., 1994[165]; Gough and Drury, 1996[166]; Smyth and Carroll, 1995[167]; St. Leger et al., 1997[168]
	Senegal dove *(Streptopelia senegalensis)*	Columbid circovirus (CoCV)	Bilaterally symmetrical feather loss and dystrophy	Pass et al., 1994[169]; Raidal and Riddoch, 1997[170]
Coraciiformes	Rainbow bee-eater *(Merops ornatus)*	95.6% similarity to PBFDV	Dystrophic flight feathers with thickened feather shafts, broken remiges, bilaterally symmetric feather loss of primary feathers	Sarker et al., 2015[171]
Galliformes	Common pheasant *(Phasianus colchicus)*	Unknown	Circovirus-like particles found in a commercial pheasant flock, no clinical information retrieved	Terregino et al., 2001[172]

Continued

TABLE 32.1

Circovirus Infections in Nonpsittacine Birds—cont'd

Taxonomic Order	Species	Type of Circovirus	Clinical Features	Reference
Passeriformes	Canary *(Serinus canaria)*	Canary circovirus (CaCV)	"Black spot" of neonatal canaries with coelomic distension, gallbladder congestion, failure to thrive and mortality (<7 days). Grossly normal feathering though microscopic changes have been identified	Goldsmith, 1995[173]; Phenix et al., 2001[174]; Todd et al., 2001[175]; Sheykhi et al., 2018[176]
	Gouldian finch *(Chloebia gouldiae)*	Finch circovirus (FiCV)	Progressive beak deformities (elongated beaks with rough surface), feather loss, mortality of young chicks showing weakness, lethargy and ruffling of the feathers	Shivaprasad et al., 2005[177]; Todd et al., 2007[178]; Circella et al., 2014[179]
	Zebra finch *(Poephila castanotis)*	Zebra finch circovirus (ZfiCV)	Mild to severe disease with high mortality caused by secondary infections (commonly *Macrorhabdus ornithogaster*) in nestlings, fledglings, and adult birds. Feathering abnormalities and feather loss can also be observed	Mysore et al., 1995[180]; Rinder et al., 2015[181]; Rinder et al., 2017[182]
	Common starling *(Sturnus vulgaris)*, and spotless starling *(Sturnus unicolor)*	Starling circovirus (StCV)	Detected in apparently healthy and diseased starlings during a mortality outbreak	Johne et al., 2006[183]
	Australian raven *(Corvus coronoides)*	Raven circovirus (RaCV)	Progressive feather defects in juveniles, bilateral feather depigmentation (white feathers) and dystrophy	Stewart et al., 2006[184]
Sphenisciformes	Adélie penguin (*Pygoscelis adeliae*)	Penguin circovirus (PenCV)	Feather loss in juvenile birds	Morandini et al., 2019[185]; Levi et al., 2020[186]
	Chinstrap penguin *(Pygoscelis antarcticus)*	Penguin circovirus (PenCV)	Feather loss in juvenile birds	Levi et al., 2020[186]
Strigiformes	Powerful owl *(Ninox strenua)*, tawny owl *(Strix aluco)*	Spillover BFDV from lorikeets? Tawny owl-associated circovirus (ToCV)	Feather lesions similar to PBFD, possible spillover from BFDV in lorikeets (prey species for the powerful owl); detected in the spleen of a tawny owl during routine evaluation	Sarker et al., 2015[187]; Legnardi et al., 2022[188]
Struthioniformes	Ostrich *(Struthio camelus)*	Circovirus detected on PCR in various tissues from young chicks, but not on histopathology; circovirus-like particles observed in the gut contents by negative contrast electron microscopy, but unknown whether this was of plant or animal origin	Early to late embryonic death, increased mortality and wasting in chicks; circovirus particles were also discovered in the gut contents of an ostrich; clinical presentation of this bird is otherwise unknown	Els and Josling, 1998[189]; Eisenberg et al., 2003[190]
Suliformes	New Zealand king shag *(Leucocarbo carunculatus)*	Suspected viral infection, but not confirmed	Juvenile bird with feather dystrophy, consisting of swollen and flaky calamus of outer four primaries, as well as some primaries that were missing or broken near the base	Melville and Schuckard, 2021[191]

PBFDV, Psittacine beak and feather disease virus; *PCR*, polymerase chain reaction.

observed mostly in juvenile birds, leading to immunosuppression and susceptibility to secondary infections. Upon histopathology, characteristic basophilic inclusion bodies can be found in various organs and tissues (e.g., bursa of Fabricius, liver, spleen). Feather dystrophy and (symmetrical) feather loss, if present, resemble the abnormalities as have been described for psittacine birds.

Avian polyomavirus. Avian polyomavirus (APV) is a nonenveloped virus that is relatively environmentally stable. While all species of parrots can be affected, disease is most common in macaws (*Ara* spp.), conures (*Aratinga* spp.), eclectus parrots, and caiques (*Pionites* spp.). In contrast, disease is rare in Grey parrots, cockatoos, and cockatiels. Passeriformes, including canaries, grass finches, goldfinches, and greenfinches, can also be infected (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).

In affected budgerigars, which are the primary reservoir of polyomavirus, signs include decreased hatchability and embryonic death, abdominal distension, subcutaneous hemorrhages, and feather dysplasia that may resolve after several months (see Chapter 41). Neurological signs may be seen if the cerebellum is affected. Over the course of the disease, primary and secondary feathers on the wings and tail are lost or fail to erupt. In the budgerigar fancy, birds with this presentation are referred to as runners or creepers, and the condition is known as French molt (Figure 32.40).

In other species, the virus typically causes sudden death (especially in chicks <15 days old) at around 10 to 14 days after exposure. In larger parrots, most deaths occur between 20 and 140 days old.

In estrildid finches (grass finches), the virus causes acute mortality in 2- to 3-day-old fledglings, young adults, and mature finches. Survivors may demonstrate poor feather development and misshapen lower beaks.[143]

A presumptive diagnosis is usually based on the history, clinical findings, and pathological features. Definite diagnosis requires serology or PCR testing of cloacal swabs, blood, or tissues. However, due to the short viremia and irregular shedding, many false-negative results are obtained. Definite diagnosis may therefore sometimes require histopathologic examination of tissue samples which identifies the presence of basophilic intranuclear inclusion bodies in various tissues (e.g., liver, spleen, kidney, and feather follicles).[192] No specific treatment for polyomavirus infection currently exists. A commercially available polyomavirus vaccine is available in the United States to protect nestlings of large parrots in aviaries. To prevent the spread of the disease, hygiene and quarantine measures like those discussed for PBFD are recommended. PCR testing will help to identify viremic and/or shedding birds. Temporary cessation of breeding in flocks of affected budgerigars can also help to reduce horizontal and vertical transmission of the disease (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).

Bacterial infections. Folliculitis (inflammation of the feather follicles) can develop in response to invasion with (secondary) bacterial pathogens. Bacteria that have been implicated in folliculitis include *Staphylococcus* spp., *Aeromonas* spp., and *Mycobacterium* spp.[193,194] Birds with folliculitis typically present with perifollicular swelling and erythema. Often, multiple feathers are affected. Birds may furthermore show signs of pruritus and/or pain, which may be expressed by restlessness, shivering, and/or feather-damaging behavior (FDB). If the bacterial organisms invade the skin, signs of dermatitis can also be present (Figure 32.41). To identify which bacteria are involved, an affected feather may be pulled and aseptically opened, after which the pulp may be cytologically examined using specific stains (e.g., Gram stain) and/or cultured. Alternatively, histologic examination of feather follicle biopsies may be considered. Choice of antibiotics is preferably based on results of a culture and sensitivity testing.

Fungal infections. Fungal infections affecting the feathers are uncommon but can be seen in all avian species. Fungal species involved include *Candida albicans*, *Malassezia pachydermatis*, dermatophytes (*Microsporum gallinae*, *M. gypseum*, *Trichophyton verrucosum*, other *Trichophyton* spp.), *Cryptococcus*



FIGURE 32.40 Avian polyomavirus infection in a juvenile budgerigar *(Melopsittacus undulatus)*. Known as "French molt" this condition causes the loss of remiges and rectrices, rendering the bird unable to fly (they are known as "runners" or "creepers").

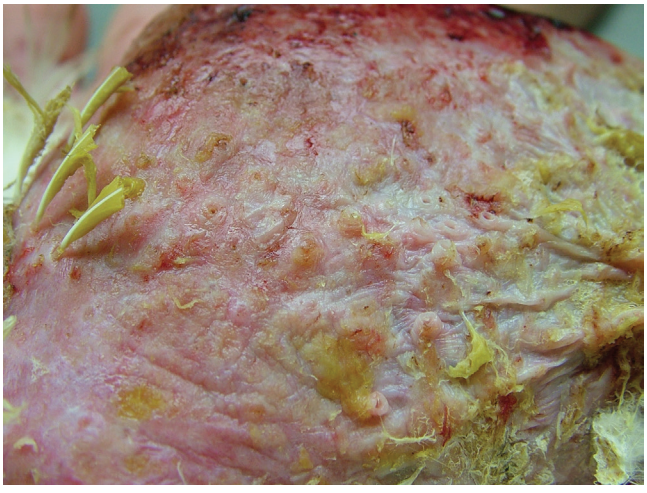


FIGURE 32.41 Bacterial dermatitis developing following a dog bite in a galah *(Eolophus roseicapilla)*. (Photo courtesy Bob Doneley.)

bacillisporus (formerly *C. neoformans* var. *gattii*) and *C. neoformans* var. *grubii* (formerly *C. neoformans* var. *neoformans* serotype A), and *Aspergillus* spp.[90]

Candida causes lesions around the commissures of the mouth and nares and occasionally around feather follicles on the head, back, and ventral abdomen. *Malassezia* results in generalized pruritus, sometimes with crusting and folliculitis.[90] Dermatophytes are a slow-spreading infection causing scabs, crusts, and alopecia on the body and thin-skinned areas of the head and upper beak, and a rough, porous appearance of the podotheca. In gallinaceous birds the characteristic scaly, crusty lesions of the wattle, comb, and legs are known as "favus" (Figure 32.42).[90] *Cryptococcus* has been reported to cause subcutaneous nodules on the head and body and raised nodular lesions on the face and beak (see Chapter 21).[90] Cutaneous aspergillosis has been associated with focal ulcerative pruritic lesions.[90]

Diagnosis generally requires biopsy and culture. Cytology must be used with caution: feather dust stains similarly to yeast organisms and can be nearly impossible to distinguish from yeast bodies. In affected birds, a systemic treatment with antifungal drugs such as voriconazole, itraconazole, fluconazole, clotrimazole, and terbinafine is often initiated, though topical therapy (e.g., miconazole) may be considered if lesions are localized. Depending on the pathogen involved, treatment periods of 3 weeks or more are required to clear the infection.

External parasites. Ectoparasites are commonly found on wild and aviary birds, but somewhat less common on companion birds. These parasites may inhabit the skin and/or feathers and are usually detected upon microscopic examination of the pulp and/or vane of a damaged or developing feather.

Mites are the most important and commonly encountered external parasites of birds. Adult mites have eight legs; the larval stage has six legs.[143]



FIGURE 32.42 Ringworm (or favus) in a chicken *(Gallus gallus domesticus)* showing feather loss and characteristic skin changes that give the appearance that flour is sprinkled over the bird. Ringworm or dermatophytosis in chickens is usually caused by the dermatophytic fungus *Trichophyton megninii*, although infections with *Trichophyton simii, Microsporum gypseum,* and *Microsporum gallinae* have also been reported. (Photo courtesy Bob Doneley.)

Knemidocoptes pilae (scaly face mite). Many species of mites cause mange in birds. The most common are *K. mutans* of poultry[195,196] and *K. pilae* in budgerigars,[197,198] other psittacines[199–202] and canaries.[91,203] The mites spend their entire life on the bird. The lesions can occur on any featherless part of the skin and are characterized by raised honeycombed encrustations on the cere, beak, eyelids, and feet (see Chapter 31). Occasionally it becomes generalized and affects feather follicles.[143] If left untreated for a longer time, severe and permanent beak deformities can occur, which could interfere with eating and may warrant regular trimming.[204] Occurrence of beak keratoacanthomas has also been associated with former *Knemidocoptes* infection.[205] Diagnosis can readily be made by means of cytology of superficial and deep skin scrapings of affected areas. Treatment consists of use of avermectins (e.g., ivermectin, selamectin), which generally results in clinical resolution of signs and negative scrapings within 2 to 4 weeks.[198,206,207] Fipronil is also effective against scaly mites.[207] In chickens, topical application of petrolatum products is commonly practiced by owners.[208]

Dermanyssus gallinae (red or roost mites). Red mites *(Dermanyssus gallinae)* live in the environment where they can survive long periods without feeding. The mite feeds on the host's blood at night and leaves it in the day to hide in cracks and crevices of the enclosure. The mites can cause skin irritation, scaliness, and anemia (especially in juveniles or small birds). Heavy infestations may kill chicks in the nest box. Because the mite is not permanently present on its host, it can be difficult to confirm the diagnosis. Elimination of the mites usually requires treatment of the host, contact birds, and environment with synthetic acaricides that have activity against *D. gallinae* (e.g., fluralaner, phoxim[209–211]; see Chapter 9). Other measures for control include the use of predatory mites (mainly in subclinical, mild infestations),[212] silica dusts, and changing the enclosure design such that hiding places for the mites are minimized by using slatted floors and laying nest floors with more open structures.[213–215]

Ornithonyssus spp. (fowl mite). Fowl mites (*Ornithonyssus* spp.) are host-dependent blood-sucking mites which remain on the bird, where they congregate at the eyelids and vent. They may cause anemia, pruritus, and damaged plumage. In contrast to red mites, fowl mites are easily observed on the bird. Over-the-counter insecticides, which are available in both powder and spray application, are effective to eliminate the parasites. Oral or injectable (200 μg/kg) ivermectin can also be used. Recently, fluralaner (Exzolt, MSD Animal Health, Macquarie Park, Australia) was registered as an in-water medication for poultry for ectoparasite treatment.

Epidermoptic mites. The group of epidermoptic mites includes *Myialges* spp., *Epidermotes* spp., and *Microlichus* spp. The hippoboscid fly may carry them mechanically. They cause depluming dermatitis with scale formation in passerines.[89,90] Diagnosis is based on the skin lesions, associated with the presence of mites. Treatment is like that of other mites (see Chapter 9).

Quill mites. The group of quill mites includes *Syringophilus* spp., *Dermoglyphus* spp., *Pterolichus* spp., *Analges* spp., and *Harporhynchus* spp. Their entire life cycle is spent on the host. They cause partial or complete feather loss. Quill mites predominantly live and reproduce inside the feather quills where they feed on the available tissue and secretions. When present

in large numbers, they may cause damage to the feathers by destroying the medulla from the quill to the rachis. Affected quills tend to break or bleed easily and may change in appearance from transparent to opaque. Although most quill mites primarily feed on the keratin and medulla of the feather quills, quill wall mites (e.g., *Syringophilus* spp.) primarily feed on feather follicle fluid and outer, nonkeratinized layers of the feather germ of developing feathers, thereby triggering hyperkeratosis of the feather sheath. They can be found by examining the pulp material within a developing or damaged feather.[9,10] Treatment consists of use of topical or parenteral avermectins (e.g., ivermectin, selamectin, moxidectin) and/or other topical acaricides (e.g., permethrin, pyrethrin, fipronil; see Chapter 9).[216] Alternatively, fluralaner, which was recently registered for use in poultry in several countries, can be added to the drinking water.

Feather mites. Feather mites (e.g., *Protolichus*, *Dubininia* spp.) live between the barbs of the contour, wing, and tail feathers and primarily feed on the feather fragments and skin debris (Figure 32.43). Feather mites are generally considered nonpathogenic but may cause problems when present in high numbers, in non-host-adapted species, or in birds with a compromised immune system. The mites, which may be identified as tiny dark spots of grains of sand on the ventral surface of feathers, may cause irritation (leading to FDB) and induce feather lesions such as darkened areas, dots or stripes on bright feathers, or bright dots and stripes on dark feathers. Diagnosis and treatment are like that described for quill mites.

Lice. Lice are six-legged, wingless, crawling external parasites with chewing mouthparts (note: sucking lice do not infect birds). They are species-specific and usually cause little feather damage.[90] There are many hundreds of species of lice found on birds, including the following:

- Biting lice of parrots include *Neopsittaconirmus*, *Psittaconirmus*, *Eomenopon*, and *Pacifimenopon*.
- Biting lice of pigeons include the Philopteridae and Menoponidae families.
- Biting lice of raptors include *Mallophaga* spp.
- Biting lice of chickens include Menoponidae lice (*Menacanthus* spp., *Menopon gallinae*), *Oxylipeurus* spp., *Goniodes* spp., *Cuclotogaster* spp., and *Lipeurus* spp.

Lice usually live on just the one host. Eggs are attached to feather shafts and hatch in 4 to 7 days. Nymphs go through three molts before maturing. The adults feed on skin scales, feather debris, and hair, and, in large numbers, may cause pruritus and poor feather quality. Diagnosis is made by direct visualization of the lice, nymphs, or eggs. As lice undergo a complete life cycle on the host, weekly treatments with topical acaricides (e.g., permethrin, fipronil), or fluralaner in the drinking water, usually suffice to eliminate the infection.



FIGURE 32.43 Unknown species of lice on the feathers of a budgerigar *(Melopsittacus undulatus)*. These are common and rarely cause problems. (Photo courtesy Bob Doneley.)

CHANGES IN FEATHER COLOR

Color in birds is the result of a combination of feather structure (affecting the passage or reflection of light) and a variety of pigments—melanins, carotenoids, and porphyrins (see section on Feather Color). The colors that these pigments produce are modified by how they are laid down in the feather; the presence, absence, and interactions between each other; and the air cells in the feather (affecting the passage of light). Anything that affects these combinations can change the color of the feathers (see also Box 32.3; Figure 32.44).

Genetically Acquired Color Mutations

Genetically acquired color mutations are often highly sought after by aviculturists and are the result of selective breeding. Most of these mutations are obvious at fledging, but some develop in intensity as the birds go through successive molts. The bird's genetic makeup may affect the deposition of feather pigments directly or indirectly (e.g., through the production of enzymes that alter dietary carotenoids).[218]

Molting

Molting may affect feather coloration. Hormonal influences (e.g., sex hormones, thyroxine) can induce a molt at the beginning and end of the breeding season and, in some species, the coloration of the birds may change dramatically just prior to the breeding season (the prenuptial molt)—an important social signal to other birds. Molting is, however, an expensive exercise requiring a lot of energy and resources. Consequently, not all avian species will show dramatic changes in coloration.

Black Marks

Black marks are commonly seen in green- or blue-colored companion birds that are given a lot of attention, involving physical and hand contact (Figure 32.45). The black marks are due to the owner's hand oils, passed on when cuddling the bird, and may be due to either a harmless fungal growth or, again, a change in reflected light. They are not seen on birds that produce powder down (cockatoos and Grey parrots).

Black/Brown Scalloping

Black or brown scalloping on the margins of feathers is often associated with a delayed or imminent molt. The ends of the vane become worn and tattered and lose their structure, that is, the barbules are no longer interlocking. This changes reflective color and gives a black or brown scalloping effect (Figure 32.46).

BOX 32.3 NOMENCLATURE FOR (GENETIC) COLOR ABNORMALITIES IN BIRDS[217]

Color Abnormality	Synonym(s)	Morphologic Appearance	Cause	Consequences
Total amelanism	(Total, complete, perfect, pure, or true) albinism, complete albinism, all white.	Absence of all melanin from the plumage, eyes, and skin, which results in an all-white plumage, pink eyes, and light, often pink, skin if other pigments are also lacking; if present, other pigments remain unaffected.	Genetic mutation blocking the production or activity of tyrosinase, a key enzyme in the synthesis of melanin pigment. Usually a recessive trait, which can both be autosomal and sex-linked.	Absence of melanin leads to light-sensitivity and reduced visual acuity, which can affect food-locating capacity and increase the risk of bird falling victim to predators, traffic or other hazards; may also affect attractiveness to potential mates, and reduce fitness as feathers are less resilient in absence of melanin.
Partial amelanism	(Partial, semi- or incomplete) albinism, leucism, schizochroism, piebaldism, white spotting, abnormal white, and Pealea phenomenon; individuals partial amelanism have been dubbed pied, splashed, or variegated.	Abnormal absence of all melanin from parts of the plumage, skin, eyes, or all three areas; expression can vary from a single white spot on a feather to an all-white plumage. Melanin usually remains to a certain extent in the eyes, aiding in distinction from total amelanism.	Diminished tyrosinase activity, resulting from both hereditary (genetic) and nonhereditary (environmental) factors. Hereditary forms are nonreversible and can be recessive or dominant, sex-linked or autosomal. Nonhereditary forms are reversible and can result from, e.g., malnutrition, intoxications, injury, (systemic) diseases, parasitic infections, hormonal influences (e.g., testosterone), shock, and old age.	Mostly similar effects to total amelanism except for reduced visual acuity as melanin pigment is often maintained in the eye.
Hypomelanism (total or partial)	Leucism, (partial, imperfect, dilute) albinism, dilution, dilutism, ghosting, paling, isabellism/isabellinism, chlorism/chlorochroism; individuals with the condition are referred to as faded, frosted, or bleached.	Abnormal reduction of melanin concentration from the plumage, skin, eyes, or all three areas, resulting in a faded appearance. Extent of pigment dilution is variable, ranging from slightly pale to nearly white plumage; distinct patterns (e.g., tail bands, eye lines) are usually retained.	Can be hereditary (recessive or dominant) or nonhereditary; one or both types of melanin can be affected.	Effects on survival and reproduction are likely to vary dependent on the species involved and the type and extent of the condition.
Aeumelanism	Schizochroism, schizochromatism, schizomelanism, imperfect albinism, and isabellism; individuals with this condition are referred to as buff, fawn, or cinnamon.	Abnormal absence of eumelanin from the plumage, skin, eyes, or all three areas, resulting in a pale plumage in which the melanic areas are usually some shade of fawn, brown, or rufous. Other nonmelanin pigments are unaffected.	Inherited as a sex-linked, recessive trait, thereby more common in heterozygous females.	Effects on reproduction and survival in the wild are likely to vary, but no deleterious effects proven.

BOX 32.3 NOMENCLATURE FOR (GENETIC) COLOR ABNORMALITIES IN BIRDS—cont'd

Color Abnormality	Synonym(s)	Morphologic Appearance	Cause	Consequences
Aphaeomelanism	Schizochroism, schizochromatism, schizomelanism, imperfect albinism, and isabellism; individuals with this condition are referred to as gray or silver.	Abnormal absence of phaeomelanin from the plumage, skin, eyes, or all three areas, resulting in a pale plumage in which the melanic areas are usually some shade of black or gray, often ash-gray. Other nonmelanin pigments are unaffected.	Inherited as an autosomal recessive trait with equal distribution among males and females.	Effects on reproduction and survival in the wild are likely to vary, but no deleterious effects proven.
Hypermelanism	Melanism, nigrism.	Abnormally high melanin concentration in the plumage, skin, eyes, or all three areas, resulting in a (fully or partially) darker than normal plumage.	Increase in concentration of one melanin type (hypereumelanism or hyperphaeomelanism). Both hereditary (usually autosomal, recessive, or dominant) and nonhereditary forms exist.	Effects on reproduction and survival in the wild are likely to vary, with nonhereditary forms (e.g., due to malnutrition) likely associated with reduced fitness. Certain hereditary forms could be advantageous (e.g., black morphs are less prone to bacterial degradation of feathers).
Carotenism (Figure 32.44)	Xanthism, xanthochroism, flavism, erythrism, cyanism, (partial) albinism; individuals with carotenism are referred to as lutinos.	Abnormal carotenoid pigmentation; occurs only in species that normally display carotenoids in their plumage or unfeathered areas.	Can result from one or more of four causes: (1) change in the normal distribution or extent of carotenoid pigments; (2) increase or decrease in carotenoid concentration, resulting in a change in color or color intensity; (3) change in carotenoid pigment type and therefore a change in color; and (4) total absence of carotenoids from all or part of the plumage or skin. Often results from dietary factors during or just prior to molt, but can also result from a genetic mutation disrupting the carotenoid metabolism.	Effects on survival and reproduction are likely to vary.

Malnutrition

Malnutrition will affect both the feather structure and the amount and type of pigments in the feather. Dietary deficiencies can result in a lack of carotenoids or enzymes to modify them and result in the yellowing of green plumage or fading of feather color. Choline and riboflavin deficiencies in cockatiels can cause achromatosis of primary feathers, and a deficiency in tyrosine or copper can interfere with melanin production causing dark feathers to become lighter. Malnutrition can also lead to changes in feather structure and therefore reflective color. It is not only nutritional deficiencies that can affect coloration; dietary excesses in some hand-rearing formulations and pelleted foods may be associated with yellow feathers and bands in eclectus parrots of both sexes (Figure 32.47). Most nutrition-related changes in feather color will correct after a diet change and the following molt.[90]

Viral Diseases

Viral diseases such as PBFD can also affect feather color through changing reflective color. Green birds such as



FIGURE 32.44 Yellow-naped Amazon parrot *(Amazona auropalliata)* with genetic color mutation resulting in complete absence of carotenoid pigment from the plumage, hence providing the bird with a blue-white rather than green-yellow-colored plumage. (Photo courtesy Yvonne van Zeeland/Nico Schoemaker.)



FIGURE 32.46 Worn margins on feathers are often associated with a delayed or imminent molt. The ends of the vane become worn and tattered and lose their structure, that is, the barbules are no longer interlocking. (Photo courtesy Bob Doneley.)



FIGURE 32.45 This moustache parrot *(Psittacula alexandri)* shows two color changes. The first is the genetically selected color change to blue (color mutation). The second is the black scalloping on the feathers associated with human hand oils after prolonged handling. (Photo courtesy Bob Doneley.)

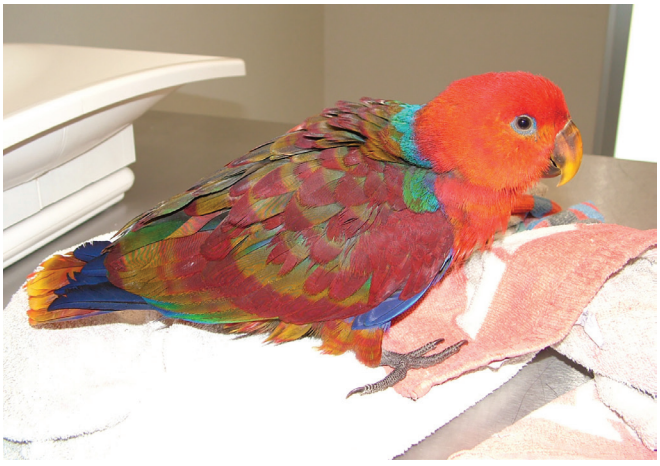


FIGURE 32.47 Dietary excesses in some hand-rearing formulations and pelleted foods may be associated with yellow feathers and bands in eclectus parrots *(Eclectus roratus)* of both sexes. (Photo courtesy Bob Doneley.)

lorikeets develop yellow patches; blue feathers in color mutation birds turn white; gray feathers in Grey parrots may turn red (Figure 32.48).[143]

Systemic Disease

Systemic diseases can affect feather coloration. Liver disease and hypothyroidism may affect the deposition of pigments in the feathers, altering their color. A lack of normal preening behaviors in an unwell bird may lead to structural changes, often giving the feathers a dull appearance.

FEATHER LOSS

In most companion birds presented with feather loss, the loss of feathers is the result from the bird (or a cage mate) pulling out its feathers. This behavior and its underlying medical and behavioral causes are considered in the next section (Feather-Damaging Behavior). Feather loss may occasionally also occur in the absence of this behavior. In most of these cases, the underlying disease damages the feathers or follicles in such a way that they are easily lost or shed.

The differential diagnosis for feather loss without the presence of FDB should include the following[92,193,219–221]:

- Normal apteria or normal molt (not recognized as such by the inexperienced parrot owner).



FIGURE 32.48 (A) Grey parrot with red discoloration of several of the coverts. This type of color change has been associated with various conditions including liver disease, psittacine beak and feather disease (PBFD), and malnutrition. However, it can also be seen in normal young Grey parrots and in areas of scarred skin. (B) A postmortem specimen of a lesser vasa *Coracopsis nigra* shows many randomly distributed white feathers. This bird contracted PBFD while being hand-reared with a group of other birds, including cockatoos. It died soon after fledging. (A, Photo courtesy Nico Schoemaker/Yvonne van Zeeland. B, Photo courtesy Nigel Harcourt-Brown.)

- Excessive or irregular molt induced by malnutrition or irregular photoperiod.
- Prior damage to the feather follicle resulting in cessation of feather growth.
- Genetic conditions (e.g., baldness in lutino cockatiels).
- Obesity, resulting in alopecia over the breast (particularly in budgerigars).
- Ectoparasites (e.g., *Knemidocoptes* spp., feather and quill mites, lice).
- Clinical manifestation of PBFD or polyomavirus infection.
- Mycotic infections, including candidiasis, *Malassezia* dermatitis, and dermatophytosis.
- Bacterial dermatitis and abscesses.
- Neoplastic skin disease including xanthomas.
- Systemic disease including hepatopathies and nephropathies.
- Endocrinopathies such as hypothyroidism.

Often, the feather loss will remain localized (e.g., in case of obesity or neoplastic disease), but generalized feather loss may also occur (e.g., in case of PBFD). Based on the distribution of feather loss certain diseases may be ruled out. In addition, diseases may be ruled out based on the presence or absence of skin damage or skin lesions.

Diagnostic workup of patients presented with feather loss should always begin with a thorough history and full physical and dermatologic examination. Additional diagnostic testing may be indicated based on the likely differential diagnoses established. In addition to specific treatment based on a diagnosis, contributing factors such as suboptimal diet, housing, environment, and management may be corrected as well.

Feather-Damaging Behavior

FDB can be broadly grouped into FDB inflicted by other birds and self-inflicted FDB.

Feather-damaging behavior inflicted by other birds

FDB inflicted by other birds is most seen in parrots, finches, and poultry. The following behaviors have been observed:

- Nestling parrots that have their feathers chewed by the parents, perhaps as a learned behavior (the parents were feather-picked themselves as chicks) or to remove the chicks from the nest so the hen can lay again. Mild cases involve removal of feathers, usually from the back, dorsal wings, and head. Severe cases will see the parent(s) remove wing tips or toes, or even kill the nestling. Affected chicks may be best removed for hand-rearing. They should not be used for breeding if possible.
- Breeding pairs that pull feathers from the head and face of their mate (Figure 32.49), often at the beginning of the breeding season, perhaps as the result of mismatched libido and readiness to breed. Both hens and cocks will pluck feathers from their mate.
- Overcrowding of enclosures holding finches, budgerigars, *Agapornis* spp., and *Neophema* spp. (particularly in pet shops) can lead to feather loss on the head and face, perhaps as a stress response by the birds inflicting the damage. Affected birds and, if possible, the offending bird(s) should be removed. Stocking density and stress factors need to be addressed and corrected. Foraging activities and toys should be provided and strategically enriched for birds that



FIGURE 32.49 Green-winged macaw *(Ara chloropterus)* with feather loss on the head as a result of plucking by its partner. (Photo courtesy Nico Schoemaker/Yvonne van Zeeland. Reprinted from van Zeeland YRA, Friedman SG, Bergman L: Behavior. In Speer BL, editor: *Current Therapy in Avian Medicine and Surgery*, Vol 1, St. Louis, MO, 2016, Elsevier, pp 177–251.)



FIGURE 32.50 (A) Excessive feather pecking and (B) cannibalism are commonly seen in poultry because of overcrowding. Similar injuries may also occur in other avian species when returning a recently deflighted individual back into the flock. (A, Photos courtesy Bas Rodenburg. B, Photos courtesy Elske de Haas.)

appear to be indulging in this behavior due to a lack of environmental enrichment.

- Poultry that are overcrowded will frequently display FDB toward other chickens, including the removal of feathers on the backs and pericloacal regions (Figure 32.50). Again, stocking density and stress need to be addressed.

Self-inflicted feather-damaging behavior

FDB, also referred to as feather-destructive behavior, feather plucking, feather picking, or pterotillomania,[222–224] has been reported in approximately 10% to 15% of captive parrots (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). FDB includes the chewing, plucking, biting, or pulling of feathers,[225,226] which can inflict more or less extensive damage to feathers in areas that are easily accessible to the beak (i.e., neck, chest, flanks, inner thighs, and wings; Figures 32.51 and 32.52).[222,227] While consequences of this self-inflicted feather damage may seemingly be merely aesthetic, medical issues can arise due to alterations to the birds' thermoregulatory abilities and metabolic demands and/or the development of hemorrhage and/or (secondary) infections.[227–229]

Although FDB may be noted in all psittacine species, it appears to be most common in Grey parrots, cockatoos, and eclectus parrots, with reported prevalence of up to 40%.[230] In contrast, it is less common in Amazon parrots (*Amazona* spp.), cockatiels, and budgerigars.[231–233]

Determining whether the feather damage is self-inflicted or due to a medically or environmentally related condition that causes loss or damage to the bird's plumage (irrespective of its behavior) can be difficult. First, birds are often left unobserved throughout a specific portion of the day, which limits the owner's ability to properly observe the bird's behavior. Second, it can be difficult for the owner to distinguish FDB from normal preening behavior, as both may follow the same pattern (Figure 32.53).[234]

In birds with self-inflicted feather damage or loss, it will be important to identify whether the behavior is primarily related to a medical condition that results in FDB due to the associated pruritus, pain, or other discomfort (which thereby acts as an antecedent to the behavior); whether that the behavior relates to an inadequate husbandry, management, and/or nutrition that affects the feather quality (hence signaling the behavior); or if there is a psychological underpinning to the behavior which stems from an attempt to deal with a suboptimal living environment (so-called *maladaptive behavior*) or an abnormal psyche or altered brain neurochemistry (i.e., an

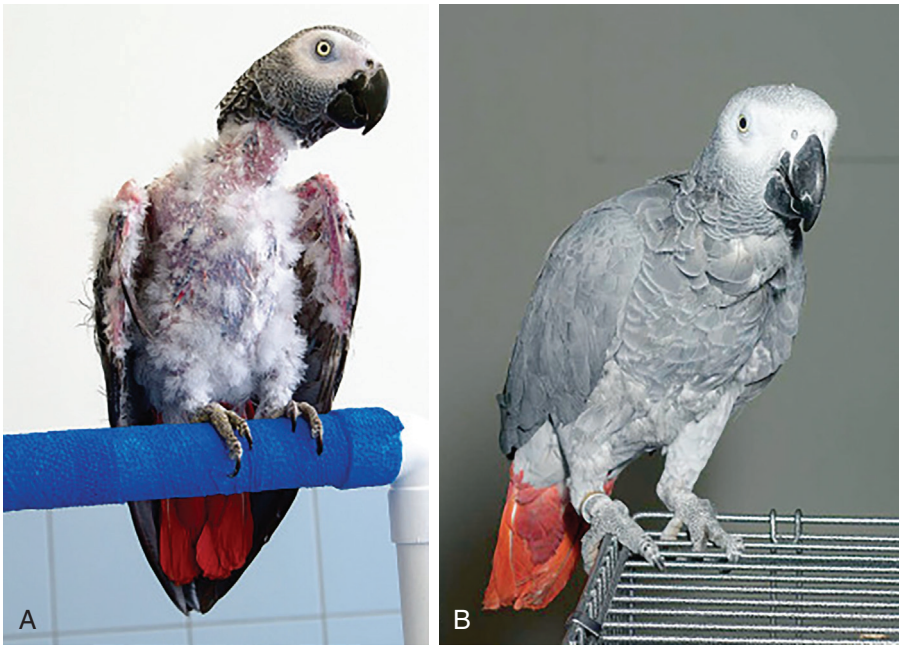


FIGURE 32.51 These two Grey parrots *(Psittacus erithacus)* demonstrate the typical appearance of a bird with self-inflicted feather-damaging behavior, as indicated by their normally feathered head. Severity of the feather damage may vary from case to case, resulting in (A) more or (B) less extensive loss of or damage to feathers. (Photo courtesy Nico Schoemaker/Yvonne van Zeeland. Reprinted from Paul-Murphy J, Wensley Koch V, Briscoe JA, et al: Advancements in management of the welfare of avian species. In Speer BL, editor: *Current Therapy in Avian Medicine and Surgery*, Vol 1, St. Louis, MO, 2016, Elsevier, pp 669–718.)

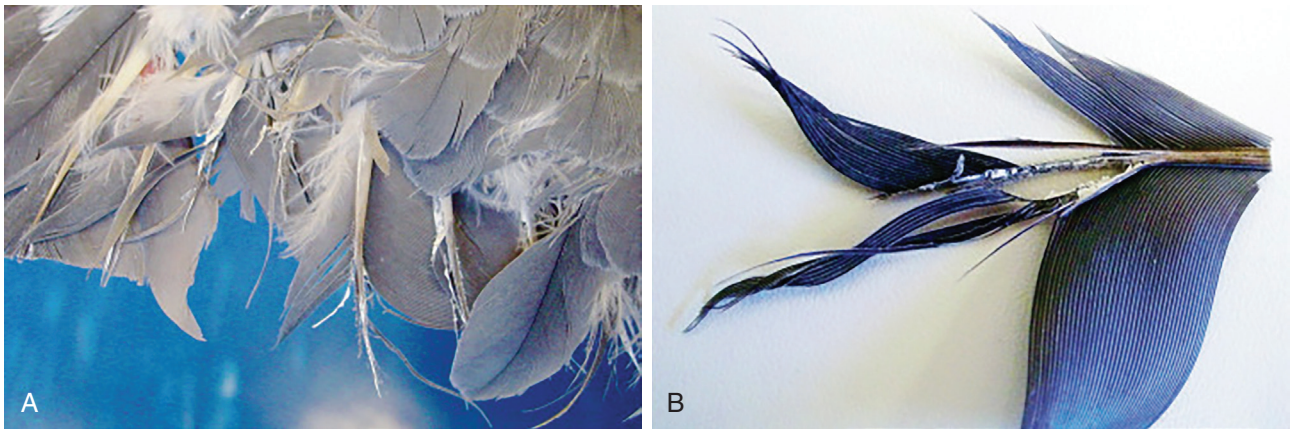


FIGURE 32.52 (A, B) Chewed and frayed feathers often have an irregular appearance, as can be seen in these feathers. (Photo courtesy Nico Schoemaker/Yvonne van Zeeland. From van Zeeland YRA, Friedman SG, Bergman L. Behavior. In Speer BL, editor: *Current Therapy in Avian Medicine and Surgery*, Vol 1, St. Louis, MO, 2016, Elsevier, pp 177–251.)

abnormal repetitive behavior or *malfunctional behavior*). Determining an exact cause often proves challenging, especially since multiple factors usually play a role and these behaviors do not reside in simple linear causality. These factors can make it challenging to effectively treat and reduce or eliminate the behavior.

Etiological Considerations

FDB often is a multifactorial problem, in which various medical, genetic, neurobiologic, and socioenvironmental factors can play a role (Table 32.2).[235] Numerous medical conditions have been associated with FDB, but in most cases it is difficult to prove a causal relationship. In essence, any disease or condition that causes pain, discomfort, anxiety, irritation, and/or pruritus could result in the development of FDB, which in turn can become a reinforced behavior due to a perceived consequential value that results from the behavior. Such conditions may both include primary feather and skin diseases as well as systemic diseases (Table 32.2).[193,233,235–243] In case of systemic disease, feather damage may either be



FIGURE 32.53 Grey parrot *(Psittacus erithacus)* in the process of removing a tail feather. Without the owner directly witnessing the removal of a feather, it might be hard to distinguish feather-damaging behavior from normal preening. (From van Zeeland YRA, Friedman SG, Bergman L: Behavior. In Speer BL, editor: *Current Therapy in Avian Medicine and Surgery*, Vol 1, St. Louis, MO, 2016, Elsevier, pp 177–251.)

TABLE 32.2

Factors Suggested to Be Involved in Feather-Damaging Behavior in Parrots

Environmental/Extrinsic Factors	Medical Factors	Intrinsic (Psychological) Factors
◆ Inadequate enrichment, lack of opportunities to perform species-specific behaviors, e.g., foraging, social interaction, flying ◆ Lack of choice or control over the environment ◆ Sudden changes to the environment ◆ Exposure to aversive, threatening stimuli (e.g., predators, novelty) ◆ Small cage or poor cage design with little space for the parrot to move around or fly ◆ Social isolation ◆ Overcrowding ◆ Reinforcement by actions of the owner ◆ Airborne and/or topical toxins, including cigarette smoke, scented candles, air fresheners, hand lotions, and creams ◆ Low humidity levels, lack of bathing opportunities ◆ Abnormal photoperiod ◆ Poor wing trim ◆ Trauma	◆ Ectoparasites (e.g., feather or quill mites, lice) ◆ Bacterial or fungal dermatitis/folliculitis (e.g., *Staphylococcus, Aspergillus, Malassezia*) ◆ Chlamydiosis ◆ Viral infections (e.g., polyomavirus, PBFD, avian bornavirus) ◆ Skin neoplasia (e.g., xanthoma, lipoma, SCC) ◆ Hypersensitivity, skin allergy ◆ Airsacculitis, pneumonia ◆ Cardiovascular disease (e.g., atherosclerosis) ◆ Liver and/or renal disease ◆ Hypocalcemia ◆ Endocrinopathy: hypothyroidism/diabetes mellitus ◆ Reproductive disease (e.g., egg binding) ◆ Heavy metal toxicosis (e.g., lead, zinc) ◆ Nutritional deficiencies (e.g., hypovitaminosis A) and/or dietary imbalances ◆ Gastrointestinal disease, e.g., colic, endoparasitism (particularly giardiasis in cockatiels) ◆ Obesity ◆ Orthopedic disorders (e.g., osteosarcoma, fracture, osteomyelitis, osteoarthritis) ◆ Neuropathy (e.g., spinal disorders)	◆ Hand-rearing and imprinting on humans ◆ (Sexual) hormones ◆ Boredom ◆ Frustration ◆ Stress ◆ Anxiety ◆ Attention seeking behavior ◆ Abnormal repetitive behavior resulting from neurotransmitter deficiencies and/or excesses (e.g., serotonin, dopamine, endorphins), similar to obsessive-compulsive or impulsive disorders

PBFD, Psittacine beak and feather disease.

Modified from van Zeeland YRA, Friedman SG, Bergman L: Behavior. In Speer BL, editor: *Current Therapy in Avian Medicine and Surgery*, Vol 1, St. Louis, MO, 2016, Elsevier, pp 177–251.

diffuse and generalized or localized directly over the region of discomfort. Renal disease, for example, may appear to induce FDB in the region of the synsacrum,[244,245] whereas hepatic disease may either induce feather damage that is limited to the ventral portion of the body or follows a more diffuse, generalized pattern.[138,246]

A commonly encountered risk factor for FDB also includes inappropriate husbandry, management, or nutrition (Table 32.3). A small cage or poor cage design may cause damage to the feathers, in particular the primaries and tail feathers. As a result, the bird may remove the damaged feathers, which should be considered as normal behavior.[236] Other environmental risk factors that have been implicated in FDB include nutritional deficiencies and dietary imbalances; airborne and topical toxins; low humidity levels and abnormal photoperiods, which can all affect the quality of the plumage and thereby stimulate the bird chew, pluck, or over preen its feathers.[221,235,236]

Almost regardless of whether a medical or environmental basis can or cannot be found for FDB, behavioral and/or psychological factors should always be considered. In general, two possibilities should be taken into consideration when a psychogenic FDB is suspected: (1) the behavior concerns maladaptive behavior, which results from an attempt of the animal to adapt to a suboptimal, inadequate living environment that either lacks appropriate stimuli to allow normal, species-typical behavior(s) or exposes the bird to aversive stimuli; and (2) the behavior represents an *abnormal repetitive* or *malfunctional behavior* which results from an abnormal psychology, brain development, or neurochemistry (potentially induced due to long-term exposure to an inadequate living environment).[247,248] Although the two are not separate entities per se and may even represent consecutive phases of the same disorder, the ability to make a distinction between the two may be of particular importance when considering the success of future therapeutic interventions. Whereas maladaptive behavior may benefit from changes to the environment that help to optimize the bird's living conditions, malfunctional behavior may show a lack of response to these measures and will more likely require the use of psychopharmaceutic drugs to reduce the behavior.

The list of factors that contribute to the development and maintenance of psychogenic FDB is numerous and includes

TABLE 32.3

Diagnostic Tests to Be Performed in Birds With Feather Abnormalities, Including Their Indications

Diagnostic Test	Indication(s)
Complete blood count and biochemistry	Hepatopathy, nephropathy, generalized infection or inflammatory process, diabetes mellitus, hypocalcemia
Toxicology	Suspected lead or zinc toxicosis. Collect heparinized whole blood (lead) or plasma/serum in nonrubber plastic or glass tubes
TSH stimulation test	Hypothyroidism
Fecal cytology (including wet mount and/or flotation)	Giardiasis (common in cockatiels), helminth infection, candidiasis, *Macrorhabdus ornithogaster* infection (avian gastric yeast), bacterial gastroenteritis
Radiology	Heavy metal intoxication, reproductive disorder (e.g., egg binding), hepato-, spleno- or renomegaly, proventricular dilatation disease, pneumonia, airsacculitis, neoplastic conditions, musculoskeletal disease (e.g., osteoarthritis, osteomyelitis, fractures, osteosarcoma)
Ultrasound	Hepatomegaly, reproductive disorders (e.g., egg peritonitis, cystic ovary), neoplastic conditions, cardiac disease, ascites
Endoscopy	Airsacculitis, hepato- or nephropathy, splenomegaly, pancreatic disorders, reproductive disease
Skin scrapings	Ectoparasites, in particular mites (e.g., *Knemodicoptes*)
Impression smear, swab cytology, or tape strip	Bacterial or fungal dermatitis, dermatophytosis, *Malassezia*, *Candida*, ectoparasites (e.g., feather mites, lice), pox virus
Fine needle aspirate	Skin neoplasia, xanthomatosis, feather follicle cyst, hematoma, bacterial dermatitis or abscess
Feather digest (using potassium hydroxide)	Ectoparasites (quill mites)
Feather pulp cytology	Bacterial or fungal folliculitis, PBFD or polyomavirus infection, quill mites
Culture	Bacterial or fungal dermatitis, folliculitis
Skin and/or feather follicle biopsy (histopathology)	Various infectious, inflammatory, and/or neoplastic skin diseases, e.g., PBFD, polyomavirus, bacterial and fungal folliculitis, quill mite infestation, xanthomatosis, squamous cell carcinoma, feather follicle cysts
Intradermal skin testing	Hypersensitivity reactions, allergic skin disease. Thus far not found to be reliable due to the bird's diminished reaction to histamine
Tests for specific causative agents	◆ PCR testing on whole blood, feather pulp or tissue for PBFDV ◆ PCR testing on fecal swab or tissue for presence of polyomavirus ◆ PCR on cloacal swab/feces and/or serologic testing for ABV ◆ PCR on conjunctival/choanal/cloacal swab and/or serologic testing for *Chlamydia psittaci*

ABV, Avian bornavirus; *PBFDV*, psittacine beak and feather disease virus; *PCR*, polymerase chain reaction; *TSH*, thyroid-stimulating hormone.
Reprinted from van Zeeland YRA, Schoemaker NJ: Plumage disorders in psittacine birds - part 2: feather-damaging behavior, *Eur J Compan Animal Pract* 24(2):24–36, 2014, with permission.

many socioenvironmental factors (Table 32.2).[233,235,249–253] In addition, the behavior may be exacerbated by inappropriate responses by the owner (e.g., punishing or attempting to distract the bird while it is damaging its feathers) as these responses may serve to reinforce the behavior.[254,255] Intrinsic reinforcements of the behavior should also be considered. Neurotransmitter deficiencies and/or excesses (e.g., dopamine, serotonin, endorphin) and a genetic background have also been proposed.[235,256]

FDB has particularly been linked to the lack of foraging opportunities in a captive environment. As a result, time spent foraging is drastically decreased: whereas wild parrots spend up to 6 hours daily on searching, selecting, and manipulating food,[257] captive birds usually consume their daily food ration within 30 to 72 minutes.[258,259] The lack of appropriate target stimuli to engage in species-specific foraging behaviors may subsequently lead to the onset of behavioral problems such as FDB.[227,251] Although the onset of this behavior may in part be the result of the altered time budget of captive parrots, some studies have demonstrated that parrots are motivated to forage and will contra-freeload (i.e., work for food even when identical food is freely available), thereby suggesting that foraging may be a "behavioral need."[260–262]

For most other factors that have been implicated in FDB, a causal relationship is less clearly established, thereby demonstrating the use for further epidemiologic and experimental studies into the etiology and risk factors associated with FDB.

Diagnostic Approach

In general, the diagnostic workup of FDB is primarily aimed at identifying or ruling out any medical or environmental factors that may be involved. For this purpose, a thorough history and complete physical examination are deemed essential. During the physical examination, the self-inflicted nature of the feather damage and/or loss may be confirmed by the absence of feather abnormalities on the head, which is inaccessible to the bird's own beak.[222] In addition to the history taking and physical examination, a thorough dermatologic examination of the skin and feathers is warranted, after which diagnostic skin and feather samples may be collected (Table 32.3). If an underlying systemic cause is suspected, diagnostic workup may be further expanded with, for example, a hematologic and/or biochemical blood panel, urinalysis, diagnostic imaging, and/or endoscopy (Table 32.3).[235,263] Intradermal skin testing for diagnosis of allergic skin disease has been described, but thus far been found unreliable in part due to the birds diminished reaction to histamine.[264,265] Definite diagnosis of allergic skin disease may therefore be difficult, although the collection of paired skin biopsies from affected and unaffected areas of the same patient may identify the presence of inflammation consistent with delayed-type hypersensitivity reaction.[266,267]

Any and each diagnostic test should always be carefully weighed for its merit and necessity for the particular case to prevent a plethora of tests from being run as this can pose health and welfare risks for the bird and be a financial burden to the owner. Should the history, physical examination, and additional tests fail to identify feather damage related to the physical environment or a medical problem that is likely to function as a strong antecedent for the behavior, a psychological or primary behavioral origin of the disorder becomes likely. It then becomes important to identify the potential signals (antecedents) and reinforcing factors (consequences) that may result in the behavior being maintained.[255] This can be a difficult and time-consuming task that could be further complicated by the limited reliability and accuracy of the owners' observations and their willingness to learn and commit to environmental adaptations, offering enrichment and behavior modification techniques.[235]

Therapeutic Considerations

Management of FDB can be challenging, especially in chronic cases, where it can be difficult to determine the antecedents and consequences that are associated with FDB, and ritualization of the behavior may have occurred, resulting in the behavior occurring irrespective of the initiating events. It is therefore important to initiate treatment as soon as possible, whereby the approach will largely depend on the findings of the history, physical examination, and diagnostic tests. An initial therapeutic plan will often be aimed at correction of the diet and modification of the bird's housing and living conditions to address any environmental factors that may be involved.

If any medical issues are encountered, these should be appropriately addressed, which may include the use of topical and/or systemic antibiotics, antifungals, and antiparasitic drugs to treat any underlying parasitic or infectious disease. In case allergies are suspected (e.g., based on histopathological findings that indicate a delayed hypersensitivity response), dietary and/or environmental modifications should be made to decrease or eliminate exposure to the suspected allergen(s), if and where possible. In addition, the use of antihistamines (e.g., hydroxyzine hydrochloride, 2 mg/kg PO q8h)[268] and/or corticosteroids may be considered. However, as histamine does not seem to be involved in the inflammatory response in birds, the value of antihistamines in the control of allergies remains questionable. Similarly, corticosteroids should be used with caution in birds because of their potential to induce profound immunosuppression which renders the bird susceptible to develop secondary infections in the absence of appropriate (preventive) antibiosis and antifungal treatment.

Promoting a more stimulating environment by means of social contact, perches, chewing toys, puzzle feeders, and other forms of environmental enrichment may be considered as an important part of the treatment regimen to alter the behavior (see Chapter 47 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).[235] In particular foraging enrichment has been shown to effectively reduce FDB.[224,234,242] Providing such enrichment may be as simple as providing complicated food items such as corn on the cob, pineapples, or pomegranates, providing food in larger chunks or pellets, using multiple feeding stations, scattering the food through the enclosure and/or mixing it with inedible it ems.[235,259,269] Owners may also use paper bags, cardboard boxes, plastic bottles, and other materials to create their own foraging toys or, alternatively, buy one or more of the more complicated foraging devices and puzzle feeders that have become commercially available throughout the past years. Although most of these foraging enrichments appear effective

to significantly increase foraging time of captive parrots, they do not appear capable of naturalizing foraging times to levels comparable with those of wild conspecifics (i.e., 4–6 hours per day).[269] New, more effective foraging enrichments may therefore need to be developed and tested for their efficacy to naturalize foraging behavior and reduce abnormal behavior.

In addition to providing environmental enrichment, behavior modification techniques such as differential reinforcement of other behaviors may be employed to alter the behavior of the bird (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).[232,255,270] Training may furthermore also help to provide the bird with a mentally stimulating challenge or task, provided the owner is able and willing to employ the techniques in a proper and consequent manner.

Other treatments that have been used to treat (psychogenic) FDB include the use of Elizabethan collars and neck braces, fabric "ponchos," "jackets," or "vests," and/or local application of foul-tasting substances (Figures 32.54 and 32.55).[235] It should, however, be remembered that these interventions are primarily aimed at preventing the behavior rather than eliminating the underlying causes. Even their use as a tool to help "break habitual FDB," which was considered an acceptable approach in the past, is nowadays highly questionable from an ethical standpoint as it could lead to "learned helplessness" through "flooding" (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). However, sometimes collars, braces, or ponchos may be necessary to stop the bird from self-mutilating. When opting to use one of these devices, care should be taken to select the device that will be effective yet poses the least amount of burden to the bird from both a physical and mental perspective. If and where possible, devices should be designed such that as much of the normal behavior is possible. For example, pieces of tape can be stuck to a collar or brace, and ponchos can be made from multiple layers of lightweight bird-friendly cloths of which the upper layers are cut into strips to allow preening activity. When placing a collar, poncho, or brace, one should also take into consideration that many birds respond well to the placement; as a result, administering tranquilizers such as midazolam (0.3–0.5 mg/kg IM) could be helpful to facilitate placement, while also ensuring adequate monitoring and supportive care (e.g., gavage feeding and fluid therapy in birds that will not eat and drink) until the bird is properly acclimatized to the device.

Pharmacologic intervention has also been proposed to treat (chronic) FDB. In the authors' opinion, this type of intervention is primarily indicated in those cases that appear refractory to treatment with behavior modification therapy and environmental changes. Options that can be considered include (1) anxiolytic drugs such as diazepam[271]; (2) antipsychotic drugs such as the dopamine antagonist haloperidol[272,273]; (3) tricyclic antidepressants such as amitriptyline, clomipramine, and doxepin[271,274,275]; (4) serotoninergic reuptake inhibitors such as paroxetine and fluoxetine[271,276,277]; and (5) opioid antagonists such as naltrexone (Appendix 1).[271,278] In cases of suspected sexual or hormonally related FDB (e.g., seasonal occurrence and presence of [hyper]sexual and nesting behaviors) treatment may be initiated with a depot gonadotropin-releasing hormone (GnRH) such as deslorelin or leuprolide acetate, or medroxyprogesterone acetate.[279] For more in-depth information on the various psychoactive drugs and their use in birds, the reader is referred to reviews by Seibert (2007),[271] van Zeeland (2018),[280] and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior.

The preferred drug to be used in a given case will be the one that is most likely to be effective in treating the behavioral problem but poses the least risk of adverse side effects. This not only requires knowledge of the mechanism of action, (contra)indications, and side effects of the various classes of drugs available but also warrants an accurate analysis and diagnosis of the underlying motivation for FDB prior to initiating a treatment, and a thorough monitoring and follow-up with detailed instructions being provided to the owner on what can be expected (Box 32.4). It should furthermore be emphasized that, while psychoactive drugs can be useful in the treatment of FDB, they are not expected to provide a cure on their own. The likelihood of beneficial outcomes following drug therapy is greatest when using these as an adjunct therapy to appropriate behavior modification therapy, environmental adjustments, and adequate treatment of any concurrent medical illnesses. When thoughtfully applied, medications are likely to benefit the treatment plan because they can help attenuate an animal's response and increase its receptiveness to the environment. In general, starting at a low dose and gradually titrating it to effect (or until adverse effects are observed) is recommended. Unless a patient exhibits adverse effects, treatment should be attempted for at least the time period that is needed for the drugs to take effect (i.e., 1 week for benzodiazepines and 1 month for most other drugs). If, after this period, insufficient response without adverse effects is seen, the dose can be gradually increased. Should the maximum dose be reached, or adverse effects encountered without adequate resolution of FDB, switching to another drug (from the same or a different class) could be attempted following (gradual) weaning of the original drug and allowing a drug-free interval of approximately 2 weeks before initiating the new medication.

Monitoring and Follow-up

To evaluate whether the treatment has the desired (and no adverse) effects, regular monitoring and follow-up are recommended. Ensure that regular contact with the owner is established to discuss the effects of the interventions, as well as challenges encountered during their implementation. Although the owner is able to directly observe the bird's behavior,[275] this method may not always provide reliable results since it can be difficult to distinguish normal preening from abnormal FDB and bouts of FDB may be missed as these may occur during the night.[234] As such, it could be important to educate the owner to, for example, evaluate (changes in) the number of feathers found on the bottom of the enclosure. In addition, regular checkups in the clinic are advised to evaluate the bird's plumage condition, and adjust the plan, if necessary. To objectively evaluate the changes in plumage condition, feather scoring systems (Box 32.5) and (serial) photographs can be useful.[251,281]



FIGURE 32.54 (A) Many different types of Elizabethan collars are available for parrots and are preferably only used as a component of treatment if the parrot is mutilating itself. The collar this Grey parrot *(Psittacus erithacus)* is wearing has an extension that prevents the bird from reaching even the most distal areas of its body. A great disadvantage is that these collars commonly weigh around 20% of the body weight of the birds and prevent normal preening behavior and other important maintenance behaviors of the bird. (B) In this Grey parrot *(Psittacus erithacus)* a ball-shaped collar is used to prevent the parrot from chewing on the esophageal tube, which was placed because of an extensive crop burn. The great advantage of this "ball" collar is that the curvature of the neck fits within the inner side of the ball. These collars are therefore often better tolerated compared with the one shown in (A). The disadvantage is that the birds can still reach the feet and parts of the wings when wearing these types of collars. Neither of these mechanical blocking devices should be viewed as acceptable substitutes for appropriate pain management of these types of wounds and for promoting alternative chewing behaviors. (C) Besides the commercially available Elizabethan collars, home-made collars are also frequently used. The collar that this lovebird is wearing was made out of insulation tubing that is used to protect warm-water pipes. These collars are not only very inexpensive, but they are easy to make, lightweight, effective, and well tolerated by the birds. (A–C, Photos courtesy Nico Schoemaker/Yvonne van Zeeland. From van Zeeland YRA, Friedman SG, Bergman L: Behavior. In Speer BL, editor: *Current Therapy in Avian Medicine and Surgery*, Vol 1, St. Louis, MO, 2016, Elsevier, pp 177–251.)



FIGURE 32.55 (A) Alternatives to the use of Elizabethan collars are "ponchos" or "socks," which owners frequently fabricate themselves. Alternatively, socks can also be purchased online and customized to the size and color of the parrot, as is the case with this sulfur-crested cockatoo *(Cacatua sulphurea)*. These garments provide a safe, nonrestrictive, and comfortable alternative to collars. Moreover, a sock helps protect against heat loss that may occur as a result of lack of insulating contour and down feathers. Birds generally tolerate socks surprisingly well. (B) In case of self-injurious behavior to the wings, socks will not suffice. However, a jacket, such as the one that was made by the owner of this Grey parrot *(Psittacus erithacus)*, may be helpful to protect the wing area from further damage inflicted by the parrot's beak. (A, Photo courtesy Suzanne Freeman [www.thesockbuddy.com]. B, Photo courtesy Nico Schoemaker/Yvonne van Zeeland. From van Zeeland YRA, Friedman SG, Bergman L: Behavior. In Speer BL, editor: *Current Therapy in Avian Medicine and Surgery*, Vol 1, St. Louis, MO, 2016, Elsevier, pp 177–251.)

BOX 32.4 CHECKLIST FOR ADMINISTERING PSYCHOACTIVE DRUGS[280]

Veterinarians should ask themselves the following questions when considering the use of pharmacologic intervention for behavior modification and ensure all these can be answered with yes before initiating treatment.

1. Has a thorough behavioral and medical workup been performed to identify the potential underlying causes for the problem behavior? Has a working diagnosis or differential diagnoses been established?
2. Have the potential underlying medical issues and environmental stressors been adequately dealt with? Has or will an appropriate behavior modification plan be(en) put into place?
3. Is it possible for the owner to medicate the bird without causing additional stress that may exacerbate the existing problems?
4. Does the drug of choice's mechanism of action correspond with the hypothesized neuropathophysiologic processes that underlie the behavioral problem (e.g., reduction of anxiety, compulsiveness, or hormonal influence)?
5. Did the medical workup reveal no contraindications for use of the medication? If the bird is receiving any other medication, is there no interaction with these drugs that warrants caution or adjustment of the dose?
6. Has the owner been informed properly about effects of the therapy and the potential risks (i.e., side effects) of the treatment?
7. Has a proper monitoring and follow-up protocol been set in place (e.g., frequency of rechecks, monitoring of plasma concentrations, evaluation for presence of side effects)?

Reprinted from van Zeeland Y: Medication for behavior modification in birds, *Vet Clin North Am Exot Anim Pract* 21(1), 115–149, 2018.

BOX 32.5 FEATHER SCORING SYSTEM FOR ASSESSING PLUMAGE CONDITION IN BIRDS

(A) Score Determination Table for Coverts and Down Feathers; Used for Chest/Neck/Flank, Back, Legs, and Dorsal and Ventral Surface of Wings

	Down Feathers			
Coverts	**No Down Removed**	**<50% of Down Removed**	**>50% of Down Removed**	**All Down Removed**
All coverts intact	100	85	70	60
Fraying or breakage	95	80	65	55
<25% of coverts removed	90	75	60	50
25%–50% of coverts removed	80	65	50	40
50%–75% of coverts removed	70	55	40	30
75%–90% of coverts removed	60	45	30	20
>90% of coverts removed	50	35	20	10

The percentage of damage to the covert and down feathers is assessed for each body part separately.
Deduct 10 points from the score if skin damage is present.
Total body plumage score (0–100)[a] = 0.25 × chest/flank + 0.17 × back + 0.10 × legs + 0.28 × dorsal wings + 0.20 × (ventral wings).
[a]To determine the total body plumage score, the scores for each body part are corrected for their relative body surface percentage, similar to scoring systems used in human burn victims. These percentages (expressed as % of the total body surface area excluding the surface area of the head and unfeathered parts of the legs) were determined in six Grey parrots. Mean (± SD) values for the various body parts were 25 ± 1.2% (chest/neck/flank), 17 ± 1.5% (back), 10 ± 1.2% (legs), 28 ± 2.2% (dorsal wing surface, up to the level of the tertiaries), and 20 ± 1.9% (ventral wing surface, up to the level of the tertiaries).

(B) Score Determination for Flight Feathers; Used for Tail, Primary and Secondary Feathers (Wings)

Score	**Description**
0	Flight feather with signs of fraying and/or breakage over >50% of the original length
1	Flight feather with signs of fraying and/or breakage over <50% of the original length
2	Flight feather with little or no damage present

Damage to individual flight feathers is assessed.
Total flight feather score (0–100)[b] = (primary + secondary feathers left wing) + (primary + secondary feathers right wing) + (tail feathers).
[b]The maximum score is dependent on total number of flight feathers of the bird. In general, each wing has 10 primary feathers and 10 secondary feathers (remiges), whereas the tail has 10–12 flight feathers (rectrices). As each individual flight feather is awarded a score from 0 to 2, the score will range from 0 to 40 for each wing and from 0 to 20 (or 0–24 in the case of 12 tail feathers) for the tail, respectively.

SUMMARY

A variety of different conditions may affect the plumage of birds, resulting in various feather abnormalities, including changes to the feather structure, coloration, and growth. Feather loss, particularly related to self-inflicted pulling, plucking, biting, chewing, or fraying of the feathers, is common in psittacine species and can be a difficult and frustrating problem to deal with due to the complex interplay between medical (i.e., dermatologic as well as systemic diseases), environmental, nutritional, psychological, and genetic factors. Due to the myriad of factors that can be involved, any condition affecting the plumage of a bird warrants thorough behavioral and medical workup, whereby treatment is targeted at eliminating all the risk factors involved. Proper education of clients is also important, as it may take time before the effects of the treatment will be visible (i.e., several weeks to months in case of feather loss, or—in case of feather abnormalities—until the next molt, when the new feathers regrow).

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CHAPTER 33

Pododermatitis

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INTRODUCTION

Pododermatitis is the most frequently encountered foot disease of captive birds and a cause of significant welfare concerns. Pododermatitis is rarely seen in free-living birds, and consequently, management of cases frequently requires identification and correction of husbandry, nutritional and/or environmental deficiencies that have causal roles in the condition. Several bird groups appear predisposed, with the taxonomic orders Falconiformes, Anseriformes, Sphenisciformes, and Phoenicopteriformes seemingly being particularly susceptible.

Pododermatitis can be simply defined as inflammation of the skin of the foot. In avian medicine it is considered a degenerative, inflammatory condition of the plantar surface of the foot, most commonly involving the plantar metatarsal pad or plantar digital pads. It is best considered a disease syndrome—where disruption of the plantar epithelium allows microbial invasion and secondary infection. If not identified quickly and managed effectively, pododermatitis can progress and affect deeper tissues, resulting in tendon necrosis and osteomyelitis of the distal tarsometatarsus and digital bones. This chapter provides an overview of the etiological and pathophysiological considerations, diagnostic workup, and treatment of pododermatitis in birds. As knowledge of the anatomical and physiological characteristics of this region is essential for gaining a thorough understanding of the pathology and the seriousness of its effects, these will first be discussed.

ANATOMY AND PHYSIOLOGY

There are a number of species-specific differences between the anatomy of avian feet; however, some similarities exist between all birds. These anatomical similarities and differences will be discussed in the later sections.

Epidermis

The skin covering the feet of birds is constructed of heavily modified dermis and epidermis (Figure 33.1). The epidermis is avascular and thin and consists of two layers:

- *Stratum corneum*—the most superficial, horny layer
- *Stratum germinativum*—this layer is split into three distinct layers:
  - *Stratum basale*—the deepest layer, which is separated from the dermis by a basal membrane. Dividing cells in the *stratum basale* move superficially forming the *stratum intermedium*.
  - *Stratum intermedium*—these epidermal cells are held together by desmosomal junctions. Nutrients are transported from the vascular dermis and transported within the extracellular fluid, which is found between the cell plasma membranes.
  - *Stratum transitivum*—the most superficial layer of the *stratum germinativum*. This transitional layer contains keratohyalin granules found in the cytoplasm, closely associated with the cell nucleus. In the most superficial layer of *stratum transitivum*, keratinization occurs below the horny *stratum corneum*.[1]

In the domestic fowl, at their thinnest, the *stratum corneum* and *stratum germinativum* are only two to three cells thick. However, it is substantially thicker over the featherless areas of the body, such as the face, and is at its thickest in the podotheca of the foot. In most species the transition to modified thicker skin starts at the region of the intertarsal joint—although some variation exists. The degree of cornification of the modified skin depends on the species—with terrestrial birds having heavily cornified structure, while in water birds it is softer and more flexible.[2]

Dermis

The dermis is found directly under the epidermis and is composed of connective tissue. It is separated from the subcutaneous tissue by a layer of elastic connective tissue—*lamina elastica*. This layer enables the dermis to remain flexible and pliable, which is needed for flight, particularly in the axillary region. The superficial layers of the dermis are characterized by increased numbers of fibroblasts. The superficial compact layer of dermis is made up of dense network of collagen—predominately with horizontal orientation and elastic fibers. On microscopy, this collagen structure is more organized and structured in birds compared to mammals. In the deep loose layer, the connective layer is less structured, and adipose cells and lymphatic vessels are present.[3]

Podotheca

The integumentary covering of the feet, termed the podotheca, is a layer of scales from approximately the distal tibiotarsus to the ends of the digits. While the podotheca is heavily cornified in land birds, it is softer and more flexible in water birds. Feathers tend to diminish distally on the legs as scales arise and become prominent, the transition usually occurring

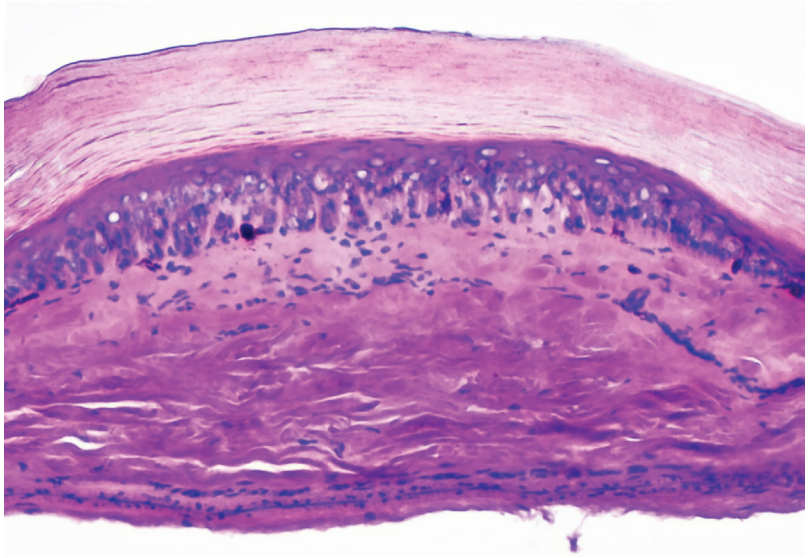


FIGURE 33.1 Scaly skin from lateral tarsometatarsal region and removed to the underlying bone. Frozen section cut 6- to 9-μm thick, manually stained with hematoxylin and eosin (slender-billed parakeet, *Enicognathus leptorhynchus*).

about the intertarsal joint (but varying greatly between species). Eagles and some other eagle-like raptors, such as the ferruginous buzzard *(Buteo regalis)*, are feathered below the intertarsal joint—often described as being "booted." Feathers and scales are variously situated in relation to each other in parts of the foot and among birds, but they never intergrade.

Scales are flat, rounded, or conical raised thickenings of highly keratinized epidermis, separated by inward folds of thinner, less keratinized epidermis (Figure 33.2).[3] They vary in size, shape, amount of overlap, and degree of fusion on different parts of the foot, not to mention among species. The main types of scales that can be distinguished are as follows:

- *scutes* (large scales)
- *scutella* (intermediate scales)
- *reticula* (small scales)

Scutes are seen most frequently on the dorsal aspect of the tarsometatarsus distal to the intertarsal joint. The plantar surfaces (and occasionally the anterior surface) of the tarsometatarsus and the dorsal surface of the digits are typically covered by scutella scales—being more rectangular and more regularly arranged than those on the remaining surfaces (reticulate scales). Metatarsal scales, not needing flexibility, are often fused, especially on the anterior surface. Overlapping or imbrication of scales is species specific—being found in kingfishers (Alcedinidae) and woodpeckers (Picidae).

The scale types differ embryologically and histochemically as well as morphologically. Scutellate (scutes and scutella) scales contain beta-keratin, similar to the rhamphotheca and the claws, whereas reticulate scales are composed of alpha-keratin. These differences indicate that they have different evolutionary histories. Scutellate scales, being very similar to crocodilian scales, appear to have come directly from reptilian scutes, whereas reticulate scales are apparently a later-derived form with different properties.[2]

The reticulate scales on the plantar aspect of the digits and the distal end of the tarsus cover the thick plantar and digital pads. These pads, formed from connective tissue, provide protection to the flexor tendons and joints and are designed to withstand compression, especially in terrestrial species. They underlie the joints and are separated by transverse furrows or gaps, which open and close as the digits are bent. The

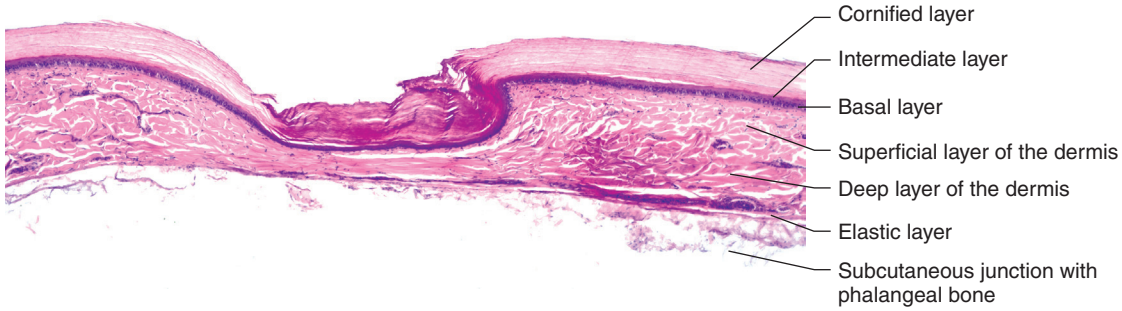


FIGURE 33.2 Section of full-thickness skin showing two small scutes (hard keratin) and their more flexible (soft keratin) junction. Lateral digit of Harris' hawk *(Parabuteo unicinctus)*. Frozen section cut 6- to 9-μm thick, manually stained with hematoxylin and eosin.

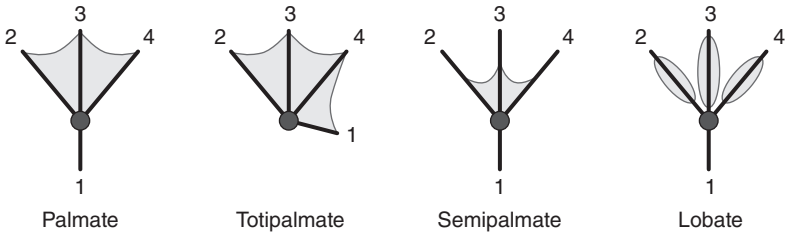


FIGURE 33.3 Type of feet webbing recognized in birds: (A) palmate, (B) totipalmate, (C) semipalmate, and (D) lobate.

pads and papillae vary morphologically and histologically among the digits and among species according to the length of the digits, their need for support, and the natural history of the bird.

Young woodpeckers, barbets, toucans, kingfishers, jacamars, and trogons develop an extra-thick pad that is located on the distal-ventral aspect of the tarsus. During the nesting period, while confined to the nest cavity, they rest on this pad and shed it when they fledge (see Chapter 44, Figure 44.8).[2]

Most waterbirds have a palmate foot structure and have a web present between the digits that is formed from reticular scales, resulting in paddle-like feet (Figure 33.3). This web is composed of thin, flexible, leathery integument. Pelecaniformes have full webs connecting all four digits (totipalmate); Anseriformes, Procellariiformes, lari, loons, and alcids have full webs between the three forward digits (palmate); and plovers and some sandpipers have basal webs between the forward digits (semipalmated). Grebes, rails, and finfoots (Heliornithidae) accomplish the same end with flat lobes on independent digits (lobate).

Ptarmigans have digit feathering that increases the surface area of the foot, acting as natural snowshoes that allow easier ambulation in winter climates. For example, the winter plumage of willow grouse *(Lagopus lagopus)* includes digit feathering that increases the surface area of the foot fourfold, thereby reducing the sinkage into snow by 50%, thereby also saving considerable energy. Black grouse *(Tetrao tetrix)* and capercaillie *(T. urogallus)* have pectinations (combs), which are extensions of the foot scales on the digits that also increase the surface area of the feet. Digit feathering often develops in autumn and molts out in spring.[1,4]

Claws

Avian claws are formed over the germinal epidermis at the terminal phalanx of each digit. They share many similarities with the rhamphotheca (beak covering) with both having a highly keratinized structure that contains hydroxyapatite crystals (see Chapter 31). A digit's claw is composed of a dorsal plate *(scutum dorsale)* that is the hardest, containing heavy deposits of beta-keratin plus calcium salts and curves downward at the tip and sides, and a plantar plate *(scutum plantare)* that fills the space between the sides underneath (Figure 33.4).[3] The curved shape of the claw comes from the faster dorsal growth in comparison to the plantar plate and has advantages for grasping prey, perching, and climbing. Digit claws vary in length, curvature, and pointedness in relation to their usage and the substrate where a bird lives. For example, claws are less developed in diving and water birds, whereas they are much larger in Falconiformes, and are



FIGURE 33.4 Sagittal section of a steppe eagle *(Aquila nipalensis)* claw.

flattened in grebes, contributing to the foot paddle. Claws have pectinate (comb) edges, used for grooming feathers, in nightjars, herons, frigatebirds, and pratincoles.[2] The same function is suggested to be present in hawks, in which the claw on digit 3 has a flatter profile that may serve as an adaptation for grooming feathers.

Digits

The foot of a "typical" avian species has four digits. While no birds have more than this number, several species have fewer. The ostrich is the only species that has just two digits—with digits 1 and 2 being lost. Other ratites, many waders, petrels, auks, and woodpeckers have three digits, with digit 1 being vestigial or absent. In kingfishers, it may be the second digit that has been lost in development.

The arrangement of digits depends on function and is highly species specific (Figure 33.5). Among the arrangements that can be distinguished are:

- Anisodactyl feet—three digits pointed forward, one digit (digit 1) pointing back; this arrangement is seen in, for example, passerines (Passeriformes), gallinaceous birds (Galliformes), and Falconiformes (falcons, caracaras).
- Zygodactyl feet—two digits (digits 2 and 3) pointing forward; two digits (digits 1 and 4) pointing back; this arrangement is seen in toucans (Rhamphastidae), parrots

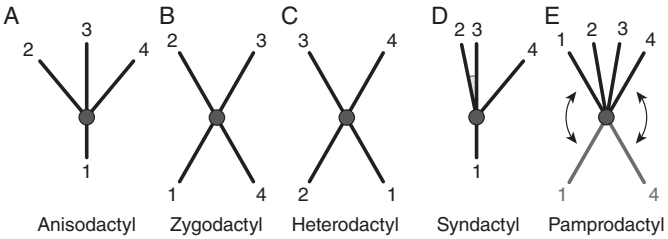


FIGURE 33.5 Digital arrangements of the feet in birds: (A) anisodactyl, (B) zygodactyl, (C) heterodactyl, (D) syndactyl, and (E) pamprodactyl.

(Psittaciformes), and cuckoos (Cuculidae) and is beneficial for climbing and grasping. Owls (Strigiformes), osprey (Pandionidae), and turacos (Musophagidae) have a variation on zygodactyl feet where digit 4 can face either forward or back.

- Heterodactyl feet—this arrangement is much like that for zygodactyl feet, but here digits 1 and 2 face backward, as is the case in trogons (Trogonidae).
- Syndactyl feet—here, digits 3 and 4 are partly united, as can be seen in Coraciiformes (kingfishers, bee-eaters, rollers, motmots, and todies.), and hornbills (Bucerotidae).
- Pamprodactyl feet—all four digits face forward in an adaptation for climbing; this arrangement is seen in mousebirds (Coliidae) and swifts (Apodidae).

Feet can further be classified as grasping, walking/wading, or swimming feet, based on the function to which they have specialized.

Grasping Feet

Grasping feet are adapted for perching or holding prey or food items. The function is most common in species with anisodactyl and zygodactyl feet. In the anisodactyl feet, the hallux (digit 1) is fully opposable to provide a firm grip, whereas the grip in zygodactyl feet is developed further due to two pairs of opposable digits.

Walking/Wading Feet

In species with walking/wading feet, digit 1 is often absent or vestigial, leading to the foot having limited or no grasping capacity. In wading birds, full or partly webbed feet can be seen. In certain species, the digits may be elongated to increase the ground/surface contact and spread the load.

Swimming Feet

Swimming feet all have a significant development of webbing or lobes. Some species have totipalmate feet, that is, all four digits are fully webbed, whereas others have palmate feet in which digit 1 is vestigial or absent and the other three digits are webbed. Grebes and coots have broad lobes on the side of the four digits, forming a lobate foot, but lack a web that joins the digits.[1]

PATHOPHYSIOLOGY OF PODODERMATITIS

Regardless of the species, pododermatitis typically starts with mechanical breach of skin surface. Most of these cases occur due to pressure and/or abrasion, which results in an area of avascular damage. The initial ischemic insult progresses to the development of necrosis and inflammation of the soft tissues, leading to the release of inflammatory mediators. This not only aggravates the lesion but also causes thrombosis of small blood vessels and provides a point of entry for pathogenic, commensal, or opportunist bacteria which then lead to further inflammation and tissue damage. In grade 1 and early grade 2 diseases, which are characterized by inflammation, erythema, scabs, and moist lesions, bacterial infection has yet to develop. As such, these cases are mostly amenable to treatment.

Hyperkeratosis appears when the lesion becomes chronic, reducing the epithelium thickness followed by tissue ulceration. Lesions can also advance toward the subcutaneous tissue and cause secondary infections, most notably of *Staphylococcus* spp. In animals in which the condition is left untreated, osteomyelitis, tendinopathies, and arthritis may develop. At this stage, the prognosis is unfavorable and can also lead to systemic pathologies such as endocarditis and polyarthritis.

Recognizing the factors that predispose to pododermatitis is essential. Factors known to increase the risk of pododermatitis include excess weight or obesity, larger-sized birds, inappropriate perching (i.e., size, texture, materials), substrate or bedding, inactivity or sedentary lifestyle, malnutrition (e.g., hypervitaminosis or hypovitaminosis A in psittacine birds, biotin deficiency in turkeys), and lack of cage hygiene. In addition, birds with unilateral lameness (e.g., arthritis, fracture) may develop pododermatitis on the contralateral leg due to a shift in weight bearing. Trauma can also predispose to development of pododermatitis, as primary penetration allows direct inoculation of bacteria into the foot or creates a wound that serves as an entry point for bacteria. Examples include talon penetration (in raptors), bite injuries, or puncture wounds from sharp objects. Finally, pododermatitis has been linked to repeated exposure to nicotine residues on the hands of a smoker, strong cleaners/disinfectants, or allergens, which all can cause local irritation and dermatitis of the feet.[5]

CLASSIFICATION OF PODODERMATITIS

No classification scheme for pododermatitis in birds has been universally accepted. However, the most used ones involve grading from either 1 to 5 or 1 to 6, with 1 representing the least severe condition, and 5 or 6 constituting "end-stage" disease. A scheme originally proposed for classifying raptor pododermatitis is commonly used (see Box 33.1 and Figure 33.6) and can be adapted and applied to other species.[6] In addition, specific classification schemes have been developed for certain species such as flamingo[7] and poultry.[8]

HISTORY AND CLINICAL EXAMINATION

A combination of signalment, clinical examination, diagnostic imaging, and microbiology is frequently employed in the diagnosis of pododermatitis. A clinical history and examination are important to determine the severity of the pododermatitis and identify any predisposing factors that contributed to its development. This information is essential to determine an appropriate treatment plan and establish a prognosis.

History should include information about the diet (including any treats or supplements provided), housing/enclosure, perches (if applicable), substrate/bedding, cleaning and disinfection protocols, and exercise. In addition, attention should be paid to possible trauma or medical conditions that could lead to unilateral lameness or altered weight bearing.

BOX 33.1 GRADING SCHEME FOR PODODERMATITIS (FIGURE 33.6A–E)

Grade 1: Erythema and smoothing of the skin on the plantar aspect of the feet. Flattening of the papillae. Slight discoloration, most frequently a darkening of the skin, can also be seen.

Grade 2: Hyperkeratosis and thickening of the plantar skin may be seen. Localized inflammation and the beginning of necrosis. Crusts may be seen.

Grade 3: Moderate to severe hyperkeratosis. The feet will often feel warm to touch, with moderate to severe swelling and mild to moderate pain. The necrotic and hyperkeratotic core penetrates the skin to full thickness, and a caseous abscess forms.

Grade 4: Infection spreads to adjacent soft tissues, including joints and tendons. Tenosynovitis is often present, resulting in ascending infections and inflammation extending from the foot toward the tarsometatarsus. Swelling will commonly be seen on the dorsal and plantar aspect of the feet. Mild to moderate pain can be observed.

Grade 5: Involvement of skeletal structures. Osteomyelitis and/or septic arthropathies of one or more of the intertarsal, tarsophalangeal, and interphalangeal joints. Marked swelling with moderate to severe pain is often observed.



FIGURE 33.6 Examples of various grades of pododermatitis. (A) Focal hyperemia and loss of integrity of the epidermis in grade 1 pododermatitis. (B) Grade 2 pododermatitis affecting the digital pad of digit 2. (C) Grade 3 pododermatitis in a snowy owl *(Bubo scandiacus)*. (D) Grade 4 pododermatitis in which the infection has spread to the underlying tendons and bony structures. (E) Grade 5 pododermatitis in which septic arthritis has resulted in significantly impeded function of the foot. (Photos A and B courtesy Neil Forbes.)

When obtaining a history regarding the enclosure or perches, asking the client to provide photographs and videos is very useful. Where possible, on-site examination of the bird in its environment should be performed as the bird's perching preferences and behavior can help identify more subtle risk factors. In nocturnal species such as owls it may be necessary to observe the birds during the night. Night vision web-cameras or closed circuit television (CCTV) can help to facilitate this procedure.

A systematically performed physical examination is helpful to discover concurrent diseases or predisposing factors for pododermatitis. First, the patient should be observed in its cage or carrier, whereby any changes in gait or weight bearing, as well as abnormal posture while perching should be noted.

To facilitate the clinical examination, sedation or anesthesia may be required, especially in untrained or fractious patients (see Chapter 46); however, in many species, manual restraint may suffice. A weight and body condition score should be obtained and compared to previous values, if applicable. Physical examination findings may include erythema, erosions or ulcerations, swelling, lameness, reluctance or inability to move, and pain on palpation. Lesions are most commonly found on the metatarsal pad or digital pads and should be graded according to their severity (see section on Classification of Pododermatitis). Photographs can be helpful to monitor progression. Aside from a visual inspection of the lesions, the feet should be palpated for swelling, crepitus, and range of movement of the joints. Gloves should always be worn—firstly to prevent inoculation of skin bacteria into lesions, but also to protect the examining veterinarian from exposure to bacteria. In addition to examining the affected foot, the clinician should always examine the other leg and foot closely to evaluate for the presence of problems on the contralateral leg leading to a shift in weight bearing.

Differential diagnoses for pododermatitis include the following:

- trauma (soft tissue, fracture, dislocations);
- gout;
- septic arthropathy (hematological spread).

With the majority of these, there will not be disruption to the plantar skin, thereby facilitating the establishment of a diagnosis.

DIAGNOSTIC TECHNIQUES

To determine whether infection has spread to underlying tissues and differentiate between grade 3 and 5 pododermatitis, imaging will often be required. Radiography or computed tomography (CT) imaging is best used to assess skeletal changes (e.g., osteomyelitis, Figure 33.7; see also Chapter 2). Moreover, these allow the clinician to assess injuries that may contribute to uneven weight distribution between the feet (e.g., degenerative joint disease, previous misaligned fracture healing, metabolic bone disease, intraabdominal disease–causing sciatic nerve compression). Ultrasound, on the other hand, can be used to assess soft tissue structures such as tendon sheaths and tendons—particularly in larger birds, though the scales may hinder proper visualization of the structures (Figure 33.8). CT or magnetic resonance imaging (MRI) may

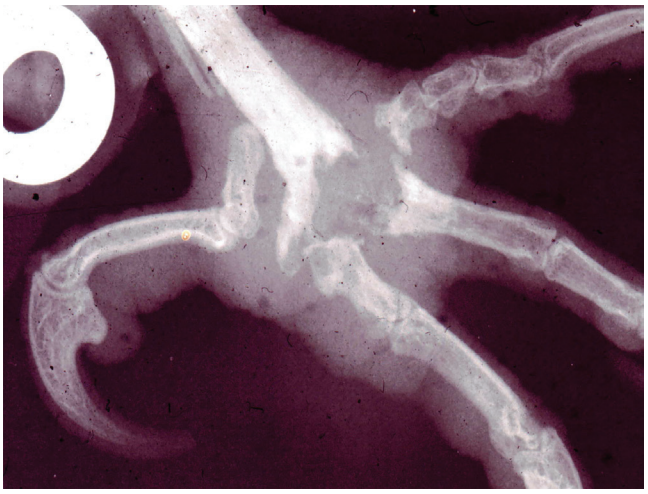


FIGURE 33.7 Radiograph showing severe osteomyelitis in a case of a grade 5 pododermatitis.

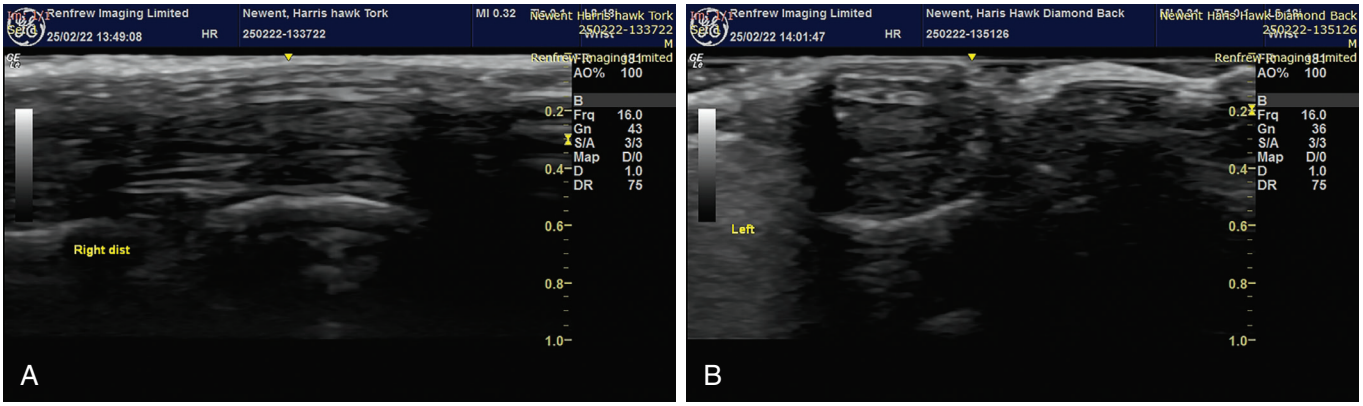


FIGURE 33.8 Longitudinal (A) and transverse (B) images of a normal gastrocnemius tendon in a Harris' hawk *(Parabuteo unicinctus)*.

also be used to detect more subtle bone and soft tissue pathologies.

From the lesions, material can also be collected for cytology and/or culture. Cytological samples should be stained with a Gram stain as this can help with the selection of first-line antimicrobial therapy while awaiting culture results (see Chapter 7). Culture and sensitivity performed on a deep portion of the lesion or a piece of infected tissue will generally lead to the best results. Frequently, *Staphylococcus aureus* is isolated from such lesion, though other bacteria, such as *Escherichia coli*, *Corynebacterium* spp., *Pseudomonas* spp., and yeasts are regularly cultured from the lesions as well.[9–12]

A complete blood cell (CBC) count and biochemistry panel can be performed to identify concurrent diseases or predisposing factors. If blood work is performed as part of a complete clinical investigation in the more advanced stage of disease, there is frequently elevation of the white blood cell (WBC) count with a heterophilia. Fibrinogen is also elevated to boost clotting (hyperfibrinogenemia).

TREATMENT OF PODODERMATITIS

Management of pododermatitis will generally require a multimodal approach. Dependent on the grade of pododermatitis, a combination of medical and surgical interventions may be needed that aim at relieving pressure on the affected area(s), reducing swelling, establishing drainage (as needed), and treating any secondary infections and associated pain and inflammation (Table 33.1). Regardless of the severity and species, treatment should aim at correcting the underlying causes. Concurrent disease conditions as well as any stress and/or anxiety should also be addressed.

REVIEW AND CORRECTION OF HUSBANDRY AND MANAGEMENT

Treatment of pododermatitis is doomed to fail unless the underlying causal management problem or problems are identified and corrected. While a clinical history can be very useful, nothing is quite as beneficial as an on-site examination of the housing. The detail is vitally important, and subtle problems can easily be missed in conversation. Where an on-site evaluation is not possible, photographs and videos can be used. Time-lapse footage of birds in an aviary space or enclosure can be useful to assess the proportion of time spent at certain locations, or on certain perches. It may allow assessment of group dynamics which may be a factor in the onset of pododermatitis.

TABLE 33.1

Summary of Grade and Treatment Options for Pododermatitis in Avian Species[a]

Grade	Identification	Treatment Options	Prognosis
1	◆ Erythema/smoothing skin ◆ Flattened papillae ◆ Discoloration—either darkening/pink erythema	◆ Environmental/husbandry changes ◆ Pressure-relieving dressings/casts ◆ Topical treatments (emollients, footbaths/soaks) ◆ No indication for antibiotics	Excellent if underlying cause can be identified and corrected
2	◆ Hyperkeratosis/thickening plantar skin ◆ Localized inflammation ◆ Early necrosis ◆ Crusts	◆ As above for grade 1 disease ◆ Systemic antiinflammatories ◆ Topical antibiotics ◆ Systemic antibiotics (rare cases where secondary bacterial colonization is likely) ◆ Surgery considered in refractory cases	Good
3	◆ Progressed hyperkeratosis ◆ Diffuse swelling feet ◆ Mild to moderate pain ◆ Necrotic and hyperkeratotic core ◆ Abscess formation	◆ As for grades 1 and 2 ◆ Surgical debridement necrotic/avascular tissues ◆ Remove caseous material and pyogenic membranes ◆ Systemic antibiotics based on culture ◆ Topical antibiotics, that is, absorbable calcium sulfate impregnated beads ◆ Topical dressings promoting granulation	Fair
4	◆ Spread of infection into tendon sheaths, joints, and adjacent tissues	◆ Flush tendon sheaths and surgical debridement as required ◆ Advanced imaging food can help with surgical planning ◆ Local/regional antibiotics particularly beneficial ◆ Regional limb perfusion antibiotic administration ◆ Long courses of treatment required where bone and synovial structures effected	Guarded
5	◆ Involvement of skeletal structures ◆ Osteomyelitis and/or septic arthropathies ◆ Marked swelling with moderate to severe pain is often observed	◆ Viability and ethics of treating need to be assessed ◆ Even if infection is controlled long-term secondary degenerative joint disease will affect welfare ◆ Euthanasia may need to be considered	Poor

[a]Species-specific variations are discussed in section on Species-Specific Considerations.

Reversal of early stage pododermatitis is often possible following correction of the underlying predisposing factors. These may include dietary changes, stimulating exercise, frequent exposure to sunlight, improved cleanliness, and adjusting the enclosure design. For animals that are overweight, a weight loss program should be initiated which includes measures to stimulate the bird to exercise and move around (e.g., by offering foraging enrichment) as well as a reduction of the daily caloric intake. For psittacine birds, a gradual transition from a seed-based diet to a formulated diet with additional fresh fruits and vegetables that contain vitamin A (e.g., peppers, squash, sweet potatoes) is commonly advised. Birds of prey should be fed with a varied (supplemented) whole-prey diet, whereas for poultry and waterfowl a commercial maintenance diet appropriate for the species is recommended.

Perches of variable sizes and types should be offered to perching birds and rotated on regular basis to encourage birds to perch on different surfaces. Surfaces should be kept clean and dry and can be wrapped with safe, soft materials such as cotton padding or foam covered with Vetrap bandaging tape (3M, St. Paul, MN, USA) during the management of pododermatitis. For birds of prey, shape and configuration of the perch should be appropriate for the species involved. For example, falcons should be housed on flat surfaces (e.g., shelves covered with artificial turf), whereas eagles, hawks, and owls are preferably housed on round or elliptical perches (e.g., made of hemp or sisal rope).[13] For waterfowl, it is essential to provide access to water for swimming, which must be kept clean and free of organic matter. For most species, a large pond with a minimum depth of 2 feet should be provided, though for the diving species, a depth of at least 4 feet is recommended.[14] Gradual slopes in and out of the water are important, as is the provision of islands for resting, and areas of shade and sunlight.

TOPICAL MEDICATION

Many topical products have been used in the treatment of pododermatitis, albeit without little scientific evidence to promote the use of one product over another.

Examples include silver sulfadiazine 1% cream, Collasate postoperative dressing (PRN Pharmacal, Pensacola, FL, USA), and nontoxic antimicrobial products such as triple-antibiotic ointment (Neosporin; Johnson & Johnson Consumer Companies, Inc., New Brunswick, NJ, USA; Table 33.2; see also Chapter 36).[15] Whenever using topical antibiotics, care should be taken to avoid products that are toxic following ingestion. Similarly, oil-based products can potentially affect feather quality and impair thermoregulation. Topical corticosteroids should be avoided because of their immunosuppressive effects and delayed wound healing.

SYSTEMIC ANTIBIOTICS

Systemic antibiotics may be indicated in case of persistent or deep infections. Ideally, the choice of antibiotic is based on the results of a culture and sensitivity testing, but if this is unavailable, a broad-spectrum antibiotic with activity against both gram-positive and gram-negative bacteria can be selected. An antibiotic that is able to penetrate tendons, joints, and/or bones should be selected in case of tendinitis, arthritis, and/or osteomyelitis (see Chapters 6 and 7).

TABLE 33.2

Commonly Used Topical Treatments for Pododermatitis[15–19]

Active Ingredient	Mode of Action	Use
Manuka—methylglyoxal	High osmolarity—reducing edema and inflammation. Gluconic acid production contributes to low pH and antibiotic activity. Hydrogen peroxide production. Provides a moist and favorable environment for wound healing.	Useful particularly with resistant bacterial infections. Activity against biofilm-producing organisms such as *Staphylococcus aureus*.
Silver	Compromising phosphomannose isomerase and components of the microbial electron transport system as well as impairing some DNA function of microorganisms.	Multispectral topical antimicrobial action. Care delay wound healing with extended use.
Dimethyl sulfoxide (DMSO)	Changes the scope of penetration of chemical compounds in biological membranes. Increases membrane fluidity.	Addition of DMSO in the dressing is suggested to facilitate the penetration of topical antibiotics and may help to reduce swelling.
Sodium fusidate	Fusidic acid is a bacteriostatic antibiotic with bactericidal properties at higher concentrations. Its mode of action is via inhibiting bacterial protein synthesis. The ointment base reduces the amount of dry, hyperkeratotic skin.	Antibacterial, particularly against *Staphylococcus* spp. infections. Reported to penetrate intact skin.
Povidone iodine	Action against both gram-positive and gram-negative bacteria. Ability to penetrate biofilms with minimal cytotoxicity. Oxidative damage of bacterial cell structures.	For either footbaths or topical wound cleansing.
Benzalkonium chloride, polyhexanide (F10 Barrier Ointment)	Broad-spectrum antimicrobial properties from disruption of cell membranes.	Topical administration to the foot or incorporated in footbaths or lavage solutions.
Yeast extract and shark liver oil	Shark liver oils contain alkylglycerols which modulate the inflammatory response, enhancing macrophage activation.	Found in topical human hemorrhoid preparations, applied to the plantar skin to treat low-grade pododermatitis (grades 1–2).

ANALGESIA

Analgesia is important to provide for patients with pododermatitis, as even the superficial lesions can be painful. Meloxicam is a commonly used analgesic, but dependent on the severity, opioids such as butorphanol and tramadol may be considered (see Chapter 35).

REGIONAL LIMB PERFUSION

Regional limb perfusion administration of antibiotic treatments has recently received significant attention in avian species (see Chapter 6, Figure 6.5; see also Chapter 36). Relative ease of intravenous access into the medial metatarsal vein makes this technique achievable in medium to large species. The higher tissue concentrations achieved locally are particularly useful where resistant infections are present or when using antibacterials that have greater risk of systemic toxicity (e.g., aminoglycosides).[20] Amikacin has been used in cranes. Ceftiofur regional limb perfusions have been investigated experimentally in chickens, and showed synovial concentrations of ceftiofur to exceed therapeutic concentrations for most pathogenic bacteria.[21,22]

A tourniquet or manual vessel occlusion is performed proximal to the site of infection, following which an infusion of intravenous antibacterials is performed distal to the tourniquet for approximately 15 minutes. Most commonly, cannulation of the medial metatarsal vein and placement of the tourniquet are performed just proximal to the intertarsal joint. In large species, placement of a digital cannula may be possible.

LASER THERAPY

Laser therapy has been used in a number of species as an adjunctive treatment for pododermatitis. The difficulty of setting up control trials and multimodal treatment of most patients make it difficult to demonstrate its added value, although empirical case reports have shown promising results.[23–25]

MEDICAL VERSUS SURGICAL INTERVENTION

Low-grade (grade 1 or 2) pododermatitis is frequently managed with husbandry changes and topical therapies, with or without the addition of pressure-relieving dressings (Figure 33.9). Overall aims of this treatment are to:

- provide analgesia;
- control swelling and inflammation;
- prevent formation of (further) necrotic tissue;
- prevent progression of pododermatitis to higher-grade disease;
- prevent colonization of pathogens;
- promote granulation and healing (thus avoiding cytotoxic therapies).

Higher-grade (i.e., grades 3–5) pododermatitis frequently requires more extensive therapy to be successful (Figure 33.10). Surgical intervention is often required and aims to:

- debride necrotic and avascular tissues and remove caseous material and pyogenic membranes;
- minimize disruption to the normal foot structure;
- allow primary closure, where possible; many cases, however, will require healing by granulation and secondary intention;
- expedite healing and allow the limb to return to full function as soon as possible;
- prevent spread of infection into bone, tendons, tendon sheaths, or joints.



FIGURE 33.9 Grade 1–2 pododermatitis in a small falcon following 1 week of topical treatment and correction of husbandry. Callus is starting to lift at the periphery.



FIGURE 33.10 Severe grade 3 digit pododermatitis. Progression can lead to avulsion of the digital flexor tendon.

In severe cases, with extensive involvement of underlying tissues and functional limb dysfunction, amputation of the affected leg may be considered. However, in wild birds or birds of prey, the procedure would not be recommended.

Surgical debridement will vary significantly from case to case. The species, chronicity of disease, previous treatment attempted, importance of (remaining) limb functionality, and so on will all affect how a surgeon manages a case. The timing of surgery is important. Premature surgery will lead to unnecessary disruption of the foot architecture (Figure 33.11) versus a surgery that is delayed for too long leading to progression of lesions and development of irreversible septic arthropathies, osteomyelitis, or tendinitis with risk of tendon rupture. Typical surgical steps involved in most cases are summarized in Box 33.2 and Figure 33.12.

FOOT DRESSINGS

Bandages or foot dressings are required in most, except the milder cases. There is a wide range of foot dressings utilized in the management of pododermatitis (Figure 33.13). The technique selection will depend on species, lesion location and severity, and—to a degree—on personal preference. There is very little published material in which techniques are compared, hindering the ability to provide evidence-based guidance on the efficacy of



FIGURE 33.11 Brite-Vue enhanced computed tomography images of the anatomy and unique vascular supply of the foot of various bird species. (A) Bald eagle *(Haliaeetus leucocephalus),* dorsal and ventral view; (B) African goose *(Anser cygnoides),* dorsal and plantar view; (C) pigeon *(Columba livia domestica),* dorsal view.

Continued



FIGURE 33.11, cont'd (D) Grey parrot *(Psittacus erithacus)*, dorsal and plantar view; (E) macaroni penguin *(Eudyptes chrysolophus)*, dorsal and ventral view; (F) yellow-billed stork *(Mycteria ibis)*, medial and lateral view. In order to prevent excessive disruption of the normal architecture, adequate knowledge and understanding of the species-specific anatomy of the foot, including its nervous and vascular supply, are important to help plan an optimal surgical approach. (Images courtesy Dr. Scott Echols and Avian Studios, www.AvianStudios.com.)

BOX 33.2 STEPWISE SURGICAL PROCEDURE FOR TREATMENT OF PODODERMATITIS

1. Assess plantar skin and plan surgery (Figure 33.12A). If possible, incisions should be made to reduce tension and allow primary healing.
2. Surgical preparation of skin—the preference is chlorhexidine, although povidone iodine can be used (see *Current Therapy in Avian Medicine and Surgery Vol 1, Chapter 21, Surgery*). A toothbrush can be useful to remove debris from between scales. If no crust is present, samples for culture and sensitivity should be taken before surgical preparation of the foot.
3. Crusts, if present, are removed, following which samples can be taken for bacterial and fungal culture. Culture swabs should be sampled from deep tissue to prevent culturing of surface commensals (Figure 33.12B)
4. A temporary tourniquet can be placed proximal to the intertarsal joint and left on for a maximum of 10 minutes per foot. This helps to prevent blood contamination of the surgical field and allows easier visualization of the structures of the foot.
5. Necrotic material and caseous material with associated pyogenic membrane are removed to leave a healthy, bleeding wound bed (Figure 33.12C). Where present, the pyogenic membrane should be removed in its entirety, and ideally be left intact. A small Volkmann spoon can be useful for curetting material. The surgeon's aim is for one single and thorough surgical procedure rather than leaving contaminated tissue and requiring repeat procedures. Care should be taken to maintain the structural integrity of the foot and minimize formation of scar tissue (Figure 33.12D).
6. The wound bed is soaked and/or lavaged with warm saline or lactated Ringer's solution. Alternative irrigation solutions include dilute povidone iodine and dilute benzalkonium chloride (F10 sc). These would preferably only be used in the initial phases of treatment as these solutions can be cytotoxic to fibroblasts and may reduce white blood cell viability and phagocytic efficiency, hence leading to delayed wound healing (see Chapter 36).

BOX 33.2 STEPWISE SURGICAL PROCEDURE FOR TREATMENT OF PODODERMATITIS—cont'd

7. Small (2–4 mm) antibiotic-impregnated beads can be used to locally obtain a high concentration of antibiotics, which generally will exceed the minimum inhibitory concentration (MIC) of the organisms involved. Commonly used antibiotics for this purpose include aminoglycosides, cephalosporins, penicillins, fluoroquinolones, and clindamycin, with the choice depending on culture and sensitivity of the lesion.[26] If antibiotic-impregnated beads are placed, they should be sited so that they can ideally be removed from a dorsal incision rather than requiring a repeat disruption to the plantar skin. The surgeon should accurately record where they have been placed to help expedite removal, once needed. Modern alternatives of absorbable calcium sulfate beads (Kerrier LLC) do not require removal and are preferable to Polymethyl methacrylate (PMMA) when available. Antibiotic-impregnated substrates are most useful in cases with grade 3 and higher-grade pododermatitis or those with resistant bacterial infections where high concentrations of antibiotics are required to overcome the MIC. Local antibiotic therapy is also considered appropriate in cases sensitive to potentially toxic antibiotics (e.g., aminoglycosides).
8. Avascular and devitalized skin should be removed and, where possible, apposition of the skin should be accomplished with monofilament interrupted sutures to allow first intention healing (Figure 33.12E). In some cases, a tension-relieving suture pattern (mattress or "far-near-near-far") suture may be required.

different dressings. In general, the aims of a foot dressing or casting are to:

- relieve pressure on pododermatitis lesions;
- prevent desiccation and provide favorable conditions for granulation and healing;
- shield the wound from contamination and prevent microbial contamination;
- in some cases, allow continued application of topical therapies;
- avoid softening and thinning of the epidermis.

With early pododermatitis lesions (e.g., flattening of the papilla), liquid bandages, cyanoacrylate skin protectants, or skin toughners (e.g., camphor and benzoin) can be placed on the lesion. In the case of more extensive lesions, bandaging can be applied to the lesion. Such bandages are generally



FIGURE 33.12 (A) An adult female steppe eagle *(Aquila nipalensis)* was presented with chronic bilateral pododermatitis. (B) After a 5-day course of enrofloxacin, the bird was anesthetized and the scab removed to allow the foot and tendons to be assessed. Once cleaned, the foot lesions were covered with propylene glycol, salicyclic acid (Dermisol, Zoetis) and bandaged with a nonadherent dressing that was held in place with zinc oxide tape. (C) Once the wounds had started to granulate with no signs of purulent discharge from the tendon sheaths, the bird underwent surgery to close the wounds. Excess granulation tissue and fibrous tissue were removed. (D) During surgery, the wound was regularly assessed to ensure that the skin edges would oppose without undue tension and that the shape of the foot was returned to as close to normal as possible.

Continued



FIGURE 33.12, cont'd (E) Polydioxanone suture swaged onto a round-bodied needle was used to repair the wound. Initially, a series of mattress sutures were placed to reduce the dead space as well as the tension on the skin edges. These sutures were subsequently opposed by simple interrupted sutures. (F) The wounds were covered with a sheet of hydrophilic (Granuflex) dressing to help keep the wound clean and reduce tension and movement on the suture line. The dressing was held in place with zinc oxide tape. If this bird had been a falcon, a foot cast would have been used, but hawks are known to healwith just a well-padded perch. The bird was exercised by flying on a creance daily. (G) Dressings were changed every 7 to 14 days. The sutures remained present until the wound looked healed. This process can take up to several weeks to complete. (H) After 8 weeks, the skin was healed but still quite thin and easily injured. A small wound was still present over an area where the foot had not been returned to the correct shape. However, with time and care, this wound eventually healed. This case clearly demonstrates that it may take a long time for the foot surface and function to return to normal. Once unbandaged, lanolin-based hand cream can be applied to the dry skin to allow it to return to normal faster.

composed of three layers: (1) a primary layer that contacts the wound, (2) a secondary layer that has absorptive properties, and (3) a tertiary layer to hold the other layers in place (see Chapter 36).

The primary layer of the dressing must be carefully chosen to provide the ideal environment for wound healing and greatly depends on the stage of healing (see Chapter 36). In raptors, for example, wet-to-dry bandages or a seton with ball bandage are used in the initial stage to facilitate drainage of the wound, followed by a ball bandage without the seton in the granulation stage, polypropylene shoes in the early healing stage, and an interdigital bandage in the later stages of healing. A lightly absorbent, nonadhesive dressing such as Melolin (Smith & Nephew, Watford, UK) is preferred by the author in the initial treatment as it is of particular use where mild, moderate discharge is present from the wound. Hydrocolloid dressings have been recommended; however, these should only be used in noninfected wounds. Their use can promote a moist wound environment that has been proposed to increase the risk of secondary mycotic infections. Hydrocolloid-type dressings may also lead to softening of the skin surrounding the wound—particularly if used for protracted periods of time. Hydrocolloid dressings (e.g., Granuflex, DuoDERM - Convatec, Deeside, UK) help to promote epithelialization and protect and strengthen the suture line (see Chapter 36).

Pressure-relieving foot casts in their many different forms should be used where feasible (Figures 33.13–33.15). Ongoing pressure at the lesion or surgical site is a major reason for poor resolution and wound healing. One of the authors' preferred techniques is a "snowshoe" style dressing constructed of yoga matting (Figure 33.14A). Portions of the snowshoe mat can be cut away full or partial thickness to remove pressure away from the tissue below (Figure 33.14B). Later in the treatment course where a primary dressing is not required,

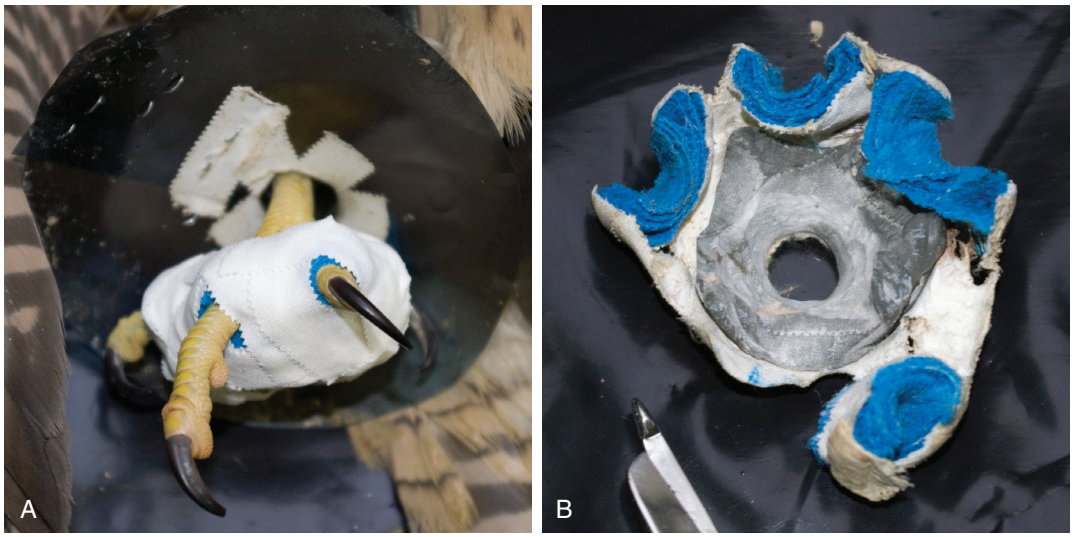


FIGURE 33.13 A saker/gyr falcon hybrid *(Falco cherrug × F. rusticolus)* had surgery for bilateral bumblefoot. For this bird, foot casts were made from car-body filler that was modeled in the correct shape using a Dremel drill with a 9.5-mm grindstone. The toes were padded with sections of loosely wrapped conforming bandage of approximately 1-cm width. As this type of bandage compacts and tightens with time, it must be replaced at least every 10 days. The bird interfered with the bandages, which was prevented by an "Elizabethan collar" on each leg, in this case old radiography film (A). A view of the contact surface of the dressing following removal from the birds foot (B).

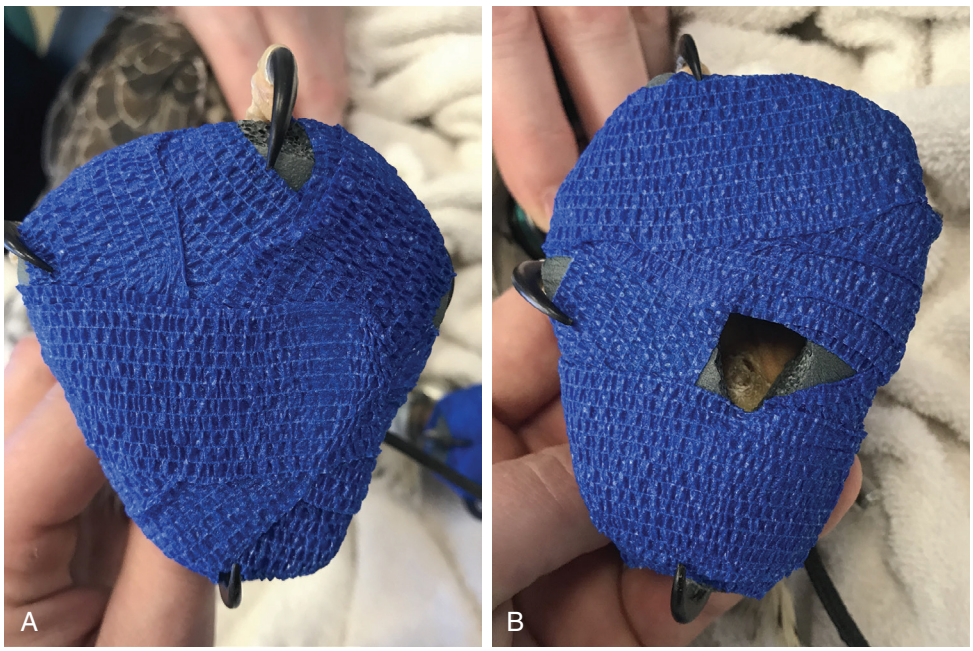


FIGURE 33.14 (A) Pressure-relieving dressing constructed of 12-mm-thick "yoga mat" and cohesive bandage. (B) A central "cutaway" in the yoga mat dressing allows direct application of topical treatments to the central callus of the (grade 2) pododermatitis lesion.

this can also facilitate direct examination and application of topical medication while keeping a dressing in place.

The client/owner will need to keep any foot dressings clean and dry. Should dressings become wet or contaminated or slip out of position, these would need to be changed as soon as possible. Dressings should initially be changed daily to two to three times per week and at least every 5 to 7 days in the later stages of wound healing. Suture removal should be carefully planned and is often best performed in a staged removal (which is why interrupted suture patterns are preferred) to allow the sutures to continue to provide some support for 1 to 2 weeks after wound healing has completed. After removing dressings, the healed skin should be monitored closely (i.e., at least every 4 weeks) for several months

for evidence of recurrence. Intervals of reexaminations would be case specific, but intervals of at least every 4 weeks for the first 3 months are recommended as recurrence is common, and treatment is far more successful if recurrence is quickly identified.



FIGURE 33.15 Doughnut dressing can also be used to relieve pressure from a pododermatitis lesion. (Photo courtesy Neil Forbes.)

While this general treatment guide can be applied to most captive avian species, species-specific treatment recommendations exist and are summarized in the following sections.

SPECIES-SPECIFIC CONSIDERATIONS

Falconiformes

Introduction

The Falconiformes, together with the Strigiformes, integrate a group of birds commonly referred to as birds of prey or raptors. This term originates from the Latin word "*rapere*," meaning to size or grab, and describes how these birds hunt using the large, strong talons and sharp, hooked beaks.

The order of the Falconiformes comprises five families, that is, Cathartidae, Pandionidae, Accipitridae, Sagittariidae, and Falconidae (Box 33.3).

Birds of prey are among some of the most popular species kept in captivity. Many species are maintained in zoological collections and used in exhibitions, others are maintained in breeding centers, as part of propagation and reintroduction programs, while others are bred intensively to supply the needs of the falconry market worldwide (see Chapter 55). This trade involves the Harris' hawk *(Parabuteo unicinctus)*, the goshawk *(Accipiter gentilis)*, as well as falcon species such as the

BOX 33.3 OVERVIEW OF THE VARIOUS FAMILIES BELONGING TO THE ORDER OF FALCONIFORMES[27]

Family	Characteristics
Cathartidae (New World vultures)	◆ Comprises 3 genera, 7 species, and 13 taxa ◆ Medium to large diurnal birds of prey, with bare heads, a powerful hooked beak, and large wingspan to allow gliding and soaring in search of carrion ◆ Habitat: widely variable, ranging from desert, grasslands, and savannas to lowland tropical forests to high mountains of North America, Central America, and South America ◆ Largest species: California condor *(Gymnogyps californianus)* with a weight of 8.0–14.0 kg, length of 1.17–1.34 m, and a wingspan of 2.7 m ◆ Smallest species: lesser yellow-headed vulture *(Cathartes burrovianus)*, with a weight of 950–2550 g, a body length of 0.58–0.66 m, and a wingspan of 1.6 m
Pandionidae	◆ Comprises one single species: osprey *(Pandion haliaetus)*, a relatively large, piscivorous bird of prey ◆ The male and female weigh between 1.2 and 1.6 kg and between 1.6 and 2.0 kg, respectively, and have a body length between 55 and 58 cm and a wingspan of 1.45–1.70 m
Accipitridae	◆ Hawks, eagles, and Old World vultures ◆ Comprises 237 species, 64 genera, and 535 taxa ◆ Worldwide distribution ◆ Largest species: lappet-faced vulture *(Torgos tracheliotus)*, with a weight of 5.4–9.4 kg, a body length of 1.15 m, and a wingspan of 2.80 m ◆ Smallest species: pearl kite *(Gampsonyx swainsonii)* from Central America and South America, of which the male weighs approximately 94 g, has a body length that measures 20–28 cm, and has a wingspan of 54 cm
Sagittariidae	◆ Comprises one single species: the secretary bird *(Sagittarius serpentarius),* a large bird of prey which is characterized by long legs, a loose crest of long feathers, and long central tail feathers ◆ Secretary birds weigh 2.3–4.3 kg, are 1.25–1.50 m height, and have a wingspan of 2.12 m
Falconidae	◆ Small- and medium-sized birds of prey (caracaras to falcons) ◆ Comprises 10 genera, 61 species, and 149 taxa ◆ Worldwide distribution; birds inhabit all geographical regions of the world with the exception of the Antarctic ◆ Largest species: gyrfalcon *(Falco rusticolus).* The male and female of this species weigh 0.96–1.32 and 1.26–2.10 kg, respectively, are 0.48- to 0.60-m long, and have a wingspan of 1.20–13.5 m ◆ Smallest species: black-thighed falconet *(Microhierax fringillarius)* from Southeast Asia, which weighs 28–55 g, has a body length of 14–17 cm, and a wingspan of 30–34 cm

gyrfalcon *(Falco rusticolus)*, the saker falcon *(Falco cherrug)*, the peregrine falcon *(Falco peregrinus)*, and their hybrids (particularly gyr-peregrine hybrids and gyr-saker hybrids), which are mainly destined for the markets of the Middle East, where they are used extensively for hunting and, more recently, for falcon races.

Pododermatitis is a condition commonly encountered in captive birds of prey, particularly the larger species.[28–30] Different etiological factors have been proposed as responsible for the development of pododermatitis in birds of prey including increased body weight, inadequate perches, lack of exercise, sudden cessation of exercise, bruising of the plantar surface of the feet, unequal weight bearing on one foot, overgrown talons,[28–30] and inadequate diets (Figures 33.16 and 33.17).[31] Members of the Falconiformes, but in particular the gyrfalcon *(Falco rusticolus)*, and saker falcon *(Falco cherrug)* are more likely to develop pododermatitis (Figure 33.16).[32] Owls are the next most commonly presented, with larger species such as snowy owls *(Bubo scandiacus)* particularly susceptible. Hawks and eagles have a significantly lower prevalence and typically only develop significant pododermatitis due to concurrent injury or significant husbandry limitations (Figure 33.17A).

Pathophysiology

Pododermatitis in birds of prey begins, in almost every case, with focal to diffused pressure sores to the weight-bearing surface of the plantar pad affecting one or both feet. Pressure sores develop gradually, causing devitalization of the integrity of the epidermis and dermis, and eventually the hypodermis (Figure 33.18).[33] This is mainly caused by diminishing blood circulation due to reduced exercise and activity,[34] increased body weight of the bird, inadequate perches, trauma from bating repeatedly on hard surfaces, and dietary regimens.[35] The resulting ischemia ultimately leads to necrosis and the formation of scabbing and, in many cases, callus of different diameters with associated swelling, increased focal temperature, and pain (Figure 33.19).[36] Invading bacterial infection



FIGURE 33.16 Mild reddening and flattening of the epithelium of the plantar pad in a gyrfalcon *(Falco rusticolus)* at the beginning of the training season. This is the result of standing for long periods of time on falconry stands during training sessions, even if lined with artificial turf.



FIGURE 33.17 (A) Puncture wound due to overgrown talons in a hawk. (B) Dorsal digit 1 pododermatitis lesion which developed as a result of poorly fitted and conditioned anklets. The leather had become hard and abrasive. (A, From Blair J: Bumblefoot: a comparison of clinical presentation and treatment of pododermatitis in rabbits, rodents, and birds, *Vet Clin North Am Exot Anim Pract* 16(3):715–735, 2013, with permission.)



FIGURE 33.18 Grade 1 pododermatitis in a peregrine falcon *(Falco peregrinus).* Lesions often develop gradually as a result of diminished circulation, which leads to devitalization and loss of integrity of the epidermis, dermis, and eventually the hypodermis. (Photo courtesy Neil Forbes.)



FIGURE 33.19 Stage 3 bilateral bumblefoot in a saker falcon *(Falco cherrug)* with bilateral bumblefoot. The right plantar pad exhibits extensive necrosis of the epithelium with a large superficial scab and the left foot, smaller scab with the formation of clavus. Even such chronic cases respond well to conservative treatment without the need for any surgical intervention.

occurs as a sequela to focal necrosis. Bacteria commonly isolated from plantar and interdigital abscesses in birds of prey include *S. aureus*, *E. coli*, *Klebsiella*, and *Proteus* spp.[37] Secondary mycotic infection has been reported and appears to be much more prevalent in warm, humid climates (Figure 33.20).

Clinical and pathological findings

Different classifications of the stages of pododermatitis have been proposed. This has resulted in classifications distinguishing pododermatitis into three,[28] four,[38] or five grades or stages,[6] dependent on the severity of clinical and pathological findings. Currently, the most accepted grading system for pododermatitis in birds of prey is the five-stage classification as developed by Remple (see also Table 33.3).[39]



FIGURE 33.20 Infection with *Candida albicans* of the plantar pad in a saker falcon *(Falco cherrug).* The falcon was operated for stage 2 bumblefoot and a dressing was applied. The bandage and the dressing were not replaced as often as they should have with the resulting infection. Such infections are responsible for a number of nonhealing bumblefoot surgical cases but generally respond well to the use of topical antifungal agents.

TABLE 33.3

Pododermatitis Grading Scheme for Birds of Prey by Remple (2006)[39]

Stage	Characteristics
1	◆ Mild reddening of the plantar epithelium ◆ Flattening of the papillae of the weight-bearing surfaces, and plantar pad in particular
2	◆ Mild hyperkeratosis ◆ Necrosis ◆ Formation of scabbing Mild swelling and pain
3	◆ Moderate to severe hyperkeratosis ◆ Necrotic epithelium ◆ Development of medium to large clavi (corns) ◆ Formation of caseous abscess with related moderate to severe swelling ◆ Mild to moderate pain
4	◆ Spreading infection to the tendon sheaths and tendons with ascending infection to the intertarsal joints ◆ Mild to moderate pain
5	◆ Infection affecting the bones including osteomyelitis and osteolysis and/or septic arthritis of the phalangeal and/or tarsometatarsal joints ◆ Severe swelling ◆ Moderate to severe pain

Treatment and prevention

It is always preferable to prevent pododermatitis in captive birds of prey by eliminating or minimizing the causative etiologies rather than having to attempt treatment (Figure 33.21). Different techniques and approaches for the surgical resolution of pododermatitis in birds of prey, and falcons in particular, have been described over the years.[26,28,30,38–41] Prior to surgery, it is essential that a full assessment of the blood



FIGURE 33.21 Early stage 2 bumblefoot in a saker falcon *(Falco cherrug)* with mild to moderate formation of scabs in both the plantar and digital pads. Such cases respond well to improved husbandry allowing the falcon to stand for long periods of time on clean, soft sand such as washed marine sand alternating with salt/disinfectant footbaths.



FIGURE 33.22 Foot secured in surgical restraint. (Photo courtesy Neil Forbes.)

supply and integrity of the surgical site is carried out as it is vital to create edges with full thickness and with adequate blood supply to ensure healing. In all cases, surgical resolution of pododermatitis involves an incision around the scab and clavi and excision of all necrotic tissue through surgical debridement and curettage of the subcutaneous and interdigital spaces. The collection of bacteriological swabs of purulent caseous material for culture and antibiotic-sensitive testing is important to institute adequate antibiotic therapy. Gram stain cytology can also be used to guide first-line therapy. Given the common etiologic agents involved, amoxicillin and clavulanic acid is commonly the first choice of treatment for the authors, pending results of sensitivity testing. Necrosis and inflammation reduce the blood supply to the area thereby limiting the delivery of systemic antibiotics to the region. This, together with granuloma formation, further shields the pathogens from systemic antibiotics.[31] Remple[39] described a four-step approach for the surgical resolution of pododermatitis in falcons. This approach includes the use of systemic antibacterial therapy, intraoperative antibacterial administration, surgical debridement, and postoperative foot casting.[39] The use of custom-made operating tables with foot support to secure the digits intraoperatively may be useful to facilitate the surgical procedure, if available (Figure 33.22).[39,42] Surgical intervention, as described under section Medical Versus Surgical Intervention, is possible in stage 4 pododermatitis cases but with a reserved prognosis. Stage 5 pododermatitis cases, in contrast, are often candidates for euthanasia on humane grounds as digital function will likely remain permanently impaired, with chronic discomfort.

Daily footbaths using a 5% salt solution together with a disinfectant, for example, chlorhexidine or quaternary ammonium compounds, are also beneficial. These can be prepared by procuring an 8-inch diameter plastic tray, such as those used for pot plants. This is firmly secured to a falconry stand with a heavy base to which the bird is tethered. Daily foot bathing can only be performed realistically in tame falconry birds. The footbath is filled with about 1 inch of the solution and the bird is left to stand for 30 to 60 minutes twice daily. A piece of AstroTurf can be placed at the bottom of the tray for comfort. It is important to ensure that birds are fed the correct diet. Any deficiencies identified should be corrected, although these are uncommon if whole carcasses are fed. Balanced vitamin supplements containing vitamin A and biotin have been proposed, with the latter being largely extrapolated from studies in poultry.[43]

Eagles commonly present with pododermatitis, which has been anecdotally suggested to be related to hyperkeratosis and accumulation of flaking skin (Figure 33.23). This accumulation in turn can lead to fissures and secondary infection. For these cases, the authors have found the use of lanolin-based hand cream or ointments to be beneficial.



FIGURE 33.23 Resolving pododermatitis, right foot, following surgical debridement and primary healing in an eagle. On the left foot, a grade 2 pododermatitis with central callus is present. In eagles, these lesions are anecdotally suggested to be related to hyperkeratosis and accumulation of flaking skin. Early stages may respond favorably to lanolin-based topical creams or ointments.

Various forms of foot casting have been proposed for the postoperative care of pododermatitis in birds of prey.[38,41,44,45] The earliest form of foot casting in the postoperative care of pododermatitis in birds of prey included the use of thermoplastic bandaging tape.[38,40] This was followed by custom-made shoes manufactured from bandaging and epoxy-resin[44] or from automotive body filler[41] or the use of composite silicone dental impression material (Figure 33.24).[45] High-grade foam used in the construction of yoga matting or Hexalite is inexpensive, readily available casting materials that are commonly used and favored by the authors.

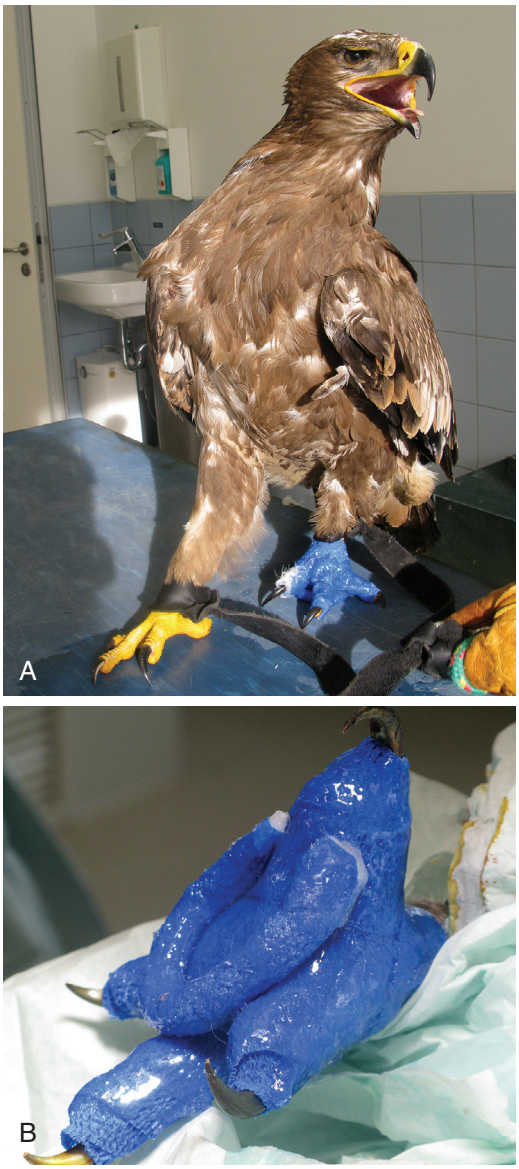


FIGURE 33.24 (A) A 1-year-old female golden eagle *(Aquila chrysaetos)* was presented for bumblefoot. Postsurgically, a custom-made shoe, manufactured from bandaging material and epoxy-resin, was placed around the left foot and was left in place for 2 weeks. After removal of the bandage the wound had almost completely healed. (B) Close-up of the bandage. On the plantar surface a "doughnut" ring was placed to alleviate pressure from the surgical site.

The use of antibacterials, antiinflammatories, and antifungal agents, if necessary, is an essential component in the treatment of pododermatitis in birds of prey.[36] Antifungals are only used in case of cytological or culture evidence of secondary mycotic infection. The use of intralesional antibacterial therapy in pododermatitis in the form of antibiotic-impregnated[26] calcium sulfate beads, or antibacterial gels[46] appears to be effective in controlling localized infections together with systematic antibiotics. This concept was originated from the use of antibiotic-impregnated polymethyl methacrylate beads in the treatment of chronic osteomyelitis and cellulitis in a juvenile bald eagle *(Haliaeetus leucocephalus)*.[47] Beads should be removed after treatment to prevent them from acting as a foreign body and potentially decreasing foot comfort. If beads can be removed from a dorsal-lateral/medial approach, this is preferred as it prevents the need for a further plantar surgical wound.

Dressings and bandaging are also essential in the postoperative care of cases of pododermatitis in birds of prey.[48] Zsivanovits and Monks[31] suggested that the addition of dimethyl sulfoxide (DMSO) in the dressing facilitates the penetration of topical antibiotics and helps reduce swelling. However, *Candida albicans* is a yeast commonly invading surgical sites covered with dressings and protected by bandaging if sterility of the surgical site was not preserved or if the dressing was not changed and replaced with more frequency. While not proven, the authors suggested that secondary fungal overgrowth of bandaged pododermatitis lesions is more common in hot and humid climates. Recent studies in cadaver peregrine falcon *(Falco peregrinus)* feet have used force pressure sensors to suggest that snowshoe dressings are preferable to interdigital bandages at relieving pressure from the plantar pad.[49] However, it has yet to be determined if this finding is repeatable in live birds and clinical cases.

Some species of raptors, particularly those considered more intelligent species such as Harris' hawks and caracara (*Caracara* spp.), will often chew and remove protective and supportive foot dressings. A smooth, round orthotic formed from dental acrylic (Kooliner, GC Europe, Leuven, Belgium) and molded around the tip of the maxillary beak limits a raptor's ability to pick successfully at dressings (Figure 33.25).[50] As this will affect a bird's ability to tear at the food, hand feeding or chopping of the food will be required. One author has used a film/plastic disk fixed around the tarsometatarsus to reduce the incidence of self-trauma.

Exercise, perching, and environment are all important factors in managing pododermatitis in raptors. Excessive body condition, inactivity, and inappropriate perching all predispose and will delay recovery. If possible, a raptor should be flown and/or exercised while under treatment as this promotes circulation and ensures body condition is managed. If daily treatment (particularly topical administration of medication) is required, it may be appropriate to tether a (trained) raptor during treatment (see Chapter 55). However, as minimal welfare intrusiveness is the goal, each case needs to be individually assessed for its potential adverse effects on the patient. Perching should be critically analyzed. If pressure-related pododermatitis has developed, then perching changes will be required—and if these are not made recurrent disease will almost certainly occur. Molting, which typically occurs in the spring/summer at the end of the breeding season, leads to



FIGURE 33.25 Dental acrylic maxillary blob is used to prevent dressing interference. This technique appears particularly useful in Harris' hawks *(Parabuteo unicinctus)* to prevent picking at a foot dressing.



FIGURE 33.26 Image of the skin 21 days postoperatively and 7 days after suture removal following surgical debridement of grade 3 pododermatitis in a Chaco owl *(Strix chacoensis)*.

reduced activity in captive birds. The authors have seen a pattern where birds who have been affected by pododermatitis had an increased chance of reoccurrence if free lofted in a pen to molt. Encouraging owners to continue to exercise their birds during the molt may thus help prevent this problem.

Conclusion

Pododermatitis in birds of prey is only observed in captive birds used in captive breeding programs and those used for falconry and is best described as a management-related medical condition. Surgical failure and recurrence of pododermatitis in birds of prey are high and have been attributed to the failure of the operator to recognize the granulomatous nature of this condition and the etiological causes.[39] A four-stage therapeutic approach has been proposed as the most successful in resolving pododermatitis in birds of prey.[39] This includes (1) systemic antibacterial therapy, (2) direct intralesional antibacterial therapy, (3) surgical debridement, and (4) postoperative protective foot casting.[39] When managing the condition, the anatomy and structure of the plantar aspect of the foot should be returned as close to the normal shape as possible, with as little scar tissue as feasible, which helps prevent recurrence of the condition (Figure 33.26).

In almost every case of stage 1 and stage 2, and in many cases of stage 3 pododermatitis, the best intervention is not to intervene surgically and to opt for conservative treatment (Figure 33.27). This is probably the most indicated form of therapy in the long term.

Anseriformes

Introduction

Incorporated within the order Anseriformes are three families: Anhimidae, Anatidae, and Anseranatidae. The family Anatidae contains over 170 species of waterfowl including ducks, geese, and swans, which are the most common Anseriformes kept in captivity. The pygmy geese (*Nettapus* spp.) are the smallest, weighing only 300 g, whereas the trumpeter swan *(Cygnus buccinator)* is the largest of the waterfowl and can weigh up to 17 kg. Anatidae are found all over the world apart from the Arctic.

Different waterfowl use different feeding strategies: some spending the majority of time on water, with others spending significant periods on land. This needs to be considered when evaluating husbandry and environmental conditions in cases of pododermatitis. Birds whose wild ancestors predominately spent time on the water in their natural range would be at high risk for pododermatitis if they are forced into terrestrial living and unable to freely spend time on water.

Dabblers are waterfowl that feed primarily on the surface of water or graze under shallow water. Traditionally, this group is assigned to ducks from the subfamily Anatinae. These birds rarely dive and tend to have their legs placed more centrally on their body, walk well on land, and even feed terrestrially. Mallards *(Anas platyrhynchos)*, teals (*Anas* spp.), widgeons (*Mareca* spp.), and most swans (*Cygnus* spp.) are dabblers.

Divers, as their name suggests, feed primarily under the water. Their legs and feet are situated more caudally, and they spend little or no time on the ground. Examples include the ruddy duck *(Oxyura jamaicensis)*, marbled teal *(Marmaronetta angustirostris)*, and the rest of the *Aythya* genus. Captive management schemes that result in having these species spending excessive periods on land is a risk factor for developing pododermatitis.

The most common grazers are geese (*Anser* spp.). These birds are terrestrial spending most of the time on land. In the wild, geese are frequently found grazing arable land where



FIGURE 33.27 Grade 2 pododermatitis before (A) and directly following (B) 2 weeks of treatment with topical Preparation H and changed perching. In early stage pododermatitis, conservative treatment can lead to full resolution (C).

they can be considered a pest species such as Canada geese *(Branta canadensis)* in Europe.

The final classification is perching ducks—which typically are long legged and are adapted to allow perching or roosting in trees. Mandarin *(Aix galericulata)* and wood ducks *(Aix sponsa)* are typical examples.[51]

General risk factors for pododermatitis in waterfowl include housing with excessive areas of hard standing (e.g., concrete), abrasive surfaces, interspecies disturbance/bullying and introducing new birds to existing group (leads to increased exercise/chasing), poor hygiene, excessive body condition, and skeletal deformities leading to uneven pressure.

Pathophysiology

Waterfowl have palmate feet to help them swim. The first digit or hallux is vestigial, and the birds have a relatively large plantar pad.

In captivity, one or more husbandry or environmental factors are often responsible for the development of pododermatitis. Among these are a lack of opportunity for swimming, obesity, contralateral limb injury causing uneven weight bearing, and standing on hard/abrasive substrate (concrete/compacted earth).[52] The disrupted epidermis and presence of gradual avascular necrosis, predominately affecting the plantar pad but on occasions also the digital pads, provide favorable conditions for microbial entry and allow for invasion of secondary pathogens. In the authors' experience, this most commonly includes bacteria such as *E. coli*, *Staphylococcus* spp., *Proteus* spp., *Aeromonas* spp., and *Pseudomonas aeruginosa*, although occasionally fungal organisms (e.g., *C. albicans*) are also involved. Poor hygiene also predisposes to such secondary infections.

Clinical and pathological findings

Pododermatitis in Anseriformes can be graded into five classifications, as has been described for raptors (Table 33.3). However, many authors simplify the classification system into three grades—mild, moderate, and severe (Table 33.4)[52]—as treatment options vary little between grades 1 and 2 or grades 4 and 5. It is very common to find grade 1 or mild pododermatitis as an apparent "asymptomatic" finding in captive waterfowl, with the birds showing no apparent discomfort and the condition only being noted on close inspection of the foot. Educating owners and keepers to closely monitor foot health and instigate measures before the disease progresses beyond this stage is vital in preventing more serious diseases. In early disease, hyperemia and erythema are associated with a shiny appearance of the metatarsal and digital pads. As disease progresses to moderate disease, fissures and large callous formation of the foot pads are particularly common as hyperkeratosis develops and lesions often become proliferative (Figures 33.28 and 33.29).

Treatment and prevention

Collection of a thorough history is vital when managing a case of pododermatitis in waterfowl. Any potential husbandry deficiencies should be identified and corrected. On-site visits can be very useful—if not, photographs or videos of the enclosure/housing can be used. In cases where substrate is too hard, sharp, or abrasive, this should be corrected. In many cases, natural substrates such as grass or sand are preferable to

TABLE 33.4

Three-Grade System for Pododermatitis in Waterfowl

Mild		Hyperkeratosis and erythema but no Infection
Moderate		Callous and infection but no skeletal involvement
Severe		Osteomyelitis



FIGURE 33.28 Grade 3 pododermatitis with hyperkeratosis, moderate swelling, and central caseous core in a Sebastopol goose *(Anser anser domesticus)*. In waterfowl, the plantar pad is most commonly affected, though on some occasions the digital pads can be involved too.



FIGURE 33.29 Radiograph shows periosteal reaction and severe soft tissue swelling of the proximal phalanx of digit 4 in a domestic goose *(Anser anser domesticus)* resulting from severe, grade 3 pododermatitis. Morphologic appearance is shown in Table 33.4.

artificial materials.[31] The birds should be observed to assess the duration of time in water and on land, ensuring it is appropriate for the species. If water is not being utilized by the birds, this warrants further attention and investigation; factors such as excessive stocking density, flock hierarchy, (aerial and terrestrial) predators, and multispecies interaction can affect

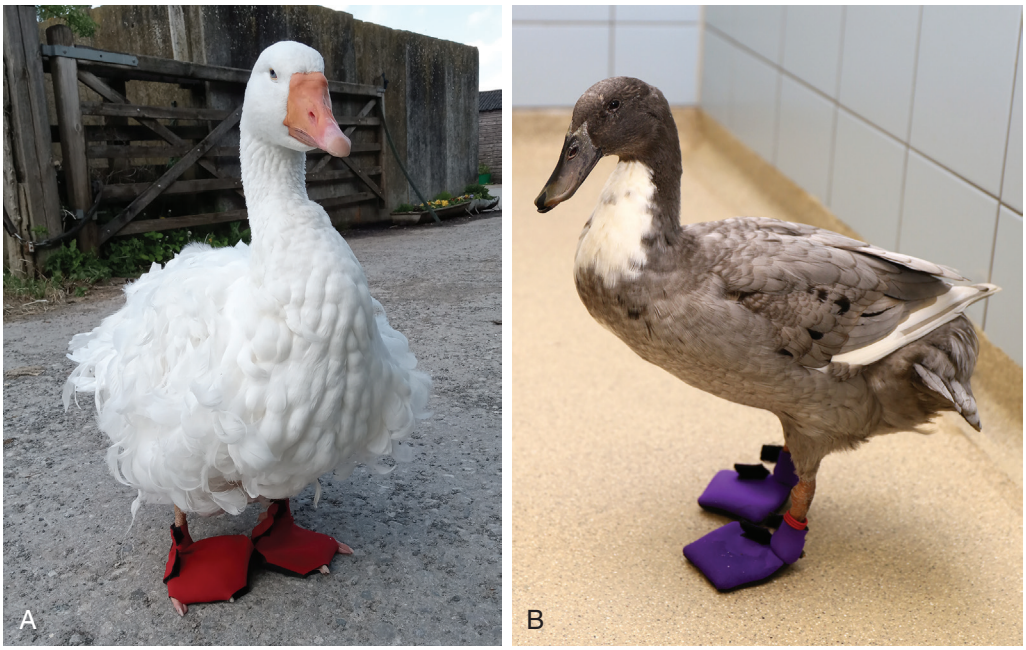


FIGURE 33.30 Sebastopol goose *(Anser anser domesticus)* (A) and female Indian runner duck *(Anas platyrhynchos)* (B) wearing removable waterproof neoprene protective shoes (Birdy Bootie, Crazy K Farm Pet and Poultry, USA). Such neoprene shoes are particularly useful in waterfowl where contact with hard abrasive surfaces like concrete is possible. In addition, the shoes can be beneficial to alleviate pressure in the case of (chronic) bumblefoot, as was the case in the Indian runner duck.

the use of available swimming water. Diet should be assessed and supplemented or converted if required. Vitamin A deficiency has been associated with chronic epithelial conditions such as pododermatitis and is associated with feeding a high-grain diet.[53] The body's condition should be assessed, and feeding should be addressed in case of obesity.

If disease has progressed beyond mild/grade 1 lesions, husbandry modifications alone are unlikely to be effective to resolve the pododermatitis. In such instances, topical foot treatments with agents such as chlorhexidine, povidone iodine, and magnesium sulfate solutions can be utilized. However, little data exist to support the use of one over the other. Topical antimicrobial treatments such as silver sulfadiazine may be sufficient in early cases, but systemic therapy is required for moderate and severe cases. Systemic antimicrobials should be selected based on culture and sensitivity, where possible. Bimodal antibacterial therapy (e.g., aminoglycosides combined with penicillamine or trimethoprim-sulfonamide) may be indicated, although restrictions may apply in food-producing animals. Pressure-relieving dressings such as doughnut type or ball bandages can help the skin to heal and keep the area clean and free from environmental contamination. However, maintaining dressings can be particularly challenging in waterfowl that need regular access to bathing facilities for maintaining plumage quality. If ambulation on hard and/or abrasive surfaces cannot be avoided, neoprene boots can be fastened to the feet to protect the plantar and digital pads. These are used frequently, particularly by owners of heavier bodied waterfowl. Velcro fasteners allow easy fitting and removal. These are commercially available in the US (Birdy Bootie, Crazy K Farm Pet and Poultry; crazy-k-farm.myshopify.com; Figure 33.30). One big advantage of neoprene is that it does not need to be removed before swimming. Commercial and home-made devices can be made to the correct size for bird feet. Each bird should ideally have two pairs so that one can be washed and disinfected while the other is in use, as this will help prevent the boot from becoming a fomite for infection. Allowing birds to continue to swim takes weight off the feet and may thus expedite lesion resolution. In advanced cases, surgical debridement may be required to debride necrotic tissue and remove caseous material with its associated pyogenic membrane. The decision to operate on waterfowl is a difficult one, as surgical wounds can be challenging to heal in aquatic birds. Modern waterproof dressings or artificial skins can be applied to surgical wounds. Equally, delaying surgery can allow the progression to deeper infection, osteomyelitis, and septic arthropathy. Following surgery, a period of minimally 5 days, in which swimming is avoided, is recommended to allow the open wound to heal and prevent secondary contamination of the wound bed. Dependent on the complexity of the case, this period could be extended. Given the well-developed medial metatarsal vein and featherless skin below the intertarsal joint, limb perfusion antibiotics are also an excellent option to consider in Anseriformes (see section on Regional Limb Perfusion and Chapter 36).

A technique has been presented for the surgical lavage of chronic septic arthritis lesions in the foot of Anseriformes. The procedure is done separately to planter approach to the pododermatitis lesion to avoid causing disruption to the blood supply. A dorsal approach is made to the affected interphalangeal joint(s)—or tarsometatarsal phalangeal joint (Figure 33.31). Inspissated material is curetted, and samples



FIGURE 33.31 Pekin duck *(Anas platyrhynchos)* with severe grade pododermatitis of the plantar pad, and concurrent osteomyelitis and septic arthritis (A). Such cases can be successfully treated with surgical lavage. For this purpose, the affected joint is accessed from (latero)dorsal, following which the necrotic/purulent debris can be removed from the joint using a curette. If needed, multiple incisions can be made. Once all debris is removed, a small silicone tube (e.g., butterfly catheter tubing) is tunneled through the joint and sutured in place to enable repeated flushing of the joint (B). A protective layer of bandage can be used to prevent the bird from chewing the tubing. Flushing of the joint for several days will help clear the joint infection and can result in complete resolution of the infection (C). (Photos courtesy Scott Echols and Avian Studios.)

are collected for cytology and culture. Sterile plastic giving set tubing with a luer attachment and preplaced ports is sutured in place between the surgical sites allowing repeated lavage to be performed over several days.

Galliformes

Introduction

The order Galliformes contains 290 species across five families. The most common in captivity are Phasianidae (including the domestic fowl, quails, partridges, pheasants, turkeys, peafowl, and grouse), Odontophoridae (New World quail), and Numididae (guineafowl). Cracidae (including chachalacas and curassows) and Megapodiidae (megapodes) are certainly less common.

Galliformes are kept in captivity for many reasons; the largest percentage of captive birds are domestic poultry kept for food production, typically on a commercial scale; however, backyard poultry keeping has boomed in recent years. Galliformes are also kept in zoological collections or as ornamental pets. Husbandry varies widely from commercial environments to more naturalistic enclosures. Within commercial poultry there is a move toward free range production—although cage and barn systems still exist in many countries.

Husbandry will vary depending on the species being housed. The majority of commonly kept poultry (chickens, guinea fowl, turkey) roost at night and will require appropriate daytime floor-level substrate and suitable perching, both having an effect on foot health. When choosing floor substrate, often a compromise between easy to clean/biosecure options and comfort for birds is required.

As well as being a significant welfare concern in commercial poultry, pododermatitis can cause severe economic losses during production. Severe cases can lead to secondary infections, poor carcass development, and carcass rejection at slaughter. Chicken feet are a commonly exported byproduct of broiler production but will be rejected where pododermatitis is present.

Pathophysiology

As in many other species, galliform pododermatitis features hyperemia, hyperkeratosis, and avascular necrosis of the epidermis. If disease is allowed to progress, ulceration and inflammation of the subcutaneous tissues occur with or without abscessation and fistula formation. Rather than pressure necrosis due to poor perching being the primary etiology, as seen in many avian species, it has been proposed that frequently, poultry disease is caused because of contact dermatitis due to poor bedding/substrate. Lesions have, on occasions, been termed ammonia burns and are believed to be caused by a combination of moist litter and chemical irritation of the skin due to a high ammonia content of the litter. Organic material in urine and feces accumulating in litter is converted to ammonia under anaerobic conditions and to nitrate under aerobic conditions. Wet substrates and a humid environment will provide conditions that favor attachment of litter to the feet and formation of ammonia due to the anaerobic conditions.[54] Bedding materials with sharp edges (large particle size wood chips, chopped straw, etc.) may contribute to foot pad dermatitis (FPD) through their abrasive action, however.[55]

Proven risk factors for development of pododermatitis include:

- bedding deeper than 5 cm;
- wet litter;
- use of drinking cups instead of nipple drinkers;
- winter weather;
- poor ventilation;
- genetic susceptibility (within lines of commercial chickens).[56,57]

Broiler birds are at higher risk of pododermatitis due to their high body condition scores and conformation. Pododermatitis incidence and severity were found to be increased following feeding with high-protein diets in broilers. When managing litter ammonia levels associated with higher stocking density, sodium bisulfate used as a litter amendment reduced volatile ammonia levels and significantly lowered the incidence and severity of pododermatitis. Male broilers have been shown to be more predisposed to pododermatitis than females.[58]

Many lesions show no histological evidence of bacterial or fungal infection.[55] Where secondary bacterial infection occurs, *S. aureus* (68%), *Enterococcus faecalis* (14%), and *E. coli* (9%) have been shown to be most prevalent.[59] Secondary mycotic pathogens are also reported, with *Cryptococcus* spp. and other opportunistic fungal pathogens have been cultured from pododermatitis lesions.[12] Most pathogens (whether bacterial or fungal) will be secondary to the underlying cause of pododermatitis. However, they require appropriate treatment—ideally based on culture and sensitivity for successful management. Choice may be limited due to antibiotic restrictions that apply to poultry. Topical treatment with betadine combined with trimethoprim-sulfonamide or amoxicillin-clavulanic acid is a common first-line treatment used in poultry by the authors.

Clinical and pathological findings

Pododermatitis lesions in Galliformes are described similarly to other avian species (see Table 33.3). Lesions can be varied, with hyperkeratosis, abrasion, ulceration, necrosis, and abscess formation, with or without the formation of a fistula canal being most common (Figure 33.32).[60] In commercial poultry, similar lesions are also seen on the hock and overlying the pectoral muscle and have been associated with wet/high ammonia litter and high body condition in boiler birds.[61]



FIGURE 33.32 Various grades of pododermatitis in the domestic chicken *(Gallus domesticus)*. (A) Mild, grade 1 pododermatitis with a small surface lesion and hyperkeratosis of the plantar foot pad. (B) Moderate, grade 2 pododermatitis with a medium-sized lesion, with erosion of the surface and central callus formation. (C) Severe, grade 3 pododermatitis with significant soft tissue swelling and central callus formation. These lesions are usually large, deep, and ulcerated.

One of the commonly utilized foot scoring systems was described by Berg[62] who classified pododermatitis lesions in poultry into a three-point system:

- 0—no lesion;
- 1—mild erythematous lesion;
- 2—severe erosive lesions.

Many other systems exist with three-, four-, and six-grade systems being proposed.[63,64] Visual scoring systems are unable to assess the depth of lesions—and it is suspected that depth is more significantly correlated with lesion discomfort than width. Depth is more readily assessed microscopically. However, this renders this type of grading scheme more suited to post-slaughter assessment than for companion birds such as the five-grade histological scoring system.

A recent publication described the attempts that were made to validate a four-point grading scheme by comparing macroscopic and microscopic findings of pododermatitis lesions in poultry (Box 33.4). Results of this study showed presence of a strong correlation between lesion width and lesion depth, and between hyperkeratosis and lesion depth. In addition, the study evaluated intra- and interrater reliability, which showed the greatest variability between assessors when having to distinguish between grade 3 and 4 lesions. This strengthens the arguments for use of a three-grade system.[8]

In commercial production units, automated foot scoring systems now exist which are less subjective and shown to be more accurate than human scoring. Thermography has shown to be more sensitive than visual examination alone at detecting early or subclinical pododermatitis lesions (see Chapter 3).[65]

Treatment and prevention

If a deficiency in substrate or environment is noted, this should be corrected. A study investigating substrate in the broiler industry found that substrate that was either highly absorbent (e.g., wood shavings) or quick-draining and easily able to release moisture (e.g., rice husks) provided the best results regarding preventing pododermatitis.[55] Good litter management and hygiene will help to reduce the risk of secondary infection. Wood or paper-based bedding materials designed for horses that are low dust, hypoallergenic, and resist mold growth can be effective choices.

As with other species, obesity and limb injury resulting in uneven weight distribution to the feet can predispose to pododermatitis. Backyard poultry in particular are kept up to much greater ages than commercial poultry.[66] Degenerative arthropathies can alter weight bearing and predispose to pododermatitis lesions. Exercise can be useful to both stimulate blood flow to the feet and contribute to weight loss, which removes the load on the feet.[67]

BOX 33.4 FOUR-POINT GRADING SCHEME FOR VISUAL SCORING OF PODODERMATITIS IN GALLIFORMES

Score 1: No lesion, no hyperkeratosis, no ulcers (original score 0).
Score 2: Small lesion on the surface (original score 1).
Score 3: Medium lesion, with erosions only in individual cases (original score 2a).
Score 4: Large and deep lesion, ulcers, and scars (original score 2b).

Many pododermatitis lesions—particularly low grade—are not secondarily infected. Litter management, pressure-relieving dressings, and topical antiseptics/antimicrobials can be used. Where secondary bacterial or mycotic infections are identified, treatment should be based on culture and sensitivity. Long-term antimicrobial administration has been facilitated in poultry by using a vascular access port.[68] Intravenous and intraosseous regional limb perfusion has also been tested in poultry and is another method of delivering high concentrations of antibacterials to the foot while decreasing the risk of systemic side effects.[69]

Surgical management should be restricted for high-grade lesions where necrotic tissue and caseous abscess require removal. Lesions should be debrided with care so as not to damage important soft tissue structures such as flexor tendons but should also remove pyogenic membranes as completely as possible. Primary closure following debridement is preferable to allow more rapid healing—but is not possible where insufficient skin is present—and healing by secondary intention may be required. Advanced flaps have been used in Falconiformes and can be adapted for use in Galliformes.

The use of vitamin and mineral supplementation has been investigated for their role in preventing pododermatitis lesions. The picture is unclear, with early studies showing biotin supplementation as potentially effective in reducing onset of lesions,[70] whereas in turkey poults, biotin supplementation made no difference to lesions.[71]

Psittacine and Passerine Birds

Introduction

Psittacine birds are very popular cage and aviary birds. They are equipped with strong legs, upright stance, and zygodactyl foot structure, giving them a strong grip and ease of perching. There are nearly 400 species of psittacine birds, weighing from <50 g up to 2 kg. Psittacine birds are known for their social nature and high levels of intelligence and are commonly kept within the home, resulting in potential exposure to some unnatural substrates on which to perch, and feeding of unlimited quantities of energy-rich diets, which predisposes to obesity and nutritional deficiencies that can increase the risk for pododermatitis.

The order of passerine birds covers over 50% of known avian species. Only a small number of species are kept in captivity—these being common aviary birds in private and zoological collections. Canaries *(Serinus canaria)* and finches (*Fringillidae* spp.) are the most frequently kept passerine species. They have an anisodactyl foot structure to give stable perching. Corvids, in particular the common raven *(Corvus corax)* are the largest passerines—weighing up to 2 kg body weight, with the smallest passerines weighing only a few grams. The taxonomic orders of Psittaciformes and Passeriformes include a large number of species that each have their unique, species-typical behaviors, but the heavy bodied birds (e.g., *Amazona* spp.) that are prone to a more sedentary lifestyle when maintained in captivity are predisposed to pododermatitis.

Pathophysiology

The most common cause of pododermatitis is poor perching or cage floor substrate. Perches with too narrow or too wide diameters can lead to poor weight distribution on the foot.

Typical recommendations include a diameter size where the foot is wrapped for approximately two-thirds to three-fourths around the perch. It is advisable to have variable perching surface and diameters within this range to provide choice and variety for the bird. Smooth perches that tend to create focal pressure are also linked to the development of lesions. An area of pressure necrosis develops, the reticulate scales become noticeably smooth and flattened, and marked erythema is seen. Sandpaper perches, while commonly recommended to keep nails short, may create areas of pressure and avascular necrosis on the skin, particularly in smaller passerine birds.[72,73] As lesions progress, ulceration and hyperkeratosis are frequently seen. Many birds will prefer a particular perch location over an appropriate perch type or size, requiring perch rotation. For communally housed birds, care should be taken to ensure that sufficient perching space is provided.

Dynamics between groups of housed birds can lead to subordinate birds being forced off the most comfortable perching. Overgrown claws can lead to punctures of the plantar surface or altered weight bearing on the feet. Many owners use sandpaper or concrete perches with the aim of correcting overlong claws, but these may exacerbate pododermatitis due to abrasion causing irritation and erythema. Sandpaper cage floor coverings are commonly used with small passerines and may exacerbate foot disease.

Both psittacine and passerine birds are prone to malnutrition in captivity. Seed-based diets have been shown to be deficient in vitamin A.[74] Hypovitaminosis A in turn can predispose to hyperkeratosis, loss of skin elasticity, and a weakened immune system.[75]

As with many avian species, excessive weight is a factor. Large bodied psittacine birds, for example, *Amazona* spp. appear predisposed to the development of pododermatitis.[76] Pain associated with lesions often leads to further reduction of activity and worsened situation. Many pet psittacine birds suffer from obesity due to a combination of excess nutrition and inactivity. Wing clipping of birds (a largely outdated practice) prevents flight, which is one of the most effective forms of exercise. As birds enter the geriatric life stage, the prevalence of osteoarthritis significantly increases. The resulting uneven use of the pelvic limb and feet is another leading cause of pododermatitis.

Clinical and pathological findings

Although pododermatitis is a frequent clinical finding in psittacine birds and occasionally passerine birds, no specific grading or classification of lesions has been accepted for either order. Most clinicians therefore adopt the 1 to 5 scale as used in Falconiformes (see Table 33.3), although it is uncommon to see advanced grade 4 to 5 lesions in psittacine and passerine birds.

Two lesion types seem to predominate in low-grade psittacine pododermatitis, empirically with some species developing one more frequently than the other. *Amazona* spp., in particular, seem to develop thinning of the skin with a loss of keratinization (Figure 33.33A). *Psittacus* spp., in contrast, develop hyperkeratosis and fissures (Figure 33.33B).

In psittacine and some passerine birds, plantar foot lesions are frequently accompanied by lesions on the plantar aspect of the intertarsal joint. Birds with sore feet, arthritis, or inadequate perching will frequently sit down on their hocks, predisposing to the tarsal lesions (Figure 33.34).



FIGURE 33.33 Typical low-grade pododermatitis lesions in parrots include thinning of the skin with loss of keratinization, as is seen in an Amazon parrot (*Amazona* spp., A), and hyperkeratosis and fissures, as seen in a conure (*Pyrrhura* spp., B).

Treatment and prevention

Treatment involves correcting underlying husbandry deficiencies and managing the lesions. Perching should be selected to provide a choice of varying diameters. Generally, the digits should grasp roughly two-thirds to three-fourths around the perch. Natural surfaces such as fruit tree branches promote better foot health then smooth doweling type perches. In some cases, it may be necessary to wrap the perch with soft padded material (e.g., Vetrap) to relieve the pressure. In some instances, pressure-relieving bandages (as used in raptors) may be required. Perch rotation can encourage perching on different surfaces.[73] Perch and substrate hygiene should be kept optimal to reduce the chance of secondary bacterial infection. Remote observation is key to investigate the bird's use of perches as well as its behavior in its environment, based on which behavior change can be instigated to promote appropriate use of perches,



FIGURE 33.34 Pododermatitis affecting the hock in a Senegal parrot *(Poicephalus senegalus)*. Such lesions are typically seen in birds with arthritis or painful feet but can also result from inadequate perching.

if required (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).

The prevalence of pododermatitis in psittacine birds increases with age. Arthritis, particularly affecting the stifle and intertarsal joints of psittacine birds, affects weight bearing. Modified perches may be required for geriatric birds—some manage better on shelf perches, or with padded perching surfaces.

If weight is identified as a contributing factor to the pododermatitis, a gradual weight loss program will be required. Dietary conversion away from a nutrient-deficient diet, or addition of reliable supplementation, may also need to be considered. Environmental enrichment should be assessed and implemented. Enriched birds will be more active, less sedentary, and likely have improved body conditions. Foot toys, foraging toys, and the provision of a foraging tree encourage the bird to move around the enclosure (see Chapter 47).[77]

Psittacine and passerine birds with pododermatitis very rarely require surgery as most birds will respond to medical treatment. Topical or, in more advanced cases, systemic antibiotics can be used and follow guidelines such as listed for Falconiformes. Effective analgesia is vital and reduces the risk of self-mutilation.

One complicating factor to consider when treating psittacine birds is that they will frequently interfere with and remove dressings. In cases where dressings are required, strategies may therefore need to be employed to prevent interference. While (temporary) collar placement can help to effectively prevent bandage destruction, the use of less-intrusive options that redirect the behavior (e.g., distractor tabs, enrichment of chewing/foraging behaviors) is highly preferred and should be used as the first-choice method, whenever possible.

Sphenisciformes

Introduction

Penguins are a group of flightless aquatic birds belonging to the order of the Sphenisciformes living primarily in the Southern Hemisphere, with the exception of the Galapagos penguin *(Spheniscus mendiculus)*, which lives north of the equator. The order of the Sphenisciformes comprises one family, the Spheniscidae, comprising 6 genera, 17 species, and 26 taxa. Penguins are among the most popular birds kept in zoological collections. The largest living species is the emperor penguin *(Aptenodytes forsteri)* weighing 19 to 46 kg and standing 1.12 to 1.15 m in height, whereas the smallest species is the little blue or fairy penguin *(Eudyptula minor)* that weighs on average 1 kg and stands 0.40 to 0.45 m in height.[78]

Penguins are pelagic birds that spend up to 75% of their lives living in the open sea, coming to shore only during the breeding and molting seasons.[78] In captivity, however, penguins are confined mostly to small enclosures provided with ponds and a beach and rockery with concrete flooring. Under such conditions, penguins are predisposed to pododermatitis due to the sedentary lifestyle, reduced time swimming, prolonged length of time spent standing on hard and abrasive flooring, and nutritional deficiencies.[79–82]

Pathophysiology

Penguins possess palmate webbed feet with four digits, but the first digit or hallux is only vestigial, and a relatively large and voluminous plantar pad. The tarsometatarsi are short, and the legs are placed far back on the body, which makes the birds stand upright.[78] The increased pressure of the weight of the bird on the plantar pad of the foot makes penguins more predisposed to develop pododermatitis.[79]

In captivity, penguins develop pododermatitis as a result of gradual avascular necrosis of the weight-bearing surfaces, mainly of the plantar pad and, less commonly, of the digital pads. This is mainly due to the lack of exercise and the result of standing on hard and abrasive flooring for long periods of time. Other predisposing factors include poor floor hygiene and nutritional deficiencies.[79,82] While concrete surfaces are commonly listed as a risk factor, pododermatitis is a seemingly rare phenomenon in certain zoos, despite the penguins being housed in a concrete surfaced enclosure. Possibly, this is due to the unlimited access to a large, long swimming pool that promotes swimming activity (Adam Naylor, personal communication, 2023). As such, these observations emphasize the multifactorial origin of the condition.

Clinical and pathological findings

Pododermatitis of penguins is a progressive clinical disease affecting the weight-bearing plantar pad of the foot and digital pads. The condition in penguins is characterized by the presence of flattened and thinner epithelium, hypertrophy of the epithelium, superficial to deep ulcerations leading to clavi, single central caseous to multiple digital abscesses, mild to severe swelling, increased localized temperature, and mild to severe pain (Figure 33.35). In penguins, the three-stage classification has been favored by several authors as this seems to conform well to the clinical and pathological findings commonly observed in individuals from the different penguin species maintained in captivity (Table 33.5).[79,81]

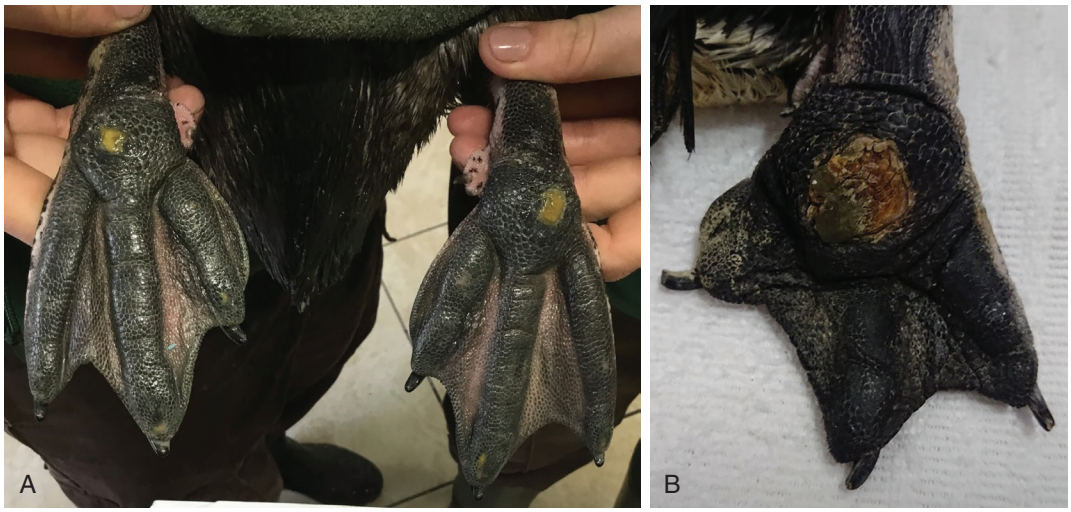


FIGURE 33.35 (A) Bilateral, mild to moderate ulcers on the plantar pad of a 5-year-old captive Humboldt penguin *(Spheniscus humboldti)*. This could be considered a transition stage between grade 1 and grade 2 bumblefoot. This type of bumblefoot should be treated as early as possible to stop developing into a more serious condition. (B) Severe and extensive necrosis of the plantar pad with the formation of deep infected clavus in an African penguin *(Spheniscus demersus)* admitted to a rescue and rehabilitation center. Such rescue centers should provide different flooring surfaces and increase activity in order to prevent the occurrence of bumblefoot and delay releasing individuals back to the wild.

TABLE 33.5

Three-Stage Classification Scheme for Pododermatitis in Penguins

Stage 1	Flattening and thinning of the epithelium of the weight-bearing surface, most commonly, of the plantar pad. Hypertrophy of the epithelium leading to clavi can also be present.[79]
Stage 2	Presence of infection of the dermis and soft tissue of the plantar pad with a variety of bacteria. Foot temperature can be increased and mild to moderate pain and subsequent lameness are usually present.
Stage 3	(Chronic) infection which not only affects the pad but also extends to the tendons, bones, and/or joints.[79]

The lack of uniform and universal description and classification of pododermatitis in penguins has made identification of early detection of disease difficult, resulting in the delay in the establishment of adequate therapy measures.[82] It has also been suggested[79] that the etiological factors leading to the development of pododermatitis in penguins may be similar to those described in detail by Harcourt-Brown[36] in raptors.

Treatment and prevention

Over the years, different treatment protocols have been undertaken, including cleansing the feet with a 2% chlorhexidine solution, footbaths with chamomile tea followed by the application of a cream containing DMSO, escin 20% solution, dexamethasone, prednisolone, and lidocaine hydrochloride.[81] The possible immunosupressive affects of steroidal medication must be considered before prescribing, even when used topically. In addition, other more invasive protocols have included the use of surgery and debridement, followed by bandaging and dressing containing a topical solution of enrofloxacin, DMSO, and dexamethasone and the use of neoprene shoes fitted with Velcro straps for a period of up to 3 months to ease the healing process (Figure 33.36).[79]

Currently, environmental enrichment seems to be the most popular long-term option to manage pododermatitis in collections of captive penguins (Figure 33.37).[81,83–85] Measures introduced into the environment and changes into the daily routine included reinforcing the penguins to stay one additional hour in the water through the provision of play objects such as colored balls and rings, ice blocks with fish, natural shells, and live fish, gradually introducing such items on a daily basis.[81] Another measure is the placement of structures within the enclosure and stimulation for the penguins to climb or swim through these. This has been used with different degrees of success to increase the time spent in the water.[84]

Behavioral husbandry has also been proposed as an aid to manage pododermatitis in captive penguins.[86] This consists mainly of reinforcement of voluntary participation by individuals in their routine care and using operant conditioning through the development and reinforcement of desirable human-animal interactive relationships.[86]

Phoenicopteriformes

Introduction

Flamingos are large birds with long necks and legs and a distinct pink plumage. One unique anatomical feature particular to flamingos is the downcurved beak adapted for filtering feeding. The neck and the legs are longer in relation to the body size than in any other group of birds. The extreme

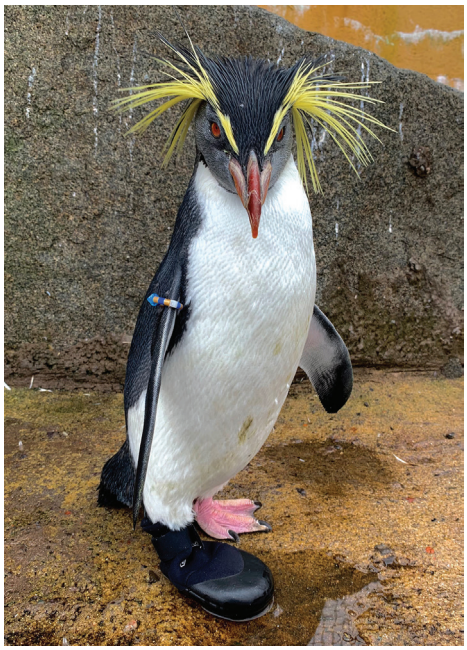


FIGURE 33.36 In penguins, neoprene protective boots that are commercially sold for small dogs are a suitable option for providing cushioning for mild pododermatitis lesions and for additional protection of dressings that may be applied in cases of severe pododermatitis or following surgery. In this elderly rockhopper penguin *(Eudyptes chrysocome)* the boot was placed unilaterally to aid healing of a pododermatitis lesion that was suspected to have developed as a consequence of osteoarthritis and associated abnormal posture and uneven weight bearing. The bird was restricted from swimming while wearing the boot. (Photo courtesy Adam Naylor, Royal Zoological Society of Scotland, Edinburgh Zoo.)



FIGURE 33.37 A captive 27-year-old African penguin *(Spheniscus demersus)* was previously affected with severe, stage 2 bilateral bumblefoot. The feet developed extensive necrosis of the plantar pad occupying over 50% of the total surface after years of standing on hard and abrasive flooring. The image shows the feet greatly improved after 4 months of environmental enrichment and improved behavioral husbandry.

length of the legs and the reduced feathering on the tarsometatarsi allow flamingos to wade deeper than any other wading bird, allowing them to go as far as the lower abdomen. The order Phoenicopteriformes is composed of one single family, the Phoenicopteridae, comprising three genera, five species, and six taxa. Flamingos have a worldwide distribution with the exception of Australasia and the Antarctic. In some species males are larger than females, but in some species the size difference is minimal. The largest species is the greater flamingo *(Phoenicopterus ruber)* weighing 2.1 to 4.1 kg and standing 1.20 to 1.45 m in height. The smallest species is the lesser flamingo *(Phoeniconaias minor)* that weighs 1.5 to 2.0 kg and stands 0.80 to 0.90 m in height.[87]

Flamingos are one of the most popular birds maintained in bird gardens and zoological collections worldwide. In a survey carried out in European zoos in 2013, 65% out of 139 members of the International Species Inventory System (ISIS) held greater flamingos, representing a total of 5,500 birds.[88]

Flamingos live and thrive in medium to large colonies of hundreds and thousands of individuals in highly specialized habitats consisting of mangrove swamps, shallow lagoons, and lakes with pH value as high as 10.5 and very often, with twice the salinity of seawater.[87]

Pododermatitis did not seem to occur in free-living flamingos as observed in the Camargue, France, in different studies.[89,90] In captivity, flamingos are housed in open enclosures provided with medium-sized shallow ponds and beaches. Due to convenience and to ease the cleaning procedures, most water ponds and beaches for housing flamingos are built of rough and abrasive concrete surfaces. The type of flooring appears to be a possible contributing factor in the development of pododermatitis.[89,91,92] Flamingos housed in collections north of the 53rd degree latitude are viewed to be at a higher risk of developing pododermatitis since the birds are maintained mostly indoors due to the prevailing climatic conditions.[7]

Pathophysiology

The feet of flamingos are small in relation to the size of the body and are provided with four digits, except for the Andean flamingo *(Phoenicoparrus andinus)* and the James's flamingos *(Phoenicoparrus jamesi)* in which the hallux or first digit is absent. There is a relatively large and thick interdigital web between the three front digits starting from the joint between the first and second phalanx. This type of foot webbing (palmate) provides support and allows flamingos to walk across soft mud and silt.[87]

Pododermatitis in flamingos develops gradually. Hyperkeratosis in the proximal pads of the digits is first commonly observed, reflecting an increased weight bearing in such areas. In a study carried out in European and American zoological collections, 100% of the flamingos included in the study were affected by such hyperkeratosis in the digital pads.[7] Hyperkeratosis of the digital pads does not appear to cause pain or discomfort to the bird. However, in the same study, up to 87% of the flamingos presented fissures with up to 46% showing deep fissures (Figure 33.38). There is no clinical information to support this, but it is believed fissures may be painful and may allow a port of entry to bacteria.[7] The presence of nodular growths was relatively small (19%), with only 6% of such birds showing severe nodular growths (Figure 33.39).[7]



FIGURE 33.38 A lesser flamingo *(Phoeniconaias minor)* showing bilateral superficial and deep fissures in all digital pads classified as stage 2 with fissures substage 1 and fissures substage 2. This bird was maintained in a rescue and rehabilitation center on concrete flooring. Such cracks are believed to be painful and serve as port of entry to bacteria.

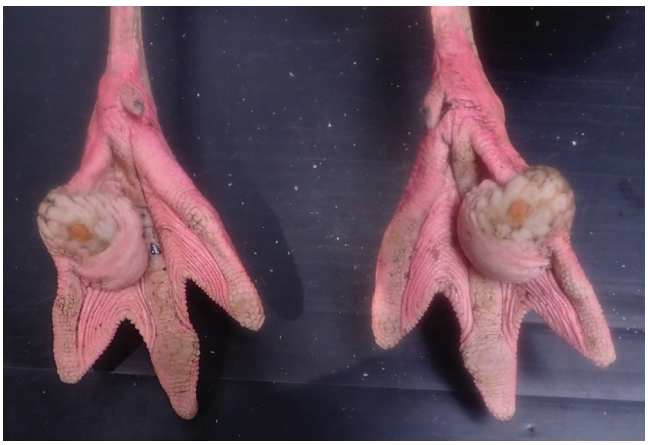


FIGURE 33.39 Greater flamingo *(Phoenicopterus roseus)* with a large abscess in the plantar pad. Such cases should undergo surgery to debride all necrotic tissue and to attempt first intention healing. Postoperative care of such cases can be challenging as the feet have to be kept dry at all times.

Papillomatous growths were observed in 46% of all the flamingos examined, with only 11% of the birds showing severe growths (Figure 33.40).[7]

In a study carried out at Basel Zoo in Switzerland, a novel bacterium was isolated from skin scrapings from several flamingos. The organism was identified as *Arsenicicoccus dermatophilus*.[93] In addition, *Micrococcus*-like bacteria invading the stratum corneum and exocytosis of heterophils into the epidermis were observed in a group of captive juvenile flamingos.



FIGURE 33.40 Greater flamingo *(Phoenicopterus roseus)* with stage 4, substage 2 bumblefoot showing cauliflower papillomatous growths on the plantar pads of the feet. Such cases of severe bumblefoot require radical excision surgery and long postoperative care with a moderate prognosis.

The exocytosis was attributed to reaction to the presence of the bacteria.[90]

It has long been suspected that papillomavirus could be involved in the development of papillomatous growths in the feet of flamingos. In support of this hypothesis, virus-like particles were observed in a northern gannet *(Morus bassanus)* with proliferative pododermatitis[94] and in several waterfowl species with verrucous hyperkeratotic pododermatitis.[95] Similarly, poxvirus has been associated with wart-like cutaneous growths in Chilean flamingos.[96] In a study using the polymerase chain reaction (PCR) carried out in 20 samples obtained from flamingos at Basel Zoo, no evidence was found for the presence of papillomavirus or poxvirus.[90] However, herpesvirus DNA of an unknown strain, but 69% identical to gaviid herpesvirus 1, was amplified in three samples obtained from flamingos affected with cauliflower papillomatous growths and fissures.[90]

Clinical and pathological findings

Pododermatitis in flamingos is classified into four stages, with two substages each according to the abovementioned pathological observations on the plantar pad and digits (Table 33.6).[7,89]

Treatment and prevention

Comparative studies carried out in zoological collections in France and Switzerland have shown that flamingos maintained in ponds provided with natural flooring have significantly less severe pododermatitis presentation than those kept on concrete flooring.[7,89] There are few species-specific recommendations for the treatment of lesions in flamingos. As a result, treatment of pododermatitis in flamingos is largely extrapolated from general treatment guidelines as listed for other species.

The exact etiology of pododermatitis in flamingos has not been identified to date; however, the type of flooring (concrete) used in ponds and dietary deficiencies, in particular dietary zinc, have been identified as possible causes of development of pododermatitis in captive flamingos.[89,91,92] Access to sunlit areas within the enclosure helps to increase natural

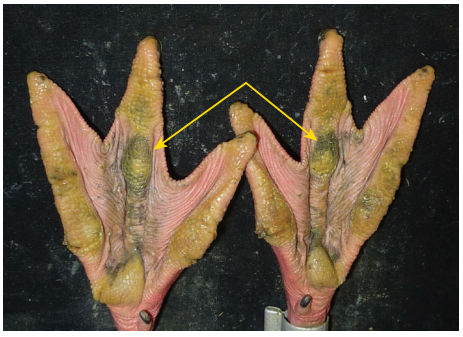
TABLE 33.6

Classification Scheme for Pododermatitis in Flamingos[7,89]

Stage 1 pododermatitis: hyperkeratosis



Substage 1: slight hyperkeratosis



Substage 2: marked hyperkeratosis

Stage 2 pododermatitis: fissures



Substage 1: superficial fissures

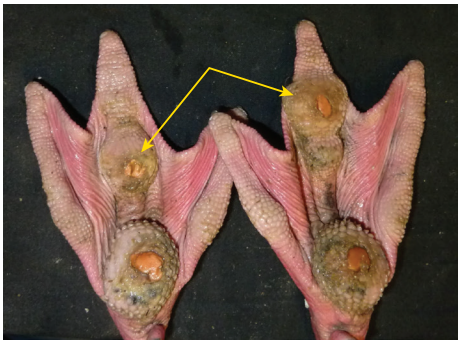


Substage 2: deep fissures

Stage 3 pododermatitis: nodules



Substage 1: nodules without ulceration



Substage 2: nodules with ulceration

Stage 4 pododermatitis: papillomatous growths



Substage 1: finger-like papillomatous growths



Substage 2: cauliflower-like growths

behaviors, including foraging and swimming, with a potential benefit to foot health.

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SECTION 8 THE TRAUMA PATIENT

CHAPTER 34

Triage and Stabilization

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INTRODUCTION

Birds presenting after trauma may show behaviors ranging in extremes from normal to increased activity and responsiveness to nonresponsive. Due to innate tendencies to hide signs of illness, many birds with catastrophic injuries can appear bright and alert, including (in the authors' experience) those with multiple fractures and even complete beak avulsion. It must be assumed that despite appearances, any bird with evidence of trauma is experiencing pain, and appropriate analgesia is a mandatory part of immediate treatment.

ASSESSING THE PATIENT—INITIAL HANDLING AND TREATMENT

Examination

All trauma patients should be observed at rest, undisturbed, prior to handling if possible. Evaluate the bird's ability to move about the enclosure, body position, respiratory effort (open-beak breathing or tail bobbing can be signs of increased respiratory effort), fluffed feathers, and overall level of responsiveness. Take note of color and consistency of droppings, any regurgitated material, and the presence of any blood or discharge. Observe the bird for normal behaviors in relation to the species and environment; for example, a bird of prey or wild bird should be very alert and not expected to eat, whereas a parrot may exhibit preening and eating. Injured birds still aware of their surroundings may experience stress with handling, which can cause rapid decompensation. During any handling and restraint, constantly look for indications that the bird should be released at once and the examination or procedure postponed; these include increasing dyspnea, inability to grasp, increasing weakness, and decreased response to stimuli. These birds should be offered oxygen and warmth while formulating a plan for the next steps.

Physical examination is best performed systematically, thoroughly, and in a timely fashion to minimize stress. It may be safest to perform the examination in stages if the patient is unstable. Have all materials, including any needed for the examination or diagnostics, organized prior to handling the bird. Work in a small room with locking doors and covered windows to minimize the potential for additional trauma with any escapes. Darkening the room can reduce movement of particularly fractious patients to facilitate initial capture. An appropriate scale (1-g increments of measure for small birds, with a range of over 1 kg to accommodate larger species) should be used to determine the weight of the patient prior to or immediately following the examination. While some birds will willingly step onto a tared perch, placing the bird in a small container for weighing is preferable to avoid further injury in cases of trauma.

Evaluation of the head should include examination of the external ear canals, nares, eyes and surrounding tissues, beak, and the oral cavity including mucus membranes and choanal slit. The beak is a common site for trauma and should be closely inspected for fractures or bleeding, particularly from the tip of the rhinotheca. Rhinothecal tip fractures can be incomplete and difficult to detect (Figure 34.1). Head trauma can result in ocular trauma, hemorrhage, or bruising (Figure 34.2). In birds of prey or in cases of head trauma, perform a fundic examination to evaluate for retinal detachment, hemorrhage, or other indicators of trauma. If the bird's condition permits, separate the feathers over the skull and assess for bruising, and palpate the orbital rim for fractures. Palpate the crop at the base of the neck and note any distention or evidence of wounds. Body condition should be assessed by palpation of the pectoral muscling and palpation of the body for any excess fat stores. The abdominal region between the ventral keel and the pelvis should be gently palpated for any evidence of distention. Wings and legs should be evaluated for symmetry, bruising, or instability, along with

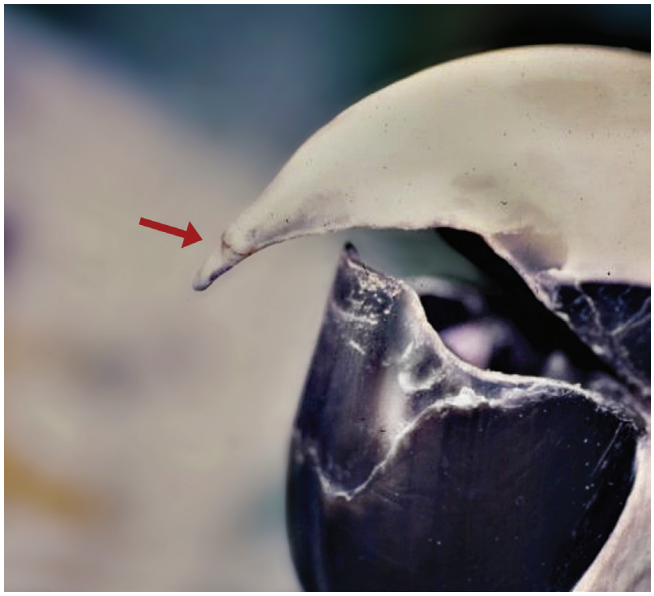


FIGURE 34.1 Traumatic rhinotheca tip incomplete fracture *(arrow)*. These fractures often require careful close inspection to detect.



FIGURE 34.2 A budgerigar *(Melopsittacus undulatus)* with suspected head trauma. Note marked depression and ruffling of the feathers of the head.

the overall quality of the skin and feathers. The presence of a wing droop or leg lameness warrants further investigation (Figure 34.3). Auscultation of the heart is best performed over the keel while the respiratory tract can best be auscultated across the dorsum. Note any heart murmur, arrhythmia, or increased respiratory sounds. Any crackles, wheezes, or clicking can indicate pathology as respiratory sounds are normally minimal. Note that there can be a normal clicking sound in some birds from the uncinate processes (projections off the caudal aspect of each rib) hitting one another—this is most common in cockatiels in the authors' experience. Ruptured air sac, fracture of pneumatic bones, and penetrating bite wounds can result in subcutaneous emphysema, which can produce a crackling sound and crepitus when palpating the skin. Skin



FIGURE 34.3 A subtle right-wing droop in a double yellow-headed Amazon parrot *(Amazona ocrocephala oratrix)* with suspected wing trauma. Wing carriage in a normal bird is symmetrical at rest.

turgor can be assessed by gently pinching or lifting the eyelids—if the skin stays tented, this can be a sign of dehydration. Observe and palpate the brachial artery for strength, regularity, and quality; this may be difficult to evaluate in smaller species. The oral cavity can be dry and pigmented in many species, so evaluation of the cloacal mucosa can give a better idea of mucous membrane color and hydration. Perfusion status can be assessed by refill time of the basilic vein. Complete neurologic examination can be challenging in some patients but should be attempted for any bird with suspected head trauma (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 9, Clinical avian neurology and neuroanatomy). At any point in time, a stressed bird can be given a break or placed in oxygen with the remainder of the examination completed later. Evaluate the recovery of the bird and note excessive recovery time, dyspnea, or weakness. Overweight birds and birds with coelomic distention can easily become overheated and may display panting and holding the wings away from their body to facilitate cooling during recovery. Be cognizant of other conditions that may contribute to overheating such as sun exposure when injured, especially for outdoor birds and wildlife.

Common Trauma Presentations

Birds are relatively fragile when compared to the size of their human companions and other traditional pets that may live in the same household. Common traumatic injuries include bite wounds, collision injuries with walls, windows, and ceiling fans (especially in free-flying birds), compression injuries from entrapment in furniture or closing doors, and blunt

trauma from falls in nonflighted, often overweight birds. Even birds that are maintained in cages are not immune to predators; there are many anecdotal reports of cats and even wildlife such as raccoons pulling parrots, poultry, and exhibit birds through cage bars resulting in severe wounds and even traumatic amputation of limbs. Immediate life-threatening conditions include moderate to severe hemorrhage, upper airway and pulmonary trauma, and internal organ damage and bleeding.

Analgesia is important for the trauma patient and is presented in Chapter 35 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 20, Recognition, Assessment, and Management of Pain in Birds.

ASSESSING THE PATIENT—BASIC LIFE SUPPORT

Before taking a complete history, determine if the bird needs immediate care. As in mammals, the ABCs (airway, breathing, cardiovascular status) should be immediately assessed in avian patients with trauma. Stabilization efforts should be made in situations of hemorrhage, respiratory distress, severe neurologic signs including seizures, open fractures, or if the bird is weak and/or on the bottom of the enclosure. If immediate care is needed, place the bird in a warm and oxygenated incubator, ideally 85°F to 90°F (29°C–32°C). Supportive care measures including fluids, nutritional support, analgesics, or antibiotic therapy can stabilize the patient and allow for additional diagnostics and treatment later. Avoid high temperature if head trauma is suspected.

The nonresponsive patient may require basic life support (BLS) and should be evaluated immediately, and cardiopulmonary resuscitation (CPR) rendered if necessary. Guidelines for BLS in birds are based on those considered most effective in other veterinary patients, with adaptations made for significant anatomic and physiologic differences (Table 34.1).[1] The Reassessment Campaign on Veterinary Resuscitation (RECOVER) campaign is an initiative of the Veterinary Emergency and Critical Care Society (VECCS) and the American College of Veterinary Emergency and Critical Care (ACVECC) to continually assess and review scientific literature in an effort to provide evidence-based and consensus guidelines for CPR for dogs and cats.[2] While RECOVER does not include data for nontraditional pet species, the guidelines are an excellent starting point for improving CPR efforts in special species, including birds. A recent study described CPR outcomes in hospitalized birds.[3] The study included 41 birds that experienced arrest either during anesthesia or during handling or hospitalization. Various CPR techniques were attempted, including ventilation, external compressions, vascular access, fluid administration, and drug administration. There were three documented cases of return of spontaneous circulation (ROSC) and one cardiopulmonary arrest (CPA) survivor. Two of the survivors were undergoing minor procedures, and one a more invasive coelomic surgical procedure. Protocols used to successfully resuscitate the patients differed in all three patients. Because of this high variability and low number of successful CPR attempts, it is currently not possible to form specific recommendations intended to improve success in future cases; however, the low number of successful attempts does demonstrate that while chances for ROSC are poor, positive outcomes demonstrate that coordinated and vigorous attempts should be made.[3] Resuscitation wishes should be discussed in advance for critical patients and CPR efforts made accordingly. Successful resuscitation requires staff preparedness, which includes frequent CPR drills, and consolidating all necessary equipment into specific designated areas of the hospital with available oxygen and where CPR is most likely to take place.

For birds, specialized equipment includes multiple sizes of endotracheal tubes (1.0 mm and larger) and tight-fitting masks for ventilation, emergency drugs, and appropriately sized needles and syringes. Modified red rubber tubes, 18- to 22-gauge (G) intravenous (IV) catheters (with stylet removed), or other tubing may be used for intubation of small

TABLE 34.1

Basic Life Support for Birds

If intubated*	If using forced mask ventilation*	
For a total of 2 min: Cardiac compressions: (>120/min) Continuous ventilation (10/min)	**For a total of 2 min** Cardiac compressions: (>120/min) Total 30 compressions Stop compressions, give 2 breaths	a. Check oral cavity for mucus or obstructions b. Discontinue inhalant anesthetics if applicable c. Reverse injectable agents if applicable
Check heart rate (5 sec)	Check heart rate (5 sec)	
For a total of 2 min: Cardiac compressions: (>120/min) Continuous ventilation (10/min)	**For a total of 2 min** Cardiac Compressions: (>120/min) Total 30 compressions Stop compressions, give 2 breaths	**Positions** Calling code Ventilation Chest compressions Catheter ER drugs Records Owner communication
Check heart rate (5 sec) Administer ER drugs	Check heart rate (5 sec) Administer ER drugs	
Repeat from the top	**Repeat from the top**	

*In cases of tracheal obstruction, place an airsac tube to provide airway support.

ER, Emergency room.

birds and should be prepared in advance and kept with standard endotracheal tubes for easy access in an emergency.

All equipment for BLS is kept in a designated "crash cart," which is separate from regular hospital supplies, clearly marked, and regularly inspected. Crash carts are sealed and dated with tape. When the cart is opened, supplies are replaced when the BLS event ends and resealed to ensure it is ready for the next emergency. A summary of useful equipment for the avian crash cart is presented in (Box 34.1).

BOX 34.1 SUGGESTED SUPPLIES FOR THE AVIAN EMERGENCY AND CRITICAL CARE "CRASH CART"

Drugs

Epinephrine (1:1000, 1 mg/mL)
Vasopressin (20 U/mL)
Atropine (0.54 mg/mL)
Naloxone (0.4 mg/mL)
Flumazenil (0.1 mg/mL)
Atipamezole (5 mg/mL)
Lidocaine 2%
Hypertonic saline 7.3%
50% Dextrose
Furosemide
± Norepinephrine
± Dobutamine
± Dopamine
± Calcium gluconate

Supplies

ET tubes (appropriate size for birds, 1.0–4.0 mm)
Tight-fitting anesthetic face masks (various sized appropriate for birds)
Sterile cotton tip applicators
Syringes, needles, flush, catheters (appropriate size for birds)
Suture (sizes 5-0 to 2-0, absorbable and nonabsorbable)
IO catheters (25-18-G hypodermic needles or small, short spinal needles with stylet)
ECG with atraumatic clips and/or small ECG pads
Capnograph
Prewarmed fluids (replacement crystalloids-LRS, saline, etc.), hypertonic saline, hydroxyethyl starch (e.g., hetastarch)
Air sac tube (commercial or made from shortened endotracheal tubes)
Bandaging supplies (basic vet wrap, rolled gauze, cast padding, tape [cut to size])
Tourniquet (rubber band, hemostat, or small commercial product)

Ideal Equipment Available in Close Proximity

O_2 flow meter + humidifier and/or oxygen cage (with heat and humidity control)
Multiparameter monitor with ECG, BP, $ETCO_2$, Pulse-OX
Doppler unit (used to monitor heart directly or for BP with appropriate cuff sizes)
Glucometer, PCV tubes, slides for blood smear
Ideal: point-of-care ultrasound unit

The crash cart is positioned in close proximity to an oxygen source. Crash cart contents should be replaced immediately after use and inspected regularly.

BP, Blood pressure; *ECG*, electrocardiogram; *ET*, endotracheal; *$ETCO_2$*, end-tidal carbon dioxide; *LRS*, lactated Ringer's solution; *PCV*, packed cell volume; *Pulse-OX*, pulse oximeter.

Respiratory Arrest

Birds are relatively easy to intubate, as the position of the glottis is clearly visible at the base of the tongue (Figure 34.4). Birds under 100 g are more challenging to intubate due to size. A small endotracheal tube can be fashioned from a 3.5- or 5-mm red rubber tube in small birds if an appropriately sized tube is not readily available. The glottis of chickens and other poultry is relatively deep and should be gently pushed rostrally to visualize it. Cotton-tipped applicators are very useful to help remove mucus and debris from the oral cavity and in some cases depress or advance the rostral portion of the tongue. Avoid inflation of cuffed endotracheal tubes in birds as their complete tracheal rings predispose them to stricture. If intubation is complicated and takes more time than anticipated, consider forced mask ventilation with a tight-fitting mask. A variety of masks with small openings to create a tight seal are required for forced mask ventilation in birds. A syringe case can also be modified to fit over the beak and nares and sometimes is necessary in some species. In both cases, a ventilation rate of 15 to 20 breaths per minute has been suggested, but the ideal ventilatory rate for birds in the face of respiratory arrest has not been determined.

In cases of suspected tracheal injury, avulsion, or obstruction (as indicated by the presence of stridor, open-beak breathing, and increased inspiratory effort), an air sac tube should be placed (see section on Respiratory Distress and Chapter 29).

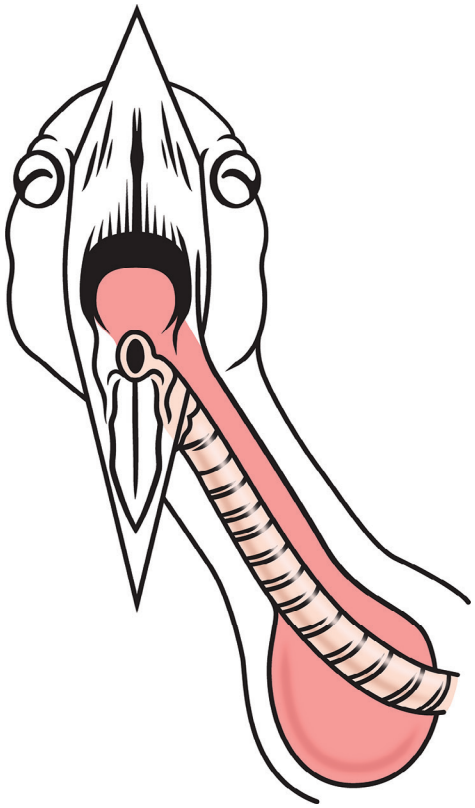


FIGURE 34.4 Line drawing of the general placement of the glottis in relation to the tongue.

Cardiac Arrest

If cardiac arrest is identified, start cardiac compressions. Note that there are limited data on the efficacy of compressions in birds because of the large keel of flighted birds and the high compression rates that are likely required. Chest compressions in birds may work on the principle of a thoracic pump, increasing the intrathoracic pressure to encourage blood flow out of the heart and great vessels. The wide sternum "shields" the heart and the coracoids prevent depressing the cranial sternum during traditional cardiac compressions. Therefore, adopting a different technique than mammals for cardiac compression may be more effective. A recent study evaluating the arterial blood pressure during three different types of cardiac compressions was performed in chickens.[4] Cardiac compression techniques include keel compressions, lateral compressions, and keel compressions with interposed caudal coelomic compressions (ICCC). Keel compressions with ICCC consistently resulted in higher arterial blood pressures compared to lateral compressions or keel compressions alone. Although this study was performed in poultry, keel compressions with ICCC should be considered in psittacine species. When compressions are attempted, more than 120 compressions/minute have been suggested. When performing compressions in the intubated patient, compressions and ventilation can be performed simultaneously. When using forced mask ventilation, discontinue compressions temporarily during ventilation (Table 34.1)

Cardiac activity should be monitored with electrocardiography (ECG) and effectiveness of compressions with a Doppler probe. ECG leads should be attached to the skin at the base of both wings and both thighs. This can be done using hypodermic needles, paper clips, gel-soaked gauzes, or self-adhesive patches. The Doppler probe can be placed near the proximal ulna or medial tibiotarsus, with a cuff for blood pressure monitoring placed on the humerus or femur, respectively. The width of the cuff should be 30% to 40% of the circumference of the limb. Indirect blood pressure measurements in birds are not very accurate; however, they can be used to monitor trends. Every 1 to 2 minutes, discontinue compressions and assess for ROSC via the ECG, Doppler probe, and auscultation.

Epinephrine and atropine have been described for use in birds, but specific research focusing on ideal dosages is lacking.[5] Table 34.2 lists some suggested dosages. Ideal route of administration is IV, or in case of vascular collapse, rapidly place an intraosseous (IO) catheter, which is discussed in greater detail in the section on Vascular Access.

Once cardiac and respiratory arrest are resolved, evaluate for shock and begin appropriate treatment.

SEDATION FOR THE TRAUMATIZED PATIENT

Many traumatized but responsive birds benefit from low-dose sedation to decrease anxiety; this is of particular benefit for the bird in respiratory distress. The most described sedation protocol combines midazolam (0.25–2 mg/kg) with butorphanol (1–2 mg/kg), which provides the added benefit of analgesia.[6] Another option is lower-dose midazolam and butorphanol with the addition of 1 mg/kg alfaxalone intramuscularly (IM). Dosages are chosen based on overall patient condition, with lower dosages chosen for sicker patients. The authors prefer the delivery of sedation drugs in a trauma patient by combining them into a single syringe and injecting IM using minimal handling techniques. Intranasal administration of midazolam and midazolam-butorphanol has been described and may be useful in some situations as well.[7]

Analgesia is discussed in detail in Chapter 35 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 20, Recognition, Assessment, and Management of Pain in Birds.

Vascular Access

Vascular access is required for effective delivery of emergency drugs, for blood transfusion, and to treat shock. Ideally, oral or subcutaneous fluids should be reserved for stable, standing patients that are suspected to be less than 5% dehydrated. However, the risk of catheter placement due to stress and handling in some patients may outweigh benefits. Therefore, it may be beneficial to administer subcutaneous fluids along with other stabilization measures prior to catheterization. Oral fluids should not be administered if there is significant risk of aspiration, in a recumbent patient or in patients with evidence of marked gastrointestinal dysfunction. Vascular access in birds can be performed via two routes: IV and IO; the choice depends on patient size and condition and personal preference.

Intravenous catheters are more difficult to maintain than IO catheters due to the thin skin of the bird. Locations for IV catheters include the jugular vein, basilic (or ulnar) vein, or the medial metatarsal vein (Figure 34.5A–C). The area for catheter placement is plucked and scrubbed. A small catheter (generally 24- to 26-G over-the-needle catheter) is guided into the vein and secured to the skin with glue or tape sutured

TABLE 34.2

Cardiopulmonary Resuscitation (CPR) Drug Dosages Suggested for Use in Birds

Drug	Dose	Concentration	Route
Epinephrine	0.01 mg/kg	1:1000, 1 mg/mL	IM, IV, IO, intratracheal
Atropine	0.02 mg/kg	0.54 mg/mL	IM, IV, IO
50% Dextrose	0.25 mL/kg	1:1 dilution with saline	IV
Calcium gluconate	50–150 mg/kg	10%, 100 mg/mL	IV (bolus slowly)
Doxapram	2.0 mg/kg	20 mg/mL	IV, IO
Vasopressin	0.8 U/kg	20 U/mL	IV, IO
Naloxone	2.0 mg	0.4 mg/mL	IV

Dosages are based on extrapolation from other species and anecdotal reports.
IM, Intramuscular, *IO*, intraosseous, *IV*, intravenous.

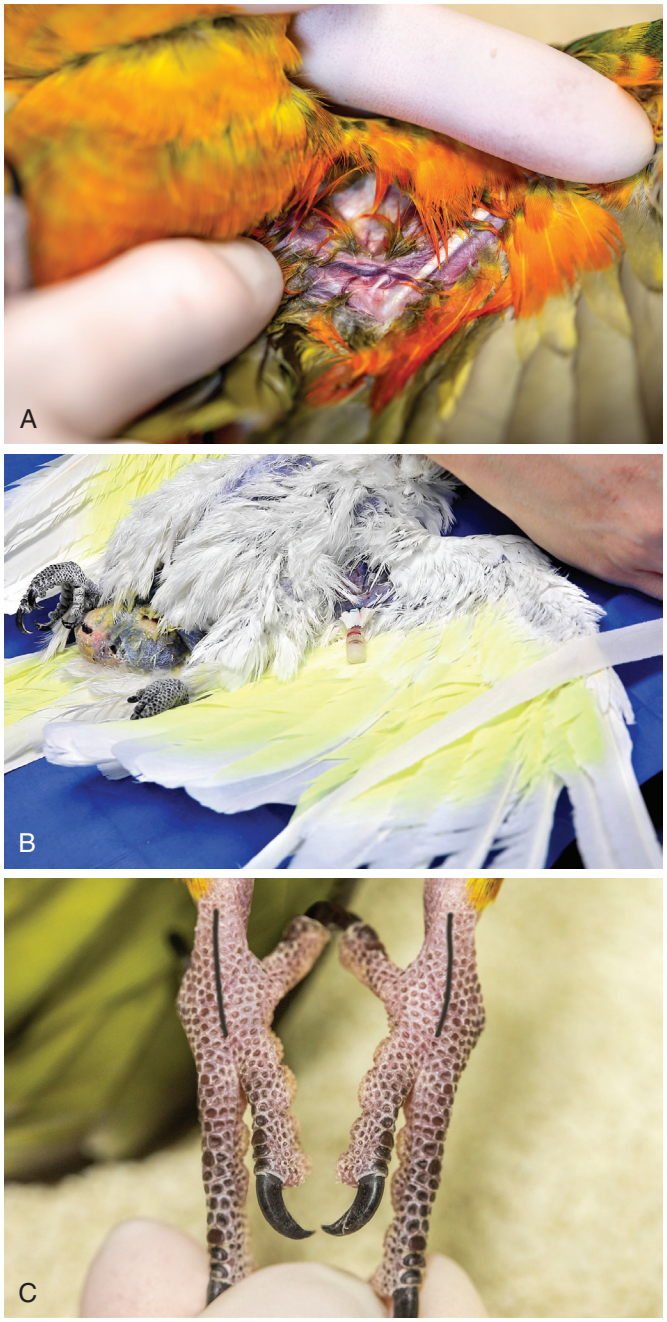


FIGURE 34.5 (A) The location of the left ulnar or basilic vein in a conure (*Aratinga* sp.). (B) An anesthetized cockatoo with a catheter placed in the left ulnar or basilic vein. A butterfly-style tape is applied to the catheter and is sutured to skin. (C) Location of the medial metatarsal veins, which can be used to collect a very small amount of blood (e.g., for evaluation of packed cell volume [PCV]) or in larger birds for placement of an IV catheter.

to the skin. A small piece of transparent film dressing (Tegaderm; 3M Company, Maplewood, MN, USA) followed by a figure-of-eight bandage can be used to protect and secure basilic vein catheters. A neck wrap can be used to help secure jugular catheters. Catheters in the medial metatarsal vein are easy to place and maintain in larger birds and generally wrapped in place. A syringe pump or fluid pump that can



FIGURE 34.6 Postsurgical image of a bird after amputation of the left leg that was crushed by another household pet. Note the use of intradermal sutures, and the placement of an IO catheter in the right tibiotarsus for intermittent fluid administration, and, if needed, delivery of emergency drugs.

administer small volumes is ideal to control slow delivery of fluids in smaller birds. In very large patients, such as larger poultry, regular drips may also be safely used.

Intraosseous catheters can be placed either in the distal ulna or in the proximal tibiotarsus through the cnemial crest (Figure 34.6; see also Box 19.1 in *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 19, Anesthesia). A potential complication of IO catheters is osteomyelitis; therefore, sterile surgical preparation of the area is essential. Analgesia is important, as placement is uncomfortable. The site (skin, subcutaneous tissue, and periosteum) can be blocked with lidocaine at 2 mg/kg prior to catheter placement. Medium- to large-sized birds can accommodate a 20- to 22-G, 1.5 to 2.5-inch spinal needle, or similarly sized hypodermic needle. Smaller size hypodermic needles (25–27 G) are used in smaller birds. A stylet can be made for the hypodermic needle using sterile cerclage wire or 3-0 to 6-0 stainless steel suture. Stylets prevent obstruction of the needle lumen with cortical bone.

FLUID RESUSCITATION

Most studies evaluating fluid resuscitation in avian patients use chickens and ducks as a model.[8] There is very little information on ideal fluid requirements for birds in shock. One study compared the effects of three fluid types (hetastarch [HES], a hemoglobin-based oxygen carrier [Hemospan; Sangart Inc., New York, NY, USA], and whole blood) for resuscitation in chickens *(Gallus gallus domesticus)* after removal of 50% of their blood volume via phlebotomy under general anesthesia. Four of 10 birds in the nontreated control group died, whereas 2 of 10 birds in the whole-blood group died, and 3 birds died in both the HES and Hemospan groups.[9] While results were not considered statistically significant in any group, mortality was lowest in the group treated with whole blood. During blood removal, arterial blood pressure decreased in all groups and increased in the three treatment groups.[7] The drop in packed cell volume (PCV) created by phlebotomy was corrected in 7 days in all groups.

Current anecdotal recommendations include the following: for early decompensatory phase, give crystalloid bolus

(10 mL/kg) and HES bolus (hydroxyethyl starch, 3–5 mL/kg IV or IO). In the decompensatory phase, give hypertonic saline solution (HSS) bolus (hypertonic saline, 3–5 mL/kg IV or IO) over 10 minutes. The rapid effects of hypertonic saline can be maintained with the addition of HES, which can be given at 3 mL/kg IV or IO if needed. Warm the patient and administer crystalloids (10 mL/kg) and HES (3–5 mL/kg) bolus IV or IO. All fluids administered should be warm. Repeat for three to four boluses or until blood pressure is greater than 90 mm Hg and/or there is improvement in clinical markers (mentation, capillary refill time [CRT], mucus membrane color). If PCV is under 20% or the patient is hypoproteinemic, consider blood transfusion.

Crystalloids are the preferred treatment for dehydration. The fluid deficit equals the degree of dehydration (%) multiplied by the body weight *in grams*. Fluid deficit can be replaced over 6 to 8 hours in acute losses and over 12 to 24 hours in chronic losses in birds without cardiopulmonary compromise.[1] Up to 50 mL/kg can be administered as an IV bolus. Recommended maintenance dose of fluids is 40 to 60 mL/kg/day (depending on species).

Whole blood is indicated when albumin, antithrombin, coagulation factors, platelets, or red blood cells are required. It should be kept in mind that blood products do not promote blood flow as well as acellular fluids (e.g., HES, crystalloids); the density of erythrocytes impedes the ability of blood products to promote blood flow (a viscosity effect). Therefore, blood is rarely used for initial resuscitation unless the patient is exsanguinating or there is coagulopathy.

As in other species, continued blood loss, nonregenerative anemia in a weak, symptomatic patient with PCV 12% to 15% or below, and clotting disorders (such as rodenticide toxicosis) are indicators used to determine the potential need for a whole-blood transfusion. Whole blood can be administered at 10 to 20 mL/kg via the IV or IO route. Blood donors can be homologous or heterologous species. Limited studies in selected species suggest that the lifespan of red cells transfused between homologous species is greater than that between heterologous species; for example, 1 to 13.1 days for homologous transfusion between pigeons *(Columba livia domestica)* and cockatiels *(Nymphicus hollandicus)* but 12 hours for pigeon to red-tailed hawk *(Buteo jamaicensis)*.[10] Some practices maintain a relationship with a local bird rescue and keep a list of clients willing to bring in patients for blood donation in exchange for clinic credit.

Blood collection is performed in the carefully restrained or sedated healthy donor via the jugular vein, using a needle and syringe, or butterfly catheter. Collect blood into syringes with heparin (2 U/mL whole blood) or sodium citrate at product-recommended doses. As blood typing in birds is impractical, and in most cases multiple donors are not available, volume to transfuse is usually limited by the amount that can be safely collected from a single healthy donor, generally 10% of the body weight. Formulas to predict posttransfusion PCV in anemic dogs have been proposed and may be useful for birds as well (Box 34.2).[11]

While general recommendations are to administer blood over 1 to 4 hours, when need is urgent, the entire volume can be administered over 10 to 15 minutes, or even quicker in cases of massive hemorrhage. Use a pediatric microfilter to remove most of the aggregated debris.

BOX 34.2 PROPOSED FORMULAS TO PREDICT POSTTRANSFUSION-PACKED CELL VOLUME IN ANEMIC DOGS THAT MAY BE USEFUL FOR BIRDS[11]

Formula 1:
Volume to be transfused [VT] [mL] = 1 mL × % PCV rise × kg body weight [BW]

Formula 2:
VT [mL] = 2 mL × % PCV rise × kg BW

Formula 3:
VT [mL] = 90 mL × kg BW × [(desired PCV − Patient PCV)/PCV of donor blood]

Formula 4:
VT [mL] = 1.5 mL × % PCV rise × kg BW

BP, Blood pressure; *BW,* body weight; *PCV,* packed cell volume.

Administer blood transfusions within 4 hours of collection to prevent the growth of bacteria, according to standards set by the American Association of Blood Banks.[10]

Cross matching donor and recipient blood has been proposed to prevent transfusion reaction. However, in most cases the need for transfusion is urgent, and it is rare that there are multiple donors from which to choose. A single transfusion is unlikely to produce a serious reaction, and risk should be weighed against the risk of foregoing blood transfusion.[10]

SPECIFIC TRAUMATIC CONDITIONS

Respiratory Distress

Traumatic injuries may produce respiratory compromise in several ways. Tracheal trauma impairs airflow to the lungs, and diseases that produce hemocoelom or pulmonary hemorrhage directly impact the function of the lungs and air sacs. Blunt trauma can produce pulmonary contusions or hemorrhage. Suboptimal oxygenation can be produced by profound anemia or hypovolemia resulting in poor circulation. Rest in a warm oxygenated enclosure is important prior to any additional handling or treatment.

Classification of respiratory patterns can be very helpful to distinguish between upper and lower airway disease. As in other species, birds with traumatic injury of the nasal cavities or trachea often show inspiratory distress, while birds with traumatic injury to the coelom that produces respiratory compromise may present with expiratory distress.

Birds with tracheal injury or obstruction due to hemorrhage often present with an audible wheeze or "squeak" upon inspiration. Some may exhibit a voice change or exhibit neck stretching and open-mouthed gasping. Severe cases of suspected tracheal obstruction not responding well to oxygen therapy require emergent air sac cannula placement (see Chapter 29). These birds also may greatly benefit from the use of conscious sedation, or gas anesthesia, which supplies beneficial oxygen as well (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 19, Anesthesia).

Air sac cannulas can be made from red rubber tubes or standard small endotracheal tubes (Figure 34.7A). The diameter of the tube is selected based on the approximate size of

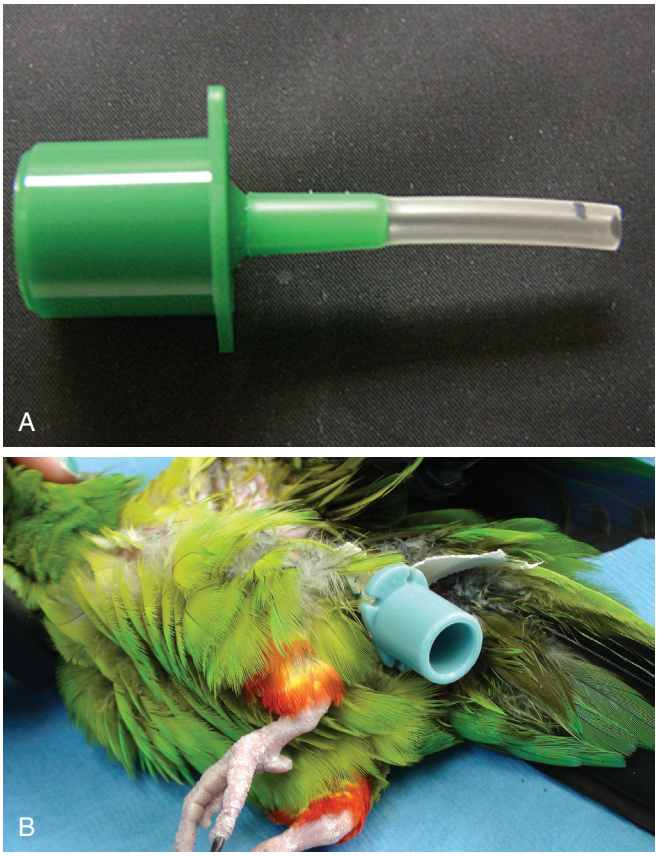


FIGURE 34.7 (A) A shortened endotracheal tube for use as an air sac cannula. (B)The cannula in place in an anesthetized bird.

the trachea in the species. A standard cuffed 3.0 mm inside diameter endotracheal tube can be used in larger birds. The position for the tube is the same as for an endoscopic approach to the caudal thoracic and abdominal air sacs. A small skin incision is made over the triangle created by the cranial muscle mass of the femur, ventral to the synsacrum and caudal to the last rib. The body wall is exposed by blunt dissection and penetrated with a pair of hemostats and the air sac cannula is inserted between the last two ribs or just caudal to the last rib (Figure 34.7B). The bird should immediately begin to breathe through the tube if placed correctly. A filter, such as the removed inner filter from a respirator mask, can be placed over the end of the tube to prevent entry of particulate matter.

After placement, test for airflow with cotton strands or a feather. Suture in place using a purse string and finger-trap suture technique. Patency should be tested again once the sutures are in place. Stay sutures can be used to attach the tube to the back; most birds do not chew these tubes, but an e-collar can be applied if necessary.

Once a patent airway has been established and the patient is stable, tracheal obstruction or trauma can be addressed. Birds can be anesthetized and maintained under anesthesia delivering inhalant gas and oxygen via the air sac cannula. Obstruction due to hemorrhage may require gentle flushing and aspiration of the trachea with an appropriately sized small red rubber catheter. One author (A.M.L.) removed a seed lodged in the midtrachea of a cockatiel by forcing it out with air injected proximal to the obstruction using a 27-G needle and 1-mL syringe directly through the wall of the trachea. These techniques may be useful for birds with more rostral obstructions due to hemorrhage.

Tracheal trauma may be amenable to tracheal surgery techniques described for other conditions in birds, such as tracheal stenosis (see Chapter 29). While tracheal surgery is often linked to poor outcomes, there are numerous reported cases of successful tracheal resection and anastomosis in larger bird species and a cockatiel.[12,13] One case report described the placement of a custom-made, nitinol wire stent in an Eclectus parrot *(Eclectus roratus)* suffering from multiple tracheal strictures spanning a length of 2.6 cm after repeated attempts to widen the trachea using fluoroscopic-guided balloon dilation were unsuccessful. Following stent placement, the bird continued to have intermittent episodes of mild to moderate dyspnea and eventually died because of complications that were suggestive of tracheomalacia.[14] While tracheal injuries often carry a good prognosis (provided the injury is not too extensive), injury to the syrinx carries a much poorer prognosis due to the complexity of the structure and difficulties related to surgical access.

In patients that have undergone airsacculotomy, air sac tubes should not be left in place for longer than 5 days and should be checked frequently to assure patency. To test the patient to see if an air sac tube is still required, plug the air sac tube with an adapter, end of a syringe, or other material appropriately sized to functionally block the opening. Observe the bird for several hours for dyspnea or distress. If no change in respiration occurs, and the patient breathes normally, then the air sac tube can be removed. Culture the tip of the air sac tube when removed. It is not necessary to close or cover the site of air sac tube placement.

Air Sac Rupture

Air sac rupture can occur in association with trauma, such as bite wounds or trauma, but can also be a result of underlying disease conditions such as airsacculitis. Air sac rupture may cause local subcutaneous emphysema, particularly in Amazon parrots (*Amazona* spp.), macaws (*Ara* spp.), and cockatiels. Needle aspiration of the air can aid in a diagnosis and reduce air accumulation, which may not reform in some cases. However, chronic recurring air accumulation may require creation of a temporary opening. In acute cases where air rapidly reaccumulates after aspiration, apply a local analgesic block, and make a small slit into the skin to allow air to escape; such incisions may remain patent for short periods of time.

Indwelling stents for palliative removal of air have been described as an option. Surgical means to address air sac hyperinflation or rupture are also described in Chapter 29.

It is possible for birds to develop persistent air sac hyperinflation or rupture without long-term complications.

Coelomic Disease

The most commonly encountered coelomic cavities with disease include those in the abdominal region, intestinal peritoneal, and hepatic peritoneal cavities, although pericardial and even pleural disease processes are also described (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 8, Pleura, Pericardium, and Peritoneum: The Coelomic Cavities of Birds and Their Relationship to the Lung–Air sac System). Ascites may be a result of trauma. Fluid may be urine/urates,

due to urinary tract injury, bile, or blood secondary to injury or rupture of the liver, kidney, or spleen. Larger accumulations may cause pronounced respiratory signs due to compression of the air sacs. Ultrasound or transillumination of the coelom is often useful to confirm the presence of fluid and may give information on the nature of the traumatic injury as well as the most optimal point to attempt centesis. Aspiration of coelomic fluid (preferably ultrasound guided) is often utilized to remove ascitic fluid from nontraumatic causes but is of questionable utility in traumatized birds. Aspirate only in the central third of the coelom to avoid lateral penetration and leakage of fluids into the air sacs. The feathers of the ventral abdomen are moved away from the aspiration site and the skin is aseptically prepared. A 27- or 25-G (butterfly) needle or catheter is inserted at the midline and directed to the right side of the peritoneal cavity (thereby preventing damage to the proventriculus) or into the fluid-filled region as identified by ultrasound or transillumination. Aspirate as much fluid as possible for complete analysis and to resolve the bird's dyspnea. Ascites resulting from egg yolk peritonitis can be viscous and may require a larger needle (22 G or larger). Continued hemorrhage, recurrence, or leakage of other fluids may be an indication for exploratory surgery (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 21, Surgery).

Head and Spinal Trauma

Flight injuries are a common cause of head trauma in pet birds. Patients with head trauma can present with evidence of bruising or bleeding, lethargy, and altered consciousness. There are several reports of Horner syndrome in birds, one in a parrot, and several in wild birds of prey (Figure 34.8).[15] Birds presented with unilateral ptosis and changes in the position and mobility of feathers. A case in a red-bellied parrot *(Poicephalus rufiventris)* was thought to be secondary to trauma to the base of the neck.[16] These birds should be placed in an oxygen-enriched, quiet, dark, well-padded environment. Additional thermal support is not recommended if intracranial hemorrhage is suspected, except to prevent hypothermia. Vascular access is ideal with placement of an IV or IO catheter. Hypovolemia can be managed with low-volume resuscitation (3 mL/kg) over 10 minutes with hypertonic saline with or without colloids. For cases of suspected increased intracranial pressure, hypertonic saline at 3 mg/kg IV or mannitol (0.25–1.0 g/kg) IV or IO as a slow bolus can be considered and given every 4 to 6 hours for a total of three doses in 24 hours. Maintenance fluid support should be reduced by 35% to 50% in these cases. Head elevation by 30% is recommended for recumbent patients. Analgesia, including opioids, can be given but should be used cautiously to avoid respiratory depression. If hydrated, meloxicam (0.5 mg/kg q12h) may decrease cerebral edema and intracranial pressure (see also Chapter 35 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 20, Recognition, Assessment, and Management of Pain in Birds). Benzodiazepines can be used to control seizures. Imaging including computed tomography (CT) scan can be performed once the patient is stable to evaluate for beak or skull fractures (see Chapter 2). The use of steroids with head trauma is not recommended, especially in birds that may be more sensitive to the effects of corticosteroids. Tranexamic acid (TXA) has been proposed in trauma, but too little information



FIGURE 34.8 (A) Left-sided Horner syndrome in a great horned owl *(Bubo virginianus)*. Marked ptosis of the left upper eyelid can be seen *(asterisk)*. Feather asymmetry is mainly characterized by depressed ear tuft feathers on the left side *(arrow)*. (B) Left-sided Horner syndrome in a northern goshawk *(Accipiter gentilis)*. Ptosis of the left upper eyelid *(white arrow)*, along with elevation of the lower eyelid *(black arrow)* can be appreciated. Feather asymmetry in the Accipitriformes and Falconiformes in this study was attributable to an erection of the head feathers on the affected side *(star)*. (From LaChance MK, Fitzgerald G, Lair S, Desmarchelier MR: Horner syndrome in birds of prey, *J Avian Med Surg* 33(4): 381–387, 2019.)

exists as to its utility in animals at this point. As potential side effects include seizures, thromboembolism, hypotension, and anaphylaxis, use of TXA is not without risk. Ophthalmologic examination of both anterior and posterior segments should occur upon patient stabilization. Topical and/or systemic administration of antibiotics is indicated for corneal ulceration or perforation. Topical ophthalmic nonsteroidal antiinflammatory drugs (NSAIDs) can be used to treat uveitis without corneal ulceration.

Spinal cord injuries can be caused by fractures, subluxation, or luxation of the spine (Figures 34.9 and 34.10). This may be difficult to detect radiographically but may demonstrate an air lucency in the affected area, particularly when the injury is caused by an altercation with an animal. Careful physical examination by gently wetting the area of the spine



FIGURE 34.9 Coronal slice CT image of a spinal fracture *(black arrow)* in a Quaker parrot. The *white arrow* demonstrates pulmonary contusions.

may demonstrate bruising in the affected areas (Figure 34.11). Hemorrhage, inflammation, edema, and ischemia secondary to the trauma may cause further damage. Serial neurologic examination is essential for monitoring recovery (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 9, Clinical avian neurology and neuroanatomy). Although prognosis for recovery is likely to be poor if deep pain is absent, one of the authors (J.G.) managed a case of spinal trauma in a great horned owl with absent deep pain for over a week that made a full recovery with cage rest. While rigid bandaging techniques are unlikely to be helpful in most cases, they can be beneficial in cases of cervical injury. Strict activity restriction is essential for spinal fractures. As with head trauma, there is no indication that steroids are warranted to manage avian spinal trauma but NSAIDs along with general supportive care measures are indicated. Surgical stabilization of spinal fractures has not been investigated in birds.

Fractures

Fractures can occur in association with flying accidents or crushing trauma and when pathology of the bone is present. Fractures should be immobilized in stable patients to prevent further trauma. Open fractures should be cleaned and reduced, taking care in the case of pneumatic bones not to introduce fluids into the air sacs. Antibiotic therapy should be initiated. The figure-of-eight bandage can be useful to manage wing fractures distal to the humerus, soft tissue injuries, and luxations. If there is a humeral or pectoral girdle fracture, a body wrap should be placed. A modified Robert Jones bandage (birds >200 g) or Altman tape splint (birds <200 g) can be used to stabilize fractures distal to the femur. Once the bandage is in place, the bird should be monitored to make sure there is no evidence of increased stress, dyspnea, or chewing/destroying the bandage. Appropriate fracture repair should follow when warranted (see Chapter 37).

Hemorrhage

Hemorrhage can be internal or external. Common sites of external bleeding include broken blood feathers, injured nails or beak, lacerations, and trauma to the ventral carpus from repeated beating of wings against the sides of the cage or another surface during an episode of panic. It should be noted that blood from the ventral wings will transfer to feathers on the lateral aspect of the body wall. When blood is seen in this location, the ventral wings should be inspected carefully. It may be necessary to thoroughly clean blood clots to identify if ongoing bleeding is present.

Broken blood feather

The larger primary rectrices and remiges can bleed significantly if the calamus is fractured during the time they are actively emerging. Small defects may effectively clot and require no additional therapy. Larger fractures may bleed significantly and require intervention. The entire feather can be gently removed from the follicle by grasping the base of the feather with hemostats; this procedure causes significant discomfort and requires appropriate analgesia. In some cases, simple ligation may be helpful as well. If the feather is removed, blood will continue to emerge from the follicle; direct pressure is recommended. Avoid applying styptic powders or other irritating substances into the feather follicles or surrounding skin. If hemorrhage from the follicle continues, tissue adhesive or a small absorbable suture can be placed to temporarily close the follicle.

Nail injury

Toes may become traumatized in toys, on cages, or by conspecifics. Styptic powder or cautery can be used on bleeding nail tips. In the case of complete nail avulsion, the exposed tissue should be cleaned and covered with a light bandage or tissue adhesive.

Beak trauma

Beak trauma can be seen in cases of head trauma or falls, toy entrapment, and bites from conspecifics. Simple rhinothecal tip fractures can be managed with manual pressure, styptic powder, or cautery. Small incomplete fractures may be managed by applying layers of surgical glue and baking soda. It should be kept in mind some bleeding rostral rhinothecal tomial fractures may be missed as blood may wick into the oral cavity and/or the bird continually licks the site. More complex fractures or avulsions may require surgical or acrylic stabilization. Analgesia is important and some birds may benefit from temporary gavage feeding or being fed a softer diet. See Chapter 31 for further details on management of beak injuries.

Laceration/other

Skin wounds can be self-inflicted or caused by conspecifics, falls, and predators. Wing tip trauma can occur with episodes of night fright and falls. Open wounds should be cleaned and closed if fresh and noncontaminated. Some wounds cannot be

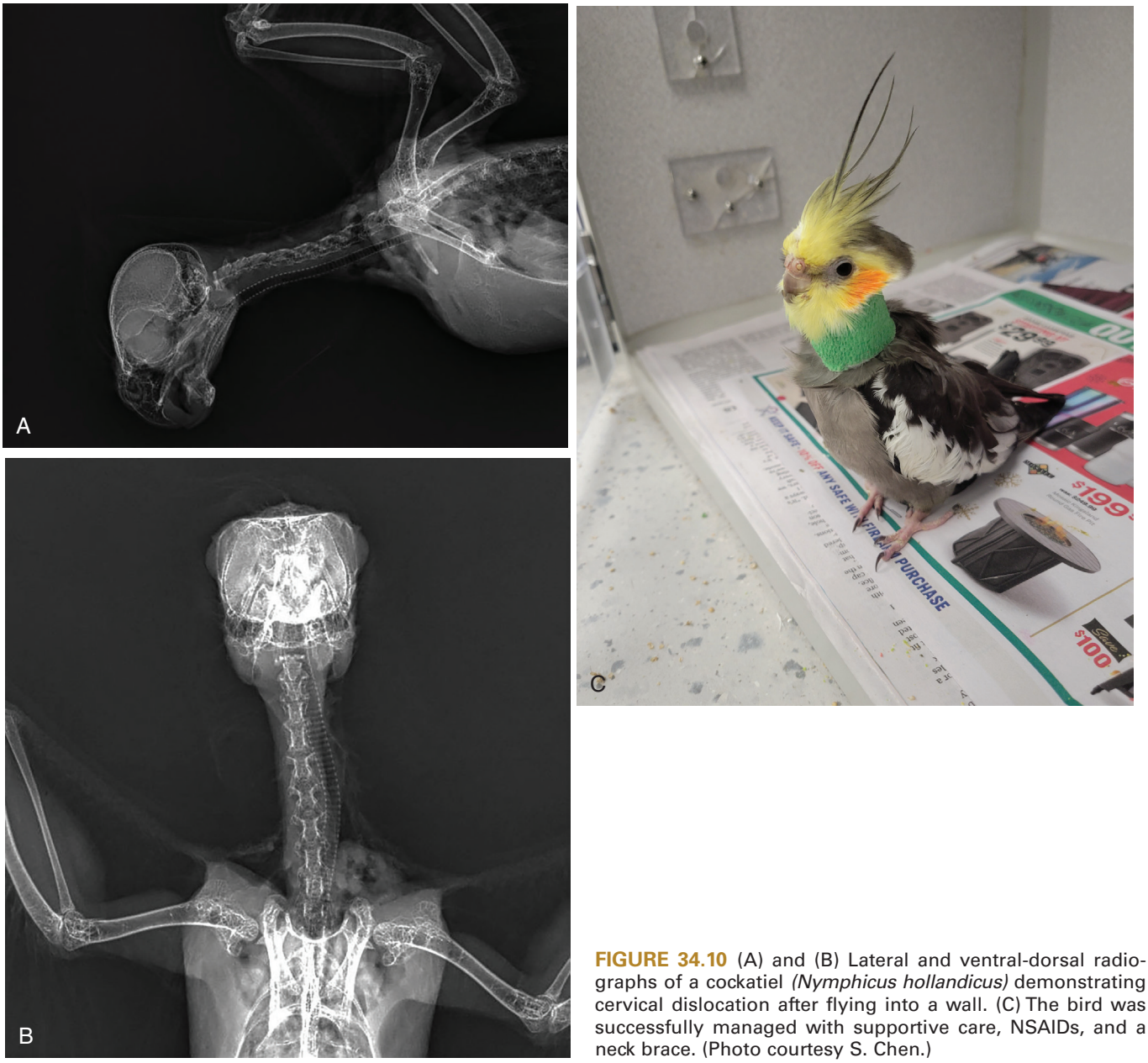


FIGURE 34.10 (A) and (B) Lateral and ventral-dorsal radiographs of a cockatiel *(Nymphicus hollandicus)* demonstrating cervical dislocation after flying into a wall. (C) The bird was successfully managed with supportive care, NSAIDs, and a neck brace. (Photo courtesy S. Chen.)

managed by primary closure, and collars or bandages may be required to protect the wound while healing (see Chapter 36).

Constriction injury

Constriction injuries can occur to an extremity from a leg band (Figure 34.12) or trauma from toys, strings, or nesting material, injuries, and skin hyperplasia secondary to malnutrition (see Chapter 41). Magnification can be helpful when removing constricting material and great care should be taken to avoid causing further injury. Kras leg band cutters (Vet Specialty Products, Fort Lauderdale, FL, USA), hemostats, wire cutters, or bolt cutters may be used to remove metal bands, taking care not to injure the leg. Appropriate wound care should follow removal of the constricting material. In some cases, constriction may be severe enough to cause loss of extremities. Pentoxifylline and/or peripheral vasodilators may be beneficial in acute constriction injury.

Soft tissue injuries

Bite wounds

Bite wounds can be minor to catastrophic and include coelomic cavity penetration or traumatic limb amputation. Bites can come from household pets, or for birds housed outside, wildlife predators such as raccoons and birds of prey (Figure 34.13). Of particular concern is the danger of septicemia from bite or scratch wounds from cats. Broad-spectrum antibacterial therapy should be initiated immediately, even if wounds appear to be minor (see Chapter 7 and Chapter 36). Bacterial septicemia is difficult to treat in any species. For these patients, IV antibiotics are recommended. One author uses piperacillin-tazobactam at



FIGURE 34.11 Close-up of the soft tissue adjacent to the spine in a bird with suspected spinal trauma. Dampening the feathers can help detect bruising.

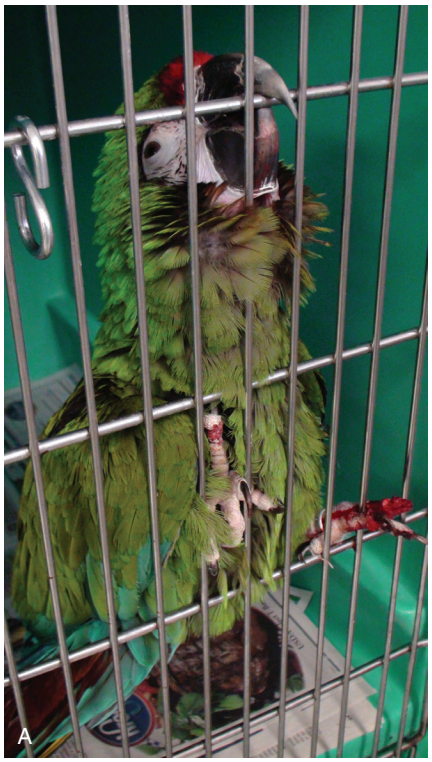




FIGURE 34.12 Constricting leg band injury of the left tarsometatarsal region in a cockatiel *(Nymphicus hollandicus)*. Once the aluminum band was cut transversely, it was able to be bent and removed from the leg without further injury including fracture occurring. With protective bandaging, pain management, and wound care, this injury ultimately healed well. (Photo courtesy Brian Speer.)



FIGURE 34.13 (A) Traumatic amputation of all toes of one foot due to a predator injury. A raccoon grabbed the bird through the cage bars when the bird was inadvertently left outside overnight. (B) A pet chicken *(Gallus gallus domesticus)* with a very large soft tissue wound caused by a predator. Despite the size of the wound, general wound care and surgical repair were successful. (A, Courtesy Angela Lennox. B, Courtesy Crystal Matt.)



FIGURE 34.14 (A) Traumatic tail avulsion in a Grey parrot *(Psittacus erithacus)* due to a falling injury. Note the position of the injury *(bottom arrow)* in relation to the vent *(top arrow)*. (B, C) Repair of a traumatic ventral tail avulsion injury in a cockatiel *(Nymphicus hollandicus)* that occurred several weeks prior to presentation. There is significant granulation tissue that required trimming prior to suturing and wound closure. Nonabsorbable suture was used in a mattress suture pattern to reduce tension. The procedure was performed in the sedated patient with a lidocaine/bupivacaine local block. (A, Permission Amy Boxberger, DVM. B, C, Courtesy Angela Lennox.)

100 mg/kg IV, IO, or, if vascular access is unavailable, IM every 4 to 6 hours for these cases. A pharmacokinetic study in Hispaniolan Amazon parrots *(Amazona ventralis)* demonstrated 87 mg/kg delivered IM q3–4h produced blood levels likely useful for susceptible infections.[17] Additional information on antibiotic therapy can be found in Chapter 6 and Chapter 7. Wound management may necessitate flushing, bandaging, suturing, or fracture repair; care should be taken to avoid flushing wounds that could connect to the air sacs or lungs.

Frostbite/freezing injuries

Most injuries of this type are seen in birds normally housed outdoors in colder climates, in particular, pet poultry. It can also be seen in birds that accidentally escape outdoors. Immediate therapy should focus on analgesia and acute management of tissue trauma, which can be minor (margins of the comb in poultry) to severe (loss of limbs). The use of pentoxifylline and/or peripheral vasodilators may be considered.

Traumatic tail avulsion

Falling and crashing injuries can cause avulsion of the tail and tearing of the tail base skin caudal to the vent. Some of these are the result of improper wing trimming (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 22, Advancements in Management of the Welfare of Avian Species). Owners often note bleeding from the tail area, and veterinarians may mistake the skin injury for trauma to the vent itself (Figure 34.14A). This injury appears most often in cockatiels but can happen in any bird, especially those that are overweight, with an inappropriately severe wing clip or other issue impairing flight. Some owners report intermittent bleeding as the scab forms and then is disrupted.

Traumatic tail avulsion is best repaired surgically. If the injury is not repaired, considerable granulation tissue often forms, preventing healing by second intention. The granulation tissue is trimmed, the skin margins freshened, and the injury sutured using nonabsorbable sutures and individual

mattress sutures taking relatively large bites of adjacent tissue (Figure 34.14B, C). In some cases, suture can be placed around the base of the primary rectrices to help reduce tension. A splint made from a tongue depressor spanning the tail and back may be applied over the dorsal aspect of the tail during the healing phase to prevent dehiscence during the healing period. Strict cage rest and prevention of additional falls are critical to prevent reinjury during the 2- to 3-week healing period. It should be noted there may be a link to future feather-destructive behavior and self-mutilation of the area, which may require eventual amputation.

Keel injuries

Hard falls can produce injuries to the skin and bone of the keel, even causing bone necrosis (Figure 34.15). Most injuries are in overweight birds, or those unable to perform a controlled landing (improper wing clip, wing injuries). Tension over the keel and movement during flapping results in poor healing by second intention, and most benefit from removal of necrotic skin and bone and suturing with tension-relieving suture patterns.

Thermal burns

Thermal burns can occur in household birds exposed to open flames or any hot surface. Aggressive supportive care is essential as there are substantial fluid and protein losses through burns. Analgesia and antimicrobial therapy are necessary during the healing process, as well as wound care. Silver sulfadiazine is beneficial for care of thermal burns, as well as frequent bandage changes. Hydrocolloid bandages may be useful but must be changed once to twice daily, and their use should be discontinued in the presence of infection. Feathers in the surrounding area should be cut rather than removed (unless caring for a wild bird) to prevent further contamination of the affected areas and allow for ease of wound care. Avian skin has the capacity to epithelialize over large areas once the burned areas have granulated. Skin grafts may be a viable option in some situations (see Chapter 36).



FIGURE 34.15 An umbrella white cockatoo *(Cacatua alba)* with a traumatic keel wound following a fall to the ground. (Courtesy L. Thielen.)

Electrocution injuries

Most electrocution injuries in household birds are from chewing on wires. Injuries may be difficult to detect in the acute phase but present as oral cavity swelling progressing to necrosis. Birds may be unwilling to eat, and supportive care may consist of tube feeding or in severe cases an ingluviotomy tube. Electrocution injuries in wild birds are addressed in more detail in Chapter 50.

Crop burns

Crop burns are addressed in Chapter 41.

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CHAPTER 35

Advances in Management of Pain in Birds

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Pain management is one of the areas of avian medicine that has advanced substantially in recent years. This chapter focuses on the most significant therapeutic advancements in pain management in birds and is presented as a continuation of a previous chapter on recognition, assessment, and management of pain in birds published in *Current Therapy in Avian Medicine and Surgery*, Vol 1.

OPIOID DRUGS

Opioids are mostly used for the treatment of moderate-to-severe pain. They act as agonists, partial agonists, mixed agonists/antagonists, or antagonists by reversibly and selectively binding to μ, κ, δ, and nociceptive receptors in the central nervous system (CNS) and peripheral nervous system. While distribution, number, and type of opioid receptors in the brainstem and spinal cord appear to have been conserved across species, more variation seemingly exists in other parts of the brain such as the forebrain, which could account for variation in the effects of opioids seen clinically. Recent studies looking at opioid receptors in chickens *(Gallus gallus domesticus)*,[1] pigeons *(Columba livia domestica)*, and cockatiels, *(Nymphicus hollandicus)*[2] together with a large number of recent studies on opioids in birds, are challenging the paradigm that κ-opioid receptor agonists (e.g., butorphanol, nalbuphine) are the opioids of choice, finding that in some species μ-opioid receptor agonists (e.g., morphine, hydromorphone, methadone, fentanyl, buprenorphine) might actually be the better choice when treating pain in some species. The results of these studies are described in the following paragraphs. Interestingly, when looking at the structure of opioid receptors (μ, κ, δ) in the peregrine falcon *(Falco peregrinus)*, snowy owl *(Bubo scandiacus)*, and blue-fronted Amazon parrot *(Amazona aestiva)*, nucleotide homologies with humans ranging from 77% to 82% were found, which might account for some of the differences seen in the efficacy of these drugs.[3] Most importantly, a large variability in the pharmacokinetics (PK) and pharmacodynamics (PD) between species, and in some cases also age and sex, has made it clear that generalizations within the class Aves and extrapolation between species need to be exercised with caution. Common opioids that have been studied and used in birds include morphine, hydromorphone, methadone, fentanyl, buprenorphine, and butorphanol (Table 35.1).

Morphine

Morphine is a μ-opioid receptor agonist that has been infrequently used in avian medicine because some of the early studies with this drug in domestic fowl yielded conflicting results.[19] In a recent study evaluating the analgesic efficacy of morphine, lame broiler chickens underwent an obstacle course (OC) and latency-to-lie (LTL) test before injection, and at 30 minutes and 2 hours after injection, to assess their walking ability and their standing ability.[4] Injection of 2 mg/kg IV of morphine resulted in an increased time to finish the OC as sedation occurred, resulting in the chicks tending to sit down rather than walk and confounding assessment of its analgesic effects. The authors hypothesized that morphine might produce analgesia without sedation at lower doses while requiring more frequent administration due to its shorter elimination half-life. However, further studies are warranted.

Hydromorphone

Hydromorphone is a μ-opioid receptor agonist with a potency of approximately five to seven times that of morphine in humans.[28] Hydromorphone is administered by the parenteral, transmucosal, and epidural routes for the management of both acute and chronic pain.[28] A recent study evaluated the thermal antinociceptive effects of hydromorphone hydrochloride following IM administration to orange-winged Amazon parrots *(Amazona amazonica)*.[5] Hydromorphone at 1 and 2 mg/kg significantly increased the thermal foot withdrawal response at 0.5, 1.5, and 3 hours, and 1.5 and 6 hours, respectively, compared with the response after saline administration. In a follow-up study to evaluate the PKs of hydromorphone hydrochloride following intravenous and intramuscular administration of 1 mg/kg, plasma hydromorphone concentrations in orange-winged Amazon parrots were maintained above 1 ng/mL for 6 hours in 6/7 and 8/8 for the IV and IM routes, respectively.[6] The IM mean bioavailability was 97.6%. The mean maximum concentration (C_{max}) was 179.1 ng/mL at 17 minutes after IM administration. The mean terminal half-lives were 1.45 and 1.74 hours after IV and IM administration. This study indicates that hydromorphone hydrochloride has a positive PK profile in Amazon parrots, with a large volume of distribution and high bioavailability after IM administration. However, clearance from the plasma is rapid, resulting in a short terminal half-life, which may require frequent dosing to

TABLE 35.1

Opioid and Opioid-Like Analgesics Evaluated in Avian Species by Either Pharmacokinetic or Pharmacodynamic Studies

Drug	Dosage (mg/kg)	Route	Frequency (q/h)	Species	Comments	Type of Study	Reference Number
Morphine	2	IV	Single dose	Chicken *(Gallus gallus domesticus)*	Decreased time to finish the obstacle course and sedation, which confounded assessment of its analgesic effects	PD	4
Hydromorphone	0.1, 1, 2	IM	Single dose	Orange-winged Amazon parrot *(Amazona amazonica)*	Increased thermal nociceptive thresholds at 1 and 2 mg/kg	PD	5
	1	IM, IV	Single dose	Orange-winged Amazon parrot *(A. amazonica)*	Maintained above 1 ng/mL plasma concentrations for 6 hours following IM administration, with a terminal half-life of 1.74 hours	PK	6
	0.3, 0.6	IM	Single dose	Great horned owl *(Bubo virginianus)*	Increased thermal nociceptive thresholds at 0.3 and 0.6 mg/kg	PD	7
	0.6	IM, IV	Single dose	Great horned owl *(B. virginianus)*	Maintained above 1 ng/mL plasma concentrations for 9 hours following IM administration, with a terminal half-life of 1.62 hours	PK	8
Methadone	1, 3, 6	IM	Single dose	Orange-winged Amazon parrot *(A. amazonica)*	Increased thermal nociceptive thresholds at 1 and 2 mg/kg	PD	9
	6	IM, IV	Single dose	Orange-winged Amazon parrot *(A. amazonica)*	Elimination half-life of 2.29 hours	PK	10
	6	IM	Single dose	Chicken *(Gallus gallus domesticus)*	Decreased MAC of isoflurane by 30% at 15 minutes after administration	PD	11
	6	IM	Single dose	Chicken *(Gallus gallus domesticus)*	Elimination half-life of 2.95 hours	PK	12
Fentanyl	25 μg/hour	TD patch	Single dose	Chicken *(Gallus gallus domesticus)*	Large interindividual differences. Maintained plasma concentrations above 0.2–1.2 ng/mL for 72 hours	PK	13
	5	TD solution	Single dose	Helmeted guineafowl *(Numida meleagris)*	Maintained plasma concentrations above 0.6 ng/mL for 7 days	PK	14
Buprenorphine sustained-release	1.8	SC, IM	Single dose	American kestrel *(Falco sparverius)*	Maintained plasma concentrations above 1 ng/mL for 48 hours and elimination half-life of 11.1 and 13.5 hours after SC and IM administration	PK	15
	1.8	IM	Single dose	American kestrel *(Falco sparverius)*	Increased thermal nociceptive thresholds for at least 24 hours	PD	16
Buprenorphine high-concentration	0.3, 1.8	SC	Single dose	Red-tailed hawk *(Buteo jamaicensis)*	Plasma concentrations were maintained above 1 ng/mL for at least 24 and 48–72 hours, with an elimination half-life of elimination half-life was 6.23 and 7.84 hours for the low and high dose, respectively	PK	17
	1.8	SC	Single dose	American flamingo *(Phoenicopterus ruber)*	Plasma concentrations were maintained above 1 ng/mL for at least 48–72 hours, with an elimination half-life of 12.6 hours	PK	18
	0.1, 1, 2	SC	Single dose	Orange-winged Amazon parrot *(A. amazonica)*	Increased thermal nociceptive threshold at 2 mg/kg	PD	Unpublished

Continued

TABLE 35.1

Opioid and Opioid-Like Analgesics Evaluated in Avian Species by Either Pharmacokinetic or Pharmacodynamic Studies—cont'd

Drug	Dosage (mg/kg)	Route	Frequency (q/h)	Species	Comments	Type of Study	Reference Number
Butorphanol	2	IV	Single dose	Chicken *(Gallus gallus domesticus)*	Decreased time to finish the obstacle course	PD	19
	5	IM	Single dose	Hispaniolan Amazon parrot *(Amazona ventralis)*	Increased thermal nociceptive threshold for 30 minutes	PD	20
Butorphanol-P407 25%	12.5	SC	Single dose	Hispaniolan Amazon parrot *(A. ventralis)*	Plasma concentrations of butorphanol remained above 100 ng/mL for more than 3 hours but less than 8 hours and an elimination half-life of 3.41 hours	PK	21
	12.5	SC	Single dose	Hispaniolan Amazon parrot *(A. ventralis)*	Did not increase the thermal nociceptive thresholds	PD	20
	247 μg/kg/hour	SC osmotic pump	CRI		Plasma concentrations remained above 60 ng/mL for 7 days in most birds	PK	22
Tramadol	30	PO	Single dose	Muscovy duck *(Cairina moschata)*	Improved a number of variables in induced acute arthritis model as measured by ground-reactive forces	PD	23
	30	PO	Single dose	Muscovy duck *(C. moschata)*	Elimination half-life of 3.95 hours	PK	24
	10	PO	Single dose	African penguin *(Spheniscus demersus)*	Elimination half-life for tramadol was 7.3 hours. Suggests q24h administration	PK	25
	5, 15, 30	IM	Single dose	Harris' hawk *(Parabuteo unicinctus)*	No effects following 5 mg/kg, but 15 and 30 mg/kg resulted in electrical nociceptive effects and mild to moderate sedation	PD	26
Tapentadol	30	PO	Single dose	Hispaniolan Amazon parrot *(A. ventralis)*	Detectable plasma concentrations in only 2/7 birds and elimination half-life of 24.8 minutes. Limited use at this dose and route	PK	27

CRI, Constant rate infusion; *IM,* intramuscular; *IV,* intravenous; *MAC,* mininum anesthethic concentration; *PD,* pharmacodynamic; *PK,* pharmacokinetic; *PO,* orally; *q/h,* every hour; *SC,* subcutaneous; *TD,* transdermal.

provide adequate analgesia. In addition, during the study into the antinociceptive effects, significant agitation, nausea-like behavior (including vomiting in one bird), ataxia, and pupillary constriction were observed following administration of the 1 and 2 mg/kg hydromorphone doses. Thus, while hydromorphone at 1 and 2 mg/kg IM appears to have desired analgesic effects, especially for severe pain management, the presence of adverse side effects warrants consideration of lower doses for clinical use in orange-winged Amazon parrots. In great horned owls *(Bubo virginianus)*, the PKs following IM administration of 0.6 mg/kg showed high bioavailability and rapid elimination.[8] Mean C_{max} was 225.46 ± 0.2 ng/mL at 13 minutes after IM injection. The mean terminal half-lives were 1.35 and 1.62 hours after IV and IM administration, respectively. The metabolite H3G was readily measured shortly after administration by both routes. This study was the first to document the presence of the metabolite H3G in avian species, which suggests similar hydromorphone metabolism as in mammals. In a follow-up study in the same species, hydromorphone at 0.3 and 0.6 mg/kg resulted in significant thermal antinociceptive effects for up to 3 and 1.5 hours, respectively, and mild to moderate sedation.[7] Two owls were observed tremoring after administration of the 0.6 mg/kg dose, which was not noted after the 0.5-hour timepoint; no other adverse effects were identified. These studies offer scientific evidence to support the use of hydromorphone in great horned owls for pain management.

Methadone

Methadone is a μ-opioid receptor agonist that acts similarly to morphine with regard to its degree of analgesia and duration of action in small animals.[29] However, it is also a noncompetitive inhibitor of *N*-methyl-D-aspartate (NMDA) receptors and can reduce reuptake of norepinephrine and serotonin, which may contribute to its analgesic effects. Due to these additional mechanisms of action, it is potentially more efficacious than

other μ-agonists (e.g., morphine), particularly for neuropathic or chronic pain.[29] Methadone is more lipid-soluble than morphine and approximately 1 to 1.5 times as potent. It does not cause significant histamine release when administered intravenously and has been shown to cause less excitation or vomiting than some other μ-receptor agonists.[29]

In hens, methadone at the dose of 6 mg/kg IM decreased the isoflurane minimum anesthetic concentrations by 30% at 15 minutes after administration. At this dose, in spontaneous ventilated hens, methadone produced mild respiratory acidosis, decreased heart rate, and increased systemic blood pressure, with occasional cardiac arrhythmias.[11] In the same study, hens under isoflurane anesthesia and using the same 6 mg/kg dose, the PK parameters were determined with an elimination half-life of 177 minutes and a C_{max} of 950 ng/mL at 18.3 minutes after IM administration. The bioavailability was estimated to be 79% for IM administration.[12]

In orange-winged Amazon parrots, methadone demonstrated comparable PKs, with a mean bioavailability of 85.7% and mean C_{max} of 515 ng/mL at 25 minutes after IM administration.[10] Mean plasma methadone concentrations were 393 ± 135 ng/mL at 0.5 hours and 242 ± 73 ng/mL at 1.5 hours. Mean terminal half-life was short (2.35 and 2.29 hours for IV and IM administration, respectively), indicating frequent dosing may be needed to maintain plasma concentrations above the analgesic threshold. A follow-up study, evaluating the thermal antinociceptive effects,[9] showed that methadone, administered at 6 mg/kg IM, resulted in a significant increase in thermal threshold in orange-winged Amazon parrots when compared to saline administration, while lower doses of 1 and 3 mg/kg did not yield a significant effect. In the same study, methadone caused significant agitation, ataxia, and nausea-like behavior when compared to saline administration. Hence, while methadone at 6 mg/kg IM appears to have the desired analgesic effects, the presence of these adverse effects warrants consideration of lower doses for clinical use in orange-winged Amazon parrots. In addition, while the PK properties appeared comparable between chickens and orange-winged Amazon parrots, the potential for species differences in drug metabolism and response warrants further studies to evaluate the analgesic and adverse effects of methadone in avian species.

Fentanyl

Fentanyl is a short-acting μ-opioid receptor agonist often administered as a continuous rate infusion or as a transdermal patch for severe pain. The patch delivery theoretically achieves and maintains steady-state therapeutic plasma levels more readily than bolus administration, being less stressful than continuous rate infusion, which requires IV access. In a study including chickens of Brahma, Delaware Redstar, and Wyandotte breeds, the plasma concentrations of fentanyl were evaluated following the application of 25 μg/hour transdermal fentanyl patches (TFP).[13] The TFPs were in place for 72 hours over the left iliopsoas muscle after plucking the feathers. There was a significant degree of interindividual difference in maximum plasma fentanyl concentrations that was attributed to absorption variability, but all chickens reached the human target plasma concentrations of 0.2 to 1.2 ng/mL within 2 to 4 hours and had a rapid decrease following the removal of the patch. Based on data from this PK study, fentanyl transdermal application of patches for chickens targeted to provide 8 to 10 μg/kg per hour of fentanyl resulted in fentanyl plasma concentration within the range considered therapeutic in people for 72 hours.[13] However, because of substantial individual variation in absorption, results from longer applications would likely be inconsistent among patients, hence warranting further studies to determine effective and safe plasma concentrations of fentanyl in this and other bird species.

Another study evaluated the effects of a transdermal fentanyl solution in helmeted guineafowl *(Numida meleagris)*.[14] The solution was placed on the skin in the interscapular region at 5 mg/kg, which resulted in mean plasma concentrations above those considered analgesic for dogs (0.6 ng/mL) from 4 hours through at least 7 days with no evidence of adverse effects or change in behavior. Unfortunately, this commercial formulation had been discontinued at the time of writing this chapter.

Buprenorphine

Buprenorphine hydrochloride has a slow onset of action with a longer duration than other opioid drugs and a unique and complex pharmacological profile. Buprenorphine is thought to act as a μ-receptor agonist, but its κ-receptor activities are less well defined. In a PK study in American kestrels *(Falco sparverius)*, subcutaneous (SC) and IM administration of 1.8 mg/kg of Buprenorphine SR-LAB (Wildlife Pharmaceuticals, Windsor, CO, USA) were both characterized by rapid absorption.[15] Forty-eight hours after both IM and SC administration, mean plasma concentrations remained above target concentrations of >1 ng/mL. The elimination half-life was 13.5 and 11.1 hours, respectively, which is nine times longer than the half-life of 1.5 hours reported in a similar study in American kestrels that received 0.6 mg/kg IM of buprenorphine HCl.[30] This difference was expected due to the sustained-release properties of this formulation and the higher dose administered than in the prior study. Subcutaneous hematomas in three of the birds that received Buprenorphine SR-LAB SC were attributed to traumatic administration but resolved without intervention. A follow-up study evaluating the thermal antinociceptive effects of this formulation in the same species concluded that 1.8 mg/kg IM could require administration for at least every 24 hours to manage pain, although the duration of action could be longer depending on the severity and type of pain, adjunctive therapy, and the individual response.[16]

In red-tailed hawks *(Buteo jamaicensis)*, the PKs of two doses of a concentrated formulation of buprenorphine (Simbadol; Zoetis, Kalamazoo, MI, USA) were evaluated after SC administration.[17] Maximum buprenorphine concentration was achieved at approximately 5 and 15 minutes for the 0.3 and 1.8 mg/kg doses, and plasma concentrations were maintained above 1 ng/mL for at least 24 and 48 to 72 hours, respectively. The mean elimination half-life was 6.23 and 7.84 hours for the low and high doses, respectively. Baseline sedation scores were significantly lower than all other time points for each individual, with return to near baseline by 24 to 48 hours, consistent with mild to moderate sedation. No adverse effects were noted in any birds. The use of this concentrated buprenorphine formulation might help mitigate stress related to repeated handling and administration of treatments in raptors while providing consistent pain management. An

additional benefit is that, for the same dose, this formulation is more cost-effective than the standard buprenorphine formulation which is a very important factor for wildlife rehabilitation. A similar PK study in American flamingos *(Phoenicopterus ruber)* following administration of 1.8 mg/kg SC of the same concentrated formulation showed a longer terminal half-life of 12.6 hours, maintaining above target plasma concentrations in other species for 48 to 72 hours.[18] Additional studies are needed to evaluate the efficacy of this opioid in flamingos.

In orange-winged Amazon parrots, the thermal antinociceptive effects of a concentrated formulation of buprenorphine (Simbadol) after SC administration were evaluated.[31] Buprenorphine at 2 mg/kg resulted in a small but significant increase in thermal threshold when compared to saline, while lower doses of 0.1 and 1 mg/kg did not result in a significant effect. No significant effect of buprenorphine on the agitation-sedation score was observed at any of the doses evaluated. These results provide evidence of weak efficacy at lower doses for pain management in this species, like previous findings in the cockatiels, and highlight the differences in efficacy with the raptor species evaluated, which is an efficacious opioid drug. Further studies are needed to fully determine the analgesic and adverse effects of buprenorphine in psittacine birds and warrant its evaluation in other species.

Butorphanol

Butorphanol is a mixed κ-opioid receptor agonist and μ-opioid receptor antagonist opioid drug. This opioid has historically been one of the most commonly used opioid analgesics in birds for moderate and severe pain, despite many studies suggesting that it requires very frequent dosing.[19] In a recent study evaluating the analgesic efficacy of butorphanol, lame broiler chickens underwent an OC and LTL test before injection at 30 minutes and 2 hours after injection to assess their walking ability and their standing ability.[4] Following the injection of 2 mg/kg IV butorphanol, chickens finished the OC faster, and the authors concluded that it may be analgesic in chickens for up to 2 hours.

In Hispaniolan Amazon parrots *(Amazona ventralis)*, the thermal antinociceptive effects of a formulation containing butorphanol in a 25% poloxamer 407 base (But-P407 25%; Sigma-Aldrich, Oakville, ON, Canada) were evaluated following SC administration.[21] P407 (Sigma-Aldrich) is a thermosensitive hydrogel, a compound that exhibits a property known as reverse gelation. P407 is liquid at room temperature, allowing easy mixing with therapeutic agents and routine handling for injection. Once injected into homeothermic animals, micellar packing forms at the warmer temperature of the body and the compound becomes a gel. This micellar packing is responsible for the high viscosity, partial rigidity, and slow dissolution of the gel, which makes it a highly effective sustained-release system for both hydrophilic and hydrophobic drugs. Butorphanol was well absorbed from the But-P407 25% with a maximal plasma butorphanol concentration reached at 90 minutes. Plasma concentrations of butorphanol remained above 100 ng/mL for 3 and up to 8 hours with no noted adverse effects. The mean elimination half-life was 3.41 hours, with a very high interindividual variability, which is almost five times longer than the half-life reported in the same species for the standard butorphanol tartrate formulation.[32] A dosage of 12.5 mg/kg SC would theoretically provide analgesia for 3 to 8 hours. Unfortunately, those theoretical analgesic effects could not be confirmed in a later thermal antinociceptive study in orange-winged Amazon parrots as no significant difference was identified between the results for But-P407 and those for either the control treatment (saline solution or P407).[20] Only some mild sedation was noted following But-P407 administration. In the same study, the thermal antinociceptive effects of the standard butorphanol tartrate formulation were also evaluated following the administration of 5 mg/kg. At this time, a significant but short-lived increase in thermal threshold was noted, but only at the 30-minute timepoint after butorphanol tartrate (vs saline solution) administration and not at 1.5, 3, or 6 hours. These results with the standard butorphanol tartrate formulation confirm the previous findings evidencing the very short duration of action of this drug in psittacine birds and together with the other recent findings with hydromorphone in orange-winged Amazon parrots, the historical paradigm that κ-opioid receptor agonists–μ-opioid receptor antagonists are the opioids of choice for management of acute pain in psittacines, warrants reevaluation.

In common peafowl *(Pavo cristatus)*, the PKs of butorphanol administered via an osmotic pump, a novel delivery method, were assessed.[22] Two osmotic pumps containing 2 mL butorphanol at a concentration depending on body weight of the bird and administering the drug at a rate of 247 μg/kg/hour each were surgically implanted in 12 birds (two pumps per bird) and removed 7 days later. The osmotic pumps used in the study were 5.1 cm in length and weighed 5.1 g. Plasma butorphanol concentration was measured before implantation SC in the left inguinal region, for several hours to days after implantation, and 3 and 6 hours after the implant removal. In most birds, the plasma concentration reached 60 ng/mL after 24 hours, stayed above this threshold for 7 days, and then decreased rapidly after implant removal. There was no evidence of sedation or adverse effect. These results indicate that osmotic pumps may provide a promising new technique for pain management in large birds, although it should be noted that deep sedation or general anesthesia will likely be required for implantation and removal.

Tramadol

Tramadol hydrochloride is an analgesic drug often used in avian medicine. It is a synthetic μ-opioid receptor agonist that appears to have variable efficacy depending on the species. The efficacy is thought to be primarily related to the production of the *O*-desmethyltramadol metabolite (M1). This metabolite acts centrally over the opioid receptor and is thought to have almost 200 times higher affinity for this receptor than the parent drug. Tramadol also has effects on the κ- and δ-opioid receptors, as well as inhibiting reuptake of norepinephrine and serotonin, and is used for moderate pain. In Muscovy ducks *(Cairina moschata domestica)*, tramadol at 30 mg/kg PO was effective in improving a number of variables in a temporary arthritis model induced by an injection of 3% monosodium urate solution into the intertarsal joint and assessed by ground-reactive forces measured by a pressure-sensitive walkway (PSW) system.[23] A PK study of the same investigators in the same species following administration of tramadol at 30 mg/kg PO showed that it has an elimination half-life of 3.95 hours with a C_{max} of 780 ng/mL at 1.16 hours.[24]

In this study, it was also demonstrated that Muscovy ducks readily produce the M1 metabolite and maintain it well above the concentrations that are known to be therapeutic for humans and other avian species.

In African penguins *(Spheniscus demersus)*, the PK parameters were determined following 10 mg/kg PO administration.[25] The elimination half-life for tramadol was 7.3 hours, which is significantly longer than other species reported, with a C_{max} of 815 ng/mL at 4.6 hours. Tramadol and M1 concentrations were determined and were consistent with therapeutic concentrations in humans through 12 hours in 9/15 birds for tramadol and 36 hours in 14/15 birds for M1. Based on plasma concentrations alone, the authors concluded that an oral dose of 10 mg/kg of tramadol once daily could be used.

In Harris' hawks *(Parabuteo unicinctus)*, the electrical antinociceptive effects of tramadol were evaluated.[26] No effects were noted following 5 mg/kg IM, but 15 and 30 mg/kg IM resulted in electrical nociceptive effects and mild to moderate sedation.

Tapentadol

Tapentadol is an analgesic agent that acts as both a μ-opioid receptor agonist and a norepinephrine reuptake inhibitor. It is a common therapeutic agent in human medicine for managing acute and chronic pain, and it is now being investigated for use in veterinary medicine. The PKs of tapentadol were evaluated in Hispaniolan Amazon parrots following a single 30 mg/kg PO of compounded 5 mg/mL tapentadol suspension.[27] Detectable plasma concentrations were identified in only 2/7 birds, and the bird with the highest plasma levels had a maximum concentration of 143 ng/mL and a half-life of 24.8 minutes. The low oral bioavailability and short half-life of this drug in Hispaniolan Amazon parrots suggest that this drug would be of limited clinical use in this species, but further studies are recommended in other avian species to evaluate its analgesic potential.

NONSTEROIDAL ANTIINFLAMMATORY DRUGS

Nonsteroidal antiinflammatory drugs (NSAIDs) have different mechanisms of action. The cyclooxygenase (COX) inhibitors act through enzyme cyclooxygenase 1 and/or 2 inhibition, helping to relieve mild to moderate pain as a result of inflammation. Newer COX inhibitors focus their action on COX-2 inhibition while sparing COX-1 inhibition to limit the inhibition of physiologically important prostaglandins. A new class of NSAIDs has recently appeared on the market and has a different mechanism of action through prostaglandin E_2 (PGE_2) receptor antagonism. NSAIDs are often used in the postoperative period and in patients suffering from trauma or inflammation of musculoskeletal tissues. Aside from their effects in patients with acute pain, NSAIDs are helpful in alleviating chronic pain such as that experienced by patients with osteoarthritis. Commonly used NSAIDs in birds include meloxicam, carprofen, ketoprofen, celecoxib, and piroxicam (Table 35.2), which each have a distinct COX-1/COX-2 ratio resulting in differences in efficacy and toxicity.

Meloxicam

Meloxicam is the most widely used antiinflammatory medication today in avian practice. Meloxicam is a COX-2-selective NSAID that may demonstrate more COX-1 selectivity at higher dosages.[47,48] Meloxicam is currently available as an oral tablet, oral suspension, and injectable formulation. Single-dose PK studies of meloxicam in several breeds of chickens,[39,40,47] ostriches *(Struthio camelus)*, emus *(Dromaius novaehollandiae)*,[47,49] mallard ducks *(Anas platyrhynchos)*,[48] Pekin ducks *(Anas platyrhynchos)*,[50] Bilgorajska geese *(Anser anser)*,[51] brown pelicans *(Pelecanus occidentalis)*,[36] turkeys (*Meleagris* spp.),[48] pigeons,[48,52] zebra finches *(Taeniopygia guttata)*,[53] ring-necked parakeets *(Psittacula krameri)*,[54] Hispaniolan Amazon parrots,[55] Grey parrots *(Psittacus erithacus)*,[56] cockatiels,[34,57] flamingos *(Phoeniconaias minor, P. ruber, Phoenicopterus ruber ruber)*,[58–60] African penguins,[37] red-tailed hawks, great horned owls,[61] and multiple vulture species *(Gyps africanus, Gyps coprotheres, Torgos tracheliotos*, and *Neophron percnopterus)*[62] have been published.

Differences in dosage, bioavailability, and elimination half-lives most likely contribute to the significant clinical differences seen with the use of this NSAID. For example, the elimination half-life of meloxicam in chickens and pigeons is three times as long as that of mallard ducks, ostriches, and turkeys.[48] There are even differences between breeds of chickens. Jing Hong layers can be dosed at 1 mg/kg every 16.9 hours.[63] Recently, it was found that white leghorn chickens have a shorter terminal half-life of meloxicam compared to Bantam Cochin and Wyandotte breeds including a shorter residue time in eggs.[40,41] Hence, it has been recommended to reduce the dosing frequency of meloxicam of 1 mg/kg orally to once daily in Bantam Cochin and Wyandotte chickens, compared with the twice daily dosing frequency in leghorn chickens. The authors of a study evaluating the PK of 1 mg/kg PO meloxicam in Pekin ducks were surprised as to how different PK values were between Pekin and mallard ducks, finding that the $t_{1/2el}$ of meloxicam in Pekin ducks was 20 times longer than in mallard ducks.[48,50] Following 1 mg/kg PO administration of meloxicam, the elimination half-life and bioavailability of meloxicam in Hispaniolan Amazon parrots were 15.8 ± 8.6 hours and 49% to 75%, respectively.[55] However, in Grey parrots following 1 mg/kg PO meloxicam, the elimination half-life was twice as long as that of the Hispaniolan Amazon parrots with significantly lower oral bioavailability (38.1% ± 3.6%).[56] In cockatiels following 1 mg/kg PO meloxicam of the commercial formulation, it was found to have only 11% oral bioavailability and an elimination half-life of less than 1 hour.[34] The highly variable bioavailability in avian species[55,56,61] is quite different than that in mammals, in which it usually exceeds 85%.[34] A possible explanation could be variability in proventricular pH among species.[56] In a multiple-dose PK study performed in Grey parrots, three different dosages of meloxicam (1 mg/kg IM q24h, 1 mg/kg PO q24h, and 1.6 mg/kg PO q24h) were evaluated. For all routes and dosages, plasma concentrations were maintained at values greater than the effective analgesic concentrations described for other avian species. In addition, evidence of moderate drug accumulation was found with both IM and PO routes of administration.[38] The more PK studies of meloxicam in different avian species, the more it shows the importance of evaluating PKs for each target species.

TABLE 35.2

NSAIDs Evaluated in Avian Species by Pharmacokinetic, Pharmacodynamic Studies, Toxicologic, or Clinical Studies

Drug	Dosage (mg/kg)	Route	Frequency (q/h)	Species	Comments	Type of Study	Reference Number
Carprofen	5	IV, IM, PO	Single dose	White-backed vulture *(Gyps africanus)*		PK, PD	33
Celecoxib	10	IV, PO	Single dose	Cockatiel *(Nymphicus hollandicus)*		PK	34
Mavacoxib	4	IV, PO	Single dose	Cockatiel *(Nymphicus hollandicus)*	Very long half-life	PK	34
	6	PO	Single dose	Caribbean flamingo *(Phoenicopterus ruber)*	Very high interindividual variability	PK	35
Meloxicam	0.2	PO	Single dose	Brown pelican *(Pelecanus occidentalis)*	Longest half-life of any avian species to date	PK	36
	0.5, 1	IM and PO, respectively	Single dose	African penguin *(Spheniscus demersus)*	0.5 mg/kg IM and 1 mg/kg PO results in concentrations above the target for approximately 24 and 60 hours, respectively	PK	37
	1	IM, PO	Multiple dose, q24h for 12 and 7 days, respectively	Grey parrot *(Psittacus erithacus)*	Moderate bioaccumulation following IM administration	PK	38
	1	IV, PO	Single dose	Cockatiel *(Nymphicus hollandicus)*	Very low (11%) oral bioavailability and shorter half-life than other psittacines	PK	34
	1	PO	Multiple dose q12h for 5 days	White Leghorn chicken *(Gallus gallus domesticus)*	Twice daily dosing needed; 2-week withdrawal time should be adequate to avoid drug residues in eggs	PD	39
	1	PO	Single dose	Wyandotte chicken; Bantam Cochin chicken *(Gallus gallus domesticus)*	Once daily dosing only	PK	40, 41
Sustained-release meloxicam	3	SC	Single dose	Hispaniolan Amazon parrot *(Amazona ventralis)*	Very high interindividual variability. Remained above target plasma concentrations for 12–96 hours	PK	42
	3	SC	Single dose	American flamingo *(Phoenicopterus ruber)*	Remained above target plasma concentrations for 0.5–4 hours	PK	43
Piroxicam	0.5, 1	PO	Single dose	Brolga crane *(Antigone rubicunda)*		PK	44
	1–2	IM	Multiple dose 1 q12h for 4 days	Chicken *(Gallus gallus domesticus)*		PD	45
Robenacoxib	4	PO	Single dose	Domestic goose *(Anser anser domesticus)*	Very short elimination half-life; not recommended for use in this species	PK	46

IV, Intravenous; *PD,* pharmacodynamic; *PK,* pharmacokinetic; *PO,* orally; *q/h,* every hour.

In chickens, dosing of meloxicam 1 mg/kg PO q12h for 5 days resulted in meloxicam residues in eggs in the yolks and whites after 8 and 3 days, respectively.[39] Similarly, a web-based interactive physiologically based pharmacokinetic (iPBPK) model for meloxicam laying hens determined that 22 and 4 days were necessary to clear meloxicam from yolks and whites, respectively.[64] Meloxicam was also identified in 2/3 unfertilized eggs of captive bearded vultures *(Gypaetus barbatus)* after breeding failure, but its implication is unclear.[65] A very recent study evaluating the PK of meloxicam in ovarian follicles and eggs showed much higher concentrations of meloxicam in the ovarian follicles than in the yolk and whites. On the basis of these results, a 2-week withdrawal time should be adequate to avoid drug residues in eggs meant for consumption.[39,66]

A sustained-release formulation of meloxicam (Meloxicam SR; Wildlife Pharmaceuticals, Windsor, CO, USA) has been evaluated in Hispaniolan Amazon parrots[42] and American flamingos *(P. ruber)*.[43] In Hispaniolan Amazon parrots, this formulation results in a rapid rise to peak concentrations and maintains above target plasma concentrations for 12 to 96 hours after a single dose of 3 mg/kg SC, likely due to interindividual variability. Because of these highly variable results, the use of this formulation in this species cannot be recommended until further PK, safety, and pharmacogenomic evaluations are performed to establish accurate dosing recommendations and to understand the high PK variability. In American flamingos, the sustained-release meloxicam formulation also had a rapid rise to peak plasma concentration, but it remained above target plasma concentrations for only 0.5 to 4 hours after a single subcutaneous dose of 3 mg/kg. Based on these results, the formulation would not extend the interval between treatments as compared to the regular formulation of meloxicam in this species.

Since higher doses of meloxicam can induce COX-1 enzyme activity, this drug has the potential to induce the adverse effects of gastrointestinal, renal, and cardiac toxicity seen in mammals, but reports of intoxication at doses used in avian practice are minimal. In a number of studies evaluating the adverse effects of meloxicam in budgerigars *(Melopsittacus undulatus)*; 0.1 mg/kg IM),[67] Grey parrots (0.5 mg/kg IM),[68] zebra finches (1–2 mg/kg PO),[53] domestic pigeons (2 mg/kg IM),[69] Japanese quail *(Coturnix japonica*; 2 mg/kg IM),[70] American kestrels (2–20 mg/kg PO),[71] and Hispaniolan Amazon parrots (1.6 mg/kg PO),[72] no significant renal, gastrointestinal, or hemostatic adverse effects were reported at the dosages evaluated. In contrast, 2/9 American kestrels had gastric ulceration after a dose of 20 mg/kg.[71] Plasma and urine *N*-acetyl-β-D-glucosaminidase activities were significantly increased in Grey parrots at the end of meloxicam treatment at 1.6 mg/kg PO every 24 hours for 7 days.[38]

Elimination half-lives of meloxicam are highly variable with some species such as the cockatiel being quite rapid,[34] which likely explains the lack of adverse effects seen in many avian species with this drug. However, the elimination half-lives in Grey parrots and African penguins are comparably very much longer, at 31.4 ± 4.6 and 25.8 hours, respectively, for oral dosing.[37,56] Anecdotal reports of visceral gout and gastrointestinal ulceration in brown pelicans using dosages commonly used in other species prompted a PK study using a single dose of 0.2 mg/kg PO, where the elimination half-life was 36 hours, the highest reported to date in any species heightening suspicion that adverse effects including renal toxicity may be attributed to this.[36]

The renal effects of NSAIDs have been of particular concern in the Asian vultures *(Gyps bengalensis*, *G. indicus*, and *G. tenuirostris)*.[73,74] These species are more sensitive to the adverse renal effects of several NSAIDs, with the exception of meloxicam where the elimination half-life is <1 hour.[75] However, recently, meloxicam was detected in eggs of captive bearded vultures.[65] Despite meloxicam's large therapeutic range and relative safety compared with other NSAIDs, these new data demonstrate that dose extrapolation is not a suitable method for meloxicam dose determination in avian species. Species-specific PK, efficacy, and toxicity studies are necessary to determine the appropriate dosage.

Celecoxib, Mavacoxib, and Robenacoxib

Celecoxib, mavacoxib, and robenacoxib are COX-2 selective as opposed to meloxicam which is COX-2 preferential, inhibiting both COX-1 and COX-2. Celecoxib in humans is used for acute pain, osteoarthritis, rheumatoid arthritis, and spondylitis. Its use in veterinary medicine is off-label in the United States and many other countries; it is not approved for use in any veterinary species. Nevertheless, celecoxib has been used for years in dosages of 10 mg/kg PO every 24 hours to reduce inflammation associated with avian ganglioneuritis.[76] A recent study in cockatiels using this dose showed no alterations in clinical presentation, viral shedding, gross lesions, histopathology, or viral distribution following experimental inoculation with parrot bornavirus 2 (PaBV-2) (see Chapter 14).[77] However, birds were infected with a large viral load, which induced severe disease, thereby making it difficult to determine whether the drug has any efficacy against the natural inflammation induced by this disease. Anecdotally, suggestions have been made to use even higher doses of 30 to 40 mg/kg divided PO every 12 hours, then long term 15 to 20 mg/kg PO every 12 hours for gastrointestinal signs, and up to 60 to 80 mg/kg divided PO every 12 hours for CNS involvement with ABV-associated ganglioneuritis.[78]

Mavacoxib administered orally has a long mean half-life of 15.5 days in dogs, allowing for monthly dosing after two loading doses administered apart.[79] Plasma concentrations of 0.4 μg/mL mavacoxib were associated with efficacy in reducing osteoarthritis in dogs.[80]

The single-dose PKs and oral bioavailability of celecoxib 10 mg/kg PO and mavacoxib 4 mg/kg PO were evaluated in the cockatiel.[34] Mavacoxib was completely absorbed (bioavailability = 111%), but celecoxib only had a mean oral bioavailability of 56%.[34] Bioavailability of the coxibs is often directly associated with feeding status; the presence of food has been shown to increase absorption of both of these NSAIDs.[81] The cockatiels in this study were fed ad libitum. A further study in cockatiels with ad libitum feeding and additional gavage feedings during the study showed that changes in body temperature, mentation, and posture were less pronounced with a single dose of mavacoxib 4 mg/kg PO compared to celecoxib 10 mg/kg at the tested dosages.[57] This is likely associated with the higher oral bioavailability of mavacoxib in cockatiels compared to celecoxib and meloxicam. Plasma concentrations of 0.4 μg/mL mavacoxib, found to be effective in osteoarthritic dogs,[82] were maintained for 1000 hours in the cockatiels in the aforementioned

studies,[34,57] whereas Caribbean flamingos *(Phoenicopterus ruber ruber)* maintained plasma concentrations of >0.4 μg/mL for 120 hours after a single dose of 6 mg/kg[35] Elimination half-lives in cockatiels (135 hours)[34] and Caribbean flamingos (74.5 hours)[35] were not as long as in dogs but are still much longer than most NSAID elimination half-lives in birds, therefore warranting additional studies to determine dosing frequencies, efficacy, and safety of these drugs in birds.[35]

Robenacoxib is also a long-acting NSAID that has been used anecdotally IM at 2 to 10 mg/kg once weekly, then monthly[83] in birds with clinical avian ganglioneuritis[84] and West Nile virus–induced CNS disease (B. Speer, personal communication, March 2, 2021). Robenacoxib is available as tablets and in an injectable formulation currently in the United States. There is only a single PK study of robenacoxib in birds to date. Based on the results from the PK study in domestic geese *(Anser anser domesticus)* at 4 mg/kg PO, it is suggested not to use this drug in this species due to a very short elimination half-life.[46]

Carprofen

Carprofen is considered a weak COX inhibitor at therapeutic doses, which may explain its apparent wide margin of safety. However, despite its weak COX inhibition, carprofen does exhibit good antiinflammatory activity, which may in part be achieved through other pathways. Even though it is believed that carprofen ingested by *Gyps* vultures does not pose a toxic risk like diclofenac, there are still concerns about its safety in these birds. In a recent PK and toxicokinetic study in white-backed vultures *(G. africanus)*, 5 mg/kg was administered IV, IM, and PO for the PK study, and a subset of birds were administered the dosage found at injection sites in cattle (64 mg/kg) for the toxicology study. Carprofen was well tolerated at 5 mg/kg via all routes of administration, but oral bioavailability was only 42%.[33] One vulture died in the toxicology study with elevations of uric acid and potassium identified, similar to the toxicity found with diclofenac and ketoprofen in *Gyps* vultures. The authors suggest that carprofen should not be used in domesticated ungulates in areas where carcasses are accessible or provided to vultures at supplementary feeding sites.[33]

Ketoprofen and Piroxicam

Ketoprofen is a potent nonselective COX-1 inhibitor that has been used extensively in small animal medicine, in particular because of its excellent oral availability. Very little new data exist on the use of this COX-1 preferential NSAID. Ketoprofen and meloxicam were detected in the eggs of captive-reared bearded vultures.[65] Two studies evaluated the effects of an extraordinarily high single-dose (50 mg/kg IM) ketoprofen as a premedication to ketamine and midazolam in pigeons and found no significant differences in clinical findings, biochemistry analytes, or histopathology between the groups.[85,86] Regardless of their findings, the extremely high dosing of this drug should be reviewed in multidose studies with the utmost caution before considering it for use.

Piroxicam is a nonselective NSAID used for its antiinflammatory properties and its value as a chemopreventative and antitumor agent. It has a much higher potency against COX-1 than COX-2. Similar to ketoprofen, little new studies have been published on its use in birds. In chickens, intramuscular administration of 1 to 2 mg/kg of piroxicam twice daily for 4 days failed to result in gastrointestinal lesions consistent with toxicity.[45] Piroxicam has been used in cranes for more than 20 years to treat a variety of conditions including trauma, inflammation, and postoperative pain with minimal observed side effects.[44] A PK study in brolga cranes *(Grus rubicunda)* evaluated two single doses of 0.5 and 1 mg/kg PO, finding that daily administration might be appropriate for this species based on plasma concentrations.[44]

Grapiprant

Grapiprant is a PGE_2 receptor antagonist NSAID approved for use in dogs in the United States. Grapiprant binds to the EP4 receptor, blocking the sensitization of sensory neurons and stimulation of inflammation, mediated by the PGE_2 receptor. It does not rely on the inhibition of COX enzymes and preserves functions associated with COX enzyme pathways. Oral administration of grapiprant at 30 mg/kg to fasted red-tailed hawks achieved plasma concentrations approximately 100 times the canine therapeutic concentrations, but it is still unknown whether these concentrations are analgesic or could be toxic in hawks.[87] While adverse effects were not identified in the main study, two birds did regurgitate at 45 mg/kg in the pilot study.[87] Grapiprant administered at the same dose to nonfasted red-tailed hawks had delayed and decreased absorption with only 50% of birds reaching the targeted canine concentrations for a much shorter time, significantly lower compared to the fasted PK study (M.G. Hawkins, personal communication, December 9, 2024). Further studies to evaluate the PKs of grapiprant at either higher doses or increased frequency with food and multidose administration are underway.

LOCAL ANESTHETICS

Local anesthetics reversibly bind to Na^+ channels and block impulse conduction, thereby making them favorable for use during surgical procedures to prevent pain conduction and sensitization of the CNS. The duration of action depends on the molecular properties and lipid solubility of the drug. Following local application, the anesthetic will be quickly absorbed into the blood stream, increasing the potential for toxic reactions. Typically, local anesthetics are administered via local line or splash blocks (Table 35.3).

Lidocaine

Lidocaine is an amide local anesthetic that decreases neuronal excitability by blocking voltage-dependent sodium channels, thus preventing the conduction of action potentials that mediate nociception. Empirically, dosages of 2 to 3 mg/kg have been recommended, although previous studies in chickens and mallard ducks showed no toxic effects following intravenous administration of 6 or 15 mg/kg for brachial plexus block, respectively. In more recent studies, the effects of lidocaine were evaluated in 2-kg broiler chickens during spinal anesthesia (also called subarachnoid or intrathecal block). During this study, lidocaine was administered in doses of 0.5, 1, and 2 mg/kg (2% preservative-free lidocaine, made up to 0.2 mL in saline) between the intervertebral space between synsacrum and first free coccygeal vertebra (synsacrococcygeal space).[88] Analgesia was tested by pinching the skin of the pericloacal area with thumb forceps to determine the onset and duration of analgesia. The interval

TABLE 35.3

Local Anesthetics Evaluated in Avian Species by Either Pharmacokinetic or Pharmacodynamic Studies

Drug	Dosage (mg/kg)	Route	Frequency (q/h)	Species	Comments	Type of Study	Reference Number
Lidocaine	0.5, 1, 2	IT	Single administration	Chicken *(Gallus gallus domesticus)*	Onset of pericloacal anesthesia 1–2 minutes, and duration of effect 4.5, 11.3, and 21.3 minutes, respectively	PD	88
Bupivacaine	1.33, 1.96	IV	Single administration	Chicken *(Gallus gallus domesticus)*	1.96 mg/kg IV bupivacaine associated with a 50% probability of a clinically relevant change in MAP and HR, whereas 1.33 mg/kg IV bupivacaine is associated with a 1% probability of causing the same effect	PD	89
Bupivacaine	0.1, 0.25, 0.5	IT	Single administration	Chicken *(Gallus gallus domesticus)*	Onset of pericloacal anesthesia 9, 4.33, and 3.33 minutes, respectively and duration of effect was 11.18 and 54 minutes, respectively	PD	90

HR, Heart rate; *IT,* intrathecal; *IV,* intravenous; *MAP,* mean arterial pressure; *PD,* pharmacodynamic.

to onset of analgesia was not significantly different for all doses of lidocaine (1.5, 2, and 1.3 minutes for 0.5, 1, and 2 mg/kg). Duration of analgesia was significantly longer following injection with 2 mg/kg than 0.5 or 1 mg/kg lidocaine (21.3, 4.5, and 11.3 minutes). In a follow-up study, using the same spinal anesthesia technique in broiler chickens of the same size, spinal anesthesia was evaluated following administration of 2 mg/kg lidocaine.[90] The onset to effect and the duration of action were calculated based on the pinch test at 1.3 and 17.8 minutes, respectively. Considering the results of the studies evaluating spinal anesthesia in chickens and the disorders that affect the cloaca, this technique would be indicated to provide local anesthesia in this species. However, caution should be exercised in other species, especially the smaller ones in which this technique has yet to be evaluated.

Bupivacaine

Bupivacaine is an amide local anesthetic that decreases neuronal excitability by blocking voltage-dependent sodium channels, thus preventing the conduction of action potentials that mediate nociception. Because it is a long-acting local anesthetic, bupivacaine can be useful for perioperative purposes. In recent studies, the cardiovascular tolerance of bupivacaine was evaluated in chickens anesthetized with isoflurane.[89] In this study, it was determined that 1.96 mg/kg IV bupivacaine is associated with a 50% probability of a clinically relevant change in MAP and HR, whereas 1.33 mg/kg IV bupivacaine is associated with a 1% probability of causing the same effect. The effects of bupivacaine have also been evaluated in spinal anesthesia following administration of 0.1, 0.25, and 0.5 mg/kg (0.5% bupivacaine hydrochloride, made up 0.2 mL in saline) in 2-kg broiler chickens.[90] The onset to effect calculated based on the pinch test was 9, 4.33, and 3.33 minutes for the 0.1, 0.25, and 0.5 mg/kg, respectively. The duration of action of the 0.5 mg/kg dose (54 minutes) was significantly longer than that of any other groups (11 and 18 minutes for the 0.1 and 0.25 mg/kg, respectively). Based on these results and comparing them with those of lidocaine use, bupivacaine would be preferred to lidocaine when using this technique in chickens.

OTHER DRUGS (TABLE 35.4)

Gabapentin

Gabapentin is an analog of the neurotransmitter gamma-aminobutyric acid (GABA) that binds to voltage-gated calcium channels acting presynaptically to decrease the release of excitatory neurotransmitters, thereby being helpful to treat neuropathic pain. Until recently, few studies had evaluated its use in avian species, but in the last years, several PK studies have been published. In these studies, a compounded formulation was used to avoid potential risks associated with xylitol in the commercially available oral suspension of gabapentin for humans. In great horned owls following administration of an oral dose of 11 mg/kg PO, the C_{max}, T_{max} (time to maximum concentration), and elimination half-life were 6.17 μg/mL, 51.43 minutes, and 4.4 hours, respectively. In this study, plasma gabapentin concentrations were maintained above 2 μg/mL for 9.6 hours, suggesting that gabapentin administered orally every 8 hours may be appropriate in great horned owls.[91] In Hispaniolan Amazon parrots, oral bioavailability was estimated to be 80% to 89%.[92] Two and three birds, respectively, received 10 and 30 mg/kg PO, with a C_{max}, T_{max}, and elimination half-life of 7.29 and 16.08 μg/dL, 2 and 2.5 hours, and 5.41 and 3.74, respectively. Mild transient sedation was noted after intravenous injection of gabapentin that resolved after 20 to 30 minutes. From that study the authors concluded that 15 mg/kg every 8 hours would be a starting point for oral dosing in Hispaniolan Amazon parrots based on effective plasma concentrations reported for human patients. However, additional studies were considered necessary

TABLE 35.4

Other Drugs Evaluated in Avian Species by Either Pharmacokinetic or Pharmacodynamic Studies

Drug	Dosage (mg/kg)	Route	Frequency (q/h)	Species	Comments	Type of Study	Reference Number
Gabapentin	11	PO	Single dose	Great horned owl *(Bubo virginianus)*	Plasma concentrations maintained above 2 μg/mL for 9.6 hours. Suggests q8h administration	PK	91
	10, 30	PO	Single dose	Hispaniolan Amazon parrot *(Amazona ventralis)*	Elimination half-life was 5.41 and 3.74 hours, respectively. Suggests 15 mg/kg q8h	PK	92
	15, 25	PO	Single dose	Caribbean flamingo *(Phoenicopterus ruber ruber)*	Elimination half-life of 3.39 and 4.46 hours, respectively. Suggests administration of 25 mg/kg q12h, but some individuals could need higher doses	PK	93
Amantadine	10	PO	Single dose	Orange-winged Amazon parrot *(Amazona amazonica)*	Elimination half-life of 23.2 hours. Reaches plasma concentrations associated with toxicity in humans	PK	94
	5	PO	Multiple dose, q24h for 7 days	Orange-winged Amazon parrot *(A. amazonica)*	Elimination half-life of 21.5 hours. Suggests q24h administration	PK	94
Cannabidiol	60, 120	PO	Single dose	Hispaniolan Amazon parrot *(A. ventralis)*	Very high interindividual variability. Elimination half-life of 1.28 hours at the higher dose evaluated	PK	95
CBD, CBDA, THC, THCA, CBG, CBGA, and cannabichromene	Dosing based on CBD/CBDA 30/32.5 mg/kg	PO	Single and multiple dose q12h for 7 days	Orange-winged Amazon parrot *(Amazona amazonica)*	In the multiple-dose study, elimination half-life of 8.6 and 6.3 hours for CBD and CBDA, respectively. Suggests q12h administration		96

CBD, Cannabidiol; *CBDA,* cannabidiolic acid; *CBG,* cannabigerol; *CBGA,* cannabigerolic acid; *IV,* intravenous; *PD,* pharmacodynamic; *PK,* pharmacokinetic; *PO,* orally; *THC,* Δ-tetrahydrocannabinol 9; *THCA,* tetrahydrocannabinolic acid.

to demonstrate that these suggested dosages would be effective. In a more recent study in Caribbean flamingoes, following administration of a 15 and 25 mg/kg dose, the C_{max}, T_{max}, and elimination half-life were 13.23 and 24.48 μg/mL, 0.50 and 0.56 hours, and 3.39 and 4.46 hours, respectively.[93] Based on the results of this study, gabapentin dosed at 25 mg/kg orally in most Caribbean flamingos was considered likely to maintain plasma concentrations above the therapeutic range established for humans for approximately 12 hours; however, dosages as high as 37 mg/kg orally every 12 hours may be needed in some individuals.

Amantadine

Amantadine is an antiviral medication that increases dopamine in the CNS and weakly inhibits the NMDA receptors that contribute to chronic pain.[97] Amantadine is synergistic with NSAIDs, opioids, and gabapentin/pregabalin. In humans, amantadine has been shown to decrease opioid use in postoperative patients, decrease pain in postherpetic neuralgia, and aid in neuropathic analgesia.[98,99] Amantadine orally administered to dogs at 3 to 5 mg/kg every 24 hours in conjunction with meloxicam showed significant improvement in the management of osteoarthritis pain refractory to meloxicam alone in dogs.[100] In orange-winged Amazon parrots, the single- and multiple-dose PK profiles were evaluated following 10 mg/kg PO once and 5 mg/kg PO q24h for 7 days.[94] The heart rate, respiratory rate, behavior, and urofeces were monitored. Mean ± SD maximum plasma concentration, time to maximum plasma concentration, half-life, and area under the curve from the last dose to infinity were 1174 ± 186 ng/mL, 3.75 hours, 23.2 hours, and 38.6 hours/μg/mL, respectively, for the single-dose study and 1185 ± 270 ng/mL, 3.0 hours, 21.5 hours, and 26.3 hours/μg/mL, respectively, for the multiple-dose study at steady-state. No obvious adverse effects were observed. Based on these results, once daily administration of amantadine at 5 mg/kg orally in orange-winged Amazon parrots is able to maintain above target plasma concentration with weak accumulation. For long-term administration, especially for other species, it is recommended to check plasma concentrations over time. Further studies evaluating the safety and efficacy of amantadine in orange-winged Amazon parrots and other avian species are warranted. It is also important to remember that the use of amantadine in chickens, ducks, and turkeys is prohibited in the United States by the Federal Drug Administration.

Cannabidiol and Other Cannabinoids

Cannabidiol (CBD) oil, derived from hemp, is the primary nonpsychoactive cannabinoid derived from the cannabis plant *(Cannabis sativa)* that can interact with the endocannabinoid system. Cannabinoids have been evaluated in other species for their multiple effects including pain relief and antiinflammatory properties, making them an attractive therapeutic option in birds with chronic pain. The PKs of a CBD oral formulation (50 mg/mL; Canna Companion, Mill Creek, WA, USA) have been evaluated in Hispaniolan Amazon parrots.[95] The C_{max} were 213 and 562 ng/mL at 0.5 and 4 hours with an elimination half-life elimination of 1.28 hours for the 60 and 120 mg/kg doses evaluated. The highly variable results and short half-life of the drug in Hispaniolan Amazon parrots, even at high doses, suggest that this formulation would have to be administered at least three to four times daily. More recently, a commercial hemp *(Cannabis sativa)* extract (100 mg/mL; Ellevet, South Portland, ME, USA) that contained CBD, cannabidiolic acid (CBDA), Δ-tetrahydrocannabinol 9 (THC), tetrahydrocannabinolic acid (THCA), cannabigerol (CBG), cannabigerolic acid (CBGA), and cannabichromene (45.2, 49, 1.86, 0.56, 0.69. 1.21, and 1.53 mg/mL, respectively) was evaluated in orange-winged Amazon parrots in a single- and multiple-dose PK and safety study at a dose of 30/32.5 mg/kg PO of CBD/CBDA.[96] The half-life in the multiple-dose study was 8.6 and 6.3 hours for CBD and CBDA, respectively. Interesting differences in the metabolism of some of the cannabinoids when compared to other species studied were found. The results from that study were encouraging for a q12h administration, based on plasma concentrations and limited safety data in the 7-day multiple-dose study. Evaluation of other formulations and in other species is recommended to further characterize the analgesic potential of CBD, CBDA, and other cannabinoids in birds.

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CHAPTER 36

Wound Management

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INTRODUCTION

Avian patients are frequently presented with skin wounds and injury to the underlying tissue. These injuries may be the most common problem affecting birds treated by veterinarians. Wounds affecting the skin and/or underlying tissue may present or be classified as abrasions, lacerations, crushing trauma, self-trauma, infections, thermal injuries, or limb amputations. Initial therapeutic efforts should address blood loss, shock, and pain in addition to rapid treatment of the affected area(s). These treatments increase probability of patient stabilization, timely injury care, and appropriate wound management, hence increasing the chance for a complete recovery based on the initial prognosis.

An important component to obtain the most expeditious resolution for dermal wounds in birds is understanding the pathophysiology of wound healing and determining which treatment options are best for each case. New treatment options and products are routinely being introduced, often providing improved healing modalities but making a clinician's decision for treatment difficult. Knowledge of wound healing principles will help the veterinarian determine an appropriate treatment regimen for the patient. The information presented here should aid clinicians in understanding these principles and common products used when treating avian skin wounds to have the greatest chance for successful resolution of these cases.

AVIAN SKIN

The anatomical structure of avian skin is like that of mammals, in that there is an outer epidermis and inner dermis; however, a bird's skin is generally much thinner and more delicate. The epidermis varies in thickness throughout the body and is avascular. It is routinely three to five cells thick, but in certain areas, such as the legs and face, will be much thicker.[1] The epidermis is composed of two layers: the stratum germinativum (also referred to as stratum basale) and stratum corneum.[2] The stratum germinativum is the site of cellular growth where keratinocytes change in form and function then migrate upwards to the outer cornified layer, or stratum corneum.[1] The dermis contains connective tissue with blood vessels, nerves, tendons, muscles, and feather follicles. It consists of two layers, the superficial and deep. The deep dermal layer is further divided into compact and loose layers, with the loose layer sitting at the base of the dermis, and attaches to the subdermis where it is infiltrated with fat and apterial muscles.[2]

STAGES OF HEALING

Wound healing is an organized process that generally has three to four phases, including the hemostasis (coagulation), inflammatory and debridement, proliferation, and maturation (remodeling) phases (Figure 36.1).[3] These phases often overlap, creating variable distinction between the categories. While hemostasis is often separate, it can also be considered part of the inflammatory phase. These stages are similar between mammals and birds. Hemostasis occurs immediately after the injury, causing vasoconstriction and activation of the clotting cascade in order to form a fibrin clot. This is followed by vasodilation within 30 minutes. The release of proinflammatory cytokines causes recruitment of neutrophils, endothelial cells, and fibroblasts to the wound. After 24 to 48 hours, the inflammatory phase causes increased capillary permeability to allow for neutrophils, macrophages, and lymphocytes to infiltrate the wound. This can last for up to 2 weeks and often overlaps with the debridement phase. Once debridement is accomplished, the proliferative phase begins, usually 3 to 4 days following the original injury. A granulation bed develops through the combination of fibroblasts, new blood vessels, and fibrous tissue. Keratinocytes proliferate and mature to form a new epidermal layer. The final phase, maturation (remodeling), typically occurs 1 week after the original injury and can continue for weeks to months. The newly formed collagen fibers remodel and strengthen the newly formed tissue.[3]

Several factors dictate wound healing, including level of bacterial contamination, extent of tissue damage or necrosis, and patient's healing ability.[4] Traumatic wounds that require initial and/or repeated cleaning and debridement take longer to heal compared to aseptic, surgically repaired wounds. In avian patients, wounds of distal extremities with a reduced vascular supply and wounds located over joints, the axilla, and the patagium tend to heal more slowly. Other systemic factors can also impair healing and should be addressed. As wounds exude large amounts of protein daily, if a patient is already hypoproteinemic or has inadequate nutrition, they may break down protein for energy and further deplete their body's ability to heal. Additionally, patients recovering from soft tissue

Phase	Time frame	Cells involved	Function of activity	Cellular and bio-physiologic events
Hemostasis (coagulation)	Immediate	Platelets	Clotting	Vascular constriction Platelet aggregation, degranulation, and fibrin formation (thrombus)
Inflammatory/ debridement	Days 1–4	Neutrophils Macrophages	Phagocytosis	Neutrophil infiltration Monocyte infiltration, and differentiation to macrophages Lymphocyte infiltration
Proliferation	Days 4–21	Macrophages Lymphocytes Angiocytes Neurocytes Fibroblasts	Fill defect Reestablish skin function Closure	Reepithelialization Angiogenesis Collagen synthesis
Maturation (remodeling)	Day 21–2 years	Fibrocysts	Develop tensile strength	Collagen remodeling Vascular maturation and regression

FIGURE 36.1 The stages of wound healing are similar between birds and mammals and are an organized process that involves three to four phases. (Modified from Stacey M: Why don't wounds heal? *Wounds Int* 7:16–21, 2016.)

wounds will have higher daily water intake requirements in order to provide moisture at the surface of the wound and help white blood cell migration to the injury site. It is important to address hypoproteinemia, poor nutrition, and dehydration in any avian patient presenting with injuries as these conditions may delay healing. Other conflicting factors include systemic or chronic disease, especially chronic anemia or cardiovascular disease. Patients suffering from these conditions may have compromised blood flow to or decreased oxygenation of the wound, which both can delay the healing process.

WOUND EVALUATION

Obtaining a thorough history when patients present with a soft tissue injury is critical for appropriate treatment of the wounds and should include the cause and period of time that has expired since the initial injury. This information will help dictate treatment, including decontamination and closure, if necessary, in addition to patient stabilization.

When a patient is presented with a skin wound that may have underlying soft tissue involvement, it is important to determine the overall condition of the avian patient. The bird should be stabilized prior to any prolonged and/or stressful treatment efforts, which may include direct treatment of the wounds. During initial stabilization, the wound should be examined for hemorrhage, which should be immediately addressed, in addition to providing therapeutics, including pain medications (see Chapters 34 and 35). Temporary dressings can be placed over the affected area until further treatment can be performed (Figure 36.2). Once the patient is able to withstand prolonged handling, the wound can be appropriately treated. Major considerations when evaluating wounds include wound type, vascular supply, contamination, possible infection, presence of exudate (type, volume, and consistency), condition of the surrounding/underlying tissues, dimensions (length, width, and depth), location, and, if known, cause of injury. Fractures and luxations can be a common result of traumatic injuries and should also be addressed (Figure 36.3). For more information on managing fractures, see Chapter 37. Aseptic technique should always be practiced when treating these wounds. To determine the extent of injury, removal via plucking or cutting of overlying feathers to produce 2 to 3 cm of exposed, healthy skin, surrounding the affected area should be performed. Pulling feathers from torn or lacerated skin should be done cautiously as it easily results in tearing of the



FIGURE 36.2 Temporary bandage placed over a wound on the dorsal aspect of the left carpal joint in a Quaker parrot *(Myiopsitta monachus)*.



FIGURE 36.3 Traumatic wound of the right elbow joint of a domestic cockatiel *(Nymphicus hollandicus)*. The right radius and ulna were also fractured.

skin, particularly in species and regions where it is particularly thin, thereby increasing the extent of the injury, especially in waterfowl. Cutting feathers with sharp scissors, in contrast, is generally less traumatic and stressful.

Therapeutic tools and agents used to stabilize critical patient are generally available in most hospitals and include catheters (22- to 26-gauge intravenous catheters, 1.5-inch 22-gauge spinal needles for intraosseous catheters), crystalloid and colloidal fluids, iron dextran (Watson Laboratories Inc., Corona, CA, USA), and a nutritional critical care formula (Emeraid; Lafeber Co., Cornell, IL, USA; see also Chapter 34 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 17, Critical Care). Analgesic compounds are a beneficial addition for most veterinary patients, and for the avian patient this has been no exception (see Chapter 35 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 20, Recognition, Assessment, and Management of Pain in Birds). Meloxicam (Metacam; Boehringer Ingelheim, St. Joseph, MO, USA), a nonsteroidal antiinflammatory, and butorphanol tartrate (Torbugesic; Zoetis, Parsippany, NJ, USA), a mixed agonist-antagonist, are two of the most commonly used analgesic compounds for avian patients. Meloxicam is available in both oral suspensions and a 5-mg/mL injectable form. For many hospitalized patients and common companion species, injectable 10 mg/mL butorphanol is the pain medication of choice due to its agonist activity at kappa opioid receptor, which is more prominent in the avian forebrain and midbrain.[5] Based on pharmacokinetic studies, frequent (every 2–3 hours) intramuscular or intravenous administration at 5 mg/kg is recommended. Oral administration was found to be an ineffective route, as <10% of the drug was available in Hispaniolan Amazon parrots *(Amazona ventralis)*.[6] Topical analgesia can also be beneficial when managing pain associated with wounds, especially when given prior to debridement. Lidocaine is a common local anesthetic that can be injected subcutaneously or applied topically. It comes in both a 5% jelly and ointment preparation, which can be applied directly to a wound for analgesia within 3 to 5 minutes of contact. Injectable lidocaine can also be administered subcutaneously surrounding a wound in a ring block fashion. The high acidity of lidocaine will often cause a burning or stinging sensation when administered in this route, so it is often recommended to buffer the solution with sodium bicarbonate to decrease the irritating side effects. Analgesic effects of lidocaine can last as long as 2 hours. Bupivacaine, another local anesthetic, can provide analgesic for up to 8 hours; however, its onset of action is much longer (5–10 minutes) compared to lidocaine. For more information on analgesia in birds, see Chapter 35 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 20, Recognition, Assessment, and Management of Pain in Birds.

Like mammals, avian patients may succumb to bacterial sepsis from wounds without antibiotic treatment, depending on the source of the wound. Common aerobic bacterial isolates in avian wounds include *Pasteurella multocida*, *Streptococcus* spp., *Staphylococcus* spp., *Moraxella* spp., and *Neisseria* spp. Common anaerobes include *Fusobacterium* spp., *Bacteroides* spp., *Porphyromonas* spp., and *Prevotella* spp. Antibiotic treatment choice should ideally be based on culture and sensitivity testing, but preemptive treatment considering these agents and the type of wound present is critical. More information on the use of antimicrobials in birds can be found in Section 3: Therapeutic Intervention. The antibacterial, antifungal, and antiparasitic drugs used to treat both systemic and topical wounds are like those found in most veterinary hospitals. Avian patients often tolerate oral fluid medications better than pill or tablet forms. Exceptions to this include medications that carry a bitter or poor taste, such as metronidazole hydrochloride compared to the more palatable metronidazole benzoate. If a veterinary hospital does not have common antibiotic agents formulated in an oral suspension form or if a clinician desires added flavoring to help with palatability, a compounding pharmacy, such as Kingswood Compounding Pharmacy in Australia, Diamondback Drugs or Roadrunner Pharmacy in the United States, or Pharmacy Lab in the UK, should be contacted. More information on compounding can be found in Chapter 5.

LAVAGE

Depending on the wound, cleansing and decontamination can be achieved with a moistened gauze sponge and/or rigorous lavage with sterile isotonic fluids (e.g., saline, Plasma-Lyte, lactated Ringer's solution). This not only removes debris from the tissue but also helps to rehydrate the exposed dermis and underlying tissue. It is important to note that heat support may be necessary while lavaging wounds to prevent hypothermia in avian patients, especially smaller species, which can be prone to rapid heat loss when wet.

In contaminated wounds, either 0.05% chlorhexidine or 0.05% povidone-iodine antiseptic solution may be used for

lavage. In a study assessing the healing times of induced open wounds in blue-fronted Amazon parrots *(Amazona aestiva)* with varying treatments applied every other day,[7] saline and chlorhexidine solution were found to have significantly shorter healing time compared to vitamin B_5 ointment, castor oil, and a control group. The chlorhexidine group also showed the highest neovascularization production and fibroplasia compared to the other study groups. Similar results were seen in an earlier study in the same species where application of chlorhexidine every other day showed more efficient wound healing compared to vitamin B_5 ointment, castor oil, saline, and laser therapy.[8] It is important to note that both studies used noncontaminated induced wounds. Similarly, a review of clinical trials assessing the effectiveness of povidone-iodine in the treatment of wounds in humans found that it does not prolong or interfere with healing.[9]

Advancements in products for wound management include the advent of Tricide (Molecular Therapeutics, LLC, Athens, GA, USA). This product is a chelating agent that acts as an antimicrobial potentiator and is used to flush the wound bed and control infection. Tricide creates holes in the bacterial cell wall, which allows the introduction of antibacterial drugs into the organism. Tricide can be prepared with normal saline or distilled water, has a shelf life based on the package instructions, and mixes well with multiple antibiotic and antifungal drugs. Solutions containing this product have been shown to be effective against multidrug-resistant bacteria cultured from human burn patients and from companion animals and help to reduce the concentration of antimicrobials necessary to achieve the minimum inhibitory concentration (MIC).[10] As it causes direct physical damage to the bacteria, there is decreased likelihood of resistance to the added antimicrobial agent. To date, however, no studies have assessed its efficacy in avian wounds. See Table 36.1 for a list of lavage products and their properties.

DEBRIDEMENT

To stimulate and encourage healing, removal of nonviable and necrotic tissue and/or foreign debris is the next step in wound treatment after cleansing has been performed. This can be accomplished through either surgical or mechanical means and should typically be done only during the inflammatory phase of wound healing.[3] Surgical debridement includes targeted removal of nonviable tissue and solidified exudative material with instrumentation (e.g., scissors, forceps). Ideally, the tissue debridement is performed under general anesthesia or sedation and local anesthesia, but this decision should be based on clinical assessment of the wound and the stability of the patient. Exposed tissue, including tendons, ligaments, blood vessels, and nerves, should be grossly examined to determine viability, but debrided as minimally as possible to maintain function (Figure 36.4). This process is often staged, debriding obvious nonviable tissue while leaving questionable tissue, and bandaging the area. This process is repeated daily until any and all tissues that have been determined to be avascular or obviously nonviable are removed.

Mechanical debridement is accomplished through gentle rubbing of a healing wound with a saline or antiseptic soaked gauze sponge or by placing a wet-to-dry or dry-to-dry bandage over the affected area. With this process, debridement



FIGURE 36.4 Surgical debridement of a leg wound exposes vital connective tissue, vessels, and nerves that supply the distal limb. Structures should be grossly examined to determine viability and debrided as minimally as possible at the onset of treatment.

TABLE 36.1

Lavage Agents Used to Treat Contaminated Wounds in Avian Patients

Product	Properties	Toxicity
0.05% povidone-iodine	Broad-spectrum antimicrobial, penetrates biofilms, anti-inflammatory, nonirritating.[11]	Low cytotoxicity, but may cause mild irritation if exposed to eyes.
0.05% chlorhexidine	Broad-spectrum antimicrobial, expedited healing time.	Associated with corneal damage and severe pain if exposed to eyes. Higher concentrations can cause tissue toxicity,[12] inhibition of granulation tissue,[13] and temporary decreased tensile strength.[14]
Isotonic fluid (saline, Plasma-Lyte, lactated Ringer's solution)	Rehydrates exposed dermis and underlying tissue. Little to no antimicrobial properties.	Tolerated well in most tissues.
Tricide	Antimicrobial potentiator that is mixed with saline or distilled water for topical application or bathing. May also be mixed with multiple antibiotics or antifungals and results in decreased development resistance.	Serious ocular irritation can occur. Proper protective equipment should be worn with use.

is nonselective and removes debris in addition to the superficial layer of the wound bed. Removal of dressings may be painful and typically requires changing multiple times daily for the first few days.[3] Other forms of debridement include enzymatic or autolytic processes, but these are rarely used. Commercial enzymatic debridement products often contain collagenase or papain-urea.[3] These products selectively reduce necrotic tissue and liquefy coagulum and bacterial biofilm.[15] Enzymatic ointments and gels are often expensive, have to be used judiciously due to possible damage to viable tissue, and are not recommended for large wounds or long-term management.[3] Collagenase-based preparations are the most common type of enzymatic debriding agent. Collagenase is water-soluble proteinase derived from bacteria such as *Clostridium histolyticum* and *Vibrio* spp. that has a specific activity against collagen in nonviable cells, meaning that it does not break down healthy tissue. In several canine and feline case studies with nonhealing wounds, granulation tissue was observed within 2 days of beginning treatment with the collagenase preparation Iruxol Mono (Smith & Nephew Ltd., London, UK), and all successfully healed. However, further research is required to compare the efficacy of this treatment with other wound management techniques as well as determine the efficacy and safety in avian patients.[16]

Maggot infestation (myiasis) can be a problem in wounds, especially during the summer months or in regions where it is commonly warm and humid.[17] Removal of the fly larvae is necessary to prevent further contamination and provide a healthy environment for wound healing. Maggots may be removed manually, but in large wounds the time to do this should be weighed against patient stability. Nitenpyram, the active ingredient of Capstar (Elanco Animal Health, Greenfield, IN, USA), is an effective treatment for myiasis. No pharmacokinetic studies have yet been performed on birds, but the authors have used it successfully in avian patients. It may be used topically or orally at 1 mg/kg and usually kills larvae within 1 to 2 days. Ivermectin at 0.2 to 0.4 mg/kg may also be used orally or subcutaneously, but no studies evaluating the efficacy of treating myiasis in birds are available at this time.

CLOSURE

Depending on the degree of wound contamination, patient status, and amount of soft tissue loss surrounding the wound, wounds may be treated with primary, secondary, or tertiary intention closure. Primary closure of the wound is the apposition of skin edges using suture or an adhesive (e.g., tissue glue) at or close to the time of presentation. There has been a general belief in an "8-hour golden period" where primary closure can be performed following injury.[18] Degree of contamination should be considered a larger factor to determine management of wounds, with delayed primary closure (tertiary intention) being preferred in soiled or heavily contaminated wounds, such as cat bites. This treatment has been found to reduce wound infection rates and hospital stays in human patients with contaminated wounds[19] and should be implemented in avian patients with similar history. Secondary intention closure allows the wound to heal through development of a granulation bed at the wound site. As previously mentioned, tertiary intention healing, or delayed primary intention closure, is when a wound is purposefully left open to allow for treatment and cleaning over an extended period (usually between 3 and 5 days) as well as to allow demarcation between viable and nonviable tissue, then closed using primary intention techniques (Figure 36.5). Closure with



FIGURE 36.5 Tertiary closure of a neck wound in a wild lesser scaup *(Aythya affinis)*. The patient was presented to the Wildlife Hospital of Louisiana with a traumatic neck wound of unknown origin that had developed a large eschar (A). After several days of topical treatment, the wound was surgically debrided, cleaned, and closed under general anesthesia (B).

absorbable suture (2-0 to 5-0) with an atraumatic tapered needle can be used to close muscle and subcutaneous tissue.[17] In the case of crop wounds, tertiary wound closure can be beneficial in reducing the risk of infection at the wound bed and stricture as the area heals. It is important to provide systemic support, such as gavage feeding through a pharyngostomy feeding tube.

SKIN GRAFTS AND FLAPS

In larger birds, such as raptors, the use of skin flaps or grafts can be considered to close large wounds. Advancement, or pedicle, flaps involve sliding or rotating skin over an adjacent defect (Figures 36.6 and 36.7). The area is elevated and moved, keeping a portion attached to the donor site in order to



FIGURE 36.6 Serial images of cervical advancement flap procedure in a 46-year-old green-winged macaw *(Ara chloropterus)* with a chronic, nonhealing 9-month-old wound at the occipital region. (A) Anesthetized bird in ventral recumbency and the firmly adhered dry eschar is visible. Blood pressure cuff is visible over the right humerus as a part of anesthetic monitoring setup. (B) Using iris scissors and jeweler's forceps, the eschar is progressively elevated and removed. (C) Following removal of the eschar, the exposed caudal portion of the skull is visible, covered by the remaining periosteum; granulation tissue is present peripherally. (D) Using jeweler's forceps to minimize iatrogenic damage to the skin, construction of the advancement flap is commenced, and Metzenbaum scissors are used for progressive blunt-blunt dissection. Linear incisions are advanced caudally down the dorsal cervical region, in slightly diverging angles, bilaterally. (E) A small rent was inadvertently created in the left cervicocephalic air sac and sutured closed prior to continuing with dissection. If possible, care should be taken to minimize opening this air sac and leading to leakage and subcutaneous emphysema as a postoperative complication. Good intraoperative magnification, appropriate surgical instruments, and delicate tissue handling skills are helpful. (F) The advancement flap is initially tacked in its designated position. It is important to assure that there is minimal tissue tension to minimize risks of dehiscence, particularly at the cranial-most attachment sites.

Continued

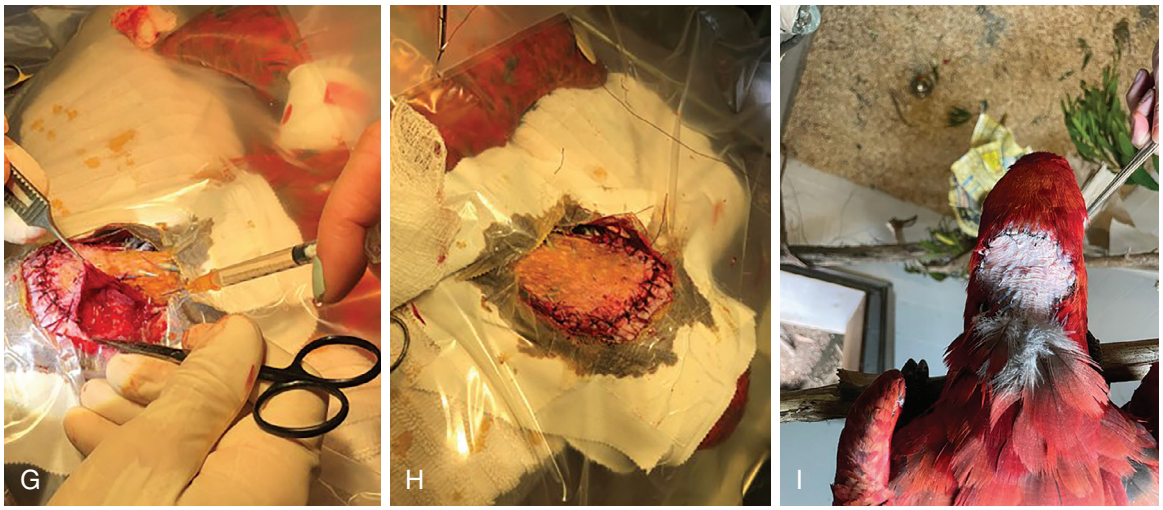


FIGURE 36.6, cont'd (G) A series of simple interrupted sutures are visible, as intraoperative bupivacaine is applied during closure. (H) The completed, closed wound. (I) The healed wound 3 weeks following surgery. (Images courtesy Brian Speer.)



FIGURE 36.7 Images of preoperative and two postoperative repairs of a large irregular occipital wound in a domestic chicken *(Gallus gallus domesticus)*. (A) The anesthetized patient is positioned in ventral recumbency. A caudal aspect of the comb is visibly missing, because of the predator attack that caused the injury. (B) Left lateral view of the patient immediately postoperatively. Care was needed to keep the ear openings patent and effectively restored and to not provide excessive dorsal tension that would cause elevation of the eyelids. (C) Right lateral view of the same patient, immediately postoperatively. (Images courtesy Brian Speer.)

maintain blood supply to the subdermal plexus. With this intact intrinsic blood supply, properly designed flaps have a greater chance of viability compared to grafts. Skin flaps also provide immediate coverage and as a result, contraction and epithelialization of the wound do not affect healing, as they would with open wounds. They also decrease the possibility of excessive scarring, contracture, poor or prolonged healing, or nonhealing, which could occur if left to heal via second intention closure.[20] However, rotation of feather growth is a consideration with flaps as the direction of the pterylae may change with placement. Using these in areas of high tension is cautioned as it carries a high risk of failure. Flaps that incorporate a direct cutaneous artery are known as axial pattern flaps. These can be used to cover a larger area of tissue and ensure flap survival via the flap's own blood supply. Skin flaps also provide immediate coverage and as a result, contraction and epithelialization of the wound do not affect healing, as it would with open wounds. They also decrease the possibility of excessive scarring, contracture, poor or prolonged healing, or nonhealing, which could occur if left to heal via second intention closure.[20] The published use of skin flaps have been used in closure of wounds in a great horned owl *(Bubo virginianus)* with a large open wound on the distal antebrachium,[21] to cover degloving wounds on the head of northern goshawks[11] *(Accipiter gentilis)* and a red-tailed hawk[22] *(Buteo jamaicensis)*.

Free skin grafts are used in cases with significant tissue loss such as burns or degloving injuries. They can be full- or partial-thickness segments of skin removed from one area and placed over the wound. Grafts can be harvested from the same individual (autogenic graft), from another animal of the same species (allogenic graft), or from a different species (xenogenic graft). Once placed, they must develop a new blood supply in order to survive. Eventually, new vessels grow into the graft and from the skin along the margins of the wound. As avian skin is thin, revascularization is easily facilitated.[23] Since grafts often contain full-thickness skin and subcutaneous tissue, they can also provide normal feather coverage if they contain pterylae.[22] The provided graft is often fashioned in a split mesh pattern to allow drainage, which prevents seroma formation as well as to provide greater coverage for larger or uneven wounds.[20] Skin grafts are only practical in larger avian patients as grafts with smaller surface area are at greater risk of failure. Successful use of full-thickness skin grafts has been reported in great horned owls,[24] an ostrich[12] *(Struthio camelus)*, and a wild bald eagle[13] *(Haliaeetus leucocephalus)*. The bald eagle presented with large eschar formations on the dorsal head and right stifle likely caused by electrical injury. A full-thickness skin graft was harvested from the bird's medial inguinal skin fold of the left pelvic limb. Ten days following the procedure, approximately 90% of the graft had been accepted and remained healthy (Figure 36.8).

PRODUCTS AVAILABLE FOR TOPICAL WOUND MANAGEMENT

Topical therapeutic products, including antimicrobial ointments/creams, may be applied, depending on the nature of the injury and advantages of using the product for that

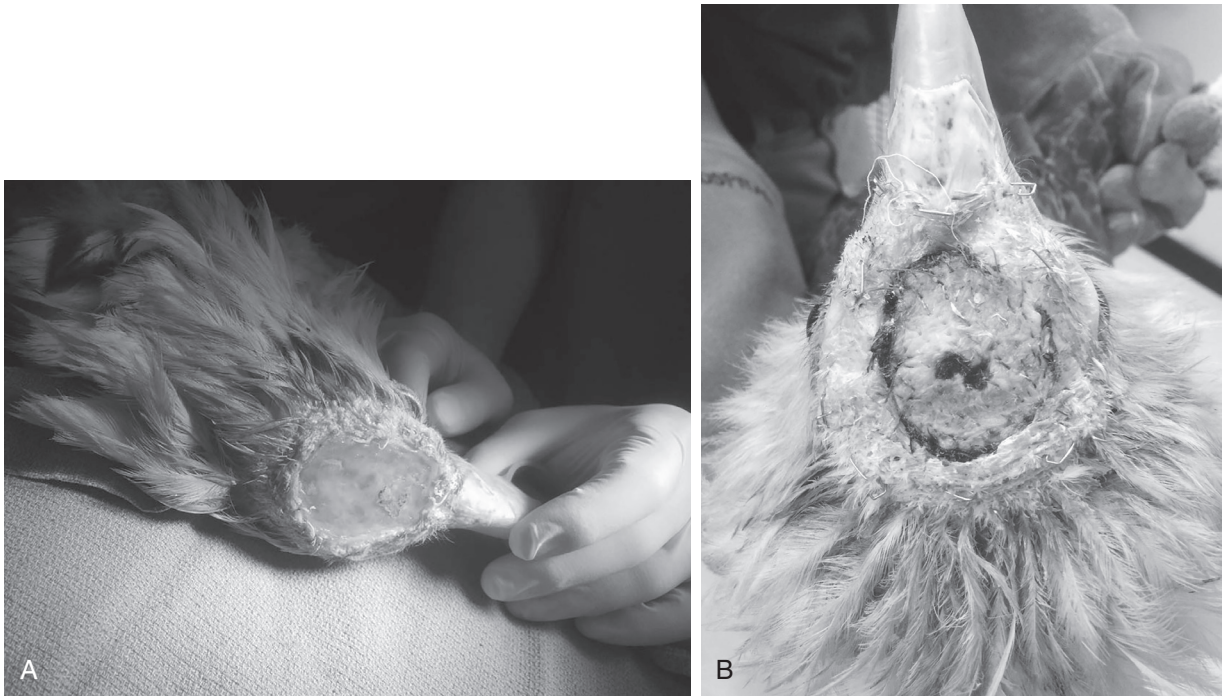


FIGURE 36.8 Use of a skin graft in a bald eagle *(Haliaeetus leucocephalus)*. The animal presented with eschar formation on the head (A). The area was debrided and a full-thickness skin graft from the medial left leg was used to cover the defect. Ten days following the procedure, there was approximately 90% acceptance of the graft (B).[23] (From Kane LP, Shrader TC, Stice RC: Successful, full-thickness skin graft in a bald eagle (*Haliaeetus leucocephalus*), *J Avian Med Surg* 33(2):184–188, 2019.)

particular case. When prescribing a topical ointment/cream for avian use, caution must always be applied. Owners need to be educated on the application process, using only a small amount to treat the lesion to reduce the possibility of feather matting. Topical ointments containing corticosteroids are not recommended due the high potential for severe adverse side effects. In addition to having glucocorticoid activity, corticosterone also has significant mineralocorticoid activity, causing increased potassium retention and glomerular filtration.[14] Documented side effects of corticosteroids in birds include suppression of cell-mediated and humoral immunity,[25] lymphopenia and granulocytosis,[26] rendering birds more susceptible to aspergillosis. In addition to immune suppression, birds given corticosteroids may also develop polyuria, polydipsia, decreased growth, hyperphagia, increased gluconeogenesis and protein catabolism causing lipemia, hypercholesterolemia, excess hepatic glucose deposition, steroid-induced diabetes mellitus, and glucosuria. As systemic absorption is facilitated through the thin avian skin, topical treatment with corticosteroid-containing products can readily lead to increased blood levels and associated side effects. A study evaluating the suppressive effects of glucocorticoids on the pituitary-adrenocortical system in pigeons found that topical glucocorticoid containing dimethyl sulfoxide resulted in systemic effects, though less pronounced than oral or ophthalmic application. However, when glucocorticoid-containing cream without dimethyl sulfoxide was used, there was no suppression of the pituitary-adrenocortical system, suggesting that local effects on the skin may be maintained.[27] Regardless, these products should be used judiciously. If topical corticosteroids are considered necessary in the treatment, every other day application is recommended to prevent secondary infections. Commonly used topical products are listed in Table 36.2 and discussed below.

Acemannan Freeze-Dried Gel (Carravet)

Acemannan Hydrogel, or Carravet (Carravet, Palmetto, FL, USA), is found in wound dressings, which can be applied directly to low-to-medium exudative wounds in birds. Acemannan is a bioactive polysaccharide that promotes tissue repair. It has

TABLE 36.2

Topical Products Used to Treat Dermal Wounds in Avian Patients

Product	Properties	Toxicity
Acemannan freeze-dried gel	Antimicrobial, promotes tissue repair, can be used on low-medium exudating wounds. Must be maintained with a bandage.	Nontoxic, nonirritating.
Borate-based biological glass	Promotes angiogenesis and healing. Must be maintained with a bandage.	Nontoxic, nonirritating.
Chlorhexidine 0.05%	Broad-spectrum antimicrobial, expedited healing time.	Associated with corneal damage and severe pain if exposed to eyes. Higher concentrations can cause tissue toxicity,[12] inhibition of granulation tissue,[13] and temporary decreased tensile strength.[14]
Hydrogel wound dressings	Maintains a moist environment, promotes autolytic debridement, and may improve healing time.	Nontoxic, nonirritating.
Hydrogel silver antimicrobial wound gel	Broad-spectrum antimicrobial, absorbs and retains wound exudate, prevents penetration of environmental bacterial, provides cooling and soothing benefits, prevents dressings from adhering to wound.	Nonirritating, nonharmful to granulation tissue.
Hydrolysate of type I collagen	Provides a matrix for tissue regeneration and moist environment, promotes autolysis.	Nontoxic, nonirritating.
Hypochlorous acid	Antimicrobial, provides moisture to wound.	Nontoxic, nonirritating.
	Slow-release antimicrobial properties for up to 7 days,[28] maintains moisture in wound, promotes cell growth and neovascularization.[29]	Nontoxic, nonirritating, low risk for toxicity to healthy tissue
Manuka honey	Broad-spectrum antimicrobial, can be used in high-exudate wounds, may stimulate the immune response, suppress inflammation, and promote debridement of the wound.	In vitro studies have shown cytotoxicity to human cells with prolonged use.[30]
Povidone-iodine cream 5%	Broad-spectrum antimicrobial, penetrates biofilms, low bacterial resistance, antiinflammatory.[11]	Low cytotoxicity, but may cause mild irritation if exposed to eyes.
Sea salt and lysozyme spray/foam	All-natural, antimicrobial, provides minerals to enhance cell growth and tissue repair.	Nontoxic.
Silver sulfadiazine cream 1%	Bactericidal and bacteriostatic properties.	Does not appear irritating, but may cause impaired reepithelialization, hypertrophic scar formation and cytotoxic effects on fibroblast cells with prolonged use, especially at higher concentrations (2%).
Triple antibiotic ointment	Antibacterial (neomycin, bacitracin, and polymyxin), but overuse may cause resistance.	May cause local irritation.

been shown to significantly accelerate skin wound closure and cell proliferation in experimentally induced skin wounds in mice.[31] This product should be applied to an aseptically cleansed wound and covered with a bandage.

Borate-Based Biological Glass (RediHeal)

RediHeal (Avalon Medical, Stillwater, MN, USA), a borate-based biological glass, is placed directly on a wound to quickly react with tissue fluids. Upon reacting with moist tissue, trace elements are released that promote a strong angiogenic response. By improving the blood flow to damaged tissue, oxygenation is increased, thereby promoting the healing process. This product must be maintained in place with a semiocclusive bandage and can be changed daily.

Chlorhexidine 0.05%

Due to its high bactericidal properties, chlorhexidine is a commonly used disinfectant and antiseptic solution. When used in wound treatments, stock solution should be diluted to a much weaker concentration. Chlorhexidine has been found to accelerate wound healing in full-thickness wounds in beagles[32] and has been found to be relatively safe at lower concentrations (0.02%) for use as a surgical wound irrigation solution. Only the higher concentration (0.05%) has been shown to cause slight tissue toxicity in rats.[30] Other studies have found chlorhexidine to cause inhibition of granulation tissue in guinea pigs[33] and decreased tensile strength of wounds in rats.[34] In rats, however, this appeared to be temporary as strength improved over time and chlorhexidine-treated wounds had decreased healing time.[28] Chlorhexidine-treated open wounds in blue-fronted Amazon parrots had significantly shorter healing time as well as increased neovascularization and fibroplasia.[8] It should be avoided on facial wounds, though, as contact with the eyes can cause corneal damage and severe pain.[35] Otherwise, chlorhexidine appears to be relatively safe with little effect on the wound healing process.

Hydrogel Wound Dressings (Intrasite, Purilon Gel, Nu-Gel, Granugel, Derma GeL)

Intrasite (Smith & Nephew, London, England, UK), Purilon gel (Coloplast, Minneapolis, MN, USA), Nu-Gel (Systagenix, Skipton, England, UK), Granugel (Convatec, West Yorkshire, England, UK), and Veterinus Derma GeL (Maximilian Zenho & Co, Brussels, Belgium) are topical amorphous hydrogel products. Hydrogel is able to maintain a moist environment in the wound by providing moisture to dehydrated tissue. It can also rehydrate necrotic tissue, which facilitates autolytic debridement and loosen tissue. The hydrophilic residue is then able to absorb moisture, slough, and exudate by swelling extensively without changing its gelatinous structure.[36] As it absorbs fluid, it becomes less viscous but remains nonadherent, therefore not harming viable tissue or skin surrounding the wound. These properties make it an acceptable topical dressing for wounds during every stage of the management process. Both Derma GeL and Intrasite have shown bacteriostatic and fungistatic activity in vitro against *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Candida albicans*.[37] Improved healing time has been documented with many of these products when compared to traditional saline dressings.[36] Each product comes with a provided application system, which is typically available in a range of sizes to help with application in awkward wound sites.

Hydrogel Silver Antimicrobial Wound Gel (SilvaSorb, Gemcore360°, Normlgel Ag)

Hydrogel silver antimicrobial wound gels, such as SilvaSorb (Medline, Northfield, IL, USA), Gemcore360° (Gemco Medical, Hudson, OH, USA), and Normlgel Ag (Molnlycke Health Care, Brooklyn, NY, USA), are wound dressings that utilizes ionic silver and is indicated for moist wounds that will respond to an antimicrobial barrier. This product is nonirritating, not harmful to granulation tissue, and absorbs and retains wound exudate, which in turn promotes fibroblast proliferation and keratinocyte migration.[38] The tight mesh of hydrogels prevents environmental bacteria from reaching the wound, while the broad-spectrum antimicrobial hydrogel product releases ionic silver contained within a controlled manner for up to 3 days. High water content of the hydrogel not only provides a cooling and soothing benefit but also prevents the dressing from sticking to the wound, causing less pain and discomfort with removal.[38]

Hydrolysate of Type 1 Collagen (Collasate, Collasate Silver Topical Dressings)

Collasate and Collasate silver topical dressings (PRN Pharmacal, Pensacola, FL, USA) are products that provide the healing benefits of collagen for wounds, including providing a matrix for cellular colonization and tissue regeneration,[39] reduction in bleeding, protecting the wound bed, providing a moist healing environment, and promoting natural autolysis. In a group of humans with diabetic foot ulcers that were treated with collagen-containing wound products, patients had faster healing time compared to those given hyaluronic acid-medicated gauze. They also complained less about pain and itch.[40] This product is available in both a spray and gel preparation, with an additional gel formula that contains silver oxide. The added silver oxide provides antibacterial properties and helps maintain a moist wound environment.

Hypochlorous Acid (Vetericyn)

Hypochlorous acid is the active ingredient in many wound care products made by Vetericyn (Rialto, CA, USA) which are promoted as a next generation line of animal health care that offers an advanced alternative to saline solutions. Hypochlorous acid is an antimicrobial agent that is safe on all tissues. Many veterinarians appear to be very happy with the clinical results of this product when treating avian wounds. Hypochlorous acid is nontoxic, nonirritating, and safe to use on birds and around the face. These products come in spray form that can be used by the owner to treat a bird through the cage.

Ionic and Metallic Silver Absorbable Wound Matrix (Microlyte Ag)

Microlyte Matrix (Imbed Bioscience, Madison, WI, USA) is a thin, transparent, dissolvable gel fiber wound dressing, which provides a slow-release antimicrobial barrier for up to 7 days. The thin dressing allows it to conform to the wound bed while the silver-containing gel provides antimicrobial activity. A micrometer-thick-soluble film of polyvinylalcohol that laminates the fiber dissolves when placed on a moist wound, leaving the silver-loaded film immobilized on the wound bed and maintaining a moist environment.[41] This conformational contact with the wound bed enables localized delivery of silver. In vivo studies using mice models have shown significant decrease

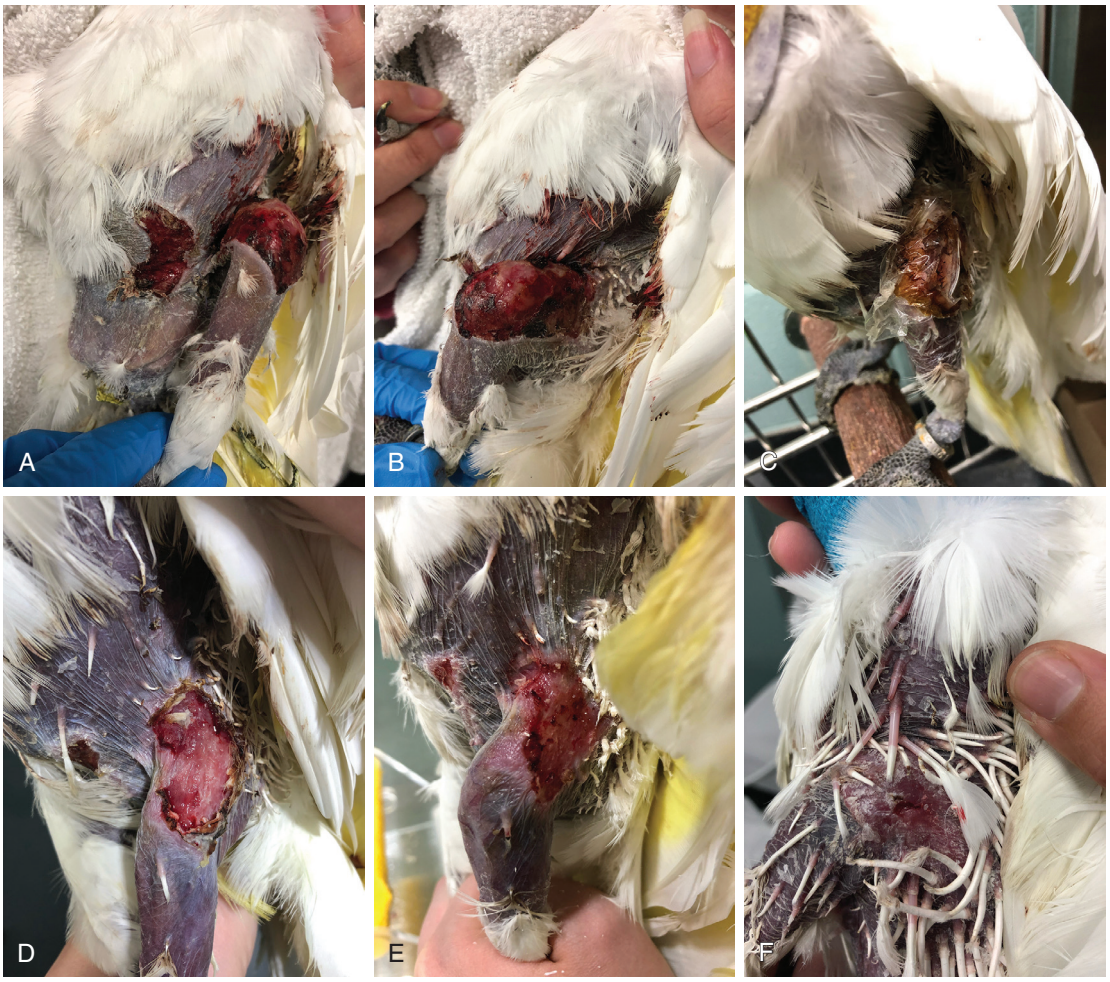


FIGURE 36.9 Severe degloving wound from self-trauma of a surgical incision in an umbrella cockatoo *(Cacatua alba)* (A and B). After 3 weeks of delayed healing with other topical products, an ionic and metallic silver absorbable wound matrix (Microlyte Ag) was applied (C) which after one application showed improved granulation (D) and more rapid healing after only 2 weeks of treatment (E). After 7 weeks following initial presentation, the wound had completely healed (F).

in bacterial burden beneath the applied dressing at 72 hours after application to contaminated wounds.[42] Studies also indicate that the matrix allows for cell growth and neovascularization, promoting faster healing time.[43] This product should be applied to a moistened wound bed. As the product is dissolvable, it does not need to be removed prior to reapplication. The authors have used this product with success in slow-healing wounds (Figure 36.9).

Manuka Honey (Medihoney, Vetramil, Dermiel)

Manuka honey and honey-containing ointments, such as Medihoney (Derma Sciences, Princeton, NJ, USA), Vetramil (Bfactory Health Products B.V. Mooresvilled, NC, USA), and Dermiel (AST Farma, Oudewater, Netherlands) have gained popularity in veterinary medicine. Although honey has been promoted as having broad-spectrum antimicrobial activity, there are considerable differences in this benefit between honey products. Manuka honey nectar contains dihydroxyacetone, which converts into methylglyoxal in the honey product. This compound prevents the inactivation of hydrogen peroxide that is produced at the wound site by enzymes present in blood.[39] Even when used in a wound environment of substantial exudate, manuka honey appears to still inhibit the growth of bacteria.[44] The high osmolarity of honey also helps to draw out fluid from bacteria and the wound bed to create an outflow of lymph. There is also strong evidence that manuka honey has bioactivities that stimulate an immune response, suppress inflammation, and promote rapid autolytic debridement at the injury site. Results of studies comparing treatment of burn wounds in humans show that honey dressings promote better wound healing than silver sulfadiazine (SSD) products.[45] The results of in vitro studies have indicated that, when combined with certain antibiotics, manuka honey may provide synergistic effects against the growth of methicillin-resistant *Staphylococcus aureus*.[46] Using the Unique Manuka Factor (UMF) rating system implemented by the manuka honey producers, the product should have a minimum rating of >10 UMF to be considered potent enough to be therapeutic. Normal, table honey has a UMF of 0–5. While difficulty in obtaining manuka honey products may lead some practitioners to consider commercially produced table honey, it is important to know why this is not

an appropriate substitute. Table honey is heated to eradicate *Clostridium botulinum* spores, which exist in the environment. However, this process also alters the peroxide activity, removing most of the medically beneficial properties. Medical-grade honey, instead, is treated with gamma irradiation, which destroys spores but also has no adverse impact on its beneficial properties. More so, when compared to medical-grade honey, a variety of table honeys were shown to have lower antibacterial activity and contain a wide range of microbial species.[47] Locally made and unprocessed honey may exhibit antibacterial effects as they still contain hydrogen peroxide, though these properties may vary with geographical location and floral source.[48]

Manuka honey should be used judiciously, as in vitro studies with human cells have shown time-dependent cytotoxicity. This may be due to the osmotic effect of honey, which also provides its antibacterial property.[49]

Povidone-Iodine 5%

Povidone-iodine is an antimicrobial agent that has long been used as an antiseptic and wound healing treatment. Povidone-iodine has many properties that make it a favorable topical treatment for wounds, including broad spectrum of activity, ability to penetrate biofilms, lack of associated bacterial resistance, antiinflammatory properties, low cytotoxicity, and good tolerability.[34] In a human comparison study with a triple antibiotic cream and 5% povidone-iodine cream, both were nonirritating to human skin while reducing bacterial counts and had significantly faster healing times than if no treatment was utilized.[50] In a study evaluating treatment options for *S. aureus* infections in rat models with prosthetic grafts, povidone-iodine use resulted in a 90.73% decrease in *S. aureus* colonies in wounds after 4 days of treatment.[51] In human studies, povidone-iodine has been shown to have low cytotoxicity to the skin. These studies also suggest that it may counteract inflammation elicited by both pathogens and the host response.[52]

Povidone-iodine comes in a liquid, spray, cream, and ointment. According to Betadine (iNova Pharmaceuticals, Chatswood, NSW, Australia), povidone-iodine ointment is more concentrated than the cream and does not spread as easily, suiting smaller skin wounds or infections that concentrate in one area.[53] Although this is not a novel product and may not often be considered for wound treatment, it remains an effective topical option.

Sea Salt and Lysozyme Spray/Foam (Vet-Aid)

These wound care products by Vet-Aid (Vet Aid, Stuart, FL, USA) are promoted as an all-natural sprays and foams that can be used to clean and heal wounds without tissue or cellular toxicity. The goal of the product is to provide antimicrobial activity while providing essential minerals to enhance cell growth and tissue repair resulting in a faster healing time as well as reducing inflammation in and around the affected healing site.

Silver Sulfadiazine Cream 1% (Silvadene, Flammazine)

SSD cream 1%, such as Silvadene (Pfizer, Kalamazoo, MI, USA) or Flammazine (A. Menarini, Florence, Tuscany, Italy) is a white, water-miscible cream containing the antimicrobial agent SSD in micronized form, providing both bactericidal and bacteriostatic properties. Clinically this product appears to aid in rapid wound healing without being irritating; however, based on clinical research studies, SSD should be used with care due to the potential for impaired reepithelialization, greater hypertrophic scar formation, and cytotoxic effect on dermal fibroblast. In a single study,[29] 24 rabbits were placed in four groups and subsequently treated with a base cream, 0.01% SSD, 0.1% SSD, and 1% SSD on similarly produced wounds. At day 7 following creation of the wounds and succeeding treatment, half the rabbits were euthanized with the remainder being sacrificed on day 28 to evaluate the epithelial gap and granulation area of the affected areas.[29] At day 28, the 0.1% and 1% SSD groups both had significantly larger hypertrophic epithelial gaps and hypertrophic scar formation than the base cream group. No difference in granulation tissue development was noted between treatment groups. A study[54] focusing on a reptilian species, the green iguana *(Iguana iguana)*, showed that SSD does provide beneficial antimicrobial and healing properties with no delay in healing time; however, this treatment may not be any more advantageous compared to other wound treatments. A 14-day course of 1% SSD on induced wounds showed no significant histological differences in ulceration, bacterial infection, reepithelialization, necrosis, inflammation, fibrosis, and collagen maturity when compared to laser treatment, a topical antimicrobial product, or no treatment.[54] A study that investigated the effect of 2% SSD on the wound healing process and dermal mechanical strength of a 15-day treated lesion in rabbit ear skin found that there was impaired dermal regeneration and decreased mechanical strength of the repaired tissue.[55] It was suggested that the impaired healing was due to the cytotoxic effect of the SSD cream on dermal cells.[55] The belief that SSD cream is cytotoxic to dermal cells was substantiated through the fact that the investigators determined SSD to be highly cytotoxic to cultured human dermal fibroblast cells.[55] As stated previously, low-concentrated SSD creams (1%) do not appear to adversely affect the formation of granulation tissue and can aid in the control and prevention of infection.[54,55] Nevertheless, there are potential adverse side effects with this product, especially at higher concentrations (2%), which demands judicious use by the medical professionals that are treating avian patients with SSD cream.

Triple Antibiotic Ointment (Neosporin, Perrigo Triple Antibiotic Cream)

Triple antibiotic ointment, like Neosporin (Johnson & Johnson, New Brunswick, NJ, USA) or Perrigo triple antibiotic cream (Perrigo, Allegan, MI, USA), is recommended for use in minor skin wounds and contains three antibiotic agents: neomycin, bacitracin, and polymyxin. The ointment should be applied two to three times a day, ensuring to use only small amounts of the product at any one time. When applied properly, only the surface of the wound surrounding the skin should glisten. If any ointment can be visualized, then the excess should be removed or gently massaged into the wound. A primary bandage can be applied over the wound, if necessary, following application of the triple antibiotic ointment.

BANDAGING

There are many reasons to apply a bandage to wounds. These range from protection of the wound site to maintaining a wound treatment in place. For whatever reasons a veterinarian feels a bandage is necessary, careful thought should occur regarding necessity and benefit prior to application. Birds present a unique anatomical challenge for clinicians regarding bandaging and maintaining dressings. Unlike the forelimb of

a mammal, wings can be difficult to bandage with their folding capability. Unique techniques have been developed to keep bandages on wings, such as figure-of-eight and body wraps. For more information on these types of bandages, see Chapter 37. Birds also have strong beaks capable of tearing through strong adhesives and bandaging material. "Distractor tabs" are often placed, which are benign pieces of tape or other material coming off a bandage to create a distraction from patients chewing off their bandage.

Wound dressings are categorized into three layers, with one or all of the layers being applied over the wound. The first, or primary, layer is the product that contacts the wound surface, which may be either an adherent or nonadherent dressing. Nonadherent dressings (such as Adaptic; Ethicon Inc., Somerville, NJ, USA) prevent tissue desiccation and cause minimal trauma. Adherent dressings use wide mesh material to allow fluid and debris to become incorporated into the bandage, which is then removed at bandage changes. However, this is a nonselective process and may damage healthy tissue if inappropriately used. Adherent dressings are classified as dry-to-dry, wet-to-dry, or wet-to-wet based on composition. Dry-to-dry consist of dry gauze applied to the wound. If placed on wounds with exudate, these may be painful to remove but enable significant tissue debridement. Wet-to-dry bandages are made with saline-moistened gauze placed directly on the wound. These can also be painful to remove but result in less tissue desiccation than dry-to-dry bandages.[23] Wet-to-wet bandages tend to damage tissue beds by keeping the healing tissue too moist. Occlusive primary bandages that are impermeable to body fluid and air have been reported to have a decreased healing time and better cosmetic result,[56] likely because they allow vital proteins and cytokines to be retained in the wound bed. This facilitates autolytic debridement within the wound and promotes healing.

The secondary layer provides support to the limb, pads the wound, holds the primary contact layer in place, and may also absorb fluid.[23] This layer is typically composed of cast padding, roll cotton, or gauze sponges. In avian species, secondary bandages are often minimized to reduce bulk and weight in relation to the patient's size.

The tertiary layer holds the primary and secondary layers in place, protects them from environmental contamination, and provides pressure.[23,56] There are a number of bandage materials available for use on an avian patient and most are commonly used for dogs and cats. These include Vetrap (3M, Saint Paul, MN, USA), Elastikon (Johnson & Johnson Medical Inc., New Brunswick, NJ, USA), and white cloth tape (waterproof and nonwaterproof). The more common bandaging products used on avian patients are listed in Table 36.3 and described below.

Cellulose Acetate Fabric With Petrolatum Emulsion or Paraffin-Impregnated Gauze (Adaptic, Jelonet, Respectively)

Adaptic (Johnson & Johnson, New Brunswick, NJ, USA) and Jelonet (Smith & Nephew, London, England, UK) are types of nonadherent dressings made of tight mesh, which allows for the dressing to be cut to size, though they also come in a variety of sizes. The mesh allows exudate from the wound to be absorbed by secondary dressing placed over the mesh.

Cyanoacrylate Tissue Adhesive (Vetbond)

Cyanoacrylate tissue adhesive like Vetbond (3M, St. Paul, MN, USA) bonds tissues together to form a flexible barrier and also acts as a hemostatic agent. Once contacting the body,

TABLE 36.3

Bandaging Materials Used to Dress Wounds in Avian Patients

Type	Properties	Type of Wound Best for
Dry-to-dry	Adherent, significant tissue debridement.	Heavily necrotic wounds, may be painful to remove.
Wet-to-dry	Adherent, significant tissue debridement with less tissue desiccation.	Heavily necrotic wounds, may be painful to remove.
Wet-to-wet	Adherent, may damage tissue beds by keeping it too moist.	Not recommended.
Cellulose acetate fabric with petrolatum emulsion or paraffin-impregnated gauze	Nonadherent, nonabsorptive, protects wound.	Nonexudative wounds during the proliferative phase; may be used as a primary layer with absorptive secondary layer in low to high exudative wounds.
Cyanoacrylate tissue adhesive	Forms long-lasting transparent bandage, protects wound, semiocclusive.	Noncontaminated wounds, wounds of small size or in areas difficult to bandage.
Hydrocolloid dressings	Adhesive, occlusive, impermeable to moisture and oxygen, maintains a moist environment, absorbs fluid and exudate.	Noncontaminated wounds, dry wounds, low to moderate exudative wounds.
Semiocclusive polyurethane membrane coated with acrylic adhesive	Adherent, semiocclusive, nonabsorptive, prevents external contamination, maintains moist environment.	Noncontaminated wounds, no to low exudative wounds. May be used as secondary layer with absorptive primary layer in high-exudate wounds.
Specialized nonadherent gauze pads	Nonadherent, absorptive.	Dry to lightly draining wounds.
Tilapia skin	Nonadherent, nonabsorptive, prevents loss of moisture, contains collagen that accelerates healing process.	Sterile burns or degloving injuries.

the liquid transforms into a solid in order to seal wounds within seconds of application and prevent external contamination. The blue dye contained in the product increases visibility during application. It will naturally slough off over the course of 5 to 7 days. This helps eliminate the need for overbearing bandages that may stress out the animal. It is especially effective for areas that are difficult to bandage, noninfected wounds, and surgical incision sites. Care should be used when applying, as the adhesive should only be applied to the dermal surface and not directly to exposed subcutaneous tissue. The quick drying time can also cause external objects, including surgical instruments, gloves, feathers, drapes, and so on, to easily stick to the product and the patient. The area should be completely clean prior to application. Skin edges should be appropriately apposed and using a new applicator tip, the tissue adhesive is applied in single drops along the apposed margins.

Hydrocolloid Dressings (Allevyn, DuoDERM, Medihoney)

Allevyn (Smith & Nephew, London, England, UK), DuoDERM (Convatec, West Yorkshire, England, UK) and Medihoney (Derma Sciences, Princeton, NJ, USA) are adhesive, opaque, and impermeable hydrocolloid dressings. These occlusive dressings provide a moist and insulating healing environment for uncontaminated wounds which allows the body's own enzymes to heal the area. The hydrophilic dressings absorb fluid and exudate from the wound surface to produce a gelatinous, moist cover for the healing surface. They may require additional bandaging material to hold in place despite their adhesive covering.

Semiocclusive Polyurethane Membrane Coated With Acrylic Adhesive (Tegaderm)

Tegaderm (3M Medical Solutions, St. Paul, MN, USA) is a thin semiocclusive polyurethane membrane coated with a layer of acrylic adhesive. The polyurethane membrane protects the wound from external contamination while reducing moisture loss from the exposed tissue. The wound environment promoted by this dressing prevents eschar formation, thereby increasing epidermal regeneration. Due to its adhesive nature, the product can be applied as a primary contact layer bandage alone, which provides good visualization of the wound. The thin material allows it to be contoured to fit small patients and wounds in difficult locations. Its beneficial adherent properties can also cause difficulty when applying, as the product may adhere to itself. This can cause frustration for the attending clinician or personnel who is applying the product. Based on the author's experience, once the wound is prepared, it is easiest to remove the paper lining and then apply the adherent side to the area to be covered prior. Once applied, the paper tabs can be removed, ensuring to apply pressure to the bandage margins as they are removed. This product may be used at home by clients for long-term wound management, but proper instructions on application should be provided. There are many products available that can be used as primary contact layer bandages including high-performance foam, Ag mesh dressing with silver, hydrocolloid adhesive dressings, alginate dressing, alginate Ag silver dressing, hydrogel wound filler, and nonadherent contact layer.

Specialized Nonadherent Gauze Pads (Telfa, Melolin)

Telfa (Covidien, Dublin, Ireland) and Melolin (Smith & Nephew, London, England, UK) are types of specialized nonadherent gauze pads that can be used as primary or secondary dressings. A polymer coating on both sides of the dressing prevents the product from adhering to the wound bed if placed directly on it, unlike gauze. These come in a variety of sizes and variations.

TILAPIA SKIN

Application of Nile tilapia skin *(Oreochromis niloticus)* is an emerging advancement in wound management. Previously seen as a fish-processing byproduct, tilapia skin has been found to greatly improve healing time in cases of skin burns or degloving injuries. Tilapia skin has low toxicity and is well tolerated in mammalian patients. The collagen nanofibers contained in the skin promote skin regeneration by inducing keratinocyte differentiation and collagen synthesis by dermal fibroblasts.[57] Additionally, it acts as a barrier from excessive exudate and fluid loss, prevents bacterial invasion, and is easily removed for changing purposes. The tilapia skin is prepared with sterilization and irradiation, so it can last up to 2 years. It is applied as an occlusive dressing, often sutured in place, and left for up to 10 days. Its application may be limited to larger avian patients with large enough surface area to accept. Though reported use of this dressing is limited in avian patients, clinicians should consider this product for large sterile skin wounds. For more information on the use of tilapia skin, see Chapter 53.

THERAPEUTIC MODALITIES FOR WOUND TREATMENT

Laser Therapy and Photobiomodulation

Red and near-infrared light wavelengths are directed over the wound area for increased therapeutic effect. The light interacts with chromophores in cells, causing upregulation or downregulation of some biochemical processes. Wavelengths in the range of 400 nm (violet) to 1100 nm (near infrared) can result in photobiomodulation.[58] The mechanism of action of photobiomodulation is not yet completely understood but has been shown to promote improved healing time, provide analgesia at the wound site, increase circulation, and decrease edema. Low-level laser therapy has been shown to reduce inflammation and significantly increase collagen deposition in open wounds in rats, with the best results seen with daily application of 16 J/cm^2.[59] It has even resulted in superior wound contraction in full-thickness burns in rats compared to topical treatment of 2% SSD.[60] Pododermatitis in Magellanic penguins *(Spheniscus magellanicus)* showed better healing rates and times after surgical debridement with laser therapy than in a group treated with antiinflammatories and antibiotics.[61] Multiple treatments are usually required to achieve the most beneficial effect of increasing the metabolic activity of cells surrounding the wound to stimulate healing. For smaller avian patients, lower therapeutic doses are recommended compared to larger species and are listed in Table 36.4.

TABLE 36.4

Suggested Doses for Laser Therapy in the Treatment of Avian Wounds[58]

Dose	Usage
1 J/cm^2	Surgical wound healing and postoperative pain Dermal burns
2 J/cm^2	Lacerations
2–4 J/cm^2	Trauma site inflammation and pain Minimize scar formation and increase healing from self-mutilation
3–4 J/cm^2	Pododermatitis
4 J/cm^2	Deep wounds
4–6 J/cm^2	Reduce patagial extension restriction following trauma

Due to the risk of retinal damage, operators and patients should have their eyes protected. Caution should be used to prevent tissue overheating and overuse in pigmented areas, which will absorb more light. Neoplastic or infected tissue may become overstimulated with laser therapy and should also be avoided. Treatment is also not recommended over open fontanels, hemorrhagic tissue, or in pregnant patients.[58]

Ultrasonography

The delivery of ultrasonic waves to tissue provokes thermal and nonthermal effects that may stimulate soft tissue healing. The sound waves interact with large protein molecules (such as collagen), causing increased skin temperature, which results in increased extensibility and flexibility of collagen-rich scar tissues and blood flow.[58] This may be beneficial in cases of chronic inflammation. Appropriate selection of frequency, intensity, duty cycle, and duration of ultrasonic waves delivered are important considerations. With conventional therapy, high frequency (1.1–3.3 MHz) and intensity (0.1–3.0 W/cm^2) ultrasonic waves are delivered to the target area in a continuous or pulsed mode.[58] Pulsed mode may be more advantageous in providing mechanical effects over thermal ones. Therapeutic ultrasonography, when used in rat models, decreased necrosis of skin flaps and increased levels of endothelial growth factor in skin margins compared to nontreated animals.[62] Similar results have also been reported in rabbits with skin grafts.[63]

Negative Pressure Wound Therapy

Negative pressure wound therapy (NPWT) is promoted to improve wound perfusion, reduce interstitial edema, promote the formation of granulation tissue, decrease the formation of substances that inhibit wound healing, and diminish bacterial contamination of the injury site. Studies evaluating their use in wound treatment in rabbits[64,65] and chelonians[66–68] have shown that NPWT can be effective at reducing edema and promoting a healthier wound environment causing expedited healing. No reports of their use in an avian species have been published at this time. Small portable units including the vacuum-assisted closure (VAC) device (KCI, San Antonio, TX, USA) can be used for larger birds. NPWT is not practicable for small avian patients because their body surface area is not often large enough to achieve a negative pressure wound environment over the affected area; however, NPWT remains a therapeutic option to treat avian wounds and it is up to the attending veterinarian to determine if the patient in question would benefit from this treatment and if it would be possible to use.

Hyperbaric Oxygen Therapy

Hyperbaric oxygen therapy (HBOT), up to this time, appears to be a rarely used modality to treat avian wounds due to availability and cost. Recently there has been an increase in HBOT chambers manufactured specifically for the pet market, and this treatment option may become increasingly popular for avian wound care soon. The patient's natural healing process is enhanced by inhaling 100% oxygen in a chamber where atmospheric pressure is increased and controlled. Through HBOT supplemental oxygen infiltrates injured areas of the body, improving the antimicrobial activity of the white blood cells, reducing swelling, and promoting angiogenesis. It may also have some bactericidal activity. HBOT has shown improved healing times in full- and partial-thickness wounds in rats,[69] full-thickness wounds in mice,[70] and second-degree burns in guinea pigs.[71] Preliminary results evaluating the effects of HBOT on healthy domestic chickens by the author showed no adverse effects on the chicken's overall health.

Regional Limb Perfusion

Regional limb perfusion is a technique where antibiotics are administered intravenously to a selected region of a limb through the venous system. A tourniquet is placed, often for no more than 15 minutes, to diminish systemic absorption of the medication. Concentrations of ceftiofur sodium in synovial fluids exceeded therapeutic thresholds when administered via intravenous regional limb perfusion in an avian model,[72] indicating that this technique allows for a high local concentration within deep tissue. This technique has been routinely used in veterinary medicine in a variety of species, including horses, rabbits, and elephants. The first reported use in an avian species showed improvement in lameness and size of lesions associated with pododermatitis in a chicken with intravenous amikacin.[73] Intravenous regional limb perfusion should be considered an effective technique for treatment of contaminated wounds and other infections of the distal aspect of limbs in birds. For more information, see Chapters 6 and 33.

Hydrosurgery

Hydrosurgery uses a device that forces saline solution through a narrow window at high velocity, creating a localized vacuum. This allows for dead tissue to be removed while also diluting the local bacterial load. The suction permits targeted tissue to be cut while aspirating debris from the site. It has been described as a debridement technique for burns, venous leg ulcers, and pressure ulcers in human medicine. Studies in human trials found no significant shortening of wound healing time with hydrosurgery compared to traditional techniques and no difference in quantitative bacterial load.[74] It also does not allow for hemostasis, limiting its use for large and hemorrhagic wounds. It may be effective on dry fibrin and necrotic tissue though. In vitro studies using equine muscle showed that hydrosurgery reduced the load of *S. aureus* by 99.7%, which was higher than saline irrigation with sharp debridement (87.4%), sharp debridement (82.2%), and saline irrigation (46.0%).[75] At the time of publication, the only reported clinical case of hydrosurgery in veterinary medicine was the use in a beluga whale.[76] Its application may be considered in the future for the contamination of necrotic wounds in avian patients.

Coblation

Coblation uses bipolar radiofrequency to energize molecules via a conductive solution surrounding an active electrode,

which creates a precisely focused plasma field. The glow discharge plasma produces chemically active radical species from the dissociation of water, breaking molecular bonds and causing tissue dissolution. Unlike hydrotherapy, there is no risk of spreading aerosolized bacteria. It also provides a hemostatic effect, which may be beneficial for chronic inflammatory wounds.[74] There have been no human-controlled trials assessing its effectiveness in wound healing in humans published to date, but in vitro studies have shown significant microbicidal effects. Its application in veterinary medicine has also not been reported for the purpose of wound management, as this remains an emerging new technology for clinicians.

SUMMARY

Wound management is an important part of avian veterinary practice. Birds are often presented with skin injuries that are the result of many causes. The presented wounds need to be assessed and treated in a proper manner in order to promote a rapid healing process (see Table 36.5 for examples of common avian wounds and their recommended treatment). It is important to remember that upon initial presentation, the bird must be stabilized before extensive wound treatment can take place. A temporary bandage can be applied to protect and reduce further trauma to the affected area as the patient gains strength prior to properly managing the injury. There are many treatment products available to the veterinarian for wound management and to ensure optimum healing outcome, knowledge of both wound healing and the products used is essential. A flow chart to facilitate decision-making when managing wounds in the avian patient can be seen in Figure 36.10.

TABLE 36.5

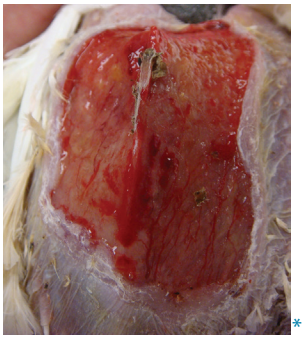
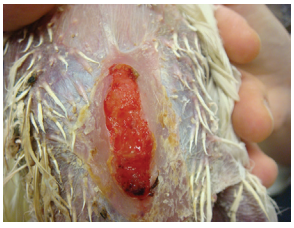
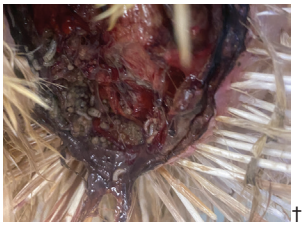
Examples of Common Avian Wounds and Recommended Treatment

Example	Type of Wound	Recommended Treatment
 *	No exudate, poor granulation or agranular tissue (pale, gray/brown)	Primary or secondary closure Dressings that provide antimicrobial and exudate-management Focus on nutrition to help grow stronger tissue
 **	Low exudate	Topical lavage Primary or secondary closure Dressings that provide moisture to prevent desiccation
 *	High exudate (soft, gelatinous)	Topical lavage to reduce bacterial load ± wound debridement Secondary or tertiary closure Dressings should provide direct pressure and manage moisture and pathogens

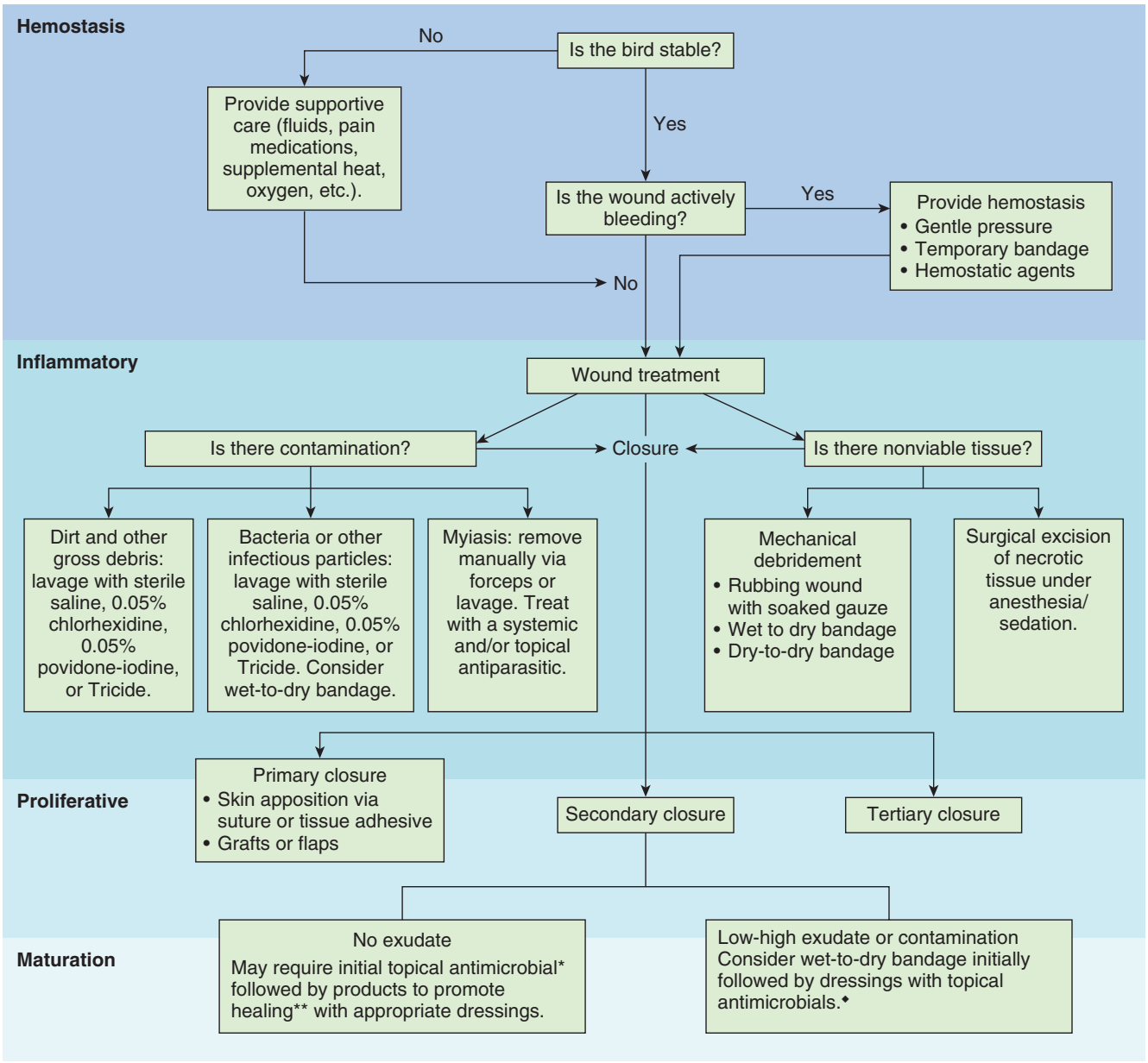
Continued

TABLE 36.5

Examples of Common Avian Wounds and Recommended Treatment—cont'd

Example	Type of Wound	Recommended Treatment
*	Necrotic (black, nonlistening) with or without plaque formation	Wound debridement (surgical or mechanical) Secondary or tertiary closure Consider dressings that aid in autolytic debridement
*	Granulation tissue (firm and pink-to-beef-reddish color)	Debridement and management of exudate product Dressings that ensure minimally moist and protective environment
	Epithelialized tissue (pale, pink/mauve tissue usually found at the edge of wounds)	Secondary closure Dressings that provide from exposure and excessive moisture
†	Myiasis	Manual removal followed by topical lavage Systemic or topical antiparasitics Secondary or tertiary closure Dressings that provide continued antimicrobial treatment

*Photos courtesy Kyra Berg; **photo courtesy Tiana Royer; ***photo courtesy Yvonne van Zeeland and Nico Schoemaker; †from Gomez-Puerta LA, Cribillero NG, Silva W, Ayala P: Cloacal myiasis by *Lucilia* spp. (Diptera: Calliphoridae) in a rooster *(Gallus gallus domesticus)* and two Harris's hawks *(Parabuteo unicinctus)*, *Parasitol Int* 83:102363, 2021.



*Topical products for no exudate wounds:
Chlorhexidine 0.05%
Hypochlorous acid
Povidone-iodine 5%
Silver sulfadiazine cream 1% (judiciously)
Sea salt and lysozyme spray/foam
Triple antibiotic ointment (judiciously)

◆Topical products for low-high exudate wounds:
Acemannan gel
Chlorhexidine 0.05%
Hydrogel silver wound gel
Hydrogel
Hydrolysate of type I collagen
Hypochlorous acid
Manuka honey
Povidone-iodine 5%
Silver sulfadiazine cream 1% (judiciously)
Sea salt and lysozyme spray/foam
Triple antibiotic ointment (judiciously)

**Topical products that promote healing:
Borate-based biological class
Ionic and metallic silver absorbable wound matrix
Hydrogel
Hydrolysate of type I collagen

FIGURE 36.10 Decision-making schematic for wound management.

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CHAPTER 37

Management of Fractures and Luxations

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INTRODUCTION

Successful management of fractures and luxations in the avian patient requires an understanding of orthopedic principles, an appreciation of the unique anatomy of birds, a comprehensive evaluation of the presenting patient, and clear outcome objectives. Musculoskeletal injuries are common in avian medicine and while the proximate cause of most fractures and luxations is traumatic, there are many potential underlying contributing factors that are reflected in the diversity of avian species and their reasons for being in captivity. Wild birds being treated in rehabilitation centers present with different comorbidities and overall health status than caged birds living as companion animals. Exotic and domestic bird species held for display or education in zoos and nature centers represent a third type of avian patient with a wide variety of health histories, ranging from a captive-bred exhibition bird to an ambassador animal that is permanently injured and nonreleasable. Nutritional status will vary based on their life history as will overall health and exercise levels. Each of these groups has a different role to fill and those histories influence treatment, prognosis, and acceptable outcomes. Successful outcomes are defined by the requirements the bird needs for survival or its purpose in its specific setting (e.g., free-flight program) as well as the bar set for acceptable long-term welfare.

Many of the most common orthopedic conditions, including fractures, luxations, and degenerative joint disease, are secondary to trauma with or without an underlying pathological disorder such as metabolic bone disease, neoplasia, or malnutrition.[1] Because avian long bones have brittle cortices and minimal soft tissue covering, birds present with a higher level of comminution than humans, dogs, or cats.[1] Fractures often occur in pet birds as a result of trauma from falling, impacting a window or ceiling fan, being stepped on, or being the victims of an encounter with a dog, cat, or other predator.[2] Trauma can also be seen secondary to weakness, ataxia, or seizures in companion birds with other health issues such as atherosclerosis, bornavirus infection, and disease or lead toxicosis. In addition, malnutrition and poor bone quality often contribute to companion bird fractures, especially in laying hens on poor diets. Poor bone density may also contribute to wing fractures that occur secondary to restraint in psittacines. These fractures are likely to be lower energy (lower impact) than those suffered by wild birds, with less soft tissue damage and less comminution. Free-living wild birds, however, are more likely to suffer high-energy fractures with significant soft tissue damage and highly fragmented bones. In addition, they are more likely to have concurrent traumatic issues such as eye damage, head trauma, and multiple limb fractures.

In general, companion and aviary birds are more likely to present with pelvic limb fractures than wing fractures.[1,3] Small caged birds such as canaries have been reported to have a very high prevalence of tibiotarsal fractures.[4] Wild raptors, however, have a higher incidence of pectoral limb breaks with approximately 80% of their reported fractures involving the wing (The Raptor Center [University of Minnesota] unpublished data).[5] Reports of commonly seen luxations are correlated with fracture locations: pet and aviary birds are more susceptible to femorotibial and coxofemoral luxations while wild birds have a higher incidence of elbow and shoulder girdle luxations (The Raptor Center, unpublished data).[6] Understanding the differences between avian populations related to the most likely cause of injury, underlying health conditions, and potential concurrent injuries will influence the clinician's diagnostic and treatment plan for the avian trauma patient.

Different populations of birds also have different desired final outcomes. For wild birds, the goal is full restoration of flight and function so that the bird can be returned to its natural existence. For a companion bird, full restoration is still the ideal goal, although it may be less critical. When full restoration of function is not possible for whatever reason, it may be appropriate to set a goal of returning a patient to a comfortable and pain-free life. With food supplied daily by a provider and often no requirement for flight, captive birds may have acceptable welfare despite less-than-perfect restoration of function. Ultimately, the decisions made, and actions taken on presentation of a fracture case will impact the long-term outcome and set the stage for the quality of the bird's welfare.

Management of orthopedic conditions encompasses the care that is provided from initial presentation through active fracture treatment and extends after fracture healing with physical therapy and management changes. The likelihood of a successful outcome is increased when the clinician has an appreciation of the unique characteristics of birds, a strong understanding and appropriate application of orthopedic principles, and clear, appropriate objectives for patient outcomes.

ANATOMICAL CONSIDERATIONS

The evolution of flight has resulted in a skeleton that is very lightweight while at the same time strong enough to withstand the stresses of flight, including takeoff and landing. Many of the bones in the skeleton of birds are fused, resulting in a smaller total number of bones than other vertebrates and providing extra stability for flight.[7] This increased rigidity to the skeletal system also predisposes to traumatic fractures. Although the avian skeletal system is quite strong, the cortices of the long bones are substantially thinner relative to bone diameter than in similarly sized mammals. Because of these thin and brittle cortices, bones often shatter on impact and fractures are frequently comminuted. Another complication of the thin cortices of bird bones is they have less holding capacity for fixation than mammalian bones.[2]

Other structural characteristics of note in avian patients are the presence of pneumatized bones in many bird species and the bipedal nature of movement. Most, but not all, birds have pneumatized bones, which communicate with the respiratory system. This communication is recognized as a potential pathway for the movement of pathogens between a traumatized limb and the respiratory system and must be considered during treatment. Diving birds have less pneumatization of bones than birds that spend more time flying. In general, pneumatic bones do not secure fixation as well as solid bones, another consideration that the clinician should take into account when preparing a treatment plan. The bipedal nature of birds means that wings do not need to be weight-bearing during recovery/fracture healing, an advantage when determining the optimal treatment plan for a wing fracture. With pelvic limb fractures, however, the clinician has to take into account the requirement for almost immediate weight-bearing or use of the injured leg when developing a plan.[8]

In addition to unique bone structure relative to most mammals, birds have a minimal amount of soft tissue covering their long bones. With very thin skin, minimal subdermal tissue, and centralized musculature, many long-bone fractures are open, potentially resulting in contamination, devascularization, and loss of bone viability. This paucity of soft tissue protection can have a substantial impact on both treatment approach and prognosis. It is critical to lubricate, protect, and support/stabilize exposed bone fragments during triage to avoid devitalization and bone loss. If soft tissue connections are preserved, birds have a strong ability to incorporate their bone fragments into a healing fracture. Although the need to preserve long-bone length makes it intriguing, successful bone grafts have not been commonly used in birds as they have very little cancellous bone and there are few other options.[2]

The propatagium is a soft tissue structure critical for flight that may be damaged during traumatic events. Severe contraction of the propatagium occurs when the elastic fibers forming it are replaced by fibrous scar tissue after a traumatic event. This type of scarring can also be seen when wing fractures are treated with figure-of-eight bandages and/or not provided adequate physical therapy.[8] As the loss of patagial elasticity and function can prevent return to full flight capacity despite adequate fracture healing, particular attention needs to be paid to conserving and protecting this structure in birds for which restoration of wing function is critical to their survival.

In hens, derangements of calcium metabolism associated with egg laying can present unique challenges. The primary function of medullary bone in avian long bones is to serve as a calcium reservoir for the rapid movement of calcium required by hens. As an eggshell represents up to 10% of the total body calcium, the demand of egg laying can complicate bone healing by redirecting the calcium needed for bone healing to egg formation, especially in a malnourished hen.[9] During the hyperostotic bone phase of calcium mobilization the radiographic appearance of the bone changes, and the placement of an intramedullary (IM) pin may not be possible.

FRACTURE HEALING

Relative to mammals, avian bone healing can be quite rapid.[10] Uncomplicated humeral and femoral fractures can heal in 3 to 4 weeks in adults and even more quickly in juvenile birds (Table 37.1). As a rule of thumb, the more distal long bones have slower healing. Overall outcomes will be improved if normal joint movement, range of motion, and bird mobility can be maintained during the healing process, goals most easily achieved with surgical fixation. In an experimental setting, it took 5 weeks for a radius/ulna fracture to heal with a surgical approach while similar fractures took 8 weeks when treated with coaptation.[11] More recently, a retrospective study in raptors showed that conservatively managed cases healed more quickly than surgically managed ones, although this may have been biased by case selection (i.e., more complicated cases were more likely to be surgically managed).[12] Complicating conditions such as open fractures, severely comminuted fractures, infection, patient comorbidities, and very distal limb fractures will all contribute to delayed healing times.

Because the predominant source of hematogenous nutrition is associated with the periosteal blood supply, disruption of this system may result in delayed healing or a nonunion.[2] While free bone fragments without a blood supply have a high risk of necrosis and sequestrum formation, the presence of soft tissue attachments indicates that fragments may be incorporated into the healing process.[13] For these reasons,

TABLE 37.1

General Healing Times for Uncomplicated (e.g., Simple Transverse, Closed) Versus Complicated (e.g., Comminuted, Open) Fractures

Fracture	Uncomplicated (in Weeks)	Complicated (in Weeks)
Coracoid, clavicle, scapula	3–4	4–6
Humerus	3–4	4–6
Radius and ulna	4–5	6–8
Metacarpal and phalanges	5–6	7–10
Femur	3–4	4–6
Tibiotarsus	4–5	6–8
Tarsometatarsus and digits	6–8	10–12

Note these are general guidelines, and each fracture will heal at a different pace depending on the fracture itself, the complication, how quickly and effectively the complication is addressed, and the overall health status of the individual patient.

minimization of tissue handling and avoiding disruption of fragments are key concepts.

How bone healing progresses is a function of adequate nutrition (blood supply) and the amount of (or lack of) motion between fragments. The concept of interfragmentary strain has been used to explain direct and indirect bone healing.[14] When fixation is complete and there is no movement or gap between fragments, direct or primary healing occurs and there is minimal callus formation. This requires a minimum of 50% alignment and contact between the fragments as osteoclasts, osteoblasts, and osteoid production cannot cross any gaps. Healing is hard to assess radiographically as there is only a gradual loss of detail to fracture lines. With primary healing, it can take many months for sufficient remodeling to return bone to its original, functional strength. In contrast, if there is any gap present between fragments at the fracture site, bone healing occurs indirectly, starting with the formation of a hematoma and progressing through inflammation, soft callus formation, deposition of osteoid, and mineralization. Eventually, with the formation of woven bone, a stiff callus is formed. Successful healing is based on the amount of interfragmentary gap strain. While this results in a strong and successful union, without adequate rigidity in the early phases, there may be too much gap strain, which can result in a fibrous union rather than a bony callus.[15]

Over time, the approach to surgical fixation of mammalian fractures has progressed from open reduction and rigid fixation through mechanical stabilization to a biologically friendlier approach stressing preservation of blood supply and gentle tissue handling (OBDNT—"Open But Do Not Touch"). This progression has continued in recent years to a new philosophical approach known as minimally invasive osteosynthesis (MIO), in which the fracture site is not opened, preserving the initial hematoma, and promoting earlier healing. MIO preserves the biological environment and emphasizes longitudinal and axial alignment of bones rather than perfect structural anatomy, as well as focuses on optimal construct stability and integrity rather than rigid interfragmentary stability.[16] This philosophy is well aligned with the experiences and teachings of Dr. Patrick Redig of the University of Minnesota's Raptor Center, a pioneer in avian orthopedics who has stressed soft tissue–preserving approaches to avian orthopedics for decades. Redig's development and use of the tie-in fixator (TIF), also known as an external skeletal fixator-intramedullary tie-in (ESF-IM tie-in) followed the OBDNT approach and recommended a closed (minimally invasive) approach whenever possible. Although supported by clinical outcome and evidence-based medicine, this minimally invasive fracture repair method does have some downsides, including the need for excellent knowledge of anatomy and early intervention to align fragments and maintain limb length, an option that is not always available with avian patients.[17]

DECISION-MAKING PROCESS FOR FRACTURE TREATMENT

Timely and appropriate decision-making on fracture treatment is fundamental to consistently getting to the best possible outcomes. The earliest choices made are often the most influential on outcome and need to take into consideration the treatment goals, the overall patient, and the specifics of the fracture presentation. The clinician should have clear objectives based on the "role" of the patient. Is it a wild bird that absolutely needs full function before release? Is it a companion bird who has a caregiver to provide for it and ensure good welfare? Or is it a bird that is on display or used for educational purposes? Depending on the environment that the bird must function in posthealing, acceptable outcome goals may vary. Ideally, the goal for all cases would be a return to full function, which often dictates whether a surgical or nonsurgical treatment approach is utilized. In situations where full function is not a feasible outcome and the bird's long-term care can be provided for, it is important to take prognosis for long-term comfort (lack of pain) and the ability to ensure good welfare into consideration when determining a treatment approach. All potential outcomes are enhanced by choosing an approach that maximizes healing while minimizing secondary morbidity.

EVALUATING THE PATIENT: A HOLISTIC APPROACH FROM INITIAL ASSESSMENT THROUGH TO FINAL OUTCOME

Given the wide diversity of birds, the signalment of the presenting case is important. Species variations can have an extremely significant impact on decision-making. Psittacine species are more resilient than raptors and will adapt to life with a limb amputation in ways that raptors rarely do. The number of pneumatized bones is correlated with size and ecological behavior and varies by species. In general, the number of pneumatic bones is increased in species such as albatross, condors, and vultures that soar while diving birds have a reduction in number. Hawks and falcons typically have pneumatized humeri and femurs while the monk parakeet and cockatiel have only pneumatized humeri. Loons, penguins, and cormorants are considered to have apneumatic bones.[18] These anatomical variations may restrict the choices on surgical fixation devices. In addition, patient size also matters. Many of the orthopedic techniques used in birds were developed for raptors weighing a kilogram or more. The TIF, a very versatile device, can be easily adapted to birds under 100 g but is not realistic for a 20-g passerine.[2] Age and sex also influence outcomes with the bones of juvenile birds healing much more quickly than adults and calcium metabolism affecting the bones of laying hens.

The clinician often has more treatment options with captive birds than with wild birds, where a successful outcome is defined by the ability to survive in the wild, and some decisions, such as conditions necessitating euthanasia, are defined by regulatory agencies such as the United States Fish and Wildlife Service (FWS), Migratory Bird Treaty Act (MBTA) and Code of Federal Regulations (CFR). Among captive birds, living environments also vary tremendously. Typically maintained in some type of enclosure, these can be anything from individual cages to large, free-flight aviaries. These long-term husbandry and housing conditions influence choices on acceptable outcome compromises. Diet and nutrition, on the other hand, are important considerations for treatment and prognosis. While wild birds often have far more complex fractures and a higher bar for acceptable outcomes, they almost always have better bone integrity and nutrition levels than companion birds, providing more options on fixation devices and better prognosis for healing.

In addition to the husbandry issues mentioned earlier and the outcome considerations dictated by the bird's activities

and living circumstances, the patient's overall health status influences treatment decisions. Comorbidities may be the result of lifestyle (poor nutrition, limited exercise) over the years, secondary injuries associated with trauma, or simply related to aging and geriatrics. Of note for wild birds is the high incidence of ocular impairment associated with trauma. Studies have shown that 14% to 28% of raptors admitted to wildlife rehabilitation centers have ocular damage, which may affect decisions regarding releasability.[19] A complete ophthalmological examination and visual assessment should be done as part of the admission process as visual compromise may impact survival odds. In the authors' experience, ipsilateral trauma is common in birds admitted with chronic ocular lesions, possibly indicating a compromised ability to avoid injury. Additionally, wild birds may also have significant internal traumatic injuries that may delay their stability for a prolonged anesthetic event associated with surgical fixation.

ORTHOPEDIC EVALUATION AND PLANNING THE REPAIR

When evaluating a fracture patient's treatment options and prognosis, it is essential to understand the characteristics of a fracture and its anatomical location in order to achieve successful outcomes.[20] The type of fracture, bone integrity, and degree of fracture displacement should be assessed with a minimum of two orthogonal radiographic views; additional views such as the H-view for shoulder girdle fractures and oblique views for femoral head fractures can be very helpful (Figures 37.1 and 37.2).[21,22]

The common classification systems for fractures, which often include causal factors, location, morphology, and severity, are helpful tools in characterizing fractures as the nature of the fracture has direct implications for the choice of treatment (Table 37.2; Figure 37.3).[20,23] When planning the construction of a fixation device, the type of fracture is a key consideration. Transverse fractures often can load share with a fixation device while oblique, segmental, and comminuted fractures are unlikely to have an initial capacity for load sharing and require a much stronger construct for fixation. The clinician must also consider the location of the fracture, nearby structures, and the ability to place fixation without damaging joints or other structures. Mid-diaphyseal long-bone fractures are less likely than periarticular fractures to be associated with joint damage, which often leads to chronic degenerative joint disease, a complication that may prevent a wild bird from being successfully released or require long-term pain management for a permanently captive bird. Loss of joint function is likely

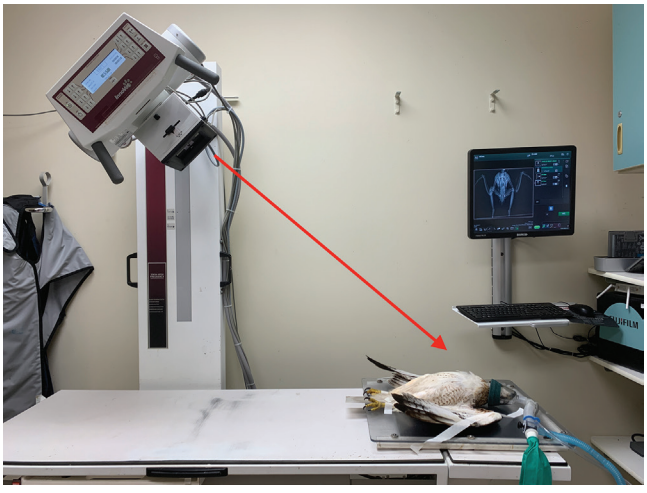


FIGURE 37.1 Setting the radiographic machine with the beam centered on the thoracic inlet at a 45-degree angle to the patient in dorsal recumbency (H-view) avoids superimposition of shoulder girdle bones and may allow clearer visualization of fractures.

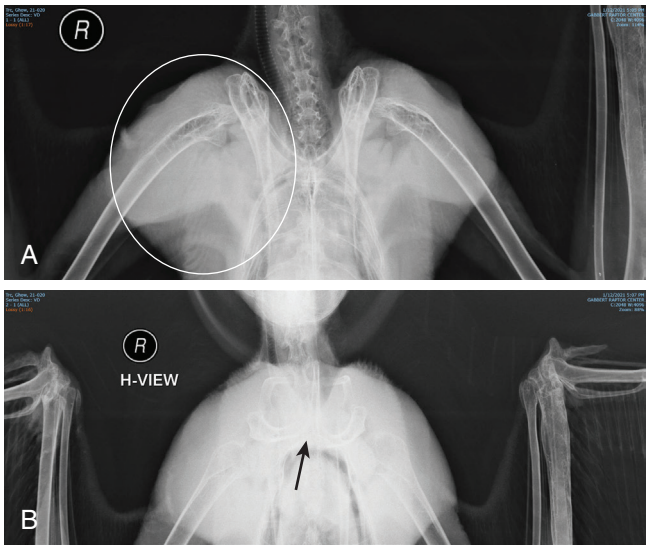


FIGURE 37.2 Radiographic images of an adult female great horned owl *(Bubo virginianus)* admitted to The Raptor Center at the University of Minnesota after being observed on the side of the road unable to fly. Image (A) is a ventral-dorsal radiograph taken on admit showing an apparently normal shoulder girdle. There is mild to moderate soft tissue swelling of the right shoulder *(white circle)*. Image (B) is an H-view taken on admit showing the right proximal coracoid luxation. Note the asymmetry where the coracoid bones meet the keel along the midline *(black arrow)*.

TABLE 37.2

Example of an Adaption of AOVET Fracture Classification System Using a Three-Digit Code

Bone	Location	Severity/Load Sharing Capacity
1 Humerus	1 Proximal	A Simple
2 Radius/ulna	2 Diaphyseal	B Wedge
3 Femur	3 Distal	C Complex
4 Tibiotarsus		
5 Metacarpus		
6 Metatarsus		
7 Coracoid		
8 Scapula		
9 Clavicle		

AOVET, is branch of the AO Foundation, which was originally named for a Swiss study group, *Arbeitsgemeinschaft für Osteosynthesefragen* (Association of the Study of Internal Fixation).



FIGURE 37.3 (A) Radiograph of a great horned owl *(Bubo virginianus)* with a transverse midshaft humeral fracture. Fracture classification based on bone (1: humerus), location (2: mid-diaphyseal), and complexity (A: simple transverse): 12A. (B) Radiograph of a great horned owl with comminuted radial/ulnar fractures. Fracture classification based on bone (2: radius/ulna), location (2: mid-diaphyseal), and complexity (C: comminuted): 22C.

to occur if a fracture is closer to the joint than 1.5 times the diameter of the bone.[24] The location of the fracture relative to muscle/tendon attachment/insertion sites will change the types and degree of forces applied to that fracture site and may change treatment decisions. Additionally, it may be hard to build adequate fixation in situations with small fragments and strong muscle attachments, requiring creative construct development to meet the goal of early and complete rigidity.

While there is a minimal force required to break a bone, the avian patient may experience much greater forces than this during a traumatic event. Fractures resulting from high-energy impacts are more likely to be highly comminuted with more devastating soft tissue compromise than those associated with low-energy events as energy is transferred to and absorbed by the patient's tissues. The forces experienced when a free-flying raptor is hit by a moving car are likely much greater than a companion bird flying into a stationary object (although malnutrition and poor bone integrity may complicate that latter situation). These high-energy fractures are more difficult to treat, slower to heal, and carry a poorer prognosis for complete recovery. Once again, successful outcomes are more likely to be achieved with strong adherence to the fundamental goals of early and adequate rigidity while minimizing additional soft tissue damage.

Tools in the Toolbox

A major goal in fracture treatment is to capitalize on the body's ability to heal the bone while minimizing complications. In planning the clinician's approach to fracture management, decision-making should be based on the ability to meet the following objectives, which inform best practices[2,7,23]:

- Establish early and effective rigidity
- Maintain anatomical alignment longitudinally and axially, as well as bone length
- Return limb to normal function and range of motion as quickly as possible
- Preserve and protect soft tissues
- Where possible, provide load sharing between the fixation device and the bone
- Reduce morbidity and prevent iatrogenic damage
- Promote patient mobility and comfort

After initial patient stabilization, the clinician should make an early decision whether the definitive treatment will be surgical intervention or conservative management. A fracture is considered stabilized when the forces of bending, rotation, shear, tension, and axial compression are neutralized. The ability to achieve most of the orthopedic objectives listed earlier in any fracture case positively correlates with the robustness of the treatment approach. The level of robustness required for maximum healing potential is dependent on the load sharing capacity of the bone, the construction of the stabilization device, and the integrity of the materials; the fixation must have the strength, duration, and capacity to counteract the forces of bending, rotation, and compression. A highly comminuted fracture in a weight-bearing limb of a large-bodied bird requires a fixation device with much more integrity and stability than a simple transverse fracture in a small bird. While this concept may seem intuitively obvious, it is fundamental to achieving a successful outcome. Failure to establish early, rigid stabilization with adequate fixation leads to a host of complications including delayed healing, nonunions, and fracture disease (compromised limb function).

Treatment options for fracture management vary in their ability to resist these forces. In order of increasing robustness, treatment options include cage rest, external coaptation, tape splint, IM pin, type I ESF, type II ESF, and the TIF (Table 37.3).

Cage rest

Very few avian fractures have satisfactory outcomes with just cage rest, which is mostly used as a supplement to coaptation or surgical fixation. Pelvic, spinal, and shoulder girdle fractures on small birds may do well with just cage rest. Management tools to enhance cage rest include reduced lighting to decrease activity and smooth-sided enclosures with removal of perches to prevent climbing.

TABLE 37.3

Tools for Managing Fractures

Cage rest/ confinement	◆ Very small birds ◆ Injuries not amenable to bandaging/ surgery (pelvic fractures)
Body wrap	◆ Initial or temporary fracture immobilization of wing fractures ◆ Coracoid/shoulder girdle fractures/ luxations ◆ Supplementary to wing wrap, figure-of-eight, or curved-edge splint
Wing wrap	◆ Initial or temporary fracture immobilization of wing fractures ◆ Nondisplaced wing fractures
Figure-of-eight	◆ Initial or temporary fracture immobilization of antebrachial fractures ◆ Immobilization of elbow and distal wing
Curved-edge splint	◆ Initial or temporary fracture immobilization ◆ Metacarpal fractures
Tape splints (Altman)	◆ Initial or temporary fracture immobilization ◆ Tibiotarsal fractures (birds <300 g) ◆ Metatarsal fractures (birds <300 g) ◆ Digit fractures (birds <300 g) ◆ Intertarsal joint luxations (birds <300 g)
Metatarsal and ball bandages	◆ Initial or temporary fracture immobilization ◆ Metatarsal fractures (birds <300 g) ◆ Digit fractures (birds <300 g)
Intramedullary pin	◆ Radius fracture ◆ Small bird long bones ◆ Immature birds
Type I ESF	◆ Ulna fractures ◆ Metacarpal fractures
Type II ESF	◆ Distal tibiotarsal fractures ◆ Tarsometatarsal fractures
TIF	◆ Humerus ◆ Ulna ◆ Femur ◆ Tibiotarsus
FESSA system	◆ Used as external bar for type I ESF, TIF ◆ Requires excellent alignment
Circular fixators	◆ Tibiotarsal ◆ Tarsometatarsal ◆ Distraction osteogenesis to increase bone length
Bone plates[a]	◆ Humerus ◆ Femoral ◆ Tibiotarsal ◆ Coracoid

[a]Primarily reported as single case studies.
ESF, External skeletal fixator; *FESSA,* Fixateur Externe du Service de Santé des Armées; *TIF,* tie-in-in fixator.

Coaptation

Bandaging techniques are very useful for temporary fixation to prevent additional trauma and pain prior to definitive treatment. They often result in poorer outcomes and complications when used alone as bandaging is often bulky and requires prolonged immobilization of joints. As a definitive fixation tool, coaptation is best suited for very small birds, nondisplaced fractures, shoulder girdle fractures, phalangeal fractures, and single bone fractures of the antebrachium. Coaptation may be a good option in situations where full return to function is not required, when surgical risk is too great, or when there is minimal displacement, and owners are not willing to spend the money for surgery. Commonly used bandaging techniques include body wrap (Figure 37.4), wing wrap, figure-of-eight, modified figure-of-eight (Figure 37.5), tibiotarsal tape splints (Figure 37.6), and foot wraps/ball bandages.[20,25] Curved-edge splints are a good choice for many metacarpal fractures and tape splints may be used for definitive fixation for lower-leg fractures of smaller birds (Figure 37.7).

Surgical fracture management

A variety of devices and approaches can be applied surgically and used to provide definitive fixation. An IM pin is relatively easy to insert into a long bone and provides excellent opposition to bending forces. Because of its inability to resist torsional, compressive, and tensile forces, the IM pin is typically combined with coaptation for increased stability. On the other hand, type I, II, and III ESF devices are very good at resisting rotational, compressive, and tensile forces, while having moderate capacity to oppose bending forces (Figure 37.8). The TIF combines these two approaches to create a very versatile and effective system for long-bone fixation (Figure 37.9). The TIF construct consists of an IM pin, two or more ESF pins, and an external connecting bar that integrates these components into one unit, resulting in a robust device that resists all relevant forces. The external connecting bar can be created from acrylic (Technovit; Jorvet, Loveland, CO; Acrylix; IMEX, Dallas, TX, USA) using a Penrose drain or small plastic tubing as a mold or epoxy putty (Epoxy ESF Putty; Jorvet, Loveland, CO, USA).[7,20] In addition, the Fixateur Externe du Service de Santé des Armées (FESSA) system is commercially available and has been used successfully in both ESF and TIF styles.[26,27] Plates and nails have been used with varying degrees of success in repairing avian fractures with available information deriving primarily from individual case reports, including one in which a locking compression plate was applied externally and used as an ESF connector bar.[28] Limitations include lack of suitably sized plates and nails to accommodate the wide range of bone sizes in avian patients, lack of strength of the small plates to accommodate applied forces, morbidity associated with their placement, and expense.[20] More recently, adaptations of circular fixators have been used for definitive fixation as well as scaffolding for distraction osteogenesis to reestablish long-bone length.[29–31]

The robustness of each of these methods can be enhanced by clinical choices in application. For instance, while the materials and design used in a bandage or a splint will determine its ability to resist movement at the fracture site, these tools inherently reduce the patient's ability to use the limb during fracture healing. The surgically applied constructs are significantly stronger when applied correctly and meet the goal of allowing a quicker return to function. These devices can be reinforced by adding additional components. While an appropriately applied ESF requires a minimum of two pins in each fragment for stabilization of the fracture, adding additional ESF pins will increase the strength and rigidity of

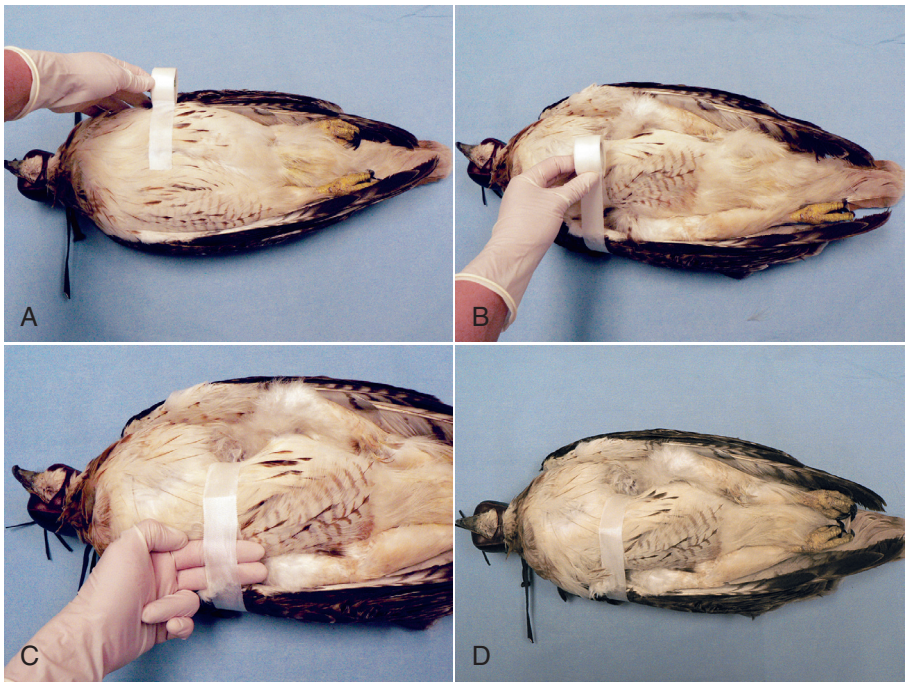


FIGURE 37.4 Application of a body wrap. Beginning mid-keel (A), the tape is passed away from the injured wing (B) and wrapped once around the body with the second wrap incorporating the injured wing (C). Final wrap should be snug, but not tight (D).

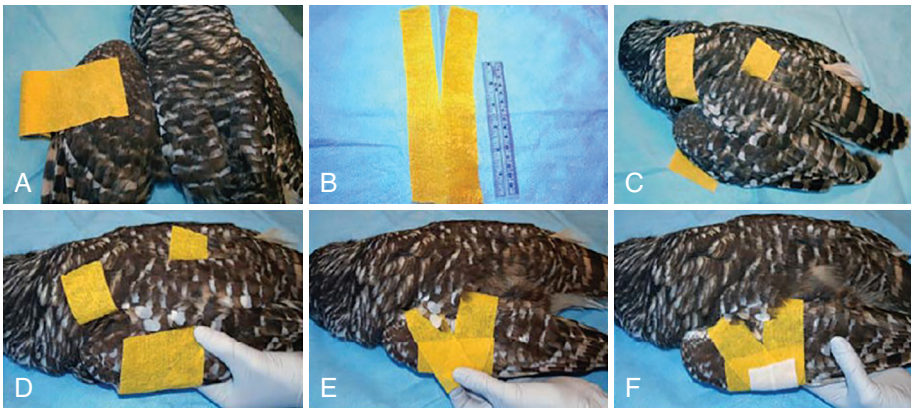


FIGURE 37.5 A modified figure-of-eight wrap. (A) Use a piece of self-adhesive bandage material wide enough to cover approximately two-thirds of the dorsal surface of the wing between the elbow and the wrist and long enough to overlap by 4–6 inches (10–15 cm) when wrapped around tucked wing. (B) Cut a slit in the self-adhesive bandage material that is parallel to the longest length of the bandage material. The slit should extend approximately half of the length of the bandage material. A ruler can be used to gauge the length of the slit if desired. The length of the slit should be approximately equal to the width of the wing from humerus to major metacarpal bone as measured on the dorsal surface of the wing when folded in a normal perching position. (C) Position the self-adhesive bandage material on the affected wing. One length of the cut bandage material is positioned in the axillary region proximal to the elbow joint and passing from the ventral to dorsal surface of the wing. The second length of cut bandage material is passed from the ventral surface of the wing, cranial/anterior to the humerus and over the patagium to rest on the dorsal surface of the wing. The uncut piece of self-adhesive bandage material is placed on the ventral surface of the antebrachium in the region of the radius/ulna, and is passed around the distal aspect of the wing over the major metacarpal bone onto the dorsal surface of the wing. (D) As the bandage is placed, tuck the wing into a normal perching position. (E) The bandage is secured in place by bringing the cut pieces of the bandage material together to join the single intact piece of self-adhesive bandage material on the dorsal surface of the wing and the bandage material is adhered together to secure the wing in place. (F) A piece of tape can be used to help secure the bandage. When appropriately placed, this modified figure of 8 bandage is able to immobilize both the elbow and the wrist joint, and is conceptually easier for some practitioners to place.



FIGURE 37.6 Application of a tape splint to a tibiotarsus/tarsometatarsus. After preparing the leg (A), place a light gauze wrap around the leg (B) and sandwich between two pieces of tape, ensuring no loose gauze strands are exposed (C, D). Using a hemostat, crimp the tape snug to the leg (E) and trim the excess tape (F). Final splint may be hardened with a coating of cyanoacrylate. (Images courtesy Y.R.A. van Zeeland.)

the device. Similarly, although a TIF requires only two ESF pins in its construction, additional ESF pins will add to its robustness, as will using a larger external bar for the tie-in.[32] The limitation to these choices is the ability of the bird to handle the size and weight of the fixation construct. Meeting both the objectives related to robust fixation and the goal of permitting patient mobility, comfort, and maximum range of motion for joints during healing often requires a clinical decision of surgical fixation (Figures 37.10 and 37.11).

While conservative management is best practice for some fractures such as shoulder girdle or pelvic fractures, for others (e.g., humeral fractures) a requirement for normal flight necessitates surgical fixation. In many situations, however, either coaptation or surgical fixation may be a practical option, a decision that requires thoughtful consideration. What are the goals for the patient? Is its long-term living environment (release to the wild, exhibit, or pet) determinant in the decision? Does the patient require return to full function? Are there future concerns for additional problems or

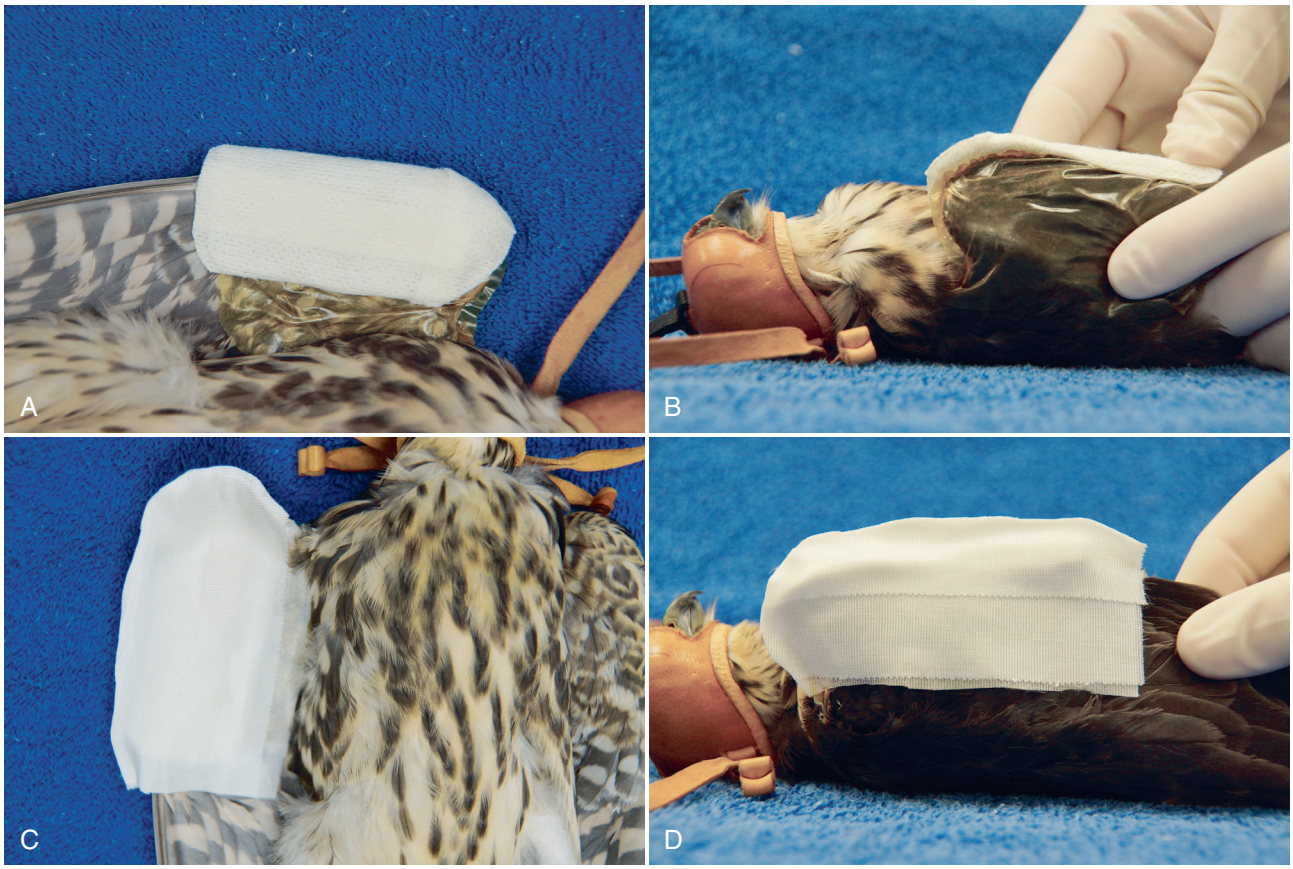


FIGURE 37.7 Curved-edge splint made from moldable thermoplastic. A piece of thermoplastic is cut to the length of the metacarpus and molded to the ventral aspect of the bone (A) with a right-angle bend covering the leading edge of the bone (B). The splint is sandwiched in place using overlapping pieces of adhesive tape applied ventrally (C) and dorsally (D).

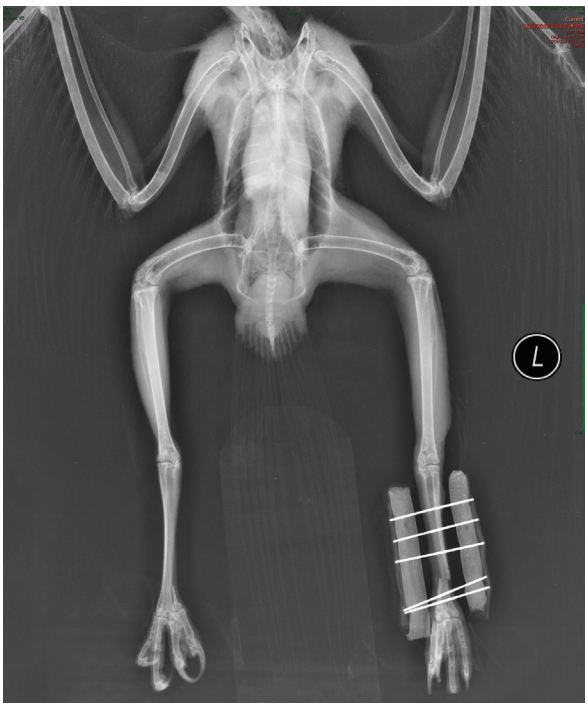


FIGURE 37.8 Type II external skeletal fixator (ESF). A type II ESF applied to the tarsometatarsus of a broad-winged hawk.

complications? What are the resources available for treatment, including financial and equipment resources, clinical knowledge and experience, and surgical skills in avian fracture management?[20] For many patients, the question of whether there are adequate postoperative nursing care and rehabilitation facilities is important to consider.

The question of limb amputation as a salvage procedure and the ramifications of that decision can be complex. As previously noted, some avian groups such as psittacine species adapt far better to limb amputations than others. Characteristics such as species, biology, husbandry, and behavior all influence this decision. With wild birds, the options for amputation are limited in the United States; the Code of Federal Regulations proscribes euthanasia for any wild bird requiring an amputation of wing above the elbow or any part of a leg or foot other than a toe. A waiver can be requested by a veterinarian for wild birds being maintained in permanent captivity, although prior to requesting this waiver, it is critical that the veterinarian understands the ramifications of the decision in that species and the implications to the specific patient's welfare and is not basing the decision on whether or not the surgery can technically be done.

Triage and Treatment

Upon presentation for traumatic injury, the patient should be evaluated completely, including signalment, history, physical

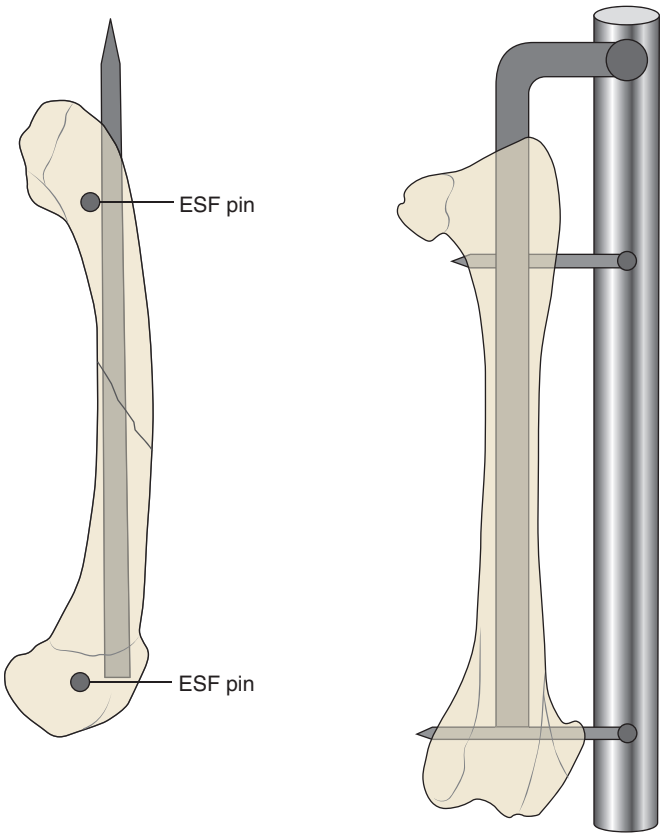


FIGURE 37.9 Diagram of a tie-in fixator (TIF), which uses an acrylic bar to connect an intramedullary (IM) pin with a type I external skeletal fixator (ESF), applied to a femur.

examination, orthopedic assessment, and radiography with stabilization of the patient and its immediate critical care concerns being prioritized, including supportive care and initiation of medications for antibiosis and analgesia (see also Chapters 34 and 35). In the earliest stages of diagnosis and treatment, the long-term prognosis for wild birds should be evaluated as quickly as possible to avoid unnecessary suffering when the prognosis is poor. As most avian fracture presentations are emergency situations or acute traumas, these patients must first and foremost be considered critical care patients. While fracture stabilization and soft tissue care are essential to minimize morbidity, careful consideration must be given to what the patient can withstand upon initial presentation. Preserving the patient's life is the principal consideration. A holistic approach should be used, addressing immediate critical care concerns, assessing the overall health status of the patient, and constantly evaluating the patient's condition and ability to withstand additional handling.

There are some actions, however, that—if taken early—will set the stage for successful outcomes. Attention to the preservation of soft tissue and bone during preliminary treatment of the avian patient is a key component of optimizing healing potential. Many avian long-bone fractures are open due to the paucity of soft tissues. Debridement of wounds should be performed without excessive manipulation to preserve soft tissues and avoid compromising circulation to bone fragments. Protection of the bone ends protruding through the skin should be done as quickly as possible through coverage with a water-based, water-soluble lubricant (K-Y Jelly; Reckitt Benckiser, NJ) or saline-moistened gauze as the first bandage layer (no direct contact between dry gauze and bone;

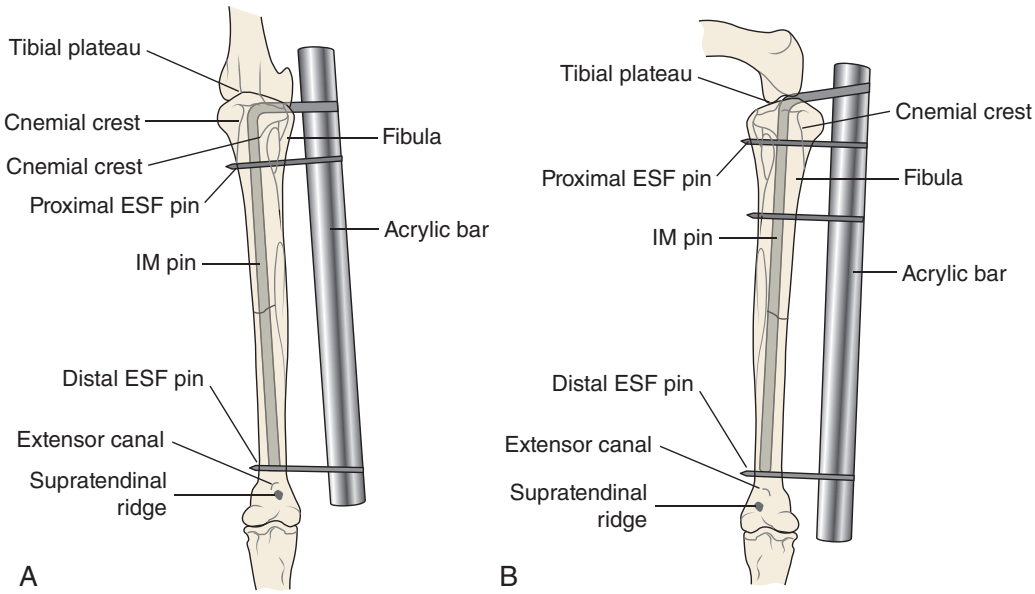


FIGURE 37.10 The robustness of an external skeletal fixator (ESF) or a tie-in fixator (TIF) can be increased by the addition of ESF pins. A standard TIF with one ESF pin in each fragment (A) is strengthened by adding additional ESF pins in one or more fragments. (B) Note, the IM inserts through the tibial plateau with the stifle in normal standing flexion and does not enter the femur.



FIGURE 37.11 Radiographic example of adding additional robustness with extra external skeletal fixator (ESF) pins on bilateral bald eagle *(Haliaeetus leucocephalus)* tibiotarsal fractures.



FIGURE 37.12 An open fracture with exposed bone showing loss of soft tissue attachments, darkened bone color, and a demarcation line reflecting loss of viability.

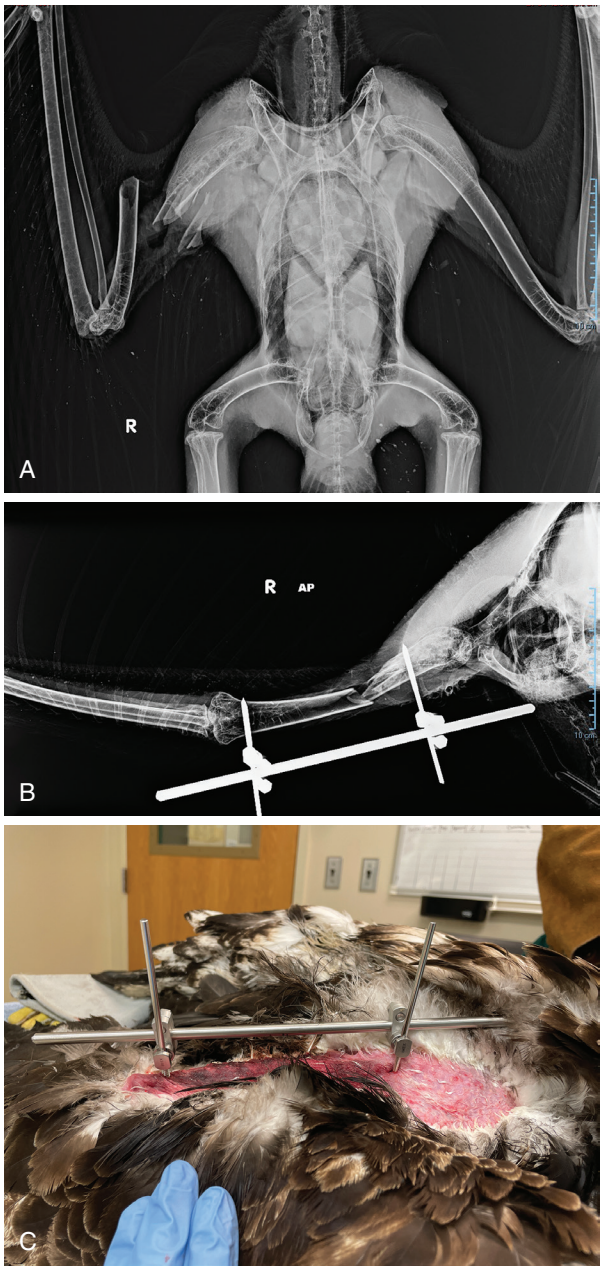


FIGURE 37.13 Use of a type I external skeletal fixator (ESF) as a temporary fixator on a bald eagle *(Haliaeetus leucocephalus).* (A) Intake radiograph showing a comminuted, mid-diaphyseal humeral fracture. (B) Caudal-cranial radiograph taken after placement of the temporary type I ESF and (C) image of fixation in place on the patient.

see Chapter 36). The treatment of the exposed bone ends will prevent this tissue from becoming devitalized and preserve bone integrity. Especially in wild birds, exposed bone is often already nonviable (Figure 37.12) and this should be taken into triage consideration as loss of long-bone length can impair flight mechanics, preventing full restoration of flight and releasability.

Stabilizing the fracture often occurs in two stages—temporary stabilization upon presentation of the patient with definitive repair after stabilization and full assessment of the patient has occurred. Preliminary treatment of orthopedic trauma should include reduction and stabilization of fractures and luxation. Coaptation methods such as body wraps, wing wraps, and leg splints are important presurgically to stabilize the fracture site and minimize additional soft tissue damage. Temporary stabilization with a type I ESF (Figure 37.13) is another option that can be applied relatively quickly in the unstable traumatized patient to protect the fracture site until the patient is stable enough for surgery. The two-pin, type I ESF helps prevent muscle contracture that can be challenging to overcome when

surgery is delayed and can help prevent the fracture from reopening in the presurgical period. If the ESF pins are well placed and still well seated at the time of surgery, they can be incorporated into the final fixation when using a TIF or ESF.

APPROACHES TO FRACTURE MANAGEMENT WITH SPECIES CONSIDERATIONS (TABLE 37.4)

Pectoral Limb

Shoulder girdle (Figure 37.14)

The coracoid, a key structural element in the avian shoulder girdle, is often fractured or luxated because of a frontal collision with an object. The force required to break this bone is significant, so the clinician should look carefully for concurrent trauma elsewhere. A key component in determining prognosis is the integrity of the shoulder joint. Despite sometimes dramatic displacement of the shoulder joint secondary to trauma, most birds will have a good prognosis if the actual joint is intact. If the fracture (or luxation) involves the joint, the prognosis for return to flight is poor (Figure 37.15).

TABLE 37.4

Management Techniques by Bone in Order of Decreasing Strength and Robustness

Bone	Temporary Stabilization	Definitive Fixation
Coracoid	Body wrap Cage rest	Body wrap Cage rest
Humerus	Type I ESF (two ESF pins) Body wrap	Tie-in fixator IM pin Coaptation[a]
Ulna	Figure-of-eight bandage Modified figure-of-eight wrap Body wrap	Tie-in fixator Type I ESF (four or more ESF pins) IM pin Coaptation (if radius intact)
Radius	Figure-of-eight bandage Modified figure-of-eight wrap Body wrap	IM pin Coaptation (if ulna intact)
Metacarpus	Splint Figure-of-eight wrap with body wrap	Curved-edge splint Type I fixator (four or more ESF pins)
Femur	Cage rest (well-padded)	Tie-in fixator IM pin Splint/cage rest
Tibiotarsus	ESF-IM tie-in, type II fixator	Tie-in fixator Type II fixator Tape splint
Metatarsus	Padded Splint	Type II ESF Tape splint
Phalanges	Coaptation Splint Ball bandage	Splint Ball bandage Thermoplastic/Styrofoam shoe

[a]Not generally recommended by authors for definitive fixation.
ESF, External skeletal fixators; *IM*, intramedullary.

While there are reports of successful surgical fixation of the coracoid in larger birds such as eagles, conservative management has been associated with an excellent prognosis for successful outcome in all sized birds and is the recommended approach.[7,33,34] Conservative management consists of cage rest combined with limiting wing motion on the affected side when possible. In raptors, successful outcomes are consistently achieved using a body wrap for 2 to 3 weeks with twice weekly physical therapy, and antiinflammatory and joint support medications.

Humerus (Figure 37.16)

Humeral fractures most often occur in the middle third of the diaphysis, frequently in the area where the muscle mass tapers off into more tendinous structures. A key characteristic influencing the approach to treatment is that the powerful flight muscles that attach to the proximal end of the humerus provide a formidable distraction force, resulting in fractures that are rarely even remotely aligned (unless partial or greenstick). Most commonly oblique, these fractures are frequently open with the sharp end of the proximal fragment projecting through the skin on the dorsal surface of the wing and the distal fragment exposed on the ventral surface. The radial nerve, which crosses from caudal to cranial in the midshaft portion of the bone, is often close to the fracture site and must be preserved during treatment. Because of the marked fragment distraction associated with humeral fractures, they almost invariably require a surgical approach and internal fixation if the bird is to return to normal function and flight. If treated with conservative management (coaptation), the outcome is, at best, a misaligned fracture and often a nonunion due to the inability to adequately stabilize the fracture with coaptation alone due to the strong forces applied by the proximal flight musculature.

Coaptation is important in the presurgical period. While the recommendation to use a figure-of-eight bandage on humeral fractures can be found in the literature, this technique is inadequate alone as it increases the distractive forces at the fracture site. A body wrap is simpler to apply, adequately stabilizes the joints above and below the fracture, and can be effective for short-term stabilization prior to surgical fixation. A TIF is well suited for definitive fixation of most humeral fractures. While it is an advantage that the pectoral limb does not need to be weight-bearing during healing, there are times when extra ESF pins are recommended to strengthen the construct (e.g., large birds, highly comminuted fractures). In smaller birds with uncomplicated fractures, a simple IM pin combined with external coaptation may be adequate. With extremely proximal or distal (within two to three bone diameters of condyles) humeral fractures, the short fragment may not provide enough purchase for an IM pin, necessitating that the surgeon employ a more involved construct such as a cross-pin technique or a tension band approach.[7]

Antebrachium (Figure 37.17)

The approach to treating fractures of either the radius or the ulna depends on the integrity of the other bone as well as the long-term objectives. External coaptation may provide satisfactory results, especially in small birds, if only one antebrachial bone is fractured. While a figure-of-eight bandage

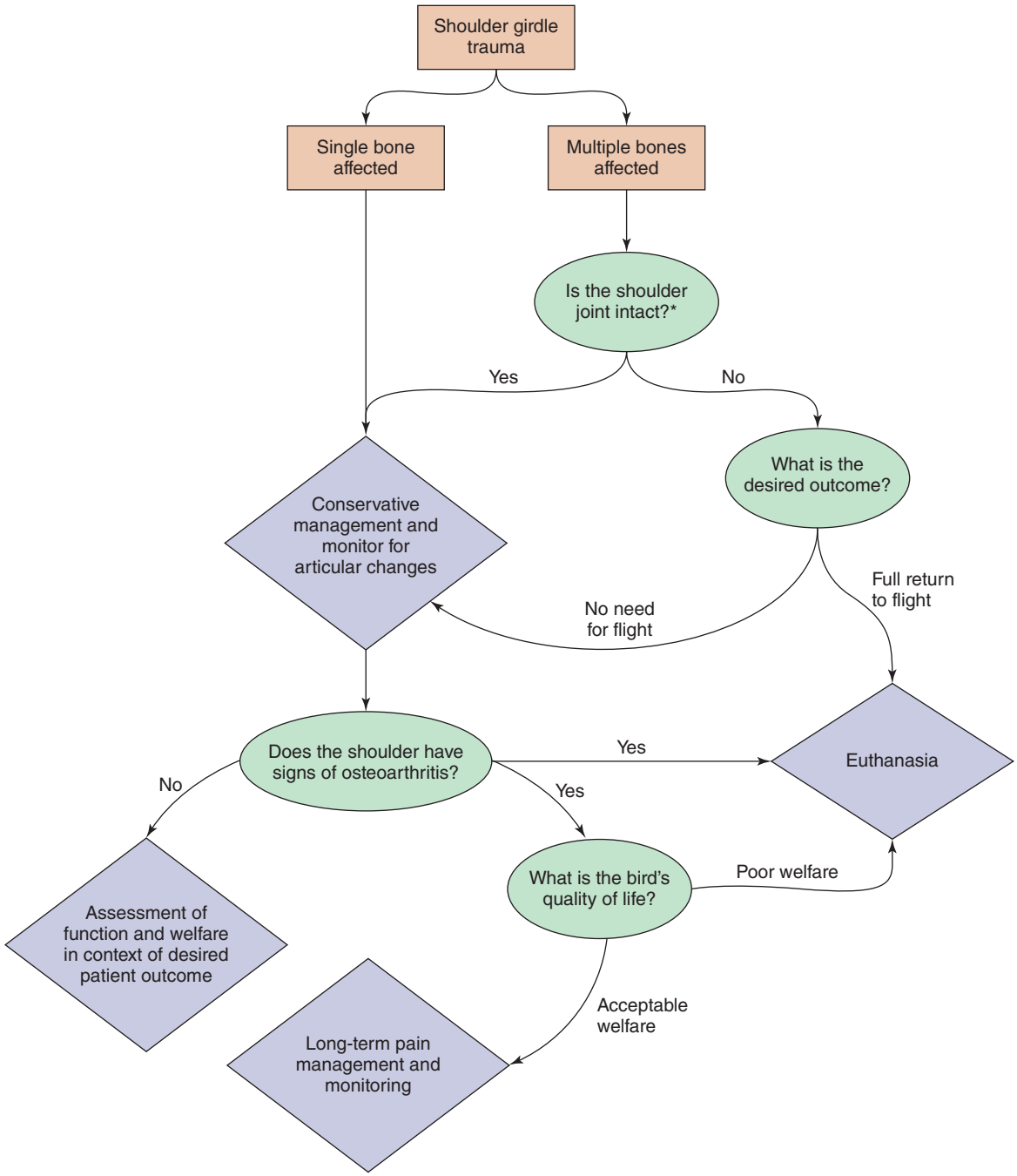


FIGURE 37.14 Decision tree to help guide the avian practitioner through the clinical decision-making process when presented with a bird with shoulder girdle trauma.

provides relatively good stabilization in this situation, its use carries a risk of complications such as reduced range of motion or patagial contracture.[2,20] These risks have been reduced by the use of a modified figure-of-eight bandage. In fractures involving one bone in the distal third of the antebrachium, the risk of synostosis developing can be reduced with a surgical approach utilizing internal fixation (IM pin in radius, TIF on ulna). In raptors, with an ulna fracture and intact radius, both conservative and surgical treatment approaches have been shown to be effective, with conservative management being used for chronic fractures with good alignment.[12]

When both antebrachial bones are fractured, some amount of internal fixation is typically required for return to normal function. Inserting an IM pin into the radius may be adequate with coaptation if the ulna is minimally displaced. However, the relatively small diameter of the radius necessitates a small diameter IM pin, which is often not rigid enough to provide adequate support even with additional coaptation when both the radius and ulna are fractured. Surgical fixation of the ulna in these situations, using a TIF, is still recommended if possible as it will result in greater integrity of the fixation and shorter healing times.[7]

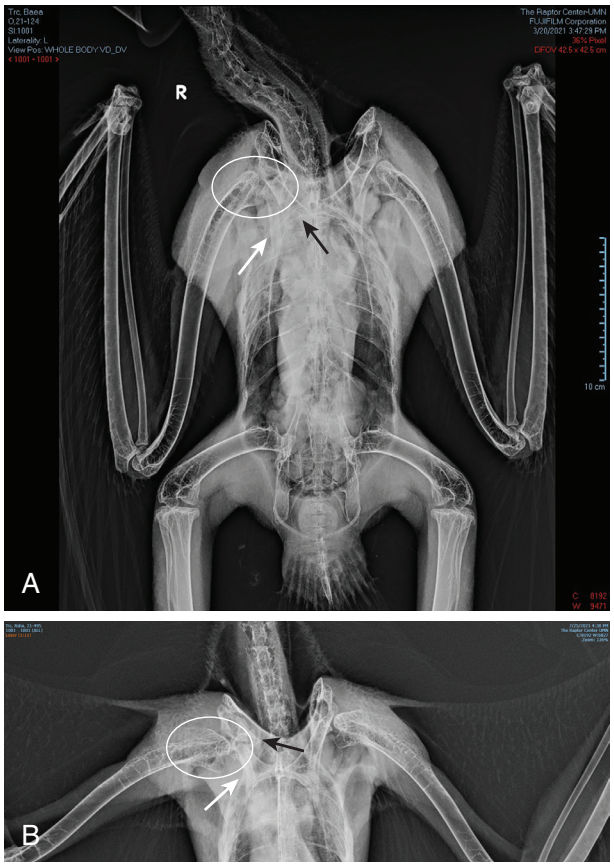


FIGURE 37.15 Radiographic image of a bald eagle (*Haliaeetus leucocephalus*) with a proximal right coracoid luxation (*black arrow*), a right scapula luxation (*white arrow*)—the scapula is displaced caudally relative to the normal right side, and a right clavicular fracture. (A) Note that the orientation of the articular surfaces of the humeral head relative to the scapular head has been preserved *(white circle)*. (B) Radiograph of a red-shouldered hawk *(Buteo lineatus)* with a transverse mid-diaphyseal right coracoid fracture *(black arrow)*, and a right scapula luxation *(white arrow)*. Note that the orientation of the articular surfaces of the humeral head relative to the scapular head has changed *(white circle)*.

Carpus/metacarpus (Figure 37.18)

Especially in wild birds, carpal trauma is often high energy with fractures of the major metacarpal having significant comminution. With even less soft tissue than other areas, open fractures and luxations in the carpal/metacarpal area can be challenging. In one study on metacarpal fractures in raptors, management with a moldable (curved-edge) splint supplemented with coaptation was shown to have good outcomes with relatively quick healing (21 days).[35] The authors have had good success with both curved-edge splints and type I ESF fixation. Carpal luxation has the best prognosis when stabilized after reduction with a type I ESF.[7] Chronic luxations are very difficult to reduce and often lead to reduced range of motion or ankylosis.[36] Whether stabilizing with a curved-edge splint or a fixation device, it is important to provide additional support with a body wrap when applying any weight to the distal wing.

Pelvic Limb (Figure 37.19)

Pelvis

Fractures of the pelvic girdle are most common in wild birds and often associated with car strikes or other major trauma. Frequently, there are multiple bones involved due to the high level of trauma. Treatment is cage rest with close attention to neurological integrity for prognosis. Involvement of the caudal synsacrum may result in abnormal tail position. In gulls (Laridae), fractures of the vertebral column caudal to the acetabulum carry a poor prognosis for return to flight. The successful use of a tail splint has been reported by taping the primary and secondary flight feathers of one or both wings together, then taping the tail to the wing tips.[37]

Femur

The avian femur is closely associated with the body wall, which provides substantial support. Because of this, femur fractures are less common than other leg fractures in birds. Stabilization with coaptation can be difficult, although the use of a spica splint has been recommended.[7] In almost all species, a TIF can be used with materials scaled as needed for the size of the bird. Smaller birds may do well with a simple IM pin for fixation, while in very small birds, a K-wire can be used as an IM pin. While malalignments are common, small birds may have successful outcomes with simple cage rest. In small-bodied altricial chicks, the femur may achieve adult length within the first week of life. In these birds, the incorporation of ESF pins to secure rotational stability may not interfere with growth. Chick bones may be amenable to piercing by hypodermic needles to use as ESF pins. Passerine femurs will typically accept a 25- or 27-gauge (0.020 inches, 0.5 mm or 0.0164 inches, 0.42 mm) 1.5 inches (3.8 cm) needle as an IM pin with the same size needles as ESF pins. A small amount of epoxy putty on a thin piece of wooden coffee stir stick works well as an ESF. Pinning these tiny fractures prevents the chick from growing up with a permanent disability.

Tibiotarsus

The tibiotarsus is among the most fractured bones in birds, being especially common in both pet birds and falconry birds. In psittacine species, tibiotarsal fractures occur most frequently in the mid to distal portions of the diaphysis while in raptors, they are mostly found in the proximal third of the bone. These fractures are generally closed and carry a relatively good prognosis. External coaptation is recommended for smaller birds weighing less than 300 g and tape splints were shown to have good outcomes in 92% of cases in one study (86 total cases).[2,20,38] For larger birds, surgical fixation is recommended, with Bueno and Redig reporting success outcomes in 84% of 37 cases following surgical reduction and the application of a TIF.[39]

Tarsometatarsus

There is tremendous variability in the confirmation of the tarsometatarsus among species, which is reflected in treatment approaches. For example, psittacine birds have a rounded tarsometatarsus, while raptors have a C-shaped bone with the powerful flexor tendons traveling in the plantar concavity. As with the tibiotarsus, tape splint fixation of tarsometatarsal fractures in smaller psittacines is generally associated with good outcomes. Larger psittacines and most raptors will benefit from

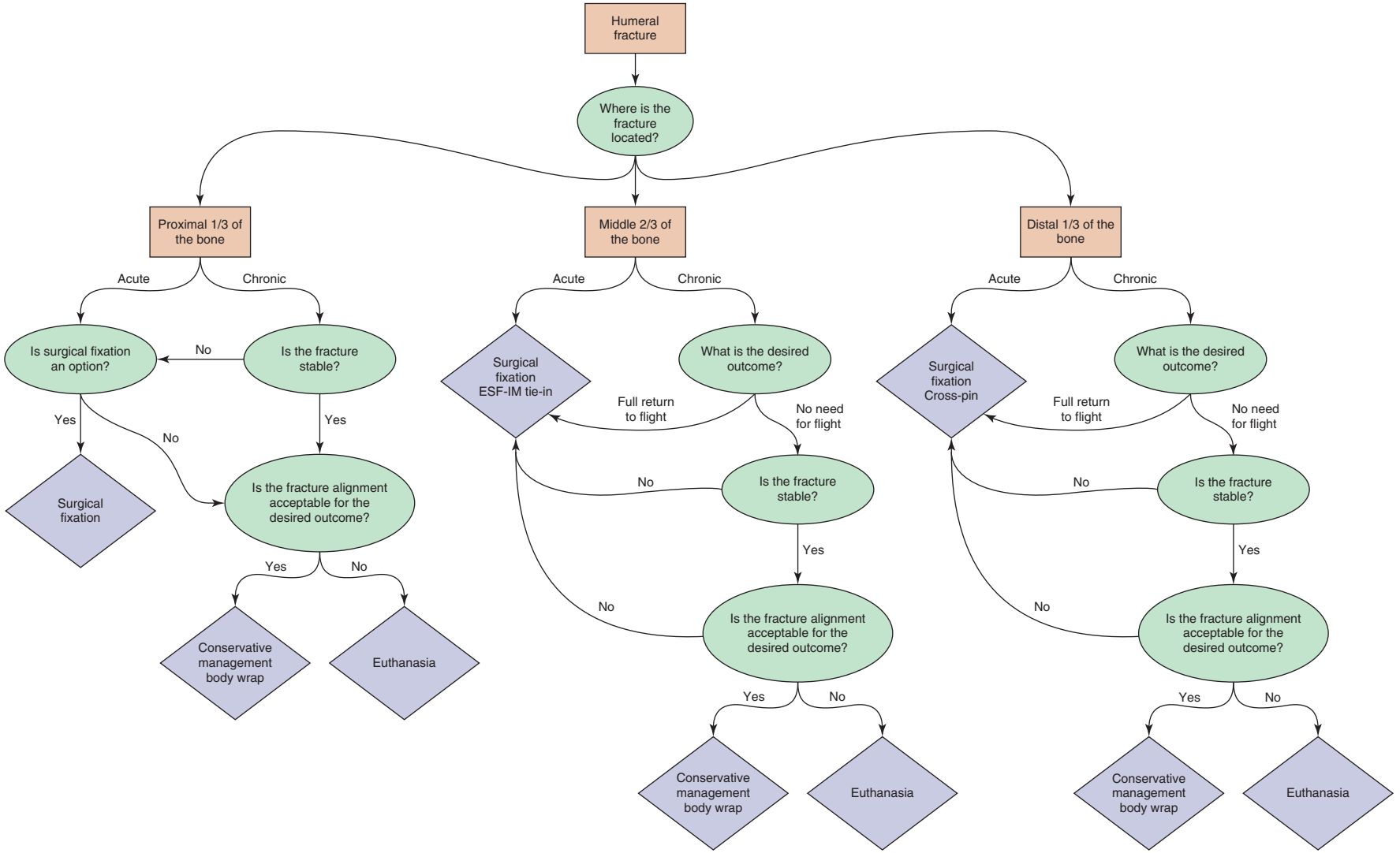


FIGURE 37.16 Decision tree to help guide the avian practitioner through the clinical decision-making process when presented with a bird with humeral trauma. When assessing if the fracture alignment is acceptable for the desired outcome, longitudinal and rotational alignment should be considered, as well as if there is a significant loss of bone length. *ESF*, External skeletal fixator; *IM*, intramedullary.

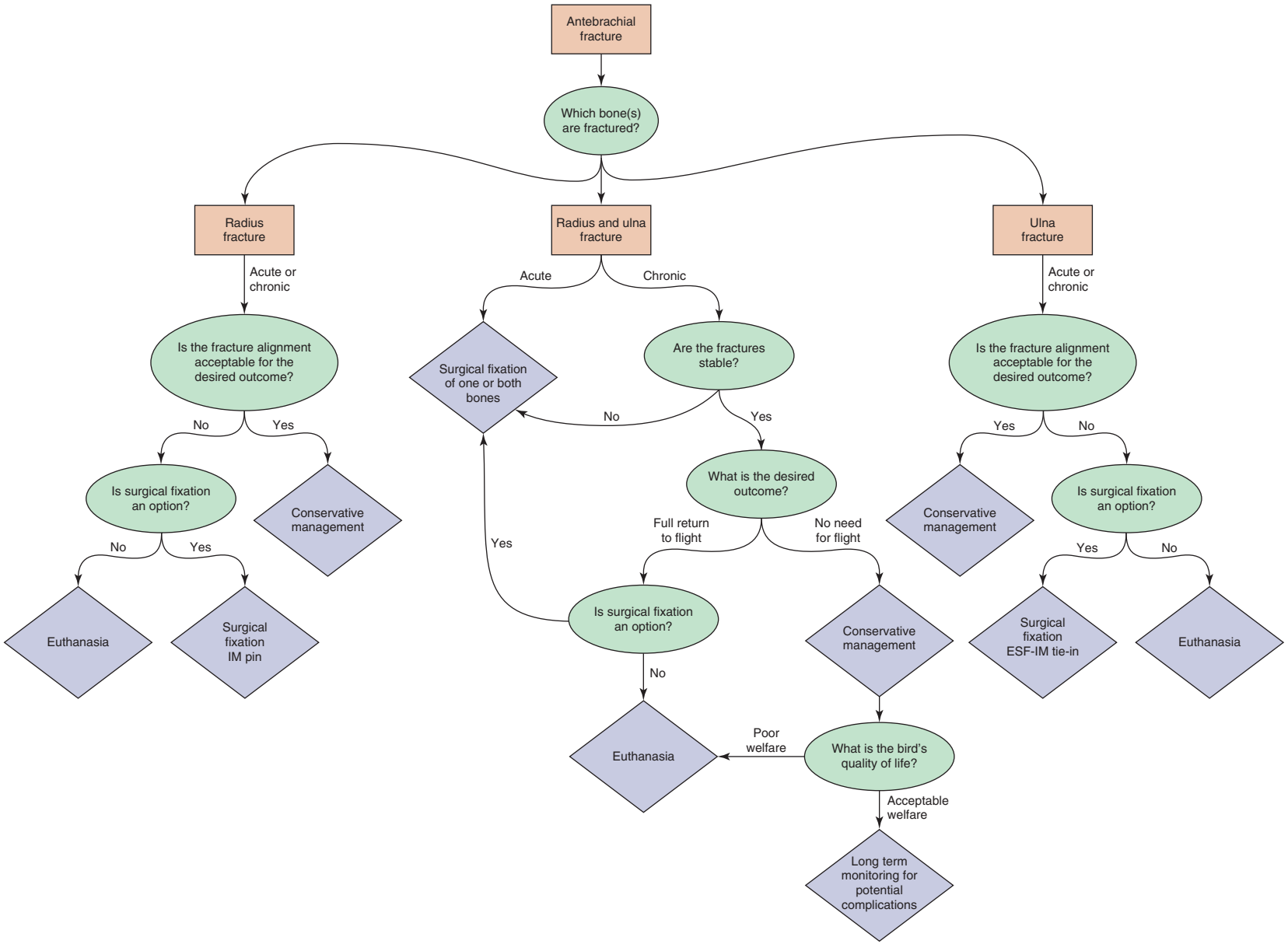


FIGURE 37.17 Decision tree to help guide the avian practitioner through the clinical decision-making process when presented with a bird with an antebrachial fracture. For a radius or ulna fracture where the other bone is intact, the fracture fragments need at least 50% alignment for the fracture to heal successfully with conservative management. *ESF*, External skeletal fixator; *IM*, intramedullary.

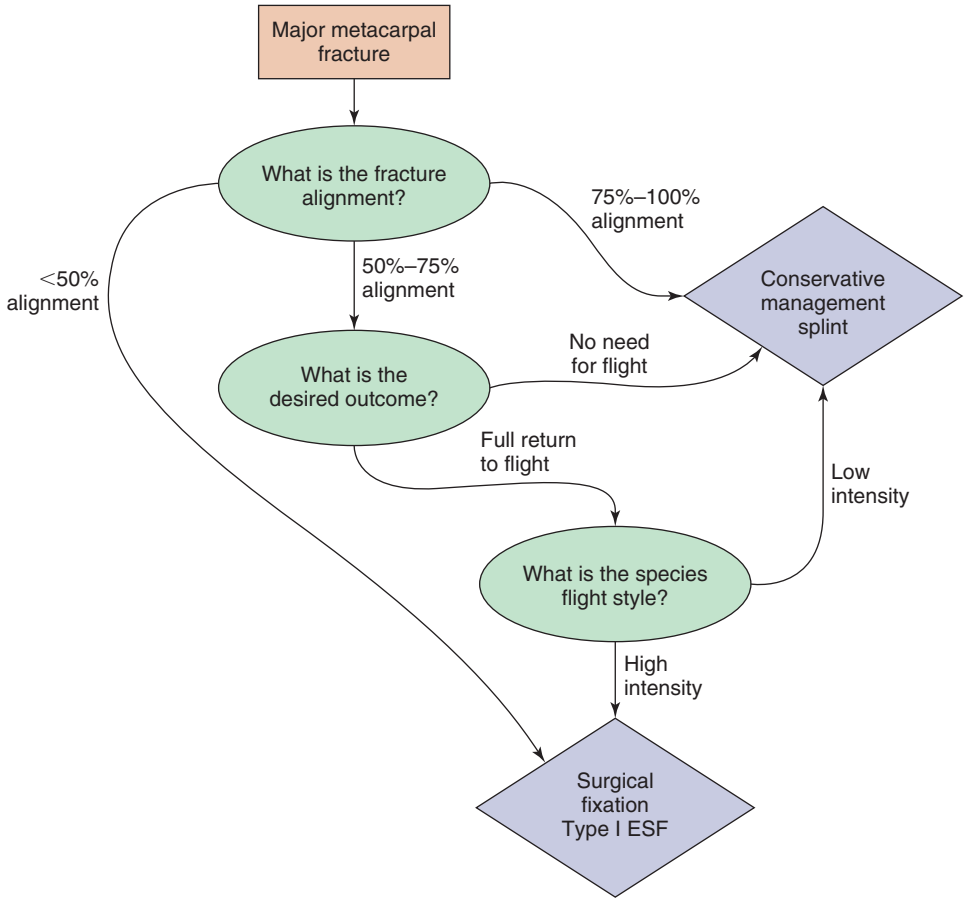


FIGURE 37.18 Decision tree to help guide the avian practitioner through the clinical decision-making process when presented with a bird with a major metacarpal fracture. *ESF,* External skeletal fixator.

surgical fixation utilizing a type II ESF as the placement of an IM pin is difficult in both groups. Another option is the placement of an L-shaped splint along the plantar side of the foot, leaving the toes to function freely and thus preventing immobilization of the flexor tendons during callous formation.

Galliformes, passerines, and rails have a tarsometatarsus that is amenable to stiff splints that provide rigid immobilization to fracture fragments without restraining adjacent joints; these splints can often be quite snug without risk of a tourniquet effect as the tarsometatarsus does not include much soft tissue in these species. Pelicans (Pelicanidae) and herons (Ardeidae), however, have enough muscle on this section of the leg that it does not allow for stiff coaptation to be applied due to risks of marked edema.[37] While raptors have primarily tendons in this area, the power of their flexor tendons limits the use of splints or coaptation for tarsometatarsal fractures.

Waterfowl are amenable to an external coaptation "boot" for tarsometatarsus fractures and have an excellent prognosis for rapid healing and release or full return to function even when the metatarsals have fractured free of each other or the tarsometatarsus (Figure 37.20).[40] Oblique metatarsal fractures may pierce the skin to become an open fracture underneath splints, but the simple application of a hydrocolloid bandage over the area prior to splinting often mitigates this concern. These boots allow the bird to walk, run, and swim while the fracture is healing. Restricting movement of the hock is not necessary if the fracture fragments are well restrained by the boot and external coaptation of the tarsometatarsus fragments. While oftentimes these splints will violate the principle of stabilizing joints above and below the fracture, evidence-based medicine demonstrates they often work.[40] The boot assists the bird in maintaining the foot in a normal walking position. Never splint a mallard *(Anas platyrhynchos)* or domestic goose *(Anser anser domesticus)* foot in a nonergonomic position as recovery to full function is much less likely; if the foot joint results in poor range of motion after external coaptation in a standing position, the bird will still be able to comfortably stand and walk.

Phalanges

Fractures of the digits usually respond well to conservative management in most species. In parrots, where trauma is often a result of inadequate grooming and long nails getting tangled in cage furnishings or other objects, cage rest is recommended if the fracture is closed. Immobilization of toes through splinting may result in formation of adhesions and reduced range of motion. If the fracture is open, as is often the case with bites from cage mates, there is a risk of osteomyelitis, and amputation has been recommended as a consideration. In other species, natural history and management should be considered prior to amputation. For instance, amputation of the hallux in

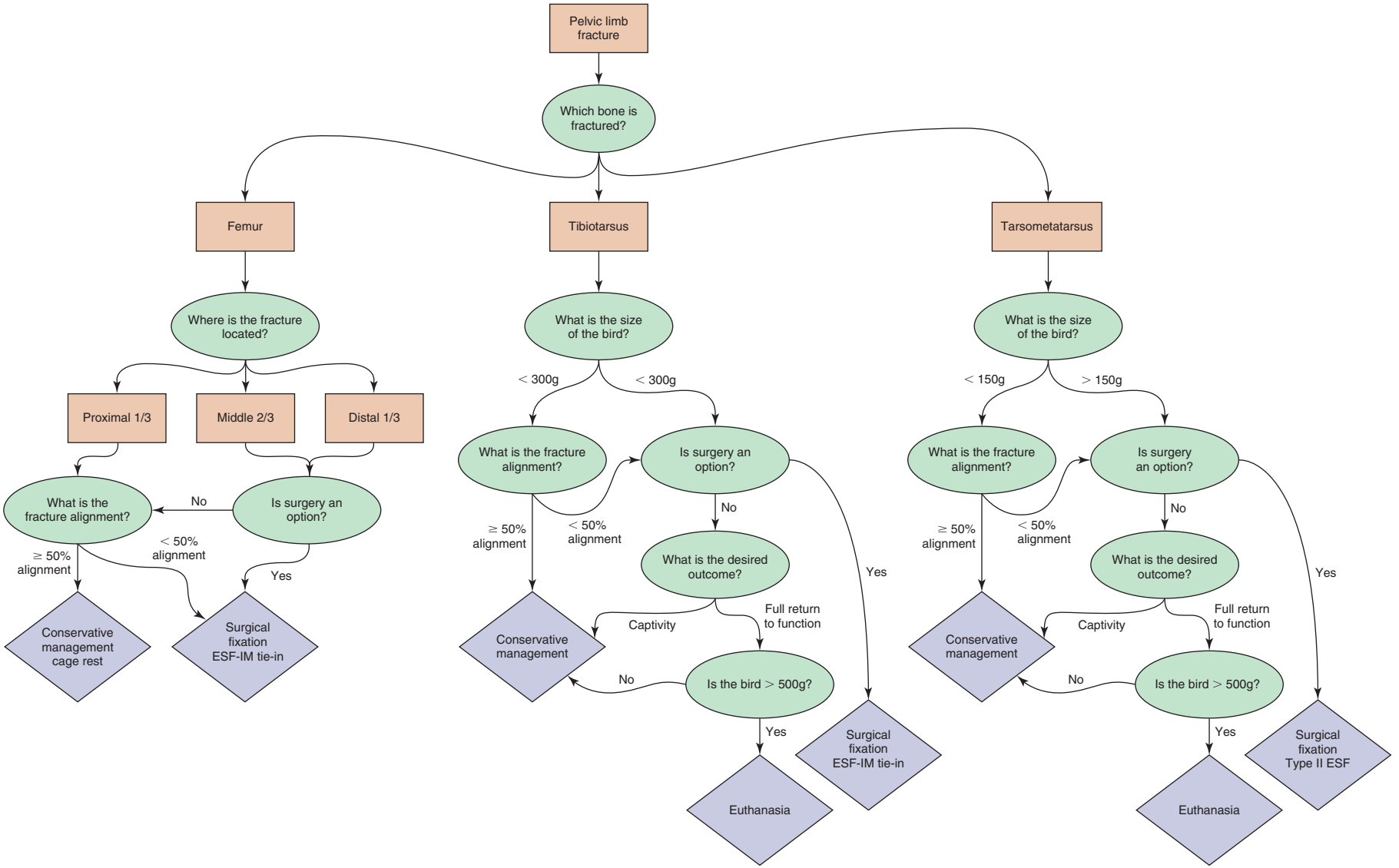


FIGURE 37.19 Decision tree to help guide the avian practitioner through the clinical decision-making process when presented with a bird with a pelvic limb fracture. *ESF*, External skeletal fixator; *IM*, intramedullary.

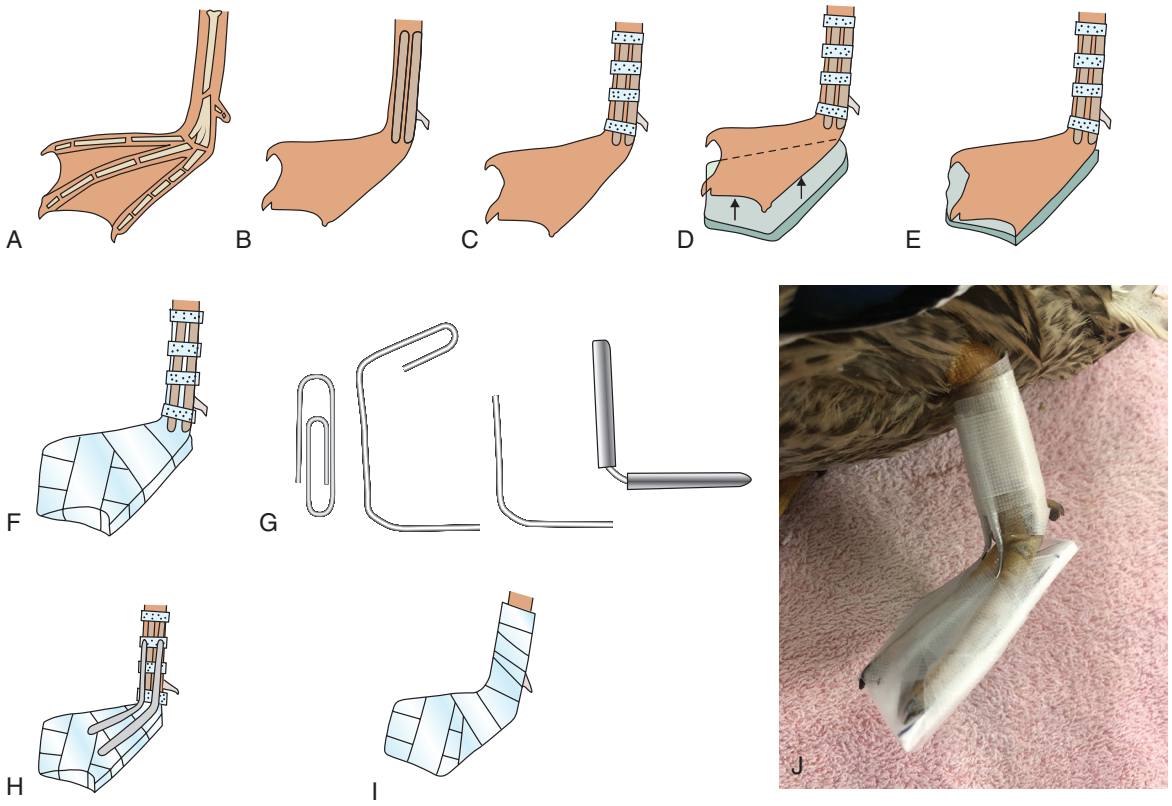


FIGURE 37.20 Application of a waterfowl tarsometatarsal "walking boot" for a closed, distal tarsometatarsal fracture (A). Swimming is also potentially allowable with this boot. Splint the fractured tarsometatarsus in a standing position. If fracture is oblique with sharp ends, or extra padding is indicated, apply a layer of hydrocolloid dressing to leg before applying splints. The fracture is stabilized with two narrow wooden splints covered with layers of plastic tape (Transpore, 3M, St. Paul MN). This clear tape allows observation of the foot during healing and stays on well in water. (B) Tape splints to lateral and front sides of the leg, avoiding medial metatarsus and associated vasculature and snugly fitting one piece of tape distal to digit I (C). Trace foot to create shoe from packaging foam (D, E) and secure to bottom of foot by taping toes in place (F). Create L-shaped braces from metal paperclips (G), cover with layers of tape and secure one between digits II and III, and the other between digits III and IV (H). Adjust the angles for comfortable standing posture. Tape snugly to front of tarsometatarsus and between toes (I). Wrap foot with yet more Transpore tape, leaving toe tips exposed to monitor swelling as possible (J). Inspect for tape loosening daily, replace as needed. Remove boot weekly to monitor progress. The shoe may not need to be replaced at 1 week, and the fracture should be stable to palpation in 2 to 3 weeks. (Images courtesy Rebecca Duerr.)

a raptor is never recommended due to its importance in hunting strategies and concerns with subsequent pododermatitis and/or balance concerns. Acceptability of amputation of other toes on raptors depends on their hunting style. For example, digit II is critical in buteos and owls (Strigiformes), while digit III is more important in accipiters. Gulls can handle amputation of digit II or IV (not both) while the webbed feet of waterfowl are often evaluated on ability to maintain surface area.

Splinting is the recommended method for treatment of most phalangeal fractures.[7] A basic ball bandage can be used to immobilize digits and remove weight-bearing in raptors or psittacines (Figure 37.21). Other methods used include incorporating a fitted "shoe" made from thermoplastic, hexalite, or open-cell foam into the splint (Figure 37.22). Shoes can be donut shaped (Figure 37.23). Once healing is well underway, the splint can be replaced with a thick interdigitating bandage extending the length of the involved digit.

Splinting is also recommended for most other avian species although the diversity of birds creates slightly different considerations for different taxa. Splints can be made from a variety of materials, including rigid plastic, Styrofoam, or padded aluminum; cardboard is typically not recommended as it holds water.[41] The splint is shaped so that it can be applied to the plantar surface of the foot and the toes taped in place to immobilize and secure the shoe to the foot. Routine rechecks (one to two times per week) are recommended with the splint being removed to allow examination of the plantar foot surface of the foot for development of pododermatitis. Waterfowl provide some additional challenges with their webbed feet and their predisposition to pododermatitis and keel lesions when maintained on land. In addition to the application of a splint to the foot, housing and management are particularly important.

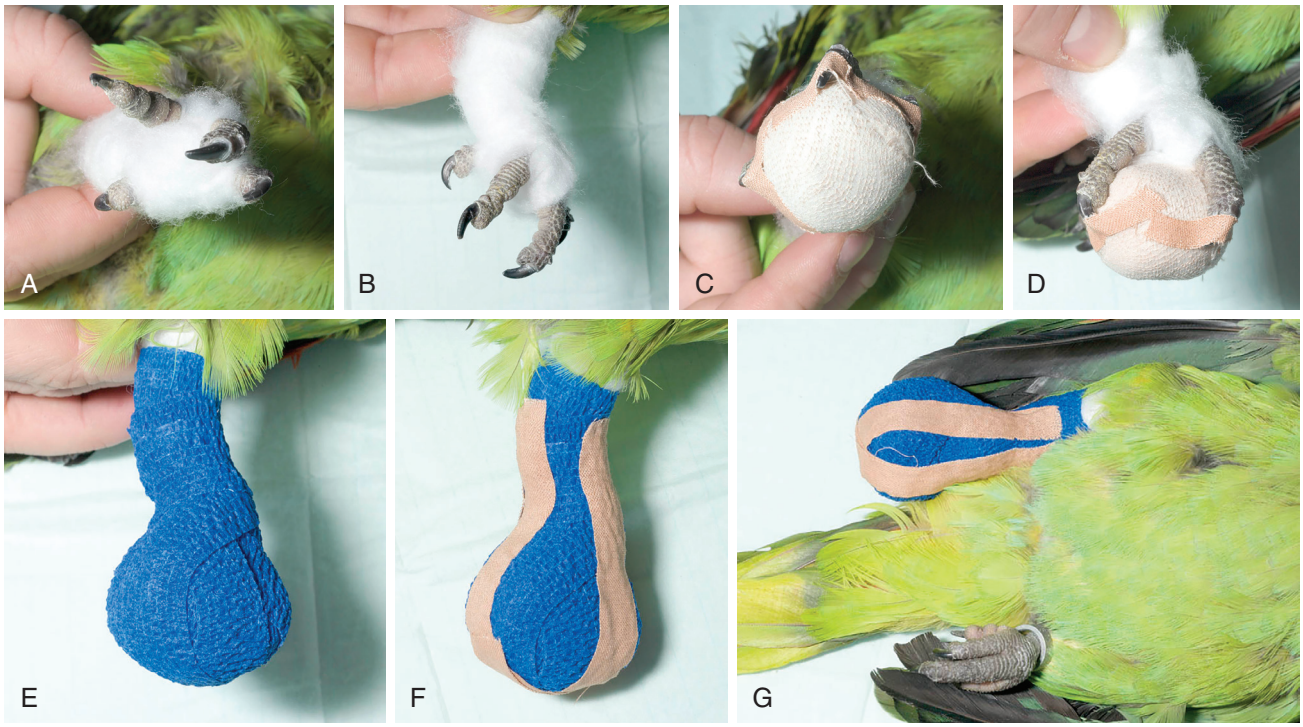


FIGURE 37.21 A ball bandage on a parrot foot. Apply a layer of padding to foot and leg (A, B) and then place a ball of cotton wrapped in gauze on the plantar aspect of the foot and tape the toes to the ball (C, D). Wrap the foot in self-adherent wrap, extending up tarsometatarsus to provide support (E) and apply tape strips to hold in place (F). Finished ball bandage on a parrot (G). (Images courtesy Y.R.A. van Zeeland.)

Facial Fractures/Skull

The diverse taxa of birds differ markedly in facial structure, form, and function. The diversity of morphology of bills has a profound bearing on both the types of injuries sustained and treatments. The beak shows wide variation in the number and location of hinges in both upper and lower bills (see Chapter 31). Lifestyle also has a large impact on feasibility of treatment for many fractures, and housing needs must be kept in mind during care lest the treatment predisposes to further problems. External fixators modified to adapt to beak confirmation are the most common form of surgical fixation. These fixators and other hardware can easily become entrapped in netting or other aspects of housing or handling equipment.

Birds that have heavily keratinized bills, especially those with heavily keratinized gnathotheca (lower bill) such as gulls, many passerines, and parrots are prone to problems with keratin overgrowth of the opposite bill when a malocclusion is present. In the wild, these birds are likely to develop progressive rhamphothecal overgrowth abnormalities that will ultimately cause the bird to starve to death. In captivity, these species often require rhamphothecal shaping procedures as part of ongoing routine care, or corrective surgical procedures of the primary malocclusion problem, if possible (see Chapter 31). Consequently, if portions of the bill are missing in these species, the bird is not a candidate for wild release. These cases are also often not suitable for placement; as per USFWS regulations, wild birds must be able to apprehend food to be a candidate for placement. When the tip of the upper bill is missing, species vary in how much can be missing and retain the potential for regrowth of adjacent keratin to fill in a gap to achieve normal occlusion; several months may be needed to ascertain whether regrowth will result in a bird potentially suitable for release. Species with fleshy bills such as ducks, pelicans, cormorants (*Phalacrocorax* spp.), and others often adapt well to bill defects as long the defect does not impair food apprehension. Prostheses are not appropriate for birds intended for wild release due to their need for regular maintenance and adjustments.

Spinal Fractures

In most birds, there is an evolutionary fusion of thoracic vertebrae into the notarium and thoracic, lumbar, sacral, and caudal vertebrae into the synsacrum, which provides a rigid and lightweight structure for flight. In many species, there is a free thoracic vertebra located between the notarium and synsacrum. Because this intervertebral joint is more vulnerable than the rest of the rigid spinal column, it is often the site of severe trauma. When nondisplaced, visualization of these lesions on survey radiographs can be very difficult. If neurological deficits are severe, the prognosis for full recovery is poor and wild birds are often humanely euthanized. For wild birds with mild deficits or captive birds that do not require full return to function, conservative management consists of maintaining in an enclosure with thick padding and monitoring for cloacal output (gentle flushing or evacuation as needed). If the trauma occurred within 8 hours of admission, administration of high-dose methylprednisolone sodium succinate (Solu-Medrol; Pfizer Inc, NY) may be helpful.

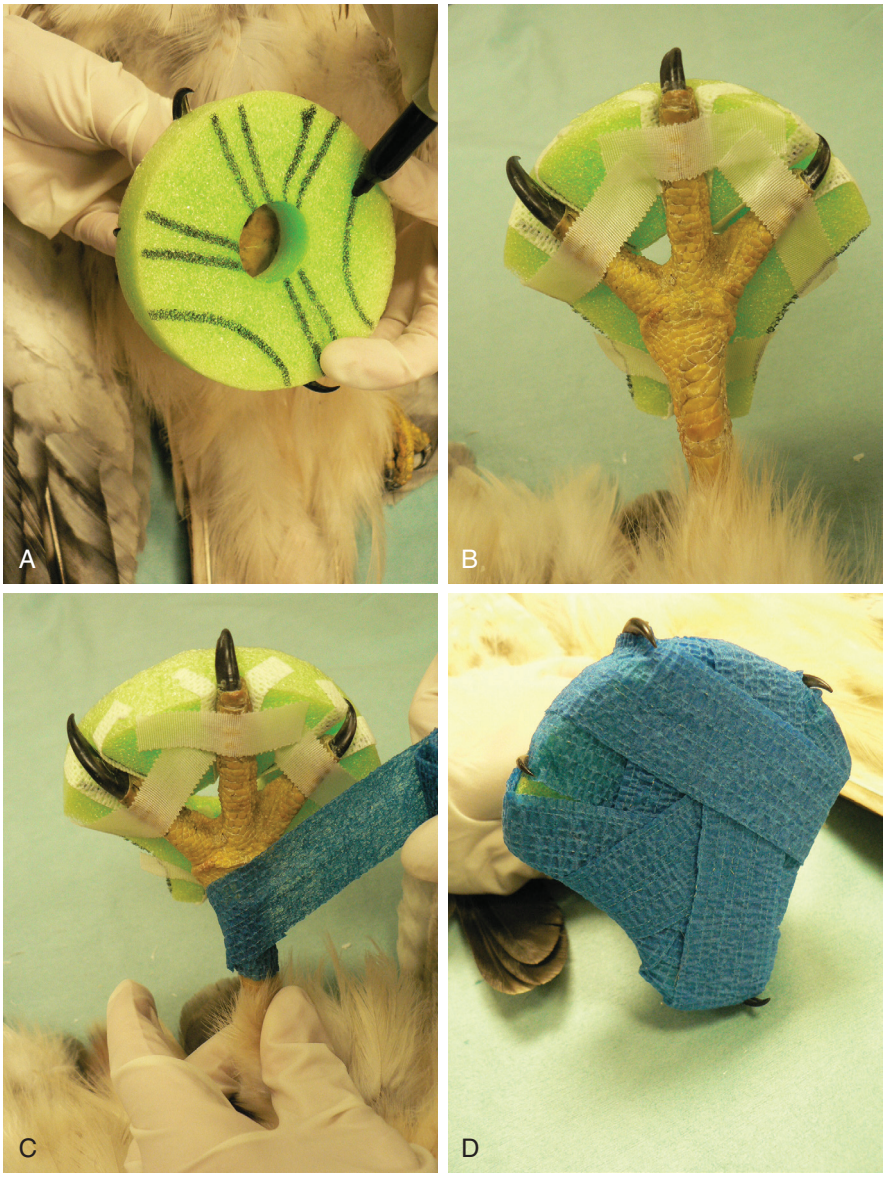


FIGURE 37.22 Creating a raptor "shoe" from a foam disk. Normal toe position is sketched on disk (A) and channels cut out for positioning toes, which are taped in place (B). The foot is then wrapped in self-adherent wrap, extending up the metatarsus for support (C) and covering the whole shoe (D). Optional materials include making shoes from thermoplastic or hexalite (see Chapter 33).

Luxations

Shoulder girdle

Coracoid luxations are treated similarly to coracoid fractures, with conservative management focusing on cage rest, coaptation, and physical therapy. Proximal coracoid fractures at the sternal juncture carry a better prognosis than distal luxations where the coracoid articulates with the scapula, clavicle, and humerus in the authors' experience. If the integrity of the shoulder joint is altered, the bird has a poorer prognosis for full restoration of flight.

Elbow

While some success has been reported with caudodorsal luxations on the elbow, the prognosis for these cases is usually poor either because of the inability to maintain reduction, loss of range of motion, or because of the development of significant long-term degenerative joint disease. Successful outcomes are most likely to be achieved with early intervention with quick reduction of the luxation. Once reduced, the joint needs to be protected from reluxation either with coaptation or with a transarticular ESF fixator while it heals. Regular physical therapy using passive range of motion exercises (anesthesia recommended) is also needed. Lateral imbrication of the joint done after the initial swelling has resolved will help to stabilize the joint.[42]

Metacarpal-phalangeal

While there are few reported treatments for luxation of this joint, joint arthrodesis had a successful outcome with full restoration of flight in a prairie falcon. A type I ESF was used to stabilize the joint during healing.[43]

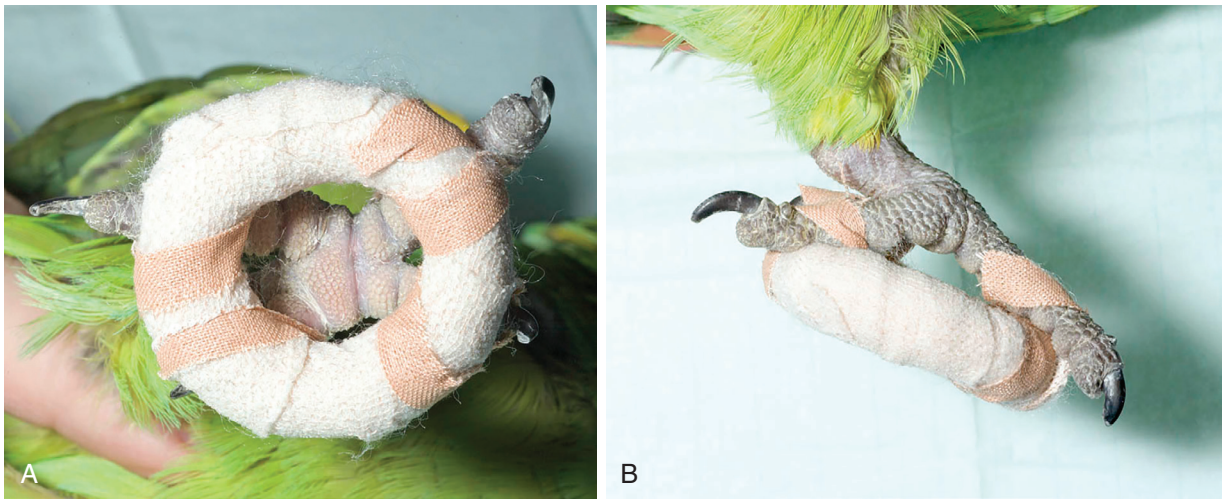


FIGURE 37.23 (A) A donut shoe on the plantar aspect of a parrot foot and (B) lateral view showing toes taped in place. (Images courtesy Y.R.A. van Zeeland.)

Hip

Femoral head luxations occur with the capital physeal ligament between the femoral head and the acetabulum being ruptured. Small birds may do well with closed reduction and cage rest while a femoral head and neck ostectomy has been used successfully with larger birds.[2,22]

Stifle

Treatment of stifle luxations involves reduction and stabilization while the traumatized tissues heal. A transarticular ESF with an acrylic bar connection can be used to stabilize the stifle and hold it in a flexed perching position.[44] Another reported method involves the placement of IM pins in both the femur and tibiotarsus with both pins projecting at the stifle. These pins were bent where they protruded and connected with acrylic to maintain alignment of the joint during healing.[45]

SUMMARY

The broad diversity in patients and injuries seen in avian practice requires that the clinicians have a strong understanding of the guiding principles of orthopedics and the unique challenges presented by birds. Getting to successful outcomes requires a variety of tools, as well as creativity and adaptability in using them. Given the limited number of controlled studies and emphasis on case studies in the literature, the value of evidence-based medicine cannot be overstated. Choosing the right approach in any specific case requires a systematic approach to decision-making that is based on the patient and its circumstances, the resources available, and the ability to apply those resources to construct adequate stabilization for fracture healing.

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CHAPTER 38

From Fertilization to Hatch: Egg and Embryonic Development

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INTRODUCTION

The most striking difference between mammalian and avian reproductive strategy is that of oviposition: birds lay eggs, mammals do not. It has long been a puzzle why "birds constitute the only vertebrate class that is both rich in species number and exclusively oviparous."[1] The absence of avian viviparity has frequently been explained by citing morphological or physiological factors that presumably are incompatible with live birth.[2] Whatever the reasons for maintenance of oviparity, their success is obvious, judging from the large number of species of birds that have evolved and their colonization of all parts of the world. The number of known bird species is more than double the number of mammals. One can also look at birds as successful competitors with mammals by adapting incubation behavior, shell structure, and shell porosity to varied environmental conditions to deliver hatchlings that are behaviorally, functionally, and in gross composition like mammalian neonates. Some of the key differences between the reproductive systems of birds and mammals are displayed in Table 38.1. Common differences in physiologic features are displayed in Box 38.1.

There are also many differences between avian and mammalian reproduction. For example, mammals exhibit physiological monospermy, in that only a single sperm enters the ovum. Monospermy is ensured by a variety of mechanisms, which are collectively known as the block to polyspermy. However, in birds, many fish, amphibians, and reptiles, the principal feature of fertilization is physiological polyspermy, which involves penetration of the ovum by many sperm.[4] Following ovulation, the ovum is transported to the infundibulum of the female reproductive tract where multiple spermatozoa then encounter the inner vitelline layer. Soon after contact with this layer, an outer vitelline layer is quickly assembled, trapping many spermatozoa, and preventing additional spermatozoa from entering. Once a single sperm enters the germinal disk (GD) and fuses with the female oocyte, the other supernumerary sperm degenerates.

PHYSIOLOGY OF EGG LAYING

The major hormones involved in the ovulation are gonadotropin-releasing hormone-1 (GnRH-1) and luteinizing hormone (LH).[5] Follicle-stimulating hormone (FSH), which is an important hormone in the ovulation in mammals, is involved in the differentiation of granulosa cells and induction of steroidogenesis in the prehierarchical follicles. However, in contrast to mammals, it is not directly involved in ovulation. Estrogens and testosterone need to be present to allow ovulation to occur, but neither of these hormones can induce ovulation on their own. The role of corticosteroids during ovulation is unclear, but their presence is required in order for ovulation to occur. Progesterone is present during ovulation with an increase 4 to 6 hours prior to ovulation, produced by the granulosa layer of the largest preovulatory follicle F1, and during the egg development. Progesterone receptors in the oviduct stimulate the hyperplasia of the epithelial cells and the tubular gland cells. Progesterone plays a role in the production of avidin, a protein that is involved in the defense mechanism of the uterus and egg, the contraction of the myometrium, and the shell formation. Estrogen during egg development is involved in the synthesis of ovalbumin, ovotransferrin, ovomucoid, and lysozyme in the magnum. During oviposition, the smooth muscle of the oviduct needs to be relaxed, which occurs under the influence of several hormones including prostaglandins, acetylcholine, galanin, oxytocin, arginine vasotocin (AVT), and angiotensin II. In laying hens, the administration of oxytocin and AVT induces premature oviposition.

ANATOMY OF THE FEMALE REPRODUCTIVE TRACT AND FORMATION OF THE EGG

In contrast to mammals, the reproductive tract in birds allows internal fertilization, while further development of the ovum

TABLE 38.1

Key Differences Between the Avian and Mammalian Reproductive Systems

	Avian	Mammalian
Definition	Permits internal fertilization Allows the development of the embryo in the external environment	Permits internal fertilization Development of the embryo inside the body Produces offspring directly
Male organs	Testes and epididymis, ductus deferens, seminal glomera, phallus (in some species)	Scrotum, testes, epididymis, vas deferens, ejaculatory duct, penis, seminal vesicles, prostate gland, Cowper's gland
Female organs	Ovary and oviduct	Ovaries, oviducts, uterus, vagina
Nourishment of the embryo	By the ovum itself	Through the placenta
Birth of offspring	Oviposition and egg hatch in the external environment	Delivery of fully developed offspring

BOX 38.1 SOME DISTINGUISHING FEATURES OF BIRD EMBRYOS AND NEONATES IN COMPARISON WITH MAMMALS

1. Avian embryos are maintained at temperature of about 36°C (96.8°F) and mammalian embryos at 37°C (98.6°F).
2. During the late stages of development, the O_2 and CO_2 tensions of the embryonic and extraembryonic blood vessels in avian embryos attain values like those in the mammalian fetus. In both, the acid-base status is essentially that of the adult, characterized by a plasma PCO_2 of 30–40 torr and a pH of 7.4–7.5, the hallmark of warm-blooded vertebrates.
3. Both birds and mammals deliver equivalent neonates, which range from altricial species, born naked and blind, to precocial species, born furred or feathered, and able to walk and thermoregulate.
4. The relative water content of neonatal birds is inversely proportional to their state of maturity and, in birds, varies from approximately 85% in altricial species to approximately 65% in the most precocial species.[2] The percentage of the body composed of water in a term human neonate is 75% (40% extracellular fluid, 35% intracellular fluid). Term newborns usually lose 5% to 10% of their weight in the first week of life, almost all of which is water loss.[3]

occurs outside of the body. This requires that all the nutrients that are needed for the embryo to develop to be included in the egg before it is laid. As a result, egg formation is a complex process that is highly dependent on the functionality of the ovary and oviduct. While two bilaterally symmetrical ovaries and oviducts develop in the avian embryo, in nearly all species, the right ovary and oviduct regress leaving only the left side functional in adult life. Exceptions include the kiwi (Apterygiformes) and some birds of prey from the families Cathartidae, Accipitridae, and Falconidae, in which the right oviduct is commonly vestigial or nonfunctional, but two functional ovaries are present.[6]

The left ovary is located in the intestinal peritoneal cavity, cranial to the left kidney and adjacent to the adrenal gland.[6] The ovary, which is attached to the left kidney and body wall by the mesovarian ligament, is less compact compared to mammals, with a poorly defined medulla and cortex. In young hens, the ovary is barely visible, but as the hen matures, follicles start to develop within the ovary giving it the appearance of a small bunch of grapes. Primary oocytes visible on the surface of the ovary are termed follicles, which pertain to the primary oocyte and its membranous covering. The age at which this functional development occurs depends on the life span of the species. For example, many of the larger psittacine birds become reproductively active between 4 and 7 years of age, whereas smaller species often become sexually mature well before 3 years of age. During the breeding season, the follicles undergo rapid growth, during which yolk and protein produced by the liver accumulate in the oocyte or ovum, giving it its characteristic yellow color. Yolk is made in the liver and is hematogenously delivered to the developing follicle. After completion of the rapid growth period, the primary oocyte undergoes two maturation divisions. The mature follicle consists of white yolk, yellow yolk, and the GD. These are surrounded by the vitelline membrane. As in most other vertebrates, the ovum is ovulated with its nucleus at metaphase of the second meiotic division. In the chicken the voluminous 3- to 4-cm-diameter ovum is primarily comprised of yellow yolk. On its surface is a 3- to 5-mm-diameter GD, composed of white yolk that forms the animal pole. The GD contains the female complement of genetic material as well as most cytoplasmic organelles.[7,8]

Following ovulation, the ovum passes through the entire length of the oviduct, where the components of the egg are secreted and deposited from respective parts of the oviduct. The contributions of the respective segmental portions of the oviduct in the ovulatory cycle are highlighted in Box 38.2 and Figure 38.1.

The left oviduct is attached to the dorsal body wall by the dorsal ligament and has three general primary functions: egg formation; sperm selection, storage, and transport; and it is the site of fertilization and early embryonic development. Glandular development within the oviduct results in a thickening of its walls that differentiate it into five functional regions associated with egg formation. These portions are termed the infundibulum, magnum, isthmus, uterus, and vagina.[6]

The infundibulum is the funnel-shaped structure at the proximal end of the oviduct along with a very small portion of

BOX 38.2 CONTRIBUTIONS OF OVIDUCTAL SEGMENTS IN EGG FORMATION

1. **Infundibulum.** Following ovulation, the ovum is engulfed by the infundibulum where it resides for approximately 18 minutes (range, 15–30 minutes in the domestic fowl). Fertilization and formation of the chalaziferous membranes occur in the infundibulum.
2. **Magnum.** The ovum next passes to the largest portion of the oviduct, the magnum (a length of 33 cm in the chicken), where most of the albumen is formed. Movement of the ovum through the magnum is primarily via peristaltic action of longitudinal circular layers of muscle. The ovum remains in the magnum approximately 2–3 hours in the domestic fowl.
3. **Isthmus.** The isthmus is clearly demarcated from the magnum. Both inner and outer shell membranes are formed during the 1- to 2-hour (mean time, 1 hour and 14 minutes in the domestic fowl) passage through the isthmus.
4. **Shell gland (uterus).** The shell gland (uterus) is characterized by a prominent longitudinal muscle layer lined medially with both tubular and unicellular goblet cells. Prior to calcification, the egg takes up salts and water into the albumen from the tubular glands, a process termed "plumping." The ovum remains in the shell gland for 20–26 hours. Calcification of the egg occurs over a period of 15 hours, while the pigment of the shell is formed during the last 5 hours in the domestic fowl.
5. **Vagina.** The vagina is separated from the shell gland by the uterovaginal sphincter muscle and terminates at the cloaca. The vagina has no role in the formation of the egg, but, in coordination with the shell gland, participates in the expulsion of the egg.

Modified from Johnson AL: Reproduction in the female. In: Sturkie PD, editor: *Avian physiology*, ed 4, New York, Springer-Verlag, 1986, pp 403–431.

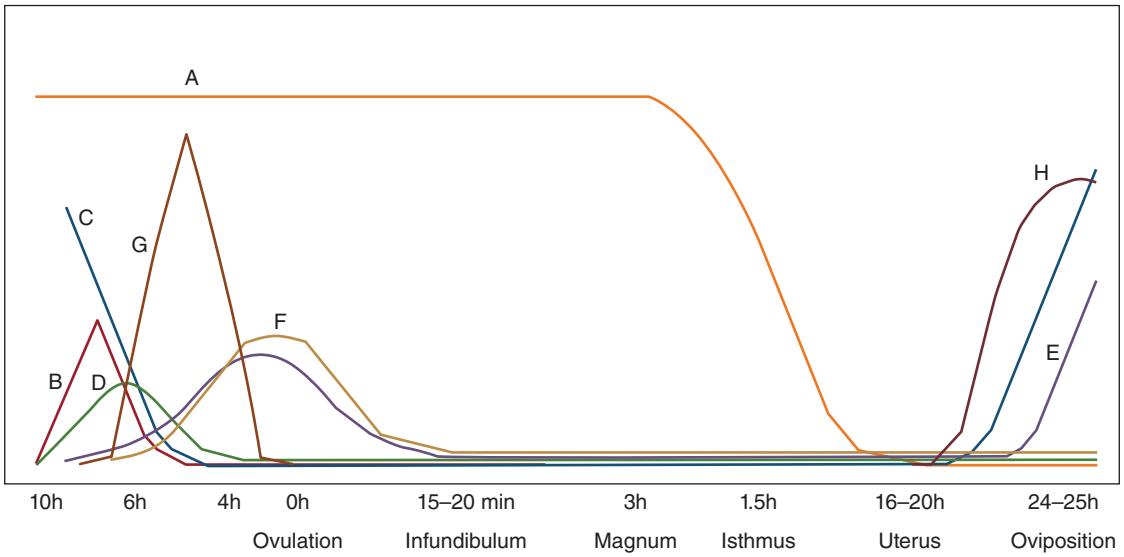


FIGURE 38.1 P. Schnitzer: Scheme of hormone involvement during oviposition: *A*, medullary bone formation with osteoblast activity; *B*, testosterone; *C*, prolactin; *D*, progesterone; *E*, estradiol; *F*, corticosterone; *G*, LH peak; *H*, prostaglandins (PGF2α, PGE1, PGE2), angiotensin II, galanin, acetylcholine, arginine, vasotocin, and oxytocin. *LH,* Luteinizing hormone; *PG*, prostaglandin.

the tubular-shaped proximal oviduct. The funnel-shaped portion of the infundibulum forms an elongated slit that faces the ovary. The ovulated secondary oocyte is literally swallowed or "caught" by the funnel portion of the infundibulum, regardless of the presence or absence of spermatozoa for the purpose of fertilization. This "catching" process is facilitated by the adjacent air sac that tightly encloses the ovary and forms the "ovarian pocket," leaving only the direction of the infundibulum for easy movement of the secondary oocyte. Fertilization of the ovum occurs within 15 minutes of ovulation and presumably occurs in the infundibular oviduct. Formation of the outer layers of the yolk membrane probably begins in the tubular portion of the infundibulum. These outer layers of yolk membrane are termed the chalaziferous layer of albumen and the chalazae. The chalazae are spiral shaped and made of albumen-like proteins that form the attenuated continuous layer and the more extensive outer perivitelline layer (OPVL). This fibrous investment strengthens the perivitelline layer (PVL) complex supporting the megalecithal ovum in its transit through the oviduct.

The magnum is the longest and most coiled portion of the oviduct. It is distinguished by its greater external diameter and markedly thicker wall caused by the presence of numerous secretory glands that account for the prominent mucosal folds of the magnum. These glands *(glandulae magni)* secrete the thick albumen protein (ovalbumin, ovotransferrin, ovomucoid, ovoglobulins, and lysozymes) around the ovum. The stimulus to secrete albumen may be mechanical, arising from passage of the ovum along the magnum. Smooth muscle contractions peristaltically move the ovum along the oviduct. In a reproductively

active bird, the magnum undergoes tremendous enlargement. The added length of the enlarged magnum causes a folding of the oviduct upon itself. The division between the magnum and the isthmus is marked by a thin, translucent line that can be seen on the mucosal surface with the unaided eye in domestic fowl. This region of the oviduct is short and has less prominent mucosal folds than the magnum.

The isthmus consists of two sections. The more proximal section is nonglandular with no folds, while the distal part, also termed red isthmus, is glandular with sulfur-containing keratin-type protein–producing glands *(glandulae isthmi)*. These proteins form the outer and inner membranes, are composed of 10% collagens (types I, V, and X) and 70% to 75% proteins and glycoproteins, and are semipermeable allowing exchange of gases and water while retaining the albumen loosely around the ovum and albumen.

The uterus (shell gland) is initially a similar diameter as the isthmus, but rapidly expands to form a pouch that retains the egg during the entire portion of egg formation. The density in the lamina propria of glands is higher in the uterus or shell gland. The egg remains for up to 20 hours in the uterus or shell gland in the domestic fowl. The glands *(glandulae uterinae)* add water to the albumen and the different layers of the eggshell. Calcium secretion starts approximately 7 hours after ovulation. Microcrystals of calcite and calcium carbonate are added to form the inner mammillary cone layer and the palisades.

The short terminal portion of the oviduct immediately proximal to its junction with the urodeum is the vagina. The strong muscle of the vaginal wall and a well-developed muscular sphincter *(m. sphincter vaginae)* at the uterovaginal junction (UVJ) serve to expel the egg during oviposition. The vaginal glands store sperm in the tubular utero-vaginal sperm host glands *(fossulae spermaticae)*.

The cuticle is the outer layer of the eggshell and is composed of an inner mineralized layer (inorganic phosphorus, hydroxyapatite crystals) and an outer layer of organic matrix (glycoproteins, polysaccharides, lipids). It plays a role in controlling water exchange by repelling water and limiting microbial colonization of the eggshell surface; it is likely the last portion of the egg to be formed. It covers the pores of the egg but is still able to allow gas and water exchange. Despite the knowledge of its existence and its function for at least 130 years, there remains confusion about its formation, its relationship with protoporphyrin pigmentation of eggs, and where it is formed. According to Wilson et al., the cuticle is deposited in the uterus instead of the vagina.[9] Its deposition is susceptible to environmental stressors and is specifically linked to steps of the normal endocrine cascade that occur immediately prior to oviposition. However, premature oviposition induced by AVT, one of the final endocrine events prior to oviposition, will not result in the deposition of cuticle, even if AVT is administered close to the expected time of oviposition. This indicates that deposition of the cuticle is a specific event that is distinct from other events in eggshell formation and is neither directly related to the organic matrix of the eggshell nor to pigment deposition (although these processes may partially overlap).

The cuticle can be found on eggs from many avian species, with its thickness greater on eggs laid in nests with a greater microbial challenge, particularly those of aquatic birds. The presence of cuticular mineralized nanospheres has been suggested to be correlated with the wetness and warmness of the nest for the species, and the reports of eggs with no cuticle are from species such as the budgerigar (*Melopsittacus undulatus*), which are from drier environments. Thus there is an apparent relationship between damp environments and presence of a cuticle.[9] Following oviposition, the two layers of the shell membrane separate to form the air cell near the blunt end of the egg.

The "ovulatory cycle," encompassing ovulation, egg formation, and finally oviposition, in the domestic fowl is about 24 to 25 hours in duration and is repeated with each egg in a clutch. If the ovum was fertilized in the infundibulum, which generally occurs within 15 minutes of ovulation, the initial cleavage furrows would appear in about 5 to 7 hours after sperm penetrates the ovum, around the time that the egg resides in the isthmus or uterus. During this time, the blastoderm undergoes further cleavage divisions and consists of 30,000 to 60,000 cells at the time of oviposition, depending on the species.[4,10]

ANATOMY AND COMPOSITION OF THE EGG

The egg is composed of five fundamental parts: yolk, white, chalazae, shell membranes, and shell (Figure 38.2).[6] At the time of oviposition, the egg must contain all the needed nutrients to support the health and development of the embryo. The breeding pair, and especially the female, needs adequate nutrition to be able to put all nutrients in the egg. An important saying of the aviculturist is: "The egg is what the hen eats!" The whole egg consists of 9% to 11% eggshell, 60% to 63% albumen, and 28% to 29% yolk. The composition of the yolk is approximately 33% lipids, 17% proteins, and 48% water. Albumen is composed of 88% water and 10% protein, 54% of which is ovalbumin, 13% ovotransferrin, 11% ovomucoid, 8% ovoglobulins, 3.5% lysozyme, 3% beta-ovomucin, and 1.5% alpha-ovomucin.[5]

Different supplements and drugs have been shown in chickens to cause alterations in the content of their eggs. Their effects on the hatchling are not fully understood and remain unclear at the present time (Box 38.3).

Aside from supporting the development of the embryo, the egg serves an important role in the protection of the embryo, acting as a barrier against physical, chemical, and biological threats. The different components of the eggshell, membranes, and egg white create a physical and chemical barrier, which protect the embryo from pressure, heat, osmotic stressors, and microbial colonization and infection. Ovotransferrin, lysozyme, and P-NAGase are the primary components acting as a chemical barrier within the egg white.[5]

SPERM SELECTION, STORAGE, AND TRANSPORT

In hens that have mated naturally or have been subjected to artificial insemination (AI), a relatively small number of spermatozoa are transported to storage sites at the proximal end of the vagina, a region referred to as the UVJ. In the surface epithelium of this UVJ, discrete tubular structures, known as sperm storage tubules (SSTs), can be found, which are populated

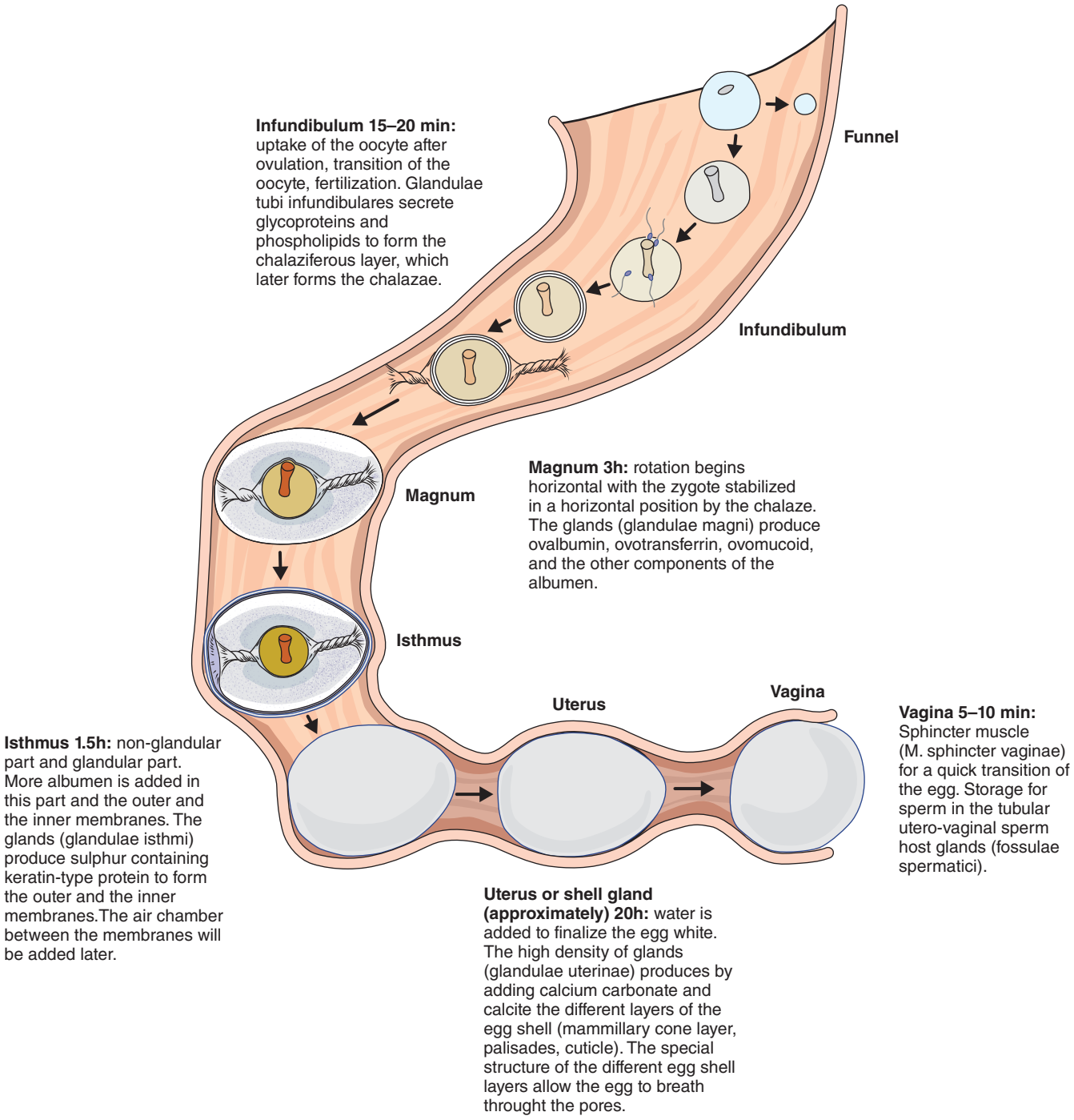


FIGURE 38.2 Diagrammatic representation of the formation of the egg.

by the spermatozoa that successfully traverse the vagina. Generally, these spermatozoa are distinct in both mobility characteristics and surface membrane composition compared to those that are unsuccessful in entering the SST. Depending on the bird species, spermatozoa are stored for variable lengths of time in the SST before their release and ascension into the infundibulum (Box 38.4, Figure 38.2), which is the site of fertilization and presumably also a secondary sperm storage site. Sperm transport is probably a combination of spermatozoal mobility and activity of the ciliated epithelium lining the oviductal mucosa. Most spermatozoa that are transferred into the vagina at copulation or AI fail to populate the SST. As a result, the maximum number of sperm cells in the SST remains below 10 million, even if hens are inseminated with higher numbers of up to 150 to 350 million spermatozoa. Obviously, the vagina orchestrates an intense sperm selection process. The mechanisms behind such selection remain to be elucidated.[4]

The spermatozoa that reach the UVJ and enter the SST can be viewed as "selected" sperm. It appears that acceptance

BOX 38.3 SUPPLEMENTS AND TREATMENTS AND THEIR KNOWN EFFECTS ON EGG COMPOSITION

Supplements/Treatments Prior to Oviposition	Alterations in Egg Composition
Garlic powder at 10 g/kg	Increase of egg weight, decrease in egg yolk cholesterol concentration
Flaxseed supplementation	Increase in albumen and decrease in yolk content of the egg
Soy oil supplementation	Increase of egg weight, increase in albumen, and decrease in yolk content of the egg, reduced shell thickness
Enrofloxacin treatment	Reduced IgY concentration in the egg
Choline and methionine supplementation	Increase of egg weight, increase in phospholipid content of the egg yolk
Grape seed extract	Increase in egg size and shell elasticity, decreased steroid production without affecting fertility
Vitamin E, carotenoids, selenium	Decreased susceptibility to lipid peroxidation, improved antioxidant system of hatchlings

BOX 38.4 SPERM STORAGE DURATION IN SELECTED BIRD SPECIES

Common Name	Scientific Name	Sperm Storage Duration (Days)
Ring-necked pheasant	*Phasianus colchicus*	21
Japanese quail	*Coturnix japonica*	6.3
Domestic chicken	*Gallus gallus domesticus*	12
Turkey	*Meleagris gallopavo*	42
Willow ptarmigan	*Lagopus lagopus*	7.8
Northern bobwhite	*Colinus virginianus*	8.3
Helmeted guineafowl	*Numida meleagris*	7
Graylag goose	*Anser anser*	9.7
Mallard	*Anas platyrhynchos* (wild)	9.9
Domestic duck	*Anas platyrhynchos* (var. domestica)	10.3
Pigeon	*Columba livia*	8
African collared dove	*Streptopelia roseogrisea*	8
Budgerigar	*Melopsittacus undulatus*	11
Zebra finch	*Taeniopygia guttata*	10
White-rumped munia	*Lonchura striata*	8
American kestrel	*Falco sparverius*	8.1

Modified from Birkhead TR, Moller AT: Numbers and size of sperm storage tubules and the duration of sperm storage in birds: a comparative study, *Biol J Linn Soc* 45:363–372, 1992.

of spermatozoa in the SST and interaction with the ovum is in part controlled by the hen,[11] but the underlying mechanisms as well as factors that favor transport of some of these spermatozoa over others currently remain unknown.[12]

In mammals, ejaculated spermatozoa are progressively motile yet do not have an immediate capacity for fertilization. In the case of in vivo fertilization, this ability is gained after residing in the female reproductive tract, whereas it can also develop following incubation in an appropriate medium in case of in vitro fertilization (IVF). The physiological changes that render the spermatozoa competent to fertilize are referred to as capacitation.[13,14] It is generally believed that capacitation of sperm is not essential for fertilization in birds.[15] This was primarily based on observations that avian ova could be fertilized in vitro by freshly ejaculated sperm that had not been previously treated with a special capacitation medium.[16–18] Moreover, it was shown in vivo that avian spermatozoa do not need to reside long in the female reproductive tract to be competent for fertilization since fertilized eggs could be observed within 15 minutes after AI into the vagina[19] or infundibulum.[10] However, while freshly ejaculated sperm can fertilize an ovum in vitro, the spermatozoa that fertilize a daily succession of ova have often resided for days or weeks in the oviductal SST. Bakst et al. suggested that sperm residing within the SST are quiescent, with motility, metabolism, and enzyme systems reversibly suppressed ("decapacitated" in the mammalian sense).[12] Upon release from the SST and prior to their ascension to the fertilization site, the oviductal environment was suggested to reactivate or recapacitate the spermatozoa. However, more recently, Nixon et al. demonstrated in Japanese quail *(Coturnix japonica)* that spermatozoa did not need a real capacitation to fertilize the ovum.[20] However, their research did suggest that hemoglobin is secreted into the quail epididymis to support sperm metabolism, aid in motility, and/or act as an antioxidant.

FORMATION AND DEVELOPMENT OF THE EMBRYO

The avian embryo has a long history as a subject of embryological study. The egg is universally available, and the embryo is readily accessible because it is a self-supplying system and does not depend on the mother for its nutrition, unlike the embryo of placental mammals. The first clear-cut account of chick embryology was given by Hippocrates (460–377 BCE). He is quoted by Needham[21] as follows: "Take 20 eggs or more and give them to 2 or 3 hens to incubate, then each day from the second onwards till the time of hatching take an egg, break it, and examine it ...." This orderly and systematic approach is meaningful even today. Aristotle (382–322 BCE), who also followed this system, appears to have been the first to dissect the chick embryo.[22]

According to Moran[23] and De Oliveira et al.,[24] embryonic development can be divided into three major phases as follows:

1. **Establishment of germ.** This phase includes the first week of incubation, which is characterized by the formation of egg compartments (amnion, chorion, allantois, and yolk sac) that support the survival of the growing embryo. Oxygen supply may be limited due to immature blood cells and an underdeveloped chorionic vascular system, so metabolic energy is provided by anaerobic glycolysis of the limited amounts of glucose present in the egg.[23,25]

2. **Embryo completion.** This phase of embryonic development is characterized by a fully developed chorioallantois, which assures adequate oxygen-carbon dioxide exchange and supports rapid embryonic growth.[23] Establishment of chorioallantoic respiration occurs around day 8 of embryonic development in the domestic chicken *(Gallus gallus domesticus)*.[26] Chick embryos are structurally completed around 14 days of incubation, whereas 22 days of incubation are needed to complete embryo development in the turkey *(Meleagris gallopavo)*. As the embryo approaches full body mass, maximum chorioallantoic respiration capacity is reached in what is called the plateau stage of oxygen consumption.[27]
3. **Emergence.** This last phase is characterized by oral consumption of the amnion by the embryo, accumulation of glycogen reserves in muscle and liver tissues and glycogenolysis, initiation of pulmonary respiration, abdominal internalization of remaining yolk, shell pipping, and emergence.[23,28–33] During this period, dramatic physiological and metabolic changes occur, and any disturbances during this period may markedly affect embryonic survival and later performance.[32,34,35]

The normal stages of morphological development in the domestic fowl embryo have been described in detail by Hamburger and Hamilton,[36] Hamilton,[37] and Romanoff.[38] A good description of the chicken embryo development is reported online by Hill in 2020,[39] and the Hamburger and Hamilton (HH) development stages are reported in Box 38.5. Figure 38.3 demonstrates the embryonic development of a cockatiel.

BOX 38.5 HAMBURGER AND HAMILTON DEVELOPMENTAL STAGES IN CHICKEN BY DAY OF INCUBATION

Hamburger and Hamilton Stages	Age	Identification of Stages
Before Laying		
Early cleavage	3.5–4.5 hours	Shell membrane of egg formed in isthmus of oviduct
During cleavage		Germ wall formed from marginal periblast
Late cleavage	4.5–24.0 hours	Shell of egg formed in uterus
After Laying		
1		Preprimitive streak (embryonic shield)
2	6–7 hours	Initial primitive streak, 0.3- to 0.5-mm long
3	12–13 hours	Intermediate primitive streak
4	18–19 hours	Definitive primitive streak, 1.88-mm long
5	19–22 hours	Head process (notochord)
6	23–25 hours	Head fold
7	23–26 hours	1 somite; neural folds
7–8	ca. 23–26 hours	1–3 somites; coelom
8	26–29 hours	4 somites; blood islands
9	29–33 hours	7 somites; primary optic vesicles
9+ to 10–	ca. 33 hours	8–9 somites; anterior amniotic fold
10	33–38 hours	10 somites; 3 primary brain vesicles
11	40–45 hours	13 somites; 5 neuromeres of hindbrain
12	45–49 hours	16 somites; telencephalon
13	48–52 hours	19 somites; atrioventricular canal
13+ to 14–	ca. 50–52 hours	20–21 somites; tail bud
14	50–53 hours	22 somites; trunk flexure; visceral arches I and II, clefts 1 and 2
14+ to 15–	ca. 50–54 hours	23 somites; premandibular head cavities
15	50–55 hours	24–27 somites; visceral arch III, cleft 3
16	51–56 hours	26–28 somites; wing bud; posterior amniotic fold; trigeminal ganglion (CN V) close lateral head ectoderm
17	52–64 hours	29–32 somites; leg bud; epiphysis
18	3 days	30–36 somites extending beyond the level of leg bud; allantois
19	3.0–3.5 days	37–40 somites extending into tail; maxillary process
20	3.0–3.5 days	40–43 somites; rotation completed; eye pigment
21	3.5 days	43–44 somites; visceral arch IV, cleft 4
22	3.5–4.0 days	Somites extend to tip of tail
23	4 days	Dorsal contour from hindbrain to tail is a curved line
24	4.5 days	Toe plate, eye primordium reaches optic cup stage
25	4.5–5.0 days	Elbow and knee joints
26	5 days	First 3 toes
27	5.0–5.5 days	Beak
28	5.5–6.0 days	3 digits, 4 toes
29	6.0–6.5 days	Rudiment of fifth toe
30	6.5–7.0 days	Feather germs; scleral papillae; egg tooth

Continued

BOX 38.5 HAMBURGER AND HAMILTON DEVELOPMENTAL STAGES IN CHICKEN BY DAY OF INCUBATION—cont'd

Hamburger and Hamilton Stages	Age	Identification of Stages
31	7.0–7.5 days	Web between first and second digits
32	7.5 days	Anterior tip of mandible has reached beak
33	7.5–8.0 days	Web on radial margin of wing and first digit
34	8 days	Nictitating membrane
35	8.5–9.0 days	Phalanges in toes
36	10 days	Length of third toe from tip to middle of metatarsal joint = 5.4 ± 0.3 mm; length of beak from anterior angle of nostril to tip of bill = 2.5 mm; primordium of comb; labial groove; uropygial gland
37	11 days	Length of third toe = 7.4 ± 0.3 mm; length of beak = 3.0 mm
38	12 days	Length of third toe = 8.4 ± 0.3 mm; length of beak = 3.1 mm
39	13 days	Length of third toe = 9.8 ± 0.3 mm; length of beak = 3.5 mm
40	14 days	Length of beak = 4.0 mm; length of third toe = 12.7 ± 0.5 mm
41	15 days	Length of beak from anterior angle of nostril to tip of upper bill = 4.5 mm; length of third toe = 14.9 ± 0.8 mm
42	16 days	Length of beak = 4.8 mm; length of third toe = 16.7 ± 0.8 mm
43	17 days	Length of beak = 5.0 mm; length of third toe = 18.6 ± 0.8 mm
44	18 days	Length of beak = 5.7 mm; length of third toe = 20.4 ± 0.8 mm
45	19–20 days	Yolk sac half enclosed in body cavity; chorioallantoic membrane contains less blood and is "sticky" in living embryo
46	20–21 days	Newly hatched chick

Modified from https://embryology.med.unsw.edu.au/embryology/index.php/Chicken_stages.

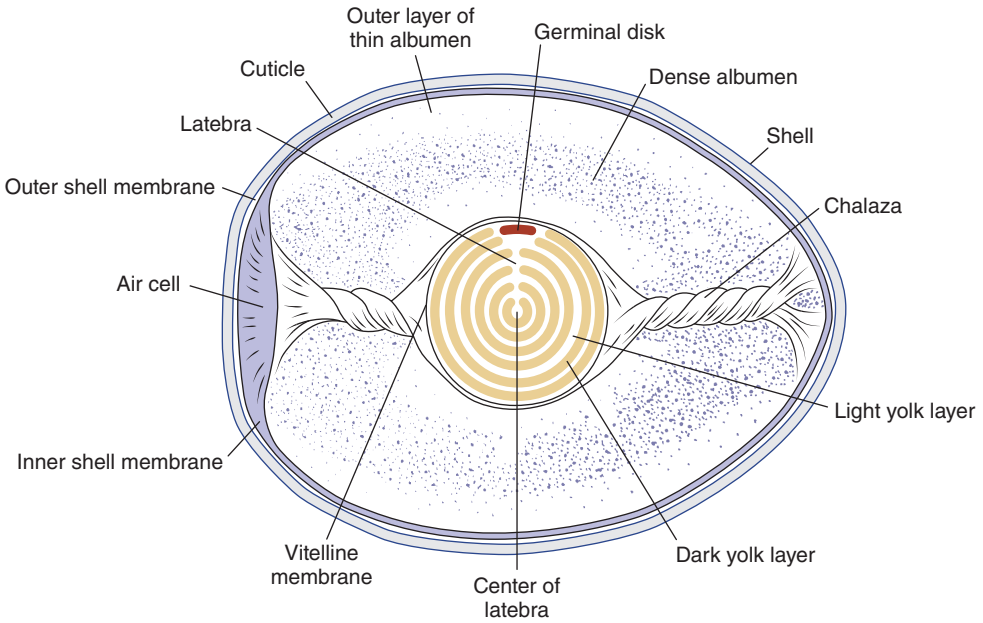


FIGURE 38.3 Diagram of the anatomy of the egg.

HATCHING

Many significant changes in metabolism occur during the last days of incubation as the embryo prepares for and ultimately hatches successfully. Hatching is a complex process, and not surprisingly, there can be a peak of embryonic mortality seen during the hatch. Unlike what is seen in mammals, essentially all the stimuli are generated by the embryo itself, though it can respond to stimuli from both the parents and the other members of the clutch.[40] However, which of these signals trigger shifts in metabolism and hormone release during this critical process has not been completely elucidated. Nevertheless, it has been suggested that thyroid hormones together with glucocorticoids play an important role.[41] Uncovering these mechanisms may yield creative ways to manipulate physiological or nutritional conditions to improve hatchability, survival, and vigor of hatchlings.[24]

There are three major events in the sequence of hatching: 1) the onset of pulmonary respiration; 2) pipping (i.e., the creation of a single point of fracture in the shell, usually in that overlying the air space), and 3) the emergence of the hatchling from the egg. However, underlying these more obvious events are many others, such as maturation of muscles, changes in the circulation, and withdrawal of the yolk sac, which all occur in a particular sequence to help ensure a successful outcome of incubation.

The egg, with its hard shell, does not enable embryonic ventilatory movements, and thus there is no convective gas exchange until the embryo's lungs begin to function. Early in incubation, prior to the formation of the heart and even initially after the detection of embryonic heartbeats, oxygen can be adequately supplied from the environment to the embryo through diffusion. However, when gas transport by diffusion alone becomes inadequate, blood convection begins to meet oxygen demand during development, with three different gas exchangers sequentially functioning in the egg: the area vasculosa, the chorioallantois, and the lungs.

The area vasculosa is a well-vascularized region of the yolk sac that fans out from the embryo and rapidly expands around the yolk during the third to fifth days of incubation (Figure 38.4).[42] Starting at day 2, the blood vessels of the yolk sac connect with the dorsal aorta of the chick embryo, following which blood begins to circulate through the embryo and area vasculosa. The fine reticulation of the vitelline vessels serves as the main source of gas exchange during this first period until the time that the chorioallantois connects with the inner shell membrane at approximately day 6. At that time, the respiratory function is taken over by the outer surface of the chorioallantois, the chorioallantoic membrane (CAM).[43] At this point in time, gas exchange takes place by diffusion across the porous shell, the shell membranes, and capillary blood of the CAM. The CAM continues to function as the main gas exchanger during the prenatal development until the embryo pips it and the inner shell membrane (internal pipping) and it degenerates.[43] Once the shell is broken by the chick (external pipping), the lung ventilation gradually takes over and the chick starts breathing in environmental air. Unique to birds and reptiles is the passing through a period where both the embryonic and adult respiratory surfaces, the chorioallantois and the lungs, function side by side. This period is conveniently described as the parafetal period, whereas the chick at this stage of development is referred to as the parafetus.[44]

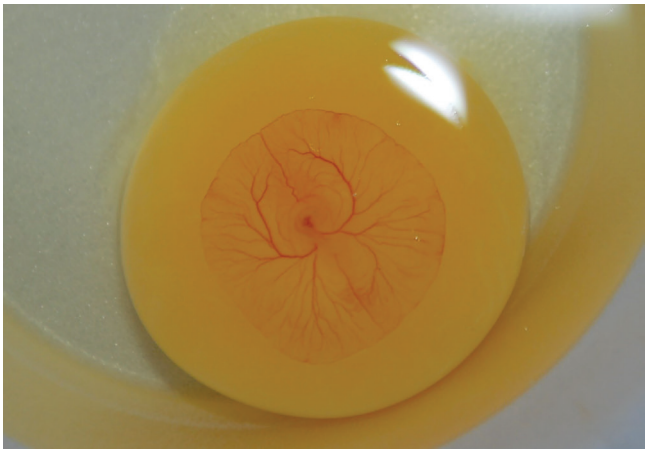


FIGURE 38.4 In this fertile egg, the area vasculosa is visible, as it rapidly expands out and over the yolk. (Photo courtesy Steve Duncan http://www.avianresources.com/.)

A prerequisite for breathing is the removal of the fluid present within the respiratory tract. This fluid primarily comprises amniotic fluid, but within the lungs an ultrafiltrate of the embryo's blood may also be present. The uptake of amniotic fluid, by active imbibition, generally starts when 70% of the incubation period has elapsed and is completed just prior to the initiation of breathing (i.e., approximately 1 day before hatching in domestic fowl).[40]

Early attempts to identify the pulmonary stimulus for respiratory movement indicated that it is probably gaseous and that possibly a high partial pressure of carbon dioxide in the arterial blood ($PaCO_2$) is the responsible trigger. This was primarily based on the observation that respiratory movements could be initiated by a 12-mm Hg PCO_2 rise in the atmosphere surrounding the egg whereas a reduction of 68 mm Hg in PO_2 was required to produce the same effect.[45] However, other authors have argued that either enhanced PCO_2 or reduced PO_2 in the space surrounding the egg, or the combination of both, can be responsible for stimulating breathing.[46] However, perforating or ventilating the air space fails to delay the onset of embryonic breathing, which renders this an unlikely possibility. Later work has shown that, toward the end of the embryonic period, the $PaCO_2$ may reach a very high level (~60 mm Hg),[47,48] while PaO_2 is relatively low (~20 mm Hg).

Sound stimuli also have a role in the initiation of breathing and hatching. For example, the hatching process can be substantially advanced by a "clicking" sound. This suggests that under certain circumstances the gaseous stimulus for breathing can either be initiated earlier or overridden by the sound stimuli. Similarly, an earlier initiation of pulmonary respiration by sound stimulation has been observed in the fowl, duck *(Anas platyrhynchos)*, goose *(Anser anser)*, and Japanese quail, but not in the bobwhite quail *(Colinus virginianus)*.[49,50] However, despite sound stimulation leading to an earlier initiation of breathing, no clear differences in $PaCO_2$ or PaO_2 could be identified.[51] In the end, it is now a common opinion that although a high $PaCO_2$ is one of the strongest stimuli to start the pulmonary activity, there are other triggers to respiration that play an important role in the onset of active breathing.

EGG WEIGHT LOSS

Throughout the development of the embryo, the egg steadily loses water by transpiration through the CAM. This water loss is a linear and consistent event throughout the entire incubation process. Because of this loss of water and the loss of yolk fats metabolized during development, the egg is much lighter at hatching than when it was laid. In most avian species, the weight loss is approximately 15% of the original weight of the egg (Figure 38.5). The eggshell, too, is thinner than when it was laid because the chick has absorbed much of the calcium from the inner shell lining. In the imminent hatching period, the chick absorbs the remainder of the yolk sac into the abdominal cavity and begins to swallow any amniotic fluid that remains.[24] During this period, there is an escalated rate of weight loss, as shown in the weight loss graph

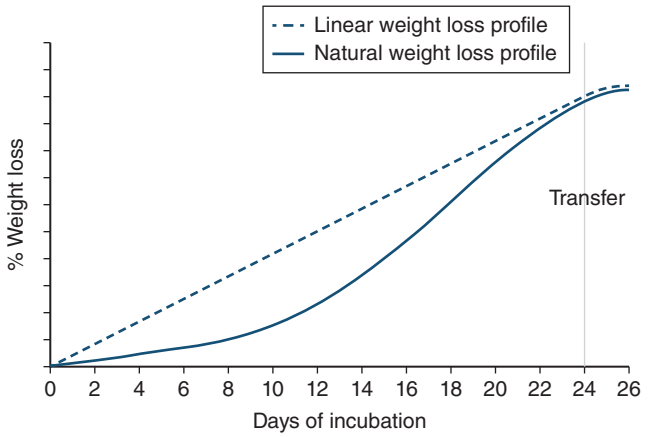


FIGURE 38.5 Natural weight loss of an egg as compared to direct linear weight loss over the incubation period. Weight loss of an egg during a 26-day incubation period, showing a completely linear weight loss of 15% *(dashed line)*, as compared to actual egg weight loss *(solid line)*. Although weight loss ultimately is the same, it is not a completely linear process over the entire course of incubation.

in Figure 38.5. Both excessive and inadequate weight loss can lead to embryonic morbidity and mortality (see Chapter 40).

PIPPING AND THE HATCHING PROCESS

In birds, pipping involves the process of breaking open the eggshell. Two specialized structures that are only found in hatchlings aid the chick in its struggle to break open the shell. The first is a small, sharp "egg tooth" that develops on the dorsal tip of the rhinotheca and is used to "pip" and "cut" out of the shell. The second is a substantial enlargement of the complexus muscle (pipping muscle) that is in the proximal dorsal cervical region and helps to brace the neck and cushion the head as the chick forces the egg tooth through the shell (Figure 38.6).[52] After hatching, most of the fluid within the



FIGURE 38.6 One-day-old hyacinth macaw chick *(Anodorhynchus hyacinthinus)*. The complexus muscle is identified *(black arrow)*, and the egg tooth is marked with the *red arrow*. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

complexus muscle is reabsorbed, and this muscle continues to function as an extender of the head in most adult birds. The egg tooth is also lost in the first few weeks following hatching.[24]

Immediately prior to the start of the hatching process, the air cell expands to encompass approximately 20% to 30% of the total internal egg volume. This change can be noted by candling and is referred to as the "draw down." At the start of hatching, the beak of the embryo penetrates the inner shell membrane to gain access to the air cell (i.e., the internal pip), and the lungs become functional by inspiring air from the air cell.

The external pip occurs when the chick cracks or cuts the outer shell membrane and shell. In fowl, this occurs approximately 8 or 9 hours after breathing is established. At this point in the hatching process, the lungs are meeting about 40% of the total oxygen requirements of the parafetus, and the parafetus might be expected to face increasingly hypoxic conditions. Just prior to pipping, the PO_2 in the air space may fall to as low as 60 mm Hg, while the PCO_2 may reach 60 mm Hg.[53] Pipping, like breathing, seems to result from rising PCO_2 in the air cell. Nevertheless, stimuli other than oxygen and carbon dioxide also seem to play a role in the external pipping process, and at current there is consensus that the hatching stimulus is not gaseous alone. The suggestion for hormonal involvement was strengthened by the observation that thyroid hormones accelerate the hatching process and specifically stimulate active hatching.[54–56] It has also been suggested that progesterone is the hormone responsible,[57] though Oppenheim has been unable to repeat the experiments to support this claim.[56] The musculus complexus has also been ascribed an important role in the hatching processes.[58–60] This muscle shows marked development just before hatching begins and acts to raise the head of the parafetus, presumably providing most of the force necessary for breaking down the shell. The timing of its maturation seems to be unique but attempts to explain this timing have thus far largely failed.[58–60]

At the onset of breathing, the oxygen requirements of the parafetus are met by the combined activities of the chorioallantois and lungs. During hatching, the oxygen requirements of the parafetus further rise. Typically, a domestic fowl parafetus consumes 25 mL/hour prior to breathing, 35 mL/hour 1 prior to active hatching, and approximately 40 mL/hour at the time of emergence from the shell. After this, a further substantial rise occurs.

The parafetus's requirement for carbohydrates also increases markedly during the hatching process, and a dramatic mobilization of glycogen stores has been recorded shortly before breathing commences. It is likely that the glucose that is made available through mobilization of these glycogen stores is necessary for the proper functioning of the central nervous system. A marked rise in the glycogen stores of the brain has been noted to occur at this time.[61] During this period, the overall respiratory quotient does not show any increase, however, remaining at about 0.7, consistent with an absence of hypoxia during the bulk of the hatching process.[40] A transient period of mild hypoxia is indicated by the fall in cardiac glycogen stores at the time of pipping.[62]

General activity, including that of the "hatching" muscle *(musculus complexus)*, increases as the parafetus moves further into the air space. Eventually, the beak contacts the calcareous

shell, and the enhanced muscular activity leads the egg tooth—the reinforced tip of the upper beak (see Thiebold and Bucher)[52]—to penetrate it. Once the shell is pipped, the parafetus enters a more quiescent period, at least in terms of physical activity, which persists until 3 or 4 hours before the chick emerges from the shell. The final activity of the parafetus is directed toward cutting round the shell and the emergence of the chick.

In domestic fowl, a period of about 20 hours elapses between pipping and hatching. In nondomestic bird species, this time frame from internal pipping to hatch can normally range from 6 to up to 48 hours. Most parrot species normally hatch within a 24-hour period from internal pip to completed hatch (see Chapter 40, Table 40.2). The hatching process is discussed in Chapter 40.

TRANSMISSION OF DISEASE THROUGH THE EGG

Infectious agents can be transmitted to the embryo vertically, or horizontally through the eggshell barrier at some point following oviposition or during incubation. Transmission of an infectious agent from a parent to an egg during fertilization, during development in the oviduct, or immediately after laying is termed vertical transmission. Once the egg is in the nest, some agents can pass through the eggshell upon contact with contaminated feces, urates, or bedding, termed horizontal transmission.[63]

Most of the information about transmission of avian pathogens we know or speculate from the data available about the infectious diseases of poultry. In poultry, vertically transmitted viral agents include chicken anemia virus, egg drop syndrome 1976 virus, fowl adenovirus, avian encephalomyelitis virus, avian leukosis viruses (genetically transmitted retroviruses), and potentially avian influenza and Newcastle disease viruses (but these usually kill the embryo and do not make it to the next generation) (P. Groves, personal communication, 2020).[64] Bacterial agents capable of vertical transmission include *Mycoplasma* (*Mycoplasma gallisepticum*, *M. synoviae*, and *M. meleagridis*), *Salmonella* (*Salmonella pullorum* and *S. gallinarum* classically, but also *S. enteritidis* and *S. typhimurium*), and *Ornithobacterium rhinotracheale*.

In other avian species, we have a lesser understanding of how and when infectious agents are transmitted to the embryo or to the chick. Apart from the assumption that some pathogens behave in the same way in nonpoultry species as they do in poultry, there are some specific infectious agents that have been reported to be transmitted vertically.

Avian Bornavirus

The first evidence to support the presence of vertical transmission was found in 2011 when the embryos of avian bornavirus (ABV)-positive psittacine parents were tested positive for ABV-RNA in the brain (see Chapter 14 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Similarly, 10 eggs out of 61 eggs obtained from a psittacine flock with proventricular dilatation disease (PDD)-affected birds within it contained ABV-RNA either in the yolk or in the brain of two embryos. However, all embryos developed uneventfully, and no inflammation typical for PDD was seen, demonstrating that vertical transmission of the virus will not automatically lead to development of disease, and may—if at all—only occur when certain, currently unknown cofactors are present. Therefore, from clinical observation, the route of transmission, at least the circumstances of a successful transmission, is currently not fully understood, though vertical transmission has recently been suggested to play a role in development of carriers (see Chapter 14 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Nevertheless, many factors still remain poorly understood, including the abilities and means by which ABV can overcome skin or mucosal barriers, as well as the influence of viral factors (e.g., pathogenicity of different genotypes) and host factors (e.g., immunosuppression, incompetency in juvenile birds, or other immunologic variables) in the development of disease. However, both the irregular horizontal transmission and the vertical transmission of ABV open large potential opportunities in preventing further spread of the virus and clearing flocks of ABV.[65–67]

Chlamydiosis

Vertical transmission through the egg has been demonstrated in the domestic duck, in the black-headed gull *(Chroicocephalus (Larus) ridibundus)*, in the budgerigar *(Melopsittacus undulatus)*, and is suspected to occur in the turkey.[68]

Psittacine Circovirus (Psittacine Beak and Feather Disease Virus [PBFDV])

There is no experimental evidence that has conclusively confirmed vertical transmission rather than horizontal transmission to the embryo via cloacal secretions and nesting material. If vertical transmission occurs, it is unlikely to be a significant mechanism for circovirus maintenance in populations. Vertical transmission is less likely as its presence would be more likely to be a deep force for virus-host coevolution.[69]

Avian/Psittacine Polyomavirus

Experimental data and observations with the natural disease suggest that polyomavirus transmission in budgerigars may occur by both horizontal and vertical routes (both among members of a flock and between generations from parent to offspring). Vertical transmission of polyomavirus has been confirmed in budgerigars but has not been confirmed to occur in other psittacine birds.[70]

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CHAPTER 39

Natural Incubation

Alessandro Melillo

INTRODUCTION

Oviparity or egg laying is the most common reproductive strategy in animals: invertebrates, fish, amphibians, and reptiles, like birds, produce eggs in which the embryo develops. Unlike other zoological groups such as crocodiles, lizards and tortoises, birds generally do not abandon their eggs with trust that the physical and chemical parameters provided by their resting environment will enable the development environment. Instead, birds actively create the correct conditions for the growth of the embryo around their eggs. This process is defined as *incubation* and corresponds to the gestation of mammals. Unlike mammals, the developing embryos are exposed to additional risks outside of the female's body, but at the same time, this allows for joint efforts to be made by both sexes in the care for their offspring (in addition to the obvious advantage of not burdening the female with pregnancy).[1]

INCUBATION AND BROODING

Incubation is the process by which oviparous animals hatch their eggs. For most species, either or both parents typically sit on the eggs, heating and moving them: this action is defined as *brooding*.[1] Exceptions to this type of incubation are the Australian megapodes (order Galliformes, family Megapodidae), which build a kind of incubator from rotting plant material which in turn allows for fermentation to produce the heat necessary for the development of the embryo.[1] Crab plovers *(Dromas ardeola)*, on the other hand, make partial use of heat from the sun to cut down on parental care.[2] Brooding duties vary among species. As examples, in emu *(Dromaius novaehollandiae)* only the male is responsible for brooding, whereas for the orders Galliformes, Anseriformes, and several Passeriformes, hens are primarily responsible for brooding, and both genders are involved in brooding for almost all Psittaciformes and Columbiformes.[1,3] In the parasitic bird species (e.g., common cuckoo, *Cuculus canorus*), instead of the biological parents sitting on the eggs, brooding is conducted by other species.[1]

Brooding behavior is mainly signaled by prolactin. Changes in this hormone will result in behavior changes, with most birds becoming more shy or secretive.[4] However, certain individuals or species are known to become more aggressive and fiercely defensive of their nesting site (Figure 39.1).[4] Aside from prolactin, visualization of the clutch of eggs in the nest serves as another antecedent to brooding behavior. Birds in general show a strong attraction for their eggs and egg-shaped objects, although species differences exist. Sphenisciformes (penguins), for example, attend their eggs, but in the absence of them, may also incubate stones. Passeriformes, on the other hand, are usually more bonded to the nest and remain indifferent to the eggs when they are outside of the nest itself.[1]

Certain species begin to brood as soon as the first egg is laid, to protect it from environmental factors. This occurs in birds of the orders Falconiformes, Strigiformes, Psittaciformes, and Pelicaniformes (family Ardeidae: herons; family Pelicanidae: pelicans).[1] A consequence of this is that chicks will hatch at different ages, and hence will have different strengths. Particularly in birds of prey (e.g., eagles), this results in the older and more vigorous chicks being better positioned to successfully compete for food brought by the parents, while their younger siblings starve or are even eaten by them. Conversely, should something unexpected happen to the eggs or the older chick(s), the younger chick(s) will be capable of functioning as a reserve, preventing a total failure of the brood. In other bird groups, the ability to respectively focus on more sophisticated parenteral care for all hatchlings of the clutch (Psittaciformes) or produce different densities of crop milk (Columbiformes) is usually beneficial to allow all chicks to grow.[1,3,5] In contrast to the aforementioned bird orders, Galliformes, Anseriformes, and most Passeriformes start brooding after deposition of the last egg, so that all chicks hatch at a similar time.[1] This is particularly useful for species with precocial offspring (Galliformes, Anseriformes) because the chicks can all start to follow the parents simultaneously (see Chapter 45).

Brooding not merely involves "sitting on eggs" but is an active process designed to transfer heat to the eggs. This is accomplished through "incubation plaques" or "brooding patches," which are areas of thickened and highly vascularized featherless skin on the ventral body surface that are in direct contact with the eggs.[1,5] Down feathers fall from pterylous tracts or are selectively plucked before laying the first egg; then capillary blood flow to the dermis is selectively increased leading to local edema under the influence of estradiol (which induces molting of feathers) and prolactin (which modifies vascularization of the area).[1,4,5] After brooding is completed, the patches will regress. Brooding patches have different distributions among species. In Columbiformes, Falconiformes, and many songbirds there is only one (median) incubation patch which corresponds to the central apterium.[1] Conversely, there are two incubation patches in auks (Charadriiformes; Alcidae), and Galliformes; and gulls (Charadriiformes, Laridae) have three patches in total. Other groups of birds, including ducks and geese



FIGURE 39.1 A blue-and-gold macaw *(Ara ararauna)* broods and protects her clutch of three eggs in her artificial nest box. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

(Anseriformes), do not possess incubation patches.[1] Instead, these species actively pull feathers from their chest region to allow the exposed skin to be placed in contact with the eggs. Gannets (Pelicaniformes, Sulidae) wrap their webbed feet, which are richly vascularized, around the egg, and the emperor penguin *(Aptenodytes forsteri)* places a single egg on its feet and then covers it with a special skin fold.[1]

The temperature at the level of the brooding patch is close to 40°C (104°F) and induces a temperature of 34°C (93.2°F) on the egg. However, as only part of the egg is in direct contact with the brood patch, a temperature gradient develops between the parts that are in direct contact with the body and the part that rests in the nest. In addition, temperature differences can be seen between the eggs that occupy a central position in the nest versus those that are positioned more to the periphery. To facilitate uniform distribution of heat and gas exchange through the pores of the shell, the eggs are therefore also periodically turned by the brooding parent. In addition, the eggs' position within the nest is also varied and rotated. The frequency with which these activities are performed is highly dependent on the species. Combined, these parental actions all are designed to have direct benefit in aiding optimal embryo development.

Birds that nest outdoors may also need to protect their eggs from overheating by the sun. This may be accomplished by standing over the nest and opening wings as an umbrella, as is seen in sandgrouse (*Pterocles* spp.).[1] The humidity that the eggs are being brooded at can be adjusted through the addition of green leaves to the nest, bathing, and moistening plumage before returning to the nest, or regurgitating water directly on the eggs. Some species cover the nest with leaves when they leave, either for the purpose of camouflage or to reduce fluctuation of incubation parameters.[1] Those species that nest in cavities can rely on the benefits of a more stable microenvironment provided by the nesting cavity.

Eggs are known to withstand drops in temperature, particularly at the beginning of incubation.[6] This durability is often necessary to allow the brooding parent to get away in case of danger or to search for food. In species where both parents share the brooding duties, eggs are rarely, if ever, left alone. However, in species where a single parent broods, the eggs must endure periodic temperature changes, directly proportional to the resistance to fasting and continual brooding demands. For example, hummingbirds (Apodiformes) leave the nest every few minutes, while the albatross (Procellariiformes, Diomedeidae) can brood continually for weeks before being replaced by its mate. The emperor penguin can brood for up to 2 months. Brooding birds are aware of the environmental temperature fluctuations and will leave the nest less often in cold weather.[1]

For species in which both parents brood, the division of incubation duties across the parents can vary. In these species, individuals will take turns at intervals measured in hours or days. The exchange of duties is often associated with characteristic innate behavioral displays of the species.[1] Among Columbiformes, the male broods during the day and the female at night, while the opposite occurs in ostriches *(Struthio camelus)*. In Passeriformes, generally the female has a predominant role in brooding, while the male feeds her and guards the nest. However, in some passeriform species, the male also actively participates in incubation. In other taxonomic groups, for example in Galliformes, incubation and brooding activities are almost entirely left up to the female. These hens typically fast for the entire incubation period or leave the nest minimally to feed. In the rhea (Rheiformes, Rheidae), the male is exclusively responsible for incubation and brooding. Exclusive male incubation and brooding responsibilities are also noted in the little spotted kiwi *(Apteryx owenii)* and brown kiwi *(Apteryx mantelli)*, the emperor penguin, jacanas (*Jacana* spp.), and polyandric species ((e.g., Charadriiformes).[7] The red-legged partridge *(Alectoris rufa)* shows a quite peculiar behavior: the female lays two consecutive broods, leaving the male in charge of one and herself of the second. In hoatzin *(Opisthocomus hoazin)*, other individuals (mainly males) help their parents with the brooding of later clutches. Incubation periods vary from 12 days in many small passerine species to 65 days in the emperor penguin, and up to 80 days in the brown kiwi and some great albatross species (*Diomedia* spp.).[1]

THE NEST

A fundamental condition for the reproductive success in birds is a nest: this is the location where the eggs are laid and incubated and where altricial chicks dwell until they are fully developed. The nests of birds can in essence be very simple—a few twigs piled up like those built by most Columbiformes, or a hole in the gravel as created by different wader birds. Some birds, such as tropical terns *(Gygis alba)*, do not even use a nest but instead deposit their single egg balanced on a branch. Alternatively, nests can be wonderfully complex, such as those of the penduline tit (*Remiz* spp., *Anthoscopus* spp.) or the African weavers (*Ploceus* spp.).[1] Most parrot species nest in cavities, usually in tree trunks but also in clay walls, termite mounds, or cactus drums. An exception is the monk parakeets *(Myiopsitta monachus)*, which breed in colonies and build large complex spherical nests of branches, hanging from trees or artificial structures.[3,6]

In captive breeding conditions, the provision of a nesting site that is appropriate and acceptable to the species and concurrently allows for individual preferences is often key to success.[3,5] Geese (*Anser* and *Branta* spp.) and several ducks

(*Anas* spp.), for example, build a rudimentary nest of grasses under a shelter (e.g., barrel, dog house, or simple wooden plank) but may also do so in the grass or the bushes. Many other ducks, such as the wood and mandarin ducks (*Aix* spp.), require real nest boxes raised at least 0.5 m (~1.64 feet) from the ground. In Anseriformes, incubation is reserved exclusively for females, but males of different species remain near the nest and protect it.[5]

Parrots are generally cavity nesters and in captivity typically require different types of nest boxes.[3,6] As is seen in most of the cavity-nesting species, parrots lay white eggs, often two to four in number. Smaller parrot species such as lovebirds (*Agapornis* sp.) or budgerigars *(Melopsittacus undulatus)* commonly lay even more eggs per clutch. As the larger parrot species can be very destructive toward wooden nests, many breeders use metal containers or metal-lined nest boxes. This solution is not ideal, as those chewing and destructive behaviors are a component of the adaptive reproductive strategy for many species. The pair's demonstrated desire to change the environment to make it suitable for breeding is important for many to effectively synchronize reproductive endocrine biology. For many psittacine species, including members of the Cacatuidae family and the *Pionus* and *Eclectus* genera, the process of enlarging and modifying the entry of the nest is an important component of nest site preparation to help achieve and synchronize reproductive status of the breeding pair. For this reason, aviculturists often apply slats of wood or bark to partially occlude the entrance to the nest to meet this need while preventing excessive damage to the nest box. Perhaps even more appropriate is the provision of multiple nests in different shapes, sizes, and locations, and made of different materials, to allow the pair to choose a preferred nesting site. The main styles of nest boxes are vertical, horizontal, and L-shaped (both vertical and horizontal; Figures 39.2–39.5). Vertical nests should have an internal ladder made from wire mesh under the entrance hole to prevent birds from jumping inside and breaking the eggs. Alternatively, nests can be hung



FIGURE 39.2 A vertical nest box design. This red-headed Amazon hen *(Amazona viridigenalis)* looks in on her chicks from the wire provided as a ladder below her entry hole in a vertically designed nest entrance approximately 1 m (3 feet) higher up. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)



FIGURE 39.3 Vertical nest box design with demonstration of the vertical wire ladder extending from the nest entrance to the actual nesting site for the eggs and chicks. This nest design can be placed vertically as depicted, horizontally, or even diagonally. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)



FIGURE 39.4 Diagonal placement of the vertical nest box design for a breeding pair of red-and-green macaws *(Ara chloropterus)*. The nest site entrance is on the elevated end to the right, and the selected nesting site is typically in the lower corner on the left. An access to the nest site for inspection can be seen on the lower outer wall. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

on the diagonal, but those in vertical L-shapes are more often preferred by most pairs, and equally effective. Horizontal nests are often preferred by smaller-sized species.

Few species, including the budgerigar, lay on the bare wood; however, many others will require some kind of padding. This padding can be transported by the birds from external sources. Lovebirds, as an example, transport willow bark, dry grass, palm leaves, and other frayed plant materials. Padding for the nesting site can be produced within the nesting site itself or by allowing the birds to chew on the inside



FIGURE 39.5 Construction of a vertical L-shaped nest box. The entrance is elevated at the top, with a wire ladder provided to allow the birds to access the lower nesting chamber at the bottom. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

walls of the nest or other provided wood items (Figure 39.6). To avoid damage to the nest box, softwood shingles can be screwed to the inner walls for the birds to chew. The floor of the nest box can also be covered with wood chips, crumbled bark, or coconut fiber.[3,5]

Some psittacine species have special nesting needs. The palm cockatoo *(Probosciger aterrimus)* nests in old hollow trees



FIGURE 39.7 A view down into a natural, vertically positioned log nesting site, as may be utilized for breeding the palm cockatoo *(Probosciger aterrimus)* and some other species. Eggs and chicks are visible at the bottom. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

that are open at the top and will rarely accept the more common or traditional parrot nesting boxes. There are testimonials of pairs that have nested in bins or barrels, but in general they require natural nests made of hewn logs (Figure 39.7). Other unique reproductive strategies of species include those that lay a single egg and build a nest of branches within the cavity. In the wild, keas *(Nestor notabilis)* nest in cavities of rocks. In captivity, they usually prefer to lay their eggs on the ground rather than using regular nest boxes. A simple compromise may be to place a nest box at about 0.9 m (3 feet) to the side on the ground and cover it with stones or build the structure with cement blocks. A fundamental element for the

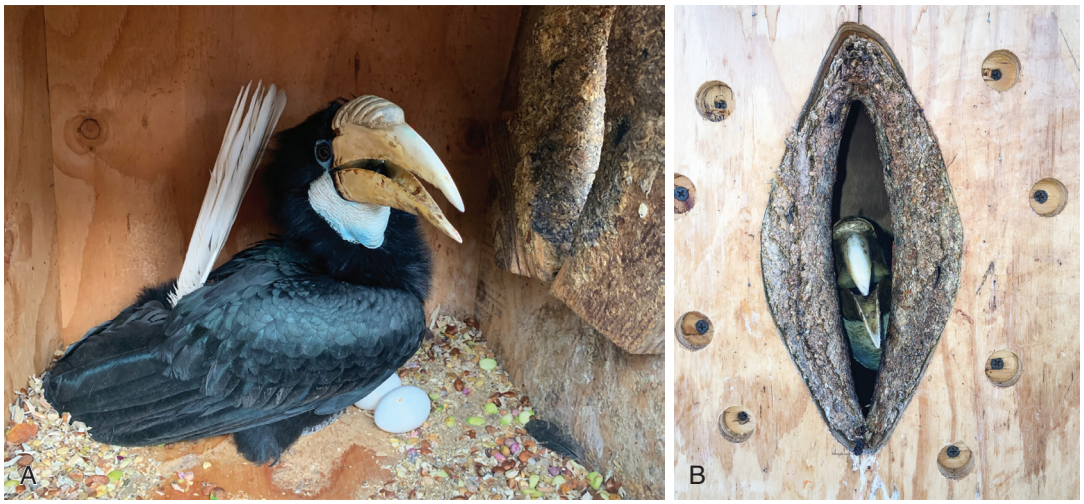


FIGURE 39.6 (A) Papuan or Blyth's hornbill *(Rhyticeros plicatus)* hen in an artificially constructed nesting site. The wood around the entrance was added to give a larger surface for the bird to pack additional material onto. The material packed inside and outside of the entrance is mostly feces, fruit pulp, and some fibrous material such as wood shavings. The nest entrance is low enough for the female, and later the babies, to back up to and squirt droppings out so that they do not accumulate in the nest. The fruit pits seen on the floor of the nest are mostly loquat pits *(Eriobotrya japonica)*, which are often fruiting while the species generally go to nest. (B) An exterior view of the same hen in the nesting site. (Photos courtesy Steve Duncan, http://www.avianresources.com/.)

nest of the kea is a tunnel for access, which may be simulated by placing a concrete or wooden pipe of about 50 cm (20 inches) in diameter and 50 to 100 cm (20–40 inches) in length in front of the nest opening. The tunnel allows the female to prevent the male from getting into the nest. The red-headed lovebird *(Agapornis pullarius)* also has special nesting needs that involve nesting in termite mounds. To induce nesting for this species in captivity, nest boxes should be filled with sheets of cork through which the female can dig a tunnel to access the nest chamber.[3]

Columbiformes build very rudimentary nests and are often content to accumulate a few sprigs over an intersection of branches or in a basket. The domestic pigeon *(Columba livia)*, which nests in cavities, will take advantage of almost any hollow structure including holes in the wall, mezzanines, balconies, or cassettes. More exotic species of doves and pigeons generally nest in bushes and trees and will assemble collected twigs on intersections of branches or in baskets. Several species are slow and reluctant to start nesting and breeding, but once the right conditions are identified, most will reproduce well in captivity. Male and female Columbiformes share brooding responsibilities, with the male usually brooding during the day and the female at night. The incubation period is often short in Columbiformes, lasting only around 2 weeks, and begins immediately after the deposition of the first egg, so that chicks hatch at different ages.[5]

Among Passeriformes, there are vastly different incubation strategies. In the commonly bred finch species, which includes the canary *(Serinus canaria)*, the female builds a cupped nest using plant material, which she lines with (horse) hair, and other soft materials. Estrildidae, which includes the Australian finches and the small African waxbills, build nests in cavities. These differences in nesting strategy are reflected in the appearance of the eggs: whereas finch eggs are colored gray and pale blue streaked with red brown, the eggs of cavity-nest builders are white in color, and chicks typically have luminescent growths around and inside their beak that serve as helpful cues for their parents to feed them. Crows (Corvidae) build bulky nests together in trees, but only the female will incubate the speckled, green-to-brown eggs. Titmice (Paridae) and nuthatches (*Sitta* sp.) are cavity nesters but often build an elaborate and padded nest. Similarly, mousebirds (*Colius* and *Urocolius* spp.), belonging to the ancient order of Coliiformes, build a cup shaped twig nest in thick bushes and trees, and will accept proper sized baskets in captivity (Figure 39.8). Among Leiothrichidae, the Pekin robin *(Leiothrix lutea)* is quite commonly bred. In this species, the male is typically responsible for building the nest, which is suspended from a horizontal branch and created from dried herbs and lined with herbs and animal hair. In captivity, these birds will accept baskets or net hammocks but are not interested in nest boxes of any kind. The female incubates the lightly spotted, pale greenish to bluish eggs during the night, whereas male and female frequently take turns during the day. Among Sturnidae, the hill mynah *(Gracula religiosa)* will use either vertical or horizontal boxes, building a messy nest from leaves, dry grasses, or other materials. Incubation of the pale blue, dark spotted eggs is carried out by both pair members and lasts 14 days. The Bali mynah *(Leucopsar rothschildi)* is bred in a similar manner.[5]



FIGURE 39.8 This white-backed mousebird *(Colius colius)* is shown nesting in an open basket. The nest is typically hidden in a thicket. Nestlings are fed by both parents as well as helpers, which are usually young birds from previous clutches. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

REPRODUCTIVE PARASITISM

Some bird species rely on pairs of an entirely different species to incubate their eggs and raise the young, referred to as brood parasitism.[1] Often the parasitic species destroys the eggs of the host so that their young are the only ones being cared for, but occasionally the eggs of the host are left alone to allow the foster parents to brood both egg types and raise the chicks together in the same nest. Nest parasitism is reported in Cuculiformes (Cuculidae), Piciformes (Indicatoridae), Passeriformes (Icteridae, Ploceidae), and Anseriformes (Anatidae). Within the order Anseriformes, only the South American black-headed duck *(Heteronetta atricapilla) shows nest parasitism, whereby the female* lays eggs in the nests of other ducks (especially the rosy-billed pochard, *Netta peposaca*), swans, coots, gulls, ibis, and other species. The ducklings, which are already independent a few hours after birth, move away from the host's nest once hatched and continue to live by themselves. Among Cuculidae, European species are highly specialized in brood parasitism, while almost all the American species reproduce normally. After mating, the female of the common cuckoo searches for nests under construction by songbirds; identification of an available nest site subsequently triggers ovulation. A single female can produce a dozen eggs at intervals of 48 hours and deposits one egg per parasitized nest at a time that foster parents leave their nest unattended. The egg is either laid directly in the nest or laid onto the ground and then transported to the targeted nest by the female using her beak. Once the egg is in the nest, the female cuckoo continues observing the nest to ensure that her eggs are incubated by the foster parents. If this does not occur

(e.g., the nest is abandoned, which can happen if the cuckoo is spotted by the nesting pair), the female may move them to another nest. Following incubation of the egg, the cuckoo chick develops more rapidly than the other eggs, and will hatch approximately 1 day before the other chicks. A few hours after hatching, the cuckoo chick develops an intense cutaneous reflex because of contact with the skin of the back, following which everything that meets its back is raised and expelled from the nest, including the eggs and other chicks. In other parasitic species this does not occur, but the host's chicks are still doomed to starvation, given the rapid development of the parasite that monopolizes the parent's attention for food. Small passerines are extremely diligent in their efforts to feed the rapidly growing cuckoo, most likely due to the super-stimulus provided by its giant red beak. Commonly, parasitic species lay their eggs in the nests of species that produce eggs very similar to theirs. As a result, distinctly defined populations exist that parasitize different species and allow them to coexist in the same territory. African parasitic species include members from the order Piciformes, which parasitize other woodpeckers, but also starlings (Sturnidae), bee-eaters (Coraciiformes, Meropidae), and hoopoes (*Upupa* spp.). Chicks have a sharp beak and, by the second day of life, use this to kill the adoptive siblings to remain the only chick in the nest. Among Ploceidae, the African widows (*Vidua* spp.) have developed a less bloody, parasitic behavior on several species of waxbills. Widows have many similarities with the species that they parasitize, up to incorporation of part of their song in their own singing. In this group, the eggs are always white, but the interspecific mimicry is perfectly expressed by the luminescent wattles in the chick's oral cavity, which perfectly alings with the chosen waxbill's pattern. This is essential because, unlike cuckoo chicks, the small widow chicks do not kill the waxbill offsprong but are rather raised together by the host species' parents.[1]

The captive breeding strategy and modified version of reproductive parasitism is the use of foster parents to support reproduction in species that are not yet well adapted to captivity. The best-known example, which is linked to the case of widows, is that of the society finch *(Lonchura striata* var. *domestica)*, which is used as a foster parent for many species of Australian, Asian, and even African finches and waxbills. The society finch is not a true, naturally occurring species but rather a domestic bird with selected genetic contributions from several species of the genus *Lonchura*. This hybrid nature was responsible for the loss of conditioning of the bird to a precise oral cavity color pattern to cue feeding behaviors, so the society finches willingly feed chicks of different species, even mixed in the same nest. However, some African finch species remain too different, and may therefore not be willingly fed by society finch foster parents. Other finch species that may be used as foster parents include the canary and the Mexican finch *(Carpodacus mexicanus)*. Small poultry breeds, often Silkie or bantams, have been used to brood and raise pheasant chicks. While the use of foster parents improves the production of species that are more difficult to breed successfully in captivity, concerns are raised regarding the risk of inadvertently selecting for genetic variants that lack reproductive instinct. This has been noted in many gallinaceous species. In addition, fostering by other species may increase the risk for transmission of infectious agents from foster parents to the chicks. An example of concern is the transmission of *Cochlosoma* from society finches to fostered Gouldian finch chicks (*Chloebia gouldiae*; see also Chapter 24).[5] Because of this risk of exposure from an asymptomatic species to a fostered but vulnerable species, caution is advised prior to instituting this practice.

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CHAPTER 40

Artificial Incubation

Petra Schnitzer

Aristotle reports first on artificial incubation dating back to 400 BCE, where the Egyptians created the first incubator to hatch the eggs of birds. The use of these ancient methods remained present until the use of electricity and the invention of the thermometer around 1881, when incubators could be designed and produced commercially.[1] Hatching and rearing birds without their parents has turned out to be a highly successful and potentially lucrative procedure, for the simple reason that removal of eggs from the nest for artificial incubation and hand-rearing the chicks leads to more clutches and eggs being produced each year by the breeding pair, hence increasing the number of offspring. The second advantage of artificial incubation is associated with the opportunity for improved infectious disease control. With the help of artificial incubation, it is feasible to eradicate horizontally transmitted diseases from a bird collection. Other benefits to aviculture include the higher profit for breeders that are producing birds for the commercial or pet industry such as chickens or parrots and the positive effects on programs working toward conservation of endangered species.[2,3] Common reasons for artificial incubation are listed in Box 40.1.

Although artificial incubation and hand-rearing techniques may have beneficial effects, potential adverse consequences should also be considered. For example, the breeding pair may be more easily exploited and suffer from compromised welfare because of overproduction and a potential resultant loss of breeding lifespan or through learned aversive responses to perceived loss of eggs or chicks over time. Similarly, hand-rearing may have deleterious consequences for the chicks. Current information pertinent to optimal welfare and breeding indicates that, compared to hand-rearing, leaving the chick with its natural parents, or foster parents, may be beneficial—if not optimal—for young birds. Several studies have shown that hand-reared birds are more prone to developing behavior problems such as feather-damaging behavior and may no longer mate with conspecifics, build nests, or incubate the eggs (see also *Current Therapy in Avian Medicine and Therapy*, Vol 1, Chapter 5, Behavior).[4,5] Hence, parent rearing is important to develop normal (reproductive) behavior, which is especially important for conservation programs and for the purpose of replacing aging breeding birds in a breeding facility. While hand-rearing traditionally has been performed to produce tame birds that are well socialized to humans, parent-raised birds can make good yet presumably behaviorally better-balanced pets, especially if co-parenting strategies have been applied.[4–7] Depending on the species (altricial or precocial), the chick either needs to be hand-reared after hatching or introduced to a bird pair for parent rearing.

ARTIFICIAL INCUBATION—PLANNING AND EQUIPMENT

When implementing artificial incubation in practice, a mid- to long-term breeding plan including a properly laid-out nursery and adequately functioning incubators is required. The layout, traffic flow pattern, and design, as well as the numbers of incubators to be utilized will vary immensely, depending on the species, the numbers of eggs anticipated to be incubated, and the anticipated variation in temperature/humidity gradients that will be required.[2] In some select situations, like the fostering of chicks under a different hen, eggs can be incubated under a different hen. As an example, brooding chickens have been used to incubate the eggs of several other species.

The ideal incubator serves as a "room within a room" and should be able to provide the desired incubation parameters necessary for the species (Figure 40.1). The room in which the incubator is placed should be thermostatically and humidity-controlled so that the climate can be kept constant, independent of daily climactic conditions. Without this type of control, fluctuations should be expected within the incubator unit.[8] The unit should be constructed with nonporous materials for two reasons: optimal temperature and humidity control, and opportunity to effectively clean and sterilize the unit.

In those facilities that maintain separate incubation and nursery areas, a general floor plan should be established for the incubation area. This floor plan should allow a specific traffic pattern to promote the efficient use of time and materials while also ensuring a passive barrier against waste and contamination. The so-called "spaghetti test" is a helpful aid toward establishing a fluid floor plan. By tracing the daily activity that will occur within a building for normal work tasks on a schematic map of the facility, the amount of overlap and inefficiency of motion will become evident in a floor plan/entire facility analysis. If the projected foot traffic begins to resemble a "spaghetti ball," the relative risk of cross-contamination and motion/time inefficiency should dictate a change in the blueprint (see Chapter 11).[9]

Before one can commence artificial incubation, they must understand and acknowledge that this may be a full-time job that can be extremely demanding. Box 40.2 highlights important considerations prior to initiating an artificial incubation effort. As a part of the plan for artificial incubation, additional factors should be considered and included during the planning phase (Box 40.3).

While there is no "gold standard" for a good incubation plan, there are basic principles and goals of incubation, and if

BOX 40.1 REASONS TO IMPLEMENT ARTIFICIAL BREEDING IN A BREEDING FACILITY OR ZOO

- To increase the production of eggs and chicks.
- Species conservation, particularly of species that have high biological value. Many zoos and breeding facilities participate in reintroduction and recovery programs for endangered species (e.g., California condor [*Gymnogyps californianus*], whooping crane [*Grus americana*], Spix's macaw [*Cyanopsitta spixii*]; see also *Current Therapy in Avian Medicine and Therapy*, Vol 1, Chapter 23, Conservation of Avian Species), which often requires a high number of offspring to be produced for release into the wild.
- High financial value associated with increased production. This can either be related to a high volume of chicks produced (e.g., commercial poultry industry) or related to a high financial value of the individual chicks produced. For example, chicks of certain species that are harder to breed in captivity, such as the hyacinth macaw *(Anodorhynchus hyacinthinus)*, or gyrfalcons *(Falco rusticolus)*, fetch a high price and breeders may want to increase the number of offspring to generate more income.
- Impaired ability or inability of a given pair to rear chicks successfully. Some breeding pairs may have a history of breaking the eggs, abandoning the nest, or not caring for or showing aggression toward their chicks after hatch. Additionally, death or illness of one or both parents may warrant artificial incubation and hand-rearing to save the chicks or eggs.
- Inexperience of a breeding pair. A first-time breeding pair of a particularly important or valuable species may not yet have the skills to incubate and rear chicks. To guarantee that at least some chicks survive, the breeder may choose to collect some eggs from the first clutch for artificial incubation.



FIGURE 40.1 Two incubators being used in a separate temperature- and humidity-controlled room. The central incubator is currently in use for psittacine species, and its bottom tray is being concurrently used as a hatcher. The incubator on the right is being maintained as a backup for cracked and repaired eggs or in the case of a functional problem with the central one currently in use. Other incubators are being used for other species groups with different incubation requirements.

the aviculturist does not follow the rules, the obtained results could be disappointing. A reasonable goal for a successful artificial incubation in psittacine species is that 90% of the fertile eggs should hatch, and 90% of the hatchlings should reach the weaning age (L. Crosta/P. Schnitzer, personal communication, 2024).

RECORD-KEEPING

Accurate data collection is essential for the aviculturist or veterinarian to enable objective and rational evaluation of the incubation results and allow for appropriate and correct management decisions to be made. Especially when artificial incubation is unsuccessful or achieved results are below the target goals, record review and analysis are of the greatest importance.

BOX 40.2 IMPORTANT CONSIDERATIONS PRIOR TO ARTIFICIAL INCUBATION

1. Financial investment: incubators, hatchers, brooders, furniture, and special rooms need to be purchased, constructed, and installed.
2. Time is required to monitor incubator performance and to assess and record egg and embryo/hatchling data.
3. Time is required to maintain and improve knowledge and skills related to the latest information, concepts, and discoveries.
4. Time is needed for record-keeping of the climatic conditions (room temperature, humidity), hygiene and disinfection, and observations related to the immediate environment.
5. Considerable time and facility investment are required for the care of the chicks, particularly when incubating altricial species (though to a lesser degree also a prerequisite for the precocial species). Chick care largely includes hand-rearing and socialization, as well as introduction to foster parents (in rare exceptions).

BOX 40.3 ADDITIONAL VARIABLES TO DISCUSS AND INCORPORATE INTO AN ARTIFICIAL INCUBATION PLAN

- What is the main role of the aviculturist?
- What level of experience with artificial incubation and hand-rearing do they have?
- How much time do they have to spend on both activities?
- How many breeding pairs are in the facility? How many of these will be used for artificial incubation?
- What species are present in the collection? Will incubation requirements for their eggs necessitate different incubators and settings?
- Are the breeding birds experienced in rearing chicks, or are they first-time breeders?
- Are there any specific disease concerns present that might lead to different breeding techniques in the breeding facility (e.g., diseases that are not transmitted vertically, hence increasing suitability of artificial incubation to eliminate disease from the flock)? (See also Chapters 11 and 12)



FIGURE 40.2 An egg has been pulled from the nest for artificial incubation, marked with ID for the breeding pair, as well as the order that the eggs were laid. (Photo courtesy Lorenzo Crosta.)

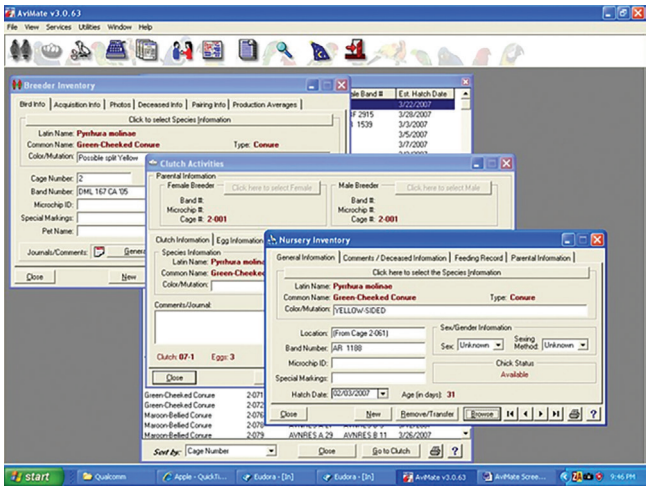


FIGURE 40.3 A computer screen capture of an example of an avicultural record-keeping system. Note screens for breeder activities, clutch activities, and nursery inventory displayed; each has several subset tabs for the information categories available. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

Records to be maintained in avicultural settings can be divided into four main areas, including general information, adults, hatchlings, and incubation. Factors including the aviculturist's mission statement, record-taking abilities, and attention to detail determine the desired and required information to be collected and recorded in the system. Excessive record-keeping can potentially be as detrimental as incomplete record-keeping, as excessive efforts to record data that have no practical or applicable value can amount to wasted time and additional fatigue and frustration on the aviculturist's part.[2]

Incubation records maintained may include identification of the specific incubator used, incubation temperature and humidity parameters, turning rate and degree, date on which the egg was laid, date on which the egg was set and incubation commenced, egg weight at setting and at hatch, expected and actual hatch dates, hatch intervals within the clutch, hatching observations, chick weight at hatch, egg candling observations such as fertility, vascularity, dead-in-shell (early, mid, and late embryonic mortality), and any and all diagnostics performed, including the results. The data obtained from these incubation records relate to both parental reproductive and systemic health, as well as incubation management by the aviculturist. The egg identification (ID) can be written on the eggshell with a blunt soft pencil (Figure 40.2). The advantage of using a pencil is that no toxic substances will be released inside the egg, and most egg disinfectants do not wash the notes away. Generally, data recorded on the eggshell will include date of lay, species, and ID (number of the pair, the female, or aviary). Elsewhere, other data are recorded, either in a notebook or computer-based record-keeping system (Figure 40.3). A Windows-based avicultural record-keeping program that is commercially available and can be easily downloaded is AviMate.

EGG STORAGE

Storing the eggs is common practice in the poultry industry, but it is also applied in breeding facilities for psittacine and other avian species. Most aviculturists will set eggs into the incubator on the same day that the egg is harvested from the nest. In part, this decision results from lack of knowledge regarding the specific date of lay. If the eggs are not yet set by the parents, and incubation has not started embryogenesis significantly, storage can be utilized to synchronize hatch groups. Allowing the eggs to rest prior to incubation, if possible, can also increase hatchability of fertile eggs. However, once incubation has started (either naturally or artificially), the eggs should not be cooled, as the risk of embryonic mortality increases significantly upon warming, cooling, and then warming the egg again. Storage between 2 and 5 days seems to yield optimal results, whereas short or excessively long storage times tend to reduce hatchability.[10]

Freshly laid eggs should be examined for dirt, abnormal shape, or cracks in the shell. If the eggs are dirty, the dirt can be gently brushed off using a fine bristle brush, but only when the eggs are dry. Routine use of water for cleaning psittacine eggs is generally discouraged (unlike in poultry species), as it can damage the cuticle, which functions as the outermost protective layer of the eggshell, thereby increasing the risk of microbial contamination. In case of a cracked shell, the egg should preferably be discarded, but in case of valuable eggs (e.g., vulnerable species), repair of the egg can be considered (Figure 40.4). Several techniques for eggshell repair have been utilized, with variable results. Glue, candle wax, and even cling wrap can be used. It is important to remember that by sealing the crack, the egg pores will also be closed, hence altering the egg's respiration and metabolism of the embryo. For this reason, focal application of repair patches is advised, as opposed to coverage of a larger surface area of the egg. A cracked egg that has been repaired needs to be handled as if it is an infected egg and should ideally be kept in a separate incubator.

Psittacine eggs are placed horizontally in a clean container and laid on a soft, clean substrate (e.g., millet seeds, dry sand, trays specifically designed for egg storing). The eggs can then

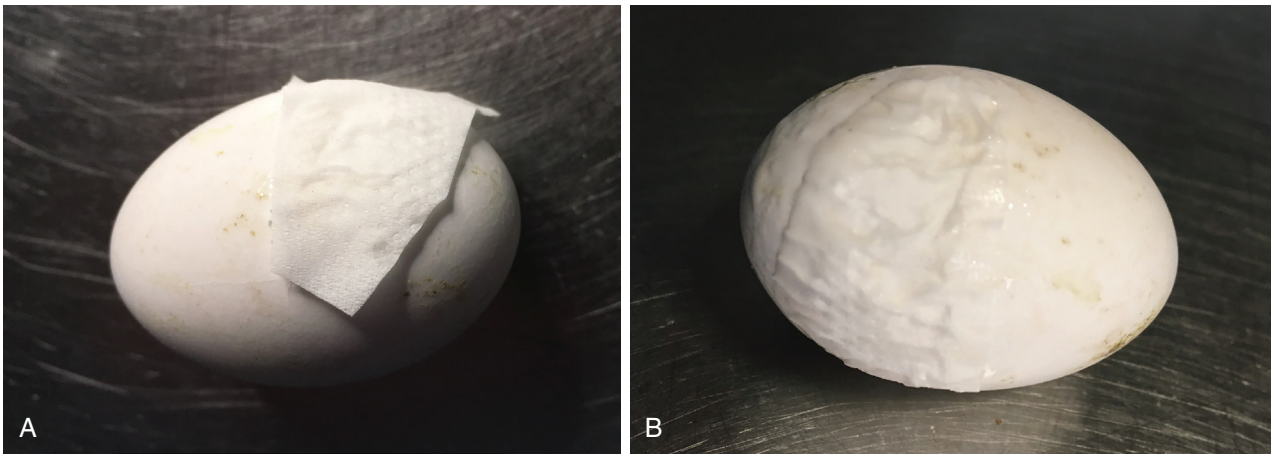


FIGURE 40.4 Egg shell crack repair. Prior to repair, the shell is lightly cleaned (A). Tissue paper is then applied to the specific area of the cracked shell and then sealed with white glue (B). Repaired eggs should be housed in a separate incubator, and regarded as if they are contaminated, to reduce risk of contamination of the primary incubator and eggs within it. (Photos courtesy Steve Duncan, http://www.avianresources.com/.)

be stored at 13°C to 16°C (55.4–60.8°F). During the initial period of egg cooling in the nest or storage site, the air cell develops following separation of the inner and outer shell membranes. Minimal handling, movement, or vibrations of the eggs during both storage and incubation are important, as any disturbance during these early phases of incubation and embryo development can predispose to early embryonic death and malposition.

INCUBATORS

Commercial incubators are available in varying brands, models, and prices. Independent of the chosen model, the incubator needs to be stable, capable of incubating eggs of different sizes, and capable of monitoring the inner temperature and maintaining it constant (Box 40.4). For these machines to operate optimally, it is recommended to keep them in an air-conditioned room with constant temperature and humidity—the so-called "room within a room" concept (Figure 40.1). In addition, it is recommended to place additional thermometers and hygrometers in the center of the incubator for close monitoring, even if a built-in unit is present. Heat and humidity are circulated by fans, which provide proper air circulation and ensure a more even temperature throughout the incubator. Generally, a common goal in artificial incubation is to provide a more uniform and even distribution of heat and humidity, as opposed to the variations that may be seen with natural incubation. Studies performed in broilers report a discrepancy from 0.4°C to 3.0°C in fan-heated incubators.[11] Depending on the egg size, how full the incubator is or is not, and the distance from the fan, late embryonic mortality can increase because of inadequate air circulation and uneven distribution of temperature and humidity.[11] Units with low air turnover should be expected to vary in their ability to distribute temperature evenly throughout the unit, particularly when the unit becomes progressively filled with eggs. Alternatively, incubators that have been altered to improve ventilation may have a higher temperature variability because of increased heat loss.[11]

New ideas and concepts are constantly developing on artificial incubation and one of these concepts includes imitation of the natural incubation process. The "brood patch" that is present in hens ensures transfer of heat to the egg once the hen is sitting on the nest, following which a thermal gradient is generated between the lower and upper parts of the egg. Additionally, when the hen leaves the nest, the eggs will cool down and be susceptible to environmental temperatures. This causes a contraction and expansion of the egg content, or "egg breathing," which seemingly improves the metabolism of the egg and the embryo. To mimic this natural process and physiologic temperature fluctuations, incubators were designed with a broad heater on the ceiling of the unit (representing the brood patch) to improve the metabolism and development of the embryo. Whether or not these types of incubators indeed improve the hatchability of the eggs currently is unknown due to the lack of published evidence comparing the older and newer methods. However, it is likely that these newer incubator designs are at least equally good as the traditional ones, and the concept of using these is nowadays widely popular and supported in avicultural circles.

BOX 40.4 IMPORTANT PARAMETERS WHEN SELECTING AND EVALUATING AN INCUBATOR FOR ITS SUITABILITY

- Amount of useable inner space
- Types of egg trays, including their versatility for the anticipated sizes of eggs to be hatched
- Ease of cleaning and maintenance
- The ease and practicality of use
- Degree of incubator mass: the bigger the mass of the incubator, the greater the degree of stability of temperature and humidity inside it
- Availability of technical assistance

BOX 40.5 COMMON REASONS FOR EMPLOYING MULTIPLE INCUBATORS

- Eggs of different sizes may need different trays for optimal air and heat circulation around them.
- Broken but repaired eggs should be considered contaminated and should be incubated separately. These eggs can "sweat" or produce foam, or even explode during incubation.
- Eggs from different species can have different incubation parameter requirements for relative humidity and temperature.
- Eggs may require different numbers of turnings per day. Small eggs generally require a higher number of turnings per day compared to larger eggs.
- A controlled number of eggs may be needed per machine to facilitate optimal incubation.
- An incubator may break down, requiring the availability of a backup device.

Regardless of the type or model that is used, there are several reasons why it is recommended to have more than one incubator available and running at the same time (Box 40.5).

It is important to verify proper functioning of the incubator(s) at least 1 month prior to the onset of the breeding season to provide adequate time to address any issues in case problems are encountered. Incubators should be capable of running and reaching the desired humidity and temperatures consistently at least 2 weeks prior to placing the eggs in the device. Regular maintenance is advised and should include checking whether the different components are functioning adequately, including the wet bulb, thermometer, hygrometer, timers, and turning mechanisms. Incubators (and brooders) should also be carefully cleaned and disinfected using nontoxic substances (available as a liquid solution or fog/mist), at least once at the end of the breeding season (see Chapter 12). The fan of the incubator should receive extra attention as it can easily harbor and distribute pathogens the following season unless cleaned well.

INCUBATION

Successful artificial incubation is highly dependent on the skills of the aviculturist and the attention paid by them to detailed record-keeping, care, and monitoring of the equipment; precision of the egg handling technique; and perseverance. For many aviculturists, it can be very satisfying to have control over the development and hatching of the chicks. However, it can also be very frustrating and emotionally challenging if embryonic mortality is high.[12]

The incubation period for the eggs depends on the species involved and is like the incubation time by the parents. For example, incubation times vary from 12 to 15 days for the zebra finch *(Taeniopygia guttata)* and from 28 to 30 days for blue-fronted Amazon parrot *(Amazona aestiva)*, and are around 35 days for the gyrfalcon *(Falco rusticolus)* and around 42 days for the ostrich *(Struthio camelus)*.[2,8]

Three key factors that are essential for successful artificial incubation include temperature, humidity, and turning frequency of the eggs. It is important that all these factors approach natural circumstances and are maintained constant in the incubator through careful monitoring by the operator.

TEMPERATURE

The incubator's temperature settings are paramount for proper embryonic development and control the speed of embryonic development. Generally, the higher the temperature, the faster the rate of embryonic development. From this perspective, incubation times will be shorter with higher temperatures and longer with lower temperatures. However, temperature extremes in both directions can result in higher rates of embryonic mortality. A balance to ensure optimal embryonic development is the goal. For most avian species, the optimal temperature ranges between 36.9°C (98.42°F) and 37.5°C (99.5°F). In most parrots and some birds of prey, the optimal temperature is 37.2°C (98.96°F) ± 0.1°C (0.2°F). Individual testimonials of temperatures ranging from 37.2°C to 37.4°C (99.0–99.3°F) have proven successful for psittacine egg incubation. While incubating the eggs it is important that the heat produced is consistent and uniformly distributed within the unit.

HUMIDITY

The humidity in the incubator regulates the transpiration of the egg. As a result of water evaporation from the egg, weight loss will normally occur during the incubation process. This process is critical for normal growth and hatching of the embryo. Humidity is traditionally measured with the use of both wet bulb and dry bulb thermometer in the incubator. The wet bulb temperature is the lowest temperature to which air can be cooled by the evaporation of water into the air at a constant pressure. The dry bulb temperature is the ambient temperature. The difference between these two temperatures is a measure of the humidity of the air. Alternatively, humidity can be measured in the incubator with the use of hygrometers that are strategically placed within. Excessively high humidity during incubation will lead to inadequate weight loss and to weak and edematous embryos, commonly called "wet chicks." These "wet chicks" are predisposed to have more difficulty hatching and a higher rate of mortality at or immediately following hatch. Generally, chicks from precocial species will show the classic signs of edema, whereas chicks from altricial species will more commonly show a weaker than normal hatch, increased tendency to develop splay leg, and possibly develop immunosuppression-linked problems. Conversely, an excessively low humidity may result in excessive weight loss, and a dehydrated and weak embryo, referred to as "dry" or "sticky chick" (Figure 40.5). Like wet chicks, these embryos are weakened and predisposed to difficulties at hatch and increased rates of mortality.

As humidity is critical to ensure normal embryonic development and hatch, this parameter should be carefully monitored throughout the incubation process. Humidity is generally expressed as relative humidity (RH) and expressed as a percentage. In addition to evaluating the RH in the room and incubator, the weight loss of the eggs can be calculated. This can have value in the diagnosis and management of suspected humidity issues in the psittacine hatchery. Should deviations from the desired RH or weight loss curve be observed, immediate action can be taken to adjust and reach the desired

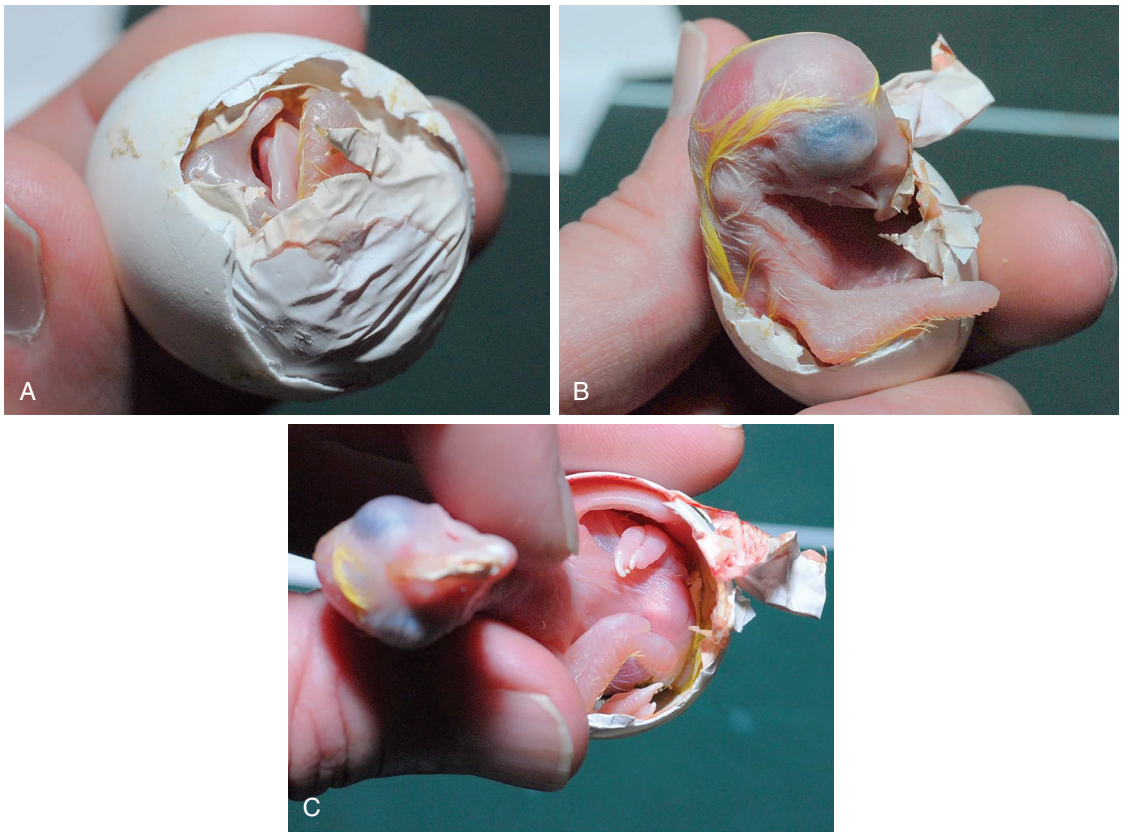


FIGURE 40.5 Assisted hatch of a cockatoo chick, which had lost excessive weight during incubation and was "dry." Note the desiccated shell membranes (A), slightly dry appearing skin (B), and the tight adherence of the chick to the inner shell walls (C). (Photos courtesy Steve Duncan, http://www.avianresources.com/.)

target humidity in the incubator. Methods with which to adjust the RH are displayed in Box 40.6.[2]

Artificial incubation is a dynamic process. The author recommends what she sees most prevalent in psittacine aviculture, which is to have a goal of RH below 40% in the first half of the incubation period, whereas RH should remain below 45% in the second half of the incubation period. The goal is to end up with a weight loss within the appropriate weight loss range at the end of the incubation. To ensure that weight loss remains within the optimal range, parameters need to be observed daily and adjusted, as indicated (Box 40.7). The "right" humidity settings are the ones that work optimally in a specific incubator and in a specific location or environment. As this can be highly dependent on the environmental circumstances, every aviculturist should be encouraged to investigate which parameters work best in their situation. Careful monitoring of the eggs' weight loss during incubation is recommended to aid in identifying optimal incubation parameters for this reason (Box 40.8). Throughout incubation, the rate of weight loss follows a linear trend, which makes it easy to verify whether the RH is correct or whether it needs adjustment. From the day that the egg is laid until internal pipping, it should normally lose approximately 14% to 16% of its initial weight, depending on the species involved (Table 40.1).[2,13]

The most common egg weight loss management problem encountered during incubation is that the weight loss is

BOX 40.6 AVICULTURAL TECHNIQUES TO ADJUST THE RELATIVE HUMIDITY WITHIN AN INCUBATOR

- Use of a small (built-in) container with distilled water within the incubator unit.
- Reduction or increase of the airflow inside the incubator through adjusting the fan speed.
  This method will also alter the ratio between air and water. However, in the authors' experience, this method is generally not advised, as reduced airflow can result in reduction of oxygen concentration available to the eggs as well as slow down the removal of carbon dioxide produced by their metabolism. Similarly, decreased fan speed can reduce effectiveness of temperature circulation within the incubator.
- Use a humidifier or a dehumidifier in the incubation room.
  Generally, incubators are very effective at adding humidity to their internal environments, but less so in reducing it. Dehumidification of the air in the incubation room, to be circulated within the incubators themselves, can optimize incubator function. This is especially relevant when lower relative humidity incubation parameters are desired.
- Introduce (nonfertile) chicken eggs into the incubator.
  This addition can help to maintain humidity within the incubator. When using chicken eggs, it is preferred to use good quality eggs of which the origin is known, as contaminated or infected eggs may pose a risk for increased embryonic mortality and failure to hatch.

BOX 40.7 INDIVIDUAL EGG PARAMETERS TO MONITOR ON A DAILY TO WEEKLY BASIS

1. Temperature
2. Relative humidity (RH)
3. Weight of the egg/weight loss of the egg
4. Damage to the eggshell
5. Candling observations

BOX 40.8 EXAMPLE OF WEIGHT LOSS CALCULATION OF AN EGG DURING ARTIFICIAL INCUBATION

Egg weight losses can be calculated using the following formula:
Example:
Blue-and-gold macaw *(Ara ararauna)*
Egg weight at egg laying: 26 g
Current weight: 24.05 g
Incubation time: 28 days

1. Calculate the grams lost per day of incubation
   (Starting weight – Current weight)/Days incubated = Weight loss/day

$$\frac{26.00\ \text{g} - 24.05\ \text{g}}{14\ \text{days}} = \frac{1.95\ \text{g}}{14\ \text{days}} = 0.14\ \text{g/day}$$

2. Calculate anticipated weight loss for the total incubation period
   Daily weight loss/Day × Number of days of incubation period for the species
   Example:

$$0.14\ \text{g} \times 28\ \text{days} = 3.9\ \text{g}$$

3. Calculate anticipated weight loss as a percentage of the original weight
   Anticipated weight loss/Starting weight × 100

$$\frac{3.9\ \text{g}}{26.0\ \text{g}} = 0.15 \times 100 = 15\%$$

inadequate. Should the rate of weight loss be uniform for all eggs in the incubator, the RH will need to be reduced. However, should weight loss be inadequate for an individual egg, whereas other eggs from the same incubator have a weight loss that is within the desired range, a more specific approach tailored to the individual egg would need to be employed. To increase the rate of transpiration and resultant weight loss, a variety of different techniques can be applied. For example, to increase loss of moisture from the egg, one to two small (1.5–2 mm) holes can be drilled in the shell (1.5–2 mm) over the air cell. Alternatively, fine sandpaper can be used to abrade the cuticle in a select area, which will open the shell pores and increase the rate of transpiration. Conversely, when there is excessive weight loss observed in an individual egg, select portions of the shell may be covered, cautiously, with nontoxic materials. These corrective procedures should always be reserved for the second half of the incubation period and only be performed by an experienced operator.[2,14]

EGG POSITIONING AND TURNING

During natural incubation, birds turn their eggs several times a day. Dependent on the species involved, eggs are artificially incubated in either a horizontal position (e.g., most psittacine species and birds of prey) or a vertical position, with the air cell pointed upward (e.g., poultry and ratite species). Turning of the eggs is mandatory to allow for uniform development of the vascular supply of the embryo throughout the embryonic membranes.[15] Without this web of vessels, the developing embryo will become deprived of adequate oxygen exchange, which may potentially result in its death. General poultry recommendations for egg turnings are to perform 90-degree turn, minimally four times per day.[16] Turning of the eggs can be accomplished mechanically or manually but should always be performed smoothly and carefully, as rough turning can be associated with an increase in early embryonic mortality. When using an automatic turning system, careful monitoring and evaluation of the turning processes are advised to ensure the process occurs smoothly.

TABLE 40.1

Differences in Egg Weight Loss Recommended During Incubation in Some Selected Psittacine Species[2]

Common Name	Species	Egg Weight Loss (%)
COCKATOOS		
Citron-crested cockatoo	*Cacatua (sulphurea) citrinocristata*	13–17
Galah	*Eolophus roseicapilla*	15–18
Little corella	*Cacatua sanguinea*	14–16
Major Mitchell's cockatoo	*Lophochroa leadbeateri*	12–15
Moluccan or salmon-crested cockatoo	*Cacatua moluccensis*	16–20
Sulphur-crested cockatoo	*Cacatua galerita*	16–18
Sulphur-crested cockatoo (Eleonora)	*Cacatua galerita eleonora*	15–18
Yellow-crested cockatoo (Parvula)	*Cacatua sulphurea parvula*	13–15
Triton sulphur-crested cockatoo	*Cacatua galerita triton*	15–20
Yellow-crested cockatoo	*Cacatua sulphurea*	13–15
Umbrella or white cockatoo	*Cacatua alba*	16–18
MACAWS		
Blue-and-gold macaw	*Ara ararauna*	15–19
Buffon's macaw or great military macaw	*Ara ambiguus*	16–20

Continued

TABLE 40.1

Differences in Egg Weight Loss Recommended During Incubation in Some Selected Psittacine Species—cont'd

Common Name	Species	Egg Weight Loss (%)
Blue-throated macaw or Caninde macaw	*Ara glaucogularis*	17–20
Green-winged macaw	*Ara chloropterus*	17–20
Hyacinth macaw	*Anodorhynchus hyacinthinus*	17–20
Military macaw	*Ara militaris*	16–20
Red-fronted macaw	*Ara rubrogenys*	14–16
Scarlet macaw	*Ara macao*	15–19
OTHER		
Eclectus parrot	*Eclectus roratus*	14–17
Rose-ringed parakeet	*Psittacula krameri manillensis*	9–12

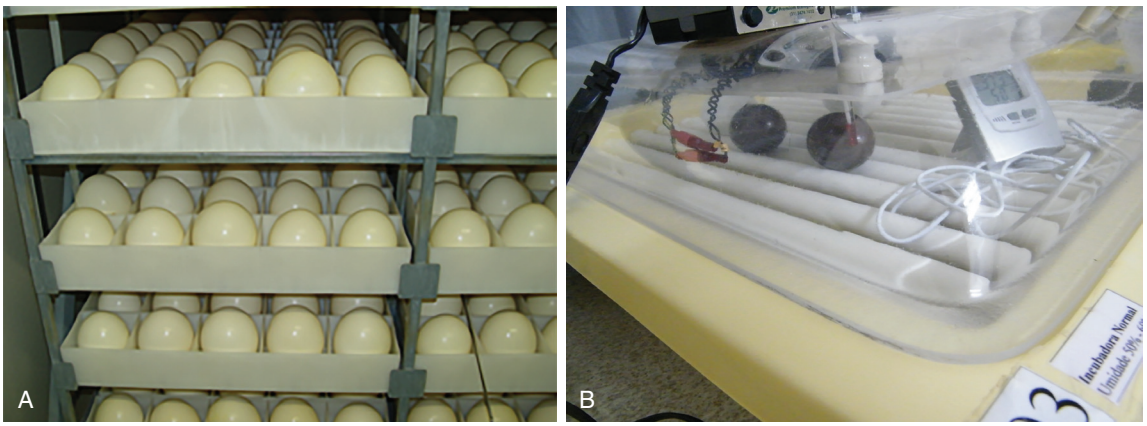
There are two basic methods by which a mechanical incubator may turn the eggs: rotation and tilting (Figure 40.6). Rotating incubators have trays with rollers of different sizes, and the eggs are positioned horizontally to allow the eggs to turn when the rollers move, which will prevent problems with chorioallantoic membrane development and malpositioning of the embryo.[17] When using tilting as a method to turn, eggs are positioned vertically with the air chamber up (note that this is not considered favorable for psittacine eggs). During the process, trays will tilt 45 degrees each way, hence having a 90-degree range of movement in total.

Manual turning of the eggs can be employed exclusively, or in addition to what is provided by the incubator. The use of an automated turning method combined with manual turning can be useful to help avoid inadequate turning–associated problems should the incubator mechanism fail to turn the eggs without the deficit being recognized. When manually turning the eggs, care needs to be taken to avoid contamination by dirty hands (i.e., wash hands carefully, or wear gloves) and to avoid forceful or rapid movements of the eggs. Inadvertent mechanical damage to the shells or developing embryonic membranes can occur due to forceful manual turning, and should the egg have already been turned mechanically, it is also possible to manually return the egg back to its original position.

CANDLING

Candling is used to evaluate eggs for evidence of fertility, to estimate the age of the embryo, and to assess the health of the developing embryo. Two different candling techniques that are commonly used are light candling and electronic or digital candling.

Light candling is the classic candling technique. Traditionally, this type of candling is done in a darkened room with a bright focal light source (e.g., lamp, torch). By holding a bright light up to the egg, the shell's contents can be



FIGURES 40.6 (A) Ostrich *(Struthio camelus)* eggs in a incubator. These vertically positioned eggs with their air cells upward are tilted 45 degrees to the right and then to the left, accounting for a 90-degree turning, at a minimum of four times q24h period. (B) Horizontally positioned eggs for incubation may be placed between the rollers in this incubator, which turns the eggs 45 degrees on their longitudinal axis each way, also accounting for a 90-degree turning roughly four times q24h. During the hatching phase of incubation, turning is not continued, regardless of the manner of incubation up to that point. (A, Photo courtesy Brian Speer; B, photo courtesy Lorenzo Crosta.)

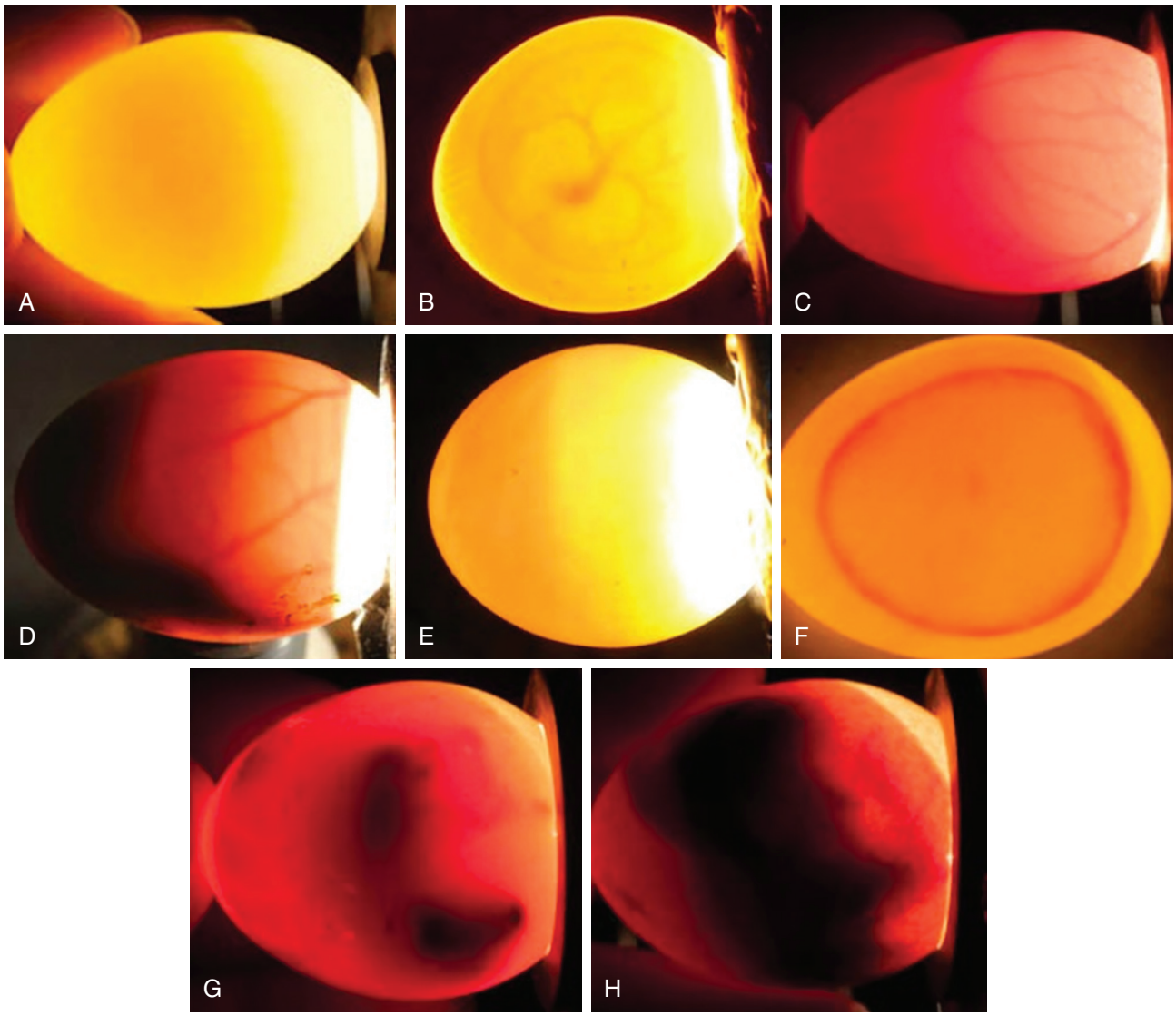


FIGURE 40.7 Examples of healthy embryonic development (A–D) as compared to embryonic mortality candling findings (dead-in-shell) (E–H). (A) Newly laid fertile egg. At this stage, vascular development should not be visible, but the normal central location of the yolk can be appreciated. (B) Early normal embryonic and vascular development at 4 to 5 days of incubation. (C) Continued vascular development at 14 days of incubation. (D) Maturing embryo and vascular development at 21 days of incubation. (E) Early embryonic mortality in the first few days of incubation. Note the apical location of the yolk in the left end of the egg, and stratified density of yolk. (F) Embryonic mortality demonstrated at days 4 to 5, with a classic "blood ring" visible due to disruption and degeneration of early vessels in the developing chorioallantoic membrane. (G) Embryonic mortality at 14 days of age. Abnormal vessel development is apparent, and there is no Doppler detection of a fetal heartbeat noted. (H) Embryonic mortality at day 21 of incubation. There are no visible vessels in the chorioallantoic membrane, the embryo is free-floating within the egg, there are gas bubbles visible slightly to the left of the embryo's shadow, and there is no Doppler detection of a fetal heartbeat noted. (Photos courtesy Steve Duncan, http://www.avianresources.com/.)

illuminated, allowing the embryo and blood vessels to be identified. An experienced operator is not only able to recognize fertility but can also estimate the approximate age of the embryo (Figure 40.7). Depending on the species, fertility can often be assessed after 4 to 7 days, when the developing blood vessels of the chorioallantoic membranes are clearly visible (Figure 40.8). Other signs that indicate a healthy, developing embryo are the increased air cell size, and altered shape and activity of the embryo, which increase right before hatching (draw down). With early embryonic death, a dark band will usually be visible (Figure 40.9).[18]

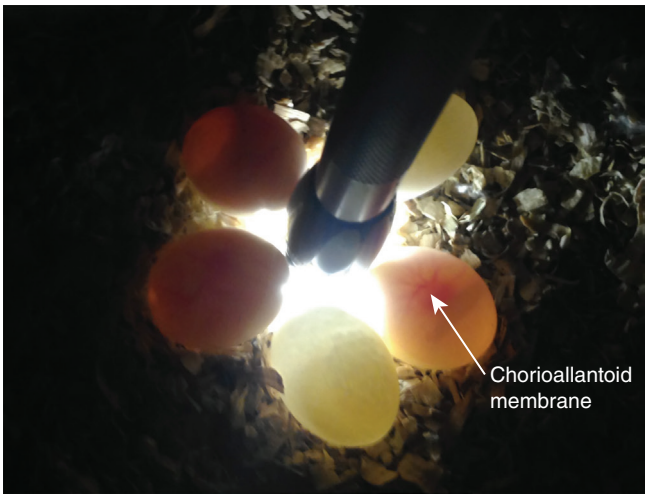


FIGURE 40.8 Candling can be performed in the nest as well as during artificial incubation procedures. This image shows the appearance of three fertile and two infertile eggs in a clutch. Bubbles visible in the lowest egg indicates the presence of a ruptured air cell, or faulty inner shell membrane, and this egg was removed from the clutch. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

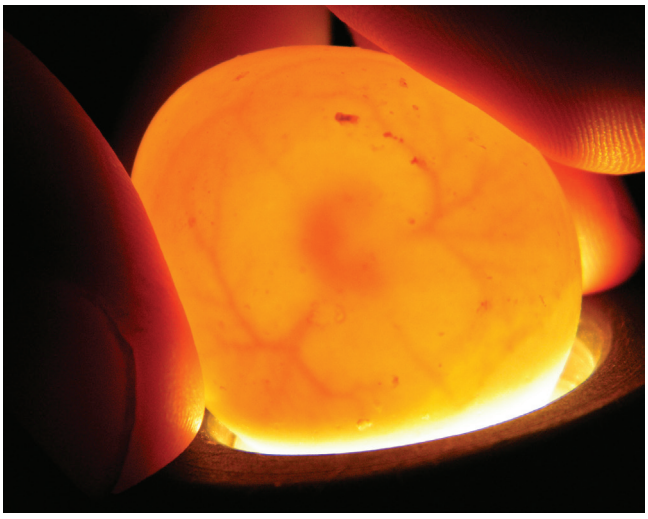


FIGURE 40.9 A dark band that surrounds the embryo in a candled egg usually indicates early embryonic death. (Photo courtesy Lorenzo Crosta)

An alternative and newer method of candling, which has been developed over the past decade, is electronic or digital candling. The monitors (e.g., Buddy; Avian Biotech International, Tallahassee, FL, USA and Bird Buddy; Avitronics, Truro, UK) used for this procedure use infrared transmitters and sound sensors to amplify the cardiovascular signal of the embryo, which can be detected a few days after the start of incubation. Although these instruments can provide valuable information about the embryo, its health, and development, they should likely never be presumed to be entirely capable of replacing classic candling techniques, as direct visualization of developing embryonic membranes as well as their cardiovascular signals both have value in assessment of embryonic health.

THE NORMAL HATCH

Depending on the species, the hatching process begins about 48 to 72 hours after the internal pipping process, where the beak of the chick penetrates the egg's air cell (Figure 40.10). Once the chick is breathing the air of the air chamber, it starts to vocalize. Following the internal pip, the egg should be transferred to the hatcher. The temperature in the hatcher (37.0–37.1°C [98.6–98.8°F]) is slightly lower than in the incubator, whereas the humidity is slightly higher (RH 50%–60%). During the hatching process, the egg is no longer turned. Many aviculturists recommend placing the hatching egg in a single container within the hatcher.[19]

The external pip occurs when the chick cracks or cuts the outer shell membrane and shell. The external pipping and completion of the hatch can take anywhere from 24 to 72 hours, dependent on the species involved (Table 40.2). During the hatching process, the chick will continue to cut the shell and progressively break out of the egg (Figure 40.11).

MALPOSITIONS AND THE ASSISTED HATCH

Malpositions of the embryo are often classified using poultry systems, which recognize seven different positions (Figure 40.12). Malpositions I and II are the most typically associated with hatching failure and will generally—though not exclusively—require hatching assistance.[18,20] Causes for these malpositions as well as the associated mortality rates are listed in Table 40.3. There are some variations from the poultry normal chick position noted in psittacine species, in which the head is rarely positioned underneath the wing.[21] The more commonly observed malpositions noted at one psittacine breeding facility are shown in Figure 40.13.[22]

Determining when assistance is needed for the hatch can be a challenge, and experience is required to make the right decision. Some malpositions, if identified by candling or other chick monitoring methods, may indicate a need for assistance (Table 40.3; Figures 40.14–40.16). Generally, assistance may be required when the chick's voice starts to get weaker or the heart rate is slower, which can be observed well with an electronic candling device. A hole should be made over the air chamber, to facilitate breathing for the chicks that are failing to start the external pip. Box 40.9 lists the general steps to perform this procedure. Often, this will be all the assistance that is required to help convert a stressful hatch into a successful one.

Box 40.10 provides an overview of common problems to be encountered during artificial incubation and their causes. Some of the differential diagnostic considerations, particularly regarding causality, are included.

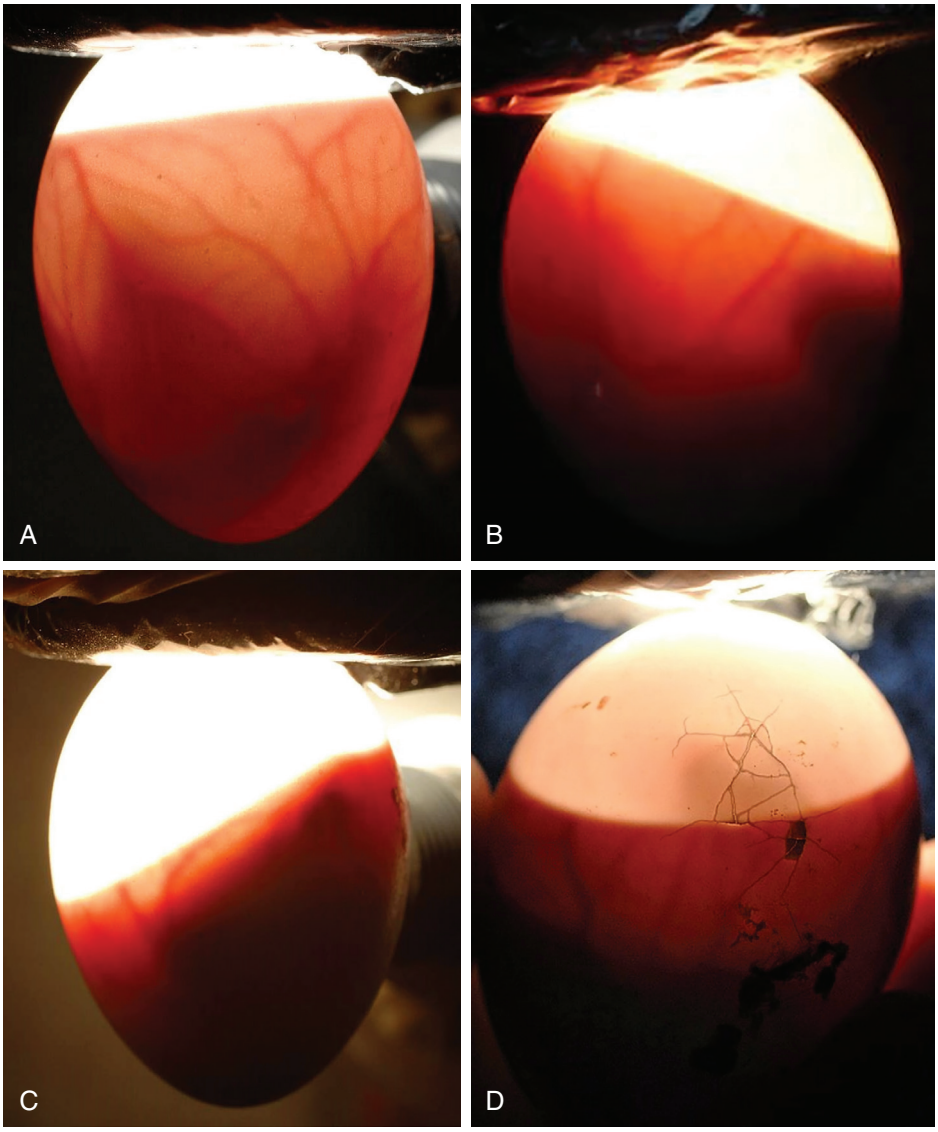


FIGURE 40.10 Candling of the normal internal aspects of the initial hatching process. (A) Embryo is visible with normal vascular development and a clear air cell at the blunt end of the egg. (B) Further development and initial retraction of vessels of the chorioallantoic membrane. There has been a slight increase in the size of the air cell, and its surface interface with the embryonic membranes is angled. (C) Draw down, where the embryo shifts its position in the egg, raising the inner shell membrane asymmetrically, in preparation for the internal pip into the air cell, and significantly altering its silhouette. (D) Internal pip. The shadow of the chick's beak is visible within the air cell. The cracks on the surface of the shell were sustained earlier and repaired and are not related to the hatching process. (Photos courtesy Steve Duncan, http://www.avianresources.com/.)

TABLE 40.2

Incubation Period and Time From Internal Pipping to Hatch in Selected Psittacine Species[2,18]

Common Name	Species	Total Incubation Period (Days)	Pip to Hatch Interval (Hours)
Amazon, blue-fronted	*Amazona aestiva*	26	24–48
Amazon, double yellow-headed	*Amazona oratrix*	28–29	24–48
Amazon, green-cheeked or red crowned	*Amazona viridigenalis*	24–26	24–48
Amazon, lilac-crowned	*Amazona finschi*	26	24–48
Amazon, red-lored	*Amazona autumnalis*	25–26	24–48

Continued

TABLE 40.2

Incubation Period and Time From Internal Pipping to Hatch in Selected Psittacine Species—cont'd

Common Name	Species	Total Incubation Period (Days)	Pip to Hatch Interval (Hours)
Amazon, white-fronted	*Amazona albifrons*	24	24–48
Amazon, yellow-crowned	*Amazona ochrocephala ochrocephala*	28–29	24–48
Amazon, yellow-naped	*Amazona auropalliata*	28–29	24–48
Budgerigar	*Melopsittacus undulatus*	18	24–36
Caique species	*Pionites* spp.	25	24–48
Cockatiel	*Nymphicus hollandicus*	21	24–48
Cockatoo, citron-crested	*Cacatua (sulphurea) citrinocristata*	25–26	36–48
Cockatoo, sulphur-crested (Eleonora)	*Cacatua galerita eleonora*	26–27	24–48
Cockatoo, galah	*Eolophus roseicapilla*	22–24	24–36
Cockatoo, goffin's	*Cacatua goffiniana*	28	24–72
Cockatoo, little corella	*Cacatua sanguinea*	23–24	36–48
Cockatoo, Major Mitchell's	*Lophochroa leadbeateri*	26	24–48
Cockatoo, Moluccan	*Cacatua moluccensis*	28–29	24–48
Cockatoo, palm	*Probosciger aterrimus*	28–30	48–96
Cockatoo, red-tailed black	*Calyptorhynchus banksii*	30	24–48
Cockatoo, red-vented	*Cacatua haematuropygia*	24	24–48
Cockatoo, sulphur-crested	*Cacatua galerita galerita*	27–28	24–48
Cockatoo, triton	*Cacatua galerita triton*	27–28	24–48
Cockatoo, yellow-crested	*Cacatua sulphurea*	24–25	24–48
Cockatoo, umbrella	*Cacatua alba*	28	24–48
Conure, *Aratinga* spp.	*Aratinga* spp.	24	24–48
Conure, golden	*Guaruba guarouba*	30	24–48
Conure, *Pyrrhura* spp.	*Pyrrhura* spp.	23	24–48
Lory, black	*Chalcopsitta atra atra*	27	24–48
Lory, blue-streaked	*Eos reticulata*	27	24–48
Lory, red	*Eos bornea bornea*	27	24–48
Lory, chattering	*Lorius garrulus flavopalliatus*	27	24–48
Lorikeet, Goldie's	*Glossoptilus goldiei*	24	24–48
Lovebird species	*Agapornis* spp.	22	24–48
Macaw, blue-and-gold	*Ara ararauna*	26	24–48
Macaw, blue-throated	*Ara glaucogularis*	26	24–48
Macaw, Buffon's or great military	*Ara ambiguus*	26	24–48
Macaw, green-winged	*Ara chloropterus*	26	24–48
Macaw, hyacinth	*Anodorynchus hyacinthinus*	26–28	24–48
Macaw, military	*Ara militaris*	26	24–48
Macaw, red-fronted	*Ara rubrogenys*	26	24–48
Macaw, scarlet	*Ara macao*	26	24–48
Macaw, severe	*Ara severus*	28	24–72
Macaw, yellow-collared	*Primolius auricollis*	24–26	24–48
Parakeet, Alexandrine	*Psittacula eupatria*	24–25	24–48
Parakeet, canary-winged	*Brotogerus versicolurus*	22	24–48
Parakeet, Derbyan	*Psittacula derbiana*	26	24–48
Parakeet, edbreasted	*Psittacula alexandri fasciata*	25–26	24–48
Parakeet, *Neophema* spp.	*Neophema* spp.	18	24–48
Parakeet, grey-cheeked	*Brotogeris pyrrhoptera*	22	24–48
Parakeet, monk	*Myiopsitta monachus*	23	24–48
Parakeet, rose-ringed	*Psittacula krameri*	23–24	24–36
Parrot, grey	*Psittacus erithacus*	26–28	24–72
Parrot, Timneh	*Psittacus timneh*	26	24–48
Parrot, Senegal	*Poicephalus senegalus*	24–25	24–48
Parrot, Meyer's	*Poicephalus meyeri*	24–25	24–48
Parrot, Jardine's	*Poicephalus gulielmi*	25–26	24–48
Parrot, Amboina king	*Alisterus amboinensis*	20–21	24–36
Parrot, Australian king	*Alisterus scapularis*	20–21	24–48
Parrot, green-winged king	*Alisterus chloropterus*	20	24–48
Parrot, Eclectus	*Eclectus roratus*	28	24–72
Parrot, blue-naped	*Tanygnathus lucionensis*	26	24–48
Parrot, thick-billed	*Rhynchopsitta pachyrhyncha*	28	24–48
Parrotlet species	*Forpus* spp.	19	23–36

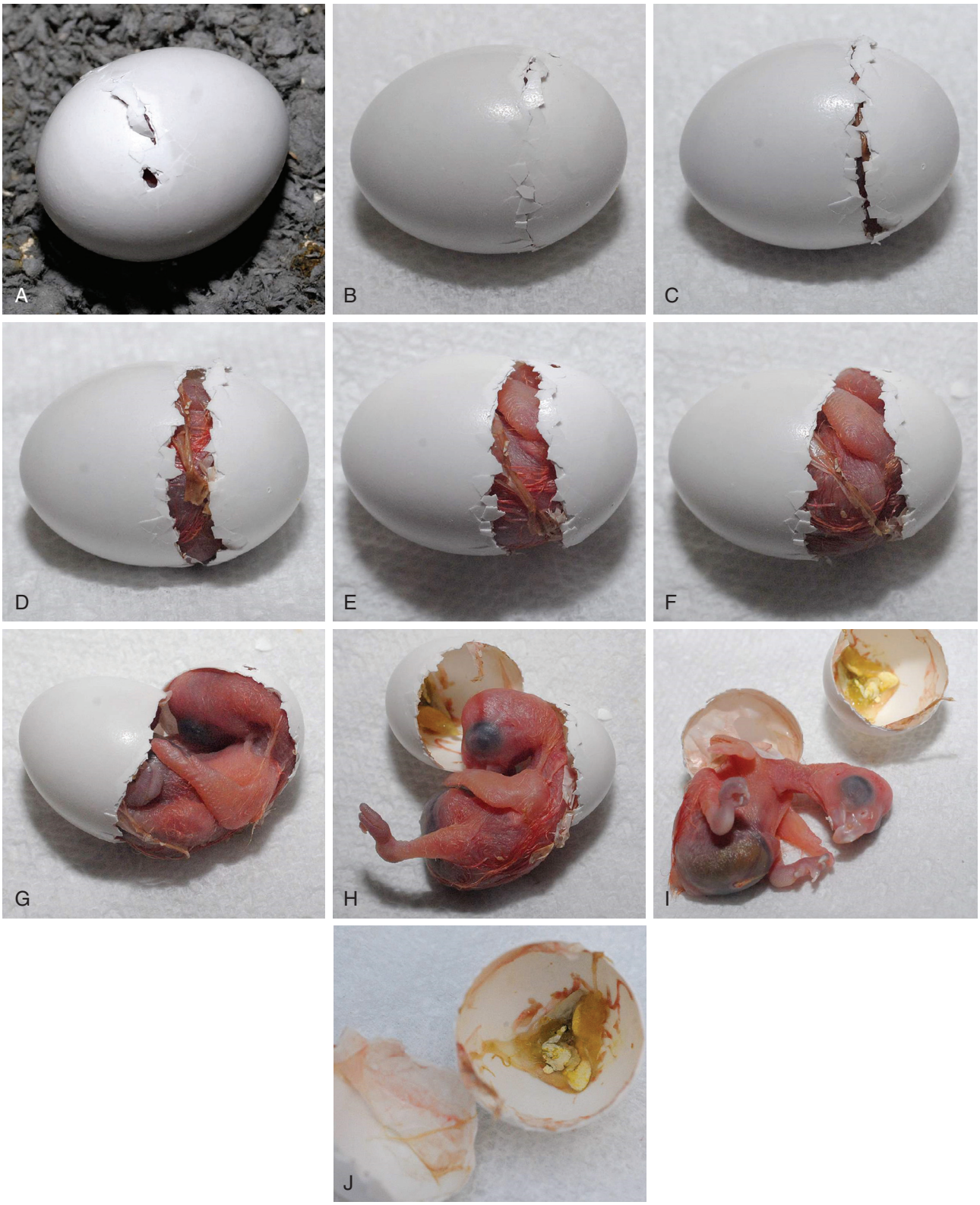


FIGURE 40.11 (A–J) A normal hatching sequence for a galah chick *(Eolophus roseicapilla).* Following a resting period after the internal pip, the chick perforates the external shell. The shell is progressively cut by the egg tooth and contractions of the complexus muscle, producing a functional cap that the chick is then able to push off and free itself from the egg. Note the apparent normal hydration of the chick as compared to the dry chick depicted in Figure 40.5. (Photos courtesy Steve Duncan, http://www.avianresources.com/.)

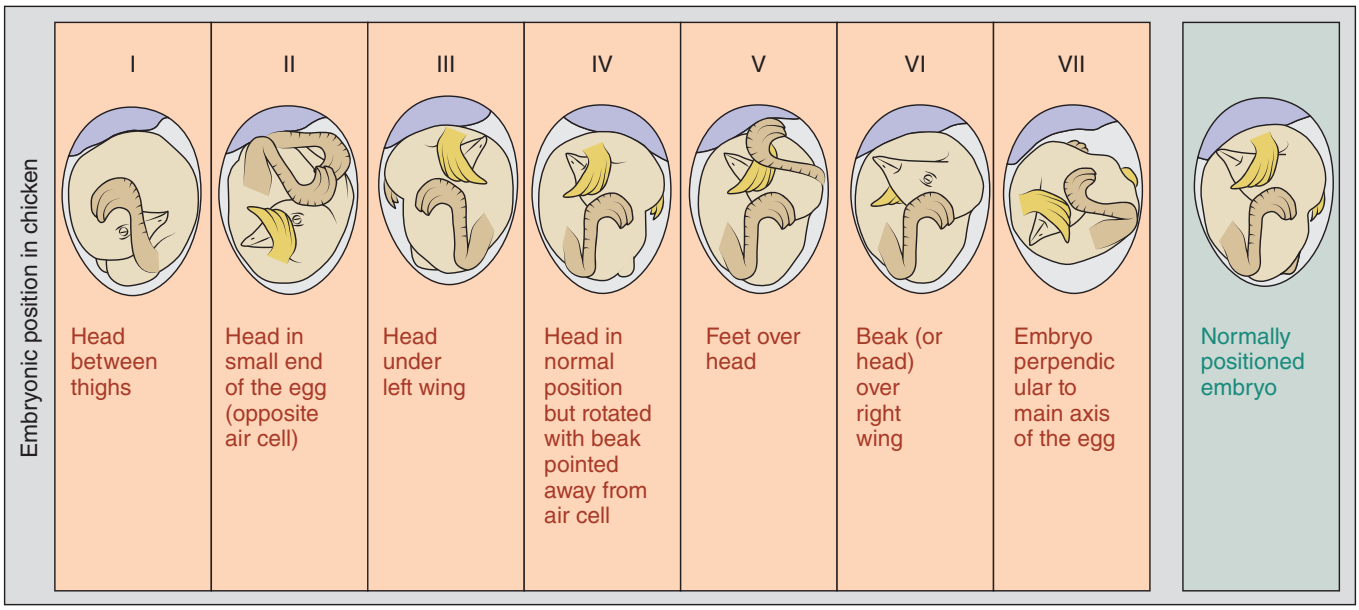


FIGURE 40.12 Embryonic malpositions in the domestic fowl. (Modified from Zhang Y: *Studies on chicken hatchability and its relation with egg yolk metabolites* (Ph.D. thesis), Gottingen, Germany, 2017, Gottingen University.[20])

TABLE 40.3

Normal Position and Embryonic Malpositions of the Domestic Fowl, Their Potential Lethality, and Causes

Position	Description	Lethality	Cause
Normal	Head at large end near air cell, head tucked under right wing upside down. Psittacines tend not to have the head tucked under the wing.	N/A	N/A
I	Head between thighs. Failure to lift and turn head right in last trimester of incubation.	Lethal	High incubation temperature
II	Head at the small end of egg opposite to the air cell, chick upside down.	50% lethal, assisted hatch often required	Incubator egg position incorrect, low incubation temperature
III	Head under left wing, rotates head left (not right). Body rotated on egg long axis.	Commonly lethal	Incubator egg position incorrect, incorrect incubation temperature, parental malnutrition
IV	Beak (egg tooth) directed away from air cell.	Slightly reduced low hatchability, assisted hatch may be required	Incubator egg position incorrect
V	Feet over head.	Commonly lethal, as embryo cannot kick or rotate for hatching	
VI	Head over right wing. Head in same plane as wing. (This is a normal position for psittacines.)	Slightly reduced hatchability (domestic fowl), assisted hatch may be required	Parental malnutrition
VII	Embryo perpendicular to the main axis of the egg.	Reduced, assisted hatch may be required	Incubator egg position incorrect

Modified from Zhang Y: *Studies on chicken hatchability and its relation with egg yolk metabolites* (Ph.D. thesis), Germany, 2017, Gottingen University.[20]

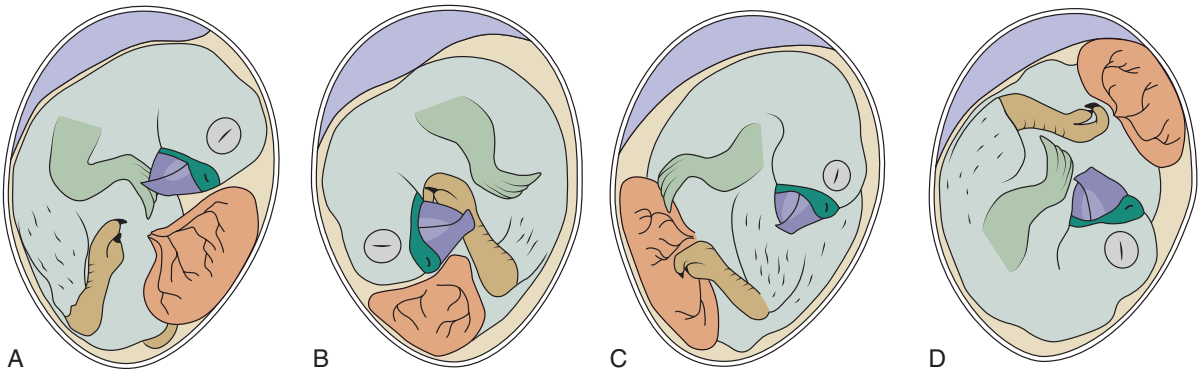


FIGURE 40.13 Common malpositions observed in psittacine embryos, as described by the Avicultural Breeding and Research Center.[21] (A) Position of the parrot embryo that most closely resembles the normal position of the chicken embryo. Unlike the chicken, the head is rarely underneath the wing. (B) Beak is rotated away from the air cell. Equivalent to malposition IV in the domestic fowl. (C) Breast is positioned toward the air cell, with the head turned back. (D) Head is in the small end of the egg (breech). Equivalent to malposition II in the domestic fowl.

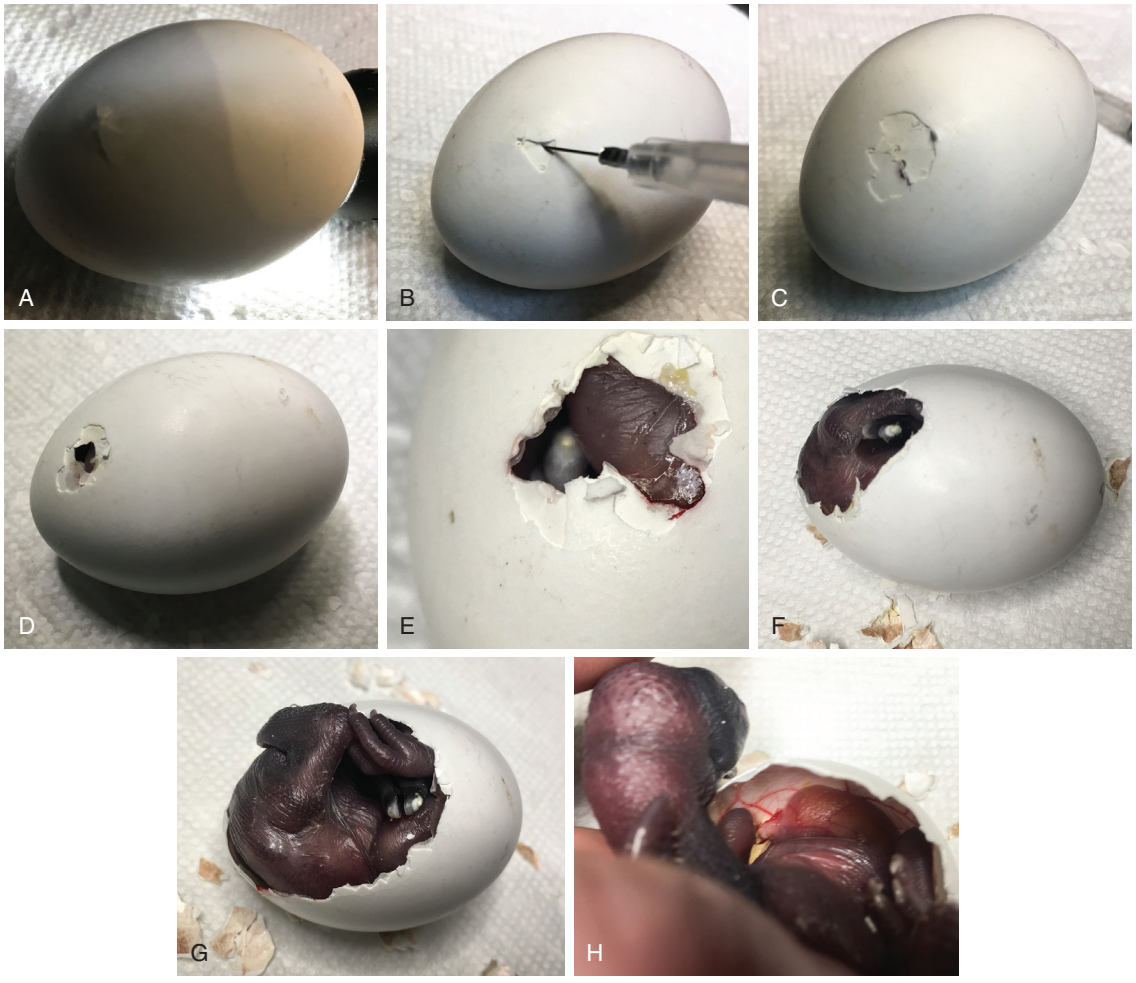


FIGURE 40.14 Assisted hatch in a blue crowned pigeon *(Goura cristata)*. (A) The chick has externally pipped well below the air cell. (B) Air is injected underneath the inner shell membrane to provide air to the chick. (C–E) Some shell is progressively removed to allow the underlying chorioallantoic membrane to dry for several hours, so it can then be opened to provide air to the head and beak. (F) With the chorioallantoic membrane and later some shell opened over the tip of the beak to allow continual airflow, the chick is allowed to rest while the yolk is retracted, and the lungs take over respiration. (G) Additional shell is removed as the blood vessels recede. (H) Full retraction of yolk is confirmed before the chick is removed from the remaining shell. (Photos courtesy Steve Duncan, http://www.avianresources.com/.)

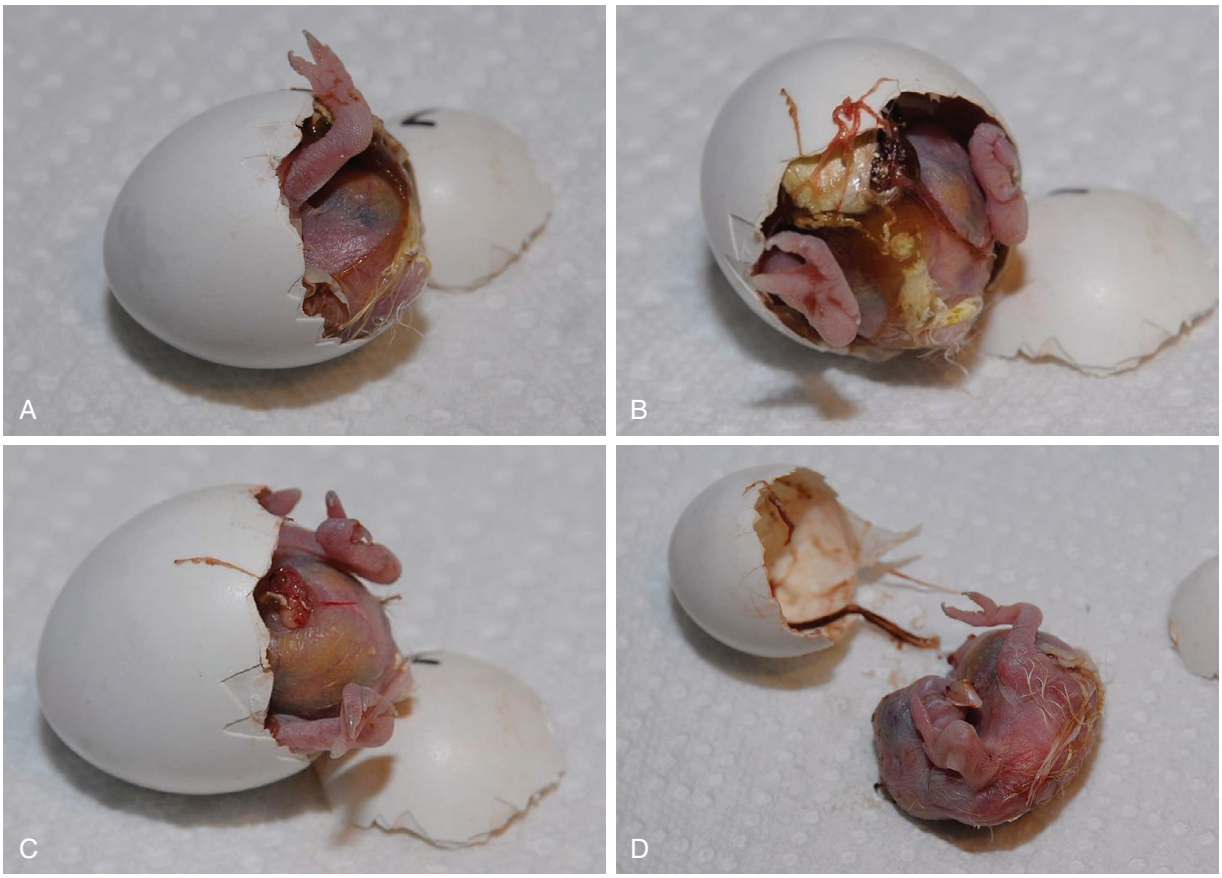


FIGURE 40.15 Assisted hatch in a malposition II umbrella cockatoo *(Cacatua alba)*. (A) Position of the chick was verified by opening the air cell and inner shell membranes, and a source of air provided to the chick's head in the narrow end of the egg. (B and C) A period of 2 to 4 hours was needed to allow for vessels of the chorioallantoic membrane to retract as well as for the yolk sac (visible in B) to be fully internalized. (D) Successful delivery of chick from the shell. (Photos courtesy Steve Duncan, http://www.avianresources.com/.)

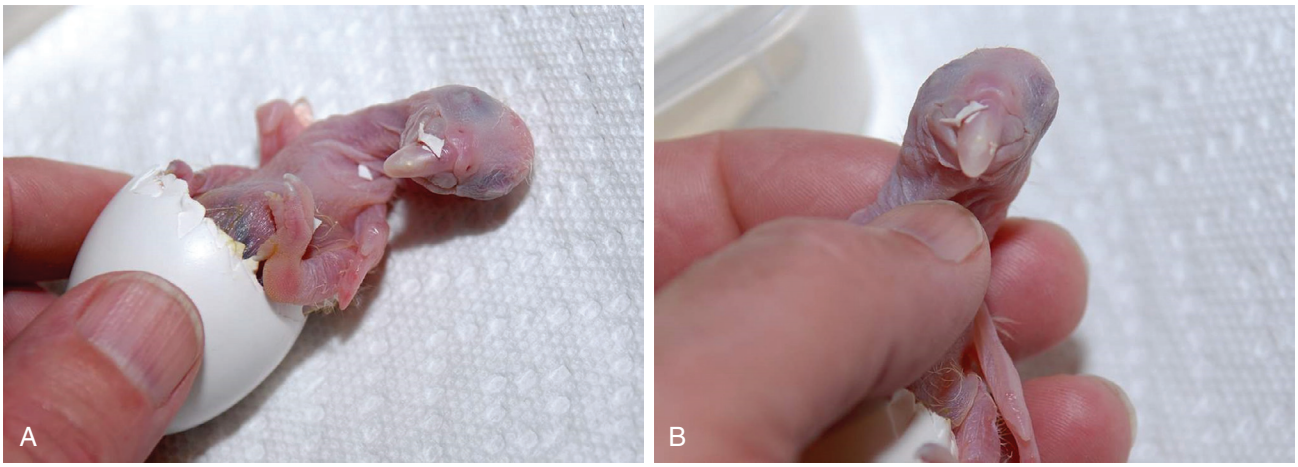


FIGURE 40.16 Completion of an assisted hatch of a green-winged macaw *(Ara chloroptерus)* from malposition II. The small end of the egg, away from the air cell, was perforated to provide air to the chick, and ultimately a cap of shell removed to enable a successful hatch. (B) Note the deformity of the head, with a flattening on the right side. This deformity rapidly self-corrected over the first 24 hours after hatch. (Photos courtesy Steve Duncan, http://www.avianresources.com/.)

BOX 40.9 STEPWISE BASIC PROCEDURES FOR PERFORMING AN ASSISTED HATCH

1. Maintain the egg in the same position as during incubation.
2. Using a sterilized needle or fine drill bit, drill a hole over the air chamber which is located at the blunt side of egg.
3. Using small forceps, take away small parts of eggshell until the hole is big enough to allow good visualization of the chick. Please note that the chick is likely covered by the shell membranes and/or portions of the chorioallantoic membrane.
4. Use a cotton-tipped applicator, or a small paintbrush soaked with distilled water or sterile saline solution, to moisten the membranes.
5. If there is a visible net of small blood vessels in the chorioallantoic membrane, the chick is not ready to hatch. In this case, place it back in the hatcher and WAIT!
6. If blood vessels are not visible, the membranes can be broken using a soaked applicator following which the bill of the chick should be located.
7. Repeat the same action after 1 hour, widening the hole on the membranes and maintaining them adequately moisturized.
8. Keep the chick moist, particularly around the bill.
9. Stimulate the chick to move and to free itself from the membranes with a gentle pressure of the applicator. At this point, vocalization is a good sign!
10. Always check the area around the umbilicus before freeing the chick completely to assure that the yolk sac has been adequately resorbed into the body and is no longer present externally.
11. When the chick is out of the eggshell, in some cases it may be necessary to ligate and remove a fully exteriorized yolk sac.
12. Disinfect the umbilicus topically as indicated and leave the chick in the hatcher for few hours to dry.

Considerable variation in timing and details may be needed, based on specifics of an individual case.

BOX 40.10 GUIDELINES FOR TROUBLESHOOTING PROBLEMS WITH INCUBATION AND THE HATCH[16,22,23]

Observation	Possible Causes
Early embryonic mortality	Delayed egg collection
	High egg storage temperature
	Long egg storage time
	Infected eggs
	Breeder malnutrition
	Rough egg handling
Mid embryonic mortality	Breeder malnutrition
	Infected eggs
	Inadequate egg turning
	Rough egg handling
Late embryonic mortality	Breeder malnutrition
	Inadequate ventilation
	Infected eggs
	Fluctuating incubation temperature
No internal pip	Weak chicks
	Dry chicks
	Wet chicks
	Malposition
	Infected egg
Internal pip and death	Hatcher temperature too high
	Hatcher temperature too low
	Hatcher humidity too high
	Inadequate ventilation
	Lack of social stimulation
Early hatch	Incubation temperature too high
	Incubation humidity too low
	Small egg size
Late hatch	Incubation temperature too low
	Large egg size
Malpositions	Inappropriate egg position
	Inadequate egg turning
	Rough egg handling
	Breeder malnutrition
Malformed chicks	Incubation temperature too high
	Breeder malnutrition
	Rough egg handling
	Chick malposition
	Genetic mutations
	Teratogens
"Wet chicks"	Inadequate air circulation
	Excessive humidity
	Large egg size
	Thick eggshells
	Low eggshell porosity
	Breeder malnutrition
Small chick size	Incubation humidity too low
	Small sized eggs
	Thin eggshells
	Porous eggshells
	Genetic predisposition
"Sticky chicks"	Incubation humidity too low
Externalized yolk sacs	Assisted hatching (premature)
	Infected eggs
	Breeder malnutrition
	Wet chicks
	Dry chicks
	Incubation temperature too high
	Fluctuating incubator temperatures
Yolk sac infections	Assisted hatching
	Inadequate umbilical care
	Unsanitary hatcher
	Infected eggs
	Inadequate egg harvesting technique

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CHAPTER 41

Pediatrics

Lorenzo Crosta • Brian L. Speer • Susan L. Clubb

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INTRODUCTION

Evolution has perfected avian parental neonatal care. As humans, we have tried to mimic the natural biological care of neonates and tried to provide for their needs to produce healthy viable young birds. Under ideal circumstances, leaving chicks in the nest with their parents would be the best choice for neonatal care.[1] Natural parenting is preferable if environmental conditions are acceptable to the parents, nutrition is adequate, and the pair is compatible and experienced. However, to enhance socialization for birds designated for the pet trade and to increase the number of chicks produced, aviculturists may elect to remove (pull) chicks from the nest for hand-rearing. In addition, chicks may be removed from the nest if parental care is inadequate, environmental conditions are hazardous for the chicks, or to increase the number of chicks produced by a breeding pair.[1,2]

In broad terms, precocial *(nidifugous)* chicks hatch from the egg with open eyes and thick coats of natal down. These chicks are strong enough to leave the nest within 1 or 2 days. Most shorebirds, ratites, game birds, gallinaceous birds, and waterfowl have precocial chicks. These species tend to have larger eggs and have a more abundant yolk sac that contains all the nutrients that the chick needs to survive the first few days of its life. Altricial chicks *(nidicolous)*, on the other hand, are hatched blind and virtually naked. They require long periods of feeding and parental care before they are strong enough to leave the nest on their own. Most tree-nesting and cavity-nesting birds have altricial young, which includes the psittacine birds.

Instead of a sharp dividing line between precocial and altricial hatchlings, a gradient can be recognized, with a wider variability in the degree or level of precociality recognized among birds (see also Chapter 45).[3] In birds, four levels of precociality are recognized, with megapodes (e.g., brush turkey, [*Alectura lathami*], Australian malleefowl [*Leipoa ocellata*]) representing precocial level 1 (superprecocial), which includes chicks that are totally independent of their parents. Precocial level 2 includes species of which the young follow their parents but find their own food (e.g., ducks, shorebirds), whereas birds from precocial level 3 have young that follow their parents and are shown food (e.g., game birds) and those of precocial level 4 have parents that feed the young that trail after them (e.g., grebes, rails). The so-called semiprecocial species includes birds such as gulls and terns of which the (nidifugous) chicks can leave the nest soon after hatching but stay in the vicinity of the nest while being fed by the parents. In semialtricial species, the downy chicks are also fed by their parents but incapable of leaving the nest. Chicks are either classified as semialtricial 1, which includes species of which chicks hatch with the eyes open (e.g., hawks, herons), or as semialtricial 2, which includes species of which chicks hatch with the eyes closed (e.g., owls). Finally, altricial species includes the species hatching with little to no down and closed eyes that are incapable of leaving the nest and fed by their parents (e.g., psittacine birds, passerines). These classifications are displayed in Table 45.1.

FACTORS INFLUENCING SUCCESSFUL PARENT-REARING IN AVICULTURE

Parental experience in raising chicks greatly influences the success or failure in the natural breeding and chick rearing process. Other factors essential for a successful outcome of this process include careful selection of breeding stock, proper genetic management, careful behavioral pairing, and a strong effort to provide an optimal environment for the breeding pair(s).[1,2]

With the notable exception of monk parakeets/quaker parrots *(Myiopsitta monachus)* that nest communally in large, growing nest structures constructed from twigs and sticks, most parrot species nest in tree hollows or cavities in cliffs, rock outcrops, or riverbanks (Figure 41.1; see also Chapter 39). In captivity, nest boxes are provided to mimic these locations, and their design (size, shape) and placement are critical factors that will attract the pair to use the nest box, which should provide comfort for the nesting pair while also enabling proper incubation of the eggs and rearing of the chicks. The substrate provided in the nest is also important and must be sufficiently attractive to the pair to encourage breeding and laying. Breeding pairs often discard nest material and may ultimately lay their eggs directly on the floor of the nest box. This can lead to damage to the shells, excessive movement of the eggs and hence to suboptimal incubation, embryonic mortality, and increased chick morbidity (e.g., developmental problems, leg deformities) and mortality (see also Chapter 40).

Environmental temperature extremes, the presence of excessive moisture in the nest, or dehydration of the chicks



FIGURE 41.1 Feral Quaker parrot *(Myiopsitta monachus)* communal nest on an electrical power pole. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

can be challenging for parents, and if they are unsuccessful in adequately supporting the needs of their fertile eggs or developing chicks, these types of environmental fluctuations can result in morbidity, mortality, and ultimately failure to reproduce (see also Chapter 39).[1,2] Parental care includes providing contact warmth for chicks and physically regulating their hydration status. In tropical climates, temperature fluctuations in the breeding and nesting sites are minimal. However, the majority of commercial and hobby psittacine aviculture is conducted in temperate climates, which are more prone to temperature fluctuations and possibly even extreme shifts in temperature.[1,2] Warmth is essential for embryonic development and survival, as well as for neonatal chicks. A devoted breeding pair can ensure adequate warmth even in reasonable temperature extremes. Most parrot species provide substrate for the nest by modifying the walls of the nest cavity. Some species such as peach-faced lovebirds *(Agapornis roseicollis)* or Goffin's cockatoos *(Cacatua goffini)* may carry vegetation to the nest tucked into their feathers. Nesting season is typically in the dry season to avoid rain flooding the nest, while chick rearing is typically timed to occur when climatic conditions are suitable for food availability for chicks. Pairs breeding under human care are free of these environmental challenges.

Predation, a major cause of nest failure in the wild, is more controllable in captivity. However, despite management controls that may be in place, rodents, snakes, raccoons, mites, etc. may enter the nest and harm the eggs and chicks. Additionally, predators or rodents can cause disturbance at night, which causes stress and affects the breeding pair's confidence regarding their own safety and their ability to safely rear their offspring. If birds are being bred in flocked setting, sharing the enclosure with other breeding pairs, there can be increased chances for aggression or competition for nests from other birds, with subsequent destruction of eggs or chicks. Coparenting by previous offspring or other conspecifics may help the pair in care of the chicks but can alternatively also result in jealousy or squabbling over eggs or chicks, possibly resulting in damage or abandonment.

The strength of the pair bond of the breeding pair is a key element for the breeding success of many species. Hence, an aviculturist should have a sound understanding of the nesting behaviors of each pair as this will increase the chances of a successful breeding outcome. Preferably, formation of a breeding pair should involve free mate selection, especially if this is important to the species. The aviculturist is recommended to evaluate for behavior signs indicative of a strong pair bond such as allofeeding, allopreening, and larger proportions of time spent in proximity to the partner (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). A strong pair bond facilitates the pair's ability to devote care to the eggs and chicks and increases the chances of success. Often, placid or fearful behavior is replaced by aggressive protective behaviors by one or both parents once the nesting cycle has begun. Some pairs become so aggressively protective that they may inadvertently damage or destroy eggs or chicks during or following nest inspection.

Egg destruction by one or both parents is a common cause of nest failure. This behavior is most often attributed to the male, which, upon entering the nest, breaks and often consumes the eggs. This behavior, if not identified, may lead the aviculturist to incorrectly assume the female is not producing eggs. Female birds have been observed eating eggs immediately following oviposition. Some birds can clean up every fragment of eggs, leaving no visible evidence that there were, in fact, eggs present. Some of these problem pairs can still be successful if the male is removed from the nest after copulation and prior to oviposition, although accurate timing for such a maneuver can be challenging. Egg drop boxes, which are designed to allow a fresh egg to roll into a protected drawer under the floor of the nest box, provide another possibility to save some of these threatened eggs (Figure 41.2). Alternatively, the aviculturist can check the nest frequently, particularly in the early morning when most eggs are laid or monitor oviposition by camera to ensure the eggs can be collected before they are damaged.

Water quality is important for the health of breeding pairs and neonates as water systems can be a common source of many pathogens. Automatic watering systems may easily become contaminated with gram-negative bacteria such as fecal coliforms or waterborne bacteria such as *Pseudomonas aeruginosa*. Overexposure and colonization by these organisms can lead to disease of both the parents and their chicks, including enteritis, sepsis, and death.[1] Common management strategies typically focus on regular flushing and replacement of water in the pipes of an automated system, resulting in a reduction of net bacterial counts noted at any point in time.

Nest cleanliness and quality are also important.[1] When nesting in their natural habitats, the success of a breeding pair is influenced by nest site availability and competitive ability of the pair to defend their nest, climatic factors, and predation or disruption by other creatures or organisms. In captivity these factors are much more efficiently controlled

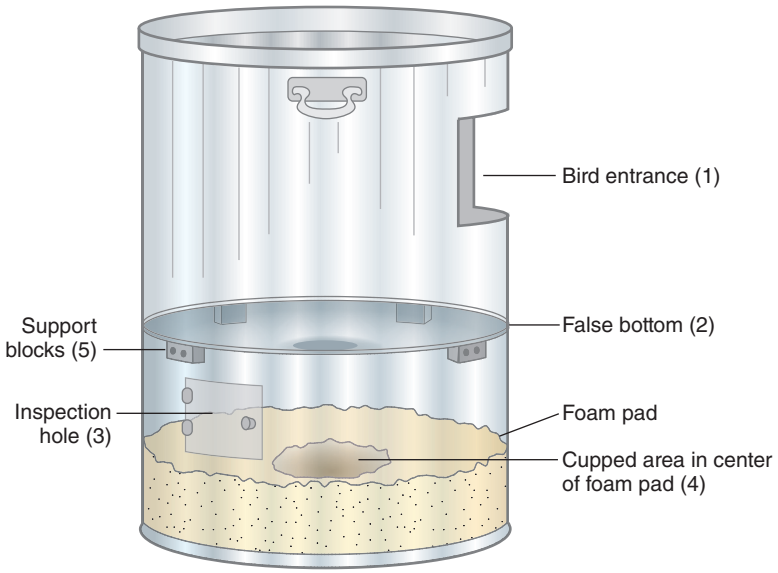


FIGURE 41.2 Nest box designs for breeding birds which have a tendency or predisposition to destroy their eggs. In this schematic, a fall-through design with a false bottom (2) mounted to support blocks (5) allows freshly laid eggs to fall down into a cupped area in the center of a foam pad (4), from where the eggs can be safely removed from the nest via the inspection hole (3) prior to being damaged or destroyed. (Photo from Abramson J, Speer BL, Thomsen JB, editors: *The Large Macaws*, Fort Bragg, CA, 1995, Raintree Publications, with permission.)

for by the design and placement of nest, the selection of nesting materials and substrates, and husbandry. Ensuring efficient techniques to clean the nest limits contamination with microorganisms, hence increasing the chances of successful breeding. Regular nest site inspection, accompanied by removal of damp materials and feces and followed by replacement of clean material, can significantly reduce pathogen buildup and potential risks.

Breeding under human care provides the opportunity to control and minimize many of the challenges faced by pairs nesting in the wild.[1,2] The educated and experienced aviculturist understands proper nest construction, size, shape, and placement, and its protection, and maintenance. Effective management of these variables removes many aspects of chance from the nesting cycle, hence, improving the probability of success. Ideally the environment should be designed such that the pair feels safe and free of disturbance, has proper perching for successful copulation, and plentiful of opportunities to provide proper parental care and nutrition.

Parental injuries to chicks in the nest typically occur with young or inexperienced pairs or when the parents do not feel secure in their nesting environment.[1,2] Pairs may not feed adequately, either in volume or frequency, resulting in stunting or even death of their chicks. Parents may nip the toes or wing tips of their chicks or sit excessively on the chicks in the nest, causing damage such as folding leg fractures, splay leg or other leg deformities, or back injuries. Young or inexperienced breeding pairs may remove, rearrange, or discard bedding from the nest, forcing the chicks to rest on hard, flat surfaces, which can lead to leg injuries and deformities. Panicked parents may also injure chicks upon hastily entering the nest following a perceived or real threat (Figure 41.3). Highly productive parents may also pluck or bite their chick's feathers once the pin feathers begin to emerge, which is largely viewed as an attempt to drive the chicks from the nest, so they can lay more eggs.

FACTORS INFLUENCING SUCCESS IN HAND-REARING CHICKS IN AVICULTURE

The Closed Aviary Concept

The closed aviary concept most likely serves as the single most unifying theme in modern aviculture. In the conceptually closed aviary, both clinical and subclinical diseases are controlled and monitored through adherence to the basic principles of designated areas and controlled flow of humans, animals, and materials. The concept of traffic control is used to reduce avicultural variables during or resulting from movements within designated areas of the aviary as well as within the aviary itself. Minimizing variables leads to reduced problems and maximizes the potential for attaining production goals.[4–6]

Coherence to the basic tenets of the closed aviary concept is an absolute requirement for the successful implementation of a flock health program. Without closed aviary management, the flock health program is easily violated, making disease incidence a random and unpredictable event. Realistically, such open facilities without coherence to closed aviary principles can only be managed on a crisis intervention basis, an approach which fundamentally undermines planned reproductive flock management (see also Figure 11.2).[6]

When chicks are being hand-raised, their dependency on their environment and nutritional needs dictates careful management, proper equipment technique, and adherence to procedural details. To provide altricial chicks with a microenvironment that meets their needs, brooder units that are

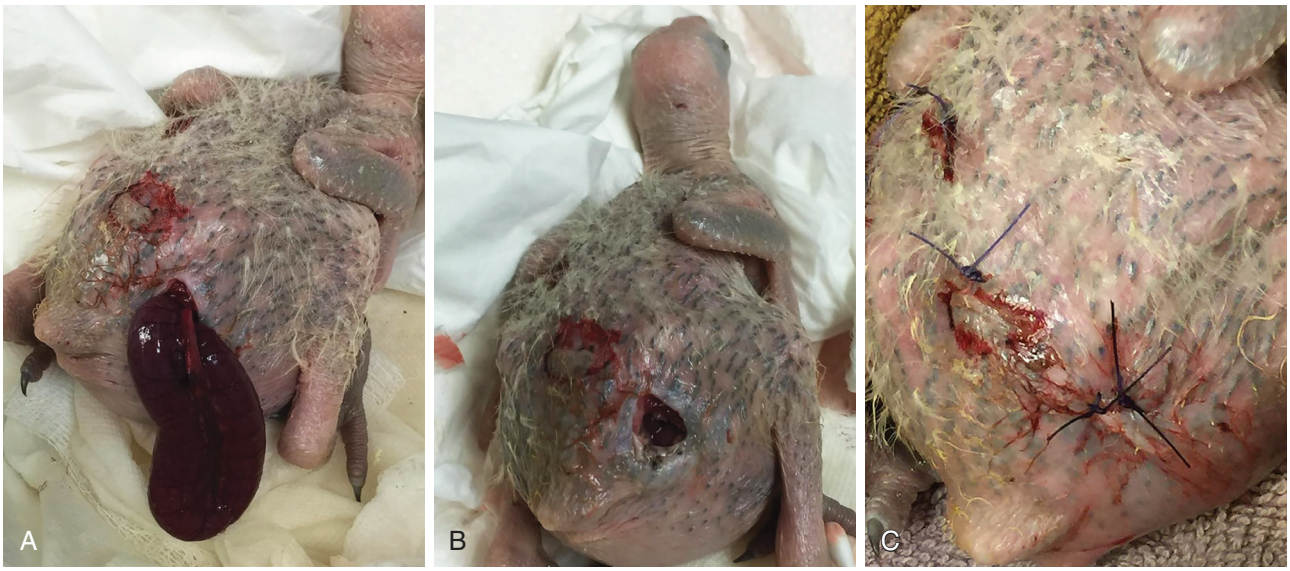


FIGURE 41.3 Parent-induced traumatic bite wound injury to a mealy Amazon chick *(Amazona farinosa)* during nest site inspection. (A) The lower penetrating wound has resulted in the protrusion of the duodenal ascending and descending loops, and their associated pancreatic heads. Surprisingly, when cleaned and inspected, there were no lacerations to the intestine itself or to the bones of the pelvis or appendicular skeleton. (B) Following cleaning, the intestine was replaced into the peritoneal cavity, and (C) the skin and body wall wounds were sutured. This chick recovered fully and was successfully hand-reared. (Photos courtesy Brian Speer.)

adjusted accordingly for the species and age of the chicks are required. In addition, appropriate nutritional support should be provided. High-quality and seasoned experience is probably the single most important factor responsible for successful artificial rearing of chicks. The American Federation of Aviculture (AFA) has developed a Fundamentals of Aviculture course, which is available online (https://afabirds.org/2018_WordPress/afa-fundamentals-of-aviculture-online-courses/) through a grant from Pet Care Trust and made possible by funding and other support from the Schubot Exotic Bird Health Center and the College of Veterinary Medicine at Texas A&M University.

Record Keeping

Chick records are perhaps the most dynamic and time-consuming records to keep, maintain, and analyze. The aviculturist may record parentage, date of hatch, daily or periodic weights, date eyes opened, age banded, identification, size of band, peak weight, and age at peak, weaning food started, perching, fledging, and weaning dates, sex determination method and results, any medical disorders encountered, and therapies administered. An example of a computerized record-keeping system is provided in Figure 41.4. Obviously, most facilities do not have the manpower or the time to record all the information listed previously, but the chick records give vital clues as to pediatric management. Remember, for most aviculturists and their attending veterinarians, increasing the numbers of viable chicks is the main production goal.[6]

Hand-Rearing or Fostering of Chicks

The ideal age to remove chicks from the nest to continue rearing in human care will depend on many factors, including the experience of the hand-feeder, availability of proper supplies and equipment, and experience of the chick's parents. The hobbyist aviculturist, wishing to produce future breeding stock, will often leave the chicks in the nest for natural fledging so chicks can learn about normal parenting techniques first hand. For those breeders who intend to hand-rear chicks for sale into the pet market or aim to optimize production, the decision may be made to pull chicks at various ages. Alternatively, compromised or at-risk chicks may be moved to nests of more successful parents to increase survival and production. Such fostering techniques offer a powerful avicultural tool when dealing with naive parents, which are often less successful in rearing their chicks, or when attempting to provide a rest period for extremely productive pairs. Likewise, many pairs will continue to be excellent parents after their own reproductive senescence.

Techniques for hand-rearing will vary by the species, the ages of the chicks, and the aviculturist (Figure 41.5). In the past, failures or challenges in neonatal care were mostly related to lack of knowledge regarding the specific neonatal nutritional and environmental needs. In recent years, however, many commercial hand-rearing formulas have been developed that meet the nutritional needs of commonly bred psittacine species. The feeding frequency and volumes for hand-rearing neonates are of particular importance (see Table 41.1 and Appendix 4A–E). Failure to provide enough food either due to inadequate numbers or volumes of feedings or due to inadequate concentration of nutrients can result in early stunting due to caloric deficiencies. The procedural challenges often depend on the stamina of the hand-feeder, because the needs of the chick must be balanced with those of the human caregiver to get adequate rest. Biphasic sleep cycles may help the

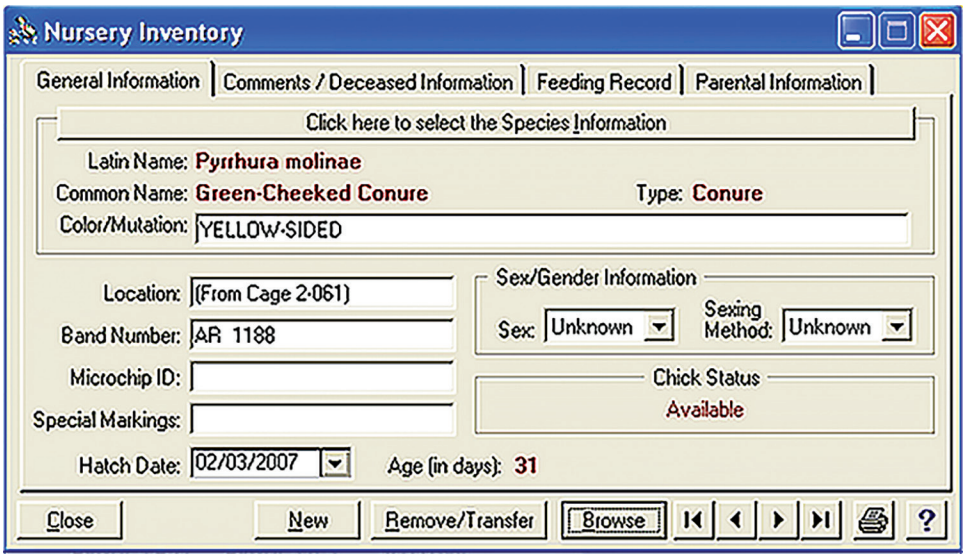


FIGURE 41.4 An example of a computerized record-keeping sheet for chick and nursery management data. Tabs for general information, comments or causes of death if appropriate, hand-feeding records, and parenting information can be seen. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

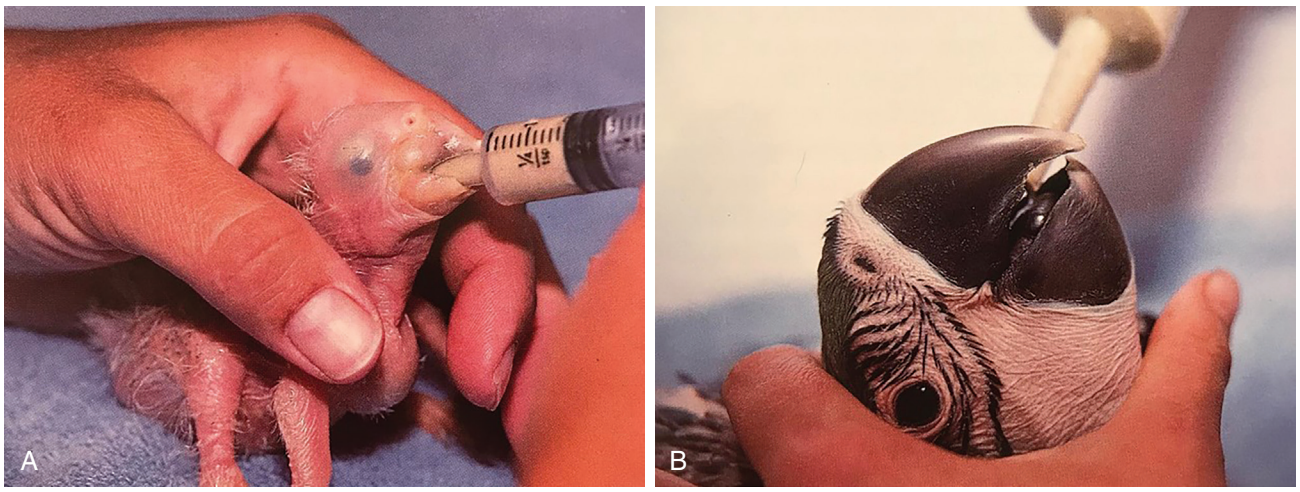


FIGURE 41.5 Hand-feeding neonatal (A) and nestling (B) macaw chicks. While stabilizing the head of the chick with the hand holding the syringe being used, risk of trauma during the pumping feeding behavior of the chick can be minimized. The chick is fed to the point of satiation, frequently filling the crop and the lower cervical esophagus. Current psittacine hand-feeding practices tend to use thicker formulas and to also not allow the crop to fully empty between feedings. (Photos from Abramson J, Speer BL, Thomsen JB, editors: *The Large Macaws*, Fort Bragg, 1995, Raintree Publications, with permission.)

human caregiver to meet a cycle of night feedings without risking inadequate sleep and associated health challenges. The prior debate as to whether the parents feed during the night has been resolved with nest cameras—the parents do feed during the night and in some species, this is done very frequently. Many highly successful hand-feeders will feed chicks every 2 hours initially with resultant rapid growth, mirroring growth rates of parent-fed chicks. Other breeders who hand-feed neonatal chicks for an entire breeding season have success feeding them using a schedule where the last feeding is administered at 11 or 12 p.m., while the first feeding the next day is provided at 6 or 7 a.m., allowing the caregiver to get adequate sleep.

Brooders

The function of a brooder is to provide an artificial environment for feeding and maintaining chicks immediately following hatching. A stable and warm environment is essential to successful hand-rearing, and newly hatched chicks should be maintained initially at incubation/hatcher temperatures,

TABLE 41.1

Feeding Frequency for Hand-Reared Psittacines

Developmental Stage	Feeding Frequency (Times per Day)
Chicks of 1–5 days old	6–10
Chicks with eyes closed	4–6
Chicks with eyes opened	3–4
Chicks with feathers emerging	2–3

Note: Chicks younger than a week may benefit from around-the-clock feeding; for older chicks feeding throughout the night is not mandatory/needed.

TABLE 41.2

Suggested Ambient Temperature Ranges for Psittacine Chicks

Recent hatchlings	33–34.5°C/92–94°F
Unfeathered chicks	32–33°C/90–92°F
Chicks with some pin feathers	29.5–32°C/85–90°F
Fully feathered chicks	24–26.5°C/75–80°F
Weaned chicks	20–24°C/68–75°F

Note that the actual temperature should be adjusted according to the needs of the individual chick.
Adapted and reprinted from: Harrison GJ, Harrison LR, Ritchie BW: *Avian Medicine: Principles and Application*, Lakeworth, 1994, Winger's Publishing.

following which temperatures can gradually be lowered as the chick matures (Table 41.2). Consistent and accurate temperature and humidity control is a key requirement for brooders, as it is for incubators. The design of these units should incorporate reliable and uniform control of temperature and humidity. A practical way to accomplish this is to have numerous brooders and to move the chicks to brooders with ever-lower environmental temperatures as they mature until they are sufficiently feathered to be maintained comfortably at room temperature. The cohousing with other chicks in such brooders can aid tremendously in both maintenance of body temperature as well as maintenance of normal hydration and provides bodily contact and auditory and visual stimulation that normally is received from siblings or parents. As such, companionship can help reduce stress-related issues, secondary opportunistic infections, and developmental abnormalities.

Many day-old chicks are initially placed individually in small cups or other plastic containers with paper towel substrates beneath them (Figure 41.6). This type of housing allows for individual attention to the chicks, while particular attention can be paid toward sanitation and cleanliness. Not all hatched chicks are placed in these types of environments, however. Some are maintained as a functional group or clutch together, and some chicks may be more comfortable if they are on shavings or other natural substrates. It is important to observe the behavior of the chicks as this helps to identify whether issues arise from an inadequate environment. Comfortable chicks will normally nestle calmly together, be relaxed, not show any panting, and will be warm to the touch.



FIGURE 41.6 Chicks are shown housed in a brooder, which provides uniform temperature and humidity for them at their young age. Room lighting is maintained dim. Separate groups or clutches can be housed within the same brooder, as demonstrated with the individual chick on the *left*, a clutch of two *centrally*, and a clutch of three on the *right*. Shared containers and brooders can facilitate early neonatal socialization development. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

Chicks that are hypothermic will be lethargic and feel cool to the touch. Hyperthermic chicks, on the other hand, will often be restless, redder in color, and will vocalize more. Most parrot species are nest cavity breeders, and their chicks generally are not exposed to much natural sunlight during the early aspects of their lives. In addition, providing a darkened recess into which a chick can retreat is consistent with their natural biology, and recommendable. As a result, it is recommended to maintain the lighting of the nursery and brooder environment comparatively dim, until the chicks become more developed and active.

An air circulation system with a low rate of air exchange or a passive airflow/heat delivery system is believed to be desirable to maintain an optimal brooder environment for newly hatched psittacine and other altricial chicks. A higher air turnover is more commonly used when managing precocial chicks.[7] Proper brooder temperature is essential to the well-being of the chick, especially if housed singly. A brooder that is too hot can result in dehydration, delayed gastrointestinal transit times, stunting, and other problems. A brooder that is too cold can also cause delayed gastrointestinal transit problems, while inadequate humidity is a common contributor to dehydration-related issues. Generally, humidity is maintained as high as possible with newly hatched altricial chicks and is decreased proportionately to common room conditions as natal down and contour feathers emerge. Brooders should be nonporous and easy to clean and disinfect. Substrate material that the chicks are maintained on should be clean (Figure 41.7). The ability to provide stable footing for the growing chick is an important consideration in substrate selection, and judicious concern for possible consumption of shavings by these chicks is needed. Extremely young chicks are frequently reared on toweling or fine shavings, whereas wood shavings are more commonly used in older chicks. Nest materials that should be avoided include those from aromatic woods including cedar,



FIGURE 41.7 A clutch of young green-cheeked conures *(Pyrrhura molinae)* are housed together on commercially manufactured paper product bedding as an entire clutch, in a single brooder. At this age of development, with significantly reduced bare skin exposed, brooder temperature and humidity are reduced as compared to what the chicks were maintained at while younger. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

redwood, and eucalyptus shavings, as well as soil, peat, and moist leaves, due to their potential to promote contamination and overgrowth of *Aspergillus* spores more easily as well as irritate the respiratory epithelium.

Diets

There are several formulated diets designed for hand-rearing psittacine chicks. These products are easily obtained from commercial bird retail stores or directly through online ordering sources in most parts of the world. Commercially manufactured hand-feeding products are utilized much more frequently in psittacine aviculture than home-made diets and recipes at the present time. Commercial formulas generally contain 16% to 26% protein, 3% to 16% fat, and 2% to 10% fiber. Some selected commercial diets and their compared guaranteed analysis are displayed in Table 41.3. Hand-feeding formulas should always be prepared fresh at the time of each feeding, using water heated to a temperature of 40°C to 41°C (103–105°F), according to the manufacturer's recommendations (Figure 41.8). Formula should be heated and mixed well, with particular attention to allowing uniform heat throughout it, as opposed to pockets that may be hotter than others, as may be seen in microwave-heated formula that is not stirred well before use. Excessively liquid diets can easily predispose to caloric deprivation of the chick. Excessively thick diets or cold formulas may predispose to delayed gastrointestinal transit time, dehydration, and secondary problems should the chick not already be in acceptable homeostasis (Table 41.3). Both extremes can result in stunting, or delayed growth, in the chicks, as well as secondary opportunistic bacterial and fungal infections, although thinner diets with reduced caloric content tend to pose greater risk in most opinions.

Hand-Feeding Equipment and Technique

Most psittacine chicks will have a variable degree of a feeding or "pumping" reflex. In most circumstances, the beak does not need to be held during hand-feeding, and by doing so, a hand-feeder may increase the risk of iatrogenic trauma to the immature beak tissues as a result of inadvertent pressure or bruising. Instead, by stabilizing the bird's head with the hand

TABLE 41.3
Selected Commercial Hand-Feeding Diets and Their Guaranteed Analyses

	Hagen Tropican	Harrisons Bird Diets Juvenile Formula	Kaytee Exact	Lafeber Company Nutri-Start	Roudybush Handfeeding Diet (Formula 3)
Protein (min)	22%	18%	22%	18.5%	21%
Fat (min)	11%	11%	9%	6.0%	7%
Fiber (max)	3.5%	4%	5%	1.5%	5.5%
Moisture (max)	9%	10%	10%	10.0%	12%
Ash (min)	—	4.2%	—		—
Calcium (min)	1.0%	0.9%	—	0.99%	—
Phosphorus (min)	0.8%	0.4%	—	0.55%	—
Iron (max)	85 mg/kg	70–100 mg/kg		139.1 mg/kg	
Vitamin A (min)	5500 IU/kg	5000 IU/kg		8137 IU/kg	
Vitamin D_3 (min)	550 IU/kg	1650 IU/kg		1293 IU/kg	
Vitamin E (min)	220 IU/kg	450 IU/kg		69.11 IU/kg	
Beta-carotene (min)	0.45 mg/kg	—			
Omega-3 fatty acids (min)	0.03%		0.5%	—	
Omega-6 fatty acids (min)	0.25%			—	
Folic acid		4.5 mg/kg		2.85 mg/kg	
Vitamin K		4.4 mg/kg		2.43 mg/kg	
Vitamin B_1		25 mg/kg		6.93 mg/kg	
Vitamin B_2		20 mg/kg		13.47 mg/kg	
Vitamin B_6		25 mg/kg		8.1 mg/kg	
Vitamin B_{12}		80 mg/kg		4.99 mg/kg	
Biotin		700 mg/kg		0.31 mg/kg	
Niacin		125 mg/kg		111.10 mg/kg	



FIGURE 41.8 Preparation of hand-feeding formula for a group of psittacine chicks. Note the relatively thick appearance of the mixture. This can be functionally made thinner if needed by adding a small amount of pancreatic digestive enzymes to the mixture, while retaining its total caloric content per milliliter. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

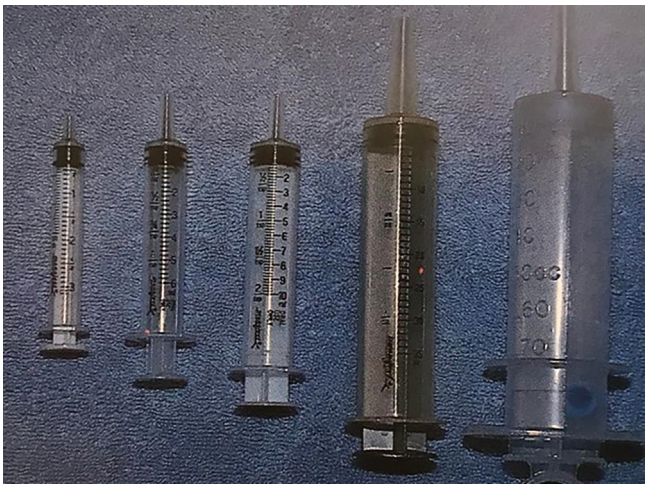


FIGURE 41.9 Catheter tip syringes of a variety of sizes that may be used for hand-feeding. Here, 3, 6, 10, 35, and 70 mL syringes are shown. In some circumstances, catheter-tipped syringes may also be used. (Photo from Abramson J, Speer BL, Thomsen JB, editors: *The Large Macaws*, 1995, Raintree Publications, with permission.)

holding the syringe, there will be reduced risk of injury (Figure 41.5). Variably sized luer-lock and catheter tip syringes (3–60 mL) are commonly used for hand-feeding psittacine chicks (Figure 41.9). Red rubber catheters or other soft rubber tubing can be used for tube or gavage feeding, if needed. Alternatively, manufactured metal gavage tubes can be used for feeding psittacine chicks. All items should be carefully cleaned and disinfected after use to minimize transfer of infectious disease agents as well as opportunistic contaminants.

The specific hand-feeding techniques that are used are aimed at supporting the head and neck of the bird, soliciting a feeding reflex, and delivering formula at a rate consistent with the swallowing rate of the chick. It is important to support the chick and feeding syringe concurrently, in a manner that prevents bruising or trauma to the pharyngeal tissues, or the rictal phalanges of the developing maxillary beak as traumatic or bruising injuries can lead to asymmetrical beak growth, resulting in lateral beak deformities or other problems (see also Chapter 31). A common, but currently outdated, requirement for hand-feeding young parrots was to allow the crop to completely empty between feedings. This practice predisposes many chicks to hunger, stress, inadequate caloric intake, and stunting. Parent-fed psittacine chicks are known to vocalize for parent attention and feeding prior to complete emptying of their crops. The more current hand-feeding techniques attempt to simulate this way of feeding and do not allow the crop to be empty in between feedings.

The volume of hand-feeding formula to be fed is dependent primarily on the crop capacity of the chick. A general rule of thumb is to feed approximately 10% of the chick's body weight with each feeding. Typically, the crop is filled to or close to the point of visualizing the formula coming up the upper cervical esophagus, which starts at the caudal aspect of the oral cavity and ends at the crop, down the right side of the neck. There is no specific requirement for feeding on the right side of the bird's mouth, contrary to common and popularly held belief (Figure 41.10). The frequency of hand-feeding also varies considerably depending on the species and age of the chicks but is dictated primarily by the chick's crop motility and growth rates (see Table 41.1 and Appendix 4A–E). The hand-rearing of some species, however, remains challenging. An excellent source of additional hand-rearing information for a large range of avian taxa is *Hand-Rearing Birds* by Duerr and Gage.[8]

The biggest variables in technique details are with the schedule of feedings and the percentage of solids of the food or formula, which influences caloric content. Monitoring the chicks while hand-feeding them will provide clues as



FIGURE 41.10 One common myth about hand-feeding young parrot chicks is that they must be fed from one side (typically the right) as opposed to the other because the cervical esophagus often lies on that side. In these two recently fed chicks, the filled crops and lower cervical esophagus can be seen on opposing sides of the necks. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

BOX 41.1 EVALUATION OF A CHICK'S WEIGHT

Chick weight at X days of age divided by "normal" weight for X days of age × 100 = Percentage of "normal"

to whether they are receiving adequate nutrition. Properly fed chicks will not beg constantly to be fed but are rather relaxed and will nestle together. They will be full-bodied and not appear desperate at feeding time. In addition, it is recommended to monitor chick weights during hand-feeding and to compare with established normal developmental parameters for the species as a point of reference, particularly if the hand-feeder is less experienced with a particular species.[5,9]

Parent-reared chicks are typically fed fluids along with the ever-increasing size of chunky foods by their parents rather than a liquid diet. An innovative aviculturist found this to be the secret to reducing intestinal stasis in palm cockatoo *(Probosciger aterrimus)* chicks, generally believed to be one of the most challenging psittacine chicks to artificially rear. This aviculturist developed a mixture of dried corn, nuts, seeds, and other grains, which was processed at home and fed to the chicks, initially starting with a finely ground mixture and gradually increasing the particle size to a chunkier mixture once the chicks matured.

One manner of utilizing a set of normal weight gain data in monitoring chick growth is illustrated in Box 41.1. This calculation can be serially evaluated during the hand-feeding process to assess if the weight gain represented by grams corresponds with this chick's comparison with normal. By not only monitoring the daily gain in grams for the hand-fed chicks but also monitoring the chick's relative percentage of normal, the hand-feeder is better positioned to detect developmental anomalies earlier. As a result, management changes can be made in a timelier manner, if needed, to have more opportunity and time to guide the chick's development back to normal. This highlights the importance of comparing the relative weight gain of chicks against a set of normal data for the species, as this may help a hand-feeder to identify subclinical developmental deficits that may otherwise remain undetected in a chick that is gaining weight (Box 41.2 and Figure 41.11). Appendix 4A–E present daily weights, growth rates, and food consumption for commonly bred psittacine birds.

Weaning And Fledging

Weaning refers to the maturation of a chick to the point where it can feed on its own, maintaining its body weight, often for a defined period of days to a few weeks. The weaned bird may or may not be fully fledged (i.e., capable of flight and leaving the nest) and will often still require parental and flock social interactions for continued acquisition of a full repertoire of adult behaviors. The age of fledging is variable among the many species of (psittacine) birds. In general, the larger the species, the longer the weaning and fledging period will be. As the chicks develop, their inquisitiveness, contour and flight feathers, and motor skills develop (Figure 41.12). During this key developmental time, fledgling chicks should be offered a variety of foodstuffs that help them to learn to eat. In general, the maintenance-type diet that is planned for these birds is the primary "weaning" food offered. During the weaning period, birds are allowed to become more active, to socialize and interact with other conspecifics, or even individuals of other species, when appropriate (Figure 41.13). They are also allowed to develop their foraging skills, to learn to accept different food items, and to still have a comfortable nesting environment to retreat to. Hand-feeding formula is still offered, but should the chick reject the feeding, no further efforts should be made to forcibly feed this. Ultimately, chicks will gradually accept less hand-feeding formula, to ultimately reject hand-feeding entirely. Some of the time-honored, but by many current standards antiquated, forced weaning techniques described in the past can be quite stressful to parrot chicks, resulting in secondary problems including but not limited to starvation, chronic stress, and increased susceptibility to opportunistic infections. Furthermore, these force-weaning techniques often downplay the importance of socialization, physical activity, and exploration aspects of the weaning and fledging period for the chick. During the fledging and weaning period, a normal drop in body weight of up to 15% can be observed in most parrot species. Typically, this weight is

BOX 41.2 AN EXAMPLE WHERE RELATIVE STATUS OF NORMAL DEVELOPMENT PERCENTAGE IS USED IN THE RECOGNITION OF AND MANAGEMENT OF STUNTING IN A BLUE-AND-GOLD MACAW *(ARA ARARAUNA)* CHICK

At 7 days old	Weight = 44 g Difference = 97% of normal	"Normal weight" = 47.3 g
At 14 days old	Weight = 89 g Difference = 68% of normal	"Normal weight" = 130.3 g
Management corrections: Increase from four feedings per day to eight per day, maintaining the same volume per feeding. Increase brooder humidity.		
At 21 days old	Weight = 229 g Difference = 75% of normal	"Normal weight" = 306.6 g
At 28 days old	Weight = 498 g Difference = 95% of normal	"Normal weight" = 522.2 g

Using a calculation of relative percentage of normal, the chick is identified as dropping from 67% normal at day 7 of age to 68% of normal at day 14 of age. Following management corrections, the chick is restored to 95% of normal weight by day 28.[10] Management changes that were instituted are inserted at the point of their implementation at day 14 and include an increase in feeding frequency of feeding but maintenance of the same volume per feeding, and an increase in brooder relative humidity.

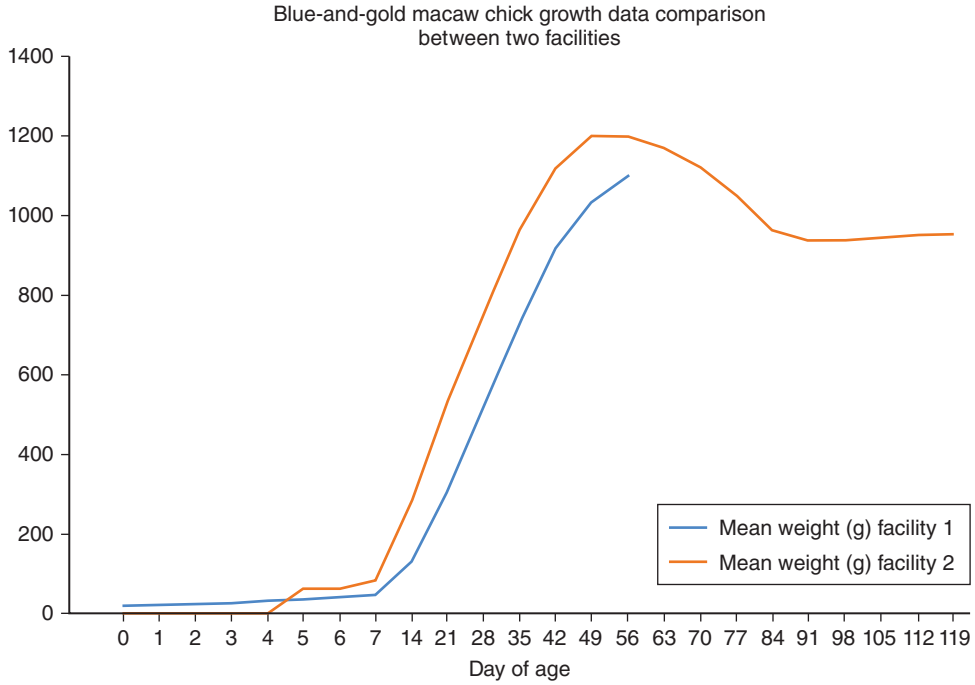


FIGURE 41.11 Example hand-feeding data and mean body weights by age for one species (blue-and-gold macaw, *Ara ararauna*) from two different avicultural sources. In source 1, the chicks were hand-raised from day 1, whereas in source 2, they were hand-reared starting at day 5 or older. Although the growth curves are similar in overall appearance, the chicks of facility 2 weigh more at any specific day of age as compared to those of facility 1. The greater the disparity from an optimal weight at any point of development, the greater the probability of the development of stunting-associated problems.[11,12]



FIGURE 41.12 Normal growth in scarlet macaw chicks *(Ara macao). From left to right:* 19 days old (315 g), 48 days old (1045 g), 17 days old (186 g), and 37 days old (965 g). (Photo courtesy Brian Speer. From Abramson J: Captive breeding and conservation. In Abramson J, Speer BL, Thomsen JB, editors: *The Large Macaws*, Fort Bragg, 1995, Raintree Publications.[5])



FIGURE 41.13 Grey parrot *(Psittacus erithacus)* chicks, being raised as a group and approaching an age at which weaning behaviors may be initially seen. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)

regained over the next 6 months to a year of life for the medium-to-larger parrot species and earlier for the smaller species. During these formative months, socialization by the bird's parents or other conspecifics is important and likely plays a key role in the learned behaviors as the bird approaches adulthood and beyond (see also *Current Therapy in Avian Medicine and Surgery*, Volume 1, Chapter 5).

PEDIATRIC PROBLEMS IN DEVELOPING PSITTACINE CHICKS

It is important for the practitioner, and even more so for the avicultural veterinarian, to be familiar with the equipment, procedures, and techniques used during artificial incubation and hand-rearing and understand the natural biology of normal chick development. An understanding of the differences between a normal chick and a chick that is developing problems will enable timely recognition and implementation of a successful intervention plan. This level of requisite familiarity goes to a species level: even closely related species may develop in a

different way, and these differences can limit the ability of the caretaker, or the veterinarian, to act in a timely manner.[7,9,13]

Clinical Examination

Several parameters can be used to assess the health and the general condition of a chick, although monitoring its growth and weight gain rates generally is the easiest way to detect abnormalities.[9]

After a chick has passed the first critical weeks of its life, a more careful and thorough examination can be performed, which will increasingly resemble the examination as performed in an adult bird. In fact, one of the biggest advantages of these young birds is their fast growth, which allows them to approximate their adult size in just a few weeks and makes life easier for the avian veterinarian.

Physical examination of the chick can typically be performed with minimal restraint. While performing the examination, it should be ensured that the chick is kept warm. The crop should be palpated at the start of the examination. If food is present in the crop, the chick should be handled with care to reduce the risk of regurgitation and/or aspiration.[14]

The clinical examination of a young chick follows the same manner as is used for any veterinary clinical examination and consists of three parts: (1) obtaining a meaningful history; (2) performing a thorough physical examination, including initial observations of the environment and the chick's behavior; and (3) selecting appropriate ancillary tests, if and when indicated. While the desired information collected during the clinical examination of a young bird does not differ much from adult birds or across species, the importance attributed to the various parameters can vary considerably.[7,9,13]

Signalment

Familiarity with the species and its normal developmental phases is important, since different species, even when belonging to the same taxonomic order or family, may develop differently. A complete and accurate description of the animal is therefore important, as well as double-checking client-reported information. The owner or caretaker of a young chick may belong to one of three main categories, that is, aviculturist, dealer (which includes owners and employees of pet shops), and private owners who have recently acquired a young bird. Presently, in several countries and individual states within the United States, it is forbidden to sell unweaned birds. This rule is often ignored and violations are rarely enforced. As a result, in practice, it is still common to have inexperienced owners present these immature chicks for veterinary care. Clients will have different experiences, perspectives, and expectations, and unweaned chicks may particularly be at risk to develop management-related health problems in the hands of an inexperienced owner.

History

The history of a young bird should include details regarding the living environment, diet, medical history, and—if possible—the reproductive history of the parents. A thorough reproductive history of the parents and of the whole breeding center, if possible, may help uncover inappropriate management procedures, nutritional errors, and possible presence of subclinical infectious diseases circulating in the environment. Using this information, the veterinarian may be able to follow a more targeted approach, limiting the selection of diagnostic tests to merely include those that are indicated in the given situation.[7,9,13] It is also important to evaluate whether and what medication is currently given as this can affect the course of disease and the ability to identify pathogens involved with the disease process. Especially aviculturists, breeders, and pet retailers will frequently have initiated a treatment based on clinical signs they have observed or presume to be present prior to visiting a veterinarian. This is especially the case in countries where antibacterials are available as over-the-counter drugs, though this type of practice is changing in many countries. In 2015, ultimately taking in effect in 2018, the state of California was the first state in the United States to ban the over-the-counter sale of antibacterial drugs and require a veterinary prescription for their use.

The history should also include a description of the main or presenting complaint(s). In many cases this can be challenging, as clinical signs are often not as clear in birds as they are in their mammalian counterparts. This is often attributed to the fact that birds need to hide signs of disease to avoid becoming an easy target for predators. For altricial chicks, however, masking of clinical signs is typically not needed.[13] This reality, to a reasonable degree, could simplify certain aspects of the diagnostic process.

General Appearance

Assessing the general appearance of a young chick can be challenging due to the differences in the development across species.[9] As a result, mistakes can easily be made when assessing the age of a chick, the development of its plumage, and recognizing the early signs of stunting.

Parameters that should be taken into consideration, when performing a physical examination on a young bird include the following:

- **Symmetry—**Any asymmetry should be noted. Some asymmetries may, however, be normal. For example, the crop predominantly lies on the right side, although the reverse can also be seen (Figure 41.10).
- **Muscle condition and hydration status—**Using one of the standard scoring methods, which typically involve a 5- or 9-point scale system in birds, the muscle condition score (a term intended to be more specific than an overall body condition score) can be assessed. It should be kept in mind that, in young chicks, muscle mass loss is best assessed at the epaxial musculature over the back, rather than at the pectorals. Hydration status can generally be assessed by evaluating skin turgor, with loss of skin elasticity often being the first sign of dehydration. Dehydration can occur quickly in a chick. Clinical signs of dehydration may include a dull red appearance of the chick's skin and there may be a decrease in the amount of urine being passed.[7,9,10,13]
- **Mentation and reaction to environmental stimuli—**Chicks should normally be bright and alert and raise their head in response to any activity that could signal food. Generally, behavioral parameters are assessed qualitatively and should take into account species-specific responses. Some behaviors, such as the reaction toward a perceived predator, are innate and species-specific. As a result, the reaction of a young macaw facing a human (predator) will be different from the one displayed by a cockatoo of the same age. Comparison of the behavior with other chicks



FIGURE 41.14 (A) A newly hatched Buffon's macaw chick *(Ara ambiguus)*, resting appropriately on a cloth substrate. There is symmetrical placement of the feet, a vertical angle of the tibiotarsi, and relaxed posture of the wings. (B) A stunted 12-day-old Calico hybrid macaw *(Ara chloropterus* × *Ara militaris).* This chick weighs approximately 68% of projected normal developmental weight. Note the inability to stand plantigrade and bear weight on the feet, elevated wing posture, and that the left leg is abnormally folded inward beneath the body of the chick. (A, Photo courtesy Brian Speer. From Abramson J: Captive breeding and conservation. In Abramson J, Speer BL, Thomsen JB, editors: *The Large Macaws*, Fort Bragg, 1995, Raintree Publications. B, Photo courtesy Brian Speer.)

from the same species and age may be useful in case of uncertainty of the normalcy of the response.[7,9,10,13]

- **Posture** is also important and can vary somewhat among species. Nestling chicks will sleep most of the time and are most active when hungry. They may sleep in any position and will generally sit on their hocks rather than on their feet until weaning. When evaluating the posture, one should specifically look for signs of incoordination, abnormal limb placement, leaning toward one side, and inability to lift the head properly (Figure 41.14). All of these signs indicate possible physical weakness, musculoskeletal problems, or neurological problems.

Physical Examination—Vital Signs

During the hands-on physical examination, several vital parameters should be evaluated in relation to presenting complaint, history, and current health status. The most important parameters that the veterinarian should evaluate include the following:

- **Body weight—**The body weight is important data to gather on any bird. However, equally—if not more—important is the growth rate of the chick, and how it compares to published ranges for chicks of the same species and age or to observed growth rates of chicks of the same species and age within the same breeding facility.[9,12,13] Typically, four phases can be recognized in the growth curve of altricial birds: (1) an initial lag growth; (2) a steady log phase; (3) a plateau phase just prior to weaning; and (4) a weaning phase, during which the chick loses some weight as a result of loss of body fat and gain of muscle mass (Figure 41.11). When the growth rate of the chick deviates significantly from the curve, further evaluation to identify the underlying cause for the abnormal growth is important, even in the absence of clear signs of disease.[13]
- **Body temperature/environmental temperature—**While measuring the body temperature is usually not considered a useful tool in the health assessment of a bird, the environmental temperature plays a critical role in (particular stages of) the life of altricial chicks. As a result, it is deemed important to inquire about the housing conditions and environmental temperature and assess whether these are appropriate for the chick, given its age. During the examination, a chick that is too cold may feel cold to the touch, often will have gut stasis, and will be relatively inactive (Figure 41.15). A chick that is too hot may separate from other chicks, may be breathing at a more rapid rate, and feel excessively warm to the touch.
- **Head and neck—**The head and face should be evaluated for symmetry. The eyes should be assessed for size, position, and presence of discharge. Sunken eyes and microphthalmia occur more frequently in birds than in mammals and may potentially be related to toxicosis. The beak and nares should be evaluated for symmetry and deformities, such as scissors beak and prognathism, which are still common issues in hand-reared chicks (see also Chapter 31). Conformation of the nares and the presence of nasal discharge must also be considered.[9,13] The oral cavity should be examined for moistness and the presence of plaques or other lesions.[14]
- **The crop (ingluvies)** is a tubular organ which is an enlarged part of the cervical esophagus. Among bird species and their taxonomic orders, there are significant variations



FIGURE 41.15 Two hybrid macaw chicks *(Ara chloropterus* × *Ara militaris)* being supported and monitored in their brooder. These chicks were initially stunted and hypothermic, and this photo was taken after the second day of corrective husbandry maneuvers. The relative humidity and temperature in Fahrenheit are displayed on the portable digital monitor in the *left upper corner*. The chicks were moved from individual containers to a common shared one, and toweling was used strategically to help them rest in a more normal posture. Additional humidity is being facilitated for the chicks by the use of water baths with cotton balls in the *upper right* and *lower left corners* of the photo. (Photo courtesy Brian Speer.)



FIGURE 41.16 Galah chick *(Eolophus roseicapilla)* that is 8 to 10 weeks of age, weighing 157 g. This body weight is approximately 50% of normal weight for this age and species, and significant stunting is present. The crop is enlarged, and palpably thin walled. Although it is capable of emptying, the time required to do so is significantly delayed. The clinical complaint of slow crop-emptying time was the purpose of presentation by the hand-feeder. (Photo courtesy Brenna Fitzgerald.)

in crop size and structure. Depending on its anatomy, it is either classified as a rudimentary crop (e.g., Anseriformes) that is long and narrow and only occupies a small space; as a "false crop" (e.g., griffon vulture, *Gyps fulvus*) that presents as a simple diverticulum of the esophagus; or a "real crop" (e.g., Galliformes) that is well developed and has a characteristic round-shaped bilobed enlargement.[15] Other species (e.g., owls, ostriches, cassowaries, kiwis, rheas, and many piscivorous species) lack a crop, and if a crop is present, this must be observed for wall muscular contractions. In addition, inquiries should be made on when the chick was last fed and how much was fed, as this will help to determine whether the crop is emptying normally.[14] Additionally, the skin covering the crop should be palpated, while paying specific attention to the crop wall texture and thickness (Figure 41.16). These are good indicators of the health of the crop, as well as of the hydration status and overall condition of the chick.

- **Trunk and limbs—**The chick's body should also be inspected for symmetry. In addition each limb and joint should be carefully palpated for abnormalities in angulation, deformities, swelling, bleeding, bony protrusions, obvious fractures or joint luxations, range of motion, or muscular atrophy. The condition of the feet and positioning of the digits should also be evaluated, and the vertebral column palpated to assess whether deviations or deformities are present. In the author's experience, kyphosis or scoliosis seems to be a common spinal deformity in young psittacines.[13]
- **Heart rate—**The heart rate in birds ranges from 50 to >615 bpm.[16,17] Most psittacine birds will have a heart rate over 250 bpm which can be difficult to accurately count and assess. Practical and reliable ways to measure the heart rate are the electrocardiogram (ECG) and echocardiography, although in the latter case the ultrasound should be equipped with a Doppler function.[18] The veterinarian will therefore more often evaluate the rhythm, rather than the raw number of beats per minute. A Doppler flow detector can be a useful tool for this purpose as well and seems to cause less stress for the patient. A pulse can be appreciated by palpating or placing a Doppler probe on the basilic (or ulnar, or wing), the jugular vein, or over the caudal sternum. In addition, the heart should be auscultated for the presence of murmurs, though these can be particularly difficult to appreciate if the heart rate is high.
- **Respiratory tract auscultation—**Auscultation is an important component of the clinical examination of every species, but auscultation may be problematic in young birds due to their relatively smaller size. One issue may be the stethoscope used: the standard 45-mm bell of instruments used for dogs and cats is far too large and heavy to perform an accurate and sensitive auscultation in a young and small bird. Therefore stethoscopes specifically

designed for pediatrics or neonatology, with a 15- to 25-mm bell, should be used. Unfortunately, even with these smaller stethoscopes it can be difficult to locate the sounds precisely. In the experience of the authors, whenever an abnormal sound is diagnosed with a simple auscultation, the abnormal finding should be investigated with the help of additional diagnostic maneuvers, including—but not limited to—radiographic or computed tomographic (CT) imaging. An altered respiration pattern may also be a normal response to stress in some parrot species. For example, chicks belonging to the genus *Pionus* will deepen and accelerate their breaths when fearful. While this observation does not exclude the presence of a respiratory disease process, it must be taken into account as a normal response to a stress.[13]

- **Abdominal region—**Although birds do not have a true abdominal cavity, the term abdomen is often used to indicate the body cavity region that contains the gut (intestinal peritoneal cavity), the liver (hepatic peritoneal cavities), gonads, spleen, and kidneys. The abdomen should be evaluated for signs of distention, deformity, displacement, symmetry, and bruising (Figure 41.17). In chicks, the umbilicus should be inspected thoroughly: omphalitis is a well-known disease in young poultry,[19] and altricial chicks can suffer from an umbilical infection, which can cause severe and generalized disease. In case of omphalitis, the umbilical scar may appear moist, exudative, and not completely scarred.

In addition to the aforementioned parameters, there are many other indicators of adequate health and growth that should be taken into consideration when assessing the physical condition of a chick. Some of these parameters can be specific for a certain species (e.g., ability to swim in water birds), whereas others are more generalized across species.



FIGURE 41.17 Normal appearance of the abdominal region of a newly hatched psittacine chick. The umbilicus appears clean and dry and is not protruding. Transillumination of the abdominal region may reveal the still-absorbing yolk sac within. The ventriculus is visible cranial and left laterally positioned to the umbilicus, and intestinal loops can be seen in the caudal ventral abdominal region. (Photo courtesy Steve Duncan, http://www.avianresources.com/.)



FIGURE 41.18 Normal developmental phases in blue-and-gold macaw chicks *(Ara ararauna). Clockwise from the top:* 76 days old (1010 g), 33 days old (924 g), and 19 days old (456 g). (From Abramson J: Captive breeding and conservation. In Abramson J, Speer BL, Thomsen JB, editors: *The Large Macaws*, Fort Bragg, 1995, Raintree Publications, with permission.)

Veterinarians are recommended to pay specific attention to at least the following parameters:

- **Normal plumage development**—Normal plumage development can vary considerably among species. Even within the order Psittaciformes, the time at which the normal plumage develops will vary according to the size of the species (Figure 41.18). Generally, the smaller species will develop, wean, and fledge at a younger age, with their plumage maturing quicker and sooner than larger species. However, even for species that are of an approximately similar size (e.g., cockatoos and macaws), differences can be noted in plumage development.
- **Feeding response—**The feeding response is largely an innate behavior and is considered a very important parameter to observe when assessing the young chick. There are considerable species differences in the way that adult birds will feed their chicks, as well as the way in which chicks respond to the environmental stimuli signaling the presence of food. For example, chicks of most Columbiformes (pigeons and doves) will not open their mouth while awaiting their parents to feed them, but they will rather introduce their beak into the parent's throat and "suck" the food directly from their parent's crop. Within the order Psittaciformes, macaw and *Eclectus (Eclectus roratus)* chicks are known to show a strong feeding response, literally "pumping" with their neck and head, while Grey parrot *(Psittacus erithacus)* chicks display much milder feeding behaviors. These species differences must be taken into account when assessing the feeding response, as a feeding response that is weaker than normal could be a nonspecific sign of disease. Hence, awareness of and familiarity with normal feeding behaviors for the species and individual are important.
- **Normal production and appearance of droppings—**The urofeces of chicks differ from the feces of the adult bird, particularly when the chicks are being hand-fed. The higher water content of the hand-feeding formula results in relatively loose feces and a relative increase in the amount of urine passed. The color of the feces may vary,

depending on the (hand-feeding) diet fed to the chick. Depending on the type of substrate on which the chick is kept, it may be more or less difficult to evaluate the quality of the feces: most beddings are hygroscopic and will absorb most of the water from the stools, making them appear drier than they actually are. As the chick grows and develops, the feces will gradually start to resemble the droppings of adult birds, thereby facilitating the evaluation of the droppings as may be expected with adults.

Routine Evaluations of the Chicks in the Nursery

Pediatric evaluations not only involve the medical assessment of single ill chicks but also the examination and monitoring of the other chicks in the nursery. The performance of the entire population within a nursery can provide insight into to management of the facility and identify possible opportunities for change that could reduce the prevalence and incidence of presenting problems. Regardless of the nursery location being in a breeding center, in a zoo, or in a private breeding collection, routine evaluations of the nursery will allow the clinician and aviculturist to obtain a detailed history of every chick and identify deviations from normality. These in turn can lead to earlier intervention, quicker isolation of potentially contagious conditions, and more successful treatment outcomes.[13]

Following discussion with the aviculturist/breeder, the veterinarian may consider testing the chicks, even the clinically healthy ones, for the pathogens most commonly encountered in the species of interest. Knowledge of pathogens that have been identified as causing problems in the facility in the past is also helpful in selecting which tests to perform. Common pathogens that are known to cause disease in nurseries include *Chlamydia psittaci*, pathogenic and opportunistic bacteria and fungi, and some select viruses including psittacine beak and feather disease (PBFD) virus, avian orthobornavirus, and polyomavirus. Testing the healthy-appearing chicks may highlight the presence of these primary pathogens, which can aid in facilitating appropriate flock management interventional actions. In addition, environmental opportunistic pathogens can be identified and more proactively managed. Water and food used in the nursery may also be routinely tested for the presence of potential opportunistic pathogens, or if indicated. There are reports of pathogens transmitted through the food, such as *Bacillus cereus* infections.[20,21] Other pathogens, such as *Pseudomonas aeruginosa*, *Cryptosporidium*, and influenza A virus, are associated with water sources.[22] Other bacterial disease outbreaks in a nursery, such as infections with methicillin-resistant *Staphylococcus aureus*, may be linked to human infections.[23–25]

The Critically Ill Chick

Young chicks can decompensate and die very quickly when seriously ill. For this reason, symptomatic treatments often must be started immediately after identification of true illness in a chick. In most cases, test results will be available well after when it becomes clear whether the initial course of treatment is successful or not. Nevertheless, appropriate diagnostic samples should be collected before initiating treatment as it allows treatment to be adjusted based on the obtained results, as well as provides a presumptive or working diagnosis for other chicks with similar clinical signs in the same facility.[13]

Typically, critically ill chicks will show signs of hypothermia and dehydration. Dehydration can occur quickly in a chick, and, if chronic, can potentially lead to several health problems, including but not limited to the development of constricted toe syndrome, stunting and its sequelae, and some forms of renal disease. Clinical signs of dehydration may include a dull red appearance of the chick's skin and there may be a decrease in the amount of urine being passed. If detected, rehydrate the chick by feeding electrolyte solutions, by subcutaneous fluid administration, but most importantly by contact rehydration with other clutch mates in an environment with increased humidity. Typically, a critically ill chick will look dehydrated and cold and will feel cold to the touch. Further clinical signs that are very common with sick chicks are inability to pass urofeces, delayed or lack of crop emptying, a crop with gas being produced within it or swallowed, and oculonasal discharge.

Most commonly fluids will be administered subcutaneously, and this can be safely done also in relatively small chicks, but some cases may require intravenous (IV) or intraosseus (IO) fluid administration. Fluid deficit can be estimated by the clinical status of the patient:

1. Loss of skin elasticity corresponds to an approximate 5% dehydration;
2. Skin tenting and loss of the normal sheen corresponds to 10% to 12% dehydration;
3. A depressed, or comatose chick with signs indicative of shock corresponds to 12% to 15% dehydration.[26]

Since bacteria can have a role in many of the pediatric cases encountered, regardless that many are secondary or opportunistic pathogens, initial antibacterial treatment may be indicated. Efforts to restore hydration and provide warmth are often indicated. In some settings, antifungal treatment may be indicated to avoid opportunistic overgrowth or invasion by yeast or other fungi.

Food is often offered with normal feeding procedures if the chick can pass it from the crop and through the remainder of the gastrointestinal (GI) tract. Risk of regurgitation or excessive crop distension should result in alterations in the volumes offered, the frequency of feedings, and/or the caloric density of the formula being used.

In case of crop stasis, the crop can be washed with warm saline solution (NaCl 0.9%) or lactated Ringer's solution. A small amount of saline solution may be left in the crop, after it has been carefully washed. In one author's experience, administering an infusion of fennel seeds can also help well. This "fennel tea" can also be used as a replacement for the fluid being used to reconstitute the feeding formula, for the first days after the resolution of the stasis. A thick mixture of hand-feeding formula may be used in some cases, particularly where it is assessed that meeting the chick's caloric and nutritional needs is an immediate need. Thick formula can be rendered more functionally thin by the addition of a small amount of pancreatic enzymes. This maneuver can allow a thinner formula which may pass easier, but retains caloric density to meet the nutritional needs of the chick.[13]

PEDIATRIC DIAGNOSTICS

Clinical Pathology

In general, immature birds have lower packed cell volume (PCV) (20%–30%), lower total protein (1–3 g/dL, 10–30 g/L), and lower albumen (1–2 g/dL, 10–20 g/L) as compared to adults of their species. Higher concentrations of alkaline phosphatase are described in younger chicks. Significant and progressive increases with age in biochemically determined values are seen in total calcium, urea, creatinine, uric acid, cholesterol, glucose, aspartate aminotransferase (AST), total protein, albumin, globulin, and prealbumin.[9,27] In the Spix's macaw *(Cyanopsitta spixii)*, adult and juvenile birds differed in mean concentrations of glucose, total protein, amylase, cholesterol, and phosphorus.[27] In the orange-winged Amazon parrot *(Amazona amazonica)*, concentrations of total protein, bile acids, phosphorus, total cholesterol, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol were significantly higher in adult compared with juvenile birds, while uric acid concentration was significantly higher in juveniles.[28] In wild nestling scarlet macaws *(Ara macao)*, albumin, aspartate aminotransferase, globulin, glucose, total protein, sodium, and uric acid levels increased with nestling age to adulthood.[29] Creatine kinase, phosphorus, and potassium concentrations decreased with age to adulthood.[29] Calcium concentrations did not change between nestlings and adults.[29] Similar observations have been reported in other avian species, including white storks *(Ciconia ciconia)*,[30] captive white-naped cranes *(Grus vipio)*,[31] ring-necked pheasants *(Phasianus colchicus)*,[32] and free-living Swainson's hawks *(Buteo swainsoni)*,[33] and likely reflect physiologic and developmental changes as nestlings mature.

Hematologically, young birds typically will have higher estimated or total white blood cell (WBC) counts as compared to their adult counterparts. WBC, mean cell volume (MCV), relative and absolute heterophil percentage, and relative and absolute monocyte percentage progressively decrease with age and relative and absolute lymphocyte counts increase with age.[9,27] Young ostriches *(Struthio camelus)* had significantly lower concentrations of hematocrit, hemoglobin concentration, calcium, and magnesium, and higher levels of total protein and potassium, than the adult individuals.[34] Similar hematologic observations have been reported in white storks,[30] captive white-naped cranes,[31] and ring-necked pheasants.[32]

Microbiology

Although normal fecal flora is generally presumed to predominately include gram-positive bacterial organisms (*Lactobacillus*, *Corynebacterium*, *Staphylococcus*, nonhemolytic *Streptococcus* and *Bacillus* spp.), the presence or absence of other organisms does not necessarily represent infectants or the absence of infection. Hand-fed chicks establish their intestinal microflora by ingestion of bacteria present in their environment and diet. It has been suggested that the prophylactic usage of antifungal drugs and the supplementation of *Lactobacillus* is not necessary for establishment of what should be deemed to be normal microflora.[9] Cytologic as well as microbiological isolates need to be interpreted considering the clinical setting, prior to being used solely to justify and refine antibacterial treatments.[13]

Molecular Diagnostics

As with their adult counterparts, polymerase chain reaction (PCR) testing can provide rapid, sensitive, and specific identification of many pediatric pathogens. As with microbiology findings mentioned previously, PCR test results should be critically interpreted regarding the clinical presentation and the management strategies being implemented for the nursery.

Radiography

In immature psittacine bird species, the proventriculus and ventriculus typically are much larger in size as compared to their adult counterparts, and these organs may fill most of the intestinal peritoneal cavity. The hepatic silhouette, as a result, may be increased. The subpulmonary air sacs as a result are more compressed and less apparent. In immature birds, the epiphyseal centers of ossification (the avian equivalent of the mammalian growth plates) of the appendicular and axial skeleton are open and appear as diaphyseal, lucent transverse lines. In the appendicular and axial skeleton, the epiphyseal centers are open and immature.

PEDIATRIC PHARMACOLOGIC CONSIDERATIONS

There is no current pharmacokinetic data available for drugs administered to hatchlings, nestlings, or fledglings. Because of the differences between precocial and altricial chick anatomy and physiology as compared to their adult counterparts, significant differences are likely to exist, which may influence therapeutic decisions made. In humans, the absorption, distribution, metabolism, and elimination of many drugs differ among premature infants, full-term infants, older children, and adults. Similar differences have been observed at different developmental stages in domestic animal species. Many of these treatment principles, extrapolated in part from other species as well as clinical practice experience, can be in making treatment decisions.

Drug Absorption

The two factors influencing the absorption of drugs from the gastrointestinal tract are pH-dependent passive diffusion and gastric emptying time.[35] Human infants at birth have gastric pH levels ranging from 6 to 8, which decrease to 1 to 3 within 24 hours. As a result, high serum concentrations of acid-labile drugs, such as penicillin, ampicillin, and nafcillin, after oral administration in premature infants can be explained by their higher gastric pH. In birds, pH, storage function, and emptying pattern of the crop influence the drug blood concentrations.[35] In chickens and pigeons, there are indications that bioavailability of some macrolide antibiotics is reduced by *Lactobacillus* crop flora because of inactivation.[36,37] In addition, percutaneous drug absorption may be substantially increased in newly hatched birds, particularly in altricial species, as is the case with human newborn infants, because of an underdeveloped epidermal barrier and increased skin hydration.

TABLE 41.4

Leghorn Chicken *(Gallus gallus domesticus)* Fluid Volumes at 1 Week of Age Compared With 32 and 55 Weeks of Age[38]

Fluid Volume Measured	1 Week Old	32 or 55 Weeks of Age
Total body water	72.4%	52.9% (55 weeks)
Total extracellular water	61.0%	26.2% (32 weeks)
Interstitial water	52.3%	21.7% (32 weeks)
Blood volume	12.0%	6.5% (32 weeks)
Plasma volume	8.7%	4.6% (32 weeks)

Distribution

The total body water as a percentage of the total body weight in a precocial hatchling is estimated to be 30% to 40% higher than in adult birds. At the age of 55 weeks, the total body water in white Leghorn *(Gallus gallus domesticus)* females was 52.9% of body weight, whereas at 1 week it was 72.4%.[38] Extracellular fluid volume is also markedly different in young animals; the extracellular fluid volume may account for 50% to 25% in juveniles and 20% in adults. In white Leghorn chickens, total extracellular water, interstitial water, blood volume, and plasma volume changed from 61.0%, 52.3%, 12.0%, and 8.7% of body weight, respectively, at 1 week to 26.2%, 21.7%, 6.5%, and 4.6%, respectively, at 32 weeks of age (Table 41.4).[38] Generally, the distribution volume for gentamicin in juvenile mammals is 50% to 80% larger than in adult mammals.[39] The same situation can be expected in birds, regarding water-soluble drug distribution volumes.[40]

Metabolism

Drug metabolism is substantially slower in infants compared with older children and adults. Drugs that require hepatic hydroxylation and/or glucuronide formation for elimination, such as chloramphenicol, sulfonamides, and flumequine in birds,[41] are expected to have longer half-lives in nestlings than in adults. Significant differences may exist in maturation of the various pathways of metabolism between precocial and altricial birds. Generally, precocial chicks may be compared with young children, whereas altricial chicks may more fairly be compared to neonatal mammals or even premature patients, pharmacotherapeutically.[41]

Drugs and their metabolites are often eliminated by the kidney. The processes of glomerular filtration, tubular secretion, and tubular reabsorption determine the efficiency of renal excretion. These processes may take several weeks to 1 year after birth to develop fully in humans.[42] There is inconsistency among species noted in renal elimination rates of types of drugs studied in newborns. The rate of elimination of gentamycin and neomycin in calves was not influenced by age, whereas the rate of oxytetracycline elimination was reduced significantly in studies of younger calves.[43]

In addition to pharmacokinetic differences, factors related to drug efficacy and toxicity should be considered.[41] Risk of chloramphenicol toxicity is increased in a newborn infant because of immature metabolism and enhanced bioavailability. Similarly, propylene glycol, which is added to many injectable drugs, including phenobarbital, diazepam, vitamin D, and ivermectin, to increase their stability, can cause hyperosmolality in infants.[44]

PEDIATRIC PROBLEMS

Understanding the Not-Doing-Well Chick

Incubation-related problems can affect the neonate after hatch. An egg that is incubated at improper relative humidity may hatch in a state of either over or underhydration. When detected, overhydration of the newly hatched chick can be corrected by lowering the brooder/environmental humidity. Dehydration may be addressed by increasing the brooder/environmental humidity or by providing supplemental fluids. Brooder humidity can be increased by the addition of damp, warm substrates beneath the chick, allowing for increased surface absorption of fluids. Supplemental fluids can be provided by oral administration of balanced electrolyte solutions. Care must be taken with oral electrolytes, as overhydration can occur. If present, this may be recognized by the presence of ventral edema and a relatively bloated translucent appearance in the chick.

Prior to hatch, the yolk sac should retract completely into the abdomen (Figure 41.17). Prematurely assisted hatches may result in exposure of the unretracted yolk or may interrupt the process prior to its completion, leaving a poor umbilical seal. Preferably, the chick is left in its shell until all these tissues and fluids are properly absorbed. Exposed yolk sacs may be protected by a light adhesive bandage until they are completely retracted and in some circumstances, may be tied off and even removed if indicated by likely contamination or known or suspected infection. Yolk sac infection may occur either via vertical transmission or through acquired contamination and infection. Vertical transmission may be related to either ovarian or oviductal infections. Omphalitis-causing bacteria may be identified by aerobic bacterial culture of the yolk sac in a dead chick or suggested by cloacal bacterial culture of the neonate. Alternatively, other eggs from the same hen may also be cultured, lending increased suspicion of the hen's contribution to these organisms' presence by repeated isolations of the same organism. A chick may also contract a bacterial infection of its yolk sac from a contaminated incubator or hatcher or through contaminated hands and instruments during assisted hatch, hence the necessity of excellent incubator and hatcher hygiene. If diagnosed in a timely enough manner, surgical omphalectomy offers the best prognosis (Figure 41.19).

Developmental abnormalities may be visibly apparent (Figure 41.20), or there may be undetectable genetic developmental abnormalities which may affect the viability of the neonate. Parental nutritional deficiencies are well known in poultry chicks; however, these are not well documented in

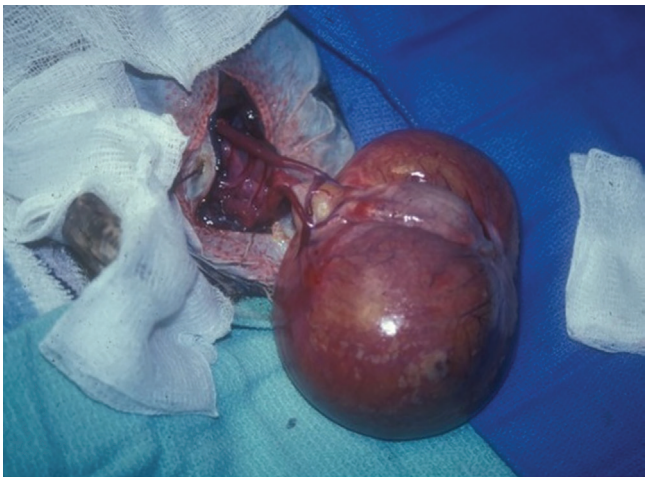


FIGURE 41.19 Surgical omphalectomy in an ostrich *(Struthio camelus)* chick. *Staphylococcus* sp. was isolated, and the infection was believed to be associated with opportunistic contamination during the hatching process, as the same organism was identified in the hatcher.



FIGURE 41.20 A large defect is visible involving the right nare, the lateral aspects of the maxillary beak, the right jugal, and the right pterygoid of this young blue-and-gold macaw *(Ara ararauna)* which was present at hatch. Although this deformity is congenital (present at hatch), its cause remained unclear. In this case, an acquired problem during incubation prior to hatch was suspected.

psittacine chicks. A lethal gene has been described and linked to the red factor gene. This gene is expressed in the F2 generation, when breeding two red factor birds together. Clinical demonstration of the central nervous system clinical signs appears around the time of weaning and continues until death.[1] Other documented congenital abnormalities include bilateral anophthalmia in a budgerigar *(Melopsittacus undulatus)*, varying degrees of cryptophthalmos and ankyloblepharon in cockatiels *(Nymphicus hollandicus)*, extrahepatic biliary cysts in a Grey parrot, and familial cataracts in scarlet macaws.[7]

Many presentations of problems within the realm of psittacine pediatrics tend to describe the ailment and its subsequent treatment. There is usually a mention of "secondary disease" and primary managerial problems, but there often is overall less emphasis on the underlying etiopathogenesis of many of these problems, as well as how to sort out the problem's origins, starting with the diagnosis. The information in Table 41.5 is provided to stimulate thought as to the underlying causes (hence true cure potential for individuals as well as management prevention for populations) that could be relevant for selected pediatric conditions. Keep in mind that pediatric medicine, in many ways, is an aspect of avicultural or flock medicine. The diagnosis and individual bird diagnoses lead toward potentially other thoughts and actions from the astute veterinary clinician.

INFECTIOUS DISEASES OF PSITTACINE CHICKS

Viral Infections

Polyomavirus

Psittacine polyomavirus infection was initially described as "budgerigar fledgling disease" causing high mortality of neonatal budgerigars in the nest.[45] Polyomaviruses are now recognized to infect and cause disease in a number of nonbudgerigar psittacine species and can vary in clinical signs among species and age groups. As a nonenveloped DNA virus, it is persistent in the environment and resistant to many disinfectants. Largely, infection and disease are seen in pediatric populations. In most cases, older psittacine birds (budgerigars over 1 month and nonbudgerigar species over 5 months) exposed to avian polyomavirus seroconvert and remain clinically normal.[46] The incubation period has not been evaluated in many species, but broad estimates of up to 15 days postexposure to onset of clinical signs, depending on age of the host, amount of viral exposure, and other host factors. It is believed that in the polyoma naive breeding and nursery population the risk of death and disease may be the highest risk. Clinical signs of infection and disease are outlined in Box 41.3.

There is no specific treatment for the disease, and the reported mortality rates have varied from 27% to 41% of the at-risk young birds in outbreaks.[46] Common gross necropsy findings include pallor of the skeletal muscle, hepatomegaly, subcutaneous hemorrhage or on the serosal surface of organs, and splenomegaly (Figure 41.21). Diagnosis is supported by history and gross necropsy findings and is confirmed with the identification of intranuclear inclusion bodies in several affected tissues at necropsy. Virus isolation or PCR testing can be used to further confirm diagnosis at necropsy. Antemortem screening for evidence of viral infection or exposure can be accomplished through serology or PCR testing of feces and cloacal swabs. Whole blood samples alone are generally viewed as a less sensitive means with which to screen for polyomavirus infection as compared to a combination of cloacal swab with whole blood. Disease prevention is accomplished through a combination of methods: high biosecurity in the nursery,

TABLE 41.5

Differential Diagnostic Approach to Common Pediatric Presentations[5]

Clinical Presentation	Differential Diagnostic Thoughts
Crop stasis	Dehydration
	Formula too hot
	Formula too cold
	Hypomotility related to stunting (see #9)
	Malnutrition
	Primary or secondary infectious disease (viral, bacterial, fungal)
	"Sour crop" as a secondary complication
	Brooder temperature too high
	Brooder temperature too low
	Brooder humidity too low
	Exogenous stressors (light, etc.)
	Foreign body obstruction of GI tract
	Normal variation in motility during 24-hour period
	Toxicoses
Regurgitation	Systemic disease
	Crop stasis (see #1)
	Normal behavior for species and age (weaning behavior)
	Inadequate feeding practices
	Excessive feeding, formula too hot or cold
	Imbalanced brooder environment
	Chicks too warm, too cold
	Localized ingluvial infections
Sinusitis	Inadequate brooder air quality
	Excessive temperature, poor ventilation, low humidity
	Foreign objects in nares
	Primary or secondary infectious disease (viral, bacterial, fungal)
	Inadequate feeding protocols
"Splay leg"	Inadequate brooder substrate
	Inappropriate nest/brooder design
	Lack of adequate nesting material
	Wet chicks
	Weak chicks
	Inattentive nursery management
Tibiotarsal rotation	"Splay leg"
	Traumatic injury
	Inattentive nursery management
	Inappropriate brooder substrate
Anteroflexed P1/P4 (zygodactylous species)	Inappropriate brooder substrate
	Malnutrition
	Inattentive nursery management
	Traumatic injury
Lateral beak deformity, mandibular prognathism	Likely multifactorial
	Hand-feeding trauma
	Malnutrition
	Sinusitis
"Diarrhea"	Normal fecal character for age and species
	Bacterial imbalance/dysbiosis
	Enteritis due to primary or secondary (bacterial, viral, fungal) infections of the GI tract
	Over medication (can result in dysbiosis)
	Malnutrition
	Hypermotility
	Hypomotility
	Pansystemic disease
	Endoparasitism
Stunting	Malnutrition
	Inadequate volume fed per day
	Inadequate calories fed per day
	Crop stasis (see #1)
	Inattentive nursery management
	Excessive brooder temperature
	Inadequate brooder temperature
	Inexperienced hand-feeders
	Inadequate adult breeder parenting
	Large interval between hatches
	Aviary disturbances
	Infectious parental disease
	Infectious disease (primary or secondary)

efforts to maintain a pathogen-specific population through serial testing and management procedures, and vaccination (Psittimune APV; Creative Science, Ballwin, MO, USA).

Psittacine beak and feather disease

PBFD was frequently seen in cockatoos during the years of highest importation into the United States (1975–1992) and remains endemic in wild parrot populations in Australia, Asia and Africa. In the decades following, a circovirus associated with the disease was identified. As with polyomavirus, psittacine circovirus is a nonenveloped DNA virus, and it is also persistent in the environment and resistant to many disinfectants (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). There are over 200 clades of different beak and feather disease virus genotypes currently described, and all psittacine birds are viewed to be susceptible to a diversity of these clades, with no one genotype considered more virulent than another.[47] A large number of susceptible species to infection and disease are listed, but the vast majority are Old World psittacine species (i.e. parrots originating from Africa, Eurasia or Australia). Conversely, naturally occurring infections and disease in New World parrots (i.e., species originating from North, Central, and South America) are rarely encountered. Although disease is described in adult birds, it is generally believed that infection and early onset of disease likely occur in subadults.[48] The incubation period has also not been evaluated in many species, but dystrophic feathers have been noted in experimentally infected galahs *(Eolophus roseicapilla)*, Grey parrots, umbrella cockatoos *(Cacatua alba)*, and sulphur-crested cockatoos *(Cacatua galerita)* at 21 to 47 days postinfection. The severity and onset of feather dysplasia are dependent on the phase of molt that the infected birds may be in; a bird that is not currently molting may not show classic

BOX 41.3 CLINICAL SIGNS ASSOCIATED WITH PEDIATRIC POLYOMAVIRUS INFECTIONS

General signs

1. Lethargy
2. Delayed crop-emptying time
3. Regurgitation
4. Lack of a normal feeding response

Budgerigars

1. Acute deaths in nestlings
2. Feather dystrophy in fledglings

Large psittacine species

1. Acute death (neonates within 12–24 hours after onset of signs)
2. Subcutaneous and systemic hemorrhage

Finches

1. Acute deaths (fledglings)
2. Beak keratin growth abnormalities (fledglings)

From Ritchie BW: *Avian Viruses: Function and Control*, Lake Worth, 1999, Wingers Publishing, pp 127–170.

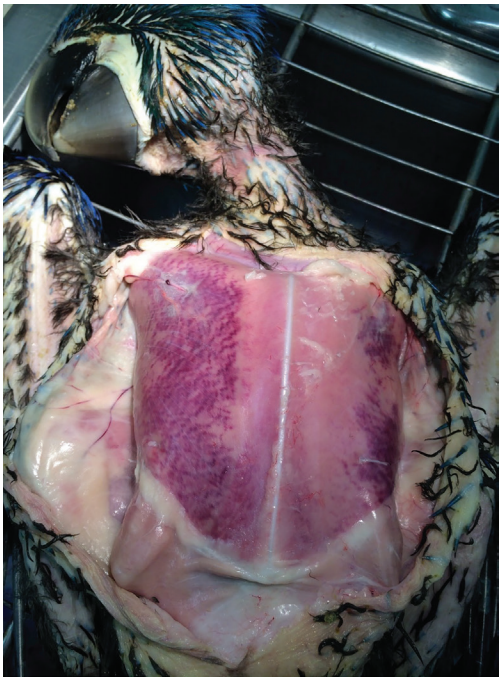


FIGURE 41.21 Ecchymotic hemorrhages are visible in the pectoral musculature of this hyacinth macaw *(Anodorhynchus hyacinthinus)* chick that died of polyomaviral infection. Hemorrhagic lesions were also present in the conjunctivae and over the brain beneath the skull cap. (Photo courtesy Susan Clubb.)

feather dystrophy signs until the next molt occurs.[48] Clinical signs of infection and disease vary among species and age groups. Peracute infections are typically presented with a rapid onset of debilitation and death, often with death occurring prior to the development of other clinical signs. Grey parrots may develop a necrotizing hepatopathy or nonregenerative



FIGURE 41.22 Psittacine beak and feather disease (PBFD, chronic form) in a young, approximately 6-month-old lovebird *(Agapornis roseicollis)*. Progressive feather dystrophy and loss are apparent, involving the contour feathers of the head and neck region shown. With magnification at examination, the dystrophic contour feathers were able to be more clearly identified. It was not clear if the rostral rhinothecal tomial overgrowth noted here is directly related to the disease. (Photo courtesy Brian Speer.)

anemia and granulocytopenia.[49] In the acute form of disease, pinched, clubbed, and dystrophic contour, down and powder down feathers are well described. Chronic disease is characterized by the progressive development of generalized feather loss and continued dystrophy. Beak and nail lesions may be noted in some species (Figure 41.22), typically in cockatoos affected by the chronic form of disease.[48] There is no specific treatment for infection or disease. Diagnosis is supported by physical examination findings and is confirmed with the identification of intranuclear or intracytoplasmic inclusion bodies in several affected tissues at skin biopsy or necropsy. PCR testing can be used to further confirm diagnosis and can be performed on biopsy samples or whole blood samples, as persistent viremia is believed to be a common characteristic in actively infected birds. Serologic testing is available in Australia only. Paired with a reduction in wild-caught bird trade, the use of targeted testing and removal of infected birds from breeding facilities appear to have aided in significantly reducing the prevalence of disease in captively managed collections in many parts of the world.[48] PBFD continues to affect wild psittacine populations in Asia, Africa, and Australia and can be a significant conservation issue in some species such as the red-vented or Philippine cockatoo *(Cacatua haematuropygia)* and the Cape parrot *(Poicephalus robustus)*.[50,51] There is no commercial vaccine available.[47,48]

Pox

Poxvirus can be transmitted from wild or other infected birds via mosquitoes and other biting arthropods to parrot chicks in the nest (see also Chapter 15). Pox infections overall are more commonly encountered in other taxonomic groups of young birds (Columbiformes, Galliformes) as compared to Psittaciformes, although disease is reported in many bird groups



FIGURE 41.23 Cutaneous pox in an ostrich *(Struthio camelus)* chick. (Photo courtesy Brian Speer.)

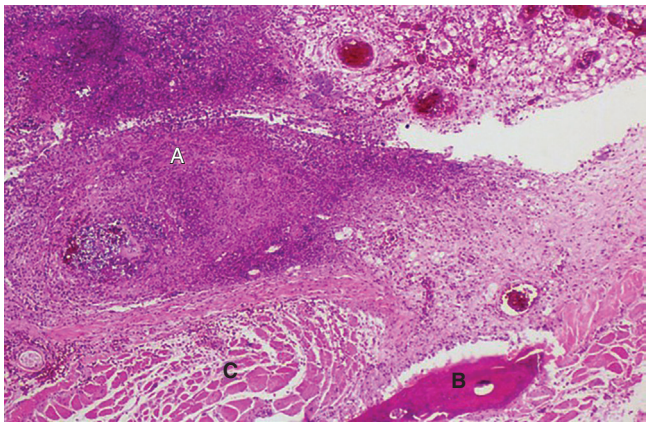


FIGURE 41.24 Histologic section of a young cockatiel *(Nymphicus hollandicus)* chick that likely died of complications from *Bordetella avium* infection (lockjaw). Note the extensive necrotizing myositis *(A)*. A fragment of bone involved in the reaction is also present *(B)*. Relatively normal skeletal muscle is also present *(C)*. These lesions may be secondary to infection that originated in the nasal cavity or sinuses, but they have also been associated with oropharyngeal trauma during hand-feeding. (Photo courtesy Robert Schmidt.)

(Figure 41.23). Infection can be transmitted by direct contact as well as fomites. Infected chicks may show cutaneous or diphtheritic manifestations, characterized by raised lesions on the skin, eyelids, inside the mouth, and on the exterior surfaces of the beak, feet, and in some cases lesions over much of the body. These cutaneous lesions will eventually scab over and when the birds recover. Secondary infections can complicate recovery, and diphtheric lesions may carry a more guarded to poor prognosis as compared to cutaneous lesions for this reason.

Proventricular dilatation disease (avian ganglioneuritis)

Proventricular dilatation disease, associated with avian bornavirus can cause disease in chicks as young at 20 days old (see also Chapter 14 and *Current Therapy in Avian Medicine and Surgery*, Volume 1, Chapter 2).[52,53]

Other viral infections

A novel metapneumovirus was identified by metagenomic sequencing in monk parakeets and associated with a disease outbreak in a psittacine nursery facility. Clinical signs included acute death and nonspecific signs of ill-thrift. Gross necropsy revealed yellow-colored livers with petechia, suggestive of an acute hepatic necrosis.[54] Avian adenoviral infections can function as comorbidities, increasing the prevalence of other infections or at times can function in the role of a primary pathogen.[55]

Bacterial Infections

Common environmental bacteria, particularly coliforms, are potential pathogens for neonates, especially if husbandry is not optimal. Many, but not all, of these organisms can become opportunistic infectants, often secondary to primary host and environmental factors that may be present. *Escherichia coli* and *Klebsiella pneumoniae* are commonly encountered environmental contaminants and infectants in many large commercial or zoological collection psittacine nurseries. The source of these bacteria often defy detection, and they may emerge and be found to be problematic even in the presence of exceptional hygiene. Common sources for consideration of contamination include the human caretaker, the cleanliness of the environment, the hand-feeding formula being used, water, and vertically passed organisms from the hen or her nesting environment. *Pseudomonas aeruginosa* and similar hydrophilic bacteria present a challenge to neonates because of their ubiquity in water sources and ability to form biofilms, which not only support their resistance to environmental factors but also protect other bacteria such as coliforms. These bacteria present no danger to humans unless exposed people are immunocompromised. Intestinal disease and sepsis are the most common clinical signs noted in chicks; however, *Bordetella avium* has been associated with infection of ciliated respiratory epithelium in turkeys *(Meleagris gallopavo)* and other poultry as well as a "lockjaw" syndrome in neonatal cockatiels and other psittacine species (see also Chapter 31).

Asymptomatic infection with *B. avium* has been demonstrated in adult breeding pairs and shedding of the organism in their oral secretions to their chicks is a common route of transmission.[56–58] Depending on the chicks' age as well as other management-related variables (timing of recognition of the problem, nutritional status of the chick, and hand-feeding practices), the affected chicks may die of acute pneumonia or may develop a disease progression characterized by coughing, sneezing, serous nasal discharge, progressing to prokinetic muscular rigidity "lockjaw" in chicks roughly 4 to 6 weeks old. The bacterium invades from the nasal mucosa into the suborbital sinus and directly into the prokinetic musculature. Inflammation at these sites causes the prokinetic rigidity and loss of range of motion of the associated joints manifested as "lockjaw" (Figure 41.24). Often, there is a characteristic raised lower lid and glassy appearance to the eyes. Affected chicks endlessly beg for food while being unable to open their jaws to accept formula from adult birds or hand-feeders. Some



FIGURE 41.25 Dilated hemorrhagic and edematous intestines, typical of what is seen on the serosal surface in cases of bacterial enteritis. (Photo courtesy Robert Schmidt.)

successful treatments have been noted, where paired antiinflammatory treatments, pain management, physical therapy, and antibacterial therapy were all utilized.[59] In a similar manner of clinical presentation, *Enterococcus* spp. believed to be a part of the psittacine normal gastrointestinal flora, invaded through mucosal injuries into the adjacent subcutaneous tissues. Susceptibility to this specific bacterial invasion is anecdotally believed to be seen in cockatiels.[57]

Clostridium spp. organisms are common components of the normal gastrointestinal flora and can often overgrow in numbers secondary to antibacterial therapy or other factors including stunting, dehydration, or delayed GI transit problems. *Clostridium* spp. overgrowth can result in primary or secondary intestinal disease in neonates, fecal malodor, gastrointestinal discomfort/distention, and including necrotizing enteritis and/or typhlocolitis (Figure 41.25). In general, most disease processes in neonates reduce intestinal transit time, which can result in overgrowth of anaerobic organisms, and as a result, not all clinical signs a chick may present necessarily have to have a direct causal relationship with the bacterial organism suspected or identified.[60]

Chlamydia psittaci infection and disease appear to be no longer nearly as common in US aviculture as compared to prior years in the experience of many US veterinarians, but neonatal infection with *C. psittaci* could be significant in other countries (see also Chapter 18 and *Current Therapy in Avian Medicine and Surgery*, Volume 1, Chapter 2). The apparent reduction in prevalence of chlamydiosis noted in US aviculture is attributed to the implementation of increased screening of breeding stock as well as young birds entering commercial trade. *Staphylococcus* spp. skin infections occur periodically in neonates and may be associated with damp nests, high relative humidity, or infection secondary to insect bites.

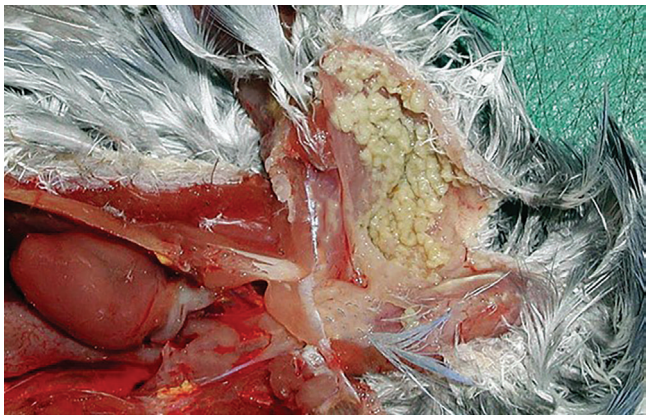


FIGURE 41.26 Ingluvial candidiasis. Note the "Turkish towel" appearance of the thickened and discolored mucosal surface. (Photo courtesy Robert Schmidt.)

Fungal Infections

Yeast infections

Yeast, most commonly *Candida albicans*, are another common component of normal intestinal flora which can become secondary pathogens in the face of antibacterial therapy or other factors as with *Clostridium* spp. Other *Candida* species are beginning to be increasingly recognized in birds as well. *Candida glabrata* and *Candida krusei* have been reported.[61] If husbandry is suboptimal, *Candida* can become a significant and sometimes aggressive pathogen. Mycotic ingluvitis is a superinfection of the mucosal surface of the crop (Figure 41.26), and infections also can occur in the intestinal tract. These intestinal tract infections can cause gastrointestinal stasis, reduce adsorption of nutrients, lead to stunting, and in advanced cases result in death. Prophylactic antifungal therapy in some situations is recommended at any time a neonate is given antibacterial therapy. Antifungals commonly used in neonates include fluconazole, ketoconazole, and nystatin (see also Chapter 8).[62]

Aspergillosis

Aspergillosis can occur if chicks are severely immunocompromised (e.g., in case of acute PBFD), or when exposed to large numbers of *Aspergillus* spores in the environment. This may occur if bedding is not changed with reasonable frequency and becomes contaminated. Cleaning this contaminated bedding in the same airspace as chicks can result in aerosolization of spores and excessive spore inhalation. Contamination of air conditioners that may be being used or hatcher and brooder fans and filters can also result in dissemination of dangerous amounts of airborne fungal spores in the nursery environment (see also *Current Therapy in Avian Medicine and Surgery*, Volume 1, Chapter 2).

Parasitic Infections

Cryptosporidiosis

Cryptosporidium spp. associated with enteric disease has been reported in outbreaks in a psittacine nursery.[63] Utilization of PCR for screening of neonatal chicks when removed from the nest has demonstrated that this may be a significant emerging pathogen in psittacines as well as being potentially zoonotic (see also *Current Therapy in Avian Medicine and Surgery*, Volume 1, Chapter 2).[64] *Cryptosporidium* infection and disease

have been reported in young passerine species,[65] waterfowl,[66] and bobwhite quail *(Colinus virginianus)*.[67–69]

Dermanyssus infestation

Red mites (*Dermanyssus* spp.) occasionally infest a nest box and can cause mortality of chicks due to blood loss. These nocturnal mites will typically not be visible during the day, hiding in cracks and crevices in the nest. They emerge at night to bite their host resulting in anemia and death. Adults will often be seen scratching or showing pruritic behaviors. Control requires extensive cleaning and spraying of insecticides in all parts of the aviary. Preferably the nest box will be burned (if wooden). Perches should be replaced, sprayed with insecticides, or baked. The addition of 5% carbaryl or permethrin powder to nest box substrate can help to control these parasites (see also Chapter 9).[1]

NONINFECTIOUS DISEASES

Nutritional problems are typically the most common noninfectious category disorder noted in psittacine chicks in their first 30 days of life. Metabolic bone disease, stunting, gastrointestinal disorders, and increased susceptibility to opportunistic infections are encountered. For psittacines, the commercially available formulas will meet the nutritional requirements for most species if mixed and administered properly. Percentage solids, the ratio of water to dry formula, is a critical factor in success. Most companies' directions for feeding their product typically show a more dilute formula for younger birds, with increasing percentage solids until it reaches around 24% to 25% solids (Table 41.3). To check mixing consistency divide the weight of the dry formula by the weight of the mixed formula in grams.[1,9,70]

Husbandry-Associated Problems

Crop burns

Thermal burns of the ingluvies result from feeding baby bird formula that is too hot (Figure 41.27). This can often occur when a microwave oven is used to heat the formula (not recommended because of the formation of hot spots within the formula).[14] Mild cases may result in red and inflamed skin around the crop and may have no other outwardly evident clinical signs other than a transient shift in feeding behaviors from what was normally seen. Second- and third-degree burns will be acutely inflamed and blistered, and these injuries may lead to tissue necrosis and fistula formation. In many cases, birds may be presented with food draining from a fistula through the crop wall and skin. Diagnosis is based on history, clinical signs, and physical examination findings.

Medical management often includes the use of antimicrobials for secondary infectants that may be present, supplemental parenteral fluids, antiinflammatory drugs and other pain management strategies, and nutritional support. Surgical repair is often required but should be postponed until the burned area is well demarcated (usually several days). In such cases, placement of a gastroesophageal feeding tube should be considered to ensure adequate nutritional intake. Surgical repair involves debridement of devitalized tissues, separating the crop wall from overlying skin, and closing the two layers separately. Prognosis is good if there is sufficient crop wall remaining for closure and the esophagus is intact.[1,26] Note that not all thermal burns to the crop need to be addressed with surgery at the time of initial presentation. Instead, healing by secondary attempt while ensuring appropriate supportive care in the meantime. An example of such a staged repair of a crop burn is represented in Figure 41.28.



FIGURE 41.27 Crop burn. There is a centrally necrotic area through which formula can be seen leaking from a fistula. Although there is a clear demarcation between necrotic and live tissue apparent at the bottom of the lesion, the lateral and upper aspects are not fully differentiated, supporting a need to delay surgical repair if possible. (Photo courtesy Susan Clubb.)

Aspiration of formula

Aspiration pneumonia is one of the most common causes of respiratory disease in hand-fed psittacine birds (Figure 41.29). Chicks can aspirate while being fed large quantities of liquid formula, especially when being fed by an inexperienced or inattentive feeder.[14] Aspiration of formula can result in instant asphyxiation and death if the volume of formula introduced into the trachea is sufficient to occlude the bronchi. Less severe aspirations can also occur when a chick inhales small amounts of formula resulting in the development of pneumonia. Clinical signs include increased respiration, respiratory distress, poor feeding response, and depression.[14] The most practical way to prevent aspiration is by conditioning the chick to accept the food rapidly, which is the natural way in which the parents feed the chick. When the chick is pumping to accept food, the glottis is closed preventing aspiration. Inexperienced hand-feeders often feel "safer" dribbling the food slowly into the chick's mouth. At some point in time, the chick must open the glottis to breathe and may aspirate. Treatment consists of oxygen therapy, nebulization, antibacterials, antifungals, warmth, supplemental fluids, and antiinflammatory drugs.[14]

Oropharyngeal trauma

Punctures of the pharynx may occur if the hand-feeder is using a small, firm tip on the hand-feeding syringe or is using a syringe with a sharp point. If the chick is bobbing to accept

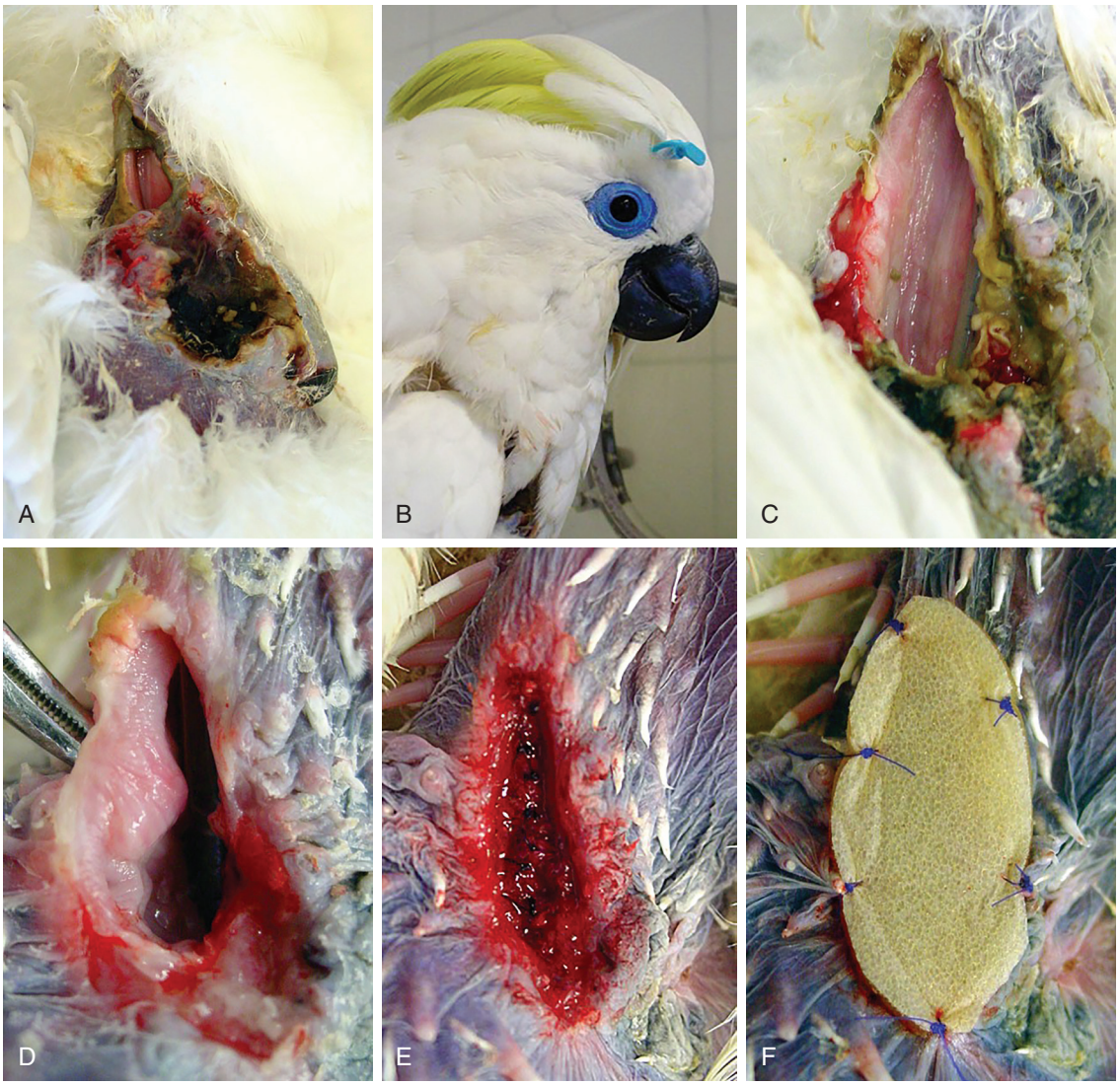


FIGURE 41.28 (A) Primary crop burn in a blue-eyed cockatoo *(Cacatua ophthalmica)*, with over 50% of the crop wall destroyed and large necrotic lesion ventrally at the initial day of presentation. (B) Placement of esophageal feeding tube attached to the head to bypass wound and allow provision of liquid formula until the necrotic tissues are better declared. (C) Wound appearance 1 week later. Demarcation is present in the proximal area, but the distal area is still not fully demarcated. (D) At 3 weeks after initial presentation, the wound had clearly decreased in size and was now well demarcated. (E) Crop wall could be closed with single monofilament sutures. (F) Wound was covered with hydrocolloid dressing (DuoDerm, Convatec, London. UK) to let allow for healing by second intent. (Photos courtesy of Nico Schoemaker/Yvonne van Zeeland.)

the formula from the syringe, the caudal pharyngeal tissue may be punctured or lacerated, and food material can be deposited subcutaneously into tissue of the neck. A relatively sudden onset of marked swelling in the gular region and upper neck is a commonly noted clinical sign, often due to cellulitis and infection/inflammation in the region. When recognized, this is an urgent situation, often requiring surgical exploration, cleaning of the area, irrigation, and repair of the inciting injury. If not addressed in a successful or timely manner, death is likely to occur due to toxemia and sepsis within 24 hours.[1] In addition to caudal pharyngeal injuries, punctures of the esophagus can also occur when tube feeding chicks. These injuries typically occur approximately halfway down the esophagus, where it is narrow, and the cervical spine has a normal flexure. The swelling that is noted will be in the lower neck and sometimes in the region of the crop. As mentioned earlier, when recognized, this is an emergency, often requiring surgical resolution within 24 hours to prevent toxemia and sepsis, which significantly reduce the chances of survival.[1,26]

Hepatic rupture

Hepatic rupture can occur when a chick is lifted with fingers surrounding the body and applying pressure to the liver or if there is a fall and abrupt blow sustained to the area (Figure 41.30). There



FIGURE 41.29 Aspiration and foreign body pneumonia. The lungs appear edematous and discolored. Hand-feeding formula can be seen in the adjacent air sacs grossly. (Photo courtesy Robert Schmidt.)

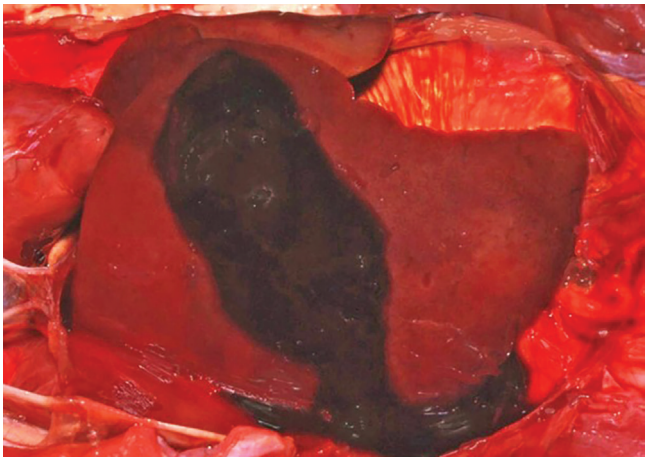


FIGURE 41.30 Hepatic rupture and grossly visible hemorrhage. Chicks may suffer acute death or present weak and pale. Some birds may survive with supportive care. (Photo courtesy Robert Schmidt.)

may be some underlying nutritional deficiency associated with this disorder, as one of the contributing authors has observed it primarily when chicks were being fed a formula made from primate biscuits.[26]

Obesity

Some species, typically large cockatoos, can have a tendency toward obesity which can result in fatty liver disease if they are given excessive amounts of food (Figure 41.31). Chicks with hepatic lipidosis typically are still being hand-fed and are overweight for their age. Affected chicks often exhibit marked signs of respiratory distress, largely due to reduced air sac space and increased vulnerability to hyperthermia. These birds must be handled gently and minimally. A cooler environment that is oxygen supplemented is often the best first step. By greatly reducing the quantity of crop food per feeding, adjusting the caloric content of the formula downward, many chicks can begin to stabilize and improve within days.[14]



FIGURE 41.31 Obesity in a sulphur-crested cockatoo *(Cacatua galerita galerita)* chick. The bird is standing in a more erect posture and gaping to breathe due to the significantly filled intestinal peritoneal cavity (largely hepatomegaly) resulting in air sac impingement and ventilatory impairment. Initial treatments included cooling the bird paired with passive oxygen support, followed by significantly reducing the calorie density of hand-feeding formula that was fed. (Photo courtesy Brian Speer.)

Gastrointestinal Problems

Ingluvial hypomotility or stasis and the development of "sour crop"

Crop atony, crop stasis, or failure of the crop to empty is a common clinical sign, often related to intestinal stasis or delayed GI emptying in young chicks. Under normal circumstances, the crop of the chick should be (nearly) empty in the morning before the first feeding, indicating effective GI transit at the start of the day. Failure of the crop to empty can predispose chicks to overgrowth of bacteria and fungi (Table 41.5).[1,70,71]

Should the ingluvies be delayed in its emptying time, it does not contract properly. As a result, some food content remains in the deepest part of the organ, which in turn sets the stage for opportunistic bacterial and/or yeast overgrowth. Acidity produced by some of the proliferating organisms can further augment development of a so-called "sour crop." These changes lead to continued alterations of the normal crop microflora, setting the stage for further secondary crop dysbiosis, infection and dysfunction.[13,26] This cycle continues to feed into itself, leading to progressive gross overdistension of the crop, marked acidity and sour odor of the content, secondary infections, and inflammatory changes, and to loss of body condition or failure of the chick to develop normally because of dehydration and malnutrition. However, most systemic and physiologic disease processes of young psittacines can result in slow gastrointestinal transit time and delayed

crop emptying,[26] which potentially sets stage for progressive development of "sour crop" and continued delayed emptying.

Crop stasis can be a sign of general illness for many infectious diseases, often associated with secondary and common bacterial and mycotic (*Candida* spp.) infections. An aerobic culture and possibly antimicrobial therapy pending culture results are often common steps in early intervention for affected chicks. Intestinal stasis can also result from other disease processes such as kidney disease, liver disease, or congenital malformations.[1,26,70,71]

Crop atony can result from overfeeding and resultant stretching of the crop musculature. The volume of formula per feeding for most species typically does not exceed 10% of body weight (1 mL formula equals 1 g). Some species can easily accept 15% of body weight per feeding. An easy way to calculate this is to divide the weight of the chick by 10 and administer that volume of formula in milliliters. It is not clear if an optimally developing chick can truly be overfed to the point of stretching the ingluvies, or if those changes are predisposed by subclinical stunting (see section on Stunting).

Treatment typically focuses on maintaining hydration or rehydrating the chick, as ingluvial motility can both be (partially) caused by dehydration and result from it. Hydration of a chick affected by crop stasis should be maintained by either increasing the amount of water content in the hand-rearing formula or by administering fluids parenterally. In case of signs suggestive of dehydration, rehydration may be accomplished by oral or parenteral administration of fluids, allowing for correction of the fluid deficit within 24 to 48 hours. The crop may be evacuated and lavaged, if indicated. Antibacterial and/or antifungal treatments may be indicated. In some cases where the crop is thin walled and markedly distended, a supportive bandage (crop bra) may be used.[13,26] Such bras can easily be prepared using baby socks or Vetwrap (3M Animal Care Products, St. Paul, MN, USA; Figure 41.32). Alternatively, if the crop is severely overstretched, reduction surgery may be necessary.

Gastrointestinal foreign body

Foreign body consumption and impaction usually occur when chicks ingest their bedding. This most often occurs if the chick

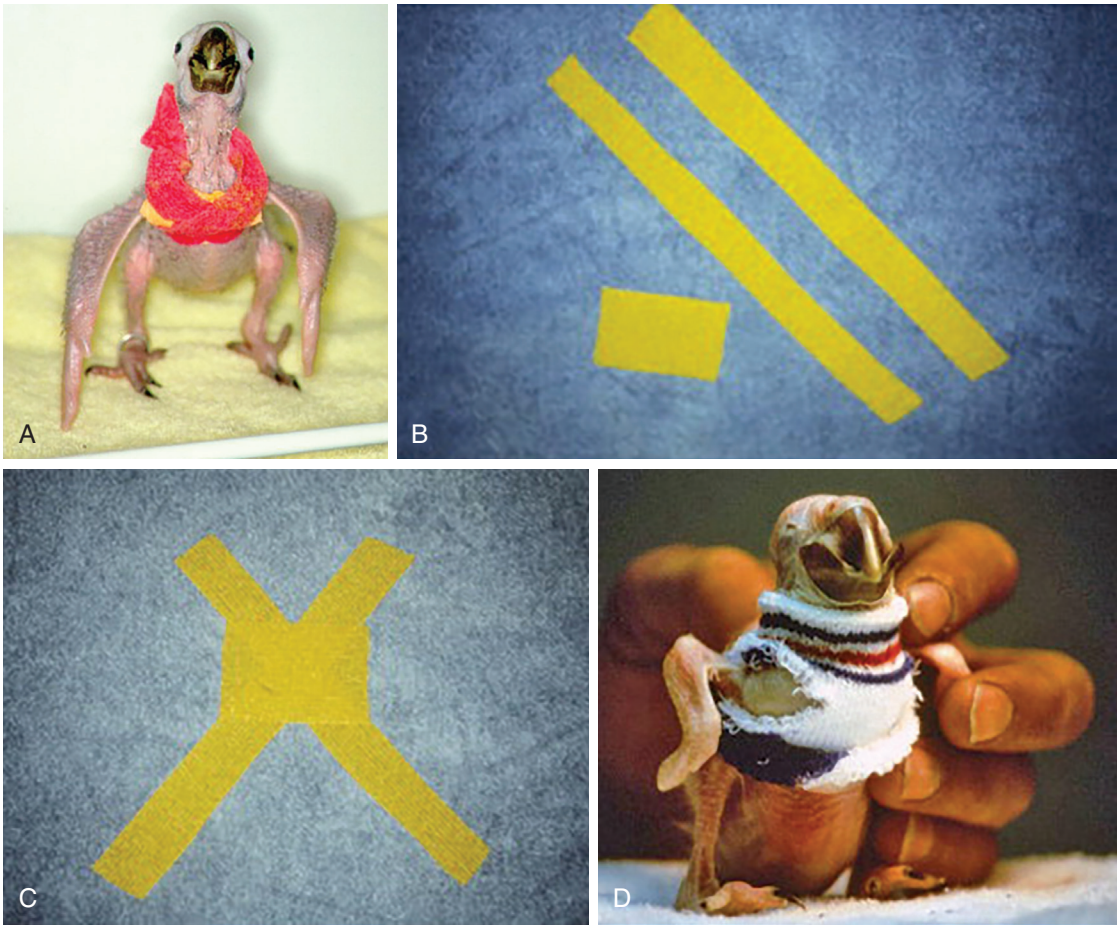


FIGURE 41.32 (A) A crop bra can be used to support an overstretched crop, as demonstrated in this slightly thin and dehydrated macaw chick. This self-made crop bra was prepared from (B) two strips of in-house of VetWrap (3M Animal Care Products, St. Paul, MN, USA) that are subsequently crossed. (C) A horizontal rectangular piece of VetWrap is placed across the area where the strips cross over. (D) As an alternative, crop bras can be made from baby socks. (A–C, From Romagnano A: Psittacine incubation and pediatrics, *Vet Clin North Am Exot Anim Pract* 15(2):163–182, 2012, with permission; D, From Worell AB: Current trends in avian pediatrics, *J Exot Pet Med* 21(2):115–123, 2012, with permission.)

does not get enough food to satiate its hunger or if there is an underlying disease process that causes pica. Bedding and brooder substrate are normally made of tiny particles that can fill the crop and can also proceed further, along the GI tract, causing problems, including more distal obstructions. Obstructions with these smaller substrate particles are more challenging to treat, as the endoscopic procedure can be long and risky, and a crop lavage can be dangerous in a small chick. In the experience of the authors, it is often safer, quicker, and less stressful to perform an ingluviotomy and retrieve the small foreign bodies directly from the crop.[13,14] Chicks may also accidentally swallow feeding tubes that are being used in their hand-feeding (Figure 41.33). Feeding tubes can be disconnected from the syringe and involuntarily swallowed by the chick. Long feeding tubes that are still in the crop can usually be retrieved from the beak, either manually or using a grasping forceps. Shorter tubes tend to reach the proventriculus quickly, but can often be retrieved with the help of an endoscope. Not all foreign bodies require endosurgical or surgical removal, and management details are dependent on clinical status and circumstances of the chick.

Intestinal intussusception

Intussusception may occur and may be confused with cloacal prolapse, although the tissue is usually jejunum and is tubular and red rather than round and pink (Figure 41.34). Intussusception is typically associated with bacterial enteritis and is usually fatal.[9,14] If detected in a timely enough manner, successful treatment requires surgery, with the potential need for resection and anastomosis should there be avascular or necrotic intestine involved.

Regurgitation

Regurgitation may occur as the result of management-associated problems or may be secondary to several primary or secondary medical conditions.[72] As a chick starts to wean, the crop naturally begins to reduce in its volume and cannot hold the previous volumes of food it was previously receiving. Refusal of formula or even regurgitation following hand-feeding may occur if the food is too cold, the chick is starting to wean, the chick does not like the taste of the food, or if the chick becomes excessively stressed or fearful. Excessive begging for hand-feeding formula can be associated with malnutrition, inadequate caloric intake, a hand-feeding formula containing an inadequate (too low) percentage solids, or continuous light cycles with no dark period for sleep. If fed while begging constantly and in the absence of correction of possible underlying antecedents, regurgitation can result. Begging behaviors may also become reinforced, as the result of suboptimal socialization during the weaning period. As an example, young cockatoos may learn to beg for food whenever they see a person, seeking attention even though the bird may be satiated. Feeding these birds large hand-feeding formula volumes can also result in regurgitation. Regurgitation is seen in young chicks secondary to many medical conditions that result in delayed crop-emptying capabilities. These may include bacterial or yeast enteropathies, foreign body ingestion and partial obstruction, renal disease, and polyoma virus infection.



FIGURE 41.33 Lateral radiographic image showing a rubber feeding tube lodged in the proventriculus and ventriculus of a 2-year-old yellow-naped Amazon *(Amazona auropalliata)* with a chronic history of periodic regurgitation and nonspecific signs of ill-thrift. This tube had apparently been present in the bird since prior to weaning and sale as a pet, without causing more significant life-threatening complications. The tube was removed endoscopically through an ingluviotomy incision. (Photo courtesy Brian Speer.)



FIGURE 41.34 Colonic intussusception exteriorized out of the cloaca in a young hand-feeding chick. This was an acute onset and fatal event for the chick. (Photo courtesy Susan Clubb.)

Developmental Problems

Stunting

Stunting is defined as a reversible or permanent process that stops or slows down growth or development (Figure 41.35).[10,72] In many species, stunting is most frequently recognized and described when advanced and more permanent or severe changes are present in chicks.[72] Criteria that can be utilized to define stunting in psittacine chicks are itemized in Box 41.4. Some of these clinical signs are obviously more advanced than others.

Likely, one of the earliest observations should be expected to be subnormal weight gain. Items 2 through 8 in Box 41.4 can be easily viewed as secondary effects, the result of delayed weight gain and stress. Early recognition of problems can facilitate earlier, less involved, and more successful intervention. There are many causes of stunting, but generally these causes will include any factor that interferes with the homeostasis of the chick in its environment.[72]At its onset, stunting most likely starts with variation in adequacy of nutritional and environmental factors, with infectious causes being less likely as a primary causal role.[10] Common nutritional factors include inadequate formulas that do not meet nutritional or caloric requirements of the chicks, feeding inadequate volumes of formula, and inadequate feeding frequencies. A brooder environment with too much or too little heat or inadequate humidity all contribute to stunting. Opportunistic bacterial



FIGURE 41.35 Marked stunting in a blue-and-gold macaw chick *(Ara ararauna)*, 48 days of age, weighing 29 g. At this age, the eyes should be open, the egg tooth should have been shed, contour feathers should be emerging, and the skull should be more symmetrically shaped. Note the extremely thin skin, allowing the crop content to be easily seen, and the dark, dehydrated musculature to be seen beneath it. Normal weight for this species at this age would be 1192 g, with this individual being 2.5% of normal. This bird's progressive stunting and debilitation were obviously not recognized in a timely enough manner to allow for effective medical or nutritional interventions to be implemented.

BOX 41.4 CLINICAL SIGNS OF STUNTING IN PSITTACINE CHICKS

1. Subnormal weight gain for age and species (see Appendix 4A–E)
2. Reduced muscle mass and subcutaneous fat deposits
3. Thin elbows and toes
4. Malproportioned body—large head in proportion to the rest of the body
5. Delayed and abnormal feathering in particular areas and pterylae
6. Chronic, recurrent microbial infections
7. Constant vocalization and begging for food
8. General illness, including sluggish behavior, dehydration, and pale skin color

infections most often develop secondary to chick dehydration and stress and often are identified after the stunting of the chick has already begun. More severely stunted chicks should be expected to manifest the highest number of serious secondary effects. These include crop atony and stasis, bacterial and fungal infections, orthopedic deformities, beak deformities (mandibular prognathism, scissors or lateral maxillary beak deformities), and mortality.[10] Survivors typically have lower adult body weights than chicks that were only mildly stunted or not stunted at all. Chicks with mild-to-moderate degrees of stunting demonstrate an overall lower prevalence of aforementioned secondary effects (Figure 41.36).[10]

There are significant variations in normal developmental parameters available for reference, dependent on the age of initiation of hand-feeding, if the chicks were parent-fed prior

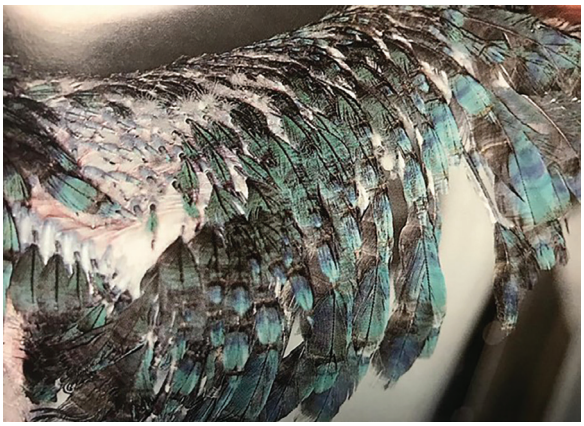


FIGURE 41.36 True stress bars of the primary remiges and dorsal wing coverts in a stunted blue-and-gold macaw chick *(Ara ararauna)*. The absence of segments of the normal development of the barbules produces visible "bars" in the feathers. Unlike feathers where the developing rachis may have been mechanically bent or injured with a palpable bend in it, the rachis of these physiologically induced stress bars have no associated palpable changes in their rachis. Normal plumage development can be seen in Figures 41.11, 41.12, and 41.13. (From Abramson J: Captive breeding and conservation. In Abramson J, Speer BL, Thomsen JB, editors: *The Large Macaws*, Fort Bragg, CA, 1995, Raintree Publications, with permission.)

to being transitioned to hand-rearing, the diet being fed, volume of formula being fed, and the frequency of feedings, and brooder temperature and humidity.[9,10,11,,73] Not all these data are available to allow easy cross comparison between datasets. A comparison of growth between two references is visibly displayed in Figure 41.11. Regardless, these data, when employed as a part of a nursery monitoring system, can allow earlier detection of weight developmental problems. By calculating the percentage of a specific chick with a normal set of data for that species and age, a percentage of "normal" can be determined. What these types of calculations provide is a comparison of growing chicks and their relative rates of growth, rather than merely weights. Almost all chicks, even those that are stunting, still will continue to gain weight, and that can mislead conclusions that it is developing properly. These trends, monitored over time, can allow for a more strategic intervention and reassessment. An example is provided in Boxes 41.2 and 41.2. This type of scenario illustrates that early recognition of subclinical stunting readily allows for simple and often successful corrective maneuvers.

The clinical management of stunting, when recognized, is dependent on the severity of the problem. Initially, if only mild stunting is present, correction of feeding volume/frequency is often sufficient to correct the problem. Chicks may be evaluated for bacterial or yeast opportunistic infections or microbial dysbiosis, but they do not necessarily always require treatment for all isolates recovered. When more significant problems are noted, including but not limited to ingluvial hypomotility, secondary infections, or other more advanced issues, the microbial isolates must more often be individually addressed. Concurrently, efforts to help these chicks move a higher caloric density food in volume per day through their GI tract must be maintained. Supportive measures to achieve this goal typically include an increase in

relative humidity of the brooder, dietary caloric content modifications, and alterations in the utilized feeding volumes and frequencies (Box 41.2).

Constricted toe syndrome

Constricted toe syndrome is believed by some to be a sign of at least regional dehydration in chicks, although a clear causal relationship has not been made. Affected chicks develop a progressive annual constriction typically around digits, leading to progressive edema and swelling of the distal aspect of the digit, and if untreated, avascular necrosis and loss of the digit (Figure 41.37). Common corrective maneuvers are done by placing a damp washcloth under the bedding of the chick so that the moisture and humidity rise upward to the feet and body of the chick, without directly getting the chick wet. This approach is the most natural and minimally intrusive way of correcting particularly regional if not systemic fluid imbalance in the chick, and the most effective way to reverse some early constricted toe syndrome issues, if not prevent them. By regularly massaging these constrictions and digits during each hand-feeding, often some of these cases may be reversed if detected early. More severe cases may require surgical intervention to prevent autoamputation. In such cases, by making a small lateral incision across the constrictions with some lifting of the cut sections of the constrictions can be helpful. Following surgical intervention, keeping the toes clean and providing surface moisture are essential to success. In particularly severe cases, repeated surgical intervention may be required. Amputation of severely affected toes that have become avascular may be required.[1,12]



FIGURE 41.37 (A) Constricted toe of the left fourth digit in a young macaw. Massage of the digit during each hand-feeding was unsuccessful, and this constrictive lesion was surgically released by incisions laterally on each side, followed by protective bandaging and massage afterward. This digit healed and appeared grossly normal when the bird was fully grown. (B) Constricted toe of the left first digit in a younger macaw chick. This lesion was more advanced at the time of its recognition, and although the digit was not lost due to avascular necrosis, it remained deformed. (From Abramson J: Captive breeding and conservation. In Abramson J, Speer BL, Thomsen JB, editors: *The Large Macaws*, Fort Bragg, 1995, Raintree Publications, with permission.)

Orthopedic Developmental Problems

Leg deformities

Appendicular skeletal deformities most often are described in the pelvic limbs, although in generalized cases of metabolic bone disease, several bones of the appendicular skeleton as well as the axial skeleton can be affected. Valgus position and/or rotation are the most frequent issues, leading to the common, sweeping, description of "splay leg" (Figure 41.38).

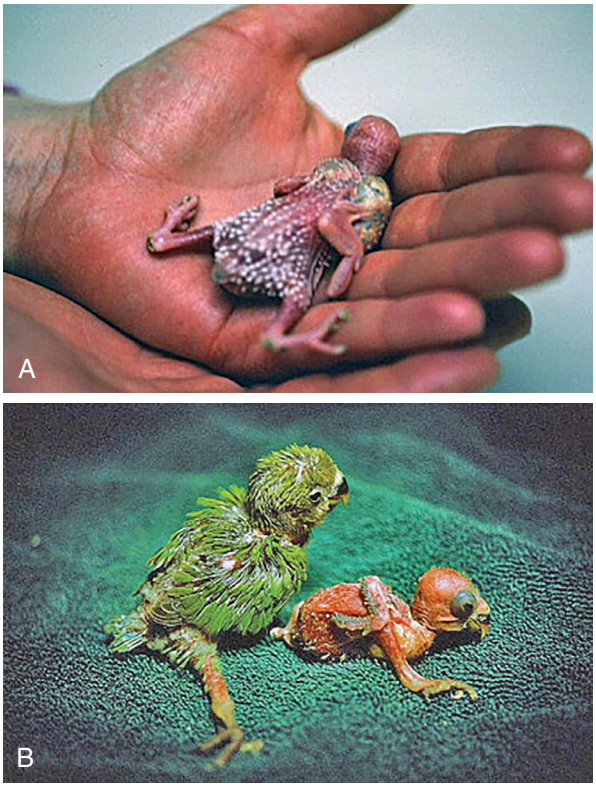


FIGURE 41.38 (A) Splay leg, also referred to as spraddle leg, in a baby budgerigar *(Melopsittacus undulatus)* and (B) angular limb deformity in a clutch of peach-faced lovebirds *(Agapornis roseicollis)*. These limb deformities are suspected to result from inappropriate substrate that prevents the bird from holding its legs in an anatomically correct position, dietary imbalances leading to nutritional secondary hyperparathyroidism, possible genetic influences, and/or other developmental problems during incubation. If caught early, conservative management using hobbles (Figure 41.39) can help to successfully manage these cases. (From Worell AB: Current trends in avian pediatrics, *J Exot Pet Med* 21(2):115–123, with permission.)

Splay leg or valgus deformities are believed to occur predominately in the nest but may also occur in the nursery. Specific deformities under this umbrella descriptor may include folding fractures or rotational deformities of the femur, tibiotarsus, and luxations of the knee. Bone deformities can occur either unilaterally or bilaterally and involve a single bone or several individual bones and their associated joints. Slipped extensor tendon of the intertarsal joint, or laxity of the collateral ligaments of the knee can also be seen, either independent or as a sequela to aforementioned bone deformities. Due to these changes, the chick is not able to stand in a normal bipedal manner, and long-term welfare may be compromised. Inadequate bedding or a parent who sits too tight on the chicks may also cause leg deformities. Similarly, chicks fed diets with improper calcium/phosphorus/vitamin D_3 levels may be predisposed, as are obese chicks that may have difficulty keeping the feet in a proper position.

A variety of different approaches have been used to address these problem(s). The use of selective bandaging, splints, hobbles, and physical therapy are the less invasive treatments that may be best applied prior to full bone growth of the chick (Figure 41.39).[26] However, the results obtained using these methods are often not as satisfactory as desired.[74] Only in mild cases, nonsurgical techniques can give good results, and they are typically achieved in very young chicks that are still growing, allowing more effective guidance of bone growth and alignment. If taking action in an early stage, leg deformities can be corrected using a variety of hobbles, taping the legs to a pad between the legs, U-shaped splints fixed to the lateral side of both legs, or simply keeping the chick packed with



FIGURE 41.39 Baby birds with (A) tape splint, (B) "snowshoe" made from tongue depressor, and (C) and (D) "snowboard" for treatment of splay leg. This type of hobbling will help to properly position the legs, whereby care must be taken that the toes face forward. For the best results, hobbling of either kind must be changed frequently to prevent soiling and ensure that the hobble is adapted to the growth of the chick. (A and B, From Worell AB: Current trends in avian pediatrics, *J Exot Pet Med* 21(2):115–123, 2012, with permission; C and D, From Romagnano A: Psittacine incubation and pediatrics, *Vet Clin North Am Exot Anim Pract* 15(2):163–182, with permission.)

paper towels or other materials that help to move the legs into a normal position. Careful monitoring of the chick during these corrective maneuvers is required, as changes in strategy and details should be expected as time progresses during these treatments.[13,75] Folding fractures of the long bones of the appendicular skeleton tend to be observed primarily in nest-raised chicks, in which their parents are receiving a diet deficient in calcium, such as a diet based primarily on sunflower seeds. Clinical rickets results (nutritional secondary hyperparathyroidism) and may be only first detected by noting the development of these skeletal deformities. If discovered early, affected chicks can usually be digitally straightened and stabilized with a split or supportive bandaging. These supports must be changed often, as with continued growth of the chick, they can become excessively tight, quickly.

Surgical approaches, including dome, transverse, oblique, or wedge osteotomy, can guarantee better results but are much more invasive.[76–78] These techniques are typically employed once the bird has attained full skeletal growth and maturity. A closed predrilled technique gives a good alternative to manage this problem in young birds and potentially also in adult patients. After the ostectomy, or osteotomy, depending on the case, a lightweight, customized cast made of aluminum can be applied, but this is limited to the smallest species. Larger birds will be treated with the application of an external fixator (Figure 41.40).[79] Tibiotarsal derotational osteotomies are typically the most common procedures. A step-by-step description for a derotational osteotomy of a valgus tibiotarsal rotation is provided in Box 41.5.



FIGURE 41.40 Postoperative ventrodorsal radiographic image of a 16-week-old domestic turkey *(Meleagris gallopavo domesticus)* following a derotational osteotomy procedure, where the intramedullary pin was preplaced as described earlier, the fracture created in a functionally closed manner, and then the pin advanced. There originally was a valgus 90-degree rotational deformity of the tibiotarsus. An extraskeletal type I tie-in fixator was then applied. Note that the intermedullary pin is less-than-optimally inserted in the distal tibiotarsus. This young turkey was functionally healed and the external skeletal fixator (ESF) type I tie-in fixator was partially deconstructed starting at week 4 postoperatively and completely removed at week 6. There was an excellent return to normal form and function achieved. (Photo courtesy Brian Speer.)

Hyperextension or luxation of the knee

In some young chicks the knee can become hyperextended or even luxated (Figure 41.41). This is most often seen as a unilateral problem; however, bilateral presentations can occur. Typically, the lower leg and foot distal to the knee are extended cranially while the intertarsal joint is hyperextended. Ligament and tendon contraction can cause the joint to become fixed in its abnormal position, rendering a poorer prognosis for more chronic luxations. An additional compounding variable is that additional bony injury can easily be induced during the manipulation process in attempts to reduce these luxations due to the poor ossification of the associated appendicular skeleton.[26] Application of a type I transarticular extraskeletal fixator can provide stability for some of these luxations in older chicks with adequately supportive bone density.[7]

Digit deformities

Crooked toes may occur due to luxation or subluxation of the interphalangeal joints, primarily of the third and second digits, although the first and fourth digits can be affected as well. (Sub)luxation may occur following placement of the chick on a hard or smooth surface, resulting in the nails becoming a pressure point and causing the last phalanx to luxate. If diagnosed early the nail can be trimmed, the toe stabilized by bandaging methods, and the chick placed in deeper bedding.[14,26] Anteroflexion of the fourth digit is another digit abnormality seen. If discovered early, this growth deformity is easily corrected by taping the toe in a normal position. In young birds, the foot can be bandaged with the toe pointing backward in the normal position for several days. Older chicks may need prolonged bandaging.[14]

Deformities of the axial skeleton

Deformities of the thoracic or cervical spine can occur in chicks resulting in scoliosis and other vertebral malformations of the spine. Rickets (nutritional hyperparathyroidism) is a common hypothesized cause, as well as trauma. In early detected cases, aggressive pain management, paired with some neck supportive bandaging may help select cases.[14,26] A spastic cervical muscular contraction is also noted in some psittacine chicks, which at least initially is not accompanied by vertebral malformation. A common loose avicultural descriptor for this condition is "wry neck." Conservative management approaches are common, and although some birds can be returned to an almost normal situation, many survive with moderate to sometimes more severe neck deformities.

Beak deformities

Malformations of the beaks are believed to most often occur in chicks which are suboptimal in development. Lateral maxillary beak deviations (scissors beak) deformities and mandibular prognathism are the most encountered in psittacine chicks. When detected early, there are several corrective options that can be implemented. Beak deformities and their corrective maneuvers are discussed in-depth in Chapter 31.

BOX 41.5 EXAMINATION FINDINGS SUPPORTIVE OF THE PRESENCE OF A VALGUS TIBIOTARSAL ROTATIONAL DEFORMITY AND PROCEDURAL DETAILS

The procedural indication can be verified typically at the examination. With the bird in dorsal recumbency, grasp the tip of the third digit, and flex the intertarsal joint completely. In the absence of tibiotarsal rotation, the toenail of that third digit should touch and align with the cnemial crest of the proximal tibiotarsus. In the presence of a valgus tibiotarsal rotational deformity, it will be positioned laterally to that structure. A similar examination of the opposite leg can be performed for comparison.

1. Drive an intramuscular (IM) pin normograde into the tibiotarsus, down to just proximal to the site where the fracture is intended to be induced. Typically, this will be done at the distal one-third location, where there is less muscle/tendon in the way, and the tibiotarsus can be cleanly palpated. The size of the pin needs to be smaller than the generic two-thirds of the inner diameter of the bone, as enough room is needed for the lateral threaded pins to get transcortical purchase around that IM pin.
2. Using a larger IM nonthreaded pin, that is about one-half to two-thirds the diameter of that bone, manually drill from the lateral cortex through the medullary cavity until the tip can be palpated penetrating the medial cortex. Withdraw, and repeat another one to two times at the same location, but different angles. The goal is to functionally weaken the cortex so that pressure can be applied manually and the bone be "cracked", inducing a functional fracture. Done well, there is still considerable periosteum that remains intact.
3. Without displacing the induced fracture, advance the prestarted IM pin down to the metaphysis/epiphysis of the tibiotarsus. This will provide good rigid linear stability. Measure to ensure that the pin is seated at appropriate length into the metaphysis/epiphysis of the bone.
4. The proximal portion of the pin that is exiting is bent 180 degrees and used to provide the rigidity for an IM tie-in. If possible, make sure that the pin is long enough to allow an adequate amount to extend down the length of that bone after bending it.
5. Clear plastic tubing, approximately the same inner diameter of the transcortical distance, can be placed over this bent pin, and provide the framework which will serve to hold the threaded pins that are placed, at least two above and two below the induced fracture.
6. The threaded pins are placed so that their points can be palpated just emerging from the medial cortex, without perforating the skin.
7. Cut the cross pins to allow them to be placed up and into predrilled holes in the plastic tubing and out its lateral aspect.
8. Once the pins are all set, the entire apparatus can be filled with methyl methacrylate or similar product and allowed to solidify. Spray a small amount of isopropyl alcohol on the apparatus, if needed, to reduce the heat that is being generated and transferred into the bone should an exothermic reaction be present.
9. Alternatively, in smaller species, the IM tie-in can be incorporated into a tape bandage or splint, without the use of threaded pins.
10. Functional healing will usually take about 3 weeks, after which the lateral pins can selectively be removed with the IM pin allowed to remain another week or two, depending on how the bird is moving and doing overall.



FIGURE 41.41 Luxation of the right knee in a young cockatoo chick. The proximal tibiotarsus is displaced cranially, and the distal epiphysis of the femur is caudal to the tibiotarsus. Some birds may be able to have these luxations reduced under general anesthesia by carefully stretching the associated musculature and manipulating the proximal tibiotarsus back into its proper alignment on the distal femur. (Photo courtesy Susan Clubb.)

Choanal Atresia

Choanal atresia is described most often in Grey parrots but has also been documented in the umbrella cockatoo.[80] The specific detailed anatomic deformities behind this developmental problem have not been definitively clarified yet. Clinical signs are increased mucus accumulation and possible infection in the nares and sinuses. Blunted choanal papilla may be detected on oral examination. Diagnosis is aided by endoscopic examination of the choana, paired with an inability of fluid flushed through the nares to be passed freely into the choana (see also Chapter 31). CT imaging may be helpful in localizing the specific location of the obstruction. Atresia can be treated by creating an opening in the choana through the nares with an intramedullary pin. A red rubber feeding tube is then threaded through the nares, out the choana, and back behind the head of the bird and is left in place for 2 to 3 weeks. This procedure is usually performed in stages, beginning with a small tube first and following with a larger tube (see also Chapters 29 and 31).[80]

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CHAPTER 42

Raptor Incubation and Pediatrics

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INCUBATION AND HATCHING PROBLEMS

Incubation

A full description of avian incubation is beyond the scope of this chapter. For this, the reader is referred to Chapters 39 and 40. Owing to the high value of many raptor species, incubation is carried out to high standards in large commercial breeding projects, particularly those breeding gyrfalcons and their hybrids. Incubation parameters (time-to-pip and Kw constants) for a range of raptor species are listed in Table 42.1 (courtesy David Le Mesurier, unpublished data). This information has been extracted from the Avian Information Management System (AIMS) (www.visualaims.com; courtesy David Le Mesurier). AIMS is an incubation management system that will calculate the humidity needed to set incubators in order to achieve the correct percentage of weight loss during incubation. The desired weight loss to pip is 15% from the fresh weight. The Kw coefficient is used to calculate the fresh weight of an egg using the egg length and breadth (in mm). Note that Kw in Table 42.1 is Kw $\times 10^7$

$$\text{Fresh weight} = \text{Kw} \times (\text{egg length [mm]} \times (\text{egg breadth})^2)$$

Eggs from large falcon species are normally incubated at 37.3°C, although some breeders will incubate at 37.4°C for smaller species such as merlins (*Falco columbarius*) (D. Le Mesurier, personal communication, 2022). Different temperatures will be appropriate for contact incubators and still air machines; for example, in a contact incubator 38°C is appropriate, measured at the top of the egg. The humidity used depends on weight loss, so it is important to calculate the weight loss and adjust the humidity accordingly. Programs such as AIMS calculate the humidity eggs need to be moved to during the incubation process after they are weighed. In the absence of a fresh egg weight, humidity can be started at 45%.

Suboptimal temperatures and humidity can directly cause incubation failure. Figure 42.1 summarizes the main causes of hatching failure. Dataloggers are readily available and represent an inexpensive and versatile tool that can be used by vets and aviculturists to monitor and manage both incubators and the environment within the incubation room.[1] Small changes in temperature have profound effects on incubation. For example, a small difference of 0.5°C can result in a 12-hour delay in hatching, while a temperature less than 35°C will cause deformed toes and feet in chickens. The effects of low temperature are most severe in the first third of incubation.

Aviculturists are used to managing weight loss in eggs because there is a higher probability of hatching if the loss is controlled between 12% and 17% of the initial egg mass. Chicks that lose too much water have a decreased probability of hatching, and if they do hatch, they will be dehydrated.[2] Chicks that lose too little water will also have problems hatching and can end up puffy and edematous.

When hatchability falls below expected percentages, the incubation facility and its management should be investigated. Well-run breeding facilities will have records, not only incubation data for each individual egg but also records of incubators and the incubation room. The key environmental conditions to monitor are temperature and relative humidity, both within incubators and within the incubation room. Inadequate control of humidity in incubation rooms can result in knock-on problems to the eggs in an incubator. Figure 42.2 shows a fragment of data from an incubation room. While the temperature of the incubation room was stable, the relative humidity was too high, which had knock-on effects on the weight loss and hatchability of the falcon eggs within the incubators. The incubation room was in a facility in Wales during a period of very wet weather, and the problem was solved by installing dehumidifiers.

Dataloggers can be used to map temperature and humidity profiles in incubators, and it is good practice to map incubators before starting to incubate valuable eggs (Figure 42.3). Figure 42.4 shows the danger spots in a commonly used incubator. Temperatures in the corners and in front of the fan ventilator were greater than 1°C higher than in the "safe" zone of the incubator. In this case, the solution was simply to avoid putting eggs in the hot zones.

Hatching Problems

Most experienced raptor breeders deal with hatching problems without seeking veterinary intervention. Heck and Konkel[3] provide useful practical tips on when to intervene and when to be patient when chicks appear to be delayed in hatching. The "pip," defined as the first crack in the shell made by the embryo, is the first stage in the hatching process, and approximately 24 to 48 hours before, candling will reveal that the air cell expands and "draws down" on one side of the egg (see also Chapter 40, where the normal hatching process is discussed). Occasionally, intervention will be required to save chicks that are struggling to hatch, which—if done timely and by an experienced aviculturist—will have a

TABLE 42.1

Incubation Parameters (Time-to-Pip and Kw Constants) for a Range of Raptor Species

Common Name	Kw × 10^{-7}	Kw Std × 10^{-7}	*n* Kw	Pip Time (Days)	Pip Std	*n* Pip
Eurasian goshawk *(Accipiter gentilis)*	5554	58.00294666	36	32.72	0.33	5
Black sparrowhawk *(Accipiter melanoleucus)*	5432	171.3187362	8	31.05	0.93	2
Red-tailed hawk *(Buteo jamaicensis)*	5563	19	2	32.17	1.11	8
Bald eagle *(Haliaeetus leucocephalus)*	5430	60.85202427	17	33.8	0.2	2
African fish eagle *(Haliaeetus vocifer)*	5480	93.20005365	4	32.11	0.35	2
Black kite *(Milvus migrans)*	5489	135.7746294	40	29.1	1.14	19
Harris' hawk *(Parabuteo unicinctus)*	5506	70.49900265	16	30.74	0.87	18
American black vulture *(Coragyps atratus)*	5466	90.80232651	16	37.7	1	2
Lanner falcon *(Falco biarmicus)*	5509	83.73157897	153	30.69	1.19	17
Saker falcon *(Falco cherrug)*	5513	67.86917114	305	31.47	0.89	94
Red-headed falcon *(Falco chicquera)*	5431	98.8198922	16	29.12	2.16	4
Merlin *(Falco columbarius)*	5480	81.63705883	8	31.04	1.3	3
Lugger falcon *(Falco jugger)*	5504	117.2949047	13	32.12	1.21	4
Barbary falcon *(Falco peregrinus pelegrinoides)*	5460	75.41230225	435	30.34	0.97	29
Peregrine falcon *(Falco peregrinus)*	5497	76.27669179	2309	30.78	0.97	123
Gyrfalcon *(Falco rusticolus)*	5492	67.59126932	1745	31.65	0.73	632
Northern eagle owl *(Bubo bubo sibiricus)*	5433	65.5762385	12	28.32	1.37	9
Great horned owl *(Bubo virginianus)*	5571	154.0072148	3	29.65	0.53	2

The pip time is the time to pip from the day the egg is set in the incubator. *n* pip is the sample size for pip times for each species. *n* Kw is the sample size for the Kw species. Std is the standard deviation for the dataset. The Kw coefficient is used to calculate the fresh weight of an egg from the egg length and breadth (in mm). Note that Kw in the table is Kw × 10^7.
This information has been extracted from the Avian Information Management System (AIMS) (www.visualaims.com). Courtesy David Le Mesurier. Kw data is copyright Avian Management Services Ltd.

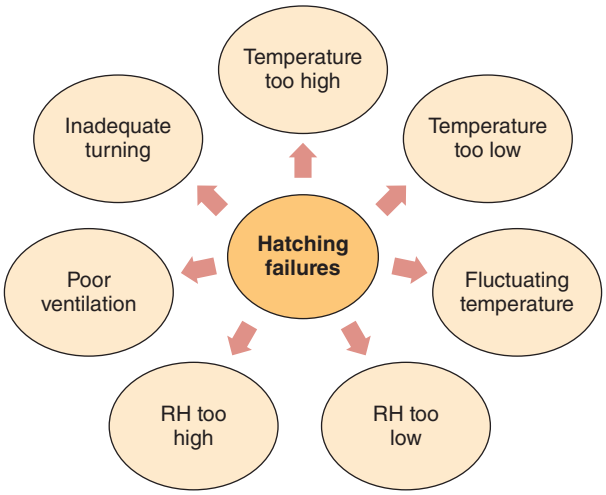


FIGURE 42.1 Causes of hatching failure. *RH,* Relative humidity.

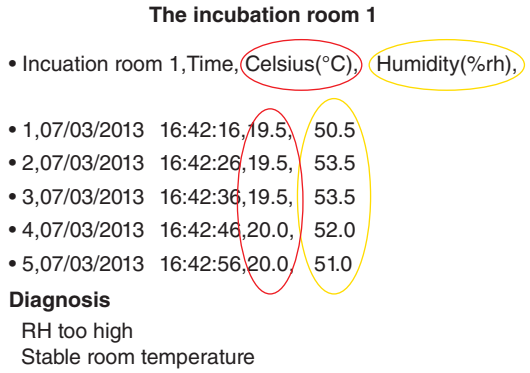


FIGURE 42.2 Fragment of data from an incubation room. *RH,* Relative humidity.

high chance of being successful. Most hatching problems are incubation related and are often linked to inadequate or excessive weight loss during incubation (see also Chapter 40). Generally, pipped eggs are left for 60 hours undisturbed before intervention.[3] The Buddy digital egg monitor (Buddy; Avian Biotech International, Tallahassee, FL, USA) is useful for checking problem eggs and to monitor heart rate. The embryo's heart rate in falcons and Eurasian goshawks *(Accipiter gentilis)* varies between 240 and 260 bpm, with an increase observed before hatching.[4] Extreme increases or decreases in heart rate can be useful indicators that an embryo is stressed, necessitating intervention. Assistance is



FIGURE 42.3 Datalogger within a dummy egg. (Photo courtesy Tom Bailey.)



FIGURE 42.4 The danger "hot spots" that are present in a commonly used incubator. (Photo courtesy Tom Bailey.)

also indicated if an embryo stops for an extended period after turning a quarter to a halfway around, or when it turns a bit but then reverses direction and returns to the pip site. Heck and Konkel[3] consider that these turning issues are often caused by the chick being too wet or having an unretracted yolk sac. Intervention may also be required if the site of the pip is too low down the egg, as this suggests poor or malpositioning of the embryo. At other times, for example, if the head is positioned at the small end of the egg, the chick may not be able to pip, which may make it hard to accurately identify where to open the egg without the use of diagnostic tools such as radiography. Fortunately, several zoological collections have the luxury of being able to radiograph an egg on site, which can lead to successful assisted hatching of malpositioned chicks by pinpointing where the head and bill are located (Figure 42.5).[5] In a review of 209 egg postmortems carried out by the author (unpublished) on large falcon species (gyrfalcons [*Falco rusticolus*], saker falcons [*Falco cherrug*], peregrine falcons [*Falco peregrines*], and peregrine-hybrid falcons) over two breeding seasons from 2012 to 2013, the commonest malpositions were (1) head in the small end of the egg (37%), (2) head above the right wing (21%), (3) head between legs (16%), (4) rotational alignment/chick positioned crossways in egg (16%), (5) head under the left wing/head twisted 180 degrees (5%), and (6) head away from the air cell because of spherical egg (5%).

If a hatcher is opened unnecessarily for too long, too frequently, or if the hatcher humidity is too low, excessive drying of the membranes may occur. Dry membranes around the pip site can conspire to prevent a chick from cutting out, causing a "stuck" chick. Generally, once an embryo has made a quarter turn after pipping, it is safe to remove it. Forceps are used to remove the shell, and a small sterile paint brush is used to "paint" saline onto the membrane to assess vascularization and check the position of the chick (Figure 42.6).

Sterile cotton buds are useful aids for manipulating delicate membranes and embryos. If blood vessels are functioning, the membranes can be moistened with saline, following which the hole can be covered with some tape, and the egg returned to the incubator before rechecking every 30 to 60 minutes.

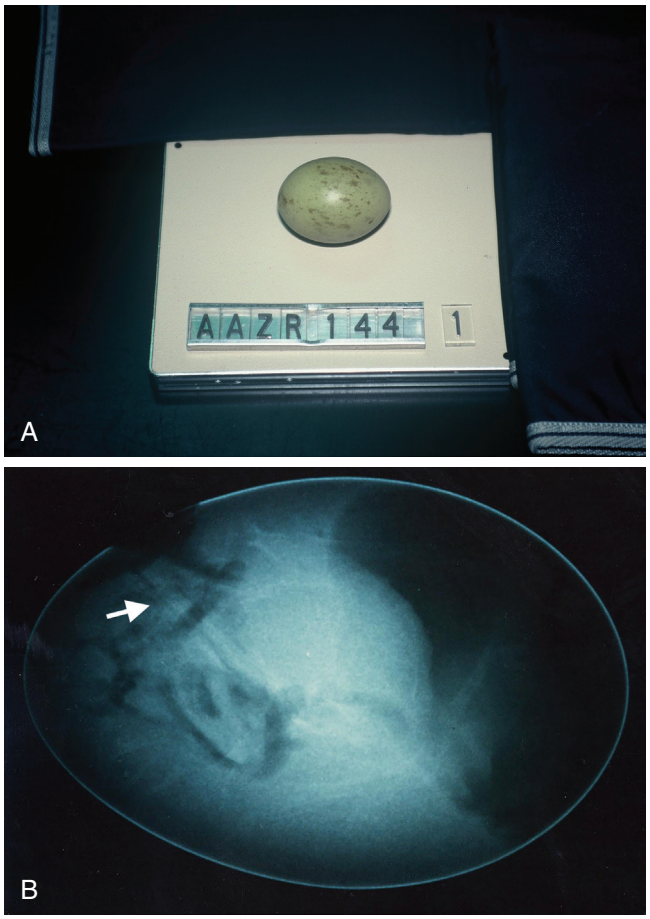


FIGURE 42.5 (A) A bustard egg is prepared for radiography. (B) Radiograph of malpositioned kori bustard *(Ardeotis kori)* with the head in the small end. The *arrow* identifies the bill. (Photo courtesy Tom Bailey.)



FIGURE 42.6 A small sterile paint brush is used to "paint" saline onto the membrane to assess vascularization and check the position of the chick. (Photo courtesy Tom Bailey.)

Chicks with partially or completely unretracted yolk sacs have a more guarded prognosis, even with veterinary assistance. Care must be taken to avoid severing any of the large blood vessels that supply the yolk sac as this may cause significant, life-threatening hemorrhage. Fine suture material should



FIGURE 42.7 Surgical closure of a small umbilical protrusion of a gyrfalcon *(Falco rusticolus)* hatchling. (Photo courtesy Tom Bailey.)

be used to tie off the yolk sac completely or to reduce the size of the yolk sac and surgically reinsert the stump into the abdomen. Generally, small protrusions or partially unretracted yolk sacs can be surgically reduced with a fair prognosis, provided that proper hygiene is practiced. The procedure is to ligate the yolk sac close to the body cavity and amputate the sac (Figure 42.7). Depending on its size, the umbilicus may need closure with one or two sutures. Correction of large unretracted yolk sacs usually carries a poor prognosis, especially if caused by interfering too early in the hatching process. Heck and Konkel[3] provide more details on dealing with hatching issues.

Chicks that have had prolonged hatching or interventions are often dehydrated and will benefit from supportive fluids (oral, subcutaneous, intravenous, or intraosseous) and from a precautionary course of antibiotics, especially if yolk sac issues are present. It is sensible practice to swab the navel of all newly hatched chicks with 1% iodine or antiseptic ointment (e.g., F10 barrier cream) to reduce the risk of infection and omphalitis.

RAPTOR PEDIATRICS

Raptor (Falconiformes and Strigiformes) chicks experience a rapid growth rate relative to their body size in the first 20 days after hatching.[6] For example, great horned owls *(Bubo virginianus)* gain 18% of their body weight every 2 days during the first 20 days following hatching.[7] The normal maturation stages of young birds of prey have been summarized by Sherrod[8] and Fox.[9] Clum et al.[10] present a series of chronological pictures of young peregrine falcons from hatch to 40 days, showing key features in the physical development of this species. Holland[11] provides useful growth and rearing data for many unusual raptor species including eagles, vultures, owls, and small falcon species. Wellfair[6] reviewed records at the International Centre for Birds of Prey (Newent, UK) and published a comparative analysis of early growth and rearing protocols in 48 species of hand-reared raptors, and this resource is available online (https://www.handrearingresource-center.com/global-partners/). Appendix 4F presents daily weights, growth rates, and food consumption for five species of raptor including lanner falcon *(Falco biarmicus)*, pygmy falcon *(Polihierax semitorquatus)*, tawny eagle *(Aquila rapax)*, merlin, and saker falcon.

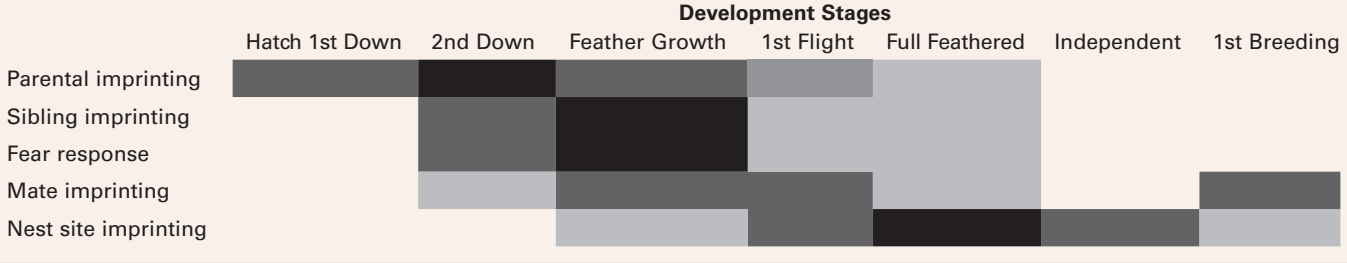
RAPTOR BEHAVIOR AND IMPRINTING

Many of the problems that arise in captive-bred birds of prey are "invisible" psychological or behavioral issues. An understanding of behavioral development is therefore important, especially since the success of a captive-bred falcon in its breeding or falconry role is—to a large extent—a consequence of the behavioral "molding" that occurs during the first 30 to 40 days of life.

Raptor behavior results from the interaction of inherited (innate), imprinted, and learned components. One piece of terminology that is important to grasp is the word "imprinting." Fox[9] defines imprinting as "a distinct genetically programmed learning mechanism in which there is a permanent attachment, during a specific sensitive period, of an innate behavior pattern to specific objects which thereafter become important elicitors of that behavior pattern." In raptors, five main types of imprinting can be identified, including (1) parent, (2) sibling, (3) fear response, (4) sexual partner, and (5) nest site and type. The sensitive times for imprinting each of these behaviors overlap chronologically (Box 42.1).

Birds of prey can be hand-reared individually by humans, group-reared with other raptors in a "crèche," reared by parent birds in an aviary secluded from human contact, or reared by a parent bird in an imprint pen with human contact, or by a combination of these methods. In the larger breeding projects, chicks are hand-reared for the first 6 to 8 days and

BOX 42.1 CHRONOLOGY OF SENSITIVE TIMES FOR IMPRINTING OF BEHAVIORS IN FALCONS



Shades represent the extent to which chicks in a particular developmental stage are susceptible to this type of imprinting. As imprinting is a gradual process, it expands across different developmental stages. The darker the color, the more susceptible the chicks are (Note: no shading means imprinting is complete). As an example, for the first row (parental imprinting), chicks are most susceptible to parental imprinting upon development of the second down, while susceptibility to imprinting on the siblings or nest is highest at later stages of development.

then placed in an aviary to be reared by surrogate parent birds. Falcon breeders with a good understanding of imprinting can hand rear a "human" imprinted raptor suitable for artificial insemination projects or—at the other end of the imprinting spectrum—a parent-reared falcon that knows it is a falcon and is imprinted on both its own species and an aviary nest site. This results in a bird that is well adapted to pair breeding in captivity. Depending on the level of imprinting and socializing, tame raptors can be produced for falconry. Such birds are well adjusted to the sights and sounds of human life (vehicles, dogs, people, etc.), which allows for greater control of the birds in the field without having to dramatically reduce the birds weight to get flying and hunting responses for falconry purposes. Unfortunately, malimprinted birds can be produced by errors in rearing, resulting in a screaming, aggressive, and "poorly mannered" bird that is unsuitable for falconry or breeding and that—in case of larger raptor species—may be dangerous to people. Readers with further interest in the development of raptor behavior are recommended to read Development and Behavior by Fox,[9] in which these developments of normal and abnormal behavior are thoroughly discussed.

GROWTH AND DEVELOPMENT

Immediate Posthatching Period

Newly hatched raptor chicks are covered with fine natal down. This density of down varies according to whether the species is from a colder or warmer climate. Most raptors have white down, but some species, such as Harris' hawks *(Parabuteo unicinctus)*, hatch with buff-colored down. The *musculus complexus* or occipital muscles at the back of the head used for pipping the shell are very pronounced and newly hatched chicks have bulging eyes (Figure 42.8). In the posthatch period, the chick struggles to lift its head and will spend most of the time resting with closed eyes. Although appearing feeble, a healthy chick will respond to the soft calls of its mother and raise its head and neck upwards and gape for food.

Days 1 to 7 after Hatching

The hatching muscle disappears within the first 2 days after hatching. During the first week of life, raptor chicks grow rapidly. The yolk sac is used up after 3 or 4 days.[9] Diurnal raptors may hatch with their eyes open or closed, but by day 2, the eyes start to open and become fully open by day 6.[12] By day 3, chicks can sit on their intertarsal joints and groom (Figure 42.9). As the chicks become more mobile, they start to feed themselves. In the wild, the chick moves toward the edge of the nest to defecate. Raptor chicks respond to any pointed object when being fed, and it is supposed that the parents bill provides the visual stimulus for the initial attempts of chicks to take the food.[13]

Days 7 to 14 Days after Hatching

After 7 to 8 days or 12 to 14 days for small falcon species and larger raptors, respectively, a second thicker coat of down grows through the natal down. This denser down enables the chick to control its own temperature and survive more easily with decreasing brooding times required.[9] In peregrine falcons, primary and tail feather sheaths start to emerge at this stage and will continue to grow at a rate of 2 to 3 mm per day.[10]

Days 14 to 21 after Hatching

By day 17, the contour feathers start to grow in peregrine falcons, which displace the secondary down, giving the chicks a fluffier appearance (Figure 42.10). This is a highly sensitive period when the chick is imprinting to its parents and siblings as well as developing appropriate fear responses.[9]

Days 21 to 28 after Hatching

By 18 days for smaller raptors or 22 to 26 days for larger species, the chicks have reached and may exceed adult body weight. Chicks can stand and walk but will still spend much time resting on their tarsi. The growing contour feathers start to become visible as they emerge through the down (Figure 42.11).



FIGURE 42.8 Newly hatched lugger falcon *(Falco jugger)*. Note the egg tooth, the prominent occipital muscles, and closed eyes. (Photo courtesy Holly Cale.)



FIGURE 42.9 Lugger falcon *(Falco jugger)* chicks between 2 and 5 days of age. Note the eye of the older chick *(middle)* is more round than slit-like. There are no signs of second down emerging anywhere on the bodies of these chicks. (Photo courtesy Holly Cale.)



FIGURE 42.10 The greater primary coverts have broken their sheaths and are visible in this image of 19-day-old lugger falcons *(Falco jugger)*. It is possible to see that some tail feathers have emerged from their sheaths as well. (Photo courtesy Holly Cale.)



FIGURE 42.11 Body contour feathers are visible through the down of these 27-day-old lugger falcons *(Falco jugger)*. (Photo courtesy Holly Cale.)

Days 28 to 35 after Hatching

The chicks are losing their down, and by day 30, peregrine falcon chicks appear to be half downy and half feathered (Figure 42.12). At this stage, chicks are generally attentive to their surroundings. The chicks will still beg and be fed by their parents, but they can feed themselves and will engage in squabbling with siblings and parents over food. The chicks are vocal and engage in robust wing flapping on the nest ledge.

At 4 weeks of age in small raptors or at 5 to 6 weeks of age in larger species, the chicks start to fly. At this stage, the feathers may have soft shafts inside the feather sockets, which in falconry parlance is referred to as not yet being "hard penned." Additionally, the skeleton is not yet mature, and it is best to keep young birds well fed at this stage to avoid any unnecessary handling as this may inadvertently damage their feathers and ossification centers.



FIGURE 42.12 By 31 days these lugger chicks *(Falco jugger)* appear to be about half downy and half feathered. (Photo courtesy Holly Cale.)

Days 35 to 42 after Hatching

At 5 weeks, peregrine falcon chicks are nearly fully feathered except for patches of down around the legs and wings and on the top of their heads. As a result, they are capable of weak flight. Full flying ability is not generally gained until the feather quills are strong and the bird is hard penned. Fledging periods for small falcons such as sparrowhawks *(Accipiter nisus)* and small falcons are short (21–25 days), but for large birds of prey, these are longer and may take up to 95 days in tropical eagles like Verreaux's eagle *(Aquila verreauxii)*.[13]

BONE GROWTH

There has been little published information on the process of ossification in raptors. Fowler[14] reviewed the literature on other avian species and published some information on the ossification of long bones in barn owls *(Tyto alba)*. This investigation confirmed that radiographically, long bone ossification in barn owls is comparable to that in chickens *(Gallus gallus domesticus)*, pigeons *(Columba livia)*, and house sparrows *(Passer domesticus)*.

All wing bones and the femur of barn owls develop without epiphyseal centers of ossification, while the distal tibiotarsus and proximal tarsometatarsus have epiphyseal centers similar to those described for chickens.[14] These epiphyseal centers resemble mammalian growth plates but are in fact the tarsal bones that have formed a center of ossified bones.[15] Harcourt-Brown[15] observed that skeletal problems in Harris' hawks can be caused by issues of metatarsal bone fusion. Damage or trauma to a single metatarsal bone is enough to slow or stop the growth on one side of the limb, resulting in deviations of the tarsometatarsus (Figure 42.13). Harcourt-Brown[15] reported seeing several Harris' hawks with deviations of the metatarsi at around 5 weeks of age.

Fowler[14] reported that in the barn owl, the humerus, radius, and ulna are completely ossified by 60 days, while the femur matures by day 50. The epiphyseal center of the distal end of the tibiotarsus and of the proximal end of the tarsometatarsus both appear on radiographs at less than 20 days,



FIGURE 42.13 Radiograph of the tarsometatarsus of a 5-week-old Harris' hawk *(Parabuteo unicinctus)* showing deviation of the tarsometatarsus, cranial caudal view. (Photo courtesy Nigel Harcourt-Brown.)



FIGURE 42.14 Lung consolidation in a juvenile falcon chick that had a *Cryptosporidium* infection of the alimentary tract and fungal pneumonia (aspergillosis). The image also shows how the tarsal bones that have formed centers of ossified bones that resemble mammalian growth plates in the distal tibiotarsus and proximal tarsometatarsus. There also appears to be a curvature of the right distal tibiotarsus. The image also clearly shows how the distal humerus and proximal radius and ulna develop without epiphyseal centers in raptors. (Photo courtesy Tom Bailey.)

and ossification is complete by day 50 (Figure 42.14).[14] Hawks are relatively slow growing compared to falcons, so there are likely to be some important differences, and a valuable area for future research would be a radiographic study of the normal ossification pattern of falcons and hawks.

NUTRITION AND DIETS

Diet quality is paramount for rearing healthy raptors successfully. Deficiencies in the quantity and quality of diet during growth can result in slowing or stunting of growth or more serious problems such as metabolic bone disease (MBD). In countries and locations where the supply of disease-free prey items cannot be assured, large breeding projects breed their own quail and rats to eliminate risks of introducing infectious diseases. A wide range of infectious agents can be contracted from feeding poorly sourced food where biosecurity or hygiene has been compromised. Diseases associated with poor-quality food include trichomoniasis, salmonellosis, colibacillosis, mycobacteriosis, campylobacteriosis, and chlamydiosis. In addition, infections with paramyxovirus infection, orthomyxovirus (influenza), and herpesvirus, as well as enteric parasites, have been reported.[16] Between 2002 and 2011, paramyxovirus infection, avian influenza, and cryptosporidiosis were diagnosed in falcon chicks by the author in a number of falcon breeding facilities in Dubai that were associated with biosecurity breakdowns in quail units from where the falcon rearing food had been sourced.

Externally sourced food should be purchased from reputable animal food wholesalers. Suppliers should have preventive medicine programs including vaccination and antiparasitic treatments as well as disease-screening protocols in place for the breeding stock and offspring. The method of killing the prey should also be known. This information allows for the prevention of food intoxications or contamination by medicines or euthanasia drugs. Nutritional analysis of the food and supplements fed to the food animals should be available for scrutiny. Cold storage facilities should have their temperatures monitored by sensors and alarms.[1] Attention should furthermore be paid to food transport and storage. Thawing and freezing during transport as well as long-term storage, even freezing, reduce the vitamin content of the food. Thiamine (vitamin B_1) deficiency can occur in fish eaters fed frozen fish caused by the activation of thiaminase, which inactivates the available vitamin.[16] Projects that produce their own prey animals should undertake health screening, which is important to perform prior to the breeding season. Representative animals should be killed and examined postmortem, and appropriate samples submitted for parasitology, microbiology, and virology. Externally sourced prey items represent one of the greatest biosecurity risks to large falcon breeding projects, and breeders should be advised to screen poultry that is used as feed at least biannually for *Salmonella*, *Campylobacter*, *Pasteurella*, *Mycoplasma*, *Chlamydia*, adenovirus, rotavirus, and routine parasitology.

Avicultural staff preparing food should follow strict hygiene protocols, and quality control should be rigorous to ensure that substandard food items are rejected and returned to wholesalers (see Box 42.2 for an example). Food should also

BOX 42.2 FOOD PREPARATION ROOM: DAILY, WEEKLY, AND MONTHLY TASK SCHEDULE

Tasks*	Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
DAILY (AFTER FOOD PREPARATION IN THE MORNING)							
Cover food with plastic/cling film and store in cold room							
Collect and dispose of feathers and gut contents from food animals separately in a bag/bin							
Clean and disinfect all food preparation equipment and return these to their designated locations							
Clean and disinfect all food preparation and other working surfaces							
Clean sinks, taps, and draining boards with disinfectant (e.g., F10)							
Check and clean walls in food preparation area and cold room appropriately							
Sweep and mop all floors							
Rinse work clothes and hang to dry on dry rack							
Clean and organize food preparation room after food has been removed in the afternoon							
WEEKLY							
Clean and soak all food tools and cleaning brushes in disinfectant (e.g., F10 for 10 minutes)							
Check freezer temperatures							
Wash all clothing and towels							
Clean restroom							
Clean and organize workshop area and entrance (including floor)							
Clean and organize aviculture equipment room							
Replace fly papers as needed; empty electronic fly zappers							
Refresh foot baths							
Empty trash							
MONTHLY							
Disinfect preparation room (e.g., F10 for 20 minutes)							
Hose and scrub preparation and cold room floors							
Clean and disinfect bucket stands and log rests							

*Tick when task has been completed.

be slowly thawed dry, to avoid food from coming into contact with thawing water, which leads to an increased bacterial load. Breeding projects that prepare large quantities of chick food will purchase freshly killed prey items and process them into chick food that can be frozen.

It can be tempting for raptor breeders to reduce costs associated with feed. This might mean feeding shot or otherwise sourced free-living animals such as pigeons to raptors, which has been associated with an increased risk of ingesting agricultural toxins and exposure to known pathogens such as *Trichomonas* spp. and herpesvirus (see also Chapter 22).[17] However, despite the well-known risks of feeding wild prey, many projects surprisingly still include wild-sourced pigeons and gamebirds as part of the chick's rearing diet.

Diets for raptor species should always include bone, which is available to the bird's digestive tract. Chick diets are often prepared by mincing skinned and eviscerated prey items such as rat or quail for medium/larger species or mice for smaller species. It is vital that larger bone or tooth fragments, particularly from rats, are removed from minced baby falcon food, especially during the first stages of life (days 1–3). One way of minimizing issues with indigestible and potential obstruction causing bone fragments (Figure 42.15) is to mince the first-stage diet twice. Casting material is generally not offered to chicks during the first 10 days of age. Overfeeding hand-reared chicks during their first week can result in crop and/or gut stasis. To avoid this, chicks should be weighed before each feeding session to monitor growth rates. In addition, food intake should be controlled, whereby chicks are generally fed the same or slightly more than their previous feeding.

Nutritional deficiencies and associated diseases can result from feeding chicks with inappropriate diets. Vitamin E deficiency in captive avian species, including raptors, has been associated with low fertility, low hatchability, immunosuppression,



FIGURE 42.15 (A) Distended gut in a chick that died because of impaction with rat bone fragments. (Photo courtesy Tom Bailey.) (B) Rat bone and tooth fragments in poorly prepared baby falcon food. (Photo courtesy Tom Bailey.)

and specific clinical abnormalities such as encephalomalacia and muscular myopathies.[18,19] Biotin (vitamin B_7) deficiency, as another example, has been associated with a reduction in hatchability without adversely affecting egg production.[11] In birds, a diagnosis of vitamin deficiency, for example, vitamin B_1/thiamine[20] or vitamin E,[18] was traditionally based on clinical signs and the clinical response to supplementation. However, now that tests measuring vitamin levels in tissues and blood are more widely available, the ability of veterinarians to diagnose deficiencies and to provide more rational supplementation can improve. If there is concern about the nutritional status of chicks, then assessment of blood vitamin levels in the parent breeding falcons may be considered. Tissues from prey animals can also be submitted for nutritional assessment when deficiencies are suspected, and routine nutritional assessment may be advisable for large commercial falcon breeding projects. Vitamins, probiotics, and calcium-vitamin D_3 supplements can be added to the food being fed to young chicks, if deficiencies or imbalances are suspected or confirmed.

PEDIATRIC DISEASES

Jones[12] provides a very useful section on raptor pediatrics. Some (common) nutritional and noninfectious diseases of raptor chicks are presented in Table 42.2.

Gastrointestinal Disorders

Impactions

Mechanical obstructions of the gastrointestinal tract are not uncommon causes of death and morbidity in raptor chicks, particularly during the first week of life. Impactions are usually caused by chicks being fed indigestible hard structures such as bone fragments or rodent teeth (Figures 42.15 and 42.17). This can occur when the chick food has been inadequately prepared or quality control is poor. Impacted chicks will show signs of starvation and gastric stasis. Chicks will become dull and may stop passing feces. Septicemia is often a sequel to impactions, particularly if sharp material perforates the gut lining. Palpation of the abdomen often demonstrates hardened ventriculus or intestine. Radiography can be helpful, if available. While surgery is possible, it is rarely performed on young raptor chicks and generally carries a poor prognosis. Parenteral rehydration and the use of motility-enhancing drugs such as metoclopramide and laxatives such as lactulose or liquid paraffin can be helpful to help resolve mild cases.

Obstructions

It is particularly important to check the crops and stomachs of chicks under 10 days old that are being hand-reared. Overfeeding and obstructions can be fatal. A chick should not be fed until its crop is empty. The stomach should be checked, which can be done by checking its lower abdomen. The stomach should feel soft and flaccid, and the overlying skin should be pinkish in color. If the area appears dark or the stomach is hard to the touch, this indicates a large amount of food remains in the alimentary tract, and thus feeding should be delayed until the stomach has sufficiently emptied. A particular problem that can cause obstructions can occur when bones from adult prey (rats or quail, for example) are inadequately ground down in mincer units for young falcons. These dense bones are poorly digestible within the neonatal stomach and can accumulate, causing gastric disturbances leading to fatal impactions. Mineral oil or lactulose are commonly used to help ease the passage of ingesta through the alimentary tract. Prokinetic agents such as cisapride or metoclopramide are also helpful to stimulate motility of the gastrointestinal tract. It is important to ensure that the chick is adequately hydrated by administering appropriate fluid therapy.

Cloacal prolapse

Cloacal prolapses are not commonly seen in raptor chicks (Figure 42.18). In other species, prolapses are associated with diarrhea, impactions, nutritional deficiencies, and tenesmus, and similar causes may be expected in birds of prey.[23] Placement of a purse-string suture or a transvent vertical suture can be helpful until the primary cause is dealt with and the tissue is viable. Cloacapexy is an alternative technique to purse-string sutures (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, for a description of the technique).

Yolk Sac Infection

Yolk sac infections, as discussed under hatching, usually occur around hatching. In case of infection, it may be possible to see an inflamed area on the yolk sac wall around the navel or around the umbilical region. As with other bacterial infections in young birds, treatment must be initiated quickly, which often involves fluid therapy to correct dehydration and appropriate antibiosis, based on culture and sensitivity results.

TABLE 42.2
Selected Nutritional and Noninfectious Diseases of Raptor Chicks

Disease	Etiology	Clinical signs	Diagnosis	Treatment and Prevention
Vitamin B deficiency	Thiamine (vitamin B_1) deficiency	Stargazing, seizures, incoordination, and opisthotonos	History, clinical signs and physical examination. Plasma vitamin levels, if possible	Vitamin B complex dose based on thiamine (vitamin B_1) content (3–30 mg/kg IM q7d or 30 mg/kg in food q48h) until signs resolve[21]
Vitamin E deficiency	Vitamin E deficiency in food caused by storage or rancidity issues	Similar signs as for vitamin B deficiency	As above	Improve diet Give selenium and vitamin E (0.06 mg/kg IM q7d or 15 mg/kg PO once[21]
Hypothermia and hyperthermia	After hatching chicks are covered in natal down and cannot maintain their own body temperature. Poor parental brooding or inadequate brood temperature can cause hypothermia. Hyperthermia occurs when heat lamps are too hot or too close to the chicks	Chicks that are too cold will huddle together and shiver. In contrast chicks that are too hot will pant and create distance to the other chicks. Hyperthermia can cause dry wrinkled reddish skin, dehydration and contribute to impactions and gut stasis	Clinical signs or cloacal temperature	Brooder temperature should be 36°C (96.8°F) and reduced by 0.3–0.6°C (0.5–1°F) per day until the chick can be kept at room temperature at around 10–14 days. Chicks should be given a temperature gradient and allowed to choose their comfortable thermal zone. Hyperthermia is prevented by careful monitoring of temperatures with infrared thermometers or dataloggers
Splay legs	Usually caused by chick being kept on slippery surface	One or both legs deviate laterally	Physical examination	Hobble legs with an adhesive conforming bandage or place padding that allows chicks to sit with legs underneath them. A substrate of fine pea gravel provides a texture that prevents splaying
Angel wing	Increased weight of the rapidly developing primary feathers causes an overextension of the carpal joint and the outer wing starts to droop	The wing twists outwards at the carpal joint and the outer wing droops	Physical examination (Figure 42.16)	Taping of affected primaries in a natural position at first sign of outward turning will correct the deformity. See Zsivanovits et al.[22] for description of correction of valgus deformity in a goshawk

Metabolic Bone Disease

MBD is a common problem in all species of young birds. Avian MBD may be seen as splayed legs, fractures, folding fractures, rickets, beak malformations, crooked toes, bowed legs, tibiotarsal rotation, and slipped tendons (Figures 42.13, 42.19, and 42.20). An excess of either calcium or phosphorus results in the sequestering of the other in the form of an insoluble calcium phosphate.[24] In the intestine, this results in less absorption and lower availability of the element involved. Calcium deficiency is a common cause of nutritional secondary hyperparathyroidism in falcon chicks and is associated with a range of growth- and calcium imbalance–related issues. Tibiotarsal rotation can result in varus deformity, which is seen as rotation of the tibiotarsal bone laterally above the hock joint rotating the foot (tarsometatarsus and toes) laterally/to the outside.

Metabolic bone disorders are covered in greater detail in veterinary texts on poultry species and long-legged birds where more research has been conducted.[25] Historically in falcons, MBD used to occur when chicks were fed bone-deficient diets, but this is now rarely the case because breeders are better informed. Despite great attention to the provision of calcium and vitamin D supplements, and minced chick diets with crushed whole bones, sporadic cases of MBD do occur. Some breeders believe that crushed rat bones may not provide enough available calcium because of the thickness of rat bones compared to quail or mouse bones. It is also considered that enteric infections causing malabsorption of minerals such as phosphorus might be contributing factors.[25]

Radiographic imaging is important in demonstrating decreased bone density, growth plate abnormalities, fractures, and other skeletal malformations. Dietary review and appropriate supplementation are important in the prevention of MBD in chicks. Some falcon breeders are also using full-spectrum lighting in rearing nurseries to help increase the conversion of vitamin D into its active form.

Viral Diseases

Avian pox, herpesviruses, paramyxoviruses, and adenoviruses can cause morbidity and mortality in young raptors (see Chapter 15 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease). Volume 1 provides information on the clinical thought processes in diagnostic



FIGURE 42.16 Desert eagle owl *(Bubo ascalaphus)* with angel wing. (Photo courtesy Tom Bailey.)



FIGURE 42.17 An accumulation of dense quail bone fragments within the stomach of a gyrfalcon *(Falco rusticolus)*. The mincer used to prepare the food had been used with too large a diameter grinding head, allowing large bone fragments to be incorporated into the baby falcon food. (Photo courtesy Tom Bailey.)

decision-making pertinent to some of these viral disorders in birds. Adenovirus infection is discussed in the following section because it has been a significant cause of mortality in some of the smaller falcon species.

Adenovirus infection

Adenovirus infections have caused significant outbreaks of mortality in breeding projects and have been confirmed in the following species: aplomado falcons *(F. femoralis)*,



FIGURE 42.18 Cloacal prolapse in a falcon chick with a *Salmonella* infection. (Photo courtesy Tom Bailey.)



FIGURE 42.19 Radiograph showing severe metabolic bone disease in a juvenile Bonelli's eagle *(Aquila fasciata)*. (Photo courtesy Tom Bailey.)

American kestrels *(F. sparverius)*, goshawks, merlins, peregrine falcons, Mauritius kestrels, orange-breasted falcons *(F. deiroleucus)*, gyrfalcon × peregrine hybrids and Taita falcons *(F. fasciinucha)*.[26] The author has suspected the disease in New Zealand falcons (*F. novaeseelandiae;* Bailey, personal observations).

Clinical signs include anorexia, diarrhea, and sudden death. The primary lesion in affected chicks is multifocal random coagulation necrosis and inflammation of the liver. Adenovirus infection is diagnosed by demonstrating intranuclear inclusion bodies in affected organs and can be confirmed by adenovirus polymerase chain reaction (PCR).[26]

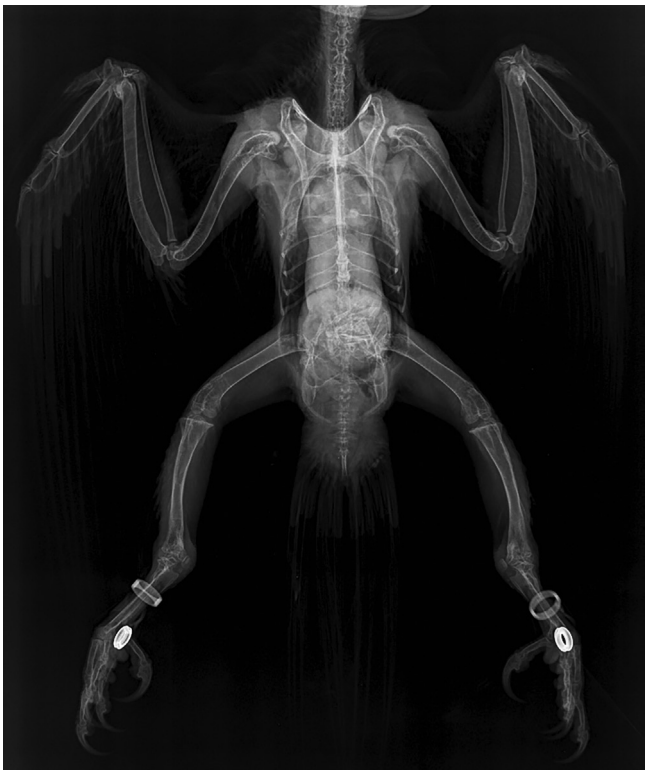


FIGURE 42.20 Radiograph of a juvenile gyrfalcon *(Falco rusticolus)* hybrid showing permanent bony changes caused by mild metabolic bone disease. Note the bowing of the femurs, tibiotarsi, radiuses, and ulnas. Although the radiographic signs appear obvious, the bird itself showed no obvious physical deformities. (Photo courtesy Tom Bailey.)



FIGURE 42.21 Feather dystrophy following suspected adenovirus infection in a New Zealand falcon *(Falco novaeseelandiae)*. (Photo courtesy Tom Bailey.)

Small falcon species seem to be most susceptible (aplomado falcons, Mauritius kestrels, New Zealand falcons), with young birds of less than 35 days of age predominantly being affected. The incubation period is approximately 6 to 10 days. Dystrophic feathers have been noted in New Zealand falcons (Figure 42.21) that were suspected to have survived adenovirus infection during the second week of life (Bailey, personal communication).

Forbes et al.[27] reported an outbreak in Mauritius kestrels and considered that turkey poults and domestic chicks fed to the birds were the source of the infection. Schrenzel et al.[26] were unable to identify the source of the adenovirus in the disease outbreaks that they described but considered that several members of the genus *Falco*, including peregrine falcons and gyrfalcons, may have been reservoirs of the virus. Contact with carrier falcons within a multispecies facility may transmit the virus to susceptible species, particularly young birds, and should therefore be prevented, if possible.

Bacterial Diseases

Bacterial enteritis can cause morbidity and mortality in falcon chicks and is influenced by many factors including intestinal flora, nutrition, environmental factors, and pathogens.

Intestinal Flora

Birds hatch from the egg with a completely sterile digestive tract, and microbes are quickly picked up within the nest or incubator.[28] The source of the microflora depends on the hygienic conditions of the environment and the presence or absence of the parents.[28] In intensive breeding projects, falcon chicks are incubated and hatched from sterilized eggs, incubators, and hatchers, and after hatching they are reared in a heavily disinfected environment. Under these circumstances, it is hard for chicks to acquire a normal intestinal microflora, which comprises useful bacteria that protect the intestine from infection with pathogenic bacteria by occupying the available attachment sites (competitive exclusion) and by creating an environment (pH and metabolites) in which pathogens cannot survive.

Bacterial organisms cultured from the upper digestive tract include *Staphylococcus aureus*, *Bacillus*, and *Corynebacterium*.[12] Bacterial organisms cultured from the lower digestive tract include *Escherichia coli*, *Enterococcus*, and *Proteus*.[12] These are "normal" flora, although they can cause problems in immunocompromised chicks. Gram-negative bacteria are the most important cause of enteric disease in young falcons and include *Salmonella* spp., *E. coli*, *Salmonella*, *Clostridium*, *Mycobacterium*, *Pasteurella*, and *Proteus*.[12] Battisti et al.[29] described neonatal mortality in a breeding center caused by salmonellosis. The main sources of *Salmonella* identified in this case were the infected poultry meat and day-old chicks that were used to feed the birds.

Staphylococcus (not *aureus*) and *Streptococcus* are considered normal flora of the raptor respiratory tract. *E. coli*, *Pseudomonas*, *Klebsiella*, *Pasteurella*, *Acinetobacter*, *Moraxella*, *Aeromonas*, and *Mycoplasma* have been isolated from chicks with respiratory



FIGURE 42.22 *Pseudomonas* sinusitis in a saker falcon *(Falco cherrug)* chick. The chick also showed neurological signs (head tilt). No viruses were isolated from this bird. (Photo courtesy Tom Bailey.)

disease (Figure 42.22).[12] Diagnosis of bacterial diseases in chicks requires clinical examination and bacterial culture of suspected lesions. Immediate antibiotic treatment and fluid therapy are warranted to treat the infection and correct dehydration.

Parasitic, Protozoal, and Fungal Diseases

Coccidial infections

Coccidian parasites that can cause health issues with young raptors include *Caryospora*, *Cryptosporidium*, *Eimeria*, *Frenkelia*, *Sarcocystis*, and *Toxoplasma gondii*.[12] While coccidia are not generally considered to be highly pathogenic in adult raptors, infections with *Caryospora* in juvenile raptors can be severe and even fatal. Clinical signs include lethargy, depression, diarrhea (sometimes with blood), poor body condition, and weight loss. The disease is readily diagnosed by the detection of oocysts in fecal samples by microscopy. Forbes[30] describes the clinical disease and control in juvenile falcons (see Chapter 9 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).

Aspergillosis

Young raptors, especially compromised or debilitated birds, are susceptible to fungal infections. Aspergillosis should therefore always be considered a differential for any young birds showing respiratory clinical signs (Figure 42.14), especially in gyrfalcons or gyrfalcon hybrids (see also Chapter 8 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 2, Infectious Disease).

Candidiasis

Infections with *Candida* spp. can cause oral and crop infections. Candidiasis in falcon chicks can also be a side effect of prolonged antibiotic therapy or poor food hygiene (see also Chapter 41).

Miscellaneous Conditions

Alula malpositions

The alula is the freely moving first digit, the bird's "thumb," and typically bears three to five small flight feathers. The alula is an aerodynamic device used when a bird lands to prevent stalling. Malpositioning of the alula occurs in gyrfalcons and gyr-hybrid chicks from 50 days old. The development of this deformity coincides with the period when young falcons are in groups of up to 30 birds in large hack pens and are hard to observe individually. When the birds are caught for sale at 70 to 90 days of age, the malposition, a ventrolateral deviation of the alula, is noted. It is most obvious when the wing is extended and can affect one or both wings (Figure 42.23). While the deformity does not appear to have any negative effect on flying performance, it is unsightly and reduces the commercial value of the falcon.

While no definitive etiology has been identified, breeders consider environmental conditions including inadequate humidity and temperature to be factors. Barbon et al.[31] reviewed surgical techniques to correct alula malpositions and reported that no technique is 100% effective. Fracturing the base of the first digit and stabilizing the alula to adjacent covert feathers using cable ties for 2 to 3 weeks seems to be a simple and



FIGURE 42.23 Gyr-hybrid falcon showing bilateral alula malpositioning. (Photo courtesy Tom Bailey.)

effective technique that is commonly employed by veterinarians working in falcon hospitals in the Middle East (Barbara Arca-Ruibal, personal communication, 2023). The technique is performed under anesthesia, followed by postoperative analgesic treatment, and appears to resolve in many cases presented between 60 and 90 days of age.

Growing skull fracture

A growing skull fracture has been reported in a red-tailed hawk *(Buteo jamaicensis)*.[32] Skull trauma to nestling raptors may lead to the development of this disorder and can potentially cause neurological signs and visual deficits. Three-dimensional computed tomographic imaging may reveal skull defects that are not readily visible on radiographs.[32] Growing skull fractures have been reported in humans, usually resulting from injury to the soft skull during the rapid growth phase of an infant's life. Nestling raptors have thin, fragile skulls, a rapid growth rate, and compete aggressively for food items; hence, this condition should be considered a potential diagnosis when unexplained neurological signs are seen in young chicks.

CONGENITAL PROBLEMS

Congenital disorders are occasionally seen, but unfortunately issues associated with inbreeding may become increasingly frequent in the future. Inbreeding has been shown to cause early embryonic death in passerine species.[33] Because of import restrictions, it is becoming increasingly difficult in Europe and North America for breeders to legally acquire wild-caught unrelated breeding stock. Given that only a few raptor breeders have good record systems and unscrupulous breeders sell related birds as unrelated, it is inevitable that the limited captive pool of birds used for breeding will become more inbred with each passing generation. Splay or spraddle legs, scoliosis, lordosis, opisthotonus, cerebellar defects, hydrocephalus (Figure 42.24), eye abnormalities, joint deviations, and stunted birds are examples of congenital and growth problems seen in psittacines and raptors.[23,34] Recently, Moore et al.[35] diagnosed photoreceptor dysplasia in peregrine falcons and peregrine falcon hybrids and suggested this to be heritable.

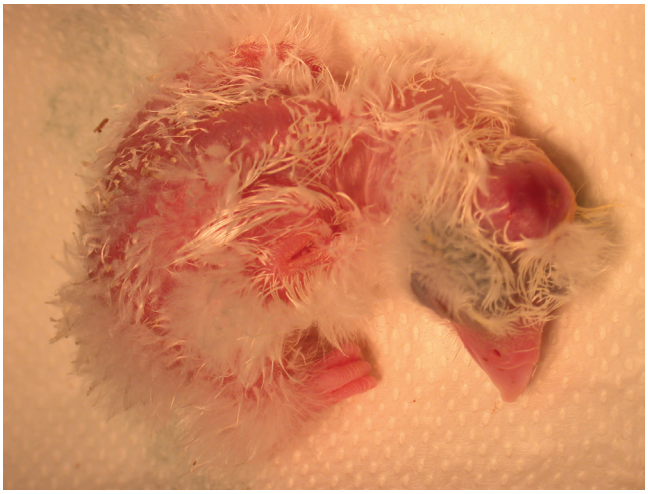


FIGURE 42.24 Hydrocephalus in a newly hatched falcon chick. (Photo courtesy Tom Bailey.)

It is likely that genetic testing will become an important tool for diagnosing genetic and inbreeding issues in the future. Relatedness can be tested through multiple gene loci comparison between birds and may be helpful to identify the genetic variety within a breeding collection, which can be an important tool for managing breeding in controlled study books for species conservation breeding programs.

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I thank Martyn Paterson from Desert Falcons and Nick Fox, Oscar Oliva, Sandor Sebastian, and Diana Durman-Walters for the avicultural knowledge they shared during the 3 years I worked at International Wildlife Consultants breeding falcons. Thanks to Jorge García de la Fuente and Barbara Arca-Ruibal for sharing their experiences of correcting alula deformities in falcons. Thanks to Holly Cale from the International Centre for Birds of Prey for sharing additional images. Information in Table 42.1 was extracted from the Avian Information Management System (AIMS) courtesy David Le Mesurier (www.visualaims.com).

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CHAPTER 43

Incubation and Pediatrics of Columbiformes (Pigeons/Doves)

Shangzhe Xie

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INTRODUCTION

There are 371 species in the family Columbidae (order Columbiformes), out of which 12 have been classified as critically endangered and 20 have been classified as endangered by the International Union for Conservation of Nature and Natural Resources (IUCN) Red List.[1] As strategies to increase the reproductive efficiency of columbids become more important in countering decreasing population numbers in the natural habitat, artificial incubation and hand-rearing may become essential for the conservation efforts of these species.

INCUBATION

The artificial incubation of columbiform eggs follows a standard protocol. The incubation temperature is maintained at 37.2°C (98.6°F) and the humidity level at 55%. The eggs are weighed daily to ensure they maintain their 15% (±3%) total weight loss from lay. The daily weight loss can be controlled by moving eggs that lose either too little or too much weight into additional incubators set at 35% and 75% humidity, respectively. The automatic turning mechanism of the egg incubator should be set at 24 turns a day, that is, once every hour, with the turn occurring over 10-minute intervals. Forty-eight hours before the anticipated hatch date and/or at the time that internal pipping is observed, the egg should be moved into a hatching incubator with rollers removed and set at 75% humidity.

HAND-REARING PROCEDURES

Columbiform chicks are altricial and therefore require hand-rearing if hatched away from the parents. Hand-rearing protocols appear relatively similar among the different columbiform species (see Appendix 4G). There are a variety of hand-rearing formulae available in the market, and those formulated for columbiform species should contain 20% to 22% crude protein and 13.14 to 13.39 MJ/kg metabolizable energy.[2,3] An example of a columbiform hand-rearing protocol is summarized in Table 43.1.

NORMAL DEVELOPMENT

The development and milestone achievements of columbiform chicks appear consistent among species. An example is provided in Table 43.2, comparing these milestones among columbiform species of different sizes. Appendix 4G provides the growth curves of various columbiform species.

COMMON PEDIATRIC PROBLEMS

Like other species of avian neonates discussed in other chapters in this section, columbiform chicks can be affected by the full range of avian pediatric problems, including crop stasis, crop thermal injuries, crop perforations, valgus deformities of the wings, splay leg, beak malformations, omphalitis, and infectious diseases. Prevention and management of these common pediatric problems are discussed in another chapter, so only the more common columbiform-specific pediatric problems or those requiring a different, columbiform-specific preventive or management approach will be discussed here.

CROP STASIS

Complete or incomplete stasis appears to be common in the first few days post-hatch and whenever there are changes to the feeding regime. The crop should not be completely empty in between feeds, as this may predispose to crop stasis. Failure of the crop to empty at a normal rate should be readily picked up by the carer, and initial diagnostics should include a review of the feeding protocol and growth chart (see also Appendix 4G), to determine if there are systemic diseases that need to be investigated. Initial diagnostic tests should include crop and fecal Gram stain cytology and culture and sensitivity testing. Hematology, biochemistry, and radiographs may also be useful if an underlying systemic disease is suspected.

The hydration of a chick affected by crop stasis should be maintained by increasing the amount of water content either in the hand-rearing formula or parenterally. A crop lavage can be performed but should be done with caution, as completely emptying the crop can exacerbate the slow clearance of crop contents. Antibiotics and/or antifungals should be prescribed based on the results of crop and fecal cytology and culture and sensitivity testing. In the author's experience, bacteria and yeast overgrowth in the crop are equally common, so assumptions should not be made without diagnostic tests being performed. Metoclopramide can also be included as part of the treatment plan, if a foreign body obstruction has been ruled out, but its efficacy in this zoological group is unknown. If the crop stasis is addressed early, before the crop is overly distended, then

TABLE 43.1

Mandai Wildlife Group, Jurong Bird Park, Breeding and Research Centre, Columbiform Hand-Rearing Protocol

Days Post-Hatch	Brooder Temperature (°C)	Feeding Frequency	Food Intake by % of Body Weight (per Feeding)	Miscellaneous
0	35	Every 2 hours (= 7× a day)	10%	Apply betadine over umbilicus 2×/day for first 2 days. Feed using cannula infusion tip on syringe, clear rubber feeding tube or crop needle depending on chick size. Ensure there is no air in syringe before feeding. Feed formula at 38°C–40°C.
3	34			
5	33	Every 3 hours (= 5× a day)	12%	
6		15%		
8		Every 4 hours (= 4× a day)	17%	
9	32			
11				Daily weight gains over 15% after this age may result in valgus deformity of the wings.
13	31			
14				Offer lightly soaked pellets/ripe papaya with no skin hanging from a cup to omnivorous columbiform species. Offer millets to granivorous columbiform species.
17	30			
21	29	Every 5 hours (= 3× a day)		Expect daily weight gains to be less than 10%.
22	28			
23		Start reducing volume of second feeding (e.g., 20 g, 10 g, 20 g)		
24	27			
25		Every 8–12 hours (= 2× a day)		
26	26			
28				Most species are starting to eat well on their own and due to fledge.
30		Every 24 hours (= 1× a day)		
35–40		Discontinue feeding if chick is readily eating on own		

there should not be any need for external physical support of the crop using a crop brace. However, a crop brace is a useful adjunct in severe cases not addressed early.

The prognosis of crop stasis in columbiform chicks is usually good if appropriate treatment is provided early and there are no underlying systemic diseases involved.

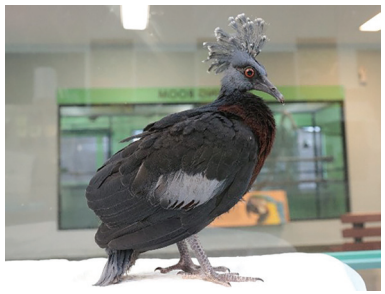
TRICHOMONIASIS

Although infrequently observed at the author's facility, *Trichomonas* spp. is a protozoan parasite (see Chapter 22) that columbiform species, particularly young ones, are susceptible to. Trichomoniasis most commonly presents as unthriftiness and high mortality in young columbiform birds, with diphtheritic membranes a common finding on physical examination (Figure 22.4A). A definitive diagnosis can be obtained by finding motile protozoa on microscopic examination of a crop wash, throat swab, and/or feces. Another presentation of trichomoniasis in young columbiform chicks is omphalitis (Figure 22.4B), which presents most commonly with very large retained and inflamed yolk sacs comprising 20% to 40% of the chick's weight.

Treatment with a nitroimidazole (metronidazole, dimetridazole, ronidazole, carnidazole) is usually effective if diagnosis is made early. Supportive treatment with antibiotics for secondary bacterial infection may also be warranted. In flocks of columbiform birds where trichomoniasis is a persistent problem, improvement of husbandry conditions and strategic treatment of the flock may be indicated, for example, during the breeding season.

TABLE 43.2

Comparison of Milestone Achievements Among the Santa Cruz Ground Dove *(Alopecoenas sanctaecrucis)*, Jambu Fruit Dove *(Ramphiculus jambu)*, and Victoria Crowned Pigeon *(Goura victoria)*

	Santa Cruz ground dove		Jambu fruit dove		Victoria crowned pigeon	
Milestone	**Number of Days Post-Hatch**	**Chick appearance**	**Number of Days Post-Hatch**	**Chick appearance**	**Number of Days Post-Hatch**	**Chick appearance**
Full yolk absorption, pin feathers present	1		1–2		1–2	
Toes uncurled, able to grasp	2		3–4		3–4	
Eyes fully open	8		8–10		5–7	
Fully grown and weaned	30–60		41–60		31–40	

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CHAPTER 44

Toucan Incubation and Pediatrics

Mathias Dislich

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INTRODUCTION

Toucans are tropical birds that are easily recognized for their prominent beaks. The general term toucan will be referred to throughout this text as synonymous with all birds that belong to the family Ramphastidae, such as aracaris, toucanets, and toucans. Toucans are found in a wide range of biomes, from tropical forests to Brazilian cerrado, and therefore they are expected to have different diets, behaviors, or even disease susceptibilities. On the other side, with a few exceptions, there is still scarce information to support species-specific recommendations for husbandry and nutrition. An evidence-based approach for medical intervention in toucans is especially hindered by the lack of controlled studies, especially those related to therapeutics, which makes extrapolation from other species a rule. Large-scale investigations are difficult to conduct, because of the small number of chicks that are bred.[1]

Toucan neonatology has many similarities with other altricial birds, such as Psittaciformes and Columbiformes. For all those birds, breeding success depends on meticulous observation, proper sanitation, and nutrition. With a few exceptions, the basic procedures regarding artificial incubation and hand-rearing are the same as for psittacines. The ability to rear toucans can be considered rare.[2] Sensitivity, carefulness, and patience are essential attributes for good birdkeepers.[3] Most difficulties in breeding toucans are not related to pairing, egg laying, and incubation but to the survival of the chicks.[3]

This section will focus on the peculiarities of Ramphastides that affect hand-rearing, the incidence of diseases, and their management in neonate and juvenile toucans. Since medical conditions that affect neonate toucans depend on the husbandry approach that is adopted, they will be presented separately for parent-reared and hand-reared chicks.

PARENT-REARED CHICKS AND COMMON PROBLEMS

Whenever opting for parent-rearing, it must be assured that nests can be easily monitored, since parent neglect and diseased chicks are common events. Incubators and brooders should always be kept ready, if immediate removal of eggs or chicks turns out to be necessary. The use of infrared cameras (Figure 44.1) has been a valuable tool to provide a noninvasive method of nest inspection, during all breeding phases.[3,4] Removal of eggs from the nest should be done with care and planned, preferably at a time when none of the parents are inside the nest, for example, during feeding time. Quickly covering the nest entrance with a net or piece of cloth usually prevents parents entering the nest, so that eggs can be safely accessed through an inspection opening. Breeding toucans can be very aggressive, so these procedures should always be performed with assistance. Chick monitoring can be done by daily inspections and frequent weighing, in the case of pairs that tolerate those interventions.[5]

The most common causes of neonatal death in parent-reared chicks are predation, food or foreign material impaction, and parasitic diseases.

Predation

Any toucan that is reared in a nestbox has the risk of being predated by rats or opossums, but most of the predation events in neonate toucans are caused by their own parents. Parental predation may be related to several factors, ranging from stress to the destruction of chicks with congenital deformities.[3] The observation that predated chicks often end up being fed to their siblings leads to the conclusion that these events were caused by the scarcity of live prey in captivity. The increased supply of insects allowed a couple with a history of predation to successfully rear chicks.[3]

Impaction

In the urge of feeding their chicks, toucans might offer them items such as bark, sticks, and pebbles, which can cause impactions. For this reason, toucans should always have enough food available to avoid any other items being offered to their offspring. During this period, toucans will favor live food (mostly insects) as the main food for their chicks. Even with a enough supply of pellets, they might not be offered at all. For this reason, the best way to prevent impaction is to guarantee that the right food items and amounts are always available. Careful planning includes contacting a trustworthy insect supplier in advance or preparing your own facilities to produce a reliable number of insects. During the breeding season, one pair of toucans can consume thousands of crickets.[4] Toucans start to feed their chicks at sunrise, so from that moment on, they need to have something to offer. One way to increase the amount of edible items in the early morning is by installing lamps to attract insects to the aviary during the night.

The way the regular food is offered must also be taken into consideration, since it should be presented in smaller pieces

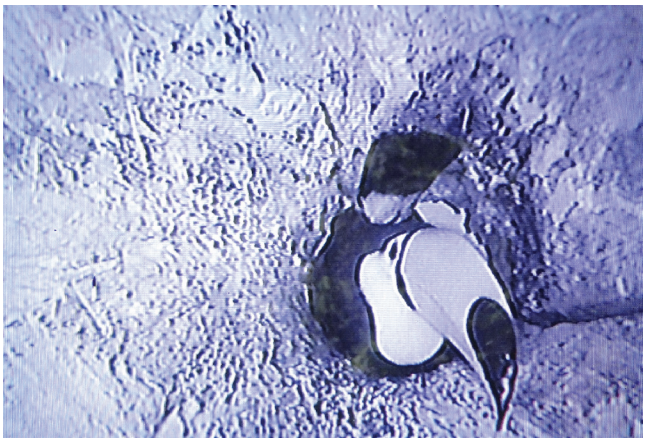


FIGURE 44.1 Infrared camera monitor view of a toco toucan *(Ramphastos toco)* hen in her nest. (Photo courtesy Mathias Dislich.)



FIGURE 44.2 Fatal ventricular impaction in a newly hatched chick; note the yolk sac is still present and quite large. (Photo courtesy Mathias Dislich.)

than usual. Fruits, such as papaya, must have their skin removed, because it can also cause obstructions (Figure 44.2).[3]

Impacted chicks generally have poor weight gains and might have soil in the feces.[2] Chicks with complete obstruction will not defecate, which is difficult to recognize when other siblings share the same nest. Any suspect case must be removed immediately from the nest and treated as an emergency. Once it has been hydrated and placed in a brooder, the production of feces is assessed. If it begs for food, a small amount of diluted hand-rearing formula can be offered. If no stools are passed after 15 minutes, between two or three drops of mineral oil can be given. Impacted toucan chicks have a poor prognosis.

Parasitic Diseases

Toucan chicks raised by their parents may be subject to ectoparasite infestations, since enclosures do not limit parasites access to nests. Some of these infestations can be intense, leading to the death of individuals.



FIGURE 44.3 Toucan chick infested with *Ornithonyssus* mites. As the chick is devoid of feathers, these mites and the skin rashes they induce can be easily identified upon visual inspection of the chick.

Mites

Mites of the genus *Ornithonyssus* can eventually parasitize toucan chicks, which become disturbed and agitated, feeding less. Consequently, they become dehydrated and do not develop well. Since *Ornithonyssus* spp. are obligate hematophagous mites, high degrees of infestation can lead to anemia,[6] which can be fatal if left unattended. Because toucan chicks are devoid of plumes, mites and skin rashes are easily identified during physical examinations (Figure 44.3). Toucan chicks have a dirty appearance, due to the accumulation of cast skins, eggs, and mite feces.[6] Infested chicks must be removed from the nest, to be stabilized and treated. The application of small amounts of propoxur (Bolfo®; Elanco, São Paulo/Brazil) with a cotton swab near the vent and the removal of the bird from the nest are generally enough.

Nests showing signs of infestation should be removed and treated with the application of propoxur powder inside or fumigated with formaldehyde tablets. It is important that, when returning to the enclosure nests, they are free of any excessive residues of those substances.

Philornis

Infestations by botflies of the genus *Philornis* are common in free-living toucan chicks and may also occur in captive birds. Adult flies are attracted by the organic matter present in the nests and lay their eggs on the skin of the nestlings. As a result, larvae burrow into the host integument, forming individual cysts between the dermis and musculature.[7] These can be found in various anatomical regions, such as the head, back, and limbs. At the end of 4 to 8 days, the larvae leave the bird and form pupae in the nest's substrate.[7] Those infestations cause discomfort, but birds do not stop feeding and development. However, on some occasions, the larvae may be found in more sensitive areas, reaching the eyeball, ear, or oral cavity, causing important lesions (Figure 44.4). Larvae are easily removed with forceps. Topical application of antibacterial ointments might be used, but normally they are not necessary.



FIGURE 44.4 Botfly-induced lesion caudal to the right nare in a young toucan chick. (Photo courtesy Mathias Dislich.)



FIGURE 44.5 Normal hatching of a toucan chick. Note the circular cutting pattern of the shell during the hatching process, as was demonstrated in psittacine chicks. (Photo courtesy Mathias Dislich.)

Endoparasites

Although it is possible that toucan chicks are infested by endoparasites, through the ingestion of insects offered by parents or contamination of feces of adults inside the nest, there are no reports of such diseases in these birds before they fledge.

HAND-REARED CHICKS AND COMMON PROBLEMS

The decision to remove eggs for artificial incubation must be weighed carefully, since both approaches have their benefits and risks, which depend on the purpose of the breeding (e.g., conservation vs commercial breeding) and on intrinsic characteristics of the facilities (equipment, local climate, risk of predation, etc.). In most institutions, those decisions are based on previous events, especially those related to the rearing experience of the breeding pairs,[1,5] but also consider other management issues such as weather conditions.[1] On the other hand, with proper husbandry, toucans can successfully rear their young.[1,8]

The removal of eggs for artificial incubation has been advised as a manner of increasing productivity,[1] since this can stimulate a second or even a third clutch. In the author's experience, this recommendation is controversial, since many toucan females will resume egg laying only in the next breeding season.

Artificial incubation of eggs can be done with the same incubators used for Psittaciformes. Eggs should be kept between 36.9°C and 37.5°C,[1,3] while relative humidity should be between 50% and 65%.[1,3] Ideally, humidity is adjusted based on an egg weight loss curve, since many factors such as eggshell quality and local climate conditions influence this parameter. A weight loss of 15% is ideal, but weight losses between 12% and 16% have been reported.[1] An accurate measurement depends on knowing the fresh egg weight, which means that the egg must be pulled out of the nest on the day it was laid. The relatively short incubation period of toucans makes any further adjustment to the curve difficult. Toucanets and aracaris have an incubation period of around 16 days, while bigger toucans have an incubation period that ranges from 16 to 18 days.[1] Risk versus benefit of egg weighing should be considered as it implies greater egg handling. Toucan eggs, especially those of small species, are delicate and have thin shells, which can easily break or suffer fissures. Toucan eggs can be easily evaluated by ovoscopy, which should be done quickly. If the ovoscope uses an incandescent light, eggs can quickly heat up, which can cause the embryo to die. Toucan eggs can be safely monitored with Buddy® (Avitronics Ltd, Norfolk/United Kingdom). Hatching occurs quickly and usually without difficulties, with pipping occurring in the middle of the egg (Figure 44.5).

Chick Rearing

As with other altricial birds, toucans are born featherless. Yet, while psittacine chicks have down feathers, toucans' chicks are completely devoid of feathers when born (Figure 44.6). Therefore special care should be taken with temperature to avoid hypothermia. Toucans should be handled as quickly as possible when outside the incubator. After hand-washing, the friction of the hand provides a warmer surface for a clinical examination. Neonate toucans can be placed inside a cupped hand so that clinical examination can be made safely (Figure 44.6). The absence of feathers facilitates inspection. It is possible to observe the yolk sac in the first few days, which is useful for the evaluation of yolk absorption. Lungs can be assessed percutaneously; congestion or hemorrhage is easily recognized.[3]

As soon as they hatch, neonate toucans should be placed in a brooder at high relative humidity, between 93% and 95%.[1] Due to previous cases of aspergillosis, the author has used lower humidity settings (between 50% and 60%). In those cases, chicks were constantly inspected because of the higher risk of dehydration due to the dryer atmosphere. Brooder temperature recommendations for neonate toucans vary from 35°C to 37°C. During all the rearing period, chicks must be observed carefully and the temperature must be adjusted according to visual cues. When it is too cold, chicks stay close to each other, shiver, or get lethargic.[8] When the temperature is too hot, they tend to extend their necks, stay away from each other, and their skin gets hyperemic.[8] Those may be mechanisms to increase temperature loss. As they grow up and the



FIGURE 44.6 Young, newly hatched toucan chick being handled with cupped hands to facilitate support of the neck and safe examination. (Photo courtesy Mathias Dislich.)



FIGURE 44.7 Single toucan chick with the head and neck being supported by rolled paper toweling during the first few days post-hatch. (Photo courtesy Mathias Dislich.)

first feathers erupt, brooder temperature can be gradually decreased.[1]

The best way to keep toucan chicks is to use deep bowls, lined with paper towels. Groups of toucan chicks provide each other warmth and support, especially in the way they rest their long necks. When only one chick is raised, a paper towel can be wrapped around the chick to provide similar support (Figure 44.7).[3] Toucan bedding should be changed often, since they defecate frequently. Constant monitoring of the stool volume, consistency, and odor is an important cue for early detection of medical conditions such as impaction and enteritis.

As soon as they are born, toucans are very active and beg for food. Neonate toucans have long necks and, when active,



FIGURE 44.8 Postmortem image of a young toucan. The large heel pads at the caudal aspects of the intertarsal joints and proximal tarsometatarsus are readily visible. These pads are shed at or about the time of fledging and weaning. (Photo courtesy Mathias Dislich.)

move them intensively, sometimes losing their balance. In order to keep their necks extended during feeding time while maintaining an upright position, they lean on the floor forming a tripod consisting of their abdomen and pelvic limbs (Figure 44.7). Toucan chicks have heel pads on the plantar surface of the intertarsal joints, which provide them with additional support (Figures 44.6 and 44.8). At the time of the fledging, they lose these appendices.

Feeding

Most differences between toucans and other bird species in hand-rearing are observed during feeding procedures. Toucans do not have a crop and have a shorter gastrointestinal tract. As a consequence, the transit time is shorter; therefore feeding intervals are shorter and meals have to be offered more frequently. Even though toucans can be considered eager feeders, accepting hand-feeding well, the time devoted to each feeding is prolonged when compared with psittacines.[1] This difference is even bigger when they grow older and start to receive solid food.

Hand-rearing protocols have been published elsewhere and can serve as a basis for the development of each facility's own protocols.[1,2]

The first day is devoted to hydration and stimulating the absorption of the yolk sac. First feedings are important to evaluate the feeding response and to adjust the feeding technique. Toucans do not have crops. To evaluate food ingestion, abdominal repletion is assessed. After feeding, the entire intestinal tract is filled with food, from the proventriculus to the intestines. The abdomen becomes rounded, and it is common for the chick to lose balance. Experience is needed to properly assess the filling of the gastrointestinal tract. Although toucan chicks usually stop feeding when full, this is not always true.[1] One-milliliter syringes are the most practical way to feed toucans in the first week, since food of different consistencies can be slowly delivered, drop by drop with full control. Since neonate toucans swing their necks when begging for food, their heads must be gently supported during feedings. After feeding, the beak is cleaned with a moistened paper towel to

remove any residues that can favor yeast or bacterial overgrowth.

At the beginning, feeding formulas start very diluted, and the amount of water is slowly decreased so that a more consistent (and energy-dense) food can be offered. The number of daily meals that must be offered depends on the energy content of the hand-rearing formula and the volume of food that the toucan chick accepts. After determining the best consistency (i.e., dilution), the number of meals and frequency can be estimated. Adjustments should be made daily, and good communication between the animal nutritionist and the nursery staff is essential. Daily weighing is essential. During protocol adjustments weighing every 12 hours is advised, because it allows the prediction of the daily weight gain and the option of additional feeds.[2] From the second or third day on, toucans are expected to gain between 8% and 12% of their weight daily for the next 2 weeks. With the inclusion of solid food, the growth rate slows down to approximately 2% to 5%, until chicks reach their adult weight (see Appendix 4H). Different growing curves have been described in which remarkable growth is considered to start just after the 10th day and extend for a longer period.[1] These differences might be attributable to husbandry and nutrition.

Most facilities use good-quality psittacine hand-rearing formulas during the initial phase.[1,3,8] There is a controversy over the use of those formulas for toucans, because of their propensity to develop iron storage disease. In nature, toucan chicks consume animal protein, and since toucan neonates are growing, they have a high demand for iron. Therefore the development of iron storage disease is unlikely in the first few weeks. The author investigated the presence of liver hemosiderosis in 13 deceased toucans, at less than 2 months of age and none had even mild iron deposits (unpublished data). Yet, once the toucan chicks start accepting food pellets, those should be the same as adult toucans receive.

Shortly after the first week, the liquid diet is slowly discontinued, and the adult diet is presented to the chicks.[1] At this age, toucan chicks already accept small pieces of food, which can be offered between regular feedings. These items include skinless fruit pieces, insects (moths, crickets), pinky mice cut-in pieces, and moistened adult pellets.[3] During this phase, toucan chicks must be closely monitored by constant evaluation of passed stools and growth rate. Since a loss in the daily weight gain is possible in the first few days, it is important to rule out the presence of any disease.[1] Over the next few days, the size of the pieces can be increased and also the amount of food. At about 2 to 3 weeks, soft food is completely discontinued. Extreme care should be taken to provide proper nutrition to the developing chicks, since developmental metabolic diseases can give rise to severe postural abnormalities. Strict commitment to feeding protocols is imperative. Toucan chicks tend to prefer fruits over other items, so that whoever is feeding them can be tempted to offer a higher proportion of those than necessary, thus unbalancing the diet. Feeding older toucan chicks requires patience, since they become more and more restless, and feeding procedures consume more time compared to parrot chicks.

Most toucan species fledge between approximately 6 and 7 weeks. At this time, they should have reached the weight of an adult or be slightly heavier. While smaller species are generally self-sufficient at around 45 days, larger species such as toco toucan become self-sufficient at about 60 days.[1]

Fungal Diseases

There is a high prevalence of fungal diseases in toucan chicks, to the point that several institutions adopt prophylactic measures for this at the beginning of the artificial rearing period.

The humid environment of the brooder chicks added to the fragile immunocompetence of the chicks favors the development of these diseases. Despite the much more contaminated environment in the nests, the incidence of fungal diseases is lower in parent-reared chicks.

Candidiasis

Candida infections are considered common in toucan chicks,[5] being observed in the oral cavity but also along the gastrointestinal tract of deceased toucans during necropsy.[1]

Since toucans do not have crops, the clinical signs of candidiasis are slightly different from those seen in parrots. Stunted growth can be observed, but in many cases, toucans die without premonitory signs. For those reasons, many institutions include in their hand-rearing protocols candidiasis prophylaxis starting from the first day. There are slight variations regarding the dosage, frequency, and length of treatment. The recommended dose is 300,000 UI/kg twice a day and should be adjusted as the bird gains weight.[3] Treatment length varies from 1 to 4 weeks.[1,2] Nystatin is considered safe for neonate birds, acting only locally. There are no reports of intoxication in toucans using those protocols.

Aspergillosis

There are few reports of aspergillosis in toucan chicks; however, in the author's experience, they are one of the main causes of death in hand-raised toucans, with a prevalence of approximately 20% (4/25). This disease was not observed in any of the parent-reared toucans that were born in the same period.

Aspergillosis can develop rapidly and is most often detected postmortem. Although it might cause stunted growth,[8] sudden death in chicks with normal development is also observed. The death of a 2-week-old toucan motivated treatment of its sibling with itraconazole. However, despite this, 1 week later, the second toucan died. At necropsy, two granulomas were observed, one in each lung. It is difficult to make an early diagnosis because the birds do not show clinical signs and the initial granulomas are too small to be detected with conventional tests, besides the risks of antifungal therapy make the prognosis of these cases very unfavorable. Contrary to nystatin, drugs recommended for the treatment of aspergillosis have a greater potential to cause intoxication, especially in neonates. For this reason, preventive protocols like those used for the control of candidiasis in toucans are not used for aspergillosis prophylaxis.

Beak Problems

Neonate toucans have short, slightly prognate beaks (Figure 44.8). This feature apparently facilitates feeding by their parents. As the chick grows, the beak gradually assumes its characteristic shape and size. Compared with parrots, juvenile toucan beaks are much softer and delicate. Until about 3 weeks of age, toucan beaks are extremely malleable.[2] During that time, extreme caution must be taken so that beaks are not traumatized during handling. It takes a few months for juvenile toucans to have their beak trabeculae completely developed and ossified.



FIGURE 44.9 Traumatically induced injury to the maxillary beak in a parent-reared toucan. (Photo courtesy Mathias Dislich.)

Beak deviations may occur for several reasons, such as syringe pressure during feeding.[2] Because of custom or ergonomics, chicks might always be fed from the same side, which forces the developing beak to deviate. Gentle handling and alternating sides when tube feeding are generally enough to prevent this condition. If this defect is observed, it can be corrected by applying gentle pressure to the tip of the bill in the direction that opposes the curvature, three or four times per day, bending the tip of the mandible about 20 to 30 degrees.[2]

Trauma

Once toucan chicks become more active, usually after the eruption of feathers,[1] care must be taken to prevent trauma. As their beaks grow and get heavier, they are more susceptible to lose balance and fall.[2] Their beak is still very soft and vulnerable to blunt trauma, which can result in hematomas. Perforation of the beak, although uncommon, can be a port of entry for bacterial disease, such as rhinosinusitis, which can be difficult to treat.

Fledging is the riskiest period for trauma accidents in juvenile toucans, because during this period they tend to be restless, have low control over their flight, and are not completely aware of their boundaries. This holds especially true for parent-reared toucans, which can damage their beaks trying to fly against the mesh (Figure 44.9). To prevent such accidents, the best strategy is to revise the enclosure furniture, such as perches and plants, and position them in a way that they obstruct direct flight attempts. Perches can be placed at different heights not too far from each other. One perch should be placed about 25 cm (10 inches) from the mesh. In this way, the fledging toucan can try short flights, slowly develop its skills, and get a better sense of its surroundings. For most species, fledging starts at about 6 weeks of age.[1,2]

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CHAPTER 45

Neonatology of Precocial Birds

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INTRODUCTION

Precocial birds are defined as those birds that are relatively mature and mobile at the time of hatching. The term *Aves precoces* was first used by Sundeval (1836).[1] This chapter will discuss the early development of the precocial bird in comparison to the altricial species with which clinicians are usually more familiar. Potential clinical consequences of these differences will also be discussed. For a full discussion, readers are referred to the excellent texts by Starck and Ricklefs (1998)[2] and O'Connor (1984).[3]

CLASSIFICATION AND SPECIES

Originally, the terms *Aves precoces* and *Aves altrices* were used to distinguish two methods of rearing. Nowadays, a wider classification of rearing strategies is recognized within the so-called altricial-precocial spectrum (see also Table 45.1; Chapters 39 and 41).[2] The naming of these strategies is based on Starck (1993),[4] although other sources may group these strategies typically as follows:

- Precocial = Superprecocial (Precocial 1) and Precocial 2 and 3
- Semiprecocial = Precocial 4 and Semiprecocial

DIFFERENCES BETWEEN PRECOCIAL AND ALTRICIAL STRATEGIES

The different strategies are defined according to the stage of development of the hatchling and the degree of parental care given, as is demonstrated in Table 45.1. From this table it may be derived that the primary diagnostic feature of precocial birds versus altricial birds is the fact that altricial birds have closed eyes at hatching. Another variable is that of thermoregulation. Altricial species are usually poikilothermic at hatching and develop endothermy at varying stages thereafter. Most precocial species demonstrate at least some degree of endothermy immediately after hatching. Other differences in post-hatch development will be discussed later in this section.

EVOLUTIONARY ADVANTAGES/ DISADVANTAGES

There is no straightforward relationship with evolutionary development of species and the rearing strategies. Both systems convey advantages and disadvantages (Tables 45.2 and 45.3) and there appears no ecological driver to these, that is, within an ecological niche many different strategies can be observed. The varying advantages would explain the development of hybrid rearing strategies in aviculture.

EMBRYOLOGY

As described earlier, the cost for a more mature chick at hatching is a longer incubation period. In the captive-rearing situation, this puts a large onus on incubation conditions, where any problems in the incubator or hatcher conditions (temperature, humidity, hygiene, etc.) are more likely to produce a compromised chick at hatching. These may include "wet" chicks, nonretracted yolk sac, or simply ill-thriving, less mobile chicks.[5] With less post-hatch development than in altricial birds, incubation problems are likely to cause higher chick mortality in precocial species.

EARLY GROWTH RATES AND DIET

Precocial birds have a slower growth rate than altricial birds *in the natural situation*. In the captive situation, the need for precocial species to move and lay down skeletal muscle quickly can be utilized in meat production of chickens and turkeys. However, consequences of this *artificially* high growth rate on the skeleton are well known. In the avicultural environment, high growth rates can lead to skeletal issues. These may include "angel wing" (Figure 45.1) and rotation or fracturing of long bones (Figure 45.2).

Contrary to most bird rearing, growth rates may actually need to be held low—for example, in buff-crested bustards *(Lophotis gindiana)*, growth rates should be held below 10%.[6] In the author's experience, in great bustards *(Otis tarda)*, reducing protein feeding and increasing fiber levels in the first week of life has produced lower rates of skeletal deformities in chicks. Olsen (2009) described 64% of fractures in young crane birds that occurred in the first 5 to 16 weeks of age, which they attributed to both a high-protein diet and lower exercise rates.[7] In addition, it was suggested that group size plays a role in growth rate, hence also affecting the incidence of skeletal deformity.[7]

Exercise rates are also implicated in other species. For example, Langgemach (2008) described positive effects on

TABLE 45.1
Levels of Precociality and Their Characteristics Recognized in Birds

Type of Development	Plumage Present?	Eyes Open?	Mobile?	Feed Themselves?	Parental Care?	Nest Attendance?	Examples
Superprecocial (precocial 1)	Yes, contour feathers	Yes	Yes	Yes	No	Leave nest	Megapodes (Megapodidae)
Precocial 2	Yes, down	Yes	Yes	Yes	Yes, brooding only (follow parents and find their own food)	Leave nest	Anatidae (ducks), Charadriiformes (plovers)
Precocial 3	Yes, down	Yes	Yes	Yes	Yes, showing food	Leave nest	Rheiformes (rheas), many Galliformes, including Meleagrididae (turkeys), Phasianidae (quail, pheasants), Numididae (guineafowl), Tetraonidae (grouse)
Precocial 4	Yes, down	Yes	Yes	Yes/No	Yes, parental feeding	Leave nest	Podicipediformes (grebes), Rallidae (rails), Gruiformes (cranes), Cracidae (guans and currasows), Turnicidae (buttonquail)
Semiprecocial	Yes, down	Yes	Yes/No	No	Yes, parental feeding	Remain in nest area	Laridae (gulls and terns), Alcidae (auks), Stercorariidae (jaegers and skuas)
Semialtricial 1	Yes, down	Yes	No	No	Yes, parental feeding	Remain in nest	Accipitriformes (hawks), Pelecaniformes (herons, pelicans), Ciconiidae (storks)
Semialtricial 2	Yes, down	No	No	No	Yes, parental feeding	Remain in nest	Strigiformes (owls)
Altricial	No	No	No	No	Yes, parental feeding	Remain in nest	Passeriformes (passerine birds), Psittaciformes (parrots)

Modified from Starck JM, Ricklefs RE, editors: *Avian growth and development: evolution within the altricial-precocial spectrum*, Oxford, UK, 1998, Oxford University Press.

TABLE 45.2
Advantages of Precocial Rearing Over Altricial Rearing

Factor	Advantages Conveyed
Self-feeding and parental fecundity	◆ Lack of parental feeding reduces feed-gathering ability as a limiting factor on reproductive rate ◆ Chicks follow their parent to the feeding site rather than their parent having to make repeated trips back to the nest ◆ Increased chick fitness may increase survival rate from predation—it is less likely for an entire brood to be predated ◆ Reduced dependence of chicks on requiring survival of both parents
Thermoregulation	Increased ability to thermoregulate increases ecological flexibility

skeletal development from early introduction to free space in first-week great bustards and exercise regimes in older chicks.[8] In the UK great bustard reintroduction program, increased exercise provision also was found to have a beneficial effect on skeletal development. The mechanism behind this is summarized by Ricklefs et al. (2008).[9] In birds, there is an inverse relationship between tissue maturation and growth rate, that is, if tissue is functional and maturing, the growth rate will be reduced. This appears to hold true for skeletal muscle in precocial birds, so it may be presumed that increased exercise in young birds (mimicking following of adults) may be reducing the growth rate of bone and muscle by prioritizing metabolic constituents to functioning of the tissues. Olsen (2009) also identified curled toe because of reduced exercise rates in

TABLE 45.3
Advantages of Altricial Rearing Over Precocial Rearing

Factor	Advantages Conveyed
Metabolic and developmental	◆ More gradual increase in metabolic intensity posthatch ◆ Delayed maturation of skeletal muscles (and effects on growth rate) allows higher growth rates to pass more quickly through vulnerable stages ◆ Lower metabolic rate allows faster growth rate ◆ Shorter egg incubation periods ◆ Increased postnatal brain development and, presumably, increased learning ability

cranes.[7] This observation also appears to be true for captive-reared great bustards in the author's experience.

THERMOREGULATION

Precocial species tend to have an increased ability to thermoregulate compared to altricial species. This may be in part due to better-developed plumage and in part due to shivering function by better-developed skeletal muscle.[3] However, these species may conversely be at increased risk for developing hypothermia than less mobile altricial chicks.

Increased mobility may increase the risk of wetting or being trapped in cooler areas (e.g., if stocking rates are high enough to lead to chicks being driven away from heat sources). As a result, water dishes for precocial chicks should be constructed such that immersion or tipping are prevented, and appropriate heat sources should be provided in various sites in an enclosure such that all chicks are able to obtain heat.

DEVELOPMENT OF THE GASTROINTESTINAL TRACT

Other than a proportionate increase in size with overall growth rate, there is little change in the structure of the precocial bird gut. This contrasts with the altricial species where the gut is comparatively underdeveloped at hatch and develops rapidly post-hatch. This—in part—may relate to the increased need for rapid growth rate in altricial species. In terms of precocial species, it does imply that the diet of the hatchling varies little from that of the adult bird, which may yet be another factor in controlling growth rate in young precocial species. It has also been noted that active, foraging young precocial birds may be at increased risk for gastrointestinal foreign bodies and impactions. As such, environments should always be carefully checked for any material that can easily be ingested.

DEVELOPMENT OF THE SPECIAL SENSES

As stated, altricial and precocial species may be distinguished by the eyes being open immediately (or within the first day in some species) after hatch. Given the advanced state of the senses in young precocial birds, light cycles and levels appropriate for adult birds may be provided from hatch. This contrasts with the situation of altricial birds where sensory flooding may occur with excess stimulation.

DEVELOPMENT OF PLUMAGE

Precocial birds (other than megapodes) are hatched with a covering of down. The first "mature" plumage will start appearing within the first 2 weeks, with primary feathers being among the earliest to emerge. The timing of this emergence varies with species.

One syndrome related to this plumage development is "angel wing." As stated earlier, this may be considered a skeletal deformity as it is typified by outward rotation of the distal wing (distal to the carpi) related to the weight of primary feathers and undercalcified bone. If identified early, corrective bandaging for a few days will be successful (Fig. 45.1). Bandages should always be changed every 3 to 5 days to allow physiotherapy to reduce the risk of joint contracture. In advanced cases, or in more mature birds, permanent changes which will preclude or reduce flight are likely.

Other than addressing underlying disease, prevention is key. Over the years, several underlying causes have been identified:

- Genetic factors[10];
- Growth rate and high-protein diets.[7] Growth rate may also be affected by group size[11] and has been related to angel wing incidence and severity[10];
- Underlying inflammatory disease. The effects of ingested T-2 toxin and antioxidants on alkaline phosphatase activity and on angel wing incidence and severity imply that underlying inflammatory processes may play a role in the incidence of angel wing in a flock.[12]

Based on the aforementioned risk factors, prevention is best achieved via growth rate control. Knowledge of the timing of primary feather development is essential, and reduction of dietary protein and growth rate around this time will affect both incidence and severity of angel wing, as the author has observed in captive great bustards.

BEHAVIORAL DEVELOPMENT

Advanced senses (especially vision) in the hatchling precocial bird enable early imprinting on parents and/or conspecifics, enabling them to follow their parents to food sources. A consequence of this is the potential for inappropriate imprinting on rearers (or other species) that may have long-term implications, especially for birds to be released into the wild. To prevent this, "dehumanizing" suits and reduced handling have been frequently used in a range of projects involving crane species and bustards (Figure 45.3; see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 23, Conservation of Avian Species).[8,13]

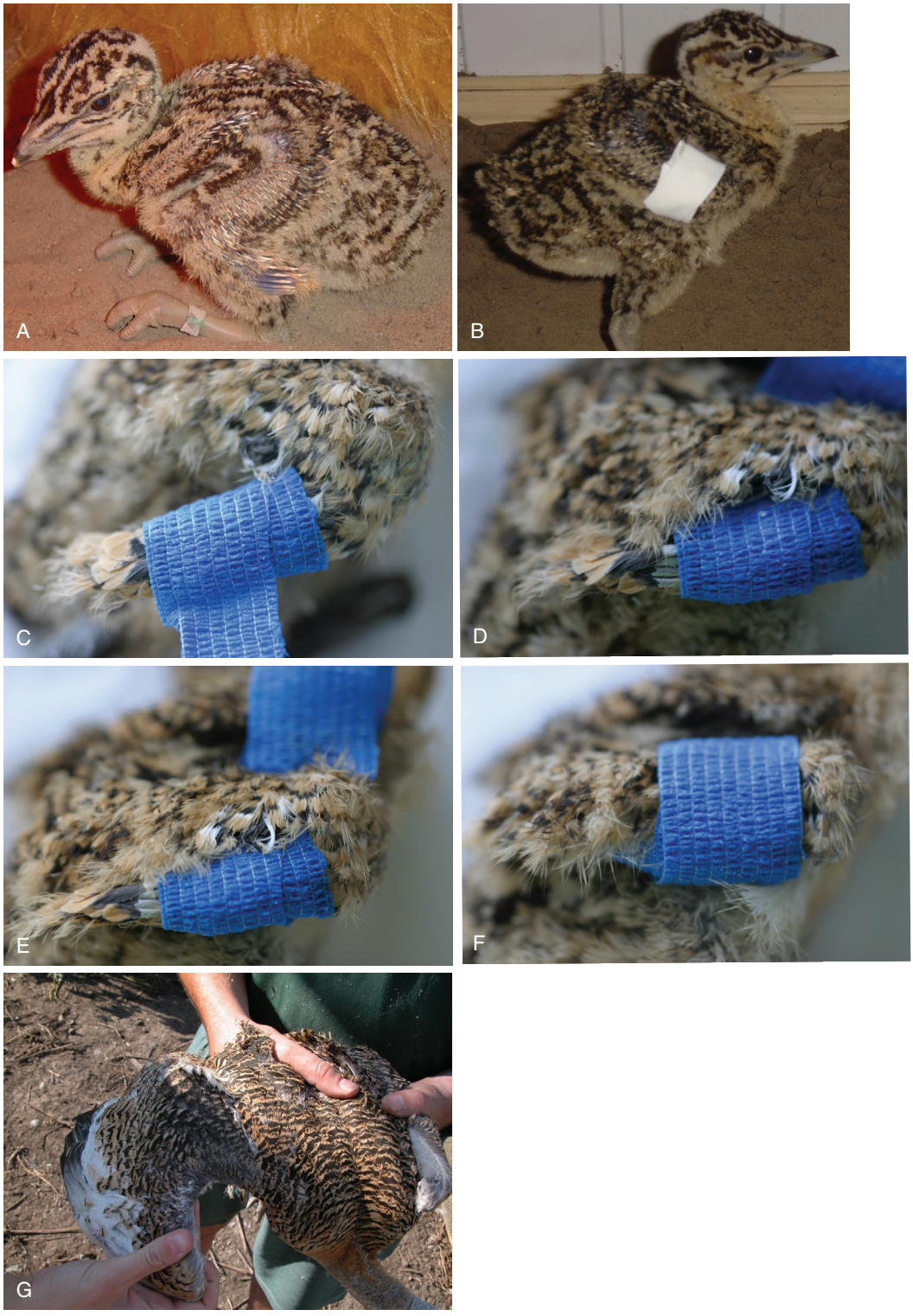


FIGURE 45.1 Excessive protein feeding in the first weeks can result in excessive development of primary feathers. Associated with poorly calcified long bones, "angel wing" can develop. Initially this appears as dropping of the distal wing (A) but will then progress to rotation of the distal wing and inability to fly. In the early stages of angel wing, a simple tape strip will suffice to hold up the distal wing and prevent rotation (B). In older chicks a heavier bandage is required. Images (C)–(F) show how the feathers are bound and the distal wing rotated medially. These bandages should be removed every 3 to 4 days until the wing is no longer dropped. When the bandage is removed, physiotherapy should be performed on the joints to prevent fibrosis of the joints and restriction of movement. Image (G) shows a great bustard *(Otis tarda)* that has been bandaged for several weeks resulting in joint fibrosis. The wing is being held at maximum extension. (Photos courtesy John Chitty.)

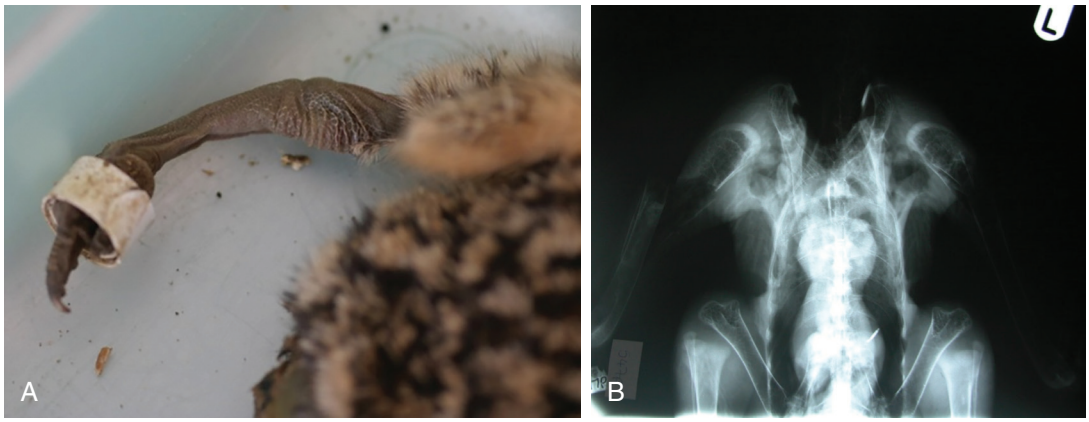


FIGURE 45.2 (A) Precocial species are generally fast-growing post-hatch compared to altricial species. Imbalances in dietary protein, calcium, phosphate, and ultraviolet/vitamin D_3 provision can result in soft, easily deformed long bones as seen in this great bustard *(Otis tarda)* chick at postmortem examination. (B) In juvenile birds this may be seen as reduced bone density and accompanying pathological fractures. This great bustard shows bilateral humeral fractures sustained during handling and associated with poor-density bones with very narrow cortices. (Photos courtesy John Chitty.)



FIGURE 45.3 Social imprinting on handlers must be avoided when rearing precocial birds if they are destined for wild release. Images (A–C) show the use of dehumanizing suits in rearing great bustard *(Otis tarda)* chicks—both for feeding and to act as attractants to stimulate normal social group following behavior and exercise. Frequent moderate exercise has been shown to be extremely beneficial in long bone growth.

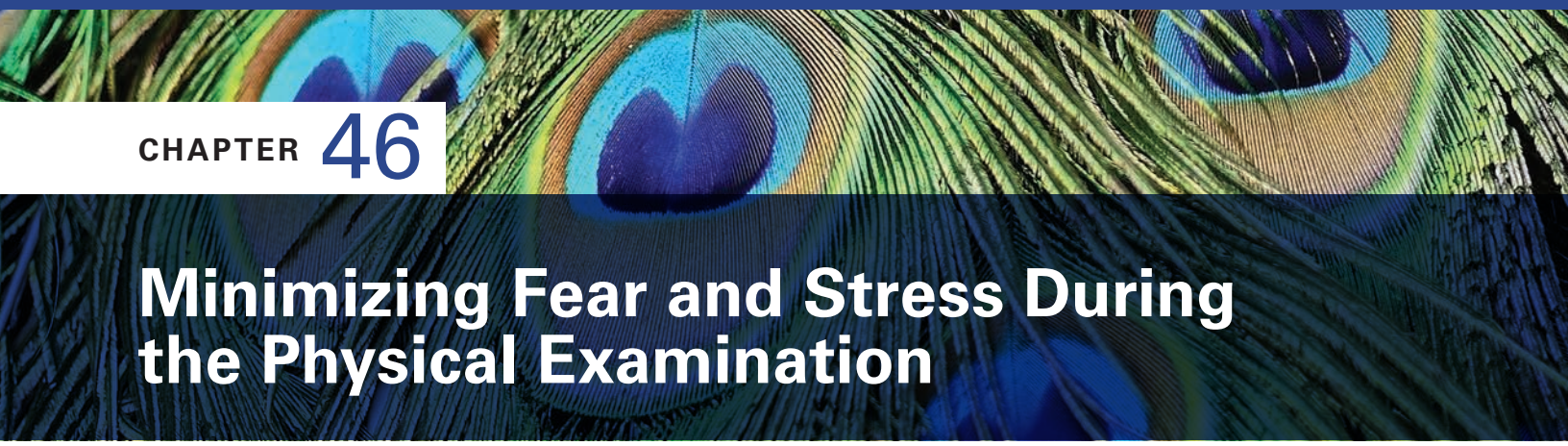
SUMMARY

While the comparative lack of "parental care" required of bird rearers may imply that precocial species have lesser needs, the converse may be true as precocial species are seemingly very sensitive to inadequacies or imbalances in the diet and exercise regimes. Behavioral needs in terms of parental imprinting may also be hard to replicate in rearing. Another problem relates to the increased difficulty of observing the more mobile precocial species in the wild, which hampers transfer of data to the captive rearer.

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CHAPTER 46

Minimizing Fear and Stress During the Physical Examination

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INTRODUCTION

Veterinary practitioners who see dogs and cats (as well as other species) have begun to embrace less stressful methods for administering veterinary examinations and treatment. This tends to result in a higher standard of care and can lead to increased compliance with medical recommendations due to improved client and patient experiences. Multiple resources exist for these practitioners (www.fearfreepets.com; https://cattledog-publishing.com; https://karenpryoracademy.com/courses/better-vet; https://www.fenzidogsportsacademy.com; etc.). Many practices specifically advertise their focus on behavioral wellness to let potential clients know their approach.

Clients are often particularly worried about the stress that their birds experience during veterinary visits, which results in many birds not being brought in for medical evaluation until the late stages of illness. By embracing the goal of minimizing stress during veterinary visits, avian medicine practitioners can improve the quality of life and longevity of their patients. Behavioral health is a crucial component of patient care that is often overlooked by busy or untrained veterinarians due to perceived time constraints or lack of knowledge.[1–3]

Avian behavior is poorly understood by most pet owners, and captive husbandry leads to a variety of issues that affect both the physiological and psychological health of birds. In fact, behavioral problems are a common reason for psittacine birds to be rehomed.[4] While succesful resolution of behavior problems often requires intervention by a knowledgeable veterinarian or behaviorist, most bird owners rather seek behavioral advice from other sources before talking to their veterinarian.[5] Many clients are likely to turn to social media where an explosion of behavior information is available, most of which is not evidence-based and can be harmful. Clients must therefore be encouraged to turn to their veterinarian as a trusted resource of information. Veterinarians can achieve this by partnering with clients to reduce stress during visits and/or providing resources to address training and behavior challenges, in addition to medical problems.

PHYSIOLOGICAL AND BEHAVIORAL IMPACT OF FEAR, ANXIETY, AND STRESS

A stress response is defined as the set of physiological and behavioral changes that occur in response to a stressor, which can be any stimulus or environmental condition that is an actual or perceived threat causing emotional or physical trauma to the animal. The goal of a stress response is to return the body to a normal baseline. Stress responses vary in intensity and may escalate quickly depending on the stressor involved. The "fight-or-flight response," which is evoked by extreme stress, creates a hormonal and chemical cascade within the body that is supposed to increase the animal's chances of surviving the aversive situation. The animal may choose to fight or flee the stressor depending on the surrounding environment, the type of stressor, and the animal's learning history.[6] Another behavior that may be seen in response to stress is the "freeze response," where the animal freezes in place to escape notice of the perceived danger.[7] Stress responses are normal but can become problematic when they are chronic in nature or if they lead to long-term changes in the animal's emotional state.

The stress response consists of two phases: the immediate response and the delayed response. The immediate response is mediated by the sympathetic nervous system and triggers epinephrine (adrenaline) and norepinephrine (noradrenaline) release from the adrenal medulla, leading to the fight-or-flight response. In contrast, the delayed response is initiated by corticosterone secretion, which originates from the adrenal cortex.[8] The amygdala initiates hormone secretion of corticotropin-releasing hormone (CRH) from the

hypothalamus in the presence of a perceived threat, which stimulates release of adrenocorticotropin-releasing hormone (ACTH) from the anterior pituitary, triggering release of glucocorticoids in the adrenal cortex. This may stimulate either a fearful or an aggressive response. The amygdala may stimulate release of additional hormones (noradrenaline, glutamate, gamma amino butyric acid [GABA], serotonin, etc.) to trigger the fight-or-flight response, a defensive state that prepares the organism to either escape or confront the threat. This hormone cascade is regulated by the hypothalamus.[9–15]

The general impact on the body in response to this hormonal cascade includes blood flow diversion toward major muscle groups and to the liver, increased cardiac output with increased arterial pressure, increased glucose release, and increased oxygen perfusion. All of these factors prepare the body to escape the (actual or perceived) threat. Some of the potential negative sequelae to fear include selective vasoconstriction to areas not critical for movement (which may lead to cardiovascular accidents or organ failure); changes in growth, reproductive function, and pain perception; altered memory, sensory, digestive, and immune function; creation of inflammatory responses in both the digestive tract and the skin; and even structural and functional brain damage. The physiological stress response impacts all body systems, and the immediate phase can occur within minutes of the inciting stimulus.[9,14,15] The immediate stress response causes heart and respiratory rates to increase, diverts blood to the muscles, and increases blood glucose. The animal may have decreased pain perception to facilitate escape from the stressor or alternatively may have increased pain perception (based on human studies).[16]

Stress affects both the ability to perform previously learned tasks and memory consolidation.[16–21] The amygdala stimulates creation and storage of fear memories in the medial prefrontal cortex. These memories are consolidated more easily when there is a strong emotion associated with the event.[18] This allows the memory to be quickly recalled when the animal is faced with similar environmental stimuli in the future. Fear in response to something painful or disturbing can sensitize an animal into a conditioned response to experience more fear when exposed to similar stimuli later on.[22] Because fear can have such a powerful long-term impact, it can be difficult to overcome the behavioral effects of even one exposure to a perceived aversive stimulus (such as a visit to a veterinary practice).

Some of the physiological responses of the body may be visible in lab work. A typical acute stress response results in a leukocytosis characterized by a mature neutrophilia/heterophilia, lymphopenia, and eosinopenia, along with hyperglycemia, although this varies with the individual. These changes are usually short lived. The delayed stress response is associated with a more chronic stress to the body, such as chronic illness, and repeated or prolonged stressful events. Effects include immunosuppression, delayed wound healing, muscle wasting, and development of behavioral disorders. Permanent brain damage or life-threatening illness associated with immunosuppression may result. Many species of birds are particularly prone to stress-induced illness. Aspergillosis secondary to immunosuppression is a common concern for captive waterfowl, penguins, and birds of prey.[15,23–28]

Fear and anxiety are emotional responses that often occur simultaneously. Fear results from the presence of a specific, known, and observable threat from which distance is desired, whereas anxiety results from the anticipation of a potential threat, which may be poorly defined or unknown and more oriented toward the future. Anxiety also results when the task requirements exceed the learner's competency. In other words, the animal is unable or unprepared to cope with the situation. It does not have behaviors in its repertoire it can use to address the current conditions.[29,30] A fear response can be triggered automatically in response to a known stressor. Animals that are afraid may not be able or willing to respond to known behavior cues and may behave differently than they would normally. Fear and anxiety can create vivid sensory memories of the circumstances (environment, people, animals, and inanimate objects) surrounding the stressor. Because these memories can be easily recalled when in similar settings, many animals begin experiencing anxiety and fear even when exposed to a milder version of the previous stressor in the future.[22]

ORIGIN AND IMPACT OF FEAR IN THE VETERINARY PRACTICE

Veterinary visits may cause fear, anxiety, and stress for a variety of reasons. Patients are subjected to an unfamiliar environment with their owners (who are typically worried/stressed) and experience uncomfortable or novel stimuli. Many birds are sensitive to their owner's emotional behavior, which can exacerbate their own stress reaction. The veterinary team can have a profound impact on these factors through carefully chosen body language and environmental management. Stress may lead to death in some avian patients, likely secondary to undiagnosed heart disease and/or an extreme fear response leading to a massive catecholamine release.

Body language reflecting fear may remain unrecognized by veterinarians and staff. Fear may be viewed as a normal part of the veterinary visit and tolerated due to time constraints and lack of knowledge of methods to mitigate it. Some birds may be perceived by owners and/or vets as "calm" and "resigned to their fate," when they are actually terrified and show a freeze response rather than attempt to escape, flee, or fight to protect themselves. Many of these patients have experienced or are experiencing "flooding." Flooding, as defined in behavior science literature, is a form of behavior therapy used in humans to treat phobias and posttraumatic stress disorder.[31–34] It involves sudden, full-intensity exposure to the aversive stimulus without means of escape until the patient no longer resists. Flooding triggers the physiological and psychological effects of the "fight-or-flight" response, with the goal of quickly habituating the patient to the aversive stimulus. While it may be effective in some situations, it is easy to misapply (especially to animals, who cannot consent to the technique). The potential behavioral impacts of flooding may include increased sensitization to the aversive stimulus, increased aggression, and learned helplessness. Learned helplessness is observed in both animals and humans who have been conditioned to expect pain, suffering, or discomfort without a means of escape. Eventually, the patient will stop trying to avoid the undesired experience, even if escape is possible. The resultant behavior may be misinterpreted as a

willing compliance with restraint, as the patient is anticipating a frightening procedure and is showing a lack of autonomy and empowerment to actively respond. Learned helplessness can lead to immunosuppression and depression.[35] Preferred alternatives to flooding are systematic desensitization, counterconditioning, and operant conditioning, which have been discussed in *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior, and will also be discussed in more detail later in this chapter.

The patient's stress decreases the quality of veterinary medicine that is possible. Repercussions include a requirement for sedation or anesthesia to accomplish procedures, problems with healing due to immunosuppression, injuries to team members or the patient, decreased staff morale, poor compliance on the owner's part, and loss of revenue for the hospital. Providing care in the least stressful manner possible empowers patients and everyone involved with their care, resulting in an improved quality of life.

Client perceptions are equally detrimental to patient care and practice revenue. Those who perceive rough handling of their birds or other unpleasant experiences during a visit are less likely to follow through with recommendations for treatment, progress examinations, and preventative care. They are also less likely to return for appropriate follow-up examinations or seek care in the future, which results in delayed diagnosis of health concerns and even death. Stress associated with forcefully administering medications to birds may lead to physiological side effects for the patient, as well as a loss of the human-animal bond and reduced quality of life for both the pet and the client. In one study,[36] 26% of dog owners and 38% of cat owners said that their pets hate going to the vet. This percentage is likely significantly higher for bird owners.

BENEFITS OF A LOW-STRESS VETERINARY EXPERIENCE

Veterinary teams can ensure increased client compliance with medical recommendations, improved staff morale, and more enjoyable patient interactions if they provide medical care in an environment that reduces patient and client stress to the greatest degree possible. Some of the benefits of sensitive handling include improved patient care, increased accuracy in physical examination findings, and reduced injuries both to veterinary staff and patients.[37] This approach also leads to improvements in client trust and retention, which increases referrals and hospital revenue.[38]

The goal of the veterinary visit should be to provide a minimally stressful experience while laying the groundwork for improved future experiences. Sensitivity to avian behavior and body language, combined with skillful application of operant conditioning or systematic desensitization and counterconditioning, can often result in minimal restraint examinations. Depending on the patient's stress threshold, sedation may be a better choice compared to restraint.

CHALLENGES ASSOCIATED WITH PROVIDING LOW-STRESS AVIAN MEDICINE

There are some additional challenges associated with providing minimal stress veterinary visits for birds compared to dogs and cats. Awareness of these issues allows veterinary staff to accommodate and circumvent them.

While most clients will be very happy to see an increased focus on behavioral wellness for birds, some may have reservations regarding this change in approach. The need to slow down the speed of appointments may be seen as an unnecessary waste of time. Recommendations to use sedation to reduce fear may be viewed as an unacceptable risk, as clients may confuse conscious sedation with brief anesthesia and not fully understand why this is preferable. Respectfully and thoroughly explaining the benefits of the changed approach can make a huge difference in the client's willingness to trust and cooperate.

Many birds are not brought into veterinary practices until they are in advanced stages of illness. Reasons for this include both the avian tendency to hide clinical signs of illness until decompensation occurs and a lack of understanding on the client's part regarding the value of preventative care. Thus, many birds have their first veterinary visit after several decades of life. Even if birds are not visibly displaying signs of illness, they often have underlying disease present. Most birds presenting to a veterinary practice need to have some form of diagnostic testing performed because of these factors, and every patient should be treated as if they may have a serious health problem. This reality limits the usefulness of some strategies recommended in Fear Free™ dog and cat medicine, including postponing sample collection in favor of prescribing anti-anxiety medications that can be administered at home or waiting until a behavior has been trained using positive reinforcement.

Many birds display neophobic responses, defined as a fear of new things. This survival mechanism helps them to avoid attack from predators or being poisoned by an unknown, toxic food item. This response makes it difficult for birds to tolerate veterinary visits, which involve many unfamiliar stimuli, including tools and other items from which escape is often prevented. The use of operant conditioning or systematic desensitization and counterconditioning can teach birds that these aspects of the visit can be experienced without undue stress (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). However, it can be challenging to overcome a neophobic response within the short amount of time allotted for a typical veterinary visit.

Many clients do not provide their companion parrots with intentional behavior guidance or training. Thus, their birds have no previous experience responding to cues or receiving reinforcement for desired behavior. The lack of familiarity with this process may retard initial efforts during a first vet visit. In addition, these untrained parrots lack the resilience that birds who receive regular training possess. Many of these birds are also poorly socialized to new people and environments.

Reading bird body language is an involved and complex skill that is not commonly taught in veterinary curricula. As a result, it is common for veterinarians and veterinary staff to misinterpret or disregard signals presented by avian patients. In addition, many of these patients can fly, which provides a uniquely difficult challenge when it comes to providing force-free care and keeping the patient, client, and staff safe. Finally, many of these patients have strong memories associated with previous aversive experiences at veterinary offices, which continue to influence their behaviors for years later.

Despite these factors, providing minimally stressful avian veterinary visits is absolutely possible and should be considered standard of care. The best way to accomplish this is through adequate education (both of staff and clients), appointment preparation, and thorough understanding of both avian body language and the science of behavior change.

NECESSARY SKILLS TO MASTER

Veterinarians must develop a variety of skills in order to accomplish low-stress veterinary visits, including knowledge, confidence, technical competence, compassion, client communication, patience, and flexible case management.

Knowledge is the most important of these skills. A knowledge of the behavior and body language of parrots will lead to accurate recognition of the stress in the patient (see Figure 46.1 and Box 46.1 for examples) and allows for behavior questions to be answered for the client. An excellent resource for helping clinicians to recognize signs of fear and stress in multiple species of birds can be found in the Avian Fear Free™ Online Modules. Up-to-date medical knowledge will permit the problem to be handled in the most efficient manner, eliminating excessive trial and error. Clients will feel more confident in a veterinarian who can identify the species of their bird on sight and can easily communicate their medical and behavioral recommendations.



FIGURE 46.1 Moluccan cockatoo *(Cacatua moluccensis)* displaying signs of arousal, including a tall and erect body posture, open beak with vocalization, and feathers slicked to body but erect around the head and crest. Recognition of these initial signs of arousal is important to prevent further escalation of the behavior.

Clinicians who are confident in their ability to practice high-quality avian medicine will be less stressed and elicit less stress from the staff, clients, and patients. Confident clinicians are better able to handle birds safely, assess the medical issues at hand, and address the concerns of the client. However, confidence must be gained honestly through time and repetition; false confidence when interacting with avian patients can increase the risk of injuries to patient and veterinary staff.

Handling birds safely is a mechanical skill that should be actively developed, both in a clinical setting and in a social interaction setting. Confidence in technical skills can be gained by attending wet labs, practicing techniques on models or cadavers, and finally by applying techniques to live birds. Skilled bird handlers improve the examination experience for the client, veterinarian, and patient.

Compassion is an important part of ethical practice of veterinary medicine. It is easy to be compassionate toward a tame, talking parrot; it is harder to have the same compassion for a terrified bird that is biting and screaming. It can also be difficult to have compassion for some clients when there are significant barriers to productive communication, including values mismatches, lack of understanding, and personality conflicts. Until the clinician can overcome these challenges, they will not be successful at practicing low-stress avian medicine.

The combination of knowledge, confidence, and compassion allows the practitioner to develop a balanced practice philosophy. Aggressive diagnostic and therapeutic measures are necessary at times, while a conservative approach may be more appropriate in other situations. The client should be made a part of this decision process. Both sides of issues should be presented, including risks, costs, and all other relevant factors. This requires excellent client communication to avoid misunderstandings and set appropriate expectations.

Patience is also essential to this process. Clients may take a while to make decisions. Patients may take some time to calm down before another step can be taken. Rushing through any phase can increase errors and worsen patient stress. All the above requires adequate time to be set aside in the appointment schedule and a commitment to patience on the part of the veterinary staff.

Even after a plan has been approved and instituted, clinicians must be willing to alter their plan based on the changing needs of the patient. Patient stress levels, worsening clinical conditions, and availability of equipment are all factors that may require adjustments to a treatment plan. A competent clinician will monitor these and make changes as necessary.

BEHAVIOR DEFINITIONS AND TECHNIQUES

Veterinarians are presented with a difficult challenge of attending to the immediate medical needs of the animal, while trying to minimize impact on behavior and maintaining the human-animal bond. It is helpful to understand some basic principles and strategies that influence behavior, in particular ones that can facilitate overcoming fear responses (Table 46.1).

Habituation occurs when exposing an animal to stimuli without regard to how the stimulus influences behavior. Upon repeated exposure to a (mild aversive) stimulus, a decrease in response may be seen as a naturally occurring phenomenon.

BOX 46.1 EXAMPLES OF SIGNS THAT CAN POTENTIALLY INDICATE STRESS, ANXIETY, AND/OR FEAR IN BIRDS[a]

Facial expressions

- Eye pinning or flashing (particularly in psittacine birds)
- Wide open eyes
- Slight opening of the beak
- Gaping mouth (beak is wide open for an extended period, in birds of prey)
- Raised head or crest feathers

Note that birds have minimal muscles that can contribute to facial expressions, hence providing limited options to recognize stress, fear, or anxiety from these.

Postural changes

- Freezing, tensing up with feathers tight against the body
- Standing tall, elongating the body
- Wings spread and held away from the body
- Fanned tail
- Leaning away from the feared person or object
- Withdrawal in a corner
- Lifting foot toward the feared person or object (psittacine birds)
- Firming of the grip with the feet (in birds of prey)
- Crouching or cowering

Behavioral changes

- Absence or cessation of comfort behaviors, e.g., preening, social interaction, eating
- Darting looks across the examination room to try and escape or fly away
- Vision oriented toward the perceived threat, following its every movement
- Increased alertness to stimuli
- Chewing on the leg band, feet, or toes (psittacine birds, in particular grey parrots [*Psittacus erithacus*])
- Feather plucking
- Shivering, shaking, trembling
- Headshaking, pacing, rocking back and forth, or other stereotypic/repetitive behaviors
- Quivering or flapping of the wings
- Hitting or beating with the wings in an attempt to escape or avoid capture
- Kicking with the legs (particularly in backyard poultry and waterfowl)
- Struggling profusely (when restrained)
- Moving, leaping, or flying away from the feared person or object
- Flying around frantically in the cage (particularly in small birds)
- Thrashing or biting on cage bars
- Rolling onto the back (usually last defense mechanism in psittacine birds)
- Backward flip, falling off the perch or hand/glove (birds of prey, psittacine birds)
- Bating (jumping of the perch to the length of the leash, in birds of prey)
- Biting (often quick, short nip) or lunging toward the feared person or object (usually in situations when there is no more option to escape)

Physiological changes

- Increased respiratory rate, panting, breathing with open beak (e.g., poultry, birds of prey)
- Gular flutter (feathers at throat flutter in and out, e.g., in owls)
- Increased heart rate, tachycardia
- Hyperthermia
- Bleeding from the eyes (grey parrots) or nares
- Copious feather loss (e.g., pigeons, turacos)
- Seizure activity (e.g., grey parrots, cockatiels)
- Collapse or sudden death due to cardiac arrest (in extremely stressed birds, particularly passerine species)
- Elimination of feces and urine; loose, watery droppings

Changes in vocalization

- Decreased vocalizations, extreme silence
- Excessive vocalization, e.g., screaming, screeching, squeaking, squawking
- Repetitive chirps or alarm calls, e.g., high-pitched "twittering" in Harris' hawks *[Parabuteo unicinctus]*, or "kek-kek-kek" sounds in accipiters
- Growling or hissing
- Snorting and wheezing (Pionus parrots [*Pionus* spp.])
- Clacking with the beak (particularly in owls)

[a]Note that body language and behavior differ across bird species. In addition, different behavioral intentions may underlie the same behavior. Hence, when interpreting the behavior of a bird, the species-typical patterns and context should be kept in mind.

However, when the fear response to the stimulus is too strong, habituation cannot be relied on to change behavior, as it is ineffective at overcoming the fear response. For example, a parrot may never set foot on a new perch placed in the habitat through the process of habituation alone.

Systematic desensitization is the process of exposing the animal to a stimulus in a graduated manner, so that it does not evoke a substantial fear response. This requires close and thorough observation and understanding of body language. The observation of the slightest body language indicative of a fear response is information that the bird has reached its threshold and that the item evoking a fear response must be placed at a greater distance from the patient. A very observant practitioner can use this approach to slowly introduce a towel to a bird that is showing a fear response to that object. This can provide a more optimal option for introducing a towel for restraint compared to immediately capturing the bird with the towel (which will also lead to an increased fear response to the towel on future occasions; see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).

Systematic desensitization is even more effective when used in conjunction with the association of desired items or experiences such as preferred food items, desired touch, toys, and so on. This process is known as counter conditioning. The intention is to pair desired experiences and items (preferably in conjunction with systematic desensitization) with the introduction of the stimulus. Counter conditioning is used to change the perception of an experience or situation from aversive to appetitive and can allow practitioners to progress rapidly through steps of introducing novel items such as dosing syringes, stethoscopes, nail clippers, and other medical instruments. Again, sensitivity to bird body language is critical. There is a common tendency to push for the result as opposed

TABLE 46.1
Behavior Definitions[39–44]

Term	Definition	Key Points
Positive reinforcement	A learning process in which the presentation of a stimulus following a behavior increases the probability or likelihood of that response.	For example, delivering preferred food items to a patient after stepping onto a scale.
Negative reinforcement	A learning process in which the removal of a stimulus following a behavior increases the probability or likelihood of that response.	For example, removing pressure when the bird relaxes during restraint to increase presentation of calm behavior.
Positive punishment	A learning process in which the presentation of a stimulus following a behavior decreases the probability or likelihood of that response.	For example, a "wobble correction" (destabilizing a perching surface) when a parrot attempts to bite to decrease biting behaviors.
Negative punishment	A learning process in which the removal of a stimulus following a behavior decreases the probability or likelihood of that response.	For example, removing fingers from the mouth of a young parrot when gentle mouthing becomes too painful for human fingers.
Reinforcer	The stimulus (aka consequence) that increases or maintains the behavior.	For example, a desired food item. Only becomes a reinforcer if behavior change occurs; otherwise, it is a "potential reinforcer."
Threshold	The point at which conditions become so aversive that a bird is unable to exhibit normal behaviors and responses.	See "Under threshold" and "Over threshold."
Under threshold	The bird is experiencing minimal stress and showing normal behaviors.	This is the goal throughout the veterinary examination.
Over threshold	Following a significant stressor, behaviors observed may include escape, avoidance, increased rate of respiration, and no receptivity to food or other appetitive.	Time is required to recover from this level of stress. Anxiolytic medication may be required short term. Training is necessary to achieve long-term goals of stress-free medical care.
Habituation	Decreased response to a stimulus following repeated exposure to the stimulus. If this is done without regard to how it influences behavior, it is not always successful.	Always occurring, but not usually successful if the fear response is too strong. Flooding is an extreme version of intentional habituation.
Systematic desensitization	Exposing the bird to a stimulus in a graduated manner so as not to evoke a substantial fear response.	Can work alone with good sensitivity to body language. Works better when paired with counterconditioning.
Counterconditioning	Pairing a desired object, experience, or stimulus with a previous experience or situation to change the perception from aversive to appetitive (or vice versa).	For example, a bird can be taught to engage with towels instead of fearing them by pairing towels with desired food items.
Flooding	Exposing the bird to what it fears all at once at the fullest intensity until it no longer resists.	Flooded patients appear to be compliant, but the flooding process is stress inducing for the patient. Not recommended.
Sensitization	The process of becoming highly sensitive to specific events or situations.	May be triggered by unskilled application of systematic desensitization.
Stimulus fading	Gradually introducing or removing a stimulus that may evoke a desired or undesired response.	When done skillfully, this can be used to introduce items such as towels and medical instruments without evoking fear responses.

to focus on the process, which can result in sensitization. The patient will then react with a fear response to stimuli associated with the procedure earlier in the process.

For those individuals who are receptive, positive reinforcement can be used to train voluntary cooperation in behaviors that facilitate examinations such as stepping onto a scale, accepting oral medications from a syringe (Figure 46.2), allowing wrapping in a towel, loading into a transport container, stepping onto strangers, and more. This is the preferred approach when possible. This involves shaping behavior with successive approximations and reinforcing each step with desired consequences (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). Ideally, clients should be encouraged to train their birds at home in advance and generalize those behaviors to many locations. Since many clients do not train or have the skillsets to train, it is important to demonstrate and provide proper guidance and resources for clients to achieve the goals that are set and/or refer them to qualified behavior consultants or animal trainers to help during this process. However, even if prior training has not occurred, some patients are receptive to learning at the veterinary practice, either during an appointment or while hospitalized or boarding (Figure 46.3). Take advantage of these opportunities when possible.



FIGURE 46.2 Many birds, in particular parrots, can readily be trained to participate in medical procedures using shaping and positive reinforcement. This brown-headed parrot *(Poicephalus cryptoxanthus)*, for example, has been trained to voluntarily accept medication from a syringe.



FIGURE 46.3 Training for medical behaviors can be implemented while the bird is hospitalized. Initially, training can be done through the bars of the enclosure to reduce the risk of escape or exhibition of aggressive behavior, as performed here with the white-bellied caique *(Pionites leukogaster)*.

Other behavior modification techniques involve the use of negative reinforcement, negative punishment, positive reinforcement, and positive punishment (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). Each of these techniques has the potential to achieve a desired result. However, there are many undesirable consequences that can occur, particularly associated with positive punishment. The use of positive punishment is not recommended in a veterinary setting.

PREPARING FOR THE LOW-STRESS VETERINARY VISIT

Once the tools for good "perch-side" manner have been developed, the next step is to develop a comprehensive strategy for minimizing the stress of the entire veterinary experience.

Scheduling Guidelines

Realistic scheduling guidelines are the first step in achieving our goal of stress-free visits. Appointment times should be more generous than those used for dogs and cats, since these patients often take more time. Overbooking often forces veterinarians to rush through the visit. In these situations, client education and the necessary, patient-centered approach to the examination are often curtailed in the interest of time (see Chapter 1 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 24, Practice Management).

Since many ill birds are much sicker than the client originally understood when making the call, some flexibility in the schedule will be necessary. A best practice is to include in each day's schedule a few emergency slots for critical same-day appointments. Sick birds cannot wait to be seen.

Generally, appointments are best scheduled early in the day, specifically in the situation where conscious sedation may be needed to accomplish the visit goals. Moreover, sick birds may require hospitalization, which can allow more to be accomplished following admission when the client comes in early as opposed to at the end of the day.

For routine appointments, the client service representative (CSR) must schedule skillfully to attain the goals of a low-stress appointment. The arrival of the client should not coincide with that of especially loud patients or during exceptionally busy times, such as the mornings of surgery days. Some thought should be given also to matching patient needs with the talents of specific technicians or assistants. For example, certain team members may be more skilled at client education or with handling large macaws. If conscious sedation is anticipated based on previous visits, access to oxygen anesthesia equipment should be given consideration as well.

This type of scheduling may limit the number of patients that can be handled each day. Fees may need to be adjusted accordingly (see Chapter 1).

Team Member Roles

It is vital that all team members be fully committed to the goal of a minimally stressful examination and fully trained in their roles. They must deliver to the client a consistent message about the value of this approach. Having a handout that describes the goals of the visit and standard practices, which can be available online or given to the client at check-in, may help achieve greater understanding and buy-in. Readers are referred to Fear Free™ website (https://fearfreepets.com/), which has resources and handouts available for hospitals to provide to clients. Team members must also work together closely to meet the needs of individual patients, practicing flexibility in scheduling when needed.

Adopting low-stress practices practice-wide can be difficult, especially given the different backgrounds and experiences that team members may have. Since the success of the

visit is dependent upon so many factors and the cooperation of so many different team members, it is best to have for reference a printed resource of job descriptions and hospital protocols, as well as client instructions and scripts for scheduling appointments.

The CSR is the first team member to convey appropriate expectations to the new client regarding the minimal stress approach and is responsible for facilitating the client experience from start to finish. When scheduling appointments, the CSR must describe the practical details for a typical appointment, gather pertinent client/patient information, and provide initial coaching for transporting the patient to the practice in a stress-free manner. If there is any question about meeting any special needs that the client or patient might have, the CSR will also consult with appropriate team members to decide upon the best scheduling options. The CSR will also make sure that the process of entering and exiting the building is as smooth as possible.

The role of the technician or assistant is also multifocal. Any technicians and assistants who will be handling avian appointments must maintain communication with receptionists and veterinarians to streamline visit flow and optimize patient care. Additionally, a large part of the technician's role is that of client education, including discussions about training challenges and behavior modification.

The technician is responsible for restraining patients, administering anesthesia (if indicated), and performing procedures/treatments in a low-stress manner. They are also responsible for providing care for hospitalized patients and being a patient advocate by recognizing and addressing patient distress. Technicians may also be responsible for providing training to patients while they are hospitalized or during office visits. They may be expected to help clients develop and implement training plans to cover both cooperative medical care and participation in normal husbandry.

The veterinarian plays an overarching role in establishing the practice as one that has committed to low-stress medicine. The practice needs a strong mission statement by which team members can measure the approach they intend to take with a patient or client. The veterinarian must lead by example, providing to their staff a clear duplicatable image of what low-stress medicine looks like when interacting with patients. This will indicate the vet's own commitment, which sets the stage for the veterinarian to demand 100% commitment to the force-free approach among other team members.

The veterinarian is responsible for educating both team members and clients about the body language signals that indicate unacceptable levels of stress in a patient. The veterinarian also conveys information about both the harmful physical and behavioral ramifications of stress. The veterinarian must also be instrumental in developing an in-house training plan so that all team members have the opportunity to learn the skills necessary for a low-stress examination and client interaction. Hands-on training with live birds will be necessary for this. Options may include using birds owned by staff members, those currently in sanctuary or fostering situations, or even birds owned by consenting clients. Technicians and assistants should not be allowed to participate in restraint or care until they have demonstrated proficiency in reading body language and basic handling skills.

During the examination itself, the veterinarian has an opportunity to communicate value to the client regarding the force-free approach and to discuss specific appointment details. The veterinarian then manages the flow of the appointment and is responsible for making medical recommendations.

The veterinarian then performs an examination, making every effort to avoid eliciting stress in the patient. The vet will evaluate the need for sedation and, if necessary, develop a sedation or anesthetic protocol. The vet will also have a driving role in establishing a plan for the best system for collecting diagnostic samples that are indicated and achieving other diagnostic processes in order to minimize stress for the patient.

At the end of the examination, the veterinarian will help the client to develop an ongoing plan for behavioral wellness, including the training of any behaviors that will make future examinations less stressful. A last step will then be to enter into the patient record information relating to the degree of stress observed for the patient during that visit, what was done to mitigate this, and a plan for future examinations.

Team Member Training

Ongoing training for each team member must be a priority and is best conducted in practice during staff meetings. Establishing the practice of mandatory regular staff meetings is important. Identify which time of the week is slowest for the practice and schedule a 1- to 2-hour meeting at this time. The benefits of the information sharing and training that take place during these meetings will far outweigh the disadvantages of being able to schedule every possible appointment slot.

The first part of the meeting can be dedicated to practice business according to a previously agreed-upon agenda. The bulk of the meeting can then be spent in smaller groups training team members using role play, hands-on training with healthy birds, or for any other skills necessary to accomplish the goal of a satisfactory visit. The most skilled team members can be chosen to help those needing to build skills. For this purpose, "Awesome Visit" role plays, which involve each level of team membership, can be exceptionally helpful.

In addition, team members should be encouraged to attend any applicable continuing education, including the Fear Free™ Avian Modules. Continuing education should be paid for by the practice and wages paid for attendance, which helps team members to understand the importance of their roles. The individual should be expected to return and provide a summary of their learning to the entire team during a subsequent meeting.

Each day, before appointments begin, the CSR, technicians, assistants, and veterinarians who will be handling appointments should meet to discuss the cases of the day. This affords them an opportunity to brainstorm any individualized approach that might benefit any special needs of clients or patients. A brief end-of-day meeting enables staff to evaluate their performance and look for opportunities for improved case management. These "all staff rounds" provide a valuable training opportunity and will optimize staff engagement.

When all team members are trained to the highest level, practices can truly be integrated to offer best practices from the start to finish of every appointment. A consistent staff mindset and clinical approach is critical to both effective client education and patient care. Team members who are

self-assured in discussing behavior and training and in handling birds will inspire client loyalty and confidence.

Scheduling the Appointment

When a client contacts the practice to set up an appointment for their bird to be examined, the CSR must first assess the possibility of the patient being ill. This is often the case, since routine preventative care visits are still relatively uncommon for companion parrots in many practice settings. If illness is suspected, the patient should ideally be examined the same day, since birds tend to hide symptoms of illness until they are in an advanced stage of disease. For these cases, the focus of the CSR should be on helping the client bring the bird into the hospital for assessment as soon as possible. While there are still many ways to minimize the stress of sick birds (see sections on Restraint of Poorly Socialized or Birds Showing Extreme Fear or Aggressive Behavior, and Conscious Sedation as Part of the Low-Stress Avian Examination), birds that are sick are unlikely to respond well to training procedures and postponing a visit until a bird is trained is not an option.

The approach to scheduling an appointment for a healthy bird is different. It is important for the CSR to educate the client regarding the goals for the visit and establish appropriate expectations. This includes the fact that the appointment may take more time than they are used to spending at a veterinary visit, and the examination itself may proceed more slowly to keep the patient's stress levels as low as possible. The client should receive instructions on getting the patient to the practice in as stress-free a manner as possible (emailed handouts are an excellent way to accomplish this efficiently; see Box 46.2), including which types of carriers are appropriate for the species. Birds should always be transported in an appropriate carrier (not free within a vehicle on a client's shoulder) for a variety of safety reasons. The CSR should also inform the client that the bird must remain in the carrier while being transported from the car to the practice, while waiting in the lobby, and while acclimating to the examination room (see

BOX 46.2 EXAMPLE OF A CLIENT HANDOUT WITH INSTRUCTIONS ON HOW TO MINIMIZE STRESS DURING TRANSPORTATION

Tips for transporting your bird to the veterinary practice

Transportation to the veterinary clinic may be a stressful event for your beloved bird. To maximize safety and minimize the amount of stress involved, we recommend you do the following:

- Make sure to use a comfortable, safe, and secure carrier.
  - A carrier should be appropriate to the size of your bird. For small birds, you may choose to transport them in their own cage if it fits in your vehicle, as the familiar surroundings will cause the least amount of stress. Alternatively, you can use a small cage or box that has sufficient air holes to provide adequate air flow and ventilation. For larger birds, a well-ventilated carrier with front opening is preferred. The opening must be large enough to facilitate entry and exit from the carrier, but not so large that the bird can easily escape.
  - To prevent escape during the ride, the enclosure should preferably be constructed of nondestructible materials (e.g., wire, acrylic, or hard plastic) and have a secure closure. For parrots that are known escape artists, the use of an extra lock is recommended! If you are using a cardboard box to transport a small bird, remember that they can chew their way out of the box, so be sure to always monitor them and leave them in the box for the least amount of time possible!
  - To ensure your bird sits comfortably during the ride, the carrier or cage should be fitted with an adequately sized perch that offers a sturdy, stable grip so that your bird can maintain its balance during the trip. If your bird is suffering from trauma, weakness, seizures, or other conditions that hinder its ability to perch, a flat-bottomed container or box without perches is more appropriate to use. All toys and swings should be removed to prevent injury to your bird. Food or treats can be provided during the trip (unless you have been advised otherwise) in a dish that is securely attached to the carrier or cage. Water dishes should be emptied to prevent spillage; during long trips, ice cubes or fresh foods that contain moisture can be provided as an alternative water source. Make sure that there is a towel, absorbent padding, or paper on the bottom of the enclosure as this will allow for collection of droppings that can provide helpful information during the visit.
  - **NEVER allow your bird to roam freely in the car while you are driving as this is neither safe for you nor for your bird!**
- Unless your bird is curious or accustomed to regular travel in an uncovered carrier or cage, it should be covered during the ride. This will help to reduce the level of stress (especially for nervous or fearful birds!) and car sickness. Once in the examination room, the cover can be removed to allow your bird to acclimatize to the new surroundings.
- Place the carrier or cage on the floor of your car and prevent it from sliding during transport. If placed on a seat, use a towel to keep the base horizontal and secure it safely with a car seat belt to prevent movement or shifting.
- Protect your bird from the elements! Be sure to consider the weather conditions during the travel. If it is cold or damp outside, prewarm your car and cover the cage or carrier using towels or blankets. When it is hot outside, be sure to provide adequate ventilation or fresh air and never leave your bird unattended in the car as you may risk overheating it.
- A carrier ideally should be a familiar space where your bird feels safe and comfortable. If possible, allow your bird to get acquainted with the carrier by placing it in clear view for at least 1 week prior to the visit. You can offer highly favored treats or toys in the carrier to motivate and train them to voluntarily step into the carrier. Alternatively, you can consider using the carrier as a "night cage" where your bird sleeps every night, so that entering and exiting the cage becomes a daily routine.
- Do not forget to bring items that can be of importance during the veterinary visit, such as your bird's food, medication, or favored toys. If your bird has ingested something that you suspect to be toxic, do not forget to bring these items either, or take pictures so that information can readily be retrieved.

sections on Hospital and Examination Room Preparation; The Lobby Experience; and The Low-Stress Examination).

CSRs should advise clients to bring preferred food items, small toys, familiar towels, and/or other items that can be used during the visit. In some cases, it may be appropriate to recommend withholding the bird's preferred food item for the week prior to the visit to increase receptiveness to this potential reinforcer. The CSR should also ask the client about any known aversive stimuli (previous experienced objects, colors, handling methods, etc.) that may result in increased patient distress. These aversive stimuli should be clearly flagged in the medical record so that the clinician and staff can adjust their plan accordingly.

Before the close of the conversation, the CSR should encourage the new client to complete the necessary paperwork in advance of the visit (online, via email, or by obtaining a hard copy directly from the hospital) and request that the client send it in (e.g., via email) photos (and/or video) of cage with surroundings, nighttime cage, play spaces, and so on. This will minimize time spent in the receiving area.

Technicians and veterinarians should provide support to the receptionist as needed during the scheduling process. This may take the shape of brainstorming the best appointment time (including fitting patients into the schedule on an emergency basis), or how best to meet the special needs of either the client or the patient. In some cases, these team members may need to have an additional phone conversation with clients to clarify details of the appointment or to consult on ways to get the bird into a carrier in a low-stress manner.

Offering wellness plans to clients with avian patients may help increase compliance with preventative health care. It is helpful to include additional office visits as part of the wellness package to be used as training sessions and habituate the bird to the practice environment and staff.

Hospital and Examination Room Preparation

The setup of the hospital and the examination rooms can contribute greatly to the success of the visit. A crowded waiting room is not a good place for a sick or nervous bird. Ideally, the patient should spend minimal time in the reception area and move straight into the examination room upon arrival.

If this is not possible, the bird should wait either in an avian-specific reception area or in the client's vehicle until the examination room is available, environmental conditions permitting. If neither of these options is acceptable or available, reception areas dedicated for cats are preferable to those used for dogs. Partially covering the carrier with a towel to decrease visibility may help decrease fear responses in some birds. Similarly, ensuring that the carrier can be placed higher up rather than on the ground can help to alleviate stress.

Examination rooms should be in a quiet area of the hospital, where the patient is not disturbed by the sound of slamming doors or the banging of wagging tails against a wall. Examination rooms with windows carry the risk of potential injury (if a bird flies into the window) and may be a source of aversive visual stimuli for some patients. There are various options available to make the window less of a risk (decals, shades/blinds, commercial window coatings, etc.) although their degree of efficacy may vary.

Preparation of the examination room prior to the visit is necessary. This should be done with the expectation that the entire examination will take place there in the presence of the client.

Patients can be disturbed by the opening and closing of drawers and the movement of equipment. All items that might be used during the visit (including materials for diagnostic sampling, grooming, etc.) should therefore be out and ready in their intended location for use prior to the entrance of the patient into the room. A laminated photo of the correct setup arrangement can be kept in a drawer to serve as a reference for newer team members. Once the room has been set up, team members should communicate with each other that it is "In Use" to avoid disrupting flow of the appointment.

If the client has brought their own scale or towels, these can be substituted as needed when the client enters the room and before the patient exits the carrier.

Included in the examination room setup should be the presence of potential food reinforcers (Tables 46.2 and 46.3). A variety of preferred food items should be available and selected based on the species of bird being examined (Figure 46.4). Another helpful reinforcer to have on hand for parrots is small foot toys; these can be as simple as a paper towel tied in a knot or some tongue depressors zip-tied together (Figures 46.5 and 45.6).

The Lobby Experience

The CSR should greet clients as they arrive and direct them to transport the bird's carrier into the practice in a stable fashion (not swinging, held in an upright position, not bumping the carrier against any objects, etc.). Ideally, the client should hold the carrier by its base instead of grasping it by a handle to facilitate this. It may be necessary to help some clients enter the building by holding doors open for them, particularly if the carrier is bulky.

Once the bird is inside the practice, CSRs should advise clients to place the carrier in a location that decreases stress, such as on an elevated surface in a quiet area. Placing carriers on the floor increases stress for most perching birds, as it places them at eye level for many other animals entering the lobby and may increase the possibility of collisions with the carrier from foot traffic. Species-specific exceptions may exist (large poultry, for example). As previously mentioned, a towel can be used to cover the carrier if this appears to reduce patient stress, and the amount of time spent in the receiving area should be minimized.

Ideally, the bird should be moved directly from the client's vehicle into the examination room and allowed to habituate for several minutes before exiting their carrier. The CSR should give a cursory glance at the bird to judge the level of stress that the bird might be displaying. If stress levels seem elevated, the CSR should ask for assistance from a technician or an assistant.

THE LOW-STRESS EXAMINATION

The examination is generally a significant source of stress during the veterinary visit. Thoughtful preparation will minimize the length of the examination, resulting in reduced stress. This includes prior setup of the examination room as described previously. Obtaining husbandry information and a

TABLE 46.2
Potential Reinforcers for Parrots

Potential Food Reinforcers for Parrots	Comments
Sunflower seeds in the shell—low oil preferred	Some parrots do not recognize shelled sunflower seeds.
Shelled sunflower seeds	Preferred by many parrot species.
Almond pieces	Preferred by many parrot species.
Walnut pieces	Preferred by some individual parrots.
Pistachio pieces	Preferred by some individual parrots.
Pine nuts—out of the shell	Preferred by many larger parrot species.
Millet spray	Preferred by many smaller parrot species. Conure-sized and smaller. Of the large parrots, Eclectus species often respond to millet.
Macadamia pieces	Preferred by hyacinth macaws.
Safflower seeds	These bitter seeds are often preferred by parrots in the genus *Poicephalus* and some conures.
Unsulfured papaya bits	Moluccan *(Cacatua moluccensis)* and umbrella cockatoos *(Cacatua alba)* and some Amazon parrots (*Amazona* spp.) often respond to this food item.
Peanut pieces	Preferred by many parrot species—however must be used with caution due to aflatoxicosis risk.
Lorikeet nectar, grapes, and apples	Preferred by lories and lorikeets.
Nut butters (almond, cashew)	Can be helpful for masking oral medications.
Parrot hand-feeding formula	Can be helpful for masking oral medications.
Human baby food (banana, winter squash)	Can be helpful for masking oral medications.
Potential Nonfood Reinforcers for Parrots	**Comments**
Touch, head scratches, toys	Evaluate on a case-by-case basis for individual response.

TABLE 46.3
Potential Reinforcers for Non-Parrots

Potential Food Reinforcers for Non-Parrots	Comments
Fresh corn, thawed frozen corn, cracked corn	Preferred by chickens and other poultry
Live mealworms, dried mealworms, crickets	Preferred by chickens and other poultry
Shelled sunflower seeds, nut pieces, vegetables, meat, cooked egg	Preferred by chickens and other poultry
Mice, pinkies, fuzzies	Hawks, owls, eagles
Beef heart	Caracara, vulture
Quail, fresh pigeon	Falcons
Peanut pieces	Pigeons



FIGURE 46.4 Different bird species respond to different types of food items. Smaller birds such as cockatiels *(Nymphicus hollandicus)* and budgerigars *(Melopsittacus undulatus)* often prefer millet spray. Even if a bird is not trained, they can potentially be lured onto scales and into crates to avoid the stress associated with a capture and restraint.

medical history (including flight status) can be accomplished at the same time as a hands-off assessment of the bird's clinical state and behavior (posture, reaction to unknown individuals, vocalizations, symmetry and position in cage, respiration rate, and effort). If the bird is not in a carrier, make note of how the bird is interacting with the client, and consider the best way to approach the bird. These observations may be adequate to determine the need for conscious sedation before the examination starts in some patients.

The goal should be to have the bird restrained for the shortest amount of time possible (if required at all), and restraint should be characterized as optimizing welfare (maximizing benefits and minimizing harms). Veterinarians and team members who are experienced in reading avian body language and accurately interpreting hands-off examination findings are much more likely to be able to successfully handle and treat birds. Careful observation, gentle handling, and conscious sedation (when indicated) all improve safety of patients.

Birds can display a variety of behavioral and physical changes secondary to stress. If noted, some of these symptoms require an immediate shift to a hands-off approach, and others may even require oxygen support. If signs such as aggression, panic vocalizations, red tears (in grey parrots; Figure 46.7), profuse feather loss (in Columbiformes and Musophagiformes;



FIGURE 46.5 Parrots that are not interested in food may respond to small toys as potential reinforcers for desired behaviors. It is recommended to keep a supply of small bird-safe destructible items on hand for this reason.



FIGURE 46.7 Occasionally, grey and Timneh parrots (*Psittacus erithacus* and *Psittacus timneh*) may secrete red, bloody tears while being manually restrained for physical examination or diagnostic sampling. While the exact cause is not known, it has been hypothesized that a rise in blood pressure due to stress from handling could be involved, especially in already nervous animals. Screening for underlying pathologies, such as chronic respiratory infections, cardiovascular disease, or ocular disease, may be indicated, but in the majority of cases, workup may be unrewarding with no underlying cause identified. (Photo courtesy Richard Jones, Avian Veterinary Services.)



FIGURE 46.6 This Moluccan cockatoo *(Cacatua moluccensis)* is playing with a foot toy created in an examination room out of two tongue depressors and some VetWrap. Toys can be an excellent reinforcer for some birds.



FIGURE 46.8 This dove lost all her tail feathers secondary to stress of capture. This is a common defensive mechanism for Columbiformes, Musophagiformes, and other species.

Figure 46.8), open mouth breathing (in absence of respiratory disease), hyperthermia, tachycardia, tachypnea, and hypertension are noted, release the bird from restraint and consider a different approach, potentially including conscious sedation.

Signs of extreme stress include dyspnea, changes in mentation, seizures, and collapse. If you see any of these extreme symptoms, the patient may be on the verge of going into cardiovascular collapse secondary to underlying disease combined with extreme stress. The patient should be **immediately** placed in an oxygen chamber and handling should be minimized thereafter. If actions are taken quickly enough, the bird may recover (at least in the short term). Further supportive care, such as IV catheterization and fluid therapy, may need to be delayed until the patient has stabilized in oxygen, unless the patient has gone into full cardiorespiratory arrest and cardiopulmonary resuscitation (CPR) is indicated

(see Chapter 34). Birds that experience a collapse like this are critically ill; their owners must be prepared for the possibility of death at any moment. These patients must be handled with extreme caution, and procedures (including further examination and supportive care) must be staged carefully in order of importance.

Clinical History and Relevance to Low-stress Veterinary Medicine

Collecting a thorough history, including husbandry and diet information, is critical to success with avian cases. Interpretation of the history prior to the examination will allow for a more efficient and tailored diagnostic approach, often reducing the number of diagnostic modalities required to reach a diagnosis.

Obtaining information regarding the patients' prior veterinary experiences is extremely helpful for minimizing patient stress. This may include handling techniques (by the client and previous veterinary staff) and exposure to relevant objects/situations (how the patient has reacted to approach by unfamiliar people, whether the bird has experienced pleasant interactions with towels, etc.). An example of the depth and breadth required for an avian behavioral history is provided in (Box 46.3 is online only and can also Box 5.3 from *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). Although it seems that routine visits may not require as much detail in the history, it is all relevant.

Knowing how often the bird is handled and *how* the bird is handled is important for the initial approach to handling the bird in the hospital. Companion birds experience a wide variety of home situations. Some birds stay in a cage all the time; some birds are brought out only on a stick/perch; some birds are handled by their owners routinely and may be more willing to step up to unknown individuals. However, just because a bird is handled a certain way by their owner, does not mean that it will respond the same way with an unfamiliar person. Ultimately, the method of approach to the bird should be chosen based on the bird's behavior in the examination room.

It is ideal when clients have previous knowledge of how to train their parrots to perform behaviors using positive reinforcement. This allows staff to more easily explain how to incorporate training for medical behaviors into the case management plan. If a client lacks such knowledge, the treatment plan will need to incorporate the demonstration of shaping plans for targeting, taking oral medications, stepping onto a scale, going into a crate, and so on.

Obtaining a thorough history can be accomplished by sending a technician into the room to speak with the client prior to the veterinarian's entrance, by having the veterinarian collect the history during the examination, or by having the client complete a form online prior to the appointment. There are advantages and disadvantages to each approach.

Having the technician take the history can serve to create rapport with the client, ferret out details and impressions important for the veterinarian's success with each unique patient, allow the patient more time to habituate to the examination room, and buy the veterinarian time to complete the last examination. This approach does, however, take additional time and may be a disadvantage in a tightly packed schedule.

There are similar benefits and challenges to having the clinician take the history. It can be difficult for some veterinarians to get all the information necessary in a timely manner before jumping into the examination. However, this approach may be more efficient in some clinicians' hands because the hands-off examination, development of client rapport, and patient acclimatization to the clinician can occur simultaneously.

Having the client complete the form electronically allows the information to be entered more quickly into the patient record and to be reviewed prior to the appointment. Such a review may facilitate a more efficient and tailored diagnostic approach, as well as increased success with handling. On the other hand, clients often do not provide information in as much detail as needed when filling out a generic form and/or may lack the technical skills required. Therefore, all client-completed forms should be edited for clarity/details by veterinary staff at the time of the appointment.

Wording on the form must be precise to collect sufficient detail. Some questions should be open-ended. Others should offer multiple-choice options that will identify common husbandry concerns. For example, when asking about UV light exposure, including "through a window" as an option (in addition to time outdoors, exposure to a UV light, etc.) will provide an opportunity to discuss how to improve effective exposure.

Interacting with a Bird during the Hands-off Examination

Observation of the unrestrained patient prior to handling is a critical part of the physical examination. Not only can many important health concerns be identified, but also the temperament of the bird can be evaluated, including receptiveness to handling.

When initially approaching a bird, consider utilizing sidelong glances rather than a direct gaze, as this may increase patient comfort. During this process, the clinician can begin to associate desired items or experiences with their presence. Allowing the bird some time to habituate can be helpful to some birds if they are under threshold. Talking may be viewed as a desired experience by some parrots; for others it can create high levels of arousal that can quickly evoke aggressive behavior. It should be used with caution. Preferred food items and desired toys can be helpful to reinforce voluntary cooperation (Figure 46.9).

Birds are very alert to changes in the surrounding environment and have a reputation for exhibiting unexpected fear responses in examination room settings. Potential triggers for these behaviors may range from something visible outside a window to an insect, a color, pattern, or style of clothing, examination room furniture, or something that reminds the bird of a past aversive experience (objects, people, or other stimuli). Careful observation may allow staff members to identify stimuli that evoke fear responses, but this is not always possible due to the subtlety of certain stimuli and the fact that many of these patients have incomplete behavioral histories. In many cases, the response may be rooted in neophobia rather than an aversive history with a stimulus. Collecting data over time and recording reactions in the behavioral medical record can sometimes reveal patterns or trends and allow identification of stimuli that evoke fear responses. If a stimulus cannot be

BOX 46.3 AVIAN BEHAVIORAL HISTORY FORM

Date of consultation:

OWNER INFORMATION:

Name:
Address:
Phone: ..... (day) ..... (evening)
Email:
Who is the bird's primary caregiver?

BIRD INFORMATION:

Name of the bird:
Species:
Age: ... years/... months Unknown
Sex: Male/Female/Unknown
If the sex is known, how was this determined?

HOUSEHOLD INFORMATION:

List all people in household and people who interact with bird on a regular basis:

Name	Age	Relationship (e.g., Spouse, Son, Daughter, Pet Sitter, Housecleaning Service Workers)

List other birds and/or pets in household (list these in the order in which they came into household; include the patient in this chronologic list as well):

Name	Species/Breed	Current Age	Age when Acquired	Sex

Provide a brief description of the interaction between the birds (e.g., do they have vocal/visual/physical contact, share a cage)

ACQUISITION AND REARING HISTORY:

Reason for acquisition:
Acquired from (circle the answer that is applicable):
Breeder/Pet Store/Previous Home (Rehomed bird)/Own bred/Shelter/Other (describe):
If your bird is a re-homed bird, describe briefly below what is known about its previous home environment (e.g., married couple? children? length of time in home)
Your bird was (circle the answer that is applicable):
Bred in captivity/Wild caught/Unknown
Hand-raised/Parent-raised /Unknown rearing method
If hand-raised, by whom? (e.g., breeder/you/unknown)
Raised with: siblings/similar-aged birds but different species/birds of other ages/unknown
At what age was your bird separated from its parents?
At what age was your bird rehomed to its new home?
Briefly describe the weaning process and stimuli (e.g., social contact and enrichment) the bird was confronted by during early life:

HUSBANDRY:

Cage(s) and playpen:
- Size (approximate dimensions) and materials of which the cage is made
- When used (e.g., sleeping, daytime, outside)
- Location(s) (i.e., inside/outside, where in the house and where in the room)
- Cleaning (frequency, products, procedure of cleaning)
- Furniture in cage (include information on which types of enrichment are provided, which of these are used, frequency and way in which these are used, and how often these are changed, if applicable):
  - Perches and other climbing materials/toys
  - Bowls
  - Chewing toys/materials
  - Puzzle toys
  - Foot toys
  - Nest boxes
  - Other

NUTRITION:

- Diet: % fed (offered and % actually eaten)
  - Main diet
  - Treats and supplements (including which and how often provided)
  - How fed (bowl, foraging enrichment)
  - Water source (how provided and frequency of changing)

SLEEP RHYTHM AND LIGHTING:

- Hours awake/asleep
- Hours light/dark

Cage covered: Yes/No
What type of lighting is available to your bird (e.g., daylight, UV-light, fluorescent light)?

Continued

BOX 46.3 AVIAN BEHAVIORAL HISTORY FORM—cont'd

FEATHER CARE (BATHING/MISTING/CLIPPING):

- Do you provide your bird bathing/misting opportunities: Yes/No
  - If yes:
    - How often?
    - Method?
    - Bird's response to bathing/misting?
- Are wings routinely clipped? Yes/No
  - If yes, how is the clipping performed?
- Does your bird come outside? Yes/No
  - If yes, how often and where? (e.g., garden, park, friends/family)

HUMAN/BIRD INTERACTION:

- What tricks or commands does your bird know?
- Provide a brief description of the interaction between the bird and the people in the household (e.g., how often does the bird interact with the different people, characterize the interaction [cuddling/physical contact, playing/training, care and feeding]).

DAILY ROUTINE:

- Describe a typical day in your bird's life, starting with when and where bird wakes up in the morning. Be sure to include when, where, how, and who feeds bird, exercise/training/play (what type, how much, where), where bird is when people are not at home, where bird sleeps at night, and anything else you think is important (use additional pages as needed):

BEHAVIOR OF YOUR BIRD:

- Give a short description of your bird's behavior to the various people/animals/stimuli as mentioned as follows:
  - How does your bird react to its daily caregiver?
  - How does the bird react to familiar people (other than the daily caregiver)?
  - How does the bird react to unfamiliar people?
  - How does your parrot respond to other parrots?
  - How does the bird react to new items or toys?
  - How does the bird respond in a new environment (other than its familiar environment)?
- Does your bird allow petting?
  - If yes, does it actively ask for it (e.g., by offering its head) or not?
- How does your parrot respond to handling (e.g., using a towel)?

PROBLEM BEHAVIORS:

Indicate whether bird displays any of the following behaviors:

Behavior	No	Yes (Indicate Frequency in Next Columns)	Daily	Weekly	Monthly
Chronic egg laying					
Chewing/biting/tearing its own skin (self-mutilation)					
Damaging its own feathers (feather-damaging behavior)					
Destructive chewing of furniture or other materials in the environment					
Screaming					
Rubbing cloaca/vent on objects or people					
Regurgitating					
Repetitive behaviors such as somersaulting or weaving					
Hiding/trembling/trying to get away from people or other animals (fearful behavior)					
Lunging at/biting people or other animals					
Other (provide a description)					

MEDICAL HISTORY:

Does your bird have any known medical conditions or physical problems?
If yes, describe these briefly.
Does your bird take any medication?
If yes, describe which, what dose, and how often?
Details regarding your bird's problem behavior:

- What is the behavior problem for which you are seeking advice?
- Did a specific event prompt you to address this problem now?
  - If yes, describe this briefly.
- When did you first notice the problem, and what did the behavior "look like" at that time?
- When the problem first began, how frequently did it occur?
- How often does it occur now?
- Do you feel that there were any inciting factors?
  - If yes, describe these briefly.
- How has the problem changed or developed over time?
- When does the problem behavior occur now?
- Do you feel that there are any specific situations that seem to "trigger" the behavior?
  - If yes, describe these briefly.

BOX 46.3 AVIAN BEHAVIORAL HISTORY FORM—cont'd

Date of most recent incident of behavior:
Description of most recent incident of behavior:
How do you respond when your bird performs this problem behavior?

What, if anything, have you done to try to treat the problem (e.g., medical treatment, behavioral therapy, medications, supplements, herbs)? Indicate if the attempted treatment made the problem better, worse, or if the behavior remained unchanged.

Treatment Attempted	Behavior Better	Behavior Worse	No Change in Behavior



FIGURE 46.9 A food lure can be used during an examination to view a wound without restraint.



FIGURE 46.10 Stationing to obtain an accurate body weight.

definitively identified and removed or desensitized/counter-conditioned, conscious sedation may be necessary to prevent escalation of the fear response.

Obtaining a body weight from a bird should be done prior to restraint, if possible, as it allows the clinician to quickly calculate dosages of sedatives if deemed necessary. There are a variety of techniques for obtaining a body weight from a bird, including asking the bird to step onto a perch placed on the scale, standing on the scale itself, and weighing the bird in its own carrier or a separate box to prevent escape (Figure 46.10). In general, veterinary staff should seek to make this process as easy as possible for the bird. It is important that the surface the bird is standing on is not slick and that the bird does not have to be in an uncomfortable position to stand on the perch (including not touching its tail on anything). If the bird is not well socialized, obtaining a body weight should be delayed until the veterinarian enters the room to minimize the risk of unnecessary stress.

Low-stress Handling and Restraint

> *"By restraining pets in a forceful, crude, or unskilled manner, you could be breaking your promise to do no harm. Such handling can make pets behaviorally worse and even lead to aggression and, ultimately, euthanasia."*
>
> **Dr. Sophia Yin**

Based on what the client (and patient) have experienced in the past, they will anticipate the examination to unfold in a certain way, such as a low-stress examination or the traditional "capture and restraint." Veterinary staff need to be aware of this, communicate clearly to set appropriate (possibly novel) expectations, and be sensitive to body language of both clients and pets to maximize success. Information offered about the bird's preferences for handling can help save time and reduce stress for the bird and should be entered in the medical record.

Avian species (and individuals within species) differ in their need for restraint for various procedures. Many well-socialized birds (parrots, poultry, birds of prey) will tolerate the entire or majority of the examination with minimal restraint. The focus of each veterinary-patient interaction should be to utilize the most optimal method of restraint to safely obtain diagnostic information. When choosing the restraint technique to be used with a patient, it is important to take into consideration possible sources of injury to veterinary staff, which varies depending on the species of the patient.

Parrot bites can be painful and even debilitating. Although they do not have the grip strength compared to raptors, sharp nails on a parrot can cause punctures and lacerations to the handlers. Loud vocalizations of some species can be damaging to the ears. Poultry may kick or strike with their wings or spurs. Some raptors species have powerful grasping feet that can cause severe injuries, and some species may also bite (eagles, owls, etc.). For these reasons, proper protective equipment is essential for certain species.

Ear protection should ideally be worn by those that will be within a meter or so of certain bird species (especially macaws and cockatoos) to prevent hearing loss secondary to loud vocalizations, should this be an issue with a particular patient. Consider ear plugs rather than headphone-style hearing protection, as they are unobtrusive and can minimize the possibility of creating a fear response. Thick leather gloves should be avoided in all non-raptor species. Although they may provide some protection against bites and scratches, leather gloves allow minimal dexterity for needed procedures and may create a fear response. Additionally, a large parrot bite has substantial crushing power and a bite through a glove can still be quite painful.

When an animal is presented with an aversive stimulus that evokes a fear response, in most cases the animal chooses to escape. When escape is not an option, an animal may default to aggressive behavior to cause the aversive stimulus to go away. Aggressive behavior should not be "expected" in veterinary practices, although biting, lunging, and other types of aggressive behavior may be noted when the patient is exhibiting escape/avoidance behavior or fear response. Staff or owner behavior is typically the primary antecedent for bites or strikes. Coercive handling often creates aggressive responses. Providing choice in interactions and pairing interactions with desired consequences is a much better approach.

BOX 46.4 EXAMPLES OF SUBTLE BODY LANGUAGE DISPLAYED BY PARROTS THAT, IF IGNORED, CAN LEAD TO A BITE

- Pinning or flashing eye pupils
- (Slight) opening of the beak
- Raising or flattening of the (crest, head, or body) feathers
- Tensing up of the body, rigid body posture
- Spreading and arching of the wings away from the body
- Tail fanning
- Leaning away and then toward the hand
- Rocking back and forth
- Crouching
- Vocalizations, e.g., hissing, growling

TABLE 46.4

Injuries Resulting From Improper Restraint and Stress Responses

Suffocation	Birds must expand their keel bone in order to breathe normally. Placing pressure around the body of a bird during restraint can lead to suffocation.
Blunt trauma	Attempts at escape from aversive stimuli may lead to blunt trauma from flying into windows or walls or falling to the floor. Even skilled fliers may be injured due to their heightened emotional states, but unskilled fliers are at the highest risk for injury.
Musculoskeletal injuries	Many birds kept as pets have osteoporosis from inadequate dietary calcium and lack of exposure to natural sunlight, increasing their risk for pathologic fractures. Capture myopathy can occur in any bird but is a higher risk for certain species (long-legged wading birds, waterfowl, and ratites).
Hemorrhage, wounds, bruising	Broken blood feathers or toenails, wounds on carpi resulting from wing flapping, bruising on the cheeks (particularly in macaws and grey parrots) from excessive force during restraint.
Feather loss	Many bird species (especially Columbiformes and Musophagiformes) will lose feathers as a distraction ploy when faced with a perceived predator attack—may impact thermoregulation/flight in addition to patient appearance.
Seizures and/or cardiac arrest	Particularly common in small birds with other underlying health problems.

Another common cause for injuries is inaccurate interpretation of avian body language and poor management of the environment. True unprovoked aggressive behavior is rare. Birds are more likely to bite when subtle body language presented as a precursor to a bite has been repeatedly ignored (see Box 46.4 for examples), and compliance is gained via coercion.

If physical restraint is necessary for the individual patient or stage of the veterinary visit, it must be administered skillfully to avoid patient injury. Improper restraint and patient stress responses can lead to life-threatening injuries, which are documented in Table 46.4.

Tools for low-stress restraint

Most veterinarians utilize towels as a method of restraint. Towels are inexpensive, soft, yielding, and effective at preventing staff injury when used competently. They can also be very well tolerated by many birds when they are thoughtfully implemented. Birds can be easily trained to accept and even seek out contact with towels using positive reinforcement. Every bird owner has access to towels for training at home. Towel size, thickness, flexibility, and material type can be tailored to the individual patient/species.

Towels provide excellent handling dexterity and protection of staff hands against bites or grasping injuries and for most avian species. Although bites can easily penetrate even a thick towel, a towel can provide a visual barrier that does not allow the bird to purposefully bite a hand. Only portions of the towel without hands underneath should be placed in a position to be bitten by the patient. By holding a bird's wings against the body, towels also prevent wing strikes and inadvertent patient injuries. In addition, parrots will often chew on towels, which can be a useful distraction during the examination.

One concern with towel restraint methods is that a towel can trap heat close to the restrained bird. This can lead to significant overheating, often resulting in the bird panting as an early symptom. Close monitoring of the bird's responses is therefore warranted during restraint, and prolonged restraint should be avoided.

Hooding is another technique to minimize avian stress, most often used in raptors and Struthioniformes. The hood is a leather or cloth "cap" custom-designed to fit over the bird's head. It covers the eyes and ears, limiting visual and auditory stimuli, while leaving the beak and nares exposed (Figure 46.11A and B). Limiting stimuli can reduce observable fear responses and make it possible to examine some hooded birds with little to no restraint. Individual raptors will have different histories with hooding. Birds with a great deal of experience with hooding, especially paired with positive reinforcement, can present as quite relaxed when wearing a hood, as well as when it is removed and put in place again. While decreasing visual stimuli can reduce expression of the fight-or-flight response, it should not be an excuse to replace training with coercive handling.

Thick leather gloves may be used as a perch for trained falconry birds to facilitate handling. These birds may also have jesses attached to their legs, which can be useful to help keep them safely on the glove and provide some protection from footing injuries during minimally invasive handling (Figure 46.11C).

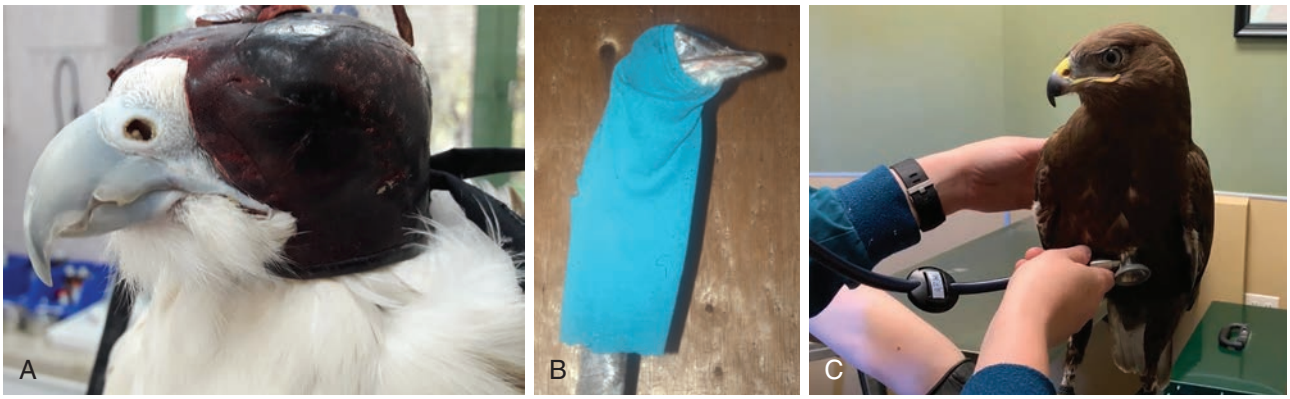


FIGURE 46.11 (A) Hooding is a technique that is commonly applied in raptors and Struthioniformes. By covering the eyes and ears, exposure to visual and auditory stimuli can be limited, hence preventing a stress response. (B) For this adult ostrich hen *(Struthio camelus)*, a functional hood was created using the cutoff end of sweat bottom pants. (C) A ferruginous hawk *(Buteo regalis)* is examined on the falconer's glove. The jesses are held tightly to prevent a footing injury to the veterinarian during auscultation.

Handling and towel restraint techniques

Unfortunately, many avian patients have a history of coercive handling at the veterinary office or in the hands of groomers, which will affect their behavioral responses regardless of the chosen approach to the contemporary examination. Traditionally, parrots have been rapidly grabbed from behind with a towel or gloved hand. This method often evokes a severe fear response and should be avoided for most patients, as veterinary professionals have options to provide more optimal restraint procedures.

There are a variety of handling and restraint techniques available that may have varying degrees of efficacy for each individual bird, depending on its learning history and individual preferences. The method(s) used should be recorded in the medical record along with the relative success or failure of the technique. This allows the best method to be used on subsequent visits. Keep in mind that some birds have multiple methods that may "work" depending on the circumstances surrounding their visit, so there may need to be additional stipulations within the medical record for behavior cues to watch for to modify your approach.

Ideally, the bird should remain in the carrier until handling by the veterinary staff needs to commence. There are several benefits to this approach. First, it minimizes the risk of a patient injury due to a startle response (as it cannot fly into a window or wall). Second, it usually allows the patient to be picked up in the safest manner, preventing escape. Third, leaving the bird in the carrier allows the bird time to habituate to the examination room while confined to the (relative) safety of its carrier. Finally, it allows the veterinarian to perform a hands-off examination with the bird in a minimally stressful way and reduces examination artifact caused by exercise from flying around the room or stress associated with capture. However, some pet owners will remove birds from carriers despite recommendations of veterinary staff.

The techniques described in the following section can be modified for socialized birds that are exiting their carriers or have already left the carriers. Birds that have already been removed from the carrier should be transferred to a perch the bird is more likely to leave (such as the examination table, or the top of the bird's carrier) prior to asking them to step up, rather than asking them to step up while perched on the client's hand or shoulder. Some parrots may be more likely to bite the veterinary team member, or exhibit redirected aggressive behavior, if they are seated with their owner.

Handling and towel introduction in the well-socialized parrot. Picking up a well-socialized parrot is typically straightforward. These birds show mild or zero signs of stress and are familiar with the step-up behavior, regardless of whether they are within their carrier or on another perch. The bird is offered a hand to step onto; in general, the hand should be turned sideways to offer an easier perching surface, rather than the palm pointed upward. This would not apply for birds that have been trained to step onto a flat upward facing palm, or to very large or non-psittacine perching birds (such as chickens). While most birds prefer to step up onto a hand in front of them, some prefer to step onto a hand behind or to the side of them. Some clients may know their bird's preference, so be sure to ask. A flexible approach that is responsive to body language of the patient is essential.

In some cases, birds may respond better to standing on an examination table or a perch for the examination, rather than perching on a hand or being restrained in a towel. It is possible to perform entire examinations with no restraint in well-socialized parrots and many poultry. The most intrusive parts of the examination should be saved for last, as this may push birds over threshold and lead to the need for restraint for the remainder of the examination. This varies with species, but in general, parts of the examination that require utilization of tools near the bird (stethoscope, specula, etc.), coelomic palpation, and cloacal examination tend to be most intrusive.

Once the examination reaches the point where restraint is necessary, one of the most common techniques for getting the bird into the towel starts with them perching on the restrainer's hand. Depending on the individual patient, it may be safest to grasp the toes by the thumb to prevent the bird from jumping or flying off (Figure 46.12). Be aware that this step may be poorly received by some birds and precipitate a



FIGURE 46.12 The handler is restraining the toes of this umbrella cockatoo *(Cacatua alba)* with her thumb prior to attempting towel restraint. This method can help prevent injuries in flighted birds but may lead to injuries to the handler as the bird may try to bite.

startle response or a bite. If the bird is not tolerant of this, a different approach is needed. It may be possible to get the bird wrapped in a towel without concurrent foot restraint, although injuries may occur if the bird is flighted. Other options include having the bird standing on a perch or standing on a towel-covered hand. In extreme cases, some birds may need to have their restraint experience initiated from within the carrier.

After the bird is comfortably in hand, the bird is then brought close to the veterinary staff member's body, and systematic desensitization is used to bring the towel up from the side or below the bird gradually. This means the practitioner pays close attention to the bird's body language and moves the towel so that it does not evoke an excessive fear response. The towel moves in increments to slowly cover the bird's body and head as opposed to moving up and down repeatedly, which may evoke escape and avoidance. Negative reinforcement can also be used in which calm body language causes the towel to be removed. It is important to start far enough away that calm responses can be emitted. This distance is gradually decreased as calm body language increases. Moving too quickly may cause the animal to become sensitized to the towel's approach. This may require the practitioner to start over, try again later, or consider another technique. While it may seem that this would take an excessively long time to accomplish, in many cases it takes less than a minute; in general, the slower/more gradual the approach to start with, the faster and better the results.

When the towel is almost touching the bird, the bird's neck can be gently grasped from behind. The towel is then wrapped around the body for restraint of the wings and feet. The bird should be held vertically in a natural standing posture, as this is less stressful than lying on the back and will minimize exacerbation of medical conditions that affect breathing. If possible, allow parrots to grasp something in their feet (a hand, a perch, etc.) during restraint, like standing on a perch. The restraint technique may need to be shifted multiple times within the examination room based on the patient's behavior.

While this method is successful for many birds, it is far from the only technique available and could result in patient or staff injury if applied to certain patients. For example, birds that show fear responses or aggressive responses to hands; birds that have phobic responses to the presence of a towel; birds that are poorly socialized; and bird species other than parrots would respond poorly to this approach. In the following paragraphs, several alternatives are explored.

Alternative methods of towel introduction in parrots. Fear responses to the hand can be very common depending on reinforcement history (grabbed by hands/gloves, fallen off a hand, etc.). Parrots with this history and behavior response may prefer to step to a toweled hand. To start "The Potholder" technique, cover the hand with one end of the towel and leave the rest of the towel hanging down (Figure 46.13A). For large parrots, the towel can be folded at the end to provide two layers over the hand as well as to pull it up over the forearm. Once the bird has stepped up to the toweled hand, the handler gently holds the parrot's toe to keep the bird on the towel. Use systematic desensitization or negative reinforcement to cover the bird with the towel as described in the first technique.

This technique can be modified into "The Butler" technique for birds that show a fear response to towels but are willing to step to a bare hand (Figure 46.13B). Place one end of the towel over the handler's forearm, letting the remaining length of the towel to hang down. Ask the bird to step up using the same hand as the towel. Next, decide if it is needed to gently hold onto the bird's toes or bring the towel forward to cover the handler's hand, which will prevent the handler from being bitten. Drape the towel slowly, as described previously, covering the entire bird with the towel.

For patients that prefer to step to the towel and respond to tactile contact as a desired consequence, we can use "The Toga" technique (Figure 46.13C). The handler drapes the towel over their shoulder, covering the front of their body. With either hand, ask the bird to step up and offer desired touch. Bring the bird in against your body and the towel while cuddling and with the free hand slowly bring either end of the towel around the bird. Many birds will accept (and enjoy) this type of restraint, provided they like tactile contact. Food can be substituted for tactile reinforcers if the bird is receptive to food in the veterinary setting (Figure 46.14).

When interacting with the patient and client in the examination room, some parrots will not step off the perch for a towel, hand, or dowel. These birds might do well with "The Yoda" technique, in which the handler uses stimulus fading (gradually introducing the towel so that it does not draw the bird's attention) to slowly bring the towel behind the perched bird and wraps the towel around the bird (Figure 46.13D). The handler's arms are on either side of the bird, limiting its movements. The handler may talk to the bird while this happens or remain quiet depending on the individual patient. This technique may be particularly effective for parrots that seek to acquire attention from people, as they can receive



FIGURE 46.13 Toweling techniques for handling parrots. (A) The "Potholder" technique encompasses covering of the hand with one end of the towel (thereby protecting the hand), while the rest is left hanging down and is gently draped over the bird while holding its toes after it has stepped onto the towel. It is often used in birds that will step to the towel but not to the hand. (B) The "Butler" technique is very similar to "The Potholder"; only here the end of the towel is positioned over the handler's forearm rather than held in the hand. This will often work well for birds that will step onto the hand voluntarily but may be nervous about the towel. Once the bird has stepped up, the towel can be moved up to cover the hand and then gently lifted and draped over the bird, as done during "The Potholder." (C) For birds that like to cuddle and prefer to step to the hand or towel, "The Toga" technique can be used whereby the handler drapes the towel over the shoulder and drapes it gently over the bird once it has stepped onto the hand and is being cuddled. (D) The "Yoda" technique often works well for birds that are glued to the perch. Talking to the bird during the procedure can help keep them focused while the handler slowly brings the towel up from behind and then gently wraps it around the bird. (E) The "Lap Dance" technique works well for parrots that are responsive to attention and touch as desired consequences and are tolerant of standing on the lap of the handler while the towel is gently draped over the bird from the sides or behind. Like "The Yoda," talking to the bird may help calm it down.



FIGURE 46.14 Some birds, like this Goffin's cockatoo *(Cacatua goffini),* will accept food reinforcers during a restraint experience.

copious amounts of attention as a desired consequence while the towel approaches them. Keep in mind that the heightened state of arousal that is often triggered by bouncing up and down, vocalizing, and so on, may trigger undesired aggressive responses with a parrot.

"The Lap Dance" technique works very well for parrots that respond to both attention and tactile contact as desired consequences and are tolerant of standing on the lap of the handler (Figure 46.13E). While the bird is perched on the lap of the handler, slowly bring the towel toward the handler's knees. Deliver tactile or other appetitives while the towel is brought toward the bird and draped over the body. The bird is corralled against the handler's body and the handler's arms help limit the bird's movement, making it easy to gently cover the bird.

It is also possible to combine techniques, such as "The Potholder" with "The Toga," where one end of the towel is draped over the shoulder, and the other end covers the hand that is offered to the bird. This provides a little more protection for the restrainer's hand. Additionally, working with a bird in a corner of the room can functionally replace arms on either side of the patient (as in "The Yoda" or "The Lap Dance") while using a different technique, such as "The Toga." The combinations are limited only by your creativity. Videos of these techniques can be viewed at https://animaltrainingfundamentals.com/vet-text-videos/.

Restraint of poorly socialized or birds showing extreme fear or aggressive behavior. In some cases, it may be preferable to shape a restraint experience for a bird from within the carrier rather than allowing the patient to exit the carrier unrestrained. This is especially true for some flighted birds, birds that are known to panic and drop to the floor (risking injuries), and birds showing aggressive behavior (biting, lunging, etc.) that is unchanged with alterations of the environment and time to habituate. Standard pet carriers can have narrow front openings, increasing the difficulty of these restraint efforts; carriers with an opening on the top are safer and easier to use.

The towel should be slowly moved toward the bird while blocking the entrance to the crate to prevent escape. Some birds tolerate this very well (especially if the towel is slowly lowered onto them from above). Parrots often have a strong grasp on the cage's ventilation openings and will have to be carefully separated from the carrier. Some parrots (particularly Amazon species [*Amazona* spp.]) will flip onto their backs as part of a defensive strategy when approached by a towel, making it much more difficult to access the back of their neck. A technique that is useful in these cases is to approach the bird with several layers of bunched-up towel between your hands and the parrot. Bring the ends of the towel around the sides of the bird, slowly enveloping them, while placing gentle pressure on the sides of the neck to keep the head restrained. Once the bird is in hand, the bunched-up towels should be replaced with a more traditional single towel wrap around the back of the neck for better control.

Small birds, such as finches and canaries, may require more strategic planning for safe acquisition of restraint. These birds are typically severely frightened on presentation and are agile fliers. Chasing these patients around their cage is exceptionally stressful for them and may lead to them escaping into the examination room.

For birds brought in their own cage, you may need to start by taking out the perches and toys inside, which work against you as obstacles when trying to get the bird in hand. Once the perches are out, assess if you can take the top or bottom off of the cage. Cover the large opening with the towel and slowly bring the towel to the other end, allowing you to restrain the bird. By removing obstacles from the cage, the bird is not able to hide and avoid the towel, which generally shortens the timeline for acquiring effective restraint. Additionally, slowly bringing the towel in through a larger opening like the top or bottom of the cage will minimize stress from the patient being chased around the cage. The handler may find that laying the cage on its side is more helpful with this process.

Another method to restrain these small birds with minimal chasing is to mentally note the position of the bird, completely darken the room, and grasp the bird in the dark. When the lights come back on, the bird is in hand and already restrained for the tasks required. While this is most certainly still stressful, the elimination of the chase can shorten the duration of the stress event. This approach, if frequently repeated, could induce fear of the dark for the bird or precipitate stress-induced seizures, so it should be avoided if possible. The authors only recommend its use when prolonged attempts at acquiring restraint would be dangerous for the patient. Researchers at Stanford University have expanded upon this technique by incorporating inexpensive night vision goggles and using infrared lights (like techniques used to calm down poultry in commercial farms) that have reduced and/or eliminated stress of restraint procedures for regular health checks of psittacine birds participating in scientific studies.

Different birds respond to restraint in different ways. Some will fare better if handled for several brief periods of time, with handling and restraint repeated as needed to finish

the examination. Others are difficult to get restrained initially and therefore should have everything done during one episode of restraint. Flexibility in restraint decisions is essential.

Training in the Examination Room

When a bird moves away from something, this is often described as a fear response. When the bird cannot get the distance it desires, the bird may respond with aggressive behavior to drive the aversive stimulus away. In both situations, the attempt to gain distance is maintained by negative reinforcement. The bird's behavior (either escape or aggression) is increased by the removal of an aversive stimulus. Understanding that negative reinforcement is maintaining the fear response or aggressive behavior is important. This is because this information can be used to shape new desired responses. Distance can be provided as an outcome for calm and relaxed behavior.[45] For this strategy to be successful, the animal must be calm and relaxed at the outset of the session. The aversive stimuli must be presented from far enough away that it does not evoke a response. For example, a person may hold a towel in their hands and stand 5 feet away from the bird. The person would then approach the bird until a slight response is observed, such as if the bird looks at the towel. At this moment the person with the towel stops moving and waits for the bird to habituate to their presence and present any desired behavior, such as engaging in preening or other acceptable activities. When this is observed, the person with the towel calmly takes a few steps away from the bird. In some cases of history of extreme fear responses, it is better to walk away even before any slight response is observed. This process is repeated and with each repetition the person with the towel attempts to get closer before a response is observed.

When applied well, negative reinforcement can quickly teach a bird to present comfortable body language around a towel. Because the bird is now relaxed around a towel, this can then be transitioned to using positive reinforcement by providing desired food items, preferred touch, or experiences for interactions with the towel. This same strategy can be applied to syringes, medical instruments, new people, and so on. This is also helpful for teaching a bird that relaxing when restrained is the desired behavior. When a bird struggles, pressure remains constant. When the bird relaxes, pressure can be removed slightly. This can be gradually implemented so that very little pressure is required to maintain restraint resulting in a less-coercive restraint procedure. In both examples, the procedures teach the bird it has the power to cause the aversive situation to change by changing its behavior.

Conscious Sedation as Part of the Low-stress Avian Examination

Conscious sedation is a safe and effective option for minimizing the impact of fear and stress on avian patients. This underutilized tool should be considered in many contexts, even with debilitated patients. Ill birds should not be subjected to intense fear, anxiety, or stress and the associated physiological and psychological effects. Both benzodiazepines and opioids are very safe for the great majority of patients. Dosages of medications and route of administration can be altered if indicated to tailor the sedation protocol to the patient's condition and level of stress.

Unfortunately, many birds arrive at veterinary practices already so far past their stress threshold that it would take hours to hit the "reset" button, and neither the client nor the clinician can move forward at the pace the bird needs due to the client's schedule, other patients needing care, and so on. Rescheduling appointments for fearful patients may not be possible if the bird is presenting for illness and due to the infrequency of routine wellness visits in avian patients. The best option to help these patients while minimizing the effects of fear is often conscious sedation.

The decision to sedate a bird should be based on the individual birds' body language in response to environmental stimuli. In many cases, how the veterinarian and veterinary team handle the bird can make a huge impact on whether sedation is needed. For example, a mildly fearful bird that is given time to habituate to a new situation, paired with systematic and counter conditioning, may become less fearful and allow a procedure to be performed without restraint (Figure 46.15). However, if the bird is immediately restrained in a towel, the fear response is likely to escalate so far past threshold that no additional unrestrained procedures may be possible that day.

Bird owners may be fearful of sedation due to concerns about perceived risks, appearance of sedated birds, past aversive experiences, and perceived time constraints. If they have never had a pet sedated before, they typically will not have a frame of reference for the benefits of sedation and may not appreciate the positive impact this can have on their bird's experience. It is important for veterinarians to be able to clearly communicate their reasons for recommending sedation with bird owners in a way that demonstrates sensitivity to their fears and confidence in the medical recommendation. Some helpful strategies are listed as follows.

Many pet owners are unaware that stress can have severe long-term consequences to their pet's physical and emotional



FIGURE 46.15 Counterconditioning techniques by provision of food reinforcers can facilitate voluntary cooperation during the physical examination, as shown in this male eclectus parrot *(Eclectus roratus)*.

health and welfare. It usually is not difficult to convince the bird owner that their pet is experiencing fear, anxiety, and/or stress during the veterinary visit, as this is typically already one of their biggest concerns. The concept that sedation with benzodiazepines can decrease the development of fear memories, as well as the "needle-less" aspect of intranasal (IN) administration, is often very attractive to pet owners. Referring to sedatives as "anti-anxiety medications" can make them sound more palatable to many clients, as "sedatives" or "drugs" often carry a negative connotation. Comparing the use of anti-anxiety medications in the veterinary practice to the use of diazepam (Valium) or midazolam (Versed) for fearful humans in dentistry offices may also help the veterinarian connect to the client.

Some clients appreciate learning the difference between sedation and anesthesia, especially the safety and reversibility of the drugs utilized; however, reviewing this information in detail may prove more frightening for others. Discussing anesthetic/sedation monitoring protocols and staff expertise with bird sedation/anesthesia can help alleviate many client concerns. It may be helpful to mention how many avian sedation procedures are performed on average per week/per month and the rate of positive outcomes to illustrate your staff's competence.

Veterinarians must accurately and thoroughly explain the sedation procedure itself, as well as how the bird will look while sedated and during/after sedation reversal. This includes discussing duration of effects and warning clients that it may take several hours for the bird to begin behaving "normally" again, especially if there is underlying metabolic dysfunction (although this is unlikely with sedation reversal). Clients should also understand that each bird responds differently to individual dosages of sedatives and be aware of the range of "normal responses," which can range from completely unaffected to laying on their back and minimally responsive (Figure 46.16). They should also be advised that if this is the first time their bird has been sedated, the dose will be selected based on the patient's overall demeanor and perceived level of fear, anxiety, and stress; the patient's reaction to the chosen dosage will allow the veterinary team to tailor future doses to the patient.

Veterinarians should have a clear picture of their personal/ethical boundaries regarding willingness to forego sedation and attempt sample collection versus refusing to perform procedures without sedation in these patients. We hold responsibility as the medical experts to stand our ground on standards of care.

The goal of conscious sedation is to have the patient be standing, but very relaxed and calm. Most used protocols in birds utilize either midazolam alone or a combination of midazolam and butorphanol. These drugs are fully reversible, can be administered either IN or intramuscular (IM), and have a consistent onset of action. The depth of sedation achieved varies depending on the patient and dose.

Midazolam is a benzodiazepine drug with a wide safe dosage range in the medical literature.[46–53] The dosage range that is most helpful for reducing stress in most avian patients is between 0.5 and 3.0 mg/kg. Lower doses are more effective if administered IM or in combination with butorphanol. IN administration typically requires higher dosages to obtain the same effect. Dosages over 3 mg/kg should only be



FIGURE 46.16 While sedation helps to reduce the level of stress experienced by the bird, the level of sedation may not always be sufficient to fully eliminate all responses. This mildly sedated macaw is showing signs of stress related to towel restraint, including panting, ruffled feathers, and drooped wings.

administered IN. Onset of action is consistently less than 5 minutes postadministration, regardless of the route of administration; duration of action ranges from 20 minutes to 2 hours.[46] Midazolam does not have any analgesic properties but may prevent creation of additional fear memories due to its amnesiac properties.[54] Side effects include transient ataxia, disinhibition of aggression, nausea, transient dysphoria following reversal, and increased appetite.[46,55–57] A mild monocytosis may be seen on hematology associated with this drug (Kendall Harr, unpublished data, 2018).[14] Undesirable side effects, such as disinhibition of aggression or nausea, are typically resolved following reversal with flumazenil (0.025-0.1 mg/kg). These events should be recorded in the medical record and a different protocol or dosage should be considered in the future.

Butorphanol is rarely effective as a sole sedative but can enhance the effects of midazolam and functions as an effective and safe analgesic in many avian species.[46,50–53] It is an opioid analgesic with partial agonist/antagonist activity at the mu receptor and partial agonist activity at the kappa receptor. Administration of 1.0 to 4.0 mg/kg butorphanol IM or IN in conjunction with midazolam should be considered for birds undergoing potentially uncomfortable/painful procedures, or for birds that are not adequately sedated with midazolam alone.[52] This combination sedation protocol typically results in smoother induction and recovery in many species, particularly macaws. Onset of duration is less than 5 minutes, and duration of action ranges from 2 to 4 hours. Butorphanol is reversible with naloxone 0.04 mg/kg IN or IM. Side effects of butorphanol may include transient ataxia, excitement,

respiratory depression, and mildly decreased blood pressure and heart rate. Butorphanol should be avoided in cases of central nervous system trauma and dysfunction, as this may hinder the neurological assessment and can induce respiratory depression, thereby exacerbating cerebral hypoxia and raising intracranial pressure.[53]

IM administration of sedatives typically results in a rapid onset of more marked sedation compared to intranasal administration, and this route of administration is preferable for radiographic imaging or more delicate procedures. IN administration is often preferred by clients as it does not require any needle punctures, but many birds tolerate small-volume IM injections surprisingly well. IN administration may be easier to accomplish with less restraint in some patients and carries a lower risk of bleeding or bruising. Syringes should only be used without the needle present to avoid accidental damage to the patient's nares during administration. Patients may sneeze or show mild upper respiratory congestion immediately after intranasal administration.

IN midazolam (2.0 mg/kg) provides excellent standing sedation for most patients that are experiencing mild to moderate stress, allowing physical examination, phlebotomy, and other minor procedures to be performed without distress to the patient. Lower doses may be adequate for some patients (such as cockatoos, or very sick patients). The addition of 1.0 to 2.0 mg/kg butorphanol should be considered if the procedure has the potential to cause discomfort to the patient, if the patient is highly stressed, or if the procedure requires a heavier level of sedation (diagnostic imaging, for example). Administration of midazolam 2.0 mg/kg ± butorphanol 2.0 mg/kg IN usually results in a bird that can stand, although perching may not be possible. IM administration at these same doses tends to result in birds with a much higher level of sedation; some patients may be ventrally or dorsally recumbent following onset of effect. This can be frightening for clients to observe, and veterinarians should provide a detailed description of the possible range of symptoms that their pet might exhibit prior to any sedation event to help prepare them for this appearance.

Following the physical examination and/or sample collection, midazolam can be reversed with flumazenil. The dosage range of flumazenil is 0.025 to 0.1 mg/kg, and the medication can be administered IN or IM. IM administration may lead to dysphoric or other undesirable responses, especially if serious underlying medical conditions exist; IN administration typically results in smoother recoveries. A 0.05 mg/kg dose administered IN is effective for most patients. Since the half-time for flumazenil is shorter than that of midazolam and diazepam, this procedure can be repeated if reversal is inadequate. If butorphanol is used in the sedation protocol, reversal with naloxone is usually unnecessary.

Sometimes it may be preferable to discharge the patient without reversing the sedative to allow a more gradual recovery and decrease stress of the trip home. This may be appropriate if the sedation was extremely mild, if transport is historically excessively stressful for the patient, if the client typically has difficulty transporting the patient from the carrier into the home enclosure, or if the patient's medical condition would benefit from administration of anxiolytics or analgesics. Any decision to avoid sedation reversal must be discussed thoroughly with the client, including potential complications and benefits. One of the potential risks of this decision is the patient may become more sedated after leaving the practice due to drops in adrenaline levels; this could precipitate a fall or injury. Discharging these patients in a flat-bottomed, dark enclosure without bars to discourage the patient from either attempting to perch or hang on the side of the cage is advisable to reduce this risk. Cardboard disposable pet carriers may serve admirably as long as they are too sedated to attempt chewing through the cardboard and escaping. It may be beneficial for the practice to keep a few flat-bottomed plastic carriers available to loan out to clients for the trip home for birds that are more awake or facing a longer car ride; these carriers can be returned to the practice later and reused.

Sedated birds should not be discharged in the same enclosure as other birds until the effects of the sedative have fully worn off, as abnormal behavior associated with sedation may precipitate aggressive responses from others.

Other sedation/anesthetic options

Other reported IN or IM sedatives in birds include diazepam, xylazine, detomidine, dexmedetomidine, and ketamine.[46,50,51,53,55,56,58–61] However, all these drugs have disadvantages compared to midazolam and butorphanol, and effectiveness/safety varies significantly. They should not be considered first-line choices for sedation in avian patients.

General anesthesia may be more appropriate than conscious sedation for some patients and some medical procedures, including prolonged or delicate procedures (e.g., splinting a fractured bone), and surgery. The most frequently used anesthetic agents in birds are isoflurane or sevoflurane gas.[53,62,63] Both of these anesthetics are effective in birds and relatively safe. However, there are more risks of serious side effects such as hypotension, respiratory depression, and arrhythmias compared to conscious sedation. Additionally, gas anesthesia does not provide analgesia.

At-home sedation?

A mainstay of Fear Free™ dog and cat medicine is to have pet owners administer sedatives at home so that the animal does not experience increasing fear, anxiety, and stress on the way to the veterinary practice. In an ideal world, this would be an option for bird owners, as well. However, there are several reasons why this is logistically challenging at the present time. Most bird owners do not seek veterinary care until their patient is sick, and as such frequently do not have the valid veterinary-client-patient relationship legally necessary for medication dispensation. Bird owners may not have an ability to accurately assess the health status of their pet, which would influence the safety of medication administration, and many bird owners are unable to handle their birds safely or administer medications to them. The most obvious reason is there are currently no proven, safe, consistently effective oral sedation options for birds. Options that may be considered in the future include gabapentin and tramadol, but appropriate dosages are unknown and may vary significantly depending on species. If a valid veterinary-client-patient relationship exists, and the client can safely/effectively handle their bird, a single dose of midazolam can be dispensed for intranasal use at home prior to transport to the hospital.

Diagnostic Sample Collection

An organized and thoughtful approach to a diagnostic protocol will help to reduce patient stress. Developing a list of differentials for the current symptoms provides a targeted list of indicated diagnostic tests. This prevents the bird from being subjected to unnecessary procedures. In larger birds, it may be desirable to draw a larger blood sample than needed for initial testing. This allows the practitioner to avoid collecting additional blood samples if further testing is indicated by the results. It will also prevent any alteration of the test results secondary to early supportive care and treatments. This is not possible for smaller birds and the decision to do so must be evaluated considering the patient's stability.

In some cases, it may be beneficial to postpone sample collection to allow for the client to spend time training the patient to voluntarily accept restraint and potentially participate willingly in medical procedures. For example, a healthy, young parrot that is presenting for a wellness examination and is owned by a dedicated pet owner with an interest in training and behavior could have routine screening phlebotomy postponed until a training protocol has been implemented. It may be challenging to obtain compliance in these situations, so building value for this process with client education and providing appropriate resources and support for training is essential.

Developing a Treatment Plan

When designing a treatment plan, the most effective therapy with the minimum of undesirable effects should always be selected. One of these undesirable effects is increased stress. Intravenous or intraosseous fluid therapy is highly effective in many situations but can be stressful. Subcutaneous fluid therapy may be preferred in many patients, especially small birds. When selecting drugs, those with the longest dosing intervals should be selected if all other factors are equal. Some injectable drugs are more painful than others. Some oral drugs have extremely unpleasant flavors. Some suspensions require a huge volume to be administered. All of these factors should be considered when the plan is developed.

Outpatient treatment is usually less stressful than inpatient treatment. However, if the client will be medicating the bird at home and has not previously trained this behavior, the veterinary staff must help them develop a plan for this to be accomplished and demonstrate how to administer the medications safely and effectively (Box 46.5). In general, hospitalization should be reserved for patients that require treatments that are beyond the client's capabilities.

BOX 46.5 METHODS FOR SAFE AND EFFECTIVE ADMINISTRATION OF MEDICATION BY BIRD OWNERS

- Training the bird to voluntarily accept medication from a syringe
  - Use a syringe that glides easily. Fingers should be wrapped around the syringe, while the plunger is pressed with the thumb for optimal control.
  - Habituate the bird to the syringe by leaving it in sight at a distance where it does not elicit a fear response. Reinforce engagement with the dosing syringe. This can include voluntarily approaching the syringe or allowing it to be brought closer to the bird. Potential reinforcers can include preferred food items, toys, attention, or praise.
  - Target training using positive reinforcement can be used to teach the bird to touch the tip of the syringe with its beak or tongue. The syringe should be held at a distance where the bird can barely touch it with its tongue (which will help prevent it from biting the syringe). If a bird does not reach for the syringe, the desired treat or toy may be positioned near the tip of the syringe to prompt desired responses.
  - Once the bird is fluently touching the tip of the syringe, add water to the syringe. Reinforce for touching the syringe with water in it. Next, a drop of water can be suspended from the syringe. Reinforce for drinking the drop of water from the syringe. If the bird shakes its head following touching the droplet of water, go back to the syringe training without the drop of water and rebuild reinforcement history.
  - Once the bird readily accepts the droplet of water, gradually increase the volume of fluids delivered before reinforcing.
  - Expand the procedure to include delivering palatable fluids from the syringe. Reinforce for taking these fluids from the syringe. Follow this step by offering less-palatable fluids from the syringe such as diluted apple cider vinegar. Start with small amounts first and gradually build the amount upon acceptance.
  - Preferably, this procedure is trained prior to the need for medication.
- Use of food vehicles to hide medication, which the bird readily accepts. It is recommended to prepare the food away from the bird, as this may increase the chances that the food is accepted. The best vehicles mix easily or act as an absorbent, have high perceived value for the animal, and are small enough to encourage rapid consumption, yet large enough to hide the taste of medication. Examples of commonly used vehicles in birds include the following:
  - Parrots: hand-feeding formula, human baby foods, peanut butter, oatmeal, winter squash, sweet potato, small pieces of fruit (e.g., banana, grape) or fruit juice, yogurt, scrambled eggs, small pieces of bread, crackers, or pastries soaked in liquid medication
  - Poultry or waterfowl: grapes, cheese, tomatoes, bread
  - Birds of prey: prey animals
- Compounded drugs can be flavored to help mask unpleasant tasting medications to facilitate acceptance. For medications of which the taste cannot be effectively masked, hiding in vehicles is not recommended as the bird may start avoiding the preferred food. In such instances it is better to administer the medication directly, followed by provision of a favored treat upon acceptance of the medication. Use of gentle handling techniques and toweling is recommended, and owners should be taught and demonstrated on how to apply these techniques at home.

Hospitalization may be required in situations where owners are hesitant and unsure as to whether they are capable of delivery of medication. In cases where owners are willing to attempt medication at home, a follow-up consultation by phone is recommended to evaluate the success of treatment.

Hospitalization

All birds experience some degree of increased stress when away from their home environment. Stressful stimuli include unfamiliar sights and sounds, restraint required for medical treatment, and hospital housing conditions. Consider the natural history of the species when designing housing for birds in your avian ward. Some species such as parrots and raptors may prefer housing that is elevated, while other species such as large waterfowl may prefer enclosures lower to the ground. The barking of dogs, the sight of cats or even larger birds, and excessive staff foot traffic can all increase the stress of hospitalized birds. Wards should be kept separate from canine and feline wards and hospital cages should face in a direction that allows minimal visual contact from one cage to another (see also Chapter 1). Providing a visual barrier, such as a towel or newsprint taped to the front of the incubator or cage, gives the bird the ability to hide from staff attention while still allowing regular patient evaluation. Providing soft background noises (such as a white noise machine or classical music) to balance out sudden noises is also recommended. Some companion birds may respond favorably to social interactions with hospital staff members independent of procedures. Birds should have access to their regular diet while hospitalized to increase their willingness to eat. Clients should be encouraged to visit the bird in the hospital and potentially to bring in other familiar items from home that may increase comfort (toys, perches, caging, etc.; Figure 46.17). Some hospitalized birds will do better if housed with a companion bird; however, sick birds may also be traumatized or bullied by conspecifics, so this should be evaluated on a case-by-case basis.

Pain Management

One of the primary responsibilities of the veterinarian is to reduce pain and suffering. Pain should be controlled by the selection of the least painful treatment option, by preventing self-inflicted trauma, and by the judicious use of analgesic drugs when indicated (see Chapter 35 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 20, Recognition, Assessment, and Management of Pain in Birds).



FIGURE 46.17 Enrichment opportunities are important for hospitalized avian patients. When hospitalization is anticipated, clients should be encouraged to bring familiar items such as toys, perches, towels, cages, and so on, from home along with the visit to increase comfort. Even the boarding of a cage mate or companion can be considered to minimize stress during the hospitalization period. However, as sick birds may also be bullied or traumatized by conspecifics, this should be evaluated on a case-by-case basis.

Grooming

Birds can develop significant problems because of inappropriate grooming. Excessive wing trims can cause birds to be clumsy, frequently resulting in injuries to the beak tip, sternum, wings, and tail. The subsequent pain can lead to excessive levels of stress in these birds. Techniques for trimming wings are somewhat controversial, but clearly the cutting of all the primary and secondary feathers is excessive (Figure 46.18; see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 22, Advancements in Management of the Welfare of Avian Species). Cutting the nails short enough to induce bleeding is a painful procedure that can make birds more fearful of restraint. Beak trimming is never required in a healthy bird. The stress associated with beak trimming can be life-threatening, especially considering the possibility of a severe underlying disease process. For this reason, strong efforts should be made to attempt to determine and treat the underlying cause for the beak abnormalities to minimize ongoing risk (see Chapter 31).

POSTEXAMINATION PRACTICES

If an examination resulted in excessive stress to the patient, consider short-term supplementation with omega-3 fatty acids, which has been shown to be neuroprotective following traumatic brain injury in rats.[64–68] The authors recommend the use of omega-3 fatty acid supplementation (VetOmega, North Salt Lake, UT, 84054, USA) at a dose of 0.22 to 0.44 mL/kg orally once daily for 3 to 5 days. Benefits of prescribing this treatment must be weighed against the stress of administration. This supplement may be administered on a preferred food item to minimize stress.

The CSR should check the client out in a calm place (ideally, the examination room). If examination room checkout is not possible, the client should be escorted to a quiet place in the receiving area with the same precautions that were in place for check-in. If the weather permits, the patient could even be returned to the car and the client return to the practice for checkout.

Upon completion of the veterinary visit, team members should plan to reach out to the pet owner within 24 to 48 hours to investigate their perceptions of the visit. This information should include the following questions:

1. How is your bird doing today?
2. How do you think your bird responded to the veterinary visit?
3. If applicable: How did your bird respond to the sedation? How long were the effects noted? Do you think it helped reduce your bird's stress level?
4. Is there anything we could have done better?

Other questions may also be appropriate depending on the procedures that were performed during the visit and the individual bird's medical condition.

Management of the Medical Record

The medical record is generally centered around medical information, typically using the SOAP (Subjective, Objective, Assess-

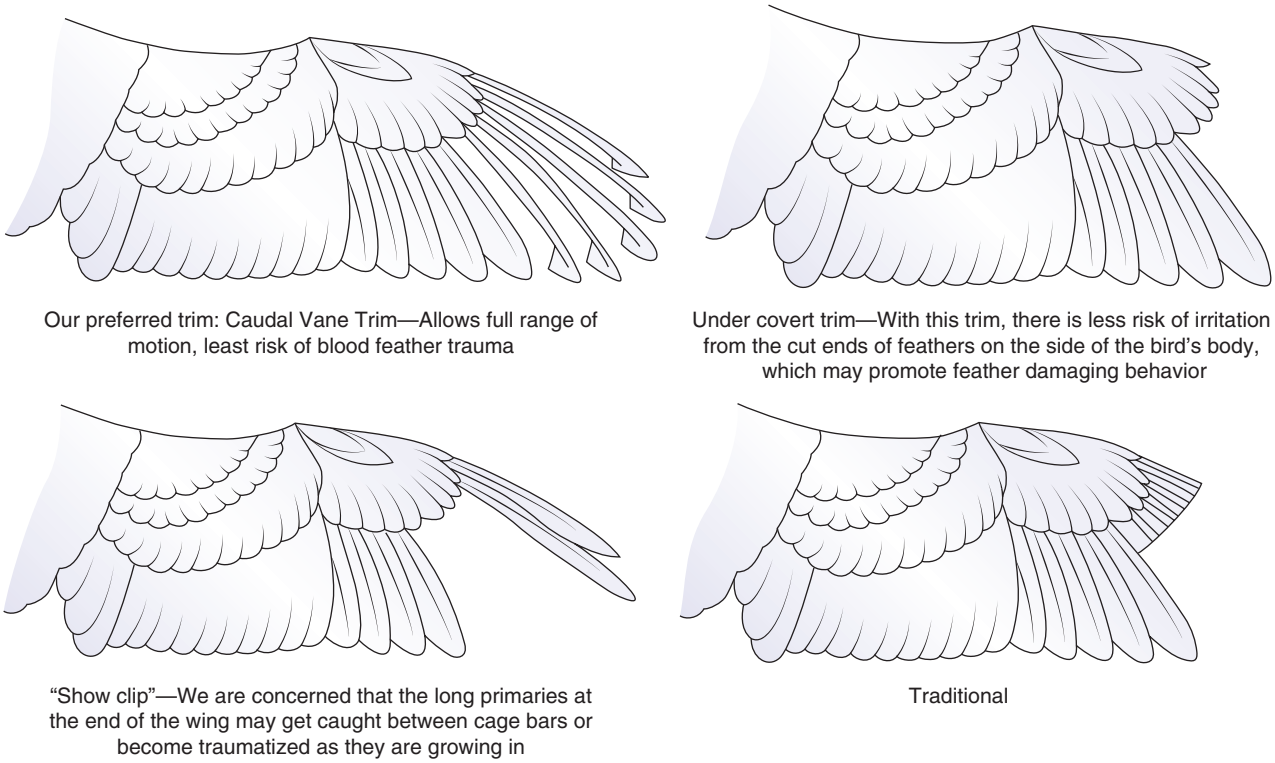


FIGURE 46.18 Wing trim preference form.

ment, Plan) format. Including details about behavior in the medical record is an excellent way to provide consistent interactions from visit to visit and from one staff member to another. It also provides a way to track progress with training goals. Information that should be added to the record includes the following:

1. Information about the patient's demeanor during history gathering
2. How the patient reacts to staff
3. Presence of towel (if needed), and how it was used and responded to during the visit
4. Flight ability
5. How well the client can manage handling the patient in the examination room versus at home

There are many practice management software options that allow development of an Electronic Medical Record (EMR). These typically provide options to create templates for medical notes, which can be used to create consistency of information gathering/reporting. While behavior information can be included with each SOAP, some clinicians prefer to place it within an individual behavior note/alert that staff can see quickly. Regardless of the chosen method, training of doctors and staff to use the system consistently is required for maximum efficacy. It is critical for both detailed notes to be recorded from each visit, as well as previous information reviewed prior to stepping into an examination room with a bird that has been seen before.

FACILITATING STRESS-FREE FOLLOW-UP CARE

Follow-up care may require training of clients and patients to facilitate proper administration of treatment protocols. Veterinary staff should facilitate this by following these steps:

- List treatment protocols required for patient follow-up care.
- Identify patient treatment protocols that can benefit from on-site training by veterinary team.
- Implement training to gain voluntary cooperation of the patient prior to discharge (oral medications, scale training, etc.).
- Identify patient treatment protocols that will require maintenance by the client and therefore client education.
- Identify additional behaviors that will further reduce or eliminate stress for future medical care of the patient.
- Identify and prioritize behavior goals list for the patient.
- Provide education/educational resources for the client to facilitate achieving these goals.
- Create follow-up plan with the client to facilitate success and compliance.

FOUNDATION BEHAVIOR GOALS

Helping clients further develop the behavioral repertoire of their companion birds is an important opportunity to reduce or eliminate stress for veterinary care. Veterinary professionals can enhance the bond between practice and client by providing guidance, resources, and support to achieve these goals. Healthy patients can often be trained during a well-bird examination to present behaviors such as targeting, voluntarily stepping onto a scale, and to accept fluid from a dosing syringe. This can demonstrate how quickly a behavior can be trained that can impact cooperation in medical care (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).

Clients can be given the opportunity to practice under the guidance of the veterinary professional to ensure success and gain confidence. Self-study educational resources such as handouts, videos, books, and referrals to additional materials can be provided to continue making progress at home. This can lead to training additional behaviors to facilitate stress-free medical care, such as allowing restraint in a towel and voluntarily loading into a crate or other transport container (Figure 46.19).

While ultimately improving animal welfare is the incentive for implementing a training protocol, client compliance is not guaranteed. Veterinary professionals can help increase participation by reinforcing clients. This can be done by featuring clients (with permission) on social media, in the waiting room video feed, or bulletin board. Clients can also be offered items such as free dosing syringes, product samples, or discount coupons after successfully training a behavior.

It can also be helpful to take an incremental approach to building a repertoire of behaviors. Allow clients to have success with one behavior. Celebrate this success, before providing tools and resources to work on the next behavior. Overwhelming a client may lead to inaction instead of enthusiasm.

Depending on the patient, some of these behaviors can be trained while on site. These behaviors may include target training, touching the tip of a dosing syringe, and standing on a scale. Many of these behaviors can be shaped and trained, while also administering treatments with restraint for hospitalized patients. A key to facilitate this requires careful attention to reading patient behavior and minimizing the severity and degree of disempowerment or fear generated during treatments as well as daily reassessment of patient response to treatment procedures, particularly when the patient has worked through desensitization and counterconditioning and may also be employed for patients during the process of ongoing treatment of medical problems. Other patients require an investment from the client, which can be supported with educational resources provided or referred by the veterinary staff. Periodic rechecks by phone or in-practice ensure the client's success in their training progress by addressing questions and offering feedback/reinforcement for the client. Depending on what staff is available to provide these training checkups, the veterinary hospital can enlist the help of a trainer as well.



FIGURE 46.19 Transport containers come in a wide variety of designs. Creative thinking can lead to clever approaches for teaching birds to voluntarily enter for desired items to help reduce or eliminate stress.

FOSTERING GROWTH IN THE INDUSTRY

Veterinary medicine and evidence-based animal training technology are two disciplines with a common goal of attending to high standards in animal welfare. Because of this, we have seen tremendous growth in training birds to cooperate in medical care in recent years. Progressive veterinary professionals may find it advantageous to have team members skilled in training birds for behaviors beyond the basics. This can include behaviors such as the following:

- Nail trimming (Figure 46.20)
- Tactile examination (Figure 46.15)
- Voluntary auscultation
- Voluntary injections
- Voluntary intranasal drug administration
- Voluntary mask induction (Figure 46.21)
- Voluntary phlebotomy

Behaviors are commonly trained by breaking them down into small steps or approximations. These are described as shaping plans.

See the following list for a Voluntary Injection Training Shaping Plan[69]:

- Reinforce for stationing in a specific location or perch
- Build duration for stationing
- Use stimulus fading to introduce syringe without needle to pectoral muscle
- Transition to touching the body first and delivering reinforcers afterward



FIGURE 46.20 Training birds to cooperate in grooming procedures can be a realistic goal for clients. Veterinary professionals can facilitate by providing instruction and/or directing clients toward trusted resources that focus on force-free training.



FIGURE 46.21 By breaking down complex behaviors into small steps or approximations, even more complex medical behaviors can be taught through the process of shaping and positive reinforcement. For example, this brown-headed parrot *(Poicephalus cryptoxanthus)* has learned to voluntarily place its head into the anesthetic mask through target training.

- Add capped needle to syringe and repeat process of introducing syringe
- Repeat process using a dull metal object such as an open paper clip
- Repeat process using a toothpick
- Repeat process using a blunted needle on syringe
- Repeat process using a rubber band and snap against pectoral muscle (increase intensity gradually)
- Pierce skin with sharp needle on syringe
- Inject a small amount of saline

To train a bird to allow injections, begin by reinforcing for staying in a particular location for duration. This can be a designated perch or mat on the floor or table. For birds of prey trained to sit on the glove, this behavior can also be accomplished while the bird is perched on a gauntlet.

As with introducing nail trimmers, towels, etc., a critical step is introducing the syringe to the pectoral muscle in a manner that does not evoke a fear response. A good method is to offer preferred food items and slowly moving the syringe closer while observing body language (stimulus fading). Once introduced successfully, transition to touching first and reinforcing after.

The subsequent steps of this shaping plan are designed to teach a bird to tolerate many different sensations including different levels of pain. Before proceeding to any new sensation, the bird's body language should be relaxed and comfortable while experiencing being touched with the current object. Snapping the chest with a rubber band simulates the sting of some medications. The intensity of this can be increased gradually.

By the time you are ready to pierce the skin with a sharp sterile needle, the bird will have experienced many different sensations and remained calm and relaxed throughout the process.

Piercing the skin and/or injecting saline solution should only be done one time during a session and only once every few weeks to maintain the behavior. The location should change each time to avoid injecting into the same location within the same muscle.

Select clients may also show enthusiasm for training beyond the basics. Those team members skilled in training can foster client growth and development. The more support an eager client has to help teach their bird to cooperate in medical care, the more potential to reduce or eliminate stress for future health care. Additional benefits include that the client is more likely to collect data such as regular weights, preventative care is more easily accomplished, and illness may be detected earlier.

Step-by-step instructions to train the behaviors described in this chapter can be found at these outlets:

Stress Free Veterinary Care for Parrots by Barbara Heidenreich https://vimeo.com/ondemand/parrotvetcare

Train Your Parrot for the Veterinary Exam by Barbara Heidenreich https://vimeo.com/ondemand/parrotvetexam/336936924

Training Hand Injections Course www.AnimalTrainingFundamentals.com (Animal Training membership site for professionals by Barbara Heidenreich)

Fear Free™ Pets Avian Course www.FearFreePets.com

Veterinary Clinics of North America: Exotic Animal Training and Learning, An Introduction to the Application of Science Based Training Technology https://www.vetexotic.theclinics.com/article/S1094-9194(12)00061-8/fulltext

CHOOSING A TRAINER OR BEHAVIOR CONSULTANT

Current technology allows information to be easily shared across many platforms. Many clients look to the Internet for advice when seeking assistance for a companion animal behavior problem or training challenge. The veterinary professional can be an important resource for guiding clients toward quality information that teaches best practices. Animal training/behavior consulting is not a regulated industry. Therefore, it requires critical thinking to evaluate credentials. Ideally the veterinary professional can make a trainer/behavior consultant recommendation based on a working relationship, known use of force-free training strategies, client testimonials, and proven results.

As with any skill-based profession, it can be challenging to evaluate an individual's expertise. Many biographical sketches include words such as "professional," "certified," "published," "international speaker," "President," "Director," "CEO," "emeritus," "PhD," and a long list of memberships to professional organizations, speaking engagements or other potentially exaggerated credentials. For many, these words are accurate descriptions of their qualifications. For others these may require further exploration.

Important questions to ask when evaluating certifications include: What was measured? Was knowledge or practical application skills measured? Who did the measuring? How often is this measured to maintain certification? Are courses required? Must they show case work? Is testing required? Must the applicant reapply regularly? And even with certification, does it prove this person can address your needs?

Important questions to ask when evaluating a PhD include: Is the degree relevant to this field? Does he or she have practical application experience? Does he or she run a lab, practice, do research? Does he or she oversee/produce

graduate students? Does he or she teach in a university? Has he or she published in peer-reviewed journals? What professorship rank did he or she achieve?

Words like "international speaker," "president," "CEO," 'Director," "Emeritus," and "all species" are often used to exaggerate one's credentials or experience. An international speaker may have only presented in two countries. Titles can be self-appointed, especially if the person in question owns the company. Emeritus is often inferred to mean a distinguished professor who left a legacy with a university and still holds the title. However, a retired PhD without such a contribution may request to use this designation as well. Saying one trains "all species" is quite a statement, and hopefully a red flag for a critical thinker.

There is a difference from being an expert and being *perceived* as an expert. Experts do not require degrees, titles, certifications, years accrued, memberships to professional organizations, popularity, Internet visibility, although they may certainly have these things too. They are less likely to exaggerate credentials or misrepresent experience. Experts can produce solutions to problems rapidly based on experience with many prior situations. It becomes intuitive from so much practice of putting all the tools of practical application together to get results over and repeatedly.

To help with the process of choosing a trainer, invite them to give your hospital a "Lunch and Learn" to introduce themselves and allow staff members to learn and ask questions. A good working relationship with a trainer should have good communication between veterinarian, trainer, and pet owner. This includes training and behavior notes and SOAPs in the patient's record from both veterinarian and trainer, like adding records from a referring veterinarian.

Animal training/behavior consulting is a practical application–based skill that is also rooted in learning science. Read biographical sketches with a critical eye. But most importantly choose an animal trainer/behavior consultant who demonstrates the following criteria[70]:

- Your animal trainer/behavior consultant should be science- and evidence-based. This means they understand the science of behavior analysis. They will be able to communicate to you about topics such as operant conditioning, the four quadrants, classical conditioning, counter conditioning, and systematic desensitization. Your consultant should provide data-driven resources and also be interested in collecting, measuring, and analyzing data when providing interventions.
- Your animal trainer/behavior consultant should be focused on effective, efficient, and optimal (maximizes benefits/minimizes harms) intervention strategies (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). This means they understand which behavior modification principles and procedures to choose to effectively address behavior challenges and reach behavior goals while at the same time maintaining high standards in animal welfare.
- Your animal trainer/behavior consultant should be able to demonstrate excellent practical application skills. This can include a sensitivity to animal body language, an ability to respond in a manner that creates a relaxed and comfortable animal, excellent mechanical skills (timing, observational skills), ability to arrange environments for success, etc.
- Your animal trainer/behavior consultant should be knowledgeable about the natural history of the species. They should understand ethology, phylogeny, modal action patterns, and their role in species-specific behavior.
- Your animal trainer/behavior consultant should show transparency and integrity, meaning they will openly discuss behavior intervention methodologies and their limitations and not misrepresent their experience.

RESOURCES FOR CLIENTS

Resources for Parrot Behavior Problems from Barbara Heidenreich www.ParrotBehaviorProblems.com

Stress Free Veterinary Care for Parrots by Barbara Heidenreich https://vimeo.com/ondemand/parrotvetcare

Train Your Parrot for the Veterinary Exam by Barbara Heidenreich https://vimeo.com/ondemand/parrotvetexam/

IAABC International Association of Animal Behavior Consultants, Find an animal behavior consultant directory https://iaabc.org/consultants

Avian Behavior Lab https://avianbehaviorlab.com/

Science-based articles on behavior, Susan Friedman, PhD http://www.behaviorworks.org/htm/articles_behavior_change.html

Pamela Clark, CPBC, Behavior and Training Consulting Services http://pamelaclarkonline.com; http://blog pamelaclarkonine.com;; pamelaclarkcvt@gmail.com

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CHAPTER 47

Strategic Use of Enrichment: What, When, and How

Kenneth R. Welle • Melody Hennigh • Brian L. Speer • Yvonne R. A. van Zeeland

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INTRODUCTION

Taking any animal into the home carries a great deal of responsibility. While the basic needs of food, water, and shelter are obvious, it is also necessary to provide for the more abstract needs of companion animals. Psittacine birds are intelligent and socially complex animals that can suffer physical and behavioral consequences when these needs are not met. So much so that the suitability of parrots as companion animals is sometimes questioned.[1] Captivity, by necessity, restricts some natural behaviors and, in response, alters time allotment to others, setting the stage for development of new (displacement) behaviors to fill the void. A wide variety of behavior problems are prevalent in the companion parrot population, including biting, screaming, feather destructive behavior, and other abnormal repetitive behaviors (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).[2] Enrichment is often seen as means to prevent these undesirable behaviors. Additionally, enrichment increases possibilities for animals to display appropriate, species-typical behaviors, allows them to exert control over the environment, and increases their adaptability to environmental changes, thereby reducing inappropriate fear responses and stress (Box 47.1).[3–7]

Enrichment is defined as the act of improving the quality of something, or the act of making it better. In the case of captive animals, this means making their life better. Enrichment in the broadest sense involves creating a life that provides for all physical, psychological, and emotional needs of an animal. The larger the behavioral spectrum an animal has, the more challenging but critical this enrichment will be. In this chapter we will explore some methods of enrichment that are appropriate for companion parrots. Some will involve trying to replicate natural behavioral opportunities for these birds, while others will involve replacement behaviors that can benefit the bird rather than creating further stresses.[5,8–11]

TIME BUDGETS

One of the challenges of enrichment is the drastic alteration of the time budget in captivity compared to the time budget of a wild conspecific.[12,13] Although time budgets for wild animals can vary considerably across individuals, species, and throughout the year, some things are reasonable to assume. For instance, tropical (equatorial) birds would typically have a light cycle approximating 12 hours of light and 12 hours of dark. This reduces the range of time that birds will be active. Those inhabiting ranges farther from the equator will have day length that fluctuates with the season. In these species, the changing day length is often involved in regulation of certain endocrine processes, such as reproduction and molting. Companion birds are often tied to the daily schedule of their caretakers. In many cases, this creates a consistently long photoperiod. Long days are inherently more difficult to fill with enriching activities for these birds. Additionally, long day length is often linked to the breeding season for seasonal, long-day breeders. This stimulus can create further stress and frustration when appropriate conditions for breeding are not available. One relatively simple measure to prevent this is to reduce light cycles to somewhat short, consistent duration throughout the year. While this can be challenging with some schedules, it is usually much easier to accomplish than creating adequate activities for an active, intelligent parrot.

The daylight hours may be utilized for feeding, socializing, performing self-maintenance, moving to and from feeding areas, and playing. Of course, many of these usages overlap significantly. Time cannot always be strictly budgeted into one category. Feeding, self-maintenance, and play are all social activities, so a given hour may be budgeted to more than one category and the total time budget would exceed 24 hours. In captivity, some of these activities are necessarily restricted. This void will sometimes force a shift toward other activities or create pathologic displacement behaviors such as stereotypies, apathy, or self-damaging behaviors. Much of the goal of enrichment is to manage the time budget to recreate the activities of a wild bird or to replace proportions of them with healthy, stimulating, and appropriate activities.[14–16] Figures 47.1 to 47.3 show some sample time budgets.

Photoperiod

Most psittacine birds come from tropical to subtropical latitudes. The photoperiod in these areas varies minimally and will always be close to 12 hours of light and 12 hours of dark. By comparison, most pet birds have prolonged day lengths. This affects the time budget significantly by reducing the amount of dark, quiet time when the bird would be sleeping. As in humans, it can be assumed that inadequate sleep would be a significant stress to these birds. Additionally, it creates a greater void in the time budget that must be filled in some way. Although photoperiod is highly critical for signaling

BOX 47.1 POSSIBLE GOALS WHEN DEVELOPING AN ENRICHMENT PLAN

- Prevention or reduction of problem behaviors, including abnormal repetitive or stereotypic behavior.
- Offer choices that enable the animal to exercise control over the environment (empowering autonomy).
- Promote expression of natural, species-typical behaviors (e.g., foraging behavior).
- Stimulate activity and physical fitness, offer opportunities for exercise.
- Provide mental stimulation by increasing complexity of the environment and/or stimulating problem-solving capabilities.
- Increase variability and diversity in behavior.
- Increase (positive) utilization of the enclosure or living environment.
- Increase the ability of the animal to cope with changes and challenges (increased adaptability and resiliency).
- Stimulate a healthy owner-bird relationship.

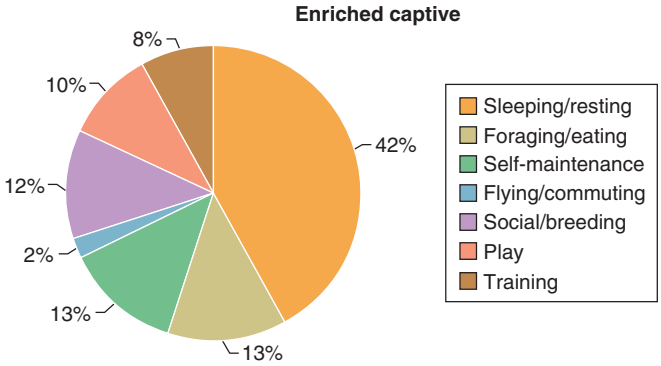


FIGURE 47.3 This graph represents a bird with an enrichment program in place. The sleep time is increased to 10 hours daily. A robust foraging program increases the foraging time to 3 hours daily. Training occupies 2 hours daily and the bird engages in flight exercises for 30 minutes daily. Ample opportunities for play and social interactions are made available.

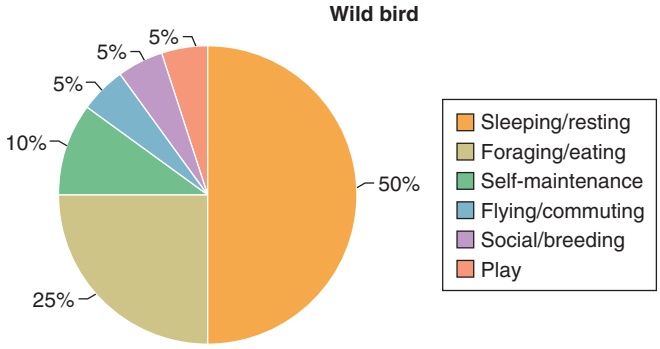


FIGURE 47.1 Sample time budget for a wild bird. While time budgets vary greatly between species and even seasonally within a species, some generalizations can be made. Many psittacine birds are tropical to subtropical, meaning that their light cycles will tend to be around 12 hours of light and 12 hours of darkness. Foraging for food, preening and other self-maintenance behaviors, play, and flying make up the remainder of the time. Nearly all of these behaviors are done socially.

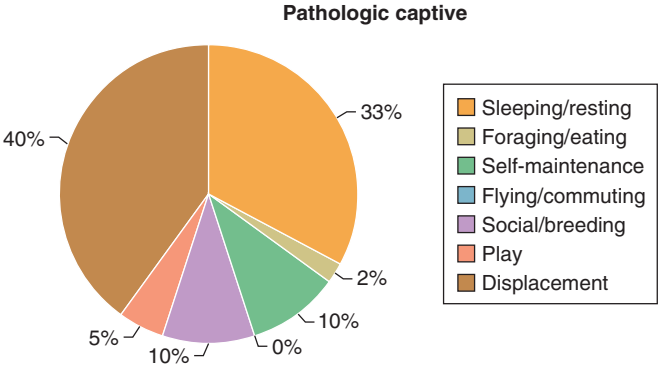


FIGURE 47.2 Sample of a pathologically skewed time budget for a captive bird. This bird gets only 8 hours of darkness each night. Foraging time is negligible. If other behaviors remain similar to wild birds, there remains 40% of the time for displacement behaviors to occur. Displacement behaviors often are pathologic, although there are beneficial displacement behaviors as well.

reproductive cycles in temperate species, in tropical species these effects will be variable. Other parameters such as food abundance, rain, and availability of a partner and/or nest site may have a larger effect in these cases. When there is photoperiod dependence, it is primarily dependent on changes in the light cycles rather than the actual day length, with prolonged exposure to constant photoperiod inducing photorefractoriness during which the physiological and behavioral changes normally induced by prolonged photoperiod are no longer recognized.[17] Thus, whenever possible, maintaining a consistent cycle of 12 hours of light and 12 hours of dark is preferred over a constantly changing light:dark schedule. While this rarely is practical for those with typical work schedules, the goal should be to stay as close to this schedule as possible.

ENERGY BUDGETS

Most birds are naturally high-energy creatures. The various activities of wild birds require a high caloric input. Flight is the most obvious high-energy activity. During flight, oxygen consumption goes up by about 1300%, and birds generally have energy requirements that are 50% to 75% higher than an equivalent-sized mammal.[18] In managed care, flight is generally restricted at least to some degree, sometimes via feather trimming and other times as a consequence of confinement to enclosures inadequate in size for flight. Additionally, many of these birds are provided unlimited access to high-energy diets. The resulting caloric surplus can either be manifested in an overabundance of active energy or in deposition of adipose. Although providing an outlet for flight may be the most efficient means of utilizing this energy in a healthy way, a variety of other activities can be utilized to fill some of the void. Enrichment can be chosen to encourage low-, moderate-, and high-energy activities. Ideally, a combination of all of these should be incorporated into the strategy. The ratio should be tailored to the physical condition of the bird. Table 47.1 lists some examples of high-, moderate-, and low-energy activities of parrots.

TABLE 47.1
Examples of High-, Moderate-, and Low-Energy Activities for Parrots

High-Energy Activities	Moderate-Energy Activities	Low-Energy Activities
Flight	Climbing	Chewing
Wing flapping	Swinging	Vocalization
Hop/flap	Bathing	Puzzles
Swing/flap	Walking	Preening

POPULATION-BASED STUDY OF ENRICHMENT

For enrichment to be applied scientifically, specific enrichments must be investigated to determine whether they are having the desired effects. This can be very challenging, given the highly variable individual preferences between species and even between conspecific individuals. A variety of studies have investigated the behavioral and physiologic effects of enrichment, enrichment preferences, and motivation to utilize enrichments. Most of these studies are applied at the population level and provide a good framework for the design of enrichment programs. Some of these studies suggest that birds show a great deal of motivation to work for their food, a concept called contrafreeloading.[19] This motivation suggests that foraging, in particular, is an essential component of parrot welfare. Table 47.2 lists some studies that have investigated enrichment.

BEHAVIORAL ANALYTICAL ASSESSMENT OF ENRICHMENT

Behavioral analytical methods may be an alternative method of assessing enrichment at the individual level. Single-subject design (SSD) evaluations of behavioral interventions determine the efficacy of an intervention in an individual subject. Since welfare itself is assessed at the individual level, this may be an appropriate way to evaluate enrichment programs.[50] After all, although a population study can determine what the color most favored by the majority of the group may be, that information is relatively useless in the individual as it may have different preferences. For veterinarians counseling clients, a "study of one" is certainly the most appropriate method of assessment. There is a great deal of literature on human physical and behavioral disorders using this methodology.[51–55] Studies often track behaviors at baseline, after the institution of an interventional program, and sometimes again after the reversal of the interventional components.[50] Whether or not reversal is used is dependent on whether the changed behavior is unlikely to revert back as with a trained or learned behavior, and whether it is humane or ethical to reverse an intervention that is improving the welfare of the subject.

TYPES OF ENRICHMENT

There are five generally accepted categories of enrichment: social, occupational, physical, sensory, and nutritional.[56] Social and sensory enrichment are self-explanatory. Occupational enrichment is the provision of activities or challenges. Physical enrichment is the enhancement of the physical environment. Nutritional or foraging enrichment involves both the type and the presentation of food. Some types of enrichment can fit into more than one category. For example, training a bird provides both social and occupational enrichment. When designing and implementing an enrichment plan, as many of these categories as practical should be incorporated.

SOCIAL ENRICHMENT

Most parrot species are obligately social animals.[23,57] While it is common to remark about birds being "spoiled" because they demand constant attention, it is unnatural for most parrots to be alone. The only time most parrots are ever alone in the wild is when incubating eggs. At other times, they would be with their parents, offspring, clutch mates, juvenile flock, adult flock, or their mate. While flock size and composition may differ across species and seasons, being alone is generally unnatural, except for solitary species such as the kakapo *(Strigops habrotilus)*. Flocking is primarily a protective mechanism to reduce the pressures of predation. Being within a group allows for multiple sentinels looking for signs of danger. A group of birds can "mob" a predator and chase it away. Even thinking less altruistically, a bird in a group of 10 birds has a 10% chance of being the target of a predator, while an individual bird has a 100% chance.

Despite their importance, social skills must be partly learned. While many of the social behaviors do not need to be learned initially, they do need to be modified and improved by learning. Early socialization starts with parental care. Even before hatching, the parent birds start interacting with a chick. Like mammals, the parents will stimulate defecation and carry the waste out of the nest initially. Parental care is constant and involves feeding, preening, vocalization, and other interactions. This is not readily replicated in incubator-hatched and hand-raised chicks. Most species will also have clutch mates in the nest with them. This also helps them learn social skills. Once they fledge, there still is a great deal of parental care and teaching. Many parrots also have a crèche system, a flock of juvenile birds that move about together and learn about the environment, feeding, predators, and other important life skills.[58] One of these skills is how to interact with other birds without conflict.

Hand-raised birds have somewhat displaced imprinting. While filial imprinting on humans may have some early advantages, sexual imprinting on humans can have severe behavioral effects. Birds that are sexually imprinted on humans often pair bond with humans and will present aggressive behavior toward others, can redirect aggressive behavior to their perceived mate under certain conditions, or will defend a perceived nest territory.[59] Other birds may become frustrated and engage in self-damaging behaviors such as feather-damaging behavior (FDB).[60] In addition, birds that are completely imprinted on humans may exhibit poor socialization with other birds. Many bird owners have multiple birds, and one with poor social skills may either attack or be frightened of and attacked by other birds. Even if no aggressive behavior is displayed, some of these birds will be uninterested in socialization with other birds. These challenges can be addressed; however, it can take extensive work under the guidance of a

TABLE 47.2

Overview of Studies Investigating the Effect of Enrichment in Psittacine Birds

Species	*n*	Enrichment Type	Duration	Parameters	Outcome	Reference
Orange-winged Amazon parrot *(Amazona amazonica)*	21	Social enrichment (conspecific companionship)	12 m	Stereotypy development, feather-damaging behavior, fearfulness, aggression	Pair housing resulted in higher activity and more diverse behavior. Stereotypies reduced; fear responses modified	Meehan et al., 2003[20]
Orange-winged Amazon parrot *(A. amazonica)*	19	Social enrichment (different rearing environment: parent reared ± human interaction, human reared)	12 m	Latency to feed and fearfulness	Hand-reared birds less neophobic until 6 m of age. At 1 y similar fear levels in all groups	Fox and Millam, 2004[21]
Orange-winged Amazon parrot *(A. amazonica)*	64	Social enrichment (neighboring conspecifics); foraging and physical enrichment	N/A	Stereotypies, feather-damaging behavior	Parrots with more neighbors show less stereotypies; proximity to the door associated with feather-damaging behavior	Garner et al., 2006[22]
Grey parrot *(Psittacus erithacus)*	45	Social enrichment (social isolation)	N/A	Telomere length (chromosome stability)	Telomere length shortened significantly in socially isolated birds, suggestive of exposure to chronic stress and reduced life span	Aydinonat et al., 2014[23]
Mixed species	18	Social enrichment (video calling other birds)	N/A	Engagement of the parrot; perceived benefits by the caretaker	Parrots exhibited high motivation and intentionality to use the system and caretakers all reported perceived benefits	Kleinberger et al., 2023[24]
Buffon's macaw *(Ara ambiguus)* Green-winged macaw *(Ara chloropterus)*	2 2	Cognitive enrichment (behavioral training for artificial insemination procedures)	15 w	Behavioral activity, stereotypies	Training did not result in stress or affected reproductive behaviors in *A. ambiguous*; female *A. chloroptera* stressed, male did not complete training	Leblanc et al., 2011[25]
Ornate lorikeet *(Trichoglossus ornatus)* Josephines lori *(Charmosyna josefinae)* Fairy lorikeet *(Charmosyna pulchella pulchella)* Hooded parrot *(Psephotus dissimilis)* Coconut lorikeet *(Trichoglossus haematodus moluccans)* Mulga parrot *(Psephotus varius)* Orange-billed lorikeet *(Neopsittacus pullicauda)* West Papuan lorikeet *(Charmosyna papou goliathina)* Eastern bluebonnet *(Northiella haematogaster)* Red-rumped parrot *(Psephotus) haematonotus)*	14 10 12 14 12 14 14 14 14 14 14	Physical/cognitive enrichment (novel objects: rope, cotton mop and two small plastic blue tiles)	2 d	Latency to touch novel object, number of objects touched, duration of exploration	Resident species showed earlier exploration; exploration positively correlated with feather damaging	Mettke-Hofmann et al., 2000[26]

Continued

TABLE 47.2

Overview of Studies Investigating the Effect of Enrichment in Psittacine Birds—cont'd

Species	*n*	Enrichment Type	Duration	Parameters	Outcome	Reference
Orange-winged Amazon parrot *(A. amazonica)*	8–10	Physical enrichment (wooden and rawhide cubes, different colors)	>13 d	Preferences for enrichment characteristics	Color, hardness, size, and material, all influence use of the enrichment device; preferences appeared to reflect properties of foods native to Amazon's natural habitat	Kim et al., 2009[27]
Orange-winged Amazon parrot *(A. amazonica)*	12	Physical enrichment (ropes of different colors and sizes)	57 d	Preference for enrichment characteristics	Item properties may trigger different motivation behaviors; nonfrayed rope preferred; sex-specific preferences for length and diameter	Webb et al., 2010[28]
Cockatiel *(Nymphicus hollandicus)*	18	Physical enrichment (wooden sticks with a hole, iron rings with beads)	3 m	Behavior changes	Enrichment resulted in less time spent on eating, preening, locomotory behaviors, less undesirable activities, and improved fitness	Assis et al., 2016[3]
Grey parrot *(P. erithacus)*	3	Sensory enrichment (acoustic stimuli)	N/A	Behavioral changes, preferences for music	Musical preferences appeared influenced by personality; differences in behavior dependent on the acoustic environment	Péron et al., 2012[29]
Grey parrot *(P. erithacus)*	15	Sensory enrichment (acoustic stimuli)	N/A	Behavior observations, musical preferences	Parrots intrinsically participated in the experiments and provided positive feedback to the devices; preferences for certain music types observed	Gupfinger and Kaltenbrunner, 2017[30]
Grey parrot *(P. erithacus)*	15	Sensory enrichment (acoustic stimuli, musical enrichment)	Three test phases of 3–4 d over 3 m	Behavior observations, musical preferences	Preference for continuous beat to single notes, but no obvious preference for faster or slower beats; individual preferences exist. Birds voluntarily and positively interacted with home-made musical instruments	Gupfinger and Kaltenbrunner, 2020[31]
Yellow-headed Amazon parrot *(A. oratrix)* Grey parrot *(P. erithacus)* Hyacinth macaw *(Anodorhynchus hyacinthinus)* Scarlet macaw *(Ara macao)* Military macaw *(A. militaris)* Blue-and-gold macaw *(A. ararauna)*	1 2 1 2 2 2	Sensory enrichment (auditory enrichment), social enrichment (rearing method, social environment)	36 d	Behavior (vocalizations, stereotypies, enrichment interaction)	Exposure to talking radio and pop music significantly reduced psittacine vocalization; hand-reared birds showed more stereotypy and interacted less with enrichment than parent reared; solo-housed birds showed more stereotypy than group-housed birds	Williams et al., 2017[32]

Red-fronted macaw *(Ara rubrogenys)* Thick-billed parrot *(Rhynchopsitta pachyrhyncha)* Green-winged macaw *(A. chloropterus)* Chattering lori *(Lorius garrulus)*	4 2 2 2	Foraging enrichment (length of wood with two variations in food distribution)	4–5 d	Preference on enrichment's food supply	More appropriate species-typical behaviors induced following provision of foraging enrichment	Coulton et al., 1997[10]
Grey parrot *(P. erithacus)*	18	Foraging enrichment (puzzle feeders)	2 m	Feather-damaging behavior	Pipe feeder increased foraging times and improved feather condition	Lumeij and Hommers, 2008[33]
Grey parrot *(P. erithacus)* Healthy Feather damaged	22 11 11	Foraging enrichment	1 m	Contrafreeloading	Contrafreeloading displayed by both healthy and feather-damaging birds; birds with feather-damaging behavior displayed less contrafreeloading	van Zeeland, 2009[19]
Grey parrot *(P. erithacus)*	12	Foraging enrichment	>12 w	Foraging activity	9/11 enrichments significantly increased foraging activity	van Zeeland et al., 2013[16]
Red-tailed black cockatoo *(Calyptorhynchus banksii)*	6	Foraging enrichment (sliced cucumber, fresh grass, baffle cages, and millet disks)	25 d	Oral stereotypic behavior, foraging activity	All foraging enrichment increased median percentage of time spent foraging compared to control. 2/4 enrichments (grass, millet disks) significantly decreased oral repetitive behaviors	Fangmeier et al., 2020[34]
Orange-winged Amazon parrot *(A. amazonica)*	30	Social enrichment (pair separation), physical enrichment (misting, nest hole restriction, larger nest boxes), nutritional enrichment (fruits)	2 y	Egg laying	Parrots' sexual activity stimulated by enrichment protocols	Millam et al., 1995[35]
Crimson-bellied conure *(Pyrrhura perlata)*	10	Foraging enrichment, physical enrichment (addition of perches)	24 w	Feather-damaging behavior	Stabilization of feather-damaging behavior during study, natural and edible materials more successful	van Hoek and King, 1997[36]
Orange-winged Amazon parrot *(A. amazonica)*	16	Physical (increased cage complexity), foraging enrichment	1 y	Responses to novelty and human handlers	Enrichment reduced fear and motivation for interaction/exploration	Meehan and Mench, 2002[11]
Orange-winged Amazon parrot *(A. amazonica)*	16	Physical (increased cage complexity), foraging enrichment	48 w	Feather-damaging behavior	Feather damaging prevented and reduced following provision of enrichment	Meehan et al., 2003[37]
Orange-winged Amazon parrot *(A. amazonica)*	16	Physical (increased cage complexity), foraging enrichment	48 w	Development of stereotypic behavior	Stereotypies prevented following enrichment	Meehan et al., 2004[38]

Continued

TABLE 47.2

Overview of Studies Investigating the Effect of Enrichment in Psittacine Birds—cont'd

Species	*n*	Enrichment Type	Duration	Parameters	Outcome	Reference
Nanday conure (Aratinga nenday) Social enrichment birds	48 27	Physical enrichment, social enrichment, handling by humans	6 w	Novel object, novel conspecific, open field, emergence, latency to feed, learning tests, and behavioral assessment	Enrichment protocols reduced fear levels and increased exploration in several tests	Luescher and Sheehan, 2005[39]
Cockatiel *(Nymphicus hollandicus)*	16	Physical enrichment (collard green stalks)	20 d	Behavior changes	Stalks increased the feed intake and decreased sleep activities	Carvalho et al., 2017[40]
Red-winged parrot *(Aprosmictus erythropterus)* Jandaya conure *(Aratinga jandaya)* Sun conure *(Aratinga solstitialis)* Blue-winged or Malabar parakeet *(Psittacula columboides)* Green-winged macaw *(Ara chloropterus)* Rose-ringed parakeet *(Psittacula roseate)* Red-breasted parakeet *(Psittacula alexandri)*	2 1 1 6 1 6 1	Physical enrichment (ladder, continuous rings, mirror, rope with knots); foraging enrichment (skewers with fruits)	8 d	Preference and use of the items	Ladder was highly preferred; rope with knots was least preferred	Prathipa et al., 2017[41]
Cockatiel (Nymphicus hollandicus)	12	Physical enrichment, substrate (earth, sand, wood shavings, shallow and deep water)	Three periods of 1 w	Preference and behavior	Wood shavings, earth, and sand were preferred as substrates; no bathing behavior was observed	Stevens et al., 2021[42]
Orange-winged Amazon parrot *(A. amazonica)*	34	Foraging devices; toys	25 w	Novel object test	Rotation of devices more effective than enrichment itself to reduce fear	Fox and Millam, 2007[43]
Orange-winged Amazon parrot *(A. amazonica)*	6–12	Foraging enrichment	>4 w	Behavior (foraging), pellet preference	Birds preferred oversized pellets, which naturalized foraging times. Feeding of oversized pellets reduced destruction of wooden cubes (act as foraging substitutes?). Podomandibulation reduced upon feeding regular pellets	Rozek et al., 2010[15]

Orange-winged Amazon parrot *(A. amazonica)*	10	Foraging enrichment	7 m	Motivation for pellet types, destructible cubes	Oversized pellets are highly preferred over regular pellets; chewing/biting of wooden cubes reduced upon provision of larger-sized pellets	Rozek and Millam, 2011[44]
White-eyed conure *(Aratinga leucophthalma)*	10	Foraging enrichment	7 m	Behavior (abnormal behavior)	Reduced abnormal behavior (nonsignificant)	de Andrade and de Azevedo, 2011[5]
Budgerigar *(Melopsittacus undulatus)*	36	Physical and social enrichment	8 m	Behavior (stereotypies)	Stereotypies reduced following interaction with multiple partners	Polverino et al., 2015[45]
Orange-winged Amazon parrot (A. amazonica)	13	Physical, foraging, and social enrichment (handlers)	40 w	Development of abnormal behaviors	Deprivation of enrichment worsened feather condition and increased duration of stereotypies	Cussen and Mench, 2015[46]
White-eyed conure *(Aratinga leucophthalma)*	12	Sensory, foraging and physical enrichment (multiple items)	3 w	Feather-damaging behavior	Enrichment was more effective than drug treatment	Telles et al., 2015[47]
Sulphur-crested cockatoo *(Cacatua galerita)*	2	Cognitive enrichment (training), social enrichment (human contact), foraging enrichment in combination with haloperidol	N/A	Abnormal behavior, feather score, fecal corticosterone	Reduced abnormal behavior and improved feather score; decreased fecal corticosterone concentrations	Peng et al., 2014[48]
Blue-and-gold macaw *(Ara ararauna)*	22	Physical, occupational, and food enrichment	6 m	Behavior analysis, fecal corticosterone	Biting and pacing as well as vocalization, preening, and feather bristling decreased significantly during the enrichment phase, whereas locomotion increased; no differences observed in corticosterone metabolites in the droppings	de Almeida et al., 2018[49]

d, Day; *m*, month; *N/A*, not applicable/not available; *w*, week; *y*, year.



qualified behavior consultant well-versed in training technology based in the science of behavior analysis. Hence, the parrot's previous experiences and rearing history need to be considered when weighing the risks and benefits of the addition of a new bird to the household (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).

Some studies have shown that parent-raised birds that are socialized by people while developing have pet qualities exceeding those of hand-reared birds.[59,61] This can be slightly challenging to accomplish but should be considered the ideal practice. Breeding birds can be trained to station outside of the nest box so that the nest can be inspected, and the young birds handled. Alternately, parent-raised birds can be trained using positive reinforcement techniques after fledging. Minimally, having birds raised with clutch mates or other babies will at least allow them to imprint on both humans and birds. (see Section 9: Embryology, Neonatology and Pediatrics, and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).[61]

Assuming a bird has well-developed social skills, the next concern is providing them with opportunities to socially interact with humans, conspecifics, or other animals. While bird owners like to believe they are providing this for their pets, in most cases, the social contact they provide is inadequate. Socializing a parrot is more than a full-time job. The ideal solution is that birds be kept in a more normal social grouping, especially those that have been reared by their parents and/or with other birds. Compatible birds, housed together, can provide 24-hour socialization for each other. To be compatible, they must have some social skills with other birds. Cohoused birds also should be size and temperament matched (Figure 47.4). Although it can occasionally work, housing large birds with small birds can be a dangerous situation. Larger birds can easily tear the beak or a toe off a smaller one. Likewise, a loud bird may be a source of stress for a nervous or quiet bird. In a study on assessment of personality traits to aid in social pairing of orange-winged Amazon parrots *(Amazona amazonica)*, pairing success was greatest for social-guarded, social-nervous, or nervous-nervous parrot pairs.[62] Another study in white-eyed conures *(Psittacara leucophthalmus)* also indicated effects of temperamental traits on behavior of group-housed individuals. While bold parakeets spent more time on preening, showed more negative social interactions, and tended to spend less time interacting with the environment, vigilant parakeets showed lower levels of positive social interactions and spent less time on locomotion.[63] Both studies therefore indicate that temperament could be useful to determine whether or not individuals could be housed together, thereby avoiding unnecessary disputes and injuries. In general, introductions are easiest with very young birds, and most birds that grow up together remain compatible, provided they do not conflict over a mate or resources. If breeding is not desired, the birds should be the same sex.[20] Although there are exceptions, breeding often will have some undesired effects on the companion characteristics of a parrot during the breeding season. Most breeding pairs will defend the nest territory from all others, resulting in aggressive behavior. If breeding is desired, the pair can be allowed to defend their territory. They should, however, be conditioned to allow nest cavity inspection daily and should be trained to allow normal care procedures (via stationing or other methods) but be left undisturbed otherwise.

The obvious benefit of having birds in pairs or groups is that it corrects for some of the limitations of human companionship.



FIGURE 47.4 Compatible, size-matched birds can be cohoused if the enclosure size permits. These young male blue-and-gold *(Ara ararauna)* and green-winged macaw *(Ara chloropterus)* were hand-raised together. As they mature, greater monitoring for aggressive behavior may be needed. Birds housed in smaller cages may not be able to be cohoused. They can be housed in adjacent cages and allowed play time together when supervised.

Companion birds can be together all the time, reducing the reliance on the owner for companionship and protection. In addition, the benefits of companionship from a conspecific have been well studied, with several studies indicating pair or group housing to increase overall activity and behavioral diversity while lowering the incidence of stereotypic behavior and fear responses.[20,32,45] As such, social housing with a companion may reduce problem behaviors such as nuisance vocalization, stereotypies, or FDB that commonly occur when birds rely too much on the owners' presence and attention.[63] Contact with other birds may vary from limited vocal and/or visual contact (e.g., when birds are placed in the same or an adjacent room or aviary) to more intense contact (e.g., when the cages are placed side by side, the birds are physically housed together or are allowed to interact with each other outside of their cages). Mutual preening is considered important to promote social bonds.

Recently, researchers also developed an approach to allow single-housed parrots to engage in video calling other parrots.[24] During a pilot study with 18 birds, most of the birds showed high motivation and intentionality to use the system, while caretakers all reported benefits from these calls, some arguably life transformative (e.g., learning to forage or fly by watching others). Such options may therefore be worth exploring for highly motivated caretakers with a single-bird household. Alternatively, owners may sometimes provide a "dummy bird," mirror, or mirror-like toy in the cage to serve as a substitute for social interactions, particularly for smaller birds such as budgerigars *(Melopsittacus undulatus)*, cockatiels *(Nymphicus hollandicus)*, and lovebirds (*Agapornis* spp.). Indeed, some studies have shown the beneficial effects in parrots and other birds, where birds

exhibited extensive interaction, more playful behavior, and reduced stress levels upon exposure to a mirror.[64–66] Others, however, have suggested a potential negative effect on behaviors through negatively affecting food intake and nesting activity while increasing occurrence of aggression (to conspecifics or the mirror) or leading to maladaptive behaviors such as pair bonding with the mirror.[67–71] These potential advantages and disadvantages should be discussed with the owner so that an informed decision can be made on the provision of mirrors to any pet bird.

Breeding and sexual behaviors are not desired in singly housed birds. Nevertheless, these behaviors may inadvertently be encouraged by the owner. For most wild birds, breeding occurs during a defined, short season, which is signaled by a changing photoperiod, rainy season, presence of a mate, availability of certain food items, or simply food surplus.[72–75] Many of these key stimuli are present in the captive situation. Pet birds typically are overfed on high-calorie diets. Numerous birds are offered nest boxes, "snuggle" tube perches, or are allowed access to cabinets or other hollow structures that the bird may perceive as a potential nest site. Many owners interact with their birds in ways that signal pair bond formation and reinforcement in parrots. Reproductive instinct is strong in most species and adding in these potent stimuli can suffice to initiate the breeding process. To discourage reproductive behaviors, several strategies can be employed (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 12, Reproduction). Caloric intake should be carefully controlled. This is especially effective for suppressing egg laying since most hens will increase food consumption in preparation for egg laying. Foraging can give the bird the impression that food supply is limited, even if higher amounts of food are offered. Owners should try and restrict physical contact with the bird, specifically the type of contact that simulates courtship or mating behaviors. Alternative activities that stimulate social interaction, such as training, playing, and other active games, should be encouraged to replace much of the "cuddle" time. The petting that is done should be brief, used to reinforce good behaviors, and limited to nonsexual locations of the body. The advice of touching only above the shoulders is appropriate (Figure 47.5). For those species that respond to photoperiod, modifications can be made to regulate the day:night cycle, with a relatively short and consistent day length being most desired. Access to any potential nest sites or nesting material should be restricted. Several of these interventions comprise antecedent arrangement interventions to prevent stimulation of reproductive behavior. However, merely focusing on removal of antecedents for reproductive behavior could create a void for other problem behaviors to occur. Hence, where possible reduction or elimination of reproductive activity should not solely rely on antecedent arrangement but preferably be combined with positive reinforcement of alternative, appropriate behaviors. Aside from foraging and (nonphysical) social interaction, this may include stimulation of exploratory behavior by offering new toys or enrichment, regular replacement of toys, and/or rearrangement of the layout and design of the enclosure and living environment.

Of course, human companionship can be a critical form of enrichment for many birds. A recent study in blue-and-gold macaws (*Ara ararauna*; $n = 5$) used a classical separation-reunion paradigm where birds were temporarily placed in an unfamiliar environment with a familiar caregiver (union; 2 minutes) to then be left alone (separation; 2 minutes) and subsequently be reunited with its caregiver again (reunion; 2 minutes) and found birds to show facial indicators of positive affect (higher crown and nape feather heights; increased redness of the skin; higher frequency of contact-seeking behaviors; eye pinning) during the union and reunion phases compared to the separation phase during which the birds expressed long-distance contact calls.[76] These results indicate that the presence of a human companion can be beneficial to parrot welfare. It should be noted that appropriate interactions can greatly benefit both the bird and the human in this relationship. Noncontact interaction can be a large part of this relationship. Being in the visual and auditory range of human "flock" members appears to be comforting to most human-imprinted birds. The location of cages, play stands, and other living spaces of birds should allow the bird to remain in view of daily household activities. While doing so, the character or temperament of the bird should be carefully considered. For example, an anxious, nervous bird that is known to respond fearfully to novel objects or people may warrant the enclosure to be positioned on the side, near a wall, to provide a sense of security and option to retreat from social activities, whereas a more central location is preferred for a bird that is seemingly curious and "loves" attention. A continuous vocal connection can help reassure a bird that the flock is near. By positively reinforcing the lower-volume vocalizations, and the owner keeping their responses low in volume as well, some of the louder nuisance vocalizations can be minimized. When the owner is away, radios or televisions or other types of background noises may provide the single pet parrot with vocal stimulation. Verbal games seem to be



FIGURE 47.5 Although relished by many birds, excessive petting, cuddling, and close physical contact can simulate pair bonding and lead to a variety of health and behavioral problems. Alternative activities like positive reinforcement training can provide occupational and social enrichment simultaneously.

enjoyed by many birds. Parrots, especially Grey parrots *(Psittacus erithacus)*, have been shown to have considerable skills in language and this can be used to advantage.[77,78] Direct physical contact interactions can serve as social enrichment as well. A human hand or arm can serve as a mode of transportation for birds within a home. This can be coupled with verbal interaction and limited petting, playing, and training activities. Many of these interactions then double as occupational enrichment.

Social interactions with other species in the home also provide enrichment, and literally, any species that can offer the opportunity for birds to interact in a desirable manner for both should be considered as an option that can offer favorable benefits for both. Extreme caution should be taken with predatory species, however, as severe or fatal injury can occur very rapidly. Likewise, a large parrot could severely injure a smaller animal. Careful observation and supervision are critical to make these introductions safe and successful.

OCCUPATIONAL ENRICHMENT

Occupational enrichment is activity based. It involves encouraging either physical or mental activity to the subject animal. This can include any number of activities. Mental stimulation can be provided through cognitive enrichment, which includes activities such as training, playing games, or offering puzzle feeders that the animal must manipulate to obtain food. Moreover, novel objects such as unusual foods, novel toys, and/or unusual scents can be used to stimulate exploration behaviors. Recent studies have also demonstrated the opportunity to teach letter-speech associations (so-called grapheme-phoneme correspondence) to parrots as well as the ability to read and implement computerized technology (e.g., touchscreen devices, tablet-based computer boards, video calling) in parrot enrichment, thereby significantly expanding the available options to engage parrots in activities that align with their higher-cognitive capabilities.[24,79–83]

Physical stimulation, on the other hand, can be promoted by providing the animal with opportunities to exercise (i.e., flying, climbing, swinging, and swaying). While some of these activities can be encouraged by the physical environment and performed independently by the parrot, others are brought about during direct interaction with humans.

Positive Reinforcement Training

Owners will sometimes claim their parrots are seemingly difficult to train. This is perplexing, given their intelligence. Part of the reason is likely to be the typical feeding practices for pet birds. Although words, touch, or attention may be effective at times, at other times, it can be difficult to predict exactly what an individual parrot will find reinforcing. As a consequence, training parrots has long involved less desirable methods that involve coercion and force rather than positive reinforcement. Mostly these techniques were used in good faith, but there is substantial evidence of fallout from coercion.[84] As a result, training using positive reinforcement has nowadays become the standard (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).[85] Positive reinforcement can involve a diversity of primary and secondary reinforcers, which are provided to the bird upon it displaying a desirable behavior. Primary reinforcers, which include resources such as food, water, sleep, and shelter, often work well because of their biological relevance to the animal, especially if the motivation for obtaining these resources is high. For example, a parrot that is hungry (not starving!) becomes infinitely more trainable as food becomes a valuable resource to them. However, secondary reinforcers, such as toys, touch, and attention, which initially begin as neutral stimuli but become conditioned when paired with primary reinforcers, can also make for suitable reinforcers once the bird perceives these as highly desired items and experiences.

Positive reinforcement training provides a means for owners to communicate effectively with their birds and to allow the birds a degree of choice in what behavior they will exhibit. Undesirable behaviors can be replaced with desirable behaviors that are subsequently reinforced (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). In addition, training through positive reinforcement techniques has been shown to reduce abnormal behaviors such as stereotypies and FDB in a limited number of birds, though the effect was highly dependent on the individual.[25,48] Although the process of training is relatively simple, the devil is in the details. The ability to read the body language of the bird, developing timing with delivery of a bridging stimulus and/or reinforcers, and even presenting cues in a manner that does not appear threatening requires practice. Box 47.2 contains a sample instruction sheet for helping clients understand how to train their birds.

Additional training for the owner is strongly recommended. There are many great books, videos, and online resources that can be used to improve training skills (see Chapter 46). One of the most difficult aspects to master is being patient. Pushing a reluctant bird too quickly is likely to hinder learning of the behavior, even if they initially perform the behavior. The fact that they were uncomfortable doing the behavior will make them avoid it in the future. This is particularly difficult when transitioning from traditional training techniques to positive reinforcement behavior.[86]

The behaviors that are trained are far less important than the training itself, as training by itself provides an excellent opportunity for clients to engage their birds without stimulating reproductive behaviors. However, training some husbandry behaviors can greatly reduce stresses that will occasionally occur for any pet bird. If a bird allows the owner to file the nails daily with a fingernail file, then trimming by restraint of the bird will rarely, if ever, be required. Training a bird to defecate in appropriate locations will make it easier to integrate them into normal household routines, while recall is an important behavior to teach to flighted birds to ensure their safe return. Training can also focus on behaviors that are relevant to the veterinary examination, thereby helping to reduce stress from restraint or administering medication (see Chapter 46). For example, training a bird to allow gentle restraint in a towel will facilitate veterinary examinations or other procedures, while training a bird to drink juice or almond milk from a syringe or other device will facilitate delivery of medication if an illness occurs.[87]

Model/Rival Training and Observational Learning

While positive reinforcement training should be used when behaviors need to be taught efficiently, casual interactions can

BOX 47.2 INSTRUCTIONS TO CLIENTS REGARDING TRAINING OF PARROTS

Tips to Create an Eager Student

- Start a foraging program (see Box 47.4).
- Limit a highly favored food item to a small quantity and use it only for training.
- Break up the food to be used as a reward into small morsels (1/4 inch, 0.63 cm or less for a medium- to larger-sized parrot, and even smaller sizes for smaller species).
- Take away all food for about 1–2 hours before training sessions.
- Stop sessions before the bird loses interest in the treats.

Definitions

- Reinforcer: The consequence for presenting behavior. In force-free training, reinforcers tend to be things or activities the animal seeks to acquire or values. This can include items such as preferred food, access to enrichment items, touch, companionship, and, in some cases, verbal responses from caregivers. Desired items should only be considered true reinforcers to a particular bird once it has been demonstrated that they actually increase or maintain behaviors in that bird (and not because an owner, behavior consultant, or trainer perceives it to be one).[86]
  - Food items
  - Food toys
  - Access to a preferred person
- Target: A common tool in animal training is a target. A target can be an item (e.g., stick, light, pen), hand, or spot that the animal needs to touch with a part of its body (e.g., beak, tongue, head, wing, foot). The action of orienting the body part toward a target is called targeting. Once an animal has learned to target, it is possible to use the target to direct the animal where to go or where to place a body part. This allows the trainer to give the animal information and create action that can be reinforced without having to handle or manipulate the animal. For example, another way to train a parrot to lift its foot for nail trimming is to train it to touch a target with its foot.[86]
  - Often the trainer's hand or a colored stick or wand
- Shaping: To train more complex behaviors, it is recommended to break these up into smaller, more easily achievable behaviors or actions that will gradually build toward the more complex behavior. Once these smaller actions or approximations toward the desired behavior are identified, a trainer can start reinforcing these until the behavior is shown fluently. Next, the trainer focuses on building more behavior using targets, showing reinforcers, or careful observation of the animal's action. If the animal makes any action that builds upon the previous action, the trainer reinforces that action. Each approximation that is successfully reinforced brings the animal closer to presenting the desired final behavior.[86]
- Bridging stimulus (bridge or marker): The reinforcer is the tool that communicates to the animal that it has presented behavior desired by the trainer. It is important that delivery of the reinforcer is paired in direct conjunction with the presentation of the desired action. A bridging stimulus is a sound or a signal that can be used to help mark behavior when it is difficult to deliver reinforcers promptly.[86]
  - Clickers
  - Verbal (e.g., "good")
- Cue: Cues are sounds or signals that indicate the opportunity exists to present an action and earn reinforcers. Cues acquire their value by being paired with the action followed by the reinforcer. Sounds and signals are meaningless as cues without this pairing.[86]
  - Verbal (e.g., "step up")
  - Hand signals
- Jackpot: Providing a better (larger amount or more preferred consequence) for performance of a behavior or an approximation that is particularly challenging or novel for the animal. If this is offered during the shaping process, the jackpot should not be so great that it disrupts the flow of the session.

A Brief Overview of How to Train a Behavior Using Shaping

- Find a way to create an action toward your desired goal behavior you can reinforce by showing food, using a target, and/or arranging an inviting environment.
- Gradually build upon this action by reinforcing small approximations toward your desired goal behavior.
- Build reinforcement history for your desired goal behavior.
- Once reinforced behaviors will be repeated and become predictable, insert your cue right before the behavior is offered to create the association with your desired cue.
- Fade out reinforcing for offering and only reinforce when cued.

also be used to teach behaviors. This method, the model/rival method, uses social cues and observational learning to teach the subject.[88–90] In dogs, model/rival training was as effective in teaching object names as positive reinforcement training.[89] In parrots, model/rival training has been used primarily for teaching language skills and involved a teacher and a model/rival doing verbal exchanges in front of the bird. The emphasis is on the name of a particular object. When the model/rival said the name of the object, it was handed to them. Attention is only directed to the bird when it says the name of the object, at which point the object is given to the bird. Therefore the reward is intrinsic to the object rather than any association with a food reward. Using this method, Pepperberg has been able to demonstrate a wide variety of cognitive capabilities of parrots including numeric skills, object permanence, same-different concepts, and other categorical capabilities.[91–94] Similarly, Kleinberger et al. reported birds to learn how to forage or fly from watching other parrots during parrot-parrot video calls.[24] Given the crèche systems often employed in psittacine flocks in the wild, it is highly likely that such social, observational learning is critically important in the learning of survival skills. Hence, observational learning can certainly be employed in companion birds that look to human flock members for cues on behavior. In addition to learning language, responses to various household features can be taught. For instance, a small role-play can be done in front of a stovetop. The model/rival can pretend to burn a finger and act dramatically. The teacher can scold and say, "Yeah, Dangerous!" or "Hot!" This can teach the bird that this is a dangerous place. Similarly, windows and mirrors can be shown to be solid objects. Pictures or objects can be shown to be safe and not to be feared. This method is certainly not mutually exclusive to positive reinforcement training. It just uses intrinsic, social reinforcement. It should be noted that

there may be circumstances when it may not be appropriate. In one experiment, a human-raised gibbon was being taught gestures using this method. Every time the rival performed the task and was rewarded with attention (from the favorite person), the gibbon attacked the rival.[95] It would be easy to believe this could happen with a human-imprinted cockatoo or other parrot as well. Anecdotal reports of "jealousy-based" attacks on perceived rivals for a favored person's attention are all too common in companion bird medicine. While the model/rival improves learning, it may not always be essential to have a second person or bird for observational learning to occur.[96] The mechanisms by which observational learning occurs are not always known, but it certainly can be an additional way to model behaviors in companion birds. The previous example of the stovetop can be done without the model/rival and still achieve the goal. The clinician can recommend a "house tour" game to clients. The bird is carried about the house and introduced to objects, people, and other pets while modeling appropriate responses to these items. Although the goal of these methods is to achieve improved behavior, they inherently engage the bird's mind and stimulate their curiosity. They also provide great alternatives to pair bond–simulating activities like petting and cuddling with birds.

Physical Exercise

The benefits of physical exercise are well documented (Box 47.3).[97–99] Likewise, the negative effects of inactivity are well known. As the number of aging birds increases, the frequency of certain degenerative diseases has grown by an even greater degree.[100–102] While not all these conditions are preventable, many are much less prevalent in animals that receive appropriate diet and exercise. Cardiovascular diseases, such as atherosclerosis and congestive heart failure, can be reduced with appropriate levels of physical activity.[98,99] Obesity, hepatic lipidosis, and arthritis are other conditions that may result from a severe lack of physical activity. The musculature will be stronger with regular exercise. The joints require movement to nourish the articular cartilage. Bone density improves with physical activity as well. Exercise has a great deal of psychological benefit as well.[103] In humans, it can reduce the effects of depression.[104–107] In animals, exercise can help reduce displacement behaviors.[103,104,108,109] The expenditure of energy on healthy physical activity can only help reduce the use of energy on pathologic behaviors.

As in any animal or person, a medical examination should be performed to determine the safety of a new exercise program. Certain conditions may require restriction of exercise, even if it originated because of inactivity. Such conditions may include some orthopedic conditions, atherosclerosis, and heart failure. Carefully planned exercise may still be beneficial in these cases, but they should be tailored to the individual bird and instituted very cautiously and voluntarily as forcing these birds to exercise may cause them to decompensate.

There are many types of exercise. For birds, the simplest and most obvious is flight. The largest mass of muscle in flying species is the pectoral musculature and this is the exercise they are best adapted to. There are complications to having a flighted bird as a pet. These include injuries, escapes, or difficulties with training. Most of these complications result from poor flying skills rather than flying itself and can be managed with appropriate training and flight development. Recall (flight to the handler), flight to a perch, and other such games are easy ways to promote flight activity. An enclosure that is large enough to allow flight will passively encourage this type of exercise. It is much more feasible to provide ample flying space for a budgerigar with a 25- to 30-cm wingspan than for a macaw with a wingspan of over a meter. In some cases, though, an owner may wish to keep the wings trimmed to prevent some of these complications (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 22, Advancements in Management of the Welfare of Avian Species). In these cases, providing ample wing exercise can be more challenging. Wing flapping can be encouraged but should be trained rather than forced. Dropping a hand while a bird is perched upon it will force a bird to flap. However, this can promote biting, sometimes just to hold on. It is also a force-based method and is therefore best avoided. Most birds will flap when the surface they are perched upon moves. Providing swings, flexible branches, ropes, or other movable perching surfaces is a good way to encourage some wing flapping. Often birds on a swing or rope will flap the wings to propel the rope or swing toward another foothold or beak-hold. This motion can be referred to as a flap/swing and is commonly used by parrots when there are numerous flexible perching options in large enclosures. Likewise, many will flap the wings, along with hopping to another perch. This can be referred to as a flap/hop. The flap/swing and flap/hop may be the most beneficial wing exercises for birds that do not have flight capabilities.

More so than any other avian species, parrots are also adapted to climbing. Their hooked beak and zygodactyl feet provide strong holds that allow them to climb vertical heights and grasp perches.[110–113] Parrots can hang from just the beak or from one toe and can flip back on top of a branch with a few flaps of the wings. Even fully flighted parrots will often use their climbing abilities to negotiate a maze of branches rather than fly over them. Tarzan himself would be jealous of the gymnastic capabilities of a fit parrot! To encourage these types of activities, branches, ropes, or ladders can be provided. In fact, nearly all structures or materials that will provide grip to the parrot will be suitable for this purpose.

Many birds are quite vigorous in their bathing activities. They may hop in and out of a spray of water, flap the wings, shake the tail and/or head, and generally have a great time. This can be another means of encouraging exercise.

Beak activity is a relatively low energy form of exercise. It is constant, however. Parrot owners are fully aware of the destructive capability of psittacine beaks. Some birds may initially require encouragement to chew. For this purpose, soft items like paper, cardboard, or softwoods can be provided to the bird. As beak strength improves, harder types of woods can be given. Some parrots may require a small nick or other

BOX 47.3 BENEFITS OF PROVIDING PHYSICAL EXERCISE TO PARROTS

- Increase bone density, joint, and muscle strength.
- Reduce incidence of cardiovascular diseases (atherosclerosis, congestive heart failure).
- Reduce incidence of hepatic lipidosis.
- Prevent and/or treat obesity.
- Reduce displacement and other problematic behaviors.
- Mutual enjoyment of bird and handler.

starting spot to enable the beak to get a grip on the object at first. In addition to stimulating beak activity, enrichment can also be provided to stimulate the parrot to manipulate items dexterously with its feet. This activity, referred to as podomandibulation, is often paired with chewing activity as items are often held up for the beak to engage and is yet another characteristic that is unique to parrots. To stimulate this activity, at least some of the chewing items that are provided should be loose to allow for this kind of manipulation.

Free Flight

Free flight with companion parrots is a growing hobby and has recently even been found to benefit the release of captive birds into the wild.[114–116] Internet videos show spectacular footage of birds flying and landing on their owners' hands. There are also free flight clubs, chat rooms, and other groups that allow enthusiasts to communicate about and practice this activity. Avian veterinarians may encounter individuals who are engaged in, or are planning to engage in, free flight with their birds. A basic knowledge of this activity may be helpful in advising these clients as it is an extremely appealing yet also extremely risky practice.

Benefits: As previously described, exercise for most companion birds is often limited to nonexistent. Clearly, free flight offers the greatest possibility for flight exercise in a companion bird, especially for the larger species for which adequate flight space can be difficult to provide indoors. Along with providing exercise, when expanding a bird's environment to include nearly unlimited space, a much higher degree of enrichment becomes possible. Exposure to fresh air and natural sunlight is an added benefit. Flight also provides a significant amount of mental as well as physical activity. Navigation, wind adjustments, and other fine motor skills are partly mental. Allowing these activities can promote mental development that may not be allowed to develop otherwise. One advantage of free flight is more self-serving. Most people who love birds enjoy watching their antics. Free flight expands the range of available activities to watch. The fear induced by seeing a parrot flying away pales in comparison to the thrill of watching them fly back and make a perfect landing on the hand. Free-flighted birds can be more readily involved in daily life. They can go for hikes, hang out in the yard during a picnic, or other outdoor activities. They can play in a tree that is hundreds of times the size and complexity of a commercial play stand (Figure 47.6).

Risks: There are certainly risks involved with free flight. Of course, there is risk inherent in all activities. Sending a child to school, allowing them to drive, or date, or leave the home at all, will expose them to risks. However, to eliminate all risk is also to eliminate opportunities, enjoyment, and any real semblance of a life. Most people choose to teach their children to live in our world and then trust them to do so. The same process can be applied to pet birds. The first thought most people will get when thinking about free flight is that their bird will escape and fly away. Some people seem to think that pet birds dream of escaping and will do so at the very first opportunity that they get. Certainly, the escape of a bird is devastating. It is also the last thing that most pet owners want to happen. However, most escapes are the result of the bird's inability to return rather than a lack of a desire to return. Recall requires the flying bird to navigate, turn, and land correctly. An unskilled bird can gain altitude but may have difficulty slowing, turning, landing accurately, and performing other fine motor skills. This can result in the bird flying straight away from the owner and landing only from exhaustion in a tree somewhere quite far from where the flight began. This may subsequently result in a parrot getting lost because it is lost and lacks the ability to return. One of the essential components of successful free flight is allowing a bird to learn these skills in an area where they will not get lost. Training for free flight can initially start indoors and then



FIGURE 47.6 (A) In addition to the benefits of actual flight, free-flighted birds can utilize a nearly unlimited array of environmental enrichments. This green-winged macaw *(Ara chloropterus)* is playing on a rope ladder in a large maple tree. (B) The same macaw is foraging in a white mulberry tree. (C) Two free-flighted green-winged macaws and one blue-and-gold macaw *(Ara ararauna)* unable to fly accompany their caregiver on a hike.

continue outdoors in large open expanses where there is little to land on besides the familiar owner or a perch. Further skills can be built from there. In fact, birds that are well trained to this behavior have far less risk of escape than an unskilled, untrained bird that accidentally gets out the door. There is little evidence of a homing capability in parrots as is seen in pigeons, although this may be species variable and even among pigeons may be influenced by multiple factors.[117–119]

The risk of predation is also very real. While this can be true for many pets, the small size of most companion birds makes them susceptible to a wide variety of predators. Aerial predators such as raptors are of particular concern. Raptors can prove to be a risk when a bird is in the air and on the ground or perch. Although they are not necessarily predatory toward parrots, crows, ravens, and other birds can present a risk of injuring or chasing away an otherwise well-trained bird. Predation risk cannot be entirely prevented. However, it can be mitigated by several things. First, flying a very large parrot narrows the range of predators that would attack them. For example, few Cooper's hawks *(Accipiter cooperii)* will try to attack a large macaw that is three times its own weight. Second, the flying skills of a bird will reduce the chances that it will be caught by a raptor. The bird can also learn vigilance, safe perching strategies, flock flying, and other skills that can help it evade predation. Flocking is vital to safe, free flying. Single birds are far more susceptible to predation than multiple birds, and thus flying birds alone is not generally advised. Additionally, the presence of the owner can also serve as a deterrent to predators. Often just walking toward a hawk perched on a telephone pole will cause it to vacate the area.

A fast-flying bird is going to be prone to certain accidental injuries. Indoor flying for some birds carries some of these risks as well. Even wild birds are prone to flight-related trauma (see Section 8: The Trauma Patient, and Chapter 50). Anyone who has worked in a wildlife hospital has seen broken wings, broken legs, concussive head injuries, and other casualties. These cannot always be prevented. The best way to mitigate this risk is to control and carefully select the areas where birds are flown. The complexity of the flying environment can be increased as the flight skill increases. Some environments, such as those with large plate glass windows, wind turbines, transformers, heavy vehicular traffic, and other dangerous structures, are best avoided altogether.

Responsible ways to accommodate free flight: Avian veterinarians should promote responsible pet ownership. The last thing that should be encouraged is anything that could contribute to the establishment of feral populations of nonnative species. Many of the pet bird species would not be able to survive in a cold climate. If they can, it should not be assumed that they would be harmless to the local native species or environment. Displacement of native species, distribution of introduced plant species, and destruction of agricultural and native flora are all distinct potential risks. To prevent this from happening, only well-trained birds should be flown outdoors without restraint. Additionally, these birds should be brought into a safe enclosure whenever they cannot be supervised.

Ideal candidates for free flight are birds that can fly exceptionally well. Flying clumsily across the living room is not an adequate qualification. Free flight will require the ability to turn, slow down, move up and down, and otherwise navigate in three dimensions. Fully functional wings are a prerequisite. Trimming the wings will only hinder the development of the required skills. Injuries could also inhibit adequate flight skills. It is generally recommended to start free flight training when the bird is young. Birds learn flight skills best immediately upon fledging, as this coincides with the time that they master these skills in the wild. At this time, birds will also have an innate need to stay with their perceived parent. This allows them to develop outdoor skills while still having a mental "leash" that keeps them close to the trainer. Birds that are easy to find are also helpful. A large, loud, brightly colored bird is easier to spot in a tree than a small, quiet, green-colored bird. Larger birds present less temptation to hawks, especially when flying in a flock. A group of birds is oddly more difficult for a predator to successfully attack. Flying skills, vigilance in looking for danger, and other skills require practice. These skills can be honed while flying closer to the handler, which will serve as somewhat of a deterrent to some raptors.

Free flight requires a great deal of training of both the handler as well as the bird. Classes are recommended before undertaking free flight. There are few trainers who will encourage this practice for liability reasons. However, they should be sought out and consulted prior to beginning free flight with any bird. In addition to learning how to train the bird, a prospective free flight enthusiast should learn how to retrieve a lost bird and get acquainted with the area resources for doing so. In terms of training flight, there are two phases involved.[114–116] First the bird must learn what is being asked, to then acquire the skills to accomplish the task that is being asked. Since the trainer cannot fly, they cannot teach the bird to do so. Instead, a trainer teaches the bird to come to them for a reinforcer. The bird practices the skills to accomplish this in different circumstances, starting with short hops or flights and eventually culminating in longer flights in more complex environments with air currents, sounds, and other distractions.

PHYSICAL ENRICHMENT

Physical enrichment involves increasing the usable space and complexity of the bird's living environment, including the addition of objects (e.g., toys, chewing materials), substrate, vegetation, or other structural elements (e.g., perches, swings and other climbing structures, hiding places) in the living environment. Offering this type of enrichment will positively affect the animal's behavior by increasing its general activity, locomotory, and exploratory behaviors.

Environmental Complexity

The problem with captivity is, simply put, captivity. Restricting the movements of a bird will limit the extent of exposure to the environment. How restrictive this is largely depends on the environmental complexity available to the bird. A good analogy would be incarceration of prisoners. Placing a prisoner in solitary confinement is far more restrictive than housing one in the general prison population, which is, in turn, far more restrictive than someone confined on "house arrest" with a monitoring anklet. No one likes to think of their birds as prisoners, but many companion birds are given environments far smaller and less complex than an average prisoner is afforded.

Size of the Enclosure

Size of the primary enclosure is one of the factors involved in environmental complexity. Generally, larger spaces will allow a greater diversity of activity than smaller spaces. For example, a crossover study comparing the behavior of budgerigars in

standard-sized (0.65 m^3) and larger-sized (128% and 156% of standard) aviaries demonstrated increased flight distance and more frequent flights following access to additional space, while more wing flapping and tail wagging were observed in the smaller aviaries. Following return to the larger aviaries, flight times increased, suggestive of rebound activity and providing some initial evidence that additional space and flight are beneficial to the welfare of budgerigars.[120] Conversely, extremely small spaces will restrict the available options for activity and lead to the development of locomotor stereotypies and other abnormal behaviors, although this may be affected somewhat by other factors.[45] Nevertheless some birds seem to become bewildered by an extremely large cage. This is likely to be due to early experiences or lack thereof. No parrot has a small cage in the wild, and any aversion to open space should be considered a pathological response, borne from developmental stunting. It is relatively easy to find information about the minimum cage size for birds. However, this may not be the best way to look at the equation. A better way would be to determine the maximum space that *can* be provided by the owner. Once that is determined, then the type of bird to be obtained can be chosen to match the available space. Guidelines stating that a cage should be large enough to allow for the wings to extend without touching the sides and damaging the feathers and tail are grossly inadequate as this size significantly limits the bird's ability to move around. Most birds spend 12 to 22 hours in the cage; it is impossible to allow normal behaviors such as climbing or flying in a space barely large enough to turn around in. Whether or not a bird is flighted, a flight cage, open bird room, or aviary is highly recommended to provide ample opportunity for activity.

Cage Design

In addition to the raw space, the shape and orientation of the cage are also important. Two cages of equivalent volume may have very different amounts of "effective" space. The older cages for small birds with a vertical, narrow, cylindrical shape had no more usable space for the bird than a cage of half the height. Generally rectangular cages oriented horizontally provide the most usable space for birds. As many cages are not appropriately sized, alternative housing may be appropriate for some birds. Rooms can be bird proofed so that a larger area can be available to companion birds for part of the day or even for full-time housing. This can be accomplished by paneling walls with smooth, easily cleaned materials. Steel siding designed for barns can be used for particularly destructive birds (Figure 47.7). Areas around windows, doors, attachment points for perches, toys, or feeders, and electrical outlets should be protected using safe materials. The floor will have to be lined

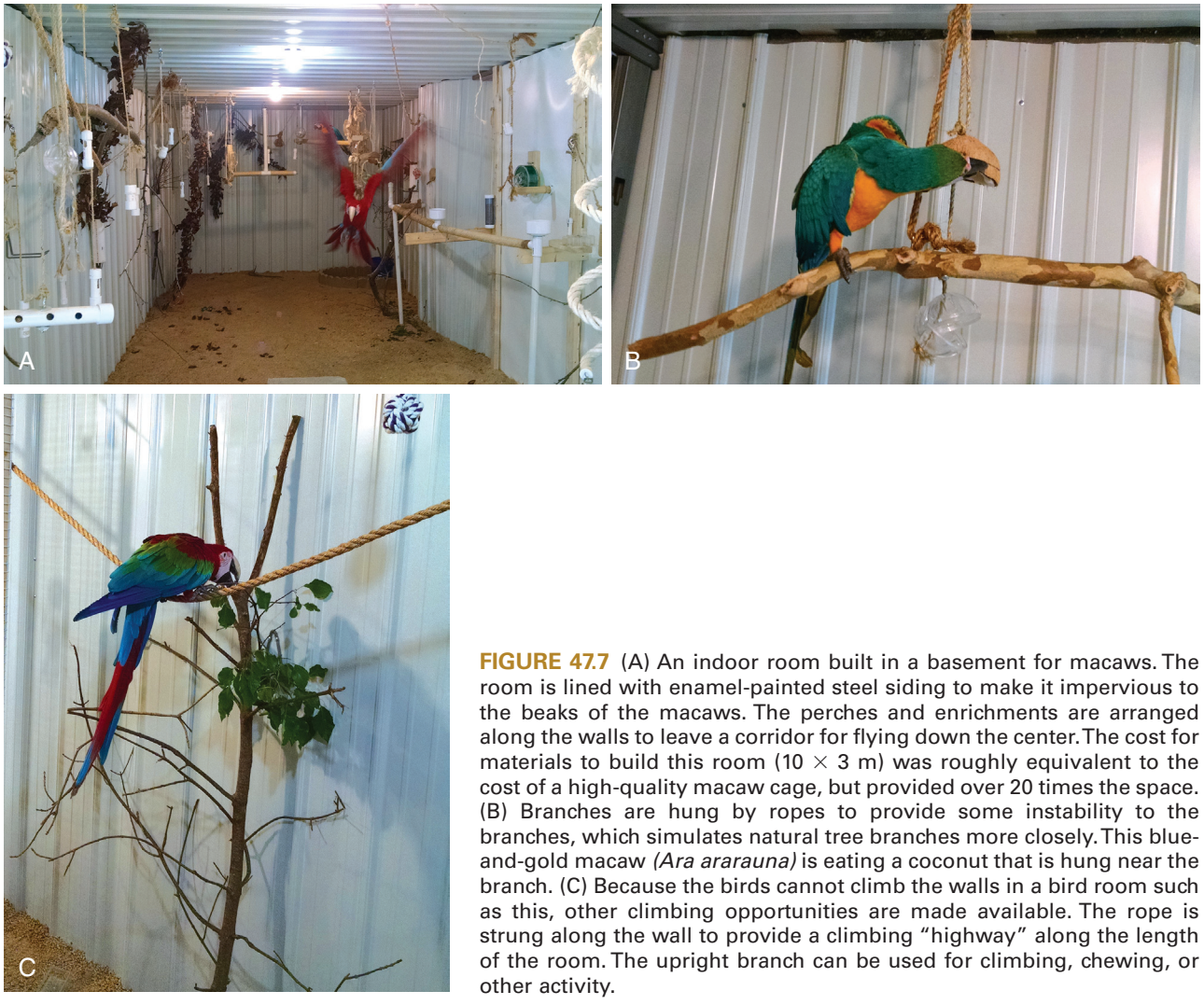


FIGURE 47.7 (A) An indoor room built in a basement for macaws. The room is lined with enamel-painted steel siding to make it impervious to the beaks of the macaws. The perches and enrichments are arranged along the walls to leave a corridor for flying down the center. The cost for materials to build this room (10 × 3 m) was roughly equivalent to the cost of a high-quality macaw cage, but provided over 20 times the space. (B) Branches are hung by ropes to provide some instability to the branches, which simulates natural tree branches more closely. This blue-and-gold macaw *(Ara ararauna)* is eating a coconut that is hung near the branch. (C) Because the birds cannot climb the walls in a bird room such as this, other climbing opportunities are made available. The rope is strung along the wall to provide a climbing "highway" along the length of the room. The upright branch can be used for climbing, chewing, or other activity.

with something easy to clean. While such rooms may require remodeling prior to resale of the home, they will provide a much richer environment for companion birds while in use. Outdoor aviaries are another means of providing additional environmental complexity (Figure 47.8). Size, construction, and furnishing of these aviaries vary substantially as does their purpose. Some provide short-term, supervised outdoor housing, while others are used for full-time housing of the birds. The purpose will dictate the requirements to some extent. Full-time housing will require protection from predators and vermin, a sturdy, beak-resistant design, and protection from the weather. Housing for supervised time can suffice with a simpler construction. Outdoor housing allows the possibility of natural sunshine, fresh air, and a much more diverse array of visual and auditory stimuli. Often, outdoor aviaries are larger than indoor cages. For flighted birds, the transport between indoor and outdoor housing areas should be carefully planned to avoid escapes. Even entry of the caretaker could allow an escape if precautions are not taken. Training a bird to enter a carrier cage for transport would be the safest and least obtrusive way to transport a bird without risk of escapes.

Surroundings

The area surrounding the primary enclosure can be a source of stress or enrichment as well. Well-socialized companion parrots benefit from locating the primary enclosure (cage) in an area of household activity. The area can also be enhanced by placing plants or other interesting items in the vicinity, keeping in mind the propensity for parrots to destroy things they can reach. Ergo, nothing valuable or dangerous should be very close to the cage. Plants can be helpful for shy birds. They can give a sense of visual isolation and security. More on this topic will be discussed under the section on Sensory Enrichment.

Alternative Housing

Play stands are a good alternative site for parrots to spend time. There are numerous arrangements that can be useful. Branches, polyvinyl chloride (PVC) pipe structures, and many



FIGURE 47.8 (A) Outdoor aviaries often provide increased space as well as greater sensory enrichment. The construction is highly dependent on the birds to be housed and the purpose of the aviary. This aviary was constructed from a powder-coated dog kennel. The spacing of the wire is too large for anything other than a very large psittacine bird. It would also not be appropriate for 24-hour daily housing because it would not exclude predators or rodents. However, it provides a pleasant place to spend a few hours outdoors, conditions permitting. Full-time housing would require substantial extra protection and the ability to control climate. A safety vestibule would be essential for flighted birds to avoid escapes as well. Smaller wire spacing is needed for smaller bird species. (B) To extend the season that the aviary can be used, wind blocking materials have been mounted on the exterior of the aviary. These two macaws are acclimated to the weather and enjoy spending time in this aviary when temperatures are as low as 0°C. (C) A smaller outdoor aviary version. This one connects to the indoor bird room in Figure 47.7. The window can be opened and the birds can climb out into the outdoor aviary. Again, this aviary is for short-term supervised activity.

other stands are commercially available or can be readily built to provide a play area for psittacids of varying sizes and activity levels (Figure 47.9). For small- to-medium-sized socialized birds, a basket can be adapted as a portable perch to allow a parrot to follow the activities of the household. Multiple cages can be helpful in some cases, especially in birds that display territorial aggression. A sleep cage will allow the primary cage to be in an area of high activity while providing a dark quiet place to sleep at night. The sleep cage can be smaller and minimally furnished. Alternately, having additional cages in other rooms may just provide a change of scenery. Commuter birds that go to the workplace with the owner have the advantage of staying with their human flock most of the time. The noncontact interaction with various people throughout the day is ideal for well-socialized birds. Travel can be either highly stressful or highly rewarding, depending on the individual

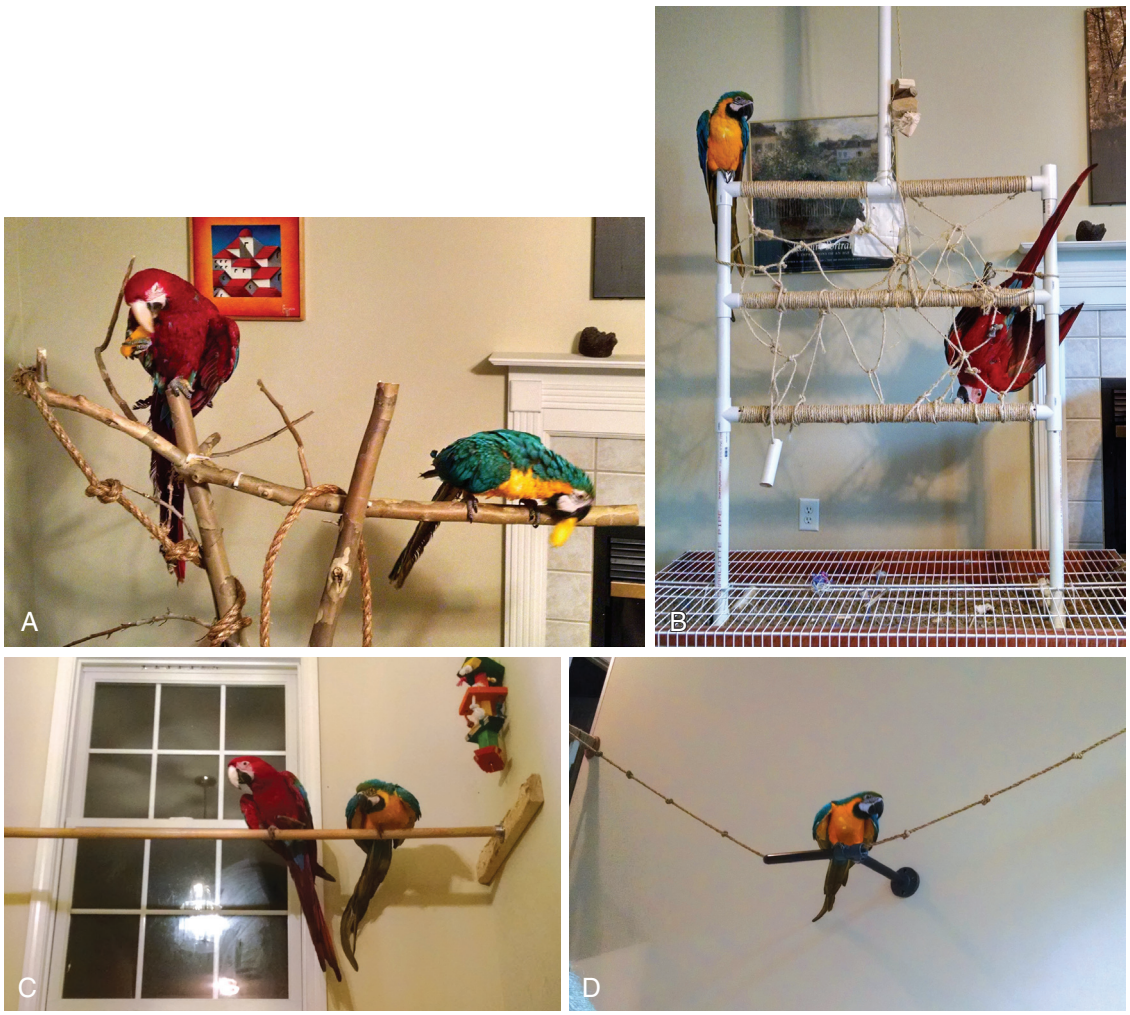


FIGURE 47.9 (A) Play stands allow birds to spend time outside of their primary enclosure. This one is constructed of several natural branches and mounted over a tub to contain messes. Enrichment items can be hung from the branches, and the height is appropriate for training sessions with the birds. (B) A simple play stand constructed of PVC pipe and sisal rope. This stand has multiple levels that allow climbing and a tray base to contain messes. This one has casters to allow it to be moved about easily. (C) This space was above a platform in the foyer of the home. It is largely inaccessible without a tall ladder for people. This was used for flight games with the birds. The birds would fly down to the handler and back up to the perch. There are toys hung at each end of the perch, and the birds could look out the window at the neighborhood. The perch was mounted far enough from the window that the birds did not damage the window frame or ingest anything from the wall. Since the perch is not reachable, this type of play area is only appropriate for birds that are adequately trained to fly to the handler. (D) Additional perches can be mounted in the home. This one has a tightrope walk that provides an additional type of play. Clearly these types of spaces must be carefully applied to prevent injury to the bird, property damage, and other factors. However, they do allow for a greater usage of home space by parrots.

Continued



FIGURE 47.9, cont'd (E) Outdoor play stands can be carefully used with free-flighted birds, harnessed birds, or effectively wing-trimmed birds. Because of the risk of terrestrial and aerial predators, and the ability of parrots to chew through a harness or leash, very close monitoring is needed for this scenario. In this case, a simple play stand is made from an aluminum and fiberglass ladder.

bird and the methods used. Generally, travel will involve more close confinement and sometimes motion sickness, difficulty in delivering water, and other issues. These factors need to be accounted for travel to be a pleasant experience. Certainly, birds that only get into the car to go to the veterinarian will have a more adverse reaction to it than those that go on frequent trips with the owner (Figure 47.10; see Chapter 46). Medical therapies such as metoclopramide or maropitant may be helpful for motion sickness.



FIGURE 47.10 Birds that travel frequently will be less likely to be stressed by it than birds that only travel to veterinary appointments. These macaws are enjoying a camping trip with their caretaker. A screened-in canopy tent provides shade and prevents fly-offs during supervised social time. The perch is a plant hanger that pushes into the ground, wrapped with rope, and hung with simple toys and foragers.

Cage Furnishings

Exploratory opportunities are another feature of avian enclosures that can influence their complexity and positively affect the bird's behavior through increasing behavioral diversity and reducing and preventing abnormal or problem behavior.[21,36,40,42] Parrots have the unique capability among birds to climb using both their feet and beaks. For birds with clipped wings, and those with wing conditions preventing flight, this is the primary mode of locomotion. Provision of cage furnishings that encourage this activity will enhance the ability to explore. Wire cages with horizontal bars provide this opportunity inherently. Additionally, wire cages allow the bird a view of the surrounding area and provide excellent ventilation. However, climbing excessively on the wire of the cage can have deleterious effects on the remiges and rectrices. The feathers often push between bars, bending the barbs back and ultimately breaking them. Alternative climbing opportunities should therefore be available (Figure 47.11). Natural branches are a good addition to most cages. In addition to providing climbing opportunities, they allow for beak activity in the form of stripping bark or chewing the wood. Branches can be oriented horizontally, diagonally, or vertically, depending on the bird's abilities and the configuration of the branches. They can also be hung rigidly between the cage sides or can swing more freely. Very few perches are rigid in nature. If natural branches are unavailable, wooden dowels, cholla cactus spines, rope perches, commercial swings, and PVC pipe can be used. PVC can be roughened with a wire brush to improve grip or can be wrapped in self-adherent bandages or natural twine or rope. Ropes can be used alone as well. The diameter should be appropriate for the size of the bird. The ways the ropes are hung are dependent on the climbing abilities of the bird. Ropes should generally be natural fibers in case the bird chews off fragments and swallows them and to prevent strands from constricting toes. Ladders offer a simple climbing opportunity as well and can be rigid or constructed of rope. Large manila cargo nets are excellent climbing furniture for large birds.



FIGURE 47.11 (A) Climbing opportunities are important for psittacine birds. Perches will be utilized both from the top and the underside by athletic birds. A combination of rigid dowels, rigid branches, springy branches, PVC, bamboo, and ropes can be utilized to provide a means of exploring the environment. Adequate space must be left open for movement of the birds. (B) An outdoor play area for a free-flighted or supervised nonflighted bird. This tree limb mounted in a base has numerous flexible branches suitable for climbing, stripping, and chewing. (C) Sisal rope–wrapped PVC perch. This perch is semiflexible. Rope-wrapped perches should be used cautiously as the fibers can sometimes catch toes. Alternately, PVC can be roughened with a wire brush, wrapped in nonadhesive bandage material, or simply left slippery. (D) This perch is bamboo, hung at a steep angle to provide a sliding option. The same method could be used with PVC pipe. There is also a springy rope perch with a bell in this photo. These are commercially available and appear to be enjoyed by many birds. In the background, numerous branches with foliage are placed to provide chewing opportunities and visual enrichment. (E) Arrangement of this indoor aviary room allows for exploration of the enclosure both by flight and full-length climbing at multiple levels. The window at the end can be connected to a rope to allow the birds to climb out into an outdoor aviary as seen in Figure 47.8C.

One type of item that should be avoided in most situations is a nesting structure. Most species of parrot are quite territorial about their nests and will display aggressive responses to people or even other birds that they would normally tolerate. This is because most species will separate from the flock for breeding. This is the time when the nest structure is usually utilized. Few parrots roost in nests during the nonbreeding seasons. For most parrot species, the nest is a hollow tree cavity. Any hollow item large enough to climb into could be perceived as a nesting structure. The potential nest need not even have an entry hole. Parrots may knock on a cabinet or box and sense the hollow sound. This may be enough to stimulate nesting behavior. Some will chew an entry hole or open a drawer to begin nesting. One possible exception to the seasonal nest usage rule may be Quaker or monk parrots *(Myiopsitta monachus)*. These birds are communal nesters, and nest occupation is not isolated to breeding activity.[121] They also build their nests, which is unique among psittacine birds. Providing these birds with small twigs will allow natural nest-building activity to occur. While this can provide a sort of occupational enrichment, it could create a problem if the bird decides to defend the nest from human caretakers, so the decision to provide nesting materials should be made on an individual basis.

Toys can provide a great deal of activity for parrots (Figure 47.12). There are numerous types of toys that are appropriate for parrots to stimulate different types of activities. Toys should be selected specifically for the species involved and the activity intended to be stimulated. Foot toys simply allow the bird to manipulate the item with the foot. This is usually combined with beak activity or foraging. Birds that would feed in trees in the wild are more likely to use foot toys than ground-feeding species like cockatiels or grass parakeets (*Neophema* spp.). Chew toys are critical for all parrots. These should be constructed of natural, nontoxic materials and should be easy enough to destroy to avoid the bird giving up on the project. Branches with the bark still on them, cardboard, and thin, softwoods are good for birds with low to moderate destructive potential. Harder woods are appropriate for very beaky birds. Long, woven strips of natural materials are available for making toys for parrots. These can be tied or woven into cage bars or other areas. Larger birds can be provided with blocks of untreated lumber, which can be cut into various sizes and presented in a variety of ways, matched to the abilities of the bird. Similarly, natural branches of bird-safe trees can be cut to serve as toys. Wood and branch toys can double as foraging toys by drilling holes in them so that food items can be wedged into them. Puzzles allow the bird to work out a problem to achieve some goal, whether that is a food item, another toy, or just a sound and provide much-needed mental stimulation. Puzzles should be simple at first, gradually increasing the complexity as the bird learns and gains confidence. Some toys provide sensory enrichment and will be discussed in that section. Toys should never crowd the cage to the extent that movement is inhibited. One or just a few of each type of toy can be provided at a given time. Like humans, birds can become bored with continued exposure to the same toys. How many people have home gyms that are collecting dust or serving as a clothing rack? Rotation of the toys will help keep the interest up. However, care must be taken to avoid frightening timid birds with new toys. Some birds will react adversely to new items being placed in the cage. In these cases, toys should be introduced outside of the cage in a nonthreatening way, thereby allowing the bird to approach the toy at its own pace. Bird toys have become somewhat of a cottage industry and costs can range from free or cheap, home-made toys to quite expensive, complex commercial toys. In birds that seem uninterested in toys, teaching or training the bird to use the toy through observational, model/rival methods can be helpful. With some toys, placing small, desirable food items in them can encourage their usage. In addition, parrots can be trained to interact with toys using positive reinforcement and shaping, whereby interactions with the toy are reinforced with food initially. Eventually, the food reinforcer can be faded out, while allowing the reinforcing qualities of engaging with the toy to take over.

Change of Scenery

No matter how interesting a home is, it is always desirable to get away from it occasionally. Even if it seems stressful at first, having exposure to varying environments is critical for pet parrots. The truth is that very few pet parrots will live their entire lives in one home. Their long life expectancy introduces the likelihood of major changes in the home situation. Death or relocation of an owner, rehoming, or other situations necessitate them to be able to adapt to a new environment. When a bird is more accustomed to various locations, this will facilitate transitions to be made smoothly. For well-adjusted parrots, a more variable environment will provide a pleasant stimulation. However, birds that are fearful of new situations may require extra time and behavior modification, including systemic desensitization and counterconditioning, to help them adjust to variations in their environment (see Chapter 46 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).[86] If a bird is afraid of leaving the vicinity of the cage, favorite treats can be given as they are carried to the edge of the comfort zone and maybe one step beyond. Close monitoring for fear responses is important and the bird should be returned to the comfort zone immediately if any signs indicating fear are observed. By adequately responding to the bird's behavior and being patient, fear responses may be significantly reduced and eventually overcome.

SENSORY ENRICHMENT

Sensory enrichment involves providing more stimulation to any of the five senses: sight, sound, touch, smell, and taste. Some of these enrichments can double as social, physical, or nutritional enrichment. For parrots, sight, smell, and touch comprise the main senses, thereby emphasizing the need for provision of visual, tactile, and auditory stimulation.

Visual Enrichment

Since avian vision is among the most acute in the animal kingdom, provision of high-quality visual stimulation is considered a critical part of any parrot enrichment program. Visual enrichment can take many forms, including provision of bright-colored toys, moving objects, videos, and mirrors or changing the position of the cage or its furnishings to change



FIGURE 47.12 (A) A sample toy for parrots. This clear plastic sphere can be opened to place food, smaller toys, or other enrichment materials inside. The bird can work these items out through the slots or open the sphere to remove the items. It can be used both as a simple toy or a foraging toy. (B) This toy was constructed using a stainless steel chain, bamboo, driftwood, and scrap, untreated dimensional lumber. Assembly is very simple. After drilling a hole in each item, the chain threads through and the toy can be hung using a stainless steel clip. Because of the tendency for parrots to destroy toys rapidly, the ability to build simple, inexpensive toys is valuable to parrot owners. (C) This bird room has larger PVC pipes mounted to the wall to allow safe fresh foliage and branches to be placed as in a vase. Here, some willow and crab apple branches are provided for chewing. They can be quickly changed when they become depleted. Because this room does not have typical bars, the hanging wire shelves were used to mount commercial toys and perches intended for parrots in cages. (D) This image shows an array of climbing and swinging toys. To the *left* is the bamboo slide and springy rope perch with a bell as shown in Figure 47.11D. There is one branch hanging from a V-shaped section of rope, and then two large branches that are bolted loosely together so that they sway from side to side when the birds move on them. These options provide exercise and build the birds' athletic ability as well as providing entertainment. The birds will flap/swing from one to the next with remarkable agility.

the parrot's line of vision and view. Since parrots are inclined to investigate and subsequently destroy items within their reach, visual stimuli need to be either out of reach, safe enough to be chewed, or both. Both visual and auditory stimulation will be affected by the environmental complexity. The activity and scenery surrounding a bird's cage should be interesting but not stressful to the bird. Cages usually should be in areas with a great deal of household activity. This allows noncontact interaction with people in the household, which provides both visual and social enrichment. In households where no one is home for most of the day, alternative stimuli should be provided. Television, other pet birds, a view of a bird feeder outside, or other interesting stimuli may be useful. As previously mentioned, plants surrounding the cage location can provide visual enrichment and a sense of security. However, care must be taken that these stimuli do not evoke fear responses, at least not on a constant basis. One may think that watching an outdoor bird feeder would be a great stimulus, but seeing birds snatched up by Cooper's hawks may be more like a horror movie to some birds. This is not to say that brief exposure to threatening stimuli is harmful to a psychologically stable bird, but this will depend on prior experience and the individual personality of the bird. Indeed, these stimuli, and the learned response to them, are essential to the survival of birds intended for release or even for those that engage in free flight.

Some toys provide visual enrichment. Mirrors give a visual representation of a bird and many budgerigars and cockatiels will spend hours talking, singing, and interacting with this image. This can sometimes go to undesirable extremes if the bird begins regurgitating to the mirror or loses interest in live avian or human companions. Birds generally have excellent color vision, although their perception is somewhat different from that of humans as they can see in the ultraviolet and fluorescent spectra.[122] Many bird toys are brightly colored, and there is some evidence that certain colors may be preferred for some types of toys. However, this may not be consistent between individuals or even between objects. Orange-winged Amazon parrots preferred wood blocks that were yellow over red, green, blue, violet, or natural colors. The same was not found with rawhide squares or sisal rope.[27] Because this preference was fairly precise for a particular object, and in a single species, it would seem advisable to provide a variety of colors to allow individual selection of preferred colors. For humans, video viewing is a common form of enrichment (or at least entertainment). There is little information about the use of this method in birds, although anecdotally, one of the authors has observed several parrots (e.g., Amazon parrots [*Amazona* spp.], Grey parrots, cockatoos [*Cacatua* spp.], caiques [*Pionites* spp.], cockatiels) favor specific video clips on YouTube. In fact, one bird was even observed to demonstrate attention-seeking behavior toward the owner, which it redirected toward the television, and once the television was turned on by the owner its behavior became calmer and focused on the video clips that would be played. Chimpanzees *(Pan troglodytes)* utilized a television in both single and group housing situations.[123] While similar studies may not exist in parrots, it appears they are able to translate three-dimensional shapes to two-dimensional representations of them.[124] Also, parrots were able to use a touchscreen device, suggesting that two-dimensional viewing is recognized.[29] However, parrots failed to learn language through watching video.[125,126] Many companion bird owners watch television with their birds. Whether this provides enrichment for the bird is a potential avenue for further studies. Pending such studies, the use of video devices for birds can be considered when access can be safely controlled to prevent damage to the device or toxic ingestion or electrical damage to the bird.

Auditory Enrichment

Auditory enrichment, which can be achieved by provision of sound-producing toys, vocalizations of other birds, animals or humans, or radio (music) or television sounds, has particular importance for psittacine birds. Parrots are known for their vocal capabilities as much as for any other reason. The term parroting is used for the mimicry that these birds are famous for. However, they can do far more than mimic. People who live with parrots have long known what science has now demonstrated. Studies in wild parrots show a great deal of vocal variation and even regional dialects.[127–132] Other studies have suggested specific purposes of natural vocalizations.[133–135] While we have only begun to understand avian vocalizations, some parrots have shown the capability to use and understand human speech.[77,94,136] Parrots also have a strong affinity for music.[31,32] They are one of the few groups of animals that have shown the ability to dance in time with music, usually preferring beats that correspond best to their own vocal timing.[137,138] Other studies showed large interindividual differences with no specific preference for specific beats, though continuous beats were preferred over single beats.[138] Provision of auditory stimuli can take many forms. The ideal situation is direct social vocal interaction. Parrots have been shown to learn vocalizations best from social engagement with the teacher.[88] This makes sense given that the purpose of the vocalizations is communication. Whenever possible, birds should be near other birds or their human family, depending on their early socialization. This allows frequent vocal interactions, even when more direct interactions are not possible. When this is not possible, other options can be used. Simple toys like bells can allow birds to make some sound. Birds can either ring the bell or in some cases use it as an echo chamber for their own vocalizations. Years ago, a toy shaped like a hat was marketed for this purpose. It provided the echo chamber for the bird and the amusing image of a bird wearing a hat to the owner. A child's xylophone could be provided to allow the bird to create sounds. Radios or media devices can be used to play a variety of sounds, including talk, bird vocalizations, nature sounds, and music. One study showed a hyacinth macaw *(Anodorhynchus hyacinthinus)* from a zoo use two interactive devices to control its sonic environment,[80] while another demonstrated that Grey parrots learned to use a touchscreen device to select music that would play when they touched an icon.[29] While it would require some setup to protect the device and the bird, and a bit of training, it allows both auditory enrichment and occupational enrichment concurrently. Other technology now allows two-way audio using pet or security cameras. Some pet cameras also allow two-way video and even dispensing of treats to allow simulation of more direct social interaction.

Tactile Enrichment

Touch can be a potent enrichment. While there is little research available on this topic in birds, there is ample evidence

of its benefits in rats.[139] This benefit may be species-specific or associated with social cues, however, and inanimate tactile sources may not be as effective.[140] There are numerous tactile stimuli that appear to be pleasant to birds. Tactile interactions between humans and birds are usually mutually enjoyable. However, care must be taken to avoid simulating the pair bonding activities when preening a bird. Human-imprinted birds can develop a variety of medical and behavioral problems when their reproductive hormones are stimulated. Most touch should be directed to the head and neck. Additionally, this should be applied sparingly with more active interactions, such as training and verbal play, used more extensively. Some birds will provide self-enjoyment by scratching their head feathers slowly with a foot. Others will use a stick or other tool to provide this stimulus. Many of the available toys have the purpose of tactile stimulation of the beak and tongue. Providing a variety of materials in toys allows the tongue and beak to sense a variety of textures. Some toys appear to have a texture that birds enjoy touching with the beak. Many parrots appear to enjoy whittling wood into small fragments, often giving as much time and attention to a piece of wood as they would a favorite food item. Even clean, nontoxic stones can be enjoyed by some parrots. This activity doubles as self-maintenance by providing excellent wear on the beak keratin. Bathing is a natural part of most species' normal grooming, and many appear to enjoy the sensation. Bathing should be provided in a way that is preferable to the bird. Some options to try would be showers, a fine mist, open shallow water, or even wet vegetation.

Olfactory Enrichment

The relatively small olfactory lobe of most avian species has led to the belief that the sense of smell is not important in birds. In truth, various bird species use olfactory signals to communicate, to find food, to navigate, and possibly for other purposes.[141–148] Unfortunately, studies on olfactory sensation in psittacine birds are scarce, with only one study published on olfactory sensitivity in kea *(Nestor notabilis)* and kaka *(Nestor meridionalis)*.[149] Though the sample size was small and responses differed across individuals, both species were able to discern scents and detect novel sense, indicating these species possess functional olfactory abilities. Taking into account that many parrots have specific scents detectable by their human companions, it would seem logical that these serve a purpose as communication chemicals. Applying this to enrichment is somewhat more challenging. Because of their sensitive respiratory system, the use of any volatile materials, candles, or other perfumes is not recommended. Applying the secretions from the uropygial gland of the same or other bird to objects may provide some olfactory stimulation. Care must be taken when collecting this secretion so that the bird does not become overstimulated. Additionally, the purpose of these cues can be associated with reproduction and their use could promote undesirable behavior as well. The use of aromatic foods and herbs can provide other olfactory sensation, although individual preferences may be challenging to identify and meet.

The number of taste buds in birds is small compared to that in mammals. However, there is ample evidence that birds are capable of distinguishing taste and observably have taste preferences. Although some food preferences may involve visual, olfactory, or tactile sensations, some do appear to involve taste. Hispaniolan Amazon parrots *(Amazona ventralis)* were able to distinguish between levels of sodium in diets, and cockatiels can detect plant chemicals in water by taste.[150,151]

Most olfactory enrichment is provided by providing birds with different flavored foods, although it is possible to provide taste or olfactory enrichment without food by rubbing a small amount of spice (e.g., cinnamon, nutmeg) onto chew toys, or exposing birds to nontoxic (natural) scents. Companion parrots observably enjoy certain food items and often vocalize their enjoyment. The challenge with providing taste enrichment is in doing so without disrupting the nutritional balance of the diet. Generally, vegetables are beneficial to the nutritional plane of companion birds. Finding vegetables that are preferred by a particular bird can be challenging sometimes but can provide both micronutrients and enrichment. Fruits are usually preferred by most parrots, but the sugar content dictates that they must be used more sparingly. Parrots do appear to enjoy peppers and while capable of sensing capsaicin, they do not appreciate it as an irritant.[152] Formulated diets have some differences in taste, and some have natural or artificial flavors added to provide some taste enrichment as well. Provision of a variety of formulated diets has some distinct advantages. In addition to the taste differences, most have mild variation in nutrients and almost always have texture, size, and visual appearances. The differences can provide enrichment, but in addition, most avian diets are manufactured by small companies, and a few have become unavailable. Birds accustomed to multiple diets will adapt much easier than those that have only eaten one type.

NUTRITIONAL OR FORAGING ENRICHMENT

Avian veterinarians have always concerned themselves with nutrition. Provision of all required nutrients is critical to the physical health of any animal and the effects of imbalanced diets are all too familiar to the profession. Nutritional balance can be achieved by giving appropriately formulated commercial diets. With extruded or pelleted diets, each bite generally has all the required nutrients. If anything, the amount provided for some nutrients (e.g., vitamin D, vitamin A, calcium) exceeds the amount that is necessary for maintenance in many birds. Other formulated diets take whole seeds and/or pellets and bind them together into a ball or cake with added supplements. Other diets comprise seed mixtures with supplements concentrated in nuggets. With these "fortified seed mixtures," all the components of the diet must be consumed to provide these same nutrients. This rarely happens since most owners feed far more of the diet than the bird will consume. This can lead to the same nutritional deficiencies generally seen with all seed diets. To best provide for nutritional needs of the bird, feeding a primarily pelleted or extruded diet as the base is recommended, with a defined small amount of unsupplemented grains or seeds added to slightly dilute the nutrients. Vegetables and fruits are added to provide vitamin precursors, antioxidants, and other phytonutrients. The total amount of food provided should be calorically appropriate for the bird. It is unnecessary for birds to have unlimited food in front of them 24 hours a day. This common practice has led to obesity, constant reproductive stimulation, and birds that are poorly

responsive to training techniques. The amount of food that a bird is eating should be determined, and if the body weight is appropriate, the food should be trimmed to exactly what is being consumed, with little or none left at the time that the food is refreshed. However, there are times in a bird's life when this amount will not be appropriate. Birds that are growing, raising offspring, or laying eggs may need additional calories or specific nutrients (i.e., calcium for egg laying), as would birds that are molting, ill, or injured, and diets for these birds should be adjusted accordingly.

Food is important for more than nutrition. In humans, food is a part of every culture. Likewise, food for parrots is more than just nutrition. Feeding takes up a large part of the day. Most birds feed socially. To fill a bowl with a uniform nugget does not provide the same opportunities for these birds. To improve the psychological benefits of the diet, several strategies can be employed. Variation of the diet is one method of making it more interesting. Even with a diet composed of 100% formulated nuggets, variation can be offered (Figure 47.13). Using several varieties not only provides some diversity but may also be a good safety precaution. There have been a small number of recalls of formulated diets over the years where a mixing error has caused dangerous excesses of some nutrients in a diet. Using a variety of types would cut the total amount of any one diet that a bird eats. Extruded diets come in many forms and shapes. These may provide the ability of the bird to hold the food in the foot and manipulate it. There are a variety of sizes of most brands of pellets as well. Even the flavors and scents of the diets vary. Some have food coloring added. Vegetables can be given whole or cut into different sized and shaped pieces. Most wild birds will have variable diets depending on the season, so temporal variation of the diet by securing seasonal produce for the birds may be beneficial (Figure 47.14).



FIGURE 47.13 Even when using formulated diets as the main component of the diet, a wide variety of physical forms can be found. Pellets and extruded diets are available in numerous sizes and shapes. Some are colored with food coloring or natural colors. Pressed, supplemented food balls and cakes are also available. This variation allows usage in various foraging devices and larger sizes encourage podomandibulation. Both increase the time spent feeding and provide challenges for a bird.



FIGURE 47.14 Provision of seasonal vegetables allows for some temporal variation in the diet. Many parrots are reluctant to try new foods, especially the first time they are offered. Sometimes with continued offering, the food will be accepted. Often, a different presentation or preparation of the food will encourage consumption. For example, soft, cooked sweet potato may be preferred over crisp, raw sweet potato.

Insectivory appears to be relatively uncommon in psittacids. Those species that do engage in eating insects generally consume larval invertebrates in plant galls or other such areas. Because of this, chasing and capturing are not major components of the process. Species that have been observed engaging in insectivory include blue-headed pionus parrot *(Pionus menstruus)*, peach-fronted conures *(Eupsittula aurea)*, Meyer's parrots *(Poicephalus meyeri)*, and keas.[153–156] Because the process of acquiring the insect prey is very much similar to that of acquiring plant-based foods, and because of the poor nutritional value of many commercially available feeder insects, provision of this food type has not been widely considered for psittacine birds. However, it may be worthwhile considering feeding insects to species for which it matches their natural diet, especially if breeding is desired. If electing to do so, insects should be fed very sparingly, and be selected for appropriate calcium-to-phosphorus ratio, or be supplemented appropriately.

In addition to variation in the types of food offered, nutritional enrichment can also be provided through variation in the way food is presented to the animal (Figure 47.15). Traditionally, parrots have their food presented to them on a silver (stainless steel) platter, with food being provided food in a single bowl situated a few inches away from a water bowl. While this is convenient for the caretaker, this type of feeding eliminates most of the parts of foraging that wild parrots would engage in, and typically results in companion birds fed this way spending only a small fraction of the time consuming food that a wild bird would in seeking, finding, and obtaining food.[73,157–160] This creates an enormous void in time that often is filled with displacement behaviors, including FDB, and oral and locomotory stereotypies. Moreover, the provision of readily available food fails to challenge the intelligent minds of parrots that are accustomed to spending energy to obtain their daily food requirement.

Foraging opportunities appear to be a requirement for healthy behavior in parrots. In fact, parrots will often preferentially consume food that they worked for, even when food is



FIGURE 47.15 (A) A simple forager can utilize two food items. In this case, a dried date is wrapped in a cabbage leaf and held together with a cardboard tube. The wrap can be readily seen and encourages usage. These will become more quickly used once the bird knows about the more desirable inner food. However, the leafy wrap is often wasted. (B) A kabob of fruits or vegetables can provide simple foraging as well. Depending on where it is located, the challenge here is just to get to it and to hold it as the food is eaten from the skewer. (C) This item was a more complex foraging option on purchase. Each chamber had a different mechanism to open it. One had a sliding door; two had hinged doors oriented in different directions and another a rotating door. In typical parrot fashion, this one was quickly broken and only the hinged door remains. It was also originally mounted on a spinning mechanism, which was also quickly broken. However, it was remounted to wood using a bolt and it still serves as a simple forager. (D) This is another simple device. It is a small cup, made from polyvinyl chloride (PVC) pipe and fittings, suspended from a rope. Food is placed inside the cup. The bird must bring the cup up to the perch, and either reach into the cup or tip it to drop the food into their mouth. PVC pipe is a durable material that holds up well to parrot beaks. (E) This forager is a step up in complexity. This one is closed at both ends and only has a single hole for the food to be removed. This one requires the bird to maneuver the pipe just right so that gravity will put the food where it can be reached with the tongue or it drops into the beak. (F) To add an extra step to (E), a small plug of wadded paper can be placed into the hole. It does not add a large challenge, but some birds will spend some time chewing up the paper before proceeding to the food extraction. Another extra step that can be added is to place a smaller-diameter pipe inside the outer pipe. The inner pipe has a similar hole drilled in it. The bird has to align the holes to remove the food. (G) A commercial foraging toy. This one has five chambers that can have food or toys added. The bird must rotate the wheel to open each chamber. (H) This is a two-stage foraging toy. Food is placed in the top. The bird must slide a lever to drop the food into the lower chamber and then must pull a rope on the bottom to release the food into the cup where they can access it. A toy like this may be too challenging for some birds, particularly those that have not had previous foraging opportunities.

readily available in an open dish (i.e., contrafreeloading).[10,19,161,162] By providing foraging opportunities, making collection of food more of a challenge, feeding time can be increased 2- to 4-fold, hence being brought closer to what would occur in the wild.[15,16,33,37,163]

The process of foraging includes numerous activities. Any of these can be manipulated to provide a challenge to a bird, although some are more difficult to manipulate than others. Some parrots travel long distances from their roosting sites to foraging sites. The location of these sites may be culturally known by the flock, or previously discovered by the individual. Upon arrival to the foraging site, the work is not done. The bird must seek and recognize the food among a great deal of inedible material. This may require further flight, climbing, hanging, or even digging or burrowing. Once localized, food can be procured by detaching it from a branch by plucking, pulling it out from in between substrate or inedible materials, or picking it up from the ground with the beak. Some food will be eaten right where it is found, but sometimes it will be transported to a safe perching site for consumption. Often, food is encased in a tough rind or shell that the bird must penetrate to get to the edible portion. Parrots will often use their powerful beaks to crack or puncture the shells or use it to peel off the casing to then extract the food item hidden within. During this process, parrots commonly manipulate the food item simultaneously with their beak, tongue, and feet, a process called podomandibulation (Figure 47.16). As a final act of foraging, the food is broken down into smaller pieces that are then consumed. Table 47.3 lists the phases of psittacine foraging including ways to manipulate these to create challenges for companion birds.

The importance of foraging for captive animals has become more obvious over the past decades.[10,16,33,34,37,44,163–165] The difference in the time budget spent on this activity in free-ranging versus companion birds may be the greatest of any activity. This may be a significant reason for the frequency of displacement behaviors. Some research has shown foraging to reduce stereotypic and FDBs in parrots.[34,44,164] For these reasons, foraging should be considered the most important enrichment activity that can be provided to any captive parrot. It is important that clients be guided on how to institute an effective foraging program. Simply telling owners to supply the bird with foraging toys will not be effective nor suffice. Foraging items should first and foremost be easy for the caretaker to fill, sanitary, safe, and appropriate for the level of the birds' skills. In addition, the various types of foraging activities should be considered (Table 47.3), which then need to be matched to the feeding behavior of the species and the individual. For instance, many popular psittacids (e.g., budgerigars, cockatiels) are primarily ground-feeding birds. They eat a lot of grass seeds and other items they find on the ground. These birds usually do not possess extensive skills in holding food in the feet or opening complicated nuts. However, they do have keen eyes for spotting food particles on the ground. Hence, for these birds, foraging is best provided by offering food in a foraging tray or filling a flat pan with shredded paper or paper litter, in which food can be sprinkled to allow the bird to search, find, and select these food particles. Tree-feeding birds, by contrast, may collect food from the ground but would take it up in the tree, where they will sit down on a branch to eat it. Sometimes they may find food items in the trees themselves, which can be mimicked by hanging food items from the top of the cage or aviary, stimulating the parrots to stretch out while perched or climb or hang on the sides or roof to reach the food. Often, tree-feeding birds will have greater dexterity with the foot and beak. They use these simultaneously to manipulate and open nuts. Several parrot species, especially the more intelligent ones, also exhibit problem-solving abilities to retrieve their food. One of the commonly used tests to investigate an animal's problem-solving abilities is the so-called string-pulling paradigm, which demonstrates the animal comprehends that an out-of-reach food item can be reached by pulling in the string attached to it. Successful completion of this task has been reported in many psittacine species, including keas, green-winged *(Ara chloroptera)*, hyacinth, Lear's macaws *(Anodorhynchus leari)*, blue-fronted Amazon parrots *(Amazona aestiva)*, Grey parrots, Goffin's cockatoos *(Cacatua goffini)*, greater vasa parrots *(Coracopsis vasa)*, yellow-crowned parakeets *(Cyanoramphus auriceps)*, and spectacled parrotlets *(Forpus conspicillatus)*.[166–175] In addition, several species (i.e., hyacinth macaw, palm cockatoo *[Probosciger aterrimus]*, sulphur-crested cockatoo *[Cacatua galerita]*, Goffin's cockatoo, Grey parrot, yellow-crowned Amazon parrot *[Amazona ocrocephala]*, greater vasa parrot, and kea) have been observed to perform even more complex tasks, such as manipulation, manufacturing, and use of sticks, pebbles, or other tools to retrieve food. As such, these birds are more adapted to common commercial foraging toys.[176–184] Regardless of the type of enrichment provided, foraging should always be introduced as a graduated program. A novice forager should not be given a complex foraging toy. Their inability to achieve rapid success may frustrate them and cause them to give up. Further



FIGURE 47.16 Podomandibulation, or the simultaneous use of the beak and the foot, is a unique feeding habit of parrots. Provision of larger food items encourages this activity and effectively increases feeding time.

TABLE 47.3
Different Activities Related to Foraging and Feeding in Psittacine Birds

Foraging Aspect	Definition/Reasoning	Provision in Human-Managed Birds
Commuting	Travel from roosting or nesting site to the site where food can be found. May be cultural or individually learned.	Place foraging opportunities in multiple areas throughout the habitat of the bird. Requires large enclosure or multiple foraging sites. Ability to fly, climb, or walk to the area needed.
Searching	Localization of food or a potential food source. Within a feeding area, multiple foci of food may be present and dispersed among the numerous inedible materials that are present in their vicinity. Some birds may use their digging or burrowing capabilities to uncover food items.	Scatter feeding is one method. Food can be hidden out of sight or mixed in with safe, inedible material such as bedding, shredded paper, wooden beads, small stones, etc. For tree-feeding species, foragers can be interspersed with other types of toys.
Selection	Birds learn to recognize and identify edible from inedible items in the environment, or differentiate between different food items. They often will be able to distinguish between an empty shell, unripe fruits, etc. and ones that are full of appropriate food. This may be identified by weight, sound, color, or other characteristics.	Companion birds can quickly learn to recognize foraging toys. They may be able to tell if there is food in them by sight, shaking them to hear the nugget, or just trying to extract. Wrapping food in paper is a common foraging option. Many birds palpate the paper to find the nugget of food. Providing a mixture of different food items is another possible alternative.
Procurement	Acquisition of a food item, which may include activities such as plucking, pulling, or pecking. Some food items would be in positions where feeding would not be safe or comfortable. The food may be carried to another perching site to consume.	Some foragers should be in challenging sites that require hanging or climbing to reach. In these cases, most birds will carry the food to a perch for consumption. Many birds like to dunk or soak pellets prior to consumption. Separating the water from the foragers will require some movement to allow this behavior.
Extraction	Many psittacine food items are encased in shells or rinds. The beak and tongue are highly skilled at peeling, puncturing and cracking these open and extracting the food items localized inside.	A limited number of shelled nuts or seeds can be provided for this purpose. Alternately, wrapping pellets in paper or other safe materials can substitute for some of this activity. Many puzzle feeders in which food can be hidden attempt to mimic this process as they require the bird to open a lid, drawer, or lock to then extract the food that is placed inside.
Processing	Rather than consuming food in whole, parrots usually break down food particles in smaller bite-sized pieces before consuming them. Larger food items are often held in the foot, while being manipulated with beak, tongue, and foot (podomandibulation). This is especially true in psittacids that feed in trees. Ground feeders are less likely to engage in this.	Provision of large nugget sizes will encourage this behavior, as would provision of whole, larger-sized fruits, vegetables, and whole nuts.

instructions for clients to train their birds how to forage are found in Box 47.4.

Initially, birds may be trained to develop foraging skills using their favorite treats to help increase their motivation to forage, to then expand to regular food items. Once a bird gets proficient with foraging, the original food dish can be eliminated, and all the food can be offered via foraging opportunities. There are unlimited ways to present the food. Sometimes the food can be hidden in other toys, in the bedding, in holes drilled in branches or perches, or in other places that the bird can explore. Some spots will not have any food, others will, which will help to simulate the natural environment and scarcity of food. Some of the foraging sites should be relatively easy, and some should be difficult. Foraging should always continue to challenge the bird. Ideally, finding and consuming the food should take up a larger portion of the day. Although it would be difficult to achieve the 6 to 8 hours of foraging wild birds may get, even 2 to 3 hours would be a huge benefit. Combining several foraging activities in one enrichment and distributing food across multiple items will be helpful to achieve this goal.[163] In addition, enrichments that will only provide access to smaller portions of the food at a time (e.g., pipe feeders with small holes that will only release a few pellets upon rolling or swinging) or those that are made of chewable materials that will require a great deal of time to chew through will be more effective in increasing foraging times in the long term compared to treasure boxes or other items that may initially require a more complex action but are readily performed once the bird knows the trick.[16]

PROMOTING SELF-MAINTENANCE BEHAVIORS

Preening and other self-maintenance behaviors are critical for health and welfare. Preening takes up a large portion of the waking hours of wild birds and helps to maintain feather

BOX 47.4 INSTRUCTIONS TO CLIENTS FOR INITIATING FORAGING ACTIVITIES

Wait until your bird is mastering one step before moving to a more challenging step.

- Step 1: Reduce the total amount of food to just what your bird finishes in a day. Measure the food before putting it in the dish. Then in 24 hours measure what is left. If there is no food after 24 hours, give a little more the next day. If there is some food remaining, further reduce the food.
- Step 2: Divide the food. Get 5–10 smaller dishes (preferably ones that fit in a ring holder). Initially put most in the original dish and just a sprinkle in each of the others. Once your bird is eating from all the dishes, start to divide the food more evenly between them.
- Step 3: Mix in some nonedible materials as distracters. Some choices would be wooden beads, paper litter, or paper scraps. Initially just put a few in each dish. Gradually add more so your bird has to carefully sort through to separate food from debris. Alternatively, you may sprinkle the food on top of the inedible materials.
- Step 4: Cover the dishes. Take a paper towel, put it over the dish, and tuck into the ring holder. If there is no ring, a rubber band or nontoxic paper tape can be used. Now your bird must chew open the dish, then sort through the distracters to find the food. Initially you may motivate your bird by only covering the dish partially with a thin strip of paper, to then gradually increase the portion of the dish that is covered. Once fully covered, a small hole can be made in the paper to help guide the bird. For more experienced birds, thicker pieces of paper or multiple layers can be considered to increase the level of complexity.
- Step 5: Wrap individual food items. Take a few food items and put these in a small piece of paper and twist the ends. Place these in the dish with the other food items and the distracters.

Other ideas:

- Try hiding a few of your bird's favorite foods on or in other toys, cardboard boxes, etc. This can encourage play and a more inquisitive nature.
- Put a food nugget in a paper cup and then roll the top of the cup around a cage bar.
- For a big challenge, hang a food item at the end of a string or rope, so the bird has to pull it up (or bite through the string to let it drop).
- There are lots of "puzzle toys" available for sale. After your bird masters the steps mentioned earlier, try a few of them to see what works best.
- Try a foraging tree. Set up a stand or tree with lots of little spots to hide a food nugget. Put food in some but not all of the spots. This keeps the bird guessing and looking.

quality, which is critical for their survival. Generally, no encouragement is required for birds to preen themselves. If there is sufficient space and the bird is healthy, preening will proceed normally. Birds housed together will allopreen as well. Although most birds like to have their head feathers preened by a conspecific or a human companion, the feathers will develop normally without this assistance. Allopreening should not be forced upon the bird in the name of removing the pin feather sheaths. Premature removal can be uncomfortable and prohibit full development of the feather.

Bathing also is an important component of skin and feather care, not just to soften and remove dirt and debris from feathers and the skin but also because it encourages preening. For many birds, bathing is an excellent and vigorous activity. Generally, the use of plain, cool-to-lukewarm water will suffice, with no need to add soaps, pesticides, or other additives, as these can potentially be harmful. Birds vary tremendously in the method of bathing. Some birds will prefer fine misting or perching in the shower for secondary spray. Some will bathe in wet grass or washed greens; others rather would like to sit in shallow water and splash around. As a result, different bathing methods should be tried until the preferred method is found.[185]

Toenails are maintained by natural wear, but normal wear produces very sharp tips. This is true with the "grooming perches," sandpaper perch covers, as well as other surfaces. Rough surfaces like these can damage the skin of the feet with prolonged exposure and should not make up a large proportion of the available perching space. Although sharp nails are normal for the bird, they are highly uncomfortable for the handler. Unless the owner wants to wear falconry gauntlets, the nails will therefore need to be kept trimmed. As previously discussed, birds can be trained to do this cooperatively (see Chapter 46).

Beaks also can be maintained by normal activity. This requires that some abrasive surfaces be made available. Using a bowl of stones as a foraging site, providing pumice stones, lava stones, and other items may be all that is required. Some birds may even take a stone (small enough to hold, too big to swallow) in the beak and scrape the stone for several minutes.

CREATING ENRICHMENT PROGRAMS

When creating enrichment programs for individual companion birds or groups of birds, a model that is recommended is the SPIDER (Setting goals, Planning, Implementing, Documenting, Evaluating, and Readjustment) model (Box 47.5).[50] The veterinarian can be involved in the goal setting, planning, and in the evaluation and readjustment phases. Some general recommendations that can be helpful for this process can be found in Box 47.6. Proper implementation furthermore relies on adequate implementation of the enrichment plan by the caregiver, who should receive clear (written) instructions on what and how enrichment should be provided to maximize chances of success.

First, the specific goals for the program should be outlined. These goals can be anything from reducing an undesirable behavior, to occupying a given proportion of the day with activities. Goals should be as concrete as possible and readily measurable. Larger, complex goals are ideally broken into smaller, achievable goals that move toward the goal. For instance, a goal of reducing FDB might be broken down into smaller goals that include an increase in foraging time, a reduction of repetitive behaviors, and an increase in bath time.

While planning the enrichment program, the available space, resources, and caretaker time should be considered. Additionally, the species' biology, the individual's temperament, and early life experiences should be carefully considered, as these in part help determine the value of environmental stimuli for the bird *(nature* vs *nurture)*. For example, the value of enrichments will be higher if these are biologically relevant to the species, hence warranting emphasizing the importance of gaining more in-depth knowledge of the species' natural habitat,

BOX 47.5 THE "SPIDER" FRAMEWORK FOR DEVELOPING AND MAINTAINING A SUCCESSFUL ENRICHMENT PLAN[50,186]

S—Setting Goals

Gain in-depth knowledge of the individual (e.g., rearing history, socialization, early life experiences) and natural history (e.g., natural habitat, dietary preferences, social structure, daily activity patterns) of the species involved, which will help determine its biological, social, and cognitive needs. Next, identify clearly which behaviors the enrichment intends to encourage or discourage. Goals should consider the typical time budget, frequency, and variety of behaviors in a given time period.

P—Planning

Create an enrichment plan to achieve the desired goals. A well-thought-out plan addresses the following aspects:

- the type of behaviors that one intends to encourage with the enrichment
- the resources needed to create the enrichment initiative
- the location, time frame, and circumstances during which the enrichment will be offered
- possible safety concerns that warrant additional monitoring

Note that all enrichment initiatives should continue to allow the animal to maintain a choice and exert control over its living environment.

I—Implementation

Actual execution of the enrichment initiative. Scheduling in a calendar-like format will help to ensure that the items are available, enrichment will be provided, and, by planning, opportunities can be created to present items in novel ways and using varied schedules.

D—Documentation

Documentation should include when, how, and what enrichment was presented, and how the animal responded to it (e.g., type, frequency, and duration of interactions).

E—Evaluation

Evaluate periodically whether the enrichment has the effects as originally intended, whether there are any safety or other concerns with its implementation, and whether benefits outweigh the costs (time and money spent on the enrichment).

R—Readjustment

Modify the enrichment plan by adjusting the enrichment or the way it is offered to optimize its effects on the behavior until the achieved goal is reached.

BOX 47.6 PRINCIPLES CRITICAL TO ACHIEVING A SUCCESSFUL AND EFFICIENT ENRICHMENT PLAN

- The enrichment plan should be holistic, that is, each of the five enrichment categories (social, cognitive, physical, sensory, and nutritional) should be represented appropriately in an enrichment plan.
- The enrichment plan should be goal driven, that is, one or more goals should be determined beforehand to direct enrichment strategies and schedules for an animal. Goals may be both general (e.g., increase exploration) and specific (e.g., encourage podomandibulation).
- The enrichment plan should be assessed both at the beginning and throughout time. Method of assessment may vary but needs to be adapted based on the behavioral goal. If the strategy is not satisfactory to meet the anticipated goal, modifications should be explored.
- The use of a planning protocol is highly recommended to ensure that the enrichment plan is goal oriented, tested, and assessed regularly.

dietary preferences, social structure, and daily activity patterns (see Box 47.7 for helpful resources to learn more about the natural history of selected species).[49,187] In addition, the level of novelty offered should be considered as neophobia and exploration have been tightly linked to species' ecology. A study on 61 parrot species with different habitats and diets indicated high and low levels of neophobia to be associated with diets consisting of insects and leaves, respectively. Additionally, species eating nuts or coming from islands showed the longest exploration times, while those eating seeds generally exhibited short exploration bouts.[26] Other studies have shown individual differences in responses to novelty based on their temperament or coping strategy, as well as long-lasting effects of rearing, socialization, and early life exposure to enrichment on novelty-related fear later in life.[11,20,21,43,46,188–190] Hence, a thorough evaluation of the bird's history and responses to enrichment are warranted. Equally important is to ensure that the plan is properly aligned with the capabilities of the individual bird. Offering flight space to a bird without full, functional wings will not be helpful. Likewise, a bird that has eaten from food dishes its entire life should not be challenged with a complex foraging apparatus from one moment to the next, but rather be gradually introduced to the act of foraging. A well-designed enrichment plan furthermore utilizes enrichment of various types and is composed of a mixture of high-, moderate-, and low-energy components, which again are tailored to the abilities, preferences, and needs of the individual bird.

Implementation should be carried out in a logical and gradual way. Too much stimulation and novelty added at once can induce undue stress in some birds. Challenges should be graduated so that each challenge given to a bird is achievable yet allows it to continue to evolve. This is not only important from the bird's perspective (which may become "frustrated" in the process) but also to keep clients engaged as they may otherwise get frustrated or complacent and stop moving the enrichment program forward. Anxious or nervous birds, particularly those that are fearful of novel objects, may require desensitization for even simple enrichments. For these, it is

BOX 47.7 HELPFUL RESOURCES TO GAIN IN-DEPTH KNOWLEDGE ON THE NATURAL HISTORY OF PSITTACINE AND OTHER AVIAN SPECIES

Books and Encyclopedias

- del Hoyo J, Elliott A, Sargatal J: *Handbook of the Birds of the World*, Barcelona, Spain, 1997, Lynx Editions.
- Parr M, Juniper T: *A Guide to the Parrots of the World*, London, UK, 2010, Bloomsbury Publishing.
- Forshaw JM: *Parrots of the World*, Princeton, 2010, Princeton University Press.
- Toft CA, Wright TF: *Parrots of the Wild: a Natural History of the World's Most Captivating Birds*, Oakland, 2015, University of California Press.
- Luescher AE: *Manual of Parrot Behavior*, Oxford, UK, 2006, Blackwell Publishing.
- Bond A, Diamond J: *Thinking Like a Parrot: Perspectives from the Wild*, Chicago, 2019, University of Chicago Press.
- Sibley DA: *The Sibley Guide to Bird Life and Behavior*, New York, 2009, Alfred A. Knopf.
- Forshaw JM: *Australian Parrots*, Brisbane, Australia, 2009, Alexander Editions.
- Forshaw JM: *Pigeons and Doves in Australia*, Clayton South, Australia, 2015, CSIRO Publishing.

Useful Websites

- *World Parrot Trust.* https://www.parrots.org/encyclopedia
- *Animal Diversity Web.* http://animaldiversity.org/
- *Cornell Lab of Ornithology.* http://www.birds.cornell.edu/home/

often best to introduce enrichment items outside of the primary enclosure to prevent accentuating the fear that would arise following immediate placement of the items in the cage.

Documentation can be challenging. Those providing care for a bird should ideally keep a detailed journal on the provided enrichment that was provided, including observations of its usage, and the bird's behavior in general. However, observations can easily be skewed by wishful thinking, available time for observation, and misinterpretation. Additionally, many behaviors of concern occur when birds are left alone. The presence of the caretaker can greatly affect the type, frequency, and intensity of various behaviors. Video recording may be one of the most useful means of documenting the usage of enrichment elements and should be encouraged, whenever possible. Many trail cameras are motion activated and can be very helpful in tracking activity at a particular location (Figure 47.17). Pet cameras and security cameras are also



FIGURE 47.17 Trail cameras like this one can be helpful for documenting enrichment efficacy. Most have motion-activated features and can take short video clips or still photos. These can be used to determine usage and other behavioral changes resulting from the enrichment provided. The camera should be mounted where it cannot be reached by the bird, and the bird should be habituated to its presence.

options. Journals and recordings can be evaluated by the clinician to determine which components of the enrichment program are the most and least useful.

Evaluation of the enrichment plans involves determining what elements are utilized, how much time they occupy, and the range of behaviors that are being displayed, including any increase or decrease in problem behaviors. To accurately assess the value of the enrichment plan, it is essential for the clinician to receive uninterpreted, objective, and accurate observations from the caretaker or to be able to review raw video footage. Comments such as "it made him hormonal" may represent misinterpretation of the root cause of any given response to an enrichment element (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).

Following evaluation, readjustments may be made, including removal or modification of poorly used elements, addition of more of the well-used elements, or addition of new elements of enrichment. Poorly used elements can be revisited later. Sometimes, moving an enrichment item that has been ignored for weeks to a new location will result in substantial interest from a bird. Additionally, most birds will become more accepting of objects after repeated exposure. As such, this method is particularly helpful for fearful birds, who learn to accept the item through the process of habituation. Foraging challenges can be increased in difficulty as the bird progresses. Additionally, an increasing proportion of the daily diet can be provided in this way. Ultimately, healthy, closely monitored birds can have all of their daily food provided via foraging opportunities. Physical exercise can and should progress as the bird develops better physical stamina and condition. Training can begin simple and gradually include more (complex) behaviors. Sensory enrichment can be adjusted to the preferences of the bird. By following this SPIDER method, the enrichment plan can gradually be adjusted to best meet the needs of the individual bird.

USE OF ENRICHMENT FOR TARGETING SPECIFIC BEHAVIOR PROBLEMS

Enrichment is frequently used as a component for the treatment of specific behavioral disorders (Box 47.8). In the

BOX 47.8 RECOMMENDED ENRICHMENT FOR TARGETING SPECIFIC PROBLEM BEHAVIORS

Problem Behavior	Type of Enrichment to be Considered for Behavior Intervention Planning
Feather-damaging behavior	Foraging and nutritional (food acquisition) enrichment
	Occupational enrichment: any type, particularly promoting beak activity
	Physical enrichment: chewing toys, bathing opportunities
	Social enrichment: conspecifics, human contact
	Sensory enrichment—novelty, auditory, sensory, tactile
Stereotypic behavior	Occupational enrichment: cognitive (oral and locomotor stereotypies), physical (flying, movement)
	Foraging and nutritional enrichment
	Physical enrichment: additional space, toys, puzzles, etc.
	Sensory enrichment, high novelty
	Social enrichment (conspecifics)
Aggression	Physical enrichment—(chewing) toys
	Foraging and nutritional enrichment
	Occupational enrichment—training, flight
	Training alternate behaviors and differentially reinforcing these
	Physical enrichment—change of environmental antecedents (territorial-related aggression)
	Social enrichment—positive interaction with multiple members from the household, stimulation flock activity, limit bonding activities (partner-related aggression)
Fear or anxiety	Sensory enrichment—acoustic
	Physical enrichment—toys (gradual introduction, limit novelty and frequency of rotation)
	Concurrent desensitization training and counterconditioning
Excessive vocalization	Social enrichment—soft contact calls
	Sensory enrichment—acoustic
	Occupational enrichment, including foraging enrichment and physical enrichment (chewing toys)
	Negative punishment (walk out of view when bird loudly vocalizes) and time-outs from positive reinforcement
Reproductive behavior	Foraging and nutritional enrichment—mimic "food scarcity"
	Sensory and physical enrichment—high novelty
	Occupational enrichment—training, redirection
	Limit some social enrichment activities (e.g., cuddling)
	Limit physical enrichment (nest boxes, nest materials, mirrors, etc.)

context of behavioral analysis, enrichment can be looked at as an antecedent arrangement or as a form of reinforcement (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). Because the presence of enrichment is not dependent on the behavior of the subject, it is considered *noncontingent* reinforcement. One of the difficulties in treating behavioral disorders is that they can sometimes be inadvertently or unknowingly reinforced. Reinforcement can come extrinsically, as in social reinforcement, or intrinsically, as in neuroendocrine signals. These reinforcers maintain the undesirable behavior and hinder the success of any behavior modification strategy. In humans, a review of 71 publications showed 265 uses of environmental enrichment, either as a standalone treatment or in combination with behavior manipulations used to either prompt or reinforce alternative behaviors or to block or restrain problem behavior.[191] When used alone, environmental enrichment was effective in 41% of the cases, hence indicating the value it can have to treat problem behavior, but when combined with manipulations of alternate or problem behaviors it increased substantially, depending on the manipulation.[191] This suggests that although environmental enrichment can be helpful, a comprehensive plan should be developed to include additional behavior modification methods.

When utilizing enrichment for decreasing problem behavior, it is best to apply the SPIDER model as discussed in the previous section. Frequent consultations will often be required as the process is fluid and necessitates continuous evaluation and readjustment. While planning the enrichment program, priority should be given to the enrichments that are deemed or known to be most effective. If no research is available to determine what would be the most effective enrichment to help treat a particular disorder, enrichments that encourage behaviors that are incompatible with the problem behavior are often going to be most efficient (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior). Clients can get complacent when problem behaviors start to diminish and may subsequently lapse in their enrichment efforts. Hence, a certain amount of encouragement and reminding may be needed at times to maintain any progress that is made.

When looking at specific examples in companion birds, FDB and stereotypies are the best studied.[33,37] Stimulation of foraging activity in particular has been proposed in the treatment of these behaviors as it is often considered that these abnormal repetitive behaviors should be regarded as displacement or redirected foraging in the absence of appropriate stimulation.[192,193] In addition, stimulation of foraging is

considered highly effective as the activity itself is incompatible with the activity of feather destructive behavior. After all, the beak cannot be used for both activities at the same time! In addition, foraging provides a distinct food reinforcement, which can be comparable to or greater than the intrinsic reinforcement received from the feather destruction. Alternative behavior manipulation in this case would be cueing a trained behavior just prior to when feather destruction is anticipated, and strongly reinforcing this behavior. While this can be helpful, most of the FDBs are done in the absence of the caregiver. Blocking or restraint of FDB is occasionally attempted using collars or other restraint devices. Unfortunately, these have significant adverse impacts on the welfare of the bird, often more severe than the feather destructive behavior itself. As such, the use of collars or restraint devices to block access to the feathers and skin should only be considered for birds with self-injurious behaviors that damage the skin or to help prevent the formation of an automatically reinforcing habit. If and when collars are used, caution is advised, and patient welfare should be monitored closely. The least restrictive option should be used to minimize any undue reduction of normal behaviors.

Repetitive behaviors are common in companion birds, particularly in the larger-brained species (e.g., cockatoos).[192] These are sometimes a consequence of antecedents and consequences that promote perpetuation of the behavior. In such cases, the problem may be resolved by rearrangement of the antecedents and by differentially reinforcing alternative behaviors.[194] Other times these movements represent stereotypies. As stereotypies are generally considered displacement behaviors, it stands to reason that provision of more normal activities would both prevent and treat these behaviors. This has proven to be the case in a variety of species, including parrots.[4,5,9,34,195–197] Selection of the enrichment type here is dependent on the form of stereotypy that has developed. It would make the most sense to utilize enrichments that promote behavior that is incompatible with the problem behavior. Additionally, enrichment that promotes high-energy activities can be helpful.

Excess vocalizations are another common complaint among those living with parrots. It is one of the most common reasons for parrots to be rehomed.[198] It should be noted that psittacine birds are inherently loud and that often the "problem behavior" is perfectly normal, just not desired by the owner. Consultation with a qualified behavior consultant can help with problem vocalizations through using applied behavior analysis (ABA) should the clinician see merit in augmenting their behavioral evaluations and action plans for a particular patient (see *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 5, Behavior).

Normal vocalizations should never be suppressed. For example, birds may produce loud vocalizations when they are fearful; hence the vocalizations are functional as an indicator of distress. Rather than suppressing these vocalizations, the fear should be dealt with by removal of the feared object or by desensitization. Similarly, birds that are hungry can produce loud vocalizations. While birds that forage for most of their diet in the wild are more likely to search their foraging sites for food than to vocalize for food, extended deprivation of food or water is likely to bring on justified complaints from the bird. Again, checking if there is food and water and providing these to the bird will in the end be more effective than trying to suppress the vocalization.

One common nuisance vocalization is the contact call. Parrots often use calls to maintain cohesion of the flock as the dense foliage in which they live precludes visual contact. While many owners construe birds making contact calls as spoiled birds "screaming for attention," they should be reminded that these contact calls are natural parts of the social behavior of many species. Bird caregivers can take the initiative of maintaining contact with lower-volume whistles, whispering, humming, or other sounds that reassure the bird that they are still nearby. Often parrots learn these vocalizations and will use them for flock contact rather than their natural loud vocalizations. Conversely, birds that vocalize loudly should not be engaged. All attention and even eye contact should be averted until the bird is quiet or produces a desired, less loud sound, which can be generously reinforced. For other types of excess vocalization, other forms of enrichment may be helpful. Physical, nutritional, and auditory enrichments appear to reduce vocalizations in general.[32,102] Auditory enrichment at low volumes may be incompatible with loud vocalizations because they cannot be heard over loud squawks.

Neophobic fear responses are common in parrots, and use of enrichment can be helpful both in preventing and treating this behavior, whereby novelty appears as important as the type of enrichment provided.[11,21,186] If tolerated by the bird, frequent changes of toys can be helpful to teach that change is normal. However, some birds are so neophobic that the changing of the enrichment item is inherently stressful. A period of habituation may be needed in these cases. New items should be introduced outside of the primary enclosure until accepted by the bird. It can then be placed in the cage and another introduced outside of the cage. The presence of other birds in the same room may also help, as long as the other bird does not evoke a fear response. Large loud birds can be stressful to a smaller or quieter bird. Otherwise, however, the presence of others may increase comfort. Some birds also respond well to having acoustic enrichment as this helps to decrease the startle response following sudden or unavoidable environmental sounds.

Aggression is a common and serious problem in parrots. The hooked beak of parrots is powerful and can inflict painful bites. Aggression can stem from a variety of causes including protection of a territory or nest site, displaced sexual behavior, or fear.[199] When aggression is directed at human handlers, training can be used as both an occupational enrichment and as a means of promoting and reinforcing alternative behaviors, and a more positive human-avian bond. While training can also be used for aggression directed at other animals, training sessions can only occur when the trainer is present. Physical separation of animals may subsequently be needed until these problems can be resolved. For territorial aggression, a change of physical environment can help. Similarly, physical and nutritional enrichment such as toys and foraging opportunities can provide alternative beak and pastime activity, hence stimulating alternative behaviors and alleviating aggression. Social enrichment in the form of positive interactions with multiple household members can reinforce calm behavior in their presence. These interactions can be "hands-off" and include activities such as talking or singing to the

bird, rather than involving interactions that risk biting or force the bird to choose between aggression and cooperation. Additionally, interactions should not simulate pair bonding, especially if the aggression is partner related.

Excess reproductive behaviors are another common behavioral issue for parrots. These frequently cross over into medical problems. Constant stimulation of reproductive hormones predisposes to excessive egg laying, regurgitation, cloacal prolapse, and other health problems. Enrichment can help to regulate some of these issues. Abundance of food is one of the cues used by some species to determine when to nest. A food foraging program reduces the perception of food abundance. If this is insufficient, the quantity of food can be further reduced. Additionally, some types of enrichment may need to be (temporarily) removed. Any enclosed spaces that could be perceived as a nest cavity may warrant removal or blocking of access. Physical contact by touching the bird should be restricted in quantity and limited to the head of the bird. Training activities should be substituted to redirect the bird's attention from reproductive postures or displays.

In conclusion, enrichment is a multidimensional process that can improve the lives of captive animals. Although there are challenges in providing enrichment for parrots, it is both critical and rewarding for the bird and the owner. The benefits can be seen in health, behavior, and overall quality of life. As advocates of avian welfare, it is critical for avian veterinarians to provide strategies for effective enrichment for their patients.

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SECTION 11 CONSERVATION AND WILDLIFE MANAGEMENT



CHAPTER 48

Introduction to Anthropogenic and Environmental Threats to Avian Wildlife

Heather W. Barron

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The industrial age has brought an increasing number of anthropogenic threats to earth's environment and the wild flora and fauna that inhabit it, birds being notable among these. In North America alone, it is estimated that 3 billion birds have been lost over the past 50 years.[1] Despite these losses, the laws that have shielded migratory birds for over a hundred years have been under political assault and have been weakened at a time when further legislation is needed to protect this valuable segment of earth's biodiversity. Many variables factor into this significant loss but include a variety of anthropogenic factors and a declining environment. As the human population exceeds 7 billion people, natural habitats are increasingly lost or rendered unsafe for avifauna. This section addresses some of the more notable and currently pressing threats to avian wildlife health, including man-made obstacles (e.g., wind turbines and windows), domestic cats, and climate change–induced threats, such as wildfires and extreme weather events. These, alongside nutrient pollution, also contribute to more environmental biotoxins (e.g., harmful algal blooms). Finally, globally roughly 400 million tons of chemicals are produced annually, a number of which are persistent and bioaccumulate.[2] Included here for consideration are petroleum products, heavy metals (e.g., lead), Superfund sites, and pesticides. It has been decades since the discovery that some organochlorine pesticides (e.g., dichlorodiphenyltrichloroethane [DDT]) caused eggshell thinning in wild birds, which led to bans on its use in some countries, such as the United States. However, DDT continues to be used in many countries and the effects of this chemical and its active breakdown products (dichlorodiphenyldichloroethane [DDD], dichlorodiphenyldichloroethylene [DDE]) linger in the environment for decades and are still causing decreased hatchability, for example, in bald eagles *(Haliaeetus leucocephalus)* in the US Great Lakes region.[3] Equally concerning is the fact that these chemicals are still currently found in 99% of humans tested.[2] With growing understanding of these toxins comes the awareness that damage may occur not just from the high-dose acute effects but also from low-dose chronic exposure, which may render these compounds into carcinogens, mutagens, endocrine-disrupting chemicals (EDCs), or have other impacts, such as behavioral modification. These effects, while perhaps less dramatic and obvious initially on the surface, are insidious, alarming, and just as damaging to wild bird population health in the end.[4,5]

The increasing convergence of animals and a growing number of people on an increasingly stressed earth has created a new dynamic in which the health of each of these entities is interconnected. The study of this combined health has become known as conservation medicine and calls for the collaborative efforts of multiple disciplines working to attain optimal "One Health" for all. Diseases that move from wildlife to people are a daily discussion for wildlife veterinarians. According to the Centers for Disease Control and Prevention (CDC), more than 6 out of every 10 infectious diseases in humans are zoonotic. However, only when there is an outbreak of avian influenza, West Nile Virus, or SARS-COV-2 does the importance of wildlife health seem to resonate with the general public. Thus there is a growing need and demand for veterinary expertise to address wildlife, and often in particular, avian health.

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CHAPTER 49

Biotoxins

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HARMFUL ALGAL BLOOMS

Algal toxins encompass a wide variety of naturally occurring toxins produced by different species of diatoms, algae, and cyanobacteria. During periods of growth or blooms, which are large concentrations of aquatic microorganisms such as protozoans and unicellular algae (e.g., dinoflagellates and diatoms), toxins may be produced that can be hazardous to animals and people. Only about 2% of blooms are known to be toxic. These blooms, often referred to as harmful algal blooms or HABs, occur worldwide. Occasionally they may discolor the water, and thus "red tides" are a common name for several types of algal blooms (Figure 49.1). In one documented occurrence, a red tide caused by a bloom of nontoxic dinoflagellate *Akashiwo sanguinea* caused debilitation and death in a variety of seabirds by producing a surfactant that impaired waterproofing of plumage (Figure 49.2).[1]

For most HABs, biomagnification occurs as the toxic organisms accumulate in the animals that ingest them, following which the toxin becomes more concentrated as it passes up through the food chain. Although these HABs have been documented for hundreds of years, growing evidence indicates that the frequency and intensity are increasing.[2] As an example, seabird "wrecks" and mass mortality events of other marine animals are being more frequently associated with HABs. HABs are a serious threat to human, wildlife, and ecosystem health and veterinary practitioners can be an important component in efforts to combat this growing problem. Common algal toxins affecting birds may include domoic acid, cyanotoxins (including saxitoxin and microcystin), and brevetoxins. Birds exhibit a wide range of sensitivities to algal toxins and symptoms vary depending upon the species of bird, the algal toxin, the dose, and the route of exposure (ingestion, dermal, or inhalation).

The occurrence of cases is not always correlative with documented HAB alerts in the area. Algal blooms may occur in another area that is not being monitored, birds may pick the toxins up along their migratory route, or blooms may occur offshore where there is no monitoring. Additionally, a bloom may have occurred several weeks earlier, and shellfish may harbor these toxins for weeks to months before the birds ingest them and are finally exposed.[3] There is also some evidence that birds learn not to eat toxic fish and shellfish over time through conditioned aversion. In the author's experience, over 90% of brevetoxin-affected seabirds are first-year birds. Several authors[4–6] have provided detailed accounts of the impacts of domoic acid (DA) on seabirds in California and Mexico, respectively. In both places, the birds killed (predominantly cormorants [*Phalacrocorax* spp.] and pelicans [*Pelecanus* spp.]) were piscivorous and naive. However, this author has also seen the same individual birds present repeatedly with brevetoxicosis over the course of a single red tide season. It is possible that, as first-year seabirds are less proficient at hunting, these younger individuals may eat affected fish out of desperation, whereas seasoned adults can afford to be more selective. Alternatively, birds may also be exposed through inhalation, although the age distribution of juveniles being affected almost exclusively argues against this. In Maine waters, it has been reported that adult eider ducks *(Somateria mollissima)* avoid toxic mussels under field conditions during annual blooms of *Alexandrium* catenella (formerly A. *tamarense*), which produce saxitoxins. In laboratory studies, eider ducks were offered toxic versus nontoxic mussel meats and they refused the toxic mussels. Those that were force-fed toxic mussel meat regurgitated the food almost immediately.[7]

It is not only wild birds that may be affected. Naar et al.[8] reported four king penguins *(Aptenodytes forsteri)* from a zoo in Kentucky died after being fed fish caught in Monterey Bay, CA, that were found to have high levels of DA. Therefore, animals in captive settings may also be at risk. However, the full impact of HABs on birds is likely underestimated since most free-ranging birds that die at sea are lost to follow-up. Shags *(Phalacrocorax aristotelis)* and other species of cormorants are documented in HAB mortality events more often than other birds, which undoubtedly is a function of their feeding habits (consumption of whole prey with no sampling or testing), choice of food items, and their coastal habitat (more likely in affected HAB zones and more visible once affected). HAB toxins should always be on the differential list for birds with clinical signs (as elucidated here) that may have consumed prey items from areas with a history of HABs. Diagnosis may be difficult. In some cases, a suspected diagnosis is made based on clinical signs exhibited by the birds and reported environmental conditions (including HAB alerts and public health advisories), which may or may not include the presence of algal mats, discoloration, and/or the detection of the algal species in the water where mortality has occurred.

Domoic Acid

DA is a structural analogue of glutamic acid produced by several related marine diatoms (microalgae) in the genus



FIGURE 49.1 Waters around Sanibel Island, FL, showing high counts of *Myrionecta rubra* algae and demonstrating that not all "red tides" are due to *Karenia brevis*. (Photo courtesy Rick Bartelson, Sanibel-Captiva Conservation Foundation.)

Pseudo-nitzschia, including *P. australis* and *P. pungens*. Blooms of this algae can cause "red tides," which may occur in marine waters throughout the world and may be caused by several different types of diatoms, not all of which are harmful.

The form of biologically available nitrogen has a significant effect on the ability of *Pseudo-nitzschia* to multiply and produce DA. Urea is much more potent at stimulating DA and DA-related toxicity than nitrates or nitrites.[9] As the end product of nitrogen metabolism, the sources of urea in marine environments may be natural (e.g., elasmobranchs and marine mammals) or anthropogenic (sewage, agricultural runoff, or fertilizers containing urea).

DA is a neurotoxin that in humans may cause a variety of neurologic signs, including short-term memory loss, leading to the initial designation of the clinical syndrome in people as amnesic shellfish poisoning (ASP). Since 1987, monitoring programs have been largely successful in preventing human incidents of ASP. However, wildlife continues to be at risk worldwide. In birds, DA primarily attacks the brain, causing lethargy, disorientation, fine motor tremors, opisthotonos, seizures, scratching (pelicans will scratch their pouch), vomiting, and death. Behavior may remain altered in affected birds for up to 2 weeks, according to one report.[10] Some evidence indicates that mitochondrial dysfunction and oxidative stress are involved in DA-induced cognitive functional impairment.[11]

DA has also been shown to cause other organ dysfunction in mammalian species and thus these pathologies may potentially also occur in birds. Cardiomyopathy has been documented in sea lions *(Zalophus californianus)* and otters *(Enhydra lutris)*.[12,13] Unstable blood pressure and cardiac arrhythmias may be observed in many species and can be suggestive of a cardiotoxin. Research in mice found that DA levels 100 times lower than

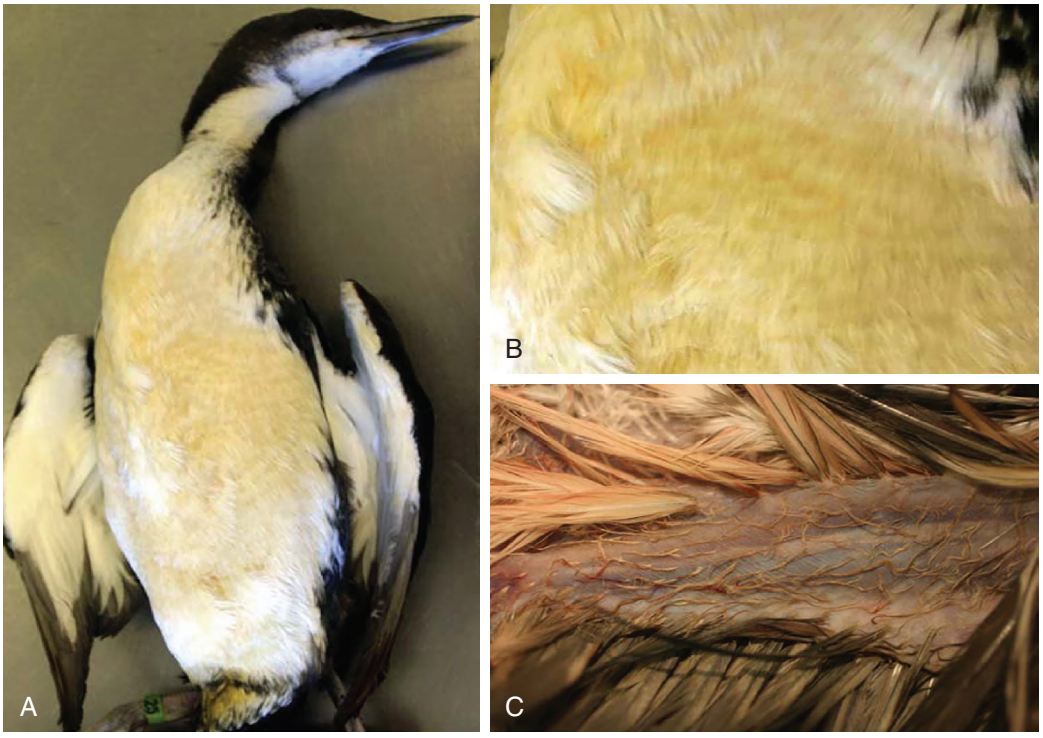


FIGURE 49.2 Dead seabirds with signs of protein-induced loss of waterproofing. (A) A Pacific loon *(Gavia pacifica)* found dead in Monterey Bay, CA, in November 2007 with yellow-green staining of ventral breast feathers typical of over 700 marine birds affected by this event. (B) Closer view of the stained feathers from (A), illustrating the yellow-green discoloration and the oily appearance of affected feathers. (C) Discolored, wet, and matted feathers on the ventral wing of a western grebe (*Aechmophorus occidentalis*) recovered during the same stranding event. (From Jessup DA, Miller MA, Ryan JP, et al: Mass stranding of marine birds caused by a surfactant-producing red tide. *PLoS One* 4(2):e4550, 2009.)

what is necessary to cause neurologic effects can cause renal damage.[14] Gastrointestinal (GI) ulcers may also be noted in affected animals. Seabirds affected by DA commonly include pelicans, murres, (*Uria* spp.), loons (*Gavia* spp.), grebes (Podicipedidae), cormorants, and shearwaters (Procellariidae). Current laboratory diagnostic methods for DA intoxication are challenging as the toxin is highly water soluble and excreted from affected animal's system within 2 to 48 hours, depending on the amount ingested and species of bird. However, DA can be measured in gastrointestinal contents, urine, or serum/plasma if samples are obtained early upon admission.

The mainstay of treatment is supportive care, including nutritional support, diazepam as needed to control seizures, and aggressive intravenous (90–120 mL/kg/day) and oral (tube, 3%–5% body weight, two to three times per day) fluid therapy. Crystalloid fluid choice is usually based on i-STAT results, but Normosol-R is a good empiric choice. As these patients are often hypoproteinemic, colloidal fluid support may also be indicated. Additionally, one study showed that the oral administration of a bioflavonoid called quercetin to DA-treated mice significantly improved their behavioral performance in a novel objective recognition and maze task.[11] Others have suggested nutraceuticals including alpha-lipoic acid (ALA), which is a precursor for glutathione. ALA has been utilized in sea lions with DA toxicosis to improve clinical neurologic signs and may be a consideration for birds.[15] Recovery may be prolonged, generally taking at least 2 weeks, and prognosis is often guarded with previous reports suggesting a 42% survival rate.[10,16]

Cyanotoxins

Cyanotoxins are toxins produced by bacteria called cyanobacteria. Also known as blue-green algae, cyanobacteria are found almost everywhere, in marine, brackish, and freshwater, and under the right conditions blooms may form mats of bright green algae with a putrid odor. There are more than 30 species of cyanobacteria that can be associated with these toxic blooms. Neurotoxic alkaloids (called anatoxins) can be produced by *Anabaena*, *Aphanizomenon*, and *Planktothrix*, while saxitoxins (also called paralytic shellfish toxins) can be produced by *Anabaena*, *Aphanizomenon*, and *Lyngbya* and form potent, presynaptic sodium channel blockers. Hepatotoxic heptapeptides called microcystins can be produced by *Anabaena*, *Microcystis*, *Nostoc*, and *Planktothrix*. Blooming cyanobacteria can produce cyanotoxins that may affect the nervous or respiratory system, liver, or skin. The toxins tend to act rapidly after ingestion, causing neurologic signs within minutes and liver hepatopathy within a few hours as the liver attempts to process these toxins. In one report, deaths of over 100 birds were associated with a microcystin toxicity and some of the affected birds (great blue herons; *Ardea herodias*) were documented to have steatitis.[16]

Typically, clinical signs in affected birds may include depression, dyspnea, vomiting, diarrhea, ataxia, opisthotonos, and seizures. Affected birds may present with blue-green algae in the plumage (Figure 49.3). Death occurs from ingestion of alkaloids leading to neurotoxicosis that produces respiratory arrest or may be induced by cyclic peptides causing acute hepatotoxicosis leading to intrahepatic hemorrhage and hypovolemic shock. Diagnosis can be presumptive based on clinical signs concurrent with area HABs and confirmed with



FIGURE 49.3 A Florida mottled duck *(Anas fulvigula)* receives treatment after presenting with neurologic signs associated with exposure to blue-green algae. In 2018, Florida declared a state of emergency due to the severity of toxic cyanobacterial blooms in Lee and other counties on the Gulf Coast.

enzyme-linked immunosorbent assay (ELISA) kits in both laboratory and field formats for microcystins/nodularin, cylindrospermopsin, and saxitoxins. Affected birds often have anemia and hypoproteinemia.[16]

Treatment is predominately aimed at supportive care, such as oxygen support, fluid therapy, control of seizures with diazepam, and correction of anemia, electrolyte, and acid/base imbalances. Preventing further absorption of toxins from the gut with activated charcoal or administration of cholestyramine may provide some benefit, but death following toxin ingestion is often rapid, and birds are typically found deceased with no real opportunity to treat. Prognosis is guarded to grave.

Brevetoxins

Brevetoxins (PbTx) are algal toxins produced by dinoflagellate *Karenia* spp. (especially *K. brevis*) that may produce "red tides." The major brevetoxin produced is PbTx-2, followed by lesser amounts of PbTx-1 and PbTx-3. Fish, birds, and mammals are all susceptible to the brevetoxins. Animals and people may be exposed by ingestion or inhalation of the toxins, which are lipid-soluble polycyclic ethers that bind to and stimulate sodium (Na+) flux through voltage-gated channels in nerve and muscle. This causes neuronal and muscle cell depolarization, leading to neurologic, gastrointestinal, respiratory, and other signs in affected individuals. In humans, ingestion leads to neurotoxic shellfish poisoning (NSP), but humans are more commonly exposed via inhalation of the toxin.

Morbidity and mortality in birds from HABs is an annual event in many places and is of concern in conservation medicine as avian populations continue to dwindle, and the length and severity of these "red tides" increase. Birds with brevetoxicosis may exhibit ataxia, tremors, depression, gastrointestinal upset (e.g., ileus, diarrhea, melena, hematochezia), bradycardia, lagophthalmos, and respiratory distress. In one report, hundreds of common murres *(Uria aalge)* dying off the coast of California from brevetoxicosis exhibited serosanguinous rhinitis and pulmonary edema as the predominant clinical signs.[17] Differential diagnoses, such as Newcastle disease (paramyxovirus; PMV-1) and botulism, are important to rule

out. Hematologic and biochemical abnormalities are common in affected animals.[18–20] Plasma protein levels may serve as a prognostic indicator in cormorants (and possibly other species) with brevetoxicosis. In one study, the probability of survival was only 50% at total protein levels of 2.3 g/dL, whereas at protein levels of ≤1 g/dL, the probability of survival was 20% or less.[20] Total protein is likely decreased due to effects of the toxin causing damage to the GI tract and subsequent protein-losing enteropathy, chronic GI blood loss, and hepatopathy. Concomitant anemia is often present and degrades the prognosis further. Affected birds are often heavily parasitized with both ecto- and endoparasites, commonly including GI nematodes, and, in some cases, tracheal worms, which may exacerbate respiratory symptoms.[21]

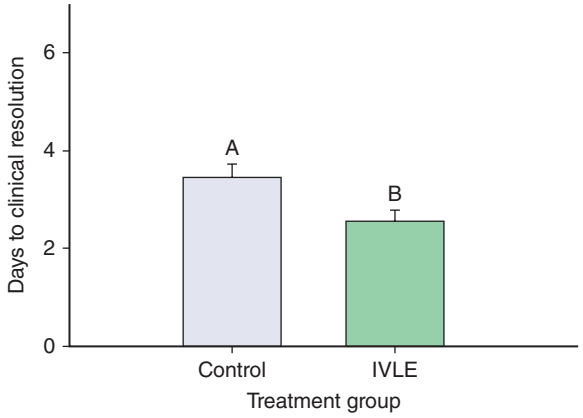
Species commonly affected with brevetoxicosis include double-crested cormorants *(Phalacrocorax auritus)*, brown pelicans *(Pelecanus occidentalis)*, great blue herons, common loons *(Gavia immer)*, and a variety of gulls (*Larus* spp.), terns (*Sterna* spp.), and other Charadriiformes. Interestingly, the species of bird may affect clinical presentation, with cormorants typically showing predominately ataxia, disorientation, and head tremors, while pelicans most commonly show weakness, depression, and GI distress (regurgitation and diarrhea). Gulls and shorebirds, such as sanderlings *(Calidris alba)*, often present with rear limb weakness or paresis. Any species may demonstrate a delayed or absent palpebral reflex, loss of vent tone, and seizures. In the author's hospital, double-crested cormorants are the predominant species presenting with brevetoxicosis during the "red tide" season, which typically runs from October to April on the Gulf Coast of Florida. Cormorants are piscivorous and opportunistic and may prey upon sick or dying fish. In one study, brevetoxin levels in freshly dead fish tissues ranged from 32 to 95,753 ng PbTx g-1. Brevetoxins in shorebird livers were also confirmed (26–1313 ng PbTx g-1) in dead birds collected opportunistically from local beaches and rehabilitation centers during the red tide event, suggesting that brevetoxin exposure is a risk factor for mortality.[22]

Brevetoxin can be assayed by using a mouse bioassay, ELISA, and antibody radioimmunoassay. In clinical veterinary medicine, diagnosis is usually through a competitive ELISA that may be run on plasma or serum (be consistent in which one is used), GI contents, or liver. Sea birds are predominately exposed to PbTx-3, a reduced form of PbTx-2 that is up to 10 times more toxic than PbTx-2 when ingested.[23] Results are expressed in nanograms per gram or milliliter as PbTx-3 equivalents and reflect the overall concentration of brevetoxins and brevetoxin-like compounds in the sample. The detection limit of the assay is approximately 5.0 ng PbTx-3 eq/g of tissue and 2.0 ng PbTx-3 eq/mL of fluid. However, higher levels are common in affected animals and values may be extrapolated through serial dilutions. Levels can be very high in fecal samples, which are easily collected and, in one study, ranged up to over 900 ng PbTx-3 eq/g) in affected cormorants.[24] Interestingly, toxin levels are not correlative with clinical presentation or survival. Toxins are processed by the liver and excreted by the kidneys and in bile.[25] Most dead birds had no significant pathologic findings at necropsy.

Treatment often includes parenteral fluids, nutritional support, antimicrobials, antiulcer medications, antiparasitic medications, blood or plasma transfusions, B vitamin and iron supplementation, ocular medications (in patients unable to blink or with corneal ulcers), manual evacuation of cloacal contents (as required), intravenous lipid emulsion therapy (ILE) (Figure 49.4), and thermal support, if indicated. Birds with active seizures can be treated with midazolam at 0.5 to 1.0 mg/kg intramuscularly or intravenously, whereas birds that were highly stressed in the hospital environment have been given diazepam at 0.5 to 2.0 mg/kg PO every 12 hours for as long as hospitalization lasted. Clearance of the toxin in birds typically takes 1 to 7 days.[26] Clearance is not necessarily more rapid after treatment with ILE, but birds' clinical signs are ameliorated much more rapidly and prognosis for survival is much greater (Figure 49.5). This is possibly because brevetoxin is a lipid-soluble toxin and the ILE solution serves as a "lipid sink" that holds the toxin bound in circulation and prevents it from reaching target tissues until it is excreted.[27]



FIGURE 49.4 Double-crested cormorants *(Phalacrocorax auritus)* receiving intravenous lipid emulsion (ILE) therapy as a treatment for brevetoxicosis. Resolution of neurologic signs was achieved within 24 hours in this case.



Kruskal Wallace test with w/ Cover-Innman Pairwise comparison was used due to the non-normal distribution of data. Test shows comparison of control and IVLE with Days it took till clinical resolution. $P = 0.030$, suggesting significance between control and IVLE. Birds in the IVLE treatment group had clinical resolution, on average, 24 hours earlier than control birds.

FIGURE 49.5 Use of intravenous lipid emulsion therapy for brevetoxicosis in double-crested cormorants *(Phalacrocorax auritus)* significantly improved time to resolution of clinical signs. (Barron H, unpublished data, 2017.)

BOTULISM

Botulism is a disease resulting from ingestion of preformed exotoxins produced by *Clostridium botulinum* and produces an ascending flaccid paralysis that is often fatal. The botulinum toxin is initially inactive when released, and bacterial or tissue proteases are needed to activate the toxin. Spores only contain about 1% of the toxin amount that vegetative cells do. Interestingly, some species of wildlife that have evolved to eat carrion are resistant to botulinum toxin. Virginia opossums *(Didelphis virginiana)* and vultures are good examples. Turkey vultures *(Cathartes aura)* are 100,000 times more resistant to type C botulinum toxin when compared to pigeons *(Columba livia)*.[28] Opossums have a proteinase inhibitor, called lethal toxin–neutralizing factor (LTNF), that occurs naturally in their blood and binds to toxins thus conferring botulinum resistance.[29]

There are four genotypically distinct groups (I–IV) and seven immunologically different types of botulinum toxins (A–G). Waterfowl die-offs are usually from type C (group III); sporadic die-offs among fish-eating birds (common loons, gulls) are from type E toxin, and domestic chickens (*Gallus gallus domesticus*) can be affected by type A (group I). *Clostridium botulinum* spores persist in the soil for years. Toxin production occurs during multiplication of the vegetative form when conditions (warm temperatures in alkaline and anaerobic environments with a source of protein) are favorable. The toxin production often takes place in decaying animal carcasses. Maggots of flies feeding on carcasses concentrate the toxin, and birds eat the highly toxic maggots. Death of the bird then perpetuates the cycle. Removal of carcasses was not shown to be helpful to remaining ducks on the lake in one study.[30] However, under some circumstances, carcass pickup, use of hazing devices to deter bird concentration in an outbreak area, and draining or flooding of fields have been used successfully for management of waterfowl botulism outbreaks.[31]

More than a million deaths have been reported in localized outbreaks, and deaths of 50,000 or more birds are common. Most outbreaks in the United States occur west of the Mississippi River in July through September but certainly there may be individuals that may be affected at any time of the year that flies are viable. The toxin affects peripheral nerves and results in an ascending flaccid paralysis of voluntary muscles, including the muscles of the neck, which explains the colloquial moniker, "limber neck" (Figure 49.6). Paralysis of the nictitating membrane may also occur. Large outbreaks of type C avian botulism at the Salton Sea documented thousands of sick and dying pelicans and other piscivorous birds. Death is often from drowning or from respiratory failure. Important differentials include infectious diseases like avian influenza, Marek disease, duck viral enteritis, and other toxins, such as algal toxins, organophosphates, lead, and castor bean toxicosis. In botulism cases, flight and leg muscles are affected first, so birds become unable to walk and fly. This helps differentiate paralysis due to lead poisoning, which is more spastic than flaccid, and in which birds lose flight ability but are still able to walk. Time to onset of clinical signs after ingestion of the toxin is dose dependent and may be hours to days.

Presumptive diagnosis of botulism is generally made from clinical signs and can be confirmed by detection of the toxin in the serum or GI contents of affected birds. In carcasses it may be more difficult to diagnose because there are no distinct gross or microscopic lesions, but maggots found in the GI tract may be suggestive. GI contents from moribund or very freshly dead birds may also be collected for toxin detection via ELISA or polymerase chain reaction (PCR) (mouse bioassay is the gold standard test but is discouraged for ethical reasons).[32,33] Samples from birds that have been dead for several hours are unreliable because *C. botulinum* toxin can result from postmortem multiplication of the organism and toxin production. Because botulinum toxins are heat labile, samples must be refrigerated or frozen as soon as possible after collection and shipped with ice packs to the testing laboratory.



FIGURE 49.6 Brown pelican (*Pelecanus occidentalis*) with probable type C botulism displaying the classic "limber neck" posture due to ascending paralysis.

Treatment with commercial botulinum antitoxin was shown to be 96.5% effective in one case study in wild birds affected in a natural setting but may be cost prohibitive.[34] Two preparations of botulinum antitoxins are available for treatment of botulism. Trivalent (A, B, E) botulinum antitoxin is derived from equine sources using whole antibodies. The second antitoxin is heptavalent (A, B, C, D, E, F, G) botulinum antitoxin, which is derived from equine antibodies that have been altered to make them less immunogenic. This antitoxin is effective against all known strains of botulism, except for strain H. Otherwise, give supportive care as described for brevetoxicosis. Recovery may take days to weeks as the receptors on the nerves regenerate, but prognosis is generally excellent if the patient survives the first couple of days. Mortality rate can vary from less than 10% up to 40% or more. In some large epornitics, a million or more wild birds may die.

Prevention involves removing decaying vegetation or carcasses to prevent maggot accumulation. In hot, alkaline farmlands in the western United States, delaying irrigation until after winter temperatures arrive can be helpful. There is no approved vaccine labeled for use in wild birds. Active immunization with inactivated type C bacterin-toxoids has been successful in pheasant operations. Additionally, two separate studies reported a vaccine labeled for use in mink was used successfully in ducks to reduce morbidity and mortality from type C botulism but was found to be less efficacious as botulinum dose increased.[35Î37]

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CHAPTER 50

Hazards Related to Man-Made Structures: Buildings, Noise, Electric Power

Glenn H. Olsen

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INTRODUCTION

Many human-made structures can be hazardous to avian wildlife. The most common hazard is one of collision, and this chapter will review problems with glass in buildings, power lines, and wind turbines as sources of collision and trauma to avian wildlife. However, avian wildlife can also be adversely affected by anthropogenic factors that effectively eliminate suitable habitats for avian wildlife. These can include noise factors associated with such things as wind turbines and construction areas, and habitat loss to large solar arrays, not to mention strip mining for coal and oil sands. All these factors have likely contributed to the estimated loss of 3 billion birds in the last five decades.[1]

BUILDING (GLASS) COLLISIONS

Building or glass collisions are estimated to include 253 million wild bird mortalities at homes (structures under three stories) versus 339 million and 508,000 wild bird mortalities, respectively, at low-rise (4–11 stories) and high-rise structures (over 11 stories) annually in the United States alone.[2] The total estimates of mortality from collisions with human-made structures range from 365 to 988 million birds annually.[2]

Humans use reflections and building cues (door or window frames, openings, angles) to help determine where glass is located and to be avoided. While this does not prevent us from occasionally walking into glass panels or doors, it does help significantly reduce the incidence of human injuries. However, birds do not perceive glass in structures in the same manner.[3] Reflections of themselves can often make birds fly aggressively at glass as they perceive the other bird as a rival. Similarly, reflections of vegetation can lead to a bird trying to fly through the glass to the perceived vegetation on the other side. Even on overcast days, glass windows on adjacent walls at the corner of a building, or on opposite sides of a narrow hallway or corridor, may lead to a bird trying to fly through from one side to the other. Historical recommendations for reducing bird/glass collisions have involved the use of predator silhouettes, decals of various forms applied to windows, and placing material such as feathers or string behind or in front of the glass (Table 50.1). However, applying scare tactics with decals or even mock-ups of large predatory birds does not always work, as a bird may perceive that it can fly past or around the obstacle or threat, and still collide with the glass.

Recent scientific studies evaluated the effectiveness of deterrents and summarized the results of flying birds in tunnels.[3] Using tunnels for birds to fly through to test bird vision is a relatively new area of avian research. Budgerigars *(Melopsittacus undulates)* flying in such tunnels have demonstrated that birds have the ability to change wing position to maneuver through narrow spaces along their flight path.[4,5]

For passerine birds, the most common group involved in glass collisions, this means any opaque pattern on the glass must have a narrow spacing relative to the bird's morphometrics. The spacing for the pattern must be less than the body measurements of the bird so the bird will not be tempted to fold its wings in and squeeze through the opening.

The Max Planck Institute in Germany has developed a method of conducting glass avoidance trials with small birds.[6] In their research, recently captured (usually with mist nets) birds are released, with a choice of flying toward plain (control) or patterned glass windows. Injury to the birds is prevented by mounting another mist net in such a manner as to prevent collision with the glass in the test windows. Each bird is tested only once before being released back into the wild. A further refinement in this technique using a testing tunnel was developed at the Hohenau-Ringelsdorf Biological Station in Hohenau, Austria, in 2004.[7] A similar tunnel for testing is currently in use at the Carnegie Museum's Powdermill Avian Research Center, Rector, PA, USA.[3] The binomial choice protocols used at such facilities are a step toward developing a rating for various glass materials, reflective coverings or coatings, and deterrents.[3]

Recommendations coming out of these research centers are used for deterrent materials that can be applied to the outside surfaces of glass (Figures 50.1 and 50.2; VVivid Architectural Window Film Solutions, 3M, St. Paul, MN, USA; Note: any use of trade, product, or firm names is for descriptive purposes only and does not imply endorsement by the US Government). The outside surface of the glass is best for the deterrent so that the deterrent is not compromised by any reflections.[3] Deterrents applied to the inside surface can only be used if the outside surface has a low reflectivity.[3] The tunnel testing procedures of deterrents with passerines in Europe and North America appear to be consistent despite the differences of avian species, latitude, and longitude, indicating that such testing procedures are useful for evaluating various methods and materials to deter avian collisions with building glass.[3] Various jurisdictions in North America are beginning

TABLE 50.1

Listing of Various Modifications Used to Reduce Bird Collisions With Glass in Buildings

Design Feature	Evaluation
Building envelope	Overall extent of glass on outer building surface; bird strikes increase as ration of glass to solid wall increases. Buildings with 25%–40% total window surface reduce bird collisions.
Fly-throughs	Eliminate all potential fly-through conditions such as two windows perpendicular to each other at building corners, narrow corridors or bridges with glass windows on either side, and outdoor glass railings.
Awnings and overhangs	Awnings and overhangs can provide visual clues to birds about windows underneath. These features also help reduce reflected light.
Exterior screens, grills, shutters, or shades	All these elements reduce visible glass, thus reducing bird collisions. These devices can be used to retrofit existing buildings.
Visual markers	Visual markers or images on the glass surfaces give birds visual clues that the glass is a solid surface. The denser the pattern, the more effective a deterrent. Markers need to be high contrast, spaced 10 × 10 cm apart or less, be at least 5-mm diameter each, and be on the exterior glass surface to be most effective.
Nonreflective glass	Not as effective as some other methods when used alone but useful in combinations such as with visual markers.
Opaque glass, translucent glass, stained glass, glass block	Excellent options to let in light but reduce bird collisions. Reduced reflectivity while appearing as a solid object to birds.
Ultraviolet glass	Birds can see in the ultraviolet (UV) spectrum. Glass that reflects or absorbs UV light (300–370 nm wavelength) will appear solid to birds while appearing clear to the human eye.
Low-reflective glass	Mirrored glass is the most dangerous glass for birds. Low-reflective glass (less than 15% reflectance) reduces some types of bird collisions but needs to be combined with visual markers to be most effective.
Angled glass	Angling glass downward from vertical by 20–40 degrees has not been shown to be an effective deterrent to bird collisions.
Interior blinds, interior screens	Sometimes required to reduce bird collisions, these can be raised or removed by subsequent building occupants.
Tinted glass	No scientific evidence that tinted glass reduces bird collisions.
Bird (raptor) decals	Scientific testing has shown these decals as ineffective in reducing bird collisions. Only effective if applied in a pattern 5–10 cm apart over the entire window surface, in which case the decals are acting as visual markers (see Visual markers).

Table developed from the book *Bird-Friendly Best Practices – Glass*, City of Toronto. https://www.toronto.ca/wp-content/uploads/2017/08/8d1c-Bird-Friendly-Best-Practices-Glass.pdf. Accessed January 7, 2022.



FIGURE 50.1 Building at USGS Eastern Ecological Science Center that had a history of previous frequent bird strikes, especially the second-story center, where the hallway between the two wings of the building had glass on both walls. This picture shows the building after all windows were covered with a film (VVivid Architectural Window Film Solutions, 3M, St. Paul, MN, USA) to deter birds from striking the glass. (Photograph by the author.)

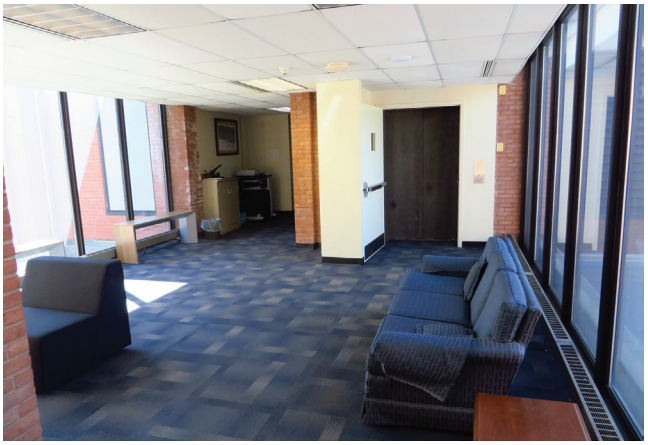


FIGURE 50.2 An inside view of a glass-lined hallway at USGS Eastern Ecological Science Center, Gabrielson Laboratory, that had a history of frequent bird collisions. This view is after a film (VVivid Architectural Window Film Solutions, 3M, St. Paul, MN, USA) was placed on outside window surfaces to deter bird collisions. (Photograph by the author.)

to require bird-friendly glass (glass that reduces or eliminates collisions) to be installed on new buildings and during major renovations of older structures. New York City recently passed legislation requiring new buildings and those undergoing major renovations meet bird-friendly standards to reduce bird strikes.[3]

NOISE

Anthropogenic noise sources can be a problem for some avian species. Noise can mask communications among members of a species, displace birds from breeding or foraging habitat, disrupt predator-prey interactions (e.g., owls hunting by sound at night), impact mate selection, and may cause injury to bird hearing.[8] Physiological responses to exposure to noise may include hearing threshold shifts, hormonal changes, and even tissue injury. Interestingly, noise may facilitate nesting success in some species as a result of the disruption of predator-prey dynamics.[9] A study in northwestern New Mexico, USA, evaluated the influence of noise on nesting community, nest density, and nesting richness in woodland habitat adjacent to natural gas wells with noise-producing compressors ("treatment") versus those that lacked noise-generating compressors ("control"). In this study, they found that noise reduced nesting by a major avian nest predator, the western scrub-jay *(Aphelocoma californica)*. However, with reduced use of the higher noise areas by the western scrub-jay, fewer nests of other species were predated, resulting in a positive effect on nesting success.[9] Nests were also significantly less parasitized by the brown-headed cowbird *(Molothrus ater)* in the noise-polluted sites as compared to control sites ($P = 0.003$).[9] However, the study also found that noise levels reduced nesting community species richness from 32 species at control sites to 21 species at high noise level sites ($R = 0.19$, $P \leq 0.001$).[9] These findings indicate that certain bird species are much less tolerant of noise pollution, resulting in a shift in species diversity in noise-polluted areas.[9]

Forest birds largely use vocalizations to communicate with one another. Typically, these vocalizations are in the range of 1.5 to 4.0 kHz, which coincides with anthropogenic machinery noise.[10] If noise from anthropogenic sources competes with male bird songs used to attract mates, then the birds in the area with noise pollution could be less likely to attract a mate, or, alternatively, the noise-polluted area could be less desirable as a breeding territory. One study examined the noise produced by compressor stations along oil and gas pipelines in the boreal forests of Alberta, Canada.[10] The authors found 92% of male ovenbirds *(Seiurus aurocapilla)* at control sites (no machinery noise) successfully paired versus only 77% ($P = 0.02$) at the noise-polluted compressor sites.[10] The noise-polluted sites were less often chosen by older adult ovenbirds (after second year [ASY]). Male ASY ovenbirds were found at 70% at control sites versus only 52% ASY male ovenbirds at noise-polluted compressor sites.[10] The reduced pair formation and, thus, nesting success of the ovenbirds at the noise-polluted sites may be attributed to the male ovenbird vocalizations not being heard or being less effective because of the background machinery noise. This study suggests that other songbird species may likewise be negatively impacted by anthropogenic noise. Other effects of anthropogenic noise can include reduced ability to detect (hear) predators approaching, changing the frequency of song to avoid the frequency of the anthropogenic noise, and the sounds made by nestlings not being heard, resulting in decreased feeding and increased nesting mortality.[10]

Another study of anthropogenic noise on wild birds in the Rattlesnake Canyon Habitat Management Area (Bureau of Land Management), New Mexico, USA, found that with western bluebirds *(Sialia mexicana)*, mountain bluebirds *(S. currucoides)*, and ash-throated flycatchers *(Myiarchus cinerascens)* there was a consistent pattern of hypocorticism in response to increases in noise amplitude.[11] This study found decreased fitness in the form of hatching success, reduced body condition of the hatched chicks, reduced feather growth (with noise levels above 70 decibels [dB]), and reduced body size of fledglings (also with noise levels above 70 dB).

In the Netherlands, a similar association between age, pairing status, and anthropogenic noise has been identified in willow warblers *(Phylloscopus trochilus)*. In this study, however, the anthropogenic noise source was automobile traffic, and the noise affected willow warblers for distances up to 200 m from the highway.[12] The US Geological Survey's Breeding Bird Survey (BBS), which is conducted each year along multiple roadside locations, also indicates that automotive noise levels along roads may lead to diminished use of the nearby habitat for breeding by many other songbird species.[13] This could be interpreted as a population decline of breeding birds in those roadside areas but may also represent an avoidance of the roadside area due to the increase in anthropogenic highway noise rather than a habitat-wide decline in species numbers.[13]

Hearing capability has been measured in about 50 of the 10,000 species of birds worldwide.[8] Many studies measure hearing ability of birds in laboratory settings or quiet field settings, while underwater hearing has been measured for a few bird species that spend most of their lives in/on water. These include penguins, gannets, sea ducks, auks, and a few other species. Sounds travel further underwater than in air and, therefore, can impact birds over a greater area. Some of the anthropogenic noises in the underwater environment (Figure 50.3) that could negatively affect sea or diving birds include propeller noise from boats and ships, construction noise, sonar activities from naval vessels, and the thrumming sounds of wind turbines transmitted down the pylon into the water.

Recently, diving bird hearing has been measured by both behavioral audiograms and auditory brainstem response (ABR), both in the air and underwater.[8] Typically, ABRs were compared to the spectrographic analysis of vocalizations for each species. The dominant vocalization frequency corresponds well with the frequency of best hearing (1–2 kHz; Figure 50.3) in most species, except the common eider *(Somateria mollissima)*. The vocalizations of common eider (0.44 kHz) were significantly lower than their frequency of best hearing (2.4 kHz).[14] Also, northern gannet *(Morus bassanus)* had comparatively poor hearing, second only to the common eider. Both species are colonial nesters, and their hearing possibly reflects their need to communicate only across short distances in a crowded breeding colony.[8] This lower hearing threshold in these two species may give them some additional protection against noise disturbance in their environment.[8]

The waterfowl species with the most sensitive hearing among the tested bird species were the lesser scaup *(Aythya affinis)* and the ruddy duck *(Oxyura jamaicensis)*.[8] These two

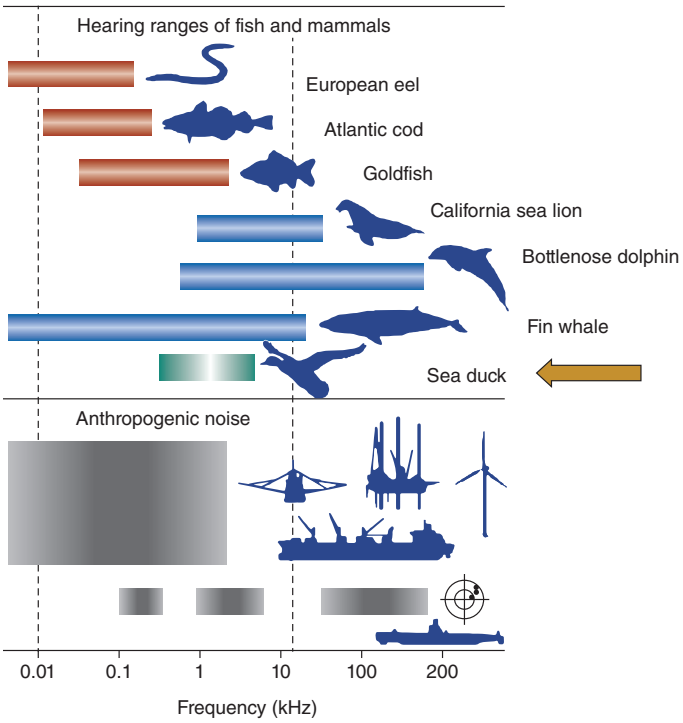


FIGURE 50.3 Hearing ranges for various animals and examples of aquatic noise relative to data collected on sea ducks. (Modified by Alicia Berlin, adapted from Slabbekoorn H, Bouton H, van Opzeeland I, et al: A noisy spring: the impact of globally rising underwater sound levels on fish, *Trends Ecol Evol* 25(7):419–427, 2010, used with permission.)

species are more often found in inland, freshwater environments that tend to be quieter than the coastal and marine habitats where wind and waves create noises. Since their hearing is most sensitive, the potential adverse impact of introduced anthropogenic noise would likely be highest for these two species. In freshwater habitats, which represent their habitat, boat traffic, construction, locating wind turbines, etc., should therefore be avoided or minimized, if possible.

ELECTRIC LINES/POWER GRID COLLISIONS

Two subsets of the electric power networks are especially troublesome for birds, that is, electric power lines and wind turbines. Concern for the detrimental effects of the electric power production grid on avian species has grown over the years. Most recently, this concern has expanded even further as wind turbines are sited with increasing frequency across the landscape (see section on Wind Turbines).

Power line or transmission line collisions and electrocutions are primarily a problem for larger bird species, including swans, cranes, and large birds of prey. To reduce power line collisions, the utility companies, the US Fish and Wildlife Service (USFWS), and the National Audubon Society, along with the University of Idaho and Clemson University, Clemson, SC, USA, created the Avian Powerline Interaction Committee (APLIC) in 1989. According to the APLIC, there are over 804,500 km (about 500,000 miles) of bulk transmission lines in



FIGURE 50.4 A dead sandhill crane *(Antigone canadensis)* found under a powerline in the Platte River Valley in Nebraska. Such findings are frequently seen in the spring when sandhill cranes and whooping cranes *(Grus americana)* use the fields adjacent to river roost sites and often fly into or from the fields in low light conditions at dawn or dusk. (Photograph by the author.)

the United States alone.[15] In addition, these electrical lines have nonconducting ground wires installed above the conducting wires, to mitigate lightning strikes that would otherwise cause power outages. These ground wires are the ones most often hit by low-flying birds as these are smaller (0.9–1.3 cm diameter) and therefore less visible than the conducting wires.[16,17] Large birds are either injured by the collision or found dead under the wires due to crashing into the ground following the collision (Figure 50.4). Estimates of avian power line mortality in the United States range up to 175 million birds annually.[17] One author[18] observed 7000 flights of various types of birds over a power line in North Dakota, USA, and counted about 1% of the birds colliding with the lines. He estimated 124 fatalities/km/year. In North America, 36% of juvenile sandhill crane *(Antigone canadensis)* mortality is attributed to power line strikes.[16] The mortality rate for red-crowned cranes *(Grus japonensis)* in Japan was 2.1% for adults and 13.4% for juveniles. Mortality from power line strikes comprised 44% of total mortality for juvenile trumpeter swans *(Cygnus buccinator)* in Wyoming, USA.[19] For Dalmatian pelicans *(Pelecanus crispus)* in Europe, the estimated mortality from power line strikes varied from 1.3% to 3.5%.[17,20] Power line strike mortality can become a biologically significant factor for small, endangered bird populations. As an example, whooping cranes *(Grus americana)*, which number less than 800 birds in the wild in North America, had a mortality rate of 12% from power line collisions in the Florida Nonmigratory Population, 17% of all mortality in the Eastern Migratory Population, and 39% of all mortality in the now extirpated Rocky Mountain Population.[17] For the largest migratory flock of whooping cranes, the Aransas-Wood Buffalo population, which migrates between northeastern Alberta, Canada, and the Texas Gulf Coast, USA, estimates of

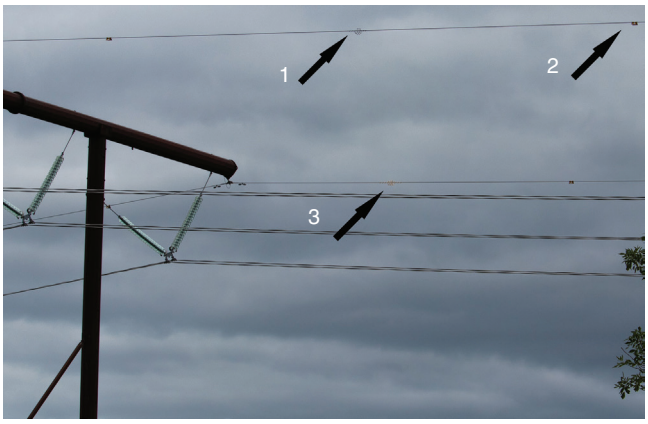


FIGURE 50.5 Three different bird flight diverters attached to power lines. *Arrows 1* and *3* are pigtails: *3* is yellow and *1* is gray. *Arrow 2* is the Powerline Sentry marker (Power Line Sentry, Wellington, CO, USA). "Hannah Jones/International Crane Foundation"

total mortality caused by power line collisions varies from 33% to 38%.[21]

Power line collisions can be related to weather conditions. Weather that causes reduced visibility (fog, precipitation, low-light conditions) or reduced maneuverability (high winds) increases the chances of a power line collision. To address concerns for these detrimental effects, power lines in areas frequented by large birds, such as wetlands and foraging areas, can be equipped with markers of high-impact polyvinyl chloride in shapes that enhance the visibility of the power line to the birds.[15] These methods include aerial marking spheres as commonly seen on power lines near airports. Other markers, including spiral vibration dampers, bird flight diverters, and swan flight diverters (Figure 50.5), have been used and shown to significantly reduce collisions.[17] Other recommendations are to (1) monitor placement and design of new power lines, avoiding areas within 2 km of roosting or foraging sites, where possible; (2) remove unnecessary power lines from areas frequented by large avian species; (3) bury new, and in some cases, older, distribution lines; and (4) use yellow-colored markers as these may be more visible to birds, especially in low-light conditions.[17] Markers on power lines have been shown to reduce bird mortality by 57% to 89% in the Netherlands,[22] 53% for nonpasserines species in South Carolina, USA,[23] and 61% to 63% for cranes and waterfowl in Colorado, USA.[24] As the use of these markers on power lines is relatively inexpensive and can be targeted for areas known to be sites of avian mortality incidents, use of these should be encouraged.

WIND TURBINES

The increased demand for affordable "clean" energy sources has expanded the use of wind turbines around the world. In the United States in the last 15 years, over 59,000 land-based wind turbines have been built in 41 states, Guam, and Puerto Rico.[25] However, wind turbines can frequently pose a threat to avian species. It is important to understand these threats and to develop feasible and cost-effective plans to minimize adverse avian events.[25]

The location of wind turbines can adversely affect raptors and other large species, which may collide with the rotating blades. The Altamont Pass Wind Resource Area in California, USA (37°43′57″N, 121°39′9″W) has caused 28 to 68 golden eagle *(Aquila chrysaetos)* collision mortality events annually.[25] These high rates of collision fatalities have only been recorded at a few wind turbine sites. The raptor species adversely affected also vary by site. Modeling has been used to try to predict potential mortality of raptors at wind turbine sites. Such modeling techniques utilize the hazardous footprint of the facility, bird exposure to the wind turbines, and collision probability.[26]

In Europe, offshore wind production has grown rapidly.[27] The United States has lagged far behind Europe in offshore wind turbine placement, with only five wind turbines operational off Block Island, RI, USA, in 2020. The rapid siting of wind turbines offshore in Europe and increased interest in such facilities in the United States has spurred the interest in studying possible effects on marine wildlife.[27] For avian wildlife, this includes collision mortality, habitat loss from avoiding wind turbine areas, possible barriers posed by wind turbine arrays to efficient migration, and increased energy expenditure by birds when avoiding these areas. Unlike land-based wind turbines where searches for avian carcasses in the vicinity of wind turbines is an established technique, counting mortalities from offshore wind turbines is very difficult. In Europe, studies before and after wind turbine construction have demonstrated avoidance behavior after the establishment of wind turbines.[27] Meta regression analysis of multiple wind farm studies shows that Anseriformes and Charadriiformes are the most negatively impacted.[28] According to this study, population numbers of long-tailed ducks *(Clangula hyemalis)*, common eider, and common or black scoter *(Melanitta nigra)* should be considered most vulnerable to negative impacts of wind farms.[28] However, not all bird species are necessarily displaced by offshore wind farms. In one study in Belgium,[29] northern gannets *(Morus bassanus)*, common guillemots *(Uria aalge)*, and razorbills *(Alca torda)* were found in significantly decreased abundance (85%, 71%, and 65%, respectively) after construction of an offshore wind farm. However, lesser black-backed gulls *(Larus fuscus)* and herring gulls *(Larus argentatus)* numbers increased significantly. Similarly, the numbers of other gull species increased, but not by significant numbers. Based on these findings, the authors speculate that offshore wind farms may offer gulls increased roosting or feeding opportunities.[29] Falconiformes, Accipitriformes, and Passeriformes also appear less negatively impacted by wind farms.[28] Nevertheless, studies of the impact of wind farms on birds thus far fail to identify if the wind farm results in an overall decline in bird populations or a decline in use due to avoidance and therefore shift to using other areas.[28] Similarly, no study has looked at the overall possible negative impact of wind farms on birds versus the negative impact of climate change from continuing to burn fossil fuels to generate electricity.

In the United States, the Federal government has jurisdiction over waters beyond three nautical miles (5.6 km) where wind turbines would be placed. During the past 10 years, the Department of Interior, Bureau of Ocean Energy Management, has funded aerial- and boat-based surveys to document marine birds in coastal waters.[27] In addition, a multiyear tracking study using satellite telemetry has focused on three key species, that is, northern gannets, red-throated loons *(Gavia stellata)*, and surf scoters

(Melanitta perspicillata).[27] All three species use the Atlantic coastal waters as their habitat for 7 months of the year (October–April) and stay within 5 km of the coast, except when migrating along the coast where there are large estuarine incursions (e.g., the openings of Chesapeake Bay and Delaware Bay).

Wind turbines are frequently located in the Great Plains, habitat to grassland bird species. Grassland birds suffer from many anthropocentric disturbances. The greatest disturbance has been the conversion of much of their native grassland habitat to row-crop agriculture. New energy development has added another source of habitat loss for this group of birds. Large sections of the prairies, starting in Illinois and Indiana, USA, and now continuing in Wyoming and other western states, have been permanently altered by strip mining for coal, primarily used in electric power generation. Other areas of the prairie, such as in Texas, USA, have experienced changes with the development of the oil industry with noise pollution, construction of roads bisecting prairie habitat, etc. Now new wind farms composed of aggregates, multiples or even hundreds of wind turbines, have permanently altered the prairie habitat, as have solar energy arrays.[25] In the Great Plains and Inter-mountain West, large developments such as these degrade habitat quality, increase noise levels, increase roads that are used by large vehicles, alter predator populations and use (roads increasingly used as predator travel corridors), and allow the introduction of invasive plants that replace the native plants whose seeds the grassland birds rely upon.[26]

OTHER ENERGY-RELATED ANTHROPOCENTRIC AVIAN WILDLIFE CONSIDERATIONS

Energy infrastructure and development can have less lethal effects on avian species than would be seen with power line or wind turbine collisions. Birds are common in most landscapes. Changes in these landscapes to accommodate energy infrastructure can lead to changes in avian behavior and even population declines.

Lesser prairie chicken *(Tympanuchus pallidicinctus)* populations in Colorado, New Mexico, Kansas, Oklahoma, and Texas have suffered population declines tied to habitat loss, caused by grassland to cropland conversion and energy development.[25,30] A major factor in grassland to cropland conversion in recent years has been the development of center-pivot irrigation systems, made possible by cheap energy and the development of transmission and distribution lines, to bring this electric power to evermore remote stretches of land. Researchers have documented the avoidance behavior of some species of grassland birds around energy production sites.[25] This has led to the development of decision-support tools that can be used for conservation decision-making and justification for compensatory mitigation in the form of prairie land purchases and preservation.[31]

Whooping cranes migrate along a 2500-mile corridor from the Texas (USA) Gulf Coast at Aransas National Wildlife Refuge to the Wood Buffalo National Park in northeastern Alberta, Canada. During this migration, they not only encounter increasing numbers of electrical transmission and distribution lines, but now also must avoid arrays of wind turbines and hundreds of square kilometers of severely disturbed habitat in the form of the Canadian oil sands mining area immediately south of their breeding grounds in Alberta. Indeed, radio telemetry studies have shown that the whooping cranes fly over this area on both northward and southward migratory trips without stopping at any rest areas. When birds must modify migration routes to avoid areas, this requires more energy to make the migration and may require stopover sites in unfamiliar territory or longer stopovers to meet energy demands for longer migration flights. Such changes can lead to predation in unfamiliar territory or reduce nesting time once arriving on the breeding grounds.

Dams to produce hydroelectric power can also permanently alter avian habitat. Loss of large stretches of natural habitat along the Missouri River in the central USA has adversely affected species such as piping plover *(Charadrius melodus)* and least tern *(Sternula antillarum)* that use the riverine sandbars as nesting habitat. Now the US Army Corps of Engineers, responsible for the dams and the river, is starting to create suitable nesting habitat for these birds as part of the Missouri River Recovery Program.[25]

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CHAPTER 51

Petroleum Products

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INTRODUCTION

Many substances fall into the category of petroleum products, from extracted crude oils to heavy fuel oils and other distillation products. Crude oil is a complex mixture that can include long straight-chain hydrocarbons, short-chain (10 carbons or fewer) hydrocarbons that can be saturated (alkanes) or unsaturated (alkenes), branched hydrocarbons, aromatic hydrocarbons including polycyclic aromatic hydrocarbons (PAHs), sulfur-containing compounds, and heavy metals.[1] Crude oil composition, and thus its toxic properties, varies between and within sources, over time due to weathering, and with commercial processing, due to addition of compounds to improve commercial characteristics.[1] Weathering may result in evaporative loss of more volatile compounds, photooxidation and polymerization, biodegradation, dissolution, and emulsion in water.[1–4] Variation of specific toxicants in petroleum products results in a wide variety of toxic effects in birds and other animals, including humans. Long-chain hydrocarbons, such as those in mineral oil, act as a cathartic and decrease gastrointestinal (GI) transit time, thus affecting hydration and electrolyte status through GI losses and directly inhibiting absorption of some fat-soluble nutrients. Short-chain hydrocarbons and smaller PAHs, as found in gasoline and kerosene, are volatile and increase the risk of direct damage to alveolar membranes due to inhalation or aspiration after oral exposure but are lost quickly due to weathering.[1,2] Larger PAHs, which are components of coal and tar, remain in weathered oil, becoming relatively concentrated.[1,5]

Spills of petroleum compounds (oil spills) are reported worldwide. Since 1907, there have been more than 140 large oil spills (>30 metric tons or ~210 barrels) that have released more than 7,000,000 metric tons (about 49,000,000 barrels) of oil into the environment.[3] These numbers do not include the countless smaller spills, which often remain undocumented.[4] Approximately 1,300,000 metric tons (about 9,100,000 barrels) of petroleum enter the world's oceans every year, including 260,000 metric tons (~1,820,000 barrels) from North America. About 45% of the worldwide input is due to natural release, 37% is associated with consumption (cars, boats, runoff from urban areas), 11% is due to petroleum transport, and 3% due to extraction activities.[6] Thousands of oil spills are reported annually in the United States, and although most are small, there have been at least 44 recorded oil spills that have released at least 1500 tons (about 10,500 barrels) of oil.[7] Since 1970, the largest international oil tanker spills on record are the 1979 Atlantic Empress spill in the West Indies (287,000 metric tons, more than 2 million barrels), the 1991 ABT Summer spill off Angolan coast (260,000 metric tons), the 1983 Castillo de Bellver spill near the South African coast (252,000 metric tons), and the 1978 Amoco Cadiz spill off the coast of France.[8] There are numerous Asian spills, which are often poorly reported in the United States, including the Sanchi spill in the East China Sea (136,000 metric tons, around 1 million barrels).[8] The Exxon Valdez in Prince Edward Sound, Alaska, by comparison, spilled 37,000 metric tons (~260,000 barrels) of oil.[8] Oil spills reported in the United States can be searched at https://incidentnews.noaa.gov/.

The largest accidental oil spill documented is the BP Deepwater Horizon (DWH) spill, which released more than 650,000 metric tons (over 4.5 million barrels) of oil into the Gulf of Mexico.[9,10] The DWH oil spill began on April 20, 2010, with an explosion and fire at the DWH drilling unit off the coast of Louisiana, killing 11 workers and injuring another 17. Oil release from the DWH drilling unit was uncontrolled for nearly 3 months, covering more than 100,000 km^2 of the surface of the Gulf of Mexico and at least 1700 km of shoreline in Louisiana, Mississippi, Alabama, and Florida.[11] The spill killed countless wildlife, including tens of thousands of adult and fledgling birds, as well as decimating the next year's recruitment. More than 3500 birds were seen visibly oiled and 8500 dead birds, including at least 93 different species, were found.[9]

Because oil spreads over aquatic surfaces and persists for a prolonged period, marine and freshwater aquatic birds that make the water surface their habitat are particularly susceptible to oiling.[1] However, other avian species may also be affected; for example, aquatic birds and other organisms are fed upon by birds of prey, and animals debilitated by oiling are more likely to be predated.[12,13] Since many birds migrate thousands of miles, a single point source can have far-reaching effects on other food chains.[13]

Heavy oiling of birds can be rapidly lethal due to both effects on plumage and systemic toxicosis. Multisystemic pathology, affecting GI, cardiovascular, renal, and immunologic function, can result from exposure to low doses of crude oil and other petroleum products and result in mortality.[9] Oiling furthermore causes considerable stress in birds, as does retrieval, cleaning, and captivity of oiled birds. While birds may be rehabilitated after exposure to lower doses,

there are detrimental effects to both egg production and hatch rate with subsequent potential to negatively impact populations, especially those that are small.[14–17] There are also significant differences in species susceptibility to oil toxicosis due to anatomic, physiologic, behavioral, and life history factors.[18] Birds that frequent an aquatic habitat are at higher risk of exposure than passerine birds, for example. Oiled pelagic birds in more temperate and arctic climates are more likely to experience depletion of energy reserves to maintain body temperature than those in more tropical climates. Among birds associated with aquatic habitats, birds that are calmer around humans, such as penguins, are more likely to survive past rehabilitation than shore birds that are anxious and stressed in captivity.

Environmental effects of individual oil spills continuously impact birds and other wildlife for many decades. Oil spills essentially sterilize beaches, killing corals and invertebrates in sediments, thereby causing long-term food chain effects. Spills destroy habitat by killing marshland plants and changing microbial populations in soils and sediments, leading to increased shoreline erosion.[10,11] Coastal mangroves, for example, prevent coastal erosion and provide important nutrients and habitat, including breeding habitat, for many species in the marine food chain. Recovery of mangrove forests and salt marshes takes decades.[12,11] Plants, fish, and invertebrates that have survived oil spills or repopulated damaged areas are continually exposed to residual petrochemicals, possibly leading to long-term impacts on aquatic food webs.[4,11] A study of seaside sparrows *(Ammodramus maritimus)* from the US Gulf Coast noted increased CYP1A5 enzyme activity, related to PAH exposure, in 2013 after Hurricane Isaac redistributed oil deposited from the 2010 DWH spill.[11]

Attempts to remediate oil spills also bring a host of problems. In situ burning, which attempts to remove oil by combustion, converts petroleum to products such as carbon dioxide, water, soot, and so on.[19] Though combustion may decrease exposure of marine birds to residues, experimental results suggest that combustion products may cause more feather damage than the original oil.[19]

Chemical dispersants, composed of mixtures of surfactants, organic solvents, and petroleum products that cause smaller droplets of oil to form in the water column, have been used to redistribute oil from the surface of water deeper into the water column, hence potentially expediting oil decomposition.[20,21] However, there are significant differences in the chemical composition of dispersants, and their toxicity must be individually evaluated. For example, Corexit 9580 is predominantly composed of petroleum distillates (volatile petroleum products), whereas Corexit 9500 has up to 30% petroleum distillates, and Corexit 9527 does not contain any petroleum distillates, based on manufacturer information.[22] Dispersants not only reduce the surface tension of the water, they bind to feathers, allowing water to pass into plumage and causing experimental eider ducks *(Somateria mollissima)* and murres *(Uria aalge)* to become waterlogged; the decreased ability to float could potentially lead to drowning deaths.[19,20] Additionally, the use of organic solvents to disperse oil results in increased bioavailability of previously hydrophobic oil particles, as well as deeper dispersion into sediments.[23,24] This results in increased measurable toxic effects in vertebrates, including sea turtles, when oil and Corexit are applied together. While this has not been explored in birds to the authors' knowledge, similar effects are expected. It is important to note that, while Corexit is currently listed on the website of the Environmental Protection Agency (EPA) as "nontoxic," evaluation of the EPA's toxicity table reveals that the Corexit 9500 and oil combination (which is the only composition relevant to field settings) has two to three times the acute toxicity as the reference toxicant, sodium dodecyl sulfate (Table 51.1).

TABLE 51.1

Toxicity of Corexit 9500

EPA-REPORTED TOXICITY DATA FOR INLAND SILVERSIDE FISH *(MENIDIA BERYLLINA)* AND MYSID SHRIMP *(MYSIDOPSIS BAHIA)*

Material Tested	Species	LC_{50} in ppm (mg/L)/time to reach 50% lethality
Corexit EC9500A	*Menidia beryllina*	25.20/96 hours
	Mysidopsis bahia	32.23/48 hours
No. 2 fuel oil	*Menidia beryllina*	10.72/96 hours
	Mysidopsis bahia	16.12/48 hours
Corexit EC9500A and No. 2 fuel oil (1:10)	*Menidia beryllina*	2.61/96 hours
	Mysidopsis bahia	3.40/48 hours
Reference toxicant (sodium dodecyl sulfate)	*Menidia beryllina*	7.07/96 hours
	Mysidopsis bahia	9.82/48 hours

LC_{50}, Median lethal concentration.
From Environmental Protection Agency. COREXIT™ EC9500A: technical product bulletin. https://www.epa.gov/emergency-response/corexittm-ec9500a; Accessed April 10, 2020.

ACUTE EFFECTS OF PETROLEUM PRODUCTS

External Oiling

External oiling is the most common form of oil exposure in birds, causing the majority of sea bird mortality and producing a number of detrimental effects.[19,25] Feathers serve important roles in flight, insulation, and, in pelagic and aquatic birds, buoyancy.[1] Even small amounts of crude oil disrupt feather structure, collapsing hooks, barbs, and barbules and affecting functionality.[25] Oiled birds also pluck feathers while preening, further disrupting the protective properties of the plumage.[26] Consequently, oil ingestion results from normal preening behavior.[25,27] Oiling can be very difficult to identify in brown birds, and underreporting of darker birds in comparison to light or white birds was documented in field reporting during the DWH oil spill (Figure 51.1). Instead of visually inspecting darker birds, it may be easier to identify potential oiling by smell, especially at distance.

Oiled feathers lose the ability to insulate and trap air, causing decreased buoyancy and allowing cold water to reach the skin. Oiled birds in cold climates can die rapidly from the resulting hypothermia.[9,27] Even in warmer climates, oiling decreases birds' thermoregulatory capabilities.[9,28] Poor thermoregulation leads to

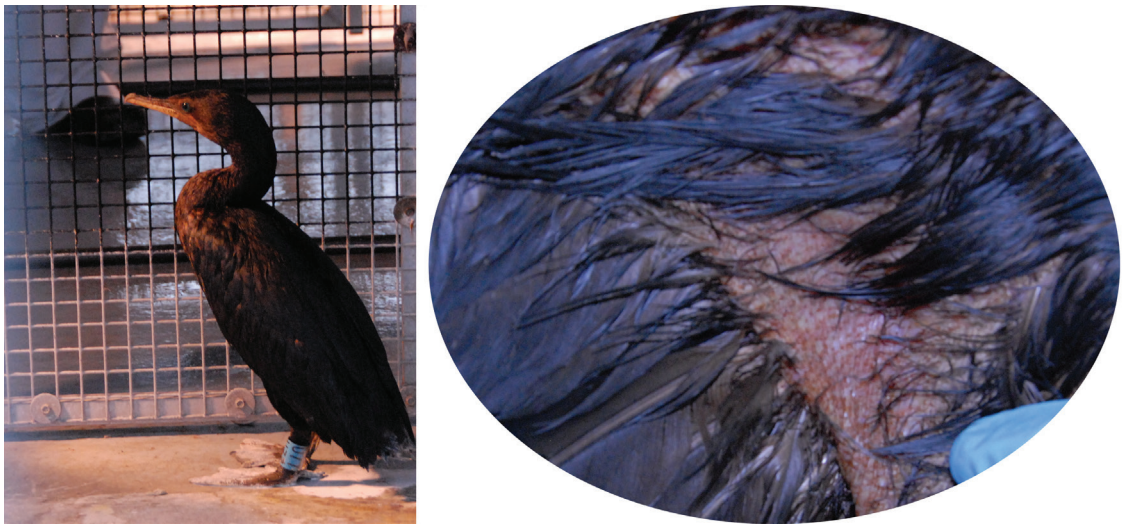


FIGURE 51.1 Cormorant (*Phalacrocorax* spp.) with <15% coverage of oil (approximately one tablespoon) applied to breast every 3 days for six total applications. It is difficult to see the oil on the brown bird at any distance even when the bird is standing in the light. Closer inspection of the axillary region *(right)* reveals significant oiling with compromised feather integrity that negatively impacts flight and allows heat loss.

increased energy demands for heat production and preening. When combined with decreased food availability, decreased time available, and decreased foraging efficiency, weight loss occurs. This, in turn, decreases reproductive and migratory capabilities.[9,28]

Oiling affected flight performance in experimental homing pigeons *(Columba livia)* and western sandpipers *(Calidris mauri)*.[25,29–31] Oiling significantly increased the time it took pigeons to travel a set distance for a month after application, mostly due to the pigeons' inability to sustain flight.[29] Sandpipers experimentally oiled with weathered MC252 had reduced take-off speed, lowered take-off angle, and flew shorter distances than controls, which would negatively influence their ability to escape predators and complete their annual migration across continents.[27,31] Experimentally oiled homing pigeons experienced increased weight loss and failed to recover the weight as quickly as control birds, possibly due to increased energy expenditures for flight and thermoregulation as well as toxic effects of preened oil on the GI tract.[25] The slowing or delay of migration due to feather damage and weight loss is likely to impact survival and reproductive capacity.[29,30]

Gastrointestinal Effects

Birds ingest oil in contaminated food and water and by preening oiled feathers. Oil ingestion during and after an oil spill is likely to occur over days to weeks.[1] Long-chain hydrocarbons have a cathartic effect when ingested; diarrhea and decreased nutrient absorption result.[32] Decreased specific nutrient absorption, including glucose transport, was noted in experimentally dosed herring gulls *(Larus smithsonianus)*.[33] Over time, these changes can lead to reduced body condition.[9] However, the cathartic action also leads to rapid elimination, which is hypothesized to decrease absorption and may reduce systemic effects of oil ingestion.[5,34] Irritation of the GI tract was associated with regurgitation in pigeons and murres.[18,35] GI hemorrhage and regurgitation were reported in oiled murres entering rehabilitation, and many birds that survived bathing later developed skin and leg lesions and respiratory problems.[35] Regurgitation increases the risk of aspiration of volatile hydrocarbons, leading to lipid pneumonia.

Pulmonary Effects

Lipid pneumonia can occur in heavily oiled birds from inhalation of oil droplets and aspiration of regurgitated, oil-contaminated material.[1] While respiratory effects were not a targeted, quantified end point of the United States Fish and Wildlife Service (USFWS) avian toxicity study, researchers noted dyspnea in orally and dermally exposed cormorants (*Phalacrocorax* spp.) while normal respiratory function was maintained by control birds throughout the study.[36] The weathered MC252 oil used in the study had significantly reduced volatile petroleum products in comparison to unweathered crude oil and, therefore, birds could have markedly increased respiratory damage when exposed to other crude oils or distillates.[2,37,38]

Neurologic/Sensory Effects

Oil exposure is associated with lethargy and obtundation in birds.[26] Cormorants dosed with artificially weathered MC252 oil, either orally or via external dosing, were lethargic. In addition, those with oral dosing kept their heads under their wings and were anorexic and unresponsive to external stimuli.[26]

Ocular damage has been reported in murres experimentally exposed to oil, dispersant, and oil plus dispersant.[21] Affected birds had conjunctivitis and many developed corneal ulcers and anterior uveitis, which in turn can affect foraging and predator evasion behavior and can lead to permanent vision loss.[21]

Reproductive/Embryotoxic Effects

Nesting birds that become even mildly oiled will transfer oil from breast feathers or feet to the surface of their eggs.[1,39] Even microliter (μL) quantities of oil on the eggshell can lead to embryonic defects, reduced egg hatchability, and increased mortality prior to fledge.[1] A study on weathered DWH crude

oil on mallard duck *(Anas platyrhynchos)* eggs found a median lethal dose of 31 μL/egg or 0.5 μL/g, though much lower lethal doses have been reported from other oil sources.[39] Absorption of PAHs through the eggshells is the most likely cause of embryonic deaths and explains the difference in toxicity between crude oil and weathered crude oil.[1] Postmortem examination of fresh embryos and hatchlings may reveal liver necrosis, renal degeneration, and generalized edema or no identifiable gross or histopathologic lesions at all.[1] Chicken eggs exposed to airborne volatile compounds from DWH crude oil had embryonic changes; volatile compounds, predominantly benzene, toluene, xylene, and ethylbenzene, caused thrombocytopenia, increased plasma osmolality, and increased CYP1A expression across several tissue types in embryonic chicks.[40]

Birds surviving oiling can have reduced egg laying, egg viability, and hatchability, possibly related to endocrine disruption.[1] Behavioral changes in the parents can be a potential cause of reduced hatchability, but structural effects on the egg, such as reduced shell thickness and yolk composition, were also reported.[41] There is evidence that oiling survivors can return to reproductive success.[1,42,43] Though there was reduced hatching success in rehabilitated little blue penguins *(Eudyptula minor)* the first season after release, no differences were detected in egg laying, hatching, or fledging success in subsequent seasons.[43]

SUBACUTE TO CHRONIC EFFECTS OF PETROLEUM PRODUCTS

Cardiovascular Effects

Chronic low-dose dermal exposure to artificially weathered MC252 oil (less than three tablespoons over 3 weeks) was associated with cardiac arrhythmias, including bradycardia and tachycardia, in cormorants.[44] No histologic changes were noted, but the hearts of both cormorants and laughing gulls *(Leucophaeus atricilla)* appeared flaccid on necropsy (Figure 51.2).[44,45] Echocardiography on oil-exposed cormorants indicated decreased systolic function and decreased left ventricular stroke volume, suggestive of dilative cardiomyopathy.[36] Cardiac isoform troponin I was elevated above the cormorant reference interval (<0.015 ng/mL) in all oil-exposed birds, indicating myocardiocyte damage.[46]

Hematologic Effects

Oxidative damage to erythrocytes, leading to Heinz body hemolytic anemia, has been reported in many avian species exposed to petrochemicals.[1,5] Even pelagic birds recovered from spill areas with no visible oiling are reported to have increased numbers of reticulocytes and Heinz bodies.[9,45] Heinz bodies were identified within 4 to 7 days of oral or dermal exposure, although erythrocyte damage can be evident already within 2 days following exposure.[1,5,32,47] However, avian erythrocyte morphology makes Heinz bodies difficult to identify by light microscopy and evaluation using electron microscopy is recommended (Figure 51.3).[47] The classic button-like, cytoplasmic projection that typically indicates Heinz body anemia in mammals does not occur in birds, even in blood samples from birds with biomarkers indicating severe oxidative damage. Therefore to use Heinz bodies as a biomarker for petroleum exposure in the field, a small amount of blood should be preserved in 2% to 3% buffered glutaraldehyde and immediately refrigerated (kept in a cool dark place). Electron microscopy specimens must be sent to a facility and technologist with experience in processing and examining blood samples. Contact a veterinary diagnostic laboratory for further information on avian cytology.

Epoxides directly interact with sulfhydryl groups on hemoglobin leading to hemoglobin precipitation in erythrocyte cytoplasm forming Heinz bodies.[47] Weakening of erythrocyte cell membranes secondary to oxidative damage, as well as the physiologic response to erythrocyte damage, culminates in hemolysis.[5] Reticulocytes (immature red blood cells) are released into circulation to compensate for erythrocyte loss, resulting in increased reticulocyte concentrations. However, this mechanism may not be sufficient to compensate for

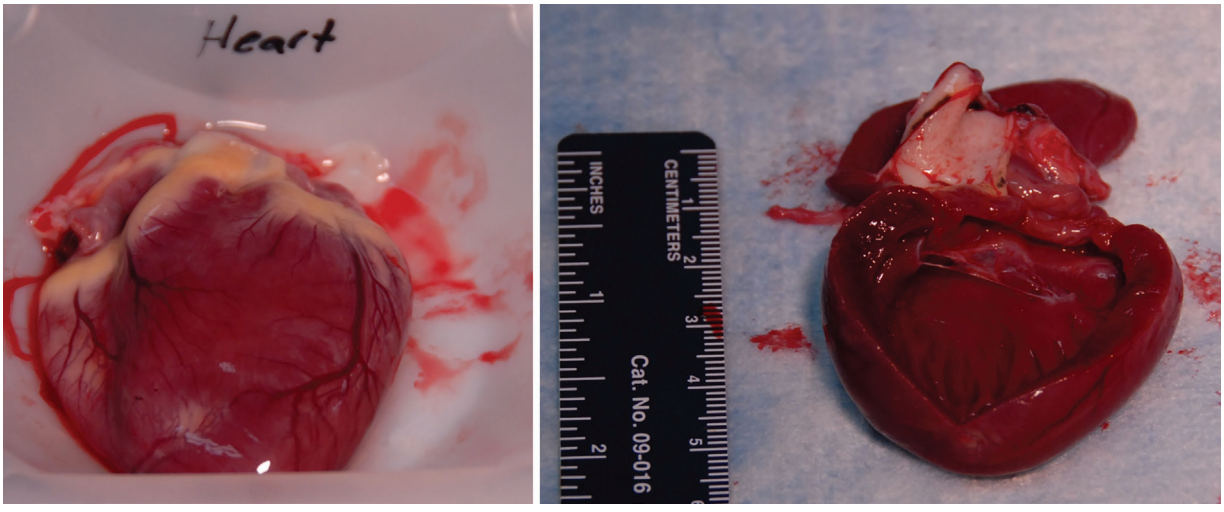


FIGURE 51.2 A flaccid heart from a cormorant (*Phalacrocorax* spp.), dermally exposed with <15% coverage of weathered MC252 crude oil over a 3-week time period. On the *left*, note the rounded shape of the whole heart that is softer rather than firm to the touch. On cut section on the *right*, thinner walls of the ventricle can be appreciated.

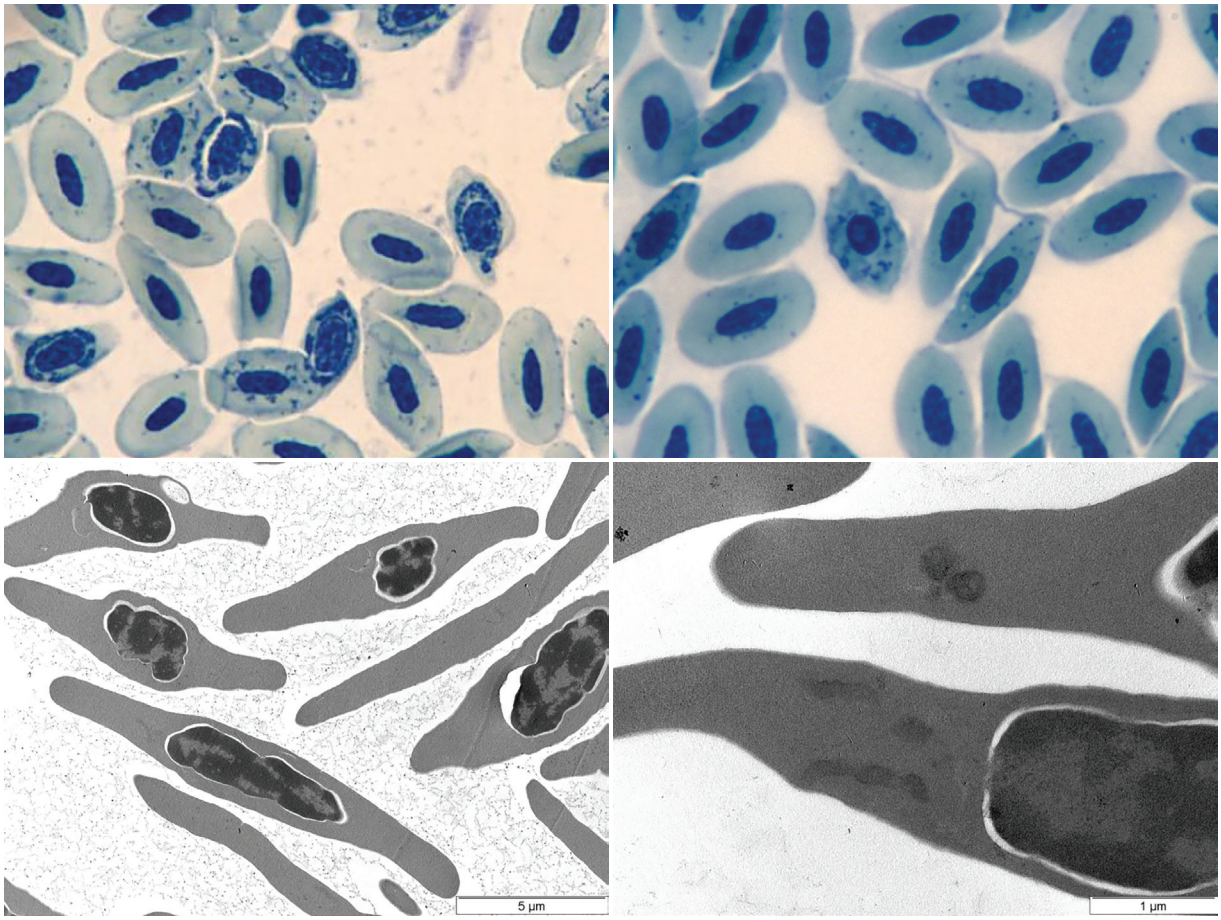


FIGURE 51.3 Reticulocytes versus Heinz bodies. The *top* images compare reticulocytes *(upper left)*, which are often found with dark blue reticulin ringing the nucleus in highly regenerative red blood cell (RBC) populations and an RBC with oxidant damage. The central crenated cell *(upper right)* has some reticulin, but fainter blue, round inclusions in the cytoplasm are consistent with Heinz bodies or denatured organelles. Light microscopy using new methylene blue stain at 1000×. Transmission electron micrograph of a normal-appearing RBC *(lower left)* from a control laughing gull exhibiting a homogeneous, gray cytoplasm with clumped chromatin in the nucleus. The white separation at the nuclear membrane and gray stippling in the background is processing artifact. A slightly higher magnification of electron-dense, darker cytoplasmic inclusions *(lower right)* found in >30% of RBCs of exposed birds only on transmission electron microscopy (TEM).

decreased oxygen-carrying capacity. While rapid acute hemolysis can cause lethal tissue hypoxia, even mild decreases in oxygen-carrying capacity impair organ function and can potentially reduce the ability of birds to fly, evade predators, and survive.[9]

Coagulopathy, suspected based on reduced blood clotting in cormorants both orally and dermally exposed to oil, was confirmed by determining activated clotting times in double-crested cormorants and pigeons that were dosed with artificially weathered MC252 crude oil. Coagulopathy results in blood loss through the GI tract (hematochezia) and other vascular leakage, which can directly cause anemia.[33,47] Often with extravascular hemorrhage (Figure 51.4), protein values will decrease.

Stress Response

Petroleum oil ingestion is reported to cause direct insult to the adrenal cortex.[9] It is hypothesized that the adrenal gland is particularly vulnerable to disruption due to its high vascularity and P450 enzyme concentrations.[48] Oiling directly and indirectly affects pituitary corticosteroid release. The immediate stress of oiling, capture, and washing likely increases endogenous corticosteroid release, leading to reduced immunocompetence and increased catabolism. After several weeks of exposure to sublethal doses of crude oil, experimental house sparrows *(Passer domesticus)* had less circulating corticosterone and cortisol.[48]

Immunologic Effects

Oil exposure increases inflammatory response in birds due to direct irritation but also causes immunosuppression from corticosteroid release.[9] Lymphopenia has been reported in oiled herring gulls and mallard ducks.[1] Cormorants in both experimental and control groups had decreases in heterophil and lymphocyte counts, suggesting that glucocorticoid release during captivity was a factor.[49] Cormorants administered approximately 8 mg/kg weathered MC252 oil had fewer



FIGURE 51.4 Coagulopathy in MC252 weathered crude oil–exposed birds was suspected at gross necropsy due to significant quantities of whole blood with no clots in the coelomic cavities.

inflammatory lesions on necropsy compared to control or low-dose birds, suggesting immunocompromise that could be the result of bone marrow suppression (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 11, Immunology).[44]

Hepatic Effects

PAHs are metabolized by cytochrome P450 enzymes (phase 1) to produce reactive epoxides, which cause oxidative stress.[9] Specifically, CYP1A enzyme activity, which is a biomarker for PAH exposure in birds and other animals, is upregulated by aryl hydrocarbon receptors (AhR).[50,11] CYP1A induction was increased 24-fold in cormorants dosed orally with artificially aged MC252 petroleum compared to control birds.[51] Other evidence of oxidative damage in experimentally dosed cormorants and laughing gulls included a linear dose-dependent increase in total hepatic glutathione concentrations.[45,51] Cormorants also had a dose-dependent linear decrease in hepatic superoxide dismutase and glutathione peroxidase activity.[51]

Hepatic failure in oil-exposed cormorants was evidenced by decreased plasma cholesterol, albumin, uric acid, and total protein plasma concentrations.[49] Hypoglycemia was present while alkaline phosphatase (ALP), alanine transaminase (ALT), aspartate aminotransferase (AST), γ-glutamyl transferase (GGT) activities, and bile acids concentration all increased in orally dosed birds; hypoglycemia and increased ALT activity also occurred in dermally exposed birds.[49] Livers from experimentally dosed laughing gulls, cormorants, and western sandpipers were grossly enlarged with increased normalized weights, but histologic lesions were not evident, suggesting that the change was due to upregulation of microsomal enzymes, edema, or undetected causes.[33,44,45] Hemosiderosis has been reported in oiled birds, but despite evidence of hemolysis and anemia, there was minimal evidence of hemosiderosis in experimental gulls and cormorants.[44,45] This finding is consistent with anemia due to hematochezia (blood loss in the GI tract).

Renal Effects

Increased plasma urea, uric acid, and phosphorus concentrations in cormorants exposed to oil dermally or orally indicated renal insufficiency.[49] Renomegaly, documented by normalized weights, was found in experimentally oil-exposed cormorants and laughing gulls in comparison to control birds.[44,45] Histologic renal lesions included renal tubular epithelial cell hypertrophy, lymphoplasmacytic interstitial nephritis, and multifocal mineralization.[44]

Musculoskeletal Effects

Muscle damage could be due to direct oxidative damage, excessive preening, or injury, and was evident in exposed cormorants as elevated plasma creatine kinase (CK) and AST activities.[49] Experimentally dosed laughing gulls had elevated plasma 3-methyl histidine, a marker of muscle wasting.[45] Oil-exposed homing pigeons had evidence of increased CK and AST activity indicating myocyte damage that worsened with the distance flown. These birds were only oiled on wing and tail tips, with controls similarly handled but painted with water instead of oil. Control birds did not exhibit CK or AST activity above the reference interval for the population. It is hypothesized that the muscular injury in the oil-exposed birds was indirect due to exertional myopathy resulting from oil-damaged flight feathers (K. Harr, personal communication, 2021).

Thyroid Effects

Thyroid function is essential for growth, reproduction, neurodevelopment, immune function, and metabolism.[52] A study of tree swallow *(Tachycineta bicolor)* nestlings in areas of oil sand mining found that thyroid activity was altered with exposure to sulfur dioxide, naphthalene, and fluorene compounds, affecting response to cold temperatures, growth, and survival outcomes.[52] Experimental cormorants with dermal exposure to artificially weathered DWH oil had thyroid follicular hyperplasia on histology.[44] Possible causes of thyroid gland upregulation include direct effects of oil exposure and, indirectly, feather damage as the thyroid is important both for increased metabolic rate in thermoregulation and for feather production.[44]

TREATMENT OF OILED BIRDS

Birds with >10% surface oiling are unlikely to survive without intervention.[40,53,54] Species vulnerability and expected response to treatment are important when making treatment decisions for oiled birds.[55] Survival rates for rehabilitated birds vary with species, type of oil, speed of response, and rehabilitation methods but can reach 98% in larger birds and penguins. Unfortunately, oil exposure and stress of captivity can cause mass mortality in other species, including common loons *(Gavia immer)* and murres. Return of rehabilitated birds to the reproductive population is variably reported.[42,43] Studies in little blue penguins, California brown pelicans *(Pelecanus occidentalis californicus)*, and other species have found decreased breeding success, and one study found reduced hatchability of eggs laid by oiled and rehabilitated penguins compared to control birds during the first breeding season after release.[41–43,56] Further viability studies to determine offspring fledge success rates are needed to assess the value of rehabilitation of oiled birds.[57] Release of birds likely to have

poor reproductive success into the ecosystem is expected to be detrimental to the overall population.

As with any other toxin exposure, general management includes stabilizing the patient, decontamination, and symptomatic and supportive care. Stabilization of oiled murres prior to washing took up to 9 days.[35] Mindful of minimizing stress in oiled birds, stabilization can include maintaining normal body temperature for the species, maintaining hydration and electrolyte balance using appropriate fluid therapy, and supplemental feeding to improve body condition in birds that have experienced weight loss.[58] Currently recognized best practices for cleaning oiled birds require successive washes with a solution of 1% to 2% Dawn dishwashing detergent (Procter and Gamble Corporation, Cincinnati, OH) and warm water (41°C, 105.8°F), followed by repeated rinsing to remove all oil and detergent residues to allow restoration of normal properties of feathers.[41,42,59] Cleaned birds should be given access to pools of clean water and time to allow proper preening and swimming behavior and restoration of the insulating and water-resistant properties of feathers, which can take anywhere from 2 to 129 days depending on the species.[35,42,43,59] It is important to consider the natural history of the species, pelagic versus freshwater, during this period. A study on rehabilitation of pelagic birds found that murres and northern fulmars *(Fulmarus glacialis)* housed with freshwater pools had declines in serum sodium and chloride concentrations, which could affect their survival.[58]

Experimental studies in murres found that structural changes to feathers caused by oiling did not improve significantly over 48 hours, and, therefore, improvement was considered unlikely without human intervention.[20] Interestingly, another study of murres affected by an oil spill off the coast of California determined that more heavily oiled birds were likely to have higher body masses and packed cell volumes than lightly oiled birds and therefore were more likely to survive to release.[35] The authors hypothesized that heavily oiled birds came to shore more quickly and were collected soon after oiling, reducing exhaustion and catabolism. Most of the murres that died during rehabilitation died soon after bathing or within 48 hours after arrival at the rehabilitation center.[35]

A study of western sandpipers found that lightly and moderately oiled birds with access to clean freshwater were able to recover from feather damage within 2 and 10 weeks, respectively.[30] A study of African penguins *(Spheniscus demersus)* found that 93% of oiled birds entering rehabilitation survived to release.[60] This success was, in part, attributed to their relative lack of stress around humans.[60] Factors that were found to decrease survival in oiled murres included low body weight (below 675 g, or 66% of the mean weight for wild murres), packed cell volumes $< 15\%$, total plasma protein < 0.8 g/dL, and body temperature < 36.1°C.[35,42,43,61]

SUMMARY

Crude oil is a complex mixture of hydrocarbons, which can be fractionated into different components with different properties. The composition of crude oil varies between different geographic sources and over time due to weathering. Therefore it can be expected that these complex cocktails of hydrocarbons, heavy metals, and aromatic compounds have a wide variety of toxic effects including, but not limited to, hypothermia, inhibition of flight and buoyancy, intestinal malabsorption, endocrine and immunologic disruptions, blood dyscrasias, cardiac damage, and multiple organ system damage. Addition of Corexit 9500, which has a significant component of volatile aromatic hydrocarbons, results in increased bioavailability of some toxic petroleum components and can result in increased biotoxicity. Studies on rehabilitated oiled birds have had variable results for survival and future reproductive competence, but the number of studies are few and often incomplete as no controls have been included. Hence, further studies are needed to properly evaluate long-term effects and prevent rehabilitation and release of nonreproductive birds into an already stressed ecosystem.

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CHAPTER 52

Anthropogenic Toxins

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INTRODUCTION

Humans have altered landscapes in remarkable ways that have improved our ability to survive and thrive as a species. Unfortunately, as we modify the world to our interests, we inevitably pollute the environment we rely on, and the consequences of these pollutants to wildlife continue to increase in parallel with our ever-expanding population. While, at times, anthropogenic pollutants can have obvious acute impacts on individuals and populations of wildlife (e.g., large-scale oil spills), more often the effects are more subtle and time-delayed. The effects of anthropogenic toxins on wildlife must certainly be evaluated at the level of individual animals, but ultimately it is more important to consider the effects at the level of populations, communities, ecosystems, and biospheres. Avian veterinarians, at the interface of human-animal-environmental health, must therefore consider the well-being of these broader communities in addition to their individual patients. Evaluating this bird's-eye view of anthropogenic toxins allows us to determine the most efficient and effective actions to prevent or remediate detrimental effects.

When approaching ecotoxicology, it is important to realize that toxicity testing under realistic environmental conditions is much more complex than testing the effects of a single chemical on a single species. The relationship of a particular toxin with a particular target species is complex and is influenced by various known and unknown environmental factors, seasonality, species and sex differences, as well as interactions with other compounds. Despite this complexity, there are numerous compounds and elements that have undeniable adverse effects. This chapter provides a summary of some well-studied anthropogenic toxins and their effects on wild birds. Rather than intending to provide clinicians with specific treatment guidelines for environmental toxin exposure, it provides an overview of the ecological consequences of anthropogenic toxins in the environment.[1] This ecotoxicological approach has importance for regulatory agencies, energy producers, agricultural industries, and individual consumers to help reduce the detrimental impact that our way of life has on wild bird populations. For further information on the treatment of intoxications, the reader is referred to *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 18, Advancements in Diagnosis and Management of Toxicologic Problems.

PESTICIDES

Pesticides are a group of chemicals used for the destruction of fungi (fungicides), bacteria (bactericides), weeds (herbicides), insects (insecticides), and rodents (rodenticides). These compounds are used on both residential and industrial scales around the world and are intended to target the physiology and survival of pest species. However, they can also have unintended consequences on nontarget species, including birds. Of these compounds, insecticides and rodenticides are the greatest risk to individuals and populations of wild birds.

It is important to realize that there is no centralized database that collects the information on avian mortality attributed to pesticides. These incidents can be difficult to detect, monitoring efforts are generally lacking, and relatively few carcasses are likely to be recovered. Furthermore, the sublethal, more protracted effects such as reduced fecundity, disturbance of behaviors, reduced prey availability, changes in migration patterns, and unrecognized effects of pesticides are rarely fully elucidated.

INSECTICIDES

Insecticides have been used for centuries to increase the productivity of crops and reduce the population of arthropod vectors of diseases that affect humans and domestic animals. However, there are numerous examples of damage to nontarget species, especially birds. These unintended consequences can be particularly problematic in part because of the staggering volume of pesticides produced and applied around the world, with the United States, European Union, China, and Brazil being the largest producers. Each year in the United States alone, 1 billion pounds (453,592,370 kg) of pesticides are used, while worldwide estimates suggest an annual use of approximately 5.6 billion pounds (2,540,117,272 kg).[2,3]

Insecticides are commonly grouped by their chemical makeup and the functional class of the active ingredient. Although there are thousands of compounds used as insecticides, those of greatest concern to bird populations include organophosphates, carbamates, and organochlorines (Table 52.1). There are hundreds of individual compounds that fall into one of these three categories.

One of the most well-known anthropogenic toxins to have a dramatic impact on bird populations was the organochlorine

TABLE 52.1
Classification of Insecticides With High Risk to Wild Bird Populations

Chemical Group	Mechanism of Action	Examples of Specific Chemicals
Organochlorines	Hyperexcitation of nerves by altering function of sodium channels and disturbing gamma-aminobutyric acid (GABA) neurotransmission	Dichlorodiphenyltrichloroethane (DDT), methoxychlor, dieldrin, chlordane, toxaphene, mirex, kepone, lindane, and benzene hexachloride
Organophosphates	Hyperstimulation of nicotinic and muscarinic receptors through inhibition of acetylcholinesterase	Monocrotophos, chlorpyrifos, dimefox, parathion, fenitrothion, fenthion, coumaphos, dichlorvos, demetox, malathion, trichlorfon
Carbamates	Hyperstimulation of nicotinic and muscarinic receptors through inhibition of acetylcholinesterase	Carbofuran, carbaryl, dimethane, pyrolan, aminocarb, aldicarb, vernolate, pebulate, butylate, methane, ferban, amoban, naban, zineb

pesticide dichlorodiphenyltrichloroethane (DDT). Organochlorines, which exert their effect via hyperexcitation of nerves by altering the function of sodium channels or by disturbing gamma-aminobutyric acid (GABA) neurotransmission, are highly lipid soluble and resistant to degradation, making this class very persistent in the environment with rapid bioaccumulation.[4] DDT was used extensively in North America during the 1940s, 1950s, and 1960s, both as an agricultural and household pesticide. The insecticide caused direct mortality in many birds. However, the more insidious effect of the compound was to reduce the transport of calcium from the bloodstream into developing eggshells, leading to eggshell thinning. The combination of widespread use, rapid bioaccumulation, and mechanism of action caused drastic declines in populations of brown pelican *(Pelecanus occidentalis)*, osprey *(Pandion haliaetus)*, peregrine falcon *(Falco peregrinus)*, and bald eagles (*Haliaeetus leucocephalus*; Figure 52.1).

The documented negative environmental effects of DDT led to most developed countries banning its agricultural use in the 1970s and 1980s, and the 2004 Stockholm Convention on Persistent Organic Pollutants put a global restriction of its use to vector control only. Despite these restrictions, DDT continues to be used for agricultural purposes in India and North Korea, and it is used for vector control in many countries in Asia, South America, and Africa to reduce the risk of vector-borne diseases such as malaria and leishmaniasis, in humans.[5] Therefore DDT still poses a risk to wild birds residing in or migrating to these countries, making hemisphere-wide conservation initiatives an important component of wildlife monitoring and management. Furthermore, because of the persistence of DDT and its breakdown products (dichlorodiphenyldichloroethylene [DDE] and dichlorodiphenyldichloroethane [DDD]), which have similar chemical and physical properties in soils and biota, the risk of exposure persists for many decades. Finally, while the use of DDT was restricted, this did not extend to all organochlorine pesticides. For example, compounds such as the miticide dicofol are still available for general use around the world; hence, their detrimental effects continue to be a concern.



FIGURE 52.1 The organochlorine pesticide dichlorodiphenyltrichloroethane (DDT), used extensively in North America during the mid-20th century, lead to declines in populations of predatory birds including the bald eagle *(Haliaeetus leucocephalus)*. (Photo courtesy Derek Courtney.)

As restrictions became more stringent on organochlorines, organophosphate and carbamate insecticides became much more commonly used. Both chemical types inhibit acetylcholinesterase, which results in hyperstimulation of nicotinic and muscarinic receptors, leading to both parasympathetic and sympathetic stimulation of the autonomic nervous system as well as central nervous system abnormalities in individuals exposed to toxic doses. Because insects and vertebrates have similar mechanisms of nerve transmission, toxicity can have lethal effects in target insects, as well as in fish, reptiles, amphibians, mammals, and birds. Birds are more sensitive to acute exposure to anticholinesterase pesticides than mammals due to more rapid binding and reduced detoxifying enzymes.[6,7]

Organophosphates are a readily available class of insecticides used extensively for agriculture, mosquito control, and residential purposes. Like organochlorines, these compounds can be toxic to vertebrates and, to a lesser extent, can bioaccumulate.[8] There are many well-documented reports of direct mortality in birds exposed to these compounds. For example, since its introduction in 1964, the organophosphate monocrotophos has directly been linked to more than 100,000 bird deaths, either through ingestion of seeds sprayed or ingestion of insects coated with the compound. Some of these mortality events can be dramatic, including the confirmed direct mortality of 5,000 Swainson's hawk *(Buteo swainsoni)* in Argentina over the course of one farming season.[9]

Some organophosphates, including monocrotophos, have been recently banned in many countries including the United States, European Union, and China. However, there are dozens of organophosphates still in heavy use, even in countries with restrictions, and some of the most dangerous are still being used around the world and continue to cause mortality in birds. Organophosphates were confirmed by the United States National Wildlife Health Center to be the cause of 335 separate mortality events, killing more than 9,000 birds in the country between 1980 and 2000.[10]

Carbamates are very similar in mechanism to organophosphates and have also been documented to cause mortality in many different species of birds. The most well-known example is carbofuran, which was estimated by the United States Environmental Protection Agency (EPA) to kill between 1 and 2 million birds annually prior to restricting the use of the granular formulation in 1994. Similarly, more than 5,000 ducks and geese were killed in five separate incidents when carbofuran was applied to alfalfa fields.[11] Sublethal effects, including reduced egg production, thinning of eggshells, and behavioral abnormalities, are triggered through exposure to carbamates, although these effects are difficult to link to pesticide exposure and quantify in wild populations.[11]

The consequences of organochlorines, organophosphates, and carbamates to individual and populations of birds cannot be understated. Fortunately, regulations have helped to ease the burden in recent years, but the use and misuse of these and other insecticides (e.g., neonicotinoids) must continually be monitored. As populations of many bird species continue to decline, we must do all we can to limit the use of these compounds whenever possible.

RODENTICIDES

Like insecticides, there are numerous compounds that are used as rodenticides. However, the most commonly used products, and therefore the greatest potential threat to wild birds, contain either anticoagulants, metal phosphides, mitochondrial toxins, or strychnine (Table 52.2). None of these compounds are species specific, and all of them have been documented to cause nontarget species toxicosis and death.

Anticoagulant rodenticides inhibit the enzyme vitamin K epoxide reductase and thus the synthesis of vitamin K, a crucial component in a number of normal clotting factors. Exposure to these compounds results in spontaneous bleeding throughout the body, leading to anemia and hypovolemia. These rodenticides are commonly categorized into first generation (e.g., warfarin), which typically require multiple exposure episodes to cause toxicosis, and second generation (e.g., brodifacoum), which are designed to cause toxicosis after a single exposure event. Of all the rodenticides, second-generation anticoagulants currently pose the greatest threat to individual birds and populations.

There are several factors that make second-generation anticoagulant rodenticides a threat to wild birds. First, they cause mortality from primary ingestion at very low doses. Second, they have historically been readily available to the general public around the world. Finally, and most importantly, they bear a high risk of secondary toxicosis (also called relay toxicosis) because of their persistence in tissues, which leads to bioaccumulation and biomagnification. Raptors, in particular, are at risk for exposure to these compounds because of their diet, and some species (e.g., owls) appear to have limited capability to metabolize these anticoagulants.[12] This results in longer elimination times compared to mammals and other birds, hence limiting their capacity for detoxification and increasing the risk for detrimental effects.

There are many reports of mortality in raptors exposed to second-generation anticoagulants, and numerous studies show widespread sublethal effects, with some demonstrating nearly ubiquitous exposure in certain regions.[13] Through opportunistic sampling of raptors, a high incidence of exposure to these rodenticides has been demonstrated in North America, Europe, Africa, Australia, and Asia. Because of the high risk of secondary toxicosis of second-generation anticoagulants, some countries, including the United States, have recently limited the availability of these compounds for use only by professional exterminators. Hopefully, this will reduce the risk of exposure of birds of prey to compounds like brodifacoum. However, an unintended consequence of these regulations is likely to be a higher incidence of exposure to other rodenticides such as zinc oxide, cholecalciferol, and bromethalin, as these products remain readily available to the general public.

The rodenticide bromethalin is a neurotoxin intended as a single-dose product. This chemical, a strong uncoupler of oxidative phosphorylation, results in nerve demyelination, spinal and cerebral edema, increased intracranial pressure, and ultimately neurologic dysfunction and death. Compared to

TABLE 52.2

Classification of Rodenticides With Risk to Wild Bird Populations

Chemical Group	Mechanism of Action	Examples of Specific Chemicals
Anticoagulants	Inhibition of the enzyme vitamin K epoxide reductase leading to spontaneous bleeding	First generation: chlorophacinone, diphacinone and warfarin Second generation: brodifacoum, bromadiolone, difenacoum, and difethialone
Bromethalin	Uncoupler of oxidative phosphorylation and neurotoxin resulting in nerve demyelination, spinal and cerebral edema, causing neurologic dysfunction	Bromethalin
Metal phosphides	Generates phosphine gas in the acidic environment of the stomach resulting in respiratory damage	Zinc phosphide
Strychnine	Inhibits the neurotransmitter glycine resulting in rigidity, seizures, and neurologic damage	Strychnine

anticoagulant rodenticide toxicosis, bromethalin toxicosis is difficult to recognize both ante- and postmortem, as the primary signs can mimic other neurotoxins (e.g., lead), infectious encephalitis (e.g., West Nile virus), or trauma. Although bromethalin toxicosis is commonly reported in domestic dogs and cats from primary exposure, there is very little data on the risk of bromethalin to wild birds, although the LD_{50} of bromethalin in birds is likely similar to that of mice.[14] Additionally, there is recent evidence of bromethalin toxicosis in free-ranging conures in an urban setting, although the source of bromethalin in these cases was not determined.[15] To date, secondary toxicosis from the ingestion of prey has not been proven. Although it is believed that this compound does not pose a high risk of relay toxicosis, there is a paucity of data on the effect of bromethalin in birds that feed on target rodent species. As increased restrictions on the use of second-generation anticoagulants come into effect, this compound will continue to become more commonly used (Figure 52.2). For these reasons, more research is needed to determine the risk of bromethalin on wildlife.

Zinc phosphide serves as rodenticide by virtue of its transition to highly toxic phosphine gas once the bait has encountered the acidic environment of the stomach. This gas causes severe irritation to the lungs and air sacs, which results in respiratory distress and death. Primary ingestion of zinc phosphide rather than secondary toxicosis is the highest risk to birds, with numerous studies indicating lethal effects to Anseriformes, Icteridae, and Galliformes allowed access to zinc phosphide pellets in experimental settings or were naturally exposed to the compound. Secondary exposure to zinc phosphide has been demonstrated to produce sublethal effects in great horned owls *(Bubo virginianus)* and bald eagles, although the risk of relay toxicosis leading to death appears to be low.[16,17]



FIGURE 52.2 Rodenticides are readily available to consumers throughout the world. In many countries, bromethalin has recently become more accessible than second-generation anticoagulant rodenticides in an attempt to reduce the risk of relay toxicosis in wildlife.

Strychnine is an indole alkaloid that, when ingested, inhibits the neurotransmitter glycine, resulting in rigidity, seizures, anoxia, and death. There are numerous reports of mortality events in dozens of bird species. Because strychnine is used around the world to target large scavenging mammal species such as coyotes and feral dogs, scavenging raptors are at particular risk. In Africa, large-scale, nontarget poisoning of vultures (e.g., bearded vulture *[Gypaetus barbatus]*, hooded vulture *[Necrosyrtes monachus]*, Cape vulture *[Gyps coprotheres]*, white-backed vulture *[G. africanus]*, lappet-faced or Nubian vulture *[Torgos tracheliotus]*, and white-headed vulture *[Trigonoceps occipitalis]*) and eagles (e.g., tawny eagle *[Aquila rapax]* and bateleur *[Terathopius ecaudatus]*) has been reported.[18] With more than half of old world vultures listed as vulnerable, threatened, or endangered, such large-scale losses can have devastating effects on populations.

Although birds typically succumb to insecticide and rodenticide toxicosis as a result of unintentional exposure, they are sometimes the target species of malicious poisoning. Hundreds of cases of malicious poisoning of raptors with these compounds are reported each year around the world.[19–21] These are preventable events. Public education about the value of scavengers in ecosystems, regulations on the availability of these products, and enforcement of existing laws protecting wild birds are paramount to reducing the incidence of this intentional and inappropriate use of pesticides.

As with insecticides, rodenticides can have enormous impact on individuals and populations of birds. Reducing the risk includes selecting compounds with low risk of relay toxicosis, limiting the access to these compounds by the general public, and selective and judicious use of rodenticides whenever possible.

HEAVY METALS

Heavy metals, so named because of their high molecular weight, are well known environmental pollutants. Although these metals are found naturally arising from weathering of certain rocks and volcanic eruptions, anthropogenic sources such as mining, smelting, agricultural use of pesticides and fertilizers, and burning fossil fuels have dramatically increased the presence of these metals in the soil, water, and air. In particular, mercury, lead, cadmium, and arsenic have been identified as metals of greatest concern to wildlife. These compounds can undergo bioaccumulation and biomagnification, putting long-lived and high trophic species at highest risk.

In experimental settings, each of these elements has been demonstrated to individually cause physiologic insult in a variety of bird species. However, because many of these compounds often occur in elevated environmental concentrations as a result of the same anthropogenic activities, it is often difficult in field studies to determine which of the compounds has the greatest impact on individuals and populations of birds. The primary exceptions to this are lead, mercury, arsenic, and, to a lesser extent, cadmium.

Lead

Although it is a naturally occurring element in the environment, lead has no functional role in biological systems and is toxic at very low levels of exposure. In birds, acute lead toxicosis causes lethargy, gastrointestinal stasis, anorexia, vomiting, diarrhea, anemia, disturbances of cellular metabolic functions, and neurologic injury leading to blindness, seizures, weakness, and death.[22–25] Chronic, low-level exposure leads to sublethal effects such as neurological damage, tissue and organ damage, and reproductive impairment.[26]

Lead has been used for centuries by humans for a variety of purposes, including plumbing, batteries, weights, solders, fusible alloys, radiation shielding, paint, and leaded gasoline. Fortunately, in many countries, lead has been banned for use in water pipes, paint, and gasoline, reducing the human risk of lead intoxication. However, despite the plethora of literature that identifies lead as a potent toxin to all vertebrates, it is still used extensively for hunting ammunition and fishing tackle throughout the world. Exposure to these sources of lead has been a known hazard to water birds (e.g., ducks, swans, and loons) for decades.[27] Upland game birds (e.g., doves and quail) and scavengers (e.g., condors and eagles) are exposed to lead from spent ammunition, with population-level consequences identified in some species.[28–31] Scavenging raptors are at particular risk, with up to 25% of bald eagles admitted to rehabilitation facilities carrying elevated lead concentrations.[32] The federally endangered California condor (*Gymnogyps californianus*; Figure 52.3) population continues to be at great risk in large part due to exposure to lead from spent ammunition found in offal piles and carcasses of shot game and pest species (see also *Current Therapy in Avian Medicine and Therapy*, Vol 1, Chapter 23, Conservation of Avian Species).[27,33]

Current data for raptors and avian scavengers demonstrate clear positive correlations of lead exposure during hunting seasons.[34] The hazard of ingested lead sinkers and fishing tackle is well documented in swans (*Cygnus* spp.) and loons (*Gavia* spp.), and restrictions on the sale or use of lead weights have been instituted in parts of the UK, Canada, the United States, and other countries in order to reduce the impact on these and other vulnerable species. Fortunately, these regulations on recreational hunting and fishing have been shown to reduce the risk of lead toxicosis, with particularly dramatic effect on waterfowl in North America and the European Union, where rates of lead shot ingestion have been reported to be reduced by up to 50% to 64% in some populations.[35,36] However, the worldwide use of lead for hunting and fishing continues to have a significant impact on wild bird populations.



FIGURE 52.3 Lead intoxication is a common cause of mortality in critically endangered California condors *(Gymnogyps californianus)*. There are fewer than 300 California condors in the wild; scientists use numbered wing tags to identify individual birds. (© Courtesy Evan McWreath mcwreath@accawv.org)

Mercury

Inorganic mercury is a toxic, naturally occurring element that has been used in a variety of applications but enters the environment predominately through the burning of fossil fuels, particularly coal, causing mercury to become airborne. This airborne mercury can travel over a range of distances, from a few feet to thousands of miles, before it falls to the ground in rain, snow, or simply by gravity, ultimately entering the soil or aquatic ecosystems. Once in water or soil, mercury enters a complex cycle, which includes bioconversion by bacteria into more soluble forms including methylmercury. The conversion to methylmercury is important both because it is more toxic than inorganic mercury and because organisms require considerably longer to eliminate methylmercury. Bacteria that produce methylmercury are either consumed by the next higher level in the food chain or the bacteria may release the methylmercury into the water where it can quickly adsorb to plankton. Bioaccumulation through the food web continues through macroinvertebrates, to herbivorous fish, to piscivorous fish, and finally to fish-eating birds and mammals, leading to increasing concentrations of methylmercury through each trophic level.

There is a large body of research demonstrating both elevated concentrations of and negative effects from mercury in both fish-eating birds and some insect-eating songbirds.[37] The accumulation of mercury in free-ranging birds is rarely lethal, but songbirds and waterfowl with blood concentrations above 5 μg/g have demonstrated reduced reproductive output, compromised immune function, and avoidance of high-energy behaviors.[37] Reduced reproductive output in particular has been demonstrated to have significant impact on local populations. For example, in the Adirondack Mountains of New York state, common loons *(Gavia immer)* with elevated blood mercury concentrations had a 32% to 56% reduction in the number of chicks fledged per year.[38]

Arsenic

Arsenic, often classified as a heavy metal, is a metalloid, having properties of both a metal and a nonmetal. It occurs naturally in soil and in several types of rock, but anthropogenic sources

including mining-related activities, coal combustion, pesticides, and wood-preserving arsenicals greatly increase the regional environmental burden.[20] Arsenic causes toxicosis primarily by inducing oxidative stress through reduction in the activity of antioxidant enzymes and production of reactive oxygen species, leading to damage to cells, tissues, and genotoxicosis.[39] The effects on birds are typically sublethal and include reduced food intake, weight loss, growth retardation of chicks, reduced reproductive success, and hepatic dysfunction.[40] Because elevated arsenic concentrations in blood and tissues occur in conjunction with elevations in other heavy metals as a direct result of the same anthropogenic sources, identifying arsenic as the definitive cause of physiologic injury in field studies is often impossible. Experimental studies, however, have shown that arsenic exposure reduces growth rates and survival in wild birds.[41]

Cadmium

Cadmium is a known carcinogen, mutagen, and teratogen that has been demonstrated to cause severe deleterious effects on freshwater invertebrates and fish. However, the effect of environmental exposure to birds is less clear, with relatively little data available compared to body of research on lead and mercury. Anthropogenic sources of cadmium include smelter fumes, burning of fossil fuels, fertilizers, and municipal wastewater, with the highest concentrations in localized regions of smelters or urban industrialized areas. Experimental studies in captive birds have demonstrated growth retardation, anemia, kidney insult, and testicular damage; however, these effects were observed at higher concentrations than in aquatic biota. Elevated cadmium concentrations have been found in the kidneys of a number of species ranging from raptors to songbirds. Additionally, renal tubule damage has been correlated to elevated cadmium concentrations in wild pelagic seabirds (e.g., Manx shearwaters *[Puffinus puffinus]*, fulmars *[Fulmarus glacialis]*, and puffins *[Fratercula arctica]*) and white-tailed ptarmigan *(Lagopus leucura)*.[42,43] Further research is needed to evaluate the population-level effects of cadmium.

Except for lead, reducing the burden of elevated heavy metal concentrations in the environment depends on decreasing human reliance on fossil fuels and careful use of fertilizers. These changes can occur at the local, regional, national, and international levels through regulation, environmentally conscious energy companies, and public demand. Individuals can reduce their contribution to heavy metal contamination by walking, biking, or taking public transportation to work or telecommuting, choosing reusable items rather than disposable, and by conserving energy in their homes and workplaces.

SUPERFUND SITES

Although anthropogenic toxins are often distributed in relatively small quantities across large regions, there are numerous examples of massive contamination in very consolidated locations. In the United States, these localized areas with massive contamination requiring a long-term response to clean up hazardous materials are often referred to as Superfund sites. The designation of Superfund sites began following the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, which authorizes



FIGURE 52.4 Environmental Protection Agency (EPA) Superfund Signage at the Clearview Landfill with covered landfill in background. There are more than 1,300 locations in the United States identified by the EPA as Superfund sites. These sites have soil and water that are heavily contaminated with volatile organic compounds, polychlorinated biphenyls, polyaromatic hydrocarbons, and heavy metals. (Photo taken January 2021, by Dwkaminski, Wikimedia Commons.)

the EPA to identify the most contaminated sites posing a threat to public and environmental health and to outline a strategy for remediation (EPA 2020). There are at least 40,000 Superfund sites distributed throughout the United States, with more than 1000 identified as severe enough to be eligible for aggressive EPA remediation efforts (Figure 52.4). Many other countries have similar programs, as localized, heavily contaminated sites are present around the world.

Superfund sites arise from a variety of sources including mines (e.g., copper, asbestos, uranium), smelters (e.g., lead), weapons manufacturing and testing facilities, waste from chemical companies, water, and soil nearby a variety of industrial and agricultural sources, and landfills where hazardous chemicals and waste were deliberately and inappropriately dumped (Figure 52.5). The list of contaminants found at these sites is long and includes metals such as lead, mercury, zinc, and arsenic, pesticides such as DDE and DDT, volatile organic compounds (e.g., benzene), polycyclic aromatic hydrocarbons (PAH), polychlorinated biphenyls (PCBs), and radioactive compounds (e.g., uranium 234, 235).

Myriad negative effects on birds and other wildlife have been documented near these heavily contaminated sites. Specifically, elevated concentrations of contaminants in wild birds near these sites have been correlated with organ dysfunction, endocrine disruption with gonadal disturbance and skewed male-female ratios, population age-structure aberrations, reduced growth rate, poor fledgling success, and reduced adult survival in dozens of species from many different families of birds.[44]

Overall, there is a great deal of public support for remediation of these heavily contaminated sites. Once remediation of a site is initiated, it can take decades and millions of dollars to restore sites to "safe" environmental states. Whenever possible, responsible parties are forced to bear the cost of cleanup. However, in many cases, there is no clearly identifiable responsible



FIGURE 52.5 The "Valley of the Drums" in Bullitt County, KY, was an uncontrolled industrial waste dump that helped prompt the passage of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, which allocated federal funding for Superfund site cleanup. Heggenhougen HK: International encyclopedia of public health. In De Rosa CT, Fay M, Keith LS, et al., editors: *Hazardous wastes*, 2008, Academic Press, pp 107–121. https://doi.org/10.1016/B978-012373960-5.00279-3.

party, or the pollution was caused by bankrupt parties. In these cases, tax money is required to support expensive cleanup efforts, and the extent of remediation depends on the appropriation of funds to the EPA from the federal government for this specific purpose.

SUMMARY

Despite their effects on birds and other wildlife, anthropogenic toxins are present throughout the environment. In some cases, remediation of localized toxins can be successful in restoring the environment to conditions that are safe to birds. Most importantly, these toxins need to be recognized and regulated by individual countries, and international cooperation for the safe and judicious use of pesticides and waste chemicals is critical to the ongoing health of our planet.

There are straightforward strategies that we can employ to reduce the negative impact of our daily lives on ecosystems. Specifically, responsible natural resource extraction, processing, and transport, proper disposal of dangerous chemicals, and reduced use of chemical pesticides are achievable goals. Good stewardship of our environment benefits not only the birds and wildlife that we cherish, but also keeps this planet a healthier place for all plants and animals, including humans. Fortunately, there are several actions that individuals can take to help reduce the risk of anthropogenic toxins to birds:

1. Encourage legislative bodies to reduce the ongoing use and volume of anthropogenic toxins in the environment. This includes restrictions on the use of lead ammunition, reducing the availability and use of pesticides (particularly carbamates and organophosphates) and rodenticides, to targeted, critical applications, demanding responsible extraction, transport, and refinement of natural resources, and requiring remediation of Superfund sites. This must be approached at the local, national, and international levels.
2. Reduce our individual contribution to these environmental toxins by choosing less toxic ammunition and fishing tackle, judicious personal use of pesticides and rodenticides, purchasing products from environmentally conscious companies, and reducing our reliance on fossil fuels.
3. Educate friends, families, clients, students, and the general public on the risks of anthropogenic toxins and alternative choices that can help improve the health of ecosystems.

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CHAPTER 53

Wildfires

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INTRODUCTION

A wildfire or bushfire by definition is an uncontrolled fire. While wildfires historically occurred in wild, unpopulated areas, they can occur anywhere and can harm homes, agriculture, humans and the animals in their path, including birds. In recent years, wildfires in North America, Australia, and Europe have gotten progressively larger causing extensive damage to life and property. Whole towns have been reduced to ash as wildfires increase in size and intensity.

Regardless of country, disaster incident response management systems are vital to have in place to ensure an organized response when any disaster incident occurs, including wildfires. In the United States, for example, the Incident Command System (ICS) manages wildfire incidents of any size and provides procedures for controlling personnel, facilities, equipment, and communications for any type of incident (wildland fire, search and rescue, structural fire). It is intended to be staffed and operated by qualified personnel from emergency services agencies; ICS may involve personnel from a variety of agencies. The National Park Service and the US Department of Agriculture (USDA) Forest Service are often first responders to these incidents and along with battalion chiefs from the local or regional fire department may become the Unified Command for the incident.

Local and regional disaster groups may be activated to assist in sheltering and evacuation of animals during a wildfire and are commonly part of the Unified Command of the ICS. Veterinarians activated during a wildfire incident should be prepared to treat a wide range of patients, sedate/anesthetize different species in the field, treat various degrees of burns, bandage wounds, and fractures, provide antibiotics and other medications as needed (and within the guidelines of the Food Animal Residue Avoidance Database [FARAD] for food-producing animals or wildlife that may be utilized as food in the United States), and provide supportive care. Wildlife is generally transferred to a local wildlife rehabilitation facility and so veterinarians working in this capacity may be called upon to provide immediate care for injured and burned wildlife.[1]

Best practices for common injuries identified during a wildfire have been established for many species but to date these have not been developed for birds. Not only are companion psittacines and passerines rescued from fires but backyard poultry, waterfowl, and wildlife are nowadays also rescued from wildfires in meaningful numbers. Disaster groups are realizing that protocols for housing and medical care still need to be established for all bird groups to have a unified response.

Disasters can quickly overwhelm health care systems. A database for tracking identification and health of animals is a critical component of effective preparation, real-time communication during disasters, and retrospective analysis.[2] The database should provide information that supports additional efforts in the recruitment of volunteers with specific skill sets (e.g., avian veterinary specialists) and to adjust daily supplies needed. A disaster database is not intended to replace individual veterinary medical records but to provide an overarching view to assess the daily veterinary needs and supplies, to help manage volunteer deployment, and to establish an objective surveillance tool for evaluation.[2] The database had specific goals of assessing the types and numbers of animals affected by the disaster, recording the precise spectrum of illness and injuries, monitoring treatment and disposition, acquiring needed veterinary supplies, and facilitating future evaluation of the response.

In November of 2018, California experienced its most destructive wildfire in history, the "Camp Fire". The fire burned over 18 days and 150,000 acres and destroyed 18,804 structures in Butte County, CA.[2] The town of Paradise was the epicenter of the inferno. The fire raged through residential and rural neighborhoods and incinerated homes and entire communities. Four days after the fire began, it was classified as a major disaster by the federal government, the worst of 16 fire disasters declared by the Federal Emergency Management Agency (FEMA) in California in 2018 alone. Over 600 backyard chickens and waterfowl were rescued—a first in California.[2] Avian species were primarily treated for burns and dehydration; ectoparasites and lameness were also commonly documented.[2] Debriefing from this incident led to the awareness that development of best practices for housing and medical care for backyard poultry and waterfowl were especially necessary for fire disaster preparedness.[2] Birds are treated at their "shelter in place" facility as much as possible without moving them, so family members and disaster workers can find and identify the animals more easily. If the severity of wounds requires more aggressive therapy and continuity of care, the birds should be transferred to a disaster veterinary facility for further care.

Wildfires are predicted to continue in the western United States and in other parts of the world into the future.

Towns in Oregon and Washington states in the United States were also devastated by wildfires in 2019 to 2020,[3] and wildfires in Australia and Brazil in those years showed not only loss of life and home but also the direct effects on endangered species and other wildlife.[4] In northern California alone in 2020 a veterinary emergency response team evaluated 315 birds in the field while "sheltering in place" and another 630 birds were brought into shelters during evacuations and from search and rescue (M.G. Hawkins, personal communication, 2024). It is therefore important to be familiar with emergency housing, patient assessment, and treatment of burns and smoke inhalation in avian species.

EMERGENCY HOUSING

Not only are companion psittacines and passerines rescued from fires, but backyard poultry and waterfowl, falconry birds, ratites, and wild birds have also been rescued. Disaster groups are realizing that protocols for housing and medical care for these various species still need to be established to have a unified response. During the 2018 Campfire in California, almost 600 backyard companion birds were rescued in addition to their companion farm animal counterparts (Figure 53.1).[2] Debriefing from this incident has led to the development of best practices for housing, biosecurity, and medical care for backyard poultry and waterfowl by the state, but there is much more to be done to develop specific protocols for the many varied avian species that have already been presented to shelters.

PATIENT ASSESSMENT

With the paucity of literature to help guide veterinarians and animal caretakers, standards of care must be derived from the human literature. Avian patients on presentation should be evaluated like other trauma-based incidents with a full physical examination including assessment for shock, dehydration, and respiratory distress (see Chapter 34). Regardless of the presence of burn wounds, it is essential to treat the patient for life-threatening systemic conditions such as shock and dehydration prior to treatment of burn wounds (Figure 53.2).[5,6] In a recent publication evaluating the occurrence of comorbidities in burned wildlife, hypovolemia and dehydration were the most common problems noted on presentation.[1] This is comparable to what is typically noted in the human literature and associated with the secondary systemic inflammation from the burn wounds creating an increase in general capillary permeability.[7] In addition, animals are often not found until several days or weeks after their initial injury and access to water is not always readily available in evacuated zones. If hypovolemic shock is noted, these animals should be treated aggressively with isotonic intravenous (IV) fluid therapy until improvement of clinical signs. At a minimum, subcutaneous fluid therapy is recommended to provide additional volume and ensure adequate hydration support. When feasible, it is ideal to collect blood for a point-of-care blood gas and chemistry panel to assess initial signs of respiratory and renal compromise.

Cardiopulmonary evaluation is essential to monitor secondary injuries from smoke inhalation. Smoke inhalation injury has been characterized by thermal and chemical damage to the upper airway, lower airway, and pulmonary parenchyma, as well as systemic toxicity.[8] Over one-third of all human burn patients suffer from smoke inhalation injury, and mortality increases 20 times when this injury is present.[9] The exact occurrence in birds is unknown, but birds can exhibit marked tachypnea and dyspnea within a few hours of smoke exposure.[10] However, in recent fires in California, few birds exhibited overt clinical signs of respiratory distress even when severe burns were present. Diagnostics in human medicine include radiographs, bronchoscopy, and computed tomography (CT), but the main diagnosis is based on history of exposure and presence of clinical signs.[8,11] Blood gas markers such as carboxyhemoglobin and methemoglobin may more objectively help in identifying the severity of respiratory compromise.[12–14] Further studies are necessary to determine how these markers may be useful prognostically in detecting smoke inhalation injury in birds.



FIGURE 53.1 (A) During the 2018 Camp wildfire in California, almost 600 backyard birds along with livestock were rescued to a nearby fairground. Protocols for avian housing and biosecurity were not yet established. Initial housing for backyard chickens, turkeys, and waterfowl used dog carriers and species were not separated. (B) Small flocks were housed in livestock stalls, often next to pigs, creating additional biosecurity issues.



FIGURE 53.2 Regardless of the presence of burn wounds, it is essential to treat the patient for life-threatening systemic conditions such as shock and dehydration prior to treatment for burns.

THERAPEUTICS

There is currently no standard consensus on the treatment of smoke inhalation injury in human medicine and even less is known in avian medicine. A variety of therapies such as oxygen support and nebulization of bronchodilators, anticoagulants, antioxidants, and pain management are used in humans.[11,15,16] Beta-2 adrenergic agonists have been used to help decrease airway resistance, improve airway fluid clearance, decrease mucosal damage, and decrease inflammatory mediators within the airways (see also Chapter 28).[11,15] Nebulization of a mucolytic agent such as *N*-acetylcysteine (NAC) has also been evaluated and shown to be helpful in facilitating removal of excess secretions by breaking down disulfide bonds. NAC also has additional beneficial anti-inflammatory and antioxidant effects. The potential side effect of nebulization of NAC is bronchospasm, and therefore nebulization in conjunction with a beta-2 adrenergic agonist is recommended.[11,17,18] The use of nebulized heparin in human clinical trials has also shown improvement and may aid in the breakdown of fibrin binding in the airways.[9,19,20] At this time, none of these treatments have been assessed in avian species, so further evaluation is warranted. Oxygen supplementation with nebulization of saline and albuterol should at a minimum be considered. Despite its use for other respiratory conditions such as asthma, corticosteroid administration is not recommended for smoke inhalation injury.[16] It may be considered for treatment of initial airway edema but should always be used with caution in birds.[10,15] Human studies have shown no significant benefit; instead corticosteroids carry an increased risk for secondary lung infection.[8,16] This has also been noted in a case report on parrots with smoke inhalation that developed secondary aspergillosis after prolonged corticosteroid administration.[10] The use of diuretics has been described in the veterinary literature but has minimal supporting evidence and may exacerbate concurrent dehydration or hypovolemia.[10,21]

A pain management plan should always be developed early in the treatment period with the knowledge that the plan may change over the course of therapy (see also Chapter 35 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 20, Recognition, Assessment, and Management of Pain in Birds). Species variability occurs due to differences in pain sensitivity, the conscious response to pain, and the physiological response to analgesic therapy. Genetic variability with response to pain has been demonstrated at the individual level and in different strains of chickens.[22] Behavioral changes can be very subtle but are often the earliest signs of pain detected by animal care staff. Behavioral change does not manifest uniformly among the many different species of birds and observers must become familiar with the full range of normal behaviors for the species. Without knowing the range of normal behavior, an observer will find it extremely difficult to detect abnormal behavior, especially in prey species, which often only demonstrate cryptic and subtle changes. The outward behavior of a companion bird acclimated to handling does not accurately reflect the physiological changes occurring with stress-related hormones, respiratory rate, or body temperature.[23] Therefore it can be inferred that a bird in pain may also mask physiological changes associated with the pain. In lieu of species-specific pain scoring sheets for birds, there is tremendous value in using a generic pain scale of 1 to 10 to evaluate a bird's pain and the response to treatment and recovery from the painful condition. Effective analgesia is expected to show a marked, easily discernable change in posture or behaviors that will effect a reliable change in the subjective pain score. If no change in pain score occurs, then the drugs, dosage, or frequency of administration need to be reevaluated for that individual patient.

Significant differences in species-specific response to analgesics have been and are continuing to be identified in studies in birds (see also Chapter 35 and *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 20, Recognition, Assessment, and Management of Pain in Birds).[24] In addition, acceptable analgesics in food-producing birds are few and this can limit the ability of the veterinarian to administer certain pain medications. For example, in the United States there are no withdrawal times for opioid medications in food-producing birds, yet μ-opioid agonists appear to provide excellent analgesia for these patients. Often in disasters the owner of the animal is not known at the time of treatment, thus obtaining documented permission to treat the animal with the expectation of not eating the bird or its eggs in the future cannot be obtained. Withdrawal times for the use of meloxicam in laying chickens have been established and this medication is very useful for reducing inflammation as well as providing analgesia, but should also be used in moderation in the face of hypovolemia or dehydration.[25] Topical analgesics including prilocaine, lidocaine, and other compounded salves have also been found to be quite effective in reducing pain in humans; however, to our knowledge no studies have been published evaluating systemic uptake of these topical analgesics in any avian species.

A major component of smoke inhalation is also the risk of systemic toxicosis due to components of the smoke such as carbon monoxide (CO) and hydrogen cyanide (CN) toxicity (see also *Current Therapy in Avian Medicine and Surgery*, Vol 1, Chapter 18, Advancements in Diagnosis and Management of Toxicologic Problems). CO is released with the incomplete combustion of fossil fuels and biomass often seen in fire.[26]

Inhalation results in CO diffusion into erythrocytes where it binds to hemoglobin. CO directly competes with oxygen and has a 230 to 270 times greater affinity for hemoglobin, thereby displacing oxygen and resulting in carboxyhemoglobin and tissue hypoxemia.[26,27] Co-oximetry is a specialized blood gas analyzer that can detect concentrations of carboxyhemoglobin to evaluate severity[28] but it is not always widely available and standards have not been established for most avian species. Despite unknown percentages for clinical signs, sparrows were historically used after coal mine fires to detect elevated levels of CO due to their perceived increased sensitivity.[29] Increased levels of carboxyhemoglobin result in clinical signs ranging from tachypnea, nausea, neurologic abnormalities, and death.[26] The main treatment is administration of oxygen therapy to help increase oxygenation levels and facilitate displacement of the CO off the hemoglobin, and thereby decreasing its effective half-life.[30] In structural fires, CN is often a significant source of toxicosis resulting from the combustion of commercial products and incomplete combustion of natural fibers. Inhalation of cyanide results in cellular toxicity by CN binding to cytochrome oxidase within the mitochondria and halting oxidative production of ATP.[16,30,31] Clinical signs include tachypnea, dry red mucous membranes, altered mental status, seizures, and death. Definitive diagnosis for CN toxicosis can have a significant lag time due to the need for laboratory-specific assays. Treatment is often based on suspected exposure and clinical signs.[16,31] The main treatment is supportive oxygen therapy, but often antidote therapy is also required. The most commonly utilized antidote is hydroxycobalamin. This is a natural derivative of vitamin B_{12} and when administered can directly chelate CN to form cyanocobalamin (vitamin B_{12}), which can then be safely excreted renally.[16,32,33]

Due to the chaos and stress of evacuation during wildfires, patients should also be evaluated for any other traumatic injuries such as fractures, lacerations, and soft tissue injuries (see also Section 8: The Trauma Patient). Human burn patients have also been noted to have a significant decrease in overall immune function making them more susceptible to infectious disease and recrudescence of chronic viral infections. In avian species, this should also be considered and the patient should be closely monitored for additional comorbidities such as underlying bacterial, fungal, or viral conditions. Ectoparasites should also be assessed and treated accordingly (see Chapter 9). A thorough ocular examination should also be performed due to the high risk of corneal ulcers and conjunctival irritation from the smoke.

Once the patient is stabilized, the traditional steps of burn care, decontamination, wound assessment, cleansing, debridement, and dressings, should be implemented.

Decontamination

Decontamination of burn patients is essential to remove ash and associated carcinogens, as well as improve visualization for assessment of their injuries.[34] Veterinary and animal care personnel should wear gloves and wipe down patients with decontamination wipes (e.g., Hero Wipes) to minimize exposure of carcinogens. If decontamination wipes are not available, adult cleansing wipes are safe and can remove the superficial layer of soot until such a time the animal can be bathed. If no open wounds are present, the bird should be bathed with dish soap and rinsed thoroughly. This may need to be performed several times. If large wounds are present, the cleaning can be done with chlorhexidine gluconate (2%) mixed with water or saline to create a 0.05% solution and thoroughly rinsed clean.[35] Ideally all debris should be cleansed from the animal, but this should also be balanced with the level of stress this will incur.

Wound Assessment

After cleaning the area, the skin should be assessed for burns. Burn wounds can be difficult to fully classify early in appearance and can range from erythema to charred tissue. Mild burns that affect just the epidermis are often noted by redness of skin with normal texture. These are typically superficial or "first-degree" burns and generally will heal with no secondary complications. Partial-thickness or "second-degree" burns extend into the dermis. These can be noted by sloughing of the skin ranging from mild blisters (superficial partial thickness) to thickened and blackened areas (deep partial thickness). Deep partial-thickness burns can be difficult to differentiate from full-thickness burns without histopathology.[36,37] Partial-thickness burns will retain some of the feather follicles and typically heal in shorter time frame than full-thickness burns. Full-thickness burns due to high-intensity heat result in the loss of epidermis and dermis including feather follicles. These burns can extend down to muscle and bone and take months to heal depending on the location and size.[6,37] Similar to other wounds (see Chapter 36), thermal damaged skin will declare itself approximately 5 to 7 days after the injury, but burns also have a second phase that can result in further tissue loss 10 to 14 days postinjury due to areas that have suffered vascular ischemia.[36] Therefore it is essential to ensure good vascular perfusion and adequate hydration in these patients.

Cleansing

Gentle washing and thorough lavage of the wounds are essential parts of burn wound care. This can be performed via irrigation technique using saline or water. There is no proven validity in using sterile saline/water versus tap water.[38,39] The main factor is the amount of volume and mechanical irrigation that is utilized to comprehensively clean the wound of debris, microorganisms, superficial slough, exudate, and residual dressings from previous bandage changes.[5] Irrigation techniques can be performed using a syringe, a bottle with holes, IV line setup with catheter, a shower head, or a hose depending on the size and extent of the wound. Antiseptic solutions can be used in addition, but there is no evidence of beneficial impact on prevention of infection or improved healing.[40] Antiseptic cleansers such as chlorhexidine solution should be followed with extensive rinsing with saline or water to ensure no residual solution is left on the wound, as this can have cytotoxic effects on healing tissue.[41–44] The use of hypochlorous acid has also been evaluated in humans and seems to hold promise as a good antiseptic cleanser that has no residual cytotoxic effects if left on the skin.[45,46]

Debridement

Once an area of skin is thickened, blackened, with no blood flow, it is deemed an eschar. This tissue is no longer viable and should be removed.[37] Early excision is the current standard of

care in human medicine, with full removal down to viable tissue within 24 hours. Delay in removal of the eschar can result in a nidus for increasing local and systemic inflammation, infection, and ischemia. Human research has shown markedly improved morbidity and mortality with the use of early excision.[5,36,47] Removal of the eschar within 24 hours is not always possible in avian patients but should be performed as early as possible to prevent further risk of sepsis.

In human medicine, it is also the standard of care to perform escharotomies in burned areas that are at risk for increased compression that may inhibit arterial flow. Full-thickness eschars can tighten as they dry out, and the ongoing inflammation and swelling of underlying tissues can together result in severe compression of vasculature. This is most commonly seen in areas suffering from circumferential burns such as along the limbs. An escharotomy is the act of incising the full-thickness eschar to allow tissue decompression and it can be performed on each side of the circumferential burn depending on the location (Figure 53.3).[37,48,49] This procedure has not been well established in veterinary medicine and further evaluation in birds is needed. With minimal subcutaneous tissue in birds, the risk of circumferential constriction due to full-thickness burns is significant. Based on the authors' experiences, escharotomies in birds should be considered in extensive burns to the limbs noted by marked erythema, bruising, dried or dead skin, or if the toes are cool to the touch. Several ground-dwelling birds were euthanized due to secondary limb ischemia from burn constriction in recent fires and limb amputation has been noted as one of the complications seen in wildlife.[1]

Removal of the eschar and wound debridement can be performed using sterile gauze or surgical excision. The key with debridement is to remove the abnormal tissue down to tissue that is viable and has adequate blood supply.[36] Debridement is essential to not only remove the dead tissue but also to disrupt the biofilm that can develop on wounds.[50,51] Enzymatic debridement is also possible, but human products have not been fully evaluated in avian species and pain can be associated with the use of some of these products.



FIGURE 53.3 An escharotomy is the act of incising a full-thickness eschar to allow tissue decompression. It can be performed on each side of the circumferential burn, depending on the location.

Dressings

The choice of wound dressings can often be challenging and there are numerous products on the market (see Chapter 36). Unfortunately, despite the ongoing research, there is no perfect wound dressing for every individual during their entire wound healing process. Wounds must be continually assessed to determine the right dressing for the wound. The dressings should be modified depending on the wound severity (depth), location, amount of exudate, time frame since injury, and the individual patient. In addition, the cost and availability of the dressing should also be considered.[5,52–55]

The ideal dressing should provide pain relief, prevent contamination, reduce dryness, and maintain body heat. The loss of the skin barrier, depending on the depth, can leave patients vulnerable to infection and mechanical injury, susceptible to loss of fluid and protein, as well as decreased thermal regulation. Therefore dressings should be considered essential for all wounds with partial (second-degree) to full-thickness (third-degree) burns.[5,36,53] It has also been noted that keeping a burn wound covered with an appropriate dressing is one of the most effective pain management treatments due to the relief of covering exposed nerve endings.[56–58]

Primary dressings are applied directly to the wound and are often followed by secondary dressings made up of bandage material. Depending on the stage of wound healing, level of exudate, and the potential for infection, primary dressing can range from topical ointments to skin grafts. Wounds in birds are often minimally exudative and so maintaining an appropriate moisture layer is vital to healing. The most common primary dressing in veterinary medicine is a topical ointment/bandage.[53] Petroleum-based dressings such as Adaptic (Adaptic, 3M Corporation, St. Paul, MN, USA) are commonly used with one to two bandage layers to help moisturize dry wounds and create a conducive environment for healing.[59] Unfortunately, these dressings are not always ideal for avian species due to the excessive oil and they do not have any antimicrobial activity. Hydrocolloid dressings such as Intracyte gel and Duoderm are also good choices to help moisturize wounds and the structure of the polymers in some hydrocolloid topicals can assist with mild debridement.[54] Hydrocolloids can be used along with or incorporated with other agents such as honey (Medihoney HCS) or silver.[54,60] Unlike human medicine, animals are often delayed in being seen for burn care and frequently have a high probability of infection. Therefore it is recommended that the ideal topical ointment for avian should also have some antimicrobial activity. Antibiotic ointments are not recommended due to the risk of creating resistant bacteria[37] and if treating domestic poultry and waterfowl, regulations on the use of antibiotics in food-producing animals must be followed. Any topicals with corticosteroids should also be avoided due to the potential for infection in the face of burn-related immunosuppression.[61,62] The standard of care has been the use of silver sulfadiazine (SSD) for burn wounds and is still the most frequently used in

wildlife.[1] SSD does have antimicrobial properties that will penetrate through damaged tissue and makes it ideal for using in the field on eschars.[37,63] Once a wound is debrided, SSD should not be used as it has been shown in multiple studies to cause cytotoxicity and significant delays in wound healing.[63–65] If SSD is applied prior to debridement, it should be used with secondary bandaging layers so as not to be ingested by the bird. Other common antimicrobial primary dressings include honey and other silver-based wound dressings. The use of honey is increasing due to its relatively low cost and benefits in wound healing. Honey has been shown to have not only antimicrobial properties but also antiinflammatory and chemical debridement effects. It has a high sugar content that creates an osmotic gradient and it also stimulates leukocytes to aid in wound repair. Studies have also shown honey to have significantly faster healing time than SSD and other topical ointments.[66–69] Other synthetic silver-impregnated dressings are also available including Mepilex AG, Microlyte AG, and Silverlon. Many of these products have shown good antimicrobial activity, improved wound healing, and less silver cytotoxic effects.[63,70] These treatments are also labeled for >48-hour antimicrobial activity in humans[71] but have not been evaluated for duration of activity in birds.

In contrast to topicals and other synthetic primary dressings, biologic dressings have been investigated and found to provide better pain relief and improved wound healing.[5] Biologic dressings include autografts, allografts, and xenografts. The ideal wound treatment is the use of a patient's own skin (autograft) as a partial- or full-thickness graft. This is the standard of care in human burn patients but requires a fully debrided and clean wound with minimal movement postsurgery. It also creates an additional wound on the patient that will require ongoing wound care and requires surgical expertise to have the best success for graft survival.[7,63] In many circumstances, an autograft is not possible. The next alternative is an allograft, which is skin from a different individual but from the same species. These are usually donated from cadavers, sterilized, and preserved for use on human patients who cannot have an autograft.[72] Stored allografts for birds are typically not available and this makes this option unlikely. The most cost-effective option that is more readily available is the use of xenografts, skin that is harvested and preserved from another species. The most commonly used xenografts in human medicine include porcine and bovine. The benefit of the xenograft is that it not only functions as a skin barrier to provide pain relief and protection but it also provides a collagen matrix to facilitate wound healing.[73–75] The cost for several brands of xenograft has been out of the reach of the veterinary market. Recently tilapia and codfish have been utilized in several published and currently ongoing studies.[76,77] The fish xenografts are more affordable and in the authors' experience have been a good alternative for burn wounds in birds (Figure 53.4). More studies are needed, and the market of primary dressing continues to grow with new research developments in this area.

Secondary dressings are synthetic materials that can be used on top of primary dressings to add additional security or help the wound environment by absorbing excessive exudate. Dressings can include foam or silicone pads, with additional roll gauze, Vetwrap, or Elasticon, depending on the location and the need to secure the bandage. Some primary dressings can be applied without a secondary and should be considered for areas that are not conducive to full bandaging.[52,53,78]



FIGURE 53.4 A tilapia xenograft was used to cover this large wound in a northern bald ibis *(Geronticus eremita)*. Recently, tilapia and codfish skin have been used for the care of burns and other wounds in birds, increasing between-treatment intervals compared with commercial dressings.

Prognosis

Overall prognosis for avian patients with burn injuries can be difficult to determine and depends on the systemic insult and extent of injuries. In human medicine, the depth and amount of total body surface area burned are significant factors in survivability.[7,36,79] In addition, the presence of clinical smoke inhalation or other systemic comorbidities such as renal failure can greatly impact the possibility of recovery.[6,80]

Long-term recovery and the possibility of return to function should also be considered especially for wild avian patients. In a recent study of burned wildlife seen by rehabilitation centers, over 61.4% of animals with burn injuries presenting for care were avian and 41% of the animals presenting went on to full recovery and release.[1] Potential long-term complications noted included scarring, feather loss, self-trauma, sepsis, amputation, behavior issues, and pressure sores.[1] Birds can go on to have full recoveries, but the potential complications should be considered and they should be continually assessed during the recovery period. The occurrence of large-scale wildfires continues to be the new normal in many parts of the United States and throughout the world.[81,82] We are only at the beginning of learning the best ways to treat avian patients and further research is needed to continue to advance the tools needed to provide the best care for burn-injured animals.

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CHAPTER 54

Wild Birds, Cats, and One Health

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INTRODUCTION

For about 10,000 years, cats have lived alongside humans. Their relationship with prey species, including birds, is much older than that. Hunting, catching, and killing are their telos (a function, a set of activities intrinsic to an animal, evolutionarily determined and genetically imprinted, originally conceptualized by Aristotle).

The purpose of this chapter is to explore the effects of cats, particularly feral cats, on birds, bird populations (particularly sensitive species), and the ecosystems on which they (and we) depend. It includes some brief comments on the treatment of birds injured by cats and examines the role of the veterinarian in preventing impacts on wild birds. In keeping with the emphasis in this volume of *Current Veterinary Therapy in Avian Medicine and Surgery*, we discuss this in a conservation and One Health context.

HISTORY OF AN INVASIVE SPECIES

Domestic cats *(Felis catus)* have long been common in urban and suburban areas throughout the world, and the number of cats is continuously growing.[1] In contrast to dogs *(Canis familiaris)*, relatively few cat owners in the United States (25%–50%) mark or confine their cats to an indoor lifestyle,[2,3] contributing to the numbers of stray and homeless cats and to predation on birds and small mammals. Homeless domestic cats may be unfriendly, stray, or somewhat or very tame, but their impacts on wildlife and the environment are similar. Cats can have more than two litters of four or more kittens per year,[4,5] and availability of food leads to increased survival of kittens, and movement of cats across landscapes.[6] In many landscapes, particularly those devoid of predators, the high fecundity and recruitment of cats (survival until adulthood) allow for rapid increase in, and maintenance of, large populations. Where they are subsidized with food, cat densities of 229 to 523 cats/km^2 have been recorded in an urban area of the UK, far higher than native mesopredator densities (averaging 37 animals/km^2 for red foxes).[7] Cat densities have been recorded as high as over 2,000 cats/km^2 in sites in urban Rome, Jerusalem, and Ainoshima, Japan.[8]

Cats evolved in Europe, North Africa, and Asia, but no similarly dexterous and fecund mesopredator was present in North America, Australasia, or Oceania prior to human colonization. Annual surveys by the American Pet Products Association have documented that cats have surpassed dogs as the most popular companion animal in most of North America and Europe,[9] with the number of pet cats in the United States being estimated to lie between 80 and 90 million individuals.[1] The number of feral cats is unclear but estimated at approximately 480 million worldwide, with numbers in the United States and Europe estimated to be in the tens of millions,[10,11] and between 2 and 6 million in Australia.[12] Such large populations of both owned and feral cats have negative implications for wildlife and public health,[13,14] and there is broad interest from community groups, nonprofit organizations, and governmental agencies in reducing free-roaming cat populations.

Biologically effective, yet socially acceptable, management strategies for feral cats are a matter of contention in the United States and other countries.[15] In locations with imminent conservation threats due to cats (islands, protected areas, wildlife refuges, restoration areas), trapping and removal, shooting, poisoning (in Australia), and other lethal methods may be used by government agencies to manage feral cats.[16,17] Feral cat management in cities and towns has typically involved animal shelters and related organizations capturing and either socializing for the purpose of adoption, or euthanizing, unwanted feral cats. Citing a lack of success at decreasing cat populations, approximately 30 years ago animal shelters began using a new approach of trapping cats, sterilizing them, often cutting off the tip of one ear for identification, and releasing them back to the site of capture (trap-neuter-release [TNR]).[18] Most often these cats are supplementally fed, although their caregivers do not consider themselves as owners, or take the responsibility expected of owners. Today, TNR (or TNVR if vaccinations are provided) is popularly promoted by several nonprofit and lobbying organizations, despite the problems it poses for wild birds and other wildlife, environmental and public health, and the welfare of the cats themselves.[1] TNR will be examined in further detail below.

ONE HEALTH

As noted in the introduction to this section (Chapter 48), the veterinary profession has seemingly accepted One Health as

an organizing principle, even if some of our activities fail to adequately balance impacts on animals, people, and the environment. This is particularly true regarding the management of cats, and comments on One Health will be found throughout this chapter. One Health is a central concept of the Canadian Veterinary Medical Association position statement on free-roaming and abandoned feral cats.[19]

IMPACT OF FERAL CATS ON BIRD POPULATIONS

Wild bird populations deliver many critical ecosystem services, including but not limited to insect control, seed dispersal, pollination, nutrient distribution, and critical roles in many food chains, and therefore are an important environmental component of One Health. Cat predation and cat diseases are well-documented major mortality factors for birds and small animals worldwide. Ecologists and conservation biologists are most concerned with the direct impacts of high numbers of feral cats on populations of native birds and other small wildlife, as well as competition with native predators for food, and degradation of ecosystem health and stability.

Cats are instinctive hunters whose prey drive is not connected to satiety centers in the brain.[20] The number of prey that cats capture is not influenced by the number of meals provided.[21] Domestic cats continue to hunt during periods of supplemental feeding, and feeding does not decrease hunting.[22] Even well-fed cats hunt and kill wildlife (Figure 54.1).

Predation studies have shown that feral cats from colonies in Georgia, USA, exhibited more hunting behavior compared to pet cats.[23,24] Sterilization did not affect the cat's motivation to hunt.[25] Birds and other wildlife face many threats, some of which are difficult to predict and prevent (e.g., extreme weather events, emergent diseases), but impacts from invasive species like feral cats are generally predictable and preventable.

Free-living cats pose a significant population-level threat to the birds, herpetofauna, and small mammals they prey upon.[17,26,27] Cats have been implicated in extinction of 63 species on islands (40 birds, 21 mammals, and 2 reptiles),[28] but also have negative impacts on songbirds in continental environments.[7] An intensive investigation of the impact of feral cats on migratory songbirds at three study sites in Southern Michigan estimated that cats killed 0.7 to 1.4 birds per week of 23 species (including two species of conservation concern), equaling 12.5% of all species.[27] It was estimated that feral cats kill 16,000 to 47,000 birds in these three study areas per year. Over the past decades, similar rates of predation and loss have been recorded by studies all across the world.[29–31]

A broader estimation of the impact of free-roaming cats on the wildlife of the United States suggests that cats kill between 1.3 and 4.0 billion birds, and between 6.3 and 22.3 billion small mammals annually.[32] Feral cats are responsible for a greater proportion of kills than pet cats, which may be related to the increased amount of time spent outdoors. Mortality on this scale can be significant at the population, regional, or flyway level, especially for rare and sensitive species. Particularly vulnerable are species that forage on the ground (e.g., quail, woodcock, rails, thrashers, towhees) and species that nest on the ground (e.g., terns, plovers, shearwaters). These losses can have ecological consequences that are hard to reverse, even with the removal of cats. For example, the loss of seabirds (most of which are ground nesters) can result in the loss of nutrient (nitrogen and phosphorus) transport from oceanic to continental locations, a vital ecosystem service.[33] In Hawaii, feral cats have major negative impacts on endangered 'Ua'u or Hawaiian petrel (*Pterodroma sandwichensis*; Figure 54.2)[34] and are of concern to at least 13 additional threatened, endangered, or endemic birds.[35] Regardless of whether in continental or island ecosystems, cat-caused injury and mortality affect birds by altering population dynamics. Cat kills can exceed the ability of native species to



FIGURE 54.1 Well-fed cat killing a yellow-rumped warbler *(Setophaga coronata)* at a bird bath in Fair Oaks, CA, circa 2003. (Photo courtesy D.A. Jessup.)

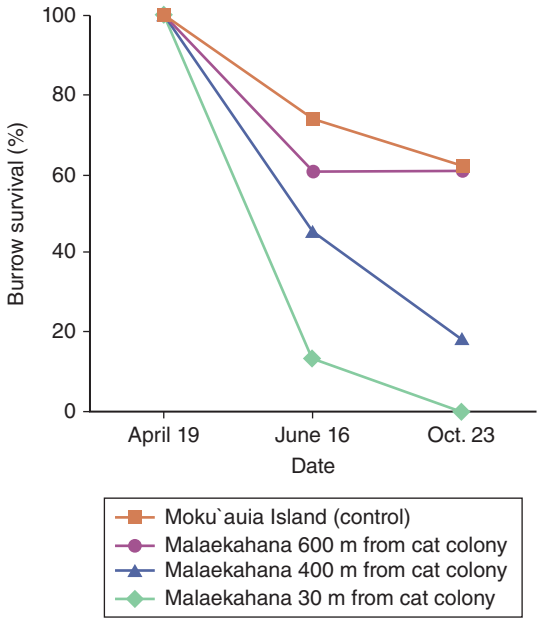


FIGURE 54.2 The serious negative impacts of feral cats on petrels and other ground-nesting seabirds in Hawaii have been well documented, in this case, the relationship between survival of petrel chicks in burrows and adjacency of feral cat colonies. Similar effects occur with other ground-nesting and ground-feeding species in both insular and continental habitats. (Courtesy D. Duffy.)

replace themselves, affecting the persistence of native wild bird populations.[36]

Cat diseases and parasites can have population-level effects on wildlife. Toxoplasmosis has been documented as a cause of death in many avian species, including passerines, doves, wild turkeys, and various diurnal and nocturnal raptor species. In Australia, the direct lethal effects of cats on endemic birds, reptiles, and small marsupials[37] are exacerbated by the high sensitivity of endemic species to lethal effects of toxoplasmosis.

IMPACT OF FERAL CATS ON WELFARE OF WILD BIRDS AND SMALL ANIMALS

Wildlife rehabilitation continues to gain popularity, with National Wildlife Rehabilitators Association (NWRA) membership growing from approximately 200 to >2,000 members since 1982. Notably, the subset of NWRA members responding to a member survey reported treating nearly 115,000 animals in 2007 alone,[38] which is certainly an underestimate given time passage and the survey's response rate. Birds and other small animals suffer from painful, debilitating, and often fatal injuries inflicted by feral cats.[39] Just as the welfare of cats is an issue for the veterinary profession, the welfare of all the species they negatively impact is also a serious welfare issue.

In 2004, feral cats were responsible for 24% of birds, 12% of mammals, and 15% of other species out of 5,669 wild animals presented over a 9-month period at a wildlife care facility in California.[11] Many of these injured wild animals were euthanized due to the nonrecoverable nature of their injuries, whereas others died from trauma or sepsis. More recently, a wildlife clinic in Tennessee reported cats to be among the top anthropogenic causes for submission of injured animals, responsible for almost half of the avian cases, of which the majority (around 70%) either died or were euthanized.[40] Interactions with cats was the second-greatest cause of small-mammal admissions to the Wildlife Center of Virginia over a 10-year period.[41] Moreover, data from 82 wildlife rehabilitation centers throughout North America found that, during a 3.5-year period, greater numbers of birds than reptiles, amphibians, or mammals were admitted for rehabilitation as a result of domestic cat attacks, and 78% of these did not survive.[42] The majority of immature animals admitted because of cat attacks also died or had to be euthanized because of the severity of injuries.[42]

When birds present for veterinary care and cat attack is suspected, standard principles of triage and emergency care should be applied (see Chapter 34). Obvious punctures and lacerations will require flushing, debridement, antimicrobials, and meticulous wound care. Cat bites often cause large skin lacerations, degloving, and/or crushing injuries. But, even if the bird is not bleeding and appears to be stable, it is imperative to perform a thorough examination to look for hidden puncture wounds by parting feathers carefully (Figure 54.3), and thoroughly searching for wet feathers, feathers caught in skin, and/or bruising. Thin and mobile avian skin can seal over puncture wounds quickly and, as a result, obscure deep wounds that can lead to sepsis, which may connect to internal injuries or massive hemorrhage, and death. Hence, if a cat bite is suspected, broad-spectrum antimicrobials are often given to prevent sequelae such as sepsis.



FIGURE 54.3 This mourning dove *(Zenaida macroura)* was admitted after a history of cat attack. Physical examination findings included multiple puncture wounds, including the one shown in the left pectoral muscle, and all the remiges on the left wing were missing. Close examination of birds attacked by cats is required as wounds may not bleed excessively, and small punctures and lacerations may only appear as wet spots or displaced feathers. (Photo courtesy Heather Barron.)

ECOLOGICAL AND ECONOMIC IMPACTS

As noted, many birds are consummate insectivores, seed dispersers, and nutrient recyclers. It is hard to overstate the negative impacts on ecosystems[43] and One Health due to anthropogenically caused declines in many bird populations. North American ecosystems and species did not evolve with medium/small feline predators (as opposed to larger bobcat, lynx, and mountain lion). As efficient small- to medium-sized predators, cats compete for prey with native mesopredators (e.g., hawks, owls, roadrunners, foxes, raccoons, skunks, opossums, weasels).[44] This predation is particularly impactful in developed, disturbed, or fragmented habitats. In places where native predators are reduced or absent, free-roaming cat numbers can increase markedly due to mesopredator release,[2] or simply by being the only mammalian predator, as is the case in a number of island ecosystems (e.g., Choiseul, Guadalupe, Hawaii, Mangere, Stephens, Stewart, etc.).

Economic impacts of events or activities (e.g., oil spills, wildfires, or development) that kill wildlife or degrade their habitat have recognized overall costs to society. One of the very few studies to consider the economic damage posed by feral and outdoor cats in the United States, conducted over 20 years ago, estimated the cost at $17 billion/year.[45] This dollar value was based on the estimated annual mortality to birds in the United States, which, at that time (1999), was conservatively estimated to be eight birds per cat per year,[46] multiplied by an estimated feral and free-ranging cat population of 71 million, and $30 per bird killed.[45] An updated

valuation of the damage caused by feral cats in the United States using a different approach estimated the cumulative costs over the past 60 years at $45.5 billion, with cats being the costliest invasive species in the nation.[47] Economic valuations may be challenged, but $30 is much lower than veterinary care costs for surviving birds, or legal penalties for harming endangered or migratory birds.[48] If updated with more current estimates of bird mortality from nonowned cats, it is between 952 million and 3 billion birds for the continental United States per year,[32] a more current economic loss estimate could range from $28.6 to $92 billion per year.[49] Importantly, this is only for birds.

Cats are the most common domestic animal source of rabies exposure,[50,51] and the species most commonly responsible for human postexposure prophylaxis (HPEP). Even though feral cats in managed colonies may receive a rabies vaccination when sterilized, they do not receive boosters, and records are generally not kept. So, when a person is bitten or scratched by a feral cat, they invariably must go through HPEP, which has significant costs ($20,000 – $30,000) and inherent medical risks. Public health agencies do not keep data on whether cat contacts resulting in rabies exposure involve feral or owned cats, but news stories over the last decade suggest this could be important and useful information. The economic costs associated with the several other cat-zoonotic diseases discussed later have never been estimated but, in aggregate, could easily approach the yearly economic losses for direct bird mortality.

ONE HEALTH AND DISEASES OF CATS

Diseases harbored by cats, particularly feral cats, can negatively impact the health of other animals and humans, posing a significant threat to One Health. Cats are carriers, reservoirs, or shedders of pathogens, including but not limited to *Bartonella henselae*, *Toxocara cati*, *Toxoplasma gondii*, typhus, tapeworms, hookworms, rabies, and feline leukemia virus.[52–55]

Toxoplasma

Toxoplasmosis has been found from the Arctic to the tropics across terrestrial, aquatic, and marine ecosystems and affects all homeotherms.[56] *T. gondii* in cat feces led directly to the extinction in the wild of the ala'la (Hawaiian crow; *Corvus hawaiiensis*).[57] Mortality due to toxoplasmosis also affected the recovery of nēnē or Hawaiian geese *(Branta sandvicensis)* and red-footed boobies *(Sula sula)*,[58] and is an important cause of death in the endangered Hawaiian monk seal *(Monachus schauinslandi)*,[59] and other listed or sensitive marine mammals including Southern sea otters *(Enhydra lutris nereis)*,[60] and two species of manatee.[61,62]

Felids are the definitive host and the only species shedding *T. gondii* oocysts in their feces. An estimated 40 million human beings are infected with *T. gondii*, and it is considered a leading cause of death attributed to foodborne illness in the United States.[63] Worldwide, approximately 40% of *T. gondii* exposures appear to be the result of oocyst contamination of water or soil, thus directly the result of cat feces in the environment. Ingestion of bradyzoites in undercooked meat accounts for about 60% of infections worldwide, but only oocysts can survive in the environment, so even ingestion of bradyzoite tissue cysts begins with fecal shedding of oocysts by felids. In the United States, most human infections are from oocysts, not ingestion of bradyzoites, based on the sporozoite-specific enzyme-linked immunosorbent assay (ELISA).[64] Feral and free-roaming cats in coastal communities can be a significant source of oocysts that wash directly into the ocean.[53]

Bartonella

B. henselae, the cause of cat scratch disease, usually results in a self-limiting regional adenopathy. But more serious sequelae are common, and it is now considered an occupational hazard for the veterinary profession. In 1994, five Florida children were hospitalized with encephalitis associated with cat scratch fever.[65] The oral flora of cats, above and beyond *B. henselae*, is notorious for causing difficult-to-treat local and regional infections (due to *Pasteurella multocida*, *Staphylococcus* spp., and *Streptococcus* spp., etc.).

Rickettsia

A daycare center at the University of Hawai'i at Mānoa was closed for 2 weeks in 2002 because of concerns about potential transmission of murine typhus *(Rickettsia typhi)* and flea *(Ctenocephalides felis)* infestations afflicting 84 children and faculty.[66] The fleas were from a feral cat colony that had grown from 100 to over 1,000 cats, despite a TNR effort.[66] Similar situations have been reported in Southern California and Texas housing complexes where high populations of subsidized feral cats and rats coexisted.[67]

Hookworms and Roundworms

In Florida, one study found approximately 75% of feral cats to be positive for the feline hookworm *Ancylostoma tubaeforme*, whereas 33% were positive for *Ancylostoma braziliense*.[68] In 2010, an outbreak of hookworm (cutaneous larva migrans) was reported in people (Figure 54.4) who had visited beaches in Miami-Dade County, FL.[69] Feces from feral cat colonies along the beachfronts were the putative source. In 2006, 22 cases of cutaneous larva migrans were reported in children at a summer camp due to cat feces in sand boxes.[70] Similar circumstances can also lead to the more serious visceral larva



FIGURE 54.4 A case of cutaneous larva migrans due to feline hookworm *(Ancylostoma)* presumed to be the result of contact with feral cats' feces on a Florida beach. (Photo courtesy Scott C. Sherman, Department of Emergency Medicine, Cook County Hospital (Stroger), Chicago, IL.)

migrans in humans caused by the migrating larval stages of the common cat roundworm, *T. cati*.

Feline Leukemia Virus

Cats can serve as reservoirs for significant pathogens that affect other wildlife, such as feline leukemia virus (FeLV), which was found to be transmitted from feral cats to the endangered Florida panther.[71] A similar case was reported in a mountain lion in California.[72] The problems *T. gondii* poses to marsupials (and many other species) have been mentioned, so establishing feral cat colonies at or adjacent to zoos is a concept whose direct (predation) and indirect (disease) consequences for captive and free-living wildlife, and public health, may be quite problematic.

OUTDOOR AND FERAL CAT MANAGEMENT

Many veterinarians advise cat owners that their animals are safer indoors. However, a double standard exists regarding feral cats as the follow-up care they receive usually falls far short of standards in feline practice. Clients with indoor cats may experience behavioral problems; hence veterinarians need to provide information on enriching cats' lives and how to provide opportunities for supervised outdoor time. In 1997, the American Bird Conservancy (ABC) launched *Cats Indoors*, a program focused on protecting both birds and cats. The ABC Cats Indoors website contains excellent updated instructions and specific recommendations.[73] An increasing number of cat owners are keeping their cats indoors or are exploring a variety of safe and sensible ways to permit them supervised or controlled outdoor time. Indoor-only cats also have fewer diseases and parasites that are harmful to cats and people.[74] Outreach has resulted in a growing number of cat and non–cat owners demanding that municipalities enact and enforce ordinances promoting responsible cat ownership, something that has been in place for dogs for decades.

"Belling the cat" has been around for hundreds of years and may have some efficacy in warning birds and small animals of the presence of a predatory feline, despite ambiguous results in published studies.[75–77] Studies show that antipredation devices may reduce (but not eliminate) the number of prey brought home.[78] The mere presence of free-roaming cats, however, can reduce avian fecundity,[79,80] and orphaning of nestlings continues. Furthermore, cat bibs, flashers, or bells on collars do not protect pet cats from vehicles, predators, fights with conspecifics, trauma, poison, or extreme weather conditions. As a result, these devices should not be considered a substitute for keeping cats indoors and may give pet owners a false sense of accomplishment.

In North America, the responsibility for the management of unowned and stray animals generally resides with city, regional, and/or county animal control agencies. Traditional methods used to deal with stray and/or feral cats include: (1) capture and return to, or reclaim by, the owner; (2) public adoption; and (3) euthanasia. It should be noted that all three options are humane and viable, with reclaim/return facilitated by routine use of RFID chips. All three options remain available, and with modern enhanced communication and networking, animal control agencies can now assist one another with "cage space" to hold cats longer, or move them to locations where adoption is more likely.

TRAP-NEUTER-RELEASE

TNR for cats gained attention in the early 1990s, concurrent with the formation of Alley Cat Allies (ACA), a national cat-focused organization that today boasts a budget of $10 million. It was first promulgated as a population reduction method that would result in the eventual elimination of outdoor cat colonies. TNR-promoting organizations subsequently changed the goal to colony stabilization, or no new kitten births. Evidence of success of even these revised outcomes has been lacking. When these more modest goals did not succeed, some TNR promoters changed from reducing the numbers of feral cats to "saving" them, to emphasize avoiding euthanasia (touted as the only alternative and part of the "no-kill" movement) and maintaining feral cats in perpetuity.

The primary reasons that TNR (and its variants) does not work on landscape or large population levels are as follows: (1) people keep abandoning intact cats, often at known TNR locations, believing they can live free and/or will be taken care of; (2) it is nearly impossible to exclude immigration by intact cats, attracted by food, from peripheral areas; (3) the cat's inherently high reproductive capacity means that immigration of intact females, and those not yet sterilized, can produce kittens faster than they can be trapped; (4) it is very difficult to capture and spay the very high percentage of cats (75%–85%) required for population stabilization for at least 5 to 10 years; and (5) many TNR caregivers get discouraged after years without reduction of the cat population and quit. Hence, TNR programs do not function as an effective means to manage a population, but largely as a means to reduce intake at shelters and prevent euthanasia of homeless, unadoptable cats.

Population modeling has shown TNR to be less effective than simple removal.[4] TNR has been practiced at Stanford University for over 35 years with no decrease in cat numbers.[11] In summary, TNR, as commonly practiced, seldom stabilizes or reduces and almost never eliminates established feral cat colonies unless significant numbers are regularly removed.[6,81,82]

Although success or failure of feral cat TNR management programs can be monitored with easily collected data and statistical analyses, this is rarely done and is strongly resisted by most TNR advocacy organizations. An assessment of two large, sophisticated feral cat TNR programs in San Diego County, CA (n = 14,452), from 1992 to 2003, and in Alachua County, FL (n = 11,822), from 1998 to 2004, using field data in a Ricker mathematical model to describe population dynamics, showed that in both counties, there was no consistent reduction in per capita growth, the population multiplier, or the proportion of female cats that were pregnant.[81] To date, no published study has refuted these findings.

A very basic problem with TNR is that it is almost entirely cat-centric, or cat advocate-centric, with little or no consideration of wildlife, people, or the triad of One Health. Specifically, TNR maintains nonnative predators in the environment, essentially in perpetuity, with no diminished capabilities for injuring and killing wildlife. TNR does not stop the movement of diseases between cats and other animals (including people), thus creating perpetual health risks. Waste and spoiled cat food can attract wild birds and scavenging and predatory



FIGURE 54.5 Feral cat feeding stations attract scavengers such as rats and raccoon (A) and wildlife predators such as fox and coyote (B) as well as birds. This type of situation encourages pathogen jumps between species and is the antithesis of the One Health concept. Raccoon, fox, and skunk are important vectors of rabies in North America. Fights over food and injuries jeopardize the health of cats and people who may subsequently encounter them. (Photos courtesy Stefan Woltmann, Clarksville, TN, USA.)

mammals, including rats, as well as skunks, foxes, coyotes, and raccoons, some of the most important wildlife vectors of rabies in North America (Figure 54.5). Cat feeders and advocates are even willing to defy law enforcement efforts to limit the damage done when leftover cat food attracts endangered species like the nēnē goose in Hawaii.[83] Concentrating cats, their urine and feces, and that of closely related species creates an environment that encourages pathogens to jump between species, the antithesis of One Health management. However, nobody, not feral cat advocacy groups, caregivers, veterinarians, or pet food retailers who facilitate TNR, or agencies of government, is willing to take responsibility for, or mitigate, these serious problems feral cat colonies create.

TNR is not considered humane for cats by many experts. The most common causes of feral cat mortality in one California county were vehicle trauma, predation, disease, and/or severe weather (Figure 54.6).[84] People for the Ethical Treatment of Animals (PETA) have called TNR "subsidized abandonment" and state that "feral cats live short, hard lives on the streets." They never die of "old age."[85] The American Veterinary Medical Association (AVMA) has stated, "Free-roaming cats may be exposed to injury, suffering, and death from vehicles; attacks from other animals; human cruelty; poisons; and traps. Additionally, these cats are more likely to be exposed to feline-specific, and zoonotic diseases, and will prey on and can negatively impact native wildlife populations."

ALTERNATIVES TO TNR OR EUTHANASIA

Feral cat advocacy groups insist that there are only two alternatives: TNR or mass euthanasia. This is a "false dichotomy," a technique used in debate. Feral cat numbers are unlikely to decline unless multiple strategies are adopted in combination. A first step must be community commitment to greater responsibility for feral cats and significantly decreasing or stopping cat reproduction. Mandatory spay-neuter laws could be a start. Recently, it has been shown that a single injection of a feline anti-Müllerian hormone (AMH) transgene was sufficient to induce long-term contraception in female cats[86] and may be able to replace surgical contraception. But to be effective in feral and free-roaming cats, advances in contraception probably need to be orally active and cat specific to protect nontarget species.

Where TNR colonies exist, focusing on removing cats for adoption is a positive step that communities can take. In addition, strongly enforced, effective licensing, identification, and confinement laws are integral. Continued financial support for animal control agencies and shelters that are tasked to



FIGURE 54.6 The most common causes of feral cat morbidity and mortality are trauma from various sources (vehicles, predators, malicious people), severe weather, and diseases and parasites. This cat, whose clipped ear identifies it as being from a TNR program, was found on a highway median strip in Brevard County, FL. It had a fractured mandibular symphysis and other injuries and would have died of thirst and starvation, or additional trauma, if not rescued. It was not "wild," but it recovered and did fine as a pet. (Photo courtesy C. Strorts.)



FIGURE 54.7 Indoor-outdoor enclosed catios are commercially available, and larger barn-like versions, like this one in Chico, CA, have been used to house feral cats where free-roaming colonies once existed.(Photo courtesy L. Winter.)

remove, socialize, and adopt animals is also essential. In the last few years, networking among shelters has enabled the movement of unadopted cats between locations, alleviating overcrowding, decreasing pressure for euthanasia, and increasing opportunities for adoption.

Pervasive and consistent public education programs are needed to encourage responsible feral cat management, as well as pet ownership by promoting: (1) reproductive control of all cats; (2) awareness of the responsibility that TNR promoters have for the health and welfare of people and other animals; (3) keeping cats indoors or supervised while outdoors; (4) methods to practically achieve this; and (5) collective responsibility for owned and unowned cats, whereby reabandoning them is considered unacceptable. In addition, cat owners need to be shown examples of devices that—with varying levels of supervision—can be used to allow cats to enjoy the outdoors without harming wildlife (e.g., pre built and commercially available cat condos, flexible cat tunnels, harnesses, cat carriers, cat strollers). One of the intermediate potential solutions for replacing free-living TNR colonies is enclosed colonies. Enclosed colonies (sometimes called sanctuaries) provide all the benefits afforded by a managed TNR colony in an enclosed, typically outdoor space (Figure 54.7) and are safer for wildlife, people, and cats themselves.[87]

WHAT CAN YOU DO?

There is a great deal any veterinarian or conservation-minded person can do to educate the public about the pernicious effects of feral and free-roaming cats on wildlife, human and environmental health, as well as actions that can be taken or encouraged. They include, but are not limited to, the following: (1) standing up for one health; (2) not doing harm; (3) demanding responsibility; (4) promoting viable solutions to reduce harm to wildlife, human, and environmental health; (5) speaking out in public forums and with clients; and (6) being informed.

Stand Up for One Health

As has been noted in this chapter, maintaining cats outdoors is hard to justify under the One Health paradigm. Although the AVMA policy statement on feral cats acknowledges several of the negative consequences of outdoor cats to wildlife, public health, and the environment, it continues to assume that TNR and "community cat" numbers will decrease, despite little evidence to demonstrate that this is occurring on meaningful scales. Veterinarians who care about balancing the health of animals, humans, and the environment need to seek and promote solutions that cause the least damage to all and that resolve problems rather than making them permanent.

Do No Harm

If asked to participate in a TNR program, consider responding "only if the 'R' is not re-abandon." The AVMA encourages keeping cats indoors, both for their own safety and for the protection of wildlife. The Wildlife Society,[88] a 10,000-member organization of professional wildlife biologists (the wildlife biology equivalent to the AVMA), and many avian, aquatic, and wildlife veterinary organizations strongly oppose TNR, primarily because of the impacts on native species.

Demand Responsibility

AVMA's policy on feral cats states: "The goal of colony management should be continual reduction and eventual elimination of the colony through attrition… For colonies not achieving attrition and posing active threats to the area in which they are residing, the AVMA does not oppose the consideration of euthanasia when conducted by qualified personnel, using appropriate humane methods as described in the AVMA Guidelines for the Euthanasia of Animals." Yet seldom are TNR colonies, once established, monitored for reduction or elimination, and veterinarians who contribute to TNR have shown no willingness to be involved in seeing that this AVMA expectation is met.

Cat caregivers and their supporters need to be held responsible for killing and injuring wildlife and for the pollution caused by TNR colonies. They need to be responsible for feces, food waste, its attraction of rats and other vermin, garbage, and for the attraction of wildlife to a potentially lethal site. Where TNR is being practiced on public land or with public funds, the vital rates of the population, success or failure in stabilizing or decreasing numbers, costs and conflicts, and the fates of the cats themselves should all be documented. Continued irresponsible behaviors, either by individuals, organizations, or officials, may need to be legally challenged, as has occurred in Los Angeles and New York. Elected officials need to be reminded that protecting wildlife health and ecosystems, public health, and property, rather than just the interests of vocal cat protectionists and organizations, is their job.

Promote Viable Solutions That Reduce Harm to Wildlife, Human, and Environmental Health

Reducing the yearly reproduction of cats and promoting all social, legal, political, or educational efforts needed to accomplish that needs to be "Job 1" wherever you stand on feral cat issues. So, consider:

- Recognizing that free-living cats are nonnative, invasive small- to medium-sized predators in North America and harmful to ecosystems that support native wildlife.
- Supporting all efforts to educate the public not to abandon their cats: THEY ARE NOT GARBAGE.

- Supporting local and regional and local animal control efforts that emphasize adoption and that do not victimize wild birds and other animals, or help them change if they do.
- Recognizing that life and death on the streets and back lots is not really a caring or humane outcome for most feral cats themselves is very damaging and cruel for small wildlife, destabilizes ecosystems, and increases public health risk for several zoonotic diseases. TNR, as currently practiced, is not compatible with a One Health perspective.
- Insisting that existing TNR efforts be monitored for success, kept away from important wildlife habitat, and improved or closed if they fail to stabilize, then reduce cat numbers.
- Supporting early trapping and taming of feral kittens for adoption.
- Supporting increased efforts to tame, hold, and place stray and formerly feral cats, optimize cooperation and movement to utilize existing cage space, and reduce pressure for euthanasia.
- Supporting "Cats Indoors" and other efforts to reduce the impacts of owned and unowned cats on wildlife, environmental and human health.
- Supporting client-owned "catios" and considering where larger-scale cat sanctuaries might be used to enclose existing feral cats groups.
- Counseling clients to protect their cats, optimize their health, and reduce the many problems created by cats that are allowed to roam free.

Speak Out in Public Forums and With Clients

Veterinarians are well-respected members of the community and can have a powerful impact on the public with whom they interact every day, and at local government meetings where feral cat management strategies are often set. Engage with others who do not share your views and hold constructive discussions. Hopefully, all groups can agree that the ultimate goal is to have cats under the care of a person; safe from vehicles, predators, and harsh weather, and with preventive medical care; and away from wildlife they kill and maim.

Be Informed

Feral cat management is newsworthy, controversial, and a hard subject to stay current on. Not all publications are based on solid science or on comprehensive review. The authors of this chapter would like to draw the reader's attention to one recent comprehensive science-based review of the management of feral cats.[89]

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SECTION 12 MEDICINE OF SPECIFIC TAXONOMIC GROUPS

CHAPTER 55

Falconry: Past, Present, and Future

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INTRODUCTION

Falconry, also called "hawking," is the hunting or taking of game with trained falcons, hawks, eagles, and in some regions even with owls, although owls are excluded from the definition of falconry. A falconer is then generally defined as someone who keeps, cares for, trains, and hunts with hawks, falcons, and other birds of prey. In 2010, the United Nations Educational, Scientific and Cultural Organization (UNESCO) through the Convention for the Safeguarding of the Intangible Cultural Heritage recognized falconry as an intangible cultural heritage.[1] Falconry is so much more than a hobby or pastime. It represents knowledge, practice, experience, culture, traditions, expressions, arts, literature, social events, craftsmanship, and comradery transmitted from generation to generation through families, mentorship, and training.[1]

Falconry transcends gender, religious, political, national, and socioeconomic boundaries and is practiced on every continent except Antarctica. The cultural diversity within falconry is evident during the International Falconry Festivals, the first of which was held in 2007 at Engelhard Estate, United Kingdom (UK), and was the largest gathering in history. This and other events bring falconers together from around the world to experience the history, pageantry, comradery, and passion of falconry. A second festival was held in the UK in 2009, then Al Ain, United Arab Emirates (UAE), in 2011, and Abu Dhabi, UAE, in 2014 and 2017 (Figure 55.1).

The origins of falconry date back to more than 2000 BCE, originating spontaneously in the Central Asian Plateau, Persia, the Middle East (Arabia), then shortly thereafter in other parts of Asia (China, Japan, Mongolia; see sections on Falconry in the Middle East—The Past and Falconry in Asia—The Past).[2–4] In these parts of the world, falconry served a utilitarian purpose—literally to put food (meat) on the table. Falconry spread to Europe, particularly due to trade between Arabia, Europe, and the Far East and reached the Mediterranean near CE 400. In Europe and the UK falconry gained popularity as a sport of nobility, yet it was practiced by all socioeconomic classes (see sections on Falconry in Europe—The Past and Falconry in the UK—The Past). Unfortunately, the development of gunpowder and firearms in the 14th century caused a significant decline in the practice of falconry as raptors were then viewed as vermin, to be shot and killed.

At the core of falconry is the concern for the health and well-being of the birds of prey that falconers use. Frederick II of Hohenstaufen, Holy Roman Emperor, King of Sicily and



FIGURE 55.1 The Parade of Nations at the 3rd International Festival of Falconry, Abu Dhabi, United Arab Emirates, 2014. Under the patronage of His Highness Sheikh Khalifa bin Zayed Al Nahyan, President of the United Arab Emirates, and organized by the Emirates Falconer's Club and the Cultural Programs and Heritage Festivals Committee, the event featured a desert camp, a scientific program, cultural events, and educational opportunities for hundreds of falconers, representing over 80 countries. (Photo courtesy Mike Dupuy, used with permission.)

Jerusalem, was considered to be the greatest of all historical falconry aficionados. His text, *De Arte Venandi cum Avibus (The Art of Falconry)*, represents one of the first scientific literary works on avian anatomy,[5] and many consider him one of the founders of ornithology. Frederick II stressed the importance of hygiene, sanitation, good nutrition, exercise, and medical therapy to maintain the health of birds used for falconry. Now, with the advent of hospitals and rehabilitation centers around the world, specifically focused on the care of free-ranging raptors and those in managed care, the behavioral, medical, and surgical management of birds of prey is a distinct discipline in the field of avian medicine and raptor conservation.

In 1968, the International Association for Falconry (IAF) and Conservation of Birds of Prey was founded in Zweibrücken Hof, Düsseldorf, Germany, during the conference of the International Council for Bird Preservation (ICBP), with the following objectives: (1) to represent the art of falconry throughout the world, (2) to preserve and encourage falconry within the context of sustainable use of wildlife, (3) to encourage conservation, the ecological and veterinary research on birds of prey, and to promote, under scientific guidance, domestic propagation for falconry, (4) to develop, maintain, and amend national and international laws, treaties, and conventions to permit the pursuit and perpetuation of falconry, (5) to require the observation of falconry, hunting, conservation and welfare laws, regulations, traditions, and culture with regard to the taking, import, export, and keeping of birds of prey, the taking of quarry species, and the right of access to land in the country concerned, and (6) to promote and uphold a positive public image of falconry with specialist organizations that regulate or otherwise affect falconry.[6]

Falconry is constantly evolving and characterized by innovation in all aspects of the activity including behavior and health care, introduction and use of new species of raptors, and innovative training methods using kites, drones, radio-controlled aircraft trailing lures, and even radio-controlled aircraft that resemble other avian species. The practice of falconry may vary around the world; however, a common theme among falconers is the preservation of natural resources, conservation of birds of prey and their habitats, and a close association with the raptors they use for this activity.

Much of the language used in falconry originates from the Latin-French language. Many falconry terms are now used in the modern English language, with many people unaware that their origin comes from falconry. Confusingly for people new to the sport, some words like "cast" have multiple definitions depending on the context used. A glossary of common falconry terms (in the English language) is included in the chapter (Table 55.1). It is important for a veterinarian treating falconry birds to have an understanding of the language used so an effective husbandry and medical history can be obtained.

TABLE 55.1
Glossary of Common Falconry Terms (in the English Language)

Term	Definition(s)
Accipiter	True hawk or shortwing. Short, rounded wings and long tail give high maneuverability.
Austringer	A person who trains and hunts with hawks (accipiters). Originates from the Latin word for goshawk—astur.
Aylmeri	Leather anklets fitted around the bird's legs with eyelets through which jesses are threaded and removed.
Bate	To fly off the fist or perch. May happen with a keen hawk baiting toward the falconer or a nervous bird baiting away from an unfamiliar object.
Bewit	Thin leather strip used to attach a bell or other equipment to the leg of a bird.
Block	Solid flat perch with a padded (usually synthetic turf) top, for falcons and eagles.
Broadwing	Birds of the *Buteo* and *Parabuteo* genera. Broad wings are ideal for soaring flight.
Cadge	A frame—traditionally wood, but now often plastic—used to carry multiple hawks into the field.
Cast	A pair or group (similar to a pack) of birds flown together—common with Harris's hawks. Restraining the bird in the hands—during examination or when fitting equipment. When a bird ejects the indigestible portion of the food—called a casting or pellet. The act of propelling a bird forward off the fist to give some extra initial forward impetus.
Cope	To trim and shape an overgrown beak or talons.
Creance	A line used during the final part of training before free flight.
Deck feathers	The central pair of tail feathers—bells and telemetry are commonly attached to these feathers.
Eyass, eyess, eyas, nyas	A bird in its first season (<1 year age).
Follow-on	A hawk moving from tree to tree or perch to perch following the falconer.
Free lofting	A hawk being kept untethered in a mews or special weathering chamber.
Glove/gauntlet	Glove used when a falcon or hawk is sitting on the falconer's fist.
Hack	A period of free flight before being caught for training. Traditionally performed with eyass birds.
Hack back	A form of soft release used to release rehabilitated wild birds following treatment.
Haggard	A bird of prey caught after at least one molt.
Halsband	Light fabric collar used to attach a bell to a hawk's neck.
Hood	Traditionally a close-fitting leather head garment used to blindfold a bird to reduce stress caused by visual stimulus during training and sometimes traveling.
Jesses	Narrow straps of leather fastened around a bird's leg. Mews jesses have a slit end to allow attachment of a swivel. Field jesses are without a slit, which minimizes the risk of tangling in branches.
Imp/imping	Process of replacing a portion of a broken or damaged feather with another for feather repair.
Mangalah	A short cuff used by Arab falconers to hold the falcon on the fist.
Manning	A period where a bird is accustomed and desensitized to environmental stimulus.

TABLE 55.1
Glossary of Common Falconry Terms (in the English Language)—cont'd

Term	Definition(s)
Mantle	Where a bird stands over food or prey with wings and tail spread to hide it.
Mews	Derived from the French word for molt—mew. Mews are aviary-type buildings where birds are kept free-lofted or to molt.
Mutes	Droppings.
Passage hawk	An immature hawk caught during its first migration.
Slip	To release a hawk from the fist in pursuit of quarry.
Swivel	Rotating metal-ringed device for joining jesses to the leash.
Tyring	A bony piece of food designed to be time-consuming to eat—requiring a hawk to pick at the food (e.g., a head of a rabbit).
Wait on	When a bird circles above the falconer waiting for the quarry to be flushed.
Water pan	Bowl/basin for bathing and drinking.

FALCONRY IN THE MIDDLE EAST—THE PAST

> *The best falconer is not the one who hunts the largest number of houbaras, but the one who has the wisdom of leaving some for tomorrow.*
>
> **HH Sheikh Zayed bin Sultan Al Nahyan, President of the UAE (1918–2004)**

In the past, Bedouins across the Middle East used to trap, train, and hunt with migratory falcons to supplement their basic diet. The falcons were then released in early spring as it was not possible to keep and feed them during the summer months. Falconry has been part of life in the desert since ancient times.

Arabian academics have been able to establish that, in the Arabian Peninsula, falconry saw its early beginnings during pre-Islamic times in the Al Jaheliya era with Al Harith bin Muawiyah bin Thor bin Kindah. According to history, one day, while Al Harith was watching a man setting up a net to trap birds, a falcon appeared and attacked a bird already caught in the net, and the falcon became entangled. Al Harith marveled at this and ordered that the falcon be brought to him. He took the falcon to his house and appointed a servant to feed and train the falcon for hunting. In a short time, Bedouins became aware of the experiences of Al Harith and his falcon, and soon the trapping and training of falcons for hunting became part of life in the desert.[7]

Then came Islam. One of the most famous hunters during the period that witnessed the rising of Islam was the renowned Hamza bin Abdulmutallab bin Hashem (54 before Hejri–3 Hejri, CE 556–625). He was greatly admired for his courage and was dubbed "The Lion of God." It is said that he embraced Islam after returning from a hunting trip while holding a falcon on his wrist.[7]

In the ensuing period, falconry continued to flourish during the Al Umauwiya era in post-Islamic times. One of the most famous falconers of this era was the Caliph Yazid bin Muawiyah bin Abisofian Al Umauwy (26–64 Hejri, CE 646–683). Abu Al Hassan Ali Al Hussein Ali Al Masaudi once said, "Yazid loved music, birds of prey, salukis and cheetahs, and was an enthusiastic hunter, a sport in which he excelled. He was the first man to carry a cheetah on horseback."[7]

Yazid, when appointed Caliph of Damascus in 60 Hejri (CE 679), ordered the construction of falconry hunting lodges for his own personal use. Subsequent caliphs thereafter used these lodges. Caliph Hisham bin Abdulmalik Al Umauwy (92–125 Hejri, CE 710–742, appointment date 105 Hejri, CE 723) was also a keen hunter. While on a hunting trip, he observed his party from an elevated point. Some hunters used birds of prey, while others hunted with cheetahs, bows, and arrows. Later he commissioned a drawing of the hunt, where he and some of his aides were portrayed by the distinguished artist Al Ghatrif bin Khudama from the tribe of Ghassan. Hisham also instructed Al Ghatrif to care for his birds of prey. He was to train and prepare them for hunting and to cure them when they were ill. Hisham also bestowed upon him the title "Hunt Master of Hisham bin Abdulmalik." A special department was set up within his court to deal with issues related to his falcons and falconry.[7]

In the Al Abbasiya era, falconry rose to new dimensions with the influence of the Persians, who were well known for their ability to train and hunt with birds of prey. During the rule of the first caliph of the Al Abbasiya era, Abu Al Abbas As Saffah Al Abbasi (104–136 Hejri, CE 722–753), falconry continued to thrive, since As Saffah was very fond of birds of prey and a devoted hunter who practiced falconry from an early age. His interest and enthusiasm for hunting were legendary and he was a source of inspiration to all his people. Yet there were other eminent caliphs in this era with a great passion for hunting that promoted falconry to the status of art.[7]

During the reign of the 5th Caliph (appointment date 170 Hejri, CE 786) Harun Al Rashid Al Abbasi (148–193 Hejri, CE 766–809), individuals were summoned to explain a particular hunting skill they had developed. Al Rashid was already a skilled hunter, but he always had a keen interest in furthering his knowledge in a continuous quest for perfection. By this time, falconry was not only a well-established entertainment of the caliphs, but it was also considered an affair of the state. During the reign of the 6th Caliph (appointment date 193 Hejri, CE 808) Mohammed Al Amin bin Harun Al Rashid Al Abbasi (170–198 Hejri, CE 786–813), 1% of the total daily budget of the caliphate was allocated to maintain the sport of falconry. Thus salaries of up to 70 dinars a day were paid to falconers and falcon keepers.[7]

The first Arabic manuscript related to falcons and falconry, *Benefits of Birds and the Comprehensive Treatment of their Diseases*,

is believed to have been written by Adham bin Mahres Al Bahili during the reign of Caliph Harun Al Rashid Al Abbasi. In one of its sections, it says, "It was natural that the keeping of birds of prey and hunting birds should not be restricted to the Caliphs but that people in all walks of life should be able to do so as well." This was a manuscript composed of 153 chapters in which issues such as diagnosis and diseases were first described. This manuscript is believed to have been enriched by the participation of Al Hajaj bin Khaytamah during the reign of the Caliph Mohammed Al Amin bin Harun Al Rashid Al Abbasi. *The Animal*, one of the most famous treatises in Arab literature on animals of all times, was written by Amro bin Bahar bin Mahboob bin Kanani Al Jahiz (163–255 Hejri, CE 779–868). In his book, the famous author mentioned that Arab falconers preferred to hunt with saker *(Falco cherrug)* and peregrine *(Falco peregrinus)* falcons rather than with Eurasian goshawks *(Accipiter gentilis)*, although the latter were much in demand by Persians, Turks, and Indians at the time.[7]

At the end of this era, between the years 490 and 601 Hejri (CE 1096 and 1204), Arab falconry extended its influence over Western Europe. In this respect, two of the most important Arab contributions to European falconry included the hood and the use of the lure.[7]

Sadly, there was a crucial interlude in the continuing contribution of Arab literature to the history of falconry with the Mogul occupation of Baghdad in 656 Hejri (CE 1258). It has been estimated that Arab writers made approximately 30 publications on falcons and falconry between 287 and 391 Hejri (CE ninth century), at the peak of Arab cultural evolution, and 700 to 803 Hejri (CE 13th century), when the Abbasid Empire collapsed.[7]

Throughout the succeeding decades, falconry continued to decline in the domains of the Ottoman and Mogul empires, but falconry prevailed deep inside the immensity of the sands of the Sheikhdoms in the Arabian Peninsula.



FIGURE 55.2 In the Middle East, falconry is part of the cultural heritage passed from generation to generation. Fast cars and fast-food restaurants in a fast-changing world have displaced customs and traditions, but falconry remains deeply rooted in the heart of Arabs as a way of communion with the past.

FALCONRY IN THE MIDDLE EAST—THE PRESENT

Falcon Species Used in Falconry in the Middle East

In the Middle East, falconry has been practiced exclusively using falcon species since ancient times. The main reason for this tradition is that some falcon species, notably the peregrine, the saker, and the lanner falcons *(Falco biarmicus)*, used to be common migrants through the region. Through trapping, training, and hunting with migrant falcon species, the art of falconry using falcons became deeply entrenched in the desert way of life (Figure 55.2).

Current species widely used in falconry in the Middle East are discussed in the following sections.

Saker falcon

The saker falcon has been the favorite falcon among Arabs in the skillful art of falconry for centuries. Arab falconers have traditionally favored this species because of its remarkable endurance in flight, great strength, and the ability to cope with the rigorous environment of the deserts of the Arabian Peninsula. As stated earlier, the first person to train and hunt with falcons during the Al Jaheliya era was Al Harith bin Muawiyah bin Thor bin Kindah. This very first falcon was a saker falcon. Similarly, when Hamza bin Abdulmutallab bin Hashem embraced Islam after returning from a hunting trip, the falcon he was carrying on his wrist was also a saker falcon.[7]

The saker falcon is the largest of the desert falcons and the second largest falcon species in the world after the gyrfalcon *(Falco rusticolus)*. The color of the plumage is highly variable depending upon the geographical origin. Generally, the head is whitish to cream with dark-streaked feathers. The face is cream with dark brown eye stripe and moustachial streak. The throat and the nape are whitish to cream. In adult birds, the dorsal plumage or mantle ranges from pale brown and dark brown to almost black and very often with cream to pale brown tips. The ventral plumage is white cream to pale brown, heavily marked by dark brown streaks. The cere and feet are grayish yellow. In contrast, juvenile birds tend to have a darker plumage and the color of the cere and feet are gray-blue. The body weight of adult males is 730 to 950 g and that of adult females is 970 to 1300 g.[8]

Peregrine falcon

The peregrine falcon is without a doubt the "Prince of the Falcons." Its beauty, grace, and extraordinary speed are legendary among falconers from all around the world. In the Middle East, however, the peregrine falcon occupies only second place among the favorite species used in Arab falconry. The name in Arabic given to the peregrine is "Ash Shaheen," which is an expression derived from the Persian language meaning "balance." This is because the peregrine falcon cannot tolerate starvation or overfeeding. In some countries of the Middle East, falconers refer to peregrines as being "more fragile than glass."[7]

The peregrine falcon has the most extensive distribution of any of the largest falcons in the world. It is generally smaller than saker falcons, but with a strong, well-built, and stocky body. Peregrines have characteristically broad-based and long-pointed wings with relatively short tails. The head is black to dark slate gray-blue with white lower cheeks and throat. The face has a wide and well-defined black moustachial stripe. The color of the plumage varies according to the geographical origin. In adults, the color of the mantle ranges from dark gray and dark blue to black with a paler blue to dark blue rump. The ventral plumage is white to pale brown with small spots on the chest and the rest crossed with bars. The cere and feet are bright yellow-orange. In juvenile birds, the mantle is dark brown with paler feather fringes. The ventral plumage has dark brown streaks from the chest down to the lower abdomen. The cere and feet are gray-blue. The body weight of peregrine falcons varies considerably in the different subspecies, but in general, the body weight of females is 750 to 1300 g and of males is 582 to 750 g.[8]

Lanner falcon

The lanner falcon is a large desert falcon and yet another favorite species used in falconry in the Middle East. However, most experienced Arab falconers consider lanners as falcons for beginners as they lack the qualities of the ideal falcon to hunt large quarry such as houbara bustards *(Chlamydotis macqueenii)*.[7]

The lanner falcon is about the same size as a peregrine falcon, but with a slightly slimmer bodily configuration. The wings are longer and broader, with rounded wing tips and a longer tail. The color and markings of the head and nape vary according to the subspecies. The North African and Near East subspecies are creamy to pale brown, without streaks, with a dark brown forehead band and eye stripe; the southwest Arabian subspecies is more red-brown with dark brown streaks at the forehead; in the European subspecies, the lower part of the crown and nape is pale red-brown. The cheeks and lower forehead are white and with a long and narrow black moustachial stripe. The mantle is barred and brown-gray in color. The rump and the upper tail are slightly paler and barred. The ventral plumage is pale cream to white with small dark brown to black spots mainly around the flanks and thighs. The cere and the feet are yellow. Juveniles have darker streaked crowns, giving a "red-headed" appearance. The mantle and lower plumage are darker and heavily streaked. The cere and feet are gray-blue. The body weight of males is 500 to 600 g and females is 700 to 900 g.[8]

Gyrfalcon

The gyrfalcon is the largest falcon in the world and a species that is still highly valued in Arab falconry. Its large size and great strength made gyrfalcons very popular among Arab falconers during the early 1970s, but their popularity soon waned when it was realized that they were not able to cope with the extreme weather conditions prevalent in the Middle East.

The gyr is a large Arctic falcon, similar in appearance to a peregrine falcon, but heavier, greater in size, and with large broad-based pointed wings and tail. The color of the plumage varies according to the geographical origin, but three basic colors are recognized: white, gray, and black. In adult dark morphs, the forehead is white with a dusky eye patch and a weak moustachial streak. The upper plumage and mantle are gray-brown with a profusion of bars and notches. The ventral plumage is basically white, heavily marked by spots and thick bars on flanks and lower abdomen. In adult pale morphs, the plumage in general is white with sparse spots, bars, or streaks across the mantle, flanks, and thighs with dark brown to black primary wing tips. In the adult bird, the cere and feet are yellow. All markings in juvenile birds are brown rather than black and the ventral plumage is streaked instead of spotted. The feathers of the upper plumage and mantle have paler fringes. The cere and feet are gray-blue. The body weight of males is 805 to 1300 g and of females is 1400 to 2100 g.[8]

Hybrid falcons

A hybrid falcon is the product of a cross between two different species of falcons. This is achieved under captive conditions and by the use of artificial insemination (AI) techniques. The production of hybrid falcons was initiated during the early 1970s across the United States and Europe. Nowadays, hybrid falcons are produced mainly in Canada, United States, Germany, and the UK to supply the demand across the Middle East. The most common and popular hybrids are gyr-peregrines and gyr-sakers.[9] In Germany, hybrids with peregrine and saker have been prohibited since 2005.

Hybrid falcons came to replace the use of pure breeds in some countries, notably the UAE, after the Convention on International Trade of Endangered Species of Wild Fauna and Flora (CITES) legislation was reinforced in the country on April 26, 2003, and the import of wild-caught falcons was prohibited (Figure 55.3).[10]

Training

Balloons and kites

One of the early forms of modern training involved the use of balloons filled with helium and kites. Balloons and kites were sent about 300 to 400 m high using a thick fishing line (1.8 mm) fixed to a stake on the ground. A lure was suspended low from the ground (e.g., 10 m) using a second nylon fishing line (0.8 mm) and a falcon would be released to catch the lure. This was elevated gradually as the falcon approached, with the aid of a pulley fitted on the balloon or the kite, and using a battery-operated winch from the ground. The falcon was made to fly higher and higher gradually until it reached the lure and the falcon would bring it to the ground, where it was rewarded. The use of balloons and kites is now obsolete.

Fixed-winged remote-controlled airplanes

Inevitably, technological innovations of the 21st century have made their mark in the way falcons are trained today. Remote-controlled fixed-winged airplanes are now widely used for training falcons in all the countries of the Middle East where falconry is practiced. These are commercially available as petrol or battery operated. The bodies are built on a skeleton made up of lightweight plywood and balsa wood *(Ochroma pyramidale)* and covered by a hardened plastic film. Various sizes of airplanes fitted with engines ranging from 0.6, 0.8, 1.2, and 1.8 cc are used (Figure 55.4). These are the equivalent values of petrol engines. The airplanes are fitted with five servos: one for the rudder and one for the elevator of the tail, two for the ailerons of the wings, and one for the release



FIGURE 55.3 Several important steps have been taken in the Middle East to preserve falconry for future generations, including adhering to the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES) and the creation of captive breeding programs to produce birds for hunting and racing and to stop relying in wild-caught falcons. Pictured here is one author's (M.P.J.) male gyrfalcon-peregrine falcon hybrid. (Photo courtesy Michael P. Jones.)



FIGURE 55.4 Fixed-wing remote-controlled operated aircraft are widely used nowadays in the training program of falcons destined for racing or hunting. This type of aircraft is commonly provided with two rechargeable batteries. One small battery is used to operate the servos of the rudder, the elevator of the tail, the ailerons of the wings, and the release mechanism, and a much larger battery to run the engine.

mechanism. The airplane carries one or two batteries depending on the size of the engine. These can maintain 5- to 8-minute flights at full throttle. An additional, much smaller, battery operates the servos. Each airplane is also fitted with a speed controller and a receiver. All the electric and electronic components of the airplane are operated by a transmitter or controller handled by an experienced operator from the ground. A train is made up using a 5-m nylon or cotton string, a lure, a parachute, and several swivels along the train to avoid entanglement. The train is fitted onto the release mechanism at the ventral body of the airplane. The training consists of allowing the falcon to chase the lure gradually at various speeds and heights and for a determined number of minutes. The falcon is then allowed to catch the lure at the end of the train; this is freed through the release mechanism by remote control, and the parachute slows the fall of the falcons. Training programs using fixed-winged aircraft include sessions for height, speed, and maneuverability. Falcons, or conversely, the airplane can be fitted with devices to read the height and the speed from the ground during flight.

The use of jet propulsion airplanes camouflaged as birds is popular in some countries in the West and by some individuals in the Middle East. However, these are mainly used in the Middle East for entertainment purposes rather than serious training as they never achieve the necessary speed to train a falcon.

Game launchers

Game launchers (Figure 55.5) are very popular in the training of retriever dogs but are also used by some parties for the training of falcons. Commercially available or purpose-built units are used. These consist of foldable arms lined with a band made up of canvas or plastic mesh and propelled by very strong springs. A prey, such as a partridge or a pheasant, is placed onto the band. The arms are then folded and held by a locking mechanism. This is operated through a single servo connected to a receiver fitted with an antenna so it can be operated at a distance of up to 400 m. A single battery operates the servo. When the locking mechanism is activated, the bird is propelled high into the air and begins to fly. The falcon



FIGURE 55.5 Game launchers are very popular in the training of retriever dogs but are also used for the training of falcons. These consist of foldable arms lined with a band made up of canvas or plastic mesh and propelled by very strong springs. A prey, in this case, a pheasant, is placed onto the band. The arms are then folded and held by a locking mechanism. This is operated through a single servo connected to a receiver fitted with an antenna so it can be operated at a distance of up to 400 m. A single battery operates the servo. When the locking mechanism is activated, the bird is propelled high up in the air and begins to fly. The falcon is then released to chase the live prey.

is then released to chase the live prey. It is common for game launchers to be placed within a pit in the ground. Therefore when the launcher is operated, the prey appears to fly out from the ground, simulating the natural escape flight of a bird flying from behind a bush.

Drones

Drones or multicopters are widely used for the training of falcons around the world and are very popular in the Middle East (Figure 55.6). Multicopters used in falconry are made up of a four- to six-arm lightweight frame carrying four to six engines fitted with propellers and speed controllers. A single large battery operates the engines, and they are fitted with a global positioning system (GPS) unit, a receiver, and servos. Multicopters need calibration in each geographic region of the world where they are used to identify the north magnetic pole. Hexacopters (six engines) are commonly fitted underneath with a box made up of aluminum or Perspex with a hatch with two doors as a floor. A live bird, such as a duck, partridge, or pheasant, is placed within the box through a loading door. The unit is sent up to the desired altitude and, through the servos, the hatch opens to release the prey. The falcon is then sent to pursue the prey. Conversely, quadcopters (four engines) are more commonly used to carry a lure and make the falcon chase it at a certain height and speed. This is particularly popular in the West.

Lures

The use of lures for the training of falcons is as old as falconry itself. In the Middle East, lures are made up using two wings or more of houbara bustard stitched together (Figures 55.7 and 55.8). A swivel is stitched in the carpal end of the wings. A nylon or cotton rope, 5 to 8 mm in diameter and



FIGURE 55.6 Drones or multicopters are widely used for the training of falcons in the Middle East. Hexacopters (six engines) are commonly provided with a single large battery to operate the engines and are fitted with a lightweight box with a hatch with two doors as a floor. A live bird, such as a pheasant, is placed within the box through a loading door. The unit is sent up to the desired altitude and, through the servos, the hatch opens to release the prey and the falcon is then sent to pursue the prey.



FIGURE 55.7 A fishing line is tied up to the jesses of a novice falcon before releasing it to a lure at the early stages of training. Fishing equipment can be placed to great use during training sessions replacing the much heavier cotton ropes widely used previously. This rope used for training is commonly called a "creance" in the Western falconry world.

approximately 1.25 m long, fixes the lure to a fishing rod. The lure is used in the same way in the East as in the West by calling and swinging the lure repeatedly to entice the falcon to come. When the falcon is close, the lure is moved swiftly, enticing the falcon to come back. This action is repeated each time the falcon dives. The distance and the number of passes are both increased gradually as the training advances.

Pigeons

Training falcons using live rock pigeons *(Columba livia)* is sadly still widely practiced in many countries in the Middle East. Commonly a pigeon is released and a falcon is sent to catch it. Pigeons are known to transmit several diseases to



FIGURE 55.8 Lure training is an essential part of the training program of any falcon or hawk used for racing or for hunting anywhere in the world. In the Middle East, a typical lure is made up of one or two pairs of houbara bustard *(Chlamydotis macqueenii)* wings tied up in a bundle. This is fixed to a short and thick fishing rod using a short 6- to 8-mm nylon rope.

falcons including trichomoniasis, inclusion body herpesvirus hepatitis, avian pox, and, notably, Newcastle disease. Collision accidents are very common when pigeons fly under cars with the falcon following at full speed and are unable to stop.

Hunting

Hunting is currently widely practiced in the Middle East and it is still enjoyed by a new generation of falconers. In the West, falconry is considered a sport, yet to Arab falconers, falconry is not a sport, but a way of life: an excuse to go back to the desert to sit with friends around a campfire and to reunite with a life that is already gone. Fast cars and fast-food restaurants in a fast-changing world have displaced customs and traditions, but falconry remains deeply rooted in the Arab cultural heritage as a way of communion with the past.

Modern Arab falconry hunting trips can be a small family affair, in which members of one or two families or a group of friends go out together for a few days. However, the hunting parties of members of the royal families can be a very large and complex concern lasting for up to 4 to 6 weeks and sometimes even longer. The size and complexity of these parties are directly related to who you are.

Falconry is still practiced in the Kingdom of Saudi Arabia. However, in countries such as Qatar, the Kingdom of Bahrain, Kuwait, and the UAE, falconry is almost nonexistent or extremely limited due to their territorial size. The Sultanate of Oman prohibited falconry and any form of hunting on its soil a long time ago.

The hunting method is simple. Generally, a group of six to eight falconers get into three to four cars or more and drive across the desert. Commonly, there is a leading car with a group of trackers walking in front, following the characteristic footprints of a houbara bustard impressed on the fine sand. When tracks are found, the trackers follow the trail until the houbara bustard flies and the falcon is released and the chase begins.

Technology from the 21st century has made a significant impact on traditional Arab falconry. In bygone days, Bedouins, on camelback, toiled for a full day to cover 30 to 45 km through the vast sands of the Arabian Peninsula in search of quarries. Today, falconers using four-wheel drive cars can cover the same distance in less than 1 hour. In days long ago, trackers used the position of the stars and planets, the signs left by the prevailing winds on the edges of the sand dunes, the marks left by the wind on the soft sand on the downwind side of a clump of grass, and the charred signs left by the fierce sun on trees and bushes in order to find their way around the desert. Nowadays, trackers can use a compass for orientation or make full use of conventional GPS technology to accurately pinpoint a particular spot in the desert or to navigate through an endless sea of sand dunes using GPS coordinates to mark the trajectory. In the old days, falconers lost many falcons flying beyond the horizon during a persistent chase of a bustard. Now, telemetry technology can track a falcon as far as 30 to 40 km away.

However, in spite of the technological innovations, falconry in the Arabian Peninsula is still as traditional as the falconers themselves. What has been described before are commodities of life, which have had the same effect in the East as in the West, but it has little or nothing to do with falconry itself.

Falcons in the Middle East are still selected and valued by the measurement of the total length from the shoulder to the tip of the tail, the girth around the shoulders, and the color of the plumage. Falcons are still fitted with the same type of falconry furniture, following the same pattern as the one designed in ancient times. Falconers still train falcons using a lure made of bustard wings tied up in a bundle as was done by their forefathers. The favorite quarry is still the elusive and well-camouflaged houbara bustard.[11–14]

Racing

With the advent of laws and legislation regulating hunting in each country and the inability of falconers to hunt in neighboring countries such as Pakistan, many falconers have turned to falcon racing as a pastime and to continue flying falcons. Falcon races are nothing new. These were discussed 30 years ago as a possibility, but plans never took off the ground until recently. Nowadays, racing is a sport widely practiced in every country in the Middle East where falconry is allowed. The races consist of allowing a falconer to release the falcon from the wrist to a calling falconer swinging a lure at a distance of 400 m. The falcon has to fly low. The winner is the falcon that is able to fly the same distance in the shortest possible time. Several categories are made using pure species and hybrids, males and females, and age.

To prove how popular falcon racing has become in the Middle East in recent years, in February 2019 the King Abdulaziz Falconry Festival in the Kingdom of Saudi Arabia was declared the world's largest falcon racing tournament ever with participation of a record 1723 falcons competing over a 10-day (January 25–February 3) period, winning a place in the *Guinness Book of Records*. The total cash prize was SR 17,630,000 (US $4,700,691.48), with the participation of falconers from the Kingdom of Saudi Arabia and other countries in the Middle East and the rest of the world.

Quarry

Houbara bustard

The houbara bustard has been the favorite quarry of Arab falconers for centuries (Figure 55.9). Its enduring strength in flight, the exquisite camouflage of its plumage that allows it to blend with its surroundings, and the diverse array of devices at its disposal to defeat the falcon are legendary among falconers in the Middle East.[7]

The houbara bustard is a medium-sized desert bird inhabiting steppes, harsh open plains, and scrub desert. There are three different subspecies of houbara bustard: the North African *(Chlamydotis undulata undulata)*, from North Mauritania to Egypt; the Canary Island *(Chlamydotis undulata fuertaventurae)*, from the Eastern Canary Isles; and the Asian species the MacQueen's bustard *(Chlamydotis macqueenii)* from the Sinai and Arabian Peninsulas, and North Caspian Sea east to Baluchistan, Afghanistan, Kazakhstan, and Mongolia. The Canary Islands and North African subspecies are virtually sedentary, while the Asian subspecies migrate from Central Asia in the autumn, southwest, wintering mainly in Pakistan, northwest India, Iran, Iraq, and the Arabian Peninsula. Houbara bustards are opportunistic omnivores, feeding on invertebrates, such as beetles and ants; plant material including leaves, flowers, and fruits; and small vertebrates, such as geckos and skinks.[15]

Asian subspecies adult males weigh 1.5 kg to 2.4 kg, and adult females 1.1 kg to 1.7 kg. The breeding season varies according to the latitude of its habitat. In the northern part of its range (Kazakhstan, Mongolia), the breeding season is between April and June, while in southern areas (Balochistan, Afghanistan), it is between March and May. The female usually lays two to four eggs. Only the female incubates the eggs, and the chicks hatch after a period of around 23 days. The chicks fledge after a period of 32 to 38 days.[15]



FIGURE 55.9 The houbara bustard *(Chlamydotis macqueenii)* has been the favorite quarry of Arab falconers for centuries. Its enduring strength in flight, the exquisite camouflage of its plumage that allows it to blend with its surroundings, and the diverse array of devices at its disposal to defeat the falcon are legendary among falconers in the Middle East. Captive breeding programs have been established in the Middle East since the mid-1980s to release captive-bred stock back into the wild and thus ensure the survival of the species for future generations.

Although the MacQueen's bustard has a wide distribution, its number has been steadily declining due to overgrazing, intensive farming, pesticides, and hunting in the countries where it breeds and by urban development, unregulated hunting, and trapping in the countries through which it migrates.

Several conservation projects were established in the Middle East as early as the late 1980s to preserve and increase the houbara bustard by captive breeding and release and habitat protection and restoration.[16]

Cape hare

The Cape hare *(Lepus capensis)* has been a favorite prey of Arab falconry for centuries. Yet the hunting of hares is regarded as a delicacy among Arab falconers since it requires highly selected falcons specially trained for that purpose.

The Cape or desert hare is a medium-sized mammal that inhabits open scrub desert and submountainous regions across the Middle East. In the Middle East, the color of its coat varies according to the time of the year. During the summer, the coat is sandy-brown in color, while during the winter, the color is grayish-brown. Hares have large ears, which act as radiators through a mechanism called thermoregulation. The ears have a vast network of blood vessels through which the circulating blood is cooled down before returning to the body. When threatened, hares have the tendency to "freeze," blending perfectly well with the surrounding habitat. Its cryptic coat also contributes to a near-perfect camouflage. It is possible for a crouching hare hiding behind a bush to go completely unnoticed by a passerby walking beside it.

Hares are more active during the early hours of the morning and late afternoon. It is mainly during the evenings that the desert hare feeds. Its diet includes leaves, grasses, and roots. Hares do not dig burrows and prefer to shelter within a thick grass patch or under a dense bush. Hares breed throughout the year giving birth to up to four litters. Each litter consists of up to four young.[17]

Stone curlew

The stone curlew *(Burhinus oedicnemus)* is also a favorite quarry among Arab falconers. Its small size makes it ideal for beginners carrying smaller falcons such as lanner falcons, or male saker, and male peregrine falcons.

The stone curlew is a medium-sized terrestrial bird inhabiting Mediterranean, steppe, mild temperate, and arid zones. There are six subspecies of stone curlew: *B. oedicnemus oedicnemus*, found across Europe, from southern England to southern Russia, and south to Siberia, Italy, the Balkans, and Caucasus; *B. oedicnemus saharae*, found on the Mediterranean Islands, Greece, Turkey, and southwest Iran, and south to North Africa; *B. oedicnemus harteri*, found in southeast Russia and central Iran, east through Kyrgysztan and Kazakhstan to the Zaisan basin; *B. oedicnemus insularum*, found on the eastern Canary Islands; *B. oedicnemus distinctus*, found on the western Canary Islands; and *B. oedicnemus indicus*, found in India and Sri Lanka to Southeast Asia. The stone curlew is not a resident species in the Arabian Peninsula, appearing only during its winter migration. It is believed that the subspecies that migrate through the Middle East are *B. oedicnemus saharae* and *B. oedicnemus harteri*.[15]

Stone curlews feed mainly at night on invertebrates such as beetles, ants, grasshoppers and crickets, flies, moth caterpillars, earthworms, and small vertebrates such as lizards, amphibians,

and mice. Adult males weigh 450 to 475 g, while females weigh 415 to 440 g. The breeding season in most of its range is from February to early June.

The female lays from one to three eggs and the chicks hatch after an incubation period of 24 to 26 days. The chicks fledge between 36 and 42 days.[15]

There are no formal conservation programs specifically devoted to hares or stone curlews in the Middle East, but the hunting of houbara bustards, hares, and stone curlews is prohibited or regulated by law in the countries of the region.

FALCONRY IN THE MIDDLE EAST—THE FUTURE

As stated previously, falconry forms an integral part of the Arab cultural heritage. As a tradition, falconry has defied time and adversity and it will continue to be part of life in the Middle East. Falcon races came to fill a vacuum left by the inability of ordinary falconers to use falcons for hunting and are attracting hundreds of falconers from different countries in the Middle East tempted by the lucrative prizes. Hunting is almost exclusively reserved for individuals of the royal families or wealthy individuals with the necessary resources to hunt in their own country or to travel abroad. Pakistan is still a very popular destination for hunters, but it is becoming more difficult to obtain the necessary permits and licenses to hunt in the country. After the collapse of the Soviet Union on December 26, 1991, some of the newly created countries, such as Kazakhstan, Azerbaijan, Uzbekistan, and Kyrgyzstan, opened the doors to selected groups of Arab hunters to hunt in some areas of their territories. This practice has continued to date. However, this practice has placed an immense pressure on the free-living population of the Asian houbara bustard as these are among some of the countries in which the species breeds.

Only by joining forces, pooling resources together, and adequate legislation could it be possible to tell if the Asian houbara bustard will continue to flourish protected in their breeding grounds and in the range of countries through which they migrate and to continue with captive breeding and release programs to ensure the survival of the species.

The mystical relationship between falcons, Arab falconers, and the houbara bustard has defied time as the tradition continues almost unchanged by time, technological advances, and commodities of the 21st century.

FALCONRY IN ASIA—THE PAST

Asia is the largest and most densely populated continent in the world covering an area of 44,579,000 km^2 (17,212,000 square miles), representing about 30% of the total land area and 8.7% of the total surface area of planet Earth. The continent is home to 4.5 billion people (June 2019) comprising approximately 60% of the total population of the world. The Asian continent is subdivided into five geographical regions, including the following countries: Central Asia (Tajikistan, Uzbekistan, Kazakhstan, Turkmenistan, and Kyrgyzstan); East Asia (China, Mongolia, North Korea, South Korea, Japan, Hong Kong, Taiwan, and Macau); South Asia (Sri Lanka, Bangladesh, India, Afghanistan, Pakistan, Bhutan, Nepal, and the Maldives); Southeast Asia (Brunei, Cambodia, Indonesia, Laos, Malaysia, Myanmar, Philippines, Singapore, Thailand, Timor Lester, Vietnam, Christmas Island, and Cocos Islands); and Western Asia (Georgia, Armenia, Azerbaijan, Turkey, Cyprus, Syria, Lebanon, Israel, Palestine, Jordan, Iraq, Iran, Kuwait, Bahrain, Qatar, Saudi Arabia, UAE, Oman, and Yemen).[18]

Some of the earliest archaeological findings on falconry are from Mesopotamia, a large area corresponding to present Iraq, Kuwait, eastern Syria, southeastern Turkey, and regions along the Turkish-Syrian and Iran-Iraq borders in Western Asia. Some of the earliest evidence of falconry can be seen in several carvings from archaeological sites in the ancient Neolithic and Hittite city of Alaca Höyük, situated in Alaca, Corum Province, in north-central Turkey and in Karatepe, a late Hittite fortress in Osmaniye Province, southern Turkey, dating 1400 to 1600 BCE. In one such carving, a large bird can be seen on the fist of a human figure holding a hare from its hind legs. In another example, a bas-relief found in the ruins of Khorsabad of the palace of the Assyrian King Sargon II in present northern Iraq, dated 722 to 705 BCE, shows a man with a small bird of prey on his fist. This image depicts the bird fitted with jesses passing between the thumb and forefinger of the holder. This is clear evidence that falconry and the use of falconry furniture was already in use in the eighth century BCE in Mesopotamia. These archaeological carvings and bas-reliefs depicting human figures carrying birds of prey fitted with falconry furniture suggest that falconry is at least 3500 years old in Mesopotamia.[19–23]

In the Korean peninsula, falconry arrived c. 220 BCE and was entangled with military and political affairs. In China, falconry also played a very important role in politics. Written records go back to 700 BCE depicting falconry as it is practiced today. The imperial time of the Chu Kingdom used falcons, eagles, and shortwings as widely used in modern falconry. This would place the beginning of falconry in the region at over 3000 years.[24]

Archaeological excavations throughout Western Asia, but particularly in Palestine, Israel, Jordan, Syria, Iraq, and Iran, in recent times are shedding new information on the possible use of birds of prey for hunting, dating back 8000 to 10,000 BCE. This evidence is in the form of bone remains found at archaeology excavations. This has been investigated by scientists from a branch of archaeology denominated zooarchaeology, the branch responsible for the study of bone remains primarily from mammals and birds found at archaeology sites. Many of the bones recovered have been identified as bones from birds of prey. Some scientists consider that these findings are remnants of food left by the inhabitants of the settlements, but the bones might actually represent the earliest evidence of training and hunting with birds of prey. Aside from bird of prey remains, these archaeological sites also harbored bone remains of prey species matching the range of prey species of current birds of prey, including gazelles (*Gazella* spp.), foxes, (*Vulpes* spp.), hares (*Lepus* spp.), partridges, francolins, and sandgrouse. Could this indicate that birds of prey were used by inhabitants of these settlements to hunt the smaller species, throwing the origin of falconry into the late Stone Age?[23]

Takagari was the art of hunting with trained goshawks, eagles, and falcons in Japanese. Due to the great cost and the time required to train and maintain the birds, it was only available to the nobility and became symbolic of their status and spirit. The sport of the samurai elite. Takagari was referred to

in the *Kojiki*, the Records of Ancient Matters, written in CE 712, the *Nihon Shoki*, the Chronicles of Japan, dated CE 720, the anthology of poems known as the *Manyoshu* from CE 759, as well as the *Genji Monogatari*, Tale of Genji. Records indicate that falconry in Japan started in the fourth century. According to writings in the *Nihon Shoki* (CE 720), falconry was first introduced by the Baekje noble Sakenokimi in CE 359 during the reign of Emperor Nintoku, the legendary emperor of the fifth century. The Japanese Emperor Saga (CE 786–842) was the 52nd emperor of Japan, reigning from CE 809 through 823. In CE 818, the emperor ordered the editing of the first surviving textbook on falconry named *Shinshuu Youkyou*.[25,26]

Maharaja Suraj Mal (CE 1707–1763) or Sujan Singh was a Hindu Jat ruler of Bharatpur Rajasthan, India. He was well known for his political knowledge, steady intellect, and clear vision. He was one of the greatest warriors and ablest statesmen ever born in India. His audacity proved a fierce foe to the Moghuls when they tried to capture Bharatpur in vain. There are ancient paintings depicting the Maharaja together with a goshawk, suggesting his interest in falconry, as with many of the Maharajas in the subcontinent at the time.[27,28]

Drawings of the Sambal tribes in the Philippines recorded in the *Boxer Codex* showed the practice of falconry. The *Boxer Codex* or *Manila Manuscript* is a manuscript written around the 1590s, containing illustrations of ethnic groups in the Philippines, ethnic groups across Southeast Asia, East Asia, and Micronesia at the time of their initial contact with the Spanish conquistadores with additional Taoist mythological deities and demons, and both real and mythological birds and animals copied from popular Chinese texts and books in circulation at the time. The Sambal people are one of the smallest of the Filipino Muslim ethnic groups. They originally came from the Island of Sangihe in Indonesia.[29]

The *Baz-Nama-Yi Nasiri* is an early treatise on Persian falconry compiled by Taymur Mirza. This is a 19th-century manual on the art and sport of hunting with birds of prey. Taymur Mirza was a member of the Qajar dynasty (CE 1785–1925) and grandson of Fath Ali Shah, Shah of Iran. The book classifies the birds used in falconry into two traditional taxonomies: "dark-eyed birds" and "yellow-eyed birds." Examples in the first category are eagles and buzzards, and in the second, various hawks, owls, and osprey. In the book, Taymur Mirza covers the husbandry of raptors, their capture, training, diet, and treatment of diseases, illustrated with examples from his own experience. It has been documented that the first personage to use birds of prey for hunting in the region was Tahmures, the son of Hushang, the third Shah of the Pishdadian dynasty of Persia, placing the birth of falconry in the region to 8000 years to 10,000 years BCE.[19–23]

Moamyn (or Moamin) was the name given in Medieval Europe to an Arabic author of a five-chapter treatise on falconry, important for early Europeans, which was most popular as translated by the Syriac Theodore of Antioch under the title *De Scientia Venandi per Aves* in common era (CE) 1240 to 1241. The treatise also contained a chapter on hunting with dogs and chapters on other related subjects such as diseases of birds. There are about 27 Latin manuscript versions of the work of Moamyn, with two versions highly illustrated throughout. The work of Moamyn is largely based on the *Kitāb al-ṭuyūr*, the Book of Birds or *Kitāb Dawari At-Tayr*, the Book of Flight Cycles (patterns) of Birds, a more extensive work by al-Ghiṭrīf ibn Qudāmah al-Ghassānī from the early ninth century.[7]

The Oghuz, Oguz, or Ghuzz Turks were western Turkic people who spoke the Oghuz languages from the Common branch of the Turkic language family. In the eighth century, they formed a tribal confederation conventionally named the Oghuz Yabgu State in Central Asia. The Oghuz, forefathers of the Turkmen people who lived 5000 years ago, had falconry symbols on the ancestral emblems, carpets, pottery, and in other archaeological findings.[19–23]

FALCONRY IN ASIA—THE PRESENT

Birds of Prey Species Used in Falconry in Asia

Eagles have played a predominant role in the traditional way falconry was practiced by the Kazakhs and Kyrgyz, in modern Kazakhstan and Kyrgyzstan across the steppes of Central Asia, and diasporas in Bayan-Ölgii Province, the west of Mongolia, and Xinjiang Province in northwest China. The most common species used in falconry were the golden eagle *(Aquila chrysaetos)* and the Eurasian goshawk.

There appears to be a great deal of controversy on the etymology of the name bāz as a favorite bird of prey used in falconry in Persian falconry. The definition of bāz in early Persian texts does not allow identification of the species as these refer vaguely to "well-known predatory or hunting species" or "a well-known species widely used by Kings and Dignitaries for hunting." In the Middle Ages, however, the name bāz was distinguished from other species such as the bāsa or sparrowhawk *(Accipiter nisus)*, the šāhān or Barbary falcon *(Falco pelegrinoides)*, or peregrine falcon according to the different authors, the čarğ/čark or saker falcon and the oqāb, traditionally accepted to be the eagle. More precise information on the bāz was provided in the *Nowrūz-nāma*, attributed to the philosopher and mathematician Omar Ḳayyām (first quarter of the 6th to 12th century). "The bāz is of numerous kinds, but the best is the white one and those with plain red or plain yellow plumage. The white one is the greediest for hunting, but it is prone to disease and ill-tempered; the yellow one is both greedy and healthy; the red one is larger and healthier than the other two kinds, but it is ill-tempered." It is now widely accepted that the bāz, as described by early Persian textbooks, corresponds to the Eurasian goshawk.[19–23]

Golden eagle

The golden eagle (Figure 55.10) is a large bird of prey measuring 66 to 102 cm in length and with a wingspan of 1.8 to 2.34 m. The wingspan of golden eagles is the fifth largest among living eagle species. Females are larger than males. In adult birds, males and females have similar plumage and are primarily dark brown, with some gray on the ventral aspect of the wings and tail and a paler golden color on the back of the crown and nape. The Asian golden eagle, Himalayan golden eagle or berkut *(Aquila chrysaetos daphanea)*, is believed to be the most commonly used in the region for falconry. This subspecies is distributed in central Kazakhstan, eastern Iran, and the easternmost Caucasus, distributed to Manchuria and central China and along the Himalayas from northern Pakistan to Bhutan and discontinuing in northeastern Myanmar (rarely ranging over into northernmost India). This subspecies is the



FIGURE 55.10 Golden eagles *(Aquila chrysaetos)* have been used for hunting in Central Asia for centuries. The quarry is mainly the Mongolian wolf *(Canis lupus chanco)* and steppe wolf *(Canis lupus campestris)*, but the tolai hare *(Lepus tolai)* is also hunted to a lesser extent. The Asian golden eagle, also called Himalayan golden eagle or berkut *(Aquila chrysaetos daphanea)*, is believed to be the most commonly used species for falconry in the region. (Photo courtesy Javier Ceballos.)

largest among all the subspecies. Males weigh 4.05 kg, while females weigh 6.35 kg on average.[8]

Eurasian goshawk

The Eurasian goshawk is a medium- to large-sized bird of prey (Figure 55.11). The scientific name in Latin, *Accipiter*, means to receive, and *gentilis* means noble or gentle, since in the Middle Ages, only the nobility was allowed to use goshawks for falconry. The Eurasian goshawk is found across most of Russia, with the exception of the tundra in the northern ranges, to the western limits of Siberia as far as Anadyr and Kamchatka peninsula. There are 9 to 10 subspecies of the Eurasian goshawks described throughout its range. The pale forms are from the boreal zones of the Palearctic. The plumage of the subspecies *A. g. albidus* is polymorphic but generally creamy to white in color. Male of this species are 46 to 61 cm in length and with a wingspan of 89 to 105 cm. The female is much larger, 58 to 69 cm in length, with a wingspan of 108 to 127 cm.[8]

Hunting

In the Kazakh and Kyrgyz languages, there are distinct names for those hunting with eagles and those hunting with other birds of prey. The Kazakh word for falconers who hunt with eagles is "búrkitshy," from "búrkit" (golden eagle), while the name for those who hunt using goshawks is "qarshyghashy," from "qarshygha" (goshawk). In Kyrgyz, the general word for falconers is "münüshkör." A falconer who specifically hunts with eagles is a "bürkütchü," from "bürküt" (golden eagle).

During the communist period, many Kazakhs fled to Mongolia, settling in Bayan-Ölgii Province and bringing with them their tradition of hunting with eagles. There are an estimated 250 eagle hunters in Bayan-Ölgii, which is located in the Altai Mountains of western Mongolia. Their falconry custom involves hunting with golden eagles on horseback, and they primarily hunt red foxes *(Vulpes vulpes)* and corsac foxes



FIGURE 55.11 The use of the Eurasian goshawk *(Accipiter gentilis)* in falconry was very popular in many East Asian countries such as India and Pakistan and in some Middle Eastern countries, notably in Iran. It is now widely accepted that the name "bāz," as described by early Persian textbooks, correspond to the Eurasian goshawk. (Photo courtesy Javier Ceballos.)

(Vulpes corsac). They use eagles to hunt foxes and hares during the cold winter months when it is easier to see the gold-colored foxes against the snow. Each October, Kazakh eagle-hunting customs are displayed at the annual Golden Eagle Festival. Although the Kazakh government has made efforts to lure the practitioners of these Kazakh traditions back to Kazakhstan, most Kazakhs have remained in Mongolia. In Turkmenistan the art of falconry differs from those in the neighboring traditions of using eagles. The practice of falconry in Turkmenistan is similar to the traditional falconry of Iran, Afghanistan, and some countries of the region, using saker falcons and tazy (saluki).[23,30]

In Pakistan, falconry is still practiced but is rapidly declining, with only a small number of falconers remaining in Punjab, Sindh, Balochistan, and Khyber Pakhtunkhwa, formerly known as the North-West Frontier Province. The different birds of prey used in falconry in Pakistan can be divided into birds with red eyes including the shikra *(Accipiter badius)*, sparrowhawk, and goshawk and birds with black eyes including peregrine, saker, and luggar falcons and merlin *(Falco columbarius)*.[31]

In present-day Turkey and Georgia, falconers are only allowed to hunt using sparrowhawks. In Turkey, there are just over 4,000 registered falconers, while in Georgia there are only just over 500 falconers. Georgia was the first state of the former Soviet Union to legalize falconry in 1967.

FALCONRY IN ASIA—THE FUTURE

The decline of the Ottoman Empire marked the decline of falconry in the Middle East, Eastern Europe, and North

Africa. The Ottoman Empire was one of the most powerful and longest-lasting dynasties in world history. Under a treaty agreement, most Ottoman territories were divided between Britain, France, Greece, and Russia. The Ottoman Empire officially ended in 1922 when the title of Ottoman Sultan was eliminated.

To the knowledge of the authors, falconry is banned in India, Jordan, Israel, and Oman. However, in most countries of Asia, falconry is still practiced and enjoyed by a new generation. In many countries of the region, falconry is organized under falconry clubs. However, it is firmly believed that falconry will continue flourishing in the different nations of the Asian region as part of the cultural heritage for future generations.

In 2010, UNESCO declared falconry a living human heritage element of 11 countries, including the UAE, Belgium, Czech Republic, France, Republic of Korea, Mongolia, Morocco, Qatar, Saudi Arabia, Spain, and Syrian Arab Republic. Austria and Hungary were added in 2012, and Germany, Italy, Kazakhstan, Pakistan, and Portugal were added in 2016. With a total of 18 countries, falconry is the largest multinational nomination on the Representative List of the Intangible Cultural Heritage of Humanity.

FALCONRY IN AUSTRALIA

In Australia, all Falconiformes and Strigiformes are protected under regional legislative acts and regulations. As such, it is illegal in all states and territories to kill, injure, take from the wild, hold captive, trade, or band any raptor without a permit issued by the relevant government authority. Permits are mostly granted for scientific, artistic, exhibition, or rehabilitation purposes, or—in rare instances—to scare, remove, or destroy raptors causing an economic nuisance or causing a threat to human safety (e.g., at airports). While the State of Victoria retains a right for citizens to apply for a falconry permit, in practice, such permits are never granted, and falconry is effectively prohibited throughout the country. Any offenses involving a raptor may lead to substantial fines of up to AU $10,0000 or up to 6 months imprisonment.[32]

FALCONRY IN EUROPE—THE PAST

A day without falconry is a wasted day.
Emperor Frederick II (CE 1194–1250)

First reports about a collaboration between men and birds of prey in Europe were given by Aristotle in antiquity, around 350 BCE, who described fowling in the marshes of Thrace with the aid of raptors.[33] He, and later Pliny, reported on men using sticks to put up little birds from bushes and reeds and falcons soaring above them to drive these birds down to the ground. In these descriptions, the fowlers caught the birds using nets and sticks and shared their prey with the falcons in a kind of hunting partnership.[33,34] However, there are no detailed descriptions of human beings training and keeping raptors for falconry until CE fourth to fifth century. Falconry was brought to Europe from Asia by migrating Gothic tribes and the nomadic Khazars of the northern Caucasus. It expanded westernmost during the reigns of the Visigoth kings Alaric I (CE 370–410), Theodoric I (CE 390–451), and Euric (CE 440–484).[34] In those days, falconry was common and practiced by large parts of the populace as an efficient way of hunting to catch nourishment for human consumption. This has been documented by the author Paulinus of Pella (CE 376–459) in his autobiographical poem entitled "Eucharisticos" (Greek for "Thanksgiving").[34] Likewise, the French bishop Sidonius Apollinaris described Emperor Avitus (CE 385–457) practicing falconry and implementing it as part of the education of young gentlemen, for it was thought to prevent illness and to demand the cultivation of personal qualities such as skill, education, and patience.[6] Besides written evidence, early pictorial testimonials of falconry were found in different parts of Europe. In the south, at Argos in Greece, a floor mosaic in the Roman "Villa of the Falconer" representing a falconry scene dates back to CE 500.[33] In Georgia and the Republic of Tuva, stone sculptures from the CE fifth and sixth centuries represent early evidences of falconry in the east.[34] In European literature of these centuries falconry has been mentioned in CE 627 to 630 by Archbishop Isidore of Seville (CE 560–636), who included a description of a falcon perching on a falconer's hand in his work *Etymologiae* (Latin for "the etymologies" or "the origins").[34]

Gradually falconry in Europe transformed from a popular way of hunting for everybody to a privilege of nobility, clergy, and people of a certain social status. Thus birds of prey were included into the Salic Law (CE fifth century), the Burgundian Law, and the Ripuarian Law (CE sixth century), and the larceny of falconry birds was punished.[33,34] In particular, the Burgundian Law covenanted that the thief of a hunting bird should get 6 ounces of flesh torn from his breast by the raptor he had stolen.[33,35] In contrast to commonality, emperors, kings, princes, and high officials of the church did not practice falconry for sustenance but for pastimes, competition, and sport. Hence, at the royal courts of Europe the quarry transformed subsequently from partridges *(Perdix perdix)* and other animals for gourmet cuisine to grey herons *(Ardea cinerea)*, common cranes *(Grus grus)*, carrion crows *(Corvus corone)*, red kites *(Milvus milvus)*, and black kites *(Milvus migrans)*, offering more spectacular and prestigious hunting events. In the ninth century in some regions privileges of aristocracy were expanded beyond death, when it was set as the right and honor of a gentleman to keep his sword and his goshawk after falling in a battle.[34] Physicians such as the magister Ububchasim de Baldach from Baghdad, who wrote the Arab medical treatise *Theatrum sanitatis* (Latin for "theater of health"), recommended in the 11th century to practice hunting and falconry in order to maintain health and fitness. At the same time, falconry should offer the greatest amount of pleasure and allow man to live in harmony with nature.[34]

Among the European monarchs, the Hohenstaufen dynasty became famous for their dedication to falconry. In the 12th century, Emperor Barbarossa (Frederick I; CE 1122–1190) was the first of the Hohenstaufen who practiced falconry by training dogs, horses, and numerous falcons. This passion was adopted by his son Emperor Henry VI (CE 1165–1167). However, the grandson of Barbarossa, Emperor Frederick II of Hohenstaufen (CE 1194–1250), should become famous as the greatest falconer of his age.[33,34] Growing up in Sicily, he was fascinated by nature, life sciences, and ornithology and appreciated the lanner falcon as his favorite bird of prey species of his home region. It is recorded that he

practiced falconry as often as possible, besides royal duties and even on the battlefield. He enjoyed hunting cranes with gyrfalcons and herons with peregrine falcons and employed Arab, German, Spanish, and English falconers at his court.[6,36] Frederick II is alleged to have said that a day without falconry should be considered to be a wasted day.[34] Following this maxim, he managed to achieve the writing of a vast and extraordinary treatise about hunting with birds of prey, published under the original title *De arte venandi cum avibus* (Latin for "from the art of hunting with birds").[33] For this purpose, he consulted falconers all around the globe to exchange experiences in order to distill the knowledge, techniques, and technologies of falconry of different cultures and geographic regions. Being able to speak nine languages including Arabic, he was able to communicate with falconers from the Middle East. Thereby he discovered the hood for falcons during a Crusade to Jerusalem (CE 1228–1230).[34] This literary oeuvre took him nearly 30 years until CE 1247 and still is a milestone in falconry literature. In total, more than 140 treatises were written in medieval Europe about falconry between the 10th and 16th centuries in Latin, Italian, Portuguese, Spanish, Catalan, Dutch, German, French, and Occitan, all offering a variety of experiences, techniques, and practical tips.[34] For example, in France falconry has been documented by Charles D'Arcussia in *La fauconnerie* (French for "the falconry") (CE 1589) and in Portugal by Diogo Fernandes Ferreira in *Arte da caça de altanería* (Portuguese for "the hunting art of falconry") (CE 1616).[34]

The demand and value of raptors increased from the early Middle Ages to the Renaissance. Birds of prey, and first of all falcons, were caught from the wild during their migration on the main passage route in Flanders, Belgium, and the Netherlands.[34,35] The small Dutch town Valkenswaard in Brabant (Dutch for "wait or look out for falcons") is a still existing testimony of the involvement of professionals and entire families in service of the courts, handing down the skills of catching falcons from generation to generation. Likewise, the monarchs of northern European countries, especially the king of Denmark, who was also for a long time ruler of Norway and Iceland, caught fledged gyrfalcons and goshawks or took them from nests and distributed them by traders across the continent.[33] They were regarded as desirable gifts between kings and princes, able to solve political discrepancies or to consolidate liaisons. Thus more than 50 different courts received these special presents. For example, in CE 1764 it is recorded that a total number of 162 falcons went to the French King ($n = 50$), the German Emperor ($n = 30$), the King of Portugal ($n = 60$), the Landgrave of Hesse ($n = 20$), and to the French Ambassador ($n = 2$). Besides European sources, European conquistadors requested exotic raptors from regions in the New World after the discovery and colonization of the Americas. Thus in CE 1492 Christopher Columbus was requested by the monarchs to send as many exotic falcons as possible to the Spanish court, and likewise, Hernán Cortés (CE 1485–1547), the Councilor of the Indies, agreed to send annually 50 hunting birds to continental Europe.[34] He had witnessed in Mexico that Aztec King Montezuma previously kept and maintained a large collection of raptors, possibly used for hunting (see also section on Falconry in Mexico), although this is debatable.[33] Another example of the high value as a diplomatic gift is the granting to nobles of the island of Malta and Tunisia by Charles the V of Germany in return for giving him one white gyrfalcon.[35] Falconry with its symbolic system was shared between most cultures and its geographical reach was extraordinary in the 17th century. Falcon trading ranged from France to Russia, from Norway to Sicily, toward Corsica, Sardinia, Balearic Islands, and across Turkey back to Asia and North Africa.

Louis XIII the Just (CE 1601–1643), King of France and Navarre, who became king shortly before his ninth birthday, seemed to be more interested in falconry than in political business and spent large amounts of time and money for this noble sport.[34] His falconry consisted of 300 birds, subdivided into six specialized equipages for hunting herons, kites, crows, waterfowl, and partridges.[6] In Germany, the Margrave Charles William Frederick of Brandenburg-Ansbach (CE 1712–1757) was one of the well-known and dedicated falconers of the 18th century. He ruled as a typical absolute monarch with a luxurious court life and extensive hunting and falconry, which earned him his nickname, the Wild Margrave. He spent at least 10% of the annual national budget on falconry, employing 43 falconers divided into heron, kite, crow, and rook hawking groups.[36] It is reported that 35,000 creatures were hunted by him and his entourage within 20 years. Additionally, Clemens August of Bavaria (CE 1700–1761), member of the Wittelsbach dynasty of Bavaria and Archbishop-elector of Cologne, assiduously practiced falconry and patronized the arts.[34] In this regard, he ordered the building of the extraordinary and quixotic palaces of Augustusburg and Falkenlust (German translation of "passion for falcons") in Brühl, North Rhine-Westphalia, both listed on the UNESCO cultural world heritage list as significant artistic representations of falconry and hunting.[34]

Two main factors led to a rapid cessation of falconry in Central Europe in the late 18th century. Firstly, the French Revolution (1789) and the associated social changes during the Age of Enlightenment/Reason led to the rejection and prohibition of everything that has been associated with the aristocracy.[34] Additionally, the development, optimization, and increased availability of firearms made raptors as hunting partners superfluous. In contrast, in these times, goshawks and other birds of prey were regarded as competitors and became victims of human hunting. Thus falconry almost faded away in the early 19th century in the entire continental Europe,[35] except a shortly expanded preservation in Holland under the patronage of Prince Alexander. There, the creation of the short-lived Loo Hawking Club (1839–1855) enabled falconers from England and Holland to join high flights in the heath around to the Palace of Het Loo in Apeldoorn until the end of the 19th century.[34]

At that time, hunting law in Germany changed fundamentally, as after the failure of the German revolution in the spring of 1848, hunting was no longer regarded as regalian right but linked to property instead. Thus in the 19th century hunting became legal for all citizens owning a certain minimum area of a hunting district.[37] Extensive, crude, and unsustainable forms of hunting broke through, calling for the implementation of a hunting code of ethics, and hence, for consideration of the notion of animal protection. In these discussions, falconry was regarded as a particularly ethical and fair method of hunting, being not focused on large hunting bags and financial revenues.[37] To "reanimate" the idea of falconry, exchanges with falconers from Great Britain and

Ireland during the First International Hunting Exhibition in Kleve in 1881 and later for various presentations in eastern Germany during the autumn of 1904 were important stimuli.[37] Some years later in 1921 the German Falcon Order (Deutscher Falkenorden [DFO]) was formed in Berlin as the first falconer association in Europe, being still the oldest and one of the largest falconer associations in the world.[6] This foundation was made by people such as the physician Dr. Friedrich Jungklaus (1875–1953), the art student Renz Waller (1895–1979), Dr. Fritz Engelmann (1874–1935), Dr. Hermann Kreyenborg (1889–1963), and ornithology Professor Johannes Thienemann (1863–1938), who were interested in the relationship between humans and animals, nature, ornithology, and art of falconry.[37] Following the German example, falconry associations were created in multiple European countries including Austria, Belgium, Bulgaria, Czech Republic, Denmark, Estonia, France, Georgia, Greece, Hungary, Ireland, Italy, Lithuania, North Macedonia, Malta, Norway, Poland, Portugal, Romania, Russia, Serbia, Slovakia, Slovenia, Spain, Sweden, Switzerland, the Netherlands, Turkey, Ukraine, and the UK. Currently most of these national falconry associations are connected under the umbrella of the IAF and Conservation of Birds of Prey, which now consists of 110 associations from 87 countries totaling 75,000 members.[6]

Besides practicing falconry, the main goals of the associations in the 20th century consisted in the protection of birds of prey. This latter has been promoted by the dramatic decline of raptors due to environmental pollutants (especially the organochlorine pesticide dichlorodiphenyltrichloroethane [DDT]) in large parts of the world during the 1960s and 1970s.[37,38] This led to attempts at captive breeding of birds of prey, which were successfully achieved in peregrine falcons in 1942/1943 by Renz Waller (Germany) for the first time. As a result of international veterinary and biological research and collaboration between scientists and falconers, large steps in captive propagation were taken in the 1970s by development and improvement of assisted breeding using artificial incubators, optimized aviary design, and the implementation of semen collection and AI. At that time, in Germany systematic breeding programs were initiated for the dramatically endangered peregrine falcon and the white-tailed eagle *(Haliaeetus albicilla)*. As a remarkable achievement, in the following four decades DFO members bred more than 2,500 peregrine falcons, of which more than 1,100 have been released into the wild via reintroduction programs to reestablish the almost extinct wild population.[37]

FALCONRY IN EUROPE—THE PRESENT

In continental Europe, various ways and fashions of falconry are practiced, defined as hunting of wild quarry in its natural habitat and state with a trained raptor. A valid falconry license after passing hunting and falconry examinations is required by law to practice falconry and to keep raptors in several European countries. Most falconers keep and train one or two raptors for hunting. The birds of prey species used for falconry include falcons for so-called "high flights" and buzzards, hawks, and eagles for so-called "low flights."

The most common and traditionally trained falcon species in several European countries is the peregrine falcon, followed by the gyrfalcon and the saker falcon. Less common is the use of lugger or lanner falcons for falconry. Hybrids obtained from cross breeding of falcon species were popular in the last decades, bred in large numbers because of the high international demand, mainly from the Middle East, but are decreasing due to prohibition of keeping and breeding most hybrid birds of prey in several European countries such as Germany. Merlins, prairie falcons *(Falco mexicanus)*, and aplomado falcons *(Falco femoralis)* are also rarely kept as hunting birds, most likely because of their limited availability due to a low number of breeders within Europe and limited import numbers. Moreover, most songbird species (e.g., different passerines such as thrush [*Turdus* spp.], starling *[Sturnus vulgaris]*, or lark [Alaudidae]), which are the usual prey of merlins and aplomado falcons, are not legalized for hunting and are protected by nature conservation law in many European countries. The previously mentioned larger falcon species are appointed to hunt upland game such as pheasants, quails, partridge, grouse, and waterfowl such as ducks. Facing a steady decline of these traditional game species in many European countries, there has been an increase in numbers of carrion crows, rooks *(Corvus frugilegus)*, Eurasian magpies *(Pica pica)*, gulls (*Larus* spp. and *Chroicocephalus ridibundus*), and smaller geese (e.g., *Alopochen aegyptiaca* and *Anser anser*) as preferred quarry for European falconers. Releasing falcons from the falconer's glove to catch prey during walking or riding is not very common anymore except in a few regions of the continent. Especially for the hunting of carrion crows, releasing the falcon from the window or roof of a moving car is more established. In contrast to gun shooting, hunting with birds of prey out of a vehicle is allowed in several European countries as long as the falconer adheres to driving laws. The falcon should follow the crow, propelling itself forward fast and catch it in pursuit flight. Alternatively, the falcon may drive the crows into a tree and hem them there. The falconer's duty is to assist his falcon by frightening the crows through shouting and making noise using rattles/flappers and thus to make them flee away from the tree. The experienced falcon will wait-on in favorable altitude ready to stoop down to the fleeing crows and catch them during nosedive or pursuit flight. This more modern way of hunting with falcons needs to be different from the traditional waiting-on techniques to hunt game such as pheasant, quail, or grouse. For this purpose, a well-trained hunting dog is crucial. Pointing dogs such as setter and pointer breeds from Germany (e.g., Munsterlander, Pudelpointer, German Shorthaired Pointer, German Longhaired Pointer, or Weimaraner), France (e.g., Epagneul Breton/French Brittany), Great Britain (e.g., English Pointer, Gordon Setter, English Setter, or Irish Setter), or Hungary (e.g., Vizsla) are widely distributed among falconers in Europe. The dogs need to be trained to freeze on point as soon as they scent the game and only to flush it out upon the falconer's signal. The signal is given when the waiting-on falcon manages to reach a suitable altitude and a promising hunting position. The falcon performs a stunning downward stoop with the wings folded tight to its body in teardrop-like form, reaching a high velocity when the game takes flight and finally catching it in a cloud of feathers—a moment which is fascinating for everyone witnessing. As this way of hunting requires perfect training of the hunting dog, perfect training of the falcon, and cautious familiarization of both animals to form a winning team with the falconer, bird of prey, and

dog, this is legitimately regarded as the supreme discipline of falconry.

With hawks and buzzards, smaller-sized mammals (e.g., rabbits, hares, and in some countries, squirrels [*Sciurus* spp.]) and birds (e.g., carrion crows, pheasants, ducks, moorhens, and small geese) are caught. An exception because of its small size and its prey (only small birds to the size of magpies, doves, or pigeons) is the sparrowhawk. The so-called "spar" has always been a popular and fascinating hunting bird in Europe. Training and keeping of the tiny, but brave and acrobatic spars require much experience because weight management is particularly difficult in this small-sized raptor. Due to nature protection laws in many European countries, the hunting quarry of sparrowhawks is strictly limited to a few common bird species such as magpies and wood pigeons *(Columba palumbus)*. Because of this, in some countries, the keeping of a sparrowhawk is currently not permitted; however, in Germany its use for falconry has been legalized since June 2018.

The most common hunting bird in Europe for centuries is the Eurasian goshawk, even though North American hawk species, especially the Harris's hawk, but also the red-tailed hawk *(Buteo jamaicensis)*, are flown in increasing numbers by European falconers. Emperor Frederick II called the goshawk a perfect bird for the kitchen, able to hunt everything that is needed for a delicious meal. With its dark red eyes, the goshawk is a formidable creature, fast and skilled to kill all prey of appropriate size. Other medium-sized species seen occasionally as hunting birds include ferruginous hawks *(Buteo regalis)* and long-legged buzzards *(Buteo rufinus)*. Hunting carrion crows with the broad-winged birds out of vehicles is usually much shorter in distance compared to the hunt with falcons. In contrast to falcons, the quarry is more diverse, including medium-sized birds and mammals. Hunting usually is performed directly off the falconer's glove while walking or from a tree or an elevated perch while having the birds follow-on and lead. The prey is scared up by the falconer or assisting persons out of bushes, grasses, or reeds, but preferably by flushing dogs (Figure 55.12). The attentive hawks are swift to react and may reach incredible acceleration to catch up rapidly to fleeing prey. In Europe the use of German (e.g., German Wirehaired Pointer, German Shorthair Pointer, German Longhaired Pointer, Pudelpointer, Weimaraner, and Munsterlander), French (e.g., Epagneul Breton/French Brittany), English (e.g., English Setter), Italian (e.g., Bracco Italiano), or Hungarian (e.g., Vizsla) flushing dog breeds is popular. If hawks and dogs are familiarized, they may form an unbeatable team (Figure 55.13). For hunting rabbits *(Oryctolagus cuniculus)* ferreting is also an established technique in Europe. Tamed ferrets *(Mustela putorius furo)* are inserted into the burrows to scare up the rabbits and make them escape to the surface, where the hawks are able to catch them. Small bells are attached to the ferret using collars to increase the rousing efficacy and to prevent the ferret from catching the rabbits by itself. Efficacy may be increased by using a dog to indicate which burrows are containing rabbits and offering more promising hunting chances.

Eagles are also commonly used in European falconry; however, their number is low compared to hawks and falcons. By far the most popular eagle species is the golden eagle, as it inhabits several mountain regions within Europe and has been used as a heraldic symbol of strength and power. Thus the golden eagle is the national symbol of several states such as Austria, Poland, Czech Republic, Russia, and Germany (even though the white-tailed eagle is most likely the real model for the heraldic symbol of Germany). In these countries, hunting with golden eagles is regularly practiced to hunt for hare (Figure 55.14), red fox, and roe deer *(Capreolus capreolus)*. Besides the golden eagle, African hawk eagles *(Aquila spilogaster)*, Bonelli's eagle *(Aquila fasciata)*, and Chilean blue eagles *(Geranoaetus melanoleucus)* are less common. The ornate hawk-eagle *(Spizaetus ornatus)*, African crowned eagle *(Stephanoaetus coronatus)*, and martial eagle *(Polemaetus bellicosus)* are rarely seen as hunting birds in Europe. Eagles are usually started from the fist during walking or more seldom riding in open fields. Prey is flushed out by the falconer or assisting persons or by flushing dogs as mentioned previously. Hunting flights



FIGURE 55.12 Hunting with goshawks or broadwings is usually performed directly off the falconer's glove while walking. The prey is scared up by the falconer or by flushing dogs out of bushes, grasses, or reeds. Here a Harris's hawk *(Parabuteo unicinctus)* and a Hungarian Vizsla support the falconer. (Photo courtesy Claas Niehues.)



FIGURE 55.13 Hawk, flushing dog, and falconer form an unbeatable team during hawking. Here the Eurasian goshawk *(Accipiter gentilis)* is taken back to the falconer's fist and rewarded by offering a pigeon wing after successfully hunting a rabbit that has been flushed out by the Hungarian Vizsla. (Photo courtesy Eva Casteel.)



FIGURE 55.14 Golden eagles *(Aquila chrysaetos)* in Europe are trained to hunt for hare, red fox, and roe deer. Here, a golden eagle caught a European brown hare *(Lepus europaeus)* and is still full of adrenaline after the hunt. (Photo courtesy Gerhard Bühler.)

of the majestic eagles are sometimes long, but powerful and formidable.

The main hunting season in Europe is during autumn, winter, and spring (usually September–March). During this time falconers go for hunting, either alone with their animals or in small groups of a few falconers. These hunts are also an integral part of falconer meetings such as the annual meetings of the national falconer associations. Except for Harris's hawks, which may be used to fly and catch as a pack, in a hunting group a previously agreed order of flights and self-discipline of the participating falconers are very important to ensure that only one bird is flying toward the quarry at any one time.[39] Otherwise one bird may injure or kill the other bird, which must be prevented. It is an unwritten ethical rule that a falconer generally should not hunt more than three creatures per hunting day to not overwhelm the hunting bird and the capacity of the habitat. Traditionally, in some countries, especially in Germany, France, and Austria, hunting-horn music is played to greet falconers and their animals when they arrive at the meets, but also when they depart to the field to hunt, and to honor the prey as it is laid out by firelight or torch at the end of the hunting day (Figure 55.15). Usually, talks, workshops, and a celebratory feast are also included in these annual meetings.

Nowadays, falconry in Europe is mainly practiced as a hobby. Only a minority of falconers are employed as professional falconers. Pest control companies (e.g., for the food industry), urban parks, sport facilities, and cemeteries, for the protection of dykes, historic buildings, or agricultural crops are some of the working tasks for professional falconers. Moreover, falconers are often in charge of bird control at airports to reduce the risk of bird collisions with airplanes. Besides this, there are multiple areas where the use of guns is prohibited or inappropriate in most European countries. At these places, falconry offers an efficient, quiet, and well-accepted alternative for hunting. Professional falconers are also employed at places with falconry displays such as wildlife parks, zoological institutions, and falconry centers. Even though "real falconry" in the sense of hunting is not



FIGURE 55.15 To honor the prey, it is laid out by firelight or torch at the end of the hunting day. Traditionally, hunting-horn music is played during this celebration in Germany, France, and Austria. (Photo courtesy Dominik Fischer.)

performed and not demonstrated to the public, methods, techniques, and equipment are used according to falconry to train and keep raptors. In several European countries, mainly Austria, France, Spain, and Germany, falconry displays are established in historic castles addressing the history and the romantic connection of knights, kings, and falconers in medieval times.[36] The history of bird of prey centers in Europe dates back to 1939 when the "Adlerwarte Berlebeck" (German for "eagle center Berlebeck") was opened to the public in Germany. This was followed by wildlife parks such as the "Wildfreigehege Hellenthal" (German for "Wildlife Park Hellenthal"), where falconry displays were first established as an integral part of zoological institutions. Nowadays, these institutions still play an important role in environmental and biological education (Figure 55.16). They offer a unique



FIGURE 55.16 Falconry displays are established at historic castles or in zoological institutions. They still play an important role in environmental and biological education and offer a unique option to transmit information about falconry, birds of prey, their current situation and threats, as well as options to protect them as part of the environment. Here a black-chested buzzard-eagle (*Geranoaetus melanoleucus*) is landing on the falconer's fist at Bird of Prey Center in Hellenthal, Germany, in front of the public and a cinereous vulture *(Aegypius monachus)*. (Photo courtesy Hermann-Josef Baumgarten.)

option to convey information about falconry, birds of prey, their current situation, and threats, as well as options to protect them as part of the environment. Therefore these institutions play an important role for falconry and birds of prey and manage to increase public acceptance and fascination for these unique and extraordinary creatures.

Captive breeding of birds of prey is practiced by European falconers, as taking birds from nature is strictly limited or completely prohibited and CITES regulations have been implemented since the late 1970s in European countries. Most falconers breed small numbers, with the offspring being intended to be given to falconer colleagues. In addition, there are a few large commercial breeders, especially of large falcons and goshawks, who sell the offspring internationally to all parts of the world. Large breeding facilities exist, for example, in Germany, Austria, and Spain, regularly breeding falcons for the Middle East. However, some raptor species are bred not commercially and donated for reintroduction projects by falconers. After the successful reintroduction of peregrine falcons in Sweden and Germany, Polish falconers are now dedicated to the reintroduction of this species in Poland and to the restoration of a tree-nesting population. Other reintroduction programs in Europe include eagles (e.g., white-tailed eagles), vultures (e.g., bearded vultures *[Gypaetus barbatus]*), and owls (e.g., Ural owls *[Strix uralensis]*). In these projects, falconers cooperate with zoos, wildlife parks, and breeding centers. Moreover, European falconers from Germany, Belgium, Spain, Austria, and the UK cooperate in international species conservation projects such as the "Project Lugger," an IAF-supported initiative to save the Asian lugger falcon (http://projectlugger.com/).

As falconers are experienced in keeping, feeding, nursing, treating, and training birds of prey, they are regularly involved in veterinary-guided rehabilitation of sick or injured raptors. In some countries, the national falconer associations run their own rehabilitation centers for this purpose. After successful medical treatment of a bird of prey, falconers are in charge of subsequent individual physiotherapy and species-specific training using falconry methods as important preparation for releasing these birds to the wild.[40] Some European falconer associations support scientific studies to optimize falconry training methods and to evaluate the true benefits of these measures for the rehabilitation of birds of prey. Currently, the veterinary knowledge that falconer-veterinarians have accumulated and published provides the basis for treatment and rehabilitation of many injured wild raptors. Furthermore, falconers contribute widely to knowledge of wild raptor ecology and management in Europe.

National initiatives for rehabilitation, species conservation, or animal welfare are coordinated and supported within Europe by the European Foundation for Falconry and Conservation (EFFC) (https://www.ef-fc.org/) or internationally by the IAF. In recognition of the intensive commitment to nature conservation in general and to birds of prey in particular, some European falconer associations, such as the DFO, have been recognized as nature conservation associations by national governments. Moreover, following the inclusion in UNESCO's Representative List of the Intangible Cultural Heritage of Humanity, falconry has been included as a living human heritage in the nationwide directory of Intangible Cultural Heritage in several European countries (as mentioned earlier: Austria, Belgium, Czech Republic, France, Germany, Hungary, Italy, Portugal, and Spain).

FALCONRY IN EUROPE—THE FUTURE

Future falconry in Europe will need to keep on focusing on conservation, animal welfare, culture, and education to be preserved as a sustainable, sophisticated, silent, and most natural art of hunting and as an incomparable, respectful, and passionate relationship to magnificent and wild living creatures.

Falconers need to continue to develop knowledge about husbandry, training and nutrition, reproduction, health, and disease in raptors and animal welfare using a strong and international cooperation with veterinarians and scientists from different disciplines. Facing large declines in both raptor species and game species, conservation will need to focus on a holistic approach involving hunters, falconers, scientists, nature conservationists, farmers, foresters, politicians, and the public to collaborate toward the common goal of environmental protection and biodiversity. Initiatives such as the IAF-supported conservation of free-ranging partridge populations ("Perdix Project") are one bullet point on the to-do list. Improvement of habitat structure, increase of food availability, and reduction of disturbances and stressors are important steps to reach this goal. Besides these measures, efforts for the individual rehabilitation of injured or sick raptors will also be an important task of future falconers in Europe.

Existing conflicts with civilization and human development (e.g., through farming and forestry, construction, pollution, disturbance, and climate change) are difficult tasks to solve in Europe. Falconry offers a unique method for sustainable use of natural resources and an ethically acceptable means of meat production by using a natural method of predation with a negligible risk of prey escaping with deadly injuries (compared to shooting) and offering a faithful alternative to intensive livestock farming. Hopefully in the future, there will still be a demand for such sustainable and ecological use of wildlife as indicated by a promising tendency in European society during recent years.

After banning several pesticides with toxic potential in the past, a current and future task is the refinement and optimization of energy sources, such as electricity transmission lines (power lines) and power plants (wind turbines) in order to reduce mortality in bird of prey populations. In this regard, the discrepancy between environmental protection attempting to decelerate climate change and species conservation is problematic. Other conflicts addressing the economy, such as the reduction of pesticides in agriculture or the use of drugs in livestock (e.g., use and deposition of the cheap but dangerous diclofenac), will require continuing attention and constant lobbying. Lobbying, transparency, and public education will also be required to positively influence legislation. To achieve reasonable legislation and guidelines on all aspects of falconry, falconers will need to be confident, but self-critical in a continuous dialogue with different stakeholders. The permission of falconry in Scandinavia will be a stage finished in the short term in Europe; this was achieved in Denmark in 2019 and will hopefully be achieved in Finland and Norway soon. Most likely, in future, falconry will meet with resistance from animal rights initiatives, which aim to terminate keeping of animals, especially of exotic and "wild" animals and try to abolish hunting. To defend falconry from

these threats, public relations activities, publications, and environmental education are most important. In a society that has drifted away from nature and the environment, this is now already crucial. However, facing future generations that did not learn to live with animals and did not get used to food production in terms of agriculture and hunting, this will be even more important to be accepted by society. In this regard, it will be important that falconers are honest and transparent, keep on explaining falconry, and always endeavor to strike the right note. Falconry has been practiced in Europe for over a millennium, but it will be a tough task handing the art and tradition of falconry down to future generations.

FALCONRY IN THE UK—THE PAST

> *As the ox hath his bow, sir, the horse his curb, and the falcon her bells, so man hath his desires.*
>
> **Shakespeare,** *As You Like It*, **Act 3, Scene 3**

Falconry—defined as the art of hunting with a trained bird of prey—is very much now considered a sporting activity in the UK. Historically a hunter would train a bird as a way of obtaining food, and food acquisition was almost certainly the origins of falconry. Historic artifacts and archaeological evidence links falconry in the UK as far back as the late sixth and early seventh century.[41] While falconry was likely practiced among all classes of people living in the UK, it is the medieval kings and nobles whose falconry pursuits we have the greatest documentary evidence. In the eighth century extracts from Cook record Aethelbald, King of Mercia, being sent a hawk and two falcons by Boniface, Archbishop of Mayence. Harold, Earl of Wessex and later King of England, is depicted with hawks on the fist in the Bayeux Tapestry dating from the 11th century. It was common practice for armies to take hawk and hounds on campaigns so knights could relax between battles.[33] A famous and frequently reproduced list of birds that could be flown according to social standing is shown in Table 55.2. Despite this list being republished frequently, there is little evidence of it being strictly adhered to, although there is probably some accuracy in the fact the rarer and more exotic raptors were only available to those of sufficient social standing.

In more recent history, falconry has become increasingly accessible. Falconry clubs traditionally provided a route into the sport and the British Falconers' Club was founded in 1927, by the surviving members of the Old Hawking Club (itself founded in 1864). The Club is the oldest and largest falconry club in the UK. A large number of smaller regional hawking/falconry clubs exist across the UK.

Falconry centers have become common across the British Isles, with the first opening in Gloucestershire in 1967. Originally termed The Falconry Centre and now named the International Centre for Birds of Prey (ICBP), it was opened by Phillip Glazier, a preeminent falconer of the time. Over the last 50 years, these falconry centers have exposed great numbers of the public to birds of prey and have helped build the popularity of falconry among the general public. Centers such as ICBP have also contributed to raptor conservation worldwide and had some of the earliest success in captive breeding.

FALCONRY IN THE UK—THE PRESENT

Rough estimates put the number of bird of prey keepers in the UK at between 15,000 and 25,000. Unlike some countries such as the United States, no license system currently exists for falconry. Many of these owners will, however, not participate in falconry in its truest definition—hunting naturalized quarry with a trained bird of prey. A number of other falconry-related activities have developed—these include display falconry, lure flying, ro-falconry, and pest control.

The number of species used in falconry in the UK is incredibly varied. Until 2020, no licenses for the wild-take of birds had been granted for several decades, and UK falconers were entirely reliant on captive breeding to provide birds for use in falconry. The justification for recent licenses was to take wild birds to ensure a healthy gene pool of birds for use in falconry.

TABLE 55.2

A Famous and Frequently Reproduced List of Birds That Could Be Flown According to Social Standing

Social Status	Raptor Flown According to Social Standing
Emperor	The eagle, vulture, and merloun
King	The ger falcon and the tercel of the ger falcon
Prince	The falcon gentle and the tercel gentle (peregrine)
Duke	The falcon of the loch (peregrine)
Earl	The falcon peregrine
Baron	The bustard (harrier?)
Knight	The sacre and the sacret
Esquire	The lanere and the laneret
Lady	The marlyon (merlin)
Young man	The hobby
Yeoman	The goshawk
Poor man	The jercel (kestrel)
Priest	The sparrowhawk
Holy water clerk	The musket
Knave or servant	The kestrel

Primarily two rearing methods are used to raise young birds—imprinting (hand-rearing) or parent-rearing. There has certainly been an increase of imprint birds being flown in recent years in the UK. Falcons, accipiters (true hawks), broadwings, and eagles are all flown. For traditional falconry, the bird selected is typically matched to the surrounding land to which the falconer has access to hunt. The UK has varied landscapes—farmland, 56%, and natural landscapes (forestry, heather moorland, grassland), 34.5%. For example, flying majestic peregrine falcons in closed country just will not work effectively, with accipiters being far more suitable in these situations.

Broadwings

Harris's hawk

By far the most popular bird in UK falconry, the Harris's hawk was originally imported from its natural range of South and Central America and southern United States in the 20th century. Its biddable nature, relative ease of training compared to more highly strung birds such as accipiters, and social behavior have made them incredibly popular. They are versatile hawks and have quickly overtaken the common buzzard *(Buteo buteo)* as the broadwing bird most commonly used in UK falconry. Their nature has also made them excellent birds when used in pest control and display falconry.

Red-tailed hawk

Another North American species, the red-tailed hawk, is a larger and more powerful hunter than the common buzzard and has a modest popularity in UK falconry. Hybrid birds are fairly common, with ferruginous hawk × red-tail hybrids being particularly popular—with flight similar to a small eagle, they are versatile hunters.

Other broadwings commonly flown include the ferruginous hawk and common buzzard.

Falcons

See section on Falconry in the Middle East—The Present.

Accipiters—Shortwings

These are primarily woodland birds and incredibly agile. Traditionally considered highly strung, modern imprinting techniques and rearing methods have been very effective when training accipiters.

Goshawk

The goshawk is the most frequently flown shortwing in UK falconry and is the classic accipiter. These are great all-round hawks, traditionally named the "cook's bird" due to their versatile nature as a hunter and ability at catching game.

European sparrowhawk

One of the most exciting birds in falconry—they are fast and without fear—often taking game far larger than themselves. They prey on a wide range of small- or medium-sized birds. Their preferred quarry—the common blackbird *(Turdus merula)*—requires a license to hunt in the UK. Sparrowhawks have high metabolic rates and careful management from the falconer is required to keep them in ultimate condition. Imprinting has greatly improved the success when hawking sparrowhawks in the UK and almost all birds now flown are hand-reared.

Other accipiters occasionally used in UK falconry include the Cooper's hawk *(Accipiter cooperi)*, black sparrowhawk *(Accipiter melanoleucus)*, and African goshawk *(Accipiter tachiro)*.

Eagles

Eagle falconry is a very specialist subsection of UK falconry. The iconic golden eagle is the most prevalent eagle in UK falconry and being a native species is best adapted to the cold weather and frequent high winds during the traditional falconry season. Typically hunting small- to medium-sized mammals, eagles can fly in both a pursuit and waiting-on style, offering contrasting but equally spectacular flying.

Training Methods

Lure flying

In the UK, the traditional method of training a falcon was to the lure. The lure—typically constructed of a leather pad—sometimes with the addition of wings, simulates prey, which the falcon must hunt. The falconer swings the lure to simulate an "evade and capture" scenario. Training in this method has the potential to become somewhat one-dimensional. As well as being used as part of fitness training, a lure can also be used to attract a falcon or hawk to be reclaimed onto the fist.

Robotic prey

In the last 5 years, simulated hunting scenarios with robotic prey have become increasingly used to train birds. Planes designed to replicate commonly flown species constructed of polystyrene are flown. The planes have internal propellers to prevent injury and allow the falcon or hawk to strike the model in the air—as opposed to planes pulling a lure, which were traditionally used. These models are increasingly used in raptor rehabilitation and have shown promise with building fitness and hunting ability for injured raptors—particularly eyass birds with limited or no hunting experience (Figure 55.17).

For the imitation of terrestrial prey species, rabbit, fox, and hare lures dragged on a pulley system are used mainly by hawk and eagle falconers. Multiple flights in a short space of time build fitness and teach birds to be observant and reactive. Flights including uphill gradients are particularly beneficial at building strength and stamina.



FIGURE 55.17 Hybrid falcon binding to a robotic houbara model during training.



FIGURE 55.18 Gyr saker falcon pursuing a robotic crow model during a race competition.

Modern machines are remote-controlled and can be very portable to allow the replication of various different hunting simulations. In comparison to traditional training this is far more mentally stimulating—particularly for accipiters and can lead to improved temperament (Figure 55.18).

Drones

To help encourage inexperienced falcons to obtain a suitable pitch (height held above the falconer before the stoop), drones are now being frequently employed. The benefit of a drone compared to a plane is it is far easier to maneuver and fly. A lure can be suspended a safe distance below the drone to attract the falcon upward. As training progresses, the drone is flown at increasing heights to encourage the bird to quickly ascend to a pitch, which provides a spectacular stoop. The best drones have covered blades.

Pest Control

Falconry birds are now routinely used for bird abatement as part of pest control activities in the UK. While traditionalists would certainly not call this falconry—with that term being saved for when a bird is used in hunting activities, pest control work has allowed people to turn flying birds from a hobby into a job. Airports, sporting stadiums, rubbish dumps, food stores, warehouses, and shopping centers, all routinely employ falconers. Harris's hawks and large falcons, particularly gyr × saker hybrids, are the most commonly used species.

The use of Harris's hawks to scare pigeons from the famous grass tennis courts at Wimbledon makes the press year on year—it has been an opportunity to promote the other positives of falconry, other than sporting activities.

Falconry Displays

For many of the UK public, the first experience they ever have of falconry birds is the displays provided at centers and game fairs throughout the UK. Although the Falconry Centre in Gloucestershire was the first, a large number of other centers have been established since. The opportunity to see birds fly close-up aims to give the public an appreciation of raptors and sympathize with their wild conservation efforts. Some of the larger falconry centers also participate in conservation breeding of critically endangered species.

FALCONRY IN THE UK—THE FUTURE

Falconry is still very popular in the UK—with most estimates suggesting a growing number of participants over the last 10 years. Current estimates are of around 25,000 UK falconers. Despite a growing popularity, it is still very much a niche sport and does not carry the same support as other field sports such as shooting and angling. The greatest threat to UK falconry comes from animal rights activists and the risk of legislation banning or severely restricting falconry activities. In 2016, falconry was inscribed by UNESCO as an Intangible Cultural Heritage of Mankind. Unfortunately, at the time of print the UK does not recognize this, and as such, the UNESCO status of falconry provides little protection to UK falconry.

Tethering of birds of prey frequently attracts criticism—and while most falconers would consider a period of tethering necessary for many species of raptor (and essential when flying large eagles or accipiters), the excessive or unnecessary tethering of birds, particularly when not regularly flown, is difficult to defend. It must be remembered that birds flown in falconry experience a freedom to perform natural behavior that can be matched by very few other captive birds.

Many falconers and falconry centers already contribute heavily to conservation. In the future, techniques developed for falconry purposes should and likely will be increasingly used in the rehabilitation and release of wild injured raptors (Figure 55.19). Captive reproduction may become an important part of conservation efforts for critically endangered species, particularly African and Asian vultures.

FALCONRY IN NORTH AMERICA—THE PAST

> *We are all entangled in this web of life. It is inescapable. Falconry is a link to our past and a guide to our future, and for some, it helps illuminate the mystery of purpose on earth.*
>
> **Ed Pitcher, 1991**

Falconry in the United States of America

Falconry is a challenging and unique sport, but it did not gain significant popularity in the United States until the early to



FIGURE 55.19 A robotic crow model converted for use as a hack station during the soft release of a rehabilitated peregrine falcon *(Falco peregrinus)*.

mid-1900s. While it began as a utilitarian activity in other regions of the world, it developed into an art form in North America. From the 18th century and to this day, success and achievement are measured more by the quality of the flight than the taking of game.[42–44] Given its effectiveness in providing table fare in many cultures worldwide, it is surprising that there is very little evidence that Native Americans practiced falconry in any form. However, there are reports that Native Americans used similar methods to those used by falconers to capture eagles for ceremonial use.[45,46] Benedict-Henry Revoil described hunting herons with Black-Foot Indians in 1844,[47] and British literature suggests that Native Americans understood the practice of taming birds and demonstrated interest in the sport.[48]

In Colonial America, there appeared to be interest in falconry, although much of the interest seemed to concentrate on exportation of raptors from the "New World" to Europe (England) in the early to mid-1600s.[45] Settlers in the Virginias (Jamestown) and Carolinas, as well as Dutch settlers in what is now New York, also expressed interest in falconry in the 1600s.[44,49] Even "thirty-five years before the Mayflower brought the Pilgrim Fathers to New England, the colonists of Sir Richard Grenville on Roanoke Island were training hawks."[50] Descriptions of falconry and its activities were scant in the 1700s and 1800s, although the second volume of the *American Turf Register and Sporting Magazine* in 1830 gave reference to the sport.[51]

The 1900s marked a significant increase in the interest of falconry in America, with several individuals supporting the increase. Benjamin Rose Hoffman of Philadelphia was interested in falconry and the British hawking method[44] and made several trips to England to participate in the events of Britain's Old Hawking Club. Paul J. Rainey, a very wealthy hunter and plantation owner in Mississippi, hired Englishman R. Bowring as a professional falconer.[44] Hoffman and Bowring communicated with each other on two occasions, in 1910 and 1911, and Bowring suggested that a falconry club was needed in the United States.[44] Born in Seattle, WA, and raised in Washington DC, a 16-year-old falconer by the name of Russell Luff Meredith wrote of his experience hunting with a sharp-shinned hawk *(Accipiter striatus)* pursuing English or house sparrows *(Passer domesticus)*.[52] Meredith, who would later graduate from the US Military Academy at West Point (Class of 1917) and retire as a US Army Air Forces Colonel (1927), is considered by many to be the "Father of American Falconry."[44] Others also wrote of their falconry experiences including Dan R. Gray of Rockdale, TN, who enjoyed hunting with a Cooper's hawk, then later a European goshawk, and sharp-shinned hawk.[53] Edward Breck in Nova Scotia was convinced that he was the first to train an American hawk and introduce falconry to the New World. Unfortunately, he would be disappointed to learn that he was not the first; Gray had written of his experiences training a Cooper's hawk prior to Beck's.[54] North American falconry experienced another surge of interest in the 1920s and 1930s, likely the result of exposure of Americans to falconry while abroad during World War I or improved economic conditions that allowed for more leisure activity.[44] One of the more important contributions to North American falconry literature was written by a non-falconer, Louis Agassiz Fuertes.[55] Fuertes' article, *Falconry: the Sport of Kings*, published in the December 1920 issue of *The National Geographic Magazine*, introduced the sport to a large audience, creating even greater interest by falconers and would-be falconers of all age groups.[44,55] In the late 1920s Captain Charles W.R. Knight, a charismatic English falconer, would travel and lecture about falconry and would fly Mr. Ramshaw, his golden eagle, during demonstrations.[44]

Even with increased interest in falconry, birds of prey were considered vermin in North America, and mass shootings of raptors occurred during the late 1920s and early 1930s, especially along their migratory routes, often occurring at Hawk Mountain, PA, and Cape May, NJ.[56] Autumn hawk shoots became tradition in North America and were the subject of *Hawks Aloft*, written by Maurice Broun,[57] who became the first keeper in what is now Hawk Mountain Sanctuary.[56] In 1934, the killing of raptors would stop at Hawk Mountain, and Hawk Mountain Sanctuary would become the nation's first refuge for birds of prey.

The 1920s and 1930s were marked by a surge in the number of falconry clubs in the United States. One of the most notable, the American Hawking Club was located in Brewster, NY. The American Hawking Club hired a falconer, Frank Levy, who was a recent graduate of Cornell University and associate of Fuertes.[44] During the early 1900s, fascination with the sport in the United States seemed to grow more from a naturalist perspective, rather than concerns about the traditionalist views of falconry noted in other parts of the world.[44] Alva G. Nye, Jr., and twin brothers Frank C. Craighead, Jr., and John J. Craighead became acquainted with Frank Levy and soon became falconers.[44] The Craigheads' father was an environmentalist, and their sister, Jean Craighead George, became a very well-known author of children's books. Her novel *My Side of the Mountain* introduces Frightful, a peregrine falcon taken by Sam, the main character, and trained to catch frogs, pheasants, rabbits, and sparrows. Frank and John Craighead would gain further prominence through their articles published in *National Geographic Magazine* and their book *Hawks in the Hand* (1939, 1997).[44]

The Peregrine Club, founded by James N. Rice II in 1936, would replace the short-lived American Hawking Club. The Peregrine Club was also short-lived but provided its members the opportunity to share ideas and information and became a blueprint for future falconry clubs in North America.[44] In 1938, Colonel Meredith proposed the formation of a national falconry club at North America's first falconry meet in Media, PA, and the Falconers Association of America was formed. Difference of opinion regarding logistics with this club led to the formation of the "Falconers Association of North America," which published the *American Falconer Journal*.[44] Unfortunately, World War II Pearl Harbor put an end to the club's activities.[44] After World War II, the Falconry Club of America was formed and grew quickly. Similar to the Falconer's Association of America, differing opinions within the Falconry Club of America lead to the formation of the North American Falconer's Association (NAFA) formed in 1961.[44] NAFA's first president was Hal Webster, a young falconer who became interested in the sport in the 1930s. Hal and his good friend Frank L. Beebe, a Canadian falconer, would be forever linked together as cofounders of NAFA as well as authors of one of the seminal texts on the natural history, capture, training techniques, and breeding of raptors

used in falconry. This text, *North American Falconry and Hunting Hawks*, was first printed in 1964 and would become required reading for anyone interested in or already practicing falconry in North America. Since its early beginnings, NAFA has represented falconry in North America on both the state and federal levels. The effectiveness of NAFA in garnering membership for North American falconers stems from the opportunities it provided its members. NAFA publishes articles written by falconers as well as provides members access to numerous articles about falconry in the *Hawk Chalk* and the annual *Journal of the North American Falconer's Association*. Social events such as regional and national field meet regularly provide falconers with the opportunity to get together.

While falconry was seemingly predominant along the eastern United States, young men and women around the country began to show interest in the sport. Of note, Bill Russell, a medical student, became interested in falconry and would later author one of America's first falconry books, *Falconry: A Handbook for Hunters*.[58] Another noteworthy individual was Fran Flint, a graduate student of Aldo Leopold, who many consider the father of wildlife management and the American conservation movement.[44] Fran would later marry Frederick Hamerstrom, and together they would provide the foundation for many of the laws and regulations protecting raptors and the recognition of falconry.[44] In 1948, Harry McElroy, a young falconer living in Houston, TX, would begin his falconry journey flying Cooper's hawks. McElroy and many others that began practicing falconry at this time would gain prominence in American falconry and become innovators of falconry training, weight management, and imprinting techniques still used to this day. In 1989, McElroy would establish a home in Mexico for nearly 3 years, hunting aplomado falcons in Querétaro, Mexico.[59] McElroy would host Mexican falconers at his home and would contribute significantly to the knowledge of falconry in Mexico.[59]

Falconry in Canada

Falconry in Canada and its territories, as in the United States, is diverse, with many of its pioneers emigrating from other countries, some specifically for falconry (Mark Williams, personal communication, 2020). Definitive accounts of falconry being practiced in the New World, what is presently Newfoundland and Labrador, are difficult to find. Falconers could have been present during early explorations of these regions. Records exits of falcons being sent back to England as early as the 1500s. British Parliament enacted a reward system in 1503, resulting in the mass transplantation of falcons, likely highly prized gyrfalcons and peregrine falcons, from the New World back to England during the 16th and 17th centuries. One of the earliest accounts of the actual practice of falconry in Canada was published in the *British Sporting Magazine* and recounts the shooting of a banded raptor in Yorkshire, England. According to the band, the owner of the hawk was the Governor of New Halifax, America, in 1762.[60] In 1850, A.E. Knox described a British Colonel Johnson known to have taken two peregrines falcons that he allowed to fly from ship to ship during the Atlantic crossing.[61] Further, in 1871 Captain F. Sandys Dugmore describes having taken a team of hawks, to include seven merlins, two goshawks, and two peregrines, from Britain to Canada aboard the HMS Serapis in 1867.[44,62] In 1935, a young 18-year-old Ben Oswald Goodman Dixon emigrated from Scotland to Canada to attend McGill University. Ben was the nephew of famed British falconer William Herbert St. Quintin.[44] John Campbell also immigrated to Canada in 1954 after serving in World War II.[44] A falconer prior to World War II, Campbell did not practice falconry again until 1954 (Mark Williams, personal communication, 2020). John Campbell, American falconer Dr. Mike Person, and other Alberta falconers pushed for legalized falconry in the Province (Mark Williams, personal communication, 2020). Importantly, Campbell was one of the first falconers to breed peregrines and merlins in the 1972/1973 season (Mark Williams, personal communication, 2020). In the early 1950s, Frank L. Beebe began to actively participate in falconry and trained his first falcon.[44] Frank Beebe gained prominence as a falconer, writer, wildlife illustrator, conservationist, and an outspoken advocate for falconry. Frank viewed raptors as a renewable resource and was critical of regulations governing falconry in North America.[44,56] He was instrumental in cofounding NAFA and coauthoring *North American Falconry and Hunting Hawks* with his good friend Hal Webster. Beebe would also author several other important contributions to North American falconry, among them are *A Falconry Manual* and the *Compleat Falconer*, and contributed significantly to our knowledge of *F. p. pealei* and gyrfalcons.

Falconry in Mexico

Prior to the arrival of the Spaniards, more specifically Hernado (or Hernán) Cortés in 1519 to "New Spain," what is now Mexico, and his subjugation of the indigenous peoples, there was no evidence that falconry was practiced in Mexico.[44] There are reports that ninth Aztec Emperor Montezuma (1466–1520), imprisoned by Cortés, may have practiced falconry; however, those accounts were misinterpreted.[44] Reportedly, Montezuma was knowledgeable about wildlife management and had a zoo with a large collection of species at Tenochtitlan.[59] Within this zoo, Montezuma was reported to favor a section containing a large assortment of raptors possibly used for falconry; however, the full veracity of this has been debated.[34,59] Historian Jose Manuel Fradejas Rueda claims that a linguistic shift led to misinterpretation of the words originally written by Cortes by later writers and historians.[63] An account from Diaz del Castillo, one of Cortes' captains and author of *The True History of the Conquest of New Spain*, in 1575 describes a firsthand encounter in which it is clear that Montezuma did not know anything about training hawks.[64] Diaz del Castillo describes Montezuma's surprise that Cortes, Diaz del Castillo, Francisco de Acevdo, and other of Cortes' captains, upon seeing a small hawk pursue prey through the garden, discussed training and hunting with the hawk.[64] Montezuma subsequently ordered the hawk captured, and another captain, Francisco de Acevedo, subsequently trained it.[44] His training of this hawk likely represents the first hawk trained in the Americas.

A sentinel moment in Mexican falconry occurred on December 9, 1531, when the peasant Juan Diego had a vision in which the Virgin Mary appeared before him.[44] The description of his vision has become a major element in Mexico's religious beliefs and the Basilica of Our Lady of Guadalupe, which was built on the site of Juan Diego's vision.

In *The Mexican Hunter or Art of Hunting Mexico*, published by Pedro Blazquez in 1868, there is mention of the sport of falconry; however, it failed to mention if falconry was actually

practiced in Mexico. Similar to other regions of the Americas, there is little evidence to suggest that falconry was practiced in New Spain until the latter half of the 20th century.[44]

As in other areas of the Americas, there is little history of falconry in Mexico after the 16th century until the mid-20th century. In 1950, the Mexican Association of Falconry emerged in Mexico City.[59] However, similar to early attempts to create formal falconry organization in North America, the Mexican Falconry Association was short lived.[59] Letters and diplomas written by its president, and likely the only member of the Association, Guillermo José Tapia, provide evidence of existence (Raul Ducoing, personal communication, 2020). One such diploma was presented to Dr. Robert M. Stabler, who was only active for approximately 2 years then disappeared with no further records of him practicing falconry in Mexico (Raul Ducoing, personal communication, 2020). While one report suggests that the Mexican Falconry Association's membership included distinguished foreign falconers,[59] one falconry historian suggests that no records exist that others knew of the Association, and its founder also disappeared in 1951 (Raul Ducoing, personal communication, 2020).

The mid to late 20th century marked a surge in the interest of falconry in Mexico. In 1960, an 18-year-old falconer by the name of Roberto Béhar Calderó began practicing falconry in Coyoacán, Mexico City, by starting with a Harris hawk, and later a golden eagle.[59] Roberto was considered the initiator of falconry in Mexico and the first to import goshawks from Europe into the country.[59] Four years later, Rafael Del Valle Reyna started practicing falconry with a roadside hawk *(Rupornis magnirostris)*, a small *Buteo* that he used with a Vizsla to hunt quail.[59] Rafael is credited as being the first Mexican falconer to train the aplomado falcon and the bat falcon *(Falco rufigularis)*, as well as contributing to the Peregrine Fund's study and conservation project of the aplomado falcon.[59] In 1967, Ing. Federico Medrano Crespo, who would later become the president of the Asociación Mexicana de Cetrería in 1985, began his falconry experience by hunting rabbits with Harris's hawks and red-tailed hawks.[59] Eduardo Bessouda, Héctor and Eduardo Appellániz, Roberto Béhar, and Enrique Guerrero began hunting rabbits with Harris's hawks and red-tailed hawks and were documented as the first written accounts of hunting with hawks in Mexico.[59] Juan Carlos Rojo G., author of the *Falconry Heritage of Mexico*, learned about falconry in 1970 and started training an American kestrel *(Falco sparverius)*.[59] Most Mexican falconers hunted with Harris's hawks and red-tailed hawks; however, the flying of longwings did not occur until 1972 when Enrique Guerroro and Eduardo Appellániz trapped the first passage peregrine falcons in Mexico.[59]

The second Mexican falconry association, the Conservation and Investigation Center for Birds of Prey, was founded by Roberto Béhar in 1973 and located in Ajusco, a half hour from Mexico City.[59] This organization had approximately 10 members, which later grew to around 20 members in 1975.[59] During the same year, an article about falconry, written by Nicolas Trillo, was published in the *Shooting Hunting and Fishing* magazine and included a photograph of a hooded peregrine falcon. This article was the first to describe modern falconry in Mexico.[59]

What is described as the second generation of Mexican falconers came along in the late 1970s. In 1977, Raul Ducoing (Figure 55.20), considered one of the pioneers of falconry in Mexico and a historian of the sport, started by flying an eyass female kestrel and a tiercel passage American kestrel.[59] In 1978, the Investigation and Conservation Association for Birds of Prey (Asociación para la Investigación y Conservación de las Aves de Presa, AIPAP) was founded by its president, Luis Carino. Luis organized the first national conference for birds of prey at the National Autonomous University of Mexico, Mexico City, Mexico, which involved more than 40 falconers.[59] A second conference was held a year later at the same location, and by the end of the decade there were approximately 50 falconers in Mexico.[59]



FIGURE 55.20 Falconer and falconry historian Raul Ducoing is considered one of the pioneers of falconry in Mexico, and he, along with others, is leading the charge for falconry standards in Mexico. (Photo courtesy Raul Ducoing, used with permission.)

During the mid to late 1980s, several falconry associations would be formed in Mexico. In 1984, Juan Carlos Rojo G., Héctor Verdejo, Jorge Carbajal, and Franciso Vázquez started a new falconry association, the Asociación Mexicana de Cetrería (AMC).[59] The year 1986 would mark the occurrence of the first captive-bred peregrine falcon in Mexico. The AMC would change its name in 1989 to the Circulo Mexicano de Cetrería (CMC) and published an international bulletin, *El Halconero*.[59] The Asociación Cetrería de Toluca, another association, was founded in 1987 by Isaías de la Rosa, José Salazar, and Mauricio González.[59] Shortly afterward, the Association of Falconers and Protectors of Birds of Prey (ACPAP) was established in 1988. An attempt was made by the CMC to encourage Mexican falconers to unite in one national falconry group; however, that did not happen. Later, the CMC would become divided, and former members of the CMC would create the Federación Mexicana de Halconeria (FEMEHA) in 1990.[59] FEMEHA, along with falconry clubs, such as the Grupo Cetreros del Valle de Mexico, would host a number of meetings for Mexican falconers.[59] During this time the number of falconers in Mexico grew dramatically, and by the end of the 1990s, there would be 300 falconers in the country.[59] Unfortunately, FEMEHA was inactivated around 1996.

The 20th century would see a surge in the number of falconry clubs in Mexico, but it would also bring some challenges to falconry in Mexico. An initiative in the Mexican

Congress sought to change legislation (Article 85) and deny the registration and possession of birds of prey in captivity and prohibit falconry.[59] Mexican falconers, with support from NAFA and the IAF, united to defend their right to own and register raptors by sending numerous letters to the Mexican legislature.[59] Deputy Guillermo Tamborrel represented Mexican falconers in Congress, discouraged changes to Article 85, and halted the initiative. The benefit of Mexican falconers uniting for a common cause was the resurgence of FEMEHA in 2010.[59] FEMEHA would produce one Mexican falconry journal, *Volatería*, in 2017 that included a history of falconry in Mexico.[59]

The Peregrine Falcon in North America

As interest in falconry seemed to grow from a naturalist perspective, the voice of falconers became even more prominent in environmental issues and the plight of the peregrine falcon, bald eagle *(Haliaeetus leucocephalus)*, and other raptors in the mid-1900s. It was falconers who first noticed the dramatic decline and near extirpation of nesting populations of peregrine falcons, especially North American migratory species including the *anatum* peregrine falcon *(F. peregrinus anatum)*, sometimes referred to as the American peregrine falcon, and the Arctic or tundra peregrine falcon *(F. p. tundrius)*. Only the third species, the Peale's peregrine *(F. p. pealei)*, appeared to have stable populations but only those nesting along the north Pacific coast in Alaska and British Columbia.[65] The United States Fish and Wildlife Service (USFWS) did not consider peregrine falcons abundant, with studies in the 1930s and 1940s estimating 500 breeding pairs in the east and 1000 breeding pairs in the west and Mexico.[65] Canadian populations of peregrine falcons are difficult to document given the remoteness and difficulty of assessing populations in some areas. By the mid-1960s, populations of breeding peregrine falcons had been nearly exterminated from their eastern range. Western peregrine falcon populations did not fare much better, with populations declining by 80% to 90% in the mid-1970s.[65] In 1965, at the University of Wisconsin, Madison, WI, Professor J. J. Hickey convened an international conference intently focused on the rapid decline of peregrine falcons. The majority of attendees were falconers. It was determined that the unregulated use of organic pesticides, in particular DDT and its metabolites and breakdown products, dichlorodiphenyldichloroethylene (DDE) and dichlorodiphenyldichloroethane (DDD), caused eggshell thinning, resulting in dramatic population declines in peregrine falcons, bald eagles, and other avian fauna.[66–70] Sadly, a similar situation was experienced in Europe. These findings, and the indiscriminate killing of birds of prey, only exacerbated the decline of the peregrine falcon in the mid-1900s. Following Hickey's conference, falconers, biologists, academicians, educators, rehabilitators, and government biologists, all concerned about the welfare of peregrine falcons, officially incorporated the nonprofit Raptor Research Foundation (RRF) on February 11, 1966.[43,44] RRF's intention was to "to stimulate the dissemination of information concerning raptorial birds among interested persons worldwide and to promote a better public understanding and appreciation of the value of birds of prey."[71] More specifically, its members wanted to assess the status of the peregrine falcon and to discuss, promote, and exchange ideas on how to breed them in captivity.[71] NAFA established an affiliate North American Peregrine Foundation for raising money to fund work on the peregrine falcons,[43] and the RRF was influential in establishing several breeding and reintroduction centers. In 1970, Tom Cade, then a falconer and professor of Ornithology at Cornell University, enlisted the help of fellow falconers Jim Weaver, Bob Berry, and Frank Bond to create The Peregrine Fund.[43,44] The Peregrine Fund developed two programs, one in the east at Cornell University, and a second under the direction of falconer Bill Burnham, in Fort Collins, CO, and in cooperation with the Colorado Division of Wildlife.[43,44] The latter would be consolidated into a single location at Boise, ID.[43] *Falcon Propagation: A Manual of Captive Breeding* describes the methods, procedures, and equipment necessary for falcon propagation.[72] Additional locations were the Raptor Center at the University of Minnesota, where Dr. Patrick Redig and Bud Turdoff coordinated reintroduction of peregrines provided by falcon breeders, mostly falconers, into the Midwest.[43] The Santa Cruz Predatory Bird Research Group, founded in 1975 by veterinarian and falconer, Jim Roush, and marine biologist Ken Norris, bred and obtained falcons for reintroduction along the Pacific coast.[44] Meanwhile, in Alberta, Richard Fyfe, while working for the Canadian Wildlife Service, supervised Camp Wainwright, a captive breeding and research station, utilizing some of the last remaining wild peregrines in the area (Mark Williams, personal communication, 2020). The latter program was disbanded in the late 1990s when government funding was discontinued. Other smaller operations in Canada included one program coordinated by John Campbell and Wayne Nelson, a program at the University of Saskatchewan at Saskatoon coordinated two falconers Lynn Oliphant and Patrick Thomson, and lastly, one at the MacDonald campus of McGill University coordinated by Dr. David Bird who was not a falconer. In total, these successful operations accounted for the release of more than 6000 peregrine falcons back into the wild since 1974.[44]

The banning of DDT was central to the recovery of peregrine falcons in North America. Most notably, captive breeding and release of peregrine falcons by falconers preceded the ban of DDT.[44] Canada banned the use of DDT in 1970. Two years later, the Environmental Protection Agency (EPA) under the authorities granted by the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) banned most uses of DDT in 1972. It is important to note that DDT may still be used in other areas of the world including some within the peregrine's migratory range. *F. p. tundrius* was delisted from the endangered species list by the USFWS in 1984. In 1999, the same designation followed for *F. p. anatum*. The conservation status of the peregrine falcon in Canada varies from "No Status" to "Endangered."[73]

The monitoring of peregrine falcon populations was initiated by Colonel Meredith, who surveyed peregrine falcons on Padre Island, TX, from the 1940s through 1965, during their autumnal migration. Padre Island would become known as a key migratory route for peregrine during their southward autumn migration and the only known staging area for *F. p. tundrius* during their northward migration.[74] Earthspan, a nonprofit organization formed in 1994, continues its biomonitoring and conservation efforts through the Padre Island Peregrine Falcon Survey, performing surveys in the fall and spring of each year. To

accomplish these surveys, Earthspan works in collaboration with Peregrine Fund, Texas Parks and Wildlife Department, the US Geological Survey, USFWS, and a number of individuals and volunteers who perform the surveys.

Legislation

North American Falconer's Association and falconry regulations

Prior to the 1950s, regulations to monitor the use and possession of raptors for falconry were essentially nonexistent. Most people interested in falconry were biologists, ecologists, and other scientists concerned with the natural history of the raptors used in falconry.[43] Thus NAFA then became a voice and advocate for not only the practice of falconry but also the conservation of birds of prey.[43]

The Migratory Bird Treaty Act (16 U.S. Code 703–712, MBTA) was originally enacted in 1916 between the United States and Great Britain on behalf of Canada. It was signed into law in 1918, and upholding the treaty with Canada; the MBTA prohibits the take (including killing, capturing, selling, trading, and transport) of protected migratory bird species without prior authorization by the Department of Interior USFWS.[75] The MBTA implements four international conservation treaties that the United States entered into with Canada in 1916, Mexico in 1936, Japan in 1972, and Russia in 1976. The law has been amended with the signing of each treaty, as well as when any of the treaties were amended, such as with Mexico in 1972 and 1976, and Canada in 1995. In 1972, raptors were added to the list of birds granted federal protection by the annex of the US MBTA with Mexico. However, there were consequences for North American falconers and falconry as a result (see section on Operation Falcon).

With growing concerns over the protection and inclusion of all raptors under the MBTA in 1972, the enactment of the Endangered Species Act of 1973, and the earlier enacted Lacey Act of 1900, NAFA's initial efforts were to acquire legal recognition of falconry as a hunting sport at a state level.[44] However, NAFA ultimately directed its attention toward federal regulation of falconry and the USFWS, Department of the Interior. The first edition of the *Code of Federal Regulations* (CFR) was published in 1938. It is divided into 50 Titles that represent broad areas subject to federal regulation. The portion of the CFR that governs falconry standards and permitting is Title 50, Part 21 (50 CFR § 21.82).[76] While this system allows regulation of falconry at both the state and federal level, state regulations may be as stringent, or more stringent than federal requirements. Canada differs from the United States in that the guardianship of its natural resources is the responsibility of each province and not their federal government. Current regulations for each state and Canadian province are found on the NAFA's website (https://www.n-a-f-a.com).

Licensure and Regulations to Practice Falconry

United States

Persons interested in becoming a falconer often seek initial advice from their state or provincial wildlife agency or inquire with their local falconry organization. Often, the agency will send a packet of information regarding how to become a falconer, as well as state requirements, and some information for preparing for a falconry examination. The state agency or local organization may then direct them to seek the assistance of a falconer. A falconer, of general class or higher, may likely agree to sponsor the person, assist them through the application and licensing process, and acquisition of required equipment, as well as help them acquire and train their first raptor. The level of commitment by sponsors may vary.

Licensure to practice falconry in the United States requires the applicant to pass, with a score of 80% or better, a qualifying state examination. Similarly, an examination is also required in Canadian provinces. For example, Ontario requires Apprentices to take the Ontario Hawking Club's falconry examination. The examination evaluates the applicant's knowledge of natural history, managed care, falconry techniques and equipment, and laws and regulations governing the sport. Once passed, the individual is not required to take the examination again unless there is a prolonged lapse in their practice of falcon, and there is reciprocity between states should a falconer move from one state to another. If passed, the Apprentice falconer may pursue acquiring the necessary equipment (e.g., appropriate leather glove, jesses, anklets, swivels, leashes, scale, an appropriate perch for the species, water pan, and a mew), all with the help of their sponsor. North American falconers are also required to obtain general hunting licenses in addition to their falconry license to pursue game.

There are three classifications of falconers in the United States. Apprentice falconers, per most state regulations, must be at least 14 years of age and have the sponsorship and support of a General- or Master-level falconer. For those states that have essentially adopted the federal falconry regulations as their state regulations, the minimum age to practice falconry is 12, although this may still vary from state to state. The falconer must remain an Apprentice for at least 2 years, and some states may limit on the number of Apprentices a sponsor may have at one time. Historically, Apprentice falconers were only allowed one bird at any time and were required to trap and train either an American kestrel, a red-shouldered hawk *(Buteo lineatus)*, or a red-tail hawk. Apprentice falconers are not permitted to take an eyass raptor from the wild and may only replace one raptor per year. General falconers must have actively practiced falconry for at least 2 years and must be at least 16 years of age; however, age requirements may vary from state to state. Apprentice and General falconers who are less than 18 years of age must have a parent or legal guardian who is legally responsible for their activities. As with Apprentice falconers, there are possession limitations for General falconers that limit possession to no more than two raptors, or replacement of two raptors per year. In addition, General falconers may not hold, keep, or transport threatened or endangered raptor species. Master falconers have been practicing falconer for at least 5 years at the level of General. Federal falconry regulations state that Master falconers may possess no more than five wild raptors, including golden eagles, and replace only two per year. Master falconers may possess any number of captive-bred raptors that are trained in the pursuit of wild game. Unless authorized by the USFWS, falconers may not possess any raptor species listed as threatened or endangered, and US falconers may not take or possess a bald eagle *(Haliaeetus leucocephalus)* for falconry. In the United States, falconers may trap passage (hatch year) raptors from the wild for use in falconry; however, exceptions exist for species such

as great horned owls *(Bubo virginianus)* and American kestrels, which may also be taken as adults at specific times during the year. Passage take of raptors is relatively new in some Canadian provinces. In the United States, all acquisitions, transfer (gift, sale, or loan), loss, or death of a raptor used for falconry are submitted to the USFWS, historically by paper and now electronically, through a Federal Fish and Wildlife Permit Application Form (Form 3-186a—Migratory Bird Acquisition and Disposition form).

Canada

In Canada, provinces differ on the minimum age requirement to practice falconry. For example, in Ontario, a person must be at least 15 years of age to practice falconer, while Saskatchewan requires falconers to be at least 12 years of age. In contrast to falconry levels in the United States, there appears to be no designation of Master falconer; instead only Apprentice and General falconers are recognized in the provinces. The apprenticeship must be sponsored by someone who has been a General falconer for a minimum of 5 years and lasts for at least 15 months, which includes two autumn hunting seasons. In Canada, reporting of acquisitions, transfers, loss, or death of a raptor used in falconry varies between provinces. In most provinces, reports are provided when they occur. In Ontario, falconers record these in a logbook and report this information once a year to authorities; however, the logbook is subject to inspection at any time.

Mexico

Wildlife management and regulations in Mexico have undergone significant and rapid changes. However, since 2001 wildlife management responsibilities have fallen under the Ministry of the Environment and Natural Resources (Secretaría de Medio Ambiente y Recursos Naturales [SEMARNAT]). In 1993, Raul Ducoing presented a proposal for falconry regulations in Mexico to the Mexican government, and this became a Norma Oficial Mexicana (NOM) for falconry, or compulsory standards and regulations. Unfortunately, the Chief Executive Officer of SEMARNAT was assassinated and the regulations were put on hold (Raul Ducoing, personal communication, 2020).

In 1998, a new initiative from Mexican wildlife authorities, including SEMARNAT and Procuraduría Federal de Protección al Ambiente (PROFEPA), was designed to govern falconry activities in Mexico. SEMARNAT develops and implements national policy on protection, restoration, and conservation of natural resources and provides permit guidance for Mexican falconers. PROFEPA provides law enforcement guidance and monitoring for illegal hunting activities. Currently, there are little to no regulations that govern falconry and very little interest from authorities or traction to push for them in Mexico.

At present, falconers in Mexico are not allowed to trap raptors from the wild as in other areas of North America. From 2005 to the present, only captive-bred or imported raptors are allowed for falconry, and importation of raptors into Mexico for falconry is difficult. Currently, Mexican falconers also have no regulations or requirements for mentorship of new falconers, or for how birds are housed or trained. Most beginner falconers are directed to hunt with Harris hawks, rather than kestrels or red-tailed hawks, as is in other areas of North America (Raul Ducoing, personal communication, 2020). Most experienced falconers in Mexico are training and hunting with Harris hawks, Cooper's hawks, and peregrine falcons (Raul Ducoing, personal communication, 2020).

Operation Falcon

No report of falconry in North America would be complete without including some discussion of Operation Falcon, an undercover operation conducted jointly by the USFWS Department of the Interior and the Canadian Department of Justice/Canadian Wildlife Service that concluded in 1984. During the 1970s, USFWS and the Canadian Wildlife Service began collecting intelligence data and became convinced that there was an illegal, multimillion-dollar, black market, international trade of valuable falcons (peregrine falcons and gyrfalcons) and their eggs from North America to Europe and the Middle East. The undercover phase of the operation culminated on June 29, 1984, and 9:00 a.m. Eastern Standard Time marked the moment USFWS and state agents descended on North American falconers executing searches, arrests, and seizures of more than 100 hawks and falcons.[44,77] Their primary targets were falconers and raptor breeders, mainly peregrine falcon breeders.[77] As per a news release from the US Department of the Interior, Operation Falcon lasted approximately 3 years, resulting in 30 arrests and estimates of as many as 400 birds illegally taken from the wild.[78] Tantamount to the Department of the Interior's news release, the National Audubon Society provided a similar release espousing the "success" of the Department of the Interior's Operation and even estimating the number of arrests to be 50 individuals with 500 birds illegally smuggled.[77,78] Ultimately, USFWS failed to prove the existence of substantial, illicit, black market trafficking of falcons in the United States.[77] Congress was informed of the abuses in Operation Falcon through a series of hearings culminating in a hearing before the House Subcommittee on Fisheries and Wildlife Conservation and the Endangered Species Act.[77] During this hearing, the USFWS Division of Law Enforcement was subject to criticism by the subcommittee members.[77] Both USFWS Law Enforcement and the National Audubon Society suffered embarrassment, and their actions gained distrust from North American falconers.[44] John Jeffery McPartlin, a falconer convicted of violating the Lacey Act, served as a sting operator and informant for the USFWS.[44,77] Operation Falcon was the subject of *The Pilgrim and the Cowboy*, which provides a good summation of this challenging time in falconry.[79]

FALCONRY IN NORTH AMERICA—THE PRESENT

In North America, falconry is the most highly regulated field sport, even compared to other methods of hunting with guns. In the United States, falconry is legal in all states except Hawaii and the District of Columbia. In Canada, falconry is not legal in every province.

Legal falconry is practiced in British Columbia, Alberta, Saskatchewan, Manitoba, Ontario, and Quebec. The New Brunswick Falconry and Austringer Association appear to be active; however, falconry appears to be illegal in this province.

The USFWS estimates approximately 6200 currently permitted falconers in the United States, approximately 65% are active with raptors on their permits. Current NAFA membership

is approximately 2000 members, 200 to 300 of which are international members. Women represent approximately 18% to 20% of membership, and the numbers are growing (Nicolle Sheldon, personal communication, 2020). There are significant numbers of underrepresented minority falconers in North America, and those numbers are growing as well; however, there is little data available to provide any statistical conclusions (Sheldon Nicolle, personal communication, 2020). In Canada, the number of falconers is considerably less than in the United States and is estimated to be between 400 and 500, with 50% or more licensed in Ontario. While Ontario has the largest number of falconers and perhaps the largest provincial club, The Saskatchewan Falconry Association is reportedly the oldest, continuously operating falconry association in North America. Significant numbers of falconers are also present in Quebec and Alberta, while Saskatchewan, Manitoba, and the eastern provinces have active but small falconry communities (Martin Geleynse, personal communication, 2020). While Mexico has approximately seven to eight falconry clubs or groups with around 950 falconers, there are 150 falconers who are actively practicing the sport, and of those, approximately 35 are Master falconers/professional falconers (Raul Ducoing, personal communication, 2020). The remainder of the 950 falconers are considered falconry enthusiasts and may practice the sport then are quickly out of falconry. Women represent approximately 0.05% of practicing falconers in Mexico (Raul Ducoing, personal communication, 2020).

Since 1962, NAFA has held field meets for its membership. These meets, which often occur over the Thanksgiving holiday, provide falconers the opportunity to share their experiences, learn from other falconers, and disseminate knowledge.

Since its inception, and for quite some time, NAFA has been the representative voice of falconers in North America and provided notable publications in the form of the annual NAFA journal and the *Hawk Chalk*. In the fall of 1995, the first volume of *American Falconry Magazine* was published. This journal is devoted to the sport of falconry, produced by practicing falconers, and highlights news and issues important to falconers including legislative information, training techniques, dogs used in falconry, falconry equipment, and veterinary care. Initially, NAFA only allowed US and Canadian falconers as members. However, in 1999, an amendment was made to NAFA's Constitution and Bylaws that allowed Mexican falconers over the age of 18 to join NAFA, thereby allowing Mexico to become a member of NAFA.

While NAFA had been the main voice of North American falconers since its inception, in 2002, the nonprofit American Falconry Conservancy (AFC) was conceived. The AFC, which also included many NAFA members, had differing views regarding falconry regulations and wild take of raptors for falconry than NAFA. AFC's mission is to protect and preserve falconry in the United States for future generations by protecting the falconer's rights as legitimate sportsmen and conservationists.[80] This would be accomplished by pursuing the broadest liberties possible that are not in conflict with legitimate conservation efforts based on sound biological and legal reasoning.[80]

Landscape

Falconry in North America is unique given the diversity of private and public landscapes on which to hunt, the diversity of raptor species used (hawks, eagles, and falcons), and the diversity of quarries available for falconers to pursue. As the third largest continent, geography varies tremendously across the United States and Canada, ranging from mountainous ranges, expansive prairies and steppes, tundra, dense forests, deserts, wetlands, and lowlands.

Raptors

Due to the dramatic differences in terrain across the United States, falconers attempt to choose the raptor species most suitable for the terrain and available quarry. As varied as the landscapes are, so are the raptors commonly used in falconry in North America. These range from the diminutive sharp-shinned hawk or American kestrel to the formidable golden eagle. Sources of raptors include captive-bred species as well as those taken as eyass or passage birds from the wild, and there are a number of raptor breeders across North America. NAFA provides a list of breeders that includes members of the North American Raptor Breeders Association (NARBA). Until recently, Canadian falconers were not allowed wild take of raptors for falconry, and regulations vary between provinces. Captive-bred birds may either be imprinted or trained when they are older.

Red-tailed hawk

Found throughout continental North America, except northeastern Canada, there are approximately 14 recognized subspecies of this large *Buteo*. Although most red-tailed hawks look similar, the Krider's *(B. j. kriderii)* is a light-colored morph of the eastern red-tailed hawk *(B. j. borealis)*, and the Harlan's *(B. j. harlani)* may be blackish-brown in color, or may also demonstrate light-colored morphs. Red-tailed hawks are one of the more popular raptors used in falconry in the United States and are often the first raptor trained by many falconers when they start. Not surprisingly, experienced falconers also choose to use red-tailed hawks, especially if quarries (e.g., squirrels) are the most accessible game in their area. Even though this is a larger raptor, it is very effective, efficient, and versatile at catching a variety of game including small mammals, waterfowl, upland game birds, and jackrabbits. Red-tailed hawks are often wild caught by falconers, as (mal)imprinted individuals may become quite aggressive toward the falconer. Surprisingly, red-tailed hawks are not commonly used in falconry in Mexico. Hybridization of red-tailed hawks with other species such as ferruginous hawks and Harris hawks has been created. These hybrids have been produced but are uncommon in the United States (see also section on Falconry in the UK—The Present). Red-tailed hawks weigh between 710 to 1550 g.[81]

Ferruginous hawk

The ferruginous hawk is the largest *Buteo* in North America. Powerfully built with a rather large gape, this hawk possesses considerable grip strength and is able to dismember its prey, swallowing large portions rather quickly. Feathered tarsi are a distinguishing characteristic of this species. Similar to red-tailed hawks, both light and dark morphs occur. Its southern range is primarily the arid and prairie regions of the southwestern United States, extending into Mexico. Its northernmost range is southern Canada, primarily Alberta and Saskatchewan. This hawk may be challenging and generally not for the novice falconer. The weight range for male and female ferruginous hawk is 980 to 2030 g.[81]

Harris's hawk

The Harris's hawk deserves a special mention as no other species of raptor has revolutionized falconry in North America and several regions around the world, compared to this extremely versatile hawk.[82] This hawk is the only member of the genus *Parabuteo*, and two subspecies of Harris's hawks are recognized. The northern *P. u. harrisi* is found in the southwestern United States, Mexico, Central America, and into South America along the western slope of the Andes from western Colombia to northwestern Peru.[81] The southern *P. u. unicinctus* is only found in South America, and its range extends from Colombia through Chile and Argentina, except for the western Pacific slope from the Panama-Colombia border, and south through Peru. The social nature of Harris's hawks has been well studied. In the wild, these hawks may hunt collaboratively and live in extended family groups. In falconry, the Harris's hawk's social hunting style allows multiple falconers and multiple hawks to hunt simultaneously. Its easy temperament, versatility, intelligence, flexibility, adaptability to hunt in a variety of conditions, and social interactions allow a comradery between falconers that is not seen with other raptors (Figure 55.21). Well suited for warm and hot climates, the one drawback, if there is any, is that this hawk does not do well in colder climates, and frostbite can be a common problem in colder climates. This hawk is extremely popular in Mexico and is the most common raptor used (see also section on Falconry in the UK—The Present). Geographic variation exists between Harris's hawks from different regions and subspecies. Reportedly, the weight range for *P. u. harrisi* can vary tremendously and range from 550 to 829 g for males to 825 to 1633 g for females,[82] but most adult females average around 1000 g. *P. u. unicinctus* are smaller.

Currently, Harris's hawks are one of the most common hawks bred and flown in falconry. In 1999, the Baywing Database was created by falconer Toby Bradshaw to allow for a global registry of captive-bred Harris's hawks (https://baywingdatabase.com/). The purpose of the Baywing Database is to collect, store, analyze, and distribute information on Harris's hawks used for falconry, and it allows falconers to register their hawk(s), generate a pedigree for any registered hawk, find a hawk's relatives, or even run a trial mating.[83]



FIGURE 55.21 "Hannibal" and "Moe" represent two extraordinary descendants of the Harris's hawks *(Parabuteo unicinctus)* "Malcom" and "White Wing"; their bloodlines are well known among North American falconers and Harris's hawk breeders. Their speed, versatility, persistence when pursuing game, sociality, ease of handling, intelligence, and impressive game records are attributed to selective breeding, parent rearing, socialization conditioning, and being provided hunting areas with abundant game. (Photo courtesy Jennifer O. Coulson, used with permission.)

Accipiters

See also the section on Falconry in the UK—The Present.

Accipiters are common in North American falconry and are represented by the northern goshawk, the Cooper's hawk, and the sharp-shinned hawk. Accipiters have the reputation of being high-strung, temperamental, and sometimes aggressive toward the falconer, requiring patience in handling. Accipiters may be taken from the wild as eyass and passage birds, although most falconers take them at a young age, preferring to imprint them.[84,85]

Northern goshawk. See also sections on Falconry in Asia—The Present and Falconry in Europe—The Present.

Formerly classified as *Accipiter gentilis* spp., the northern goshawk has recently been divided into two species, the American goshawk (*Accipiter atricapillus* spp.) and the Eurasian goshawk (*Accipiter gentilis* spp.).[86–88] *A. atricapillus atricapillus* are found throughout North America, except for the west and extreme southwest. *A. a. laingi* are found on Vancouver and Queen Charlotte Islands off the coast of British Columbia, and *A. a. apache* range from southern Arizona and New Mexico into Mexico.[81,89,90] Goshawks are highly prized in falconry for their speed, agility, and versatility. The American goshawks differ from their Eurasian counterparts in that their color is darker dorsally and there is fine dark barring ventrally. As they mature, their iris progresses from gray to yellow, then orange, and finally red as they reach 4 to 5 years of age; Eurasian goshawks retain yellow-orange irises as they mature. A number of North American falconers prefer to hunt with the somewhat larger Eurasian species of goshawk such as the Russian or Siberian goshawk (*A. g. albidus*; Figure 55.22) or Finnish goshawks *(A. g. gentilis)*. Weight range for *A. a. atricapillus* ranges from 677 to 1014 g for males and 758 to 1214 g for females.[81]

Cooper's hawk. This hawk is mid-sized between the goshawk and sharp-shinned hawks. Cooper's hawks will catch a variety of game including small birds, mammals, and even larger-winged quarry such as small ducks. However, similar to goshawks they can be quite temperamental and aggressive toward the falconer when taken as an eyass. Techniques for raising, training, and hunting with imprint Cooper's hawks are well described.[84] Cooper's hawks are one of the more commonly used species in Mexico. Weights range from 302 to 402 g for male and 479 to 678 g for female Cooper's hawks.[81]

Sharp-shinned hawk. Sharp-shinned hawks, or "sharpies" as they are often called, are the smallest of the North American accipiters, yet they have the same drive and determination as seen in their larger cousins. Similar to the other North American accipiters, most falconers will choose to imprint this hawk and use it to pursue small-winged quarry. Weights range from 87 to 114 g for male and 150 to 218 g for female sharp-shinned hawks.[81]



FIGURE 55.22 Larger goshawk species, such as this Siberian goshawk *(A. g. albidus)*, are becoming more popular among North American goshawk enthusiasts. (Photo courtesy Darryl Perkins, used with permission.)

Falcons

Both large and small falcons are very popular among North American falconers, and many of the same falcons and hybrid falcons flown in other regions of the world are used to take quarry in North America (see also section on Falconry in the Middle East—The Present). Unique to North America are the American kestrel, prairie falcon, and the aplomado falcon. Smaller species such as merlins are generally flown from the glove in direct pursuit of prey and occasionally from a modest pitch. Larger species are generally flown from higher pitches to catch feathered game, although some falconers will also use them with sight hounds, in particular lurchers, to catch ground quarry such as jackrabbits. Peregrine falcons are the most popular falcon flown in Mexico, but aplomado falcons are gaining favor.

American kestrel. The American kestrel is the smallest and, perhaps, the most colorful falcon in North America and has a wide range of distribution across the continent. Their colorful plumage is only rivaled by the aplomado falcon, of which there are several subspecies (*A. femoralis* spp.). American kestrels are sexually dimorphic. Males possess a blue-gray crown with a reddish brown nape and back that is barred. The dorsal aspect of the wings is also blue-gray in color, and the dorsal aspect of the primaries is black with white spots. The tail is also reddish brown, with a usually white tip and a black sub-terminal band. Female kestrels have a similar coloration on the head, but the back and dorsal aspect of the wings and tail are reddish brown and heavily barred. Both male and female American kestrels have a pronounced malar stripe on the side of the head. Even with its small stature, the American kestrel is a fantastic falconry raptor and is one alternative for falconers to train when they begin their falconry journey. Used to hunt primarily small birds (sparrow, starlings) and rodents, the American kestrel is well respected in falconry. American kestrels are widely distributed across North America and are able to thrive in a variety of habitats, including urban and suburban locations. Males are slightly smaller than females. American kestrels are generally 97 to 150 g in body weight.[81]

Prairie falcon. The prairie falcon is a large falcon and is similar in size to the peregrine falcon. It inhabits the more arid and open regions west of the Mississippi River, and its range extends from central Mexico northward to southern British Columbia, Alberta, Saskatchewan, and Manitoba. Free-ranging prairie falcons are capable of taking a wide variety of prey, from small passerines to black-tailed jackrabbits *(Lepus californicus)*. Also requiring patience in handling, the prairie falcon is known for its tenacity and forceful airstrikes that can incapacitate, if not kill, its intended prey from a pitch. Prairie falcons generally weigh between 420 and 1100 g.[81]

Aplomado falcon. The range of the aplomado falcon extends from southern Texas well into Central and South America. Once common in the southwest United States, this falcon is considered endangered but is now being reintroduced in south Texas. Aplomado falcons are unique in their hunting habits. While most large falcons prefer not to pursue ground quarry, the aplomado is used in falconry to pursue avian and small mammalian quarry including cottontail rabbits (*Sylvilagus* spp.). Aplomado falcons generally weigh between 208 and 460 g.[81]

Merlin. The merlin is a small falcon but larger than the American kestrel. Merlins have a large distribution including breeding and wintering areas in North America, Central and northern South America, the Caribbean, as well as North Africa and Eurasia. There are three distinct subspecies in North America including *Falco columbarius columbarius* (i.e., Taiga or tundra merlin) that breed in the boreal forest from Alaska to Newfoundland and winter in the southern United States and southward; *F. c. richardsonii* (i.e., prairie merlin) breed on the northern Great Plains and winter there and southward; and *F. c. suckleyi* (i.e., black merlin) which are resident in the wet temperate forests of coastal British Columbia and Washington, with some birds moving southward in winter.[81] A number of North American falconers enjoy pursuing small avian species with merlins. Merlins generally weigh between 129 and 236 g.[81]

Eagles

Eagle falconry is relatively new to North America and practiced by a relatively small subset of falconers. The golden eagle is the most common species used, although there are some falconers that use nonindigenous species to catch game including the African crowned eagle, the ornate hawk-eagle, and others. Jackrabbits, both black-tailed and white-tailed *(Lepus townsendii)*, and larger quarry, such as coyotes *(Canis latrans)*, are common prey for eagles (Figure 55.23). Eagles are commonly hunted either from a soar while above the falconer or directly from the fist. Golden eagles generally weigh between 3 and 6.4 kg.[81] Falconers in the United States are not permitted to take and possess a bald eagle for falconry.



FIGURE 55.23 A golden eagle (*Aquila chrysaetos*) is about to come to grips with a black-tailed jackrabbit. (Photo courtesy © Aaron Allred.)

Osprey

Ospreys are one of the most widely distributed raptors in the world, yet descriptions of their use in falconry in North America are extremely rare. However, most recently, a more detailed description of husbandry and falconry training techniques designed for ospreys became available with the publication of *Ospreys in Falconry: Lessons Learned* and an online blog that further details the authors' experience while training an osprey available at https://ospreyfalconry.com.[91] Admittedly, there is still a lot to learn about this highly specialized raptor and its future applications in falconry.

Equipment and Training

Facilities

Facility requirements vary in North America; however, in general, most falconers must possess necessary facilities and equipment (see section on Falconry in Mexico and Table 55.1) before obtaining a raptor for falconry. Most falconers maintain their raptors in an outdoor aviary known as a mew. Requirements for mew design may vary between state and province and should allow the raptor to easily extend its wings, should provide appropriately sized perches for the species, allow easy access to water, should have at least one window with vertical bars, and protect the raptor from environmental extremes and potential predators. The roof of the mew may be solid, netting, or barred to allow varying amounts of exposure to the environment while also allowing the raptor full protection. Most raptors are housed individually in mews during the hunting season, although some species may be housed communally during the off-season or if used for breeding.

A weathering area is an outdoor facility that may be attached to or separate from the mews. This structure consists of a fenced area that may be covered by a solid roof, netting, or wire. Here the raptor is given monitored exposure to the elements. Raptors are generally tethered to a perch, with access to water, while in a weathering yard. Outdoor perches of appropriate design for a falcon, hawk, or eagle are necessary.

Equipment

This list of necessary falconry equipment includes a glove (gauntlet) of appropriate size and thickness for the raptor, at least one pair of jesses made of a pliable leather or synthetic material, at least one leash of appropriate length and thickness for the species being used to hunt, at least one swivel for use with the leash and jesses, a bath container for the raptor to bathe and drink, and a scale to weigh the raptor. Most, if not all, of the equipment can be made by the falconer, usually from leather. Other equipment such as bells, which are used to help locate the raptor when near the falcon, and telemetry, a system that helps locate a lost or difficult-to-locate raptor, are extremely important. In some regions of North America, the United States, for example, hybrid falcons are required to have two transmitters when flown free. Conversely, falconers in Mexico are not required to use a transmitter for any falconry bird. Falconry telemetry consists of a transmitter that is attached to the raptor and a receiver that receives the transmitter signal. Commonly used transmitters are available in very high frequency (VHF) or ultrahigh-frequency (UHF). However, many falconers are gravitating toward the use of a more efficient way to track and recover raptors with the use of lightweight GPS transmitters that use a satellite navigation system to provide the location of raptor even up to 100 miles away from the falconer. While other GPS systems are available, the most popular system in North America, as well as other parts of the world including Europe and the Middle East, is the Marshal GPS system (Marshall Radio Telemetry, Salt Lake City, UT; Marshallradio.com). In conjunction with a smartphone, this GPS system can provide real-time location of the raptor, location of the falconer, and additional parameters such as altitude, speed, climbing rate, temperature, and distance traveled by the raptors.

Feeding and weight management

Training a raptor for falconry is a time-consuming process. Food is introduced to the raptor early in the training process, either on the glove when it is being manned, on the lure, or given directly to the raptor. Manning occurs when the raptor is in close proximity to the falconer or on the falconer's glove, and the fear of the falconer is neutralized and diminished. Traditionally, weight management and controlling the amount of food offered have been used to motivate raptors to respond to the falconer's call and pursue prey for the falconer. However, falconers are now pushing the envelope when it comes to behavioral modification and using various behavior strategies to reinforce wanted behavior, rather than always relying on maintaining a raptor's weight within a specific range. Some initial weight adjustment may be necessary early in the manning and training process, and it is important to use the scale to track the raptors weight at all stages of manning, training, and hunting; however, food deprivation to dramatically reduce the weight of the raptors is discouraged. Training is different from manning; where the latter is a course of action to get the raptor acclimated and not fearful of the presence of the falconer, training is teaching the raptor what the falconer expects in order to become a successful hunting team and the raptor accepts the falconer as a hunting partner.

Training

All of the techniques described earlier (see section on Falconry in the Middle East—The Present) for training are also used by North American falconers including lures, kites, balloons (helium), fixed-wing remote-controlled airplanes, and drones. Lures are an indispensable piece of equipment for any falconer. Classically, lures are a leather sack in which weight is added, although lures can take any form, from a prey item to a plastic bag. Lures are introduced during the training process and a variable-sized piece of food is attached to it. The raptor quickly learns that it will be rewarded when it comes to the lure, thus making it indispensable in calling a raptor back to the falconer, even from great distances or heights.

Similar to the practice of falconry in other regions of the world, kites, balloons, and fixed-wing remote-controlled airplanes can be used as tools to condition a falcon or hawk, or as a training tool to help the falcon develop a higher pitch above the falconer. For the latter, drones, kites, and balloons excel. These tools are not necessary to train a falcon; however, they can be quite useful. Drones appear to be more popular than balloons or kites, as they do not require maintaining a tank of helium or depend on wind speeds, respectively. Not surprisingly, recommendations for training a falcon with a drone vary between falconers; however, one author provides a description of the technique including types of drones, necessary equipment (e.g., parachutes and bait release systems), and training technique.[92] In short, a falcon or hawk is allowed to grab a bait or lure suspended beneath the drone by a bait release system, which holds a parachute attached to the lure. The distance of the lure or bait from the drone and the distance from the parachute to the lure vary depending on the comfort level of the raptor when approaching the drone. The distance from the drone to the lure may be as much as 20 to 25 feet. Once the falcon climbs to the drone and grabs the bait or lure, a parachute is deployed and the raptors can descend to the ground. Falcons may drift some distance from where they took the bait/lure depending on the wind speeds and the height of the drone. When using a kite or balloon, the bait is suspended in a similar fashion but without the parachute. The bait line is instead attached to the kite or balloon line, and the falcon descends to the base of the line. Falconers in more confined areas must take into consideration the amount of open space available, surrounding trees, and manmade structures (telephone poles and lines) when drone training a falcon. In the United States, the height of the drone is limited by Federal Aviation Administration (FAA) rules for small, unmanned aircraft.

Quarry

Along with its diversity of habitat, North America likely has the greatest variability of game available to falconers. The term "game" is loosely applied to any species pursued by falconers and may also include species not generally considered food for the table. For example, falconers that hunt with American kestrels, merlins, sharp-shinned hawks, and other smaller raptors often opt to pursue English or house sparrows, rock pigeons, mourning doves *(Zenaida macroura)*, starlings, or small rodents. These are offered as food to these smaller raptors or even used to feed larger raptor species. The most common species hunted fall under the categories of small game such as squirrels, cottontail rabbits, jackrabbits or hares; upland game birds such as common pheasants *(Phasianus colchicus)*, northern bobwhite quail *(Calinus virginianus virginianus)*, Gambel's quail *(Callipepla gambelii)*, California quail *(Callipepla californica)*, sharp-tailed grouse *(Tympanuchus phasianellus)*, greater prairie chicken *(Tympanuchus cupido)*, sage grouse *(Centrocercus urophasianus)*; numerous migratory species such as waterfowl (Figure 55.24); and even fur bearers.

Most falconers try to match the raptor they use with the game available in their region. However, falconers commonly travel great distances to hunt game not readily available to them near their home. Falconers that hunt ground quarry are often colloquially referred to as "dirt hawkers," while those who pursue quarry with falcons are called "longwingers"; however, they are not mutually exclusive. Some longwingers



FIGURE 55.24 (A) A falconer prepares to release his tiercel gyr × peregrine falcon hybrid for a flight on ducks. (B) One author's (M.P.J.) tiercel gyr × peregrine on a drake mallard duck. Falcons dispatch their prey quickly by severing vital structures such as the spinal cord, jugular veins, and carotid arteries. (A, Photo courtesy Shawn Hayes, used with permission. B, Photo courtesy Michael P. Jones.)

will pursue ground quarry such as jackrabbits with falcons, and "dirt hawkers" will pursue quarries traditionally reserved for falcons with various species of hawks. The term austringer has been used to describe falconers who train and hunt with accipiters, more specifically goshawks, and the term is also sometimes applied to falconers who hunt with eagles. Regardless of the quarry, North American falconers adhere to the hunting season, permit, and bag and possession limit regulations as determined by each state, tribal or territory, province, or federal regulations. Some special consideration is given to falconers in the form of extended seasons. For example, while duck season for gun hunters in Tennessee may extend into the last week in January, falconers may hunt through the end of February as part of an extended falconry duck season in the Mississippi Flyway as falconry has no impact on game species numbers. Should the falconer's bird accidentally catch a protected species, either during the closed or open hunting season, the raptor is allowed to consume the prey, but the falconer is not allowed to remove the prey's carcass from the field and must "let it lay." Some states or provinces may require reporting the date and location of incidental take of endangered or protected species.

Dogs

Dogs are often an instrumental part of the falconry team. Falconers pursuing upland game with falcons often chose pointing or versatile hunting dog breeds (e.g., English Pointers, English Setters, Spaniels, Vizslas, German Shorthaired Pointers, etc.; see also section on Falconry in Europe—The Present), while those pursuing ground quarry and squirrels may choose to use Dachshunds, various terrier breeds (e.g., Jack Russell terriers, Decker terriers, Jagdterriers, etc.), beagles, Feist and Cur breeds, as well as sight hounds or lurchers (sight hounds crossed with a working dog breed [terrier, herding breed, or scent hound]). Sight hounds such as greyhounds and lurchers are used to pursue prey such as jackrabbits under falcons, hawks, or eagles.

Falconry-Based Bird Abatement and Pest Control

See also section on Falconry in the UK—The Present.

While not considered falconry in the truest sense, a number of falconers in North America use raptors to control bird species considered pest or nuisance birds (e.g., starlings and pigeons) to reduce lost crop loss, property damage, public health concerns, and reduce airstrikes by bird at airports.

Innovation

Free of the constraints of long-established falconry traditions, North American falconers have contributed significantly to the practice of falconry through innovation and advancement of the practice of falconry.[44] These innovations include (1) establishing captive breeding methods to facilitate the production of purebred and hybrid raptors for use in falconry; (2) the development of radio telemetry systems for location and recovery of lost hawks and falcons; (3) the development of the American hood, which uses a lighter leather and demonstrates exceptional hood-making technique in delivering a hood with a beak-opening that contours to the falcon's head; (4) developing a cure for frounce (trichomoniasis); and (5) introducing the red-tailed hawk and Harris's hawk to international falconry (Kent Carnie, personal communication, 2020).[44]

FALCONRY IN NORTH AMERICA—THE FUTURE

The future of falconry looks promising, although varying degrees of optimism, pessimism, and skepticism exist. Some falconers see the eminent loss of falconry as a sport, but most falconers agree that there will be challenges to overcome. Likely, the single greatest challenge to falconry in North America will be the ever-increasing expansion of intensive agricultural practices, especially those that employ the use of pesticides and other chemicals harmful to the environment. Falconers often see firsthand the rapid loss of habitat and suitable places to train and hunt with their raptors. Falconers often reminisce of their favorite hunting areas that were once rich in game but are now lost to intensive agriculture, housing developments, and shopping malls. Habitat loss combined with anthropogenic impact and its effect on the environment, ecosystems, and biodiversity (e.g., climate change) may continue to result in declining game populations (e.g., multiple species of cottontail rabbits, snowshoe hares *[Lepus americanus]*, multiple species of quail such as the California quail, sage grouse, etc.), and game that is increasingly harder to find. Some falconers in North America believe that the future of the sport is bleak and suggest that younger generations practicing today may see the end of falconry as a field sport. Perhaps the future of falconry for some who have lost habitat and game may be the pursuit of micro-falconry, the flying of small raptors (e.g., sharp-shinned hawks, merlins, and kestrels) in a way that does not require expansive farm or woodlands to practice the sport.

Another significant challenge to the future of falconry in North America is regulations that may create an environment of mistrust between falconers and various regulatory agencies. In fact a number of falconers believe the sport should not be constrained by regulations at all. Operation Falcon represented a dark time for North American falconers. While its claims were unsubstantiated, it cast a negative light on falconry, and falconers became reluctant to openly share details of the sport in order to protect it from those that would not appreciate its value.

In 2008, the USFWS announced a major overhaul of its falconry regulations that went into effect on January 1, 2014. First, the new falconry regulations would eliminate the need for dual state/federal permit, and the USFWS would approve falconry regulations that "reflect current practices in the traditional use of raptors for sport hunting." These regulations would be administered under state, tribal, or territorial laws and regulations, with the understanding that state, tribal, or territorial laws may be more restrictive, but not less restrictive, than federal regulations. Second, USFWS established an electronic reporting system to record acquisition, transfer, or loss of raptors used for falconry. Third, the new regulations allow experienced falconers to assist federal and state-permitted wildlife rehabilitators with conditioning and release of raptors back to the wild. The Bald and Golden Eagle Protection Act, which was originally enacted to protect the bald eagle, then amended in 1962 to include golden eagles, regulates the taking, possession, and transport of eagles or their parts (inactive

or active nests, egg, feathers, blood, etc.). In part, it also provides regulations for the take and use of golden eagles for falconry in the United States.

Falconry, at best, is a fringe sport when compared to other hunting and fishing activities. Overall, members of the public are aware of the sport from something they have seen, read, or seen on social media. Even those aware of the sport, unless they know a falconer, have little understanding of the day-to-day responsibilities needed to keep, train, and hunt with raptors. Perhaps the opinion of the public is good, yet falconers must still protect and defend their passion from antihunting, animal rights organizations.

Falconers, at their core, are really conservationists practicing an ancient and highly participatory form of bird watching. For falconry to move forward, it will be necessary for NAFA, AFC, and each state's falconry association to continue their strong support of conservation and conservation efforts, to cultivate partnerships and relationships, to engage and educate the public, as well as mend fractured relationships with regulatory agencies/agents. For this "way of life" to flourish in today's society, public education must be built into our activities so that transparency rather than traditional stealth mode becomes the norm (Dr. Dave Mcruer, personal communication, 2020).

Finally, encouraging and supporting diversity and inclusion should be a driving force for falconry. Underrepresented minorities (URM) are becoming increasingly interested in falconry, and the numbers of falconers in this demographic are increasing. Unfortunately, national and falconry organizations have not historically collected demographic data regarding falconers who are members of URM groups or other demographics. Women are a significant and increasing percentage of the total numbers of falconers in NAFA, representing around 11% in the United States (Figure 55.25). For comparison, approximately 27% of members of the Ontario Hawking Club are women, and approximately 50% or more of the Québec Falconry Association's members are women (Martin Geleynse, personal communication, 2020). The increased knowledge, perspectives, insights, and talents that diversity would bring to national and state organizations and falconry would increase their strength, flexibility, adaptability, innovation, problem-solving, and ultimately performance as an organization and group.



FIGURE 55.25 Jamaica Smith with male golden eagle *(Aquila chrysaetos)*, Arizona, USA. (Credit to the photographer, Craig Golden.)

FALCONRY IN CENTRAL AND SOUTH AMERICA—THE PAST

Falconry is practiced in many Central and South American countries today, using a wide variety of native raptor species to pursue a broad range of quarry in a diverse array of habitats. South American countries that currently have active falconry clubs include Argentina, Brazil, Chile, Columbia, Cuba, Ecuador, El Salvador, Paraguay, Peru, Uruguay, and Venezuela. While the earliest records of falconry in the "New World" arise quite early, it appears that falconry died out sometime in the 17th century until a revival in the mid to late 20th century in most of these countries.

The early historical record for falconry in the Americas is somewhat scant. There is no firm evidence that falconry was practiced prior to the arrival of European explorers.[44] When Columbus arrived in the Americas in 1492, hawk bells intended for trade with locals were included among the cargo in his ships (See section on Falconry in Mexico for historical perspective).[93]

In the colonial period, much of South America was originally under Spanish control as part of the viceroyalty of Peru, founded in 1543. This vast viceroyalty initially stretched from modern-day Panama in the north to Chile and Argentina in the south. To the east was the Portuguese colony of Brazil. Garcilaso de la Vega (1539–1616) describes falconry in two different passages in his Comentarios Reales. In the first, he describes soldiers traveling with slow-moving llama caravans, taking both dogs and hawks with them to hunt during the day. A few pages later, he further describes a man from Seville who attempted to train a dark-colored falcon that resembled a "nebli"; however, he was never able to successfully take quarry with it.[94] The first illustration of a falconer in South America is a native person employed as a falconer, with hawk in hand and two dogs, in a manuscript by Guzman Pomo de Ayala (1615).[95] In a book entitled *Histórica Relación del Reino de Chile y de las Misiones y Ministerios que Ejercita en la Compañía de Jesús*, Alonso de Ovalle described the use of casts of trained falcons for hunting southern lapwings *(Vanellus chilensis)*.[96]

Further north, the viceroyalty of Spain encompassed a large area of territory extending from the southwestern United States, Mexico, Central America, and Florida. The second viceroy of Spain, Luís de Velasco, who ruled from 1550 to 1564, possessed an extremely tame falcon that was celebrated to ride upon his fist unhooded. Additionally, he kept several different species of falcons.[44] His son, Luís de Velasco II, who spent time as both viceroy of New Spain and Peru, employed a royal falconer to care for his birds.[44]

There was a great demand for hawks from the New World in the European courts (see also section on Falconry in Europe—The Past). As early as 1494, King Ferdinand II sent

a letter to Christopher Columbus ordering him to bring back as many falcons as possible to Spain.[63] In 1501, an official Royal Falcon Catcher of the Indies was appointed to help facilitate exportation of select falcons to Spain. From paintings of neotropical raptors in Europe during this period, we know that species that would have been previously unfamiliar to Europeans, such as the ornate hawk-eagle, arrived. One of the most mysterious of the species is the Aleto, whose identity has been debated among several authors, though the aplomado falcon seems the most likely.[44] In 1616, Ferreira wrote of Nebris imported from the Castillian Indes, Alettes from the Spanish Indies and Brazil, and describes a bird that sounds similar to an ornate hawk-eagle.[97] In general reference to falconry, however, he describes the "oblivion into which it has fallen" and his hope that his work may revive interest. This, however, was not to be.

Although it is not known when falconry died out in the Americas, it seems likely to have paralleled the precipitous decline of the sport in Europe. In Spain and Portugal, falconry was rarely practiced after the mid-17th century.[98] Falconry does not appear to have resurfaced in Central or South America until the 20th century. In Mexico, there was a "Mexican Association of Falconry" in 1950, which like early attempts at forming formal falconry organizations in the United States, was short-lived.[59]

FALCONRY IN CENTRAL AND SOUTH AMERICA—THE PRESENT

Today there are falconers in many Central and South American countries, with multiple active falconry clubs. There is a tremendous diversity of species available for falconry in these countries, with some neotropical species being among the most attractive. The ornate hawk-eagle, Chilean hawk *(Accipiter chilensis)*, black-and-white hawk-eagle *(Spizaetus melanoleucus)*, bicolored hawk *(Accipiter bicolor)*, collared forest falcon *(Micrastur semitorquatus)*, bat falcon, orange-breasted falcon *(Falco deiroleucus)*, and other species have been trained. Familiar species often flown in other countries include peregrine falcons, aplomado falcons, and Harris's hawks.

Falconers in these countries are frequently involved in abatement or captive breeding. Veterinarians are often involved in the care of captive birds within breeding collections. Information gathered from captive breeding or rehabilitation efforts may be important in organizing conservation efforts, especially for species declining in the wild.

FALCONRY IN CENTRAL AND SOUTH AMERICA—THE FUTURE

Similar to falconry in Mexico, falconers in Central and South America face challenges that may affect the future of the sport in these regions. Agencies that would develop and oversee regulations for falconry seem to lack interest and find it easier to prohibit the capture of wildlife than to develop formal regulations (Raul Ducoing, personal communication, 2020). Even with a number of falconers acting as consultants for their respective governments, the lack of impetus for authorities to create, vote for, and implement regulations is likely due to the low number of individuals participating in falconry (Raul Ducoing, personal communication, 2020). This is the situation in Panama, Ecuador, and Peru. However, Argentina has falconry regulations, but they are scant (Raul Ducoing, personal communication, 2020).

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CHAPTER 56

Toucans and Hornbills

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While not taxonomically related, the toucan (order Piciformes) and hornbill (order Bucerotiformes) have large beaks and skull structures that provide consistent challenges to the veterinarian. Similar concerns exist in their nutritional management with regard to iron storage disease (ISD).

TOUCANS

Janis A. Raines

TAXONOMY AND ZOOGEOGRAPHY

Toucans are classified in the order Piciformes, which is comprised of four families: Indicatoridae (honeyguides), Capitonidae (barbets), Picidae (woodpeckers), and Ramphastidae (toucans, toucanets, and aracaris). The four families share similar characteristics: zygodactyly, cavity nesting, and the adults lacking down feathers.[1–3] Ramphastidae are distinguished by their colorful beaks that are overproportioned to body size. However, the internal bill structure consists of a network of bony septa that give strength while reducing its mass.[4,5] The family Ramphastidae includes 6 genera, 34 species, and 78 taxa, with the largest genus *Pteroglossus* ("feathered tongued") containing 14 species. Ramphastids are neotropical and therefore found only in Central and South America. They inhabit varied habitats within their range, from lowland rainforests to high-altitude cloud forests. While most stay below 100 to 1200 m in altitude, some species may be found at elevations up to 2700 m. The populations are stable within the home range, with only a single species considered endangered (yellow-browed toucanet, *Aulacorhynchus huallagae*).[1,2]

ANATOMY

Serrated bill tomia is a characteristic of all toucans; however, these develop slowly and may not be noted in juveniles. A structural component found only in Ramphastidae is a modification of the nine caudal vertebrae such that their extreme range of motion allows toucans to place their tail over the head. This position, assumed by toucan species while sleeping, involves laying the beak up over the back, tucking it under the scapular feathers, and then tipping up the tail to form a "feather ball".[2] This positioning is presumed to be an adaptation for cavity dwelling, but they also are noted to assume this position on open perches. Sexual dimorphism is uncommon, with sexually dimorphic plumage seen in only two genera (*Selenidera* and *Pteroglossus*).[2] Bill length and overall body size are typically larger in sexually mature male toucans than in females, but results can be inconsistent, so DNA sexing or endoscopy is more reliable. The tongue reaches lengths up to 15 cm and is laminated along the sides, with the laminae becoming more brush-like along the tip.[2] The iris of several pale-eyed species has dark areas rostral and caudal to the dark pupil giving the appearance of a slit pupil, commonly referred to as the glitch. At least three species of toucans have a postorbital ligament, which has unknown significance. The trachea normally deviates ventrally at the thoracic inlet, which is seen radiographically (Figure 56.1).[3,6] Toucans lack an ingluvium (crop), possess a poorly muscularized ventriculus, and have no ceca. The spleen is long and slender, and this family has a gallbladder.[7,8] Toucans are not strong fliers and tend to lose altitude as they fly long distances, which is why their flight is undulating with a series of flaps alternating with glides. They make a characteristic noise while flying due to narrowed (emarginate) primary feathers along the distal edge of the wings.[1]

BEHAVIOR AND REPRODUCTION

Toucan species live in large flocks or minimally have overlapping territories. However, during the breeding season, the pairs will separate into a defended territory that can cover up to 30 hectares. Breeding cycles for toucan species in the wild correspond with the rainy seasons, so breeding behaviors may be stimulated in human care by simply providing regular heavy showers daily while simultaneously increasing food abundance. Typical breeding age (2.5–3.5 years of age) corresponds with acquisition of full adult size and plumage.

Time should be taken to pair toucans compatibly. It is recommended to house birds adjacent to one another with visual contact for some time to encourage proper bonding behavior. The time interval needed to ensure good pair bonding is very individualized and hinges on the interpersonal interactions of the birds. Regular observations can help determine the timing. After this interval, the pair can be introduced into a very large aviary with minimum dimensions of 4.6 m^2 (50 ft^2) for smaller



FIGURE 56.1 Radiograph of keel-billed toucan *(Ramphastos sulfuratus)* demonstrating normal tracheal deviation at the level of the thoracic inlet *(arrow)*.

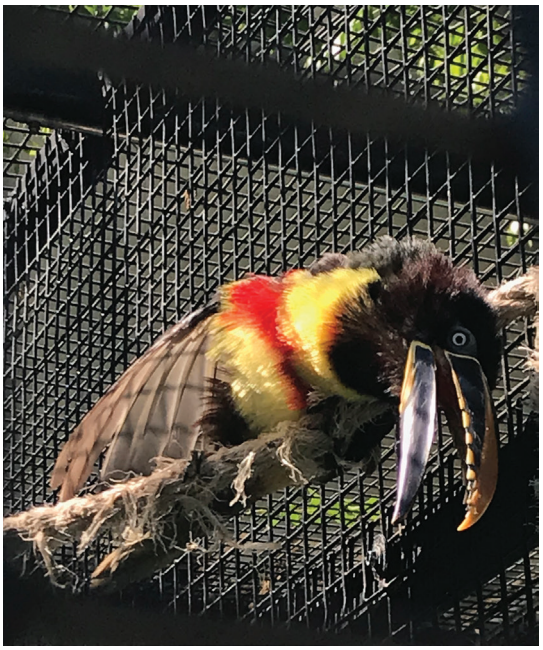


FIGURE 56.2 A chestnut-eared aracari *(Pteroglossus castanotis)* sunbathing.

species and 9.3 m^2 (100 ft^2) for larger aracaris and toucans. The aviary should contain live shrubs or structures to allow hiding locations in the advent of aggression. The aviary design should also maximize the long dimension to provide adequate flight distance. Promising signs of a successful pairing include allopreening, sitting together on a perch, feeding one another, or gentle bill tapping. Signs of aggression and incompatibility include chasing and extreme bill sparring. It is not uncommon to have fatal aggression between an established pair. Similarly, intense aggression may occur toward other pairs, requiring visual barriers between aviaries.[9]

Toucans are cavity nesters but rarely do their own excavation. Instead, they commonly use abandoned woodpecker cavities or rotted ends of branches and tree trunks. An integral part of pair bonding is the preparation of the cavity for nesting. In the wild, a breeding pair may use the same cavity for multiple years. Aviculturists can offer hollowed out palm logs to encourage breeding behavior. By filling the empty cavity of the log with wood shavings or moss, excavation behaviors can be stimulated. More basic nest boxes made of plywood can also be used, provided that nestbox dimensions are appropriate. Common dimensions for nest boxes recommended for larger toucan species measure from 70 to 100 cm in length and 20 to 30 cm in diameter, with an 8- to 10-cm-wide entrance hole. For aracaris and toucanets, the measurements are 70 to 100 cm long, 12 to 15 cm in diameter, and a 6- to 7-cm-wide entrance hole.[3] The primary difference between these is the interior dimension as this has to allow for accommodation of the longer bill length in the larger toucan species.

Ramphastids lay two to four small white eggs. Toucans can be good parents, but it is very common to pull the eggs for artificial incubation to ensure more consistent incubation parameters in captive breeding settings. This must be considered when constructing nest boxes for these species. The depth must be such that eggs (or young) can easily be reached, or an access door must be provided. If eggs are to be artificially incubated, typical parameters are 36.9 to 37.2°C (98.4–99°F) with relative humidity maintained at 65%. Incubation has an average length of 16 days. Typically, *Ramphastos* species have a slightly longer incubation (~18 days). The young are altricial and are born with tubercled heel pads, known as a tarsal-metatarsal callus. These heel pads disappear at the same time as the opening of the eyelids, at approximately 3 weeks of age. Along with a rather pendulous abdomen, this feature is thought to function as a stabilizing mechanism to keep the chicks tilted upward for easier parental feeding in the nest log. Further adaptations for a cavity lifestyle are a prognathic mandible, which begins to recede around 14 days of age, and an up-tilted uropygium. The chicks are fed throughout the day because of lack of an ingluvium. A similar schedule should be followed when chicks are being hand-reared. Feedings of small pieces of food multiple times throughout the day is most successful. Chicks typically fledge after no less than 40 days (see also Chapter 44).[9,10]

Toucans will often sunbathe after a bath by spreading feathers and draping these over a branch, which can be misconstrued as a sign of illness (Figure 56.2). Toucans also pant with an open bill to allow for cooling through evaporative action, which should not be mistaken as a sign of hyperthermia. It does bear noting that—regardless of their tropical distribution—these species can be sensitive to extreme heat, yet surprisingly tolerant of exposure to extreme cold (−6°C/20°F) as long as they are provided shelter, an adequate heat source (can be achieved using a 250 W heat bulb), and have been acclimated properly to cooler temperatures.[1,2,7]

Toucans are also mischievous both in the wild and in managed care. Their curiosity makes them both a welcome and unwelcome addition to a mixed species aviary as they have been known to destroy plantings and nests of other species. They will pick up and consume inedible objects found in their enclosures. However, as they do have the ability to regurgitate these items, foreign body impaction or gastrointestinal obstruction is not a common occurrence.[11,12]

NUTRITION

Within the varied habitats that these birds inhabit, an almost infinite variety of fruiting plants are available for feeding. While these species are primarily frugivores, it is important to remember that toucans are opportunistic omnivores. They have been known to rob nests and feed on small vertebrates, and nestlings and eggs of other toucan species as well as other avian species. The offering of insects during the nesting season can reinforce proper neonatal care. Toucans have an unusual feeding style that involves tossing a food item up in the air, catching it in an open beak, and swallowing it. Toucans are particularly prone to hemochromatosis (iron storage disease, ISD), and iron in their diet should be limited (see section on Iron Storage Disease). A variety of fruits have successfully been fed to toucans with the hypothesis that reducing citrus and high-iron–containing fruits in the diet may prevent hemochromatosis. Some aviculturists have used dietary tannin to bind dietary iron and decrease its absorption.[13] This approach has not been proven to be effective; however, it has not been found to have a detrimental effect on gastrointestinal health. In managed care, it is common practice to supplement diets with paprika or canthaxanthin to maintain bright coloration of the beak and periorbital skin. Toucans must be provided with large water-filled containers to allow both the necessary room to dip the water up with the beak as well as bathing.

MANAGED CARE AND VETERINARY CONSIDERATIONS

As detailed previously, toucans are not strong fliers, but they are very strong bounders, often moving about the aviary through leaps and hops. They are able to cause significant damage to their bill by colliding with caging walls and furniture, especially during displays or while preparing the nest log. This issue can be particularly problematic in young birds with rapidly developing beaks. Depending on the severity of bill damage, repair can be attempted using dental epoxies and, more recently, three-dimensional (3D) printing (see Chapter 31 and the section on The Hornbill Casque in Health and Disease).

Preventative medicine protocols such as those applied to other avian species can be successfully applied to ramphastids. Health screenings via blood analysis and radiographs can be useful to monitor current health status. These can be performed opportunistically, but regular fecal analysis is key to monitor parasite load. Much like hornbills, inhalant anesthesia is delivered via modified facemasks made from bags or plastic bottles (Figures 56.3 and 56.4). All other veterinary procedures can be performed similarly to other avian species, with the preferred venipuncture site being the right jugular vein.[3,14] The one caveat to this is the surgical approach to visualize the gonads, which is slightly more dorsal than that used in psittacines so as to avoid the more distended intestinal loops.[7,8]

The most common infectious diseases reported in toucans are ulcerative enteritis (*Clostridium* spp.), mycobacteriosis, herpesviral hepatitis, yersiniosis, candidiasis, aspergillosis, and capillariosis (Table 56.1).[15–17] Of these, *Capillaria* infection is probably the most common cause of mortality in toucans in managed care. Commonly, clinical signs of *Capillaria* infections include lethargy, weight loss, hypoproteinemia, and anemia. The most successful therapeutic regimen involves



FIGURE 56.3 Keel-billed toucan *(Ramphastos sulfuratus)* in modified 1-L fluid bag as facemask for anesthetic induction.



FIGURE 56.4 Modified fluid bags for use as anesthetic facemask *(above)*, and fluid container with bandaging material to contain anesthetic gas being delivered *(below)*.

removing exposure to reinfection, which may only be accomplished by hospitalization on a concrete floor, and an intensive parasite control (fenbendazole 50–100 mg/kg PO for 5–7 days, alternating with or in conjunction with ivermectin 0.2 mg/kg PO/SC/IM for 3–4 treatments given 1 week apart).[3,7,8,14]

Historically, of noninfectious diseases, diabetes mellitus was commonly reported in toucan species, but reports have decreased in more recent years.[18] It is unknown if improvements in diet and management are contributing factors to this change in incidence.

HORNBILLS

Kathryn C. Gamble

TAXONOMY AND ZOOGEOGRAPHY

Hornbills were previously included in the order Coraciiformes, but due to their limited anatomic and behavioral similarities with other birds in that group, they were reassigned to the order Bucerotiformes.[19] Together with the clade of ground hoopoes and wood hoopoes, to which hornbills have been linked ancestrally, they are included in the overarching superorder Bucerotes.[19] By their colorful oversized

TABLE 56.1

Most Commonly Reported Infectious and Noninfectious Diseases in Rhamphastidae[5,11]

Bacterial	Viral	Mycotic
◆ *Clostridium* enteritis ◆ *Mycobacteria* spp. ◆ *Yersinia pseudotuberculosis* ◆ *Salmonella carrau* ◆ *Salmonella enterica* serovar Heidelberg ◆ *Salmonella typhimurium* ◆ *Providencia* spp.	◆ Herpesvirus ◆ Cutaneous poxvirus	◆ Ventricular candidiasis ◆ *Aspergillus*

Parasitic	Noninfectious
◆ *Capillaria* ◆ Cestodiasis ◆ Hepatic hemoprotozoan cysts ◆ Biliary and intestinal trematodiasis ◆ Amoebiasis ◆ *Sarcocystis* ◆ *Giardia*	◆ Hemachromatosis ◆ Diabetes mellitus ◆ Trauma ◆ Renal neoplasia ◆ Cardiovascular disease ◆ Arteriosclerosis ◆ Pancreatic adenocarcinoma ◆ Degenerative joint disease

beaks, these strictly Old World species bear some phenotypic resemblance to the New World toucans of the order Piciformes; however, it continues no further than this feature as described elsewhere in this chapter (see section on Toucans).[19] The elaborate casques define this order and vary by each of the individual species as detailed in the section on The Hornbill Casque in Health and Disease.[19,20]

The order contains two families: Bucorvidae and Bucerotidae. The former is limited to ground hornbills within the genus *Bucorvus*. These two African *Bucorvus* species are considered the ancestral hornbill.[19] The remaining 55 species are distributed nearly evenly between 4 African and 10 Asian genera as distinguished by body size, habitat, chick-feeding strategies, and characteristic color patterns and calls (Table 56.2).[19,20] *Tockus* are considered the earliest taxonomic diversion of the African species, and *Anorrhinus* the comparable separation for the Asian species.[19,20] While African species are divided evenly between habitats of woodland and savannah or evergreen forest, only one Asian species lives in any habitat other than evergreen forest, and most Asian species are island inhabitants.[20] Interestingly, although no extant genera are found in both Asia and Africa, taxonomic bonds between the African *Bucorvus* and the Asian *Buceros* have been identified by a shared genus of *Bucerophagus (Mallophaga)* chewing lice, which suggest an earlier radiation.[19–21]

ANATOMY

Shared anatomic features of the Bucerotidae include[20,22,23]: (1) united maxillopalatine bones (desmognathous arrangement); (2) primitive stapes within the middle ear; (3) syndactylism of pedal digits 2 to 4; (4) bilobed kidneys due to the absence of the median lobe characteristic of the trilobed kidney of other birds; and (5) lack of the following structures—ceca, ambiens muscle, and stylohyoideus muscle. The beak is curved and always meets at the rostral tips where it is used dexterously to manipulate food items.[19,20] However, with its absent retractor muscle, the fleshy short tongue is not useful for food manipulation, so the beak is also used to toss these items into the caudal oral cavity for ingestion.[19,20] Due to the placement of their eyes and positioning of the beak, hornbills are able to scan overhead and see to and over their bill tip with excellent binocular vision that further facilitates food manipulation.[19] Species of *Buceros* and *Tockus* have an external directional of beak growth that parallels the inner surfaces such that smooth tomia develop, although only in the *Tockus* do the tomia actually appose.[19] In the other

TABLE 56.2

Hornbill Genera as Classified by Adult Body Size and Origin

	Small (<500 g)	Medium (500 g–2 kg)	Large (>2.5 kg)
African	*Tockus*[s,w,fo;i] (14) Jackson's *(T. jacksoni)* Red-billed *(T. erythrorhynchus)* Von der Decken's *(T. deckeni)* *Tropicranus* (1)	*Bycanistes*[fo;fr] (6) Silvery-cheeked *(B. brevis)* Trumpeter *(B, bucinator)* Brown-cheeked *(B. cylindricus)* *Ceratogymna*[fo;fr] (2)	*Bucorvus*[s;c] (2) Ground *(B. leadbeateri)*
Asian	*Ocyceros*[fo,s;fr] (3) Indian grey *(O. birostris)* *Penelopides*[fo;fr] (6) Tarictic *(P. panini)*	*Anthracoceros*[fo,w;fr] (5) Asian pied *(A. albirostris)* *Rhyticeros*[fo;fr] Wrinkled *(R. corrugatus)* Blyth's *(R. plicatus)* *Anorhinus* (1) *Berenicornis* (1) *Ptilolaemus* (2)	*Buceros*[fo;o] (3) Giant *(B. bicornis)* Rhinoceros *(B. rhinoceros)* *Rhinoplax*[fo;o] Helmeted *(R. vigil)* *Rhabdotorrhinus* *Aceros* (1)

(#) Species count.
Habitats: *fo,* forest dwellers; *s,* savanna; *w,* woodland.
Primary food habits: *c,* carnivores; *fr,* frugivorous; *i,* insectivorous; *o,* omnivorous.

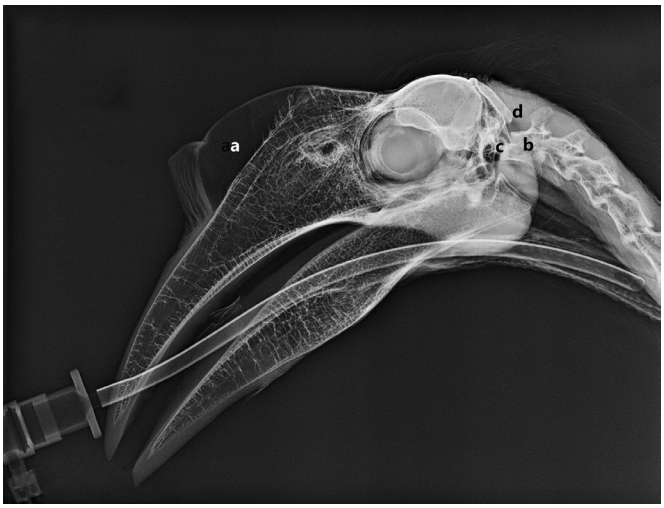


FIGURE 56.5 Axial skeleton anatomy characteristic of hornbills as represented by an adult male wrinkled hornbill, *Rhabdotorrhinus* (*Aceros*) *corrugatus,* including: *(a)* casque, *(b)* fused atlas and axis, *(c)* basio-occipital articulation, and *(d)* supraoccipital articulation. (Courtesy Kimberlee Wojick, Roger Williams Park Zoo.)

hornbill species, beak growth occurs such that jagged edges develop along the tomia for crushing and holding food items.[19] *Aceros* develop annular grooves at the caudal rhinothecal base that allow some staging of age until full sexual maturity is achieved.[19,20] Additional to the casque, and likely a result of it for needed structural support, the hornbill's axial skeleton presents two other unique features: (1) fusion of the atlas and axis and (2) two vertebral articulations with the caudal skull at the basio- and supraoccipital condyles (Figure 56.5).[19,20,23] Ground hornbills have 15 cervical vertebrae, which is considered primitive to the 14 cervical vertebrae of the other Bucerotidae; they also lack carotid arteries. Ground hornbills furthermore have a separate tendon that connects the pelvis to the femur allowing them to reach their pes to the head rather than indirectly over the wing, as is typical for many birds.[20]

Plumage coloration of hornbills is monochromatic in gray to black, and occasional brown, but patterns of white are striking and localized for visual signaling, especially during flight.[19,20] Their feathers are coarse and have a fluffed texture as more hair than plume, and palpebrae present marked cilia (eyelashes).[19,20] All hornbill species lack ventral covert feathers on both primary and secondary feathers, which reduces muffling during flight, such that larger-bodied species utilize this sound as part of their auditory signaling.[19,20] Much more resplendent than the plumage are the casque and beak, which are colorful in species-specific hues and patterns. Additionally, the *Buceros* and *Rhinoplax* species utilize their carotenoid-rich uropygial gland secretions as a cosmetic applied to their beak and casque until they are intensely orange, yellow, or red-orange.[19,20,23] Males of all species tend to be larger than the females in body size and wing span and, most dramatically, in bill length, with an 8% to 30% variation.[20] Aside from these morphometrics, patterns of the plumage and coloration of beak and casque and, in some *Buceros*, *Rhyticeros*, and *Ocyceros* species, the iris make all hornbill species sexually dimorphic as mature adults.[19,20] However, juvenile hornbills vary widely by species as to whether they resemble more the male or female parent until sexual maturity.

BEHAVIOR

Hornbills spend an inordinate amount of their behavioral and diurnal time budget preening their own plumage and that of social companions.[19] This care prominently features behaviors of bathing in open water or other sources of moisture in addition to feather manipulation by the beak. Sunbathing, sometimes employed as a warming mechanism, includes unusual, contorted posturing.[19,20] Generally heavy-bodied birds, hornbills tend to hop more than walk on the ground. Exceptions are the ground hornbills and clucking *Tockus* species that have proportionately elongated tarsi and tend to stride.

REPRODUCTION

Hornbills are long-lived with extended intervals for successful reproduction often predicated by available food resources.[19] It would be remiss to continue without description of the sealed cavity nesting strategy of all hornbills, except *Bucorvus*, which use open cavities without a seal.[19,20] This behavior has an extensive impact on the taxonomic features of anatomy, physiology, and behavior. Hornbills are seasonal breeders. African savannah and woodland species nest before the rains commence, and ground hornbills nest in later months once vegetation has grown. Asian species nest at the onset of the cool dry season. In the United States, *Buceros* breeding was documented in January through April.[24]

Extended courtship and conspecific feeding occur between adult pairs that culminate in confirmation of an identified natural cavity as the nesting site suitable to the female.[19,20,24] Repeated copulations occur during the latter stages of this period, and extended fertility of spermatozoa within the female reproductive tract of up to 3 weeks has been established.[19,20,22] Ultimately, the bred female enters the cavity and using the trowel-shaped beak, generally will complete all the entry sealing to a narrow vertical slit.[19,20,24] Material used for the seal includes soil, feces, and soft foods.[19,20,24] Specifically in *Bycanistes* and *Ceratogymna*, the male produces mud pellets from ingested dirt that are carried to the nest for the female to use. Male *Tockus* and *Anthracoceros* provide mud directly to their mate, with this material essentially being the sole product used for the seal by the latter genus.[19]

The female commences oviposition of oval eggs with pitted shells, and asynchronous incubation begins immediately due to the confines of the nest.[19,20,23] Smaller species lay up to eight eggs at daily intervals, while larger species lay fewer eggs at a longer interval of up to 5 days. Eggs are incubated for 23 to 42 days, with smaller species being at the lower and larger species at the higher end of this range.[19] Females of *Tockus*, *Buceros*, and *Bucorvus* depart the nest at the midnesting interval, then the chicks reseal the chamber opening using food and feces so that they can resume their protected growth period.[19] Similar to the female's entry, females and chicks depart in an orderly fashion from the chamber with first the head and neck, then one wing ahead of the torso, followed by the second wing, and the legs.

In the female, concurrent with the incubation of the eggs, an entire molt is initiated starting with all rectrices (10) and rapidly progressing to the remiges (10 primaries and 12 secondaries), such that for most of the incubation and nesting period, she remains flightless.[19,20] Dependent on the species, the molt is completed either at midpoint of chick rearing or at chick emergence from the nest. Males complete a more typical molt of only a few feathers at a time and on opposite sides of the body, and generally following complete emergence of the family.[19,20]

While it has often been considered from the vantage of high dependence of the female on the male in this arrangement, it must be considered that the male is equally reliant on its mate's success within the cavity to assure its contribution to the next generation. Due to the extensive work imposed by this unique nesting activity, roughly 30% of the hornbill species employ cooperative feeding and nest assistance by immature or unpaired, but related, males.[19,20]

NUTRITION AND FEEDING

Feeding strategies of the hornbills range from highly carnivorous to essentially frugivorous. Fruits are selected for their moisture content and lipid and carbohydrate components and include syconia and drupes.[19,20] Hornbills, as a rule, do not drink water but obtain water from their foods.[1] As these birds act as seed dispersers, the selected fruits separate easily from their seeds following ingestion and then either are disgorged or defecated intact.[19] All hornbills, even strict frugivores, increase their protein intake during the breeding and nesting season due to the high demands of the females' incarcerated molt and growing chicks. This diet change generally is accomplished by whole prey consumption and increased omnivory.[19,20,23] Figs (*Ficus* spp.) are emphasized in the diet when they are in season as they contain a high content of protein at 6% to 12% by weight and increased calcium.[19]

Characteristic food carriage to the female adult and nestlings is used as differentiation for genus and species[19,20]: *Bucorvus* carry multiple prey items bundled along the beak; *Tockus* carry single prey items, usually insects, at the beak tip; *Ocyceros* and most other species swallow multiple fruit items into the cranial gastrointestinal tract to be disgorged singly at the nest, although the larger hornbills that have a higher degree of omnivory will also carry individual prey items in the beak to the nest. As chicks hatch asynchronously in order of their laying, in times of reduced food resources, younger chicks may perish and serve as food for their larger, older siblings.[19,20,22]

Carnivorous hornbills are thinly spread in their habitats and considered territorial.[19,20] Some carnivorous hornbill species have developed compatible relationships with other animals in their habitats to improve success of finding prey.[19] By contrast, communal interactions are encouraged by the frugivorous feeding strategy, with sharing of food sources and less territoriality being demonstrated, except at nesting sites, which are a limited commodity.[19,20]

Regardless of the natural history, housing in managed care generally occurs in bonded pairs, or as family groups with recently emerged chicks (Figure 56.6). In managed care of *Buceros*, increased behaviors related to pair bonding, nesting, and mudding the seal were noted in successful chick production years, and a potential for increased compatibility to develop seemed evident with increased duration of the pairing.[24]



FIGURE 56.6 Family group of Jackson's hornbills *(Tockus jacksoni)* as housed in managed care. (Photograph courtesy Jan Raines, Zoo Health Management, Dallas Zoo.)



FIGURE 56.7 Abyssinian ground hornbill *(Bucorvus abyssinicus)* during "porcupine" or "pin-cushion" quill plumage stage, as imaged by author during chick rearing. (Image courtesy of Ann Knutson, Bird Department, Dallas Zoo.)

NEONATOLOGY

Following hatching, nesting periods for smaller species of hornbills are 39 to 50 days and for larger species 72 to 96 days.[19] Chicks hatch with a marked prognathism that resolves with beak elongation within the first days of life. Chick beaks also lack the species-characteristic casque, aside from a thickening in the area where future maturation will occur.[19,20] These altricial chicks maintain their eyes closed for 10 days. They are also bare of feathers for this period until suddenly they are covered in unsheathed quills over a rapid period of days (Figure 56.7), which can be concurrent with skin darkening.[19,20,23] This "porcupine stage" is replaced equally abruptly by a nearly fully feathered state as the quill sheaths are shed and the plumage unfurls in the drab color phase at one-third of the nesting interval.[19] In the first weeks of the nesting period, pockets of subcutaneous emphysema develop at the shoulders, then move and accumulate gradually across the entire torso, legs, and wings of the chick, which is maintained into adulthood as a normal finding. These chicks spend much time resting on their plantar hocks on roughened nest

substrates while confined within the small nest cavity. Prominent plantar tarsal skin callosities are present and considered to provide padding in this situation. At the end of the first third of the nesting stage, the chicks' legs will have attained the adult length for the species, which allows them to stand successfully and unassisted at the nest cavity vent to accept feeding and expel feces from the chamber interior. Flight and tail feathers are completely grown at the time of chamber emergence.[19]

MANAGED CARE AND VETERINARY CONSIDERATIONS

Confident handling of hornbills requires secure restraint of the head and casque due to their heavy nature and strength.[5] As the casque is large and the external nares are at the beak-casque interface, standard anesthetic masks should be replaced by clear plastic bags or sheaths to allow visual access to the patient but create a soft chamber around the ornamental keratin (see section on Toucans; Figures 56.3 and 56.4).[23] Peripheral venipuncture and radiographic interpretation (Figure 56.8) are impacted by the previously described subcutaneous emphysema. However, subcutaneous fluids can be administered without concern of respiratory impact or entry to the internal air sacs.

Considerations of geriatric age and events in managed care should be managed for this species as they are exceptionally long-lived, with the larger species reaching 40 to 50 years old and even smaller species routinely aged over a decade. Additionally, their high level of curiosity lends these birds to concern for foreign body ingestion. That said, no truly unique diseases to the order can be remarked aside from the species-focused squamous cell carcinoma of the giant hornbill *(Buceros bicornis)* casque, which should be monitored routinely by annual skull radiography (see section on The Hornbill Casque in Health and Disease). With a shared etiologic concern of dietary carotenoids, great vessel atherosclerosis also is featured frequently in later decades of life for giant hornbills. Due to the predominantly frugivorous nature of Asian hornbills, hemochromatosis or ISD has figured prominently in their managed care (see section on Iron Storage Disease). Otherwise, intermittent cases of bacterial, fungal, parasitic, toxicologic, or orthopedic conditions were not found at extraordinary or frequent levels aside from proventricular spiurid infections in juvenile wrinkled hornbills *(Rhabdotorrhinus (Aceros) corrugatus)*.[23]

IRON STORAGE DISEASE

Peter Sandmeier

Although Iron Storage disease (ISD) has been described in many avian species, both hornbills and especially toucans are commonly affected, leading to discussion of the disease within this chapter.

REGULATION OF IRON HOMEOSTASIS

Iron is an essential trace element but also is toxic in excess. All iron enters the body from the diet, either as heme or nonheme iron. Most heme iron is animal derived as myoglobin or hemoglobin. In contrast, nonheme iron is plant derived. As heme iron is bound within a protoporphyrin ring, its absorption is more efficient and is less affected by diet composition than nonheme iron bioavailability, which can be affected by a range of dietary constituents. The low pH of the stomach and other organic acids, such as ascorbic acid, maintains nonheme iron in its ferrous (Fe^{2+}) form, which is the soluble, more absorbable iron form. In contrast, ingested plant-derived phytates and polyphenols can bind nonheme iron and impede its absorption.

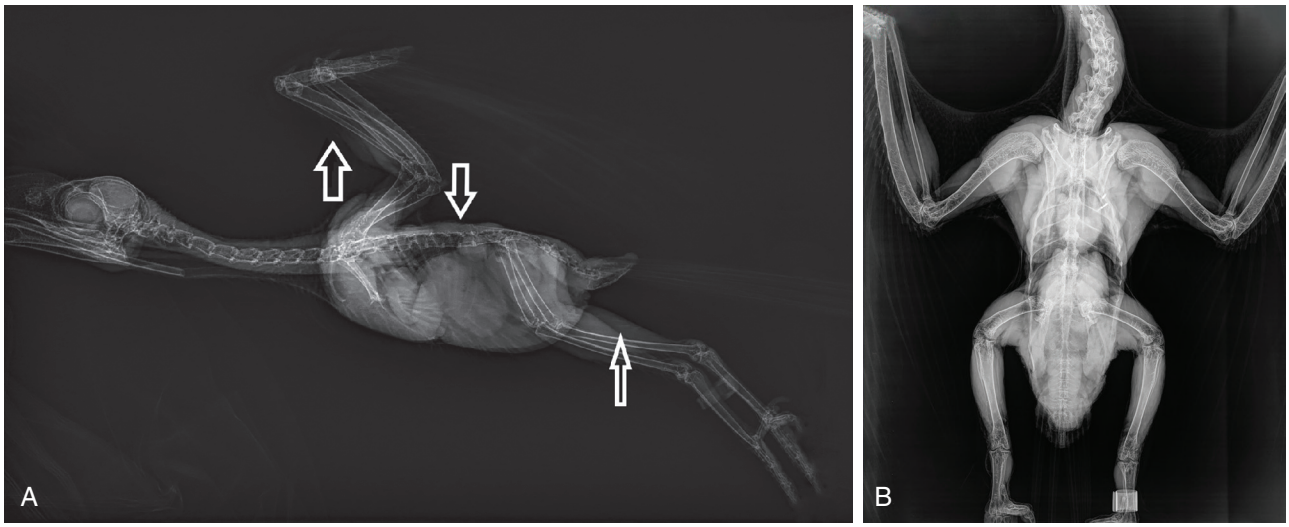


FIGURE 56.8 Subcutaneous emphysema normally present in all hornbill species to a variable degree, beginning as nestlings and maintained as adults, as shown in (A) the lateral radiograph of an adult male Von der Decken's *(Tockus deckeni)* as noted by *arrows* and (B) the ventrodorsal radiograph of an adult female Blyth's *(Rhyticeros plicatus)* hornbill. (Radiograph provided by Nancy Carpenter, Utah's Hogle Zoo, and by author, respectively.)

Iron is absorbed by enterocytes mainly in the small intestine. Nonheme iron crosses the enterocyte membrane via divalent metal-iron transporter 1 (DMT1). This transporter requires ferrous iron (Fe^{2+}) as a substrate, whereas ferric iron (Fe^{3+}) is poorly absorbed by enterocytes and needs to be reduced to ferrous iron before it can bind to DMT1 and be absorbed. If iron is not immediately required by the body, it becomes sequestered in the enterocyte cells within the iron storage protein ferritin and is lost from the body once that enterocyte is sloughed. When iron is required, it is transported across the inner enterocyte wall into the bloodstream via ferroportin 1 (FPN1).

Within the bloodstream, absorbed iron is bound to plasma transferrin. Each transferrin molecule can bind two iron atoms, becoming diferric transferrin. Normally, only approximately 30% of the iron-binding sites on the plasma transferrin are occupied, providing a considerable buffering capacity. However, in ISD, transferrin becomes saturated and the concentration of toxic nontransferrin-bound iron (NTBI) can become dangerously high.

Diferric transferrin delivers iron to cells by binding to transferrin receptor 1 (TFR1) in cell membranes. Iron that is released from transferrin moves into the cytoplasm via DMT1. Iron as NTBI also can very efficiently cross the membranes of many cell types. Once inside a cell, iron is either utilized for metabolic functions or bound to the iron-storage protein ferritin for later use. As a safety valve, cells can export iron via FPN1.

Ferritin is the major intracellular iron-storage protein. Ferritin has a large iron-binding capacity and is expressed in high quantities when intracellular iron concentrations rise, providing cells with an enormous ability to sequester iron. As the concentration of iron-laden ferritin rises within the cells, these ferritin molecules aggregate and fuse to lysosomes, resulting in Fe^{3+} cores and peptides. This hemosiderin is particularly prominent in the cells of patients with ISD. The fact that small amounts of ferritin are secreted from the cell and that the secreted amount correlates with the concentration of intracellular iron makes serum ferritin concentration a useful indicator of body iron stores used in human medicine.[25]

The regulation of iron homeostasis at the cellular level is replicated at the whole-body level. This regulation is mediated by hepcidin, a small liver-derived peptide. During iron overload, the liver increases the expression of hepcidin. Hepcidin binds to the cellular iron-exporting protein FPN1, impeding the release of iron into the circulation. In contrast, when iron demands are high, hepcidin concentrations are low, the cellular iron-exporting protein FPN1 remains free, and more iron can enter the circulation. The main iron-exporting cells include macrophages, which recycle heme-derived iron from red blood cells; hepatocytes, which are the major site of iron storage; and enterocytes, where dietary intake of iron is facilitated (Figure 56.9).[25]

Two hepcidin-like cDNAs (HP1 and HP2) have been isolated from and described in pigeons. The expression of pigeon

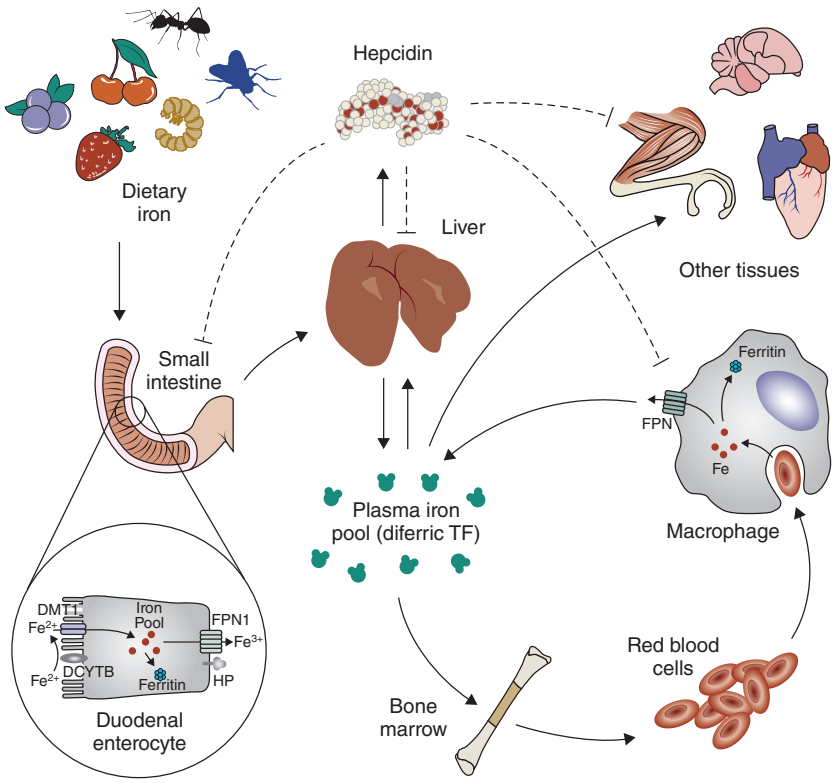


FIGURE 56.9 Body iron homeostasis. Nonheme iron enters the circulation after traversing the enterocyte. Iron binds to plasma transferrin and is distributed to tissues throughout the body. Quantitatively, most iron is used by immature red blood cells in the bone marrow for hemoglobin production. Liver-derived hepcidin binds to plasma membrane FPN1 on enterocytes, macrophages, and most body cells reducing iron absorption and impairing iron release from stores. *TF,* Transferrin.

HP1 was significantly upregulated after an iron-dextran injection, whereas the expression of HP2 did not increase, although it significantly increased after a lipopolysaccharide (LPS) injection. Therefore whereas HP1 appears to be involved in iron regulation, HP2 appears to be part of the acute phase reaction of an inflammatory response.[26]

IRON STORAGE DISEASE IN HUMANS AND OTHER MAMMALS

In humans, primary iron overload has a genetic basis resulting from mutations in genes that are involved in the transport or regulation of iron and is considered uncommon to rare. However, hemochromatosis is relatively common in white northern European males and is featured by the lack of hepcidin expression, which results in the inability to limit the absorption of dietary iron. Secondary iron overload is associated with blood transfusions that are used to treat inherited disorders such as iron-loading anemias.[27]

In other mammals with susceptibility to ISD, particularly frugivores and browsers, a similar pattern to avian species can be observed. Rather than a genetic background, a dietary component is related to the development of disease.[28]

IRON STORAGE DISEASE IN SUSCEPTIBLE AVIAN SPECIES

ISD has been described with a low incidence for many avian species in managed care. A retrospective study representing 52 genera from 13 orders reported some degree of iron accumulation in the liver of 64% of all samples, with no statistical difference in the occurrence of iron accumulation between families, orders, or origin (free ranging or managed). However, only two of the studied cases showed severe iron accumulation and clinical signs compatible with ISD.[29] Particularly susceptible or typically described species include those in the families Paradisaeidae (birds-of-paradise), Ramphastidae (toucans), Sturnidae (mynahs and starlings), Thraupidae (tanagers), Bucerotidae (hornbills), Pipridae (manakins), and Trogonidae (trogans, quetzals).[30–32] Evaluation of a large number of histopathologies in hornbills concluded that, although not as prevalent as in mynahs and toucans, frugivorous Asian hornbills, especially the genera *Aceros*, *Rhabdotorrhinus*, and *Rhyticeros* should be considered susceptible to ISD, but not necessarily the carnivorous and omnivorous species.[33]

A common characteristic of these species above is their frugivorous and, to a lesser extent, insectivorous diet. On the other hand, granivorous and omnivorous species are less likely to develop ISD.[32] Based on this observation, a common evolutionary background driven by the low iron content of their natural diet would explain the susceptibility of the affected species. However, although a low iron content of these diets can be assumed, it has only been demonstrated quantitively for free-ranging toucans.[34] Such avian species may have developed physiologic mechanisms to extract dietary iron very efficiently.[35] Higher rates of mucosal transport of iron in frugivorous mynahs than in granivorous pigeons have been demonstrated,[36] which are linked to a very high intestinal expression of iron transporters.[37]

The hypothesis that diet plays an essential role in the susceptibility of species to ISD is also supported by the fact that granivorous psittacine birds are not especially susceptible to ISD, in contrast to lories that eat predominantly nectar, fruit, and insects,[38] and red-spectacled amazons *(Amazona pretrei)* that consume a diet of mostly pine nuts containing a very low concentration of iron.[39] Similarly, among Columbidae, only frugivorous species appear to be susceptible to ISD, with the granivorous species being less commonly affected.[36]

In summary, avian species with high susceptibility to ISD that seemingly have evolved to consume diets with a low iron content appear also to have developed a very efficient iron absorption with concurrent reduced ability to downregulate iron absorption outside of their normal habitat. It seems plausible that they are not able to express hepcidin or that hepcidin does not bind to their FPH1 receptors. In managed care, these birds may develop ISD when they are fed diets containing iron concentrations that exceed their physiologic iron homeostatic capabilities[32] or containing fewer dietary components that can bind iron in the diet and impede its absorption, such as plant-derived phytates and polyphenols.

IRON STORAGE DISEASE AND INFLAMMATION

Because iron is the first limiting nutrient for the growth of many microbes in extracellular and intracellular fluids, a change in iron metabolism could offer protection against many pathogens.[32] Many ISD cases in nonsusceptible avian species occur concurrently with inflammatory diseases. A retrospective study of 180 birds representing 40 mainly nonsusceptible species showed a higher incidence of iron storage in Kupffer cells in birds that died from concurrent infectious disease than those that died from other causes.[40] In greater *(Phoenicopterus roseus)* and lesser *(P. minor)* flamingos with pododermatitis and renal amyloidosis, numerous individuals were diagnosed with ISD, suggesting that the increased iron storage is contributory to the overall inflammatory state (S. Xie, personal communication, 2020).

In mammals, the acute phase inflammatory response results in decreased absorption of iron and diversion of iron from erythropoiesis to storage sites, leading to functional iron deficiency, increased liver iron content, and subsequently to anemia. Once the inflammation is resolved, erythropoiesis resumes, stopping the anemia and reducing the liver iron.[32]

In contrast to mammals, anemia is not a hallmark of infection in avian species and plasma iron concentrations only decrease slightly during acute phase response even though liver storage increases.[32] Thus iron metabolism in avian species differs in that, during the acute phase response, diversion of extracellular iron to the liver is not accompanied by decreased absorption of iron and decreased hematopoiesis. This subsequently leads to an increased flux of iron that is deposited firstly in Kupffer cells and other phagocytes and then in hepatocytes.[32]

PATHOPHYSIOLOGY OF IRON STORAGE DISEASE

Although the mechanisms of cellular damage in avian species are not known, they are likely to be similar to those described in laboratory rodents. Iron accumulation within the lysozymes and mitochondria causes rupture and release of ionic iron, leading to oxidative damage to membranes and proteins.

Apoptosis or, in more severe cases, necrosis results. Reparative responses will cause fibrosis.[32]

DIAGNOSIS OF IRON STORAGE DISEASE IN AVIAN SPECIES

Clinical Signs

Early clinical signs of ISD are nonspecific; ISD should therefore be suspected in any diseased bird of a known susceptible species. During early stages of the disease, most patients will only show general listlessness and loss of feather glossiness.[41] Many birds will also die unexpectedly without previously noted clinical signs.[42]

As the disease progresses, clinical signs of liver disease develop, including dyspnea (due to abdominal distention as a sequela of ascites or hepatomegaly), weight loss, or depression. On thorough palpation of the coelom, ascites typically will be readily apparent, and hepatomegaly can be suspected by a flat or convex rather than concave ventral abdominal wall.[35,41]

Radiology

The most likely radiographic changes seen in birds with ISD include hepatomegaly or ascites. However, a lack of hepatomegaly does not exclude ISD. Other possible radiographic signs can include splenomegaly or cardiomegaly due to pericardial effusion.

Clinical Chemistry and Hematology

Changes in hematology or clinical pathology cannot definitively diagnose ISD. However, abnormal values that are indicative of liver damage in susceptible species make a tentative diagnosis of ISD more likely. Such changes may include increases in lactate dehydrogenase (LDH), aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma glutamyltranspeptidase (GGT), alkaline phosphatase (AP), and bile acids, although these vary by species.[43] Elevated AST, creatine phosphokinase (CK), and severe hypoproteinemia were observed in three Grey parrots *(Psittacus erithacus)* exposed to a carnivorous diet and diagnosed with ISD.[44]

Serum Iron Assays

In humans with suspected ISD, it is recommended to measure serum concentrations of ferritin and total serum iron, as well as total iron-binding capacity. Transferrin saturation is calculated by dividing the serum iron concentration by the total iron-binding capacity. However, serum ferritin concentration correlates with total body iron stores but does not correlate with the amount of iron deposited within the liver or spleen.[45]

In a case series that included 24 toucans of eight different species, iron assays of total serum iron concentration, total iron-binding capacity, and urine iron-binding capacity were all within assumed normal ranges for toucans, even though 19 of the 24 birds had liver iron concentration greater than 2000 ppm as measured by biopsy.[46] Normal amounts of hepatic iron in different managed avian species were reported to range from around 100 to 300 ppm.[46] Hence, in avian species, there seems to be no apparent correlation between serum iron assays and the amount of iron deposited in the liver.

In hornbills presented with clinical ISD, the total serum iron concentration decreased during chelation treatment, making the monitoring of total serum iron concentration a possible noninvasive method of monitoring the success of chelation therapy.[41]

Liver Pathology

Liver biopsy is the only reliable method of obtaining an antemortem definitive diagnosis of ISD. In hematoxylin and eosin–stained histological samples, iron accumulation is recognized as yellow-gold to brown granular pigment in the cytoplasm of hepatocytes.[47] In Prussian blue stains, the liver iron content stains blue in contrast to the red stain of liver parenchyma that is not loaded with iron.[41]

Histopathologically identified increased iron content in the liver without concurrent cellular damage cannot be considered a diagnosis of ISD. The changes consistent with ISD include severe iron deposits in hepatocellular and Kupffer cells, hepatic periportal macrophages, and splenic phagocytes, as well as portal bridging fibrosis, biliary hyperplasia, and mild lymphoplasmacytic, heterophilic cholangiohepatitis.[31,38] Histological evidence of ISD has also been described in other organs. Various authors describe acute myocardial necrosis,[38,44] which may in many cases be the final cause of death. Moderate to mild iron deposits also are described in the spleen, myocytes of the heart, pancreatic acinar cells, intestinal enterocytes, and renal tubuloepithelial cells.[38]

QUANTIFICATION OF LIVER IRON CONTENT

Three different methods of quantifying liver iron content have been described: chemical-toxicological analysis, quantitative image analysis (QIA), and magnetic resonance imaging (MRI).

Chemical analysis is the standard method of measuring liver iron content. Measurements are performed on a liver sample, collected by liver biopsy or on postmortem, using an atomic absorption spectrometric analysis system with a spectrometer. Results of chemical analysis are reported in mg/kg of dry matter.[41]

Quantitative image analysis is performed in formalin-fixed histologic samples embedded in paraffin and sectioned according to standard techniques. Samples are then stained with Prussian blue. The mean percentage of the blue-staining area in relation to the entire area is determined with a computer-based image analysis system. Results of QIA are reported in percent (Figures 56.10 and 56.11).[48]

Because MRI is noninvasive, it has become the method of choice in calculating liver iron content in human patients. The calculated liver iron content is reported in mg/kg tissue.[49] This method has been applied experimentally in pigeons *(Columbia livia domestica)* with induced iron overload[50] and has been used to monitor the efficacy of chelation therapy with deferiprone in two Papuan/Blyth's *(Rhyticeros plicatus)* and one long-tailed *(Tropicranus albocristatus)* hornbill with hepatic ISD (Figures 56.10 and 56.11).[41] The small size of the avian patients challenges this modality. The results obtained in the hornbills had a wide standard deviation and may be inaccurate because of severe pulsation artifacts. Although results indicated a similar reduction in liver iron content as the biopsy measurements, they did not correlate significantly with these measurements.

Further, promising as MRI measurements may be, the advantage of a noninvasive monitoring must be compared with

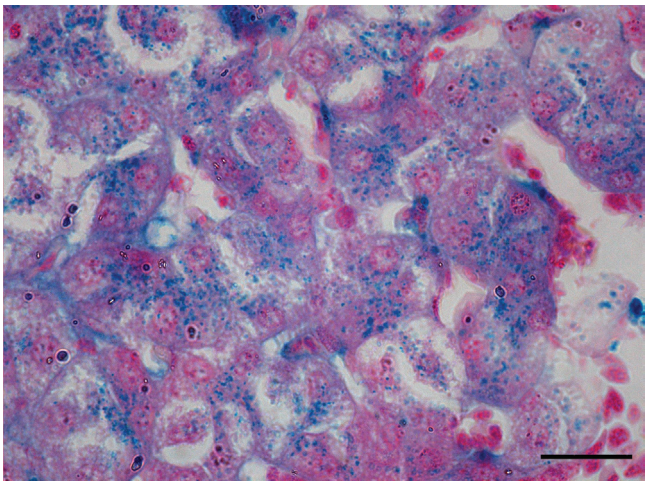


FIGURE 56.10 Prussian blue stain of a liver biopsy from a long-tailed hornbill *(Tockus albocristatus)* diagnosed with iron storage disease. Quantitative image analysis determined a hepatic iron content of 23.1%. Bar = 20 μm. Sandmeier, P., Clauss, M., Donati, O. F., Chiers, K., Kienzle, E., & Hat, J. (2012). Use of deferiprone for the treatment of hepatic iron storage disease in three hornbills. J Am Vet Med Assoc 240(1), 75–81.

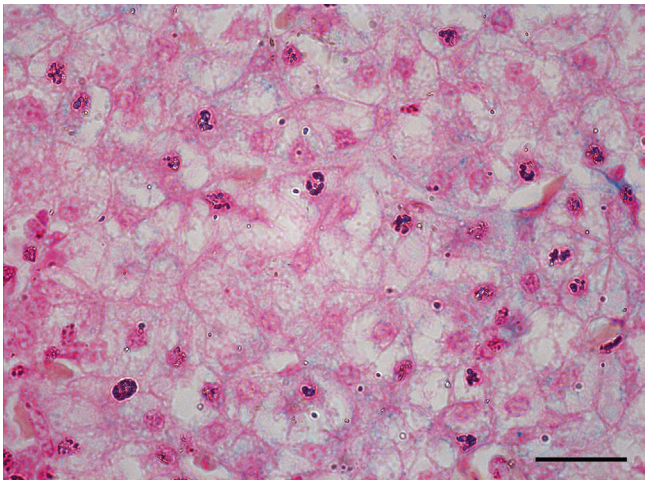


FIGURE 56.11 Prussian blue stain from the same hornbill as in Figure 56.10 after 90 days of chelation therapy with oral deferiprone. Quantitative image analysis determined a hepatic iron content of 0.7%. Bar = 20 μm. Sandmeier, P., Clauss, M., Donati, O. F., Chiers, K., Kienzle, E., & Hat, J. (2012). Use of deferiprone for the treatment of hepatic iron storage disease in three hornbills. J Am Vet Med Assoc 240(1), 75–81.

the risks of the relatively long period of general anesthesia required (60–80 minutes for MRI vs 10–15 minutes for biopsy), which is an important constraint, especially in birds of a generally reduced health status. Additional logistic challenges of longer transport routes and preparation time will increase stress for the bird related to the medical procedures. Nonetheless, with increasingly widespread availability of MRI equipment and trained personnel in veterinary referral and teaching hospitals, the use of this technology for noninvasive diagnosis of ISD in appropriately selected and monitored avian patients, particularly exotic birds of high economic value, is likely to be indicated.[41]

TREATMENT OF IRON STORAGE DISEASE

For successful treatment, it is important to be able to make a diagnosis at an early stage before the disease progresses. Three therapeutic options have been described for the treatment of ISD: phlebotomy, iron chelation therapy, and dietary modification.

Phlebotomy is the most common treatment for ISD in humans.[51] Whole blood phlebotomy performed once or twice weekly by removing a blood volume of 0.7% of the body weight is recommended. If anemia occurs, then chelation therapy with deferoxamine administered parentally or deferiprone as an oral chelator is recommended.[51] While no guideline for avian phlebotomy has been standardized, blood volumes of 0.5% to 1% of body weight have been recommended as a safe maximum.[52]

Considerations during the use of phlebotomy in avian patients would include stress, risks of repeated anesthesia, injury during capture, hematoma formation, and induction of anemia. It is prudent to closely monitor the hematocrit and hemoglobin in avian patients treated by phlebotomy.

Chelation therapy in avian patients has been described using parenteral deferoxamine (100 mg/kg q24h) as well as oral deferiprone (100 mg/kg q24h). Deferoxamine is a parental chelator that forms stable complexes with iron, preventing the iron from being involved with further reactions. It binds preferentially with ferritin and hemosiderin forms of iron but not transferrin, cytochromes, or hemoglobin. The chelated iron is metabolized by plasma membranes and is excreted in urine and feces.[53] The disadvantage of deferoxamine chelation therapy is the necessity of daily injections. Deferiprone is an orally bioavailable bidentate ligand with iron-chelating activity. Deferiprone binds to iron in a 3:1 (ligand:iron) molar ratio. As deferiprone can be administered orally, this medication represents a more viable option for long-term treatment.[41]

Dietary modification entails feeding a low-iron diet with or without adding iron-binding substances, including polyphenols such as tannins or phytates. It also should be recommended to avoid substances, such as ascorbic acid, which enhance iron absorption.[54]

In a study performed in European starlings *(Sturnus vulgaris)*, the liver iron concentration after four different treatment methods was compared among each other and to a control group of seven euthanized wild starlings.[55] After feeding a loading iron diet, all groups were fed a low-iron diet containing 25 ppm of iron. Group 1 (n = 4) was treated with the low-iron diet only. Group 2 (n = 4) was fed a phytate (inositol) and tannic acid in addition to the low-iron diet. Group 3 (n = 4) was treated with deferoxamine (100 mg/kg SC q24h) and fed the low-iron diet. Group 4 (n = 4) was treated with weekly phlebotomy and fed the low-iron diet. In groups 3 and 4, the mean liver iron concentration was significantly decreased after 16 weeks of treatment in comparison to the control group. The decrease in liver iron concentration in group 1 was small but was significant when compared to control starlings. No significant decrease in liver iron concentration occurred in group 2 when compared to the control starlings.

In a further recent study, the effect of oral deferiprone at 75 mg/kg q24h and subcutaneous deferoxamine at 100 mg/kg with or without phlebotomy was studied in healthy common mynahs *(Acridotheres tristis)* that were euthanized posttreatment and liver iron, copper, and zinc concentrations were analyzed. After 1 month, in all treatment groups, the levels of iron, copper, and zinc present in the liver were significantly reduced, but no significant difference was noted in metal concentrations between the groups significantly reduced, but no significant difference was noted in concentrations between the treatment groups. After 2 months of treatment the liver iron, copper, and zinc concentrations decreased further. However, this decrease was only significant in the group treated with deferiprone and phlebotomy. The authors concluded that all treatment options successfully reduce liver iron content within 1 month and recommend the use of deferiprone as an inexpensive oral chelator. They recommended not treating longer than 1 month, as longer-term treatment causes iron, zinc, and copper deficiency.[56]

Pharmacological studies have been performed on the oral iron chelator deferiprone in pigeons and chickens *(Gallus gallus domesticus)*. The pharmacokinetics of the two species were very similar. In pigeons and chickens, an oral administration of deferiprone at a dose of 50 mg/kg achieved plasma concentration effective for iron chelation for at least 8 hours. No significant alterations in routine hematological or biochemical parameters were noted.[53,57,58] In both species, oral chelation therapy was associated with weight loss and reduced zinc serum concentrations. In chickens, a reduction in egg production and death, most likely due to acute reduction in iron required for normal enzymatic processes, was observed.[58] Based on the half-life of deferiprone, a twice-daily dosage regimen of 50 mg/kg appears appropriate, although a single daily equivalent regimen of 75 mg/kg may be as effective. However, the potential side effects do need to be considered.[58]

In the hornbill case study, the oral administration of deferiprone at 100 mg/kg q24h was found to effectively reduce the liver iron content (Figures 56.10 and 56.11).[41]

PROPHYLAXIS OF IRON STORAGE DISEASE

Low-Iron Diet

The mainstay of ISD prophylaxis is the provision of a limited iron concentration in the diet, avoiding enhancers of iron absorption and possibly adding inhibitors of iron absorption to the diet. Current diet recommendations for ISD-susceptible avian species range from 32 to 125 mg/kg iron dry matter.[55] The diet of wild birds of paradise (Paradisaeidae) is composed primarily of fruits with 15 to 30 mg/kg iron dry matter and insects with 30 to 100 mg/kg iron dry matter.[59] Studies in mynahs suggest 19 to 25 mg/kg iron dry matter.[36] Based on these results, it appears prudent to feed ISD-susceptible avian species with a diet of 25 to 50 mg/kg iron dry matter. However, these levels are probably too low for females during egg production and for growing chicks, so seasonal diet alterations may be indicated for successful managed reproduction.

In captive Raggiana birds-of-paradise *(Paradisaea raggiana)*, elevated total iron and total iron-binding capacity could be reduced by periodically removing commercial avian pellets from their diet for 30 days at 6- to 12-month intervals, reducing the diet iron content from 55 to 42 mg/kg dry matter. This method has therefore been suggested as an effective method for lowering serum iron values without the need for handling.[59]

Adult toco toucans *(Ramphastos toco)* ($n = 2$) that had previously been diagnosed with ISD by quantitative image analysis of liver biopsies showed a significant reduction in hepatocellular hemosiderin content after 8 months of being fed a diet restricted to 100 mg/kg dietary iron.[42]

Limiting iron concentration in diets of susceptible species in captivity can be challenging. In particular, additions of red meat (e.g., in the form of mouse pups that are often not considered as a staple diet item but nevertheless offered regularly to the birds) or mineral supplements[60] are problematic because of the high content of readily available iron. Limiting iron concentrations in commercially prepared complete diets also is problematic.[61,62] Domestic feedstuffs that are low in iron are relatively rare and often expensive. In addition, more traditional feedstuffs can be highly variable in iron content and monitoring their iron concentrations adds significantly to the cost.[7] This consideration is reflected in the large variety of bird diets that are commercially available, and actual iron concentrations can vary dramatically between manufacturers' information and laboratory results[35,63] and even between different batches of the same brand of food.

Inhibitors of Iron Absorption

Based on difficulties to ensure low dietary iron content, the addition of inhibitors of iron absorption has been advocated with some success. The two main groups of iron absorption inhibitors are phytates and polyphenols. Plant-derived phytates such as inositol have been shown to inhibit iron absorption in humans at very low concentrations of 2 to 10 mg/meal.[54] Polyphenols occur in vegetables, such as chili or turmeric, and in beverages such as black tea and wine as tannins.[54]

Readily available and inexpensive additives appear to successfully inhibit iron absorption from diets containing high concentrations of iron. A high-iron diet in European starlings caused an increase in the concentration of liver iron to levels similar to birds that had died of ISD. However, this same high-iron diet supplemented with inositol and tannic acid did not cause an increase in liver iron concentrations.[64] In a further study, hepatic iron concentration increased significantly in 21 European starlings fed an iron-enriched diet, but not in 20 birds that consumed the same iron-enriched diet supplemented with black tea leaves.[65]

Enhancers of Iron Absorption

Reducing dietary components that may enhance iron absorption may also have preventative value. The enhancing effect of ascorbic acid on iron absorption has been shown convincingly in humans.[54] This effect is largely due to its potential to reduce ferric to ferrous iron. In whole fruits and vegetables, the effect is probably negated by the inhibiting effect of polyphenols. In addition to the fact that muscle tissue contains heme iron, it has also been consistently shown in humans that meat, fish, or poultry have an enhancing effect on iron absorption from vegetarian meals. The nature of this

enhancement has proved elusive. However, it appears due to cysteine-containing peptides, which, similar to ascorbic acid, could reduce and chelate iron.[54] Although the addition of 10% meat supplementation and addition of ascorbic acid at 17 mg per day did not increase liver iron concentration in European starlings,[64] it remains prudent to avoid adding meat or fruits high in ascorbic acid to the diet of ISD-susceptible avian species.

In summary, chelation therapy with orally administered deferiprone is an effective and relatively easily implemented treatment of clinically diseased patients. Chelation therapy with injectable deferoxamine is also effective but more difficult to implement due to the required daily injections. Serial phlebotomies are effective in lowering body iron content but are also associated with many restricting factors. Feeding a low-iron diet in combination with adding iron-binding substances to the diet and avoiding enhancers of iron absorption will gradually lower liver iron content but is mainly effective for subclinical cases or preventive purposes and not for the treatment of clinically diseased avian patients. It is important to be aware of increased iron requirements during times of reproduction and to prudently avoid severe iron restriction during this period.

THE HORNBILL CASQUE IN HEALTH AND DISEASE

Shangzhe Xie

The hornbill casque is a prominent organ that presents a unique focus for veterinary attention in hornbills. It is important to understand the hornbill casque in health and disease so that appropriate diagnostic and therapeutic interventions can be performed when required.

THE HORNBILL CASQUE IN HEALTH

The casque is a conspicuous bony outgrowth of the skull that is present in several taxa of birds.[66] The most well-known families of birds with casques are Casuariidae and Bucerotidae.[66,67] The shapes, sizes, and colorations of the 57 extant hornbill species vary from a simple ridge in *Tockus* species to elaborate recurved horns in *Anthracoceros* species.[68,69] The proposed functions of the casque include structural beak reinforcement for behaviors such as aerial jousting,[70] visual display to demonstrate social status or sexual maturity,[68,69] and acoustic amplification of vocalizations.[71]

In all species of hornbills except the helmeted hornbill *(Rhinoplax vigil)*, the casque is an air-filled cavity enclosed in a very thin layer of cancellous bone.[68,69,72] The bony outgrowth is covered by cornified rhamphotheca, which is often colorful and defines its visible outline.[66,67] A division of the paranasal sinus, known as the casque sinus and unique to hornbills, has been demonstrated by advanced imaging in six hornbill species.[67] Further studies are needed to demonstrate the presence of the casque sinus in other hornbill species, but it may be a route for neoplastic cells or infectious agents to extend into deeper parts of the skull or paranasal sinus system.[67]

Development of the Hornbill Casque

The casque originates from the ridge (culmen) of the maxillary rhamphotheca, which is highly vascular and enlarges with age and sexual maturity.[68,69] The development of pneumatization of hornbill casques is not well studied, but it is hypothesized in other avian species that it is caused by diverticula of the antorbital or frontal-ethmoidal sinus and develops late in ontogeny, as pneumatization is absent in the skulls of immature birds.[66]

The development of the beak and casque of hornbills generally follows the maturation of the chick, and the timeline appears consistent for both Asian and African hornbill species. The growth series of an Asian species is represented by a rhinoceros hornbill *(Buceros rhinoceros)* and an African species is represented by a Von der Decken's hornbill *(Tockus deckeni)* (Figures 56.12 and 56.13).

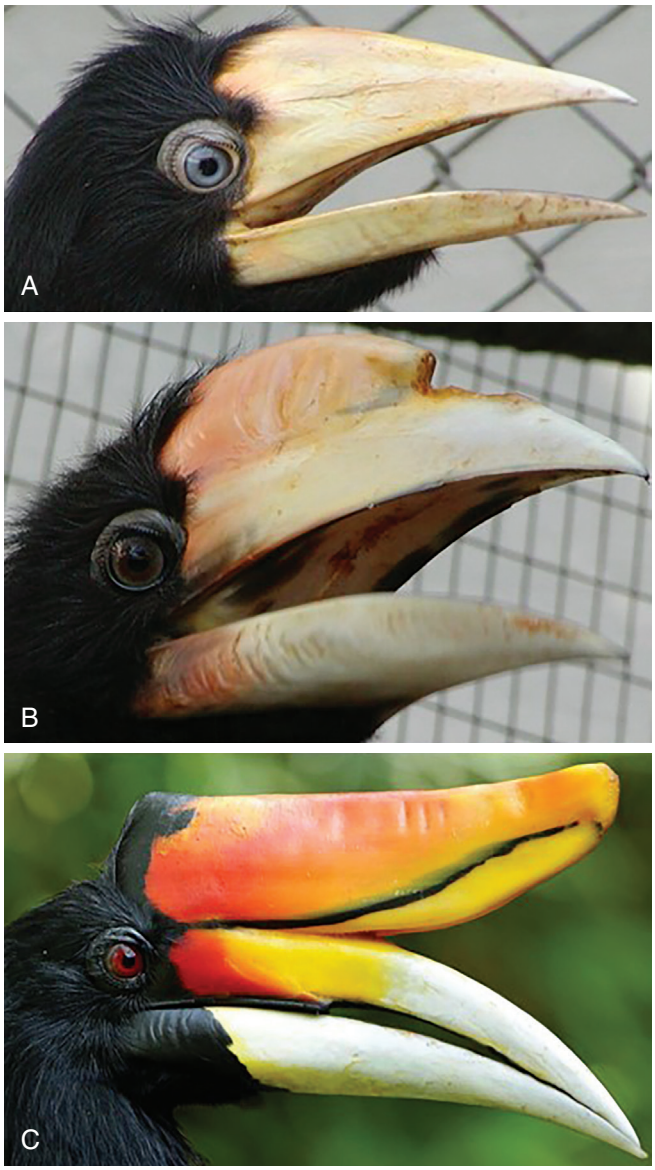


FIGURE 56.12 Growth series of a rhino hornbill *(Buceros rhinoceros)* casque at (A) emergence from nest, (B) within first year of life, (C) Buceros rhinoceros. (Photos A and B courtesy Jan Raines (Dallas Zoo); photo C courtesy Mandai Wildlife Group.)

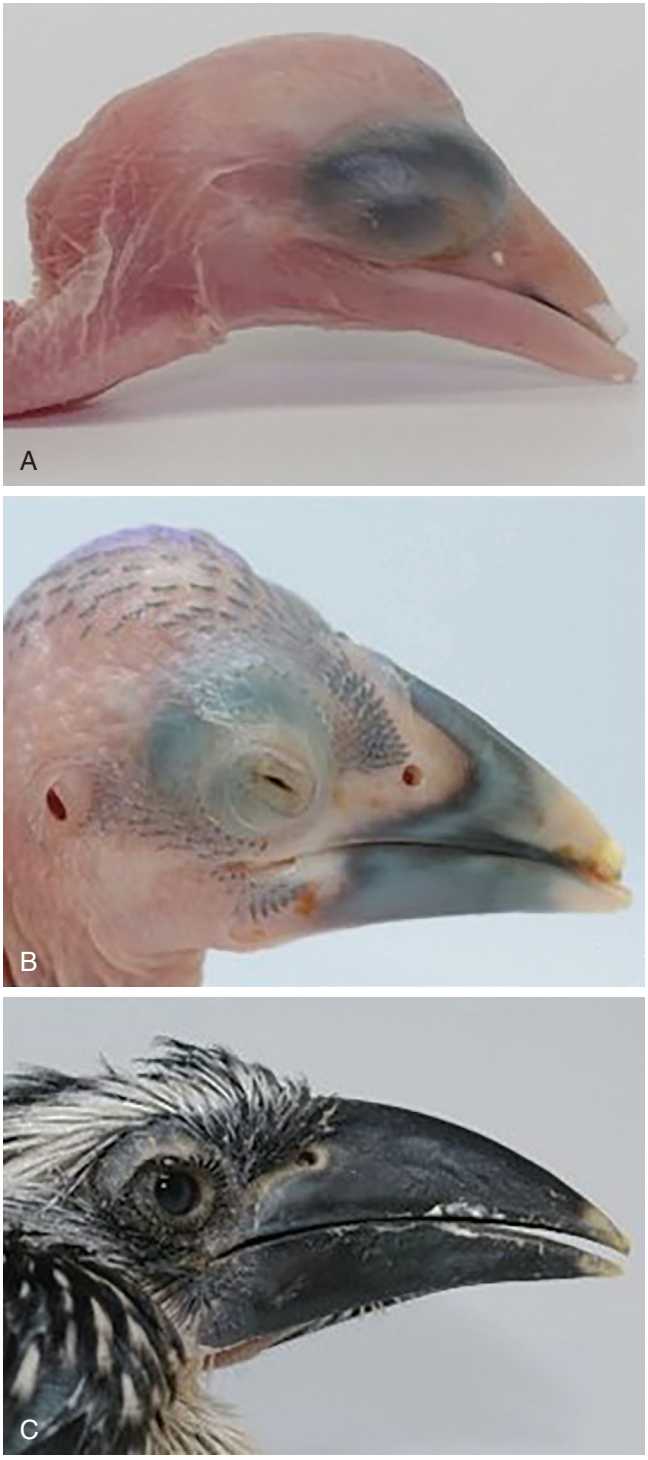


FIGURE 56.13 Growth series of a Von der Decken's hornbill *(Tockus deckeni)* casque (A) at hatch, (B) day 7, (C) day 26. (Photos courtesy Mandai Wildlife Group.)

THE HORNBILL CASQUE IN DISEASE

Common Causes of Casque Injury, Disease, and Problems

Reports of diseases of the casque are uncommon but include infection,[73] traumatic injury,[74] and squamous cell carcinoma in great hornbills.[75] In this author's experience, casque diseases and injuries are uncommon. However, communications with other veterinarians in the Southeast Asia region, where great hornbills are more commonly kept in captivity, suggest that external casque lesions similar to those that were reported in squamous cell carcinoma cases occur more frequently than the literature records. Definitive diagnoses could not be obtained for many of these cases as they were not conclusively diagnosed.



FIGURE 56.14 Lateral head radiograph of a normal adult male trumpeter hornbill *(Bycanistes bucinator)*.

Casque Diagnostic Imaging

Lateral (Figure 56.14) and dorsoventral radiographic views of the casque can be easily obtained with physical restraint of the patient. The radiographic anatomy of the casque has been described in detail,[2] but the amount of diagnostic information obtained from radiographs is usually limited. Obvious casque masses can usually be visualized on radiographs,[73,75,76] but this imaging modality does not allow differentiation between infectious and neoplastic causes of the mass.

Multislice advanced medical imaging data, particularly computed tomography (CT) (Figure 56.15) and MRI, can more accurately reflect anatomical details of the scanned region than radiographs and the images obtained can be converted to 3D volumetric models (see Chapter 2).[77] A biopsy of any masses (Figure 56.16) found can also be obtained under CT guidance.[76] Nonguided samples obtained for cytology, histopathology, and culture very frequently only obtain surface necrotic tissue, which are of limited diagnostic value. Based on these patient-specific 3D models, customized surgical tools can be created and precisely fitted to the anatomy, allowing improved surgical planning that translates to superior surgical outcomes.[78–80]

Casque Repair and Prosthetic Options

Sealing the casque after sinusotomy has been associated with infection.[73] It has been demonstrated that such infections can heal successfully by managing an open wound with extensive lavage.[73] In this instance, the casque healed by granulation initially, but eventually repneumatized.[73] Other repair techniques exist and are similar to beak repair techniques described (see Chapter 31).

In cases of squamous cell carcinoma where radical casque resection is the preferred treatment, production of a suitable casque prosthesis has historically been challenging.[81] However, 3D-printing technology has provided an improved way to produce casque prostheses.[76] The best way to preserve the

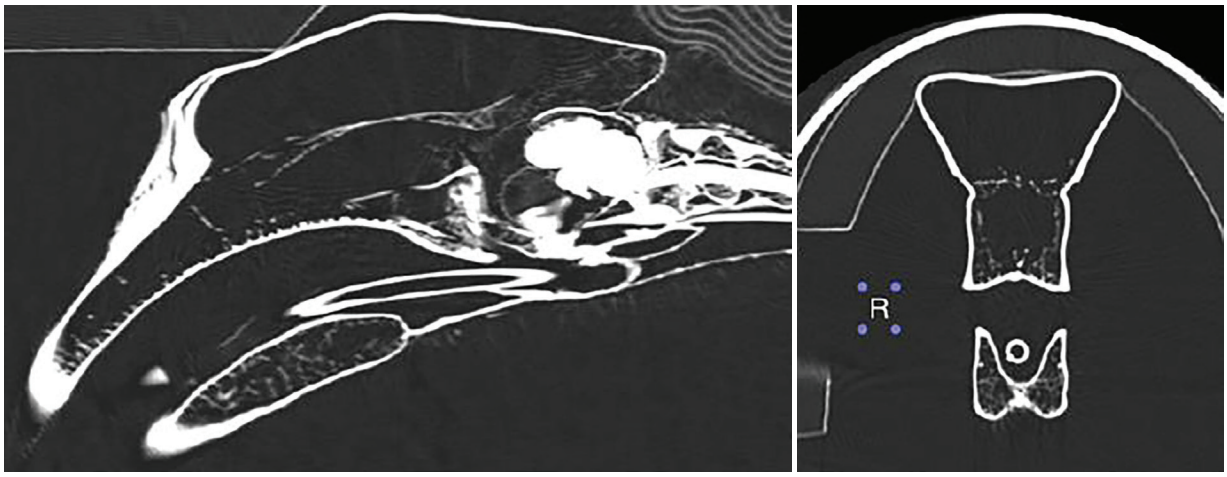


FIGURE 56.15 CT scan images of a normal adult male great hornbill *(Buceros bicornis)*.

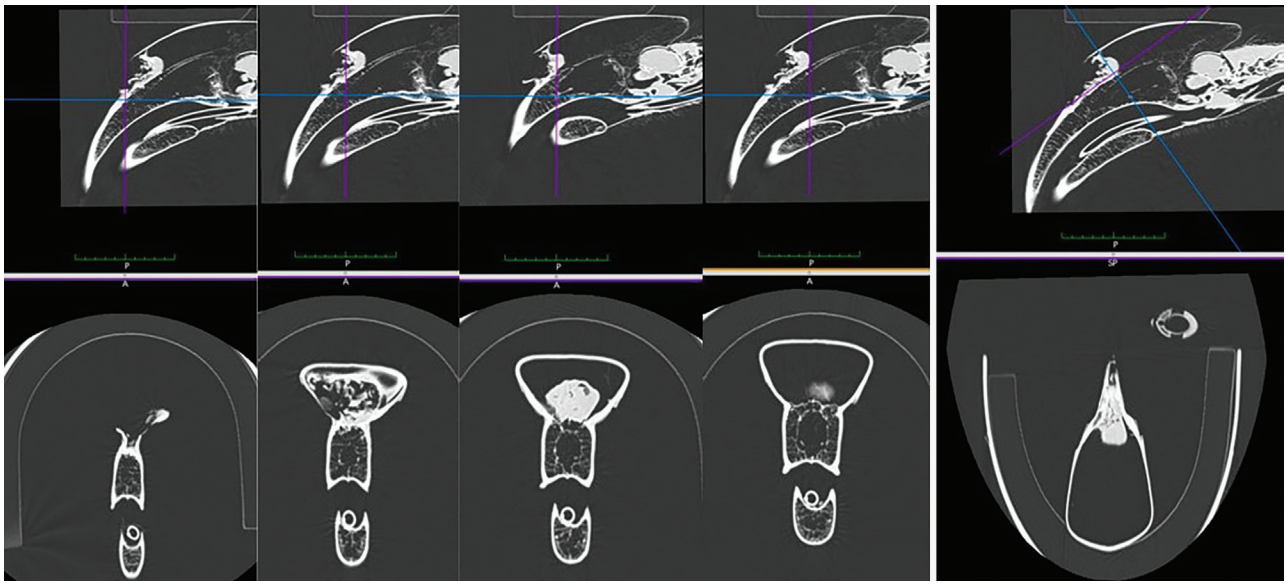


FIGURE 56.16 CT scan images of the head of an adult male great hornbill *(Buceros bicornis)* with casque squamous cell carcinoma showing the extent of the lesion in the casque in transverse and dorsal planes. Note that the right side of the head is on the left side of the images.

patient's aesthetics, retain as many original acoustic properties as possible, and minimize changes to the patient's vocalizations and balance was for the shape of the artificial 3D-printed prosthesis (Figures 56.17 and 56.18) to be identical in shape to the original beak and casque.[76] This approach can be made using CT images, which also allow production of a cutting guide (Figures 56.17 and 56.18) that fits the anatomy of the dorsal maxillary beak and casque and clearly identifies the planned surgical margins during surgery.[76] The 3D-printed prosthesis can be affixed to the maxillary beak using 2.7-mm cortical screws (DePuy-Synthes, Solothurn, Switzerland) or similar around the perimeter of the surgical margins.[76] Dental acrylic can be used to seal any remaining gaps between the prosthesis and the casque and beak (Figure 56.19), but this does not usually add any further stability to the prosthesis.[76] Such a technique (see Chapter 31) performed personally by the author and several other veterinary teams for separate cases did not result in any postoperative complication, and in the first case, no recurrence of the tumor had occurred more than 5 years after the surgery.[76] In this case, the keratin was found to have completely grown over the surgical defect and the prosthesis was no longer needed after it fell off 20 months after surgery (Figure 56.20). Follow-up radiation therapy or topical chemotherapy, which had limited success from previous reports,[81] was also not required with this technique. The success is likely due to the larger surgical margins used than in previous cases, where a suitable prosthesis could not be produced to cover a large postsurgical defect in the casque.

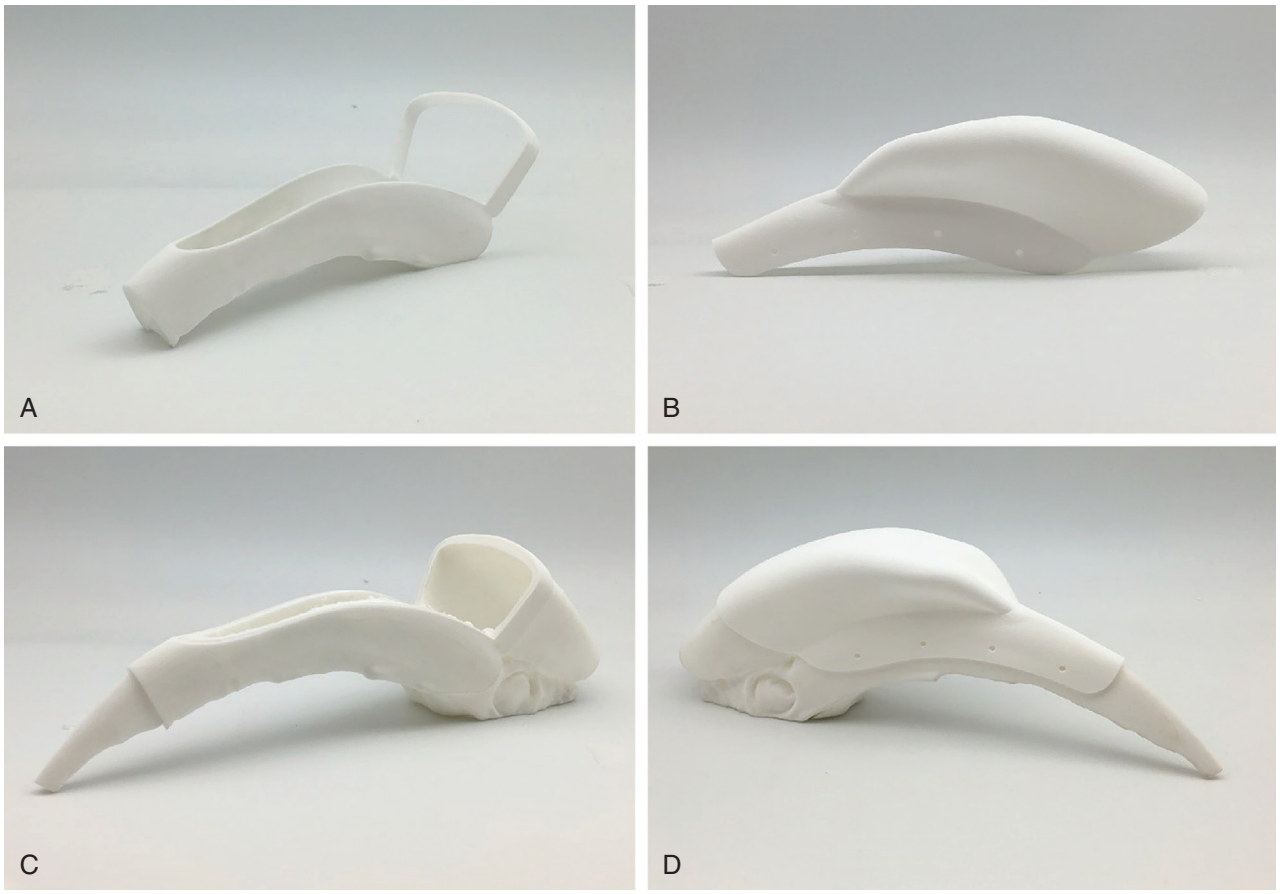


FIGURE 56.17 Three-dimensional printed models. (A) Surgical guide, (B) prosthesis, (C) surgical guide on the testing model, and (D) prosthesis on the testing model.[76]

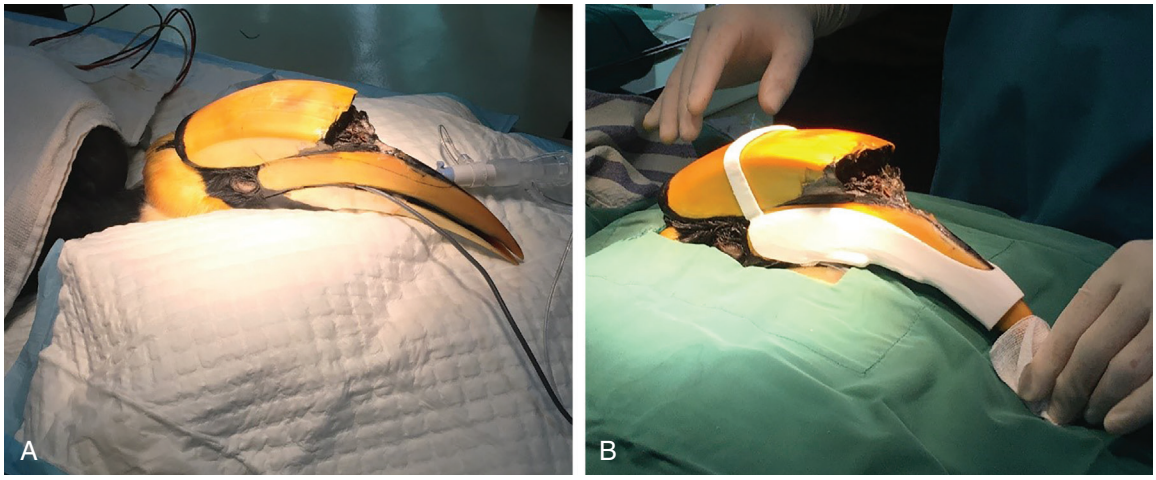


FIGURE 56.18 Casque and beak resection and replacement using a 3D-printed prosthesis of the hornbill in Figure 56.16 including (A) anesthetized patient preoperatively, (B) patient with the 3D-printed surgical guide,

Continued

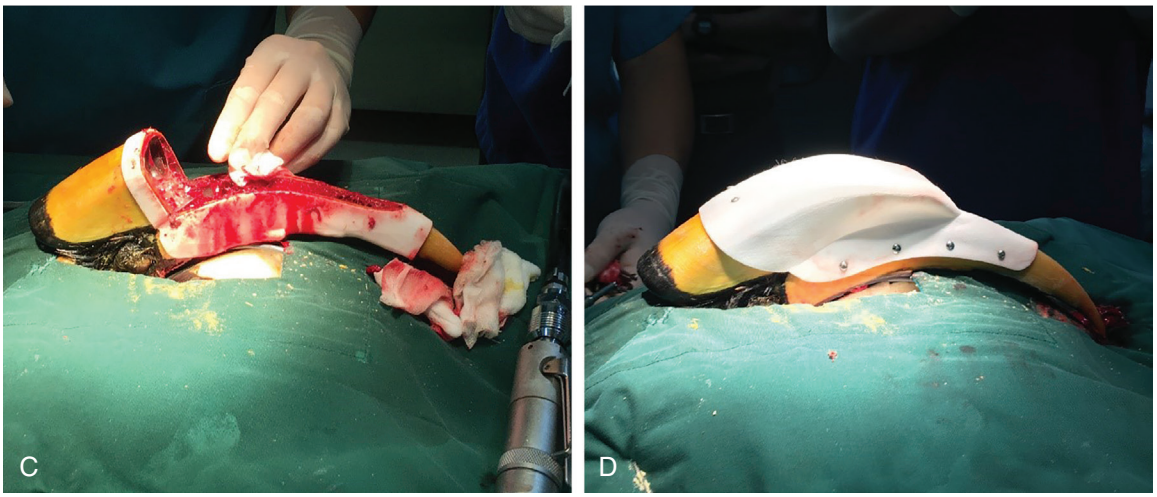


FIGURE 56.18, cont'd (C) patient with full excision of the tissue affected by carcinoma, and (D) patient with 3D-printed prosthesis secured using cortical screws to cover the surgical wound.[76]

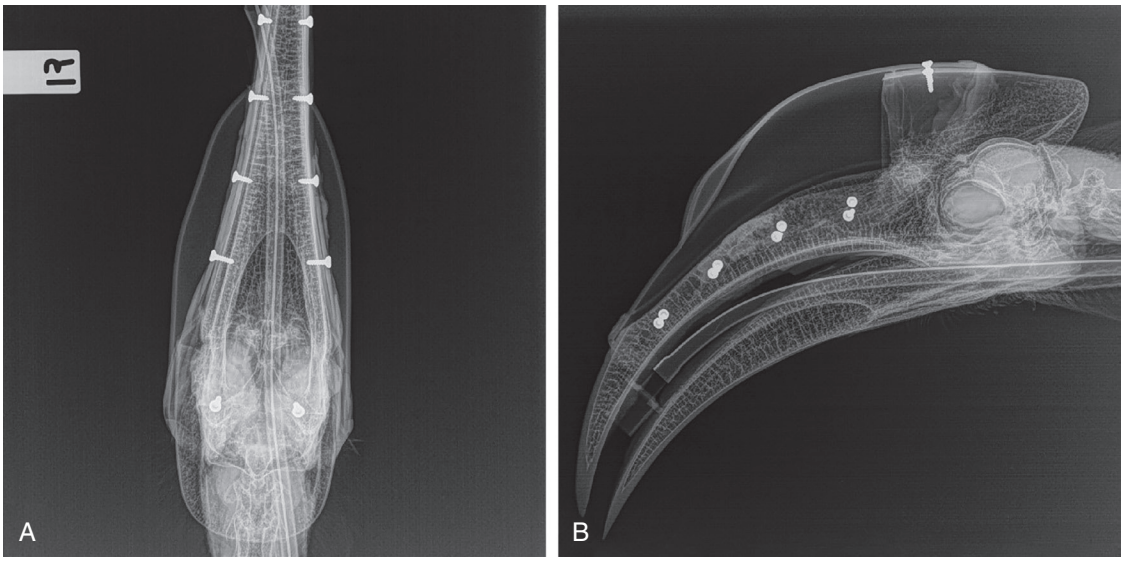


FIGURE 56.19 Postoperative radiographs from the bird of Figure 56.18: (A) dorsoventral and (B) lateral views.[76]



FIGURE 56.20 Regrowth of the keratin completely covered the surgical defect 20 months after surgery.

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Appendices

APPENDIX 1

Table of Common Drugs and Approximate Doses*

James W. Carpenter • Michelle G. Hawkins • Heather W. Barron • Rachel Baden

TABLE 1

Antibacterial Agents

Agent	Dosage	Species/Comments
Amikacin	7–20 mg/kg SC, IM, IV q8–12h	Most species; may cause myositis with IM injection; least nephrotoxic of the aminoglycosides, but renal toxicosis has been demonstrated in some species at 10 mg/kg—after 11 days; uric acid levels may be abnormal for up to 7 days after cessation; maintain hydration during use
	15 mg/kg IM q12h, IV q8h[168]	Blue-fronted Amazon parrots/PD
	15–20 mg/kg IM q8–12h[148]	Cockatiels/PD
	15–20 mg/kg IM q8h[44]	Chickens
	15–20 mg/kg IM q24h (can be divided over 2×–3× a day)[15]	Red-tailed hawks; dose remained above MIC for >12 hours for most isolates examined
	5.3 mg/kg IV[149]	Chicken/regional limb perfusion
	528 mg/L water	Ratites/egg dip
	3 g/40 packet bone cement	PMMA bead formation (1:14 ratio); same dose for all aminoglycoside beads
Amoxicillin/clavulanate (Clavamox, Pfizer)	125–250 mg/kg PO q8–12h	Most species, including pigeons, psittacines, and raptors
	125 mg/kg PO q8h[134]	Amazon parrots; area under curve increased compared to humans
	125 mg/kg PO q12h[171]	Chickens/PK; insufficient to achieve target plasma levels, no amoxicillin detected in eggs
	500 mg/L drinking water	Chickens/PD
Amoxicillin sodium	50–250 mg/kg IM, IV q8–12h	Most species, including bustards, pigeons, passerines, soft bills, and gram-positive and gram-negative bacteria
Amoxicillin trihydrate	55–200 mg/kg PO, SC, IM, IV, q4–12h	Most species/broad-spectrum bactericidal penicillin antibiotic; minimal activity for common gram-negative infections of birds; relatively low availability after oral administration
	150 mg/kg SC, IM q24h × 5 days (administer q48h with long-acting preparation)	Pigeons
	200–1500 mg/L drinking water	Pigeons, canaries, waterfowl
	1500–4500 mg/L drinking water	Psittacines
	300–600 mg/kg soft feed	Canaries, psittacines

Continued

*Although the authors attempted to verify dosages contained in this formulary, errors in the original sources or in the preparation of this chapter may have occurred. All users of this reference, therefore, should empirically evaluate all dosages to determine that they are reasonable before use. The publisher assumes no responsibility and makes no warranty with respect to results obtained from the uses or dosages listed, for any misstatement or error, negligent, or otherwise, contained in this chapter. In addition, the authors do not necessarily endorse specific products or dosages reported in this formulary. Also, the listing of a drug or commercial product in this chapter does not indicate approval by the Food and Drug Administration or the manufacturer for use in exotic animals. Drug dosages are listed in ascending order; feed and water dosages are listed last.
Most of the avian formulary information has been modified with permission from Carpenter JW, editor: *Exotic animal formulary*, ed 5, St. Louis, 2017, Saunders.

TABLE 1

Antibacterial Agents—cont'd

Agent	Dosage	Species/Comments
Ampicillin sodium	50–150 mg/kg IM q6–12h	Most species/PD
	150–200 mg/kg PO q8–12h	Amazon parrots/PD; therapeutic levels not achieved in one species at this dosage
	528 mg/L drinking water	Pigeons/PD; *Streptococcus bovis*
Ampicillin trihydrate	15–25 mg/kg SC, IM q12h	Ratites, cranes, raptors/PD
	100 mg/kg PO, IM q4–12h	Most species
	150–175 mg/kg PO, q8–12h[47]	Amazon parrots, oral dosing resulted in prolonged (up to 10 hours) but less consistent plasma levels compared to IM dosing
	1000–2000 mg/L drinking water	Galliformes, canaries/flock use
	2000–3000 mg/kg soft feed	Canaries/aviary use
Ampicillin/sulbactam (Unasyn, Pfizer)	200–300 mg/kg IM, IV q8h[54,55]	Poultry/PD
Azithromycin (Zithromax, Pfizer)	10–40 PO q24–48h	Most species/PD in cockatiels and macaws; found to be effective in eliminating *Chlamydia psittaci* infections after 21 days
	10 mg/kg PO q48h[21]	Blue-and-gold macaws/PK
	20 mg/kg PO, IM q72h[1]	Chicken/PK
	43 mg/kg PO q24h[160]	Ring-necked doves/PD, combined use with ethambutol (30 mg/kg PO q24h) and rifampin (45 mg/kg PO q24h) for 180 days; efficacy against *Mycobacterium avium* appeared poor
Azithromycin sustained-release granules (Zetamax, Pfizer)	35 mg/kg PO loading dose followed by 25 mg/kg PO q12h[207]	Pigeons/PK
Carbenicillin (Geocillin, Roerig; Pyopen, SmithKline Beecham)	100–200 mg PO, IM, IV q6–12h	Most species
Cefadroxil	100 mg/kg PO q12h	Most species
Cefazolin	25–75 mg/kg PO, IM, IV q12h	Most species
Cefotaxime (Claforan, Hoechst-Roussel)	50–100 mg/kg IM, IV q8–12h	Most species
Cefovecin (Convenia, Pfizer)	10 mg/kg SC, IM, IV q1h	Pigeons, chickens/PD; not recommended for use in birds due to short half-life; cannot be used q14d as in dogs and cats
Cefoxitin	50–100 mg/kg IM, IV q6–12h	Most species
Ceftazidime	50–100 mg/kg IM, IV q4–8h	Most species
Ceftiofur crystalline-free acid (Excede, Pfizer)[a]	10–20 mg/kg IM q3–5d[95,103,159,193,200]	Red-tailed hawks/PD; American black ducks/PD; helmeted guineafowl/PD; cattle egrets/PK; American flamingos/PK
	10 mg/kg IM q60–110h[158]	Bald eagles/PK
	20 mg/kg IM q80–160h[158]	Bald eagles/PK
	50 mg/kg IM once[187]	Ring-necked doves/PK; plasma concentrations remained above MIC for 108 hours
Ceftiofur sodium (Naxcel, Pfizer)[a]	10–50 mg/kg IM q4–12h	Most species/PD in cockatiels and one species of Amazon parrot
	2 mg/kg IV or IO[107]	Chickens/regional limb perfusion
Ceftriaxone	75–100 mg/kg IM q4–8h	Most species/PD in chickens
Cephalexin	50–125 mg/kg IM, PO q6–12h	Most species/PD mostly in poultry
Cephalothin	100 mg/kg IM, IV q6–8h	Most species/PD in poultry
Chloramphenicol palmitate (oral suspension)[a]	25–50 mg/kg PO q8h	Most species
Chloramphenicol succinate[a]	22 mg/kg IM, IV q3h	Ducks (PD), raptors
	50 mg/kg IM q6–12h	Most species; PD in macaws, conures, peafowl, eagles, geese
	200 mg/kg IM q12h × 5 days	Budgerigars/PD
Chlortetracycline (Aureomycin Soluble Powder, Cyanamid)	15–20 mg/kg PO q8h	Pigeons/PD
	100 mg/kg PO q6h	Raptors
	0.5%–1% pellets × 30–45 days	Psittacines/*Chlamydia*; reduce calcium in diet to 0.7%
	5000 mg/kg soft feed × 45 days	Chickens, turkeys/PD; medication of feed and water required to reach therapeutic level
	2500 mg/L drinking water	Most species
Ciprofloxacin (Cipro, Bayer)[a]	15–40 mg/kg PO, IM, IV q12h	Most species
	50 mg/kg PO q12h	Raptors/PD
	80 mg/kg PO q24h	Most species/*Mycobacterium*; use in combination with other agents

TABLE 1

Antibacterial Agents—cont'd

Agent	Dosage	Species/Comments
Clarithromycin (Biaxin, Abbott)	10–85 mg/kg PO q24h	Most species/*Mycobacterium*; allometrically scaled
Clindamycin	25–50 mg/kg PO q8–12h	Most species
	100 mg/kg PO q6h[111]	Pigeons/PD
	100–150 mg/kg PO q12h	Raptors; osteomyelitis
Clofazimine (Lamprene, Novartis)	1–12 mg/kg PO q12–24h	Most species/*Mycobacterium*; use in combination with other agents
Cloxacillin	100–250 mg/kg PO, IM q12–24h	Most species, including raptors; recommended for treating pododermatitis
Danofloxacin mesylate (A180, Pfizer)[a]	5–10 mg/kg PO, SC, IM, IV[28,38]	Most species
	15 mg/kg IM[165]	Californian brown pelicans/PK
Doxycycline (Vibramycin, Pfizer)	25–50 mg/kg PO q12–24h	Cockatiels/PD; *Chlamydia*; treatment for 21 and 45 days both effective; may cause regurgitation; use low end of dose range for macaws and cockatoos
	400–800 mg/L drinking water (make fresh daily)	Waterfowl, budgerigars (PD); treatment for 21 days in cockatiels with spirochete bacteria
	300 mg/kg diet (wet weight) × 42 days[62]	Cockatiels/PD; doxycycline was added to soybean oil and then mixed with a pelleted diet formulated with reduced calcium (to reduce doxycycline chelation) and a low-fat content
	1000 mg/kg feed	Large psittacines on dehulled seed (PD), macaws on corn (PD), canaries, large psittacines on soft feed (10 mg/mL syrup mixed into 29% kidney beans, 29% canned corn, 29% cooked rice, and 13% dry oatmeal cereal)
Doxycycline (Vibravenös, Pfizer)	50–100 mg/kg SC, IM q5–7d × 5–7 doses	Most species; PD in pigeons and houbara bustards
Doxycycline hyclate (Vibramycin injection, Pfizer)	25–50 mg/kg slow bolus IV q24h × 3 days	Psittacines (cardiovascular collapse associated with the propylene glycol carrier can occur after rapid IV injection)
	20 mg/kg PO, IV q24h × 3 days[201]	Laying hens/PK
	75–100 mg/kg SC, IM q5–7d	Pigeons/PD
Doxycycline (Doxirobe gel, Pharmacia)	Topical	Most species/apply to beak or pododermatitis lesions; use in conjunction with debridement; antibiotic is released for 28 days
Enrofloxacin[a]	—	Injectable formulation is highly alkaline and may cause tissue necrosis and pain; repeated IM injections are not recommended; no detectable effect on cartilage in day-old chicks; appears stable when compounded and stored at room temperature; may be administered in some food items
	7.5–9.5 mg/kg IM, IV q24h[195]	Crested caracaras/PD
	10–15 mg/kg PO, SC, IM q12h[33,192]	Most species/PD
	15 mg/kg PO, SC q24h[131]	Caribbean flamingos/PK
	15 mg/kg PO, IM, IV q12h	Raptors/PD; IV administration in owls may result in weakness, tachycardia, and vasoconstriction
	15–30 mg/kg PO, SC, IM q12–24h	Most species/PD for pulse dosing in some species
	50 mg/L drinking water	Chickens, turkeys/PD
	100–200 mg/L drinking water	Psittacines, pigeons/PD
	200–750 mg/mL drinking water	Psittacines/PD
	250–1000 mg/kg feed	Passerines; psittacines/PD
	50 mg/kg via nebulization × 4 hours (day 1, AM), then 25 mg/kg × 4 hours/day × 4 days	Muscovy, Pekin ducklings/*Riemerella (Pasteurella)*
	0.2 mg/mL saline, flush q24h × 10 days	Raptors/nasal flush
Erythromycin	10–20 PO, IM q12–24h	Most species; IM injection can cause muscle necrosis
	100 mg/kg PO	Pigeons/PD
	100–1500 mg/L drinking water	Most species; pigeons/PD

Continued

TABLE 1

Antibacterial Agents—cont'd

Agent	Dosage	Species/Comments
Ethambutol (Myambutol, Lederle)	10–30 mg/kg PO q12–24h	Most species/*Mycobacterium*; use in combination with other agents
Florfenicol[a]	30 mg/kg PO q12h[184,185]	Geese, ducks/PK
Gentamicin	2.5–5 mg/kg IM q8–12h	Most species; nephrotoxic; should be well hydrated; caution at higher doses
	5–10 mg/kg IM q8–12h	Cockatiels/PD
	5–10 mg/kg IM q4–6h	Pigeons, quail/PD
	2–3 drops ophthalmic solution intranasal q8h	Most species
Isoniazid	5–30 mg/kg PO q12–24h	Most species/*Mycobacterium*; should be used in combination with other drugs
Kanamycin (Kantrim, Fort Dodge)	10–20 mg/kg IM q12h	Most species
	13–65 mg/L drinking water	Most species; make fresh daily
Lincomycin	0.25–0.5 mL intraarticular q24h	Raptors
	25–50 mg/kg PO q12h	Most species
	50–100 mg/kg PO, IM q12–24h	Psittacines, raptors
Lincomycin/spectinomycin (LS-50 Water Soluble, Upjohn; Linco-Spectin 100 Soluble Powder, Upjohn)	50 mg/kg PO q24h	Most species
	528 mg/L drinking water	Turkey poults/PD; *Mycoplasma* airsacculitis
	750 mg/L drinking water × 3–7 days	Waterfowl
Marbofloxacin (Zeniquin, Pfizer)[a]	2–2.5 mg/kg PO q24h[74,205]	Broiler chickens and Muscovy ducks/PD
	2.5–5 mg/kg PO q24h	Blue-and-gold macaws/PD
	2–3 mg/kg IV, IO q24h	Raptors/PD
	3–12 mg/kg PO q24h	Turkeys/PD
	5 mg/kg IM, IV	Ostriches/PD
	10–15 mg/kg IV q24h[67]	Ducks/PD
	10–15 mg/kg PO, IM q12–24h	Raptors, bustards/PD
Meropenem (Merrem, Abbott)	175 mg/kg IM q24h	Pigeons/PD; broad-spectrum carbapenem antibiotic, penetrates most tissues and fluids
Metronidazole[a]	10–50 mg/kg PO q12–24h	Most species
Minocycline	10–15 mg/kg PO q12h	Penguins, raptors
	5000 mg/kg feed	Parakeets/use as antibiotic impregnated millet
Neomycin	Topical q6–12h	Use is generally limited to topical formulations for skin, eyes, and ears and, occasionally, oral treatment of enteric infections; not absorbed from GI tract
	10 mg/kg PO q8–12h	Most species
	80–264 mg/L drinking water	Waterfowl, Galliformes
	70–220 mg/kg feed × 14–21 days	Waterfowl, Galliformes/*Clostridium*, necrotizing enteritis
Norfloxacin (Noroxin, Merck; Vetriflox 20% Oral Solution, Lavet Ltd)[a]	—	Fluoroquinolone; not approved for use in food-producing birds in the United States; administration of the total daily dose to chickens over 2–4 hours (pulse dosing) has been recommended
	8 mg/kg PO q24h	Chickens/PD
	10 mg/kg PO q24h	Chickens, geese/PD
	10 mg/kg PO q6–8h	Turkeys/PD
	15 mg/kg in water over 2–4 hours	Turkeys/PD
	100 mg/L drinking water × 5 days	Chickens/PD
Novobiocin sodium	15–30 mg/kg PO q24h	Poultry/effective against some gram-positive cocci
	220–385 mg/kg feed	Poultry, waterfowl
Orbifloxacin (Orbax, Schering-Plough)[a]	15–20 mg/kg PO q24h[93]	Japanese quail/PD
Ormetoprim-sulfadimethoxine (Primor, Pfizer)	60 mg/kg PO q12h	Pigeons
	475–951 mg/L drinking water × 7–10 days	Pigeons
	200–800 mg/kg feed	Waterfowl/colibacillosis

TABLE 1

Antibacterial Agents—cont'd

Agent	Dosage	Species/Comments
Oxytetracycline	—	IM administration may cause muscle irritation or necrosis; may be useful in treating *Chlamydophila*, fowl cholera; products or foods containing Al, Ca, Mg, Fe reduce or alter absorption; outdated tetracycline is nephrotoxic
	2 mg/mL nebulization q4–6h	Parakeets/requires ultrasonic nebulizer; therapeutic concentrations of antibiotic were present in lung and trachea
	5 mg/kg SC, IM q12–24h	Chicken chicks/PD
	10 mg/kg IM q24h[69]	Chickens/PK
	20 mg/kg IM q72h (long-acting formulation)[69]	Chickens/PK
	16 mg/kg IM q24h	Great horned owls/PD
	23 mg/kg IV q6–8h or 43 mg/kg IM q24h	Pheasants/PD
	50–100 mg/kg SC, IM q2–3d	Cockatoos (PD), passerines
	58 mg/kg IM q24h	Amazon parrots/PD
	2500 mg/L drinking water and 2500 mg/kg feed	Chickens (PD), turkeys (PD), waterfowl/simultaneous medication of feed and water required to reach therapeutic level
	300 mg/kg soft feed	Psittacines
Penicillin	50,000 IU/kg IM	Waterfowl/*Erysipelas*, new duck disease
	8 g/40 g packet bone cement	PMMA beads (ratio 1:5)
Penicillin benzathine/procaine	200 mg/kg IM q24h	Most species: anecdotal reports suggest should not be used in birds <1 kg BW because of possible toxic effects
Penicillin G	6 mg/kg IV	Ostriches, emus/PD; rapidly eliminated; small volume of distribution
Piperacillin sodium	—	Extended-spectrum penicillin; not available as a veterinary product in the United States; see piperacillin/tazobactam
	100 mg/kg IM q4–6h	Red-tailed hawks, great horned owls/PD
	200 mg/kg IM q8h	Budgerigars (PD), raptors
	0.02 mL (4 mg) in macaw eggs; 0.01 mL (2 mg) in small eggs	Eggs/inject 200 mg/mL solution into air cell on days 14, 18, and 22
Piperacillin(P)/tazobactam(t) (Zosyn, Tazocin, Wyeth)	(P) 87 mg/kg + (t) 11 mg/kg IM q3–4h[22]	Hispaniolan Amazon parrots/PD
Rifabutin (Mycobutin, Pharmacia)	15–45 mg/kg PO q24h	Most species including passerines/*Mycobacterium*; use in combination with other agents
Rifampin	10–20 mg/kg PO q12–24h	Most species/*Mycobacterium*; use with other agents; may cause/be associated with hepatitis, CNS signs, depression, and vomiting
	45 mg/kg PO q24h	
Silver sulfadiazine	Topical q12–24h	Most species/burns, ulcers; Amazon foot necrosis; bandage application preferred
Streptomycin (Streptomycin Sulfate, Roerig)	—	May be nephrotoxic or neurotoxic; not recommended; consider amikacin as an alternative; *Mycobacterium*; use in combination with other agents
	20–50 mg/kg PO, IM q12–24h	Most species; chickens/PD
Sulfadimethoxine (Albon, Pfizer)	25–55 mg/kg PO q24h × 3–7 days	Raptors; loading dose at higher end × 1 day
	50 mg/kg PO q24h	Cranes
	330–400 mg/L drinking water on day 1 followed by 200–265 mg/L × 4 days	Pigeons
Tetracycline	50 mg/kg PO q8h	Most species, including passerines
	200–250 mg/kg PO q12–24h	Most species/gavage
	40–200 mg/L drinking water	Most species, including game birds
	100–600 mg/kg feed	Game birds
Tiamulin (Denagard, Novartis)	12.5 mg/kg PO × 3 days	Poultry/intestinal spirochetosis
	25–50 mg/kg PO q24h	Most species; bacteriostatic antibiotic; effective against *Mycoplasma*
Ticarcillin/clavulanate (Timentin, Smith-Kline Beecham)	100–200 mg/kg IM, IV q12h	Most species; extended-spectrum penicillin; very poor absorption if injectable given orally
Tilmicosin (Micotil 300 Injection, Provitil-powder and Pulmotil AC-liquid, Elanco)	30 mg/kg PO q24h	Poultry/PD
	100–500 mg/L drinking water × 5 days	Poultry chicks/*Mycoplasma*; macrolide; handle with caution; potentially fatal to humans

Continued

TABLE 1

Antibacterial Agents—cont'd

Agent	Dosage	Species/Comments
Tobramycin	0.25–0.5 mL intraarticular flush q24h × 7–10 days	Raptors/septic arthritis
	2.5–5 mg/kg IM q8–12h	Most species; neurotoxicity or nephrotoxicity may develop
Trimethoprim-sulfadiazine (Tribrissen, Schering-Plough; Septra, Monarch)	12–60 mg/kg PO q12h	Raptors/useful for sensitive infections in neonates
	20 mg/kg SC, IM q12h	Psittacines
	30 mg/kg PO, IM, IV q12h	Ostriches/PD
	30 mg/kg PO q8h	Psittacines; combine with pyrimethamine for treatment of sarcocystosis
	107 mg/L drinking water	Galliformes
Trimethoprim/sulfamethoxazole (Bactrim, Roche; Septra, Burroughs Wellcome)	48 mg/kg PO, IM q12h	Raptors
	60 mg/kg PO q24h	Pigeons
	100 mg/kg PO q12h	Most species, including psittacines; reduce dose if regurgitation occurs
	360–400 mg/L drinking water	Most species, including pigeons
	400 mg/kg feed	Geese
Tylosin (Tylan, Elanco)	15–30 mg/kg IM q6–12h	Most species; effective against gram-positive bacteria, *Mycoplasma*, *Chlamydia*, and *Pasteurella*; very irritating to muscles when administered IM
	50 mg/kg PO q24h	Passerines, pigeons
	60 mg/kg/day PO[45]	Muscovy ducks/PD against *Mycoplasma anatis*
	50 mg/L drinking water	Most species
	250–1000 mg/L drinking water	Most species/*Mycoplasma*

[a]These drugs are banned in food-producing species in the United States as well as some other countries. Use with caution and follow local laws regarding administration to food animals.

AM, ante meridiem, in the morning, *BW,* Body weight; *CNS,* central nervous system, *GI,* gastrointestinal, *IM,* intramuscular; *IV,* intravenous; *PD,* pharmacodynamic; *PK,* pharmacokinetic; *PMMA,* polymethyl methacrylate; *PO,* oral; *SC,* subcutaneous.

TABLE 2

Antifungal Agents

Agent	Dosage	Species/Comments
Amphotericin B	—	Fungicidal; lipid-based amphotericin B products are now available that have less toxicity than conventional desoxycholate form; *Macrorhabdus ornithogaster* (avian gastric yeast); preferred IV agent for aspergillosis; intratracheal administration for syringeal aspergilloma may cause tracheitis; potentially nephrotoxic; resistance may develop
	1.5 mg/kg IV q8h × 3–7 days	Most species
	1 mg/kg intratracheal q8–12h, dilute to 1 mL with sterile water	Psittacines, raptors/aspergillosis
	1 mg/kg intratracheal q12h × 12 days, then q48h × 5 weeks	Raptors/syringeal aspergilloma
	100–109 mg/kg PO by gavage q12h × 10–30 days	Budgerigars/avian gastric yeast; compound in simple syrup
	0.05 mg/mL sterile water	Most species/nasal flush
	0.2 mL PO q12h × 10 days	Budgerigars/avian gastric yeast; use IV formulation (5 mg/mL)
	0.25–1 mL PO q24h × 4–5 days	Raptor neonates/candidiasis; not absorbed from alimentary tract
	1000 mg/L drinking water × 10 days	Budgerigars/avian gastric yeast
	Topical	Apply 10% solution to oropharynx
	7 mg/mL saline q12h	Most species/nebulization × 15 minutes
Amphotericin B (3% cream)	Topical to affected area q12h	Most species/mycoses
Amphotericin B (A)/proteolytic nasal flush (P)	Nasal flush (A) 1 mg/kg + (P) 0.2–0.4 mL diluted in 20 mL saline q24h	Uses a commercial neomycin-chymotrypsin-trypsin-hydrocortisone ointment (Kymar, Schering-Plough); 10 mL per naris (flushed vigorously in small amounts); consider compounding without hydrocortisone to avoid any immunosuppressive side effects
Clotrimazole	—	Broad-spectrum antifungal agent; inhibits the growth of pathogenic yeasts such as *Candida albicans*; used commonly as adjunctive therapy for aspergillosis; administer via air sac, intratracheally, nebulization, or topically
	2 mg/kg intratracheal q24h × 5 days	Psittacines/syringeal aspergilloma; apply with catheter or endoscope directly into syrinx during anesthesia
	10 mg/mL saline flush	Most species/effective against *Aspergillus* at sites that can be flushed
	1% solution	Nebulization × 30–60 minutes
Enilconazole emulsion	6 mg/kg PO q12h	Eclectus parrots/glossal candidiasis
	1 mg (0.5 mL)/kg intratracheal of a 1:10 dilution q24h	Falcons/aspergillosis
	200 mg/L drinking water	Canaries/cutaneous dermatophytosis
	Topical 1:10 dilution q12h	Raptors/cutaneous aspergillosis, candidiasis
	Topical or intratracheal 1:10–1:100 dilution	Psittacines/aspergillosis, candidiasis
	0.1 mL/kg in 5 mL sterile water, nebulize × 30 minutes, 5 days on, 2 off, up to 3 months	Raptors/aspergillosis
Fluconazole (Diflucan, Pfizer)	—	Fungistatic; penetrates well into brain, cerebrospinal fluids, and eyes; *Candida*; only indicated if topical treatment (i.e., nystatin) is not feasible; water-soluble; safest therapeutic index of the azoles; may be ineffective against aspergillosis; death observed in budgerigars at 10 mg/kg PO q12h (this dose was also ineffective against avian gastric yeast with *Macrorhabdus*); compounded oral suspension stable for 14 days when refrigerated
	2–6 mg/kg PO q12–24h	Most species
	5 mg/kg PO q24h, 10 mg/kg PO q48h[151]	Cockatiels/PD
	5–15 mg/kg PO q12h × 14–60 days	Most species
	8 mg/kg PO q24h × 30 days	Psittacines/cryptococcosis
	20 mg/kg PO q48h	Psittacines/PD; mucosal, systemic yeast infections
	100 mg/L in drinking water[151]	Cockatiels/PD
	100 mg/kg soft food	Gouldian finches/candidiasis

Continued

TABLE 2

Antifungal Agents—cont'd

Agent	Dosage	Species/Comments
Flucytosine (Ancobon, Roche)	—	Fungistatic agent; penetrates well into CNS; used prophylactically in raptors (especially falcons) and waterfowl to prevent aspergillosis; may be administered as adjunctive treatment; about 50% of *Aspergillus* strains are resistant; toxicity is low; however, adverse effects may include gastrointestinal effects, hepatotoxicity, and bone marrow depression
	20–75 mg/kg PO q6–12h	Most species/generalized yeast or fungal infections
	75 mg/kg q12h × 5–7 days, then q24h × 14 days	Raptors/prophylaxis for prevention of aspergillosis
	75–120 mg/kg PO q6–12h	Most species/aspergillosis
	50–250 mg/kg feed	Psittacines, mynah birds
Griseofulvin	10 mg/kg PO q12h	Pigeons/dermatophytosis; gavage
	30–50 mg/kg q24h in drinking water	Ostriches/mycotic dermatitis
Itraconazole (Sporanox, Janssen)	—	Most species/systemic mycoses, superficial candidiasis, dermatophytosis; fungistatic; maximal oral bioavailability when taken with a full meal; commercially available suspension is recommended as a first choice; do not use compounded formulations because bulk drug is not bioavailable or stable
	2.5–5 mg/kg PO q24h	Grey parrots/anorexia, depression, and toxicity reported at higher doses in this species
	5–10 mg/kg PO q24h	Blue-fronted Amazon parrots/PD; aspergillosis; 10 mg/kg is required to achieve therapeutic concentrations in poorly perfused tissues
	5–10 mg/kg PO q12–24h × 10–14 days, then q48h	Raptors/aspergillosis prophylaxis
	5–10 mg/kg PO q12h	Passerines, waterfowl
	20 mg/kg PO q24h	Penguins/PD
	26 mg/kg PO q12h	Pigeons/PD; fungicidal levels achieved in respiratory tissue; further toxicologic studies required
	200 mg/kg feed up to 100 days	Gouldian finches/PD; dermatomycoses; beads from capsules were mixed with small amount of oil and seed
Itraconazole (Itrafungol, Elanco)	10 mg/kg PO q24h[161]	Lesser flamingos/PK
	20 mg/kg PO[206]	African penguins/PK
Ketoconazole (Nizoral, Janssen)	—	Most species/systemic mycoses (e.g., aspergillosis), candidiasis; fungistatic; less toxic than amphotericin B; more toxic than itraconazole; may be associated with potentially fatal hepatotoxicity; 20 mg/kg may cause regurgitation (discontinue for 1–2 days, then restart)
	30 mg/kg PO q12h × 7–14 days	Amazon parrots/PD
	30 mg/kg PO q12h × 7–30 days	Pigeons (PD), raptors/prophylactic in raptors for aspergillosis
	60 mg/kg PO q12h	Raptors/PD (common buzzard); aspergillosis
	200 mg/L drinking water, nectar, or soft feed × 7–14 days	Canaries, hummingbirds, Gouldian finches/dissolve crushed tablet in ½–1 tsp vinegar
Miconazole	—	Fungistatic; inhibits the growth of *Candida albicans*, *Malassezia*, and dermatophytes; injectable miconazole is not available in the United States
	5 mg/kg intratracheal q12h	Psittacines/10 mg/mL solution diluted with saline; syringeal mycoses
	10–20 mg/kg IM, IV q8–24h	Raptors, psittacines
	Topical to affected areas q12h	Cutaneous fungal infections; used in conjunction with oral itraconazole
Nystatin	—	Drug of choice for treatment of candidiasis; not systemically absorbed across intact gastrointestinal tract; oral lesions must be treated by direct contact with medication; when treating neonates, administer separately from formula to maximize concentration and contact time
	20,000–100,000 U PO q12–24h	Raptors, pigeons
	300,000–600,000 U/kg PO q8–12h	Most species
	Topical q6h	Hummingbirds/candidiasis; direct application using a cotton swab
	100,000 U/L drinking water	Canaries, finches

TABLE 2

Antifungal Agents—cont'd

Agent	Dosage	Species/Comments
Pimaricin (Natamycin, Alcon)	1 drop in affected eye q8h	Lovebirds/macrolide antifungal; keratomycosis
Silver sulfadiazine	Topical to affected areas q12–24h	Most species/bandage application preferred
Terbinafine	—	Fungicidal; questionable therapeutic potential for the treatment of aspergillosis in avian species; higher dose or use in combination with itraconazole may be more effective
	10–30 mg/kg PO q12–24h	Most species
	15 mg/kg PO q24h	Penguins/PD
	22 mg/kg PO q24h	Raptors/PD
	60 mg/kg PO q24h[52]	Amazon parrots/PD
	60 mg/kg PO q6–8h[155]	Shelducks/PK
Voriconazole (Vfend, Pfizer)	—	Most active drug against *Aspergillus*; some strains resistant; dose extrapolation difficult between species; PO and IV solutions available; may need to adjust dose for long-term treatment to maintain therapeutic concentrations; compounded suspensions stable up to 30 days at room temperature
	5 mg/kg PO q24h[99,136]	Magellanic penguins/PK, African penguins/PK
	10 mg/kg PO, IV q12h	Chickens/PD
	10 mg/kg PO q12h or 20 mg/kg q24h	Pigeons/PD
	10 mg/kg PO q8h[70]	Hawks/PD; more frequent dosing (e.g., up to q8h) may be necessary to maintain target concentrations during prolonged therapy
	12–18 mg/kg PO q12h	Grey parrots/PD
	12.5 mg/kg IM q24h[4]	Falcons/PK
	15 mg/kg PO q24h[137]	Red-tailed hawks/PK
	18 mg/kg PO q8h	Amazons/PD
	40 mg/kg PO q24h	Quail/PD
	20 mg/kg IV, PO q8–12h[106]	Ducks/PD

IM, Intramuscular; *IV,* Intravenous; *PD,* pharmacodynamic; *PK,* pharmacokinetic; *PO,* oral.

TABLE 3

Antiviral and Immunomodulating Agents[a]

Agent	Dosage	Species/Comments
Acemannan (CarraVet, Carrington Laboratories)	—	Has been demonstrated in poultry to induce macrophages to secrete interferon, tumor necrosis factor-α, and interleukins and thus may have immunostimulant, antiviral, antineoplastic, and gastrointestinal properties; used as adjuvant in some avian vaccines
	1–2 mg/kg SC or intralesional q7d	Chemotherapeutic adjunct therapy
Acyclovir (Zovirax, Burroughs Wellcome)	—	Antiviral agent; active against herpesviruses and cytomegalovirus; IM injection of the water-soluble sodium salt (IV formulation) may cause severe muscle necrosis; phlebitis and neurologic signs may occur with IV administration; most effective when administered before clinical signs begin; birds should be treated for a minimum of 7 days; the reconstituted solution is unstable and should be divided into aliquots and frozen
	10 mg/kg IM q24h × 5–14 days	Chickens/Marek disease
	20–40 mg/kg IM q12h	Psittacines/psittacine herpesvirus
	29 mg/bird PO q8h × 7 days	Pigeons/herpesvirus
	80 mg/kg PO q8h × 7 days	Quaker parakeets/PD; psittacine herpesvirus prophylaxis or treatment
	120 mg/kg PO q12h	Tragopans/PD
	330 mg/kg PO q12h × 4–7 days	Psittacine neonates/psittacine herpesvirus
	330 mg/kg PO q12h	Raptors/falcon and owl herpesvirus; may cause vomiting
	1000 mg/L drinking water	Quaker parakeets/herpesvirus; gavage
Amantadine (Symmetrel, Endo Labs)	—	Antiviral agent; inhibits replication of influenza A viruses
	1 mg/kg PO q24h × 3 weeks	Grey parrots/no effect on avian bornavirus infection
	10 mg/kg PO × 3 days pre- and 18 days postexposure	Turkeys/influenza viruses; must be administered before and during virus exposure
	25 mg/kg PO × 10 days following infection	Chickens
	100 mg/L drinking water	Chickens/can use simultaneously with killed influenza vaccine
Entecavir (Baraclude, Bristol-Myers Squibb)	—	Oral antiviral drug used in the treatment of hepatitis viruses
	1 mg/kg PO q24h × 14 days[53]	Ducklings/PD; antiviral agent; duck hepatitis B; given with duck hepatitis B virus (DHBV) deoxyribonucleic acid vaccines
Famciclovir (Famvir, Novartis)	25 mg/kg PO q12h	Ducklings/PD; antiviral agent; duck hepatitis; toxic effects were not reported
Imiquimod cream (Aldara, 3M)	Topical application	Psittacines/cloacal papillomatosis; thought to boost host cell-mediated immunity; masses decreased in size in one report, but not in another; complete remission did not occur in either
Interferon-α2a	—	Glycoprotein with immunomodulating and antiproliferative capabilities as well as antiviral activity
	30 U q24h × 5 days, 30 U 2×/week × 2 weeks, then 30 U q7d × 2 weeks	Most species
	60–240 U/kg SC, IM q12h or 300–1200 U/kg PO q12h	Most species/stock solution: mix 1 mL (3,000,000 U/mL) with 100 mL sterile water (30,000 U/mL); can freeze as 2 mL vials up to 1 year; mix 2 mL of stock into 1 L LRS (60 U/mL); refrigerate up to 3 months
Levamisole	—	Anthelmintic with immunostimulation properties; low therapeutic index (toxic reactions and deaths reported)
	1.25–2.5 mg/kg PO, IM, SC q7–14d	Most species
Oseltamivir	—	Neuraminidase inhibitor used for treatment of avian influenza
	0.5 mg/kg PO q12h × 5 days[39]	Duck, chickens/PD; administered prophylactically to healthy animals in the event of an outbreak in valuable zoological collections
Penciclovir (Denavir, Novartis)	10 mg/kg IP q24h × 12–24 weeks	Ducks/PD; antiviral agent active against herpesviruses; DHBV; viral levels were significantly reduced; no toxic effects observed; dissolve in 2 mL of 1% DMSO
Rimantadine (Flumadine, Forest)	100 mg/L drinking water	Chickens/influenza viruses; must be used before and during exposure

[a]All antiviral drugs are banned in food-producing species in the United States as well as some other countries. Use with caution and follow local laws regarding administration to food animals.

DMSO, Dimethyl sulfoxide; *IM,* intramuscular; *IV,* intravenous; *LRS,* lactated Ringer's solution; *PO,* oral; *SC,* subcutaneous.

TABLE 4

Antiparasitic Drugs

Agent	Dosage	Species/Comments
Albendazole	5–50 mg/kg PO	Broad-spectrum anthelmintic; may be toxic in keas, some Columbiformes at 50–100 mg/kg
Amprolium	13–30 mg/kg PO	Pyrimidine derivative coccidiostat; most species
Carnidazole[a]	5–50 mg/kg PO	Treatment for *Trichomonas*, *Giardia*, *Hexamita*, *Histomonas*, most species
Chloroquine phosphate	5–25 mg/kg PO	Generally used with primaquine for *Plasmodium*, *Haemoproteus*, and *Leucocytozoon*; overdose can result in death
Clazuril	3–7 mg/kg PO	Most species: drug detected in eggs after multiple dosing
Diclazuril	5–10 mg/L drinking water	Some *Eimeria* resistance in poultry documented recently; rotate for long-term prevention
Fenbendazole	10–50 mg/kg PO	Most species/anthelmintic effective against cestodes, nematodes, trematodes, *Giardia*, and acanthocephalans; toxicity documented in pigeons and doves; may be toxic to other species, including raptors, vultures, lories, storks, and pelicans
Fipronil	7.5 mg/kg; spray on skin once, repeat in 30 days PRN	Apply via pad to base of neck, tail base, and under each wing; avoid plumage during application; alcohol may create dry, brittle feathers; do not soak bird; do not exceed 7.5 mg/kg
Flubendazole	4–20 mg/kg PO once or split over 3–9 days[145]	Gray partridge/*Trichostrongylus tenuis*
	1.43 mg/kg in drinking water for 7 days[183]	Chickens/*Ascaridia galli*; birds were found to be reinfected within 7 days
	30 ppm in feed[181]	Chickens/effective against *Ascaridia galli*, *Heterakis gallinarum*, and *Capillaria*
	1.2 g/kg of feed[77]	Geese/*Cyathostoma bronchialis*; eliminated clinical signs but these recurred, and subsequent treatment with fenbendazole was effective
Imidocarb dipropionate	5–7 mg/kg IM once, repeat in 7 days	Raptors/*Babesia*; some cases require a total of 3 treatments
Ivermectin	0.2–0.4 mg/kg PO, SC, IM, topical once, can repeat in 10–14 days	Dilute with water or saline for immediate use; dilute with propylene glycol for extended use; parenteral ivermectin may be toxic to finches and budgerigars
Levamisole	1.5 mg/kg topically split between both eyes q7d for 2 doses[129]	Ostriches/*Philophthalmus gralli*
	16 mg/kg PO q24h × 3 days[177]	Chickens/reduced fecal helminth egg burden; not as effective as fenbendazole
	25–43 mg/kg PO once[68]	Brown kiwi/highly toxic; 6/9 birds died within 4 hours
	28 mg/kg PO once[57]	Chickens/*Ascaridia galli*
	28 mg/kg in drinking water provided for 8 hours[57]	Chickens/*Ascaridia galli*
Metronidazole[a]	10–30 mg/kg PO q12	Most species/antiprotozoal, including alimentary tract protozoa (especially flagellates such as *Giardia*, *Histomonas*, *Spironucleus*, and *Trichomonas*)
	30–50 mg/kg PO q24h	Most species
	100 mg/L drinking water or soft food	Canaries
	200 mg/L drinking water × 7 days	Passerines
Milbemycin oxime	2 mg/kg PO, repeat in 28 days	Galliformes
Paromomycin	—	Aminoglycoside with antiprotozoal activity
	50–100 mg/kg PO q8–24h[189]	Falcons/*Cryptosporidium baileyi*; no apparent efficacy
	100–400 ppm in feed[89]	Turkeys/*Histomonas meleagridis*; flock mortality only 20% at 400 ppm
	5 mg/kg in feed for 6 weeks[112]	Turkeys/*Histomonas meleagridis*; 2 out of 3 flocks required early culling due to high mortality rates
Ponazuril	10–20 mg/kg PO repeat in 10–14 days	Most species including pigeons, waterfowl
	25 mg/kg PO, IM, repeat in 10–14 days	Bali mynahs/cestodes

Continued

TABLE 4

Antiparasitic Drugs—cont'd

Agent	Dosage	Species/Comments
Praziquantel	—	Anthelminthic
	10 mg/kg PO q7d × 4 weeks[35]	Passerines/*Szidatitrema* trematodes; effective in most birds; those that remained positive on fecal examination after several months were then treated with 20 mg/kg IM q7d × 4 weeks
	10–100 mg/kg IM q14d[3]	Chickens/not effective against *Philophthalmus gralli*
	20 mg/kg PO once[154]	Black stilts/no difference in fecal egg counts between treated and control groups
	40–200 mg/kg IM once[14]	Common mergansers/schistosomes; 40 mg/kg was insufficient whereas 200 mg/kg reduced parasite load
	56.8–85.2 mg nebulized in 100% oxygen[35]	Blue-crowned motmots (n = 2) and fairy bluebird (n = 1)/effective at reducing total parasite burden of air sac trematodes
	Topically OU q12h (1% ointment)[25]	Greater rheas/*Philophthalmus gralli*
Primaquine	—	Hematozoa (i.e., *Plasmodium*, *Haemoproteus*, and *Leucocytozoon*); use in conjunction with chloroquine; dosage based on amount of active base rather than total tablet weight
	0.3 mg/kg PO (at 24 hours following the initial chloroquine dose) q24h × 7–10 days	Penguins and raptors/use with chloroquine (10 mg/kg at 0 hours, then 5 mg/kg at 6, 24, and 48 hours)
	0.75–1 mg/kg PO once	Most species, including raptors/*Plasmodium*; use with chloroquine (25 mg/kg at 0 hours, then 15 mg/kg at 12, 24, and 48 hours); palliative therapy
	1 mg/kg PO q7d	Use with chloroquine (10 mg/kg q7d) as a preventative regimen for birds recovering from *Plasmodium* infection
	1 mg/kg PO q24h × 2 days, repeat q7d × 3–5 treatments to prevent relapse	Raptors/*Plasmodium*; use with chloroquine (20 mg/kg IV initially, followed by 10 mg/kg PO at 6, 18, and 24 hours)
	15 mg PO[20]	African penguins/PK
Pyrimethamine (Fansidar, Roche)	—	Most species/*Atoxoplasma*, *Plasmodium*; chloroquine and primaquine are preferred; overdosage may cause hepatotoxicity; *Toxoplasma*, *Atoxoplasma*, *Sarcocystis*; may be effective for *Leucocytozoon*; supplement with folic acid
	0.25–0.5 mg/kg PO q12h × 30 days	Raptors, waterfowl/*Sarcocystis*, *Toxoplasma*
	0.5 mg/kg PO q12h × 14–28 days	Most species/use for 28 days for *Leucocytozoon* in raptors
	0.5 mg/kg PO q12h × 30 days	Waterfowl/*Sarcocystis*
	0.5–1 mg/kg PO q12h × 30 days	Eclectus, Amazon parrots/use with trimethoprim-sulfadiazine (30 mg/kg)
	1 mg/kg feed	Game birds
	100 mg/kg feed	Most species
Quinacrine HCl (Atabrine, Sanofi)	—	Most species/*Atoxoplasma*, *Plasmodium*; chloroquine and primaquine preferred; overdosage may cause hepatotoxicity
	5–10 mg/kg PO q24h × 7–10 days	Most species/use higher doses for *Atoxoplasma*, *Lankesterella*, *Plasmodium*
	26–79 mg/L drinking water × 10–21 days	Pigeons
Ronidazole[a]	2.5 mg/kg PO × 6 days	Pigeons
	6–10 mg/kg PO q24h × 6–10 days	Most species
	10–20 mg/kg PO q24h × 7 days	Pigeons
Selamectin	—	No adverse effects, including neurological signs, were seen in healthy zebra finches with doses up to 92 mg/kg
	20 mg/kg topically q19d[90]	Helmeted guineafowl/PK
	20 mg/kg topically q14d[152]	Lorikeets (*Trichoglossus* spp.)/PK
	23 mg/kg topically, repeat in 3–4 weeks	Budgerigars/*Knemidocoptes* improvement 13/14 birds at 4 weeks, no neurological signs identified but monitor for weight loss

TABLE 4

Antiparasitic Drugs—cont'd

Agent	Dosage	Species/Comments
Sulfachloropyrazine	1 g of 30% powder/L of drinking water × 5 days, off 3 days, on 5 days, then repeat cycle 4×; administer treatment 3× annually	Bali mynahs/*Atoxoplasma*; significantly reduced or totally cleared oocyst shedding for extended time; it is uncertain if the drug is safe to use when parents are feeding chicks; supplement with vitamin B_6. No adverse effects, including neurological signs, were seen in healthy zebra finches with doses up to 92 mg/kg
Sulfachloropyridazine	150–300 mg/L drinking water; 5 days/week × 2–3 weeks	Passerines, including canaries/may need to treat for months for systemic coccidiosis
	300 mg/L drinking water × 5 days, off 3 days, on 5 days, then repeat cycle 4×; administer treatment 3× annually	Passerines, including Bali mynahs/*Atoxoplasma*
	300 mg/L drinking water × 7–10 days	Pigeons
	300–1000 mg/L drinking water × 3 days, off 2 days, then repeat course	Pigeons
	400–500 mg/L drinking water × 5 days, off 2 days, on 5 days	Most species
Sulfadimethoxine	25 mg/kg PO q12h × 5 days	Raptors
	25–50 mg/kg PO q24h × 3 days	Raptors
	25–50 mg/kg PO q24h × 3 days, off 2 days, then q24h × 3 days	Raptors/*Eimeria*, *Sarcocystis*
	25–55 mg/kg PO q24h × 3–7 days	Raptors
	50 mg/kg PO once, followed by 25 mg/kg PO q24h × 7–10 days	Psittacines
	50 mg/kg PO q24h × 5 days, off 3 days, on 5 days	Pigeons/close to toxic levels
	250 mg/L drinking water × 5 days	Turkeys
	330–400 mg/L drinking water × 1 day then 200 mg/L × 4 days	Pigeons/supplement with vitamin B for 5 days
	500 mg/L drinking water × 6 days	Chickens
Sulfaquinoxaline	—	Contraindicated with dehydration, liver disease, or bone marrow suppression; gastrointestinal upset, regurgitation are common, especially in macaws; use for longer than 2 weeks may require vitamin B (folic acid) supplementation
	250 mg/L drinking water × 6 days, off 2 days, on 6 days	Turkeys
	400 mg/L (1.4 mL/L) drinking water × 6 days, off 2 days, on 6 days	Chickens
	500 mg/L (1.8 mL/L) drinking water × 6 days, off 2 days, on 6 days	Pigeons
	225 mg/kg feed continuously	Turkeys
	450 mg/kg feed continuously	Chickens
Thiabendazole	40–100 mg/kg PO q24h × 7 days	Most species
	50 mg/kg PO, repeat in 14 days	Ostriches
	100 mg/kg PO once, repeat in 10–14 days	Raptors
	100–200 mg/kg PO q12h × 10 days	Raptors/nematodes; may interfere with egg laying
	100–500 mg/kg PO once	Most species
	250–500 mg/kg PO, repeat in 10–14 days	Most species, including psittacines
	425 mg/kg feed × 14 days	Pheasants, cranes

Continued

TABLE 4

Antiparasitic Drugs—cont'd

Agent	Dosage	Species/Comments
Toltrazuril (Baycox, Bayer)	—	Has been successful in reducing mortality from *Atoxoplasma* in passerines and may affect systemic stages of the disease; not very effective against *Atoxoplasma* when given in water; bitter taste, mixing with soft drink (e.g., cola) increases palatability; 2.5% solution is very alkaline and should not be gavaged directly into the crop
	7 mg/kg PO q24h × 2–3 days	Budgerigars, raptors
	10 mg/kg PO q24h × 2 days	Raptors/preferred treatment for *Caryospora*
	10 mg/kg PO q48h × 3 treatments	Raptors/treatment of choice for coccidiosis in falcons
	12.5 mg/kg PO q24h × 14 days	Bali mynahs/*Atoxoplasma*; dosage is based on a limited number of clinical cases
	15–25 mg/kg PO q24h × 2 days	Raptors
	25 mg/kg PO q7d × 3 treatments	Raptors
	50 mg/kg PO once	Most species/*Giardia*, *Trichomonas*, *Entamoeba*
	2 mg/L drinking water × 2 consecutive days/week	Psittacines
	5 mg/L drinking water × 2 days, repeat in 14–21 days	Lories/10 mg/L administered during second course of treatment
	12.5 mg/L drinking water × 2 days	Waterfowl
	20 mg/kg in drinking water × 2 days	Pigeons
	25 mg/L drinking water × 2 days	Chickens
	25 mg/L drinking water × 2 days, repeat in 14–21 days	Cockatiels, passerines (including goldfinches, manikins, siskins)
	75 mg/L drinking water × 2 days/week × 4 weeks	Passerines
	125 mg/L drinking water × 5 days	Pigeons
	200–400 mg/kg feed	Chickens/*Histomonas*; depressed weight gain on higher dosage
Trimethoprim-sulfadiazine	5 mg/kg IM q12h	Companion birds/*Sarcocystis*; use in conjunction with pyrimethamine (0.5–1 mg/kg PO q12h × 2 days, then 0.25 mg/kg PO q12h × 30 days)
	30 mg/kg PO q8–12h	Most species, including psittacines, raptors/*Sarcocystis* (treat for at least 6 weeks); coccidia
	60 mg/kg PO, SC q12h × 3 days, off 2 days, on 3 days	Raptors, waterfowl/coccidia
	80 mg/mL drinking water (trimethoprim)/40 mg/mL water (sulfadiazine)	Canaries/*Toxoplasma gondii*
Trimethoprim-sulfamethoxazole	25 mg/kg PO q24h	Toucans, mynahs/coccidia
	30 mg/kg PO q12–24h	Passerines/antiprotozoal
	320–525 mg/L drinking water	Poultry/coccidia
	480 mg/L drinking water q24h	Pigeons/antiprotozoal; see sulfonamides

[a]These drugs are banned in food-producing species in the United States as well as some other countries. Use with caution and follow local laws regarding administration to food animals.

IM, Intramuscular; *IV,* intravenous; *OU,* both eyes, *PK,* pharmacokinetic; *PO,* oral; *PRN,* as needed; *SC,* subcutaneous.

TABLE 5

Chemical Restraint/Anesthetic/Analgesic Agents[a]

Agent	Dosage	Species/Comments
Acepromazine	0.1–0.2 mg/kg IV 0.25–0.5 mg/kg IM	Ratites/most commonly used in combination with other anesthetics; rarely used in other species
Alfaxalone (Alfaxan, Jurox Pty Ltd)	—	Neuroactive steroid, $GABA_A$ receptor antagonist
	2 mg/kg IV[190]	Greater flamingos/smoother and shorter induction than with isoflurane
	4–7.5 mg/kg IV[121]	Chickens/induction of general anesthesia; lower doses required with premedication
	10 mg/kg IM or IV[108]	Mallard ducks/insufficient anesthesia for invasive procedures
	10 mg/kg IM[48]	Budgerigars/effective sedation, adequate for short minimally invasive procedures
	10–25 mg/kg IM[196]	Quaker parrots/induction agent; longer induction with lower dose; longer recovery at higher dose; addition of midazolam 1 mg/kg to protocol decreased muscle tremors and hyperexcitation
	10–50 mg/kg SC[138]	Bengalese finches/anesthetic induction agent; reduced induction time and longer duration with higher doses; faster induction and longer duration with addition of midazolam 0.7 mg/kg SC; longer duration with butorphanol 1 mg/kg SC
	15–20 mg/kg IM[10,156]	Budgerigars/mild-to-moderate sedation at 15 mg/kg; moderate-to-profound sedation at 20 mg/kg; duration about 30 minutes at either dose; both doses caused a significant decrease in respiratory rate
Amantadine (Symmetrel, Endo Labs)	—	Increases CNS dopamine release; weak NMDA receptor antagonist; synergistic with NSAIDs, opioids, gabapentin
	5 mg/kg PO q24h[13]	Orange-winged Amazons/PK
Atipamezole	2.5×–5× alpha-2 adrenergic agonist dose IM, IV	Alpha-2 adrenergic antagonist; 1:1 volume reversal of dexmedetomidine and medetomidine is general rule; can be divided ½ IM and ½ SC
Atropine sulfate	0.01–0.02 mg/kg SC, IM, IV	Most species/premedication
	0.04–0.1 mg/kg SC, IM, IV, IO, IT	Most species/bradycardia; higher doses with CPR
Bupivacaine HCl	—	May be shorter acting in some birds; minimize dose to limit potential toxic effects
	2 mg/kg infused SC	Mallard ducks/PD; high plasma levels at 6 and 12 hours postadministration so possible for delayed toxicity
	2–8 mg/kg perineurally	Mallard ducks/variable effectiveness for brachial plexus nerve block
Buprenorphine	0.6–1.8 mg/kg IM[85]	Cockatiels/did not change thermal nociceptive threshold
	0.6 mg/kg IM, IV q6–9h[78]	American kestrels/PK
	1.8 mg/kg SC q24h[72]	Red-tailed hawks/PK
Buprenorphine SR-LAB sustained-release formulation (Wildlife Pharmaceuticals)	1.8 mg/kg SC, IM q12–72h[80,87]	American kestrels/PK
Butorphanol tartrate	—	PO bioavailability <10% in Hispaniolan Amazon parrots; do not recommend in American kestrels
	0.05–0.25 mg/kg IV	Ratites
	0.5–5 mg/kg IM, IV q1–4h	Most species, including psittacines
	2–4 mg/kg IV[50]	Guineafowl; reduced sevoflurane MAC
	3 mg/kg (premedication) + 75 μg/kg/minute IV CRI (maintenance)	Psittacines; significantly reduced isoflurane MAC
	247 μg/kg/hour via osmotic pump implanted SC[26]	Peafowl/PK
Butorphanol tartrate in poloxamer 407 gel	12.5 mg/kg SC q4–8h[109]	Hispaniolan Amazon parrots/PK; long-acting formulation has longer half-life
Dexmedetomidine HCl	25 μg/kg IM	Common buzzards/adequate restraint to prevent reaction to handling but did not allow for intubation; no arrhythmias, excitement, or major adverse effects noted; complete reversal with atipamezole
	75 μg/kg	Common kestrels/adequate restraint to prevent reaction to handling but did not allow for intubation

Continued

TABLE 5

Chemical Restraint/Anesthetic/Analgesic Agents—cont'd

Agent	Dosage	Species/Comments
Diazepam	0.05–0.5 mg/kg IV	Most species
	0.25–0.5 mg/kg IM, IV q24h × 2–3 days	Raptors/appetite stimulant
	5 mg/kg PO, IV	Ratites/sedation
	5–10 mg/kg IM[144]	Zebra finches/deep sedation for several hours; complete recovery with flumazenil
	12–15 mg/kg intranasally	Ring-necked parakeets, canaries/dose divided into each naris and given slowly; intranasal flumazenil significantly reduced recumbency time
Dobutamine	15 μg/kg/minute IV[167]	Beta-1 adrenergic agonist used to treat anesthetic-induced hypotension
Dopamine HCl	7–10 μg/kg/minute IV[167]	Inotropic vasopressor used to treat anesthetic-induced hypotension
Fentanyl citrate	0.2 mg/kg loading dose + 10–30 μg/kg/minute IV CRI	Reduced isoflurane MAC 31%–55% in a dose-related manner in red-tailed hawks; significant effects on heart rate/blood pressure in Hispaniolan Amazon parrots
	20 μg/kg IV[92]	Hispaniolan Amazon parrots/PK; significantly decreased MAC of isoflurane but caused decreased heart rate and blood pressure
Transdermal fentanyl solution (Recuvyra, Elanco)	5 mg/kg topically q7d[194]	Guineafowl/PK
Fentanyl transdermal patch (Duragesic, Janssen Pharmaceuticals)	25 μg/hour for 72 hours[34]	Chickens/PK
Flumazenil	0.02–0.05 mg/kg IM, IV	Most species
	0.05–0.25 mg/kg intranasally incorrect citation	Canaries, Hispaniolan Amazon parrots, ring-necked parakeets/dose divided evenly between nares and given slowly, significantly reduced recumbency time
Gabapentin	—	Neuropathic pain (chronic pain; often associated with traumatic and self-induced injuries); analgesic
	10 mg/kg PO q12h	Little corella/long-term analgesia (>90 days); sole analgesic for self-mutilation; no adverse effects noted
	11 mg/kg PO q12h	Prairie falcon/long-term (>90 days) analgesic adjunct to multimodal therapy for self-mutilation
	11 mg/kg PO q8h[203]	Great horned owls/PK
	15 mg/kg PO q8h[8]	Hispaniolan Amazon parrots/PK
	25 mg/kg PO q12h[18]	Caribbean flamingos/PK
	30 mg/kg PO[7]	Hispaniolan Amazon parrots/PD; additional studies are needed
Glycopyrrolate	0.01–0.02 mg/kg IM, IV	Most species/preanesthetic
Hydromorphone HCl	0.1–0.6 mg/kg IM[98]	Cockatiels/did not increase thermal withdrawal threshold despite plasma concentrations considered therapeutic for other species
	0.1–0.6 mg/kg IM[83]	American kestrels/increased thermal withdrawal threshold for up to 6 hours; appreciable sedation in several birds at 0.6 mg/kg dose
	0.6 mg/kg IM q6h[88]	American kestrels/PK
	1 mg/kg IM, IV q6h[86]	Orange-winged Amazon parrots/PK
	1–2 mg/kg IM q3–6h[82]	Orange-winged Amazon parrots/significantly increased thermal withdrawal threshold but caused agitation, ataxia, and signs of nausea
Ketamine HCl	—	Seldom used as sole agent because of poor muscle relaxation and prolonged, violent recoveries; may produce excitation or convulsions; may fail to produce general anesthesia in some species including raptors and waterfowl
	5–50 mg/kg SC, IM, IV	Smaller species require a higher dose; large birds tend to recover more slowly
	50 mg/kg IO	Pigeons/provided effective anesthesia

TABLE 5

Chemical Restraint/Anesthetic/Analgesic Agents—cont'd

Agent	Dosage	Species/Comments
Ketamine (K)/diazepam (D)	(K) 2–5 mg/kg IV + (D) 0.25 mg/kg IV	Ostriches/ketamine may be given 15–30 minutes after diazepam
	(K) 3–8 mg/kg + (D) 0.5–1 mg/kg IM	Eagles, vultures
	(K) 8–15 mg/kg + (D) 0.5–1 mg/kg IM	Falcons
	(K) 10–40 mg/kg IV + (D) 1–1.5 mg/kg IM, IV	Raptors, pigeons, waterfowl/rapid bolus may produce apnea, arrhythmia, and increased risk of death
	(K) 20 mg/kg + (D) 1 mg/kg IV	Toucans/short procedures (15–20 minutes)
	(K) 75 mg/kg IM + (D) 2.5 mg/kg IV	White leghorn cockerels/ diazepam administered 5 minutes before ketamine for typhlectomy; some limb contracture, hypothermia, hypoxia, hypercapnia
Ketamine (K)/midazolam (Mi)	(K) 10–40 mg/kg + (Mi) 0.2–2 mg/kg SC, IM	Most species, including psittacines
	(K) 40–50 mg/kg + (Mi) 3.65 mg/kg intranasally	Ring-necked parakeets/dose divided into each nare and given slowly; onset of action less than 3 minutes, dorsal recumbency for 70.7 ± 46.7 minutes, recovery times reduced with flumazenil intranasally
Ketamine (K)/midazolam (Mi)/butorphanol (B)	(Mi) 0.2 mg/kg + (B) 0.4 mg/kg IM followed by (K) 8.7 ± 0.5 mg/kg IV	Ostriches/induction; followed by intubation and isoflurane anesthesia
Lidocaine	—	Toxic effects have been documented in birds at overall lower total doses (2.7–3.3 mg/kg) than dogs; may need to dilute 1:10 in small birds
	1–3 mg/kg	Most species
	15 mg/kg + 3.8 μg/kg lidocaine perineurally	Mallard ducks/variable effectiveness for brachial plexus nerve block
Midazolam HCl	0.1–2 mg/kg IM, IV	Most species/premedication at lower doses, onset approximately 15 minutes when administered IM
	0.15 mg/kg IV	Ostriches/rapid sternal recumbency in adults
	1 mg/kg IM[120]	Budgerigars/increased food intake for 1 hour following administration
	2 mg/kg intranasally[118]	Hispaniolan Amazon parrots/mild-to-moderate sedation in 3 minutes; reduced vocalizations, struggling, and defensive behaviors for 15 minutes; reversed with intranasal flumazenil
	3 mg/kg intranasally[41]	Cockatiels/median onset of sedation 85 seconds
	3 mg/kg + butorphanol 3 mg/kg intranasally[41]	Cockatiels/improved sedation compared to midazolam alone
	2–6 mg/kg IM	Quail/PD; mild-to-heavy sedation
	5 mg/kg IV	Turkeys, chickens, ring-necked pheasants, bobwhite quail
	7.3–8.8 mg/kg intranasally[211]	Ring-necked parakeets, canaries/dose divided into each nare and given slowly; time to onset, 3 minutes; dorsal recumbency, 57.7 ± 24.4 minutes; flumazenil administered intranasally significantly reduced recovery time
	12.5–15.6 mg/kg intranasally[210]	Canaries/dose divided into each nare and given slowly; time to onset, less than 3 minutes; dorsal recumbency, 17.1 ± 5 minutes; flumazenil intranasally significantly reduced recovery time
Nalbuphine HCl	12.5 mg/kg IM q2–3h	Opioid partial κ-agonist and partial μ-antagonist; Hispaniolan Amazon parrots/PK, PD; excellent bioavailability, little sedation; rapidly cleared after IM dosing; higher dosages (25 and 50 mg/kg IM) did not significantly increase withdrawal values
Naloxone HCl	0.01 mg/kg IV	Ostriches
	2 mg IV q4–12h	Most species, including psittacines
Naltrexone HCl	300–330 mg IM, IV	Ostriches

Continued

TABLE 5

Chemical Restraint/Anesthetic/Analgesic Agents—cont'd

Agent	Dosage	Species/Comments
Propofol	—	IV sedative-hypnotic agent; give slowly for induction to minimize apnea; intubation and IPPV required
	1–5 mg/kg IV	Most species/induction
	3 mg/kg IV (induction); 0.2 mg/kg/minute IV (maintenance)	Ostriches/PD; anesthesia
	4 mg/kg IV (induction); 0.5 mg/kg/minute IV (maintenance)	Barn owl/anesthesia
	5 mg/kg IV (induction); 1 mg/kg/minute IV (maintenance)	Hispaniolan Amazon parrots/PD; recovery times (15.4 ± 15.2 minutes) were prolonged when compared with isoflurane; 6 of 10 birds had agitated recoveries; light anesthetic plane in 8 of 10 birds
Tapentadol	—	μ-Opioid receptor agonist, norepinephrine reuptake inhibitor similar to tramadol
	30 mg/kg PO[42]	Hispaniolan Amazons/PK; only 2/7 birds had detectable plasma levels
Tolazoline HCl	1 mg/kg IV	Ostriches
	15 mg/kg IV	Raptors, including vultures
Tramadol HCl	5 mg/kg PO, IV q12h	Bald eagles/similar plasma concentrations to humans for analgesia (but analgesia not evaluated); PO bioavailability in bald eagles higher than in humans and dogs; monitor for sedation and reduce dose and/or frequency PRN
	5–30 mg/kg PO[84]	American kestrels/PD; 5 mg/kg dose resulted in longest duration of action (3 hours)
	7.5 mg/kg PO	Peafowl/2 of 6 birds reached human tramadol analgesic concentrations
	8–11 mg/kg PO q12h	Red-tailed hawks/15 mg/kg PO q12h data model suggested more frequent dosing to achieve human analgesic plasma tramadol concentrations; monitor for sedation and reduce dose and/or frequency PRN
	10 mg/kg IM[49]	White-eyed parakeets/did not change MAC of isoflurane
	10 mg/kg PO q24h[104]	African penguins/PK
	30 mg/kg PO q6–8h[179,180]	Hispaniolan Amazon parrots/similar plasma concentrations to humans for analgesia
	30 mg/kg PO q12h[5]	Muscovy ducks/PK

[a]All opioid agonists and agonist-antagonists may cause respiratory depression; profound bradypnea may occur with potent opioid agonists. *CNS,* Central nervous system; *CPR,* cardiopulmonary resuscitation; *CRI,* constant rate infusion; *$GABA_A$ receptor,* gamma-amino butyric acid A receptor; *IM,* intramuscular; *IO,* intraosseous; *IPPV,* intermittent positive pressure ventilation; *IT,* intratracheal; *IV,* intravenous; *MAC,* minimum alveolar concentration; NMDA, *N*-methyl-D-aspartate; *NSAID,* nonsteroidal antiinflammatory drug; *PD,* polydipsia; *PK,* pharmacokinetic; *PO,* oral; *PRN,* as needed; *SC,* subcutaneous.

TABLE 6

Nonsteroidal Antiinflammatory Agents[a,b]

Agent	Dosage	Species/Comments
Carprofen	2–10 mg/kg SC, IM	Most species; caution should be used when administering to *Gyps* vultures; Hispaniolan Amazon parrots may need more frequent dosing recommended than q12h
	11.5 mg/kg PO[63]	Cape vultures/mild depression and lethargy noted after single dose, as well as transient (≤48 hours) increase in blood uric acid
	30 mg/kg IM	Chickens/PD; arthritis painful behaviors reduced 1 hour posttreatment only with this high dose
Celecoxib (Celebrex, Pfizer)	10–80 mg/kg PO q24h × 6–24 weeks	Psittacines/clinical proventricular dilatation disease; clinical improvement may be seen within 14 days. Higher doses of up to 80 mg/kg have been anecdotally used for neurological cases
Diclofenac		Highly toxic to *Gyps* vultures
	2.5 mg/kg IM q24h × 5 days	Chickens/3/6 birds died; significant increases in serum uric acid, ALT, AST, creatinine; extensive visceral gout on necropsy
Flunixin meglumine	1 mg/kg PO[63]	Cape vultures/mild depression and lethargy noted after single dose, as well as transient (≤48 hours) increase in blood uric acid
	5 mg/kg IV[130]	Budgerigars, Patagonian conures/PK; half-life less than 1 hour but plasma levels above the level considered antiinflammatory and analgesic
Grapiprant	30 mg/kg PO	Red-tailed hawks/PK; plasma concentrations of approximately 100 times the canine therapeutic concentrations were achieved. Toxicity at current unknown though regurgitation was seen at doses of 45 mg/kg
Ibuprofen	5–10 mg/kg PO q8–12h	Psittacines/use pediatric suspension for small birds; avoid in *Gyps* vultures
Ketoprofen	1–5 mg/kg IM q12h	Avoid in *Gyps* vultures; raptors, waterfowl
	2.5 mg/kg IM q24h × 3 or 7 days	Budgerigars/low frequency of glomerular congestion, degeneration/dilation of tubules occurred at 3–7 days treatment
	3 mg/kg IM q24h × 5 days[124]	Broiler chickens/no adverse effects noted
	5–10 mg/kg IM, IV	Waterfowl; caution in eider ducks/high mortality may be owing to high bupivacaine dose, or cumulative toxicity of bupivacaine and ketoprofen
Mavacoxib	4 mg/kg PO	Cockatiels/PK; elimination half-life of 135 hours
	6 mg/kg PO	Flamingos/PK; plasma concentrations maintained >0.4 µg/mL for 120 hours
Meloxicam	—	No reported mortalities including *Gyps* vultures, few studies evaluating renal effects of higher doses
	0.2 mg/kg PO[96]	Brown pelicans/PK; elimination half-life 36 hours
	0.5 mg/kg PO[209]	Lesser flamingos/PK; insufficient plasma levels achieved
	0.5 mg/kg IM q24h[128]	African penguins/PK
	0.95 mg/kg PO q24h[23]	Emus/PK
	1 mg/kg PO q48h[128]	African penguins/PK
	1 mg/kg PO q12h × 5 days[178]	Chickens/PK; achieved plasma levels associated with analgesia in humans; recommended egg withdrawal time is 2 weeks after last dose
	1 mg/kg PO, IM[16]	American flamingos/PK; insufficient plasma levels with oral administration; short half-life (1.83 hours) with IM administration
	1 mg/kg PO q24h[127]	Grey parrots/PK; plasma levels maintained above those considered effective for analgesia in other avian species
	1 mg/kg PO, IM, IV q12h[125]	Hispaniolan Amazon parrots/improved weight-bearing on arthritic limb compared with lower doses; PO lower bioavailability than parenteral; concentrations similar to humans for analgesia for IM, IV for only 6 hours
	1–2 mg/kg IM q24h × 4 days[123]	Zebra finches/PK
	2 mg/kg IM q12h × 14 days	Japanese quail/unremarkable histological and minimal biochemical changes
	2 mg/kg IM, PO	Cape griffon vultures/rapid metabolism and short elimination t½ suggests low potential for drug accumulation
	3 mg/kg SC sustained-release formulation[81]	Hispaniolan Amazon parrots/PK; highly variable half-life; not currently recommended
	3 mg/kg SC sustained-release formulation[173]	American flamingos/PK; parameters no different than standard formulation
	5 mg/kg PO BID × 5 days[97]	Chickens; 3/11 birds died during treatment course; 7/11 had gross and histologic evidence of gout; not recommended
	1.5–5 mg/kg transdermally (in pluronic lecithin organogel suspension)[19]	Red-tailed hawks/PK; no plasma levels detected at 1.5 or 3 mg/kg; plasma levels after 5 mg/kg much lower than previously established therapeutic levels

Continued

TABLE 6

Nonsteroidal Antiinflammatory Agents—cont'd

Agent	Dosage	Species/Comments
Phenylbutazone	1.7 mg/kg PO[63]	Cape vultures/mild depression and lethargy noted after single dose, as well as transient (≤48 hours) increase in blood uric acid
Piroxicam	—	Has been used to treat pain associated with chronic degenerative joint disease in cranes and other species
	0.5 mg/kg q12h	Psittacines
	0.5–1 mg/kg PO[102]	Brolgas/PK; insufficient sampling duration to determine half-life
	0.5–0.8 mg/kg PO q12h[c]	Whooping cranes/used for acute myopathy, chronic degenerative joint disease
Robenacoxib (Onsior, Elanco)	2–10 mg/kg IM q5–7d[d]	Psittacines/avian ganglioneuritis and proventricular dilatation disease

[a]Unless otherwise noted, drugs provide analgesic, antipyretic, and antiinflammatory effects.
[b]Nonsteroidal antiinflammatory agents may potentially cause gastrointestinal upset and hemorrhage as well as adverse renal effects ranging from fluid retention to renal failure.
[c]J. Paul-Murphy, personal communication, 2011.
[d]B.L.S. Speer, personal communication, 2022.
ALT, Alanine aminotransferase, *t½,* half life, half time, *AST,* aspartate aminotransferase; *IM,* intramuscular; *IV,* intravenous; *PD,* pharmacodynamic; *PK,* pharmacokinetic; *PO,* oral; *SC,* subcutaneous.

TABLE 7

Hormones and Steroids

Agent	Dosage	Species/Comments
Adrenocorticotropic hormone (ACTH)	1–2 U/kg IM	Psittacines/ACTH stimulation test
Arginine vasopressin	25 µg/kg IM q12h	Grey parrot/central diabetes insipidus
Arginine vasotocin	0.5–4 µg/kg IN q12h	Grey parrot/central diabetes insipidus
Calcitonin	4 U/kg IM q12h	Most species/reduce hypercalcemia (e.g., that caused by cholecalciferol rodenticide toxicity)
Human chorionic gonadotropin	500–1000 U/kg IM on days 1, 3, 7—q3–6wk PRN	Inhibits egg laying; administer on days 3 and 7 if hen lays eggs after day 1
Deslorelin acetate	—	Synthetic long-acting GnRH agonist; used to treat reproductive tract disorders or conditions exacerbated by circulating concentrations of reproductive hormones; reported to suppress ovarian carcinoma in a cockatiel[132]
	4.7-mg long-acting implant IM[17,30]	Pigeons/effective for controlling egg laying in females for at least 49 days
	4.7-mg implant SC[182]	Cockatiels/suppressed egg laying for at least 180 days
	4.7- or 9.4-mg implant placed SC intrascapularly[30,119,141]	GnRH agonist available as long-term implant; no adverse reports with use to date
	9.4-mg implant SC[140]	Japanese quail/slower onset of action and inconsistent effects compared to two 4.7 mg implants
Desmopressin	4.6 µg/kg IM q12h	Grey parrot/treatment of central diabetes insipidus
Dexamethasone[a]	0.2–1 mg/kg IM, IV once or q12–24h × 2–7 days, then q48h × 5 days	Most species, including raptors/antiinflammatory
	2–4 mg/kg IM, IV q12–24h	Most species, including ratites/shock, trauma
Dexamethasone sodium phosphate[a]	2–4 mg/kg SC, IM, IV q6h–24h	Most species/head trauma, shock, hyperthermia; higher dose for shock, head trauma, endotoxemia
Glipizide	—	Sulfonylurea antidiabetic; contraindicated in ketotic patients; patients should be maintained at trace glycosuria to prevent hypoglycemia
	0.5 mg/kg PO q12h	Cockatiels/diabetes mellitus
	1.25 mg/kg PO q24h	Most species/diabetes mellitus
Insulin	0.002 U/bird IM q12–48h	Budgerigars/NPH insulin
	0.01–0.1 U/bird IM q12–48h	Amazon parrots/NPH insulin
	0.5–3 U/kg IM	Psittacines/NPH insulin
	1.4 U/kg IM q12–24h	Cockatiels, toco toucans/NPH insulin
	2 U/bird IM	Toco toucans/ultralente or PZI; adjust dose or frequency based on glucose curves
Leuprolide acetate	—	Administration before onset of egg laying may be more successful than treatment during breeding; 1 month depot formulation generally requires q4wk treatment
	(Number of days for desired effect) × (52 or 156 µg/kg) = dosage IM	Cockatiels
	200–1000 µg/kg IM q2–6wk	Most species
	1250 µg/kg IM once	Penguins/induced molt in 1 of 2 birds dosed
Levothyroxine (L-thyroxine)	1–200 µg/kg PO q12h × 4 weeks	Psittacines, raptors
Methylprednisolone acetate[a]	200 mg/bird IM	Ratites (adults)

Continued

TABLE 7

Hormones and Steroids—cont'd

Agent	Dosage	Species/Comments
Oxytocin	0.5–10 U/kg IM	Most species; should be preceded by calcium administration for egg binding; contraindicated unless uterovaginal sphincter is well dilated and uterus is free of adhesions; used alone to stop uterine bleeding; may need to repeat injection
Prednisolone[a]	0.5–1 mg/kg IM, IV once	Most species
	1 mg/kg PO q24h[91]	Green-wing macaw/chronic lymphocytic leukemia
	1–1.25 mg/kg PO q48h	Ratites
	2 mg/kg PO q12h	Psittacines/inflammation
	2–4 mg/kg IM, IV	Raptors/shock
Prednisolone sodium succinate[a]	0.5–1 mg/kg IM, IV	Psittacines/antiinflammatory
	1.5–2 mg/kg IM q12h	Ratites/immunosuppression
	2–4 mg/kg IM, IV once	Psittacines/shock; trauma; endotoxemia; immunosuppression
	5–8.5 mg/kg IV q1h	Ratites/shock
Prednisone[a]	—	See prednisolone
Prostaglandin E2	0.02–0.1 mg/kg applied topically to the uterovaginal sphincter	Relaxes uterovaginal sphincter; lower dosage may be effective; freeze into aliquots
Prostaglandin F2α	0.02–0.1 mg/kg IM, intracloacal once	Dystocia: may be helpful when the egg is located distally and the uterovaginal sphincter is dilated; can result in uterine rupture, bronchoconstriction, hypertension, and death
Thyroid-stimulating hormone (TSH)	0.1 U IM	Cockatiels
	0.2 U/kg IM	Macaws/PD; T_4 doubled in 6 of 11 birds 4 hours after receiving TSH
	1 U/kg IM	T_4 doubled in Hispaniolan and blue-fronted Amazon parrots 6 hours after receiving TSH
	1–2 U/kg IM	Psittacines/obtain blood at 0 hours, then 4–6 hours after TSH stimulation
Trilostane	1 mg/kg q24h	Senegal parrot/Cushing disease

[a]Steroid administration may predispose birds to aspergillosis and other mycoses; administration may also be associated with the development of polyuria/polydipsia/polyphagia, increased protein catabolism, glycosuria, and diabetes mellitus; toxic levels may be attained even with topical application; administration should ideally not exceed 5 days; rapid onset, shorter-acting drugs are generally less likely to cause serious adverse effects.

GnRH, Gonadotropin-releasing hormone; *IN,* intranasal, *IM,* intramuscular; *IV,* intravenous; *NPH,* neutral protamine Hagedorn; *PD,* Pharmacodynamic; *PO,* oral; *PRN,* as needed; *PZI,* protamine zinc insulin; *SC,* subcutaneous. T_4, thyroxine

TABLE 8

Nebulization Agents[a]

Agent	Dosage	Species/Comments
N-acetyl-L-cysteine 10%–20% (Mucomyst, Bristol)	22 mg/mL sterile water until dissipated	Most species/mucolytic agent; tracheal irritation and reflex bronchoconstriction reported in mammals; use is preceded by bronchodilators in mammals; see amikacin, gentamicin, terbinafine for combinations
Amikacin	5–6 mg/mL sterile water or saline × 15 minutes q8–12h	Most species/discontinue if polyuria develops
	6 mg/mL sterile water and 1 mL acetylcysteine (20%) until dissipated q8h	Most species
Aminophylline	3 mg/mL sterile water or saline × 15 minutes	Most species/bronchial and pulmonary vasculature smooth muscle relaxation; incompatible with amikacin, cephalothin, clindamycin, erythromycin, oxytetracycline, methylprednisone, penicillin G, tetracycline; consult with specialized references for more information
Amphotericin B (Fungizone, Squibb)	—	May lead to hypokalemia; corticosteroids may exacerbate this effect; minor systemic absorption with aerosol administration; can be nebulized long term
	0.3–1 mg/mL × 15 minutes q6–12h	Most species
	7–10 mg/mL saline	Most species
Carbenicillin (Geocillin, Roerig)	20 mg/mL saline × 15 minutes q12h	Psittacines/*Pseudomonas* pneumonia; use in combination with parenteral aminoglycosides
Cefotaxime	10 mg/mL saline × 10–30 minutes q6–12h	Most species
Ceftriaxone	40 mg/mL sterile water	Poultry/PD
	40 mg/mL sterile water and DMSO	Poultry/PD; 1 g ceftriaxone in 10 mL sterile water, plus 15 mL DMSO
	200 mg/mL sterile water and DMSO	Poultry/PD; 4 g ceftriaxone in 10 mL sterile water, plus 10 mL DMSO
Clotrimazole (1%)	10 mg/mL propylene glycol or polyethylene glycol × 30–60 minutes q24h × 3 days, off 2 days, repeat PRN for up to 4 months	Most species/treatment of aspergillosis for stable patients without respiratory distress; can be toxic to psittacines at this dose; may use in combination with systemic amphotericin B, flucytosine, and itraconazole
	10% clotrimazole in propylene glycol with 5% DMSO	Raptors
Doxycycline hyclate	13 mg/mL saline	Psittacines
Enilconazole	0.2 mg/5 mL saline q12h × 21 days	Most species, including raptors, psittacines
	10 mg/mL sterile saline or water	Most species/antifungal (e.g., aspergillosis)
Enrofloxacin[b]	10 mg/mL saline	Most species
Gentamicin	3–6 mg/mL saline or sterile water and 1–2 mL acetylcysteine (20%) × 20 minutes q8h	Most species
	5 mg/mL saline × 15 minutes q8h	Most species
Lincomycin	250 mg/mL water	Most species
Miconazole	Nebulize 15 minutes q8h × 10 days	Raptors/aspergillosis
Oxytetracycline	2 mg/mL × 60 minutes q4–6h	Parakeets/PD
Piperacillin	10 mg/mL saline × 10–30 minutes q6–12h	Most species
Sodium chloride	—	Viscosity of respiratory secretions may be decreased by hydration
Spectinomycin	13 mg/mL saline	Most species
Sterile water	—	Viscosity of respiratory secretions may be decreased by hydration
Sulfadimethoxine	13 mg/mL saline	Most species
Terbinafine	500 mg and 1 mL acetyl-L-cysteine and 500 mL distilled water[46]	Psittacines/aspergillosis
Terbutaline	0.01 mg/kg with 9 mL saline	Psittacines/bronchodilation
Tylosin	10 mg/mL saline × 10–60 minutes q12h	Most species
	20 mg/mL DMSO or distilled water × 1 hour	Pigeons, quail (PD), most species
	20 mg/mL DMSO and 0.5 mL saline	Psittacines

[a]Nebulization is an adjunctive therapy indicated for rhinitis, sinusitis, tracheitis, pneumonia, airsacculitis, and syringeal aspergilloma, where there is air movement occurring in the patient's disease state; optimal particle size for deposition in the trachea is 2 to 10 μm; optimal particle size for peripheral airways is 0.5 to 5 μm; treatments of 30 to 45 minutes repeated q4–12h are recommended; caution: do not overhydrate airways.
[b]These drugs are banned in food-producing species in the United States as well as some other countries. Use with caution and follow local laws regarding administration to food animals.
DMSO, Dimethyl sulfoxide; *PD,* pharmacodynamic; *PRN,* as needed.

TABLE 9

Agents Used in the Treatment of Toxicologic Conditions

Agent	Dosage	Species/Comments
N-acetylcysteine	—	
	800 mg/kg/day in food[186]	Broiler chickens/aflatoxicosis
		Acetaminophen toxicosis; anecdotal
Bismuth subsalicylate	2–5 mg/kg PO once	Adsorbent; gavage; alternatively, can use activated charcoal
Botulinum antitoxin	0.05–1 mL/day	Treatment of botulism
Calcitonin	4 U/kg IM q12h	Most species/reduce hypercalcemia (e.g., that caused by cholecalciferol rodenticide toxicity)
Calcium EDTA (edetate calcium disodium) (CaEDTA)	—	Preferred initial chelator for lead and zinc toxicosis; may cause renal tubular necrosis in mammals; maintain hydration and monitor patient for polyuria/polydipsia; SC, IM absorbed well
	10–50 mg/kg IM q12h × 5–10 days or longer	Raptors
	20–70 mg/kg IV	Most species/empirical diagnosis; signs should resolve for up to 48 hours; diluted 1:4 in saline
	25–50 mg/kg IV q12h	Geese
	30–35 mg/kg IM, IV q8–12h × 3–5 days, off 3–4 days, repeat PRN	Most species
	40 mg/kg IM q12h	Cockatiels/PD; reduces lead levels when used alone or with DMSA
Charcoal, activated	—	Adsorbs toxins from the intestinal tract; may be mixed with hemicellulose to act as a bulk laxative to aid in the passage of ingested toxins; administration before cathartic use may help bind small particles of heavy metal; see magnesium hydroxide for combination
	52 mg/kg PO once	A component of oiled bird treatment; alternatively, may use bismuth
	2000–8000 mg/kg PO	Most species
Charcoal, activated/electrolyte slurry (ToxiBan, Vet-A-Mix)	—	Orally administered adsorbent for GI tract toxins
	50 mL/kg by gavage	Three bottles of charcoal slurry (3.75 g/kg) added to 250 mL of electrolyte solution; use in treatment of oiled birds
Deferiprone	—	Iron chelator; used in the treatment of hemochromatosis/iron storage disease
	50 mg/kg PO, IV once[197]	White leghorn chickens/PK; half-life of 3.2 hours following oral dose
	50 mg/kg PO, IV once[198]	King pigeons/PK; half-life of 2.98 hours following oral dose
	50–75 mg/kg PO q12h[199]	White leghorn chickens, king pigeons/no adverse effects noted at either dose
	75 mg/kg PO q24h[164]	Hornbills
	75 mg/kg PO q24 × 30 days[101]	Common mynahs; additional 30 days of treatment did not result in significant changes in liver iron content
	100 mg/kg PO q24h	
Deferoxamine	—	Iron chelator; used in the treatment of hemochromatosis/iron storage disease
	50 mg/kg IM q12h[66]	Chestnut-fronted macaw
	100 mg/kg SC q24h × 30 days[101]	Common mynahs; additional 30 days of treatment did not result in significant changes in liver iron content
	100 mg/kg IM q24h	

TABLE 9

Agents Used in the Treatment of Toxicologic Conditions—cont'd

Agent	Dosage	Species/Comments
Detergent (Dawn, Procter & Gamble)	1%–5% bath	Submerse bird up to mid-neck region; rinse with water; use water at 103–105°F (39–41°C) and 40–60 psi; use in treatment of oiled birds
Dimercaptosuccinic acid (DMSA)	—	Oral chelator for lead or zinc; may be effective for mercury toxicosis
	25–35 mg/kg PO q12h × 5 days/week × 3–5 weeks	Most species, including raptors/lead toxicosis
	25–35 mg/kg PO q24h × 10 days	Psittacines, raptors/lead and zinc toxicosis
	40 mg/kg PO q12h × 21 days	Cockatiels/PD; lead toxicosis; reduces lead levels when used alone or in combination with CaEDTA; 80 mg/kg resulted in death in >60% of cockatiels
Fluid therapy	—	Dehydration, shock, nutritional support, etc.
Intravenous lipid emulsion therapy	15 mL/kg/hour[114]	Domestic goose with oleander toxicosis; clinical signs resolved after 2 hours
Magnesium hydroxide (M) (Milk of Magnesia, Roxane)/activated charcoal (C)	(M) 10–12 mL + (C) 1 tsp powder	Most species/cathartic[a]; adsorbent
Oral electrolyte solutions (Pedialyte; Ross Labs)	30 mL/kg by gavage	Most species/at field stabilization site
Penicillamine	—	Preferred chelator for copper toxicosis; may be used for lead, zinc, and mercury toxicosis
	30 mg/kg PO q12h × ≥7 days	Most species/initially supplemented with CaEDTA once in severe neurological disease
	30–55 mg/kg PO q12h × 7–14 days	Most species, including raptors, waterfowl
	50–55 mg/kg PO q24h × 1–6 weeks	Most species, including psittacines, raptors/use in combination with CaEDTA for several days followed by penicillamine × 3–6 weeks
Pralidoxime	10–100 mg/kg q24–48h, or repeat once q6h	Binds to organophosphate-inactivated acetylcholinesterase; used in treatment of organophosphate or carbamate poisoning
Tea/tannins	8 g/kg of food	Reduced absorption of iron when fed to starlings; used in prevention of iron storage disease
Vitamin K_1	0.2–2.2 mg/kg IM q4–8h until stable, then q24h PO, IM × 14–28 days	Most species, including raptors/rodenticide anticoagulant toxicosis

[a]Cathartics increase gastrointestinal motility and are used to evacuate the gut and prevent absorption of toxins.
CaEDTA, edetate calcium disodium; *DMSA,* Dimercaptosuccinic acid; *EDTA,* ethylenediaminetetraacetic acid; *GI,* gastrointestinal; *IM,* intramuscular; *IV,* intravenous; *PD,* pharmacodynamic; *PK,* pharmacokinetic; *PO,* oral; *PRN,* as needed; *SC,* subcutaneous.

TABLE 10

Psychotropic Agents[a]

Agent	Dosage	Species/Comments
Amitriptyline	—	Tricyclic antidepressant; antihistaminergic action; used in the treatment of pruritic conditions (including feather-damaging behavior), anxiety disorders, and/or depression
	1–5 mg/kg PO q12–24h[191]	Most species/feather-damaging behavior, obsessive-compulsive disorders, phobias; doses up to 9 mg/kg may be required but may also cause adverse effects so titration to individual patient effect is recommended
	2 mg/kg PO q24h	Psittacines/minimum of 30 days
Buspirone	0.5 mg/kg PO q12h	Anxiolytic drug, used in the treatment of anxiety disorders
Carbamazepine	3–10 mg/kg PO q24h	Anticonvulsant; treatment of seizure disorders; may also be beneficial to treat feather-damaging behavior and anxiety- or frustration-related aggression
	166 mg/L drinking water	Most species/anticonvulsant; analgesic; may cause bone marrow suppression; combination with chlorpromazine or haloperidol recommended for the initial 2 weeks of the treatment
Chlorpromazine	—	Phenothiazine derivative; antipsychotic drug; dopamine antagonist (less specific than haloperidol); main effect is sedation, but may be useful for feather-damaging behavior as well
	0.1–0.2 mg/kg once (tranquilizer)	Cockatoos, ring-necked parakeets/use with carbamazepine after the removal of Elizabethan collar; mild sedation and decreases obsessive behaviors
	1 mL stock solution[b]/120 mL drinking water	Most species/mild sedation
	0.2–1 mL stock solution/kg q12–24h	Most species/mild sedation
Clomipramine	—	Tricyclic antidepressant; antihistamine; used, for example, in the treatment of impulsive and obsessive-compulsive disorders (ICD/OCD), depression and/or anxiety disorders
	0.5–1 mg/kg PO q12–24h	Psittacines/feather-damaging behavior; automutilation; start with low dose and gradually increase over 4–5 days
	3–5 mg/kg PO q12–24h	Psittacines/compulsive feather-damaging behavior or impulse control disorder; anxiety
	4–9.5 mg/kg PO q12h	Grey parrots/behavior interpreted as paradoxical anxiety; combine with anxiolytic therapy (buspirone)
Diazepam	—	Benzodiazepine sedative; anxiolytic/stress-associated feather picking; useful as sole agent or in combination with phenobarbital for seizure control
	0.25–0.5 mg/kg IM, IV q24h × 2–3 days	Raptors/appetite stimulant
	0.5 mg/kg PO	Passerines/calm fractious species
	0.5–0.6 mg/kg IM	Most species/facilitates acceptance of Elizabethan collar
	0.5–1(–1.5) mg/kg PO, IM, IV q8–12h	Most species/control of seizures
	2.5–4 mg/kg PO q6–8h	Psittacines/sedation
	10–20 mg/L drinking water	Most species
Diphenhydramine	—	Antihistamine; mild hypnotic effects; suspected allergic feather picking
	2–4 mg/kg PO q12h	Most species
	2 mg/L drinking water	Most species
Doxepin	—	Tricyclic antidepressant; antihistaminergic action; used in the treatment of pruritic conditions (including feather-damaging behavior), anxiety disorders, and/or depression
	0.5–1 mg/kg PO q12h	Most species/feather-damaging behavior
	1–2 mg/kg PO q12h	Psittacines/anxiety; pruritus
	1–5 mg/kg PO q24h	Psittacines/feather-damaging behavior
Fluoxetine	—	Selective serotonin reuptake inhibitor; antidepressant; adjunctive treatment for depression-induced feather picking
	0.4–4 mg/kg PO q24h	Psittacines/compulsive feather picking
	2–3 mg/kg PO q12–24h	Most species, including psittacines
	1 mg/kg PO q24h	Psittacines
	1–5 mg/kg PO q24h	Psittacines
Gabapentin	15–20 mg/kg q8–12h	Psittacines; modulating seizure activity
Haloperidol	—	Butyrophenone derivative; dopamine antagonist, antipsychotic drug
	0.1–0.2 mg/kg PO q12h	Psittacines/automutilation; feather-damaging behavior
	0.2–0.9 mg/kg PO q24h	Most species/stereotypic preening behavior
	1–2 mg/kg IM q14–21d	Most species, including psittacines

TABLE 10

Psychotropic Agents—cont'd

Agent	Dosage	Species/Comments
Hydroxyzine	—	Antihistamine; treatment of allergic responses, pruritus and pruritus-associated behavioral disorders
	2 mg/kg PO q8–12h	Most species/pruritus
	30–40 mg/L drinking water	Most species
Levetiracetam	50 mg/kg PO q8h or 100 mg/kg PO q12h[166]	Hispaniolan Amazon parrots/antiepileptic; potential use in long-term anticonvulsive treatment; drug levels should be monitored
Lorazepam	—	Benzodiazepine, tranquilizer/sedative; used in macaws with feather picking or aggression (may be used concurrently with haloperidol)
	0.1 mg/kg PO q12h	Macaws/aggression; feather-damaging behavior; use alone or with haloperidol
Naloxone	—	Opioid receptor antagonist, used in the acute treatment of stereotypies, compulsive, and self-injurious behaviors or addictions
	2 mg/kg IV	Psittacines/may be used to determine the response of stereotypic behavior to antagonist therapy; reduction of behavior should be observed within 20 minutes
Naltrexone	—	Opioid receptor antagonist, used in the acute treatment of stereotypies, compulsive and self-injurious behaviors or addictions
	1.5 mg/kg PO q8–12h	Most species/feather-damaging behavior; automutilation; contraindicated in patients with liver disease; may need to increase dosage 2× to 6× to be effective; dissolve tablet in 10 mL sterile water; preservative does not go into solution
Nortriptyline	—	Tricyclic antidepressant; may be beneficial for treating feather-damaging behavior, anxiety and/or depression. Hyperactivity commonly noted as side effect
	16 mg/L drinking water	Most species/feather-damaging behavior; seldom used; decrease dose or discontinue if hyperactivity develops; taper dose to discontinue
Paroxetine	—	Selective serotonin reuptake inhibitor, antidepressant, used in the treatment of depression, posttraumatic stress and panic disorders and ICD/OCD
	1–2 mg/kg PO q12–24h	Macaws, ibis/feather-damaging behavior; automutilation; generally, requires long-term therapy
	3 mg/kg PO q12h	Pigeons/reduce compulsive behavior
	4 mg/kg PO q12h[188]	Grey parrots/PK
Phenobarbital sodium	—	Barbiturate anticonvulsant; mild sedative effect; long-term seizure management; adjust dosage based on blood levels; may cause deep sedation and inability to perch
	1–5 mg/kg IV bolus	Most species/status epilepticus; begin at low end of dosage range and increase for refractory seizures
	2–7 mg/kg PO q8–12h	Most species/feather picking; seizures; self-mutilation
	17 mg/kg PO once[143]	Grey parrots/PK; half-life less than 2 hours
	50–80 mg/L drinking water	Most species, including Amazon parrots/idiopathic epilepsy
Potassium bromide	—	Long-term seizure management; use as sole agent or in conjunction with phenobarbital; monitor blood levels that may take up to 90 days to establish steady state; not available in approved dosage forms in North America; may be obtained from chemical companies or compounding pharmacies; for a concentration of 250 mg/mL, add distilled water as needed to 25 g of potassium bromide for a final volume of 100 mL
	25 mg/kg PO q24h	Most species
	50–80 mg/kg PO q24h	Pigeons
	75 mg/kg PO	Psittacines
Trazodone	30 mg/kg PO q24h[36]	Pigeons/PK; serotonin reuptake inhibitor, single dose resulted in plasma concentrations above therapeutic levels in humans for 20 hours
Zonisamide	20 mg/kg PO q8–12h	Psittacines/antiepileptic

[a]The use of psychotropic agents in birds is controversial because safety, efficacy, and pharmacologic effects are poorly documented; anxiolytics or tricyclic antidepressants may be useful for stereotypic behaviors or mutilation; selective serotonin reuptake inhibitors may prove helpful for explosive behaviors; consider metabolic scaling when calculating dosages; these treatments should be used as components of a structured behavior change strategy. Note: most of the dosages mentioned in this table have been derived from case reports and/or anecdotal evidence. As information on pharmacokinetics, pharmacodynamics, efficacy, and toxicity is currently lacking for most of these drugs, no specific recommendations can be made at this stage.

[b]Stock solution: 125 mg chlorpromazine in 31 mL simple syrup.

IM, Intramuscular; *IV,* intravenous; *PK,* pharmacokinetic; *PO,* oral.

TABLE 11
Nutritional/Mineral Support

Agent	Dosage	Species/Comments
Calcium	—	Recommended dietary levels[a]
	3–10 mg/kg feed (0.3%–1%)	Laying parrots
	4–8 mg/kg feed (0.4%–0.8%)	Growing Muscovy ducks
	8 mg/kg feed (0.8%)	Growing Japanese quail
	8–10 mg/kg feed (0.8%–1%)	Growing chickens
	18.8–32.5 mg/kg feed (1.88%–3.25%)	Laying chickens/3.25% recommended for hens that lay eggs daily
	22.5 mg/kg feed (2.25%)	Laying turkeys
Calcium borogluconate (10%)	50–100 mg/kg IM, IV	Psittacines/20% solution
	100–500 mg/kg SC, IV (slow) once	Raptors/hypocalcemia
Calcium glubionate	25–150 mg/kg PO q12h	Most species/hypocalcemia, calcium supplementation
Calcium gluconate (10%)	—	Hypocalcemia; dilute 1:1 with saline or sterile water for IM or IV injections
	5–10 mg/kg SC, IM, IV (slow) q12h PRN	Psittacines/hypocalcemic tetany
	10–100 mg/kg IM	Psittacines/acute presentation of hypocalcemia
	25–50 mg/kg SC, IV (slow)	Pigeons
	50–100 mg/kg IM (diluted), IV (slow)	Most species, including psittacines, pigeons, raptors
	1 mL/30 mL (3300 mg/L) drinking water	Psittacines/calcium supplementation
Calcium lactate/calcium glycerophosphate	5–10 mg/kg IM q7d PRN	Most species, including raptors/hypocalcemia
	50–100 mg/kg IV (slow bolus) once	Grey parrots
Dextrose (50%)	50–100 mg/kg IV (slow bolus) to effect	Psittacines/hypoglycemia; can dilute with fluids
	500–1000 mg/kg IV (slow bolus)	Hypoglycemia; can dilute with fluids
Essential fatty acids (omega-3, omega-6)	0.2–0.5 mL/kg PO q24h	Most species; antiinflammatory, immunomodulation
Hemicellulose (Metamucil, Searle)	Small amount on food daily	Most species/for bulk in diet; facilitates defecation in bowel deficit disorders and other conditions
	0.5 tsp/60 mL hand-feeding formula or baby food gruel	Psittacines/bulk diet to delay absorption of an ingested toxin
Iodine (Lugol's iodine)	0.2 mL/L drinking water daily	Most species/thyroid hyperplasia
	2 parts iodine + 28 parts water; 3 drops into 100 mL drinking water	Budgerigars/thyroid hyperplasia
Iron	20–40 mg/kg feed	Toucans/levels recommended for a low-iron diet
Iron dextran	10 mg/kg IM, repeat in 7–10 days PRN	Most species/iron deficiency anemia; use cautiously in species in which iron storage disease is common
Lactobacillus (Bene-Bac, Pet-Ag)	1 pinch/day/bird	Psittacines/stimulation of normal gastrointestinal flora regrowth
	1 tsp/L hand-feeding formula	Most species
Magnesium sulfate	20 mg/kg IM	Grey parrot/dietary hypomagnesemia and seizures
Pancreatic enzyme powder (Viokase-V Powder, Fort Dodge)	—	Most species/exocrine pancreatic insufficiency; maldigestion; mix with food and let stand 30 minutes
	2–5 g/kg	Most species
	$^1/_8$ tsp/kg feed	Most species
	$^1/_8$ tsp/60–120 g lightly oil-coated seed	Most species
	$^1/_8$ tsp/30–120 mL hand-feeding formula PRN	Psittacine neonates
Potassium chloride	0.5 mEq/kg PO q12h	Most species/hypokalemia
Selenium (Se)	0.05–0.1 mg Se/kg IM q3–14d	Most species/neuromuscular diseases (capture myopathy, white muscle disease, some cardiomyopathies); may be useful in some cockatiels with jaw, eyelid, and tongue paralysis
Vitamin A	200 U/kg IM	Raptor juveniles/supplemental therapy for pox infection
	2000 U/kg PO, IM	Psittacines/adjunctive therapy for pox infection
	5000 U/kg IM q24h × 14 days, then 250–1000 IU/kg q24h PO	Psittacines/adjunctive therapy for respiratory or epithelial disease
	20,000–33,000 U/kg IM q7d	Most species/hypovitaminosis A; maximum dose; improves skin healing

TABLE 11

Nutritional/Mineral Support—cont'd

Agent	Dosage	Species/Comments
Vitamin B_1 (thiamine)	—	Thiamine deficiency: requirements may be higher if thiaminase is present in diet[b]
	1–2 mg/kg PO q24h	Raptors, penguins, cranes/daily supplement
	1–2 mg/kg IM q24h	Vultures, raptors, cranes, penguins/CNS signs
	1–3 mg/kg IM q7d	Most species, including raptors
	1–50 mg/kg PO q24h × 7 days or indefinitely	Raptors
	3–30 mg/kg IM q7d	Raptors/stimulates appetite, hematopoiesis; neuromuscular disease; liver disease; supportive therapy; adjunct to sulfa therapy
	25–30 mg/kg feed or fish	Piscivorous species/recommended level of supplementation
	2850 mg/L drinking water q7d	Pigeons
Vitamin B_6 (pyridoxine)	5 mg/kg IM or 25 mg/kg PO once[161]	Maximum nonlethal dose in falcons
Vitamin B_{12} (cyanocobalamin)	0.25–0.5 mg/kg IM q7d	Most species/anemia
	2–5 mg/bird SC	Pigeons/vitamin B_{12} deficiency
Vitamin B complex	—	Usually dosed based on thiamine (see vitamin B_1)
Vitamin D_3	3300 U/kg IM q7d PRN	Most species/hypovitaminosis D_3; hypervitaminosis D may occur with excessive use
	6600 U/kg IM once	Most species
	11–30 minutes of direct sunlight/day	Chickens/sufficient for endogenous synthesis of vitamin D
Vitamin E	0.06 mg/kg IM q7d	Psittacines/hypovitaminosis E
	0.06 mg/kg IM	Ratites/prevention or treatment of capture myopathy
	15 mg/kg PO once	Raptors/PD; administer without food
	200–400 mg/bird PO q24h	Great blue herons
	100 mg/kg fish (wet basis)	Piscivorous species/recommended level of supplementation
Vitamin K_1 (phytonadione)	0.025–2.5 mg/kg IM q12h	Most species
	0.2–2.2 mg/kg IM q4–8h until stable, then q24h × 14 days	Most species/rodenticide toxicity
	2.5 mg/kg IM q24h until hemostasis, then q7d PRN	Psittacines/vitamin K responsive disorders; hematochezia; coagulopathy
	10–20 mg/kg IM q12–24h	Psittacines
	0.1 mg/kg feed	Turkeys/PD; as effective as 1–2 mg/kg in reducing plasma prothrombin time

[a]Grains and seeds commonly fed to parrots contain calcium levels of approximately 0.02% to 0.1% dry matter (DM).
[b]Food items known to contain appreciable amounts of thiaminase include clams, herring, smelt, and mackerel.
CNS, Central nervous system; *IM,* intramuscular; *IV,* intravenous; *PD,* pharmacodynamic; *PO,* oral; *PRN,* as needed; *SC,* subcutaneous.

TABLE 12

Ophthalmologic Agents

Agent	Dosage	Species/Comments
Bacitracin/neomycin/ polymyxin B sulfate	Small bead topical	Most species/antibiotic; corneal ulcers, conjunctivitis
Chloramphenicol ophthalmic drops[a]	1 drop topical q6–8h	Most species, antibiotic
Ciprofloxacin HCl[a]	1 drop topical q4–8h	Most species/antibiotic; corneal ulcers, conjunctivitis (e.g., *Chlamydophila, Mycoplasma*)
	One drop topical q12h; use in conjunction with tylosin 1 mg/mL drinking water × 21–77 days	House finches/*Mycoplasma gallisepticum* conjunctivitis
Dexamethasone (0.1%) ophthalmic drops	1 drop topical q4–8h	Most species/traumatic, noninfectious uveitis; may suppress adrenocortical function so antifungal and antibiotic prophylaxis may need to be considered
Diclofenac sodium 0.1% ophthalmic solution	1 drop OU q12h × 7 days[76]	Chickens/PK; diclofenac detectable in plasma with no biochemistry changes noted
Flurbiprofen ophthalmic solution	Topical as indicated	NSAID used in the eye to treat or prevent inflammation (i.e., uveitis, conjunctivitis)
Gentamycin sulfate	1 drop topical q4–8h	Most species/antibiotic; corneal ulcers; may cause irritation
Miconazole	1 drop topical q12h	Parrots/fungal keratitis
Neomycin/polymyxin B/ gramicidin	1 drop topical q2–8h	Most species/antibiotic; corneal ulcers; conjunctivitis
Oxytetracycline/ polymyxin B	Small bead topical	Most species/antibiotic; conjunctivitis
Prednisolone acetate (1%)	1 drop q4–8h	Raptors/traumatic anterior uveitis without corneal ulceration
Proparacaine (0.5%)	Topical	Topical anesthesia
Rocuronium bromide	—	Neuromuscular blocking agent
	0.12 mg topical in each eye[11]	European kestrels/mydriatic agent; maximal mydriasis was achieved in 90 minutes
	20 µL/eye[6,139]	Hispaniolan Amazons/first effects noted at 8 minutes; lasted 6 hours
Tetracaine (6%)	Topical	Topical anesthetic
Vecuronium	—	Neuromuscular blocking agent; mydriatic
	0.08 mg topically per eye[122,147]	European kestrels, psittacines
	0.16 mg topically per eye[113]	Double-crested cormorants

[a]These drugs are banned in food-producing species in the United States as well as some other countries. Use with caution and follow local laws regarding administration to food animals.
NSAID, Nonsteroidal antiinflammatory drug; *PK,* pharmacokinetic.

TABLE 13

Agents Used in Emergencies

Agent	Dosage	Species/Comments
Aminophylline	4 mg/kg PO q6–12h	Can give orally after initial response
	10 mg/kg IV q3h	Use for pulmonary edema
Atropine sulfate	0.2–0.5 mg/kg IM, IV, IO	Bradycardia; CPR
Calcium gluconate	50–100 mg/kg IM, IV (slow bolus)	Hypocalcemia; dilute to 50 mg/mL; hyperkalemia
Dexamethasone Na phosphate	2–6 mg/kg IM, IV q12–24h	Head trauma (until signs abate); shock (1 dose); hyperthermia (until stable); use in these conditions is controversial
Dextrose (50%)	50–100 mg/kg IV (slow bolus to effect)	Hypoglycemia; can dilute with fluids
	500–1000 mg/kg IV (slow bolus)	Hypoglycemia; can dilute with fluids
Dextran 70	10–20 mL/kg	Most species/hypovolemic shock; colloid with a t½ shorter than hetastarch
Diazepam	0.5–1 mg/kg IM, IV PRN	Seizures
Doxapram	5–20 mg/kg IM, IV, IO	Most species/respiratory depression or arrest; CPR
Epinephrine (1:1000)	0.5–1 mL/kg IM, IV, IO, intratracheal	CPR; bradycardia
Fluids	10–25 mL/kg IV, IO	Bolus over 5–7 minutes
	50–90 mL/kg fluids SC,[a] IV, IO	Most species
Hetastarch	10–15 mL/kg IV (slow) q8h × 1–4 treatments	Most species, including raptors/hypoproteinemia; hypovolemia
Mannitol	0.2–2 mg/kg IV (slow) q24h	Raptors/cerebral edema; anuric renal failure; contraindicated in intracranial bleeding
Magnesium sulfate	20 mg/kg IM once[105]	Grey parrot/treatment for seizure activity associated with hypomagnesemia (associated with hypocalcemia that is not responsive to calcium therapy)
Prednisolone Na succinate	10–20 mg/kg IM, IV q15min PRN	Historically it has been used for head trauma and CPR; use in these conditions is controversial; no longer appears to be a treatment for traumatic brain injury and may actually exacerbate mortality
	15–30 mg/kg IV	Raptors/see the previous comment
Sodium bicarbonate	1 mEq/kg q15–30min to maximum of 4 mEq/kg total dose	Metabolic acidosis
	5 mEq/kg IV, IO once	CPR
Terbutaline	0.01 mg/kg PO, IM q6h	Psittacines/α2-selective smooth muscle bronchodilator
	0.1 mg/kg PO q12–24h	Macaws, Amazon parrots/bronchodilator; obstructive pulmonary disease, pneumonitis

[a]Because of the presence of peripheral vasoconstriction, subcutaneous administration is not adequate for patients in shock.
CPR, Cardiopulmonary resuscitation; *IM,* intramuscular; *IO,* intraosseous; *IV,* intravenous; *PO,* oral; *PRN,* as needed; *SC,* subcutaneous.

TABLE 14
Cardiovascular/Respiratory Agents

Agent	Dosage	Species/Comments
Acetylsalicylic acid (aspirin)	5 mg/kg PO q48h	Most species/antithrombotic agent
Aminophylline	4–5 mg/kg PO, IM, IV q6–12h	Most species/bronchodilator; need to prepare as a suspension daily
	8–10 mg/kg PO, IM, IV q6–8h	Raptors, ratites
	10 mg/kg IV q3h, then PO after initial response	Most species
	10 mg/kg IM, IV q8–12h	Most species/for IV use, dilute in 10–20 mL saline or 5% dextrose in water and inject slowly
Atenolol	5–10 mg/kg PO q12–24h	Most species/negative inotrope, lowering blood pressure
Atorvastatin	10–20 mg/kg PO q12h	Parrots/hypercholesterolemia; use is controversial
Benazepril	0.5 mg/kg PO q24h[61,169]	Parrots/angiotensin-converting enzyme (ACE) inhibitor, vasodilation
Bromhexine	1.5–6 mg/kg IM q12–24h	Most species/expectorant
Carvedilol	1–9 mg/kg PO q12–24h[61]	Parrots/negative inotrope
Digoxin	—	Congestive heart disease: toxic reactions include depression, ataxia, vomiting, diarrhea; contraindicated with renal or liver disease; monitoring of serum digoxin, potassium, magnesium, calcium, and ECG is recommended; induced arrhythmias in pigeons at 0.2 mg/kg/day
	0.0035 mg/kg IV q24h	Turkeys
	0.0049 mg/kg IV q12h	Poultry
	0.01 mg/kg PO q24h × 6 weeks	Chickens/ascites syndrome; reduced ascites; no apparent toxicity
	0.01–0.02 mg/kg PO q12h	Psittacines, passerines, raptors/congestive heart disease
	0.02 mg/kg PO q24h × 5 days	Parakeets, sparrows (PD)/this dose led to signs of toxicity in a mynah
	0.05 mg/kg PO q24h	Quaker parakeets/PD; congestive heart failure; cardiomyopathy
	0.13 mg/L drinking water	Psittacines, passerines, raptors/congestive heart disease
Diltiazem	1–2 mg/kg PO q8–12h[37]	Most species/calcium channel blocker, used to lower blood pressure
	15 mg/kg PO q12h[202]	Chickens
Enalapril	0.25–0.5 mg/kg PO q24–48h	Most species/dilated cardiomyopathy; monitor uric acid levels; reduce dose or discontinue if concurrent renal disease
	1.25 mg/kg PO q8–12h	Pigeons, Amazon parrots/PK
	2.5–5 mg/kg PO q12h	Amazon parrot/right-sided heart failure; long-term therapy
Furosemide	—	Diuretic; overdose can cause dehydration and electrolyte abnormalities; toxicity characterized by neurologic signs and death
	0.1–2 mg/kg PO, SC, IM, IV q6–24h	Most species, including psittacines, raptors; lories are extremely sensitive
	0.15 mg/kg IM q8h	Mynahs/ascites, hemochromatosis
	0.5–2.2 mg/kg PO, IM q12–24h	Most species/cardiac disease, ascites
	2–5 mg/kg PO, IM q12–24h	Raptors/cardiac disease, pulmonary congestion
	2.5–10 mg/kg PO q12h × 7–14 days	Cockatiels, budgerigars/ascites
	40 mg/L drinking water	Most species/congestive heart failure; can be used with digoxin and ACE inhibitors
Isoxsuprine	10 mg/kg PO q12–24h[61,174]	Psittacines/vasodilator
Pentoxifylline	15–30 mg/kg q8–12h PO	Most species/peripheral arterial disease, frostbite, wing tip edema
Pimobendan (Vetmedin, Boehringer-Ingelheim)	—	Phosphodiesterase inhibitor and calcium sensitizer used in treatment of congestive heart failure
	10 mg/kg PO[79]	Hispaniolan Amazon parrots/use tablet suspension; safety and efficacy are unknown; potential use in other birds is unknown
Propranolol	0.04 mg/kg IV (slow) 0.2 mg/kg IM	Most species/supraventricular arrhythmia, atrial flutter, fibrillation
Rosuvastatin	10–25 mg/kg PO[12]	Psittacines/hypercholesterolemia; doses did not result detectable plasma concentrations
Salbutamol	0.1–0.2 mg/kg q8–12h PO, IM	Most species/bronchodilator; anecdotal information
Sildenafil	1–5 mg/kg PO q8–12h[17,61]	Parrots/phosphodiesterase inhibitor, vasodilator, used to treat pulmonary hypertension
	1 mg/kg PO q12h[31]	African penguin/used in treatment of right-sided heart failure
Spironolactone	1–2 mg/kg PO q12h[51,61,169]	Psittacines, chickens/diuretic
Terbutaline	0.01 mg/kg PO, IM q6h	Psittacines/α2-selective smooth muscle bronchodilator
	0.1 mg/kg PO q12–24h	Macaws, Amazon parrots/bronchodilator; obstructive pulmonary disease; pneumonitis
Theophylline	2 mg/kg PO q12h	Severe macaws/bronchodilation

ACE, Angiotensin-converting enzyme; *ECG,* electrocardiogram; *IM,* intramuscular; *IV,* intravenous; *PD,* pharmacodynamic; *PK,* pharmacokinetic; *PO,* oral; *SC,* subcutaneous.

TABLE 15

Dermatologic Agents

Agent	Dosage	Species/Comments
Armor All Protectant (Armor All-Protectant Corp.)	Topical to affected plumage	Most species/softens sticky trap glue-covered plumage; use detergent (Dawn) to remove Armor All
Bismuth subsalicylate (Pepto Bismol, Procter & Gamble)	1–2 mL/kg PO q12h 2–5 mL/kg PO once	Most species/weak adsorbent, demulcent Most species/a component of oiled bird treatment; alternatively can use activated charcoal
Chlorhexidine (0.05%–0.1% solution)	Topical	Presurgical skin cleaner; antiseptic
Povidone iodine	Topical, wash off within 5 minutes	Raptors/wound cleansing; antiseptic; but is infrequently used in avian dermatology owing to its drying, irritating, and staining effects
Skin-So-Soft (Avon)	Topical to affected plumage	Most species/softens and removes sticky trap glue from plumage; use Dawn dish detergent to remove Skin-So-Soft product[a]
Tincture of iodine	Topical	Most species, raptors/wounds
Trypsin-balsam of Peru-castor oil (Granulex, Pfizer)	Topical	Most species/digests necrotic tissue (may have debriding action); may have analgesic effects; may cause local inflammation and pyogenic reaction; do not use for long-term management[a]
Yeast cell derivatives (Preparation H, WhiteHall)	Topical q24h	Most species/pododermatitis; stimulation of epithelialization

[a]Many topical agents contain oils that adhere to plumage. These agents should be used sparingly and generally in nonfeathered regions to prevent losing the insulative properties of the plumage.
PO, Oral.

TABLE 16

Agents Used in the Treatment of Renal Disease

Agent	Dosage	Species/Comments
Allopurinol	10–30 mg/kg PO q12–24h	Most species/xanthine oxidase inhibitor used in the treatment of gout. Use is controversial
	25 mg/kg PO q24h[171]	Chickens/PD; reduced uric acid levels
	25 mg/kg PO q24h[115,142]	Red-tailed hawks/PD; no significant effect on plasma uric acid levels, while toxicity with secondary renal dysfunction was observed at 50 mg/kg
Colchicine	0.04–0.2 mg/kg PO q12–24h[43]	Most species/xanthine dehydrogenase inhibitor; may be used to treat amyloidosis and limit renal or hepatic fibrosis, but no controlled studies available
	0.18 mg/kg PO q24h[176]	Turkey/dose failed to influence uric acid levels after 7 days
Rasburicase	0.5 mg/kg IM[126]	Grey parrot/recombinant urate oxidase; uric acid levels significantly decreased 16–24 hours following injection
Urate oxidase/uricase (Uricozyme, Sanofi Winthrop)	100–200 U/kg IM q24h[142,172]	Red-tailed hawks, pigeons/PD; significantly lowered plasma uric acid, including postprandial plasma uric acid

IM, Intramuscular; *PD,* pharmacodynamic; *PO,* oral.

TABLE 17

Gastrointestinal and Hepatic Agents

Agent	Dosage	Species/Comments
Aluminum hydroxide	30–90 mg/kg PO q12h	Most species/antacid; phosphate binder
Cimetidine	—	Prototype histamine-2 blocker used to reduce gastrointestinal (GI) acid production
	3–10 mg/kg PO, IM, IV q8–12h	Ratites
	5 mg/kg PO, IM q8–12h	Psittacines/proventriculitis; gastric ulceration
Cisapride	0.5–1.5 mg/kg PO q8–12h	Most species/GI prokinetic agent, stimulates motility; not commercially available in the United States; can be compounded
Famotidine	1 mg/kg IM q12h[29]	Parrots/antacid; anecdotal information
Kaolin/pectin	2 mL/kg PO q6–12h	Psittacine neonates/intestinal protectant, antidiarrheal
	≤15 mL/kg PO, repeat PRN	Raptors
Lactulose	—	Reduces blood ammonia levels; does not treat liver disease
	150–650 mg/kg (0.2–1 mL/kg) PO q8–12h	Most species, including psittacines/hepatic encephalopathy
	200 mg/kg (0.3 mL/kg) PO q8–12h	Psittacine neonates
Magnesium hydroxide (M)/activated charcoal (C)	(M) 10–12 mL + (C) 1 tsp powder	Most species/cathartic; adsorbent
Magnesium sulfate (Epsom salts)	0.25–1 g/kg PO q24h × 1–2 days	Most species/purgative, cathartic; may cause lethargy; see peanut butter for combination
Mannitol	0.25–2 mg/kg q24h IV (slow bolus)	Most species/osmotic diuretic used to treat cerebral edema, especially after head trauma; may be used with furosemide
Maropitant	1 mg/kg SC, IM, PO q12–24h	Parrots/antiemetic, used to treat visceral pain, motion sickness
Metoclopramide	—	GI motility disorders; regurgitation; slow crop motility; no alterations in motility observed after a single dose of 1 mg/kg IM
	0.3 mg/kg PO, IM, IV	Most species
	0.5 mg/kg PO, IM, IV q8–12h	Most species, including psittacines/GI ileus; regurgitation
	2 mg/kg IM, IV q8–12h	Raptors, waterfowl/crop stasis; ileus
	12.5 mg/kg PO	Ratites/GI disorders
Mineral oil	5–10 mL/kg PO via gavage	Most species/cathartic; used to aid passage of grit and other foreign bodies; administer directly into the crop because oral administration may result in aspiration pneumonia; see peanut butter for combination
Mirtazapine	1 mg/kg PO q12–24h	Most species/appetite stimulant
Omeprazole	1–10 mg/kg PO q12h[27]	Most species/anecdotal information
	20–50 mg/kg PO q24h[94]	Chickens/omeprazole reduced gastroenteral toxicity at 20 mg/kg/day and almost completely prevented problems at 50 mg/kg/day
Peanut butter	Peanut butter and mineral oil (2:1)	Most species/add to diet; cathartic
	Dilute peanut butter and magnesium sulfate	Most species/add to diet; cathartic; dilute with water
Psyllium (Metamucil, Procter & Gamble)	0.5 tsp/60 mL hand-feeding formula	Most species/bulk diet; can use mineral oil as alternative or in addition to psyllium
Ranitidine	5 mg/kg PO q12h	Most species/antacid; anecdotal information
Silymarin (milk thistle)	50–75 mg/kg PO q12h	Most species/hepatic antioxidant; use in patients with liver disease and as ancillary to chemotherapy; use a low-alcohol or alcohol-free liquid formulation
Sucralfate	25 mg/kg PO q8h	Most species/oral, esophageal, gastric, duodenal ulcers; give 1–2 hours before food or other drugs; up to 200 mg/kg used anecdotally without adverse effects

GI, Gastrointestinal; *IM,* intramuscular; *IV,* intravenous; *PO,* oral; *PRN,* as needed; *SC,* subcutaneous.

TABLE 18
Chemotherapeutic Agents

Agent	Dosage	Species/Comments
L-Asparaginase	—	Antimetabolite, used in the treatment of lymphoma and immune-mediated diseases. Anaphylaxis and pancreatitis have been associated with use of L-asparaginase. Monitor patients for anaphylaxis and myelosuppression
	400 U/kg IM q7d[64]	Moluccan cockatoo/lymphosarcoma
	400 U/kg SC once[175]	Black swan/chronic lymphocytic leukemia
	1650 U/kg IM once[157]	Great horned owl/histiocytic sarcoma
Bleomycin	—	Antitumor antibiotic
	0.25 mg intralesionally as part of electrochemotherapy, 2 doses 2 weeks apart[110]	Budgerigar/epithelioid hemangioendothelioma
	0.5–1 mg/kg intralesionally as part of electrochemotherapy[146]	Cockatiel/uropygial gland squamous cell carcinoma
Carboplatin	—	Platinum product, used to treat osteosarcoma, carcinoma, other sarcomas, and mesothelioma. Monitor for myelosuppression and GI and renal toxicity. While not as toxic for the kidneys as cisplatin, caution is advised when using carboplatin in patients with renal insufficiency
	5 mg/kg IV, IO infused over 3 minutes[60,116]	Sulphur-crested cockatoos/PK Budgerigar/renal adenocarcinoma
	5 mg/kg IV[2]	ISA brown layer hens, call ducks, racing pigeons, English budgerigars/PK
	15 mg/kg IV q5wk[24]	Mallard duck/Sertoli cell tumor
	20 mg in 2 mL sesame oil intralesionally[100]	Greylag goose/atypical papilliform xanthomatous neoplasia
	125 mg/m^2 IV as a slow bolus, q14–21d[208]	Yellow-naped Amazon/bile duct carcinoma
	4.6 mg impregnated into calcium-matrix beads placed intralesionally[163]	Kori bustard/myxosarcoma
Chlorambucil	—	Alkylating agent, used most commonly for the treatment of lymphoma, lymphoblastic leukemia, and mast cell tumors. Occasionally combined with doxorubicin to treat mammary carcinoma and soft tissue sarcomas. Monitor for bone marrow depression, GI toxicity, and hepatotoxicity
	1 mg/bird PO 2×/week[133]	Pekin duck/lymphocytic leukemia
	1 mg/kg PO 2×/week[91]	Green-winged macaw/chronic lymphocytic leukemia; discontinued after 6 weeks due to thrombocytopenia
	2 mg/bird PO q48h[176]	Black swan/chronic lymphocytic leukemia
	2 mg/bird PO 2×/week[117]	Great horned owl/disseminated lymphoma; solid tumor increased in size despite therapy
	2 mg/kg PO 2×/week[73,135,153,204]	Umbrella cockatoo/cutaneous B-cell lymphoma Double yellow-headed Amazon/chronic lymphocytic leukemia; no apparent effect after 40 days Java sparrow/thymic lymphoma Cockatiel/chronic T-cell lymphocytic leukemia
Cisplatin	—	Platinum product, used to treat osteosarcoma, carcinoma, other sarcomas, and mesothelioma. Monitor for myelosuppression and GI and renal toxicity.
	0.2 mg/kg intralesionally in a sesame oil suspension[56]	African penguin/squamous cell carcinoma
	1 mg/kg IV over 1 hour[59]	Sulphur-crested cockatoos/PK
	30 mg/m^2 intralesionally (Manucy et al.)[212]	Buffon's macaw/squamous cell carcinoma
	1 mg cisplatin/cm^3 tumor intralesionally, mixed with sesame oil at a concentration of 10 mg/mL, 1×/week × 4 treatments[75]	Use powdered cisplatin rather than commercial injectable solution for better potency
Cyclophosphamide	—	Alkylating agent, used most commonly for the treatment of lymphoma, lymphoblastic leukemia, and mast cell tumors. Occasionally combined with doxorubicin to treat mammary carcinoma and soft tissue sarcomas. Side effects reported experimentally in birds include heteropenia, generalized bone marrow suppression, reversible changes in the liver and feather lesions[58,65,151]
	5 mg/kg PO q24h × 4 days, then 3 days off, then repeat[91]	Green-winged macaw/chronic lymphocytic leukemia
	10 mg/m^2 PO q24h[163]	Kori bustard/myxosarcoma
	200 mg/m^2 IO q7d[64]	Moluccan cockatoo/lymphosarcoma Great horned owl/histiocytic sarcoma

Continued

TABLE 18
Chemotherapeutic Agents—cont'd

Agent	Dosage	Species/Comments
Doxorubicin (Adriamycin)	—	Antitumor antibiotic, used for a wide variety of tumors, including osteosarcoma, mesenchymal and epithelial tumors, leukemias, and lymphomas. Can lead to severe perivascular sloughing if extravasated. Monitor for anaphylaxis, myelosuppression, GI toxicity, renal toxicity, and cardiotoxicity. Cardiomyopathy has been induced experimentally using doxorubicin in turkey poults[32]
	2 mg/kg IV[71]	Sulphur-crested cockatoos/PK
	30 mg/m^2 IO q2d[64]	Moluccan cockatoo/lymphosarcoma
	60 mg/m^2 IV q30d[40]	Blue-fronted Amazon/osteosarcoma
Lomustine (CCNU)	—	Alkylating agent
	60 mg/m^2 PO q3wk[175]	Black swan/chronic lymphocytic leukemia
Vincristine	—	Vinca alkaloid, used to treat lymphoma and other lymphoid tumors, mast cell tumors, and some sarcomas. Toxicities are usually mild, but monitoring for anaphylaxis, GI toxicity, and myelosuppression is warranted
	0.1 mg/kg IV q7–14d[153]	Umbrella cockatoo/cutaneous B-cell lymphoma
	0.5 mg/m^2 IV, then 0.75 mg/m^2 q7d × 3 treatments[133]	Pekin duck/lymphocytic leukemia and malignant lymphoma
	0.75 mg/m^2 q7d × 3 treatments[64]	Moluccan cockatoo/lymphosarcoma

GI, Gastrointestinal; *IM,* intramuscular; *IO,* intraosseous; *IV,* intravenous; *PD,* pharmacodynamic; *PK,* pharmacokinetic; *PO,* oral; *SC,* subcutaneous.

TABLE 19
Miscellaneous Agents

Agent	Dosage	Species/Comments
Anticoagulant citrate dextrose (A-C-D Solution, Sanofi)	0.15 mL/1 mL whole blood	Anticoagulant for transfusions; not effective for extended storage of whole blood; heparin can be substituted if A-C-D is not available
Citrate phosphate dextrose adenine solution (CPDA)	1 part CPDA:5 parts whole blood	Most species/anticoagulant for blood collection for transfusion; not for extended storage of whole blood
Cyclosporine	10–20 mg/kg PO q12–24h	Most species/immunosuppressant, used to treat autoimmune disorders (e.g., immune-mediated hemolytic anemia) and bornaviral-induced inflammation. Efficacy remains controversial at this time
Diphenhydramine	1–4 mg/kg PO q8–12h	Psittacines/allergic rhinitis, hypersensitivity, pruritus
	2 mg/kg IV, IO once	Cockatoos/used before chemotherapy
	2–4 mg/kg PO, IM, IV q12h	Most species/has calming effect in some anxious birds; may cause hypotension
	20–40 mg/L drinking water	Most species
Ferric subsulfate	Topical	Most species/hemostasis of bleeding nail or beak tip; will cause necrosis if used on open skin lesions
Fluids	PO, SC, IV as needed	Fluid requirement for most birds is 50 mL/kg/day
Gadopentetate dimeglumine (Magnevist, Berlex)	0.25 mmol/kg IV	Contrast agent for magnetic resonance imaging
Heparin	2 U/mL whole blood	Anticoagulant for blood transfusions
Hetastarch (Hespan, DuPont)	—	Colloid with a t½ of 25 hours; use with caution in patients suffering from congestive heart failure or renal failure
	10–15 mL/kg IV q8h × 1–4 treatments	Most species/chronic hypoproteinemia; decrease fluid treatment to ⅓–½ maintenance fluid dose
Hydroxyzine (Atarax, Roerig)	2–2.2 mg/kg PO q8h	Allergic pruritus; feather picking; self-mutilation
	34–40 mg/L drinking water	Most species/respiratory allergy; feather picking
Iohexol (Omnipaque, Sanofi Winthrop)	25–30 mL/kg PO	Cockatoos, Amazon parrots/gavage; radiographic gastrointestinal iodinated contrast media; 1:1 dilution with water can also be used
	50 mL/kg PO	Quaker parakeets, budgerigars
Pentobarbital sodium	0.2–1 mL/kg IV, ICe	Most species/euthanasia agent; birds may react unpredictably with IV administration; ICe administration is smooth, quiet
T61	0.03 mg/kg IV[9]	Most species/euthanasia agent; should only be used in unconscious or anesthetized birds

ICe, Intracoelomic; *IM,* intramuscular; *IO,* intraosseous; *IV,* intravenous; *PO,* oral; *SC,* subcutaneous.

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APPENDIX 2

Normal Clinical Pathologic Data

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TABLE 1

Hematology: Laboratory Reference Ranges for Selected Species[1–7]

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Packed Cell Volume (%) Mean	 Range	White Blood Cell Count C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	 Range	Heterophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	 Range
PSITTACIFORMES								
PSITTACIDAE								
Grey parrot	5571	Conventional			10.3	6–13	6.0667	4.635–7.519
(African grey)		S.I.			10.3	6–13	6.0667	4.635–7.519
Genus *Psittacus* sp.		%	48.2	45–53			58.9	45–73
Grey parrot	495	Conventional			12	8–15	7.236	5.520–8.880
(African grey, Congo)		S.I.			12	8–15	7.236	5.520–8.880
Psittacus erithacus		%	46.1	42–52			60.3	46–74
Timneh parrot	219	Conventional			11.3	9–13	6.486	5.085–7.91
(African grey, timneh)		S.I.			11.3	9–13	6.486	5.085–7.91
Psittacus timneh		%	47.8	45–50			57.4	45–70
Amazon	8375	Conventional			12.3	5–17	6.273	3.813–8.733
Genus *Amazona* sp.		S.I.			12.3	5–17	6.273	3.813–8.733
		%	47.6	41–53			51	31–71
Amazon, blue-fronted	828	Conventional			10.4	6–13	5.4808	3.432–7.488
Amazona aestiva		S.I.			10.4	6–13	5.4808	3.432–7.488
		%	47.7	42–53			52.7	33–72
Amazon, double yellow-headed	565	Conventional			10.4	6–13	5.5224	3.952–7.384
Amazona oratrix		S.I.			10.4	6–13	5.5224	3.952–7.384
		%	47.5	40–53			53.1	38–71
Amazon, lilac-crowned	57	Conventional			11	7–13	5.566	3.19–8.03
Amazona finschi		S.I.			11	7–13	5.566	3.19–8.03
		%	47.2	44–52			50.6	29–73
Amazon, mealy	54	Conventional			13.2	9–16	7.4844	5.016–10.032
Amazona farinosa		S.I.			13.2	9–16	7.4844	5.016–10.032
		%	49.7	42–55			56.7	38–76
Amazon, green-cheeked	206	Conventional			11.8	6–16	6.0652	3.894–8.26
(Mexican red head)		S.I.			11.8	6–16	6.0652	3.894–8.26
Amazona viridigenalis		%	46.5	40–51			51.4	33–70
Amazon, orange-winged	240	Conventional			12.2	7–16	6.3806	3.782–8.906
Amazona amazonica		S.I.			12.2	7–16	6.3806	3.782–8.906
		%	47.1	40–52			52.3	31–73
Amazon, panama	67	Conventional			11.7	8–16	6.7158	3.51–9.009
Amazona ochrocephala panamensis		S.I.			11.7	8–16	6.7158	3.51–9.009
		%	49.4	46–54			57.4	30–77
Amazon, red-lored	299	Conventional			12.3	6–16	6.2484	4.059–8.979
Amazona autumnalis		S.I.			12.3	6–16	6.2484	4.059–8.979
		%	48.1	42–55			50.8	33–73
Amazon, yellow-naped	1074	Conventional			12.4	6–17	6.4852	3.844–9.052
Amazona auropalliata		S.I.			12.4	6–17	6.4852	3.844–9.052
		%	48.4	42–55			52.3	31–73
Amazon, yellow-crowned	36	Conventional			12.7	8–15	6.4389	4.699–8.763
Amazona ochrocephala		S.I.			12.7	8–15	6.4389	4.699–8.763
		%	47.8	42–55			50.7	37–69
Budgerigar	1542	Conventional			6.7	3–10	3.7989	2.68–4.556
Melopsittacus undulatus		S.I.			6.7	3–10	3.7989	2.68–4.556
		%	51.5	44–58			56.7	40–68
Caique	30	Conventional			12	8–15	6.768	4.68–8.64
Pionites sp.		S.I.			12	8–15	6.768	4.68–8.64

Lymphocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Monocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Eosinophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Basophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
3.914	1.957–5.150	0.0309	0–0.206	0.0257	0–0.103	0.0824	0–0.103
3.914	1.957–5.150	0.0309	0–0.206	0.0257	0–0.103	0.0824	0–0.103
38	19–50	0.03	0–2	0.24	0–1	0.08	0–1
4.392	2.400–6.480	0.192	0.120–0.360	0.1608	0.120–0.240	0.0852	0–0.120
4.392	2.400–6.480	0.192	1–3	1.34	1–2	0.71	0–1
36.6	20–54	1.6	1–3	1.34	1–2	0.71	0–1
3.819	2.147–5.424	0.00452	0–0.113	0.07119	0–1.226	0.00791	0–0.113
3.819	2.147–5.424	0.00452	0–0.113	0.07119	0–1.226	0.00791	0–0.113
33.8	19–48	0.04	0–1	0.63	0–2	0.07	0–1
5.387	2.460–8.241	0.00615	0–0.246	0	0–0	0.0578	0–0.246
5.387	2.460–8.241	0.00615	0–0.246	0	0–0	0.0578	0–0.246
43.8	20–67	0.05	0–2	0	0–0	0.47	0–2
4.524	2.288–6.67	0.00208	0–0.104	0.00416	0–0.104	0.02808	0–0.104
4.524	2.288–6.67	0.00208	0–0.104	0.00416	0–0.104	0.02808	0–0.104
43.5	22–65	0.02	0–1	0.04	0–1	0.27	0–1
4.6072	2.08–6.76	0.03432	0–0.104	0.0052	0–0.104	0.0364	0–0.104
4.6072	2.08–6.76	0.03432	0–0.104	0.0052	0–0.104	0.0364	0–0.104
44.3	20–65	0.33	0–1	0.05	0–1	0.35	0–1
4.686	2.09–7.37	0.0033	0–0.11	0.0044	0–0.11	0.0319	0–0.11
4.686	2.09–7.37	0.0033	0–0.11	0.0044	0–0.11	0.0319	0–0.11
42.6	19–67	0.03	0–1	0.04	0–1	0.29	0–1
5.94	3.036–8.58	0.00264	0–0.132	0	0–0	0.01584	0–0.132
5.94	3.036–8.58	0.00264	0–0.132	0	0–0	0.01584	0–0.132
45	23–65	0.02	0–1	0	0–0	0.12	0–1
4.8852	2.478–7.67	0.00708	0–0.118	0	0–0	0.07788	0–0.236
4.8852	2.478–7.67	0.00708	0–0.118	0	0–0	0.07788	0–0.236
41.4	21–65	0.06	0–1	0	0–0	0.66	0–2
5.2582	2.44–7.808	0.00244	0–0.122	0	0–0	0.003584	0–0.224
5.2582	2.44–7.808	0.00244	0–0.122	0	0–0	0.003584	0–0.224
43.1	20–64	0.02	0–1	0	0–0	0.32	0–2
5.0193	2.691–8.19	0	0–0.117	0	0–0.117	0.01053	0–0.117
5.0193	2.691–8.19	0	0–0.117	0	0–0.117	0.01053	0–0.117
42.9	23–70	0	0–1	0	0–1	0.09	0–1
5.6949	2.706–8.118	0.00369	0–0.123	0	0–0	0.05289	0–0.246
5.6949	2.706–8.118	0.00369	0–0.123	0	0–0	0.05289	0–0.246
46.3	22–66	0.03	0–1	0	0–0	0.43	0–2
5.4684	2.48–8.308	0.00248	0–0.124	0	0–0	0.05084	0–0.248
5.4684	2.48–8.308	0.00248	0–0.124	0	0–0	0.05084	0–0.248
44.1	20–67	0.02	0–1	0	0–0	0.41	0–2
5.5372	2.286–8.001	0	0–0.127	0	0–0.127	0.03429	0–0.127
5.5372	2.286–8.001	0	0–0.127	0	0–0.127	0.03429	0–0.127
43.6	18–63	0	0–1	0	0–1	0.27	0–1
2.5259	1.474–4.02	0.00737	0–0.134	0	0–0	0.03216	0–0.134
2.5259	1.474–4.02	0.00737	0–0.134	0	0–0	0.03216	0–0.134
37.7	22–60	0.11	0–2	0	0–0	0.48	0–2
4.176	2.4–7.32	0	0–0.24	0	1–0.12	0	1–0.12
4.176	2.4–7.32	0	0–0.24	0	1–0.12	0	1–0.12

Continued

TABLE 1

Hematology: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Packed Cell Volume (%) Mean	 Range	White Blood Cell Count C Units: ×10⁹/L S.I.: ×10³/μL (%) Mean	 Range	Heterophils C Units: ×10⁹/L S.I.: ×10³/μL (%) Mean	 Range
Conure, blue-crowned *Aratinga acuticaudata*	93	Conventional S.I. %	 49.2	 40–53	7.8 7.8	14 14	4.6644 4.6644 59.8	3.354–5.772 3.354–5.772 43–74
Conure, dusky (Dusky-headed parakeet, Weddell's parakeet) *Aratinga weddellii*	29	Conventional S.I. %	 49.3	 41–55	9.9 9.9	5–13 5–13	5.7618 5.7618 58.2	4.257 4.257 43–70
Conure, gold-capped *Aratinga auricapillus*	36	Conventional S.I. %	 52.6	 50–55	10.1 10.1	6–13 6–13	5.7065 5.7065 56.5	4.444–6.969 4.444–6.969 44–69
Conure, jenday *Aratinga jandaya*	55	Conventional S.I. %	 50.6	 42–55	8.1 8.1	6–10 6–10	4.6 4.6 56.8	3.726–5.751 3.726–5.751 46–71
Conure, nanday *Aratinga nenday*	179	Conventional S.I. %	 50.1	 40–55	8.7 8.7	5–11 5–11	5.203 5.203 59.8	4.176–6.177 4.176–6.177 48–71
Conure, sun *Aratinga solstitialis*	286	Conventional S.I. %	 49.7	 42–55	9 9	6–11 6–11	4.797 4.797 53.3	3.96–6.48 3.96–6.48 44–72
Conure, green-cheeked *Pyrrhura molinae*	41	Conventional S.I. %	 50.9	 42–55	7.7 7.7	5–10 5–10	4.3736 4.3736 56.8	3.542–5.467 3.542–5.467 46–71
Conure, maroon-bellied (Red-bellied conure, brown-eared conure) *Pyrrhura frontalis*	28	Conventional S.I. %	 50.7	 46–55	10.4 10.4	7–13 7–13	5.751 5.751 55.3	4.576–7.384 4.576–7.384 44–71
Conure, cherry-headed (Red-masked parakeet) *Psittacara erythrogenys*	66	Conventional S.I. %	 49.9	 43–56	8 8	5–10 5–10	4.808 4.808 60.1	3.84–5.84 3.84–5.84 48–73
Conure, mitred *Psittacara mitratus*	74	Conventional S.I. %	 47.1	 40–52	8.1 8.1	5–11 5–11	4.795 4.795 59.2	3.564–5.832 3.564–5.832 44–72
Conure, half-moon (Orange-fronted parakeet) *Eupsittula canicularis*	29	Conventional S.I. %	 49.1	 42–54	8.2 8.2	6–10 6–10	5.2808 5.2808 64.4	4.1–5.986 4.1–5.986 50–73
Conure, Patagonian *Cyanoliseus patagonus*	57	Conventional S.I. %	 48.4	 42–55	8.8 8.8	6–11 6–11	5.262 5.262 59.8	3.872–6.336 3.872–6.336 44–72
Eclectus parrot *Eclectus roratus*	415	Conventional S.I. %	 49.5	 45–55	12.5 12.5	9–15 9–15	7.25 7.25 58	5.75–8.75 5.75–8.75 46–70
Grey-cheeked parakeet (Fire-winged parakeet) *Brotogeris pyrrhoptera*	449	Conventional S.I. %	 50.2	 45–56	8.3 8.3	4–12 4–12	4.6729 4.6729 56.3	3.735–5.644 3.735–5.644 45–68
Lory (11 genera, 53 species)	130	Conventional S.I. %	 50.6	 47–55	10.8 10.8	8–13 8–13	5.4216 5.4216 50.2	4.212–6.48 4.212–6.48 39–60
Lovebird *Agapornis* sp.	650	Conventional S.I. %	 50.5	 44–55	11.7 11.7	7–16 7–16	5.6511 5.6511 48.3	11.232 11.232 96

Lymphocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Monocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Eosinophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Basophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
2.8002	1.794–3.9	0	0–0.078	0.01326	0–0.078	0.01638	0–0.156
2.8002	1.794–3.9	0	0–0.078	0.01326	0–0.078	0.01638	0–0.156
35.9	23–50	0	0–1	0.17	0–1	0.21	0–2
3.6531	1.98–5.148	0	0–0.198	0	0–0.099	0	0–0.099
3.6531	1.98–5.148	0	0–0.198	0	0–0.099	0	0–0.099
36.9	20–52	0	0–2	0	0–1	0	0–1
3.8481	2.626–4.949	0	0–0.101	0	0–0.101	0.03333	0–0.202
3.8481	2.626–4.949	0	0–0.101	0	0–0.101	0.03333	0–0.202
38.1	26–49	0	0–1	0	0–1	0.33	0–2
3.305	1.782–4.374	0	0–0.162	0	0–0.104	0	0–0.32
3.305	1.782–4.374	0	0–0.162	0	0–0.104	0	0–0.32
40.8	22–54	0	0–2	0	0–1	0	0–4
3.036	2.088–4.089	0	0–0.087	0.096	0–0.087	0.053	0–0.174
3.036	2.088–4.089	0	0–0.087	0.096	0–0.087	0.053	0–0.174
34.9	24–47	0	0–1	0.11	0–1	0.61	0–2
3.141	1.8–4.41	0.0054	0–0.09	0	0–0	0.0684	0–0.18
3.141	1.8–4.41	0.0054	0–0.09	0	0–0	0.0684	0–0.18
34.9	20–49	0.06	0–1	0	0–0	0.76	0–2
3.0492	1.694–4.081	0	0–0.154	0	0–0.077	0	0–0.077
3.0492	1.694–4.081	0	0–0.154	0	0–0.077	0	0–0.077
39.6	22–53	0	0–2	0	0–1	0	0–1
4.222	2.496–5.824	0	0–0.028	0	0–0.081	0	0–0.104
4.222	2.496–5.824	0	0–0.028	0	0–0.081	0	0–0.104
40.6	24–56	0	0–2	0	0–1	0	0–1
2.888	1.84–4	0	0–0.16	0	0–0.08	0	0–0.08
2.888	1.84–4	0	0–0.16	0	0–0.08	0	0–0.08
36.1	23–50	0	0–2	0	0–1	0	0–1
2.9	1.863–4.05	0	0–0.162	0	0–0.081	0	0–0.081
2.9	1.863–4.05	0	0–0.162	0	0–0.081	0	0–0.081
35.8	23–50	0	0–2	0	0–1	0	0–1
2.7306	1.64–5.002	0	0–0.164	0	0–0.082	0	0–0.082
2.7306	1.64–5.002	0	0–0.164	0	0–0.082	0	0–0.082
33.3	20–51	0	0–2	0	0–1	0	0–1
2.939	1.848–4.576	0	0–0.088	0	0–0.088	0.021	0–0.088
2.939	1.848–4.576	0	0–0.088	0	0–0.088	0.021	0–0.088
33.4	21–52	0	0–1	0	0–1	0.24	0–1
5.05	2.875–7.125	0	0–0.125	0.0175	0–0.125	0.01	0–0.125
5.05	2.875–7.125	0	0–0.125	0.0175	0–0.125	0.01	0–0.125
40.4	23–57	0	0–1	0.14	0–1	0.08	0–1
2.9382	1.826–3.984	0.00415	0–0.083	0.00581	0–0.083	0.00415	0–0.083
2.9382	1.826–3.984	0.00415	0–0.083	0.00581	0–0.083	0.00415	0–0.083
35.4	22–48	0.05	0–1	0.07	0–1	0.05	0–1
4.7196	2.376–7.452	0	0–0.216	0	0–0.108	0	0–0.108
4.7196	2.376–7.452	0	0–0.216	0	0–0.108	0	0–0.108
43.7	22–69	0	0–2	0	0–1	0	0–1
4.212	2.34–6.201	0.00468	0–0.117	0.03159	0–0.234	0.02691	0–0.234
4.212	2.34–6.201	0.00468	0–0.117	0.03159	0–0.234	0.02691	0–0.234
36	20–53	0.04	0–1	0.27	0–2	0.23	0–2

Continued

TABLE 1

Hematology: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Packed Cell Volume (%) Mean	 Range	White Blood Cell Count C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	 Range	Heterophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	 Range
Macaw group	5338	Conventional			15.2	10–20	9.5912	7.6–11.4
Mostly *Ara*, some *Anodorhynchus*		S.I.			15.2	10–20	9.5912	7.6–11.4
		%	48.5	42–56			63.1	50–75
Macaw, blue-and-gold	1367	Conventional			12.8	8–16	7.7568	6.272–9.088
(Blue-and-yellow macaw)		S.I.			12.8	8–16	7.7568	6.272–9.088
Ara ararauna		%	48.7	44–55			60.6	49–71
Macaw, green-winged	347	Conventional			14.2	11–16	8.9602	6.958–10.366
(Red-and-green macaw)		S.I.			14.2	11–16	8.9602	6.958–10.366
Ara chloropterus		%	47.2	42–54			63.1	49–73
Macaw, military	88	Conventional			16.5	12–20	11.8305	9.57–14.025
Ara militaris		S.I.			16.5	12–20	11.8305	9.57–14.025
		%	46.8	44–51			71.7	58–85
Macaw, scarlet	146	Conventional			12.5	10–14	7.525	6–9.125
(Red-and-yellow macaw)		S.I.			12.5	10–14	7.525	6–9.125
Ara macao		%	49.1	45–55			60.2	48–73
Macaw, severe	104	Conventional			12.1	9–14	7.3931	5.445–9.68
(Chestnut-fronted macaw)		S.I.			12.1	9–14	7.3931	5.445–9.68
Ara severus		%	49.1	45–55			61.1	45–80
Macaw, Hahn's	51	Conventional			14.1	10–18	7.5999	5.781–10.152
(Red-shouldered macaw)		S.I.			14.1	10–18	7.5999	5.781–10.152
Diopsittaca nobilis nobilis		%	51.3	47–55			53.9	41–72
Macaw, noble	22	Conventional			14.6	8–22	7.2708	4.964–9.198
(Red-shouldered macaw)		S.I.			14.6	8–22	7.2708	4.964–9.198
Diopsittaca nobilis cumanensis		%	52.3	48–56			49.8	34–63
Macaw, hyacinth	149	Conventional			13.4	11–16	9.2862	7.504–10.72
Anodorhynchus hyacinthinus		S.I.			13.4	11–16	9.2862	7.504–10.72
		%	50.1	45–55			69.3	56–80
Macaw, Spix's	37	Conventional			9.24 ± 2.99	2.80–15.90	5.13 ± 2.50	1.3–14.1
Cyanopsitta spixii		S.I.			9.24 ± 2.99	2.80–15.90	5.13 ± 2.50	1.3–14.1
		%	46.8 ± 3.2	36.5–53.0			56.4 ± 12.9	29.0–79.3
Macaw, yellow-collared	70	Conventional			13.6	10–16	8.6904	7.344–10.064
(Golden-collared macaw)		S.I.			13.6	10–16	8.6904	7.344–10.064
Primolius auricollis		%	50.9	42–55			63.9	54–74
Parakeet, Alexandrine	18	Conventional			14.3	10–16	8.923	7.293–10.439
Psittacula eupatria		S.I.			14.3	10–16	8.923	7.293–10.439
		%	47.4	45–54			62.4	51–73
Moustache parakeet	26	Conventional			11.6	8–15	7.6676	5.8–9.164
(Rose-breasted parakeet)		S.I.			11.6	8–15	7.6676	5.8–9.164
Psittacula alexandri		%	46.1	42–51			66.1	50–79
Parakeet, ringneck	46	Conventional			11.2	8–14	6.0144	4.48–7.616
(Rose-ringed parakeet)		S.I.			11.2	8–14	6.0144	4.48–7.616
Psittacula krameri		%	49.5	45–54			53.7	40–68
Parrot, Meyers	39	Conventional			11.2	8–14	6.9328	5.376–8.4
Poicephalus meyeri		S.I.			11.2	8–14	6.9328	5.376–8.4
		%	49.9	98			61.9	48–75
Parrot, red-bellied	14	Conventional			12.6	9–16	7.5474	7.056–8.442
Poicephalus rufiventris		S.I.			12.6	9–16	7.5474	7.056–8.442
		%	48.3	45–51			59.9	56–67

Lymphocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Monocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Eosinophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Basophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
5.624	3.496–8.056	0.00456	0–0.152	0	0–0	0.01064	0–0.152
5.624	3.496–8.056	0.00456	0–0.152	0	0–0	0.01064	0–0.152
37	23–53	0.03	0–1	0	0–0	0.07	0–1
4.4416	2.304–6.784	0.00512	0–0.256			0.00512	0–0.128
4.4416	2.304–6.784	0.00512	0–0.256			0.00512	0–0.128
34.7	18–53	0.04	0–2			0.04	0–1
5.1972	3.124–8.52		0–0.284		0–0.284	0.01704	0–0.142
5.1972	3.124–8.52		0–0.284		0–0.284	0.01704	0–0.142
36.6	22–60		0–2		0–2	0.12	0–1
5.28	4.125–6.6		0–0.33		0–0.165		0–0.165
5.28	4.125–6.6		0–0.33		0–0.165		0–0.165
32	25–40		0–2		0–1		0–1
4.55	2.875–6.25	0	0–0.25	0	0–0.125	0.0175	0–0.125
4.55	2.875–6.25	0	0–0.25	0	0–0.125	0.0175	0–0.125
36.4	23–50	0	0–2	0	0–1	0.14	0–1
4.3681	3.025–5.687	0	0–0.242	0	0–0.121	0.05203	0–0.242
4.3681	3.025–5.687	0	0–0.242	0	0–0.121	0.05203	0–0.242
36.1	25–47	0	0–2	0	0–1	0.43	0–2
7.191	3.807–10.011		0–0.282		0–0.141	0	0–0.141
7.191	3.807–10.011		0–0.282		0–0.141	0	0–0.141
51	27–71		0–2		0–1	0	0–1
7.0226	4.672–9.636	0	0–0.146	0	0–0.146	0	0–0.146
7.0226	4.672–9.636	0	0–0.146	0	0–0.146	0	0–0.146
48.1	32–66	0	0–1	0	0–1	0	0–1
4.355	2.412–8.308		0–0.268		0–0.268	0.01206	0–0.134
4.355	2.412–8.308		0–0.268		0–0.268	0.01206	0–0.134
32.5	18–62		0–2		0–2	0.09	0–1
3.74 ± 1.90	0.6–8.5	0.30 ± 0.20	0.07–0.90	0.01 ± 0.03	0.05–0.13	0	0
3.74 ± 1.90	0.6–8.5	0.30 ± 0.20	0.07–0.90	0.01 ± 0.03	0.05–0.13	0	0
41.1 ± 12.9	17–67	3.1 ± 1.5	1.0–6.5	0.6 ± 0.5	0–1.0	0	0
5.1952	3.4–7.072	0	0–0.136	0	0–0.136	0	0–0.136
5.1952	3.4–7.072	0	0–0.136	0	0–0.136	0	0–0.136
38.2	25–52	0	0–1	0	0–1	0	0–1
5.22	3.861–7.007	0	0–0.143	0	0–0.143	0	0–0.143
5.22	3.861–7.007	0	0–0.143	0	0–0.143	0	0–0.143
36.5	27–49	0	0–1	0	0–1	0	0–1
4.3384	2.436–6.38	0	0–0.116	0	0–0.116	0	0–0.116
4.3384	2.436–6.38	0	0–0.116	0	0–0.116	0	0–0.116
37.4	21–55	0	0–1	0	0–1	0	0–1
4.9392	3.584–6.272	0	0–0.112	0	0–0.112	0	0–0.112
4.9392	3.584–6.272	0	0–0.112	0	0–0.112	0	0–0.112
44.1	32–56	0	0–1	0	0–1	0	0–1
3.8304	2.576–5.264	0	0–0.224	0	0–0.112	0	0–0.112
3.8304	2.576–5.264	0	0–0.224	0	0–0.112	0	0–0.112
34.2	23–47	0	0–2	0	0–1	0	0–1
5.922	4.158–9.072	0	0–0.252	0	0–0.252	0	0–0.126
5.922	4.158–9.072	0	0–0.252	0	0–0.252	0	0–0.126
47	33–72	0	0–2	0	0–2	0	0–1

Continued

TABLE 1

Hematology: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Packed Cell Volume (%) Mean	 Range	White Blood Cell Count C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	 Range	Heterophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	 Range
Parrot, thick-billed	10	Conventional			12.3	8–21	6.1623	3.936–8.364
Rhynchopsitta pachyrhyncha		S.I.			12.3	8–21	6.1623	3.936–8.364
		%	39.3	33–43			50.1	32–68
Parrotlet species	29	Conventional			8.8	6–13	5.6496	4.84–6.512
Genus *Forpus* sp.		S.I.			8.8	6–13	5.6496	4.84–6.512
		%	50.8	48–55			64.2	55–74
Pionus species group	431	Conventional			9.6	5–13	6.3168	0.48–7.104
Genus *Pionus* sp.		S.I.			9.6	5–13	6.3168	0.48–7.104
		%	49.8	45–54			65.8	5–74
Pionus, blue-headed	26	Conventional			12.6	10–16	7.4844	5.544–9.702
Pionus menstruus		S.I.			12.6	10–16	7.4844	5.544–9.702
		%	48.1	40–53			59.4	44–77
Pionus, white-capped	49	Conventional			8.8	5–11	5.3504	4.136–6.512
Pionus senilis		S.I.			8.8	5–11	5.3504	4.136–6.512
		%	50.6	47–55			60.8	47–74
Parrot, Senegal	244	Conventional			10.7	6–14	6.2702	4.708–7.811
Poicephalus senegalus		S.I.			10.7	6–14	6.2702	4.708–7.811
		%	50.6	45–60			58.6	44–73
Quaker parrot	210	Conventional			12.2	8–17	7.442	5.734–8.54
(Monk parakeet)		S.I.			12.2	8–17	7.442	5.734–8.54
Myiopsitta monachus		%	49.7	45–58			61	47–70
Rosella	21	Conventional			11.8	9–15	6.7142	5.428–8.142
Genus *Platycercus* sp.		S.I.			11.8	9–15	6.7142	5.428–8.142
		%	51	45–60			56.9	46–69
CACATUIDAE								
Cockatiel	9267	Conventional			8	5–11	4.752	3.68–5.76
Nymphicus hollandicus		S.I.			8	5–11	4.752	3.68–5.76
		%	51.2	43–57			59.4	46–72
Cockatoo group	5928	Conventional			10.4	5–13	6.2	4.68–7.49
Family Cacatuidae		S.I.			10.4	5–13	6.2	4.68–7.49
		%	45.4	40–54			59.6	45–72
Cockatoo, bare-eyed	30	Conventional			10.6	6–14	6.4978	4.346–8.162
(Little corella)		S.I.			10.6	6–14	6.4978	4.346–8.162
Cacatua sanguinea		%	48.3	43–55			61.3	41–77
Cockatoo, Goffin's	298	Conventional			10.8	7–13	6.372	4.86–7.776
(Tanimbar corella)		S.I.			10.8	7–13	6.372	4.86–7.776
Cacatua goffiniana		%	47.3	42–55			59	45–72
Cockatoo, lesser sulphur-crested	17	Conventional			12.3	15	8.364	5.412–9.717
Cacatua sulphurea		S.I.			12.3	15	8.364	5.412–9.717
		%	49.3	45–56			68	44–79
Cockatoo, Major Mitchell	14	Conventional			10		6.03	3.7–7.8
(Leadbeater's cockatoo, pink cockatoo)		S.I.			10	6–13	6.03	3.7–7.8
Lophochroa leadbeateri		%	47.9	42–53			60.3	37–78
Cockatoo, Moluccan	772	Conventional			10.6	8–12	6.201	4.664–7.526
(Salmon-crested cockatoo)		S.I.			10.6	8–12	6.201	4.664–7.526
Cacatua moluccensis		%	45.3	41–54			58.5	44–71
Cockatoo, rose-breasted	92	Conventional			12.3	8–15	7.4538	5.289–9.102
(Galah, roseate cockatoo, pink-and-grey cockatoo)		S.I.			12.3	8–15	7.4538	5.289–9.102
Eolophus roseicapillus		%	48.8	40–55			60.6	43–74

Lymphocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Monocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Eosinophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Basophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
4.797	2.46–6.888		0–0.123		0–0.123		0–0.123
4.797	2.46–6.888		0–0.123		0–0.123		0–0.123
39	20–56		0–1		0–1		0–1
3.1328	2.112–4.4	0	0–0.088	0	0–0.088	0	0–0.088
3.1328	2.112–4.4	0	0–0.088	0	0–0.088	0	0–0.088
35.6	24–50	0	0–1	0	0–1	0	0–1
3.9936	1.824–6.72	0	0–0.096	0	0–0.096	0.0144	0–0.096
3.9936	1.824–6.72	0	0–0.096	0	0–0.096	0.0144	0–0.096
41.6	19–70	0	0–1	0	0–1	0.15	0–1
4.5108	2.394–6.552	0	0–0.126	0	0–0.126	0	0–0.252
4.5108	2.394–6.552	0	0–0.126	0	0–0.126	0	0–0.252
35.8	19–52	0	0–1	0	0–1	0	0–2
3.7488	1.76–6.16	0	0–0.088	0	0–0.088	0	0–0.176
3.7488	1.76–6.16	0	0–0.088	0	0–0.088	0	0–0.176
42.6	20–70	0	0–1	0	0–1	0	0–2
4.9541	2.354–7.49	0	0–0.107	0.02675	0–0.214	0.00749	0–0.107
4.9541	2.354–7.49	0	0–0.107	0.02675	0–0.214	0.00749	0–0.107
46.3	22–70	0	0–1	0.25	0–2	0.07	0–1
4.209	2.44–7.686	0.04758	0–0.488			0.07686	0–0.366
4.209	2.44–7.686	0.04758	0–0.488			0.07686	0–0.366
34.5	20–63	0.39	0–4			0.63	0–3
5.3454	2.95–7.552	0	0–0.236	0	0–0.118	0	0–0.236
5.3454	2.95–7.552	0	0–0.236	0	0–0.118	0	0–0.236
45.3	25–64	0	0–2	0	0–1	0	0–2
3.392	2.08–4.8	0.004	0–0.08	0.0488	0–0.16	0.0096	0–0.08
3.392	2.08–4.8	0.004	0–0.08	0.0488	0–0.16	0.0096	0–0.08
42.4	26–60	0.05	0–1	0.61	0–2	0.12	0–1
3.56	2.08–5.20	0.003	0–0.2	0.033	0–0.2	0	0–0.1
3.56	2.08–5.20	0.003	0–0.2	0.033	0–0.2	0	0–0.1
34.2	20–50	0.03	0–2	0.33	0–2	0	0–1
4.0492	2.332–6.042	0	0–0.212	0.00954	0–0.106	0	0–0.106
4.0492	2.332–6.042	0	0–0.212	0.00954	0–0.106	0	0–0.106
38.2	22–57	0	0–2	0.09	0–1	0	0–1
3.8556	2.376–5.508	0.00324	0–0.108	0.0378	0–0.216	0	0–0.108
3.8556	2.376–5.508	0.00324	0–0.108	0.0378	0–0.216	0	0–0.108
35.7	22–51	0.03	0–1	0.35	0–2	0	0–1
3.936	2.583–6.888		0–0.246		0–0.246		0–0.123
3.936	2.583–6.888		0–0.246		0–0.246		0–0.123
32	21–56		0–2		0–2		0–1
3.74	2–6.2	0	0–0.02	0	0–0.01	0	0–0.01
3.74	2–6.2	0	0–0.02	0	0–0.01	0	0–0.01
37.4	20–62	0	0–2	0	0–1	0	0–1
3.6252	2.014–5.3	0.00212	0–0.106	0.02968	0–0.212	0	0–0.106
3.6252	2.014–5.3	0.00212	0–0.106	0.02968	0–0.212	0	0–0.106
34.2	19–50	0.02	0–1	0.28	0–2	0	0–1
4.2312	2.46–6.396	0	0–0.246	0.02952	0–0.123	0	0–0.123
4.2312	2.46–6.396	0	0–0.246	0.02952	0–0.123	0	0–0.123
34.3	20–52	0	0–2	0.24	0–1	0	0–1

Continued

TABLE 1

Hematology: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Packed Cell Volume (%) Mean	Packed Cell Volume (%) Range	White Blood Cell Count C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	White Blood Cell Count C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Range	Heterophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	Heterophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Range
Cockatoo, triton	544	Conventional			14.6	12–16	8.7892	6.424–10.95
Cacatua galerita triton		S.I.			14.6	12–16	8.7892	6.424–10.95
		%	45.5	42–50			60.2	44–75
Cockatoo, umbrella	951	Conventional			12.8	8–16	7.616	5.76–9.088
Cacatua alba		S.I.			12.8	8–16	7.616	5.76–9.088
		%	44.8	40–50			59.5	45–71
PASSERIFORMES								
Canary	300	Conventional			7	3–10	2.576	1.47–4.2
Serinus canaria forma domestica		S.I.			7	3–10	2.576	1.47–4.2
		%	49.9	45–56			36.8	21–60
Mynah species	44	Conventional			9.9	8–12	5.4945	4.455–6.336
Mostly *Acridotheres tristis*		S.I.			9.9	8–12	5.4945	4.455–6.336
		%	47.7	38–50			55.5	45–64
ANSERIFORMES								
Duck, domestic	73	Conventional			18.1	14–24	11.132	9.593–12.489
Anas platyrhynchos domestica		S.I.			18.1	14–24	11.132	9.593–12.489
		%	43.4	35–50			61.5	53–69
Swan	36	Conventional			13.4	11–16	7.1288	4.556–8.978
Cygnus sp.		S.I.			13.4	11–16	7.1288	4.556–8.978
		%	45.6	42–50	13.4		53.2	34–67
GALLIFORMES								
Chicken, domestic		Conventional				9–32		
Gallus gallus domesticus		S.I.				9–32		
		%		23–55				15–50
Quail		Conventional				12.5–24.6		
Coturnix spp.		S.I.				12.5–24.6		
		%		30–45				25–50
ACCIPITRIFORMES								
Combined eagle species	18	Conventional			16.9	12–21	11.762	8.788–13.52
Bald eagle		S.I.			16.9	12–21	11.762	8.788–13.52
Haliaeetus leucocephalus		%	41.9	37–48			69.6	52–80
Golden eagle *Aquila chrysaetos*								
Hawk, red-tailed	13	Conventional			19.5	15–24	9.7305	6.825–12.87
Buteo jamaicensis		S.I.			19.5	15–24	9.7305	6.825–12.87
		%	45.1	38–52			49.9	35–66
Common buzzard		Conventional			12.65 ± 6.90	10.17–15.12	4.63 ± 3.29	3.46–5.81
Buteo buteo		S.I.			12.65 ± 6.90	10.17–15.12	4.63 ± 3.29	3.46–5.81
		%	39 ± 5	38–41				
FALCONIFORMES								
Gyrfalcon	25	Conventional			7.3 ± 0.38	4.2–10.8	4.67 ± 0.34	2.31–8.85
Falco rusticolus		S.I.			7.3 ± 0.38	4.2–10.8	4.67 ± 0.34	2.31–8.85
		%	51.36 ± 0.90	44–59				
STRUTHIONIFORMES								
Emu	98	Conventional			14.9	8–21	11.7412	8.046–13.112
Dromaius novaehollandiae		S.I.			14.9	8–21	11.7412	8.046–13.112
		%	47.4	39–57			78.8	54–88

Lymphocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Monocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Eosinophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Basophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
5.5626	3.212–8.176	0	0–0.292	0	0–0.146	0	0–0.146
5.5626	3.212–8.176	0	0–0.292	0	0–0.146	0	0–0.146
38.1	22–56	0	0–2	0	0–1	0	0–1
4.416	2.56–6.528	0.00384	0–0.256	0.04864	0–0.256	0	0–0.128
4.416	2.56–6.528	0.00384	0–0.256	0.04864	0–0.256	0	0–0.128
34.5	20–51	0.03	0–2	0.38	0–2	0	0–1
3.031	1.4–4.55	0.0028	0–0.07	0.0028	1–0.07	0.0056	0–0.07
3.031	1.4–4.55	0.0028	0–0.07	0.0028	1–0.07	0.0056	0–0.07
43.3	20–65	0.04	0–1	0.04	0–1	0.08	0–1
4.0095	2.079–5.445	0	0–0.099	0.07128	0–0.396	0	0–0.099
4.0095	2.079–5.445	0	0–0.099	0.07128	0–0.396	0	0–0.099
40.5	21–55	0	0–1	0.72	0–4	0	0–1
6.588	4.887–9.05	0	0–0.362	0.0468	0–0.181	0	0–0.181
6.588	4.887–9.05	0	0–0.362	0.0468	0–0.181	0	0–0.181
36.4	27–50	0	0–2	0.26	0–1	0	0–1
5.494	3.082–8.844	0	0–0.268	0	0–0.134	0	0–0.134
5.494	3.082–8.844	0	0–0.268	0	0–0.134	0	0–0.134
41	23–66	0	0–2	0	0–1	0	0–1
	29–84		0.1–7		0–16		0–8
	50–70		0.5–3.8		0–15		0–1.5
4.039	3.042–5.746	0	0–0.507	0.197	0–0.676	0	0–0.338
4.039	3.042–5.746	0	0–0.507	0.197	0–0.676	0	0–0.338
23.9	18–34	0	0–3	1.17	0–4	0	0–2
6.6495	3.705–9.75	0	0–0.585	0	0–78	0	0–0.39
6.6495	3.705–9.75	0	0–0.585	0	0–78	0	0–0.39
34.1	19–50	0	0–3	0	0–4	0	0–2
4.52 ± 3.94	3.10–5.93	0.50 ± 0.42	0.35–0.65	2.73 ± 2.13	1.97–3.49	0.22 ± 0.19	0.14–0.39
4.52 ± 3.94	3.10–5.93	0.50 ± 0.42	0.35–0.65	2.73 ± 2.13	1.97–3.49	0.22 ± 0.19	0.14–0.39
1.43 ± 0.10	0.48–2.36	0.42 ± 0.05	0.03–0.9	0.27 ± 0.04	0–0.68	0.05 ± 0.02	0–0.29
1.43 ± 0.10	0.48–2.36	0.42 ± 0.05	0.03–0.9	0.27 ± 0.04	0–0.68	0.05 ± 0.02	0–0.29
2.9502	1.49–6.556	0.00149	0–0.149	0.447	0–0.894	0.0298	0–0.149
2.9502	1.49–6.556	0.00149	0–0.149	0.447	0–0.894	0.0298	0–0.149
19.8	10–44	0.1	0–1	3	0–6	0.2	0–1

Continued

TABLE 1

Hematology: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Packed Cell Volume (%) Mean	 Range	White Blood Cell Count C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	 Range	Heterophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%) Mean	 Range
Ostrich	140	Conventional			18.7	10–24	5.2173	10.846–16.643
Struthio camelus		S.I.			18.7	10–24	5.2173	10.846–16.643
		%	45	41–57			27.9	58–89
COLUMBIFORMES								
Dove	45	Conventional			11	9–13	5.951	4.73–6.93
Streptopelia risoria and *Geopelia cuneata*		S.I.			11	9–13	5.951	4.73–6.93
		%	47.3	92			54.1	43–63
Pigeon *Columbia livia*								
STRIGIFORMES								
Owl, great-horned	18	Conventional			17	10–25	11.764	9.52–13.43
Bubo virginianus		S.I.			17	10–25	11.764	9.52–13.43
		%	38.1	30–50			69.2	56–79
PICIFORMES								
Toucan	86	Conventional			13.5	8–18	7.0065	5.535–8.37
Ramphastos sp.		S.I.			13.5	8–18	7.0065	5.535–8.37
		%	49.8	42–60			51.9	41–62
PHOENICOPTERIFORMES								
Chilean flamingo	32	Conventional			6.65 ± 3.28	3.00–14.00	2.57 ± 1.39	1.05–6.15
Phoenicopterus chilensis		S.I.			6.65 ± 3.28	3.00–14.00	2.57 ± 1.39	1.05–6.15
		%	46 ± 5	50–58			26.7 ± 7.2	15.0–44.0
SPHENISCIFORMES								
African penguin		Conventional			17.71 ± 8.43	3.50–60		
Spheniscus demersus		S.I.			17.71 ± 8.43	3.50–60		
		%	46 ± 5.7	15–55				

Lymphocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Monocytes C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Eosinophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)		Basophils C Units: $\times 10^9$/L S.I.: $\times 10^3$/μL (%)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
4.5254	2.244–7.667	0.49368	0–0.748	0.00748	0–0.374	0.0374	0–0.374
4.5254	2.244–7.667	0.49368	0–0.748	0.00748	0–0.374	0.0374	0–0.374
24.2	12–41	2.64	0–4	0.04	0–2	0.2	0–2
5.709	4.62–6.71	0.0011	0–0.22	0	0–0.22	0	0–0.22
5.709	4.62–6.71	0.0011	0–0.22	0	0–0.22	0	0–0.22
51.9	42–61	0.1	0–2	0	0–2	0	0–2
4.76	3.4–8.16		0–0.34	0.7888	0–1.87		0–0.17
4.76	3.4–8.16		0–0.34	0.7888	0–1.87		0–0.17
28	20–48		0–2	4.64	0–11		0–1
6.8175	4.725–9.45	0	0–0.27	0.09045	0–0.405	0	0–0.135
6.8175	4.725–9.45	0	0–0.27	0.09045	0–0.405	0	0–0.135
50.5	35–70	0	0–2	0.67	0–3	0	0–1
4.91 ± 2.04	1.09–9.10	0.06 ± 0.07	0.00–0.19	0.55 ± 0.50	0.00–1.74	0.01 ± 0.02	0.00–0.08
4.91 ± 2.04	1.09–9.10	0.06 ± 0.07	0.00–0.19	0.55 ± 0.50	0.00–1.74	0.01 ± 0.02	0.00–0.08
67.4 ± 7.3	52.0–75.0	0.59 ± 0.59	0.00–2.00	5.76 ± 3.20	0.00–13.00	0.18 ± 0.39	0.00–1.00

TABLE 2
Erythrocytic Parameters[1,3]

Species Common Name (Common Name Synonyms) Scientific Name		Red Blood Cell Count (RBC) C Units: $\times 10^6/\mu L$ S.I.: $\times 10^{12}/L$		Packed Cell Volume (PCV) (%)	
		Mean	Range	Mean	Range
PSITTACIFORMES					
PSITTACIDAE					
Grey parrot	Conventional	3.27	2.84–3.62		
(African grey)	S.I.	3.27	2.84–3.62		
Genus *Psittacus* sp.	%			48.96	41.1–53.5
Amazon	Conventional	2.82	2.45–3.18		
Genus *Amazona* sp.	S.I.	2.82	2.45–3.18		
	%			47.83	41.96–52.5
Budgerigar	Conventional	4.32	3.77–4.6		
Melopsittacus undulatus	S.I.	4.32	3.77–4.6		
	%			51.45	43.4–56.1
Eclectus parrot	Conventional	2.98	2.5–3.7		
Eclectus roratus	S.I.	2.98	2.5–3.7		
	%			47.51	42.4–53.7
Lory (11 genera, 53 species)	Conventional	1.85	1.72–2.1		
	S.I.	1.85	1.72–2.1		
	%			47.4	39–57
Lovebird	Conventional	3.79	3.25–3.95		
Agapornis sp.	S.I.	3.79	3.25–3.95		
	%			48.3	41.5–55.4
Macaw group	Conventional	3.82	3.45–4.04		
Mostly *Ara* and some *Anodorhynchus*	S.I.	3.82	3.45–4.04		
	%			50.7	44.6–55.3
Parrot, Senegal	Conventional	1.8	1.70–2.17		
Poicephalus senegalus	S.I.	1.8	1.70–2.17		
	%			45	41–57
CACATUIDAE					
Cockatiel	Conventional	3.93	3.1–4.4		
Nymphicus hollandicus	S.I.	3.93	3.1–4.4		
	%			52.63	41.4–58.9
Cockatoo group	Conventional	2.84	2.44–3.34		
Family Cacatuidae	S.I.	2.84	2.44–3.34		
	%			47.1	40.2–55.12
ANSERIFORMES					
Duck, domestic	Conventional	3.61	3.2–4.01		
Anas platyrhynchos domestica	S.I.	3.61	3.2–4.01		
	%			52.06	42.3–57.7
GALLIFORMES					
Chicken, domestic	Conventional		1.3–4.5		
Gallus gallus domesticus	S.I.		1.3–4.5		
	%				23–55
Quail	Conventional		4.0–5.2		
Coturnix spp.	S.I.		4.0–5.2		
	%				30–45
STRUTHIONIFORMES					
Emu	Conventional	2.93	2.38–3.33		
Dromaius novaehollandiae	S.I.	2.93	2.38–3.33		
	%			49.38	42.2–54.6
Ostrich	Conventional	3.11	2.63–3.49		
Struthio camelus	S.I.	3.11	2.63–3.49		
	%			48.87	41.7–53.9

Hemoglobin (Hb) Conventional: g/dL S.I.: g/L		Mean Corpuscular Volume (MCV) = PCV/RBC Conventional: μm^3/cell S.I.: fl/cell		Mean Corpuscular Hemoglobin (MCH) = Hb/RBC Conventional: pg/cell S.I.: pg/cell		Mean Corpuscular Hemoglobin Concentration (MCHC) = MCH/MCV Conventional: g/dL S.I.: g/L	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
13.81 138.1	12.7–15.9 127–159	150 150	144–155 144–155	41 41	36.4–43.9 36.4–43.9	27.36 273.6	25.38–28.1 253.8–281
14.46 144.6	12.2–15.89 122–158.9	168 168	160–175 160–175	51.7 51.7	47.2–56.8 47.2–56.8	30.53 305.3	29.07–31.89 290.7–318.9
14.7 147	12.42–16.89 124.2–168.9	121.5 121.5	116–127 116–127	26.1 26.1	23.1–30.89 23.1–30.89	21.79 217.9	19.8–23.9 198–239
12.36 123.6	11.1–13.99 111–139.9	164.3 164.3	157–170 157–170	42 42	37.5–44.6 37.5–44.6	25.53 255.3	22.69–27.53 226.9–275.3
16.04 160.4	13.6–17.0 136–170	219 219	206–220 206–220	86.5 86.5	78.2–89.0 78.2–89.0	39.37 393.7	35.2–43.3 352–433
11.82 118.2	10.8–14.76 108–147.6	135.1 135.1	128–140 128–140	29.3 29.3	27.5–31.4 27.5–31.4	21.51 215.1	20.29–23.14 202.9–231.4
11.4 114	10.1–13.82 101–138.2	130.4 130.4	125–138 125–138	29 29	27.5–31.4 27.5–31.4	21.51 215.1	20.29–23.14 202.9–231.4
16.92 169.2	14–17.2 140–172	212 212	205–218 205–218	82.2 82.2	76.4–88.4 76.4–88.4	37.65 376.5	34.7–41.2 347–412
12.15 121.5	10.2–14.7 102–147	135.9 135.9	126–142 126–142	31 31	26.4–35.8 26.4–35.8	22.68 226.8	20.45–25.2 204.5–252
13.61 136.1	11.1–16.0 111–160	166.3 166.3	158–175 158–175	47.7 47.7	40.45–53.7 40.45–53.7	28.85 288.5	25.8–31.5 258–315
13.06 130.6	10.9–15.98 109–159.8	145.9 145.9	136–156 136–156	35.8 35.8	31.8–39.4 31.8–39.4	24.46 244.6	21.9–26.1 219–261
	7–18.6 70–186		100–139 100–139		25–48 25–48		20–34 200–340
	10.7–14.3 107–143		60–100 60–100		23–35 23–35		28–39 280–390
14.35 143.5	11.8–16.31 118–163.1	167.1 167.1	159–173 159–173	48.1 48.1	44.96–53.08 44.96–53.08	28.87 288.7	26.79–31.4 267.9–314
13.7 137	11.3–16.04 113–160.4	161.1 161.1	149–173 149–173	43.5 43.5	27.3–31.3 27.3–31.3	22.08 220.8	20.58–22.94 205.8–229.4

TABLE 3

Enzymes: Laboratory Reference Ranges for Selected Species[1–11]

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Lactate Dehydrogenase (U/L)		Creatine Kinase (U/L)	
		Mean	Range	Mean	Range
PSITTACIFORMES					
PSITTACIDAE					
Grey parrot (African grey) Genus *Psittacus* sp.	5571	248.2	154–378	303	140–411
Grey parrot (African grey, Congo) *Psittacus erithacus*	495	243.1	144–390	306.1	135–410
Timneh parrot (African grey, timneh) *Psittacus timneh*	219	238.5	144–402	286.3	126–420
Amazon Genus *Amazona* sp.	8375	230.4	160–368	257.1	117–425
Amazon, blue-fronted *Amazona aestiva*	828	228.3	158–366	22.5	130–417
Amazon, double yellow-headed *Amazona oratrix*	565	233.3	160–354	260.9	128–420
Amazon, lilac crown *Amazona finschi*	57	225.7	160–332	248.3	153–376
Amazon, mealy *Amazona farinosa*	54	228.8	159–381	278.5	208–348
Amazon, green-cheeked (Mexican red head) *Amazona viridigenalis*	206	246.1	145–388	2435.4	123–429
Amazon, orange-winged *Amazona amazonica*	240	251.6	148–451	278.2	120–455
Amazon, panama *Amazona ochrocephala panamensis*	67	233.5	160–387	318.6	157–458
Amazon, red-lored *Amazona autumnalis*	299	229.2	150–412	262.5	136–420
Amazon, yellow-naped *Amazona auropalliata*	1074	228.5	160–360	252.5	132–402
Amazon, yellow-crowned *Amazona ochrocephala*	36	197.6	171–265	391.8	260–490
Budgerigar *Melopsittacus undulatus*	1542	251.7	156–384	235	117–368
Caique *Pionites* sp.	30	192.3	147–270	249.3	124–384
Conure group *Aratinga and Pyrrhura* sp.	2911	292.1	216–408	262.4	153–408
Conure, blue-crowned *Aratinga acuticaudata*	93	221.6	148–380	256.3	120–344
Conure, dusky (Dusky-headed parakeet, Weddell's parakeet) *Aratinga weddellii*	29	220.1	114–328	202.6	108–375
Conure, gold-capped *Aratinga auricapillus*	36	250.1	175–387	284.9	200–384
Conure, jenday *Aratinga jandaya*	55	279.7	224–354	276.8	167–388

Aspartate Aminotransferase (U/L)		Alkaline Phosphatase (U/L)		Amylase (U/L)		Bile Acids (μmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
173.8	110–340	33.9	12–92	510.9	415–626	55.8	12–96
161.1	81–369	40.2	14–64	283.3	200–384	53	12–96
191.4	120–342	39.8	12–125	304.5	170–436	60.8	23–97
221	150–344	52	8–100	330.1	184–478	89	33–154
231.5	146–408	57.8	20–108			87	34–140
210.4	152–400	73.4	50–100	410.3	327–474	86.5	32–148
227.5	150–382	46.3	11–75			84.1	33–144
225.8	138–377			305	36–574	68	31–145
210.6	150–328	53.8	18–92			88.2	36–154
205.8	114–389	35	20–64	0	0–0	93.6	29–157
208.7	154–394	42.8	14–64	381	200–645	90.2	31–148
224.3	150–408	53.5	26–101	346.7	156–472	68.7	24–120
218.8	150–390	52.7	20–96	336.2	187–546	85.1	36–144
214.2	150–380					119	59–188
262.3	156–375	68.3	24–96	437.3	302–560	81.1	32–117
222.5	118–364			267	244–290	67.4	12–112
229.5	147–378	54.6	24–104	441.1	192–954	65.7	32–105
201.4	140–354					68	24–105
257.2	162–356					17	16–18
232.8	147–363					20.7	9–32
233.7	140–312					71	20–114

Continued

TABLE 3

Enzymes: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Lactate Dehydrogenase (U/L)		Creatine Kinase (U/L)	
		Mean	Range	Mean	Range
Conure, nanday *Aratinga nenday*	179	285.8	204–396	251.7	135–400
Conure, sun *Aratinga solstitialis*	57	257.7	188–392	280.1	153–372
Conure, green-cheeked *Pyrrhura molinae*	41	251.5	168–370	207.6	144–276
Conure, maroon-bellied (Red-bellied conure, brown-eared conure) *Pyrrhura frontalis*	28	281	204–372	201.5	111–491
Conure, cherry-headed (Red-masked parakeet) *Aratinga erythrogenys*	66	249.4	180–441	253.1	146–396
Conure, mitred *Psittacara mitratus*	74	212	140–360	193.2	136–311
Conure, half-moon (Orange-fronted parakeet) *Eupsittula canicularis*	29	222.2	104–368		
Conure, Patagonian *Cyanoliseus patagonus*	13			305.7	221–387
Conure, orange-fronted *Eupsittula canicularis*	13				
Eclectus parrot *Eclectus roratus*	415	275.3	198–386	264.3	132–410
Grey-cheeked parakeet (Fire-winged parakeet) *Brotogeris pyrrhoptera*	449	262.7	154–356	278.8	164–378
Lory (11 genera, 53 species)	130	194.6	124–302	295.6	178–396
Lovebird *Agapornis* sp.	650	277.1	225–354	250.7	160–392
Macaw group Mostly *Ara* and some *Anodorhynchus*	5338	135.3	70–220	214.7	88–361
Macaw, blue-and-gold (Blue-and-yellow macaw) *Ara ararauna*	1367	133.7	69–220	219.4	92–380
Macaw, green-winged (Red-and-green macaw) *Ara chloropterus*	347	139.9	72–224	220.9	96–368
Macaw, military *Ara militaris*	88	134.8	72–216	219.7	126–321
Macaw, scarlet (Red-and-yellow macaw) *Ara macao*	146	122.2	60–210	240.1	98–366
Macaw, severe (Chestnut-fronted macaw) *Ara severus*	104	128.9	66–210	225.7	123–356
Macaw, Hahn's (Red-shouldered macaw) *Diopsittaca nobilis nobilis*	51	184.8	132–260	292.8	242–380
Macaw, noble (Red-shouldered macaw) *Diopsittaca nobilis cumanensis*	22				

Aspartate Aminotransferase (U/L)		Alkaline Phosphatase (U/L)		Amylase (U/L)		Bile Acids (μmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
238.8	147–378	41	28–64			69.1	16–129
238.6	138–355					51.5	12–92
204	138–270					53.2	10–93
224.2	136–378						
215.9	132–385					59	32–94
189	118–342	40.4	12–78			51.7	12–96
207.2	120–324					64.2	28–138
178	120–360					47.2	15–93
219.9	144–339	72.2	32–111	622.2	562–684	70.7	30–110
324.6	189–388					53.7	81
257.5	141–369					57.3	20–97
214.5	130–360					56	12–90
121.5	65–168	50.3	12–100	421.2	239–564	48.9	7–100
118.9	64–168	60.2	32–100	407.8	239–516	52.8	27–86
116.9	62–168	59.9	30–106			44.5	15–78
123.3	76–166	41.9	17–88			66.2	24–130
120.1	74–177	30.1	16–58			68.3	21–100
129	72–170	41.7	12–76			54.3	25–82
184.1	133–216						
176.7	126–215						

Continued

TABLE 3

Enzymes: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Lactate Dehydrogenase (U/L) Mean	 Range	Creatine Kinase (U/L) Mean	 Range
Macaw, hyacinth *Anodorhynchus hyacinthinus*	149	136.3	90–208	280.2	138–369
Spix's macaw *Cyanopsitta spixii*	37	213 ± 116	104–669	536 ± 178	257–1088
Macaw, yellow-collared (Golden-collared macaw) *Primolius auricollis*	70	152	96–213	291.1	210–361
Parakeet, Alexandrine *Psittacula eupatria*	18				
Moustache parakeet (Rose-breasted parakeet) *Psittacula alexandri*	26	300	208–360		
Parakeet, ringneck (Rose-ringed parakeet) *Psittacula krameri*	46	213.9	150–318		
Parrot, Meyers *Poicephalus meyeri*	39				
Parrot, red-bellied *Poicephalus rufiventris*	14				
Parrot, Senegal *Poicephalus senegalus*	244	254.1	153–396	288	151–396
Parrot, thick-billed *Rhynchopsitta pachyrhyncha*	10	173	158–202	251.6	200–344
Parrotlet species Genus *Forpus* sp.	29				
Pionus species group Genus *Pionus* sp.	431	248.5	179–384	276.2	116–408
Pionus, blue-headed *Pionus menstruus*	26	225.3	138–384		
Pionus, white-capped *Pionus senilis*	49	228.5	157–354	219.6	106–365
Quaker parrot (Monk parakeet) *Myiopsitta monachus*	210	243	138–360	318.8	192–402
Rosella Genus *Platycercus* sp.	21	223.9	121–348		
CACATUIDAE					
Cockatiel *Nymphicus hollandicus*	9267	255	122–378	269.1	160–420
Cockatoo group Family Cacatuidae	5928	302.8	208–414	266.4	147–418
Cockatoo, bare-eyed (Little corella) *Cacatua sanguinea*	30	314.2	256–415	279.7	155–420
Cockatoo, Goffin's (Tanimbar corella) *Cacatua goffiniana*	298	329.9	198–452	281.8	144–439
Cockatoo, lesser sulphur-crested *Cacatua sulphurea*	17	285.6	173–398	281.1	157–408
Cockatoo, Major Mitchell (Leadbeater's cockatoo, pink cockatoo) *Lophochroa leadbeateri*	14	303.7	240–390	247.8	132–376

Aspartate Aminotransferase (U/L)		Alkaline Phosphatase (U/L)		Amylase (U/L)		Bile Acids (μmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
143.2	110–174	41.8	15–84			50.5	13–91
140 ± 28	87–212			257 ± 105	133–554	24 ± 12	8–64
129.4	104–150					57.9	21–93
252.7	159–303						
266.7	143–380						
178	120–360						
229.8	156–352						
245.8	176–316						
202.8	120–330					587	20–94
186.5	143–280	81.5	70–93			55	22–98
173.7	110–224						
222.3	135–358					47	15–92
241.8	148–342						
213.5	120–346						
248.6	130–380					55.3	21–90
176.3	116–258						
244.6	128–396	51.1	12–100	360.7	113–870	75.8	44–108
204.4	140–360	58.9	24–104	557.9	228–876	70	34–112
219	130–340	17.5	4–28			55	26–96
210.8	140–350	43.6	20–92	370.3	273–536	56	15–96
191.4	134–276	65	21–100	414.3	236–750	59.3	24–95
182.2	165–210	54	50–60			166	142–189

Continued

TABLE 3

Enzymes: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Lactate Dehydrogenase (U/L)		Creatine Kinase (U/L)	
		Mean	Range	Mean	Range
Cockatoo, Moluccan (Salmon-crested cockatoo) *Cacatua moluccensis*	772	304.6	192–456	272.1	148–404
Cockatoo, rose-breasted (Galah, roseate cockatoo, pink-and-grey cockatoo) *Eolophus roseicapillus*	92	320.5	161–395	228.9	120–395
Cockatoo, triton *Cacatua galerita triton*	44	269.6	118–374	296.3	164–396
Cockatoo, umbrella *Cacatua alba*	951	296.4	208–405	269.8	152–444
PASSERIFORMES					
Canary *Serinus canaria forma domestica*	300				
Mynah species Mostly *Acridotheres tristis*	44			336.6	250–414
GALLIFORMES					
Chicken, domestic *Gallus gallus domesticus*	133			578 ± 434	65–2340
Quail	60–150				
ANSERIFORMES					
Duck, domestic *Anas platyrhynchos domestica*	73	193.8	120–246	266.1	165–378
Swan *Cygnus* sp.	36	244.2	150–352	246.9	130–389
ACCIPITRIFORMES					
Combined eagle species	18	289.4	252–340	334.6	234–485
Bald eagle *Haliaeetus leucocephalus*				714 ± 421	190–1000
Golden eagle *Aquila chrysaetos*					
Common buzzard *Buteo buteo*	17–23			1219 ± 1268	20–5369
Hawk, red-tailed *Buteo jamaicensis*	13	339.8	230–398		
FALCONIFORMES					
Gyrfalcon *Falco rusticolus*	75–102				
STRUTHIONIFORMES					
Emu *Dromaius novaehollandiae*	98	778.1	318–1243	428.8	70–818
Ostrich *Struthio camelus*	140	970	408–1236	3702	800–6508
COLUMBIFORMES					
Dove *Streptopelia risoria* *Geopelia cuneata*	45	263.1	150–388	260.2	180–388
Pigeon *Columbia livia*	68–77			302 ± 195.7	151–888

Aspartate Aminotransferase (U/L)		Alkaline Phosphatase (U/L)		Amylase (U/L)		Bile Acids (µmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
189.3	136–366	60.2	21–104	542.9	228–780	66.9	30–110
264.8	150–344	65	48–82			128	101–148
162.4	120–208	75.3	54–100			67.5	24–98
204.1	140–376	68.5	38–106	632	264–876	74.5	38–114
224.4	132–351						
284.4	200–352					58.2	30–96
173 ± 43	82–311					14 ± 12	1–53
402 ± 13	243–562						
33.9	12–73					54.9	22–82
52.3	10–108	66.7	48–92			38.9	18–85
231.3	134–339						
352 ± 171	131–962						
345 ± 141	131–688	80 ± 344	26–1474				
242	110–392						
65.02 ± 49.87	18.88–184.46	508.79 ± 206.71	106.7–1053.32	53.92 ± 17.42	17.82–103.49	33.78 ± 19.49	4.64–96.44
227.2	80–380					18	2–34
447.9	226–547					21	2–30
217.7	111–372					71.8	40–106
111 ± 22.8	66–166						

Continued

TABLE 3

Enzymes: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Lactate Dehydrogenase (U/L)		Creatine Kinase (U/L)	
		Mean	Range	Mean	Range
STRIGIFORMES					
Strigiformes species	148	187 ± 367	6–3580	271 ± 653	0–3319
Owl, great-horned *Bubo virginianus*	18	282.8	142–383	286.7	252–376
PICIFORMES					
Toucan *Ramphastos* sp.	86	257.6	180–319		
PHOENICOPTERIFORMES					
Chilean flamingo *Phoenicopterus chilensis*	32	238 ± 126	91–583	541 ± 345	138–898
SPHENISCIFORMES					
African penguin *Spheniscus demersus*	108			419 ± 272	77–1554

Aspartate Aminotransferase (U/L)		Alkaline Phosphatase (U/L)		Amylase (U/L)		Bile Acids (μmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
236 ± 90	90–783			264 ± 444	33–4235		<35
196.7	104–344	39	8–102			23.5	8–54
243.3	141–340	43.3	14–88			54.4	16–86
74.7 ± 29.6	44.4–143.2	48.1 ± 15.9	138–898				
218 ± 90	90–627					9.53 ± 16.75	0.08–78.27

TABLE 4

Metabolic: Laboratory Reference Ranges for Selected Species[1–11]

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Uric Acid Conventional: (mg/dL) S.I.: (μmol/L) Mean	 Range	Glucose Conventional: (mg/dL) S.I.: (mmol/L) Mean	 Range
PSITTACIFORMES						
PSITTACIDAE						
Grey parrot (African grey) Genus *Psittacus* sp.	5571	Conventional S.I.	5.55 330.1	2.0–11 117.8–648.3	281.1 15.6	256–360 14.21–19.98
Grey parrot (African grey, Congo) *Psittacus erithacus*	495	Conventional S.I.	5.65 336.1	2.5–10 146.9–593.6	279.2 15.5	252–345 13.99–19.15
Timneh parrot (African grey, timneh) *Psittacus timneh*	219	Conventional S.I.	5.5 327.1	1.7–10 103.5–604.9	295.6 16.41	274–372 15.21–20.65
Amazon Genus *Amazona* sp.	8375	Conventional S.I.	5.1 303.3	2.2–10 132.0–618.0	286.6 15.91	246–378 13.66–20.98
Amazon, blue-fronted *Amazona aestiva*	828	Conventional S.I.	5.03 299.2	2.3–10 137.4–604.3	287.5 15.96	246–389 13.66–21.59
Amazon, double yellow-headed *Amazona oratrix*	565	Conventional S.I.	4.88 290.3	2.3–9.9 136.8–585.9	279.6 15.52	248–369 13.77–20.48
Amazon, lilac-crowned *Amazona finschi*	57	Conventional S.I.	5.65 336.1	2.6–10 155.2–612.6	292.1 16.21	245–353 13.60–19.60
Amazon, mealy *Amazona farinosa*	54	Conventional S.I.	5.27 313.5	2.3–12 138.6–699.5	264 14.65	201–337 11.16–18.71
Amazon, green-cheeked (Mexican red head) *Amazona viridigenalis*	206	Conventional S.I.	5.51 327.7	2.2–11 130.9–625.1	286.6 15.91	248–370 13.77–20.54
Amazon, orange-winged *Amazona amazonica*	240	Conventional S.I.	5.32 316.4	2.3–11 136.8–624.5	295.3 16.39	249–482 13.82–26.76
Amazon, panama *Amazona ochrocephala panamensis*	67	Conventional S.I.	5.58 331.9	2.8–10 164.8–606.1	272.4 15.12	248–330 13.77–18.32
Amazon, red-lored *Amazona autumnalis*	299	Conventional S.I.	5.23 311.1	2.2–9.9 130.9–587.1	285.9 15.87	250–388 13.88–21.54
Amazon, yellow-naped *Amazona auropalliata*	1074	Conventional S.I.	5.07 301.6	2.3–11 137.4–636.4	284.1 15.77	249–377 13.82–20.93
Amazon, yellow-crowned *Amazona ochrocephala*	36	Conventional S.I.	5.37 319.4	2.3–11 134.4–624.5	255.4 14.18	170–316 9.44–17.54
Budgerigar *Melopsittacus undulatus*	1542	Conventional S.I.	8.61 512.1	4.8–13 285.5–764.9	329.8 18.31	216–456 11.99–25.31
Caique *Pionites* sp.	30	Conventional S.I.	5.13 305.1	2.5–11 149.9–631.7	267.2 14.83	170–372 9.44–20.65
Conure group *Aratinga* and *Pyrrhura* sp.	2911	Conventional S.I.	6.58 391.4	3.0–11 178.4–665.0	291 16.15	216–418 11.99–23.20
Conure, blue-crowned *Aratinga acuticaudata*	93	Conventional S.I.	5.31 315.8	2.3–11 137.4–638.2	292.3 16.23	228–377 12.66–20.93
Conure, dusky (Dusky-headed parakeet, Weddell's parakeet) *Aratinga weddellii*	29	Conventional S.I.	7.52 447.3	3.1–11 186.2–644.2	273.5 15.18	213–350 11.82–19.43
Conure, gold-capped *Aratinga auricapillus*	36	Conventional S.I.	6.2 368.8	2.1–11 124.9–666.2	280.3 15.56	202–366 11.21–20.32
Conure, jenday *Aratinga jandaya*	55	Conventional S.I.	6.32 375.9	2.1–11 122.5–673.9	277.5 15.4	192–346 10.66–19.21

Cholesterol Conventional: (mg/dL) S.I.: (mmol/L)		Total Protein Conventional: (g/dL) S.I.: (g/L)		Albumin Conventional: (g/dL) S.I.: (g/L)		Total Calcium Conventional: (mg/dL) S.I.: (mmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
193.2 5	100–250 2.59–6.47	3.5 35.1	2.7–4.4 27–44	1.7 17	0.2–2.4 2–24	9.29 2.32	8.0–14 2.00–3.49
221 5.72	139–264 3.59–6.83	3.3 33	2.8–3.7 28–37	1.7 17	1.2–2.2 12–22	9.1 2.27	8.0–12 2.00–2.99
203.9 5.27	96–268 2.48–6.93	3.5 34.5	2.4–4.4 24–44	1.4 14	1.0–1.8 10–18	9.15 2.28	7.5–14.4 1.87–3.59
190.6 4.93	148–228 3.83–5.9	3.7 36.8	2.6–4.5 26–45	1.8 18	0.3–2.4 3–24	9.83 2.45	8.0–13.9 2.00–3.46
200 5.17	100–270 2.59–6.98	4.5 45	3.5–6.5 35–64.8	1.4 14	0.4–1.7 4–17	9.9 2.47	8.2–13.8 2.05–3.44
194.2 5.02	100–252 2.59–6.52	3.7 36.6	2.4–4.4 24–43.7	1.7 17	1.2–2.0 12–20	9.76 2.44	8.4–13.1 2.09–3.26
201.4 5.21	160–232 4.14–6.00	3.5 34.7	2.6–4.4 26–44	2.1 21	1.5–2.8 15–28	9.63 2.4	8.4–12.9 2.10–3.21
184.4 4.77	111–249 2.87–6.44	3.5 34.7	2.4–4.2 24–42	2.1 21	1.4–2.6 14–26	10.3 2.58	8.6–12.9 2.15–3.22
188.5 4.88	144–230 3.72–5.95	3.4 34.2	2.1–4.4 21–44	1.6 16	0.8–1.9 8–19	9.77 2.44	8.3–15 2.07–3.74
215.2 5.57	180–254 4.65–6.57	3.8 38.1	2.7–4.9 27–49	2 20	1.6–2.2 16–22	9.52 2.38	8.0–14.5 2.00–3.62
203.3 5.26	178–237 4.60–6.13	3.6 36.2	2.9–4.6 29–46	1.9 19	1.2–2.4 12–24	8.97 2.24	8.0–12 2.00–2.99
201.1 5.2	150–228 3.88–5.90	3.9 39	2.3–4.4 23–44.4	1.9 19	1.0–2.4 10–24	9.51 2.37	8.0–13.2 2.00–3.29
188.8 4.88	100–256 2.59–6.62	3.8 37.6	2.8–4.7 28–47	1.7 17	1.1–2.0 11–20	9.85 2.46	8.4–13.2 2.10–3.29
211.9 5.48	125–246 3.23–6.36	4 40.4	3.2–4.6 32–46	2.2 22	2.0–2.4 20–24	9.97 2.49	8.6–11.7 2.15–2.91
181.1 4.68	120–230 3.10–5.95	2.8 28.3	2.1–4.3 21–43	1.1 11	0.9–1.2 9–12	9.27 2.31	8.0–11.2 2.00–2.80
174 4.5	126–220 3.26–5.69	3.1 30.8	2.5–3.5 25–35	2.1 21	1.8–2.5 18–25	9.25 2.31	8.3–11.1 2.07–2.77
174.4 4.51	144–202 3.72–5.22	3.4 33.9	2.4–4.9 24–49	1.8 18	1.1–2.6 11–26	9.38 2.34	8.1–11.7 2.02–2.91
187.2 4.84	162–227 4.19–5.87	3.8 37.5	2.4–4.9 24–49	1.7 17	1.2–2.0 12–20	9.45 2.36	8.3–12 2.07–2.99
142.8 3.69	120–163 3.10–4.22					9.77 2.44	8.1–11.7 2.02–292
215.7 5.58	170–246 4.40–6.36	3.4 33.8	2.7–3.9 27–39			9.44 2.36	8.0–11.1 2.00–2.77
190.1 4.92	102–258 2.64–6.67					9.97 2.49	8.6–11.7 2.15–2.92

Continued

TABLE 4

Metabolic: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Uric Acid Conventional: (mg/dL) S.I.: (μmol/L) Mean	Uric Acid Range	Glucose Conventional: (mg/dL) S.I.: (mmol/L) Mean	Glucose Range
Conure, nanday *Aratinga nenday*	179	Conventional S.I.	6.48 385.4	2.3–11 137.4–660.2	290.8 16.14	231–393 12.82–21.82
Conure, sun *Aratinga solstitialis*	57	Conventional S.I.	6.66 396.1	2.2–12 131.5–688.8	273.4 15.17	167–380 9.27–21.09
Conure, green-cheeked *Pyrrhura molinae*	41	Conventional S.I.	8.11 482.4	3.2–12 189.7–742.3	288.6 16.02	215–395 11.93–21.93
Conure, maroon-bellied (Red-bellied conure, brown-eared conure) *Pyrrhura frontalis*	28	Conventional S.I.	6.16 366.4	3.0–12 178.4–738.7	300.6 16.69	261–340 14.49–18.87
Conure, cherry-headed (Red-masked parakeet) *Aratinga erythrogenys*	66	Conventional S.I.	5.8 345	2.1–11 124.9–678.7	290.8 16.14	220–384 12.21–21.32
Conure, mitred *Psittacara mitratus*	74	Conventional S.I.	5.94 353.3	2.2–13 130.9–772.1	292.6 16.24	228–370 12.66–20.54
Conure, half-moon (Orange-fronted parakeet) *Eupsittula canicularis*	29	Conventional S.I.	7.17 426.5	3.1–11 182.0–666.8	281.3 15.61	189–388 10.49–21.54
Conure, Patagonian *Cyanoliseus patagonus*	13	Conventional S.I.	5.53 328.9	2.3–11 133.8–678.1	310.7 17.25	236–389 13.10–21.59
Conure, orange-fronted *Eupsittula canicularis*	13	Conventional S.I.	6.6 392.6	3.6–11 213.5–647.1		
Eclectus parrot *Eclectus roratus*	415	Conventional S.I.	5.45 324.2	2.0–11 120.1–645.4	265.7 14.8	216–396 11.99–21.98
Grey-cheeked parakeet (Fire-winged parakeet) *Brotogeris pyrrhoptera*	449	Conventional S.I.	6.29 374.1	0.3–12 19.6–706.6	289.3 16.1	210–385 11.66–21.37
Lory (11 genera, 53 species)	130	Conventional S.I.	6.23 370.6	2.0–12 119.6–703.1	287.2 15.9	192–388 10.66–21.54
Lovebird *Agapornis* sp.	650	Conventional S.I.	7.44 442.5	3.3–11 193.9–634.1	305.7 17	220–390 12.21–21.65
Macaw group Mostly *Ara* and some *Anodorhynchus*	5338	Conventional S.I.	5.57 331.3	1.8–12 107.1–701.3	277.2 15.4	210–360 11.66–19.98
Macaw, blue-and-gold (Blue-and-yellow macaw) *Ara ararauna*	1367	Conventional S.I.	5.32 316.4	1.9–11 113.0–655.5	279.7 15.5	210–368 11.66–20.43
Macaw, green-winged (Red-and-green macaw) *Ara chloropterus*	347	Conventional S.I.	5.31 315.8	1.5–11 90.4–659.6	275.7 15.3	210–360 11.66–19.98
Macaw, military *Ara militaris*	88	Conventional S.I.	5.14 305.7	2.0–11 121.3–653.7	274.1 15.2	214–340 11.88–18.87
Macaw, scarlet (Red-and-yellow macaw) *Ara macao*	146	Conventional S.I.	5.15 306.3	2.1–11 121.9–642.4	265.7 14.8	210–333 11.66–18.48
Macaw, severe (Chestnut-fronted macaw) *Ara severus*	104	Conventional S.I.	6.51 387.2	2.2–12 129.7–701.9	278.5 15.5	220–366 12.21–20.32
Macaw, Hahn's (Red-shouldered macaw) *Diopsittaca nobilis nobilis*	51	Conventional S.I.	7.26 431.8	3.9–11 230.8–645.4	265.9 14.8	190–322 10.55–17.87

Cholesterol Conventional: (mg/dL) S.I.: (mmol/L)		Total Protein Conventional: (g/dL) S.I.: (g/L)		Albumin Conventional: (g/dL) S.I.: (g/L)		Total Calcium Conventional: (mg/dL) S.I.: (mmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
180.6 4.67	117–248 3.03–6.41	3 30.4	1.9–3.7 19–37	1.6 16	1.1–2.2 11–22	9.15 2.28	8.0–11 2.00–2.73
175.6 4.54	112–255 2.90–6.590	3.4 34.2	2.4–4.5 24–45	1.9 19	1.2–2.4 12–24	9.22 2.3	8.0–11.2 2.00–2.80
165 4.27	96–220 2.48–5.69	4.1 41	3.4–4.8 34–48	2.1 21	1.8–2.4 18–24	9.28 2.32	8.0–12.5 2.00–3.2
182.6 4.72	104–207 2.69–5.35					9.38 2.34	8.1–11.4 2.02–2.84
162.1 4.19	96–245 2.48–6.34	3.3 33.2	2.8–4.9 28–49	1.8 18	1.6–2.0 16–20	9.57 2.39	8.1–11 2.02–2.73
193.1 4.99	123–240 3.18–6.21	3 30.4	2.5–3.7 25–37.3	1.7 17	1.2–2.2 12–22	9.17 2.29	8.4–10.9 2.10–2.72
165.8 4.29	120–237 3.10–6.13					9.5 2.37	8.4–12.6 2.10–3.14
189.4 4.9	94–255 2.43–6.59	3 29.8	2.2–3.8 22–38	1.9 19	1.4–2.2 14–22	9.06 2.26	8.2–10.4 2.05–2.59
198.6 5.13	100–261 2.59–6.75	3.7 36.6	3.2–4.3 32–42.8	1.7 17	1.1–2.0 11–20	9.31 2.32	8.1–11.9 2.02–2.98
185.1 4.79	96–249 2.48–6.44					9.16 2.29	8.0–11.6 2.00–2.89
197.5 5.11	100–257 2.59–6.65	3.1 30.9	1.9–4.1 19–41	1.7 17	1.3–2.1 13–21	9.08 2.27	8.0–11.5 2.00–2.87
163.8 4.24	100–228 2.59–5.90	2.7 27.1	1.8–3.7 18–37	1.6 16	1.2–2.1 12–21	9.5 2.37	8.4–11.7 2.10–2.92
167.8 4.34	96–264 2.48–6.83	3.4 34.2	2.4–4.4 24–43.9	1.7 17	0.3–2.4 3–24	9.66 2.41	8.4–11.9 2.10–2.96
160.7 4.16	96–249 2.48–6.44	3.5 34.7	2.5–4.2 25–42	0.8 8	0.3–1.2 3–12	9.73 2.43	8.4–11.8 2.10–2.94
163.3 4.22	108–256 2.79–6.62	3.5 34.8	2.7–4.2 27–42	1.7 17	1.1–2.4 11–24	9.65 2.41	8.4–11.8 2.10–2.94
167.5 4.33	110–237 2.84–6.13	3.5 34.5	3–3.9 30–39	1.6 16	1.2–1.8 12–18	9.27 2.31	8.4–10.9 2.10–2.72
183.2 4.74	120–247 3.10–6.39	3.2 32.3	2.4–3.8 24–37.7	1.7 17	1.4–2.0 14–20	9.38 2.34	8.4–10.9 2.10–2.72
180.6 4.67	100–252 2.59–6.52	3.3 32.9	2.3–3.9 23–39	1.7 17	1.1–2.0 11–20	9.4 2.35	8.4–10.8 2.10–2.69
156.9 4.06	96–238 2.48–6.15					9.08 2.27	8.1–10.4 2.02–2.59

Continued

TABLE 4

Metabolic: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Uric Acid Conventional: (mg/dL) S.I.: (μmol/L) Mean	Uric Acid Range	Glucose Conventional: (mg/dL) S.I.: (mmol/L) Mean	Glucose Range
Macaw, noble (Red-shouldered macaw) *Diopsittaca nobilis cumanensis*	22	Conventional S.I.	6.38 379.5	2.2–10 129.7–622.2	270.8 15	231–300 12.82–16.65
Macaw, hyacinth *Anodorhynchus hyacinthinus*	149	Conventional S.I.	6.79 403.9	1.5–12 91.0–734.0	271.1 15.1	216–352 11.99–19.54
Macaw, Spix's *Cyanopsitta spixii*	37	Conventional S.I.	18 ± 7.8 315 ± 132	4.6–35.8 79–602	324 ± 50.45 18.0 ± 2.8	223.42–401.8 12.4–22.3
Macaw, yellow-collared (Golden-collared macaw) *Primolius auricollis*	70	Conventional S.I.	7.44 442.5	3.4–12 204.0–702.5	278.6 15.5	126–363 6.99–20.15
Parakeet, Alexandrine *Psittacula eupatria*	18	Conventional S.I.	7.16 425.9	4.3–11 257.5–640.0	254.3 14.1	192–308 10.66–17.10
Moustache parakeet (Rose-breasted parakeet) *Psittacula alexandri*	26	Conventional S.I.	6.81 405.1	3.1–11 185.0–624.5	284.6 15.8	225–348 12.49–19.32
Parakeet, ringneck (Rose-ringed parakeet) *Psittacula krameri*	46	Conventional S.I.	6.7 398.5	3.3–12 193.9–700.7	294.4 16.34	220–354 12.21–19.65
Parrot, Meyers *Poicephalus meyeri*	39	Conventional S.I.	5.72 340.2	2.1–11 26.1–672.1	255.5 14.2	212–294 11.7–16.32
Parrot, red-bellied *Poicephalus rufiventris*	14	Conventional S.I.	6.55 389.6	3.0–9.5 178.4–562.7	275.1 15.27	232–326 12.88–18.10
Parrot, Senegal *Poicephalus senegalus*	244	Conventional S.I.	5.95 353.9	2.3–12 134.4–707.8	288.7 16.02	256–354 14.21–19.65
Parrot, thick-billed *Rhynchopsitta pachyrhyncha*	10	Conventional S.I.	6.47 384.8	3.4–11 202.2–683.4	295 16.38	246–350 13.66–19.43
Parrotlet species Genus *Forpus* sp.	29	Conventional S.I.	8.23 489.5	4.1–12 244.5–694.1	308.7 17.13	252–384 13.99–21.32
Pionus species group Genus *Pionus* sp.	431	Conventional S.I.	5.91 351.5	2.0–12 120.7–685.2	279.5 15.51	212–368 11.77–20.43
Pionus, blue-headed *Pionus menstruus*	26	Conventional S.I.	6.11 363.4	3.1–12 184.4–710.8	276.4 15.34	200–348 11.10–19.32
Pionus, white-capped *Pionus senilis*	49	Conventional S.I.	5.62 334.3	2.1–11 126.7–662.0	282.5 15.68	221–348 12.27–19.32
Quaker parrot (Monk parakeet) *Myiopsitta monachus*	210	Conventional S.I.	6.8 404.5	2.2–12 133.2–712.6	287.9 15.98	210–395 11.66–21.93
Rosella Genus *Platycercus* sp.	21	Conventional S.I.	7.22 429.4	3.4–11 201.6–634.7	337.6 18.74	281–396 15.60–21.98
CACATUIDAE						
Cockatiel *Nymphicus hollandicus*	9267	Conventional S.I.	7.05 419.3	3.4–11 202.2–648.3	326.1 18.1	228–440 12.66–24.42
Cockatoo group Family Cacatuidae	5928	Conventional S.I.	6.69 397.9	8/3/2011 226.0–654.3	267.8 14.87	206–418 11.44–23.2
Cockatoo, bare-eyed (Little corella) *Cacatua sanguinea*	30	Conventional S.I.	5.95 353.9	3.0–11 176.1–638.2	265.5 14.74	220–378 12.21–20.98
Cockatoo, Goffin's (Tanimbar corella) *Cacatua goffiniana*	298	Conventional S.I.	6.85 407.4	3.5–11 208.2–639.4	274.2 15.22	220–412 12.21–22.87

Cholesterol Conventional: (mg/dL) S.I.: (mmol/L)		Total Protein Conventional: (g/dL) S.I.: (g/L)		Albumin Conventional: (g/dL) S.I.: (g/L)		Total Calcium Conventional: (mg/dL) S.I.: (mmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
132.9	96–216	3.1	2.6–3.4	1.7	1.5–2.2	9.37	8.2–11.4
3.44	2.48–5.59	30.5	26–34	17	15–22	2.34	2.05–2.84
351.3 ± 42	262.55–416.99	3.3 ± 0.29	2.7–4	2.1	1.48–2.82	7.21 ± 0.4	6.49–8.5
9.1 ± 1.1	6.8–10.8	33.6 ± 2.9	27.0–40.0	21.0 ± 3.1	14.8–28.2	1.88 ± 0.10	1.62–2.12
181.7	108–240	3.7	3.2–4.4	2	1.2–2.4	10.1	8.8–11.7
4.7	2.79–6.21	36.9	32–44	20	12–24	2.52	2.20–2.92
						9.13	8.0–10.9
						2.28	2.00–2.73
						9.3	8.3–10.8
						2.32	2.07–2.69
						9.21	8.4–10.4
						2.3	2.10–2.59
						9.34	8.2–10.4
						2.33	2.05–2.59
193.8	114–250	3.4	2.9–4.1	1.7	1.0–2.4	9.24	8.4–10.8
5.01	2.95–6.47	34.2	29–41.2	17	10–24	2.31	2.10–2.69
159.8	102–214	3	2.8–3.6	1.3	1.2–1.4	9.11	8.0–10.5
4.13	2.64–5.53	30.3	28–36	13	12–14	2.27	2.00–2.62
229	176–258	3.4	2.3–4.3	1.6	1.1–2.0	9.23	8.2–10.4
5.92	4.55–6.67	34.1	23–43	16	11–20	2.3	2.05–2.59
						8.93	8.0–10
						2.23	2.00–2.50
						8.94	8.2–10
						2.23	2.05–2.50
219.1	100–262	3.1	2.5–3.7	1.7	1.3–2.0	9.23	8.4–10.8
5.67	2.59–6.78	31.2	25–37	17	13–20	2.3	2.10–2.69
						9.27	8.0–10.9
						2.31	2.00–2.72
151.7	90–200	2.9	2.1–4.8	1.5	0.8–1.8	9.2	8.2–10.9
3.92	2.33–5.17	29	21–47.9	15	8–18	2.3	2.05–2.71
165.5	96–212	3.6	2.6–4.8	1.8	1.2–2.4	9.42	8.2–11.5
4.28	2.48–5.48	36.2	26–48.3	18	12–24	2.35	2.05–2.86
195.2	174–223	3.1	2.6–3.8	1.8	1.6–2.0	9.78	8.1–11.9
5.05	4.50–5.77	30.5	26–38	18	16–20	2.44	2.02–2.96
169.9	114–214	3.4	2.4–4.3	1.7	1.1–2.0	9.07	8.0–11.4
4.39	2.95–5.53	34	24–43	17	11–20	2.26	2.00–2.84

Continued

TABLE 4

Metabolic: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Uric Acid Conventional: (mg/dL) S.I.: (μmol/L) Mean	 Range	Glucose Conventional: (mg/dL) S.I.: (mmol/L) Mean	 Range
Cockatoo, lesser sulphur-crested *Cacatua sulphurea*	17	Conventional S.I.	6.76 402.1	2.1–11 123.7–638.2	245.2 13.61	138–304 7.66–16.88
Cockatoo, Major Mitchell (Leadbeater's cockatoo, pink cockatoo) *Lophochroa leadbeateri*	14	Conventional S.I.	7.4 440.2	2.2–11 130.9–672.1	301.9 16.76	242–392 13.43–21.76
Cockatoo, Moluccan (Salmon-crested cockatoo) *Cacatua moluccensis*	772	Conventional S.I.	6.05 359.9	3.2–11 190.3–632.9	259 14.38	198–424 10.99–23.54
Cockatoo, rose-breasted (Galah, roseate cockatoo, pink-and-grey cockatoo) *Eolophus roseicapillus*	92	Conventional S.I.	6.92 411.6	3.9–11 231.4–635.2	300 16.65	198–376 10.99–20.87
Cockatoo, triton *Cacatua galerita triton*	44	Conventional S.I.	5.55 330.1	2.2–11 127.9–654.9	270.3 15	200–345 11.10–19.15
Cockatoo, umbrella *Cacatua alba*	951	Conventional S.I.	6.67 396.7	3.5–11 208.2–648.3	269.4 14.95	214–426 11.88–23.65
PASSERIFORMES						
Canary *Serinus canaria forma domestica*	300	Conventional S.I.	8.68 516.3	4.1–13 246.2–750.0	297 16.49	160–360 8.88–19.98
Mynah species, Mostly *Acridotheres tristis*	44	Conventional S.I.	7.57 450.3	2.3–12 134.4–713.2	318.5 17.7	228–372 12.66–20.65
ANSERIFORMES						
Duck, domestic *Anas platyrhynchos domestica*	73	Conventional S.I.	6.39 380.1	2.0–12 119.0–701.3	206.5 11.46	127–319 7.05–17.71
Swan *Cygnus* sp.	36	Conventional S.I.	6.82 405.7	2.1–11 126.1–654.3	187.9 10.43	132–360 7.33–19.98
GALLIFORMES						
Chicken, domestic *Gallus gallus domesticus*	133	Conventional S.I.	4.2 249.8	0.9–8.9 53.53–529.37	206 11.35	174–239 9.6–13.17
Quail	60–150	Conventional S.I.	5.4 ± 0.23 320 ± 13.9	2.66–7 158–422	259.2 ± 10.8 14.4 ± 0.6	135–383.4 7.5–21.3
ACCIPITRIFORMES						
Combined eagle species Bald eagle *Haliaeetus leucocephalus* Golden eagle *Aquila chrysaetos*	18	Conventional S.I.	6.17 367	2.1–13 121.9–761.3	325.6 18.08	253–389 14.04–21.59
Common buzzard *Buteo buteo*	17–23	Conventional S.I.	48 ± 23.31 807 ± 392	8.8–93.74 149.0–1576.0	374.77 20.8 ± 4.6	147.75–482.88 8.2–26.8
Hawk, red-tailed *Buteo jamaicensis*	13	Conventional S.I.	11.1 658.4	6.3–18 375.9–1068.9	310.8 17.3	222–388 12.32–21.54
FALCONIFORMES						
Gyrfalcon *Falco rusticolus*	75–102	Conventional SI	2.1 ± 16.53 235.42 ± 278.66	0.9–50.2 15.32–844.31	372.43 ± 64.5 20.67 ± 3.58	271.35–580.72 15.06–32.23
STRUTHIONIFORMES						
Emu *Dromaius novaehollandiae*	98	Conventional S.I.	6.3 374.7	1.0–14 59.5–832.7	134.1 7.4	101–243 5.61–13.49

Cholesterol Conventional: (mg/dL) S.I.: (mmol/L)		Total Protein Conventional: (g/dL) S.I.: (g/L)		Albumin Conventional: (g/dL) S.I.: (g/L)		Total Calcium Conventional: (mg/dL) S.I.: (mmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
138.8	113–206	3.5	2.8–4.6	1.8	1.4–2.4	9.16	8.1–10.3
3.59	2.92–5.33	34.8	28–46	18	14–24	2.29	2.02–2.57
202	152–252	3.5	3–3.9			9.26	8.4–11.6
5.22	3.93–6.52	34.8	30–39			2.31	2.10–2.89
171.9	104–224	3.6	2.5–4.8	1.7	0.9–2.6	9.39	8.0–11.7
4.45	2.69–5.79	36.1	25–48	17	9–26	2.34	2.00–2.92
185	146–224	3.2	2.4–4.5	1.7	1.6–1.8	8.62	8.4–9.1
4.78	3.78–5.79	31.6	24–45	17	16–18	2.15	2.10–2.27
198.6	146–248	4	2.4–4.8	1.8	1.4–2.0	9.49	8.0–12
5.14	3.78–6.41	39.5	24–48	18	14–20	2.37	2.00–2.99
162.4	96–212	3.6	2.3–6.1	1.7	0.8–2.4	9.33	8.1–11
4.2	2.48–5.48	36	23–61	17	8–24	2.33	2.02–2.74
204.1	126–244	3.6	3–4.3	2.4	2.0–2.8	9.41	8.2–10.8
5.28	3.26–6.31	35.6	30–43	24	20–28	2.35	2.05–2.69
170.2	104–244	4.5	3.5–5.5	2	1.7–2.2	10	8.7–12.7
4.4	2.69–6.31	44.7	35–55	20	17–22	2.5	2.17–3.18
164.6	92–250	3.4	2.3–4.4	1.5	1.2–1.8	10.9	8.2–13.4
4.26	2.38–6.47	33.6	23–44	15	12–18	2.72	2.05–3.34
		5.4	3.9–7.0			17.9	≥10.9
		54	39–70			4.47	≥2.72
269 ± 9.1	158.3–382.24	3.7	2.9–4.3	1.5	1.3–1.8		
6.97 ± 0.24	4.1–9.9	36.5 ± 0.6	29.7–43.3	15.3 ± 0.2	12.6–18		
205.4	134–268					10.8	8.9–13.5
5.31	3.47–6.93					2.68	2.22–3.37
		4.6 ± 0.17	0.24 ± 9.8	2.3	1.48–2.95	11.94 ± 1.36	8.3–15.03
		46.6 ± 17.1	2.4–98.3	23.7 ± 4.1	14.8–29.5	2.98 ± 0.34	2.07–3.75
						10.6	8.4–13.8
						2.65	2.10–3.43
204.63 ± 37.84	97.3–302.7	2.7 ± 0.38	1.67–3.6	1.12	0.8–2	8.26 ± 0.76	5.53–11.02
5.3 ± 0.98	2.52–7.84	27.18 ± 3.8	16.7–36.62	11.28 ± 1.78	8.35–20.06	2.06 ± 0.19	1.38–2.75
122	68–170	3.9	3.4–4.4	1.7	1.2–2.4	11.1	8.8–12.5
3.15	1.76–4.40	39	34–44	17	12–24	2.77	2.20–3.12

Continued

TABLE 4

Metabolic: Laboratory Reference Ranges for Selected Species—cont'd

Species Common Name (Common Name Synonyms) Scientific Name	*N*	Units	Uric Acid Conventional: (mg/dL) S.I.: (μmol/L) Mean	 Range	Glucose Conventional: (mg/dL) S.I.: (mmol/L) Mean	 Range
Ostrich	140	Conventional	8.62	1.0–15	217	164–330
Struthio camelus		S.I.	512.7	59.5–892.2	12.1	9.10–18.32
COLUMBIFORMES						
Common pigeon	68–77	Conventional	3.04 ± 2.17	0.96–10	338.4 ± 20	311.4–390.6
Columba livia		S.I.	181 ± 129	57–595	18.8 ± 1.1	17.3–21.7
Dove	45	Conventional	7.25	2.5–12	299.9	164–386
Streptopelia risoria, Geopelia cuneata		S.I.	431.2	146.9–710.2	16.65	9.10–21.43
STRIGIFORMES						
Strigiformes	148		8.39 ± 4.94 499 ± 294	2.27–28.72 135–1708	340 ± 52.2 18.9 ± 2.9	192.6–547.2 10.7–30.4
Owl, great-horned	18	Conventional	8.52	3.1–12	319.1	237–390
Bubo virginianus		S.I.	506.8	183.2–703.6	17.7	13.16–21.65
PICIFORMES						
Toucan	86	Conventional	7.93	2.4–14	297.9	222–363
Ramphastos sp.		S.I.	471.7	144.5–832.7	16.53	12.32–20.15
PHOENICOPTERIFORMES						
Chilean flamingo	32	Conventional	6 ± 2.61	2.71–12	202.88 ± 40.36	134.95–274.05
Phoenicopterus chilensis		S.I.	357 ± 155	161–714	11.26 ± 2.24	7.49–15.21
SPHENISCIFORMES						
African penguin	108	Conventional	6.62 ± 3.72	1.66–22.92	212.25 ± 38.74	147.75–351.35
Spheniscus demersus		S.I.	394 ± 221.0	99–1363	11.78 ± 2.15	8.20–19.50

Cholesterol Conventional: (mg/dL) S.I.: (mmol/L)		Total Protein Conventional: (g/dL) S.I.: (g/L)		Albumin Conventional: (g/dL) S.I.: (g/L)		Total Calcium Conventional: (mg/dL) S.I.: (mmol/L)	
Mean	**Range**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
103 2.66	39–172 1.01–4.45	3.9 39	2.4–5.3 24–53	1.7 17	1.1–2.3 11–23	10.7 2.67	8.0–13.6 2.00–3.39
		3.1 31 ± 3.8	2–4 20–40	3 30 ± 2.6	2–3.5 20–35	10.02 ± 0.6 2.5 ± 0.15	8.8–12.6 2.19–3.14
219.6 5.68	124–257 3.21–6.65	2.9 28.6	2.1–3.7 21–37	1.4 14	1.2–1.6 12–16	9.41 2.35	8.0–12.6 2.00–3.14
220.08 ± 77 5.7 ± 2	127.41–837.84 3.30–21.70	3.9 ± 0.7 39 ± 7	2.4–8.2 24–82	3.1 31 ± 6	1.6–5.1 16–51	9.66 ± 1.56 2.41 ± 0.39	5.21–12.42 1.3–3.1
191.4 4.95	114–261 2.95–6.75	4 39.5	3.3–4.6 33–45.9	1.6 16	1.1–2.0 11–20	10.2 2.6	8.8–12.4 2.20–3.09
175.1 4.53	104–254 2.69–6.57	3.5 34.9	2.8–4.4 28–44.2	2.1 21	1.4–2.4 14–24	10.2 2.55	8.8–11.8 2.20–295
320.46 ± 88.8 8.3 ± 2.3	208.5–467.18 5.4–12.1	5.3 ± 0.5 53 ± 5	3.3–6.2 33–62	2.7 27.7 ± 4.4	1.8–3.5 18.2–35.9	12.42 ± 2 3.1 ± 0.5	10.02–17.23 2.5–4.3
206.9 ± 52 5.36 ± 1.36	69.5–339.77 1.80–8.80	5.9 ± 0.9 59 ± 9.61	3.4–8.3 34.0–83.0	1.9 19.3 ± 3.97	1.1–3.3 11.0–33.0	11.1 ± 3.29 2.77 ± 0.82	7.86–23.69 1.96–5.91

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APPENDIX 3

Normal Biological Data

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TABLE 1

Classification of Selected Avian Species Groups

FALCONIFORMES	
American kestrel	*Falco sparverius*
Common kestrel	*Falco tinnunculus*
Gyrfalcon	*Falco rusticolus*
Merlin	*Falco columbarius*
Peregrine falcon	*Falco peregrinus*
Prairie falcon	*Falco mexicanus*
ACCIPITRIFORMES	
Bald eagle	*Haliaeetus leucocephalus*
Black vulture	*Coragyps atratus*
Cooper's hawk	*Accipiter cooperii*
Eurasian buzzard	*Buteo buteo*
Eurasian goshawk	*Accipiter gentilis*
Eurasian (northern) sparrowhawk	*Accipiter nisus*
Golden eagle	*Aquila chrysaetos*
Harris's hawk	*Parabuteo unicinctus*
Osprey	*Pandion haliaetus*
Red-tailed hawk	*Buteo jamaicensis*
Sharp-shinned hawk	*Accipiter striatus*
Turkey vulture	*Cathartes aura*
STRIGIFORMES	
Barn owl	*Tyto alba*
Barred owl	*Strix varia*
Eurasian eagle owl	*Bubo bubo*
Great horned owl	*Bubo virginianus*
Screech owl, eastern	*Megascops asio*
Screech owl, western	*Megascops kennicottii*
Snowy owl	*Bubo scandiacus*
RATITES	
Cassowary	*Casuarius casuarius*
Emu	*Dromaius novaehollandiae*
North Island brown kiwi	*Apteryx mantelli*
Ostrich	*Struthio camelus*
Rhea	*Rhea americana*
PICIFORMES (RAMPHASTIDAE)	
Channel-billed toucan	*Ramphastos vitellinus*
Toco toucan	*Ramphastos toco*
CICONIIFORMES	
Great blue heron	*Ardea herodias*
PHOENICOPTERIFORMES	
American (Caribbean) flamingo	*Phoenicopterus ruber*
Phoenicopterus roseus	*greater flamingo*
GRUIFORMES	
Common crane	*Grus grus*
Sandhill crane	*Grus canadensis*
Whopping crane	*Grus americana*
OTIDIFORMES	
African houbara bustard	*Chlamydotis undulata*
Chlamydotis macqueenii	*Asian houbara bustard*
CORACIIFORMES	
Rufous hornbill	*Buceros hydrocorax*

TABLE 2

Biological and Physiologic Values of Selected Psittacine Birds[1–5]

Species	Body Weight (kg)	Lifespan (Years)		Temperature °C (°F)[a]
		Range	Maximum Reported	
Amazon parrot (*Amazona* spp.)	—	>50	80	—
Amazon, blue-crowned (mealy) *(Amazona farinosa guatemalae)*	540–700	4–8	—	—
Amazon, blue-fronted *(Amazona aestiva)*	361–485 (432)	—	—	—
Amazon, double yellow-headed *(A. oratrix)*	463–694 (568)	1.5 wild	19.3 wild	—
Amazon, green-cheeked (Mexican red head) *(A. viridigenalis)*	343–377 (360)	4–8 (6)	35	—
Amazon, yellow-crowned (yellow-fronted) *(Amazona ochrocephala)*	—	—	—	—
Amazon, yellow-naped *(A. auropalliata)*	476–795 (596)	—	—	—
Amazon, white-fronted (spectacled) *(A. albifrons)*	—	—	—	—
Bourke's parakeet *(Neopsephotus bourkii)*	35–50 (40)	—	—	—
Brotogeris spp.	—	—	—	—
Budgerigar *(Melopsittacus undulatus)*	26–35	Median: 5	18	—
Cockatiel *(Nymphicus hollandicus)*	80–100	Median: 10–12	36	—
Cockatoo (*Cacatua* spp.)	—	—	—	—
Cockatoo, medium	—	—	—	—
Cockatoo, large	—	—	—	—
Cockatoo, bare-eyed (little corella) *(C. sanguinea)*	331	—	—	—
Cockatoo, citron-crested *(C. sulphurea citrinocristata)*	—	—	—	—
Cockatoo, galah (rose-breasted) *(Eolophus roseicapillus)*	299	40–60	—	—
Cockatoo, greater sulphur-crested *(C.galerita galerita)*	806	—	—	—
Cockatoo, Leadbeater's (Major Mitchell) *(Lophochroa leadbeateri)*	381–474 (423)	—	—	—
Cockatoo, lesser sulphur-crested *(C. sulphurea)*	303	—	—	—
Cockatoo, Moluccan (salmon-crested) *(C. moluccensis)*	808	—	—	—
Cockatoo, palm *(Probosciger aterrimus)*	—	—	—	—
Cockatoo, triton *(C. galerita triton)*	559	—	—	—
Cockatoo, umbrella *(C. alba)*	552	—	—	—
Conure	80–100	—	—	—
Conure, small	—	—	—	—
Conure, large	—	—	—	—
Conure, blue-crowned *(Thectocercus acuticaudatus)*	—	—	—	—
Conure, golden (Queen of Bavaria) *(Guaruba guarouba)*	252–276 (262)	—	—	—

Heart Rate (bpm)	Respiratory Rate (BPM)[b]	Clutch Size	Incubation Period (Days)	Hatching Weight (g)	Fledging Age (Days)		Weaning Age (Days)	Sexual Maturity (Years)
					Parent-Raised	Hand-Raised		
—	15–45	—	24–29	—	45–60	90–120	75–90	4–6
—	—	—	—	—	—	—	—	—
—	—	—	26	—	—	—	—	—
—	—	—	28–29	—	—	—	—	—
—	—	—	26	—	—	—	—	—
—	—	—	28–29	—	—	—	—	—
—	—	—	28–29	—	—	—	—	—
—	—	—	24	—	—	—	—	—
—	—	—	—	—	—	—	—	—
—	—	—	22	—	—	—	—	—
—	60–75	4–6	16–18	—	22–26	30–40	30	6–9 months
—	40–50	4–5	18–20	—	32–38	47–52	42–49	6–12 months
—	15–40	—	—	—	—	—	—	—
—	—	—	—	—	45–60	90–120	75–100	3–4
—	—	—	—	—	60–80	120–150	95–120	5–6
—	—		23–24	—	—	—	—	—
—	—	—	25–26	—	—	—	—	—
—	—	—	22–24	—	45–55	90–120	89–90	12 months
—	—	—	24–25	—	—	—	—	—
—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—
—	—	—	28–29	—	—	—	—	—
—	—	—	28–30	—	—	—	—	—
—	—	—	27–28	—	—	—	—	—
—	—	—	28	—	—	—	—	—
—	—	—	—	—	35–40	45–70	60	2–3
—	40–50	—	—	—	—	—	—	—
—	30–45	—	—	—	—	—	—	—
—	—	—	23–24	—	—	—	—	—
—	—	—	—	—	—	—	—	—

Continued

TABLE 2

Biological and Physiologic Values of Selected Psittacine Birds—cont'd

Species	Body Weight (kg)	Lifespan (Years)		Temperature °C (°F)[a]
		Range	Maximum Reported	
Conure, nanday (black-hooded) *(Aratinga nenday)*	—	—	—	—
Conure, orange-fronted *(Eupsittula canicularis)*	—	—	—	—
Conure, Patagonian (burrowing parrot) *(Cyanoliseus patagonus)*	—	—	—	—
Conure, sun *(Aratinga solstitialis)*	120–130	Median: 19	30	—
Eclectus parrot *(Eclectus roratus)*	347–512 (432)	20–40	80	—
Grey parrot *(Psittacus erithacus)*	370–534 (407–454)	40–60 45 captivity 22.7 wild	48–60	41 (105.8)
Lory/lorikeet	—	20–30	—	—
Lorikeet, rainbow *(Trichoglossus haematodus)*	75–157	Median: 7	38	—
Lovebird	42–48	15–30	—	—
Lovebird, Fischer's *(Agapornis fischeri)*	42–58	Median: 8	32	—
Lovebird, masked (yellow-collared) *(A. personatus)*	43–47	Median: 6	24	—
Macaw	—	—	—	—
Macaw, large	—	75–100	—	—
Macaw, small	—	50–80	—	—
Macaw, blue-and-gold (blue-and-yellow) *(Ara ararauna)*	1021 995–1380 (1021)	21	48	—
Macaw, green-winged (red and green) *(A. chloropterus)*	1050–1708 (1179)	19	63	—
Macaw, hyacinth *(Anodorhynchus hyacinthinus)*	1197–1695 (1355)	22	54	—
Macaw, military *(Ara militaris)*	788–1134	Median: 20	54	—
Macaw, red-fronted *(A. rubrogenys)*	458	—	—	—
Macaw, scarlet *(A. macao)*	900–1490 (1103)	Median: 21	48	—
Pionus parrot (*Pionus* spp.)	—	—	—	—
Princess of Wales parakeet *(Polytelis alexandrae)*	100–129 (108)	—	—	—
Quaker (monk) parakeet *(Myiopsitta monachus)*	—	—	—	—
Red-crowned parakeet *(Cyanoramphus novaezelandiae)*	95–100	—	—	—
Red-rumped parakeet *(Psephotus haematonotus)*	60–69 (65)	—	—	—
Ring-necked parakeet *(Psittacula krameri)*	115	18–25	—	—
Senegal parrot *(Poicephalus senegalus)*	—	—	—	—

[a]Body temperature is not routinely measured during physical examination.
[b]Restraint can increase respiratory rate 1.5- to 2-fold.
bpm, Beats per minute; *BPM,* breaths per minute.

Heart Rate (bpm)	Respiratory Rate (BPM)[b]	Clutch Size	Incubation Period (Days)	Hatching Weight (g)	Fledging Age (Days)		Weaning Age (Days)	Sexual Maturity (Years)
					Parent-Raised	Hand-Raised		
—	—	—	21–23	—	—	—	—	—
—	—	—	30	—	—	—	—	—
—	—	—	24–25	—	—	—	—	—
—	—	4–5	23–28	—	49–56	—	—	2–3
—	—	—	26–28	—	72–80	120–150	100–110	4
340–600	24–45	2–5	26–30 Congo: 28 Timneh: 26	—	77–94	100–120	75–90	3–6
—	—	—	21–27	—	42–50	62–70	50–60	2
—	—	1–3	24–25	—	49–56	—	—	1
—	50–60	—	18–24	—	30–35	45–55	40–55	6–12 months
—	—	3–8	20–22	—	28–35	—	—	—
—	—	3–8	20–22	—	28–35	—	—	—
	20–25	—	—	—	—	—	—	—
—	—	—	26–28	—	70–80	120–150	95–120	5–7
—	—	—	23–26	—	45–60	90–120	75–90	4–6
—	—	1–3	25–27	—	84–98	—	—	3–4
—	—	2–3	26–28	—	84–98	—	—	4–5
—	—	2–3	29	31.6	98–112 (108)	—	—	5–6
—	—	2–4	28	—	91	—	—	3–4
—	—	—	—	—	—	—	—	—
—	—	1–4	28	—	84–91	—	—	4–6
—	—	—	25–26	—	—	—	—	—
—	—	—	—	—	—	—	—	—
—	—	—	23	—	—	—	—	—
—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—	—
—	—	—	22–23	—	40–45	55–65	—	3
—	—	—	24–25	—	—	—	—	—

TABLE 3

Biological and Physiologic Values of Selected Passerine Birds[1,6–38]

Family	Species	Body Weight (g)	Lifespan (Years)		Temperature °C (°F)[a]
			Range	Maximum Reported	
Bombycillidae	Cedar waxwing *(Bombycilla cedrorum)*	30–32	—	8	40–41.3 (104–106.3)
Cardinalidae	Northern cardinal *(Cardinalis cardinalis)*	33.6–64 (42–48) Males are slightly larger than females	Annual survival rates for adults have been estimated at 60%–65%	15	40–41.3 (104–106.3)
Corvidae	—	—	—	—	40–41.3 (104–106.3)
	American crow *(Corvus brachyrhynchos)*	316–620 (438–458) Males are slightly larger	—	16 wild 59 captive	—
	Blue jay *(Cyanocitta cristata)*	64.1–109 (85)	7 wild	17 wild 26.25 captive	—
Estrildidae	—	—	—	—	40–41.3 (104–106.3)
	Bengalese finch (Society or Japanese Mövchen) *(Lonchura striata domestica)*	7.5–16	4–7	—	—
	Common waxbill *(Estrilda astrild)*	7.5–16 (8.9)	4 wild	—	—
	Gouldian finch *(Erythrura gouldiae)*	7.5–35	—	—	—
	Java sparrow *(Padda oryzivora)*	7.5–35 (24–30)	—	—	—
	Zebra finch *(Taeniopygia guttata)*	7.5–16 (12)	4–7	—	—
Fringillidae	—	—	—	—	40–41.3 (104–106.3)
	American goldfinch *(Carduelis tristis)*	8.6–20.7 (12.6)	—	10	—
	Common canary (Island or Atlantic) *(Serinus canaria)*	12–30	5–15	20–25	—
	House finch *(Carpodacus mexicanus)*	16–27 (21)	—	11	—
	Purple finch *(C. purpureus)*	18–32 (25)	2 wild	14	—
Hirundinidae	Tree swallow *(Tachycineta bicolor)*	16–25 (20)	8–11 (2.7)	12	40–41.3 (104–106.3)
Mimidae	Northern mockingbird *(Mimus polyglottos)*	36.2–58 (43–49)	—	20 captivity	40–41.3 (104–106.3)
Paridae	Black-capped chickadee *(Parus atricapillus)*	8.2–14 (10.8)	2.5 wild	12	40–41.3 (104–106.3)
Passeridae	House sparrow *(Passer domesticus)*	20.1–34.5 (27.4–28)	—	13	40–41.3 (104–106.3)
Sturnidae	—	—	—	—	40–41.3 (104–106.3)
	European starling (common) *(Sturnus vulgaris)*	64–100	—	—	—
	Golden-breasted starling *(Cosmopsarus regius)*	80	—	—	—
	Hill mynah (myna) *(Gracula religiosa)*	180–270 (205)	12	20	—
Timaliidae	Pekin robin (red-billed leiothrix) *(Leiothrix lutea)*	26	—	—	40–41.3 (104–106.3)
Turdidae	American robin *(Turdus migratorius)*	64.8–85 (77)	2 (wild) Only 25% of fledged young survive to November	14	40–41.3 (104–106.3)[c]

[a]Potential diurnal variation of 2°C to 3°C.
[b]Pip-to-hatch interval is usually 24 hours or less.[33]
[c]Less than or equal to 38°C (100.4°F) is considered slightly hypothermic in the American robin.[37]
bpm, Beats per minute; *BPM,* breaths per minute.

Heart Rate (bpm)	Respiratory Rate (BPM/min)	Clutch Size	Incubation Period (Days)[b]	Hatching Weight (g)	Eyes Open (Days)	Fledging Age (Days)	Weaning Age (Days)	Sexual Maturity (Years)
—	—	2–6 (4–5)	10–17	—	Eyes remain closed in the first week	14–18 (15) fledging weight of 30 g	—	1
—	—	1–5 (3)	11–14	3.5	—	9–15	—	1
—	—	—	—	—	—	—	—	—
—	—	2–9	16–22	15.6–70	10–15 (110–210 g)	20–45	—	—
—	—	2–8	16–22	—	—	20–45	—	1
—	—	—	—	—	—	—	—	—
—	—	4–8	12–16	—	—	18–20	25–28	9–10 months
—	—	4–6	11–12	—	—	17–28	33–44	6–12 months
—	—	—	—	—	—	19–28	33–44	—
—	—	4–8	14–14	—	—	26–28 captivity 19–28 wild	33–44	—
—	—	4–6	12–16	—	—	14–28	33–44 25–28	9–10 months captivity 2.5–3 months wild
—	—	—	—	—	—	—	—	—
—	—	2–7	12–14	<1–2	—	12–18	2–7	12–14 months
265–325 resting	60–80 resting	3–5	12–14	—	—	11–17 (14)	21	<1
—	—	2–6 (4–5)	13–14	<1–2	—	12–19 (14)	—	<1
—	—	2–7	12–13	—	—	14–17	—	—
—	—	4–7	11–20 (14.5)	—	—	20–25	—	1
—	—	2–6 (4–5)	10–17	3.5–18	—	11–13 fledging weight of 32g	—	1
—	—	1–13 (6–8)	12–13	—	—	14–18	—	6 months
—	—	2–5	12–14	<1–2	—	13–17	—	—
—	—	—	—	—	—	—	—	—
—	92 resting 180 flight	4–5	10–17	5.5–30	—	20–23	—	—
—	—	—	—	—	—	—	—	—
—	—	2	14–15	—	—	30	60	2–3
—	—	—	—	—	—	—	—	—
—	—	3–5 (3.5)	10–17	5–35	—	13–15 fledging weight of 48–50 g	—	1

TABLE 4

Biological and Physiologic Values of Selected Raptors[1,39–47]

Order	Species	Body Weight (kg) Male	Female	Longevity (Years)
Accipitriformes	Bald eagle *(Haliaeetus leucocephalus)*	4.1–4.3	5.6–6.3	20–30 wild, 47 captivity
	Black vulture *(Coragyps atratus)*	2.0–2.7		25
	Cooper's hawk *(Accipiter cooperii)*	0.28–0.35	0.44–0.57	12 (20)
	Eurasian buzzard *(Buteo buteo)*	0.55–0.86	0.7–1.2	25
	Eurasian (northern) sparrowhawk *(Accipiter nisus)*	0.15–0.2	0.19–0.3	15
	Golden eagle *(Aquila chrysaetos)*	2.5–4.0	3.25–6.35	>38
	Harris's hawk *(Parabuteo unicinctus)*	0.7–1.0		20–30
	Osprey *(Pandion haliaetus)*	1.0–1.7	1.1–1.9	—
	Red-tailed hawk *(Buteo jamaicensis)*	0.69–1.15	1.0–1.35	21
	Sharp-shinned hawk *(Accipiter striatus)*	0.08–0.13	0.14–0.21	10
	Turkey vulture *(Cathartes aura)*	0.85–2.0		16
Falconiformes	American kestrel *(Falco sparverius)*	0.1–0.12	0.13–0.17	2–7
	Gyrfalcon *(Falco rusticolus)*	0.8–1.3	1.4–2.1	—
	Kestrel *(Falco tinnunculus)*	0.14–0.25	0.15–0.31	—
	Merlin *(F. columbarius)*	0.15–0.21	0.19–0.26	10–14
	Eurasian goshawk *(Accipiter gentilis)*	0.6–1.1	0.8–1.4	15–19
	Peregrine falcon *(Falco peregrinus)*	0.56–0.85	1.1–1.5	15–20
	Prairie falcon *(F. mexicanus)*	0.50–0.65	0.70–0.98	10–15
Strigiformes	Barn owl *(Tyto alba)*	0.44–0.47	0.49–0.57	>15
	Barred owl *(Strix varia)*	0.63	0.8	18
	Eurasian eagle owl *(Bubo bubo)*	1.5–2.8	1.8–4.2	50–60
	Great horned owl *(B. virginianus)*	1.3	1.7	20
	Screech owl, eastern *(Megascops asio)*	0.16–0.18	0.18–0.22	>14
	Screech owl, western *(M. kennicottii)*	0.13–0.21	0.16–0.25	>13
	Snowy owl *(Bubo scandiacus)*	1.4–2.5	1.6–2.9	28

bpm, Beats per minute; *BPM,* breaths per minute.

Heart Rate (bpm)	Respiratory Rate (BPM)	Clutch Size	Incubation Period (Days)	Interval Between Eggs (Days)	Fledging (Days)	Sexual Maturity (Years)
—	—	1–3	34–36	—	70–98	4–5
—	—	1–3	37–48	—	80–94	3
—	—	3–6	32–36	—	27–34	2
—	18	2–4	36–38	3	49–56	2–3
—	—	2–4	32–43	—	24–30	1–2
—	—	1–3	43–45	3–5	70	>5
—	—	2–5	32	2–3	43–49	>3
—	—	3–4	29–32	2–3	48–59	3
—	—	2–4	28–35	—	42–46	2
—	—	3–9+	30–33	2–3	24–27	—
—	9.2	1–3	30–40	—	66–88	—
—	—	3–7	29–31	2–3	30–31	1
—	—	3–5	34–36	—	49–56	—
—	—	3–6	27–29	1–2	27–30	1
—	—	2–7	28–32	2	30–35	2
—	—	4–6	35	2–3	35–42	>3
—	—	2–7	29–33	—	35–42	>3
—	—	3–4	29–31	—	35–42	1–2
—	—	2–9	30–31	2–3	70–75	1
—	—	2–4	28–33	—	42	—
—	—	2–4	34–36	2–3	90	2–3
—	—	1–4	30–37	—	42–63	1–3
—	—	2–6	21–30	—	—	—
—	—	3–8	32–35	—	28	—
—	—	1–3 (11)	38–41	—	16	2

TABLE 5

Biological and Physiologic Values of Selected Galliformes[1,48–59]

		Body Weight (kg)		Lifespan (Years)	
Family	**Species**	**Male**	**Female**	**Average**	**Maximum Reported**
Cracidae	Curassow			24 Great curassow	—
	Red-billed (southeast) curassow *(Crax blumenbachii)*	3.5		—	—
Meleagrididae	Turkey, wild *(Meleagris gallopavo)*	6.8–11	3.6–5.4	—	—
Numididae	Guineafowl (*Acryllium* spp.)	—		—	—
Phasianidae	Chicken, Greater prairie *(Tympanuchus cupido)*	—		—	—
	Jungle fowl, red *(Gallus gallus)*	2.58		30 captivity	—
	Domestic chicken *(G. gallus)*	2–12		25	—
	Francolin, Erckel's *(Francolinus erckelii)*	—		—	—
	Francolin, black *(Francolinus francolinus)*	—		—	—
	Peafowl, Indian *(Pavo cristatus)*	2.7–6 (4)		10–25 (16–18)	25 wild 23.2 captivity
	Pheasant, blood *(Ithaginis cruentus)*	—		—	—
	Pheasant, ring-necked or common *(Phasianus colchicus)*	1.263		10–18	—
	Quail, common *(Coturnix coturnix)*	0.070–0.155		11	—
	Quail, Japanese *(Coturnix japonica)*	0.090–0.115		6	—
	Quail, northern bobwhite *(Colinus virginianus)*	0.140–0.170		6	—
	Tragopan (*Tragopan* spp.)	—		—	—
Opisthocomidae	Hoatzin *(Opisthocomus hoazin)*	—	—	—	—
Tetraonidae	Grouse	—	—	—	—

[a]Best layers average between 250 and 280 eggs per year.
bpm, Beats per minute.

Clutch Size	Incubation Period (Days)	Fledging (Days)	Sexual Maturity (Years)
2	28–32	—	—
2, rarely 3	30.5	—	2–3
4–17 (8–15)	25–31 (28)	—	10 months–1 year
6–20	26–28	—	—
12	23–26	—	—
5–10 (4–6)	21	—	—
12–16[a]	—	—	12–20 weeks for most, 26 weeks in Jersey Giants
5–10	21–23	—	—
8–12	18–19	—	—
2–8 (max 12)	27–29	7–14	1–3
5–12	27–29	—	—
7–15 (10)	23–28 (24)	7–12	1
6–13	17–20	11	—
7–14	19–20 (17)	—	Male: 52 days Female: 63 days
—	23	14	1
2–6	28	—	—
2–3	—	—	—
2	28–32	—	—

TABLE 6

Biological and Physiologic Values of Selected Waterfowl[1,60–77]

		Body Weight (kg, mean)		Lifespan	
Family, Subfamily	**Species**	**Male**	**Female**	**Range**	**Maximum Reported**
Anatinae	American black duck *(Anas rubripes)*	(1080)		—	—
	American wigeon *(A. americana)*	538–649 (594)		—	—
	Blue-winged teal *(A. discors)*	314–387 (335–352)		—	—
	Cinnamon teal *(A. cyanoptera)*	310–355 (333)		—	—
	Eurasian teal (common) *(A. crecca)*	280		—	—
	Eurasian wigeon *(A. penelope)*	990–1160	700–800	10–15	35
	Gadwall *(A. strepera)*	587–697 (642)		—	—
	Mallard *(A. platyrhynchos)*	837–1047 (901–982)		10–15	—
	Mandarin duck *(Aix galericulata)*	428–693		10–15	—
	Mottled duck *(Anas fulvigula)*	734–860 (797)		—	—
	Muscovy duck *(Cairina moschata)*	2000–4000	1100–1500	10–15	—
	Mute swan *(Cygnus olor)*	12.2	8.9	25–30	—
	Northern pintail *(Anas acuta)*	454–1362 (612)	454–1135	—	—
	Northern shoveler *(A. clypeata)*	466–569 (518)		—	—
	Ringed teal[a] *(Callonetta leucophrys)*	360		—	—
	Wood duck *(Aix sponsa)*	548–610 (579–582)		—	—
Anhimidae	Screamers	3500–5000		—	—
Anserinae	Bar-headed goose *(Anser indicus)*	2000–3000 (2600)		15–20	—
	Black-necked swan *(Cygnus melancoryphus)*	7000		—	—
	Barnacle goose *(Branta leucopsis)*	1454–2020 (1737–2100)		—	—
		Body mass ↓ by ~25% during molt			
	Canada goose *(B. canadensis)*	—		—	—
	B. c. canadensis	3554–4628 (4091)		—	—
	B. c. occidentalis	2495–3206 (2851)		—	—
	B. c. minima	1095–1387 (1241)		—	—
	B. c. moffitti	3137–4305 (3721)		—	—
	Emperor goose *(Anser canagicus)*	1770–2233 (2002)		—	—
	Greater white-fronted goose *(A. albifrons)*	2661		—	—
	Greylag goose *(A. anser)*	2500–4100		—	—
	Hawaiian (nēnē) goose *(Branta sandvicensis)*	2200	1900	15–20	28 wild 42 captivity
		1800–2300			
	Pink-footed goose *(Anser brachyrhynchus)*	2600	2350	15–20	—
	Red-breasted goose *(Branta ruficollis)*	1300–1600	1150	15–20	—
	Snow goose *(Anser c. caerulescens)*	1710–2530 (2120)		—	—
	Trumpeter swan *(Cygnus buccinators)*	(9639)		—	—
	Tundra swan *(C. columbianus)*	(6300)		—	—
	Whooper swan *(C. cygnus)*	(8100)		—	—
Aythyinae	Canvasback *(Aythya valisineria)*	1001–1157 (1079)		—	—
	Common pochard *(A. ferina)*	—		—	—
	Lesser scaup *(A. affinis)*	629 (559–698)		—	—
	Redhead *(A. americana)*	852–1044 (948)		—	—
	Ring-necked duck *(A. collaris)*	594–666 (630)		—	—
	Tufted duck *(A. fuligula)*	589–740 (665)		10–15	
Dendrocygninae	Black-bellied whistling duck *(Dendrocygna autumnalis)*	716	705–756 (731)	—	—

Heart Rate (bpm)	Respiratory Rate (BPM)	Clutch Size	Incubation Period (Days)	Mean Body Weight of 1-Day-Old (g)	Fledging (Days)	Sexual Maturity (Years)
—	—	3–7	29–31	2–3	30–31	1
—	—	—	25	—	37–48	1
—	—	—	23	—	—	—
—	—	—	23	—	—	—
—	—	—	—	16.8	—	—
180–230	30–95	7–11	23–25	26.4	40–45	1
—	—	—	26	—	—	—
180–230	30–95	8–12	23–29	28.8	42–60	1
180–230	30–95	9–12	28–30	—	41–46	1–2
—	—	—	26	—	—	—
180–230	30–95	8–15	35	—	—	1
80–150	13–40	4–8	35–40	—	—	5
—	—	7–9	22–24	—	46–47	0.5–1
—	—	23	—	—	—	—
—	—	—	—	—	—	—
—	—	—	30	—	—	—
—	—	—	—	—	—	—
80–150 (104 ± 7)	13–40	4–6	27–30	—	55–60	2–3
—	—	—	—	—	—	—
113 ± 11	—	—	24	—	—	—
—	—	4–10	25–30	—	40–73	2–3
—	—	—	28	—	—	—
—	—	—	27	—	—	—
—	—	—	26	—	—	—
—	—	—	28	—	—	—
—	—	—	25	—	—	—
—	—	—	—	—	—	—
—	—	3–12 (4–6)	27–28	—	—	2–3
80–150	13–40	1–5 (3)	29–31	—	3 months	2–3
80–150	13–40	3–5	26–27	—	—	2
80–150	13–40	3–7	23–25	—	—	2
—	—	—	23	—	—	—
—	—	—	—	—	—	—
—	—	—	—	—	—	—
—	—	—	—	—	—	—
—	—	—	24	—	—	—
—	—	—	—	40.1	—	—
—	—	—	—	—	—	—
—	—	—	26	—	—	—
—	—	—	26	—	—	—
180–230	30–95	6–14	23–25	34.1	—	1
—	—	—	31	—	—	—

Continued

TABLE 6

Biological and Physiologic Values of Selected Waterfowl—cont'd

Family, Subfamily	Species	Body Weight (kg, mean) Male	Body Weight (kg, mean) Female	Lifespan Range	Lifespan Maximum Reported
Merginae	Common eider *(Somateria mollissima)*	2250	2120	10–15	—
		850–3025 (1800)			
	Common golden eye *(Bucephala clangula americana)*	645–687 (666)		10–15	—
	Common merganser *(Mergus merganser)*	—	1076	—	—
	Hooded merganser *(M. cucullatus)*	521–536 (529)		—	—
	Red-breasted merganser *(M. serrator)*	—		—	—
	Velvet scoter *(Melanitta fusca fusca)*	—	1316	—	—
	White-winged scoter *(M. f. deglandi)*	1091–1440 (1204–1326)		—	—
Oxyurinae	Maccoa duck *(Oxyura maccoa)*	673–761 (717)		—	—
	Ruddy duck *(O. Jamaicensis)*	508–619 (564)		—	—
Tadorninae	Common shelduck *(Tadorna tadorna)*	—	960	—	—
	Egyptian goose *(Alopochen aegyptiaca)*	1500–2250		—	—

[a]Sometimes placed in subfamily Tadorninae.
bpm, Beats per minute; *BPM,* breaths per minute.

Heart Rate (bpm)	Respiratory Rate (BPM)	Clutch Size	Incubation Period (Days)	Mean Body Weight of 1-Day-Old (g)	Fledging (Days)	Sexual Maturity (Years)
180–230	30–95	3–6	25–30 (26)	61.4	60–75	1–2
180–230	30–95	9–11	27–32 (31)	32.4	—	1
—	—	—	—	46.2	—	—
—	—	—	—	—	—	—
—		—	—	46.2	—	—
—	—	—	—	54.7	—	—
—	—	—	28	—	—	—
—	—	—	25	—	—	—
—	—	—	24	—	—	—
—	—	—	—	—	—	—
—	—	5–12	28–30	—	70	2

TABLE 7

Biological and Physiologic Values of Selected Pigeons and Doves[78–81]

Species	Body Weight (g)		Lifespan (Years)		Clutch Size	Incubation Period (Days)	Fledging (Days)	Sexual Maturity (Years)
	Male	Female	Average	Maximum Reported				
African collared dove *(Streptopelia roseogrisea)*	150–160		—	—	—	—	—	—
Common ground dove *(Columbina passerina)*	30		4–8	—	—	12 to >14	18	1
European turtle dove *(Streptopelia turtur)*	85–186		—	—	1–2	13–14	20	—
Mourning dove *(Zenaida macroura)*	96–170 (120)		1.5 wild	19.3 wild	2	13–15	12–15	85 days
Pigeon (rock dove) *(Columba livia)*	240–300 (358.7)		4–8 (6)	35	2	16–19	28–35	—
Pink pigeon *(C. mayeri)*	240–410	213–369	—	—	—	—	—	—
Southern crowned pigeon *(Goura scheepmakeri)*	2250		—	—	1	—	—	—
Spotted dove *(Streptopelia chinensis)*	146–198		—	—	2 (rarely 3)	14–16	14–15	—

TABLE 8

Biological and Physiologic Values of Other Selected Species[1,8,13,31,40,82–99]

Taxonomic Group	Species	Body Weight (kg)		Longevity (Years)		Body Temperature, °C (°F)
		Male	Female	Reported	Maximum	
Apodiformes		—	—	—	—	—
Subfamily Apodidae		—	—	—	—	—
	Alpine swift *(Tachymarptis melba)*	—		—	26	—
	Chimney swift *(Chaetura pelagica)*	0.017–0.030 (0.023)		—	14	40–41 (104–105.8), potential diurnal variation of 2–3°C
	Common swift *(Apus apus)*	—		—	21	—
Tribe Chaeturini	Spinetails	—		—	—	—
Trochilidae	Most hummingbirds	0.003–0.007		5–12 (5–8)	17 captivity	39–45 (102.2–113) As low as 14 (57.2) during torpor
	Reddish hermits *(Phaethornis ruber)*	<0.002		—	—	—
	Bee hummingbirds *(Mellisuga helenae)*	<0.002		—	—	—
	Giant hummingbirds *(Patagonia gigas)*	0.019–0.021		—	—	—
Ciconiiformes	Great blue heron *(Ardea herodias)*	2.1–2.5		15	24	—
Phoenicopteriformes	American flamingo (Caribbean) *(Phoenicopterus ruber)*	2.8	2.2	—	>60 captivity	—
Coraciiformes	Rufous hornbill *(Buceros hydrocorax)*	—	0.75	—	—	—
Gruiformes	Sandhill crane *(Grus canadensis)*	2.7–5.4		20–30	—	—
Otidiformes	Houbara bustard *(Chlamydotis undulata)*	2.2	1.2	10–15	—	—
Pelecaniformes		—	—	—	—	—
Anhingidae	Anhinga *(Anhinga anhinga)*	1.08–1.35		11.9 wild	16.4 wild	—
Fregatidae	Frigatebird, magnificent *(Fregata magnificens)*	1.2–1.815		20–30 estimated	—	—
Phalacrocoracidae	Cormorants	—		—	—	—
	Cormorant, double-crested *(Phalacrocorax auritus)*	1.2–2.5 (2.0) Males slightly larger		—	—	—
	Cormorant, guanay *(P. bougainvillii)*	1.55–3.2		—	—	—
Pelecanidae	Pelicans	Up to 15		—	—	—
	Pelican, American white *(Pelecanus erythrorhynchos)*	6.18–8.0 4.54–9.0	4.21–8.0	—	26.4 wild	—
	Pelican, Australian *(P. conspicillatus)*	4.4–6.8		15–25	50 captivity	—
	Pelican, brown *(P. occidentalis)*	2.05–5		27.83	43 wild	—
	Pelican, pink-backed *(P. rufescens)*	3.86–5.55	3.42–4.57	—	—	—
Phaethontidae	Tropicbirds	—		—	—	—
	Tropicbird, red-tailed *(Phaethon rubricauda)*	0.64–1.1		—	—	—

Heart Rate (bpm)	Respiratory Rate (BPM)	Clutch Size[a]	Incubation Period (Days)	Birth Weight (g)	Weaning Age	Fledging Age (Days)	Sexual Maturity (Years)
—	—	—	14–32	—	—	—	—
—	—	1–7 (2–3)	—	—	—	—	—
—	—	—	—	—	—	—	—
—	—	2–7	16–21	1.0–1.5	—	28–30	—
—	—	1	—	—	—	—	—
—	—	4–5	—		—		—
500–600 resting >1000 active <50, down to 30 torpor	13–40 resting 30–95 active	2	13–19	—	—	20–35	—
—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—
—	—	2	27–29	—	—	56–60	1.8
—	—	3–5 (2–7)	27–31	—	28–42 d	63–91	3–5
—	—	2–3	—	—	—	119–136	—
—	—	2 (1–3)	28–31	—	Precocial	67–75	3–6 (wild)
—	—	2–3	21–22	—	—	35–40	1–3
—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—
—	—	1–7 (3)	25–28	—	—	21–28	2
—	—	2–4	24–31	—	—	—	—
—	—	2	41	—	—	—	—
—	—	—	—	—	—	—	—
—	—	2–4	28–35	—	—	—	3–4
—	—	2	30	—	—	70–77	3
—	—	1-3 (2)	32–35 (32)	—	—	60	3–4
—	—	2-3 (3)	29–30	—	—	77	Male: 2–4; Female: 3–4
—	—	—	—	—	—	—	—
—	—	1	40–46	—	—	—	4
—	—	—	—	—	—	—	—

Continued

TABLE 8

Biological and Physiologic Values of Other Selected Species—cont'd

Taxonomic Group	Species	Body Weight (kg)		Longevity (Years)		Body Temperature, °C (°F)
		Male	Female	Reported	Maximum	
Sulidae	Boobies	—		—	—	—
	Booby, blue-footed *(Sula nebouxii)*	1.53		18 captivity	—	—
	Booby, red-footed *(S. sula)*	0.85–1.0		—	—	—
	Gannet, northern	2.932	3.067	—	21 wild	—
	(Morus bassanus)	2.2–3.6		—	—	—
Piciformes (Ramphastidae)	Channel-billed toucan *(Ramphastos vitellinus)*	0.6–0.7		20	—	—
	Toco toucan *(Ramphastos toco)*	0.59–0.76		18–20	—	—
Ratites	Emu *(Dromaius novaehollandiae)*	30–55		19	30	—
	Ostrich *(Struthio camelus)*	120–160		50	—	—
	Rhea *(Rhea americana)*	25		—	—	—

[a]Forty-eight-hour interval between egg laying.
[b]Under isoflurane anesthesia.
bpm, Beats per minute; *BPM,* breaths per minute.

Heart Rate (bpm)	Respiratory Rate (BPM)	Clutch Size[a]	Incubation Period (Days)	Birth Weight (g)	Weaning Age	Fledging Age (Days)	Sexual Maturity (Years)
—	—	1–3	40–45	—	—	—	1–6
—	—	2–6 (4)	25–30	—	—	42	—
—	—	—	—	—	—	—	—
—	—	1	42–50	—	—	120–200	3
	—	—	—	—	—	—	—
—	—	2–4 (5)	15–16	—	—	—	1
—	—	2–4	15–18	—	—	42–56	3–4
42–76 hours[b]	4–17 hours[b]	5–15	50–56	—	Precocial	—	3–5
60–72 hours[b]	6–12	10 (20)	41–43	—	Precocial	—	4
—	8.5	—	36–41	—	Precocial	—	1.5–2

TABLE 9

Values Reported for Selected Ophthalmic Diagnostic Tests in Avian Species[1,100–116]

Species	Ultrasound (mm ± STD)[a]	Intraocular Pressure (mm Hg)		Schirmer Tear Test (mm/Minute)	Phenol Red Thread Test (mm/15 Seconds ± STD)
		Applanation Tonometry	Rebound Tonometry		
American flamingo *(Phoenicopterus ruber)*	—	16.1 ± 4.2	9.5 ± 1.7	12.3 ± 4.5	24.2 ± 4.4
Axial globe length	13.8 ± 0.16	—	—	—	—
Anterior chamber depth	1.75 ± 0.05	—	—	—	—
Lens thickness	4.6 ± 0.06	—	—	—	—
Vitreous body depth	6.95 ± 0.1	—	—	—	—
Pecten dimensions	5.1 ± 0.38 length 2.2 ± 0.14 width	—	—	—	—
Bald eagle *(Haliaeetus leucocephalus)*	—	20.6 ± 2	—	14 ± 2	—
Barn owl *(Tyto alba)*	—	—	10.8 ± 3.8	—	—
Blue-fronted Amazon[b] *(Amazona aestiva)*	—	—	—	—	—
D1	1.7 ± 0.3	—	—	—	—
D2	3.5 ± 0.2	—	—	—	—
D3	7.3 ± 0.4	—	—	—	—
Axial length	12.6 ± 0.6	—	—	—	—
Common buzzard *(Buteo buteo)*	—	17.2 ± 3.53	26.9 ± 7.0	12.47 ± 2.66	—
Chicken, 3-week-old	—	—	17.5 ± 0.1	—	—
Golden eagle *(Aquila chrysaetos)*	—	21.5 ± 3	—	—	—
Great horned owl *(Bubo virginianus)*	—	10.8 ± 3.6	—	—	—
Eurasian eagle owl *(Bubo bubo)*	—	—	10.5 ± 1.6	—	—
Eurasian sparrowhawk *(Accipiter nisus)*	—	—	15.5 ± 2.5	—	—
Japanese quail (*Coturnix coturnix japonica*)	—	—	5.8 ± 0.89	—	17.5 ± 4.17
Axial globe length	7.8 ± 0.17	—	—	—	—
Anterior chamber depth	1 ± 0.13	—	—	—	—
Lens thickness	2.4 ± 0.09	—	—	—	—
Vitreous body depth	4.4 ± 0.14	—	—	—	—
Pecten dimensions	3.5 ± 0.18	—	—	—	—
Falco sp.	—	—	—	—	30.2 ± 4.6
Kestrel *(Falco tinnunculus)*	—	8.53 ± 1.59	9.8 ± 2.5	6.2 ± 3.67	—
Humboldt penguin *(Spheniscus humboldti)*	—	—	28 ± 9	9 ± 4	—
Large psittacines[c]	—	—	—	—	19.1 ± 3.3–25.5 ± 6.3 (OS) 19.8 ± 4.3–28.2 ± 6.3 (OD)
Long-eared owl *(Asio otus)*	—	—	7.8 ± 3.2	—	—
Eurasian goshawk *(Accipiter gentilis)*	—	—	18.3 ± 3.8	—	—
Brown pelican *(Pelecanus occidentalis)*	—	—	10.86 ± 1.61	5.45 ± 1.88	—
Axial globe length	20.70 ± 0.62	—	—	—	—
Anterior chamber depth	3.38 ± 0.19	—	—	—	—
Lens thickness	5.19 ± 0.23	—	—	—	—
Vitreous body depth	12.15 ± 0.53	—	—	—	—
Peregrine falcon *(Falco peregrinus)*	—	—	12.7 ± 5.8	—	—
Pigeon	—	—	19.5 ± 4.4	—	—
Red kite *(Milvus milvus)*	—	—	13.0 ± 5.5	—	—
Red-tailed hawk *(Buteo jamaicensis)*	—	20.6 ± 3.4	—	—	—
Swainson's hawk *(Buteo swainsoni)*	—	20.8 ± 2.3	—	—	—
Tawny owl *(Strix aluco)*	—	11.21 ± 3.12	9.4 ± 4.1	3.12 ± 1.92	—
Juvenile	—	—	8.7 ± 4.9	7.2	—
Adult	—	9.4 ± 1.8	11.1 ± 3.1	4.3	—
White-tailed sea eagle *(Haliaeetus albicilla)*	—	—	26.9 ± 5.8	—	—

[a]Mean values reported.
[b]Transpalpebral ocular ultrasound examinations were performed on 32 blue-fronted Amazon parrots with no history of ocular disease; measurements were taken in sagittal planes using a 10-MHz linear probe without a standoff pad; D1 is the distance between the cornea and the anterior lens capsule; D2 is the distance between the anterior and posterior lens capsule; D3 is the distance between the posterior lens capsule and the optic papilla.
[c]Group of large parrots including 22 macaws, 17 cockatoos, 10 Amazon parrots, 1 grey parrot, 1 eclectus, and 1 pionus.
OD, Right eye; *OS*, left eye; *STD*, standard deviation.

TABLE 10

Approximate Resting Respiratory Rates of Selected Avian Species and by Weight[109–111]

Species	Respiratory Rate (BPM)[a]
Finch	90–110
Canary	60–80
Budgerigar	60–75
Lovebird	50–60
Cockatiel	40–50
Small conure	40–50
Large conure	30–45
Toucan	15–45
Amazon parrot	15–45
Cockatoo	15–40
Macaw	20–25
Mallard	30–95
Raptor	10–20
Mute swan	13–40
Weight (g)	
100	40–52
200	35–50
300	30–45
400	25–30
500	20–30
1000	15–20

[a]Restraint can increase respiratory rate by 1.5× to 2× the resting rate.
BPM, Breaths per minute

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APPENDIX 4

Chick Growth Data

Brian L. Speer and Yvonne R. A. van Zeeland

A. Developmental Parameters of Selected Psittacines

Species	Day at Which Eyes First Open	Day at Which First Feathers Emerge	First Day of Perching	Last Days Unweaned
AMAZON PARROTS				
Blue-fronted *(Amazona aestiva)*	14–21	24	38–45	70–90
Blue-crowned *(Amazona farinosa)*	10–21	21	35–40	70–90
Double yellow-headed *(Amazona oratrix)*	14–21	24	38–45	70–90
Lilac-crowned *(Amazona finschi)*	10–21	21	35–40	70–90
Orange-winged *(Amazona amazonica)*	10–21	21	35–40	70–90
Red-lored *(Amazona autumnalis)*	10–21	21	35–40	70–90
White-fronted *(Amazona albifrons)*	10–21	21	35–40	70–90
Yellow-crowned *(Amazona ochrocephala)*	10–21	21	35–40	70–90
Yellow-naped *(Amazona auropalliata)*	14–21	24	38–45	70–90
COCKATOOS				
Bare-eyed *(Cacatua sanguinea)*	7–14	18–21	38–45	70–90
Citron-crested *(Cacatua sulphurea citrinocristata)*	7–14	18–21	38–45	70–90
Goffin's *(Cacatua goffini)*	7–14	18–21	38–45	70–90
Major Mitchell's *(Lophochroa leadbeateri)*	7–14	21	40–60	80–110
Moluccan *(Cacatua moluccensis)*	14–21	21	50–65	80–110
Red-vented *(Cacatua haematuropygia)*	7–14	21	38–45	70–90
Rose-breasted *(Eolophus roseicapilla)*	7–14	21	38–45	55–65
Slender-billed *(Cacatua tenuirostris)*	11–14	20	40–45	80–110
Triton *(Cacatua galerita triton)*	14–21	21	38–45	80–110
Umbrella *(Cacatua alba)*	14–21	21	50–65	80–110
MACAWS				
Blue-and-gold *(Ara ararauna)*	14–21	30–35	60–75	90–120
Buffon's *(Ara ambiguus)*	14–21	35	60–75	90–120
Green-winged *(Ara chloropterus)*	14–21	35	60–75	90–120
Hahn's *(Diopsittaca nobilis)*	14–21	35	60–75	90–120
Harlequin	14–21	35	60–75	90–120
Military *(Ara militaris)*	14–21	35	60–75	90
Red-fronted *(Ara rubrogenys)*	12–21	28–30	55–70	85–120
Scarlet *(Ara macao)*	14–21	35	60–75	90–120
Yellow-collared *(Primolius auricollis)*	10–18	21	45–55	70–90
OTHER PARROTS				
Amboina king *(Alisterus amboinensis)*	7–14	21–28	35–40	60–80
Blue-headed Pionus *(Pionus menstruus)*	10–15	20–23	40–45	
Eclectus *(Eclectus roratus)*	11–18	22–28	50–60	75–95
Grey parrot *(Psittacus erithacus)*	7–16	28	50–60	80–100
PARAKEETS				
Barraband *(Polytelis swainsonii)*	6–14	14	30–40	55–75
Barnard *(Barnardius barnardi)*	6–14	14	30–40	55–75
Princess of Wales *(Polytelis alexandrae)*	6–14	14	30–40	55–75
Rock pebbler *(Polytelis anthopeplus)*	6–14	14	30–40	55–75

[a]Data adapted and reprinted from Aviculture Institute, Placerita Canyon Road, Newhall, CA, 91321. Table 3, prepared by Swanson J: In *Proceedings of avian pediatrics seminar*, Union City, CA, 1988, pp 126.

B. Weight Range (in Grams) of Selected Hand-Reared Psittacine Chicks at the Avicultural Breeding and Research Center (ABRC), Loxahatchee, FL[a]

Birds were fed a home-made hand-feeding formula, made with primate diet. This diet consists of 1 quart primate diet plus 1 quart hot water that is soaked for 30 minutes prior to cooking it on the stove top to a soft boil or microwave for 8 minutes on high. Next, 2 tablespoons of smooth peanut butter, 4 ounces of dry infant oatmeal cereal, and ice cubes are added (approximately seven large cubes, to cool the mixture down, as needed). The mixture is then smoothened with a blender, hand mixer, or by hand, and water is added in case the formula is too thick. The temperature of the food is subsequently checked (should be 37.8–40.5°C/100–105°F). If needed, food can be frozen immediately after preparation, but any remaining food should always be discarded and not held for later feedings.

[a]Data adapted and reprinted from Schubott RM, Clubb KJ, Clubb SL: *Psittacine aviculture: perspectives, techniques and research*, Loxahatchee, FL, 1992, Aviculture Breeding and Research Center, with permission.

B.1. Weight Range of Hand-Fed Chicks of Selected Cockatoo Species (in Grams)

Age (Days)	Bare-Eyed Cockatoo *(Cacatua sanguínea)*	Citron-Crested Cockatoo *(Cacatua sulphurea citrinocristata)*	Goffin's Cockatoo *(Cacatua goffini)*	Greater Sulphur-Crested Cockatoo *(Cacatua galerita galerita)*	Lesser Sulphur-Crested Cockatoo *(Cacatua sulphurea)*	Major Mitchell's Cockatoo *(Lophochroa leadbeateri)*	Medium Sulphur-Crested Cockatoo *(Cacatua galerita eleonora)*
	(n = 21)	(n = 15)	(n = 3)	(n = 10)	(n = 22)	(n = 23)	(n = 10)
Hatch	8–14	12–15	9	18	8–15	9–13	14
1	9–14	12–16	10	20	10–15	9–14	16
2	10–16	13–18	12	22	11–16	10–15	17
3	11–18	15–20	13	24	12–19	10–17	19
4	11–21	16–23	15	28	13–23	11–19	23
5	13–26	17–30	19	33	16–27	13–22	28
6	14–32	21–32	22	39	19–32	15–26	34
7	18–33	26–37	25	46	20–39	18–32	40
8	21–37	24–43	28	53	21–49	20–36	44
9	24–50	34–48	35	64	28–55	27–45	52
10	27–58	42–59	41	73	34–63	31–53	65
11	29–71	46–69	52	88	37–74	38–61	75
12	36–79	55–80	60	101	44–83	42–71	85
13	45–88	55–96	69	115	51–94	53–80	96
14	48–103	78–101	79	129	58–105	60–92	105
15	59–112	86–114	89	145	64–116	66–107	118
16	66–123	98–127	96	161	75–126	76–119	136
17	73–133	106–142	105	173	83–140	86–134	147
18	82–144	121–152	112	187	92–156	92–150	162
19	87–160	132–166	122	205	101–173	94–164	175
20	94–169	139–183	133	223	113–188	104–179	189
21	99–186	148–197	144	235	122–203	111–188	202
24	128–221	167–249	169	293	161–239	143–226	244
28	167–265	208–305	218	385	213–282	190–270	302
35	238–328	292–372	278	525	280–338	254–329	396
42	283–364	319–431	300	658	313–380	289–395	449
49	**289–385**	**320–448**	**304**	**729**	**314–404**	**330–407**	463
56	302–375	319–437	289	750	325–390	326–391	461
63	238–346	296–428	271	757	308–373	282–364	452
70	244–322	270–397	242	718	281–345	274–356	**423**
77	260–344	290–347	251	717	267–333	283–395	406
84	268–314	308–367	265	677	229–358	299–392	408
91	263–324	300–370	258	674	221–376	279–393	402
98	280–336	282–390	260	673	236–366	275–413	408

Values in bold indicate maximum weight.

Moluccan Cockatoo *(Cacatua moluccencis)*	**Palm Cockatoo *(Probosciger aterrimus aterrimus)***	**Palm Cockatoo *(Probosciger aterrimus goliath)***	**Red-Tailed Cockatoo *(Calyptorhynchus banksii)***	**Red-Vented Cockatoo *(Cacatua haematuropygia)***	**Rose-Breasted Cockatoo *(Eolophus roseicapilla)***	**Triton Cockatoo *(Cacatua galerita triton)***	**Umbrella Cockatoo *(Cacatua alba)***
(*n* = 26)	(*n* = 10)	(*n* = 7)	(*n* = 3)	(*n* = 2)	(*n* = 17)	(*n* = 17)	(*n* = 71)
16–22	16	20	25	11	7–12	11–19	11–21
18–22	17	22	25	12	7–12	12–20	12–22
19–24	18	23	26	14	9–13	13–21	13–24
21–26	20	24	29	16	10–15	15–24	14–27
23–29	21	27	32	18	11–16	16–30	15–31
26–35	24	29	36	20	12–19	18–36	18–36
30–40	27	33	41	22	13–22	23–41	21–41
33–48	30	36	47	25	15–27	24–52	25–48
34–52	32	40	53	29	16–28	26–54	25–55
44–64	38	45	59	33	–18–37	38–67	34–66
52–74	42	50	67	38	22–43	47–76	40–77
59–84	46	54	74	46	24–51	55–88	46–89
68–95	51	60	83	54	32–57	63–100	54–104
77–108	55	66	92	63	34–68	67–118	62–117
89–123	60	73	101	72	35–77	78–134	72–133
101–139	66	80	110	81	38–90	88–147	82–148
116–155	70	85	121	90	43–101	103–163	95–164
129–176	75	91	127	99	47–115	111–177	107–178
142–198	80	98	137	110	50–126	128–191	124–193
156–217	89	105	148	122	57–132	139–206	132–214
167–242	97	113	156	133	63–141	153–218	149–234
171–261	102	121	165	143	67–146	171–231	160–253
234–330	123	146	194	176	89–161	212–277	203–313
320–433	153	194	235	230	114–181	268–362	263–400
478–596	208	271	312	283	172–257	370–456	359–533
608–706	260	338	406	313	205–301	407–542	436–606
686–790	310	370	483	323	240–305	433–569	486–646
738–855	349	410	543	338	**225–292**	**416–600**	497–663
761–875	389	**438**	**556**	N/A	220–289	406–576	**492–679**
759–885	399	489	555	N/A	209–294	381–549	474–666
753–882	417	508	527	N/A	228–285	344–518	466–637
734–900	428	544	510	N/A	237–309	356–495	440–591
699–873	443	577	485	N/A	243–302	355–491	414–586
686–826	450	577	490	N/A	248–299	365–482	404–586

B.2. Weight Range of Hand-Fed Chicks From Selected Other Psittacine Species (in Grams)

Age (Days)	Blue-and-Gold Macaw *(Ara ararauna)*	Green-Winged Macaw *(Ara chloropterus)*	Hyacinth Macaw *(Anodorhynchus hyacinthinus)*	Military Macaw *(Ara militaris)*	Scarlet Macaw *(Ara macao)*	Blue-Throated Macaw *(Ara glaucogularis)*	Red-Fronted Macaw *(Ara rubrogenys)*
	(n = 43)	(n = 10)	(n = 5)	(n = 23)	(n = 23)	(n = 24)	(n = 28)
Hatch	20–25	28	22	19–26	21–26	14–22	12–16
1	21–26	29	23	20–26	22–27	16–22	12–17
2	23–27	31	25	22–28	24–30	17–23	13–19
3	25–30	33	27	24–32	26–32	19–26	16–21
4	27–33	37	31	25–36	29–36	20–29	17–24
5	30–37	40	34	29–40	32–40	23–32	20–28
6	34–42	45	38	32–46	36–45	27–36	21–35
7	38–49	50	43	35–53	42–50	29–44	23–45
8	42–55	53	47	38–57	47–58	31–54	26–49
9	49–63	63	55	46–72	54–66	37–62	32–62
10	57–74	72	64	52–80	61–76	40–70	37–72
11	63–86	84	73	56–94	68–87	46–82	43–83
12	72–97	93	84	65–109	77–97	53–93	49–94
13	82–112	105	97	78–123	88–113	61–106	55–114
14	93–129	118	110	87–138	98–127	70–118	64–123
15	108–145	133	124	99–154	112–144	81–134	65–151
16	122–166	143	147	116–172	126–163	98–146	71–174
17	135–182	156	165	123–196	143–182	105–167	84–186
18	151–200	170	186	139–220	160–200	118–188	98–198
19	173–234	184	214	158–241	176–216	134–206	109–213
20	186–256	202	231	181–262	194–239	150–223	122–229
21	199–279	221	247	194–292	210–259	164–245	135–249
24	263–366	278	307	270–364	276–339	217–304	174–306
28	349–480	394	463	362–494	374–495	274–415	232–366
35	523–622	610	632	486–695	560–687	411–576	330–478
42	688–816	844	811	590–872	718–851	509–698	415–530
49	813–947	993	902	663–1019	861–1042	586–785	448–524
56	916–1030	1085	957	**760–1039**	**931–1072**	661–819	452–521
63	**927–1064**	1179	1107	812–975	946–1044	685–829	**413–526**
70	868–1010	**1185**	1175	795–925	914–1066	**671–833**	383–481
77	837–985	1090	1206	712–890	845–998	611–796	366–444
84	817–954	1064	**1236**	689–886	808–942	555–758	366–433
91	822–950	979	1223	668–817	760–873	552–694	353–461
98	826–922	981	1215	678–820	800–932	568–670	352–477

Values in bold indicate maximum weight.

Buffon's Macaw *(Ara ambiguous)*	Yellow-Collared Macaw *(Primolius auricollis)*	Amboina King Parrot *(Alisterus amboinensis)*	Australian King Parrot *(Alisterus scapularis)*	Eclectus Parrot *(Eclectus roratus)*	Grey Parrot *(Psittacus erithacus)*	Golden Conure *(Guaruba guarouba)*	Thick-Billed Parrot *(Rhynchopsitta pachyrhyncha)*
(*n* = 9)	(*n* = 8)	(*n* = 29)	(*n* = 16)	(*n* = 118)	(*n* = 18)	(*n* = 17)	(*n* = 6)
21	9	7–9	6–11	12–20	12–18	7–11	11
22	10	7–10	6–12	12–21	13–18	7–11	12
24	11	8–10	8–16	14–22	13–19	8–12	13
25	12	9–12	9–16	15–24	15–21	9–12	14
28	14	10–15	10–20	15–28	16–24	9–14	17
32	16	12–18	11–24	16–32	19–28	9–16	18
36	18	13–21	15–29	18–37	22–34	10–18	20
42	21	14–30	18–34	20–44	25–40	11–20	22
47	24	15–32	23–36	21–50	29–47	12–23	25
54	28	18–42	27–46	28–60	33–55	12–27	29
62	32	21–50	31–54	32–71	39–67	13–30	32
71	37	25–55	37–63	36–82	42–78	14–33	38
80	42	29–63	44–69	42–94	47–90	17–36	41
91	47	38–75	51–75	51–106	53–103	19–40	46
104	54	45–86	57–85	58–120	62–115	20–44	53
117	63	51–93	69–94	69–134	69–129	20–49	59
132	70	59–101	71–101	80–148	75–144	21–58	69
149	78	63–110	76–110	89–160	85–159	24–61	81
166	85	69–124	83–123	99–175	95–174	25–68	91
186	93	75–133	85–138	102–189	105–192	26–74	106
203	100	85–143	93–147	112–201	116–208	27–78	123
224	108	94–153	98–159	119–219	126–220	27–84	138
296	133	130–177	119–196	151–257	167–265	30–106	168
413	173	167–199	156–233	197–322	213–335	40–138	213
638	231	180–217	171–247	257–419	298–439	72–233	256
825	263	189–255	148–230	312–454	369–470	108–270	281
1037	**280**	184–215	149–198	318–477	436–501	154–307	281
1123	272	173–192	144–200	319–477	N/A	182–302	291
1129	253	168–196	156–199	**322–441**	**N/A**	**214–282**	**295**
1152	242	180–207	163–211	321–420	N/A	209–270	278
1146	220	176–200	165–202	290–444	N/A	220–257	265
1099	218	173–206	171–199	312–422	N/A	206–256	256
1091	247	177–210	175–197	292–472	N/A	208–251	261
1096	248	168–213	172–192	321–442	N/A	196–235	266

C. Weight[#] (Mean ± SD, Range) and Food Intake (in Milliliters) of Selected Hand-Reared Psittacine Chicks at the Aviculture Institute, Newhall, CA[a]

#Weight in grams; mean ± SD values are listed on the first line, range and number of chicks on the second line, and the third line lists the volumes administered per feed, respectively weaning age.

Birds were fed a hand-feeding formula prepared by mixing two cups of ground monkey chow (Zupreem Primate Dry Diet; Hill's Pet Products, Topeka, KA) with one cup of hand-feeding diet (Roudybush Hand Feeding Diet; Roudybush, Davis, CA) and one cup of Gerber oatmeal cereal (Florham Park, New Jersey). For chicks <3 days, this mixture is prepared with water in a ratio of 1:3, while for older chicks, the mixture is prepared with water in a ratio of 2:3.

SD, Standard deviation.

[a]Data adapted and reprinted from Aviculture Institute, Placerita Canyon Road, Newhall, CA, 91321. Tables 1 and 2. In *Proceedings of avian pediatrics seminar*, Union City, CA, 1988, pp 117.

C.1. Cockatoos

Age (Days) Number of Feedings	Bare-Eyed Cockatoo *(Cacatua sanguínea)*	Citron-Crested Cockatoo *(Cacatua sulphurea citrinocristata)*	Goffin's Cockatoo *(Cacatua goffini)*	Major Mitchell's Cockatoo *(Lophochroa leadbeateri)*
Day of hatch 6–8 feedings	10.3 ± 2.3 (8.0–12.5; *n* = 3) 0.5–1 mL	13.3 ± 0.9 (11.5–14.5; *n* = 9) 1–2 mL	N/A	N/A
Day 1 6–7 feedings	12.1 ± 1.4 (10.0–13.0; *n* = 4) 1–2 mL	14.2 ± 1.6 (11.5–17.0; *n* = 11) 1–2 mL	9.8 ± 0.4 (9.5–10.0; *n* = 2) 0.5–1 mL	12.2 ± 1.8 (11.0–13.5; *n* = 2) 0.5–1 mL
Day 2 6–7 feedings	12.7 ± 1.5 (10.6–15.0; *n* = 6) 1–2 mL	17.0 ± 3.3 (13.0–25.5; *n* = 14) 1–3 mL	11.2 ± 0.4 (11.0–11.5; *n* = 2) 1–2 mL	14.5 ± 1.4 (13.5–15.5; *n* = 2) 1–2 mL
Day 3 6 feedings	19.0 ± 8.4 (10.5–34.4; *n* = 9) 2–5 mL	19.6 ± 3.3 (14.5–25.5; *n* = 15) 2–4 mL	13.5 (*n* = 2) 1–2 mL	17.2 ± 2.5 (15.5–19.0; *n* = 2) 1–3 mL
Day 4 6 feedings	22.2 ± 8.2 (13.8–37.5; *n* = 9) 3–6 mL	24.1 ± 4.4 (17.0–32.5; *n* = 17) 3.6 mL	15.8 ± 0.4 (15.5–16.0; *n* = 2) 2–3 mL	20.5 ± 3.5 (18.0–23.0; *n* = 2) 2–4 mL
Day 5 6 feedings	27.2 ± 8.0 (16.4–39.5; *n* = 12) 4–8 mL	30.3 ± 11.9 (20.5–74.5; *n* = 18) 4–8 mL	18.5 ± 1.4 (17.5–19.5; *n* = 2) 3–4 mL	24.2 ± 4.6 (21.0–27.5; *n* = 2) 3–5 mL
Day 6 5–6 feedings	34.5 ± 14.3 (17.5–71.5; *n* = 15) 6–10 mL	35.6 ± 10.7 (25.5–72.0; *n* = 20) 6–10 mL	22.2 ± 1.8 (21.0–23.5; *n* = 2) 3–5 mL	29.5 ± 4.2 (26.5–32.5; *n* = 2) 4–6 mL
Day 7 5–6 feedings	40.6 ± 15.6 (19.5–70.0; *n* = 19) 6–12 mL	41.6 ± 12.8 (27.0–84.5; *n* = 21) 6–12 mL	27.5 ± 4.2 (24.5–30.0; *n* = 2) 4–6 mL	35.8 ± 5.3 (32.0–39.5; *n* = 2) 4–8 mL
Day 14 4 feedings	115.7 ± 29.4 (57.5–172.0; *n* = 25) 15–25 mL	108.4 ± 27.2 (72.5–144.0; *n* = 26) 15–20 mL	87.2 ± 6.7 (82.5–92.0; *n* = 2) 20–30 mL	105.5 ± 13.4 (96.0–115.0; *n* = 2) 15–20 mL
Day 21 3 feedings	215.5 ± 50.5 (70.0–308.0; *n* = 26) 30–40 mL	198.9 ± 41.0 (94.0–276.5; *n* = 27) 25–35 mL	180.5 ± 6.4 (176.0–185.0; *n* = 2) 20–30 mL	216.8 ± 6.0 (212.5–221.0; *n* = 2) 20–30 mL
Day 28 3 feedings	300.7 ± 39.4 (169.0–363.0; *n* = 25) 35.45 mL	292.5 ± 52.6 (188.5–397.5; *n* = 26) 35–45 mL	284.5 ± 2.1 (283.0–286.0; *n* = 2) 35–40 mL	310.0 ± 33.2 (286.5–333.5; *n* = 2) 35–45 mL
Day 35 3 feedings	347.0 ± 32.1 (274.5–418.5; *n* = 23) 45–50 mL	350.3 ± 55.5 (230.0–430.0; *n* = 25) 40–50 mL	322.8 ± 0.4 (322.5–323.0; *n* = 2) 35–40 mL	394.5 (*n* = 1) 40–45 mL
Day 42 3 feedings	361.8 ± 29.7 (313.5–411.9; *n* = 14) 45–50 mL	379.3 ± 40.4 (303.5–446.5; *n* = 18) 50–60 mL	N/A	401.0 ± 3.2 (399.0–403.5; *n* = 2) 40–45 mL
Day into cage Peak weight reached at 3 feedings	374.0 ± 27.3 (334.0–418.5; *n* = 14) 44 days 45–50 mL	420.1 ± 45.8 (342.0–502.0; *n* = 13) 45 days 50–60 mL	N/A	401.0 ± 3.2 (399.0–403.5; *n* = 2) 42 days 40–45 mL
At time of weaning	270.0 ± 22.4 (245.0–305.0; *n* = 6) 74 days	320.5 ± 51.0 (272.5–392.5; *n* = 4) 81 days	N/A	± (; *n* =) 76 days
30 Days postweaning	279.0 ± 15.1 (261.5–297.5; *n* = 4)	285.0 (*n* = 1)	N/A	± (; *n* =)

Moluccan Cockatoo *(Cacatua moluccensis)*	Red-Vented Cockatoo *(Cacatua haematuropygia)*	Rose-Breasted Cockatoo *(Eolophus roseicapilla)*	Triton Cockatoo *(Cacatua galerita triton)*	Umbrella Cockatoo *(Cacatua alba)*
18.2 ± 2.1	N/A	9.5 ± 0.9	17.8 ± 2.4	15.1 ± 1.1
(16.5–20.0; *n* = 3)		(8.5–10.5; *n* = 5)	(14.5–22.5; *n* = 9)	(13.5–17.0; *n* = 8)
1–2 mL		0.5 mL	1–3 mL	1–2 mL
19.2 ± 1.8	N/A	10.1 ± 0.7	18.7 ± 2.8	16.0 ± 1.4
(17.5–21.0; *n* = 3)		(9.0–11.0; *n* = 8)	(14.0–24.0; *n* = 14)	(14.0–18.0; *n* = 9)
2–3 mL		1 mL	2–4 mL	1–2 mL
22.3 ± 0.3	19.0	12.4 ± 2.4	21.9 ± 3.7	18.2 ± 1.7
(22.0–22.5; *n* = 3)	(*n* = 1)	(9.5–18.2; *n* = 9)	(17.0–27.5; *n* = 12)	(16.0–20.5; *n* = 9)
2–4 mL	2.5 mL	1–1.5 mL	3–4 mL	1–3 mL
25.7 ± 1.30	19.5	15.0 ± 2.7	25.7 ± 4.5	20.1 ± 2.0
(24.5–27.0; *n* = 3)	(*n* = 1)	(11.0–21.5; *n* = 13)	(17.0–27.5; *n* = 12)	(18.0–24.5; *n* = 8)
3–5 mL	3 mL	2–3 mL	3–4 mL	2–4 mL
30.3 ± 2.0	22.5	18.2 ± 4.3	30.3 ± 6.3	23.6 ± 3.0
(28.5–32.5; *n* = 3)	(*n* = 2)	(12.0–28.5; *n* = 17)	(21.0–39.5; *n* = 13)	(20.0–29.5; *n* = 9)
3–6 mL	3 mL	2–4 mL	4–8 mL	3–5 mL
34.2 ± 2.4	26.5 ± 6.1	21.6 ± 4.5	37.4 ± 7.8	28.5 ± 6.7
(31.5–36.0; *n* = 3)	(21.0–33.0; *n* = 3)	(15.0–33.8; *n* = 19)	(25.0–47.5; *n* = 15)	(21.5–44.0; *n* = 9)
4–8 mL	4 mL	3–6 mL	4–10 mL	4–8 mL
40.2 ± 2.0	31.2 ± 4.5	25.1 ± 5.4	44.9 ± 10.8	34.4 ± 7.1
(38.0–42.0; *n* = 3)	(27.0–36.0; *n* = 3)	(12.0–46.5; *n* = 22)	(28.5–64.0; *n* = 15)	(24.0–47.0; *n* = 10)
5–10 mL	4 mL	4–8 mL	6–12 mL	4–10 mL
46.7 ± 2.3	36.5 ± 6.3	30.3 ± 8.3	54.2 ± 12.8	37.5 ± 6.8
(44.5–49.0; *n* = 3)	(30.0–42.5; *n* = 3)	(13.0–46.5; *n* = 27)	(32.5–73.5; *n* = 15)	(25.2–46.5; *n* = 8)
6–10 mL	4 mL	4–8 mL	8–12 mL	6–12 mL
137.2 ± 22.4	101.2 ± 16.1	92.5 ± 22.0	155.7 ± 33.8	117.8 ± 25.3
(118.5–162.0; *n* = 3)	(81.0–120.0; *n* = 4)	(34.0–130.0; *n* = 30)	(76.0–204.0; *n* = 14)	(69.0–164.5; *n* = 10)
20–30 mL	15–25 mL	15–25 mL	25–35 mL	15–25 mL
292.7 ± 44.0	205.4 ± 27.1	187.2 ± 30.6	306.5 ± 52.0	243.9 ± 33.4
(254.0–342.5; *n* = 3)	(165.2–224.5; *n* = 4)	(88.0–235.5; *n* = 30)	(170.5–365.5; *n* = 14)	(188.7–3 00.0; *n* = 10)
35–45 mL	25–30 mL	25–35 mL	40–50 mL	40–45 mL
432.2 ± 36.6	283.2 ± 31.2	264.7 ± 25.4	447.7 ± 64.9	368.2 ± 31.5
(402.5–473.0; *n* = 3)	(241.0–315.0; *n* = 4)	(177.5–321.0; *n* = 30)	(292.0–540.0; *n* = 14)	(304.5–426.5; *n* = 10)
40–50 mL	35–40 mL	35–45 mL	50–60 mL	50–60 mL
603.7 ± 59.2	303.6 ± 47.8	315.5 ± 27.1	552.3 ± 68.7	474.5 ± 41.7
(568.0–672.0; *n* = 3)	(232.0–328.0; *n* = 4)	(262.0–376.0; *n* = 29)	(442.0–662.0; *n* = 12)	(409.5–541.0; *n* = 10)
60–70 mL	35–45 mL	40–45 mL	60–70 mL	60–70 mL
730.8 ± 66.8	317.9 ± 54.3	N/A	615.6 ± 68.9	532.5 ± 60.7
(683.5–778.0; *n* = 2)	(247.0–363.0; *n* = 4)		(557.5–724.0; *n* = 12)	(449.5–628.0; *n* = 7)
70–90 mL	35–45 mL		60–80 mL	60–80 mL
919.5 ±33.2	342.5 ± 29.0	332.3 ± 29.2	723.9 ± 79.1	630.8 ± 121.0
(896.0–943.0; *n* = 2)	(299.5–363.0; *n* = 4)	(293.0–423.0; *n* = 23)	(616.0–887.3; *n* = 9)	(470.5–764.5; *n* = 4)
56 days	46 days	40 days	55 days	56 days
80–90 mL	40–45 mL	40–45 mL	70–80 mL	70–80 mL
822.0	261.8 ± 1.1	278.8 ± 31.4	517.4 ± 65.9	518.3 ± 34.3
(*n* = 1)	(261.0–262.5; *n* = 2)	(246.0–363.0; *n* = 12)	(474.0–628.5; *n* = 7)	(481.0–548.5; *n* = 3)
99 days	80 days	61 days	101 days	105 days
N/A	288.5 ± 30.4	339.8 ± 51.1	530.9 ± 80.3	506.4 ± 66.5
	(267.0–310.0; *n* = 2)	(298.5–429.0; *n* = 6)	(448.0–648.0; *n* = 6)	(459.4–553.5; *n* = 2)

C.2. Macaws

Age (Days) Number of Feedings	Blue-and-Gold Macaw *(Ara ararauna)*	Buffon's Macaw *(Ara ambiguous)*	Green-Winged Macaw *(Ara chloropterus)*
Day of hatch	20.0 ± 3.4	22.4 ± 2.2	20.6 ± 1.9
6–8 feedings	(14.0–28.0; *n* = 69)	(18.5–26.0; *n* = 16)	(16.0–24.0; *n* = 12)
	1–2 mL	1–2 mL	1–2 mL
Day 1	21.1 ± 3.8	23.5 ± 2.7	23.6 ± 2.2
6–7 feedings	(14.5–31.0; *n* = 82)	(18.5–27.0; *n* = 17)	(19.0–27.0; *n* = 11)
	2–3 mL	2–4 mL	2–3 mL
Day 2	23.7 ± 4.0	26.2 ± 3.2	27.7 ± 2.5
6–7 feedings	(15.0–35.2; *n* = 81)	(20.5–31.0; *n* = 17)	(22.5–32.0; *n* = 13)
	2–4 mL	3–5 mL	4–6 mL
Day 3	26.6 ± 4.4	29.0 ± 3.6	32.7 ± 7.4
6 feedings	(17.5–41.2; *n* = 82)	(23.0–35.4; *n* = 16)	(24.5–56.5; *n* = 15)
	3–4 mL	4–6 mL	4–8 mL
Day 4	31.7 ± 6.0	32.8 ± 4.6	37.4 ± 7.1
6 feedings	(18.0–46.5; *n* = 87)	(25.0–40.0; *n* = 16)	(28.0–57.0; *n* = 14)
	3–6 mL	5–8 mL	6–8 mL
Day 5	35.2 ± 6.9	37.2 ± 4.7	44.1 ± 9.2
6 feedings	(21.0–61.0; *n* = 88)	(28.0–46.0; *n* = 16)	(31.0–64.0; *n* = 15)
	4–8 mL	4–5 mL	6–10 mL
Day 6	41.5 ± 10.2	42.7 ± 6.0	48.6 ± 7.9
5–6 feedings	(24.5–78.0; *n* = 92)	(31.0–54.0; *n* = 16)	(34.0–61.5; *n* = 13)
	5–10 mL	6–10 mL	8–10 mL
Day 7	47.3 ± 12.4	49.5 ± 8.1	56.9 ± 12.0
5–6 feedings	(26.0–111.0; *n* = 88)	(35.5–67.0; *n* = 16)	(37.5–88.0; *n* = 14)
	10–15 mL	10–15 mL	10–15 mL
Day 14	130.3 ± 39.9	129.6 ± 23.5	168.4 ± 41.5
4 feedings	(65.0–250.0; *n* = 98)	(86.0–166.5; *n* = 16)	(86.5–263.0; *n* = 16)
	15–25 mL	20–25 mL	20–30 mL
Day 21	306.6 ± 71.3	350.0 ± 75.6	356.0 ± 65.3
3 feedings	(114.5–476.0; *n* = 98)	(229.5–550.0; *n* = 16)	(224.5–459.5; *n* = 17)
	30–50 mL	40–50 mL	40–50 mL
Day 28	522.2 ± 82.2	610.1 ± 87.5	587.0 ± 78.6
3 feedings	(263.0–680.0; *n* = 96)	(433.5–787.0; *n* = 16)	(441.0–715.0; *n* = 17)
	60–80 mL	70–80 mL	60–70 mL
Day 35	728.0 ± 91.2	864.1 ± 84.5	801.0 ± 90.4
3 feedings	(463.0–929.5; *n* = 92)	(732.5–1061.0; *n* = 15)	(660.0–968.0; *n* = 16)
	80–100 mL	80–90 mL	70–80 mL
Day 42	917.7 ± 86.3	1074.0 ± 75.6	978.0 ± 76.5
3 feedings	(659.0–1125.0; *n* = 88)	(932.0–1220.0; *n* = 15)	(831.5–1092.0; *n* = 14)
	90–110 mL	100–110 mL	90–100 mL
Day 49	1033.3 ± 81.5	1226.6 ± 78.4	1117.7 ± 64.4
3 feedings	(774.0–1210.5; *n* = 73)	(1100.0–1372.0; *n* = 14)	(995.0–1202.0; *n* = 12)
	100–120 mL	110–120 mL	100–120 mL
Day 56	1098.7 ± 71.3	1328.0 ± 75.4	1222.5 ± 45.3
3 feedings	(927.0–1267.0; *n* = 67)	(1221.0–1442.0; *n* = 12)	(1136.5–1304.0; *n* = 10)
	120 mL	120 mL	110–120 mL
Day into cage	1114.0 ± 68.4	1355.7 ± 70.1	1283.8 ± 65.1
Peak weight reached at	(959.5–1267.0; *n* = 67)	(1279.0–1468.0; *n* = 11)	(1168.5–1376.0; *n* = 8)
3 feedings	62 days	61 days	62 days
	120 mL	120 mL	120 mL
At time of weaning	877.7 ± 67.6	958.5 ± 36.1	958.9 ± 59.2
	(701.5–1021.0; *n* = 39)	(933.0–984.0; *n* = 12)	(910.0–1036.5; *n* = 4)
	111 days	135 days	115 days
30 Days postweaning	912.4 ± 80.5	N/A	1001.5
	(721.0–1080.5; *n* = 29)		(*n* = 1)

Military Macaw *(Ara militaris)*	**Red-Fronted Macaw** *(Ara rubrogenys)*	**Scarlet Macaw** *(Ara macao)*	**Yellow-Collared Macaw** *(Primolius auricollis)*
20.4 ± 2.4	12.8 ± 0.8	20.4 ± 2.2	12.0 ± 2.3
(16.0–25.0; *n* = 14)	(11.5–13.5; *n* = 5)	(16.0–24.5; *n* = 32)	(10.0–14.5; *n* = 3)
1–2 mL	1–2 mL	1–2 mL	1–2 mL
21.1 ± 1.9	14.9 ± 3.0	21.6 ± 2.2	12.3 ± 1.9
(17.0–25.0; *n* = 16)	(13.0–21.0; *n* = 6)	(17.0–26.0; *n* = 35)	(10.5–14.6; *n* = 4)
2–3 mL	2–3 mL	2–3 mL	1–2 mL
24.0 ± 2.4	18.1 ± 3.3	25.1 ± 4.6	13.7 ± 2.4
(19.0–30.0; *n* = 20)	(15.0–23.0; *n* = 7)	(18.5–49.0; *n* = 38)	(11.0–15.8; *n* = 4)
2–3 mL	2–3 mL	2–4 mL	2–3 mL
28.1 ± 5.1	20.9 ± 2.7	27.7 ± 4.2	16.1 ± 2.9
(21.0–34.0; *n* = 22)	(17.5–25.5; *n* = 7)	(21.5–48.0; *n* = 40)	(13.5–19.5; *n* = 4)
3–5 mL	3–4 mL	3–6 mL	2–3 mL
30.7 ± 4.8	24.1 ± 2.9	31.6 ± 4.8	20.0 ± 5.0
(24.0–37.0; *n* = 22)	(19.5–24.5; *n* = 8)	(23.0–50.5; *n* = 40)	(14.5–27.0; *n* = 5)
4–6 mL	4 mL	4–6 mL	2–3 mL
33.8 ± 4.6	27.8 ± 3.4	35.9 ± 5.8	21.6 ± 4.7
(25.5–44.0; *n* = 21)	(23.5–32.5; *n* = 8)	(26.5–54.0; *n* = 40)	(15.0–27.0; *n* = 6)
5–8 mL	4–5 mL	4–8 mL	2–3 mL
48.9 ± 31.2	32.6 ± 4.3	41.3 ± 7.1	27.0 ± 8.0
(28.0–187.5; *n* = 24)	(27.5–38.0; *n* = 8)	(28.5–60.0; *n* = 40)	(16.5–44.5; *n* = 7)
6–10 mL	4–6 mL	5–10 mL	4–6 mL
56.9 ± 39.8	38.3 ± 6.0	47.8 ± 8.7	32.0 ± 8.0
(31.0–194.5; *n* = 22)	(32.5–48.5; *n* = 8)	(30.5–67.5; *n* = 40)	(19.0–43.0; *n* = 6)
10–15 mL	6–12 mL	10–15 mL	6–12 mL
161.0 ± 83.0	110.1 ± 15.9	134.8 ± 34.7	87.1 ± 10.0
(56.5–362.0; *n* = 27)	(78.6–136.0; *n* = 11)	(53.5–191.0; *n* = 40)	(55.5–110.0; *n* = 8)
20–35 mL	15–20 mL	15–25 mL	12–15 mL
320.7 ± 96.0	226.5 ± 31.4	299.5 ± 68.8	162.0 ± 20.1
(108.0–480.5; *n* = 28)	(184.0–271.5; *n* = 11)	(106.0–408.0; *n* = 41)	(117.0–216.0; *n* = 8)
35–50 mL	25–30 mL	30–50 mL	20 mL
502.9 ± 108.2	336.9 ± 42.3	506.6 ± 83.9	232.1 ± 20.3
(227.5–702.0; *n* = 28)	(285.0+422.5; *n* = 11)	(25+.0–624.5; *n* = 41)	(196.5–263.5; *n* = 8)
50–70 mL	35–40 mL	50–70 mL	30 mL
678.2 ± 102.4	431.8 ± 48.6	724.1 ± 93.0	267.8 ± 15.4
(408.0–864.0; *n* = 28)	(355.5–519.0; *n* = 9)	(515.0–863.5; *n* = 41)	(240.0–281.5; *n* = 6)
65–80 mL	50 mL	70–90 mL	35 mL
827.2 ± 88.5	492.8 ± 43.5	900.3 ± 81.1	286.8 ± 2.3
(634.0–948.0; *n* = 25)	(405.0–543.5; *n* = 8)	(738.0–1057.5; *n* = 39)	(284.5–288.5; *n* = 3)
80–90 mL	50–60 mL	80–100 mL	40 mL
921.3 ± 83.8	528.5 ± 54.9	1015.1 ± 96.4	N/A
(765.0–1115.0; *n* = 23)	(466.0–579.0; *n* = 4)	(831.0–1195.0; *n* = 35)	
80–100 mL	60 mL	90–120 mL	
932.7 ± 68.2	N/A	1095.8 ± 90.2	N/A
(824.0–1069.0; *n* = 12)		(893.0–1334.0; *n* = 33)	
90–100 mL		100–120 mL	
946.5 ± 56.2	539.5 ± 42.3	1137.3 ± 88.4	296.8 ±10.7
(862.0–1074.0; *n* = 16)	(471.0–590.5; *n* = 7)	(1024.0–1334.0; *n* = 30)	(284.5–304.0)
61 days	49 days	61 days	44 days
90–100 mL	60–70 mL	120 mL	40 mL
731.0 ± 53.2	414.4 ± 18.3	888.5 ± 77.1	N/A
(656.0–829.0; *n* = 13)	(396.0–449.5; *n* = 7)	(784.0–1027.0; *n* = 18)	
94 days	91 days	108 days	
763.0 ± 55.1	420.9 ± 14.7	913.3 ± 96.1	N/A
(648.5–830.0; *n* = 12)	(402.5–440.0; *n* = 6)	(763.0–1086.0; *n* = 13)	

C.3. Amazon Parrots, Grey Parrot, and Eclectus Parrot

Age (Days) Number of Feedings	Green-Cheeked Amazon *(Amazona viridigenalis)*	Lilac-Crowned Amazon *(Amazona finschi)*	Blue-Fronted Amazon *(Amazona aestiva)*	Yellow-Crowned Amazon *(Amazona ochrocephala)*
Day of hatch	11.2 ± 1.6	11.6 ± 1.5	14.8 ± 2.3	14.2 ± 0.6
6–8 feedings	(10.0–13.0; *n* = 4)	(10.0–13.5; *n* = 4)	(12.5–17.0; *n* = 4)	(13.5–15.0; *n* = 7)
	1–2 mL	1–2 mL	1–2 mL	1–2 mL
Day 1	14.3 ± 3.0	14.0 ± 3.1	16.6 ± 3.1	15.6 ± 1.4
6–7 feedings	(10.5–20.0; *n* = 9)	(10.5–19.0; *n* = 6)	(13.5–22.5; *n* = 7)	(13.5–17.5; *n* = 9)
	1–2 mL	2 mL	2–3 mL	2–3 mL
Day 2	16.1 ± 2.3	16.4 ± 4.9	19.3 ± 3.5	18.1 ± 1.9
6–7 feedings	(12.5–20.0; *n* = 8)	(11.5–26.5; *n* = 7)	(15.0–24.0; *n* = 8)	(15.5–22.0; *n* = 9)
	2–3 mL	2–3 mL	2–4 mL	2–3 mL
Day 3	18.1 ± 2.3	18.2 ± 3.2	23.6 ± 6.9	22.3 ± 5.4
6 feedings	(14.0–21.5; *n* = 10)	(15.0–24.0; *n* = 7)	(17.5–40.5; *n* = 9)	(15.5–34.0; *n* = 12)
	3 mL	3 mL	3–4 mL	3–5 mL
Day 4	20.1 ± 3.1	21.0 ± 3.8	28.1 ± 6.6	25–8 ± 6.5
6 feedings	(14.5–24.5; *n* = 9)	(14.5–26.0; *n* = 5)	(21.0–40.0; *n* = 10)	(18.5–39.5; *n* = 13)
	3–4 mL	3–4 mL	4–6 mL	3–6 mL
Day 5	23.6 ± 3.3	24.8 ± 4.2	35.0 ± 9.6	31.2 ± 8.0
6 feedings	(17.5–32.0; *n* = 9)	(16.5–29.0; *n* = 5)	(29.5–57.5; *n* = 12)	(22.0–45.5; *n* = 15)
	4–5 mL	4 mL	6–8 mL	4–6 mL
Day 6	29.6 ± 7.1	28.1 ± 5.1	42.3 ± 9.6	38.2 ± 9.3
5–6 feedings	(17.5–32.0; *n* = 9)	(22.0–34.5; *n* = 5)	(29.5–57.5; *n* = 12)	(26.0–55.0; *n* = 15)
	4–5 mL	4–6 mL	6–8 mL	4–8 mL
Day 7	37.4 ± 10.6	34.8 ± 5.5	49.5 ± 10.9	44.7 ± 10.0
5–6 feedings	(24.0–60.5; *n* = 12)	(26.5–40.0; *n* = 6)	(34.0–66.0; *n* = 12)	(31.5–62.5; *n* = 18)
	6–8 mL	6–8 mL	6–10 mL	5–10 mL
Day 14	103.6 ± 27.3	105.1 ± 21.4	129.4 ± 26.2	125.0 ± 23.4
4 feedings	(60.0–154.0; *n* = 12)	(82.0–142.0; *n* = 6)	(85.0–171.0; *n* = 12)	(85.5–172.0; *n* = 18)
	15–20 mL	15–20 mL	20–25 mL	15–25 mL
Day 21	208.2 ± 34.3	202.8 ± 24.6	244.8 ± 21.9	240.3 ± 26.2
3 feedings	(147.0–262.0; *n* = 12)	(178.0–244.0; *n* = 6)	(210.0–272.0; *n* = 11)	(194.5–279.0; *n* = 17)
	25–35 mL	25–35 mL	30–35 mL	25–35 mL
Day 28	296.9 ± 28.7	291.7 ± 17.3	336.8 ± 29.5	344.7 ± 25.0
3 feedings	(260.0–334.5; *n* = 12)	(265.0–309.0; *n* = 6)	(295.5–382.5; *n* = 12)	(292.5–384.5; *n* = 18)
	30–40 mL	30–40 mL	40–45 mL	40–45 mL
Day 35	332.4 ± 18.7	336.0 ± 16.5	396.3 ± 29.6	403.3 ± 23.0
3 feedings	(310.5–369.5; *n* = 11)	(307.0–350.0; *n* = 6)	(352.5–462.0; *n* = 12)	(367.0–443.5; *n* = 18)
	40–45 mL	40–45 mL	45–50 mL	45–55 mL
Day 42	318.5 ± 19.3	327.2 ± 17.5	402.2 ± 21.2	426.8 ± 32.1
3 feedings	(289.0–347.0; *n* = 6)	(312.0–347.0; *n* = 4)	(376.5–433.0; *n* = 8)	(378.5–485.5; *n* = 16)
	40–45 mL	40–45 mL	45–50 mL	50–55 mL
Day into cage	340.1 ± 14.4	346.3 ± 23.9	411.9 ± 30.6	436.2 ± 28.9
Peak weight reached at	(314.0–401.5; *n* = 11)	(309.5–370.0; *n* = 6)	(376.0–470.0; *n* = 8)	(390.0–485.5; *n* = 15)
3 feedings	40 days	42 days	42 days	43 days
	40–45 mL	40–45 mL	45–50 mL	50–60 mL
At time of weaning	264.8 ± 13.7	276.2 ± 4.4	325.2 ± 17.6	377.4 ± 29.4
	(250.0–333.0; *n* = 6)	(270.0–280.0; *n* = 4)	(314.5–351.5; *n* = 4)	(350.0–423.0; *n* = 11)
	70 days	70.2 days	71 days	74 days
30 Days postweaning	300.6 ± 17.2	293.3 ± 12.4	334.0 ± 16.2	409.8 ± 23.6
	(286.0–319.5; *n* = 4)	(279.0–301.0; *n* = 3)	(320.0–357.0; *n* = 4)	(382.0–450.5; *n* = 8)

Yellow-Naped Amazon ***(Amazona auropalliata)***	**Yellow-Headed Amazon** ***(Amazona oratrix)***	**Grey Parrot** ***(Psittacus erithacus)***	**Eclectus Parrot** ***(Eclectus roratus)***
16.2 ± 2.0	15.1 ± 2.4	13.3 ± 1.7	15.5 ± 1.7
(11.5–18.0; *n* = 16)	(11.0–20.5; *n* = 16)	(10.5–17.0; *n* = 27)	(11.5–16.7; *n* = 8)
1–2 mL	1–2 mL	0.5–1 mL	0.5–1 mL
18.8 ± 2.7	16.2 ± 2.7	14.5 ± 1.69	17.9 ± 3.5
(13.0–23.5; *n* = 17)	(11.5–22.5; *n* = 21)	(12.0–18.0; *n* = 31)	(12.5–26.5; *n* = 10)
2–3 mL	1–2 mL	1–3 mL	1–3 mL
21.3 ± 2.8	20.2 ± 5.0	16.5 ± 1.6	19.9 ± 3.6
(16.0–25.0; *n* = 16)	(14.0–36.5; *n* = 23)	(13.5–20.0; *n* = 33)	(14.0–26.0; *n* = 11)
2–4 mL	1–3 mL	2–3 mL	1–3 mL
25.4 ± 4.5	22.5 ± 4.7	19.0 ± 2.4	22.7 ± 5.6
(16.5–35.0; *n* = 18)	(16.0–35.0; *n* = 25)	(15.5–26.5; *n* = 35)	(15.5–37.0; *n* = 14)
mL	2–4 mL	2–3 mL	2–4 mL
29.7 ± 5.3	28.3 ± 7.7	21.5 ± 2.5	28.2 ± 8.4
(20.5–40.0; *n* = 17)	(17.0–43.5; *n* = 18)	(18.0–28.0; *n* = 32)	(18.0–39.5; *n* = 16)
4–8 mL	3–6 mL	3–4 mL	2–4 mL
35.6 ± 7.0	36.4 ± 13.1	25.3 ± 3.7	30.7 ± 8.0
(23.0–50.2; *n* = 18)	(19.5–69.5; *n* = 23)	(20.0–37.5; *n* = 35)	(19.0–42.0; *n* = 17)
4–10 mL	5–8 mL	3–5 mL	3–5 mL
41.5 ± 8.9	42.5 ± 14.6	29.6 ± 3.7	36.5 ± 11.3
(24.5–61.0; *n* = 18)	(22.0–77.0; *n* = 22)	(24.0–39.5; *n* = 34)	(20.5–62.8; *n* = 20)
4–12 mL	5–8 mL	4–6 mL	3–5 mL
49.1 ± 11.9	45.9 ± 14.9	34.2 ± 4.1	44.1 ± 15.7
(27.0–75.0; *n* = 18)	(25.5–89.5; *n* = 34)	(27.5–44.5; *n* = 34)	(23.0–102.0; *n* = 27)
4–12 mL	6–10 mL	4–6 mL	4–6 mL
140.4 ± 43.1	123.0 ± 34.7	99.0 ± 15.0	105.3 ± 25.3
(61.5–218.5; *n* = 19)	(75.0–199.5; *n* = 36)	(71.5–124.5; *n* = 34)	(35.0–158.5; *n* = 71)
15–25 mL	15–25 mL	10–20 mL	10–15 mL
283.2 ± 61.7	246.4 ± 41.5	193.2 ± 26.1	180.6 ± 39.7
(142.5–383.0; *n* = 19)	(203.5–322.0; *n* = 38)	(145.0–246.5; *n* = 34)	(61.0–252.0; *n* = 95)
30–40 mL	30–40 mL	25–30 mL	20–30 mL
401.6 ± 79.9	365.4 ± 46.1	283.9 ± 25.6	265.6 ± 41.9
(220.5–517.0; *n* = 19)	(229.0–450.0; *n* = 36)	(235.5–329.0; *n* = 34)	(113.5–357.5; *n* = 98)
40–50 mL	40–45 mL	30–40 mL	30–40 mL
507.1 ± 69.3	441.5 ± 54.9	356.3 ± 35.4	339.8 ± 41.6
(322.0–612.0; *n* = 13)	(272.0–579.6; *n* = 34)	(290.5–426.0; *n* = 28)	(184.5–442.0; *n* = 90)
50–55 mL	40–55 mL	40–45 mL	35–45 mL
563.6 ± 59.8	462.0 ± 56.0	416.9 ± 30.4	387.6 ± 38.7
(421.5–650.0; *n* = 10)	(315.0–559.0; *n* = 25)	(374.5–490.0; *n* = 22)	(271.0–456.5; *n* = 81)
50–60 mL	40–55 mL	40–50 mL	40–50 mL
590.9 ± 37.9	484.7 ± 48.3	468.5 ± 36.5	430.0 ± 30.8
(556.5–654.5; *n* = 7)	(388.5–565.0; *n* = 22)	(418.0–526.0; *n* = 13)	(355.0–499.5; *n* = 74)
46 days	47 days	54 days	51 days
50–60 mL	45–55 mL	45–50 mL	45–50 mL
487.5 ± 5.0	377.6 ± 40.0	422.4 ± 44.3	383.4 ± 28.7
(484.0–491.0; *n* = 2)	(304.5–396.5; *n* = 15)	(372.0–493.0; *n* = 7)	(332.5–468.5; *n* = 45)
76 days	75 days	79 days	79 days
N/A	423.3 ± 37.6	385.5 ± 27.6	388.8 ± 22.0
	(352.0–476.0; *n* = 10)	(346.5–411.0; *n* = 4)	(350.0–437.0; *n* = 36)

D. Weight Gain (in Grams) of Selected Hand-Raised Psittacine Chicks and Normal Adults

Age (Days)	Cockatiel *(Nymphicus hollandicus)*	Bare-Eyed Cockatoo *(Cacatua sanguinea)*	Citron-Crested Cockatoo *(Cacatua sulphurea citrinocristata)*	Goffin's Cockatoo *(Cacatua goffini)*	Greater Sulphur-Crested Cockatoo *(Cacatua galerita)*	Lesser Sulphur-Crested Cockatoo *(Cacatua sulphurea)*	Major Mitchell's Cockatoo *(Lophochroa leadbeateri)*
0	4–6	8–14	12–15	8–11	16–20	8–15	9–13
3	5–6	11–35	15–23	10–15	18–35	12–22	13–22
7	12–14	18–70	26–84	20–45	35–80	25–60	22–55
14	45–65	48–170	78–144	70–100	100–200	65–120	55–130
21	72–108	99–308	148–265	125–240	220–330	140–250	140–220
28	80–120	167–363	208–366	175–275	370–525	225–320	210–300
35	80–90	238–415	292–430	220–325	450–625	280–340	270–375
42	80–95	283–410	319–445	250–350	500–725	315–380	290–450
49	90–110	289–415	320–464	250–350	550–880	320–410	340–500
Adult	90–110	375	375	255	843	450	423

Age (Days)	Blue-and-Gold Macaw *(Ara ararauna)*	Blue-Throated Macaw *(Ara glaucogularis)*	Buffon's Macaw *(Ara ambiguous)*	Green-Winged Macaw *(Ara chloropterus)*	Hyacinth Macaw *(Anodorhynchus hyacinthinus)*	Military Macaw *(Ara militaris)*	Red-Fronted Macaw *(Ara rubrogenys)*	Scarlet Macaw *(Ara macao)*
0	16–27	14–22	20–26	17–28	20–27	17–26	12–16	17–26
3	25–40	19–25	25–35	30–55	25–35	24–45	18–25	25–45
7	40–100	30–45	40–70	45–80	45–75	35–170	25–45	40–65
14	90–250	70–120	100–170	100–250	110–180	85–300	70–130	90–175
21	200–450	165–250	250–500	225–450	250–400	220–425	140–250	200–400
28	350–650	275–420	450–750	400–650	450–600	360–650	230–360	380–625
35	525–900	420–600	650–900	610–900	600–750	500–800	330–470	540–800
42	670–1100	520–725	850–1100	830–1030	800–1000	600–950	405–530	720–1050
49	800–1200	600–800	1050–1350	990–1190	900–1200	680–1050	465–580	830–1150
Adult	1039	752	1290	1194	1355	925	490	1001

Weight ranges are derived from weight gain records of birds hand-raised from hatching to weaning. They are provided as suggested ranges only, as growth of an individual chick is dependent on hatch weight, body structure, sex, diet and feeding, and husbandry practices. Comparison of data from two successful nurseries has indicated that birds with widely divergent body weights can successfully wean.[1,2] Birds in the lower end of the weight range are more prone to stunting and should be carefully observed. Birds in the upper end of the weight range may shed excess weight at weaning. Data from this table should be combined with observation of the conformation and physical condition of the chick before deciding if an individual is stunted in growth.

Adapted and reprinted from Harrison GJ, Harrison LR, Ritchie BW: *Avian medicine: principles and application*, Lake Worth, 1994, Wingers Publishing.

Medium Sulphur-Crested Cockatoo *(Cacatua galerita eleonora)*	Moluccan Cockatoo *(Cacatua moluccencis)*	Red-Vented Cockatoo *(Cacatua haematuropygia)*	Rose-Breasted Cockatoo *(Eolophus roseicapilla)*	Triton Cockatoo *(Cacatua galerita triton)*	Umbrella Cockatoo *(Cacatua alba)*	Eclectus Parrot *(Eclectus roratus)*	Grey Parrot *(Psittacus erithacus)*
12–15	16–22	11	7–12	11–19	12–20	12–20	11–17
18–25	21–30	16–20	10–17	15–30	15–20	16–35	15–21
35–70	35–55	25–30	15–40	30–70	25–55	23–60	25–40
65–140	90–170	70–100	35–100	90–170	75–150	60–150	70–120
160–250	190–300	145–200	70–200	200–325	170–300	110–240	135–250
240–350	330–450	230–280	115–300	290–475	280–400	190–350	240–335
340–450	470–650	250–300	175–370	400–650	350–530	260–440	300–440
400–525	600–750	275–350	220–400	450–750	450–600	300–450	380–470
450–550	680–825	230–350	240–423	490–800	500–725	320–480	435–500
465	853	298	403	643	577	432	554

Yellow-Collared Macaw *(Primolius auricollis)*	Blue-Fronted Amazon Parrot *(Amazona aestiva)*	Green-Cheeked Amazon Parrot *(Amazona viridigenalis)*	Lilac-Crowned Amazon Parrot *(Amazona finschi)*	Yellow-Crowned Amazon Parrot *(Amazona ocrocephala)*	Yellow-Headed Amazon Parrot *(Amazona oratrix)*	Yellow-Naped Amazon Parrot *(Amazona auropalliata)*	Golden Conure *(Guaruba guarouba)*
9–15	14–17	10–14	11–13	12–15	12–21	11–18	7–11
12–20	20–25	15–22	15–20	15–33	16–30	16–35	10–12
25–35	35–60	30–50	25–35	15–55	25–50	28–75	12–23
60–90	100–170	90–135	75–140	70–170	75–200	60–200	20–25
110–160	240–280	200–250	160–240	175–260	140–300	170–360	30–100
190–240	280–370	225–310	250–300	250–360	230–450	275–500	45–150
230–280	350–420	280–350	300–350	350–440	270–580	420–600	90–240
250–290	380–440	290–350	310–350	400–480	310–560	500–650	125–270
270–300	380–430				380–565	500–650	180–310
250	432	360	360	500	568	596	262

E. Weight Records of Selected Species of Macaws[a]

Birds were reared using Raintree's hand-feeding formula for macaws. This home-made hand-feeding formula consists of a dry mix (one cup of ground Zupreem pellets; one cup of Gerber mixed cereal for babies; one cup of Tune-Up Mynah Pellets; three-fourths of a cup of raw peanuts, blanched without skin; one-fourth of a cup of macadamia nuts, raw and shelled; one cup raw sunflower seeds; ground fine using a meat grinder or high powered food processor and mixed) of which 1 pint is added to two and a half cups of purified water (lactated Ringer's for the first few days), 1 junior-size Gerber applesauce jar and two small jars of Gerber baby food (beechnut, banana, squash, garden vegetables, sweet potatoes, peaches, home-style creamed corn, carrots, and/or creamed spinach). This mixture is then heated to 39.7 to 41.4°C (103–106°F), stirred well for even heat, and tested with a thermometer prior to feeding.

[a]Adapted and reprinted from Abramson J, Speer BL, Thomsen JB: *The large macaws: their care, breeding and conservation*, Fort Bragg, 1996, Raintree publications, Fort Bragg CA; Zupreem - Shawnee KS; Florham Park, New Jersey.

E.1. Blue-and-Gold Macaw *(Ara ararauna)* Weight Records

	Weight (g)			Food Volume (mL)			Times Fed	
Age	Average	Minimum	Maximum	Sample Size	Minimum	Maximum	Minimum	Maximum
0	0	0	0	0	0	0	0	0
1	0	0	0	0	0	0	0	0
2	0	0	0	0	0	0	0	0
3	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0
5	63.0	56.0	70.0	2	5	10	12	16
6	62.7	50.0	78.0	3	5	10	12	16
7	83.3	55.0	116.0	4	8	10	12	16
8	106.3	66.0	132.0	6	9	30	8	16
9	142.0	80.0	225.0	9	10	40	6	16
10	165.5	94.0	225.0	11	14	40	6	12
11	180.4	85.0	250.0	12	10	60	6	12
12	214.7	88.0	302.0	19	10	60	4	16
13	244.0	102.0	360.0	22	10	60	4	16
14	282.6	120.0	375.0	27	15	70	4	12
15	315.3	136.0	425.0	32	15	70	4	12
16	348.7	168.0	460.0	33	20	70	4	12
17	382.2	192.0	510.0	34	25	70	4	8
18	418.6	214.0	535.0	35	35	70	4	8
19	456.4	250.0	600.0	35	40	70	4	8
20	487.3	270.0	610.0	36	40	70	4	6
21	530.1	324.0	680.0	36	40	70	4	6
22	556.4	356.0	650.0	36	60	70	4	6
23	587.2	388.0	696.0	37	60	70	4	4
24	623.9	420.0	775.0	37	60	90	4	4
25	653.4	448.0	795.0	37	60	90	4	4
26	686.9	504.0	825.0	38	60	90	4	4
27	720.9	544.0	855.0	38	60	100	4	4
28	754.1	558.0	890.0	38	60	100	4	4
29	787.4	590.0	938.0	38	60	100	4	4
30	817.7	610.0	938.0	38	60	100	4	4
31	849.9	665.0	975.0	38	60	100	4	4
32	877.1	705.0	1005.0	38	60	100	4	4
33	908.7	720.0	1030.0	38	60	100	4	4
34	937.0	745.0	1070.0	38	60	100	4	4
35	964.6	775.0	1135.0	38	70	100	4	4
36	993.4	795.0	1155.0	38	70	100	4	4
37	1020.8	845.0	1185.0	38	70	100	4	4
38	1044.5	890.0	1175.0	38	70	100	4	4
39	1072.5	920.0	1195.0	38	90	100	4	4
40	1086.8	960.0	1230.0	38	90	100	4	4
41	1110.2	1000.0	1240.0	38	90	100	4	4
42	1119.0	1004.0	1250.0	38	90	100	4	4
43	1143.6	1000.0	1300.0	38	90	100	4	4
44	1156.5	1044.0	1340.0	38	90	100	4	4
45	1169.9	1072.0	1300.0	38	90	100	4	4

E.1. Blue-and-Gold Macaw *(Ara ararauna)* Weight Records—cont'd

	Weight (g)			Food Volume (mL)			Times Fed	
Age	Average	Minimum	Maximum	Sample Size	Minimum	Maximum	Minimum	Maximum
46	1184.2	1080.0	1330.0	38	90	100	4	4
47	1187.4	1072.0	1330.0	38	90	100	4	4
48	1192.6	1066.0	1300.0	38	90	100	4	4
49	1200.2	1082.0	1330.0	38	90	100	4	4
50	1203.4	1095.0	1370.0	38	70	100	4	4
51	1206.7	1090.0	1355.0	37	70	100	4	4
52	1210.7	1100.0	1375.0	38	70	100	4	4
53	1217.0	1118.0	1355.0	38	70	100	4	4
54	1209.9	1100.0	1365.0	38	70	100	4	4
55	1204.2	1080.0	1370.0	38	70	100	3	4
56	1208.4	1085.0	1350.0	38	70	100	3	4
57	1198.4	1075.0	1305.0	37	60	100	2	4
58	1195.7	1060.0	1380.0	37	60	100	2	4
59	1197.5	1070.0	1340.0	38	60	100	2	4
60	1188.3	1065.0	1335.0	38	60	100	2	4
61	1185.8	1025.0	1304.0	36	60	100	2	4
62	1173.8	1000.0	1290.0	37	60	100	2	4
63	1170.3	1010.0	1285.0	36	60	100	2	4
64	1166.7	1005.0	1310.0	36	70	100	2	4
65	1155.6	1000.0	1300.0	36	70	100	2	4
66	1150.2	960.0	1290.0	36	70	100	2	4
67	1141.8	965.0	1265.0	37	70	100	2	4
68	1135.9	965.0	1248.0	36	70	100	2	4
69	1130.3	970.0	1235.0	37	60	100	2	4
70	1122.3	945.0	1240.0	36	60	100	2	4
71	1116.4	940.0	1248.0	36	60	70	2	4
72	1115.6	995.0	1235.0	34	60	70	2	4
73	1111.6	985.0	1255.0	34	60	70	2	4
74	1102.2	980.0	1234.0	33	60	70	2	4
75	1087.9	975.0	1222.0	33	60	70	2	4
76	1071.6	965.0	1185.0	33	60	70	1	4
77	1051.9	940.0	1170.0	33	60	70	1	4
78	1032.5	935.0	1160.0	33	60	70	1	3
79	1019.1	920.0	1150.0	32	60	70	1	3
80	1005.1	905.0	1125.0	31	60	70	1	3
81	992.1	865.0	1114.0	31	35	70	1	3
82	981.3	860.0	1110.0	32	35	70	1	3
83	970.2	885.0	1095.0	29	35	70	1	3
84	963.0	876.0	1085.0	29	35	70	1	2
85	959.4	885.0	1070.0	30	35	70	1	2
86	948.5	870.0	1100.0	29	35	70	1	2
87	940.4	870.0	1040.0	28	35	70	1	2
88	944.9	872.0	1020.0	28	35	70	1	2
89	938.6	860.0	1025.0	25	35	70	1	2
90	942.5	848.0	1030.0	26	35	70	1	2
91	937.8	848.0	1015.0	25	35	70	1	2
92	935.4	845.0	1012.0	25	35	70	1	2
93	941.0	850.0	1025.0	25	35	70	1	2
94	942.5	852.0	1005.0	22	35	70	1	2
95	947.5	800.0	1010.0	22	35	70	1	2
96	948.5	864.0	1010.0	22	35	70	1	2
97	935.8	864.0	1010.0	19	35	70	1	2
98	937.9	864.0	1030.0	18	35	70	1	2
99	943.6	862.0	1040.0	17	35	70	1	2
100	945.4	852.0	1030.0	16	35	70	1	2
101	943.9	840.0	1036.0	15	35	70	1	2
102	947.2	840.0	1056.0	14	35	70	1	2
103	943.9	856.0	1082.0	14	35	70	1	2
104	945.4	858.0	1062.0	14	35	70	1	2
105	944.2	862.0	1092.0	14	35	70	1	2

Continued

E.1. Blue-and-Gold Macaw *(Ara ararauna)* Weight Records—cont'd

	Weight (g)				Food Volume (mL)		Times Fed	
Age	Average	Minimum	Maximum	Sample Size	Minimum	Maximum	Minimum	Maximum
106	950.3	850.0	1108.0	14	35	70	1	2
107	949.8	848.0	1072.0	14	35	70	1	2
108	946.6	860.0	1070.0	14	35	70	1	2
109	952.5	862.0	1078.0	14	35	70	1	2
110	953.7	862.0	1062.0	14	35	70	1	2
111	960.1	864.0	1072.0	14	35	70	1	2
112	951.2	862.0	1050.0	13	35	70	1	2
113	952.6	844.0	1048.0	13	35	70	1	2
114	956.2	838.0	1038.0	12	35	70	1	2
115	960.2	854.0	1042.0	12	35	70	1	2
116	954.8	845.0	1025.0	12	35	70	1	2
117	962.4	840.0	1040.0	12	35	70	1	2
118	960.5	838.0	1060.0	11	35	70	1	2
119	953.1	820.0	1080.0	10	35	35	1	2
120	958.9	826.0	1070.0	9	35	35	1	1

E.2. Buffon's Macaw *(Ara ambiguus)* Weight Records

	Weight (g)							
Age	Average	Minimum	Maximum	Sample Size	Food Volume (mL)		Times Fed	
0	22.0	22.0	22.0	1	1	1	16	16
1	25.5	25.0	26.0	2	1	1	12	16
2	28.0	25.0	31.0	2	1	2	12	16
3	31.5	30.0	33.0	2	2	3	8	16
4	33.5	30.0	37.0	2	2	4	8	16
5	38.0	35.0	41.0	2	3	4	8	16
6	60.7	40.0	92.0	3	4	5	8	12
7	65.3	40.0	94.0	3	5	7	8	12
8	72.3	45.0	102.0	3	5	10	8	12
9	83.3	50.0	120.0	3	5	12	8	12
10	91.3	50.0	132.0	3	6	15	6	12
11	134.5	60.0	220.0	4	7	20	6	12
12	148.5	65.0	225.0	4	8	25	6	8
13	172.5	68.0	250.0	4	10	30	6	8
14	202.8	77.0	295.0	5	10	35	6	8
15	225.2	85.0	325.0	5	12	40	6	8
16	253.8	100.0	370.0	6	15	40	6	8
17	294.2	112.0	400.0	6	15	50	6	8
18	342.0	130.0	455.0	6	15	70	4	8
19	378.8	148.0	485.0	6	25	70	4	8
20	413.5	166.0	526.0	6	25	70	4	8
21	452.5	234.0	546.0	6	35	70	4	6
22	484.2	262.0	592.0	6	35	70	4	6
23	520.8	292.0	650.0	6	45	70	4	4
24	556.0	324.0	678.0	6	45	70	4	4
25	589.8	376.0	694.0	6	50	90	4	4
26	625.8	410.0	746.0	6	60	100	4	4
27	671.5	440.0	800.0	6	60	100	4	4
28	710.8	472.0	832.0	6	70	100	4	4
29	749.3	510.0	876.0	6	70	100	4	4
30	793.3	558.0	930.0	6	70	100	4	4
31	832.3	594.0	966.0	6	70	100	4	4
32	870.0	632.0	992.0	6	70	100	4	4
33	927.8	662.0	1040.0	6	70	100	4	4
34	959.0	728.0	1078.0	6	70	100	4	4
35	1001.2	780.0	1144.0	6	70	100	4	4
36	1021.2	804.0	1122.0	6	70	100	4	4

E.2. Buffon's Macaw *(Ara ambiguus)* Weight Records—cont'd

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
37	1054.7	866.0	1160.0	6	70	100	4	4
38	1079.5	870.0	1182.0	6	70	100	4	4
39	1109.2	948.0	1206.0	6	70	100	4	4
40	1129.8	970.0	1204.0	6	70	100	4	4
41	1149.7	995.0	1248.0	6	70	100	4	4
42	1170.7	1020.0	1260.0	6	70	100	4	4
43	1192.3	1050.0	1244.0	6	70	100	4	4
44	1217.7	1080.0	1290.0	6	70	100	4	4
45	1228.5	1120.0	1280.0	6	70	100	4	4
46	1250.5	1160.0	1325.0	6	70	100	4	4
47	1260.3	1145.0	1325.0	6	70	100	4	4
48	1294.0	1180.0	1366.0	6	70	100	4	4
49	1293.3	1190.0	1365.0	6	70	100	4	4
50	1295.8	1200.0	1370.0	6	100	100	4	4
51	1283.5	1166.0	1370.0	6	100	100	4	4
52	1283.0	1176.0	1380.0	6	100	100	4	4
53	1284.5	1180.0	1380.0	6	100	100	4	4
54	1303.0	1200.0	1390.0	6	100	100	4	4
55	1328.3	1240.0	1390.0	6	100	100	4	4
56	1315.8	1230.0	1400.0	6	100	100	4	4
57	1303.0	1218.0	1400.0	6	100	100	4	4
58	1321.0	1232.0	1430.0	6	70	100	4	4
59	1319.2	1256.0	1415.0	6	70	100	4	4
60	1294.8	1160.0	1400.0	6	70	100	4	4
61	1290.1	1184.0	1390.0	6	70	100	3	4
62	1276.0	1170.0	1370.0	6	70	100	3	4
63	1264.5	1162.0	1350.0	6	70	100	3	4
64	1268.2	1176.0	1345.0	6	70	100	3	4
65	1267.3	1166.0	1340.0	6	70	100	3	4
66	1255.0	1188.0	1330.0	6	70	100	3	4
67	1250.3	1138.0	1340.0	6	70	100	3	4
68	1248.8	1134.0	1350.0	6	70	100	3	4
69	1248.5	1186.0	1330.0	6	70	100	3	4
70	1234.5	1170.0	1310.0	6	70	100	3	4
71	1237.7	1150.0	1295.0	6	70	100	3	4
72	1227.8	1140.0	1310.0	6	70	70	3	4
73	1211.8	1130.0	1305.0	6	70	70	2	4
74	1213.7	1084.0	1310.0	6	70	70	2	4
75	1203.3	1120.0	1285.0	6	70	70	2	4
76	1191.3	1124.0	1295.0	6	70	70	2	4
77	1184.2	1110.0	1300.0	6	70	70	2	4
78	1182.5	1076.0	1280.00	6	70	70	2	4
79	1145.5	1074.0	1250.0	6	70	70	2	3
80	1130.2	1052.0	1240.0	6	70	70	2	3
81	1119.2	1052.0	1230.0	6	70	70	2	3
82	1103.2	1040.0	1225.0	6	70	70	2	3
83	1084.3	1020.0	1210.0	6	70	70	1	3
84	1051.7	1006.0	1170.0	6	70	70	1	3
85	1045.0	995.0	1140.0	6	70	70	1	3
86	1030.3	975.0	1130.0	6	70	70	1	3
87	1019.8	956.0	1115.0	6	70	70	1	3
88	1012.5	962.0	1075.0	6	70	70	1	3
89	1011.5	960.0	1065.0	6	70	70	1	2
90	1011.7	958.0	1070.0	6	70	70	1	2
91	993.5	958.0	1025.0	6	70	70	1	2
92	1005.0	962.0	1060.0	6	70	70	1	2
93	1010.3	956.0	1075.0	6	70	70	1	2
94	1009.3	960.0	1026.0	6	70	70	1	2
95	1026.7	965.0	1055.0	6	70	70	1	2
96	1009.0	974.0	1055.0	5	70	70	1	2

Continued

E.2. Buffon's Macaw *(Ara ambiguus)* Weight Records—cont'd

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
97	1009.5	974.0	1060.0	6	70	70	1	2
98	1007.3	955.0	1075.0	6	70	70	1	2
99	1019.4	946.0	1095.0	5	70	70	1	2
100	1015.4	928.0	1080.0	5	70	70	1	2
101	1009.6	934.0	1075.0	5	70	70	1	2
102	1017.6	960.0	1075.0	5	70	70	1	2
103	997.3	925.0	1070.0	6	35	70	1	2
104	1007.8	922.0	1070.0	5	35	70	1	2
105	1020.2	920.0	1080.0	5	35	70	1	2
106	1011.5	928.0	1085.0	4	35	70	1	2
107	1022.4	938.0	1090.0	5	35	70	1	2
108	1021.6	944.0	1100.0	5	35	100	1	2
109	992.0	944.0	1032.0	3	35	100	1	2
110	1020.3	995.0	1060.0	4	35	100	1	2
111	1013.0	965.0	1045.0	4	35	100	1	2
112	988.0	950.0	1030.0	3	35	100	1	2
113	1001.3	940.0	1050.0	4	35	100	1	2
114	1000.7	960.0	1062.0	3	35	100	1	2
115	991.0	960.0	1028.0	3	35	100	1	2
116	1012.8	964.0	1075.0	4	35	100	1	2
117	997.3	960.0	1040.0	3	35	70	1	2
118	999.0	965.0	1056.0	3	35	70	1	2
119	990.0	950.0	1044.0	3	35	70	2	2
120	983.7	935.0	1062.0	3	35	70	1	2

E.3. Green-Winged Macaw *(Ara chloropterus)* Weight Records

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
0	20.0	17.0	23.0	4	1	1	16	16
1	22.5	20.0	25.0	4	1	1	16	16
2	27.0	23.0	30.0	4	1	1	16	16
3	32.0	28.0	34.0	4	2	2	16	16
4	37.2	31.0	42.0	5	1	3	16	16
5	44.1	34.0	60.0	7	2	5	12	16
6	68.4	35.0	118.0	11	2	10	12	16
7	76.7	44.0	130.0	11	2	10	12	16
8	101.4	53.0	160.0	14	3	25	6	16
9	117.7	58.0	180.0	18	4	30	6	16
10	148.8	56.0	330.0	25	5	35	4	16
11	172.6	81.0	360.0	30	8	35	4	16
12	212.6	96.0	400.0	37	10	50	4	16
13	238.4	114.0	450.0	42	12	60	4	12
14	272.3	126.0	495.0	48	15	60	4	12
15	304.8	144.0	535.0	50	20	70	4	12
16	335.6	172.0	565.0	52	30	70	4	8
17	369.8	192.0	605.0	56	30	70	4	8
18	405.3	220.0	645.0	58	30	70	4	8
19	444.3	254.0	666.0	59	35	70	4	8
20	476.3	282.0	700.0	59	35	100	4	8
21	509.1	316.0	732.0	61	35	100	4	8
22	546.5	362.0	746.0	61	35	100	4	8
23	583.7	390.0	785.0	61	35	100	4	8
24	620.8	415.0	830.0	61	35	100	4	8
25	657.0	435.0	860.0	61	40	100	4	4
26	698.2	470.0	915.0	61	50	100	4	4
27	735.3	495.0	948.0	61	50	100	4	4

E.3. Green-Winged Macaw *(Ara chloropterus)* Weight Records—cont'd

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
28	769.7	515.0	990.0	61	50	100	4	4
29	810.3	555.0	1012.0	61	50	100	4	4
30	848.2	600.0	1080.0	61	60	100	4	4
31	885.0	660.0	1105.0	61	60	100	4	4
32	921.2	660.0	1134.0	61	60	100	4	4
33	956.9	705.0	1165.0	61	60	100	4	4
34	984.2	725.0	1198.0	61	60	100	4	4
35	1012.1	750.0	1224.0	61	60	100	4	4
36	1038.8	785.0	1270.0	61	60	100	4	4
37	1069.8	810.0	1300.0	61	80	100	4	4
38	1096.4	815.0	1326.0	61	80	100	4	4
39	1119.1	840.0	1355.0	61	80	100	4	4
40	1145.5	880.0	1380.0	61	80	100	4	4
41	1162.1	915.0	1365.0	61	80	120	4	4
42	1179.6	940.0	1380.0	61	80	120	3	4
43	1204.0	985.0	1426.0	61	80	120	3	4
44	1222.8	1005.0	1438.0	61	90	120	3	4
45	1240.6	1010.0	1470.0	61	90	120	3	4
46	1255.7	1050.0	1500.0	61	90	120	3	4
47	1269.6	1070.0	1512.0	60	90	120	3	4
48	1277.5	1060.0	1510.0	61	90	120	3	4
49	1290.5	1060.0	1530.0	61	90	120	3	4
50	1300.6	1115.0	1510.0	61	90	120	3	4
51	1312.4	1115.0	1575.0	61	90	120	3	4
52	1317.3	1120.0	1555.0	61	90	120	3	4
53	1324.6	1140.0	1560.0	61	90	120	3	4
54	1332.1	1140.0	1626.0	61	90	120	3	4
55	1339.6	1150.0	1575.0	61	70	120	3	4
56	1340.2	1160.0	1624.0	60	70	120	3	4
57	1346.8	1165.0	1618.0	60	70	120	3	4
58	1347.4	1155.0	1628.0	61	70	120	3	4
59	1349.6	1150.0	1658.0	61	70	120	3	4
60	1348.0	1165.0	1674.0	60	70	120	3	4
61	1348.4	1130.0	1652.0	60	70	120	3	4
62	1345.4	1130.0	1632.0	59	60	120	3	4
63	1340.2	1125.0	1650.0	58	60	120	3	4
64	1333.3	1125.0	1672.0	58	60	120	3	4
65	1329.0	1120.0	1674.0	58	60	120	3	4
66	1325.3	1130.0	1708.0	57	60	120	3	4
67	1324.8	1130.0	1680.0	56	60	120	3	4
68	1313.7	1120.0	1670.0	57	60	120	3	4
69	1301.1	1100.0	1660.0	57	60	120	2	4
70	1297.5	1100.0	1696.0	56	60	120	2	4
71	1292.2	1105.0	1690.0	53	60	100	2	4
72	1287.1	1095.0	1670.0	53	60	100	2	4
73	1277.5	1080.0	1652.0	53	60	100	2	4
74	1271.9	1060.0	1632.0	51	60	100	2	4
75	1262.2	1050.0	1650.0	52	60	100	2	4
76	1250.3	1040.0	1600.0	52	60	100	2	4
77	1242.9	1020.0	1550.0	48	60	100	2	4
78	1225.5	1024.0	1566.0	54	60	100	2	4
79	1220.5	1000.0	1544.0	49	60	70	2	4
80	1200.8	945.0	1498.0	48	60	70	2	4
81	1184.4	925.0	1470.0	49	60	70	2	4
82	1178.4	980.0	1518.0	46	60	70	2	3
83	1163.1	955.0	1440.0	49	60	70	1	3
84	1149.5	960.0	1430.0	48	60	70	2	3
85	1135.4	925.0	1410.0	48	60	70	1	3
86	1117.2	945.0	1355.0	48	60	70	1	3
87	1109.1	940.0	1360.0	46	60	70	1	3

Continued

E.3. Green-Winged Macaw *(Ara chloropterus)* Weight Records—cont'd

	Weight (g)							
Age	Average	Minimum	Maximum	Sample Size	Food Volume (mL)		Times Fed	
88	1092.1	945.0	1360.0	48	60	70	1	3
89	1088.8	900.0	1338.0	47	35	70	1	3
90	1079.4	935.0	1338.0	47	35	70	1	3
91	1067.8	940.0	1300.0	46	35	70	1	3
92	1065.0	925.0	1330.0	45	35	70	1	3
93	1060.7	930.0	1344.0	46	35	70	1	3
94	1058.9	935.0	1368.0	46	35	70	1	3
95	1055.5	915.0	1354.0	45	35	70	1	3
96	1055.1	920.0	1336.0	44	35	70	1	3
97	1050.1	930.0	1338.0	46	35	70	1	3
98	1051.4	930.0	1322.0	45	35	70	1	3
99	1041.4	910.0	1300.0	44	35	70	1	3
100	1050.9	910.0	1298.0	43	35	70	1	3
101	1051.6	900.0	1300.0	42	35	70	1	3
102	1048.1	900.0	1302.0	43	35	70	1	3
103	1057.2	905.0	1328.0	38	35	70	1	3
104	1060.5	905.0	1316.0	36	35	70	1	2
105	1062.4	855.0	1334.0	36	35	70	1	2
106	1069.4	875.0	1300.0	31	35	70	1	2
107	1072.0	915.0	1292.0	30	35	70	1	2
108	1077.0	915.0	1310.0	25	35	70	1	2
109	1068.1	910.0	1312.0	27	35	70	1	2
110	1077.7	915.0	1362.0	24	35	70	1	2
111	1071.0	920.0	1324.0	23	35	70	1	2
112	1060.0	935.0	1312.0	23	35	70	1	2
113	1058.9	900.0	1326.0	18	35	70	1	2
114	1050.6	900.0	1304.0	16	35	70	1	2
115	1047.2	925.0	1314.0	19	35	70	1	2
116	1052.2	935.0	1324.0	16	35	70	1	2
117	1051.8	915.0	1332.0	15	35	70	1	2
118	1024.1	925.0	1202.0	13	35	70	1	2
119	1041.6	945.0	1230.0	10	35	70	1	2
120	1048.4	930.0	1194.0	10	35	70	1	2

E.4. Hyacinth Macaw *(Anodorhynchus hyacinthinus)* Weight Records

	Weight (g)							
Age	Average	Minimum	Maximum	Sample Size	Food Volume (mL)		Times Fed	
0	23.4	22.0	26.0	22	0.5	1	16	16
1	25.3	22.0	29.0	22	1	1	16	16
2	28.8	26.0	33.0	23	1	2	12	16
3	34.0	29.0	41.0	25	1	3	12	16
4	39.6	35.0	52.0	25	1	4	12	16
5	46.1	40.0	63.0	28	2	5	12	16
6	53.6	43.0	78.0	29	3	6	12	16
7	64.1	49.0	94.0	29	3	7	12	16
8	74.8	55.0	110.0	31	4	10	12	16
9	88.5	60.0	140.0	31	5	13	12	16
10	104.3	68.0	160.0	32	6	20	8	16
11	120.6	75.0	188.0	32	8	20	8	16
12	137.2	85.0	218.0	33	8	25	8	16
13	160.1	106.0	250.0	33	10	30	7	16
14	180.3	118.0	280.0	34	14	35	6	16
15	206.8	130.0	312.0	34	14	50	4	12
16	237.5	140.0	330.0	33	15	60	4	12
17	267.5	165.0	380.0	33	20	70	4	12
18	302.7	185.0	430.0	33	25	70	4	12
19	332.2	210.0	460.0	33	25	70	4	12

E.4. Hyacinth Macaw *(Anodorhynchus hyacinthinus)* Weight Records—cont'd

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
20	363.3	235.0	496.0	33	30	70	4	8
21	403.5	260.0	556.0	33	35	70	4	8
22	434.4	290.0	582.0	33	35	70	4	8
23	468.9	325.0	624.0	33	35	70	4	6
24	501.8	375.0	632.0	34	35	70	4	6
25	533.4	390.0	646.0	34	35	70	4	6
26	562.5	435.0	734.0	33	60	70	4	4
27	604.6	470.0	756.0	33	60	90	4	4
28	638.3	515.0	800.0	33	60	90	4	4
29	670.7	540.0	855.0	33	60	90	4	4
30	706.8	575.0	870.0	33	60	100	4	4
31	745.8	615.0	908.0	33	60	100	4	4
32	783.4	655.0	978.0	33	60	100	4	4
33	821.4	680.0	1035.0	33	60	100	4	4
34	853.9	715.0	1050.0	33	60	100	4	4
35	887.2	755.0	1095.0	33	60	120	4	4
36	825.8	765.0	1135.0	33	60	120	4	4
37	958.8	810.0	1155.0	33	60	120	4	4
38	992.5	860.0	1205.0	33	60	140	4	4
39	1023.4	900.0	1255.0	33	70	140	3	4
40	1055.7	915.0	1280.0	33	70	140	3	4
41	1091.3	950.0	1330.0	33	70	140	3	4
42	1122.9	975.0	1385.0	32	90	140	3	4
43	1145.7	1000.0	1405.0	32	90	140	3	4
44	1171.5	1030.0	1400.0	32	90	140	3	4
45	1198.3	1070.0	1430.0	32	90	140	3	4
46	1218.5	1080.0	1450.0	32	90	140	3	4
47	1241.7	1110.0	1500.0	32	90	140	3	4
48	1265.3	1135.0	1490.0	32	90	140	3	4
49	1278.3	1160.0	1460.0	32	70	140	3	4
50	1298.5	1190.0	1470.0	32	70	140	3	4
51	1319.3	1215.0	1495.0	32	70	140	3	4
52	1329.1	1240.0	1505.0	32	90	140	3	4
53	1347.3	1250.0	1505.0	32	90	140	3	4
54	1367.3	1260.0	1525.0	32	90	140	3	4
55	1384.8	1285.0	1545.0	32	90	140	3	4
56	1396.1	1304.0	1560.0	32	90	140	3	4
57	1410.5	1315.0	1595.0	32	90	140	3	4
58	1432.9	1315.0	1590.0	32	90	140	3	4
59	1440.2	1334.0	1625.0	32	90	140	3	4
60	1448.7	1348.0	1635.0	32	90	140	3	4
61	1452.7	1354.0	1615.0	32	90	140	3	4
62	1463.3	1344.0	1640.0	32	90	140	3	4
63	1468.0	1290.0	1625.0	32	100	140	3	4
64	1475.3	1350.0	1650.0	32	100	140	3	4
65	1484.6	1375.0	1670.0	32	100	140	3	4
66	1487.2	1380.0	1650.0	21	100	140	3	4
67	1493.2	1370.0	1665.0	32	100	140	3	4
68	1490.0	1370.0	1675.0	32	100	140	3	4
69	1497.4	1386.0	1655.0	32	100	140	3	4
70	1500.8	1400.0	1665.0	32	100	140	3	4
71	1497.4	1380.0	1695.0	32	100	140	3	4
72	1497.2	1390.0	1670.0	31	100	140	3	4
73	1499.2	1386.0	1685.0	32	100	140	3	4
74	1497.5	1370.0	1640.0	31	100	140	3	4
75	1495.7	1400.0	1655.0	30	100	140	3	4
76	1501.0	1395.0	1640.0	30	100	140	3	4
77	1501.5	1385.0	1685.0	31	100	140	3	4
78	1488.8	1370.0	1620.0	29	100	140	3	4
79	1482.7	1365.0	1615.0	30	100	140	3	4

Continued

E.4. Hyacinth Macaw *(Anodorhynchus hyacinthinus)* Weight Records—cont'd

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
80	1485.6	1360.0	1625.0	30	100	140	3	4
81	1475.7	1350.0	1630.0	29	100	140	3	4
82	1470.7	1335.0	1635.0	28	100	140	3	4
83	1467.5	1345.0	1615.0	28	100	140	3	4
84	1454.8	1330.0	1615.0	30	100	140	3	4
85	1445.7	1330.0	1605.0	30	100	140	3	4
86	1439.8	1315.0	1595.0	28	100	140	2	4
87	1423.8	1295.0	1585.0	29	100	140	2	4
88	1417.1	1295.0	1575.0	29	100	140	2	4
89	1404.6	1270.0	1600.0	28	100	140	2	4
90	1396.1	1270.0	1545.0	28	100	140	2	4
91	1381.2	1225.0	1550.0	29	100	140	2	4
92	1370.9	1235.0	1555.0	29	100	140	2	4
93	1355.7	1220.0	1550.0	29	100	140	2	4
94	1342.9	1200.0	1545.0	28	100	140	2	4
95	1326.8	1185.0	1500.0	29	100	140	2	4
96	1303.2	1185.0	1460.0	28	100	140	2	3
97	1285.4	1165.0	1435.0	28	100	140	2	3
98	1269.0	1165.0	1425.0	28	100	140	2	3
99	1267.9	1175.0	1440.0	28	100	140	2	3
100	1254.3	1125.0	1420.0	28	100	140	2	3
101	1234.9	1110.0	1390.0	27	100	120	2	3
102	1220.0	1085.0	1385.0	26	90	120	2	3
103	1214.3	1100.0	1375.0	26	100	120	2	3
104	1203.5	1085.0	1345.0	27	90	140	2	3
105	1205.7	1080.0	1335.0	26	70	140	2	3
106	1204.6	1085.0	1325.0	25	70	140	2	3
107	1203.7	1090.0	1315.0	27	70	140	2	3
108	1200.0	1060.0	1320.0	25	70	140	2	3
109	1196.3	1060.0	1305.0	24	70	140	2	3
110	1200.8	1085.0	1300.0	25	70	140	2	3
111	1198.3	1060.0	1320.0	25	60	140	2	3
112	1193.8	1040.0	1335.0	24	70	140	2	3
113	1191.5	1060.0	1330.0	24	70	140	2	3
114	1199.0	1065.0	1330.0	26	60	140	2	3
115	1196.8	1080.0	1330.0	23	60	140	2	3
116	1197.0	1060.0	1335.0	23	70	140	2	3
117	1198.4	1055.0	1325.0	24	60	140	2	3
118	1198.5	1060.0	1320.0	24	60	140	2	3
119	1197.0	1065.0	1315.0	22	70	140	2	3
120	1203.0	1075.0	1310.0	21	70	140	2	3
121	1211.7	1065.0	1330.0	23	60	140	2	3
122	1196.3	1075.0	1315.0	20	70	140	2	3
123	1201.5	1050.0	1310.0	21	70	100	2	3
124	1204.1	1070.0	1325.0	20	70	100	2	3
125	1208.9	1065.0	1305.0	20	70	100	2	3
126	1202.6	1010.0	1320.0	20	70	100	2	3
127	1208.1	1040.0	1320.0	20	70	140	2	3
128	1217.1	1055.0	1355.0	22	60	140	2	3
129	1205.6	1045.0	1315.0	19	70	140	2	3
130	1219.9	1075.0	1370.0	22	60	140	2	4
131	1215.6	1075.0	1345.0	20	70	140	2	4
132	1219.0	1110.0	1335.0	20	70	140	2	4
133	1221.4	1110.0	1355.0	22	60	140	2	4
134	1224.0	1110.0	1360.0	20	70	140	2	4
135	1223.8	1090.0	1355.0	21	60	140	2	4
136	1229.0	1070.0	1350.0	23	60	140	1	4
137	1218.4	1065.0	1355.0	20	70	140	1	4
138	1219.4	1065.0	1345.0	20	70	140	1	4
139	1227.8	1080.0	1345.0	20	70	140	1	4
140	1228.6	1065.0	1345.0	20	70	100	1	4
141	1230.7	1065.0	1345.0	21	70	100	1	4

E.4. Hyacinth Macaw *(Anodorhynchus hyacinthinus)* Weight Records—cont'd

	Weight (g)							
Age	Average	Minimum	Maximum	Sample Size	Food Volume (mL)		Times Fed	
142	1234.8	1080.0	1350.0	20	60	100	1	4
143	1239.2	1120.0	1365.0	20	60	100	1	4
144	1242.4	1120.0	1375.0	21	70	100	2	4
145	1239.9	1140.0	1400.0	20	70	100	2	4
146	1243.3	1105.0	1390.0	20	60	100	1	4
147	1241.1	1135.0	1410.0	19	70	100	2	4
148	1234.5	1100.0	1400.0	20	70	100	2	2
149	1248.4	1140.0	1400.0	19	70	100	2	2
150	1253.1	1135.0	1450.0	19	70	100	2	2
151	1251.3	1140.0	1435.0	20	70	100	2	2
152	1246.1	1122.0	1425.0	19	70	100	2	2
153	1263.5	1130.0	1470.0	20	70	100	2	2
154	1260.5	1120.0	1475.0	18	70	100	2	2
155	1264.9	1140.0	1480.0	17	70	100	1	2
156	1264.2	1174.0	1485.0	18	70	100	1	2
157	1269.0	1172.0	1480.0	16	70	100	1	2
158	1265.7	1180.0	1470.0	16	35	100	1	2
159	1268.9	1170.0	1470.0	17	35	100	1	2
160	1259.1	1165.0	1470.0	17	35	70	1	2
161	1269.5	1160.0	1475.0	17	35	70	1	2
162	1276.8	1160.0	1460.0	16	35	70	1	2
163	1267.4	1121.0	1455.0	15	35	70	1	2
164	1269.1	1175.0	1445.0	15	35	70	1	2
165	1277.6	1200.0	1425.0	15	35	70	1	2
166	1281.2	1205.0	1470.0	15	35	70	1	2
167	1286.3	1180.0	1440.0	16	35	70	1	2
168	1274.7	1200.0	1440.0	15	35	70	1	2
169	1278.6	1200.0	1390.0	16	35	70	1	2
170	1284.3	1190.0	1410.0	16	35	70	1	2
171	1277.2	1190.0	1445.0	15	35	70	1	2
172	1283.1	1190.0	1465.0	15	35	70	1	2
173	1280.5	1195.0	1455.0	14	35	70	1	2
174	1288.8	1192.0	1455.0	16	35	70	1	2
175	1281.1	1195.0	1410.0	14	35	70	1	2
176	1278.3	1180.0	1430.0	14	35	70	1	2
177	1278.0	1175.0	1410.0	15	35	70	1	2
178	1277.1	1165.0	1440.0	14	35	70	1	2
179	1259.0	1155.0	1410.0	12	35	70	1	2
180	1255.5	1150.0	1415.0	11	35	35	1	1

E.5. Military Macaw *(Ara militaris)* Weight Records

	Weight (g)							
Age	Average	Minimum	Maximum	Sample Size	Food Volume (mL)		Times Fed	
0	0	0	0	0	0	0	0	0
1	0	0	0	0	0	0	0	0
2	0	0	0	0	0	0	0	0
3	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0
5	0	0	0	0	0	0	0	0
6	0	0	0	0	0	0	0	0
7	0	0	0	0	0	0	0	0
8	123.0	116.0	130.0	2	10	15	12	12
9	134.0	134.0	134.0	2	15	15	7	12
10	158.0	146.0	176.0	3	15	25	6	12
11	187.5	164.0	206.0	4	15	25	6	12
12	206.0	186.0	224.0	4	20	30	6	12
13	240.0	214.0	268.0	6	25	35	6	8

Continued

E.5. Military Macaw *(Ara militaris)* Weight Records—cont'd

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
14	267.8	248.0	286.0	8	25	40	6	8
15	301.4	280.0	320.0	9	25	40	6	8
16	333.0	304.0	366.0	9	35	70	4	8
17	350.0	228.0	422.0	11	25	70	4	8
18	393.6	276.0	470.0	11	35	70	4	8
19	413.3	256.0	500.0	12	35	70	4	8
20	449.3	304.0	538.0	12	35	70	4	8
21	465.8	320.0	540.0	13	40	70	4	8
22	510.7	366.0	594.0	13	40	70	4	8
23	536.8	388.0	608.0	13	40	70	4	8
24	570.6	436.0	645.0	13	60	70	4	4
25	607.9	458.0	665.0	13	60	70	4	4
26	635.1	498.0	715.0	13	60	70	4	4
27	665.7	536.0	745.0	13	60	70	4	4
28	695.3	570.0	770.0	13	60	70	4	4
29	725.1	615.0	805.0	13	60	90	4	4
30	747.4	616.0	830.0	13	60	90	4	4
31	772.2	672.0	865.0	13	60	90	4	4
32	800.5	712.0	915.0	13	60	90	4	4
33	818.2	732.0	900.0	13	70	90	4	4
34	840.9	752.0	930.0	13	70	90	4	4
35	865.2	762.0	945.0	13	70	90	4	4
36	879.5	788.0	950.0	13	70	90	4	4
37	892.8	804.0	960.0	13	70	90	4	4
38	911.4	830.0	996.0	13	70	90	4	4
39	931.6	852.0	1032.0	13	70	90	4	4
40	938.9	860.0	1034.0	13	70	90	4	4
41	957.9	866.0	1040.0	13	70	90	4	4
42	964.9	895.0	1032.0	13	70	90	4	4
43	977.5	920.0	1050.0	13	70	90	4	4
44	987.2	910.0	1074.0	13	70	90	4	4
45	994.4	910.0	1080.0	13	70	90	4	4
46	1001.8	932.0	1092.0	13	70	90	4	4
47	1004.4	930.0	1078.0	13	70	90	4	4
48	1006.5	930.0	1086.0	13	70	90	4	4
49	1014.5	940.0	1110.0	13	70	90	4	4
50	1015.5	950.0	1095.0	13	70	90	4	4
51	1009.4	926.0	1100.0	13	70	90	4	4
52	1008.2	938.0	1098.0	13	70	90	4	4
53	1013.8	950.0	1118.0	13	70	90	3	4
54	1010.0	950.0	1118.0	13	70	70	3	4
55	1007.3	938.0	1106.0	13	70	90	3	4
56	1008.5	935.0	1104.0	13	70	90	3	4
57	1001.3	945.0	1090.0	13	70	90	3	4
58	1003.4	925.0	1104.0	13	70	90	3	4
59	995.5	920.0	1104.0	13	70	90	3	4
60	999.7	910.0	1116.0	13	70	70	3	4
61	987.7	900.0	1092.0	13	70	70	2	4
62	987.1	930.0	1090.0	13	70	70	2	4
63	977.6	920.0	1070.0	13	70	70	2	4
64	966.3	915.0	1054.0	13	70	70	2	4
65	954.2	896.0	1062.0	13	70	70	2	4
66	948.6	888.0	1034.0	13	70	70	2	4
67	935.8	874.0	1025.0	13	70	70	2	4
68	930.4	875.0	1020.0	13	30	70	2	3
69	925.2	864.0	1025.0	13	50	70	2	3
70	915.5	850.0	1010.0	13	50	70	2	3
71	906.8	835.0	980.0	13	50	70	2	3
72	895.8	825.0	970.0	13	50	70	2	3
73	883.1	800.0	955.0	13	35	70	2	3
74	875.3	795.0	960.0	13	35	70	2	3
75	856.2	770.0	945.0	13	35	70	2	3

E.5. Military Macaw *(Ara militaris)* Weight Records—cont'd

	Weight (g)							
Age	Average	Minimum	Maximum	Sample Size	Food Volume (mL)		Times Fed	
76	840.2	755.0	942.0	13	35	70	2	3
77	830.7	750.0	934.0	13	35	70	2	2
78	815.2	715.0	890.0	13	20	70	1	2
79	797.4	715.0	870.0	13	20	70	1	2
80	786.6	700.0	864.0	13	20	70	1	2
81	778.8	705.0	858.0	13	20	60	1	2
82	771.4	695.0	870.0	13	20	60	1	2
83	771.6	690.0	842.0	13	20	60	1	2
84	766.8	685.0	840.0	13	20	60	1	2
85	766.8	680.0	838.0	13	20	60	1	2
86	769.2	675.0	845.0	13	20	60	1	2
87	770.9	675.0	836.0	13	20	60	1	2
88	773.7	670.0	864.0	13	20	35	1	2
89	776.5	675.0	840.0	13	20	35	1	2
90	774.8	680.0	862.0	12	20	35	1	2
91	767.8	670.0	836.0	12	20	35	1	2
92	770.0	682.0	844.0	12	20	35	1	2
93	775.4	700.0	846.0	11	20	35	1	2
94	772.7	688.0	838.0	11	20	35	1	2
95	773.9	710.0	844.0	11	20	35	1	2
96	778.8	715.0	845.0	10	20	35	1	2
97	781.1	700.0	870.0	10	20	35	1	2
98	779.0	710.0	860.0	10	20	35	1	2
99	775.3	714.0	850.0	10	20	35	1	2
100	776.9	710.0	855.0	9	20	35	1	2
101	770.8	702.0	830.0	9	20	35	1	2
102	771.3	706.0	825.0	8	35	35	1	1
103	778.3	702.0	875.0	7	35	35	1	1
104	762.3	700.0	820.0	6	35	35	1	1
105	783.8	694.0	840.0	4	0	0	0	0
106	770.0	678.0	836.0	3	0	0	0	0
107	774.0	694.0	828.0	3	0	0	0	0
108	767.3	680.0	838.0	3	0	0	0	0
109	790.0	712.0	850.0	3	0	0	0	0
110	778.7	700.0	838.0	3	0	0	0	0
111	788.7	712.0	872.0	3	0	0	0	0
112	784.7	710.0	848.0	3	0	0	0	0
113	783.3	700.0	838.0	3	0	0	0	0
114	777.3	712.0	838.0	3	0	0	0	0
115	781.7	714.0	830.0	3	0	0	0	0
116	796.0	716.0	866.0	3	0	0	0	0
117	796.0	720.0	868.0	3	0	0	0	0
118	788.3	715.0	860.0	3	0	0	0	0
119	792.0	712.0	862.0	3	0	0	0	0
120	800.0	714.0	852.0	3	0	0	0	0

E.6. Red-Fronted Macaw *(Ara rubrogenys)* Weight Records

	Weight (g)							
Age	Average	Minimum	Maximum	Sample Size	Food Volume (mL)		Times Fed	
0	14.5	14.0	15.0	2	0.5	0.5	16	16
1	16.5	16.0	17.0	2	0.5	0.7	16	16
2	19.0	18.0	20.0	2	0.7	0.9	16	16
3	22.5	22.0	23.0	2	1	1.5	16	16
4	28.5	27.0	30.0	2	1	2	16	16
5	33.0	30.0	36.0	2	2	2	16	16
6	37.5	35.0	40.0	2	2	4	16	16
7	45.0	40.0	50.0	2	2	5	12	16

Continued

E.6. Red-Fronted Macaw *(Ara rubrogenys)* Weight Records—cont'd

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
8	42.5	45.0	60.0	2	2	7	8	16
9	63.0	52.0	74.0	2	4	9	8	16
10	73.5	59.0	88.0	2	6	9	6	16
11	80.0	70.0	90.0	2	8	20	6	12
12	102.0	90.0	114.0	2	10	20	6	12
13	126.0	114.0	138.0	2	10	20	6	16
14	138.0	128.0	148.0	2	12	30	6	8
15	161.0	158.0	164.0	2	15	30	4	8
16	179.0	170.0	188.0	2	15	35	4	6
17	200.0	186.0	214.0	2	25	35	4	6
18	218.0	202.0	234.0	2	25	35	4	4
19	240.0	222.0	258.0	2	35	40	4	4
20	276.0	262.0	290.0	2	35	40	4	4
21	291.5	275.0	308.0	2	35	50	4	4
22	320.0	292.0	348.0	2	50	60	4	4
23	341.0	304.0	378.0	2	50	60	4	4
24	368.0	344.0	392.0	2	50	60	4	4
25	377.0	348.0	406.0	2	60	60	4	4
26	398.0	374.0	422.0	2	60	60	4	4
27	404.0	368.0	440.0	2	60	70	4	4
28	442.0	408.0	476.0	2	60	70	4	4
29	457.0	430.0	484.0	2	60	70	4	4
30	468.0	452.0	484.0	2	60	70	4	4
31	475.0	444.0	506.0	2	60	70	4	4
32	489.5	465.0	514.0	2	60	70	4	4
33	493.0	466.0	520.0	2	60	70	4	4
34	508.0	480.0	536.0	2	60	70	4	4
35	515.0	488.0	542.0	2	60	70	4	4
36	545.0	528.0	562.0	2	60	70	4	4
37	567.0	542.0	592.0	2	60	70	4	4
38	579.0	548.0	610.0	2	60	70	4	4
39	577.0	554.0	600.0	2	60	70	4	4
40	577.0	550.0	604.0	2	60	70	4	4
41	584.0	568.0	600.0	2	60	70	4	4
42	597.0	572.0	622.0	2	60	70	4	4
43	603.0	578.0	628.0	2	60	70	4	4
44	595.0	572.0	618.0	2	60	70	4	4
45	610.0	584.0	636.0	2	60	70	4	4
46	620.0	598.0	642.0	2	60	70	4	4
47	624.0	592.0	656.0	2	60	70	4	4
48	617.0	602.0	632.0	2	60	70	4	4
49	637.0	630.0	644.0	2	60	70	4	4
50	632.0	624.0	640.0	2	60	70	4	4
51	622.0	612.0	632.0	2	60	70	3	4
52	620.5	615.0	626.0	2	50	60	3	4
53	612.5	610.0	615.0	2	50	60	3	4
54	594.0	590.0	598.0	2	50	60	3	4
55	597.0	590.0	604.0	2	50	60	3	3
56	603.0	600.0	606.0	2	50	60	3	3
57	589.0	588.0	590.0	2	50	60	3	3
58	596.0	592.0	600.0	2	50	60	3	3
59	596.0	590.0	602.0	2	50	60	2	3
60	597.0	590.0	604.0	2	50	60	2	3
61	576.0	572.0	580.0	2	50	60	2	3
62	565.0	550.0	580.0	2	35	60	2	3
63	571.0	564.0	578.0	2	35	60	2	3
64	553.0	532.0	574.0	2	35	35	3	3
65	552.0	522.0	582.0	2	35	35	3	3
66	563.0	526.0	600.0	2	35	35	2	3
67	538.0	506.0	570.0	2	35	35	2	2
68	537.0	504.0	570.0	2	35	35	2	2
69	536.0	510.0	562.0	2	35	35	2	2

E.6. Red-Fronted Macaw *(Ara rubrogenys)* Weight Records—cont'd

	Weight (g)						
Age	Average	Minimum	Maximum	Sample Size	Food Volume (mL)	Times Fed	
70	506.5	505.0	508.0	2	35	35	2	2
71	501.0	500.0	502.0	2	35	35	1	2
72	492.0	484.0	500.0	2	35	35	1	2
73	477.0	472.0	482.0	2	20	35	1	2
74	479.0	472.0	486.0	2	20	35	1	2
75	469.0	458.0	480.0	2	20	35	1	2
76	467.0	444.0	490.0	2	20	35	1	2
77	458.5	435.0	482.0	2	20	35	1	1
78	567.0	434.0	500.0	2	20	35	1	1
79	459.0	430.0	488.0	2	20	35	1	1
80	460.0	430.0	490.0	2	20	35	1	1
81	456.0	428.0	484.0	2	20	35	1	1
82	461.0	434.0	488.0	2	20	20	1	1
83	469.0	430.0	508.0	2	20	20	1	1
84	451.0	420.0	482.0	2	20	20	1	1
85	157.0	432.0	482.0	2	20	20	1	1
86	463.0	442.0	484.0	2	20	20	1	1
87	461.0	444.0	478.0	2	20	20	1	1
88	454.0	434.0	474.0	2	20	20	1	1
89	436.0	436.0	436.0	1	20	20	1	1
90	434.0	434.0	434.0	1	20	20	1	1
91	430.0	430.0	430.0	1	20	20	1	1
92	435.0	435.0	435.0	1	20	20	1	1
93	440.0	440.0	440.0	1	20	20	1	1
94	445.0	445.0	445.0	1	20	20	1	1
95	445.0	445.0	445.0	1	20	20	1	1
96	440.0	440.0	440.0	1	20	20	1	1
97	435.0	435.0	435.0	1	20	20	1	1
98	445.0	445.0	445.0	1	20	20	1	1
99	440.0	440.0	440.0	1	20	20	1	1
100	435.0	435.0	435.0	1	20	20	1	1
101	440.0	440.0	440.0	1	20	20	1	1
102	440.0	440.0	440.0	1	20	20	1	1
103	445.0	445.0	445.0	1	20	20	1	1
104	450.0	450.0	450.0	1	0	0	0	0
105	440.0	440.0	440.0	1	0	0	0	0
106	440.0	440.0	440.0	1	0	0	0	0
107	440.0	440.0	440.0	1	0	0	0	0
108	435.0	435.0	435.0	1	0	0	0	0

E.7. Scarlet Macaw *(Ara macao)* Weight Records

	Weight (g)						
Age	Average	Minimum	Maximum	Sample Size	Food Volume (mL)	Times Fed	
0	19.7	15.0	24.0	6	0.5	1	16	16
1	210.6	17.0	29.0	8	1	1	16	16
2	25.8	18.0	35.0	9	1	2	12	16
3	30.2	21.0	38.0	11	1	3	8	16
4	35.2	22.0	48.0	12	1	5	8	16
5	41.2	26.0	56.0	12	1	7	8	16
6	51.3	30.0	88.0	13	2	10	8	16
7	63.8	33.0	90.0	15	3	10	8	16
8	77.4	39.0	112.0	18	4	10	8	16
9	95.6	45.0	152.0	20	4	15	8	16
10	114.1	51.0	170.0	21	5	20	8	16
11	139.5	61.0	224.0	26	5	20	6	16
12	169.4	68.0	300.0	32	6	35	4	12
13	202.4	80.0	315.0	36	10	35	4	12

Continued

E.7. Scarlet Macaw *(Ara macao)* Weight Records—cont'd

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
14	234.4	94.0	370.0	37	10	60	4	12
15	264.3	116.0	400.0	38	15	70	4	12
16	297.7	146.0	454.0	39	15	70	4	12
17	333.4	174.0	480.0	39	20	70	4	12
18	368.7	200.0	510.0	40	20	70	4	12
19	409.4	228.0	552.0	41	20	70	4	12
20	442.3	244.0	588.0	41	30	70	4	8
21	476.0	258.0	620.0	42	30	90	4	8
22	512.9	285.0	672.0	42	30	90	4	8
23	548.7	340.0	700.0	42	35	90	4	8
24	580.9	340.0	726.0	42	35	90	4	8
25	614.2	360.0	766.0	42	35	100	4	8
26	651.5	380.0	826.0	42	35	100	4	8
27	686.2	425.0	846.0	42	35	100	4	8
28	721.3	445.0	900.0	42	50	100	4	6
29	756.5	485.0	940.0	42	50	100	4	6
30	789.2	515.0	994.0	42	60	100	4	4
31	818.8	520.0	1000.0	42	60	100	4	4
32	847.9	575.0	1010.0	42	60	100	4	4
33	875.2	605.0	1050.0	42	60	100	4	4
34	901.5	635.0	1080.0	42	60	100	4	4
35	927.1	655.0	1155.0	42	60	100	4	4
36	948.6	670.0	1170.0	42	60	100	4	4
37	970.7	695.0	1170.0	42	60	100	4	4
38	990.4	705.0	1190.0	42	60	100	4	4
39	1011.5	720.0	1200.0	42	60	120	3	4
40	1032.2	740.0	1225.0	42	70	120	3	4
41	1045.9	760.0	1255.0	42	60	120	3	4
42	1066.3	765.0	1250.0	42	60	120	3	4
43	1080.9	825.0	1260.0	42	60	120	3	4
44	1098.9	820.0	1305.0	42	60	120	3	4
45	1107.7	840.0	1315.0	42	60	120	3	4
46	1118.0	855.0	1350.0	42	60	120	3	4
47	1125.6	855.0	1365.0	42	60	120	3	4
48	1133.8	860.0	1390.0	42	60	120	3	4
49	1139.6	860.0	1440.0	42	60	120	3	4
50	1151.3	940.0	1415.0	42	60	120	3	4
51	1152.1	950.0	1450.0	42	60	120	3	4
52	1155.5	965.0	1425.0	42	60	120	3	4
53	1157.5	995.0	1425.0	42	60	120	3	4
54	1154.9	975.0	1490.0	42	60	120	3	4
55	1158.2	970.0	1445.0	41	60	120	3	4
56	1150.2	960.0	1435.0	42	60	120	3	4
57	1150.5	965.0	1435.0	42	60	120	3	4
58	1145.1	970.0	1435.0	42	60	100	3	4
59	1142.7	965.0	1450.0	42	60	100	2	4
60	1141.2	955.0	1435.0	42	60	100	2	4
61	1135.6	970.0	1445.0	42	60	100	2	4
62	1131.0	960.0	1450.0	42	60	100	2	4
63	1120.3	950.0	1450.0	42	60	100	2	4
64	1116.7	920.0	1470.0	42	60	100	2	4
65	1103.1	905.0	1445.0	42	60	100	2	4
66	1099.8	915.0	1145.0	42	60	100	1	4
67	1084.5	890.0	1420.0	42	60	100	1	4
68	1079.5	890.0	1440.0	42	60	100	1	4
69	1064.5	870.0	1415.0	42	60	100	1	4
70	1057.0	865.0	1405.0	42	60	100	1	4
71	1046.0	850.0	1420.0	41	60	100	1	4
72	1036.7	830.0	1380.0	42	60	100	1	4
73	1021.2	815.0	1395.0	42	60	70	1	4
74	1007.7	810.0	1380.0	42	35	70	1	4

E.7. Scarlet Macaw *(Ara macao)* Weight Records—cont'd

Age	Weight (g)			Sample Size	Food Volume (mL)		Times Fed	
	Average	Minimum	Maximum					
75	1005.1	815.0	1355.0	42	35	70	1	4
76	983.9	780.0	1300.0	40	35	70	1	3
77	972.0	770.0	1265.0	41	35	70	1	3
78	955.1	760.0	1255.0	41	35	70	1	3
79	939.8	755.0	1230.0	41	35	70	1	2
80	926.9	750.0	1205.0	41	35	70	1	2
81	914.2	745.0	1150.0	1	30	70	1	2
82	902.5	705.0	1135.0	41	0	70	1	2
83	893.9	695.0	1130.0	41	30	70	1	2
84	883.4	685.0	1120.0	40	30	70	1	2
85	876.7	670.0	1110.0	40	30	70	1	2
86	868.9	655.0	1085.0	40	30	70	1	2
87	866.9	650.0	1090.0	39	30	70	1	2
88	867.1	655.0	1075.0	39	30	70	1	2
89	860.8	660.0	1070.0	39	30	70	1	2
90	859.4	660.0	1065.0	9	30	70	1	2
91	858.5	665.0	1040.0	3	30	70	1	2
92	858.4	665.0	1060.0	3	30	70	1	2
93	858.8	655.0	1070.0	38	30	70	1	2
94	854.9	650.0	1060.0	38	30	70	1	2
95	858.6	650.0	1125.0	37	30	70	1	2
96	856.7	660.0	1120.0		35	70	1	2
97	858.9	685.0	1135.0	37	35	70	1	2
98	854.6	675.0	1115.0	36	35	70	1	2
99	864.2	650.0	1110.0	34	35	70	1	2
100	855.6	665.0	1120.0	31	35	70	1	2
101	866.7	665.0	1135.0	29	35	70	1	2
102	865.6	665.0	1165.0	27	35	70	1	2
103	864.2	670.0	1160.0	27	35	70	1	2
104	863.0	690.0	1170.0	22	35	70	1	2
105	870.4	725.0	1145.0	21	35	35	1	2
106	872.3	715.0	1140.0	18	35	35	1	2
107	861.5	710.0	995.0	16	35	35	1	2
108	874.6	745.0	954.0	15	20	35	1	1
109	870.4	715.0	995.0	11	20	35	1	1
110	892.4	785.0	990.0	11	20	35	1	1
111	895.9	780.0	985.0	9	20	35	1	1
112	864.2	710.0	1000.0	9	20	35	1	1
113	881.8	770.0	1000.0	9	35	35	1	1
114	874.2	785.0	1015.0	8	35	35	1	1
115	873.3	770.0	1005.0	9	35	35	1	1
116	881.3	770.0	995.0	8	0	0	0	0
117	881.1	770.0	1000.0	7	0	0	0	0
118	897.3	860.0	1000.0	7	0	0	0	0
119	897.2	850.0	1015.0	7	0	0	0	0
120	891.3	840.0	1000.0	6	0	0	0	0

E.8. Blue-Throated Macaw *(Ara glaucogularis)* Weight Records

Age	Weight (g)			Sample Size
	Average	Minimum	Maximum	
0	23.3	21.0	25.0	3
1	24.6	21.0	30.0	5
2	28.0	22.0	35.0	6
3	33.3	26.0	44.0	6
4	41.5	31.0	59.0	6
5	49.5	38.0	72.0	6

Continued

E.8. Blue-Throated Macaw *(Ara glaucogularis)* Weight Records—cont'd

Age	Weight (g)			Sample Size
	Average	Minimum	Maximum	
6	63.5	46.0	96.0	6
7	77.5	52.0	116.0	6
8	92.0	60.0	139.0	6
9	110.8	72.0	166.0	6
10	126.0	80.0	182.0	6
11	148.5	92.0	222.0	6
12	165.5	103.0	246.0	6
13	191.2	118.0	284.0	6
14	209.0	134.0	306.0	6
15	235.0	142.0	340.0	6
16	254.5	165.0	364.0	6
17	276.7	182.0	386.0	6
18	306.0	206.0	420.0	6
19	337.0	230.0	458.0	6
20	361.3	252.0	488.0	6
21	392.3	274.0	550.0	6
22	421.8	302.0	568.0	6
23	447.7	330.0	600.0	6
24	467.0	346.0	628.0	6
25	498.0	368.0	658.0	6
26	519.5	385.0	678.0	6
27	542.3	406.0	706.0	6
28	560.8	428.0	716.0	6
29	589.5	463.0	740.0	6
30	607.0	484.0	756.0	6
31	629.3	502.0	760.0	6
32	646.6	524.0	786.0	6
33	662.2	542.0	800.0	6
34	681.5	579.0	818.0	6
35	707.7	602.0	826.0	6
36	715.8	618.0	824.0	6
37	732.2	623.0	860.0	6
38	738.7	664.0	848.0	6
39	737.0	650.0	856.0	6
40	731.8	654.0	822.0	6
41	740.0	689.0	816.0	6
42	761.5	724.0	810.0	6
43	757.2	712.0	800.0	6
44	763.0	724.0	800.0	6
45	770.8	730.0	821.0	6
46	763.3	710.0	800.0	6
47	765.8	716.0	825.0	6
48	768.7	712.0	840.0	6
49	770.7	718.0	856.0	6
50	774.0	724.0	859.0	6
51	774.8	725.0	862.0	6
52	780.2	734.0	857.0	6
53	778.0	712.0	852.0	6
54	780.2	710.0	882.0	6
55	782.0	720.0	860.0	6
56	772.0	712.0	838.0	6
57	775.2	700.0	821.0	6
58	764.2	708.0	832.0	6
59	770.8	716.0	835.0	6
60	760.2	712.0	826.0	6
61	743.0	702.0	780.0	6
62	745.0	705.0	790.0	6
63	737.5	675.0	800.0	6
64	722.5	672.0	810.0	6
65	722.0	674.0	808.0	6

E.8. Blue-Throated Macaw *(Ara glaucogularis)* Weight Records—cont'd

	Weight (g)			
Age	Average	Minimum	Maximum	Sample Size
66	717.0	674.0	800.0	6
67	712.5	650.0	785.0	6
68	709.7	662.0	800.0	6
69	709.7	666.0	800.0	6
70	704.7	642.0	800.0	6
71	701.3	653.0	792.0	6
72	698.5	652.0	787.0	5
73	689.2	626.0	783.0	6
74	688.5	602.0	774.0	5
75	683.2	602.0	766.0	6
76	707.3	670.0	761.0	3
77	702.0	660.0	756.0	3
78	702.7	656.0	762.0	3
79	703.7	652.0	768.0	3
80	689.3	648.0	728.0	3
81	680.7	646.0	740.0	3
82	660.7	632.0	706.0	3
83	652.3	614.0	707.0	3
84	657.3	624.0	708.0	3
85	655.7	605.0	720.0	3
86	645.0	586.0	711.0	3
87	624.0	580.0	702.0	3
88	621.0	585.0	688.0	3
89	621.3	590.0	704.0	3
90	623.3	582.0	697.0	3
91	622.0	584.0	690.0	3
92	623.0	583.0	688.0	3
93	624.3	582.0	686.0	3
94	620.0	592.0	656.0	3
95	625.0	597.0	668.0	3
96	625.3	602.0	666.0	3
97	625.3	596.0	672.0	3
98	599.0	590.0	608.0	2

F. Growth Data of Selected Species of Birds of Prey

COMMON NAME LANNER FALCON

DIET—UNLESS OTHERWISE STATED, THE DIET/PROTOCOL USED FOR THESE SPECIMENS IS THE ICBP GENERIC REARING PROTOCOL[a]

Scientific Name *Falco biarmicus*

Age (Days)	*Chick Weight (g)*			*Chick Body Weight (%)*			*Chick Intake (g)*		
Day	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*
1	16.70	26.93	33.50	2.87	7.07	14.10	0.80	2.41	6.20
2	21.00	28.21	34.80	11.38	18.61	45.35	4.80	7.04	14.10
3	25.20	31.82	39.20	9.40	22.10	30.85	5.50	9.51	12.30
4	28.60	37.31	47.80	12.78	26.83	39.76	7.70	13.12	20.00
5	34.60	43.81	54.30	13.81	29.68	38.86	13.30	17.73	26.40
6	39.30	54.85	71.80	21.36	31.99	59.66	13.90	24.05	39.80
7	46.00	66.82	88.20	20.63	32.53	44.35	16.80	28.45	37.90
8	63.30	79.23	105.00	17.10	23.75	25.43	24.50	30.48	45.70
9	73.10	91.52	123.40	32.34	36.35	40.63	37.00	46.52	65.10
10	95.50	113.85	155.50	23.47	28.19	30.71	42.30	46.73	57.60
11	113.50	133.35	176.10	22.24	25.68	30.34	38.80	53.05	71.80
12	130.00	131.45	132.90	16.54	17.94	19.34	31.50	36.70	41.90
13	136.30	142.90	149.50	26.96	34.17	41.38	55.50	62.75	70.00

COMMON NAME MERLIN

DIET—UNLESS OTHERWISE STATED, THE DIET/PROTOCOL USED FOR THESE SPECIMENS IS THE ICBP GENERIC REARING PROTOCOL[a]

Scientific Name *Falco columbarius*

Age (Days)	*Chick Weight (g)*			*Chick Body Weight (%)*			*Chick Intake*		
Day	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*
1	11.80	12.87	13.80	2.33	6.73	11.76	0.30	1.24	2.90
2	11.70	13.23	14.70	4.92	12.00	16.08	1.20	2.66	4.40
3	11.60	14.11	17.80	6.74	13.89	22.01	2.80	3.49	4.70
4	12.50	15.16	18.30	15.12	22.96	30.36	3.40	5.15	7.20
5	13.60	17.01	21.40	16.36	26.10	36.41	4.00	6.89	11.00
6	15.60	19.84	23.70	25.00	29.98	34.83	6.30	8.14	10.40
7	18.40	24.65	29.10	13.45	21.49	31.52	3.80	7.40	10.70
8	22.60	29.84	34.40	8.14	16.91	21.18	2.80	6.28	8.50
9	27.00	34.02	42.40	9.67	18.68	28.95	4.10	7.42	12.20
10	32.20	36.20	40.40	12.78	17.19	23.27	5.00	7.20	12.00
11	45.10	45.10	45.10	17.07	17.07	17.07	10.20	10.20	10.20
12	52.40	52.40	52.40	27.48	27.48	27.48	19.20	19.20	19.20

COMMON NAME PYGMY FALCON

DIET—UNLESS OTHERWISE STATED, THE DIET/PROTOCOL USED FOR THESE SPECIMENS IS THE ICBP GENERIC REARING PROTOCOL[a]

Scientific Name *Polihierax semitorquatus*

Age (Days)	*Chick Weight (g)*			*Chick Body Weight (%)*			*Chick Intake*		
Day	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*
1	5.4	5.785714	6.10	7.41	11.04	16.95	0.50	1.03	1.40
2	5.6	6.1	6.40	10.00	20.75	33.87	1.10	1.81	2.90
3	6.1	6.814286	7.30	16.92	26.39	38.36	1.20	2.64	3.90
4	7.2	7.96	9.00	19.44	26.51	32.89	2.40	3.22	4.20
5	8	9.32	10.30	21.25	28.67	39.81	3.30	4.12	5.50
6	9.2	10.675	12.10	23.36	29.84	34.71	4.10	4.88	6.00
7	11.3	12.775	15.10	10.48	23.14	29.20	3.10	5.25	7.30
8	13	14.275	16.70	20.77	25.51	31.91	3.90	5.75	7.80
9	14.9	16	17.50	20.39	30.58	46.29	4.90	7.00	8.90
10	16.2	18.7	24.70	9.88	23.48	39.64	2.70	6.28	8.50
11	16.1	21.66667	26.80	20.81	24.17	26.87	5.30	7.20	9.50
12	20.1	25.33333	31.80	12.58	19.01	24.07	7.90	10.97	16.80
13	28.7	31.55	34.40	14.63	15.46	16.28	7.10	8.35	9.60
14	31.9	34.85	37.80	8.47	13.32	18.18	3.20	6.20	9.20
15	41	41	41.00	6.10	6.10	6.10	2.50	2.50	2.50
16	42.9	42.9	42.90	7.69	7.69	7.69	3.30	3.30	3.30
17	43.4	43.4	43.40	8.99	8.99	8.99	3.90	3.90	3.90

F. Growth Data of Selected Species of Birds of Prey—cont'd

COMMON NAME SAKER FALCON

DIET—UNLESS OTHERWISE STATED, THE DIET/PROTOCOL USED FOR THESE SPECIMENS IS THE ICBP GENERIC REARING PROTOCOL[a]

Scientific Name *Falco cherrug*

Age (Days)	*Chick Weight (g)*			*Chick Body Weight (%)*			*Chick Intake*		
Day	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*
1	26.70	31.81	36.40	2.25	7.90	17.71	0.60	3.70	8.80
2	25.90	32.91	39.80	11.00	19.79	29.36	4.90	8.94	15.40
3	28.10	37.49	48.00	9.02	24.67	36.16	4.60	13.08	19.20
4	30.10	44.94	59.00	13.96	27.67	40.82	6.70	18.06	33.40
5	33.70	52.60	70.90	24.63	33.61	42.16	10.70	24.15	34.40
6	39.90	30.62	88.40	22.56	14.44	37.57	11.50	30.91	58.20
7	62.80	74.21	111.80	12.26	29.60	42.86	7.70	35.83	54.50
8	88.70	103.51	132.00	23.15	28.22	37.37	32.20	42.53	59.10
9	104.90	123.30	153.20	17.49	26.89	36.06	24.10	44.29	78.00
10	112.40	137.06	179.20	11.22	28.81	38.08	9.30	50.54	65.30
11	136.50	152.28	167.00	13.49	28.44	43.65	20.60	63.55	102.80
12	160.30	189.30	218.30	11.92	18.19	24.46	19.10	49.35	79.60
13	229.00	229.00	229.00	26.42	26.42	26.42	83.30	83.30	83.30
14	258.60	258.60	258.60	25.99	25.99	25.99	86.10	86.10	86.10
15	287.50	287.50	287.50	33.77	33.77	33.77	32.00	32.00	32.00
16	328.50	328.50	328.50	34.16	34.16	34.16	147.00	147.00	147.00
17	374.00	374.00	374.00	12.65	12.65	12.65	47.30	47.30	47.30

COMMON NAME TAWNY EAGLE

DIET—UNLESS OTHERWISE STATED, THE DIET/PROTOCOL USED FOR THESE SPECIMENS IS THE ICBP GENERIC REARING PROTOCOL[a]

Scientific Name *Aquila rapax*

Age (Days)	*Chick Weight (g)*			*Chick Body Weight % Increase*			*Chick Intake*		
Day	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*	*Min*	*Mean*	*Max*
1	39.50	59.42	65.90	1.39	5.72	20.19	0.90	4.32	16.10
2	41.30	49.26	70.40	6.49	14.10	25.76	6.90	12.82	20.90
3	48.20	54.41	77.50	8.60	16.63	26.39	8.60	17.99	25.10
4	55.70	61.17	88.00	14.16	19.51	29.72	15.30	22.83	33.30
5	67.60	68.76	101.50	16.56	20.75	32.05	19.70	29.23	38.30
6	79.00	77.52	120.80	21.38	22.97	40.65	22.80	35.73	59.20
7	92.50	104.43	159.20	17.90	22.01	35.26	30.30	38.80	52.50
8	109.40	118.59	175.00	20.11	23.69	31.81	35.30	48.91	67.60
9	131.10	164.80	199.80	6.81	19.15	27.33	18.30	41.98	76.80
10	147.50	174.88	235.70	19.39	25.39	29.84	45.30	49.93	58.30
11	187.20	214.10	252.40	25.80	39.35	58.16	76.00	108.87	165.20
12	288.60	288.60	288.60	15.14	15.14	15.14	60.70	60.70	60.70
13	311.10	311.10	311.10	16.23	16.23	16.23	69.20	69.20	69.20

[a]ICBP Generic rearing protocol (abbreviated):
In the first few days following hatch, chicks are reared in a still air incubator or brooder running at no lower than 35°C (95.0°F). Temperatures are reduced by 1°C per day until the secondary down feathers erupt and birds are able to thermoregulate or are returned to (foster) parents. Supplemental heating will generally no longer be required after 10 days with all but the smallest of species as long as the brooder room is set at 21°C (70°F) and not lower. Chicks are housed in containers that are designed to hold the legs neatly tucked under them to prevent splay leg. To prevent buildup of feces, containers should be washed daily. Unless dealing with chicks that weigh less than 10 g at hatch (e.g., African pygmy falcons), it is best to leave the first feed for at least 12 hours (or 24 hours for larger species), if the chick is not showing signs of hunger as overfeeding can easily occur. Chicks that look distended with fluid should be left without food until the stomach is flaccid and soft to the touch. For most species, a mixed diet of finely minced or chopped meat, consisting of skinned, gutted quail, rabbit, rats of which the head, wings/feet, tail, and gizzard (if applicable) are removed, and/or skinned mice or day-old-chicks, is suitable (Note that bones should be fed to all but the youngest of chicks as this is vital for ensuring adequate mineral intake and prevent metabolic bone disease). A good multivitamin supplement in powder form (e.g., Nutrobal, Vetark) and a probiotic supplement (e.g., Avipro Pediatric, Vetark) are added to the feed of most raptors (Note: Do not exceed recommended doses!), while in vultures, addition of digestive enzymes is common to aid in digestion. Using forceps, tiny pieces of minced food are offered to the chick, which should raise its head to accept feeding. Owls need to have the side of their beaks touched to take the food, whereas falcons may need to have food placed in the beak and their upper palate touched to stimulate them to close their beak and swallow. New-world vultures, in contrast, naturally feed by putting their beaks inside the parents' beak to receive regurgitated food, which can be mimicked by gently encircling the beak with finger and thumb (behind which food is presented with a forceps). All other diurnal raptors will generally attempt snatching food from a forceps that is slowly brought toward the beak at about eye level. Damping the food is recommended to facilitate feeding and prevent dehydration (Note that young raptors do not drink until they have left the nest and can reach water for themselves but do obtain additional fluids with their feed from the oral secretions that are dripped onto the food by their parents). Four feeds per day are generally sufficient to stimulate adequate growth, with the exception of the smallest chicks (<10 g). Weighing before and after every feeding is recommended to adequately evaluate food intake and growth. Casting materials to allow the young bird to form pellets for regurgitation can be introduced in the food by stopping skinning of day-old chicks at about 10 to 14 days of age for small to medium-sized birds and up to 1 month for larger birds. An exception to this rule is the small birds such as merlins, which do better without casting material until they are feathering up. Ringing of the young is recommended at 10 to 14 days of age for most species. Whenever possible, chicks should be returned to their (foster) parents at 10 to 12 days of each, provided that they are looking healthy, eating well, and have been ringed; if this is not possible, contact and handling by the human caretakers should be minimized and involve the use of puppets shaped to the color and build of the adult to prevent severe imprinting. Whenever returning a young bird to its (foster) parents, it is necessary to intensely monitor them for at least several hours, up to 1 or 2 days, to ensure that everything is going well, and continue the monitoring throughout the rearing period to ensure that everything is going well.
Full version of the ICBP protocol is available at: https://www.handrearingresourcecenter.com/.
Data as shown were generously provided by Jemima Parry-Jones at the International Centre for Birds of Prey (ICBP), Newent, Gloucestershire, UK, and analyzed by Katie Wellfair.

G. Feeding Schedule and Expected Weight Gain in Selected Columbiformes

G.1. Feeding Schedule for Rock Pigeons *(Columba livia)*

Home-made diet for squabs: mix one hard-boiled egg yolk (mashed) with 3 tablespoons of mixed baby cereal, 3 tablespoons of oatmeal, and 3 tablespoons of cornmeal.[a] Alternatively, a mixture of Emeraid (Lafeber Company, Cornell, IL) mixed with a psittacine neonatal formula (e.g., Kaytee Exact, Chilton, WI) can be used in a ratio of 1:1. Consistency of the food should gradually be increased[b]:

- Hatch to Day 2: 1 part formula to 6 parts water
- Days 2 to 5: 1 part formula to 2 or 3 parts water
- Day 5 to weaning: 1 part formula to $1^1/_3$ to 2 parts water.

Check crop and feed when nearly empty. Food expires after 1 hour: discard and mix fresh at each feeding. NOTE: DO NOT FEED COLD FOOD!

WEIGHT (g)	APROXIMATE AMOUNT (mL)	APPROXIMATE FREQUENCY OF FEEDINGS[c]
20	1–2	16
30	2.5–3.5	12
40	3–5	8
50	4–6	8
75	5–8	8
100	8–10	8
125	11–13	8
150	14–16	8
175	17–18	5–6
200	18–20	5–6
225	18–20	5–6
250+[d]	18–20	3–4

[a]Bewig M, Mitchell MM: Wildlife. In Mitchell M, Tully TN, editors: *Manual of exotic pet practice*, 2009, Elsevier, Frisco, CA, pp 493–529 (chapter 19).
[b]Pigeon and Dove Rescue UK: What to feed. https:/ /www.pigeonrescue.sirtobyservices.com/caring-for-a-baby-pigeon/whattofeed/
[c]Normally there is no need to feed chicks at night.
[d]Often, tube-feeding is no longer necessary.
Adapted from: Eilertsen N, Purdin G: Pigeons and doves. In: Duerr RS, Gage LJ, editors: *Hand-rearing birds*, ed 2, 2020, Wiley Blackwell, Hoboken, New Jersey, pp 437–456.

G.2. Feeding Schedule for Mourning Doves *(Zenaida macroura)*

Home-made diet for squabs: mix one hard-boiled egg yolk (mashed) with 3 tablespoons of mixed baby cereal, 3 tablespoons of oatmeal, and 3 tablespoons of cornmeal.[a] Alternatively, a mixture of Emeraid (Lafeber Company, Cornell, IL) mixed with a psittacine neonatal formula (e.g., Kaytee Exact, Chilton, WI) can be used in a ratio of 1:1. Consistency of the food should gradually be increased[b]:

- Hatch to Day 2: 1 part formula to 6 parts water
- Days 2 to 5: 1 part formula to 2 or 3 parts water
- Day 5 to weaning: 1 part formula to $1^1/_3$ to 2 parts water.

Check crop and feed when nearly empty. Food expires after 1 hour: discard and mix fresh at each feeding. NOTE: DO NOT FEED COLD FOOD!

WEIGHT (g)	APPROXIMATE AMOUNT (mL)	APPROXIMATE FREQUENCY OF FEEDINGS[c]
10	1	16–24
15	1.5–2	12–16
20	1.5–2.5	8
25	2–3	8
30	2.5–3.5	8
35	4	8
40–50	5	5–6
50–60	6	5–6
60–70	6–7	4–5
70–80[d]	8	3–4
90+[d]	9–10	3

[a]Bewig M, Mitchell MM: Wildlife. In Mitchell M, Tully TN, editors: *Manual of exotic pet practice*, 2009, Elsevier, Frisco, CA, pp 493–529 (chapter 19).
[b]Pigeon and Dove Rescue UK: What to feed. https:/ /www.pigeonrescue.sirtobyservices.com/caring-for-a-baby-pigeon/whattofeed/
[c]Normally there is no need to feed chicks at night.
[d]Often, tube-feeding is no longer necessary at this weight.
Adapted from: Eilertsen N, Purdin G: Pigeons and doves. In: Duerr RS, Gage LJ, editors: *Hand-rearing birds*, ed 2, 2020, Wiley Blackwell, Hoboken, New Jersey, pp 437–456.

G.3. Feeding Schedule for Eurasian Collared Doves *(Streptopelia decaocto)*

Home-made diet for squabs: mix one hard-boiled egg yolk (mashed) with 3 tablespoons of mixed baby cereal, 3 tablespoons of oatmeal, and 3 tablespoons of cornmeal.[a] Alternatively, a mixture of Emeraid (Lafeber Company, Cornell, IL) mixed with a psittacine neonatal formula (e.g., Kaytee Exact, Chilton, WI) can be used in a ratio of 1:1. Consistency of the food should gradually be increased[b]:

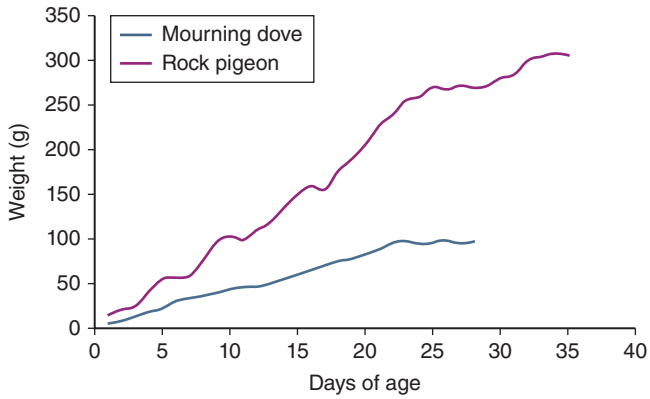
- Hatch to Day 2: 1 part formula to 6 parts water
- Days 2 to 5: 1 part formula to 2 or 3 parts water
- Day 5 to weaning: 1 part formula to $1^{1}/_{3}$ to 2 parts water.

Check crop and feed when nearly empty. Food expires after 1 hour: discard and mix fresh at each feeding. NOTE: DO NOT FEED COLD FOOD!

WEIGHT (g)	APPROXIMATE AMOUNT (mL)	APPROXIMATE FREQUENCY OF FEEDINGS[c]
15	1.5–2	16
25	2–3	12
35	4	8
40	4–5	8
45	4.5–5.5	8
50	5–6	8
55	5.5–6.5	8
60	6–7	5–6
65	6.5–7.5	5–6
70	7–8	5–6
75	8.5–9.5	5–6
80	9–10	5–6
85	9.5–10.5	5–6
90[d]	10–11	5–6
100[d]	11–12	5–6
110[d]	12–13	5–6
120[d]	13–14	5–6
130[d]	14–15	3
140[d]	15	3

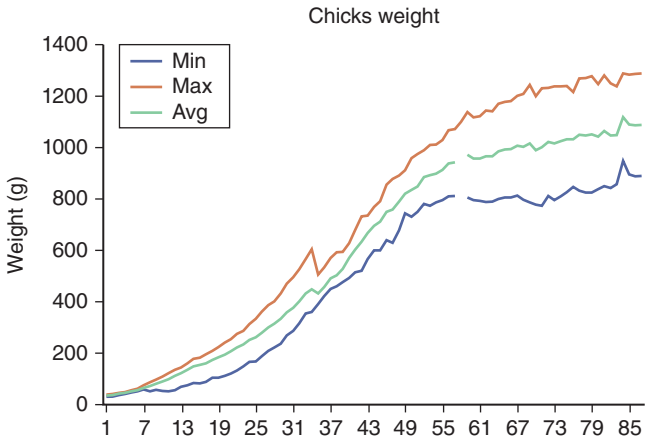
[a]Bewig M, Mitchell MM: Wildlife. In Mitchell M, Tully TN, editors: *Manual of exotic pet practice*, 2009, Elsevier, Frisco, CA, pp 493–529 (chapter 19).
[b]Pigeon and Dove Rescue UK: What to feed. https:/ /www.pigeonrescue.sirtobyservices.com/caring-for-a-baby-pigeon/whattofeed/
[c]Normally there is no need to feed chicks at night.
[d]Often, tube-feeding is no longer necessary at this weight.
Adapted from: Eilertsen N, Purdin G: Pigeons and doves. In: Duerr RS, Gage LJ, editors: *Hand-rearing birds*, ed 2, 2020, Wiley Blackwell, Hoboken, New Jersey, pp 437–456.
Information courtesy Wildlife Care Association, Sacramento, CA.

G.4. Growth Curves for Rock Pigeons and Mourning Doves



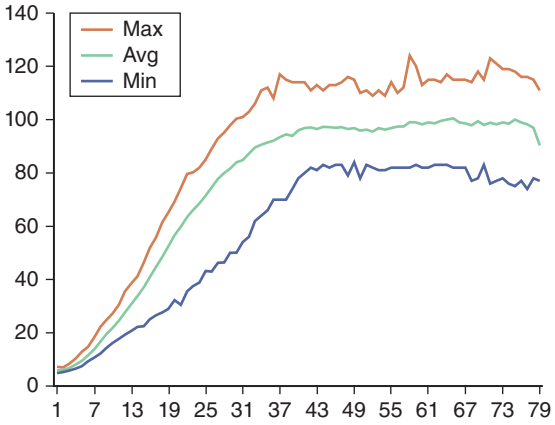
Note: Pigeon and dove chicks typically reach adult weight by 3 to 4 weeks of age. Chicks should gain weight daily until they have finished growing. Chicks should be weighed before the first feed of the day to avoid confounding from the amount of food in the crop. Images reprinted from: Eilertsen N, Purdin G. (2020). Chapter 27: Pigeons and doves. In: Duerr RS, Gage LJ, editors: *Hand-rearing birds*, ed 2, Hoboken, 2020, Wiley Blackwell Publishing, pp 437–456.

G.5. Growth Curve for Sclater's Crowned Pigeon (*Goura sclaterii; n* = 9)



Data courtesy of: Mandai Wildlife Group, Bird Paradise, Breeding and Research Centre.

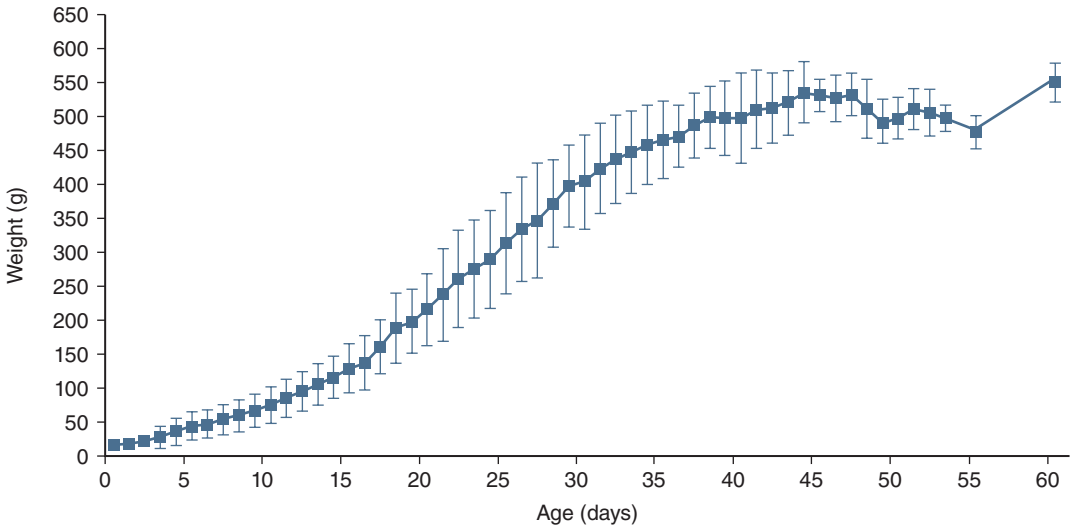
G.6. Growth Curve for Santa Cruz Ground Doves (*Pampusana sanctaecrucis; n* = 15)



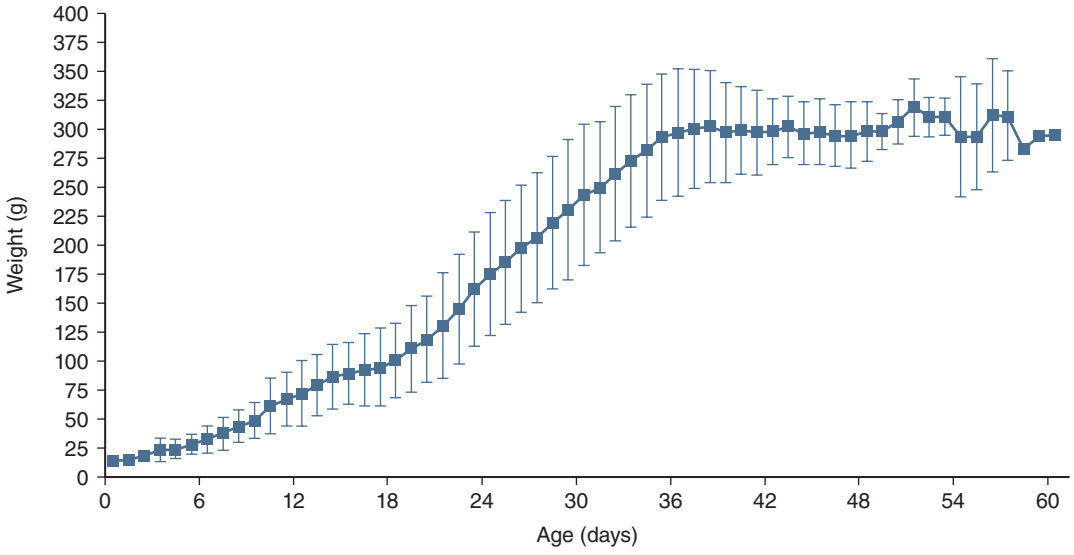
Data courtesy of: Mandai Wildlife Group, Bird Paradise, Breeding and Research Centre.

H. Growth curves for selected *Ramphastos* species

Toco toucan (*Ramphastos toco*)

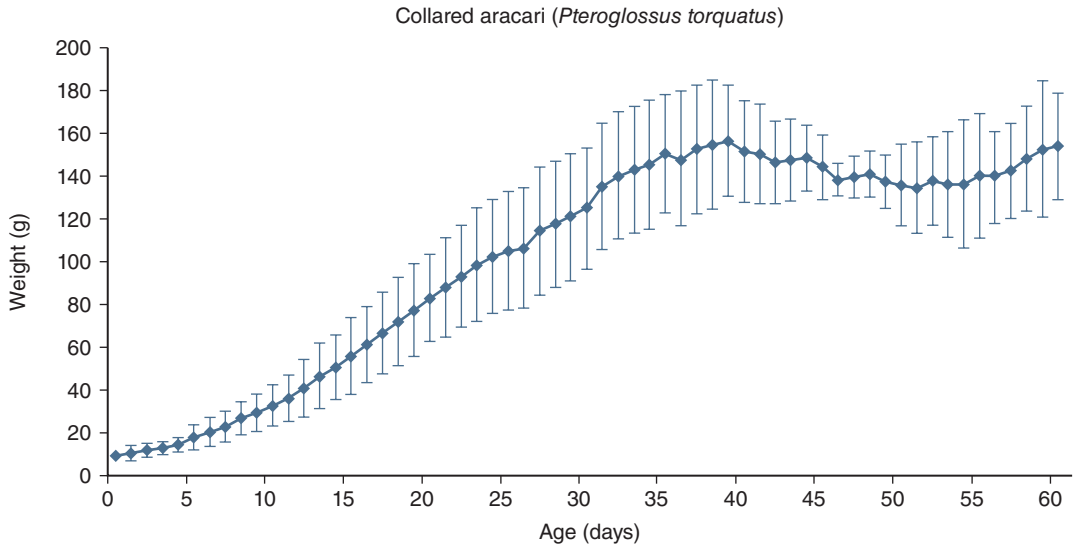


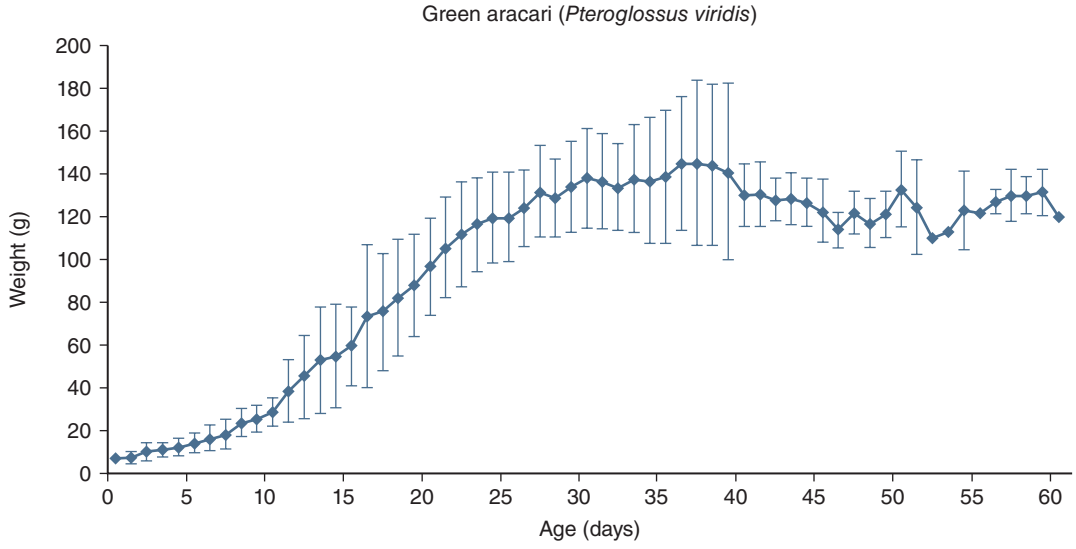
Keel-billed toucan (*Ramphastos sulfuratus*)



Continued

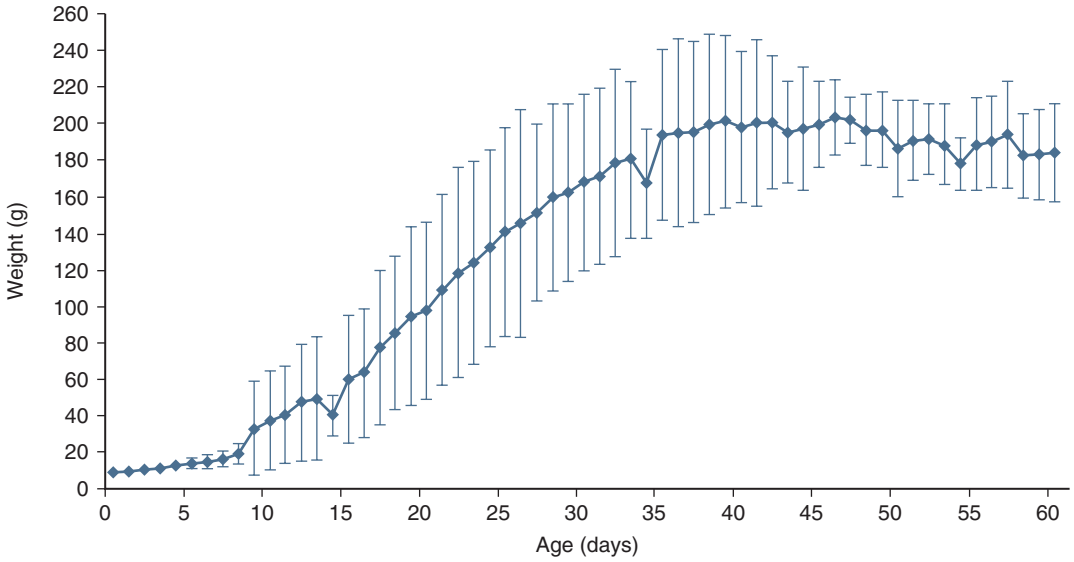
H. Growth curves for selected *Ramphastos* species—cont'd



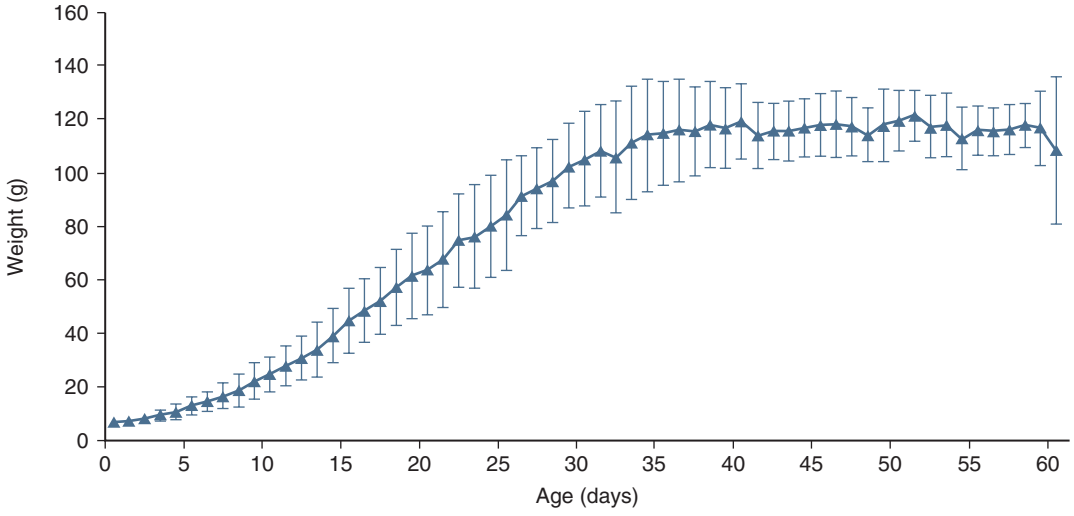


H. Growth curves for selected *Ramphastos* species—cont'd

Chestnut-eared aracari (*Pteroglossus castanotis*)



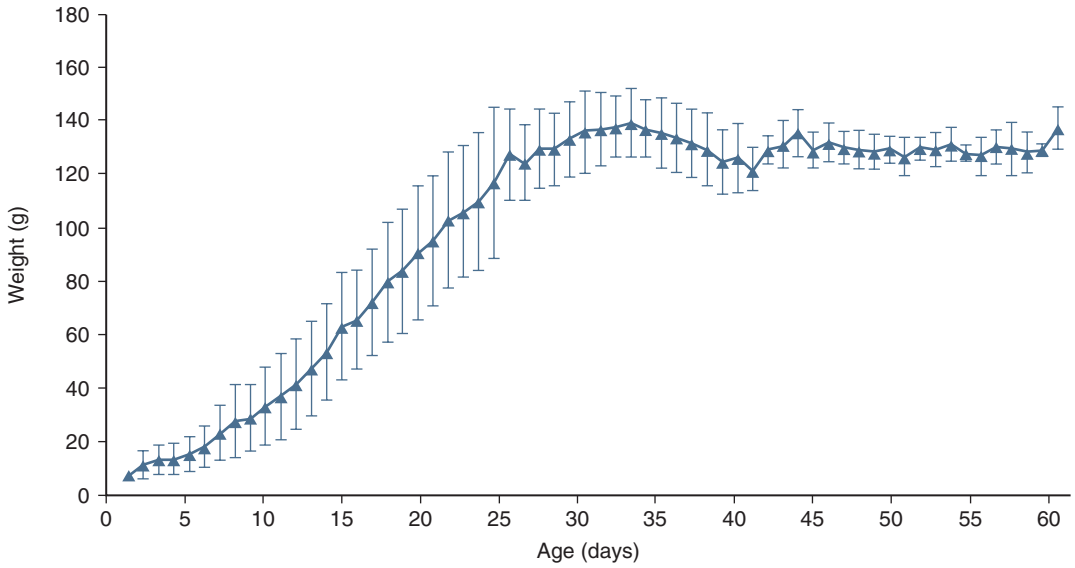
Saffron toucanet (*Pteroglossus baillon*)



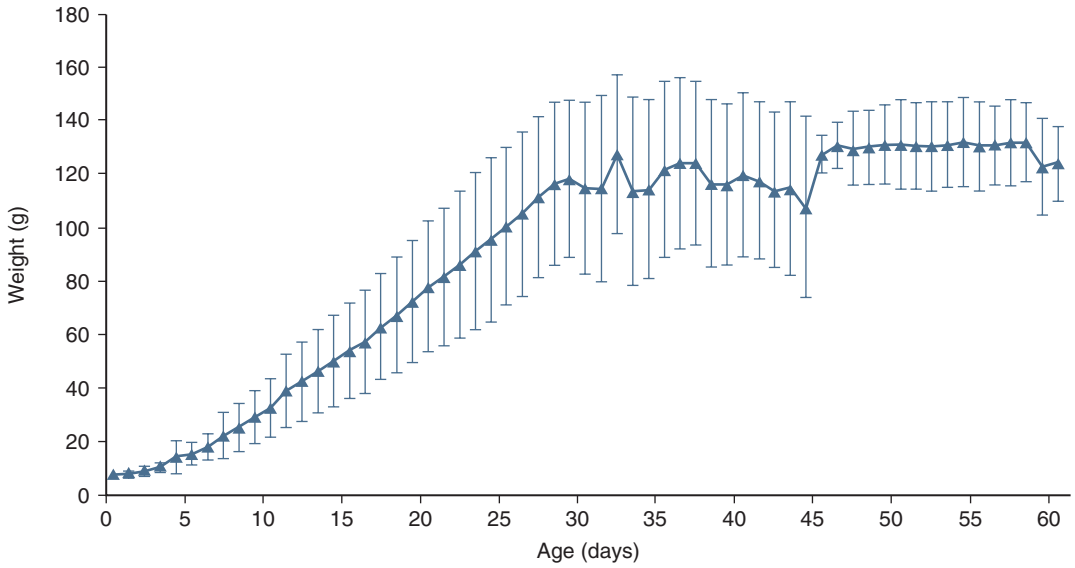
Continued

H. Growth curves for selected *Ramphastos* species—cont'd

Guyana toucanet (*Selenidera piperivora*)



Emerald toucanet (*Aulacorhynchus prasinus*)



Hand-feeding is performed using Mazuri commercial hand-feeding formula (Mazuri Exotic Animal Nutrition, PO Box 66812, St. Louis, MO 63166) mixed with distilled water to a consistency of pea soup. To this, pureed commercial canned fruit for babies is added at a ratio of 5% to 10% fruit to formula. For the first few days, initial feedings comprise no more than 5% of the bird's body weight (total amount). The amount fed is gradually increased based on the bird's increasing body weight such that by 6 weeks of age, the bird is receiving 9% to 10% of its body weight for total daily feeding. During the first 3 weeks of life, birds are fed nine times per day at 2-hour intervals. As feeding of toucan chicks requires more time (30 vs 5 minutes in a psittacine chick), this leaves a gap of 6.5 hours during the night. Reprinted from St. Leger J, Vince M, Jennings J, McKerney E, Nilson E, et al: Toucan hand-feeding and nestling growth, *Vet Clin North Am Exot Anim Pract* 15(2):183–193, 2012, with permission.

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