BACKYARD POULTRY MEDICINE AND SURGERY

A Guide for Veterinary Practitioners

Edited by **Cheryl B. Greenacre** and **Teresa Y. Morishita**

with website

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This book is dedicated to several people: my parents who taught me through example to be a lifelong learner and a keen observer; my supportive husband of over 25 years who has always been there challenging me to be my best, and to our two daughters whom we love very much; and to Dr. Branson Ritchie for providing me a start in the wonderful field of avian medicine and his willingness to share his knowledge.

Cheryl B. Greenacre

I dedicate this book to my parents, Yasuyuki and Doris Sai Kuk Morishita, for their love, laughter, support, guidance, and encouragement throughout my life.

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Foreword

Having operated an almost exclusively avian practice for over 3 decades, I've encountered practically every avian species commonly- and uncommonly- maintained as a pet. I must admit, it was initially surprising to me to observe which species are more valued by their owners. Extraordinarily rare and/or expensive specimens are sometimes regarded as nothing more than ornaments. Common, even feral, individuals have frequently exhibited as much value to their caretakers as human children do to their parents. Never should we, as veterinarians, or humans for that matter, judge the importance a pet plays in its owner's life. We, therefore, as veterinary professionals, should be adequately prepared to see and competently care for at least the majority of species we may encounter in private practice.

It is with that understanding that the veterinary profession should enthusiastically welcome the publication of *Backyard Poultry Medicine and Surgery*. Rarely does a reference text come along that covers A to Z as thoroughly as this one does. Beginning with fundamentals, such as basic husbandry, progressing through common diagnostics and diagnoses, and extending into egg maladies, and even biosecurity, the authors have outdone themselves.

Which brings me to the authors: Each is a proven authority in his or her particular subject, and readers can rest assured that the information provided here is, as much as any life science can be, indisputably accurate and authoritative.

Following the publication of this book, it would be such a waste for almost anyone seeing avian species to be without it.

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Preface

Backyard poultry are increasing in number as more people own chickens and other poultry for either companionship or small scale meat and egg production, and more municipalities allow urban and suburban poultry ownership. Practitioners are increasingly being asked to care for backyard poultry and are seeking practical information on husbandry, medicine, and surgery in order to provide state of the art medical care for their patients. The diseases and care of backyard flocks is different than that of commercial broilers, breeders or layers and information on their care can be found in scattered resources such as texts on commercial poultry, agricultural extension information, state and federal governmental websites, and lay books and websites.

With both of us having worked with backyard poultry for over 20 years, Teresa in the backyard setting and through youth groups such as 4-H as well as the commercial poultry industry setting and being a diplomate of the American College of Poultry Veterinarians; and Cheryl in an academic avian and zoological medicine setting, and being a diplomate of the American Board of Veterinary Practitioners-Avian Practice, it was a common longtime goal to someday write a book with such information readily available for other veterinary practitioners. We envisioned a book describing diseases of backyard poultry organized by body systems so that it was practical for use by veterinarians providing individual or small flock care. Many of the books available are organized by name of disease or by type of causative organism, which does not help in the initial stages of creating a differential disease listing based on the presenting clinical signs. Also, many books currently available were created to solve large flock problems in a commercial setting rather than helping the individual bird, small flock, or someone's dear pet.

We created this book to fulfill the above listed needs and asked authors from both the commercial avian field and the companion avian field to contribute their expertise. The result is a book with practical information in a usable format specific for backyard poultry. There are chapters on diseases of various body systems (gastrointestinal, respiratory, integumentary, reproductive, cardiovascular, and musculoskeletal) listing those diseases that are common to backyard poultry and those that are not so common in backyard poultry but should be known such as avian influenza and exotic Newcastle disease. There are also chapters on the laws and regulations that govern backyard poultry care, ownership, and treatment so the reader can intelligently field questions from their clientele and understand important concepts such as the difference between prohibited drug use and extra-label drug use. The reader is provided the tools for determining and understanding appropriate drug use in backyard chickens, but an exhaustive listing of drugs, doses and the appropriate circumstances for each drug's use is beyond the scope of this book. In the "Regulatory Considerations for Medication Use in Poultry" chapter the reader is referred to extensive and thorough formularies and websites.

There is information on all aspects of backyard poultry care including fundamental information on anatomy and physiology, nutrition, housing, biosecurity, vaccines, and how to identify common breeds, as well as specific information such as how to perform and interpret an echocardiogram, fecal examinations, egg diagnostics, understanding and interpreting serological and other diagnostic tests, and how to perform simple and complex surgeries. There is also a chapter specific for the diseases of waterfowl such as ducks, geese, and swans, although all chapters include information on all poultry such as turkeys, quail, pheasants, and other birds.

We hope you find the extra efforts taken to provide a practical approach to veterinary care of backyard poultry offered in this book useful. Please visit the website to view many extras including links to references and videos such as how to perform a physical examination, how to give intramuscular and subcutaneous injections, stance and attitude of chickens with Marek's disease, respiratory disease symptoms, endoscopic retrieval of coins from a duck ventriculus, physical therapy in action, and watching a chick hatch!

Cheryl B. Greenacre and Teresa Y. Morishita

Acknowledgments

Books are usually a collaborative effort and this one is no exception. Many people have directly or indirectly been involved in making this book a reality. I am thankful for a solid base in avian medicine from my veterinary education at the University of Georgia that included an entire course in poultry medicine taught by Dr. Richard (Dick) Davis as well as an entire course in companion and wild avian medicine taught by Dr. Branson Ritchie. I learned a lot from the experience and knowledge gained during my internship, residency and academic careers in avian medicine, and I learned from clients that brought me their birds asking for state of the art care. I also learned from all the interns, residents, and students I have taught along the way.

I thank each contributing author for their precious time and the hard work that went into writing these outstanding chapters. This book was a group effort and we did it! This book also benefitted from the many generous people who gave permission for their pictures to be used in this book. Thank you. I also thank our University of Tennessee College of Veterinary Medicine Librarian, Ann Viera, for the continuous stream of all things poultry she has sent my way over the last several years including books, pamphlets, journal and news articles, websites, people and even her own chickens. I also thank the wonderful staff at Wiley for all their support, especially Erica Judisch who encouraged us to write the book, and to Susan Engelken for making the book so much better.

The idea of writing this book stemmed from the need for a practical backyard poultry resource for practitioners organized by body systems to aid in the diagnosis of disease. I am very grateful that Dr. Teresa Morishita agreed to be co-editor for this book, because she contributed her invaluable knowledge, expertise and contacts in both the commercial and backyard poultry medicine fields to make this book what we both wanted it to be. It has been a pleasure working with her on this project with the common goal of providing the best care possible for backyard poultry and their owners.

Cheryl B. Greenacre

I would like to acknowledge the dedication and efforts of Sophia Alvarez, Anh Doan, and Josep Rutllant who have assisted me in completing this book, as this book would not have been made possible without their assistance. I would also like to thank Moina E. Lum and Thomas Q. Lum for their support of my efforts.

Teresa Y. Morishita

Companion website

This book is accompanied by a companion website:

www.wiley.com/go/greenacre/poultry

The website includes:

- Powerpoints of all figures from the book for downloading
- Reference lists from the book for downloading
- Web-exclusive videos

SECTION I
General Information

CHAPTER 1

Laws and Regulations Governing Backyard Poultry in the United States

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Introduction

When a veterinarian is presented with the task of caring for a client's backyard flock, many daunting obstacles will eventually become evident, including questions of legal and regulatory requirements and obligations. While the appropriateness of whether or not to extend your professional services to these clients is a personal choice, legal requirements of the veterinarian and your client are mandatory. Violations of law may have criminal consequences and regulatory violations may carry punishments of fines and/or reprimands. It is even possible for a client to have their backyard flock depopulated and/or quarantined against their will. As in almost every area of modern veterinary practice, civil liability is always a threat.

For the veterinarian, there is no substantial legal requirement specific to practicing on poultry other than state licensure. Providing standard of care to backyard poultry is the primary issue of concern and this determination falls squarely within each state's veterinary licensing body. While backyard flocks are gaining in popularity with a concurrent rise in the number of veterinarians seeing such patients, these relationships are still relatively rare within a given practice area, even in major metropolitan cities. If a standard of care complaint is lodged with a state licensing board, its members must decide whose standards you will be held against. For instance, if a small flock under your care succumbs to Marek's Disease, any commercial poultry veterinarian would consider it standard practice to have had a vaccination protocol in place. While it would seem unreasonable to hold a veterinarian who

occasionally practices on small flocks to such a standard, it is not an impossible scenario. Even if the licensing authority dismisses such a complaint, a client is still free to sue for civil damages. This sort of risk is ever-present in modern society however, and hopefully will not dissuade those inclined to enter this new and growing area of veterinary medicine.

It might also be helpful for the veterinarian to know exactly who would be defined as a specialist, or expert in poultry, backyard or otherwise. Unquestionably, boarded members of the American College of Poultry Veterinarians are considered veterinary poultry specialists and most members spend their careers managing poultry. They work in academia, government, industry, and the private sector. Most of these veterinarians are also members of the American Association of Avian Pathologists. Because most of these veterinarians are working with large commercial flocks they may not be readily accessible to most backyard poultry enthusiasts. Many veterinarians working with the occasional backyard chicken may not even be aware of their existence. It is obvious that their assistance, when sought, can be invaluable.

The largest veterinary avian community (by membership) is the Association of Avian Veterinarians (AAV). Its membership is primarily composed of veterinarians working with companion birds, but is by no means confined to it. In fact, there is no avian Family that is excluded by AAV. Historically, psittacine birds have comprised a very large percentage of the species seen by AAV members, but they have always worked with passerines (finches, canaries), ratites (ostriches), columbiformes (pigeons), and others. Backyard chicken care is a rapidly growing topic within the AAV. Some of

its members are board certified as avian specialists by the American Board of Veterinary Practitioners.

The more common legal issues for the practitioner involves our role as an advisor to our clients, informing them of their own legal responsibilities. Many clients will enthusiastically and quickly form their own back-yard flock and invest a substantial amount of time and financial resources into their new hobby without a moment's thought that they may have already grossly violated the law. To the best of our ability, it is our professional duty to at least provide them with some guidance on applicable laws, much as we inform clients of leash laws and local ordinances that may forbid certain types of pets.

Homeowners and neighborhood associations

Covenants, conditions, and restrictions (CC&Rs) are limitations and rules placed on a group of homes by a builder, developer, neighborhood association, or home-owners association. Most established neighborhoods and subdivisions, and practically all townhomes and condominiums have CC&Rs. This is the first place for a prospective backyard poultry client to look for obstacles. When clients purchase a home in a covenant-protected community they enter into a contract with the Home-owners or Neighborhood Association. The owner agrees to be bound by the restrictions contained in the community's governing documents, which include the declaration of covenants, conditions, and restrictions that are recorded with the Clerk's Office of the county in which that community is located. Those restrictions are legally binding upon all property owners in the community.

Even when a town, city, or county adopts an ordinance allowing backyard flocks, such permissiveness does NOT trump that contractual agreement between the owner and the association. So even if your client lives in a city that expressly allows (even encourages!) small backyard flocks, a prohibiting clause within the client's CC&Rs will take precedence and the client will not be able to pro-ceed with establishing their flock. The more restrictive rule applies, and HOAs can and do exist in rural settings, even within land zoned for agricultural use. Also, if the homeowner is seeking to build a coop, they must first comply with requirements for pre-approval of construc-tion of enclosures with the Homeowner's Association (HOA) before obtaining any necessary building permits from the city or county.

Some HOAs are extremely active, while others seem to be almost non-existent in reality. These neighborhood associations usually have no real policing powers but can appeal to civil courts to force compliance upon an unco-operative member. Monetary penalties can show up as a lien when a property is sold. On the other hand, HOA rules can be the easiest and least complicated to amend or create. A simple appeal directly to the HOA board or a letter of support from the neighbors bordering a potential small coop is often all that is needed to gain permission. Another common tactic is for homeowners to get themselves elected to the HOA board, which can be surprisingly easy to accomplish. Once elected, it is a simple matter to add poultry issues to the agenda and only a majority of the existing board members need be convinced of the need for a rule change.

Renters should also note that although their lease may not specifically prohibit chickens, the owner of the prop-erty is likely bound by such an agreement and subse-quently anyone occupying the property is bound by the same. Even a willing and accepting landlord may not be aware of such restrictions and a renter may risk asking for a copy of HOA rules rather than risk the demolition of a newly constructed coop and re-homing of just-bought chicks. It would be wise for a renter not to assume poul-try ownership is acceptable in the absence of specific restrictions, but inquire ahead of time.

Are backyard chickens pets or farm animals?

Because many municipal officials and members of HOAs lack agricultural knowledge, they lack a basis for understanding whether chickens can peacefully co-exist with their constituents in a cosmopolitan area [1]. Few things excite people as greatly as the goings-on in their neighborhood. It is often the case that the set of rules that apply to backyard chickens is determined by whether chickens are defined as pets or livestock, as some may believe that chicken raising and other agricultural practices involving animals simply have no place in the modern city. Some cities define chickens as domestic animals or pets, and thus subject them to the same enclosure and nuisance regulations as other domestic animals such as cats and dogs [2]. Other cities specifically define poultry as farm animals [3], and hence are subject to the same laws and regulations that apply to cattle and swine. Some owners may be shocked to find that their hometown outlaws chickens as dangerous animals, placing them in the same cate-gory as lions, tigers, bears, and sharks [4]. A novel way

to address the issue is to treat chickens as a separate category of animal, giving homeowners, city inspectors, and animal control officers clear guidelines on how to approach and handle personal flocks [5]. In at least one instance, a city allowed a homeowner to keep her chickens because the owner herself considered them to be pets and the chickens did not create a nuisance [6].

Once the HOA/neighborhood association hurdle is cleared, the next step is to review city codes and ordinances. Internet access to city records is now almost universal even in small towns and although this has simplified access, it can be bewildering to find the appropriate and applicable ordinances. Interestingly, most large American cities have at least some provision that allows for backyard poultry, and smaller jurisdictions seem to be the most restrictive [7].

Navigating city charters and ordinances

Zoning and the law of local government often are regarded as subjects that are arcane and parochial. It is best to avoid projecting what you expect to find (and where), and simply be open to acquiring local knowledge [8]. A simple internet search for "City of … … ….." usually yields the entire charter, ordinances, zoning, permitting regulations, and health codes. Once the documentation is retrieved, the process of finding pertinent material can begin as regulations may be placed in different areas of a city's codified ordinances. The first and most logical place to look is under an Animals/Animal Control heading or subsection. If chickens are addressed under a city's animal control ordinances, then further regulations concerning lot size, setbacks, or coop requirements may be conveniently located in this place. As noted above, it may be unclear as to whether backyard poultry are considered pets or livestock. If such a distinction is not clear, the opinion of the city clerk or the city's legal counsel may be sought. If an Animal Control department exists, then the advice of their department should be sought, as they are very likely the people who actually have policing authority. In many cities, this function is carried out by the police department (or sheriff if county laws are applicable).

Another place to investigate will be health codes. Features such as cleanliness, sanitation, and noise control can often be contained in the health code although the latter may be codified under a "Nuisance" section. The problem of free-roaming birds may also be addressed as a public health issue, and owners should be particularly concerned about containing their birds

as this can be especially disturbing to neighbors, and may spark complaints that could result in uninvited scrutiny. At least two cities consider escaped chickens to be illegal trespassers if they enter a neighbor's property [9]. Fly and rodent control may figure prominently in local health codes, with some cities mandating the use of insecticides [10] and others requiring fly-proof enclosures designed to "prevent the entry therein or the escape therefrom of any bee, moth or fly." [11] The cities that mention rat control usually just mandate that the coop be free of rats, although others specify the methods used to control rodents such as placing food in rat-proof containers or specifying that coops be designed to be rat-proof. Coop hygiene is another area that pops up frequently and codes may stipulate how often coops must be cleaned, while most expressly prohibit odors or offensive odors.

The issue of slaughter may also be contained in city charters and vary widely in restrictions. Most often, the slaughter of individual birds will have no state or federal inspection requirements if the meat is consumed on premises by the immediate family. However, some cities have outright bans [11] on slaughter or restrict it to a building or other structure [12], presumably so that neighbors or their children are not damaged by witnessing such actions (one city seems to be concerned about the negative effects of chickens witnessing their brethren succumb to such an end and requires that slaughter occur in an entirely separate room than the one that fowl occupy) [13]. Owners should also be aware that if backyard chickens are regarded as pets, then slaughter may run afoul of local animal cruelty laws or even draw the attention of animal rights groups even if the practice is completely legal. It should also be pointed out that some jurisdictions specifically prohibit the slaughter of chickens for religious purposes, "applicable to any cult that kills (sacrifices) animals for any type of ritual, regardless of whether or not the flesh or blood of the animal is to be consumed," [14] but exempting Kosher slaughter. At least one city expressly allows slaughter both for food and religious purposes, [15] while another bans slaughter for food purposes but allows it for religious purposes [16].

There is another issue involving carcass disposal if a bird has not been slaughtered specifically for consumption. Many jurisdictions have rules pertaining to burial of dead animals and may not allow for burial within city limits. Cremations often have legal requirements and if present may not allow for simple burning (actually, many cities and counties specifically prohibit burning of waste, which would presumably include incineration

of animal waste including bodies). Obviously, veterinarians will have arrangements for carcass disposal that chicken owners can utilize. Even submitting a whole bird for necropsy can have unforeseen consequences. Diagnostic laboratories have specific rules pertaining to reportable diseases that are diagnosed either on necropsy or other diagnostic testing. This may trigger the involvement of state or federal authorities who may dictate disposal of subsequent poultry deaths from the client's flock, either by natural death, euthanasia, or depopulation.

Roosters can present legal issues on several fronts. First, their presence may be specifically prohibited in a jurisdiction or the number of roosters allowed may be regulated [17]. There is the obvious noise problem, and clients may not be aware that roosters can and do crow at any time of day or night and do not seem to restrict their vocalizations to daybreak. Rooster crowing may trigger a noise violation, even if a city specifically allows for roosters to be kept. Some clients may also mistakenly believe that a rooster is necessary for egg production, and it may be helpful to point out that almost all commercial laying hens never encounter a rooster once they have left the hatchery. The other problem with roosters is their mere presence if an owner is only interested in egg production and has no interest in chickens for the grill or soup pot. If an owner is keeping a rooster in order to allow at least some fertilized eggs in order to replenish the flock, then half of the chicks will have no place in an egg producing flock. While many owners are successful in finding "homes" for these birds, few farmers are interested in accepting rooster "pets" and unfortunately many of these birds will end up at the local shelter (if one exists). A veterinarian may be presented with healthy rooster culls for euthanasia. Another potential legal problem exists with "rescue organizations" that claim to provide a no-kill option for unwanted chickens. While many of these operations do precisely what they claim, others are fronts for people who are hoarding animals. While hoarding increasingly appears to have a deep psychological basis, many jurisdictions are beginning to address the problem through legal prohibitions and interventions.

Zoning

Perhaps one of the most difficult areas in a municipal charter for laymen to navigate is zoning laws, as zoning cases are considered legally idiosyncratic, and thus, not subject to generalization [18]. Cities that regulate chickens through their zoning laws are much more likely to substantially restrict raising hens [19]. Generally,

zoning regulations are designed and written for experts in the areas of land development and building construction and while the language contained therein may be perfectly understandable for someone in those businesses, it may seem impenetrable to an outsider. A client must determine what zone his/her property falls within and whether that zone allows for backyard chickens. To compound the problem, the municipal employees responsible for interpreting and enforcing these codes are working with developers and builders on a daily basis and may struggle to explain the process to laypeople.

Local Zoning Boards serve as the forum where conflicting preferences over land use are articulated, disputed, and sometimes accommodated [20]. After all, zoning is "the primary legal mechanism through which the community attempts to influence the evolution of its physical structure. The community as a whole attempts to preserve that which it values, plan for that which it desires, and discourage or eradicate that which it dislikes." [21] If a city's zoning laws only allow for chickens on land zoned for agricultural use, then most urban/suburban dwellers will have no options other than requesting a zoning variance or attempt to change the law itself. Zoning laws change in response to changing community values, and the community's cultural values are affected by the structures that an earlier era first permitted and then discouraged [22]. Under the Standard Zoning Enabling Act any community that engages in zoning must set up a zoning board, which serves the function of granting variances. In its simplest form, the zoning board is authorized to grant variances from zoning regulations only when (i), the impact of the regulations constitutes an unnecessary hardship on the petitioner, (ii), granting the variance will not harm the public welfare, and (iii), the situation is unique [23].

Coop construction

Many cities will regulate how a coop should be built and maintained, specifying the dimensions of the coop, how it must be built, and exactly how it must be cleaned. Although some cities' building requirements are specific to chicken coops, many are not particular to chickens and cover any structure meant to house animals. Some HOAs and municipalities will have requirements and permits that must be obtained prior to the construction of ANY unattached structure on a property.

The most common requirement concerns the amount of space allotted to the chickens. Again, there is wide variability, but it is usually calculated on the amount of square footage available per bird anywhere between 2 square feet per bird [24] and 15 square feet per bird

[25]. Rather than set a particular amount of space per bird, one city requires that the space be twice as big as the bird [26]. A relatively recent shift in animal welfare measurement focuses on welfare outcomes rather than setting engineering standards. These requirements can be so vague as to require that the chickens not be cramped or overcrowded [27] or they may be more specific, requiring that birds have space to stand, turn around, and lie down [28] or that they must be able to move freely [29]. A few cities have requirements designed to ensure that birds are protected from the environment. These standards range from specific protection from the sun or extreme temperatures [30] to simply requiring that enclosures protect the animals from inclement weather [31]. Some ordinances are downright peculiar, requiring windows if possible [32] or prohibiting keeping chickens in cellars [33]. Some cities will also restrict how large the coop may be, capping either total square feet or a maximum height for the structure.

Space requirements

Many cities restrict raising chickens based on the lot size of the property. Some cities require a lot of an acre or more in size [34] before allowing the presence of any chickens at all. Such a requirement effectively bans backyard chickens for most people in an urban or suburban setting. Another twist is that while some cities will not have a specific requirement for the size of the lot, the lot size is used to determine the maximum number of chickens allowed. Like most local codes, the specific ordinances can vary greatly. Some cities allow for a maximum number of chickens for properties of a certain size (and under), then allow for more birds as the property size increases. This kind of step system can become somewhat intricate and be based on number of birds per square foot, per acre or division of acre, and may allow for a mixture of chickens and other animals. On the other hand, some cities appear to be very lenient in lot requirements, allowing up to 30 chickens per 240 square feet [35] (about the size of a modern bedroom). Yet one more way to regulate is to determine the number of chickens allowed based upon zoning, for example allowing a certain number of birds on property not zoned agricultural [36]. A simple, albeit arbitrary, way to limit flock size is to limit the number of chickens any household can keep, no matter the size of the property. Of the cities that use this simple method, the number of birds allowed varies from 2 [37] to 50 [38] chickens. Still other cities set a maximum number of chickens that can be owned before requiring the owner to apply for a permit [39].

Setbacks

Setbacks are an extremely common way for cities to regulate chickens, especially requirements that chickens and/or coops be kept a certain distance away from other residences or neighboring buildings. These setbacks can range from 10 feet [40] to 500 feet [41] and may be mixed with zoning requirements and/or lot size. Some cities will relax setback requirements if the client is granted permission from surrounding neighbors [42]. This can be especially useful in multi-family residences and densely constructed neighborhoods such as zero-lot-line houses in which the structure comes up to or very near to the edge of a property line (in other words, an exterior wall of one home is the lot line of the other person's property). Some cities may cite specific setbacks from the owner's own home, while others exclude any such restriction [43]. As an example of how variable such codes can be, at least two major cities frame the setback not from the structure itself, but specifically to a door or window of the structure [44]. Setbacks from structures may also not be confined to residences, but from schools, hospitals, or businesses. Grand Rapids, Michigan, places a 100 foot setback from any "dwelling unit, well, spring, stream, drainage ditch or drain." [45] Very few clients would find themselves able to escape from such tight restrictions.

More restrictive may be setbacks from property lines, no matter if a dwelling is much further away. Property line setbacks may vary from just inches [46] to many hundreds of feet [47]. As in the case with other setbacks, the rule may be relaxed if permission is granted by neighbors. In an effort to prevent direct visualization of chicken coops or possibly contain escapees, a city may prohibit coops in front yards or corner lots, [48] or have a setback from the street.

Permits

Many cities will require a permit or license in order to keep chickens. As is always the case with local laws, the permitting authority is highly variable. It may reside within a city's public health department, animal control office, inspections department, or even the city clerk. For what must truly be the ultimate in frustration, some cities do not even specify in their ordinances by what means a person actually procures a permit. Permitting fees will also vary widely as will the term of the permit; some will require annual renewals, others biennial, still others may only need to renew every five years. A few municipalities appear to have open-ended terms, either not specifying the term or being valid unless revoked. Some cities will issue a permit only with the consent of all or a percentage of neighbors that either border upon

the property or within a prescribed radius [49]. The permitting process may only apply to flocks of a certain size or if roosters will be present. Many of the permitting/licensing requirements appear to address concerns over potential complaints from neighborhood residents.

It must be noted that municipal codes and ordinances sometimes conflict with each other, creating confusion and frustration for an owner trying to be fully compliant. Animal control codes may conflict with zoning laws or with health codes [50]. This kind of discordance may pit different city departments against each other or even put into question whether a city's Board of Health has precedence over the Zoning Board. These conflicts may need to be resolved by the full City Council, and obviously may not be a priority for a busy Council. At best, it will likely not be definitively resolved quickly. As an example of the kind of confusion that can drive a client to frustration, the animal section of one city's code allowed chickens if the zoning ordinance permitted it. The zoning ordinance allowed chickens if the animal code permitted it. The city clerk resolved this vicious loop by interpreting the provisions to ban chickens entirely [51]. The contradictions that occur in local government are simply more visible and by no means preclude their presence in statewide or national forums.

Perhaps in retaliation for clients to have to navigate such a labyrinth of local laws, some homeowners are simply refusing to cooperate, or more constructively, are organizing local and regional movements to create or amend local ordinances. Commonly referred to as the "Poultry Underground," the movement gained momentum and publicity after some citizens convinced the Madison, Wisconsin, City Council to legalize backyard coops, resulting in the production of a documentary "Mad City Chickens" [51]. Websites and T-shirts frequently display slogans such as "When Chickens are Outlawed Only Outlaws Will Have Chickens."

State and national laws and regulations

Once your client has cleared the local hurdles (HOA, municipal, and county), then the next set of rules and regulations will come from state and federal authorities. It is important to realize that rules and regulations at this level are designed for commercial poultry operations and protection of public health, whereas local ordinances are also concerned with property values,

odor, noise and other "nuisance" factors. As far as the extent of involvement of state and federal authorities is concerned, if a backyard enthusiast obtains their starter chicks legally and consumes either the eggs or the meat themselves, within their own household, then it would be rare and unique for them to have any contact or problems. Even if they are breeding their own birds, as long as the chickens they produce essentially live and die on premises, there are really no state or federal entanglements to ensnare them. But if birds (live or dead) or eggs move off their property, then an entire series of hurdles must be cleared. The penalty for non-compliance can be severe – including fines and depopulation of the flock.

A backyard enthusiast must realize that the commercial poultry industry can take a very cautious view of small flocks of chickens. The problem is not a shrinking market share – at least in the United States, eggs and meat produced by a household for their own consumption has a negligible financial impact on industry regarding lost revenue from egg or meat sales at supermarkets and restaurants. The real problem is the danger of a commercial operation being quarantined or even being depopulated because a small flock of hens has been diagnosed with a highly contagious disease a few miles down the road in someone's backyard. If faced with the choice of depopulating a dozen chickens in a backyard flock versus the loss of millions of dollars of revenue because eggs or poultry cannot be transported away from the commercial operation, state and federal regulators may show little hesitation in their decision. This kind of situation is not simply an economic decision; a few backyard layers could potentially threaten the health of hundreds of thousands of hens. Commercial producers are highly protective of their very large and very expensive investment and small flocks of chickens present a credible and ever-present danger to their livelihood and the lives of their birds.

Matters become even more fraught when public health is at stake. The State has a responsibility to protect its citizens and the State takes this matter seriously. Even though there has not been a single human case of avian influenza within the United States, you would be hard pressed to find any American who has not heard of the disease. Many millions of dollars and thousands of hours of work are expended to prevent and control the entry of avian influenza into the United States. While these efforts understandably focus on large commercial operations, officials are acutely aware of the dangers that small backyard flocks present.

Movement of live poultry to a backyard flock

A client must obtain starter birds from somewhere, and that somewhere must be from a neighbor, another backyard enthusiast, a feed store, farmer's market, a local hatchery, or mail-order. It is recommended to purchase chicks from hatcheries or breeders that participate in the National Poultry Improvement Plan (NPIP), which will be described shortly. It is important for both the veterinarian and client to at least have some awareness of what the NPIP is, what it does, and why. The danger of entry of contagious diseases such as *Salmonella pullorum*-typhoid or avian influenza is a real threat - not just to your client's personal flock or their family's health, but also to public health and the commercial poultry industry. Beyond satisfying legal requirements, it is the duty of a small flock owner and the veterinarian as an advisor to prevent the entry of disease into small flocks and spread of disease to other small flocks and commercial flocks as well as protecting human health.

National poultry improvement program

The National Poultry Improvement Plan [52] was established in the early 1930s to provide a cooperative industry, state, and federal program through which new diagnostic technology could be effectively applied to the improvement of poultry and poultry products throughout the country. The development of the NPIP was initiated to eliminate pullorum disease caused by *Salmonella pullorum*, which was rampant in poultry and could cause upwards of 80% mortality in baby poultry. The program was later extended and refined to include testing and monitoring for *Salmonella typhoid*, *Salmonella enteritidis*, *Mycoplasma gallisepticum*, *Mycoplasma synoviae*, *Mycoplasma meleagridis*, and low pathogenic avian influenza. In addition, the NPIP currently includes commercial poultry, turkeys, waterfowl, exhibition poultry, backyard poultry, and game birds.

The NPIP is a voluntary program and although a particular focus is the registration of breeder flocks in order to ensure disease-free chicks, the guidelines set up by the NPIP are particularly important when birds are being transported. All states (with the exception of Hawaii) will require that poultry being imported across their state border come from flocks that either participate in the NPIP or follow the guidelines set forth for participation in the NPIP. Further, many states

will require that birds being transported *within* a state originate from an NPIP registered flock or follow the guidelines set forth for participation in the NPIP. The practical consequences of the establishment of the NPIP are simply this: although it is *possible* for a backyard enthusiast to have poultry that have never been subject to NPIP guidelines, it is not advisable as this will be the safest source for starter or replacement birds. Perhaps even more importantly, if your client is going to be moving birds off their premises to be sold, traded, or exhibited then it is highly likely that your client must either be a participant in, or follow guidelines of, the NPIP. Although the NPIP program is voluntary, every state (except Hawaii) has chosen to use NPIP guidelines in some shape or form to regulate movement of poultry into and within their state.

The technical and management provisions of the NPIP have been developed jointly by Industry members and State and Federal officials. These criteria have established standards for the evaluation of poultry with respect to freedom from NPIP diseases. Each state runs its own NPIP program; the federal government (USDA) only manages and coordinates state efforts. The NPIP website has direct links to the Official State Agencies [53] in each state (Hawaii is the only state that does not participate in the NPIP). All of the regulations for the NPIP are detailed in Title 9 of the Code of Federal Regulations (CFR) [54]. Each Official State Agency that implements the NPIP must follow the Plan as stated in the CFR but they may have their own rules and can adopt rules that are more stringent than those in NPIP.

Sources of starter or replacement birds
Neighbors, friends, or other backyard hobbyists
The supplier has the responsibility to ensure that all applicable laws have been followed before delivery of live birds. While a health certificate or similar documentation may be required for the purchaser to transport the birds to their home, these requirements are typically fulfilled by the seller. It is important to note that states have the authority not only to restrict import of animals into their state, but also transport of animals within the state. Therefore, it is conceivable that your state may have rules that restrict the movement of birds even within your own neighborhood. It would be prudent to contact your own state's animal control office to be sure that there are no state requirements. Identifying the appropriate state agency is the difficult part, as

jurisdiction varies widely and often falls across several different agencies or departments.

Feed stores, flea markets, and roadside stands

These sources very often have legal requirements, although it is not uncommon for these vendors to be completely unaware of such and can be in gross violation of existing rules. Again, it is the responsibility of the seller, not the purchaser, to be in compliance with all applicable laws and regulations, although it would be wise for the purchaser to know if health certificates or similar documentation is required while transporting the birds from the source to their home (this applies to both intrastate and interstate transport). Pragmatically and realistically, however, it is virtually impossible for state regulators to oversee and enforce rules with every possible outlet or source. These sellers have very small batches of birds, often from myriad sources, and almost always have transient supplies. Most of the year, these sources will have no animals available at all. An aspiring backyard enthusiast will have no luck acquiring birds from these sources in deep winter, only to see a glut in the spring. "Chick Days" at feed stores and flea markets are common throughout the country.

One final note must be made regarding acquiring birds from the above-mentioned local sources that has nothing to do with laws or regulations. First, your client may have no assurance that they are buying a specific breed of chicken. In fact, there is no assurance that the available chicks are even egg layers as opposed to meat-type birds (referred to as broilers within the poultry industry). Often, these chicks are hybrids. Second, except for some breeds with sex-linked traits, your client will also have no idea whether they are buying pullets or roosters. Finally, it must be pointed out that this is the simplest way of introducing severe disease into a starter flock, or more importantly into an established flock. A much safer way to acquire new birds is from the following:

Commercial hatcheries and hobby farm breeders

Virtually all commercial hatcheries, and the majority of hobby farm breeders will be participants in the NPIP. There are several advantages to this for the backyard enthusiast. First and foremost, these sources will be certified free from *Salmonella* pullorum-typhoid. Additionally, they may also be certified free from mycoplasma and avian influenza. It is absurdly easy to find out if these sources participate in the NPIP program-just ask. Most hatcheries and breeders prominently display their participation in the program and in fact, if they are transporting birds across state lines they will be required to do so. Even transportation within a state will probably have rules either requiring participation or have rules modeled upon NPIP guidelines.

There is another advantage for obtaining birds from NPIP participants regarding legal movement (transportation). Birds that are shipped from an NPIP hatchery or breeder may use a form (specifically called the VS Form 9-3) *in lieu of* a health certificate for transportation [55]. To the author's knowledge, poultry are the only species that have such an exemption from health certificates. The hatchery or breeder will typically include this form along with the chicks. As far as means of transportation for small numbers of chicks from these suppliers are concerned, the most common (and inexpensive) will be through the United States Postal Service (USPS). The USPS does not assume responsibility for ensuring that shipped birds have required documentation – that responsibility lies with the shipper. The USPS does, however, have very specific mailing and packaging requirements [56], which can be accessed from their website [57]. Of course, if a breeder or hatchery is within driving distance, your clients can simply pick up the birds themselves, although it would still be advisable to receive the VS Form 9-3. A client may be frustrated to learn that they may not be allowed to inspect the premises or meander within a commercial hatchery to observe first-hand the conditions under which chickens are raised. This has little to do with concealing practices and everything to do with biosecurity. All commercial operations are either private or corporate entities and they have every reason and authority to restrict entry onto their premises.

One final note is to point out that fertilized eggs are treated legally in the same fashion as live birds.

Transportation of poultry from a backyard flock

While discussing acquiring starter or replacement birds into your client's backyard flock, it becomes apparent that state and federal authorities highly regulate the movement of poultry, when crossing a border into a state, but also within a state. Again, these regulations are designed to protect both the public health (as in the case of avian influenza) and the health of commercial flocks (as in the case of virulent Newcastle disease). These laws and regulations may cover movement of live birds (including fertilized eggs), unfertilized eggs for consumption, and bird carcasses (meat) intended for consumption. In fact, a state may have entirely different agencies that control each separate kind of chicken or chicken product. For example, in the state of Texas, the Texas Animal Health Commission regulates the movement of live birds. At least two (possibly three,

depending on the venue and destination of the meat) different groups within the Texas Department of State Health Services are in charge of poultry meat. Both the Texas Department of Agriculture and the Department of State Health Services regulates eggs. Remember, these regulations are in addition to any restrictions that are placed at the municipal or county level. Large cities especially will usually have their own requirements, which are often in the jurisdiction of their respective Health Departments.

The transport of live birds falls squarely within most state regulations, and every state has rules and regulations governing such movement. It is not wise to assume that even giving a few birds to a neighbor in order for them to start a new backyard flock has no legal restrictions. In some states it will not matter whether a financial transaction has occurred, it is the movement itself that is regulated. Depending on the state, the rules may vary from non-existent to very stringently regulated. Often, it is the presence or absence of large commercial flocks within the state that dictates the degree of regulation and severity of penalties.

If your client is transporting live poultry on an airline, they must be aware that each individual airline will have requirements that may or may not coincide with federal and state requirements for transport. Often, these requirements will be in addition to whatever governmental regulations are applicable. In addition to their own paperwork, they will also have stringent rules on the types of containers that must be used, food and water instructions, the number and types of birds allowed, or other requirements. Individual airlines have their own set of rules that may not be applicable on another airline. Most commercial air carriers have a division that specifically handles live animals.

Health certificates/veterinary accreditation

In 1921, the U.S. Department of Agriculture (USDA) established the veterinary accreditation program so that private practitioners could assist federal veterinarians in controlling animal diseases. In 1992, the Animal Plant and Health Inspection Service (APHIS) of the USDA began managing the program nationally, but authorization of veterinarians continued on a state by state basis. Every state has an area office that can easily be obtained at the veterinary accreditation website [58]. Any veterinarian writing health certificates since this time has been familiar with the program.

In 2010, the program was enhanced as a result of threats of emerging disease; in the case of birds this has included an epizootic of exotic Newcastle disease and epizootics of West Nile virus. In the vast majority of these incursions, these epizootics have successfully been eliminated with the veterinary practitioner being the first line of defense against such catastrophic disease events [59]. The enhanced program strengthens the accredited veterinarians understanding of the program and increases their knowledge on current animal health issues. It also allows for the administration of a consistent and uniform program.

The program now has two accreditation categories (Category I and Category II) in place of a single category. Category I accreditation is designed primarily for companion animal practitioners and includes such species as dogs, cats, laboratory animals (rats, mice, gerbils, hamsters), ferrets, reptiles, and even native non-ruminant wildlife. Category II accreditation includes all animals including food and fiber species. Accredited veterinarians who wish to write health certificates for any type of bird must obtain a Category II accreditation. All birds within the Class *Aves* are included, whether they are poultry intended for food, parrots intended for human companionship, or wild birds. As many veterinarians who are seeing backyard poultry are primarily companion animal veterinarians, it is important to either apply, renew, or reinstate for the Category II classification if you wish to be able to write health certificates for your client's birds. USDA-APHIS has an easily navigable website, [60] which details requirements for accredited veterinarians and first time applicants, as well as general information for the public.

All veterinarians who were accredited before the enactment of the enhanced program have (or will have) chosen which category they wish to continue being accredited in, and have (or will have) completed supplemental training. Initial training will be required for all newly accredited veterinarians or those previously accredited veterinarians who did not renew before the deadline. All accredited veterinarians within the new enhanced program will be required to renew their accreditation every 3 years in order to maintain the program as the core of veterinary preparedness and response. Although provisions were also made for accreditation specializations, such specific rules do not exist at the time of publication.

As small backyard flocks increase in number, some clients may become interested in competing in shows and fairs. Transporting poultry to a destination where birds of differing origin are congregating compounds the possibility of dispersing disease. Therefore, almost every

state will have stringent rules regarding such movement and clients should be advised as such. Mandatory testing of individual birds for avian influenza is extremely common. Once more, the rules and the enforcing agency will differ state by state, but the state veterinarian's office should be the first place to contact. The organizers of such exhibitions are generally experienced and many of these events (such as county fairs) have been held for decades; therefore the requirements and rules for registering show animals is distributed to participants well in advance. Regarding shows, fairs, and other exhibitions, there is another consideration to keep in mind – the Animal Welfare Act (AWA).

The AWA was signed into law in 1966. It is the only federal law in the United States that regulates the care and housing of animals in research, exhibition, transport, breeding for wholesale, and by dealers. Other laws, policies, and guidelines may include additional species coverage or specifications for animal care and use, but all refer to the AWA as the minimum acceptable standard. The Act is enforced by the USDA-APHIS Animal Care program. While animals intended for food are specifically excluded from the AWA, animals that are exhibited are absolutely covered and therefore chickens that are entered in fairs, shows, and exhibitions are covered by its provisions. The AWA was amended in 2002 to include birds not bred for use in research; however the regulations have not yet been released at the time of publication, so facilities with birds used for purposes described in the AWA are not subject to enforcement action. An overview of the AWA as well as specific provisions is accessible on the USDA-APHIS Animal Care website [61].

Slaughter, processing, and distribution of poultry

The overarching law that applies to poultry slaughter and processing is the Poultry Products Inspection Act (PPIA) [62], which is administered by the Food Safety and Inspection Service (FSIS) of the United States Department of Agriculture. The PPIA was passed by Congress to ensure that only wholesome poultry that is not adulterated and not misbranded enters interstate or foreign commerce, but has been amended to extend the mandate for federal inspection to all businesses or persons that slaughter or process poultry within a state, when the State does not enforce requirements at least equal to the inspection requirements of the PPIA. Therefore, any business in any state that slaughters or processes poultry for use as human food is required

to do so under federal or state inspection, unless the slaughter or processing operations at the business meet certain exemption criteria in the Act.

Twenty seven states have their own meat inspection program (for intrastate sales) that will meet or exceed standards set forth in the PPIA. Inspection programs in states that do not have their own program are managed by the USDA, specifically the Office of Policy Evaluation and Enforcement Review [63]. Although a backyard flock may be exempt from inspection, it may be necessary for your client to apply for an exemption or follow specific criteria in order to satisfy the exemption. Exemption requirements vary, but will often require a minimum level of sanitation or other requirements. In any case, if an exemption is granted, then the rules with which the owner must comply will be clearly spelled out by the regulatory agency requiring it.

Although it was not the intent of Congress to mandate federal or state inspection of an owner's private holdings of poultry or to mandate inspections of small numbers of poultry, even owners who operate under an exemption are not exempt from all requirements of the Act. USDA-FSIS has developed a flowchart [64] to help owners determine if they qualify for an exemption, but please note that this guide only applies to poultry and not to other kinds of livestock (cattle, sheep, goats, etc.), as they fall under the requirements of the Federal Meat Inspection Act and not the PPIA. Generally, if the client is not engaged in selling poultry meat, there are no federal requirements under the PPIA. Importantly, if your client slaughters and processes less than 1000 birds a year or if they are for personal or private use, they may qualify for either a Personal Use or Producer/Grower – 1000 Limit exemption.

If your client qualifies for an exemption, then they may slaughter and process poultry without the benefit of federal inspection on a daily basis, or continuous bird-by-bird inspection and the presence of inspectors during the slaughter of poultry and processing of poultry products. However, the Act does not exempt any person slaughtering or processing poultry from the provisions requiring the manufacturing of poultry products that are not adulterated and not misbranded. Therefore, poultry must be slaughtered and processed under sanitary conditions and using procedures that produce sound, clean poultry products fit for human consumption. Specific sanitary practices are described in FSIS's Sanitation Performance Standards Compliance Guide, dated 13 October 1999 [65]. The specific sanitary practices in the document are not requirements; however, establishments that follow the guidance can

be fairly certain that they comply with the requirements in the Act.

The regulations in the PPIA require that poultry products transported or distributed in commerce bear specific information. Poultry products inspected and passed under USDA inspection at official USDA establishments must bear the official inspection legend and meet specific labeling requirements prescribed in the regulation. However, exempt poultry products cannot bear the official mark of inspection. In addition, there are specific labeling or identification requirements for exempt products to meet in lieu of bearing all required elements of a label. The information that packages of exempt poultry products must bear varies depending on the exemption and also upon each state's own regulations. In addition to labeling and packaging, states usually also have storage requirements, particularly refrigeration standards.

But even clients who qualify for both federal and state exemptions cannot donate the meat for use as human food outside of their immediate household in many states without meeting explicit criteria. In other words, your client is often not legally allowed to give away unlabeled, uninspected poultry meat to their neighbors or food pantries, although it is permissible for the neighbors to consume poultry that the client has slaughtered and processed (under sanitary conditions) on their own premises, as long as the neighbors consume the meat on the client's premises and the client does not receive money or any other type of compensation for the meal. Even then, if a family member or guest becomes ill from the meal, the owner may soon find themselves under the scrutiny of health officials.

Eggs for consumption

If selling or even giving away poultry meat seemed complicated, then the rules that may apply to eggs will seem even more so. Starting at the federal level, egg regulations will fall across several departments and agencies.

Food and Drug Administration

Egg wholesomeness and safety will fall under the authority of the Food and Drug Administration (Department of Health and Human Services). The FDA obtains its authority through both the Food, Drug, and Cosmetic Act and the Public Health Service Act and regulations will be found in Title 21 of the Code of Federal Regulations. Safety requirements, particularly regarding *Salmonella enteritidas* are exempted at the federal level for backyard flocks of less than 3000 birds [66]. However, refrigeration requirements, specifically that stored eggs be kept below 45°F, are not exempted for any operation; not even for very small flocks nor distribution from the owner's homestead [67]. It does not appear to matter whether or not commerce is involved in the transfer of eggs from the owner to another person, only that food is being provided for human consumption.

Likewise, there is a labeling requirement [68] under the authority of the FDA that seems to apply to all shell eggs, which is that all shell eggs bear the following statement: "*SAFE HANDLING INSTRUCTIONS: To prevent illness from bacteria: keep eggs refrigerated, cook eggs until yolks are firm, and cook foods containing eggs thoroughly.*" As with the refrigeration requirement, this rule appears to have no exemptions or exceptions.

Agricultural Marketing Services

Ensuring egg quality is the responsibility of this agency, which is in the United States Department of Agriculture and derives its authority from the Egg Products Inspection Act and whose regulations can be found in Title 7 of the Code of Federal Regulations. USDA-AMS surveys egg distribution to ensure that only eggs fit for human consumption (acceptable and unadulterated) are used for such purposes. This function is enforced under the AMS Shell Egg Surveillance Program, which involves quarterly inspections and sampling at egg processing facilities. There are exemptions from these requirements, specifically for producers who sell directly to consumers from their own flock, sell fewer than 30 dozen eggs, and have fewer than 3000 hens. Such eggs to be sold must not contain any more loss or leakers than allowed in the official standards for Grade B shell eggs [69]. These exemptions do not apply to restricted eggs when prohibited by state law.

Additionally, the AMS provides for uniform standards, grades, and weight classes for shell eggs through its Voluntary Grading Program [70]. This is familiar to consumers as weight classes (Jumbo, Extra Large, Large, Medium, Small, Peewee) and consumer grades (AA, A, and B). It is important to note that although this is a voluntary program of the federal government, it is a requirement in some shape or form in every state. It should not be surprising that state requirements vary wildly. For small backyard flocks, some states will not allow for the sale of ungraded eggs under any circumstances, even when sold directly from the owner to the consumer from their own home. Other states have extremely lax requirements or no requirements for grading at all if sold directly from the owner to the consumer. Eggs must generally be graded in order for eggs to be used in restaurants and retail food establishments. Most states have a mixture of requirements, such as allowing for ungraded eggs to be sold in certain

circumstances as long as the eggs are prominently displayed and labeled as being ungraded.

Some states do not allow eggs to be resold in used egg crates or cartons collected from friends or neighbors. In states that do allow this, there are almost always requirements to obliterate markings such as USDA grade shields, expiration dates, distributer information, and any other certification logos.

Food Safety and Inspection Services

This is another division of the USDA, and also derives its authority from the Egg Products Inspection Act. Its regulations can be found in Title 9 of the CFR. While the FSIS has broad authority over poultry meat (under the Poultry Products Inspection Act described earlier), the bulk of its regulatory capacity with eggs involves egg products. Egg products are those that contain dried, frozen, or liquid eggs; essentially eggs that are intended for human consumption and have been broken. While most backyard enthusiasts will not be engaged in this sort of activity, it is noteworthy to realize that these types of egg products have their own set of regulatory requirements, which are separate and distinct from whole shell eggs. Oddly enough, the FSIS also has refrigeration requirements for whole shell eggs [71], and although there is an exemption for personal use, the FDA requirement (which is the same, that is, that eggs be maintained below 45°F), has no known exemptions.

Sanitation

Finally, there may be sanitation requirements for washing or otherwise cleaning or sanitizing the eggs and these requirements can be very specific, such as a three-compartment sink necessary to wash, rinse, and sanitize equipment and eggs (with a separate sink for hand washing). Waste water must be disposed of properly. When using a municipal sewage system you may need the utility provider to sign off, certifying that the provider is approved by state and/or local authorities. Onsite sewage disposal systems (e.g., septic tanks) are usually regulated by the County Health Department, which is responsible for approving this step of the process. A residential septic system may not be suitable; your local Department of Health will determine if an additional tank is required for the processing facility. Be sure to communicate the small-scale size of the operation to the inspector.

Roadside sales and farmer's markets

Anyone considering selling their eggs or poultry meat onsite (on the owners own premises), at a roadside stand, flea market, or farmer's market should consult with their state officials to determine whether there are any inspection, storage, or labeling requirements related to their sale. Typically, the state's Department of Agriculture is the best place to start asking questions although rules may also be found within a state's Department of Health, Environmental Safety, or Consumer Safety divisions. Unfortunately, jurisdictions often overlap. Some farmer's markets are highly regulated by either the state or local authorities, and even sale from the home may require a Roadside Vendor's permit. Additional permitting may be required such as a Retail Food Establishment or Food Manufacturer's License. Flock registration for small backyard flocks is not yet universal, but more and more states are requiring this if birds (live or dead) or eggs will be leaving the owner's property, regardless of the size of the flock. If selling eggs, an additional egg license may be required by the state. Misleading advertising may be considered an offense at national and state levels. Owners should be extremely careful when using such words as "fresh," "selected," "cage-free," and so on. Use of the word "organic" has very specific legal meanings and unfortunately the definition will vary depending on the state and the product.

Live bird markets and auctions are increasingly coming under the scrutiny of both state and local health officials and the trend is to require that the birds come from NPIP certified flocks (or follow NPIP guidelines for *Salmonella Pullorum*-typhoid testing), be avian influenza tested, be from a flock registered with the state, and have record-keeping requirements. Many states are conducting regular inspections at markets and other venues to conduct surveillance testing and also to ensure compliance with all existing regulations.

Transitioning from hobby to commercial operation

If a veterinarian has a client with a rapidly growing backyard flock and is becoming concerned that they are flirting with crossing the line from a hobbyist to a commercial (albeit specialty) producer, the 3000 bird threshold would appear to be at least an easily quantified red line. Engaging in commerce itself, that is, exchanging money for birds (live or deceased) or eggs, does not in itself define a commercial producer, not even if the birds or eggs are specifically intended for consumption. Direct sales to customers, either privately or in a Farmer's Market, are exempted from food safety rules except as regulated locally through state, county, or local health codes.

Each state has its own department of agriculture that sets regulations regarding poultry, whether commercial or backyard, and a quick check on your state's website will generally yield state-specific laws and rules. In addition, each state will have a state veterinarian who should be considered as a primary reference when in doubt. The state veterinarian also often directs, manages, or is affiliated with a state animal health commission or board. The state veterinarian will be primarily involved in areas of both animal health and increasingly animal welfare.

Some states will require registration or permitting if a client is selling even small numbers of live birds, even at a roadside stand or feed store. These rules specifically target disease control, especially those diseases that could affect commercial poultry producers. Laws and regulations governing transportation of poultry are largely concerned with the same health issues – that is, the health not of humans, but of the larger commercial chicken population. Following are the diseases that are of gravest concern to state and federal officials:

Salmonella-associated pullorum and typhoid diseases

These conditions are caused by two very closely related organisms, which were once thought to be different species but have recently been classified as biovars of *Salmonella enterica* subsp. enterica. Pullorum disease is usually symptomatic only in young birds. The mortality rate varies, but it can be as high as 100%. Fowl typhoid resembles pullorum disease in young birds, but it is also a serious concern in growing and adult poultry. The control of these diseases is complicated by vertical transmission: Hens can become subclinically infected carriers, and pass the infections to their embryos in the egg. Fowl typhoid and pullorum disease have been eradicated from commercial poultry in many developed countries including the United States and Canada, but they may persist in backyard poultry flocks and game birds.

Avian influenza

State and federal officials closely monitor two types of avian influenza based on their ability to cause disease in poultry: Low pathogenicity avian influenza (LPAI) and high pathogenicity avian influenza (HPAI). LPAI naturally occurs in wild birds and can spread to domestic poultry. These strains pose little threat to human health, but the mere potential to mutate into more highly pathogenic forms has led the USDA to closely monitor both LPAI H5 and H7 strains. Broad public concern about highly pathogenic H5N1 virus has resulted in USDA efforts to very quickly respond to, and eradicate,

HPAI. It is important to note that HPAI has only been detected three times in US poultry – in 1924, 1983, and 2004. While more than 200 human cases have been reported since 2004, no strain of avian influenza detected in US poultry, either HPAI or LPAI, has caused any human illness. (see Chapters 8 and 9 for more details).

Virulent Newcastle disease

Exotic Newcastle disease is a contagious and fatal viral disease affecting all species of birds. END is so virulent that many birds die without having developed any clinical signs. END can infect and cause death even in vaccinated poultry. Mortality is up to 90% of exposed birds. USDA-APHIS is the federal agency that takes the lead in excluding END from the United States and responding to any END outbreaks that do occur (see Chapters 8 and 9).

Be cautious, not afraid

It is difficult not to be so intimidated by the labyrinth of laws, regulations, restrictions and exemptions discussed in this chapter without throwing up your hands in confusion and fear. And in fact it is possible for a well-meaning but uninformed client to find themselves in either serious trouble or face the tragedy of having their flock depopulated against their will. These rules were never intended primarily to quash backyard flocks, rather they are designed to accomplish some very simple goals, that is,

- Be a good neighbor
- Protect our poultry industry
- Protect human health.

These are worthy objectives even though it may be burdensome or even impossible for your clients to engage in their desire for a backyard flock and simultaneously fulfill their ethical and legal duties. At the very least, people should be aware and become at least minimally educated instead of pursuing such an endeavor on a whim. Stewardship of living creatures always carries responsibilities, and as veterinary professionals we should proudly carry that responsibility and pass it on to our clients.

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Common Breeds of Backyard Poultry

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General information

Chickens have coexisted with people for centuries. They have been a staple in farmyards around the world providing nutritious eggs and meat. Today chickens have reemerged as a companion animal. The modern chicken owner has discovered that hens each have a distinct personality and readily interact with humans, with the added bonus of providing delicious fresh eggs. They are given names, live in fancy coops, and when not feeling well are provided with veterinary care.

All of today's many breeds of chickens come from a single origin – the red jungle fowl of Southeast Asia. Exactly how many chicken breeds exist is not known because new varieties are continuously being developed.

The following are breeds of chickens that are easily acquired and are suitable for the urban backyard (Table 2.1). The following descriptions are only for the hens of each breed as most urban regulations do not permit roosters. This is not meant to be a complete list, but rather an example of some breeds that are kept as backyard poultry. Breeds of chickens can be divided into egg breeds, meat breeds, dual purpose breeds, and ornamental breeds [1–6]. There are several websites with complete descriptions of breeds and one of them includes a program for identifying the right breed for your needs [5,6].

Egg breeds

Ameraucanas

These are the "Easter egg" layers. Developed from the Araucana, which is a tail-less bird that lays colored eggs, the Ameraucanas are excellent egg layers with mainly blue and green colored shells (Figures 2.1–2.3). They have a calm and gentle disposition, which makes them a great choice for backyard flocks. They do well with children. These birds are very hardy and readily accept confinement. Hens weigh approximately 3 kg and have a pea comb. They come in many color combinations, but they are easily identified by the beards and tufts on their cheeks.

Hamburg

The origin of these birds is unclear with the modern varieties mainly influenced by British and Dutch breeders. There are six varieties: Golden Penciled, Silver Penciled, Golden Spangled, Silver Spangled (Figure 2.4), Black, and White. They have a spiked rose comb (Figure 2.5). The hens weigh approximately 2 kg. These birds are more flighty and love to free roam. They will need a spacious coop and would appreciate foraging in a garden. They lay small to medium white eggs, and rarely become broody. They are good fliers and in a backyard situation the wings should be kept clipped. They are not as friendly as many breeds but are certainly beautiful birds for a small backyard where the flock is not expected to interact with humans.

Lakenvelder

Several theories surround the origin of this breed. It arrived in the United States around 1900. "Lakenvelder" is Dutch for "shadow on a sheet," which describes the striking contrast of black and white feathers. They have a single comb (Figure 2.6). These birds make a beautiful flock addition. The hens are very reliable, non-broody, and medium white egg layers. They do best when given room to forage. A hen weighs about 2.5 kg. They are good fliers and should have their wings clipped when

Table 2.1 Various qualities of selected chicken breeds (lt., light; Ornam., ornamental)

Breed	Purpose	Weight (lb)	Comb	Average eggs/week	Size of eggs	Color of eggs	Docile?	Broody?
Ameraucana	egg	6–7	pea	3	medium	green/blue	yes	no
Australorp	dual	6–7	single	3	large	lt. brown	yes	no
Cochin	ornam.	8+	single	2	medium	brown	yes	yes
Cornish crosses	meat	8+	pea	1	small	lt. brown	no	yes
Delaware	dual	7–8	single	4	large	brown	yes	yes
Hamburg	egg	4–5	rose	4	small	white	no	no
Lakenvelder	egg	4–5	single	3	medium	cream	no	no
White Leghorn	egg	6–7	single	5	extra large	white	no	no
Maran	egg	7–8	single	3	large	dark brown	no	yes
Orpington	dual	7–8	single	3	large	brown	yes	yes
Orloff	dual	6–7	walnut	2	medium	lt. brown	yes	no
Polish	ornam.	4–5	V-shape	2	very small	white	yes	no
Plymouth Rock	dual	7–8	single	4	large	brown	yes	yes
Rhode Island Red	dual	7–8	single	5	extra large	brown	yes	no
Sicilian Buttercup	egg	4–5	buttercup	2	small	white	no	no
Silkie	ornam.	2	walnut	3	small	cream	yes	yes
Star	dual	6–7	single	5	large	brown	yes	no
Sussex	dual	7–8	single	4	large	lt. brown	yes	yes
Welsummer	dual	6–7	single	4	large	dark brown	no	yes
Wyandotte	dual	6–7	rose	4	large	brown	yes	yes

Sources: www.mypetchicken.com [5], www.poultrypages.com [6].

Figure 2.1 Ameraucana breed of chicken. Note the pea comb and "beard" or tuft on the cheek. (Source: Photograph courtesy of Dr. Katherine DeAnna.)

Figure 2.2 Ameraucana breed of chicken. (Source: Photograph courtesy of Dr. Katherine DeAnna.)

part of a backyard flock. The distinctive plumage and markings usually fully appear after the third molt.

Leghorn

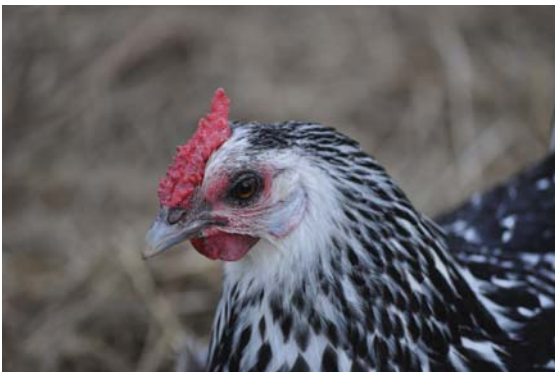The most prolific layer of eggs is the White Leghorn, and hence its popularity. The record number of eggs laid by a

Figure 2.3 Typical blue and green color to the eggs laid by Amer-acauna chickens. (Source: Photograph courtesy of Dr. Katherine DeAnna.)

Figure 2.4 Silver Spangled Hamburg breed of chicken.

Figure 2.5 Rose comb of Silver Spangled Hamburg breed of chicken.

Figure 2.6 Lakenvender breed of chicken. Note the single comb.

Figure 2.7 White Leghorn breed of hen. Note the single comb. (Source: Photograph courtesy of Dr. Cheryl Greenacre.)

chicken is by a White Leghorn: over 365 large white eggs in a single year. This very docile breed handles confine-ment well, but they can also be good foragers. Leghorns can be white, buff, black, dark wing, and brown with a single comb. Rarely do they become broody (Figure 2.7).

Maran

The Maran is a French breed developed in the town of Maran during the late 19th century. The French strain has feathered legs, while the English strain, which was

Figure 2.8 Cuckoo Maran breed of chicken. They have a single comb.

Figure 2.9 The Sicilian Buttercup breed of chicken.

developed in the 1930s, has clean legs. This breed is known for producing deep dark brown large eggs (often called the Chocolate eggers). There are several plumage varieties available. The Cuckoo Maran is one of the most common with black and white feathers, which are crossed throughout with irregular dark and light colored bars. They have a single comb and do not fly (Figure 2.8). These hens weigh approximately 3.5–4 kg. They are a good choice for a backyard flock because they tend to have a calm temperament and adapt to confinement.

Sicilian Buttercups

These were imported over 100 years ago from the Italian island of Sicily. Unlike most breeds, the hens have more impressive coloration than the roosters. This is a truly beautiful bird with a buff base color, where all feathers are marked by parallel rows of black elongated spangles, giving a spotted appearance (Figure 2.9). Another unique feature is the cup-shaped comb, which appears as two single combs connected in the back and front to make a cup shape (Figure 2.10). The comb is large and therefore more susceptible to frostbite. Buttercup hens are smaller (2.5 kg), have the ability to fly, and lay small to medium white eggs. They are very active birds that can do well in a backyard environment if worked with as young chicks.

Meat breeds

Examples of meat breeds are any of the Cornish crosses. They can reach 3 kg in 8 weeks and can reach over 4 kg after that. They lay a low number of small eggs.

Figure 2.10 The Sicilian Buttercup breed of chicken has a unique cup-shaped comb, which appears as 2 single combs that have connected in the back and front to make a cup shape.

Dual purpose breeds

Australorps

The Australorps originated in Australia in the late 1800s as a result of initial crossing between black Orpingtons and Rhode Island Reds and then other breeds. They were formerly known as Australian Black Utility Orpingtons, but in the 1920s the name was shortened to Australorps. They are very calm chickens and good layers. They are slightly smaller than Orpingtons and come in a variety of styles.

Delaware

A breed developed in the state of Delaware from a Barred Rock/New Hampshire cross. This hen is a larger bird (3 kg) that lays medium to large brown eggs. It is a beautiful bird that is almost pure white with black bearing on the hackles, wings, and tail feathers

Figure 2.11 Delaware breed of chicken.

Figure 2.12 Russian Orloff breed of chicken. They have thick feathering down the neck with a distinctive beard and muffs.

(Figure 2.11). It does not fly, has a single comb, and is non-broody. Delawares are hardy, docile, and tolerate confinement. They do well in both hot and cold climates.
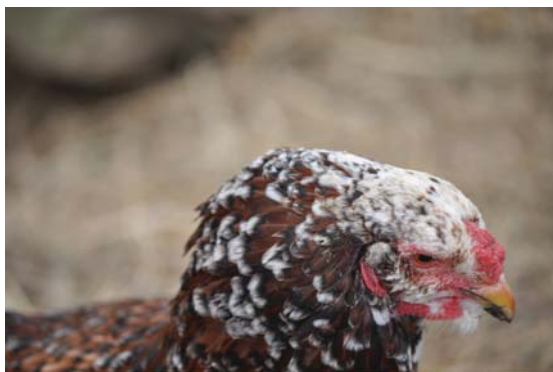
Orloff

These are the only breed in America with Russian origins; in fact they are often called Russian Orloffs. They are thought to have been developed in Russia from a Persian breed. The Orloff is a very hardy bird but can be an undependable layer. Hens lay medium, light brown eggs and do not get broody. Hens weigh in at about 3 kg. Orloffs are a tall breed and they have thick feathering down the neck with a distinctive beard and muffs (Figure 2.12). As they were developed for cold climates they have a small walnut comb as well as very small waddles and earlobes (Figure 2.13). There are several plumage varieties with the Spangled Orloff being the most common. These birds have a calm, friendly disposition and like to free roam. They are not always as readily available as some other breeds; however they make a unique addition to a backyard flock. They seem to tolerate heat, but may not be the best choice for the deep south backyard. Orloffs have done well for several years now in a Southern environment (Tennessee).

Orpingtons

This breed was developed in England and introduced to the United States in the late 1800s. Here it quickly became a popular farmyard bird for dual purpose (meat and eggs). The buff Orpington is a large (weighing 4 kg), stately bird with "golden" plumage, and a single comb (Figures 2.14, 2.15). Other colors such as lavender Orpington have developed. These birds are a friendly, affectionate breed and make wonderful pets for children. Calm, quiet, and easy to pick up and hold, they

Figure 2.13 Russian Orloff breed of chicken exhibiting a walnut comb.

Figure 2.14 Buff Orpington breed of chicken. (Source: Photograph courtesy of Phil Snow.)

Figure 2.15 Buff Orpington breed of chicken. They have a single comb. (Source: Photograph courtesy of Phil Snow.)

do well in confinement; Orpington hens are excellent layers of brown eggs. They also tolerate cold, do not fly, but can easily become broody.

Plymouth Rock (or Barred Plymouth Rock)

This American breed is one of the most popular dual purpose chickens on small farms today. They produce many large eggs. They have a typical barred black and white pattern to their plumage and a single comb.

Rhode Island Red

This dual purpose breed is of American origin and is used mostly to produce the sex-linked breeds described below.

"Sexlink" or Star chickens

A Sexlink chicken, also known as a Star chicken, is one that at the time of hatch can be sexed by color. This helps to guarantee that you have only pullets (females) for egg laying purposes. With other breeds sexing is only approximately 90% accurate. Sexlinks

Figure 2.16 "Sexlink" chickens come in several varieties such as Red Star.

come in several varieties such as Red Star (Figure 2.16), Black Star, and Golden Comets. They are hybrids and were created for production purpose. A Red Star, or Red Sexlinked, is a cross between a Rhode Island Red rooster or a New Hampshire rooster and a silver-laced Wyandotte, and Rhode Island White, or a Deleware hen. The resulting offspring will be buff or red females and white males at hatching. The Black Star, or Black SexLinked, is a cross between a Rhode Island Red rooster or a New Hampshire rooster and a Barred Rock hen. The resulting offspring will be completely black females and black with a white spot on top of the head males at hatching. The hens weigh about 3 kg. They are excellent layers and produce large brown eggs. They are also easy keepers. These birds tend to be very docile and friendly. These qualities make them an excellent choice for the backyard and for children without having to worry about acquiring a rooster.

Sussex (Speckled Sussex)

The origin of these birds remains unknown; some experts believe it arrived in England with the Romans some 2000 years ago. These birds are mostly mahogany with feathers tipped in white, which is separated from the mahogany body by a black band (Figure 2.17). They have a single comb. Hens lay well, producing light brown to brown medium eggs. They are very cold-hardy and tolerate confinement. They do not fly and hens weigh about 3 to 3.5 kg. Sussex are beautiful exhibition birds. The Sussex is calm, friendly, and curious. The Speckled Sussex is an excellent choice for backyard flock and for children. They exhibit good pet qualities.

Figure 2.17 Speckled Sussex breed of chicken. They have a single comb.

Figure 2.19 Silver-laced Wyandotte breed of chicken.
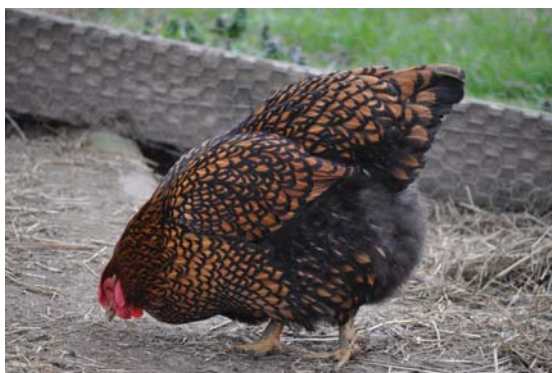
Welsummer

This is a newer breed developed in the Netherlands and imported to the United States around 1928. They have been described as "what a farmyard chicken should look like." They adapt well to any environment and are an excellent choice for more confined spaces. Hens lay well, and the large eggs are a lovely reddish brown color, often with speckles. The hens can become broody. Females are mostly reddish brown with some black stippling on some of the feathers (Figure 2.18). They have a single comb. Hens weigh around 3 kg. Welsummers have a friendly demeanor, are a calm breed, and are excellent as children's backyard birds.

Wyandottes

This is a breed developed in America. The silver-laced variety (Figure 2.19) was the original and ten colored

Figure 2.20 Golden-laced Wyandotte breed of chicken.

Figure 2.18 Welsummer breed of chicken. They have a single comb.

varieties are recognized today, such as the golden laced Wyandotte (Figure 2.20), blue-black Wyandotte to name a few (Figure 2.21). The Wyandotte has a body covered in fluffy, loose feathers, and the bird's shanks are short and set wide apart giving the birds a very rounded appearance. They have a rose comb (Figure 2.22), do not fly and do well in confinement. The hens do well in both hot and cold climates. Hens weigh about 3.5–4 kg. They lay good sized eggs that varies from light to rich brown. With the amazing array of colored varieties, these beautiful, generally calm and easy going birds make great backyard companions. A great choice when children are involved.

Figure 2.21 Blue-black-laced Wyandotte breed of chicken.

Figure 2.22 Blue-black-laced Wyandotte breed of chicken exhibiting a rose comb.

Ornamentals

Cochins

The Cochin arrived in the United States in the mid-1800s. These birds have always been popular, more for their appearance than their production. They are large birds with a distinctive curve from the neck and shoulders to the short "fluffy" tail (Figures 2.23 and 2.24). They are very docile and make wonderful pets. Cochins come in several plumage varieties and have a single comb. Generally, they are poor egg layers, but their unique appearance and friendly nature have made them as backyard favorite. They do not fly. Hens weigh around 3.5 kg.

Polish

This breed originated in Eastern Europe around the 16th century and is known for the tuft of feathers on top of
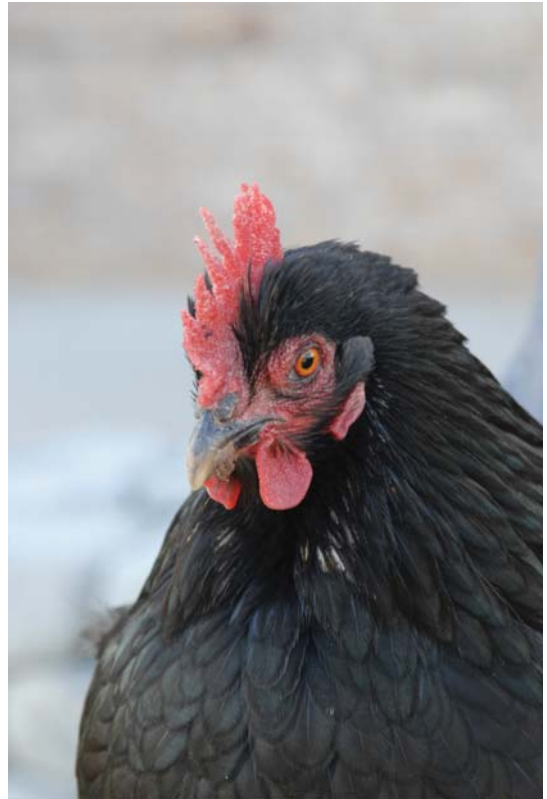
Figure 2.23 Cochin breed of chicken. Note the single comb.

Figure 2.24 Cochin breed of chicken. They are a large bird with a distinctive curve from the neck and shoulders to its short "fluffy" tail. (Source: Photograph courtesy of Phil Snow.)

the head and docile personality. They lay low numbers of small eggs.

Figure 2.25 White Bearded Bantam Silkie hen presenting for examination. Note the distinctive darkly pigmented skin, hair-like feathers, and top knot of feathers on the head. (Source: Photograph courtesy of Dr. Cheryl Greenacre.)

Silkie

Most Silkies in the United States are the bearded variety of bantam-sized Silkies. It is believed this breed originated in Asia, perhaps China, as far back as the 13th century. The feathers can be white, black, buff, blue, partridge, or gray. They are distinctive for their hair-like feathers, the feather tuft on their head, their very dark, even black skin, and for having five toes instead of the usual four (Figure 2.25).

Acknowledgments

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Basic Housing and Management

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Introduction

Raising backyard poultry can be a very pleasant experience for those involved. However, the average person is not cognizant of the time, effort, and knowledge that is required for a successful experience. In this chapter, basic management will be covered including housing, chick purchasing, brooding, daily care, and predator control options.

Housing

Design

Housing design will affect bird care, comfort, welfare, and well-being. There are numerous aspects associated with housing that need to be considered. The backyard chicken coop may be elaborate and aesthetically pleasing to the owner's eye, resembling a child's playhouse: windows, flower boxes, painted, and so on. The chicken coop might also be very simple, consisting of a cube made of 1 × 1 treated lumber with chicken wire or composed of bits and pieces of metal, wire, and wood found around the home. There is no wrong way to create the chicken coop but the design of the backyard chicken coop should be easy to clean, protect the birds from predators, and provide adequate space for the birds. The ideal structure would have a cement floor and be insulated with washable walls. This would allow the coop to be thoroughly cleaned and disinfected between each flock of birds. Typically, most chicken coops will have dirt floors, which can create problems if a disease outbreak occurs.

The backyard flock housing structures may be completely enclosed or have access to an outdoor run. Either method is acceptable, but the design of the house needs to attempt to limit the access of wild birds and predators. Further discussion on predator control can be found later in the chapter. Ideally, any openings in the chicken coop should be covered with hardware cloth and open doors should have a screen door. This will limit wild bird access because a constant supply of food and water will attract them. However, this may not be an option with an outdoor run and one must be comfortable with the liability of the increased biosecurity risk.

Ventilation

A common misconception is that the exchange of oxygen is the main reason for ventilating the chicken house. Ventilation is important for moisture removal, excess heat removal, exchange of gases produced by the litter, and providing fresh air. Spring, summer, and fall tend to see the least amount of ventilation problems occurring. However, a very common issue arises as the weather changes from warm days to cool evenings, where the owner may increase the ventilation during the day and close the house at night. There may be times that temperature swings are not accounted for, resulting in a warm day with limited ventilation. This tends to result in increased respiratory problems as a result of too much humidity in the chicken coop.

The summer months may see high temperatures, and ventilation becomes important for circulating air to help remove excessive heat in the chicken coop. In the commercial industry, ventilation is controlled by computer systems and fan numbers speeds increase to circulate the air as temperature increases. A backyard coop may open windows and doors, ideally screened, to increase the amount of air blowing into the chicken coop (natural ventilation). Another option is to use a

small fan to circulate the air (mechanical ventilation). A fan purchased at a pet supply store would work sufficiently. Again the idea is not to provide enough fan for each bird to rest in front of the air flow, but to circulate or exhaust the air within the coop.

The tradeoff between ventilation and temperature becomes apparent during the winter months. If you maximize the ventilation to remove all of the moisture within the coop, you will lose all of the warm air, resulting in an increase in input cost to maintain the temperature. On the other hand, if you maintain the temperature then you will not remove the moisture in the coop, resulting in significant management issues. Therefore, minimum ventilation should be practiced to meet both objectives: removing moisture and maintaining temperature. Minimum ventilation describes the situation where a small amount of cold air enters the chicken coop, is warmed by the temperature in the house, absorbs moisture within the coop, and is then exhausted. This can be achieved with air inlets along the roof line of the coop or, depending on the house design, a slight cracking of a window could achieve the same objective. However, the incoming air must blow toward the roof. An opening where air enters and is not directed toward the roof will result in cold air descending downward and can create an unnecessary draft in the coop. As a result, the chickens can become chilled, resulting in health issues or a higher input cost as the owner tries to heat the coop.

A chicken coop designed with insulated sidewalls and roof can result in the house becoming too tight. This means that no external air can enter the chicken coop, resulting in no air exchange. This can lead to several problems. One issue is the accumulation of moisture as a result of which condensation forms on the walls or ceiling and can result in "rain." This can lead to bedding issues that can affect bird health, as discussed later in the chapter. Additionally, the moist environment can provide opportunities for molds and other diseases to propagate. Second, excessive levels of ammonia can result from the damp litter and lack of airflow. When the ammonia levels become too high, >25 ppm, then birds can experience detrimental effects to their respiratory system and eyes.

Temperature

A chicken's body temperature falls within the range of 105–107°F with males having a higher body temperature than females. Although their body temperature is high, birds have a thermal neutral zone, or an area where they do not need to actively regulate their body temperature. The extremes, hot or cold, can be detrimental to the birds and, depending on body size, age, and breed, these zones can vary. Baby birds are going to have a higher tolerance for hotter temperatures, while older birds are more forgiving for colder temperatures. A good rule of thumb is to aim for the temperature in the chicken coop to be between 50 and 75°F. The largest consideration is whether the birds have the opportunity to get out of the weather to allow them to self-regulate their body temperature.

A ramification of extreme cold weather is frost bite. Poultry feet and head parts (combs, wattles, snoods, etc.) are most likely to become frostbitten. This can be a result of being outside for too long or having to stand, walk, or not being able to get out of snow for a long period of time. On the flipside, heat stress is a result of excessive hot weather. Birds will pant, spread their wings, increase water consumption, and decrease feed consumption in an effort to cool their bodies; proper ventilation can help reduce the chance of heat stress.

Lighting

Poultry can grow sufficiently with normal daylight and do not necessarily need any special lighting requirements. However, light intensity and duration can have an impact on birds. Light intensity can be as low as 5 lux to stimulate activity and can be as bright as desired. Very bright lights may lead to behavioral problems such as aggressiveness and bird picking. Therefore, lowering the light intensity will provide a remedy. A rule of thumb for intensity is whether you can read a newspaper at arm's length. If so, then there is sufficient light for the birds.

The other consideration is light duration. If the goal is to produce meat, the birds only need natural day length to grow. Some individuals believe that providing 23–24 hours of light results in increased performance, but poultry are like humans and need a natural dark cycle. Therefore, 16 hours of light would be the maximum suggested for meat birds.

Poultry that is being reared for egg production, table eggs or fertile eggs, are more sensitive to daylight duration. Bird biology does not vary between song birds and poultry. The increasing day length in the spring results in song birds laying eggs and hatching young. The decreasing day length in the fall signals to song birds that it is no longer a good idea to reproduce and egg cessation occurs. Poultry respond in the same way with increasing day length triggering egg production and decreasing day length stopping egg production.

A laying hen should reach a day length of 16 hours light and 8 hours dark. Egg production will naturally occur in the spring with the increasing day length,

Table 3.1 Suggested lighting programs for egg producing poultry

Age (weeks)	Light (hours)	Dark (hours)
0–18	8	16
19	9	15
20	9:30	14:30
21	10	14
22	10:30	13:30
23	11	13
24	11:30	12:30
25	12	12
26	12:30	11:30
27	13	11
28	13:30	10:30
29	14	10
30	14:30	9:30
31	15	9
32	15:30	8:30
33	16	8
34 to end of lay	16	8

Figure 3.1 Example of pine shavings being used as a substrate for this chick's enclosure. (Source: Photograph courtesy of Dr. Cheryl Greenacre.)

but artificial lighting will be needed to maintain egg production following the summer solstice. Similar to natural day lengths, a sudden jump from a short day length to a long day length should be avoided. Therefore, a suggested lighting scheme is found in Table 3.1. The important thing to remember about egg laying is that once the day length has increased and is established, any decrease in day length will result in egg cessation. For example, an owner begins to artificially increase the light in the hen house but does not set the timer correctly. The day length increases, but the birds receive 22 hours of day light for several weeks. The owner observes the mistake and wishes to decrease the length to 16 hours. Decreasing the light at this point will knock the hens out of production, so the only option is to maintain the 22 hours of daylight if eggs are desired.

Litter substrate

Many options exist when discussing litter substrate or bedding material. Ideally, the bedding material should be absorbent, loose, and fairly inexpensive. The most common substrate used is pine wood shavings (Figure 3.1). Straw, sand, shredded newspaper, crushed corn cobs, and soybean hulls are just some of the substrates that can be used. Table 3.2 summarizes the positives and negatives for some litter substrates. Poultry can be bedded with any of these materials, but management techniques may change depending on the litter substrate. Hard wood shavings should not be used with poultry due to the potential presence of fungus and molds. This can result in respiratory infections if the levels are high enough in the shavings; therefore,

Table 3.2 The positives and negatives of various litter substrates available for poultry

| Substrate	Positive	Negative
Wood shavings	Absorbent, fairly cheap	Depending on location availability may be challenging or very expensive
Straw	Cheap, abundant	Does not absorb moisture due to wax sheath on straw, but chopping into 1–2 inch pieces can help reduce this deficiency
Sand	Abundant, moisture easily drained, maintains coolness in hot weather	Hard to heat, can be costly
Newspaper	Abundant, cheap	Slippery when wet unless shredded, not very absorbent

the safest recommendation is to not use hard wood shavings.

Managing different life stages

Brooding

The management of chicks, ducklings, goslings, poults, and other baby poultry is similar with the largest difference attributed to dietary need. The brooder pen or area should be set-up 48–72 hours prior to chick arrival to ensure all equipment is functioning correctly and allow time for all environmental variables to warm to the brooding temperature. The layout of the brooding area needs to provide feed, water, and heat to ensure a good start for the baby poultry. The ideal set-up is illustrated in Figure 3.2. The heat bulb is centrally located with feed and water alternating around the heat source, providing baby poultry access to feed and water in every direction. Smaller set-ups may use a box, cattle tank, or other similar area in which to brood.

The rule of thumb for brooding is to start with a 95°F temperature for the first week and decrease by 5°F each week until the outdoor temperature is met. This may indicate that during the day heat lamps will not be needed, but at night with cooler temperatures chicks still have access to heat lamps. The heat lamp can be purchased at a pet store or local agricultural food store and is different from a 100 W light bulb. The heat lamp can be purchased to illuminate with white or red light. White light can be used, but red light tends to be most common. Observation of the chicks around the
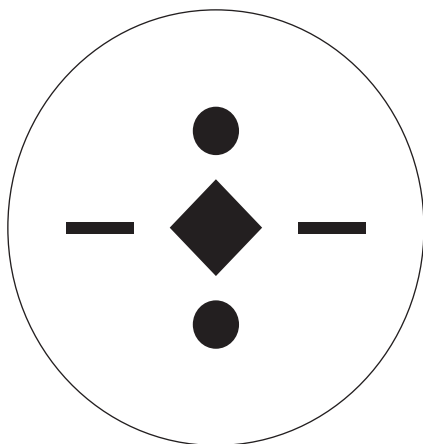
Figure 3.3 Chicks shown huddled together under a heat lamp, suggesting they are kept at too low a temperature. They should be scattered throughout the enclosure. Notice the food and waterer are placed away from the heat lamp. (Source: Photograph courtesy of Dr. Cheryl Greenacre.)

brooder can provide lots of information to the poultry enthusiast. Chicks huddled under the heat bulb are an indication that they are cold (Figure 3.3); observing chicks on the perimeter of the brooding area indicates that chicks are too hot; seeing chicks huddled together in one area of the brooder, not under the bulb, indicates there is a draft. The chicks will be most comfortable when they are evenly distributed throughout the brood area. Besides the physical location of the chicks in the brood area, the amount of noise produced by the birds will indicate whether they are cold or hot (excessive chirping by all) or comfortable (some chirping, but not all of the chicks).

Grow-out

Poultry growing results in changes to body weight and the loss of down, which is replaced by feathers. Outdoor access should be limited to birds 6 weeks of age and older. The birds will be predominantly feathered by 6 weeks and can tolerate the environment better than chicks. The feed and water should be maintained within the chicken coop and, depending on location, additional water may be placed outside during the summer months. Outside water should be located in the shade, but preferably not under a tree or bush. Wild birds will be attracted to the water and you do not want to provide a drinking source under an area where wild birds can excrete into the water.

The young poultry can be trained to re-enter the house at night by keeping the lights on in the house for an additional 20–30 minutes past sunset. The first week or two

Figure 3.2 Brooding area layout. The brooder should have waterers (●), feeders (▬), and heat lamp (◆) arranged within the brooder ring.

might require additional help in catching and placing the birds back into the chicken coop, but the birds will learn to go in at night as sunset approaches.

Adulthood

Mature chickens are easy to care for and can provide a sense of self-worth to a young or aged person. All aspects of water, feed, and temperature have been discussed previously and no additional management changes are needed with adult birds. An important item to remember is that birds establish a social hierarchy and disrupting this by removal of hens or roosters will result in aggressive fighting behavior between the birds. This is common and will resolve quickly as the new hierarchy is established.

The length of time for which an individual has backyard chickens can vary depending on the end goal (meat, eggs, or pleasure). Chickens that are producing eggs or are kept for pleasure will go through a molt period. A molt indicates that the hen biologically needs to rest from egg production and results in the growth of new feathers and cessation of eggs. Individuals may be concerned about the excessive loss of feathers or loss of egg production. A molt period will usually last 6 weeks; if the proper lighting and feeding is in place the hens will again produce eggs.

General management practices

Litter

Litter substrate was discussed earlier, but litter management was not. There is no need to remove the litter from the brooding area unless there is a water spill that soaks the bedding. New substrate can be added as needed to keep the bedding cleaner but does not require removal of the existing material. Unless there is a disease issue or government regulations that require bedding material to be cleaned every so often, the material can be removed every 6–12 months. The recommendation would be to remove litter in April and October if litter is to be removed twice. If clean-out is going to occur once, then April would be the best time. The cleaning out following the winter months allows for removal of damp material, a spring cleaning, and in the fall would provide a time to disinfect for external parasites before confining the flock to the house for the winter months. A downside to a fall clean-out is that litter material that can provide additional insulation for the winter months will be eliminated.

Feed and water

Birds need to have fresh feed and water on a daily basis. The feed should be stored in a cool dry environment inside a rodent-proof container. Plastic or metal trash cans can provide the necessary protection against rodents and other animals (raccoons, opossum, etc.) that might try to access the feed. One should only purchase enough feed that can be consumed within 30 days to ensure nutritional content and reduce the chance of spoilage. Water should be changed daily and, if needed, the waterer can be scrubbed with soapy water to remove any dirt or bacterial films that may develop. The drinking water can be chlorinated if needed, to help control bacteria and as a disinfectant, by making a stock solution (1 tbsp bleach: 0.5 gal water) and adding 2 tbsp stock solution to a gallon of water. The stock solution should be replaced weekly to ensure the effectiveness of the chlorine is not lost.

Space requirements

The commercial poultry industry has many auditing programs relating to space requirements. Every person and backyard resource has different recommendations on the necessary space required for poultry. Table 3.3 provides minimum space requirements for feeder space, waterer space, and floor space [1]. Backyard birds can easily be provided with additional space but limitations of housing structure, location (urban or country setting), or number of birds can have an impact. The general trend is to increase space provided as the birds age. The space requirements suggested in Table 3.3 do not include outdoor access and are based on medium-sized body birds.

Behavior disorders

Cannibalism

Birds are naturally aggressive and are omnivores. Chickens can become cannibalistic (peck at one another) if they are too densely populated or do not have enough resource space, that is, feeder, waterer, nestbox; incorrect lighting; abrasions or tears as a result of injury or mating; dietary deficiencies; prolapse; or meanness of a breed. As a result, the management of the flock needs to be adjusted to limit or reduce the behavior. Some potential remedies may include increasing space, dimming the lights to minimize activity, removing the wounded bird, applying an "anti-pick" compound to cover the affected

Table 3.3 Space requirements for chickens

Age (weeks)	Feeder space (linear inch)	Waterer space (linear inch)	Floor space/bird (square inch)
0–6	0.72	0.44	103
6–18	1.50	0.58	215
18 or older	3.00	0.75	377

Source: McGlone, John J. and Swanson, Janice (2010) [1].

area, or beak modification "trimming." Attempts can also be made to redirect the behavior by suspending hay slices to promote manipulation of the individual pieces of grass, broadcasting mixed grains into the litter to promote foraging behavior, or spreading grass clippings over the litter. While these are suggested remedies, each flock may react differently so several things may need to be tried until successful. Some suggest a red light may decrease pecking of others since blood or hemorrhagic areas may not induce curiosity and tempt them to peck.

Broodiness

Hens can go "broody," during which time the hen's hormones have changed her behavior, indicating time for nesting and hatching the young. A broody hen will cease egg laying, identify a nestbox or area within the coop that is her nest, and may increase aggressiveness as she attempts to protect her eggs. The best way to reduce the chances of having a broody hen is to remove eggs from the hen house on a daily basis. However, this may not always prove to be successful. A broody hen should be removed from the flock for a short period of time to break the hormonal cycle. This is achieved by eliminating her nest area and any eggs, even those laid by other hens, to prevent her from attempting to claim them as her own. Most backyard flock owners want their hens to lay eggs rather than nest and hatch them out.

Egg eating

Chickens may occasionally develop a habit of eating their own or other hen's eggs. The behavior can develop as a result of overcrowding, uneven nest space, nutritional deficiency, too bright light intensity, or disposing of cracked or broken eggs in the chicken coop. Similar to other behaviors discussed above, the behavior can be broken or redirected at times. Solutions may include frequent gathering of eggs, increasing nest availability, darkening the nests, or beak modification. If the egg eating cannot be stopped, the best approach may be to induce a molt, causing the cessation of eggs to break the

behavior and bringing the hens back into production several weeks later.

Predator control

Predator control is a must for anyone who wishes to keep backyard poultry. As was previously mentioned, the design of the chicken coop can help reduce the number of wild birds, and in some instances predators, that enter the chicken coop. A small hole where a predator can gain access or pull a chicken body part through the hole can result in a grizzly scene the following day. The best approach is to ensure all holes or other access into the chicken coop is secured to eliminate the potential for predators. Raccoons, opossums, mink, skunks, foxes, coyotes, or weasels may find ways to enter the chicken coop or pull the chickens through wire and holes to consume them. If the birds have outdoor access, a sufficient wire enclosure may be needed to protect them. A wire fence can be used for the outdoor run, but should be buried 8 inches deep to ensure that predators are unable to dig under the fence to enter the chicken yard. Additionally, shrubs and bushes can provide cover from an aerial attack by raptors, or a mesh can cover the top of the chicken yard to prevent bird of prey attacks. Depending on state laws, consultation with animal control can provide information on how best to deal with a predator attack in the backyard flock. Dogs can be responsible for what appears to be a predator attack as well. Dogs tend to kill the birds as a result of trying to play with the chickens and chickens are typically not maimed as in a predator attack.

Conclusion

Backyard poultry can be an exciting endeavor as long as proper management practices are employed. Chicken coop design can dramatically impact the effectiveness

of ventilation, temperature, and predator control. Management practices tend to be more intense early in life (brooding), but as the birds age they are more forgiving to management errors. Finally, behaviors may develop that are detrimental to the birds or eggs and need to be addressed as quickly as possible.

Reference

1 McGlone, John J. and Swanson, Janice (2010) Poultry, in *Guide for the Care and Use of Agricultural Animals in Research and Teaching*, 3rd edn, Federation of Animal Science Societies, Champaign, pp. 102–127.

Anseriforme Husbandry and Management

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Introduction

Ducks have been called "the easiest domestic birds to raise." [1] Combined with a tolerance to a variety of weather conditions, foraging and insect control abilities and resistance to numerous diseases that commonly plague chickens and other captive poultry, ducks are popular pets. Not far behind are pet geese and a distant third are captive swans. Collectively, waterfowl species are common in public and private collections as well as beloved pets. Ducks and geese are also raised for meat, eggs, and foie gras (although now outlawed in many countries) and should be considered prior to the administration of any medications.

Ducks, in particular, can be used to reduce local pest insect and water plant populations. Holderread writes that two to six ducks per acre (0.4 hectare) can be used to "control Japanese beetles, grasshoppers, snails, slugs and fire ants." [1] As a note, excessive fire ant populations can result in damage to ducks that are confined to land (see Figure 4.1). Ducks may also be used to control livestock liver flukes as they eat the snail intermediate host. Ducks are also used to clear out pest aquatic plants including duckweed (*Lemna* spp.), pondweed (*Potamogeton* spp.), green algae, skunkweed (*Chara* spp.), widgeon grass (*Ruppia maritima*), wild celery (*Vallisneria* americana), arrowhead (*Sagittaria* spp.), and more. Quantities in the region of 15–30 birds per water acre (0.4 hectare) may be needed to remove heavy plant growths, while 8–15 birds per acre can be used for maintenance control [1].

Waterfowl droppings are generally voluminous and can be both beneficial and detrimental. The obvious downside is that even a single bird can quickly contaminate a small area (land or water) and is one of many reasons that waterfowl should be provided with adequate space and, ideally, outdoor housing. On a positive note, ducks can provide readily degradable fertilizer for gardens, (as can geese) yards and, (as can swans) ponds and streams to feed fish and provide valuable nutrients to the water environment (provided water is adequately aerated, circulated and replaced). To limit damage from ducks that forage through gardens, restrict access to tender crops (lettuce, spinach, cabbage, and young plants), low hanging ripe fruits, and during irrigation [1].

Pet waterfowl generally produce acceptable noise levels in urban environments. Small flocks of waterfowl are generally quiet except when disturbed. Single waterfowl (especially geese) may be quite noisy, possibly as a result of being alone and more nervous. Of the duck species, call ducks tend to be the noisiest with Pekin breeds in second place [1]. While not entirely mute, Muscovy ducks (*Cairina moschata*) are the quietest of the domestic ducks.

General groups and features of pet waterfowl

While waterfowl are commonly classified via genetics or taxonomy, feeding and movement styles are used here: How a bird feeds and moves around helps one set up environments that best suit the animal. For example, most domestic ducks are mallards (*Anas platyrhynchos*) and are dabblers that benefit from walking on land but also feed and spend time on water. Common backyard setups for pet ducks often lack clean accessible water and many birds spend most of their time standing or

Figure 4.1 White Pekin duck (*Anas platyrhynchos*) with extensive damage to the foot webbing from fire ant bites. The lesions have healed leaving areas of missing interdigital webbing.

Figure 4.2 A gaggle consisting of two White Chinese (*Anser* cygnoides) and 1 Sebastopol (*Anser anser domesticus*) geese are walking on hard packed dirt. If no other substrate is available, this environment is conducive to foot and joint problems such as bumble foot and arthritis.

(a)

(b)

Figure 4.3 (a) This pintail drake (*Anas acuta*) is a dabbling duck. Notice the centrally placed legs which enable dabblers to walk well on land. (b) A black swan cob (*Cygnus atratus*) is dabbling on the surface of the water.

walking on hard surfaces and eat from a bowl on land (see Figure 4.2). This scenario may contribute to inactivity, obesity, arthritis, poor hygiene, pododermatitis, and more.

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. Examples of dabbling ducks include teals, widgeons, mallards, shovelers, pintails, and gadwalls (all *Anas* genus). Most swans are also dabblers. The mallard is the best known of all ducks and is the wild ancestor to all domestic ducks except the Muscovy (see Figures 4.3 a,b).

Divers are waterfowl that feed primarily under water. Ducks of this group belong to the subfamily Aythyinae.

Compared to dabblers, divers have legs placed more caudally on their bodies to help propel them underwater. However, divers tend to walk poorly on land, if at all. Examples of divers include bufflehead (*Bucephala albeola*), pochard, scaup, canvasback (*Aythya valisineria*), redhead (all *Aythya* genus), ruddy (*Oxyura jamaicensis*), and marbled (*Marmaronetta angustirostris*) ducks (see Figures 4.4 a,b).

Perchers tend to perch in trees, on top of logs or other raised surfaces. Examples of perchers include Mandarin (*Aix galericulata*), wood (*Aix sponsa*), torrent (*Merganetta armata*), maned (*Chenonetta jubata*), Hartlaub's (*Pteronetta hartlaubii*), Muscovy, and some whistling ducks (*Dendrocygna* genus), and the pygmy (*Nettapus* genus) and spur-winged geese (*Plectropterus gambensis*). Although not true of all, perchers tend to have longer legs and necks than dabblers and certainly divers (see Figures 4.5 a,b).

Grazers are primarily limited to herbivorous geese that eat terrestrial grasses, grains, and other plants. These birds are good walkers and spend a significant amount

(a)

(b)

Figure 4.4 (a) A ruddy duck drake (*Oxyura jamaicensis*) is resting on the water. As is common with divers, ruddy ducks have caudally placed legs which aid in swimming but make them poor walkers on land. (b) Another diver, the canvasback (*Aythya valisineria*), will dive to retrieve tubers, insect larvae, seeds, snails and more from the bottom substrate of waterways.

of time foraging on land. The Canada goose (*Branta canadensis*) is a good example (see Figures 4.6 a,b).

Important physical characteristics

Most domestic ducks and geese are poor or non-existent flyers – usually because they are simply too large and heavy for their wings. Domestic ducks of the same size or smaller than mallards and all wild types of waterfowl can be good flyers and precautions (pinioning, wing trims, appropriate housing) to prevent escape should be considered for captive populations.

The bill is a highly specialized organ and shows some degree of variation between different waterfowl species. Note that female mallards and their breeds often develop dark spots or streaks on their otherwise yellow to orange beak when they begin to lay. This is

(a)

(b)

Figure 4.5 (a) A wood duck drake (*Aix sponsa*) is attempting to rest on a flat piece of wood in a holding pen. Ideally hospitalized perching ducks should be provided round surfaces (such as logs) to give the option to "perch." (b) The silhouette of a spur-winged goose (*Plectropterus gambensis*) is seen perched high in a tree.

due to hormonal changes and is considered normal [1]. Mallards and other waterfowl have carotenoid pigmented beaks that may be used in mate selection [2]. The degree of coloration has also been linked to immune function [2].

Beak trimming is a practice sometimes used in commercial operations to reduce aggression and feather damage. If significant aggression and cage mate feather damaging is present, this suggests crowded or otherwise inappropriate housing conditions. The author does not recommend beak trimming but rather environmental modification to reduce animal stress.

As mentioned above, the legs of waterfowl are quite variable and are best suited to their preferred

(a)   (b)

Figure 4.6 (a) Cape Barren Goose (*Cereopsis novaehollandiae*) is casually grazing on grass. The characteristic heavy body and strong legs are common among grazers. (b) Domestic geese are classic grazers. This gaggle of mixed breeds resides in a grassy field ideal for grazers.

Figure 4.7 All captive waterfowl should be provided with soft grassy land. Grass sod strips can be seen in this backyard setting complete with two Buff geese (*Anser anser domesticus*). The grass provides forage and a soft substrate to walk upon for the geese.

environment. Divers reside primarily on water and occasionally rest on soft (grassy) land. Forcing divers to spend too much time walking on land can result in stress and leg and foot injuries. Most dabblers have legs designed for both agile swimming and walking and should be given access to both environments. Grazers and perchers generally have strong legs, making them well suited to terrestrial life. Muscovy ducks in particular have sharp talon-like claws to aid in perching. All terrestrial waterfowl environments should include soft (grassy) areas (see Figure 4.7). Perchers should also be provided with elevated rounded (logs or branches) surfaces. Chronically residing on hard substrates (packed earth, concrete, etc.), especially when combined with obesity, increases the risk of birds developing secondary pododermatitis (bumblefoot) and arthritis.

Only 3% of avian species, including waterfowl, possess a phallus [3]. Aside from external physical characteristics, most (adult) waterfowl can be sexed by identifying the phallus (or not as with females). With the bird standing or resting comfortably on its back, simply evert the cloaca and identify the phallus, which is located on the ventral floor of the cloaca and within the phallic sac (*saccus phalli*) [4] (see Figures 4.8 a,b). Sometimes the phallus can be gently palpated by inserting a lubricated gloved finger into the cloaca. Females have a smooth cloacal floor. Juvenile birds may be difficult to sex until the phallus becomes better developed.

Phallus length in waterfowl varies from 1.5 to greater than 40 cm and may be smooth and simple or highly convoluted, complete with grooves, spines, and a corkscrew shape [3]. Consequently, the female of the same species tends to have a vagina (simple to highly complex) that matches that of the male's phallus. The complexities of the phallus and vagina are positively correlated with the frequency of forced extra-pair copulations (FEPC) in the species in question. During FECP, females generally struggle and do not show receptivity (prone position with tail up) [4]. For example, the harlequin duck (*Histrionicus histrionicus*) and African goose (*Anser cygnoides*) (both of which do not engage in FECP) have short simple phalli and vaginas. The opposite is true with the long-tailed duck (*Clangula hyemalis*) and mallard (both species engage in FECP), which have long phalli and elaborate vaginas [3].

Basic behavior

One of the best known characteristics of waterfowl is their strong imprinting behavior. It has been noted that vocal imprinting (sounds encountered during

Figure 4.8 (a) When vent sexing waterfowl first expose the vent. (b) Using mild pressure slightly evert the cloaca. This Muscovy duck (*Cairina moschata*) is a hen. The phallus can be readily seen in species that engage in frequent forced extra pair copulations, such as with this Buff duck drake (*Anas platyrhynchos*), by everting the cloaca. Cr, cranial; Cd, caudal.

incubation) predates visual imprinting – at least in ducks [1]. Young waterfowl tend to readily follow the first person or animal they see, and possibly hear, at hatch. Upon reaching maturity, most waterfowl will stop this tracking behavior and integrate with others of the same species (ideal) or other birds, animals or as a single animal (not ideal).

In general, waterfowl are gentle animals and tolerate the presence of humans and other non-predatory animals well. Intra-species aggression is most common with crowding, when food or other valuable resources are limited and during mating and rearing times. Waterfowl are most aggressive towards humans when young are present and can occasionally be territorial.

Waterfowl demonstrate a pecking order much like that of chickens, with a top bird then number two, and so on. Fighting may erupt, especially when a new bird enters the flock. Generally, fights are limited in degree and intervention is only required if conflict results in serious injuries. As a means to reduce aggression among

groups of drakes, Holderread suggests "light neutering" birds by placing them in totally dark enclosures for 14–18 hours a day [1]. If such "light neutering" is used, the author recommends doing this only for a short period until the source(s) of aggression (crowding, mating season, etc.) is (are) resolved.

Common species of captive ducks and geese

Basic terminology

Several poultry organizations have set standards that define class, breed, and more for domestic ducks and geese. The American Poultry Association (APA, see www.amerpoultryassn.com), founded in Buffalo, New York, in 1873, is the oldest livestock organization in the United States. The American Bantam Association (ABA, see www.bantamclub.com) formed in 1914 also sets

standards for poultry, including ducks. The APA and ABA recognize many, but not all, of the same breeds [5]. Additional organizations set waterfowl standards, provide basic education for owners, work to preserve breeds, and more.

The "class" of duck or goose is based on weight. The APA has defined four classes of domestic duck: Bantam Duck (call, mallard, East Indie), Light Duck (magpie, Campbell, runner, Welsh Harlequin), Medium Duck (buff, Cayuga, crested, Swedish), and Heavy Duck (appleyard, Aylesbury, Muscovy, Pekin, Rouen, Saxony) (see Figures 4.9 a,b,c,d,e,f,g,h). Similarly, three classes of domestic goose are defined as follows: Light Goose (Canada, Chinese, Egyptian, Tufted Roman), Medium Goose (American Buff, pilgrim, Pomeranian, Sebastapol, Steinbacher), and Heavy Goose (African, Embden, Toulouse) [6] (see Figures 4.10 a,b,c,d,e,f). Note that the Egyptian goose (*Alopochen aegyptiacus*) is often placed in the shelduck family, placing it in between a goose and duck.

(a)   (b)   (c)

(d)   (e)   (f)

(g)   (h)

Figure 4.9 Domestic ducks can be divided into the following classes: Bantam Duck, Light Duck, Medium Duck and Heavy Duck. With the exception of the Muscovy duck (*Cairina moschata*) all other ducks are *Anas platyrhynchos*. This Gray Call drake (a) and Mallard hen in molting plumage (b) are Bantam ducks. The Chocolate Runner (c) and Khaki Campbell (drakes) (d) are Light ducks. The Buff (e, foreground) and Crested (f) are Medium ducks. This Muscovy drake (g) and White Pekin (h) varieties are heavy ducks.
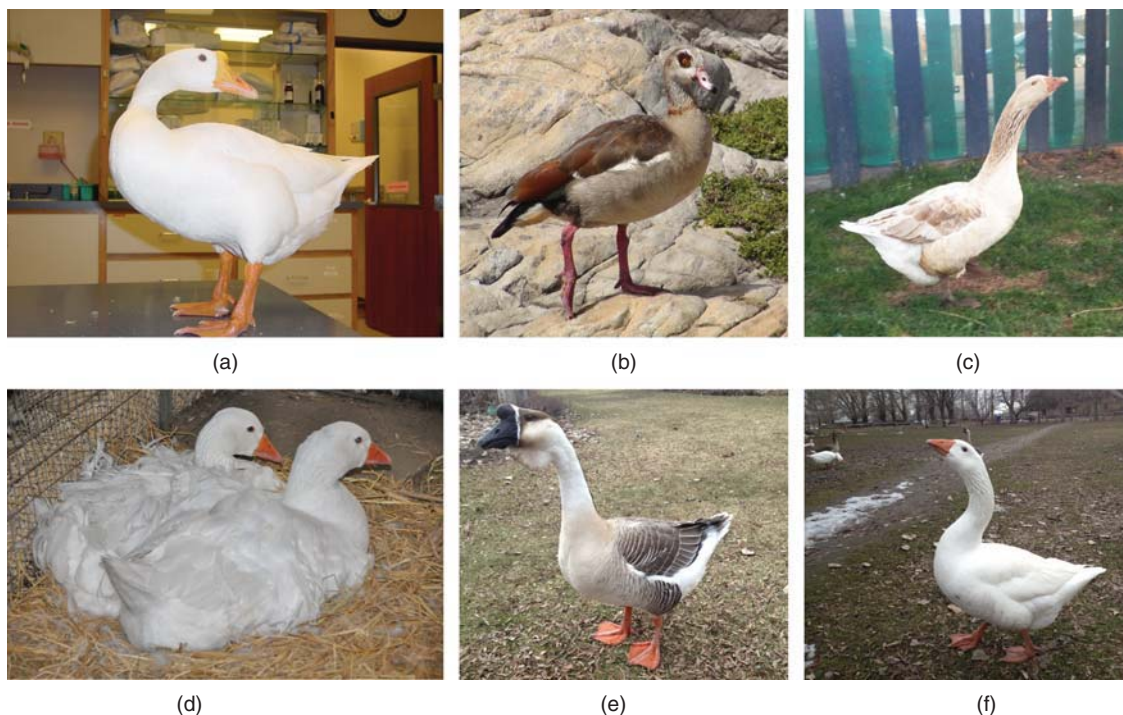
Figure 4.10 Domestic geese can be divided into the following classes: Light Goose, Medium Goose and Heavy Goose. African and Chinese geese are *Anser* cygnoides, the Egyptian goose is *Alopochen aegyptiacus* while the others are *Anser anser domesticus*. The White Chinese (a) and Egyptian (b) are Light geese. The American Buff (c) and Sebastopol (d, Courtesy of Abby Perata) are examples of Medium geese. The African (e) and Embden (f) are Heavy geese.

The "breed" of waterfowl (ducks and geese) is based on distinctive physical characteristics that were often developed over decades to millennia. As with dogs and other domestic animals, "breed type" and variation can be significant among domestic waterfowl. Breed and breed type may be distinguishable by size, body silhouette, plumage patterns and color, and more (see Figure 4.11).

The "variety" of bird is usually distinguishable by the plumage color or pattern [1]. For example, White Aylesbury ducks are white and come in one variety. However, the Runner duck comes in eight recognized varieties: blue, black, chocolate, white, penciled, gray, fawn and white and buff [1] (see Figures 4.12 a,b,c).

Finally, the "strain" of waterfowl refers to a particular breed that descends from one flock or breeding farm [1]. Birds within a strain are generally inbred to achieve specific traits, such as high egg production. These traits can actually be significantly different between strains yet still remain within the same breed. The strain is usually identified by the originator's name in the prefix. Examples of duck strains include Legarth Pekins, Horton East Indies, and Lundgren White Calls [1].

Basic terminology includes names given to males, females, and young. An adult male duck is a "drake," a female is a "hen," and a baby is a "duckling." A "mule duck" or "mule" is an infertile hybrid and most commonly refers to the offspring of a domestic mallard hen and Muscovy drake [7,8]. A "hinny duck" or "hinny" is the offspring of a domestic mallard drake and Muscovy hen [8].

An adult male goose is a "gander," the female is a "goose," and the baby is a "gosling." An adult male swan is a "cob," a female is a "pen," and a baby is a "cygnet." [5]

A group of ducks are commonly referred to as a "flock" but may also be called a "bunch," "paddling," or "raft" on water; a "safe" or "badelynge" on land or more specifically a "flock" in flight. Other names for groups of ducks include a "brace" (for a pair), "brood" (newly hatched and with their mother), and "dover." A group of geese on the ground is referred to as a "gaggle" and in fight a "skein." A group of swans is referred to as a "bevy" or

Figure 4.11 The Roman (*Anser anser domesticus*) is a breed of goose and is distinguished by the tuft of feathers on the top of its head, relatively small size with good meat to bone ratio. Contrast the Roman with another all white breed the Embden goose (figure 4.10f) which is known as being heavy and the tallest of the domestic geese.

"wedge" in flight. A "lamentation" can refer to swans on land or water.

Ducks

Domestic ducks come either from the mallard (*Anas platyrhynchos*) or Muscovy duck (*Cairina moschata*)breeds. The Pekin, Khaki Campbell, Call, Runner, Rouen, Buff, Swedish and Crested are examples of common duck breeds all believed to be descended from mallards, while Muscovy ducks (and all of their color varieties) are distinctly different.

It is believed that duck domestication began in China during the Zhou Dynasty (514–495 BC) with the Pekin duck being one of the earlier breeds [9]. Pekin ducks are the classic white-feathered, yellow billed ducks that are commonly kept as pets. Runner ducks are recognized by their vertical body posture and move with quick steps rather than waddle. Crested ducks have a unique crest on the back of their head, sometimes associated with a malformed skull and brain, intracranial fat bodies, and subsequent neurological disease [10,11]. Khaki Campbell ducks are noted for their high egg production. Ducks are most often kept as companions, display (collection) animals, and commercially for egg, meat, and foie gras production.

Several features can be used to distinguish males from females. Domestic female ducks "quack" while the males have a hoarse "cough" [1]. Mature males tend to be larger, have a curled tail (although not while molting) and are more ornately colored (for non-white breeds). *Anas*, and other, genus males have an osseous syringeal bulla that is readily seen on radiographs (see Figures 4.13 a,b,c).

Muscovy ducks are best recognized by their warty features (caruncles) on the featherless portions of the face (mature birds), tendency to perch and roost like chickens, long claws on their feet, and a hissing sound instead of a "quack" [12]. Drakes have a fleshy knob at the base of their upper bill, more pronounced facial caruncles and a short erectile crest of feathers on the top of the head. Muscovy ducks can breed with mallards; however their offspring are sterile (mule). Ducks commonly interbreed when kept with multiple species.

Geese

Domestic geese are derived either from the graylag goose (*Anser anser*) or swan goose (*Anser cygnoides*). Although the exact origins can be argued, the eastern Asian (Chinese and African) geese are generally

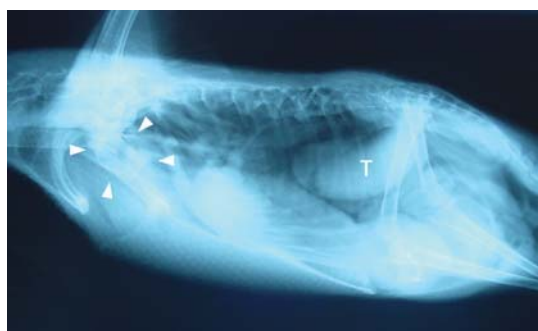(a)   (b)   (c)
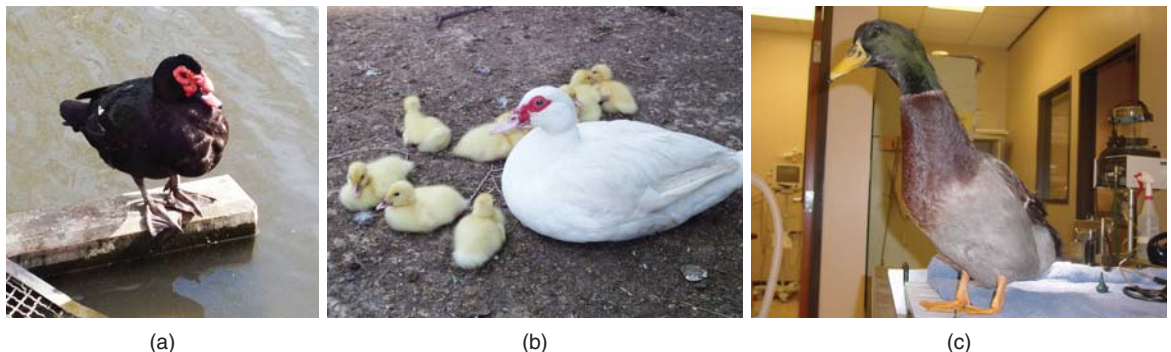
Figure 4.12 Domestic waterfowl breeds can come in many varieties. For example the Black Muscovy drake (a) and White Muscovy hen with brood of ducklings (b) are two varieties of the same breed (*Cairina moschata*). Some, such as this Rouen drake (*Anas platyrhynchos*) (c), only come in one variety.
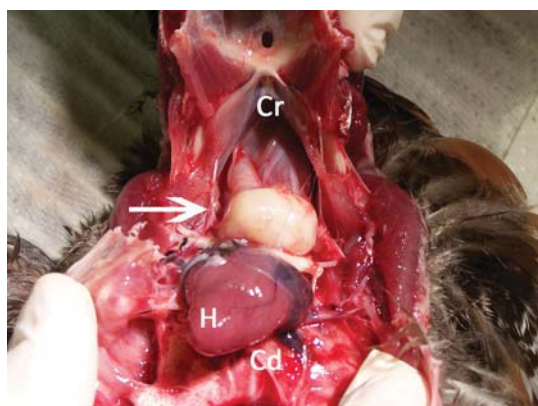
(a)
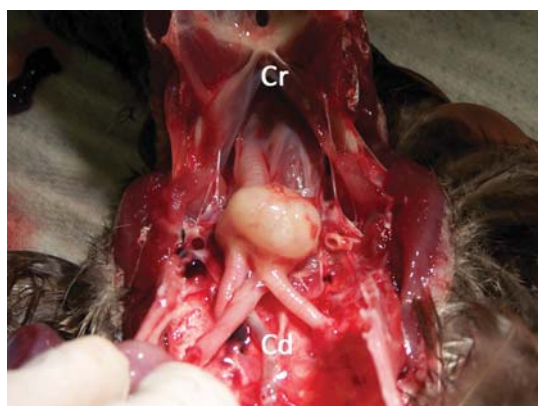
(b)

(c)

Figure 4.13 Some male ducks possess osseous syringeal bulla at the terminal end of the trachea and should be recognized as normal. (a) In this *Anas* genus duck, the sternum is reflected cranially exposing the heart (H) and osseous sryingeal bulla (arrow). (b) With the heart removed, the primary bronchi can be seen exiting the caudal aspect of the bullae. (c) This structure may be readily seen in drakes on radiographs (outlined by arrowheads). This domestic mallard drake (*Anas platyrhynchos*) was reproductively active and had enlarged testes (T). Cr, cranial; Cd, caudal.

believed to come from the swan goose and the western Asian, North African, and European (European) geese are domesticated greylags. Domestic "Chinese" and "African" geese are distinguished by the large knob at the base of the upper bill which is not present in the "European" varieties.

Common goose breeds include the Toulouse, Embden, and White Chinese and are either *Anser anser domesticus* or *Anser cygnoides* or hybrids. The Toulouse is well known for foie gras production and it's dark gray (along the back) to light gray edged (breast and ventrum) feathers. Embden is pure white with an orange bill. Chinese geese come in White and Brown varieties and have a characteristic knob at the base of the upper bill (Figure 4.14 a,b).

Swans

Mute (*Cygnus olor*), trumpeter (*Cygnus buccinator*), black (*Cygnus atratus*), black neck (*Cygnus melancoryphus*), and Coscoroba (*Coscoroba coscoroba*) swans are most commonly kept as pets. As the name implies, mute swans are less noisy than others; however they will hiss (usually defensively or aggressively), whistle, or snort.

Zoos, aviaries, and some specialized private collections may have a large number of different waterfowl species representing the approximately 150 members of the family Anseriformes. For more specific details, including breed characteristics, about common captive waterfowl, see Holderread and Ekarius [1,5].

Basic reproduction

Swans and geese tend to form strong monogamous pair bonds, whereas ducks (especially domestics) tend to be polygamous. Ducks will also breed, or at least attempt to breed, with different species of other ducks and small geese. Interestingly, the vagina of some waterfowl species has multiple blind pouches and spirals that may act as anatomical barriers to the phallus that prevent conception resulting from forced copulation [3,4]. Interspecies breeding is especially common if the duck was raised in mixed species collections. Resulting mixed species ducklings are common but are often sterile (see Figures 4.15 a,b).

Waterfowl may breed on land or water. A few species, such as the magpie goose (*Anseranas semipalmata*), Cape Barren goose (*Cereopsis novaehollandiae*) and Hawaiian goose (*Branta sandvicensis*), breed exclusively on land [3]. Most wild waterfowl tend to breed on water, while domestic ducks commonly breed anywhere convenient.

(a)

(a)

(b)

Figure 4.15 Ducks commonly interbred creating interesting offspring that are generally considered to be sterile mules. (a) American widgeon (*Anas americana*) X blue wing teal (*Anas discors*) hen. (b) Marbled teal (*Marmaronetta angustirostris*) X wood duck (Aix sponsa) drake.

(b)

Figure 4.14 (a) Characteristic of the swan goose (*Anser* cygnoides) derived eastern Asian geese, a large knob is present at the base of the upper bill as with this Brown Chinese. (b) European breeds derived from the graylag goose (*Anser anser*) lack the knob such as with this Toulouse. (Source: Photograph courtesy of Abby Perata.)

Most waterfowl lay eggs on the ground and tend to make shallow nests composed of plant matter and feathers pulled from a "brood patch" (see Figure 4.16). Wood ducks and buffleheads are examples of tree cavity nesters, but others, such as the mallard, will also opportunistically lay eggs in raised locations. Feather loss may or may not be evident over the ventral crop and breast regions. The mute (*Cygnus olor*) and other swans commonly build large raised nests from waterside vegetation (see Figure 4.17).

It is common for hens to show behavior and physical changes just prior to laying, including apparent lethargy, anorexia, ventral coelomic swelling, and a dilated vent, that may easily be mistaken for illness. Males also sometimes display lethargy and anorexia during breeding season. Once breeding season is complete and eggs have all been laid, birds generally return to normal (excluding the sitting behavior of hens).

Handling

Because of the many species variations among waterfowl, handling techniques do vary. In general, waterfowl

Figure 4.16 Nesting hens commonly pull feathers from their breast area creating a "brood patch". The feathers are often used in the nest construction as seen here with this wild mallard (*Anas platyrynchos*).

Figure 4.17 An exhibit trumpeter swan pen (*Cygnus buccinator*) sits next to her full nest. Items in the exhibit were used to create a large mound typical of many swan nests.

are easy to handle – especially domestic species. While most clinicians will be presented with individual birds, some may work with waterfowl flocks in private collections, zoos, aviaries, and through field work. The discussions below will pertain to captive waterfowl. Wild animal capture techniques are discussed in more detail elsewhere.

Most waterfowl are presented as single or paired birds in boxes, dog carriers, or simply unrestrained.

Regardless, for the safety of the animal it is always best to transport birds in a sturdy enclosure such as a dog travel crate. While they may be nervous, most domestic waterfowl will permit an examination with minimal to no handling. Some wild waterfowl are quite easy to handle when moved outside of their territory. Diving ducks generally require full handling for a complete examination simply because they don't stand or walk well and tend to rest on their breast when out of water. Of course, handling is often required for fractious animals and those requiring more detailed examinations or collections of diagnostics.

The most common defenses employed by waterfowl when restrained are clawing (especially Muscovy ducks), wing flapping, and biting. The larger birds have the greatest potential to induce damage to handlers.

Most waterfowl can be restrained by simply placing a hand or arm over the back to keep the wings tucked against the body. The head is minimally restrained and only if needed as most don't bite once the body is secured (see Figures 4.18 a,b). If needed, the handler's hand or arm can be extended around the bird to the middle of the breast. From here, the bird can be turned on its side or back to facilitate the necessary procedures. Some waterfowl will lay on their back in the crook of the handler's arm or on a flat surface (see Figures 4.19 a,b,c). Alternatively, the handler can use two hands with the palm on the back of the bird and each set of fingers extending to the bird's breast (see Figure 4.20). If the bird is in respiratory or cardiac distress, laying it on its back may exacerbate the problem and the bird should be kept upright.

With the bird secured either standing, suspended, or on its back or side, the legs may paddle freely. Small waterfowl rarely need their legs secured. Strong legs and prominent nails can result in scratch injuries to handlers. If needed, bring the restrained bird's back up against the handler's chest and use a free hand to hold the legs at the level of the tibiotarsii (see Figure 4.21). Use caution when handling legs on waterfowl with arthritis (which is common) as it may cause pain and result in more struggling.

Herding

Domestic ducks of mallard descent and domestic geese tend to form tight groups when pressured to move by predators and humans [1]. This behavior allows caretakers to direct birds to certain areas, such as night and holding pens, using simple methods. For small groups, handlers (working singly or in groups) can "herd" birds with outstretched arms, flashlights, flags, or other small visual devices (see Figure 4.22). For

(a)

(b)

Figure 4.18 (a) Most domestic waterfowl can be examined with minimal restraint. This Brown African goose (*Anser cygnoides*) with angel wing deformity is standing with no restraint in preparation for a wing bandage. (b) If needed, the restrainer's arm can be placed around or over the bird.

large flocks, consider working in (human) teams using bamboo poles, herding nets (seine, volleyball, etc.), or other devices to help span a larger area. Non-mallard ducks and wild waterfowl rarely follow these rules and swans are rarely encountered in groups, making other forms of group handing necessary.

Catching birds on water

Occasionally, birds need to be captured on open water in private collections and parks. Before attempting to capture wild waterfowl on open waterways, consult

(a)

(b)

(c)

Figure 4.19 By gently securing the wings across the back, waterfowl can be laid on their back in the crook of the handler's arm (a) or on a flat surface (b) as demonstrated on this mixed breed domestic duck (*Anas platyrhynchos*). (c) The same technique can be used with larger geese and swans such as the mute swan (Cygnus *olor*). Birds should only be kept on their back long enough to complete necessary exams and sample collection. Unless absolutely necessary, do not place distressed animals or those with cardiac or pulmonary disease on their back.

Figure 4.20   Small waterfowl, such as this Argentine ruddy duck drake (*Oxyura vittata*), can be handled by securing both wings using one (seen here) or two hands. The breast area is left unrestrained.

Figure 4.21   This mixed breed domestic duck (*Anas platyrynchos*) is being held up against the handler's chest with the bird's body facing forward. If flailing legs pose a problem, gently secure the tibiotarsi being careful to not put pressure on arthritic joints.

with local fish and game authorities as many waterfowl species are protected and capture may be considered illegal without appropriate authorization.

Assuming the animal is not trained and cannot be baited and captured at the water's edge or on land, the bird may need to be netted. Grazers and dabblers can

Figure 4.22   Mallard descent (*Anas platyrhynchos*) and domestic geese readily form tight groups when "herded" by people as shown with this small gaggle of Buff geese (*Anser anser domesticus*).

be "herded" on water using poles and nets (sometimes spanning the water and with the help of multiple people) and brought close enough to easily capture with a hand held net. Divers pose a different problem and should be carefully netted. Hand-held nets are the best but seine and other multiple person operated nets can be used as long as the netting is frequently checked to ensure the bird is not entangled unseen under water.

Swans

Swans are large birds and are generally docile unless threatened. The author finds swans easy to handle outside of their territory. In addition to biting and kicking, swans will often hit opponents using their wings which can potentially cause serious damage. If the swan is docile, minimal if any handling is needed as most will tolerate a full but gentle examination. If the swan is unruly or making threatening gestures, the wings should be secured first (the biting is minor in comparison). This is most safely accomplished by approaching swans from behind (usually with a second person in front of and in the line of sight of the bird). The wings are tucked into a normal resting position and secured while a second person performs a physical examination and any diagnostics if needed. The head is managed by simply moving the distal end of the neck away from the restrainer(s). Grabbing the neck may make the bird panic and is generally avoided. Chemical restraint may be needed for select individuals and for performing some diagnostics such as radiographs.

Basic housing

Waterfowl ideally need housing that protects them from inclement weather and predators while offering room to

freely walk around and have access to natural sunlight, fresh water for swimming and drinking, soft substrate and, especially in the case of geese, fresh grass to graze (see Figures 4.23 a,b,c,d,e,f,g). Suddenly changing the substrate may lead to waterfowl eating the new substrate (if small enough) leading to gastrointestinal impactions. While some pet waterfowl are pinioned to prevent flying, many simply don't fly and prefer to walk or swim.

Ponds can be natural or manmade ("kiddie" pool) and should contain fresh cool drinking water that allows waterfowl to completely submerge their head (as is needed to clean their nostrils and preen feathers) should be available at all times [13]. Bodies of water should be designed such that birds can easily enter and exit as needed. This generally means ensuring there is a gentle slope (natural or manmade) between the land and water. Larger ponds that are at risk of stagnation (especially in summer months) should be aerated to reduce the risk of botulism and other disease outbreaks. Waterfowl produce voluminous droppings and tend to dirty small bodies of water very rapidly. Frequent water changes are often needed.

Waterfowl incidentally consume sediment as they feed in water. "Sediment" includes non-food items that are associated with foraging behavior and includes mud, grit, and man-made objects. The bird's feeding style generally dictates how much sediment is ingested. For example, the piscivorous red-breasted merganser (*Mergus serrator*) feeds on fish within the water column and ingests less than 2% of its diet (dry matter) as sediment. In contrast, the benthos-feeding (feeds at the bottom of bodies of water) canvasback ingests 22% of its diet (dry matter) as sediment [14]. Dabblers and perchers, including the common mallard, generally ingest low amounts of sediment (less than 4% of its dry matter diet). Divers (ducks) and water grazers (geese and swans) generally ingest more sediment. To the best of the author's knowledge, sediment ingestion from land grazing has not been studied in waterfowl.

Heinz *et al.* showed that mallards experimentally fed diets containing up to 70% sediment (which was an estimated 46% consumed on a dry matter basis) had no adverse effects. When sediment reached 80% and 90% of the diet (representing 50% and 52% ingested on a dry matter basis), the birds lost a significant amount of weight [15]. While it is clear that some waterfowl (naturally and experimentally) safely ingest a large amount of sediment, the bigger concern is when the sediment contains toxins and other contaminates.

This sediment can provide nutrition, pass through unprocessed or expose the bird to environmental toxins, foreign bodies and other dangers. Areas with slow moving water and fine-textured sediment tend to be associated with elevated environmental toxins [14]. Lead ingestion is an obvious concern; however waterfowl morbidity and mortality due to other toxins in ingested sediment is well documented [14,16].

The foraging style of captive waterfowl should be considered when creating enclosures that include natural bodies of water such as streams, ponds, and lakes. Environmental sediment sampling should be considered prior to placing waterfowl at risk in natural settings. Man-made water-bearing structures in exhibits and private collections have the benefit of reducing risk of toxin exposure as long as the construction and materials are well designed and safe.

The larger the birds, the more destructive they can be to the enclosure. Mute swans typically feed in water by uprooting or cropping vegetation [14]. This normal behavior can be quite destructive to a nice planted exhibit.

Basic nutrition

General

Compared to commercial poultry, far fewer nutrition studies exist in relation to waterfowl. Commercial waterfowl diets are often formulated based on the National Research Council's (NRC) publication *Nutrient Requirements of Poultry*, 9th revised edition, published in 1994. The NRC publication offers detailed information on specific chicken diets as well as recommendations for other poultry species. However, much of the duck information is extrapolated from other species or based on limited studies in waterfowl.

For example, energy values for formulating duck diets are generally adopted from chicken bioassay data [17]. Rush *et al.* found that white Pekin duckling growth and toe ash weight were maximized with 0.95% and 0.85%, respectively, dietary calcium [18]. This is in contrast to the NRC recommendation of 0.6% dietary calcium and is just one example where published NRC guidelines may not be appropriate for all waterfowl. Rodehutscord and Dieckmann showed that young domestic ducks utilize plant and mineral phosphorous (in diets) very differently than that of same age turkeys, broilers, and quails [19]. These studies only serve to highlight nutritional differences between waterfowl (of which primarily domestic ducks have been evaluated) and chickens.

The NRC guidelines should only serve as a rough guide for waterfowl species. Some nutrients are even omitted for waterfowl. For example, there are no NRC guidelines for dietary zinc in waterfowl diets. Attia *et al.* evaluated
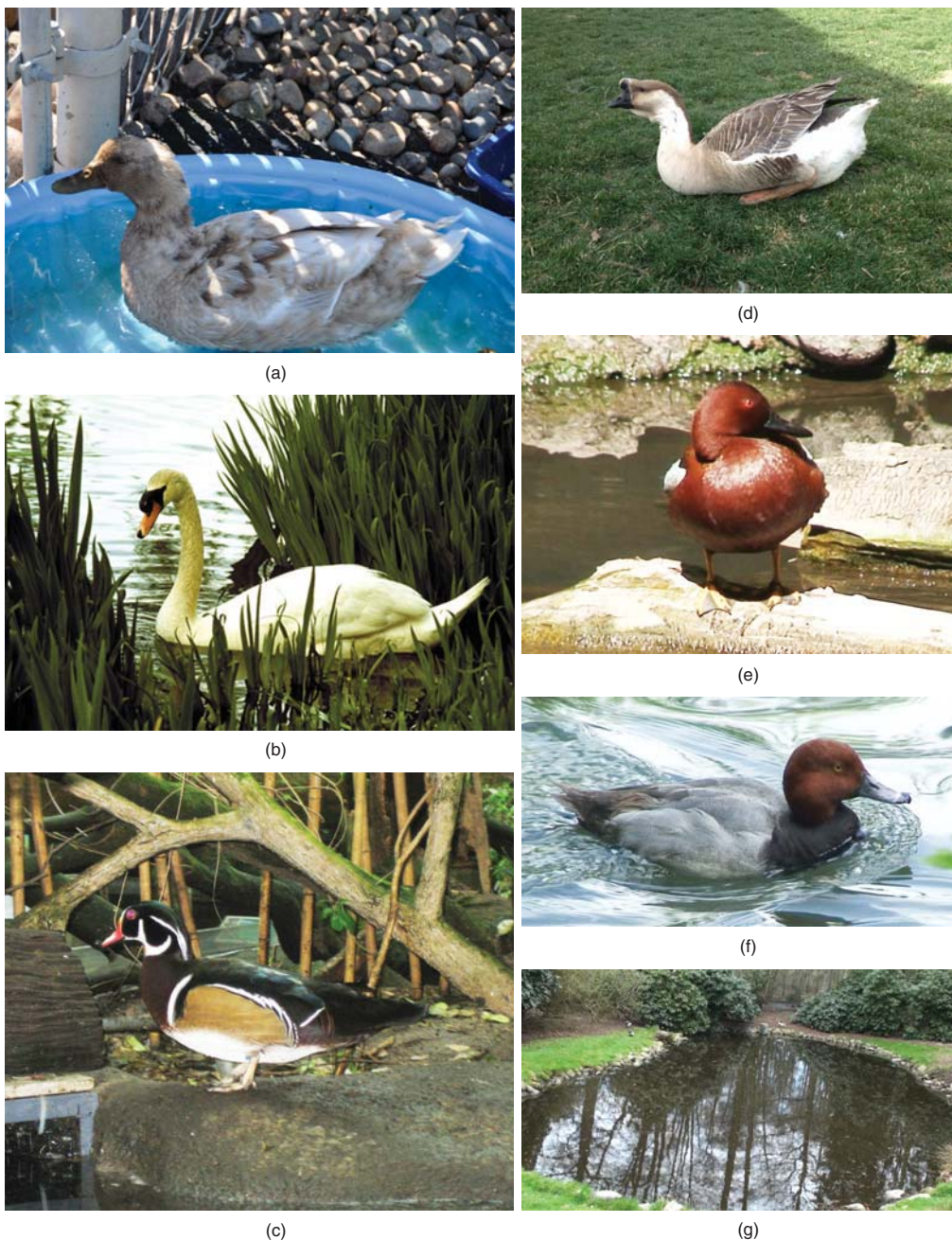
Figure 4.23 (a) Backyard ducks can be successfully provided swimming and comfort movement opportunities with a simple "kiddie" pool provided the water is kept clean as with this Khaki Campbell (*Anas platyrhynchos*). (b) Mute (*Cygnus olor*) and other swans ideally have access to water with naturally growing grasses to provide for swimming and natural forage. (c) Wood ducks (*Aix sponsa*- drake) and other perching waterfowl should be provided opportunities to perch on logs, branches or similar structures out of the water. (d) Grazers such as this Brown African goose (*Anser cygnoides*) spend most of their time on land and need grass as their primary substrate. (e) Dabblers like this cinnamon teal drake (Anas *cyanoptera*) need access to fresh water and comfortable places to rest on land. (f) Diving ducks such this redhead drake (*Aythya americana*) spend most of the their time on water and need enough depth to fully submerge their body and swim freely underwater. (g) Waterfowl ponds should have easy entrance/exit sites, some type of aeration and fresh water flow, grassy soft banks and large enough to accommodate the number of animals present.

several forms and levels of dietary zinc in white Pekin ducklings from 1 to 56 days old [20]. The authors concluded that 30 ppm was adequate for growth rate and zinc excretion in their studied birds [20].

Most of the waterfowl nutrition studies are limited to production ducks. Marie-Etancilin *et al.* noted that duck breeding is primarily directed towards fatty liver production with fat meat being a co-product [21]. Basso *et al.* stated that the most important economic factor with duck production is feed efficiency during growth (which is represented as optimal growth in the studies later in this section) [22]. As a result, our nutrition data pertinent to waterfowl are generally optimized for short-lived commercial duck production and not backyard, pet, or exhibit animals. Nevertheless, some of these studies provide insight into common nutrition-based diseases and means to improve overall health that may affect non-production birds. Specific nutrients and their additives will be discussed below.

Total energy and body scoring

Total energy requirements may be calculated by numerous means and are generally established for growth, maintenance, healing, and so on. For adult animals, energy requirements are often described with the intent to maintain an animal at a constant live weight. Cherry and Morris established that maintenance requirements for 7 genotypes of domestic drakes were 583 kJ/kgW$^{0.75}$ per day at 10°C (50°F) and 523 kJ/kgW$^{0.75}$ per day at 26°C (78.8°F) [23].

There are many factors affecting energy needs for animals including, but not limited to, health status,

reproductive activity, age, species, breed, strain, environmental conditions, activity level, and stressors. While calculating energy requirements for production animals can be important when determining feed rations and more, such calculations are not typically performed for non-production birds. The variables listed above make definitive energy requirements difficult to calculate for non-production birds.

Rather, the author relies on individual animal evaluation to determine if the bird is in an energy-positive or negative state. The author uses a combination of three methods to subjectively assess energy needs for birds (not just waterfowl); pectoral muscle score (PMS), body condition score (BCS), and health/environmental status. PMS and BCS have been described for many animals but have not been critically evaluated in birds (with the exception of budgerigars [*Melopsittacus undulatus*]). Using "yes" and "no" answers following an algorithm tree, a one to seven physical scoring system correlated with total body fat has been reported in budgerigars [24].

The systems described below are used by the author and have also not been critically evaluated or correlated with any disease or health status. As a note, advanced diagnostics such as radiographs, ultrasound, computed tomography, and magnetic resonance imaging can give a much more accurate account of muscle and fat content and can be used on conjunction with the scoring system below.

PMS is simply based on pectoral muscle mass and to a lesser degree strength (see Table 4.1). Scoring systems vary; however the author uses a one to five system

Table 4.1 Pectoral muscle score (PMS) system. Additionally, the breast muscle can be palpated to assess the softness or firmness of the pectorals. Pectoral muscles can be classified as soft (easily depressed when pressed with a finger), normal (minimally depressed when pressed with a finger) or firm (no depression when pressed with a finger). Soft pectoral muscles are most commonly associated with inactivity and obesity but may also be palpated with bruising and early localized inflammation. Firm muscles are most commonly associated with scar tissue, severe starvation and some infiltrative diseases such as sarcocystosis, granulomatous infections and neoplasia

	PMS 1	PMS 2	PMS 3	PMS 4	PMS 5
Physical Findings	Minimal pectoral muscle mass and it is concave, keel is readily palpable	Pectoral muscle mass is either linear or slightly convex, keel is readily palpable on its anterior surface	Pectoral muscle fills the breast, is slightly convex and forms an arc over the keel making a smooth transition from the left to right pectorals	pectoral muscles are convex enough to create a mild depression at the keel	Pectoral muscles form a significant convex arc and readily palpable depression at the keel commonly referred to as "cleavage"
Potential Causes	Starvation, end-stage illness, chronic disease	Disuse atrophy, chronic disease, non-specific weight loss	Normal	Normal to overweight, strong flyer	Obese

with three being average or "normal." A PMS of one means the pectoral muscle mass is minimal, concave, and the keel is easily palpable. A PMS of two indicates the keel is slightly palpable (in addition to the leading edge) and the pectoral muscle mass is either linear or slightly convex. A PMS of three indicates that the pectoral muscle fills the breast, is convex and forms an arc over the keel making a smooth transition from the left to right pectorals. A PMS of four indicates the pectoral muscles are convex enough to create a mild depression at the keel. A PMS of five indicates the pectoral muscles form a significant convex arc and readily palpable depression at the keel commonly referred to as "cleavage." The pectoral muscles may also be classified as soft (easily depressed when pressed with a finger), normal (minimally depressed when pressed with a finger), or firm (no depression when pressed with a finger).

BCS refers to the amount of fat detected in the skin, subcutaneous, and coelomic tissues (see Table 4.2). Scoring systems vary; however the author uses a one to five system with three being average or "normal." A BCS of one is only assessed during celiotomy and/or with advanced diagnostics and generally means that no dermal or subcutaneous and minimal to no intracoelomic fat is found. A BCS of two is also assessed during celiotomy and/or with advanced diagnostics and indicates that no dermal or subcutaneous but a mild amount diffuse intracoelomic fat (usually mesenteric) is present. A BCS of three can be assessed subjectively with physical exam and indicates that no dermal and minimal subcutaneous fat is present (usually along the caudal ventral coelom). A BCS of four indicates that no to minimal dermal and significant subcutaneous fat is present in one or more parts of the body (commonly along the caudal ventral coelom, breast, inner thighs, caudal dorsum [near base of tail], and neck). A BCS of five indicates dermal and significant diffuse subcutaneous fat is present.

The obvious disadvantage of this BCS system is the need for advanced diagnostics to assess scores of one and two. However, the system presented herein is designed to be used with PMS and health and environmental status considerations. At a minimum, BCS's of three to five can be accurately determined with physical exam and scores of one and two can be deduced based on other findings.

While a PMS and BCS of one often indicate severe catabolism and scores of five indicate significant energy excess, values of two to four can represent some variations of normal. For example, a non-flighted and inactive bird can have a PMS of two with soft muscle and a BCS of four with excess subcutaneous fat. So the bird in this example is overweight with decreased pectoral muscle mass due to inactivity and is very common with pet ducks. Conversely, a strong-flighted duck may have a PMS of four with normal muscle and BCS of two. This is common with healthy wild birds, especially towards the end of migration, or those in aviaries with large flight areas.

Last, health and environmental status are used when considering energy needs. For example, a clinically

Table 4.2 Body condition score (BCS) system

	BCS 1	BCS 2	BCS 3	BCS 4	BCS 5
Physical Findings	No dermal or subcutaneous and minimal to no intracoelomic fat is found*	No dermal or subcutaneous but a mild amount diffuse intracoelomic fat (usually mesenteric) is present*	No dermal and minimal subcutaneous fat is present (usually along the caudal ventral coelom)	No to minimal dermal and significant subcutaneous fat is present in one or more parts of the body (commonly along the caudal ventral coelom, breast, inner thighs and neck)	Dermal and significant diffuse subcutaneous fat is present
Potential Causes	Starvation, end-stage illness, chronic disease	Normal, chronic disease, non-specific weight loss	Normal	Overweight, 'well-conditioned'	Obese, metabolic derangement

*Can only be determined via celiotomy and/or by using advanced diagnostics such as computed tomography, magnetic resonance imaging or ultrasonography. Other findings are based on physical examination.

obese Pekin duck kept indoors will generally have lower energy requirements than a similarly obese bird kept outdoors with access to swim. Additionally, disease states can alter PMS and BCS values. For example, a bird with a wing injury will often have overall decreased PMS with more significant ipsilateral pectoral loss. Some birds with metabolic derangements (especially associated with liver and reproductive tract disorders) may have unusual fat deposits in areas not commonly seen. Because of the significant variables present, adjusting for health and environmental status are obviously the most subjective aspects of calculating energy needs for birds.

Aside from more accurate measurements using advanced diagnostics, the physical examination and daily weights often give clinicians the best assessment of a bird's energy needs. Accounting for the food in the gastrointestinal tract, weights typically fluctuate faster than PMS and BCS. PMS and BCS give clinicians better assessment of long term trends (over days to weeks).

Using PMS and BCS and by understanding the health and environmental conditions present, clinicians can better assess total energy needs for waterfowl. Caloric needs are adjusted based on the above findings. Underweight birds are provided with a more calorie dense diet with frequent feedings. Overweight birds are given fewer total calories and encouraged to exercise.

Total energy plans can be set for hospitalized birds and those in their normal environment. This may include using tube feeding products (usually ill individuals) and measured bowl (or other container) fed foods. As a note, calorie restriction in flocks may result in aggression and stress (see Section "Foraging enrichment") and should be considered prior to implementation.

Carbohydrates

Many breeds of domesticated ducks and geese have been developed for foie gras production and are predisposed to liver steatosis [25–27]. Studied mule ducks (Muscovy X Pekin) and specific breeds of domestic geese are highly susceptible to liver steatosis. Liver weight may increase 7–10-fold after just 2 weeks of overfeeding [27,28]. In a separate study, 70-day-old mule ducks were fed a carbohydrate-rich diet (corn) for 12.5 days and developed severe fatty livers. Liver weights (8% of body weight) were nearly 10-fold that of controls (1% of body weight), primarily as a result of lipid accumulation (60% of total weight). The study showed that by simply overfeeding a carbohydrate rich diet (corn), de novo hepatic lipogenesis in mule ducks predominated over dietary lipid intake to significantly alter lipid composition in hepatocytes. Liver steatosis is

likely common in certain waterfowl species (primarily domestics) because overfeeding results in intense lipogenesis that almost exclusively occurs in the liver of these birds [27,29]. Saez et al. noted that even with basal dietary conditions (non-overfed) Muscovy ducks "have a tendency for hepatic steatosis" [26].

Fatty liver disease, and fat accumulation in general, may result with excessive carbohydrate ingestion in ducks and geese [27,28,30]. Hepatic steatosis is strongly correlated with metabolic syndromes and is associated with liver injury, blood lipid metabolism, and peroxidation [30]. Species and breed determine the degree of hepatic steatosis development. As an example, liver lipids increased 85-fold and 50-fold in carbohydrate overfed Muscovy and common ducks, respectively [27]. Maize (corn) is the main component of foods used in overfeeding diets needed to achieve hepatic steatosis [26,27,31]. Ground corn, a simple carbohydrate source, is often the main ingredient in commercial waterfowl diets.

The author has noted significant obesity in geese, and especially captive ducks, fed diets high in simple carbohydrates. Just as domestic ducks and geese are predisposed to hepatic steatosis resulting from overfeeding, fatty liver is (generally) considered reversible when overfeeding is discontinued [27]. Strategies to reduce excessive body and (presumably) liver fat are to reduce dietary simple carbohydrates (corn, flour-based food, many pellet products), limit total food availability (except natural forage), encourage foraging, feed higher fiber leafy greens, increase physical enclosure space, and encourage swimming opportunities.

Reducing total pellet consumption is best accomplished in adult birds. Young waterfowl have specific dietary requirements for growth and inappropriate rations (as with reduced commercial pellets and increased other foods without the total diet being balanced) may result in serious nutritional diseases. As a result, the author recommends either feeding specific waterfowl grower pellets or using published and proven alternate diets for growing birds (at least until fully grown). The author does encourage young birds to forage on vegetation and insects (which does supplement the diet) but use pellets as the base food.

Protein

Amino acids have multiple roles in both protein and non-protein metabolism. While the many different amino acids have important roles, only methionine and arginine will be discussed in any detail here. Recommended total protein amounts will also be covered.

Methionine is especially important because it is considered the first limiting amino acid of poultry diets [32–34]. Common diets offered to waterfowl are often composed of cereal grains and soybean meal which have limited methionine content [32]. As a result these diets are usually supplemented.

Unfortunately, nutrition information in young waterfowl is scarce [35]. The methionine research is limited to production ducks and geese but does offer useful information as to growth and other factors which are important in young animals. This understanding becomes important when homemade or other non-commercial foods are fed to captive young waterfowl and should be a consideration if stunting or other problems are noted.

The NRC (1994) requirement for dietary methionine for ducks of 2–7 weeks old is 0.3% and is based on research in Muscovy ducks [33]. Jamroz et al. found that a diet containing a total of 0.40% methionine resulted in the best body weight (productive output) and ileal digestibility of cysteine and methionine for 1–21-day-old Pekin ducks [32]. Similar methionine supplementation resulted in improved body weight gain after 4 days of a duckling's life [35]. Xie et al. found that diets containing 0.377% and 0.379% methionine resulted in maximum weight gain and breast meat yield, respectively, in 21–49-day-old Pekin ducks [33]. During the same study, the authors found that the proposed methionine levels also resulted in decreased "abdominal" fat deposition as a possible result of this amino acid's effect on key enzymes of lipogenesis and lipolysis.

Wang et al. studied the effects of methionine on growing Yangzhou geese (common domestic goose in China) [34]. The authors reported that 28–42-day-old and 28–56-day-old goslings needed 4.07 g/kg and 4.14 g/kg feed, respectively, of methionine for optimal growth (maximum daily weight gain). These values are slightly higher than the dietary methionine (3.77 g/kg feed) level shown to result in maximum weight gain in 21–49-day-old white Pekin ducklings. "Abdominal" fat in the geese decreased linearly with increased methionine (as also shown in ducks). Although specific adverse effects were not elaborated upon, the authors noted that an excess of methionine resulted in an imbalanced dietary amino acid profile and altered metabolism [34].

In common with other uricotelic species, waterfowl cannot produce endogenous arginine and this amino acid must be available in the diet. The 1994 NRC arginine requirements for 0–2 and 2–7-week-old White Pekin ducks are 1.1% and 1.0% of the diet respectively [36]. Wang et al. concluded that for optimal weight gain, feed/gain, and breast meat yield of 1–21-day-old White Pekin ducklings, dietary arginine requirements should be 0.95% (9.5 g/kg), 1.16% (11.6 g/kg) and 0.99% (9.9 g/kg) of the diet respectively [36].

Wu et al. found that by adding 10 g/kg of L-arginine to a basal diet meeting NRC requirements for ducks, white Pekin ducklings gained significant weight and increased breast muscle size relative to total body weight by 5.2% and 9.9% respectively [37]. Arginine supplementation also resulted in significantly decreased skin fat and "abdominal" fat pad contents by 7.6% and 4.9% respectively. Twenty-one-day-old male and female ducks were given either a control or L-arginine supplemented diet for 3 weeks total. The basal diet contained 11.6 g/kg of arginine [37].

Other research has shown that lysine and valine requirements in starter and duck grower rations are higher than published NRC (1994) values [33]. The above findings and future research may reshape methionine, arginine, and other amino acid recommendations for young (and potentially adult) waterfowl.

By evaluating jejunal fluid contents, Zhao showed that dietary protein consumption significantly and directly altered intestinal amylase, trypsin, and chymotrypsin activity in 18-week-old Pekin ducks [38]. As protein consumption increased, so did intestinal enzyme activity. While this finding may be intuitive, it does support the concept that adequate dietary protein is required for optimal digestive enzyme activity and nutrient digestion.

Protein requirements are known to be variable between different young and aged ducks and likely between different species of waterfowl. The studies above were generally completed to determine methionine and arginine levels that result in maximum growth, which may not be an appropriate goal for display or pet birds. Also, methionine and other amino acid requirements can change based on total available protein and other nutrients in the diet. Because waterfowl (at least domestic ducks and geese) may have specific amino acid requirements (such as methionine) unique from other poultry, the author advises working with a nutritionist prior to creating a formulated diet for captive birds.

General dietary protein levels

Protein recommendations for waterfowl are more generalizations than critically studied rules. In general, commercial starter and grower diets contain 18–20% protein. Maintenance diets commonly contain 13–14% protein. Breeder and layer diets are typically between 16% and 20% protein. The higher the dietary protein content, the larger the eggs for laying birds. These

Figure 4.24 A wild Canada goose (*Branta canadensis*) with bilateral angel wing deformity. The bird was raised on a private park, developed angel wing when young, was not treated and is now a permanent resident as he can not fly.

general recommendations are in line with some popular literature such as Holderread [1].

While protein malnutrition often results in poor growth rates, excess can commonly be associated with "angel wing deformity" in young waterfowl (especially geese) (see Figure 4.24). Angel wing is pronation of the carpus. If left untreated, the wing is permanently deformed. Overfeeding and high protein diets are commonly blamed and result in rapid distal pin feather growth that outweighs the strength of the wing bones and muscles. Carpal rotation results and the affected distal wing has a "flipped up" appearance. If caught within a few days, the wing is simply taped or lightly bandaged (figure-of-eight wing bandage, or Braille bandage) into correct position and diet modified as needed (lower protein, more access to natural forage). The bandage is removed in 5–7 days or when the wing remains in normal position. Acute malformations and treatment carry an excellent prognosis. Geese are more prone to excessive protein supplementation as their natural diet generally consists of poor quality grasses and forage.

Fats and omega-3 fatty acids

Most commercial waterfowl diets are minimally supplemented with fat. Soybean oil and other plant-based oils (typically significantly higher in omega-6 than omega-3 fatty acids) are added bringing the total fat content to between 2% and 4% of the diet. The discussion below will focus on the addition of omega-3 fatty acids (O-3 FA) to supplement waterfowl diets for captive birds.

O-3 FA have been shown to have increasing roles in a variety of health and disease processes in multiple birds and mammals. Omega-3 research is limited in respect to waterfowl. However, the research completed so far in waterfowl and other avian species supports consideration of omega-3 fatty acid supplementation.

Omega-3 fatty acids have gained popularity for their anti-inflammatory, lipid-stabilizing and anti-neoplastic effects, renal protective properties, and other qualities. O-3 FAs are polyunsaturated and are designated by their first carbon-carbon double bond occurring at the third carbon from the methyl group. O-3 FAs are rich in eicosapentaenoic (EPA), docosohexaenoic (DHA) and/or linolenic acid (α-linolenic acid or ALA). DHA and EPA are considered the functional O-3 FAs as they exert the most beneficial biologic effects on body tissues [39].

Flax seed (and limited other plant sources) and menhaden (and other select fish and shellfish) oils contain predominately linolenic acid and EPA and DHA respectively, and therefore have different O-3 FA compositions. DHA and EPA are more readily incorporated into biological tissues, but also carry greater potential to create metabolic oxidative stress than linolenic acid. As a general rule, the more herbivorous the animal, the better that species is at converting ALA to DHA and EPA. Conversely, the more carnivorous the animal the poorer its ability to convert DHA and EPA from ALA.

The clinical impact of supplementation with various sources of O-3 FAs has not been clearly defined although more attention has been given to the fish oils in recent research. Nevertheless, various studies support that adding either fish oil and/or ALA to bird diets (multiple species) increases plasma levels of EPA, DHA, and (with ALA supplementation) linolenic acid and reduces arachidonic acid. These findings indicate that supplemental O-3 FAs result in incorporation into the body and in doing so exerts biologic effects. Specifically, fish oil supplementation (2% herring oil, O-3 FA source) has been shown to be relevant in Muscovy ducks [39]. Schiavone *et al.*'s work showed that supplementing Muscovy ducks with fish oil significantly altered breast muscle fatty acid composition by increasing the O-3 FA content and decreasing omega-6 fatty acid (O-6 FA) content [39].

Similarly, Liu *et al*. supplemented growing 17-week-old Shaoxing laying duck diets with one of four fat sources for 70 days total: 3 g/Kg fish oil (FO), 25 g/kg sunflower oil (SO), 30 g/kg palm oil or 20 g/kg beef tallow [30]. Serum triglycerides (FO group) and total cholesterol (FO and SO groups) were significantly decreased. Polyunsaturated fatty acids in the eggs and meat were

significantly higher with birds fed FO and SO. Meat and egg O-3 FA levels were significantly higher in the FO group [30]. These findings in ducks are similar to those shown in other animals supplemented with dietary omega-3 fatty acids.

Birds cannot manufacture linoleic acid (an O-6 FA) or ALA and must get these from their diet [40]. Depending on the species of waterfowl, ALA or EPA/DHA are likely the main sources of O-3 FAs in free-living birds. Based on research in other animals, herbivorous waterfowl (such as most geese) likely naturally consume more ALA (over DHA/EPA) in their diet as this would be the predominate source of O-3 FAs in plants. However, omnivorous and more carnivorous birds likely naturally consume a mix of O-3 FAs.

As most commercial bird foods are made from corn and soybean components, these diets are typically high in O-6 FA. Fish oils are highly unstable when manufactured in foods. Flax seeds provide a more stable source of O-3 in manufactured diets. Flax seed oil provides a more concentrated source (than flax seeds) of O-3 FA and can be added to the diet. Finally, fish oil (EPA and DHA) provides the most biologically potent form of O-3 FA and must be supplemented fresh due to its instability to heat, oxygen, and physical mixing. Based on research in multiple animals, fish oil supplementation is likely the best means to increase body EPA and DHA levels - even for herbivorous species that would naturally consume more ALA.

Bone health

While it may seem an unlikely connection, O-3 FAs contribute to several factors involving bone health. Studied 4-week-old quail fed identical diets for 7 months except for fat content (soybean oil [SBO], hydrogenated soybean oil [HSBO], chicken fat [CF] or menhaden fish oil [FO] all at 50 g/Kg of feed) had notable differences in bone parameters [41]. The ratio of O-6 FA:O-3 FA in the diets were as follows: SBO 12.55, HSBO 17.85, CF 18.47, and FO 0.66.

As O-3 FAs reduce the concentration of arachidonic acid and subsequent production of PGE_2 (the opposite is true with O-6 FA), bone formation is increased. PGE_2's long term goal is to stimulate bone resorption. As expected, quail fed FO (and HSBO) had markedly improved cortical bone thickness and density compared to the other groups. Also, quail fed FO had higher percentages of tibial ash, Ca, and P. Last, quail fed FO or HSBO had increased bone shear force compared to the other groups. All of the findings indicated bones from FO and HSBO groups were stronger. As a potential

negative, HSBO quail had more trans-fatty acids in studied tissues [41].

A similar study used 16-week-old chickens fed diets of varying O-6 FA:O-3 FA (ranging from 48.7:1 to 4.7:1) for 42 weeks. While the study did find increasing cortical bone thickness as the O-3 FA concentration increased (up to the highest O-3 FA level), other parameters such as bone strength and mineral content were similar between the different diets [42]. One of many differences is that this second study used a much lower O-3 FA content diet than in the quail study above. These findings support a role of supplemental O-3 FA in improving bone quality in young Galliformes and may similarly affect waterfowl species.

Brain development

O-3 FA, especially DHA, has been given special attention in terms of neurologic development. Studies in humans, rats, and dogs support the finding that when young are supplemented with O-3 FA, and specifically DHA, they perform better on a variety of intelligence and agility tests compared to age-matched peers given placebo. Precocial avian species, such as chickens, are known to have better developed brains and higher brain-DHA concentration than atricial species (such as swallows) at hatching [43]. These findings suggest that higher DHA brain content is correlated with a greater need for neuromuscular coordination and nerve synaptic connection in precocial species, whereas altricial species reserve DHA-brain accumulation for later as their development is slower.

Domestic duck (*Anas platyrhynchos*) and captive partridge (*Alectoris rufa*) chicks have been noted to have significantly lower brain-DHA compared to wild counterparts. The difference has been attributed to the low O-3 FA diet with the domestic species [40]. Studied 1-day-old partridge chicks showed decreased learning ability when their parents were fed low amounts of O-3 FA (fish oil) compared to chicks whose parents were fed higher amounts [44]. Although studies are limited, the findings seem to correlate well with those from other species – that O-3 FA appear to be important for neonatal brain development.

Cancer

It can easily be said that it is better to prevent cancer than work to treat cancer. The research on supplementing diets with O-3 FA and their effects on laboratory animals, dog, and human cancer prevention and treatment is extensive. In general, diets high in O-3 FA (usually through additional supplementation) are associated with a lower risk of a variety of cancers. For those

patients (humans, dogs, and some laboratory animals) with cancer, O-3 FA supplementation has been shown to reduce lipolysis and muscle degeneration ("cancer cachexia"), increase survival time and disease-free interval and improve overall quality of life (although the cancer is not "cured").

At least one study has evaluated the effects of O-3 FA supplementation in chickens with ovarian cancer. Two and a half-year-old white leghorn hens were fed a standard or 10% flaxseed-enriched diet for 1 year [45]. While the overall incidence of cancer was the same in both groups, the flaxseed-supplemented hens had fewer late stage tumors with ascites and metastasis. Additionally, the flaxseed-supplemented group maintained weight (as opposed to lost weight in the control group), had significantly lower overall mortality (from all natural causes of death), and better overall health. The authors noted that the study began after the hens had already ovulated approximately 500 times (the equivalent of a woman entering menopause) and the damage (cancer formation) may have already been done prior to starting the study. The authors are next studying the effects of O-3 FA supplementation starting in 22-week-old chicks over 4 years [45].

Kidney disease

O-3 FAs are frequently studied in mammal kidney disease models. The major renal benefits of O-3 FA supplementation appear to be centered on decreasing intra-renal inflammation, decreasing thrombosis, and improving overall renal blood flow and single nephron glomerular filtration rate. No such studies currently exist in avian models.

Nevertheless, the overwhelming literature does support the idea that O-3 FA supplementation can have renal benefits across species and should be a consideration with kidney health in waterfowl.

Obesity

Supplemental O-3 FAs have been shown to substantially affect fat deposition in many animals including birds. The effects of dietary fats and body fat were highlighted in a study of 3-week-old chickens fed identical diets for 5 weeks except for fat content (tallow, fish oil, or sunflower oil all at 80 g/Kg of feed) [46]. The fats represented saturated fat (tallow), O-3 FA (fish oil – FO), and O-6 FA (sunflower oil, linoleic acid – SO). FO chicks had significantly lower plasma triacylglycerol and total plasma cholesterol levels than the other groups. The SO group also had lower triacylglycerol levels compared to tallow, but not as significant as with the FO group. Abdominal fat pad mass was significantly lower in the

SO and FO groups (0.8% and 1.1% respectively) than those receiving tallow (2.7%). The SO and FO also had significantly increased breast muscle and subsequently breast muscle:abdominal fat than the tallow fed group. Similar findings have also been reported in humans: That FO can reduce overall body fat mass [46]. This study helps support the use of O-3 FA supplementation in birds that are overweight and has been used by the author for this purpose specifically in waterfowl.

One study involved feeding perilla seed oil (65% ALA, 14% linoleic acid, and 14% oleic acid [omega-9 fatty acid]) to 28-day-old laying Shaoxing laying ducks for a total of 50 days [47]. Compared to controls, the fatty acid-supplemented ducks had improved egg laying (without altering egg weight or feed to egg conversion ratio), altered lipid profiles (reduced serum cholesterol and triglycerides and elevated high density lipoprotein cholesterol) and down-regulation of lipogenic enzymes, and up-regulation of fatty acid catabolism enzymes in the liver [47]. All of the findings were considered positive and suggest that diets high in O-3 FA may improve hepatic fat metabolism and serum lipids in ducks as has been shown in numerous other studied animals.

Omega-3 fatty acid dosing

Specific dosing for O-3 FAs have not been established for waterfowl. A dietary O-6 FA:O-3 FA ranging from 1:5 to 15:1 has been proposed as desirable for dogs and cats with renal disease. This guideline has also been challenged and ratios of 1:1 have been proposed as ideal. Based on plasma conversion of lower O-6 FA ratios post-supplementation with O-3 FA in multiple species, it appears that at least 3–4 weeks is needed to reach "optimal" levels. Long-term supplementation (3–6 months or more) is likely appropriate if O-3 FAs are to be effectively used.

The author uses fish oil capsules (better stability than pumps or pour-on versions) to supplement waterfowl. Capsules can be fed whole or cut and squirted over fresh food daily. In general, the author supplements 300 mg (combined EPA/DHA) to small waterfowl (less than 9.0 kg) and 600 mg (combined EPA/DHA) to larger birds daily.

Vitamins

Unlike the Galliforme poultry literature, relatively few studies in ducks explore the role of micronutrients such as vitamins.

Niacin

Popular literature commonly mentions that ducks in particular have niacin (Vitamin B_3) needs above and

beyond those in more commonly kept poultry [1]. Holderread states that "young waterfowl require two or three times more niacin in their diet than chicks other than broilers." Classic signs of niacin deficiency in ducklings include bowed bones, stunted growth, and enlarged hocks. Holderread further notes that chick broiler feed has sufficient niacin for ducklings. Providing 100–150 mg niacin per gallon (3.75 L) drinking water to ducklings until 8–10 weeks old can "cure" niacin deficiency in affected birds. Alternatively two to three cups of brewers' yeast added to 10 # (4.5 kg) chicken feed will prevent niacin deficiency [1].

Wu *et al*. reported that day-old mule ducklings when fed for 3 weeks required 45 mg/kg niacin in the feed [48]. In addition, excess tryptophan compensated for niacin deficiency but the opposite was not true. Ducklings fed basal diets had poorer growth rates and bowed legs compared to those with higher levels of niacin. Maximum growth rate and absence of bowed legs was noted when ducks were fed rations containing 48 mg/kg niacin. While maximum body weight and feed efficiency was noted when 0–3-week-old ducklings were fed 48 mg/kg niacin in the feed, regression analysis predicted the minimum requirement of 45 mg/kg niacin [48].

Niacin is synthesized from tryptophan, which requires dietary pyridoxine, and much of the niacin in food stuffs is unavailable due to its form [48,49]. For example, only 30% of the niacin in corn is available to chicks. Wu consequently also found that optimal growth rate and feed efficiency in 0–3-week-old ducklings was obtained by feeding 0.23% tryptophan in the diet. Wu *et al*. concluded that niacin supplementation was needed when tryptophan levels were suboptimal but not when tryptophan was given in excess [48].

Serafin studied the dietary requirements of nicotinic acid (niacin), riboflavin (vitamin B_2), choline, and pantothenic acid (vitamin B_5) in Embden goslings [49]. Previous reports had shown than goslings and ducklings require 22–55 mg/kg feed of available niacin for optimal growth. After 2 and 3 week trials, the author determined the goslings required no more than 3.84 mg/kg and 31.2 mg/kg dietary riboflavin and nicotinic acid respectively for rapid growth and development. Dietary pantothenic acid requirement did not exceed 12.6 mg/kg and 1530 mg/kg of dietary choline was adequate to allow rapid growth and prevent perosis. Bowed legs were noted in diets with suboptimal levels of riboflavin. Choline-deficient birds developed perosis. However, no goslings developed bowed legs or perosis on nicotinic acid deficient (as low as 16.2 mg/kg

feed) diets. Deficiencies in all of the nutrients resulted in slow growth [49].

These studies highlight the complexity of the relationships between niacin, tryptophan, and naturally available niacin in the food and likely other nutrients. Niacin supplementation appears to be very safe. However, it should be noted that the limited research above shows that bowed legs and stunting in young waterfowl is not limited to niacin deficiency. If such developmental abnormalities are found, several nutrient deficiencies should be considered.

Vitamin C

Vitamin C performs many roles in the body, primarily as an antioxidant, and has been heavily studied in many animals including poultry. Specific to Jin-ding female layers ducklings, Wang *et al*. found that supplementing vitamin C at 400 mg/kg feed resulted in maximum weight gain; reduced malondialdehyde and increased superoxide dismutase and glutathione peroxidase in serum and liver; and increased serum IgA, IgG and IgM concentrations [50]. While vitamin C was supplemented at 150, 300, 400, 800, and 1400 mg/kg feed, the 400 mg amount appeared optimal. Ducklings were 1 day old at the start of the study and supplemented until 28 days of age. The base diet was formulated as per NRC (1994) guidelines and contained no vitamin C. While it is reported that adult poultry (assumption extended to ducks) are able to synthesize vitamin C, requirements are higher during stress [50]. This provocative study demonstrates how a single added nutrient can affect growth, oxidative status, and immune system function.

Enzymes

Enzyme supplements have been shown to be beneficial diet additives as a means to degrade non-starch polysaccharides (NSP) and increase energy and nitrogen retention in chickens (with multi-enzyme blends more effective than single enzymes) and other animals [51,52]. Limited work of this type has been performed in waterfowl.

Young poultry, including ducks, seem to be sensitive to the anti-nutritional effects of NSP [53]. These carbohydrates are not digested by endogenous enzymes and increase the viscosity of the gastrointestinal contents. This in turn may decrease excretion of endogenous enzymes and bile acids and have other effects that ultimately reduce digestibility of nutrients. Conventional poultry diets containing corn and/or wheat are low in NSP. However, when foods such as oats, rye, triticale, and barley replace corn and wheat, the concentration of NSP can significantly increase [53]. The theoretical

application of enzyme supplements would be to degrade dietary NSP and improve the bird's ability to digest and utilize nutrients.

Adeola *et al.* evaluated an enzyme supplement containing 7500 units protease, 44 units cellulase, and side activities of pentosanase, amylase and α-galactosidase all per gram (0 or 1 g/kg feed additive) [51]. The supplement was added to the diet of 8 or 9-week-old white Pekin drakes and feed and excreta evaluated for nitrogen, dry matter, amino acids, and energy contents. The enzyme supplementation had no effect on nitrogen, dry matter, or energy utilization but did improve limited amino acid digestibility (particularly methionine) in the ducks fed starter or grower diets [51].

Hong *et al.* added enzyme supplements (4000 units amylase, 12,000 units protease, and 1600 units xylanase all per gram) at 0.375 and 0.5 g/kg feed to White Pekin duckling diets for 42 days (starting at 3 days of age) [54]. Compared to controls, enzyme-supplemented ducklings showed a 6% to 8% gain in body weight and had improved nitrogen and amino acid retention. This all correlates to improved feed efficiency with enzyme supplementation under the conditions of the study [54].

Based on limited research, it appears that enzyme supplementation can be used to improve amino acid digestibility and weight gain when added to starter and grower diets in (at least) white Pekin ducklings. It should be emphasized that the efficacy of enzyme supplementation is based on the type and dose of enzymes used, the diet fed, and likely the animals. However, commercially available enzyme supplements are generally considered safe.

Grit

While naturally consumed by wild waterfowl, the need for grit supplementation depends on how the birds are being kept and what they are fed. Birds kept on lakes, ponds, waterways, and large open spaces will likely naturally accumulate grit in their diet. Birds that consume fibrous foods (natural forage, grasses, grains, etc.) are more likely to need grit than those eating processed commercial pellets (which are easily digestible). Some authors, such as Holderread, do recommend adding variably sized granite grit to duckling and adult duck diets [1]. However, the "need" for supplemental grit in captive waterfowl diets has not been critically evaluated.

Grit supplementation in waterfowl has been studied for non-nutrition related purposes. Grit may be used as a drug delivery system and has been specifically used to successfully provide the wildlife contraceptive nicarbazin to mallards [55]. Grit supplementation next to waterways may reduce lead shot ingestion in wild birds. It is believed that the shot particles are specifically selected by waterfowl as "grit" and not mistaken as a food item [56].

Grit type can be variably digested in birds. Mateo and Guitart found the half-life of ingested calcareous grit was 1.4 days in mallard gizzards. This compares to siliceous grit, which has a half-life of 3.1 days in mallard gizzards [56]. The implication is that calcareous grit would need to be replaced more frequently than siliceous versions to maintain functional levels in the ventriculus.

Natural zeolite and vermiculite

As the production poultry industry works to move away from drugs to improve health and animal growth, newer natural products are being used. Zeolite is "crystalline, hydrated alumino-silicate of alkali and alkaline earth cations, able to absorb water and exchange nitrogen molecules" and has experimentally been shown to reduce toxicity associated with litter ammonia and aflatoxins in chicks [57]. Vermiculite is a "clay mineral, magnesium alumino-silicate which has a high cation exchanging capacity" [57].

Khambualai *et al.* found that by supplementing a mixture of zeolite (70%), vermiculite (10%), and extracted plant enzymes (pineapple and papaya 20%) to 14-day-old farmed Aigamo ducks for 9 weeks, the experimental group birds gained significant weight over controls [57]. The experimental additive produced significant body weight gain at 0.1g, 0.5g, and 1.0g/kg feed. Based on electron microscopy observations, the authors hypothesized that the experimental mixture resulted in intestinal villi hypertrophy and activated cell proliferation, which subsequently increased nutrition absorption. The authors concluded that the experimental mixture could be added at a rate of 1 g/kg feed as a natural means to improve weight gain in (Agaimo) production ducks [57].

Ducks

In general, ducks are omnivorous and captive birds that can eat commercial pellets, live worms and other insects, fresh leafy vegetables, and some fruit. Obesity is common in waterfowl, especially ducks. Domestic ducks may be predisposed genetically to fat storage, resulting in hepatic lipidosis, coelomic fat accumulation and elevated plasma glucose, triglycerides, and cholesterol. This is especially evident when birds are overfed diets high in corn and corn flour (simple carbohydrates) [58]. Limiting high energy foods and total food quantity, increasing exercise, and providing either natural or artificial foraging opportunities help to reduce the

incidence of obesity. As such, the author typically offers about 50% of the diet as commercial pellets and the remainder as chopped dark leafy greens, free access to forage outside, worms as treats, and supplemental fish oil.

Geese

Geese are predominantly herbivorous and feed on young tender grasses, aquatic plants, and some roots, rhizomes and cultivated grains. The author typically offers about 25–50% of the diet as commercial pellets and the remainder as chopped dark leafy greens, free access to forage outside (especially grass) and supplemental fish oil.

Swans

Swans naturally primarily eat vegetation supplemented with animal matter. The author typically offers about 50% of the diet as commercial pellets and the remainder as chopped dark leafy greens, (ideally) free access to forage on water plants, and supplemental fish oil.

General comments on feeding waterfowl

Beyer *et al.* estimated the average digestibility of natural swan diets at 50% [14]. The result is a large amount of fecal matter. This is common with all waterfowl. However, the fecal matter increases in volume as pellets are decreased and fibrous foods increased.

The amount of food needed depends on multiple factors that have already been discussed. The author uses BCS and PMS (which can be taught to owners) and regular weighing (if practical) to assess caloric needs. If the bird's weight is deemed too heavy, then pellets are generally reduced until an acceptable PMS and BCS are achieved. Food quantity and caloric density generally need to be increased with inclement weather, increased activity, reproductive seasons, and illness.

Female birds often have increased nutrient (especially total calories, protein, and calcium) demands when reproductively active. Commercial waterfowl breeding diets are available and are substituted for maintenance diets during reproductive seasons. Emptied, cleaned, and dried crushed (chicken) egg shells offer a good source of calcium and are readily eaten by many waterfowl. Crushed oyster shell also works but is less commonly accepted by some birds. Special "breeder" supplements are readily available and popular with many waterfowl owners. However, no peer reviewed research has been published evaluating these supplements.

Young waterfowl are precocial and grow rapidly. They will generally eat most food items offered but are at risk of developmental nutritional disease with unbalanced diets. The author recommends feeding commercial waterfowl starter diets as the main food from 0 to 21 days of age. Commercial waterfowl grower diets are then fed starting at day 22 until the bird is 90% grown (which may be several weeks to months depending on the species). Once young waterfowl have reached most of the adult size, they can be switched over to a lower protein maintenance diet during this slower last growth phase. As a note, avoid feeding layer rations (which typically have a significantly higher calcium level) to developing waterfowl. Doing so may result in boney abnormalities, organ failure, and death.

When with their mother, young waterfowl will start foraging on a large variety of food items (in addition to pellets) within a few days of hatching. When single or otherwise without an adult role model, the author recommends supervised foraging at 1–2 weeks old to supplement their regular diet. As the young bird matures, foraging progressively makes up more and more of the diet.

Avoid medicated feed if possible. Medicated feeds for poultry are commonly used to treat parasites and are usually not necessary for waterfowl. Coccidiosis and other parasitic diseases can occasionally be seen in waterfowl and should be carefully evaluated before considering feed-based treatment.

Waterfowl pest control

Resident populations of waterfowl (especially mallards, duck hybrids, domestic geese, and Canada geese) may be found in many urban environments (see Figure 4.25). Whether availability of food, shelter, bodies of water and/or appropriate breeding grounds are present, these birds can sometimes overbreed and overstay their welcome. The end result is often significant fecal accumulation and the potential for disease transmission to other animals (especially commercial poultry operations) and even people [55].

Several methods of pest waterfowl control have been used and will only be briefly discussed here. These methods include capturing and relocating birds, euthanasia, and use of chemical contraceptives (such as nicarbazin) [55]. Additionally, as pest waterfowl nests are clearly identified, the eggs may be oiled shortly after the clutch is laid. This simple procedure requires one to paint the egg with safe consumable oils such as canola, corn, or olive oil. To ensure the eggs don't mature and hatch, first shake the egg vigorously. Then apply the oil to the addled egg and repeat 2 more times over 1 week.

Figure 4.25 Domestic and wild waterfowl may overrun local ponds and urban environments and become pests. These wild Canada geese (*Branta canadensis*) are walking on to a golf course.

Each method of waterfowl control has its pros and cons. In the United States, waterfowl are protected species and unauthorized population control may be illegal. Prior to considering any waterfowl control measures, especially euthanasia or contraceptive use, work with local and state officials.

Enrichment for captive waterfowl

Enrichment basics
In addition to plenty of room to walk around and a water source to swim in, waterfowl thrive with various forms of enrichment. Waterfowl are generally social and benefit when two or more of the same species are present. Overcrowding and lack of resources (food, water, etc.) can lead to aggression and should always be considered prior to adding new birds to a flock. Additionally, closed aviary principles apply to waterfowl as with all other birds. Foraging is another means to improve enrichment. Geese will generally graze on new grass. However, ducks tend to be more sedentary when allowed to simply feed from a bowl. Small amounts of pellets can either be placed in multiple feeding stations or foraging devices (such as "duck safe" foraging boxes) to increase the bird's effort and energy expenditure to find food. Also, leafy greens can be floated on water and worms can be placed in loose dirt or shallow water. If parasite transmission is a concern, then worms can be

placed within foraging devices to prevent parasitic ova or larva contamination.

Enrichment is defined as simply adding something to an animal's environment to improve its life and allow for species typical behaviors. The real challenge with enrichment is finding biological relevance that is practical. As its name suggests, biologically relevant enrichment is "effective" in that the animal actually uses the introduced enrichment to better its captive life. This is done in part by controlling stressors in its environment and allowing for species typical behaviors. Enrichment is only as successful as it is practical for caretakers to introduce and maintain.

What may be enriching for a mallard may not apply to a Canada goose. Because of physical, behavioral, developmental, natural history, and other differences, items that are "enriching" can vary significantly between, and even within, species. While some enrichment may be readily accepted by most of a species, such as swimming areas for ducks, others may not be regularly used, necessitating a trial and error approach.

Sometimes recognition for the need for enrichment can be challenging, especially if the waterfowl appear physically normal. Feather damaging is obvious to see. Cannibalism and feather "pecking" are serious problems recognized in captive production Muscovy ducks (*Cairina moschata*) and have resulted in the highly criticized practice of beak and claw trimming as a preventative measure [59]. Crowding and lack of access to adequate

water troughs are just two of the proposed causes for these destructive behaviors.

Per the author's observations, one of the most common abnormalities with waterfowl is physical inactivity. This may lead to obesity, arthritis, and other complications or excessive reproductive activities (leading to coelomic reproductive disease in females and masturbation, phallus disorders and aggression in males) and all are common with pet ducks. Lack of appropriate "comfort movements" as described by McKinney may be another means to assess whether or not captive waterfowl are performing normal behaviors [60]. Johnsgard gives detailed accounts of normal waterfowl behavior that may also be used to help recognize abnormal activities of captive birds [61]. Once explained, clients may recognize abnormal behaviors with their pets and be more willing to make appropriate changes (see Figures 4.26 a,b,c,d,e).

The benefits of environmental enrichment in numerous captive animal species are well documented. However, similar research is rarely conducted in captive waterfowl.

Some biologically relevant enrichment can have unintended negative consequences. Introduced items may incite fear, especially in those animals poorly socialized to experience new items in the environment. Others may result in frustration, such as when enrichment holds prized items (food) that cannot be obtained because of the animal's physical limitations. Highly valued items, such as high protein food treats for ducks, may result in aggression with group housed animals (while the same enrichment can be very beneficial to a singly housed bird). Others may result in trauma or danger to the animal such as beak lesions caused by improperly made foraging devices. As with any item introduced into an animal's environment, complications should be considered and clients prepared accordingly.

Social enrichment

Social interaction is the most effective and dynamic form of enrichment for the majority of captive animals. Due to the complexities of social enrichment, only generalizations will be made here.

Recent attention has been given to the environment and conditions surrounding the development of young animals of many species as well as the long term consequences when they are raised under unnatural or stressful situations. In general, waterfowl are precocial and rapidly recognize movement, follow it, and become socially attached (imprinted) during a short period of time after hatching (see Figure 4.27). Usually, the moving object is the bird's mother. However, many waterfowl are raised by people and their pets (dogs) for various reasons. Waterfowl recognize and associate with future mates in part based on their exposure to conspecifics when young.

Possibly the biggest concern with abnormal imprinting is that the bird may not be able to form pair bonds with its own species or attempt to, and successfully, mate/bond with a different species altogether. This is seen when ducks are raised in relatively crowded exhibits with multiple species. When they become adults, ducks of different species tend to fairly readily breed, resulting in interesting offspring. However, this does not appear to be a problem for redhead ducks (*Aythya americana*) that often lay their eggs in the nests of canvasback ducks (*Aythya valisineria*) and are raised by them. Canvasback-reared redheads later appropriately pair with their own species.

Light enrichment

As waterfowl are primarily diurnal, they would naturally be expected to receive partial or full sunlight. While most zoos and aviaries have enclosures that allow their birds to receive unfiltered sunlight, some pet waterfowl (especially ducks) are kept in areas that receive little or no sun.

The author recommends regular exposure to partial and full sunlight for captive waterfowl. The reason is subjective and based on poor general (radiographic and surgical) bone density noted in captive waterfowl. One potential cause is low natural sunlight exposure (and subsequent inadequate vitamin D production). Also, birds kept outdoors are more likely to engage in physical activity, which helps build and maintain muscle strength and mass, and bone density. The author recommends that owners allow waterfowl to go outside. Be sure that there are plenty of hide spots or shelters should the bird choose not to "sunbathe" (see Figure 4.28). Ideally, waterfowl should be left outside during the day but can be brought inside at night and during inclement weather.

Substrate enrichment and enclosure design

Proper substrate is important for healthy feet and leg joints in waterfowl. It is also important to have an enclosure that gives waterfowl appropriate space security and adequate water.

In general, waterfowl can usually be found either naturally on water, on soft (usually grassy) ground, or perched on a rounded log or branch. Bumblefoot is commonly seen in waterfowl under one or more of the following conditions; obesity, hard substrate, lack of access to water to swim in, malnutrition and

(a)

(b)

(c)

(d)

(e)

Figure 4.26 Evaluation of the presence of normal behaviors and comfort movements can be used to help assess the general well-being of captive waterfowl. Preening, as with this green-winged teal (*Anas carolinensis*) (a), and bathing, shown here with this Mandarin duck (*Aix galericulata*) (b), are examples of normal comfort movements. (c) Waterfowl should also feel secure enough to rest comfortably without disturbance as shown with this lesser Magellan goose and her goslings (*Chloephaga picta picta*). (d, e) This trumpeter swan (*Cygnus buccinator*) has enough room and water depth to dabble and completely submerge its head.

underlying disease, arthritis (resulting in placing extra weight on one leg), and foot trauma (fire ant bites, burns, etc.) Access to clean water seems obvious for waterfowl. However, research has only recently shown how important various types of water enrichments (shallow water troughs, showers, etc.) are for production ducks [59]. Even without prior experience, ducks show clear preferences for open water and use the

Figure 4.27 Hatchling waterfowl readily imprint. For proper social development, it is best to let hatchings imprint with conspecifics. As is normal, this black swan cygnet (*Cygnus atratus*) and pen are together.

Figure 4.28 Natural unfiltered sunlight is extremely important for captive waterfowl. However protection from predators and the elements should be considered. This shaded pond housing a pair of White Pekin ducks (*Anas platyrhynchos*) provides relief from heat at an private aviary in a hot desert environment.

water for drinking, foraging and feeding, locomotion, preening, and general exploration [62].

Proper space and enclosure set up can help reduce predation, aggression, sleeping/inactivity, and, possibly, inappropriate interspecies breeding. Space requirements can be highly variable and can significantly affect social dynamics depending on how many birds and what species are present in a given enclosure. Aviaries with bird aggression and predation issues may need fewer animals, more hide spots, predator control measures, visual barriers, and vertical rest spots or a combination to relieve tensions. In studied mallards, common teals (*Anas crecca*) and tufted ducks (*Aythya fuligula*), increased predation risk resulted in increased sleeping and decreased preening and foraging activities [63].

Toy enrichment
Most waterfowl do not readily play with toys. However, some ducks seem to like balls or other objects that they can push around.

Foraging enrichment
Just as with most wild animals, a significant portion of a duck's waking time is spent foraging. While it has been reported that Brent geese (*Branta bernicla*) forage an average of 3 hours a day, data on the specific amount of time most waterfowl spend foraging is lacking [64]. Ducks may not use a foraging device if the food is too difficult to obtain. Popular foraging feeders in group housing may result in stress and aggression if no other acceptable enrichment or food options are available.

Foraging devices and setups may also be used to encourage exercise and limit total food consumption. If total caloric needs are known, they can be measured and spread between multiple feeding stations or foraging devices. This is most easily accomplished with single birds or small flocks (2-4 birds) and is especially useful as a means of weight loss. Large flocks also benefit from foraging devices but often need supplemental and more traditional bowl feeding sites to reduce aggression and stress.

There are several simple types of foraging enrichment for waterfowl. For geese and some dabblers (mallards, for example) a grassy field offers the opportunity to graze (geese) or find insects (dabblers). Some dabblers will also pick at insects climbing trees and in and around logs. Of course, all ducks need water but divers live on and in water. Specific foraging devices are listed below.

Waterfowl foraging devices
Dive-to feeder
Dive-to feeders are used to feed diving waterfowl separately from dabblers and grazers. The feeding device is enclosed on top, floating on the water and close to shore. Access to the birds is gained by diving under the floating feeder, which is open. Caretakers can access the food through a hatch in the top of the cage.

Dive-to feeders can also be fitted with elevated feeding stations on the outside of the structure. The feeding stations are placed high enough off the water to only allow large dabblers to feed. This can be an effective means to feed swans and some geese while preventing ducks (especially invasive species) from eating the food (see Figures 4.29 a,b,c).

Deer feeder
Waterfowl "deer feeders" are based on old style deer feeders. They are inexpensive and relatively easy to

(a)

(b)

(c)

Figure 4.29 Dive-to feeders offer an excellent means to feed diving ducks separately from dabblers. (a) The floating rectangular box is next to shore allowing easy access to the internal feeder (IF) from a top hatch. The IF is suspended just above the water and can only be accessed by diving under the box (which has an open bottom). (b) An external feeder (EF) is elevated and placed on one side with a wire cage over the top. This allows tall birds such as swans to feed and prevents smaller birds from accessing the food from below or if on top of the box. (c) Dabblers generally do not swim under the box to gain access to the IF.

make and maintain. Deer feeders are meant to be hung and are used by tall ground-feeding birds (ratites, larger gallinaceous birds, etc.) However, waterfowl (of any size) that walk on land can also learn to use deer feeders. This is a good method to separately feed waterfowl that share time on land (such as dabbling ducks) from those that spend most of their time on water.

Use a standard 5 gallon bucket (larger and smaller versions can be made too) with a lid and handle (optional). A hole slightly larger than 2–3 times the size of the food is drilled at the bottom of the bucket. For example, if the food is 1.0 cm in diameter, make the hole 2.5 cm. Next, find about 0.5 m of dowel rod that is the same diameter as the food. Use a 1.0 cm diameter dowel in this example. On one end of the dowel attach a washer that is significantly larger than the opening on the bottom of the bucket. Use a washer that is at least 5 cm in this example. The washer is either left flat or bent upwards (away from the dowel end) on both sides. Next place the dowel through the hole at the bottom of the bucket such that the washer end is inside the bucket and the dowel is left dangling below. The bucket is filled with food, covered with the lid and suspended via the handle, rope or both. Alternatively, the bottom of the bucket can be lined with a plastic cone such that all of the food is funneled to the central hole.

The bird then must learn to hit and move the end of the dowel to create an opening large enough for food to fall past the washer and dowel, through the bottom of the bucket and on the ground.

Alternatively, ornaments can be placed on the end of the dowel such as a bell or shinny object. Attach all items well enough that the bird cannot ingest the object (see Figures 4.30 a,b,c,d,e).

Foraging logs

Foraging logs are good for birds that like to look into cavities for food. Many species of birds can learn to use foraging logs.

Use natural untreated logs or tree branches that are soft enough to drill holes with your existing equipment. The size of the logs can vary from a few inches (even using existing perches) to a few feet in diameter depending on the resources available. Logs can be oriented horizontally or vertically. These can further be rested on the ground or suspended (for perching waterfowl) (see Figure 4.31).

Small (1–2 inches) to large (3–5 inches) holes can be drilled in the wood. Metal bowls can be placed within large holes and the logs oriented horizontally. Further, large holes can be covered (bark, leaves, etc.) to make it more challenging for the birds.

(a)

(b)

(c)

(d)

(e)

Figure 4.30 Deer feeders' serve as excellent foraging devices to feed waterfowl terrestrially. (a) A standard 5 gallon bucket is most commonly used. (b) A hole about 2–3 times the size of the food is drilled at the bottom of the bucket. A dowel rod with washer hangs suspended from the bottom of the bucket. In this example, a plastic lining was added to create a funnel leading to the central hole. (c) The finished product is filled with some food and then hung at the appropriate height such that the birds can easily grab the suspended dowel. This deer feeder was designed for southern ground hornbills (*Bucorvus leadbeateri*) and has a dowel too large for waterfowl. (d) A deer feeder being used in a mixed waterfowl species collection. Notice the lettuce tied to the end of the dowel being used to target the birds. (e) An American widgeon hen (*Anas americana*) grabs the lettuce, pulling the dowel which results in a small amount of food dropping on to the ground.

Figure 4.31 Foraging logs are simple and effective foraging devices for waterfowl. This horizontal log (partially covered by a rock in the foreground) has several large drilled holes each about 12–15 cm deep and 10 cm wide. Logs can be oriented vertically or horizontally and adjusted to the physical abilities of the bird.

Figure 4.32 Foraging boxes and troughs are additional terrestrial feeding devices that work well with waterfowl. A series of 4 foraging boxes are attached to a log within an enclosure. Note the cut outs on each side of the box. These prevent injury to the bird if the lid of the box falls on the bird's head. While the boxes shown here are heavy duty, lighter construction materials work well for waterfowl.

Note: If a large enough hole is drilled (on primarily vertically oriented logs), wild birds may try to enlarge the site to create a nest cavity.

Foraging feed troughs and boxes

These devices are good for birds with poor "foraging dexterity" (especially those that do not handle food with their feet but can simply push things with their head and beak). Skilled birds can also use these devices.

Place feed troughs along the sides of the pen or along banks, trees, or other structures within the enclosure. The troughs can vary in size but for ducks and geese should generally be about 2–4 inches deep, 4–6 inches wide, and 5–7 inches long. Foraging feed boxes are simply shorter versions of the troughs. Adjust size (bigger or smaller) based on the bird. Start with the troughs on the ground and leave open. Gradually raise the troughs off the ground by attaching them to the sides of the pens. Have 6–8 feed troughs scattered at different levels around the sides of (and/or within) the pen. Use dry foods in the troughs (pellets, grains, some vegetables, and some fruit - just as long as it can easily be cleaned).

Once the birds are accustomed to eating the food from the troughs, attach a flip top cover that the birds will have to flip up to find the food within. The flip tops can be wood, metal, or durable plastic and are attached via a hinge to the back side of the trough (see Figure 4.32). Long troughs (2–5 feet long) can have several flip tops along the length of the trough. So, one section of the trough may have a flip top up while the rest of the tops are down. The goal is that the birds don't know what is in each section (which should vary on a daily basis) and will have to open the flip top to check for food. Put small amounts of high energy food items in these feeding troughs. Troughs can be situated on the ground, suspended on the sides of cages, or attached to trees. All suspended troughs or boxes should have an adjacent branch to give the bird good solid footing when opening the boxes.

Note: Some birds risk getting their head stuck between the flip top and the box. Make sure to create a cut out area on the central section of the top edges of the box. If the flip top falls down and the bird panics, the cut out depressions will allow the bird to pull its head out unharmed.

Identification

Several methods of identification are available for waterfowl. The most common are leg bands, patagial bands, neck collars, and/or microchips. Leg bands are best placed on birds a few days old. The band should be large enough to slip over the foot and rest over the tarsometatarus. Bands need to be large enough to grow into without risk of constriction and small enough to prevent the foot from slipping through within a few days. Metal crimp, plastic, and spiral wire leg bands can be placed on adult birds.

Production birds will sometimes have notches or perforations in their webbing (using a poultry "toe punch") but this is not recommended for pet birds

[1]. Pet birds are most commonly fitted with leg bands and/or are microchipped.

Wild waterfowl may be banded by governmental agencies. The birds are banded to help track animal movement, disease monitoring, and more. The most common bands used on wild waterfowl are leg, patagial, neck, nasal markers, web tags (for babies too young to place leg bands), and occasionally radio-telemetry devices. Each band generally contains information pertinent to the banding agency and may include contact information. Alternatively, one may call 1-800-327-BAND to report a bird band and get specifics on the animal. Hunters most commonly report bird band information back to the banding agency. This information is vital to understanding health and disease of wild waterfowl populations.

Pinioning and wing trims

Pinioning renders waterfowl flightless by removing the wing distal to the alula. Waterfowl are best pinioned between 2–4 days of age by simply amputating the proximal base of the major and minor metacarpal on one wing leaving the alula intact. Typically, sterile clippers and no anesthesia are used. If the bird is sexed, pinion the right wing for males and left for females [65]. Adult birds can also be pinioned. However, the procedure requires full anesthesia, pain management, and so on.

Alternatively wings may be trimmed in adult birds. The outer 5–10 primary feathers of both wings are trimmed just distal to the coverts. Avoid cutting new incoming or "blood" feathers. While wild waterfowl commonly molt twice a year, domestic birds may have more sporadic molts. Either way, the wing trim is temporary and feather regrowth should be periodically monitored.

Managing excessive egg production

Domestic ducks and geese are well-known for their high egg production. Some breeds of domestic duck including Campbell, Harlequin, Magpie, Appleyard, and, the most commonly kept pet, Pekin can produce 200–300 eggs per year [1]. In contrast, wild mallards lay approximately 5–16 eggs per clutch with one to three clutches per year depending on environmental conditions and the bird's health. With near year-round egg production, chronic nutritional and coelomic disease is common often requiring medical and sometimes

surgical treatment (see Chapter 16). The same problems are occasionally encountered in domestic geese and rarely in swans and non-domestic geese and ducks.

Factors contributing to excessive egg production include genetics, presence of mates (including stimulation by the owner), lengthened daylight (natural or artificial), adequate to excessive calories, restricted access to exercise and natural behaviors such as foraging, gonadotropin releasing hormone (GnRH) agonists, and possibly more. Understanding and identifying these factors helps to set a plan to reduce chronic egg laying and its negative effects on a bird's body.

Prior to laying, waterfowl often undergo physical changes. White plumaged ducks tend to lose color intensity in the beak, which fades to pale yellow, while the bills of colored ducks tend to darken during breeding season. Receptive ducks assume a prone position with tail up. For testing purposes only, this behavior can be induced in some by simply placing a hand on the bird's back. Also, the ventral coelom of many waterfowl noticeably distends as the reproductive tract enlarges in preparation for egg laying. The pubic bones on some ducks will also spread just prior to laying [1].

Associated risk factors and suggestions on how to reduce excessive egg laying in waterfowl are listed below. One or a combination of the described management changes may be needed to address egg over production. As a side note, some of the same management changes can be used to help reduce recurring phallus prolapses in drakes.

Genetics

Genetics is the least controllable factor. Many domestic duck and goose breeds have been inbred for decades, centuries, and in some cases millenniums. Combined with a propensity for development of fatty liver disease when fed simple carbohydrate-rich diets, persistent egg laying has been selected to meet the needs of the foie gras, meat, and egg industries. Owners should understand that while such high egg production is unnatural compared to wild counterparts, it is very common in domestic ducks and geese. The management changes proposed below are essentially working to counteract the bird's genetic drive to lay.

Stimulation from other birds and owners

Ducks and geese seem to be stimulated by other reproductively active birds and, especially ducks, by inappropriate human interaction. While placing the bird in isolation with no nest often will stop egg production, the author recommends other measures. If a flock is present, place the over productive hen or

goose with non-reproductively active birds (including non-waterfowl species) or other non-predatory animals. If the bird is paired and over production is a problem, consider splitting the pair and placing both birds with other animals as above. If the bird is single and is being stimulated by the owner (frequent petting, holding, close contact), have the owner discontinue the behavior and focus more on positive reinforcement training (clicker, trick, etc.) as the means of interaction (see Figures 4.33 a,b,c,d). All of these techniques can be stressful to the bird (and sometimes owner) and may need to be modified based on the response(s).

Light exposure

Manipulating light (day length) in poultry is a well-known means to modify egg production. The same appears to be true in production waterfowl. Holderread notes ducks are more sensitive to light changes than chickens and a reduction in as little as 15–30 minutes of light per day can result in diminished rate of lay [1]. Typically, laying will start towards the end of winter and early spring as day length increases.

A simple strategy is to reduce light exposure (including indoor lights) to mimic the shortened winter days. This strategy often requires that the bird be brought inside in a dark room. Using public weather charts, determine the sunrise and sunset times and total day length (in terms of hours of light) one month prior to the (approximate) day or week when the reproductive behavior was first noticed or suspected. Bring the bird into a lighted room with no external light visible starting at the calculated sunset time. Start by keeping lights on to match outdoor lighting. Over a period of 1–2 weeks, gradually decrease the artificial light time until no additional light is in the room. Each morning, take the bird out at the calculated
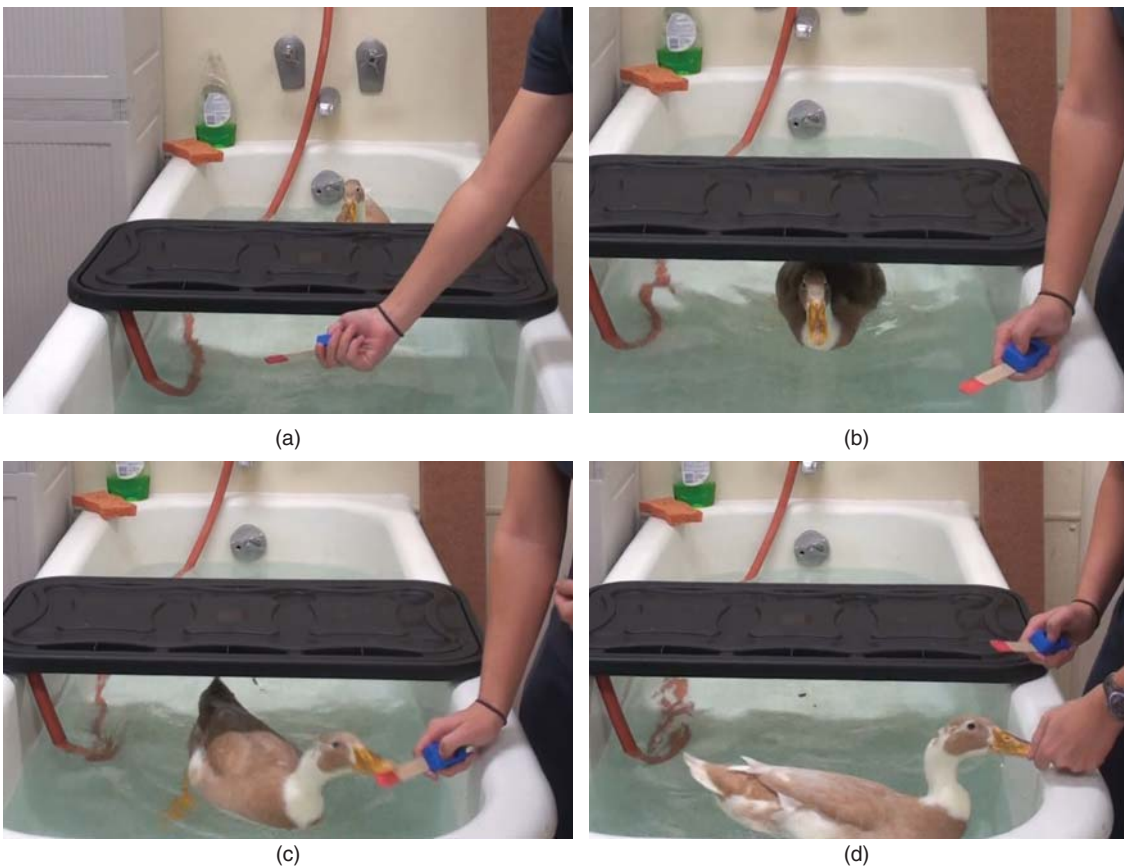
Figure 4.33 A mixed breed domestic duck (*Anas platyrynchos*) is being used to demonstrate clicker training. (a) The owner holds a clicker attached to a tongue depressor with a target. While being prompted with clicks, the duck follows the target under an obstacle (b), then touches the target (c) and finally receives a food reward (d).

sunrise time. This same pattern can be created with automatic lights for indoor birds. It is best to keep the bird with a companion(s).

The above strategy works best to prevent egg laying for female waterfowl with a known history of reproductive problems. If needed, birds can be kept in reduced light throughout the entire breeding season (which may be 6–9 months for some birds). However, once the physical and behavioral signs of reproductive activity have ceased, the bird can be returned to a full light schedule and simply monitored. If needed, the light reducing procedure above can be repeated until day length is shortening significantly in the fall.

Dietary management

With excess calories comes the energy to produce eggs. The first step is to ensure the diet is appropriate and correct dietary inadequacies as needed. Next, reduce the amount of simple carbohydrates in the diet as this energy source will be the primary driver for hepatic lipogenesis and lipoprotein production of which some is used for the eggs. Holderread notes that egg production is lowest for ducks fed whole or cracked grains, with free access to pasture and pond and normal day length compared to the same breeds fed pellets with no pond time and either natural or increased day length [1].

Significant calorie restriction, especially when combined with diminished light, can result in cessation of reproductive activity and a "forced" molt. This often involves restricting all food (but free choice water) for 3–4 days. Food is then reintroduced on the fifth day.

Intermittent fasting is described in wild waterfowl and is often needed during migration [66]. In common with some other migrating species, hepatic steatosis is a main mechanism of energy storage in waterfowl and easily occurs with excessive carbohydrate ingestion (hence the development of the foie gras industry) [27]. However, long term steatosis is not expected under natural conditions as fasting (during migration) reverses this physiologic change. Unfortunately, captive waterfowl often experience long term energy excess.

Black ducks (*Anas rubripes*) have been studied after 4-day fasts (no food) and have lost weight and shown delayed egg production but otherwise underwent no significant adverse health effects [66]. Even young (10 and 20-day-old) Pekin ducklings have been studied with 5 days of calorie restriction (just enough to maintain relatively stable body mass). The study showed that ducklings adapted to calorie restriction by decreasing resting and peak metabolic rates and returned to normal growth when free feeding resumed [67]. Collectively these studies highlight a natural adaptive response in

ducks that is common in other migratory animals and those with intermittent food supplies.

If such a fast is considered, the duck should be otherwise healthy and the owner instructed on a clear plan as to providing plenty of water, possible light restriction (outlined above) and monitoring for any problems.

Exercise and natural behaviors (foraging)

Providing access to exercise and foraging opportunities serves many purposes. First, it fulfills the need to perform natural behaviors. Doing so may also reduce or prevent abnormal behaviors such as feather damaging, inactivity, and more. Restricting access to perform normal behavior is also considered poor welfare and goes beyond the scope of this chapter. Second, exercise is needed to utilize excess fat and maintain good muscle strength and tone. The obvious end results are better weight control and reduced side-effects of a sedentary lifestyle. Additionally, strengthening legs may help stabilize and decrease the pain of arthritic joints. Last, by giving birds something to do beyond eating and minimal activity the drive for reproductive behaviors is sometimes decreased. Ideas for foraging activities are listed above. Providing access to outdoor space and water to swim in helps increase activity.

GnRH agonists

Drugs such as leuprolide acetate (Lupron Depot, TAP Pharmaceuticals, Inc, Deerfield, IL, USA) and deslorelin (Suprelorin®, Peptech Animal Health/Virbac, Australia) have recently become popular among veterinary and lay discussions. While these products have not been critically studied in waterfowl, they are being used clinically. GnRH agonists have been discussed in more detail in Chapter 16.

In the author's experience, leuprolide acetate and deslorelin induce a very short term cessation in egg laying in domestic ducks, with both reducing egg production for as little as a few days to weeks in some animals. Without other concurrent management changes as listed above, currently available GnRH agonists alone are a poor means of managing excessive egg production in over-productive ducks. This is especially true when instituted after laying has begun.

It is not clear if the dosages being used are inappropriate, the drugs themselves have little biologic effect, or if other intrinsic mechanisms easily override the effects of currently available GnRH agonists in ducks. Regardless, the author discourages use of these drugs as a sole means to manage egg overproduction in ducks and other waterfowl.

Surgery

Unless a medical emergency is present, surgery should be one of the last resorts to control excessive egg laying in waterfowl. Surgery usually consists of salpingo-hysterectomy and coelomic cleanup with or without partial ovariectomy. See Chapter 16: Soft Tissue Surgery for more details of surgical correction of diseases of the female reproductive tract. The author generally reserves surgery for oviductal obstructions, ectopic eggs, advanced yolk coelomitis, neoplasia, or other non-medically manageable reproductive tract diseases. Surgery should be performed in addition to some of the other management changes listed above.

Pre-emptive salpingohystectomy in young at-risk domestic ducks and geese has not been studied and reported at the time of writing.

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Backyard Poultry Nutrition

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Introduction

Sufficient supply of nutrients is essential to drive the long-term health and well-being of any poultry flock. Nutritional insufficiencies/deficiencies are much more commonly observed in backyard poultry than in commercial poultry production, and this occurs for numerous reasons. Foremost among these are lack of nutritional knowledge of the backyard poultry owner and their dogmatic approach, but the limited specificity of information regarding nutritional needs for non-commercial poultry strains/breeds is also important. Not surprisingly, the genetic "gap" between commercial poultry strains and that of backyard breeds has greatly increased. Current commercial livestock and poultry strains are more efficient in utilizing nutrients and the commercial feeds are better formulated to meet the requirements of the rapidly growing animal [1]. For example, nitrogen (N) and phosphorus (P) excretion per unit of live weight were 55% and 69% less respectively from a 1991 commercial broiler strain versus a 1957 commercial broiler strain fed the same diet.

Common nutritional issues in backyard poultry

While nutritional issues in any flock can be numerous, the most common issues observed (and further described herein) in backyard flocks are either a result of:

a. Insufficient water quality or amount
b. prolonged storage and degradation of vitamin efficacy
c. dilution of dietary nutrients with a "cheaper" and less nutrient rich ingredient(s)
d. feeding of the wrong life-stage diet

Insufficient water quality or amount

Water comprises 85% of young birds, 67–70% of adult birds, and 65% of the egg. Thus, water is the nutrient that is required in greatest quantity by the bird. However, quantity and quality of water is often forgotten about and overlooked. Poultry require 1.5–3.5 parts water for every 1 part of feed consumed (up to 5–6 times for waterfowl). Several factors can influence water consumption, including (but not limited to):

A. Salts
B. Dietary fiber content
C. Ambient temperature
D. Medications
E. Disease State

Generally, monitoring of water consumption can be an initial gauge of flock health, as deviations from "normal" consumption patterns often occur with initial onset of disease. Drinking water guidelines, namely the maximum can be found in Table 5.1.

Vitamin and mineral deficiencies

Typical vitamin and mineral deficiencies observed in poultry are elaborated further in Tables 5.2 and 5.3. When vitamin deficiencies have been observed, they typically have occurred as a result of either not including a vitamin premix into the diet, or utilizing a vitamin premix that is well beyond its shelf-life (and thus efficacy). In either case, fat-soluble vitamin deficiencies will present themselves prior to water-soluble vitamin deficiencies, particularly vitamin D_3. Thus, skeletal abnormalities that would be observed would include beading of the ribs, scoliosis, soft and pliable bones, keel, and beak, and rickets resulting from lack of hydroxyapatite crystallization at the growth plate in long bones such as the tibia, femur, or humerus.

Table 5.1 Suggested maximums for drinking water for poultry

Contaminant	Average concentration	Maximum acceptable concentration	Remarks
Total bacteria		<100 cfu/mL	
Total Coliforms		<50 cfu/mL	
Total hardness	60–180 ppm	110 ppm	<60 is unusually soft; >180 is very hard
pH	6.8–7.5	6.8–8.0	<6. is undesirable; <6.3 may degrade performance
Arsenic		0.2 ppm	
Calcium	60 mg/L	- - -	- - -
Cl⁻		250 ppm	
Copper	0.002 mg/L	250 mg/L	Even 14 mg/L may be detrimental if sodium level is higher than 50 mg/L
Fluorine		2.0 ppm	
Iron		500 ppm	
Lead		0.02 mg/L	Higher levels are toxic
Magnesium	14 mg/L	125 mg/L	Higher levels may have laxative effect
Mercury		0.01 ppm	
Nitrates		50 ppm	
Nitrites		10 ppm	
Sodium	32 mg/L	50 mg/L	>50 mg/L may affect performance if sulfate or chloride are high
SO$_4$		250 ppm	
Zinc		1.5 mg/L	Higher conc. are toxic

Source: Adapted in part from Carter and Sneed [2].

Backyard producers may try to produce their own feed in some cases with the thought that they could do it better and more cheaply than the commercial equivalent. One of the biggest limitations for them to do so is the availability of a vitamin and micro-mineral premix. There are some readily available that could be purchased, likely from a toll-mill that mixes poultry rations, but usually in 50 pound quantities. For most vitamin/mineral premixes, the range of inclusion typically would be between 3 and 10 pounds of vitamin/mineral premix per ton of feed. Usually, a laying hen would eat no more than $1/4$ pound of feed per day. Thus as an example, if a backyard producer had 25 hens, it would take 320 days to utilize one ton of feed. If the inclusion of vitamin/mineral premix were 5 pounds per ton, it would take $8 3/4$ years for them to get through a 50 pound bag of premix. With a maximum shelf-life of 3–6 months, it is easy to see why the birds in a smaller producer's flock may have vitamin deficiencies.

Proper and thorough mixing of all ingredients is essential to ensure the bird is able to ingest proper proportions of nutrients on a daily basis. For example, a newly hatched chick will eat enough feed in its first meal to fit on the surface of a US quarter. Thus, it is easy to see that adequate mixing (particularly of the vitamin/mineral premix) may be of particular difficulty for the small flock owner.

When considering minerals, poultry have specific requirements for calcium, phosphorus, sodium, chloride, potassium, sulfur, magnesium, iron, copper, zinc, manganese, iodine, and selenium. The three from this list that are not routinely supplemented within a mineral premix are potassium, sulfur, and magnesium as it is presumed that the feed ingredients in the rest of the diet would contain sufficient amounts to meet the birds' needs for these.

Laying hens may also experience periods of producing thin-shelled eggs for numerous reasons. In many cases, the hen will sacrifice bone resorption to readily supply the calcium needed in the egg if it is not sufficiently available in the blood during shell formation. Long-term consequences of cortical bone resorption can affect skeletal integrity, with results including keel angulation, long bone fractures, beading of ribs, and rib collapse (Figure 5.1). Supplementing additional calcium or phosphorus to the diet when the hen reaches these

Table 5.2 Vitamin requirements, deficiencies, and sources for poultry

Vitamins	Major deficiency signs (in addition to growth reduction)	National Research Council (1994) requirements (per kg feed) [3]	Good sources
Vitamin A	Drowsiness, incoordination, emaciation, ataxia, reduced vision, urate deposits in soft tissues, reduced hatchability, more susceptible to disease	C-1500 I.U. H-4000 I.U. P-4000 I.U.	Fish oils, synthetics and carotene from yellow corn and alfalfa meal
Vitamin D$_3$ (cholecalciferol)	Rickets, thin egg shells, poor reproduction	C-200 I.C.U. H-500 I.C.U. P-900 I.C.U.	Fish oils, some animal products and synthetics
Vitamin K	Hemorrhagic disease (poor blood clotting)	C-0.5 mg H-0.5 mg P-1.0 mg	Synthetics and low levels in plants
Vitamin E	Encephalomalacia, exudative diathesis and poor reproduction	C-10 mg H-5 mg P-12 mg	α-Tocopherols in plants and synthetics
Thiamine (Vitamin B$_1$)	Polyneuritis and anorexia	C-1.8 mg H-0.8 mg P-2 mg	Many natural feedstuffs
Riboflavin (Vitamin B$_2$)	Curled-toe paralysis, enlargement of sciatic nerve, poor protein utilization	C-3.6 mg H-2.2 mg P-3.6 mg	Pure substance, milk products, alfalfa meal, Brewer's yeast
Pyridoxine (Vitamin B$_6$)	Reduced nitrogen retention, dermatitis, convulsions, anemia	C-3.5 mg H-2.5 mg P - 4.5 mg	Pyridoxal & pryidoxamine in animal products Pyridoxine – whole grains Up to 40% loss w/processing & storage
Pantothenic Acid	Dermatitis on top of feet and corners of mouth, poor hatchability	C-10 mg H-2+ mg P-11 mg	Many feedstuffs, yeasts and milk products, synthetics
Nicotinic Acid (Niacin)	Pellagra-like syndrome, scaly dermatitis, hock disorders in poults	C-27 mg H-10 mg P-70 mg	Wheat products, synthetics
Choline	Perosis-like condition, fatty liver	C-1300 mg H-500 mg P-1900 mg	Soybean meal, wheat products, synthetics
Biotin	Dermatitis on bottom of feet, around vent and eyes and beak	C-150 µg H-150 µg P-200 µg	Liver meals, yeast and alfalfa meal, synthetics
Vitamin B$_{12}$ (cobalamine)	Poor livability, poor hatchability, perosis-like condition (slipped tendon)	C-9 µg H-3 µg P-3 µg	Meat and other animal products, fermentation products, synthetics
Folic Acid	Anemia, poor feathering, hock disorders in poults	C-550 µg H-350 µg P-1000 µg	Widely distributed in feedstuffs but may be limited in availability

C, Growing Chicks; H, Laying Hens; P, Growing Turkey Poults
ICU, International Chick Units (which for vitamin D$_3$ are equal to IU (International Units)

Table 5.3 Mineral requirements and deficiency symptoms in poultry

Mineral	Major deficiency signs	National Research Council recommended minimum dietary concentrations	
Calcium	Rickets, decreased activity and sensitivity, tetany, thin egg shells, poor embryonic development	Chicks (C) Hens (H) Poults (P)	0.8% 3.40% 0.55−1.2%
Phosphorus, available	Rickets, depraved appetite, weakness, thin egg shells	C H P	0.4% 0.32% 0.28−0.60%
Sodium	Softening of bones, gonadal inactivity, corneal keratinization, decreased plasma volume, decreased cardiac output	C H P	0.15% 0.15% 0.12−0.17%
Potassium	Overall muscular weakness; intestines, heart and respiratory muscles	C H P	0.4% 0.15 0.4−0.7%
Chlorine	Dehydration, hemoconcentration, low blood chloride, tetany-like syndrome	C, H, & P	0.15%
Magnesium	Anorexia, low blood Mg, disorientation, hyperirritability, tetany, reduced hatchability	C H P	600 ppm 500 ppm 600 ppm
Iron	Anemia (microcytichypochromic), inadequate respiration	C H P	80 ppm 50 ppm 50−80 ppm
Copper	Anemia, bone disorders, depigmentation of hair and feathers, cardiovascular defects	C H P	8 ppm 6 ppm 6−8 ppm
Zinc	Poor feather development, skeletal malformations, poor wound healing, impaired function of reproductive organs	C H P	40 ppm 50 ppm 40−75 ppm
Manganese	Skeletal abnormalities, decreased reproductive performance	C H P	60 ppm 30 ppm 60 ppm
Iodine	Goiter and consequences of thyroid hormone inadequacy	C H P	0.35 ppm 0.30 ppm 0.40 ppm
Selenium	Muscular dystrophy (white muscle disease), degeneration of myocardium, liver necrosis, pancreatic fibrosis	C H P	0.15 ppm 0.1 ppm 0.2 ppm

extremes will not alleviate long-term osteoporosis that has already occurred. Any additional hydroxyapatite crystallization that occurs while the hen is laying eggs and circulating estrogen content is high will occur in the medullary portion of the bone. Rather, if a molt were induced through daylight length reduction to no more than 8 hours per day, a period of cortical bone remodeling could occur. Once adequate cortical bone regeneration has occurred, daylight length could be increased and another reproductive cycle could occur (albeit for a shortened duration).

Consequences of diet dilution

Small-scale producers may also try to stretch their feed budget by purchasing a commercial diet, but diluting it directly or indirectly with other grains (often referred to as scratch grains). General symptoms that usually occur when nutrient density of feed is too low are slow growth, slow or cessation of egg production, and plausibly feather loss if the amino acid needs of the bird were not adequate. Thus, while foraging behavior of hens may be desired, provision of scratch grains or diet dilution may compromise optimal performance.

Figure 5.1 Angulation of the keel due to inadequate mineralization.

Consequences of feeding of the wrong life-stage diet (hen diet to a non-laying bird)

One of the common problems experienced with mixed age flocks is avian urolithiasis and/or gout. Urolithiasis involves the blockage of a ureter with urates. Gout occurs with renal damage and consequent high blood uric acid levels causing deposits of urates in the kidneys (renal gout), in the joints (articular gout), or on the serosal surfaces of the liver, kidney, heart, air sacs, and mesentery (visceral gout; Figure 5.2). Urolithiasis can occur as a result of numerous factors, but the most common cause is demand for egg shell production along with prior renal damage. Predisposing factors could include infectious bronchitis, feeding of excessive levels of sodium bicarbonate (producing alkaline urine and an ideal medium for kidney stone formation), an episode of severe dehydration, or more commonly a high calcium (i.e., laying hen) diet to an immature bird. Even in commercial production, transition from birds not laying (i.e., the pullet phase) to laying is difficult to manage. Poultry species are photo-sensitive, and with increasing daylight lengths from 12 to 16 hours will induce egg production if the bird is old enough and proper body conditioning occurs. Without housing where lighting is controlled it can be difficult to match the calcium needs of the bird coming into lay with proper, and not excessive, amounts of dietary calcium. Thus, the high (3.5–6%) calcium content of a laying hen diet can adversely affect the kidney in the proportion of the flock not in lay.

Changes to nutrient needs with life stages

Examples of changing nutrient and energy needs for egg-laying hens and broiler-type meat chicken strains are listed in Tables 5.4 and 5.5 respectively. Specific requirements for nutrient and energy can be found in the last National Research Council publication on the nutrient requirements for poultry, published in 1994. While there are recommended minimums for meeting birds' needs, it is important to realize that these may not be the concentrations needed for optimal or maximal productivity. Additionally, different strains of birds will also vary their intake as a result of factors such as environmental changes, disease state, and energy density of the diet. General nutrient needs for changing life phases are exemplified in Tables 5.4 (egg-laying strains) and 5.5 (broiler strains).

Diet formulation

Feed ingredients and feed additives

While the nutrient needs of the bird at different life stages are fairly well defined (more so for commercial poultry strains), there are numerous ingredients and combinations that can form an effective diet. Several ingredients, however, may contain anti-nutritional factors, toxic factors, and/or nutrient imbalances that should be limited as a proportion of the diet. Considerations of maximal inclusions of certain feed ingredients are illustrated in Table 5.6.

All feedstuffs have the potential to be contaminated with mycotoxins in the field or during storage, and thus should always be monitored. For the backyard owner, it is also important to maintain feeder hygiene. Thus, to prevent mycotoxin ingestion, make sure producers allow birds to clean up their feeders at least once a week, and never allow a bird to consume a visually moldy feed/feedstuff. Particular mycotoxins of concern and prevalence for poultry include aflatoxin, ochratoxin, T-2, and deoxynivalenol. All of these are of particular concern for young birds as they are more highly susceptible. Some of these toxins have readily

Table 5.4 Examples of changing nutrient needs for egg-laying chicken strains

	Pullet starter*	Pullet grower	Pre-lay	Hen	Rooster
Crude protein%	20.00	18.62	18.40	18.30	11.54
Metabolizable energy, kcal/kg	3010	3005	2920	2890	3140
Calcium%	1.00	1.00	2.50	4.20	0.75
Phosphorus%	0.71	0.65	0.59	0.53	0.51
Available phosphorus%	0.45	0.40	0.35	0.30	0.30
Methionine%	0.51	0.45	0.45	0.48	0.24
Methionine+Cystine%	0.85	0.77	0.77	0.79	0.47
Lysine%	1.16	1.00	1.00	1.01	0.51
Threonine%	0.77	0.72	0.71	0.71	0.44
Sodium%	0.183	0.183	0.183	0.183	0.183

*The approximate ages (depending on breed and season) to which the starter, grower, pre-lay, and laying hen diet would be fed would be 0–6, 6–16, 16–18 (time to which daylight length is increasing), 18 weeks of age (from 25–50% egg production for duration of lay) respectively.

Table 5.5 Examples of changing nutrient and energy needs for broiler-meat type chicken strains

	Broiler starter*	Broiler grower	Broiler finisher
Crude protein%	22.50	18.25	17.50
Metabolizable energy, kcal/kg	3050	3175	3225
Calcium%	0.9	0.9	0.85
Available phosphorus%	0.42	0.4	0.375
Methionine%	0.45	0.41	0.38
Methionine+Cystine%	0.88	0.83	0.75
Lysine%	1.15	1.1	1
Sodium%	0.18	0.15	0.15

*Approximate ages to which these diets would be for starter, grower, and finisher, would be 0–3, 3–6, and 6 until finishing respectively.

identifiable lesions while others do not, namely (in order of prevalence):

a. Aflatoxin – hepatic necrosis caused by free radical production, lipid peroxidation, and inhibition of RNA and protein synthesis. The liver will have a yellow/brown jaundice appearance. Aflatoxin is the only mycotoxin that can be readily adsorbed by certain feed additives (hydrated sodium/calcium alumninosilicates, bentonite, zeolites, and clinoptilotie)

b. T-2 toxin – causes contact dermatitis and can cause oral and dermal lesions within the mouth's palate, tongue, and/or corners of the mouth

c. Ochratoxin – binds to plasma proteins and causes renal damage. Ochratoxin (of all the mycotoxins) causes the most body weight loss. In chronic cases, birds will have urate deposits in their joints (articular gout) and abdominal cavity (visceral gout). (Figure 5.2) Polyuria is a common observation in excreta

d. Deoxynivalenol – does not have readily observable lesions, but rather causes damage through inhibition of protein synthesis, thus making tissues with high protein turnover more susceptible (including the small intestine, bone marrow, lymph, spleen, and thymus). Of particular concern is damage to both the innate and acquired immune system

Cereal grains

Cereal grains are the primary energy source of the diet. They include ingredients such as corn, milo (grain sorghum), wheat, barley, oats, or triticale. Fiber content of the diet should be limited to 10% to 15% at a maximum. Higher amounts of fiber can be used, but litter wetness may become an issue, and lower energy diets will have an impact on performance. Several

Table 5.6 Suggested ingredient maximums for feed ingredients for poultry for specific life stages

Ingredient	Young birds <3 wk (maximum, %)	>3 wk (maximum, %)	Pullets (maximum, %)	Laying hens (maximum, %)
Corn bran	30	30	30	30
Barley	10	20	20	20
Rice bran	15	20	20	15
Wheat bran	10	15	15	15
Peanut meal	8	10	15	10
Fish meal	5	5	8	3
Blood meal	2	2	2	2
Palm oil	2	5	12	15

Figure 5.2 Visceral gout (urolithiasis) in a laying hen.

cereal grains also contain higher amounts of non-starch polysaccharides (NSP) within the soluble fiber fraction. For example, barley is high in β-glucans (a non-starch polysaccharide), which can cause pasting of the beak and vent. In commercial poultry diets, a β-glucanase enzyme would be supplemented to improve energy utilization. Higher content of NSP in a diet also increases intestinal digesta viscosity and mucin production. In many cases this increased viscosity can predispose the bird to *Clostridium perfringens* proliferation, and if prior intestinal damage has occurred (e.g., coccidial infection), *C. perfringens* can gain a foothold to cause a condition known as necrotic enteritis. Additional issues with cereals include that of rice bran which can be high in trypsin inhibitors.

Quality of feed ingredients should always be a concern. Particularly for grain ingredients, one needs to consider weed seed contamination, as certain weeds can be high in thiaminase activity. Grain by-products are also readily available, and in the United States, include products of the dry and wet corn milling industries (gluten feed and hominy), from the brewing and distilling industries (wet or dry distiller's grains plus solubles, and wheat byproducts (wheat bran, wheat middlings, screenings). Notably, many of these byproducts have mainly utilized the starch portion of the whole-grain, and thus the fiber content may be higher as well as the protein fraction more concentrated. While the protein content may be higher, typically it is of similar profile to that of the cereal grain itself and will need a complement of amino acids from a legume and or animal byproduct meal to meet the amino acid needs of the bird.

Protein sources

These are the primary amino acid source of the diet. Plant protein sources typically come from leguminous plant seeds, and are higher in protein than cereal grains. The plant proteins' amino acid profiles complement the profile of cereal grains to help meet the amino acid needs of the bird. Often, however, these needs cannot fully be met through the combination of the two types of ingredients and supplemental amino acids must be added to the diet through addition of DL-methionine (or methyl hydroxyl analog), L-lysine-HCl, and L-threonine. Typical protein meals from plant sources include soybean meal (without hulls containing 48% protein), canola meal (low glucosinalate varieties of rapeseed meal), corn gluten meal, peanut meal, peas, safflower meal, sunflower meal, sesame meal, and/or cottonseed meal.

Full-fat soybeans can also be fed (in addition to soybean meal), and provide additional energy versus the meal. However, in both cases, raw soybeans contain a trypsin inhibitor that can be partially inactivated by heat. Proper heating is essential, as over-heating will cause a Maillard reaction, thus reducing lysine digestibility.

Peanut meal is low in methionine, lysine, and threonine, high in tannins, and the trypsin inhibitors can only be partially inactivated with heat. Cottonseed meal contains gossypol, an alkaloid, which if present in high enough levels in hens' diets can cause a discoloration to egg yolks, as well as cyclic fatty acids that may cause pink egg whites. Additional supplemental iron can be fed to partially alleviate these toxicities.

Animal by-products used as protein sources include meat and bone meal, poultry by-product meal, hydrolyzed feathermeal, fishmeal, and blood meal. Sources can be somewhat variable because of variable amounts of collagen, feathers, and hair; all of which have relatively low amino acid digestibility. Feathermeal, if processed properly by autoclaving to 145°C for 30 minutes, can have improved digestibility, but still has a relatively poor amino acid balance for poultry. Fish meal should be limited to only 2–3% in laying hens to prevent "fishy" tasting eggs. Fishmeal can also contain a thiaminase enzyme if not properly processed. It can also contain a biogenic amine (gizzerosine) known to cause gizzard (ventriculus) erosion and a condition called "black vomit." Blood meal can be a good source of lysine, but is deficient in isoleucine and can contain a high amount of sodium, which limits the amount of dietary inclusion. Proper processing is key for blood meal, as it has a greatly reduced amino acid availability if overheated (blackish in appearance).

Fats and oils

These can supply a higher caloric density to the diet as well as reduce the dustiness of the diet. Typical sources include that of animal fats, vegetable oils, animal/vegetable oil blends, and restaurant grease. In poultry, during their first two weeks of life, digestibility of saturated fats is much less than that at older ages and is thus accounted for in dietary formulation. Fats and oils need to be monitored routinely for quality control through the amount of free fatty acids, moisture content, unsaponifiable material, insoluble matter, and fatty acid stability. While unsaturated fats are more easily prone to oxidation, all fats are subject to it. Oxidation is catalyzed by any combination of trace metals, oxidative enzymes, light and/or heat. Consequences of feeding oxidized fats to birds include degradation of fat-soluble vitamins, increase in cellular membrane damage and friability, increase in intestinal enterocyte turnover rates, and reduction in xanthophyll content (and skin "bleaching"). Much of this damage occurs as a result of susceptibility to intracellular, extracellular, and membrane damage with reduced levels of antioxidants, especially vitamin E and glutathione peroxidase. Thus,

often synthetic antioxidants such as ethyoxyquin, butylated hydroxytolulene, or butylated hydroxyanisole are included with the fat/oil source to suppress lipid oxidation.

Mineral sources

Various mined sources are utilized, primarily to provide necessary macro- and micro-minerals to the diet. Calcium, phosphorus, and electrolytes are macro-minerals that are usually provided separate from a mineral premix. Laying hens in particular can have a calcium-specific appetite, and thus many backyard poultry producers will provide a certain amount of oyster-shell or limestone for the birds to consume at will. A larger particle size of either of these two feedstuffs is also important to increase the amount of retention time in the gizzard. Ideally, two-thirds of calcium supplementation (in the mixed diet or otherwise) would come from a larger particle limestone or oystershell between 0.5 and 1.0 mm. This becomes important as the egg shell is being deposited onto the shell for 15–16 hours of the 24 that it takes to make an egg after ovulation of the follicle into the oviduct. Much of this occurs during the night when the bird is not eating. Thus, a slower release of calcium from the gizzard (ventriculus) helps supply some of the calcium need of the bird and can reduce the long-term resorption of medullary and cortical bone.

Phosphorus is typically supplied from dicalcium phosphate (18% phosphorus), mono- and di-calcium phosphate blends (21% phosphorus), and through the least bioavailable source, defluorinated rock phosphate (16% phosphorus).

Electrolytes supplemented into the diet include sodium chloride (salt), sodium bicarbonate, and potassium chloride. Micro-mineral sources in the mineral premix are supplemented either as sulfates, oxides, or as chelated forms with amino acids or proteins/peptides. Additional copper sulfate may be included in the ration (up to 1 pound per ton) to aid in prevention of crop molds and as an antimicrobial.

Other feed additives

Removal of sub-therapeutic antibiotics from poultry diets in Europe and recent pressure to reduce or remove these compounds in other parts of the world has amplified interest in maintaining the specific functions that growth promoting antibiotics elicit: Improving intestinal health, improving nutrient utilization, and reducing endogenous nutrient loss resulting, in part, from innate immune responses.

Table 5.7 Example of the nutrient minimums and maximum listings on a typical feed tag

Crude protein (CP)	min 26%
Lysine	min 1.5%
Methionine	min 0.5%
Crude fat	min 6.0%
Crude fiber	max 4.0%
Calcium (Ca)	min 1.1%
Calcium	max 1.5%
Phosphorus (P)	min 0.8%
Salt	min 0.4%
Salt	max 0.5%

However, the ascribed "antibiotic replacements" utilized as feed additives have never been able to elicit the full range of physiological, microbiological, and immunological responses to those of sub-therapeutic antibiotics. Thus, poultry nutritionists can be hesitant to incorporate these categories of feed additives resulting, in part, from a) unfamiliarity, b) over-selling of plausible effects, c) documented physiological and microbiological effects *in vivo*, and d) documentation of persistence from the feed and within the intestinal tract. Where AGPs have documented effects, they have generally shown more narrow biological effects than those of sub-therapeutic antibiotics, including:

a. Organic acids (e.g., fumeric and propionic acids) – antimicrobial against Gram negative bacteria
b. Plant extracts (e.g., essential oils from oregano, thyme, cinnaminaldehyde) – varied physiological functions, including: antimicrobial, altered intestinal mucin production, reduction in intestinal "turnover"
c. Probiotics – specific pathogen(s) exclusion, immunological modulation, improved nutrient use, antimicrobial action through pH modification and bacteriocin production.

Additional feed additives can include antibiotics, coccidiostats, arsenicals, mold inhibitors, mycotoxin binders, antioxidants, pigmenting agents, and pellet binders.

Table 5.8 Examples of diet formulation for egg-laying strains of chickens

Ingredient	Pullet chick starter (%)	Pullet grower (%)	Laying hen (%)	Rooster (%)
Corn	66.8	66.8	54.35	85.93
Soybean meal (48% protein)	28.46	28.46	29.54	10.96
Soy oil	0.8	0.8	3.91	- - -
Sodium chloride (salt)	0.41	0.41	0.41	0.41
DL Methionine	0.15	0.15	0.19	0.03
Limestone	1.8	1.8	10.42	1.42
Monocalcium phosphate	1.23	1.23	0.83	0.9
Vitamin/Mineral premix	0.35	0.35	0.35	0.35

Table 5.9 Examples of diet formulation for broiler/meat strains of chickens

Ingredient	Broiler starter (%)	Broiler grower (%)	Broiler finisher (%)
Corn	57.66	63.76	66.9
Soybean meal (48% protein)	35.27	29.68	26.3
Soy oil	3	3	3.52
Sodium chloride (salt)	0.48	0.46	0.48
DL Methionine	0.24	0.21	0.12
Lysine, HCl	0.11	0.1	0.02
L-Threonine	0.06	0.04	- - -
Limestone	1.41	1.38	1.49
Monocalcium phosphate	1.42	1.02	0.82
Vitamin/Mineral premix	0.35	0.35	0.35

Interpreting a feed tag

Commercial feed that can be purchased will not have an exhaustive list of nutrient composition, as was indicated in Tables 5.4 or 5.5. Rather, it will likely contain a list of feedstuffs along with guaranteed minimums and maximums as illustrated in Table 5.7. Notably, the two most "limiting" amino acids for poultry in corn/soybean meal diets are methionine and lysine, and they are therefore listed. As mentioned previously, a multitude of feedstuff combinations can be made to meet nutrient needs at various life stages. Example rations comprised primarily of corn and soybean meal are given in Tables 5.8 (egg-type chicken strains) and 5.9 (broiler/meat chicken strains).

Summary

Nutrition is imperative to successful growth, reproductive performance, and health of the backyard flock. The fundamentals outlined in this chapter touch on the most common nutritional issues experienced by the small flock owner. More in-depth information is available in the following resources.

Further resources

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2 Carter, T.A. and Sneed, R.E. (1987) Drinking Water Quality for Poultry. PS&T Guide No. 42, Extension Poultry Science, North Carolina State University, Raleigh, NC.

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CHAPTER 6

Parasitic Diseases

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Introduction

Clinical parasitic disease in backyard poultry in general is less of an issue compared to commercially raised poultry due to the lower density of birds. When parasite-associated morbidity and mortality occurs in backyard poultry it is often a result of poor husbandry and nutrition, overcrowding, mixing of avian species, or mixing of multiple age groups. This chapter contains common clinically significant parasites of backyard poultry, particularly birds in the order Galliformes, and thus is not an exhaustive list of avian parasites. Confirmatory testing for numerous parasites may require the submission of samples to a trained parasitologist. It is important to contact the respective laboratories prior to collection and shipment of samples to ensure proper protocols are followed to maximize parasite identification (Table 6.1).

Central nervous system

Toxoplasmosis
Clinical history
Toxoplasmosis is, in theory, infectious for all granivorous, insectivorous, and carnivorous birds. Infections are reported infrequently, which is perhaps due to lack of surveillance.

Causative agent
Toxoplasma gondii is a coccidia in which felines are the definitive host.

Clinical signs and lesions
Clinical signs include incoordination, listlessness, seizures, and convulsions. Gross lesions are variable and can range from no lesions to pneumonia, encephalitis, and splenomegaly.

Transmission route
Infection occurs by ingestion of oocysts in the environment or by ingestion of tissue cysts in muscle or other organs of intermediate hosts.

Diagnostic tests
Diagnosis of infected birds can be performed using sera in the modified agglutination test (MAT). Post-mortem diagnostics include histopath examination and polymerase chain reaction (PCR) on affected tissues.

Differential diagnosis
Baylisascaris infection, avian vacuolar myelinopathy, heavy metal and pesticide toxicosis, West Nile or equine encephalitis viruses, trauma, duck plague, *Leukocytozoon* infection, avian malaria, and botulism.

Prevention and control
Limiting cat access to area near the poultry is the most effective prevention. Sulfadiazine and pyrimethamine as well as diclazuril have been effective at treating toxoplasmosis in various avian species [1,2].

Zoonotic potential
Humans can be infected by ingestion of oocysts shed by domestic or wild cats or ingestion of tissue cysts in undercooked food.

Table 6.1 List of laboratories that are known to perform parasitology tests on poultry samples

Institution	Investigator	Laboratory address	Laboratory email	Laboratory phone number	Laboratory website
University of Tennessee	Rick Gerhold	University of Tennessee, CVM, 2407 River Dr., A233 Knoxville, TN 37996-4543	rgerhold@utk.edu	865-974-5645	http://www.vet.utk .edu/diagnostic/ parasitology/
Virginia Tech University	David Lindsay	VA-MD CVM, 1410 Prices Fork Rd VA Tech Blacksburg, VA 24061	lindsayd@vt.edu	504-231-6302	http://www. vetmed.vt.edu/ org/dbsp/faculty/ lindsay.asp
Mississippi State University	Linda Pote Sue Ann Hubbard Kelli Jones	Mississippi State University, CVM, Box 9825, Mississippi State, MS 39762 Poultry Research and Diagnostic Lab. MSU/CVM, 3137 Hwy. 468 W., Pearl MS 39208	lpote@cvm.msstate.edu hubbard@mvrdl.msstate.edu kjones@mvrdl.msstate.edu	662-325-1154 601-932-6771	
Arkansas	Tom Yazwinski	Department of Animal Science, AFLS B110D, University of Arkansas, Fayetteville, AR 72701	yazwinsk@uark.edu	479-575-4398	
University of Georgia	Lorraine Fuller	Poultry Science Dept., 152 Poultry Science Bld. University of Georgia, Athens GA 30602-2772	alfuller@uga.edu	706-542-1367	
USDA, ARS	Eric Hoberg	US National Parasites Collection; Animal Parasitic Diseases, BARC-1180, 10300 Baltimore Ave., room 1180, Beltsville, MD 20705-2350	Eric.hoberg@ars.usda.gov	301-504-8588	

Balisascariasis
Name of disease
Baylisascariasis, raccoon roundworm infection, visceral larval migrans, and neural larval migrans.

Clinical history
Baylisascaris is infectious for all birds. Infections are reported infrequently; however, outbreaks have been reported in captive collections.

Causative agent
Baylisascaris procyonis is a nematode parasite of raccoons. The geographical distribution of *B. procyonis* has been increasing and the parasite has been detected in numerous locations in the United States. Animals are infected by ingesting larvated eggs from the environment.

Clinical signs and lesions
Visceral and neural larval migrans of the larvae can result in numerous CNS clinical signs similar to

Toxoplasma. Post-mortem diagnostics include histopath examination and PCR on affected tissues.

Transmission route
Raccoons and domestic dogs can serve as definitive hosts for the parasite. Aberrant hosts are infected by ingesting larvated nematode eggs from the environment.

Diagnostic tests
Post-mortem diagnostics include histopath examination and PCR on affected tissues.

Differential diagnosis
Toxoplasmosis, avian vacuolar myelinopathy, heavy metal and pesticide toxicosis, West Nile or equine encephalitis viruses, trauma, duck plague, *Leukocytozoon* infection, avian malaria, and botulism.

Prevention and control
Limiting raccoon access to the yard and making the areas unattractive to raccoons, including removing excess pet, livestock, and poultry feed as the most effective prevention. Lids need to be secured on feed containers to ensure that raccoons or other wild animals cannot gain access to the feed [3].

Zoonotic potential
Humans can be infected by ingestion of larvated eggs, which can lead to aberrant migration.

Eyes and associated structures

Oxyspiruriasis
Name of disease
Oxyspiruriasis, eye worm.

Clinical history
Oxyspirura infection has been reported infrequently in back yard poultry. Nematodes in the genus *Oxyspirura* have been found in numerous galliforms and other avian families.

Causative agent
Oxyspirura are approximately 15 mm long but can range from 8–22 mm and have a rounded anterior and pointed posterior.

Clinical signs and lesions
Infected birds can have swollen conjunctiva and birds are often seen scratching their eyes. If left untreated, the globe may be destroyed due to chronic inflammation.

Transmission route
Birds are infected by ingesting infected cockroaches.

Diagnostic tests
Diagnostics are performed by observing the nematodes in the eye or identification of nematode eggs in feces. Eggs are approximately 55–60 μm long and 45 μm wide and are embryonated in freshly defecated feces.

Differential diagnosis
Trauma, foreign body.

Prevention and control
Prevention can occur by minimizing ingestion of arthropods. Ivermectin has shown efficacy in treatment of eyeworms in galliforms [4].

Oral cavity and respiratory diseases

Trichomonosis
Name of disease
Trichomonosis, trichomoniasis, crop canker, and frounce.

Clinical history
Trichomonosis primarily affects pigeons, doves, birds of prey, domestic fowl, and birds in captive collections. The disease is frequently reported in doves and pigeons and is variably reported in other avian species.

Causative agent
The flagellated protozoal parasite *Trichomonas gallinae* is the cause of *trichomonosis*.

Clinical signs and lesions
Trichomonosis is characterized by a rapid and progressive course. The intragular region (throats) of affected birds may appear to be bulging due to the canker and fluid often accumulates in the mouth, most likely due to the inability to swallow. Affected birds may be observed attempting to aggressively ingest food; however, the mass precludes swallowing of food particles. The inability to ingest food leads to rapid weight loss and subsequent weakness and listlessness. Additionally, affected birds may be observed open-mouth breathing and gasping for air if the cankers obstruct respiration. Birds can die after 8–14 days of being infected in acute cases. Lesions initially appear as small white to yellow areas of necrosis within the oral cavity, crop, or esophagus. The cankers expand rapidly, often coalescing, to form large masses in the oral cavity and esophagus and

often lead to complete obstruction of the oral cavity and esophagus. The virulence of *T. gallinae* is variable and the parasite can be found in clinically normal as well as diseased birds, so the presence of the parasite is not indicative of disease.

Transmission route
Trichomonads are transferred from one avian host to another by direct contact or ingestion of contaminated food and water. Contaminated water is the most likely source of infection for chickens and turkeys. Predation of infected birds is a method of exposure for birds of prey.

Diagnostic tests
Confirmatory testing can be performed by acquiring swabs of the oral fluid, mucus, or canker and performing wet mount examinations by light microscopy. The trichomonads have a characteristic undulating swimming motion (see video on website). A commercial media culture packet (InPouch™ TF) has been developed by BioMed Diagnostics (White City, Oregon, United States) for the culture of bovine trichomonads, but the packets work well for culture of *T. gallinae*. PCR testing is available for samples in which live trichomonads are not available for culture.

Differential diagnosis
Gross lesions of avian trichomonosis are characteristic, but not pathognomonic. Other diseases including avian pox, candidiasis, aspergillosis, oral *Capillaria* spp. infection, and vitamin A deficiency can have similar gross findings.

Prevention and control
Routine cleaning of birdfeeders and birdbaths with a 10% bleach solution in water and disposal of wet, moldy feed is recommended to help control outbreaks. Successful treatments in early infections include metronidazole and carnidazole [5]. Treatment of birds with fulminate trichomonosis is generally unrewarding. The intestinal trichomonad, *Tetratrichomonas gallinarum* has also been found in numerous galliforms but there is no evidence of disease associated with this parasite.

Crop capillariasis
Name of disease
Crop capillariasis, thread worm infection.

Clinical history
Crop capillariasis is one of the more frequent causes of respiratory distress in quail. The parasite is found infrequently in other game birds.

Causative agent
Capillaria contorta is a thread-like nematode parasite found in the oral cavity, crop, and esophagus of affected birds.

Clinical signs and lesions
The affected birds may be observed open-mouth breathing and gasping for air. The lesions are similar to trichomonosis and often the disease is misdiagnosed as trichomonosis if conformational wet mounts of the protozoa (trichomonads) are not performed. On necropsy, numerous thread worms can be seen, especially with use of dissecting scope or magnifying lens.

Transmission route
Birds are generally infected by ingesting embryonated eggs or the earthworm vector.

Diagnostic tests
The eggs can be observed on fecal floats. The eggs are 55–60 μm long and 26–28 μm wide. They contain two polar opercula and are similar in appearance to *Trichuris* (whipworm) eggs except that the polar plugs are off-set in *Capillaria* (Figure 6.1).

The thread-like parasites are slender long worms that may be difficult to visualize grossly and may require magnifying glass or dissecting scope.
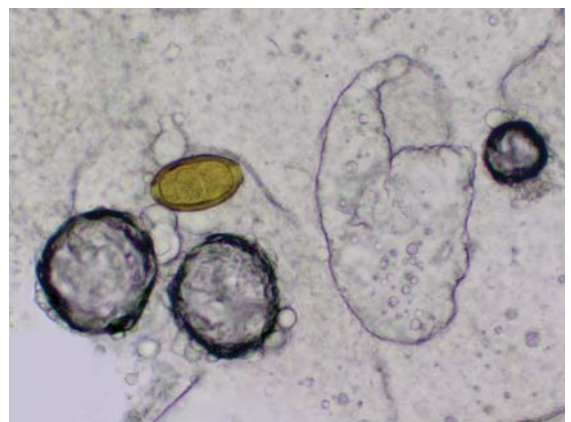
Figure 6.1 *Capillaria* eggs from sugar floatation of chicken feces(20x).

Differential diagnosis

Avian pox, candidiasis, aspergillosis, trichomonosis, and vitamin A deficiency can have similar gross lesions.

Prevention and control

The eggs are extremely environmentally resistant, thus treatment alone will not stop an outbreak. Other capillarids are found in the gastrointestinal tract and can cause weight loss and enteritis. Various antihelmentics including benzamidazole products have been successful; however, there are no approved products available, thus prescription based off-label use is indicated [6]. Prevention of infection is performed by removing feces and limiting access to earthworm vectors.

Syngamiasis
Name of disease

Gapeworm infection, gapes, tracheal worm, syngamiasis. Pheasants are most frequently affected and it is variably found in other poultry.

Causative agent

Syngamus trachea is a bright red nematode.

Clinical signs and lesions

Pheasants are most frequently affected and clinical signs include gaping and gasping, listlessness, and lethargy.

Transmission route

Eggs are passed up the trachea, swallowed and then defecated. Eggs can be concentrated by earthworms and various other invertebrates that serve as paratenic hosts.

Diagnostic tests

The eggs are approximately 80–100 µm in length with shallow polar plugs, similar to the morphology of *Capillaria* and *Trichuris*. These nematodes are easily seen on necropsy given their bright red color and the fact that the males and females are attached and form a characteristic "Y" shape (Figure 6.2).

Differential diagnosis

Avian pox, candidiasis, aspergillosis, trichomonosis, crop capillariasis, and vitamin A deficiency can have similar gross lesions and clinical signs.

Prevention and control

Treatments for gapeworm include benzimidazole antihelmentics including thiabendazole and fenbendazole [7]. In the United States, thiabendazole is registered for control of gapeworms in pheasants [6].

Figure 6.2 *Syngamus trachea* from trachea of wild turkey.

Zoonotic potential

None reported.

Dispharynxosis
Name of disease

Dispharynxosis or proventricular worm.

Clinical history

The disease is observed infrequently in ruffed grouse and other galliforms. Severity of disease is related to the number of worms infecting the bird.

Causative agent

Dispharynx nasuta is a nematode.

Clinical signs and lesions

Infected birds are often listless and thin, but have an aggressive appetite. The nematode head penetrates the lamina propria of the proventriculus leading to swelling, ulceration, inflammatory infiltrates, caseous necrosis, squamous metaplasia, hemorrhage, and destruction of proventricular glands.

Transmission route

Eggs are passed in feces and ingested by the required isopod intermediate hosts (pill bugs and sow bugs). The life cycle is completed when the birds ingest the isopod hosts.

Diagnostic tests

The nematodes are relatively short (8mm) and are generally found easily in the proventriculus on necropsy. The eggs are ellipsoidal and approximately 35 µm long and 21 µm wide and are embryonated in freshly defecated feces.

Differential diagnosis

Avian pox, candidiasis, aspergillosis, trichomonosis, and crop capillariasis.

Prevention and control

Prevention is mainly achieved by limiting feces of wild birds from accumulating near captive birds and limiting isopod intermediate hosts. Successful treatment has occurred through use of ivermectin and benzimidazoles [8].

Note: Cryptosporidiosis can cause respiratory disease but see listing under causes of diarrhea.

Causes of diarrhea

Coccidiosis
Name of disease

Coccidiosis.

Clinical history

Although, coccidiosis outbreaks have been reported from backyard poultry, the disease is most often observed in commercially raised birds. There are two reasons for this: 1) parasite replication is self-limiting given the fixed number of asexual cycles and 2) after infection, the host develops protective immunity. The disease most often occurs in immunologically naive animals or in animals that are stressed or crowded which can both result in overwhelming infections.

Causative agent

Coccidiosis is the general term given to the disease caused by the lesions and clinical signs elicited by *Eimeria* spp., which are obligate intracellular protozoal parasites that infect and replicate within the host's intestinal epithelial cells.

Clinical signs and lesions

Mild to moderately affected birds have suppressed weight gain and diarrhea. Severely affected birds are depressed, have marked diarrhea (possibly with blood), ruffled feathers, and the birds often huddle together for warmth. Significant mortality can occur in untreated flocks. The lesions and clinical signs produced by the parasites are a function of the number of ingested oocysts, the immune status and age of the host, the site of infection, concurrent infections, and other factors [9].

Clinical coccidiosis in chickens usually occurs as a result of one of three species. Lesion with *E. acervulina* causes raised white nodules in the duodenum and *E. maxima* causes hemorrhage and mucosal reddening in

Figure 6.3 Necrohemorrhagic cecal cores in a 2-week-old Americauna chick with severe coccidiosis from *Eimeria tenella*. (Source: Photograph courtesy of Dr. Shelly Newman, University of Tennessee, Department of Biological and Diagnostic Services.)

the jejunum. *Eimeria tenella* causes hemorrhagic cecal cores and often bloody feces are noted (Figure 6.3). Clinical disease in domestic turkeys occurs as a result of various different *Eimeria* species, especially *E. adenoides* (which form dry firm cecal cores), and *E. meleagrimitis* (forming petechiae and pseudomembranes in the ileum and jejunum).

Coccidia in game birds are often more prolific than those in chickens and turkeys and can reach as high as 600,000 to 2,000,000 oocysts produced per oocyst ingestion [10,11]. Oocyst production generally extends longer in game birds compared to chickens and turkeys, leading to increased environmental contamination [11]. Coccidiosis in ring-necked pheasants and chukars present with gross lesions consisting of caseous cecal cores and hemorrhagic typhlitis.

In contrast, bobwhite quail usually lack cecal cores, but instead have attenuated intestinal mucosa with marked enteritis and abundant edema.

Transmission route

Oocysts are shed in the host's feces and once outside the host, undergo sporulation. Following ingestion by another host animal, sporulated oocysts rupture, releasing sporozoites, which infect the host's epithelial cells. Sporulation occurs more rapidly in ambient temperatures >25°C. Minimum sporulation time can be as little as 24 hours in warm moist conditions.

Diagnostic tests

Oocysts can be easily found on fecal floats or intestinal scrapes from affected regions of the intestines. Oocyst

Figure 6.4 Sugar flotation generated *Eimeria* spp. oocysts from a chicken. Freshly defecated oocysts are unsporulated.

size and prepatent periods can vary depending on the *Eimeria* spp. infecting the birds [9] (Figure 6.4).

Differential diagnosis

Clostridium infection, histomonosis, salmonellosis, cryptosporidiosis, dehydration, and pesticide intoxication.

Prevention and control

Prevention of coccidiosis can be performed by removing bird feces and limiting mixing of young and older birds. Subclinical infections of coccidia can predispose birds to other parasitic and bacterial infections including *Clostridium* spp., thus proper coccidial control is important for overall health.

The development of effective chemotherapy against coccidia was a major milestone in the evolution of the poultry industry, and without the use of these anticoccidial compounds the broiler industry as we know it would not exist. Anticoccidial compounds generally fall into one of two categories. The first are polyether ionophores, which disrupt the proper intra and extracellular concentrations of the various cations, leading to cellular dysfunction in the parasite. The second group includes compounds that cause an enzymatic reaction. Ionophores generally have lower rates of resistance development compared to the enzyme reaction drugs listed above and often allow some low level cycling of the coccidia in the host leading to host immunity [12]. Anticoccidial drugs belonging to the polyether ionophores include lasalocid, salinomycin, maduramicin, monensin, narasin, lonomycin, and semduramicin. Other drugs include amprolium, clopidol, diclazuril, decoquinate, robenidine, roxarsone,

sulfadimethoxine/ormetoprin, salinomycin, semduramicin, and zoalene. The efficacy of these drugs is variable and may require some investigations to determine the most effective compound. Due to continual use of amprolium, resistance has been reported in numerous species including bobwhite quail [13]. Maxiban (narasin/nicarbazin) has been found to be toxic in turkeys and should not be used in this species. Live vaccines, consisting of infective oocysts of the important *Eimeria* species, are available for use in the poultry industry, providing an alternative to the use of anticoccidial drugs. The development of vaccines for coccidia is possible due to the fact that replication is self-limiting and infected birds develop protective cell-mediated immunity [14]. Protective immunity develops rapidly after exposure, but depends on reinfection to reinforce the developing protection. There is no confirmed cross-protection between different species of *Eimeria*, resulting in the requirement for multiple species of coccidia in vaccines. Given that *Eimeria* are species specific, separate and specific vaccine formulations are needed for each species of bird. Commercial vaccines are currently available for chickens and turkeys. Northern bobwhites and chukars administered low doses of respective host *Eimeria* spp were protected against a high dose challenge, suggesting that vaccine development may be an option in game birds [15,16].

Zoonotic potential

None. *Eimeria* are species specific.

Histomoniasis
Name of disease

Histomonosis, histomoniasis, or blackhead

Clinical history

Blackhead is considered the most important parasitic disease for turkeys and is an important cause of mortality for numerous game birds. Recently, mortality has been documented in backyard chickens [17].

Causative agent

Histomonas meleagridis is a protozoal enteric pleomorphic flagellate. It loses the flagellum once it adheres to the intestinal wall and therefore has flagellated and amoeboid stages.

Clinical signs and lesions

Clinical signs include diarrhea, often having a sulfur yellow appearance, along with non-specific findings of weight loss and ruffled feathers. Gross lesions generally
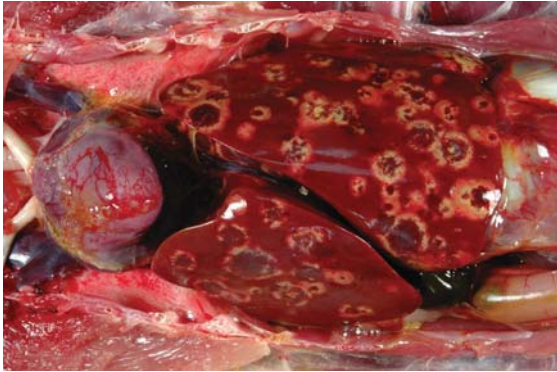
Figure 6.5 Gross histomonosis lesions in a wild turkey liver.

Figure 6.6 Histomoniasis in a turkey with cecal lumen distended by a large amount of caseous necrotic and hemorrhagic material consistent with cecal cores.

include characteristic target-shaped foci of necrosis of variable size in the liver (Figure 6.5).

The ceca are markedly thickened and the lumen is distended by a large amount of caseous necrotic and hemorrhagic material consistent with cecal cores (Figure 6.6).

Transmission route

Ringneck pheasants are the natural host for *Histomonas*; however, chickens can be patent host for the parasites including the *Heterakis* nematode that serves as a paratenic host for *Histomonas*. Earthworms can serve as second paratenic host harboring both the *Heterakis* nematode and histomonads. In high densities, transmission of histomonads can occur directly from bird to bird via cloacal drinking [18].

Diagnostic tests

On fresh carcasses, the histomonads can often be identified using saline wet mounts of swabs or scrapes taken from necrotic cores. It is important to have a heat source, such as the microscope light, to warm the histomonads. Histomonads have a single flagellum and their motion is characterized by slow agitating circular rotations. This differs from trichomonads, which have a fast undulating motion. If histopathology is performed, within the areas of necrosis and inflammation there are numerous round to oval protozoal organisms of 10–20 μm diameter. Often the protozoa are surrounded by clear vacuoles, giving a halo appearance. Histomonads similar to trichomonads autolyze soon after death, thus histopath is often unrewarding on birds that have been dead for more than 24 hrs. In addition, affected cecal contents can be inoculated into Dwyer's media and shipped to diagnostic laboratories for identification. It is important to inoculate samples into prewarmed (>30°C) media and to keep media warm during shipment to ensure survival of histomonads [19]. The addition of tropical fish shipping warmer packets to the shipping container produces an ample heat source (Beckstead and Gerhold, unpublished data).

Differential diagnosis

Coccidiosis, *Clostridium* infection, salmonellosis, cryptosporidiosis, dehydration, and pesticide intoxication.

Prevention and control

Turkeys, quail, grouse, or chukars cannot be raised in the same areas as chickens or pheasants. Additionally, the practice of raising game birds in houses previously used to house poultry tends to lead to blackhead outbreaks due to the environmental persistence of the *Heterakis* egg containing the histomonads. Nitrasone (Histostat7 Alpharma Inc. Clifton, New Jersey) has been used to prevent outbreaks; however, the drug has limited efficacy once clinical signs are evident in birds. Various antihelmentics including benzamidazole products aimed at limiting *Heterakis* development have been useful in preventing outbreaks of blackhead [20].

Zoonotic Potential

None reported.

Cryptosporidiosis
Name of disease

Cryptosporidiosis.

Clinical history

Cryptosporidiosis may be seen infrequently in pen-raised quail and other birds.

Causative agent

Cryptosporidium baileyi are coccidia organisms that invade host epithelial cells of the intestines and respiratory system.

Clinical signs and lesions

The most common clinical signs include diarrhea and dehydration. In addition to diarrhea, the parasites can infect the respiratory tract and cause respiratory disease. In respiratory infections the parasite can lead to coughing, sneezing, and dyspnea.

Transmission route

The oocysts are shed in the feces of infected birds and ingested from contaminated environments.

Diagnostic tests

The oocysts are small in size with a diameter of approximately 5 μm. The oocysts have a pink hue when polarized, but they can be difficult to observe due to their small size. Fecal samples from suspected cases should be sent to trained parasitologists for identification.

Differential diagnosis

Coccidiosis, *Clostridium* infection, salmonellosis, histomoniasis, dehydration, and pesticide intoxication.

Prevention and control

There are no known effective treatments for cryptosporidium. Removing feces and allowing the affected area to be exposed to direct sunlight is the most effective means of controlling outbreaks.

Zoonotic potential

Cryptosporidium baileyi is specific to birds. Turkeys and chickens can be infected with *C. meleagridis*, which some sources say may be synonymous with *C. parvum*, which is zoonotic [9] (see Chapter 8).

Miscellaneous parasites

Twenty-two nematode (roundworm) species belonging to various genera are found in galliforms. Ascarid eggs can be seen on fecal flotation (Figure 6.7). *Ascardia*

Figure 6.7 Mourning dove intestines impacted with ascarids.

Figure 6.8 Ascarid eggs on fecal float from chicken (10x).

dissimilis and other ascarids are frequently reported in birds in the southeastern United States. In heavy infections, ascarids can interfere with intestinal passage of food (Figure 6.8).

Ascarids are relatively large and range from 3–10 cm in length. *Heterakis* spp. are cecal nematodes and in contrast to the larger size when compared to ascarids, *Heterakis* are short worms ranging from 0.5–2.0 cm in length. *Heterakis* can cause cecal inflammation in high numbers, but more importantly it is the vector of *Histomonas meleagridis*, the cause of blackhead.

The trematodes *Athesmia heterolecithodes* and *Echinoparyphium recurvatum* have been reported to cause morbidity and mortality in galliforms. *Athesmia heterolecithodes* is found in the bile ducts of the liver and can

obstruct bile flow, resulting in enlargement and fibrosis of bile ducts. *Echinoparyphium recurvatum* is found in the small intestines and has been reported to cause severe enteritis, emaciation, anemia, and mortality in chickens and domestic turkeys. The fluke is relatively small at approximately 2.5 x 0.5 mm. Since all the trematodes require a snail intermediate host, it would be more likely to find trematodes in birds in wet coastal areas compared to birds from dry, arid areas. Limiting access to marshes and standing water is the most effective control.

In severe cases of high parasite intensity, cestode infections can interfere with intestinal passage of food, otherwise there is limited disease associated with cestodes. Sometimes proglittids can be seen in fecal floats (Figure 6.9).

Haemoproteus meleagridis, Leukocytozoon smithi, and *Plasmodium hermani, P. kempi, Plasmodium* sp. are all vector-borne protozoa that infect erythrocytes and muscle (*H. meleagridis)*, leukocytes, liver, and spleen (*L. smithi*), and erythrocytes (*P. hermani, P. kempi, Plasmodium* sp.) (Table 6.2). Proper vector control including eliminating stagnant water is needed to control these diseases.
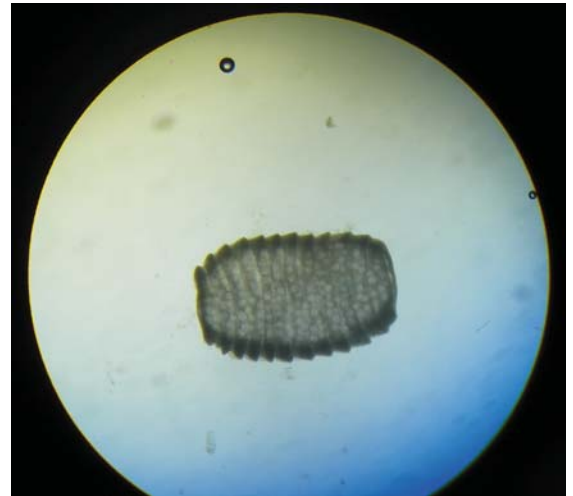
Figure 6.9 Tapeworm proglottid that just happened to be seen on a fecal float from a chicken with cestodiasis. (Source: Photograph courtesy of Cheryl Greenacre.)

Table 6.2 Important erythrocytic and leukocytic avian protozoa with corresponding vectors, organ involvement, and lesions

Protozoa	Vector	Parasite development in avian host	Lesions
Haemoproteus meleagridis	*Culicoides* spp. (midges)	Asexual development (Merogony) within tissue and capillary endothelial cells; gametocytes found in RBCs	Myositis. Grossly with areas of pallor and hemorrhage.
Leukocytozoon smithi	*Simulium* and *Prosimulium* spp. (Blackflies)	Merogony in liver and erythrocytes, gametocytes in leukocytes	Splenomegaly, hepatomegaly, tissue pallor, anemia. Liver can have central necrosis. Spleen and lung infiltrated with pigment-filled macrophages. Can have encephalitis and optic neuritis
Plasmodium hermani	*Culex nigripalpus* mosquitoes	Two stages of merogony in liver, spleen, brain, kidney, and lung followed by one stage of exoerythrocytic meronts in capillary endothelial cells. Then erythrocytic stages seen in erythrocytes.	Anemia, parasitemia, splenomegaly, hepatomegaly. Spleen and liver infiltrated with pigment-filled macrophages. +/− dilated capillary venules with edema. **Same vectors carry avian poxvirus so birds may be found with both diseases.
Plasmodium kempi	*Culex* spp. mosquitoes		
Plasmodium sp.	*Culex* spp. mosquitoes		

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Medicine and Surgery

Physical Examination, Anatomy, and Physiology

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Physical examination

Introduction

A physical examination is important for identifying any abnormalities that may be occurring in the flock and in the individual bird. The following will focus on the chicken, but the information can be applied to physical examinations of most birds. It is important to understand what is normal, including behavior, before deeming a finding abnormal. Before restraining the bird for a physical examination, obtain a thorough history.

History

Obtain signalment of the bird (breed, age, and gender) and determine its use. Is it a pet, a pet that happens to lay eggs, is it kept for egg production, for meat production, for show, or for breeding? How many chickens are owned, how long has the flock been owned, did they come from multiple sources at multiple times (i.e., is it an open or closed flock), when was the last addition to the flock, how long has this chicken been owned, where was it obtained, any previous diseases, any previous treatments? Also ask about housing, substrate, perch size and composition, and space of coop and range. Are they brought in at night? Ask about diet including type, brand, amount, where purchased, how old is the food, any treats or supplements, medicated foods? Are they offered greens and insects for enrichment? If laying, how many nest boxes are set up and what is the laying history? Last, what is the presenting complaint and how

long has it been occurring, are multiple birds affected, and is it progressive?

Restraint
Prior to restraint

Before restraining the bird, perform a visual examination of the bird and its surroundings from a distance, preferably before the bird becomes aware of you. See if the bird brightens up after it is aware of your presence as this could be indicative of a bird trying to "put on a good show" or appear healthier than it is, because birds that look sick are pecked by conspecifics or become a meal for a predator. Examine the alertness of the bird and if it is interacting with others in the flock or off in a corner by itself. Healthy birds are curious and hold their head high with bright open round eyes, whereas sick birds hold their head down with the eyelids partially closed (Figure 7.1 and Figure 7.2). Observe stance and ability to walk normally. Examine the droppings for consistency, blood, or abnormal smell. Realize that chickens have two types of feces, the more common drier droppings and the wetter cecal droppings.

Restraint

The restraint of any bird begins with controlling the weapons of that bird, for example with parrots the head is restrained first, and with raptors the talons are restrained first. Chickens flap their wings and may injure themselves or you, so first restrain their wings gently by holding the wings to the body in their normal folded position with your fingers spread wide apart to

Figure 7.1 Healthy, curious chicken with round, open, bright eyes and head up.

Figure 7.2 Sick chicken with closed eyes, head down, and a copious oculonasal discharge.

Figure 7.3 Demonstration of proper restraint in a chicken with the bird tucked under the arm with the head pointed forward. Alternatively, the head can be pointed backwards.

restrain as much of the wing as possible. Do not bend the wings backwards, as the relatively flimsy wings of chickens can be easily luxated at the shoulder or elbow. Also, remember to allow the normal up and down excursions of the keel so the chicken can breathe, because birds lack a diaphragm. The bird can then be tucked under your arm either pointing forward or backward (Figure 7.3). Placing a towel on a table is a great way to evaluate a bird at your eye level. Most chickens are very calm and usually the less restraint the better. Systematically evaluate the chicken from head to toe, but do save the oral examination for last as this is highly perturbing to the chicken (see website for video of a complete physical examination in a duck.)

Physical examination

Examine the head for symmetry of the beak, eyes, sinuses, and nostrils. Examine for discharge, crusts, scratches, scabs, swellings, and discolorations. The beak tips should come to a point. The iris should be the same color on both sides. A lighter color of one iris may be indicative of the ocular form of Marek's disease. Also, the eyes (anterior chamber or lens) should not be cloudy. Ocular discharge can appear as matted feathers along the cranial ventral lid margin. Flip the feathers cranially that cover the ear and examine the shallow ear of the chicken for discharge, blood, or parasites.

Figure 7.4 Healthy rooster with a firm, red comb. External features of the head are identified (a) blade of comb; (b) operculum; (c) crest of comb; (d) body of comb; (e) maxillary rectus; (f) wattle; (g) ear lobe; (h) external acoustic meatus; (i) points of comb.

The comb should be firm and red (Figure 7.4). A capillary refill time can be performed on the comb by digitally pressing and releasing (Figure 7.5). It should refill in about two seconds. The comb should not be pale. Occasionally the comb is flopped over to one side, which can be normal as long as this did not occur suddenly.

Alternatively, the basilic vein (cutaneous ulnar vein) located just distal to the ventral surface of the elbow can be digitally pressed as in other birds to examine refill time. Normally, when the finger is removed from the vein, refilling will be so fast that it cannot be witnessed visually. If it can be witnessed visually then the bird is considered approximately 5% dehydrated, and if one second can be counted, then the bird is about 10% dehydrated or in shock. Decreased corneal moisture exhibited by a dull surface appearance to the eye, or in severe cases, recession of the globe, is also indicative of dehydration [1].

Palpate the crop. It should feel soft and fluctuant, similar to a bean bag, and have crop movements about once

per minute. Birds lack organized lymph nodes, so normally there should be no subcutaneous masses present. Birds possess only one gland, the bilobed uropygial gland (preen gland), on the dorsal caudal area just cranial to the base of the tail.

The keel should be straight with no deviations, with a "V" shape to the pectoral muscles on either side. In general, back yard chickens normally feel thinner in the pectoral muscles and therefore normally have a more pronounced "V" shape than parrots. If "blisters," or any redness or swelling, are seen on the keel it may be indicative of a bird that has been in sternal recumbency and not walking for a period of time [2].

To auscult the heart, place the stethoscope over the breast muscle on either side of the keel (Figure 7.6). Birds have a four-chambered heart and the sounds are similar to mammals, but faster at about 140–250 beats per minute. The heart rate can increase significantly with the stress of handling. To auscult the lungs, place the stethoscope over the craniodorsal body wall. Normal respiratory rate in the chicken is about 15–30 respirations per minute, but this too can increase with

Figure 7.5 Same rooster as in Figure 7.4 after the comb has been digitally pressed and released. The "refill time" is approximately 2 seconds.

Figure 7.6 Physical examination of a chicken with a towel on the tabletop showing proper placement of stethoscope over breast muscle to auscult the heart.

the stress of handling. Temperature is typically not taken in birds because it is generally higher than the maximum for typical thermometers and there is debate over whether ill birds exhibit a fever like mammals. The normal core body temperature of a chicken is about 105.0–109.4°F (40.6–43.0°C) and can be measured with a thermistor thermometer with a small (2 mm wide) probe if needed.

The feathers should lay down flat and smooth. Conspecific aggression may result in feather loss over the dorsal head and neck area or the vent area. Lift feathers up and look at the shafts, especially under the tail near the vent and under the wings and base of primary feathers for evidence of lice and mites. Lice can be seen with the naked eye as beige oblong insects; their nit eggs look like clumps of white material at the base of feathers. Mites can cause abnormal irritated, inflamed, or pitted looking skin.

All joints should be examined externally for evidence of trauma and palpated for range of motion, absence of stiffness, crackles, and clicks. Palpate for fractures. The caudal coelomic cavity should be soft and doughy in an egg laying bird, but should not be extended, fluid filled, or hanging down between the bird's legs. Often a bird will present for lameness, when in fact it has an enlarged coelomic cavity interfering with normal ambulation. Commonly, in a normal bird only the ventriculus is palpated within the coelomic cavity, or if present, an egg. The liver should not extend past the level of the sternum.

The vent should be clean and dry. There should not be a pasty vent, a vent with white urates stuck to it, nor should there be feces around the vent. The conformation

Figure 7.7 The bird can be lifted up to examine the smooth plantar surface of the feet and symmetrical appearance of the caudal surface of the hocks (tibiotarsal-tarsometatarsal joint). This white Pekin duck has a left metatarsal and phalangeal pododermatitis as well as a swollen right hock joint.

of some egg laying birds requires the plucking of feathers around the vent to keep them clean.

The bird can then be lifted up to expose the smooth plantar surface of the feet and symmetrical appearance of the caudal surface of the hocks (tibiotarsal-tarsometatarsal joint) (Figure 7.7). Scabs, callouses, swellings, keratin overgrowth, dark areas, or cuts are abnormal on the plantar surface of the feet. The hocks should be of equal size, not swollen, and the tendon should be in the trochlear groove. The nails should be smooth, straight, and extend to just below the plantar surface when placed on a flat surface.

Last, an oral examination is performed. Gently open the mouth to evaluate the oral cavity for white plaques, masses, abnormal smell, and evaluation of the choanal slit and choanal papillae on the roof of the mouth (Figure 7.8).

Accurately weigh the bird on a gram scale in order to accurately calculate potential drug dosages or to compare to previous or later weights.

Figure 7.8 Same chicken shown in Figure 7.2 demonstrating how to open the mouth to perform an oral examination. This chicken has a severe case of pox with white oropharyngeal plaques including the tongue and choana.

Anatomy and physiology

Understanding the anatomy of a chicken allows for recognition of normal versus abnormal findings and accurate descriptions of abnormalities in the record. Only the relevant physiological differences that differ from mammals will be included in the descriptions that follow.

Body regions

The body regions divide the surface of the entire body and can be subdivided into subregions with special clinical interest [3].

Head and neck regions

Several regions can be identified on the head including nasal, nasal arch, forehead, orbital, suborbital, crown, postorbital, anterior dorsal neck, posterior dorsal neck, lateral neck, and anterior ventral neck (Figure 7.9).
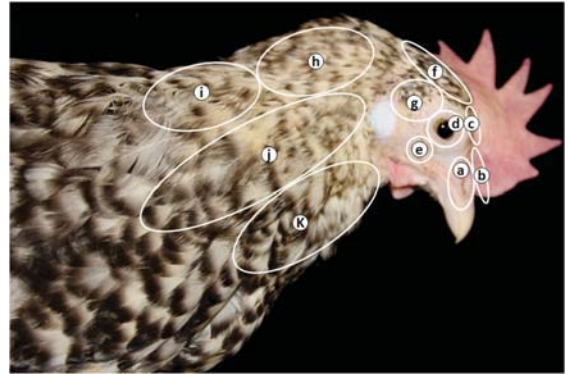
Figure 7.9 Regions of the head and neck of an adult female chicken. (a) nasal; (b) nasal arch; (c) forehead; (d) orbital; (e) suborbital; (f) crown; (g) postorbital; (h) anterior dorsal neck; (i) posterior dorsal neck; (j) lateral neck; (k) anterior ventral neck.

The natural orifices on the head (the eyes, external acoustic meatuses, nasal openings, and mouth) as well as the ornamental structures (comb, wattles, ear lobes) are frequently used to identify certain clinical signs in the diseased chicken. The ornamental structures differ in size between male and female chickens even at a young age, being more developed and much larger in size in the male (Figures 7.4 and 7.10).

External trunk and lower extremities regions

Major body regions that can be identified on a chicken include metatarsal, ankle, abdominal, knee, sternal, prolateral, wing, ventral abdomen, crop, ventral neck, lateral neck, and posterior dorsal neck regions (Figure 7.11).

Wing regions (ventral aspect)

Wing regions may be used for blood collections or examination for external parasites and abnormal feather conditions. These regions include prolateral, shoulder, upper arm, forearm, prepatagium, wrist, hand, and alular patagium [3,4] (Figure 7.12).

Skeletal anatomy

Only characteristic features of the chicken skeleton will be described here. Birds, in general, are noted for their exceptionally large sized eyes, which are accommodated by equivalently large orbits in the skull (Figure 7.13). The two bony orbits are separated from each other by an ossified partition, the interorbital septum. The quadrate bone is a very complex bone that articulates with the mandible to help in jaw suspension. It also forms the pivotal bone for the kinetic jaw mechanism.
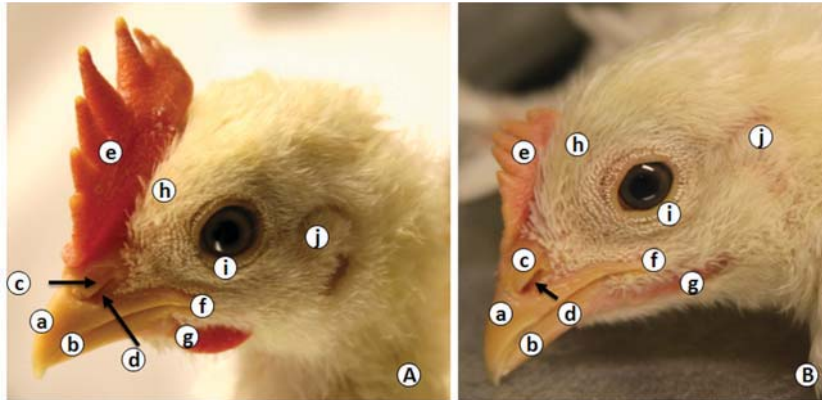
Figure 7.10 External features of the head of a five-week-old male (A) and female (B) chicks. (a) superior (maxillary) beak; (b) inferior (mandibular) beak; (c) operculum; (d) external nares; (e) comb (compare sizes between male and female of the same age); (f) maxillary rictus; (g) wattle; (h) frontal feathers, (i) eye feathers, (j) ear feathers.
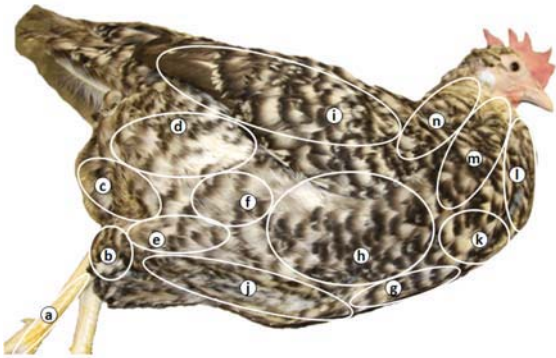
Figure 7.11 Body regions of adult female chicken. (a) metatarsal; (b) ankle; (hock) (c) abdominal; (d) prolateral; (e) shank; (f) knee; (g) sternal; (h) prolateral; (i) wing; (j) ventral abdomen; (k) crop; (l) ventral neck; (m) lateral neck; (n) posterior dorsal neck.

Figure 7.12 Regions of the ventral aspect of the wing of a four-week-old chick. (a) prolateral; (b) shoulder; (c) upper arm; (d) forearm; (e) propatagium; wrist (carpus) (f) wrist; (g) hand region (area pf major and minor metacarpal) (h) alular patagium.

The chicken has a single occipital condyle that articulates with a small and ring-shaped atlas. The cervical vertebrae have characteristic saddle-shaped articular processes. In the last two cervical vertebrae the vertebral segment of the ribs can be identified. The chicken has 4–6 thoracic vertebrae and the lumbar and sacral vertebrae are fused into a structure called the synsacrum. The caudal vertebrae are variable in number and several of them fuse to form the pygostyle (plowshare, rump post) [5,6].

There are seven pairs of true ribs. Except for the first and the last, ribs have uncinate processes which overlap the succeeding rib giving rigidity to the rib cage. In the sternum (breast bone), the keel (carina) can be identified and serves as the origin of major flight musculature (pectorals and supracoracoid). The pectoral girdle is comprised of three pairs of bones that support the wings: Fused clavicle (furcula), coracoids, and scapulae (Figure 7.14). They come together dorsally leaving a triosseal canal (foramen triosseal) through which the tendon of the supracoracoid muscle passes to insert on the humerus. It acts to elevate the humerus and the wing.

The hyoid apparatus of the chicken is unique and is composed of several segments [7]:

1. Paraglossal (entoglossal), which extends into the free portion of the tongue
2. Two cornua extended laterally from the paraglossal bone forming the wide base of the tongue
3. Rostral basibranchial (basihyal) bone lies in the fixed portion of the tongue
4. Caudal basibranchial (urohyal) bone
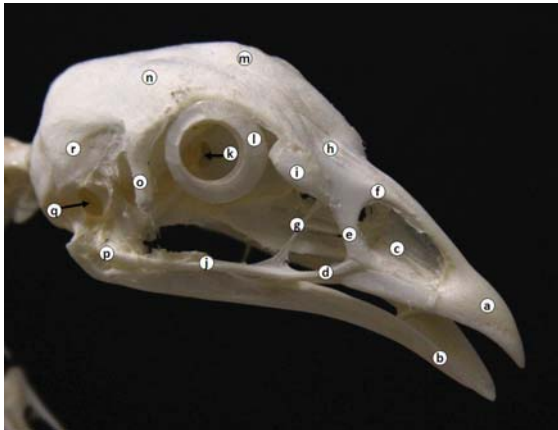
Figure 7.13 Bone of the skull of adult chicken. (a) Premaxilla; (b) mandible; (c) external nares opening (d) maxilla; (e) lateral ramus of the nasal; (f) nasal; (g) Lacrimal process (h) nasal frontal suture; (i) lacrimal (prefrontal) (j) jugal bar (k) optic foramen; (l) scleral ring (bone); (m) frontal; (n) parietal; (o) postorbital process; (p) quadrojugal; (q) external acoustic meatus; (r) squamosal.
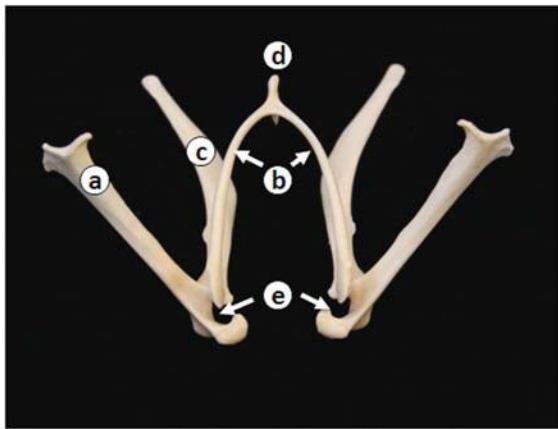
Figure 7.14 Thoracic limb girdle of the chicken. (a) coracoid; (b) clavicle; (c) scapula; (d) apophysis furculi (hypocleidum, lamina interclavicularis); (e) foramen triosseum (triosseal canal).

5. Ceratobranchial bone
6. Epibranchial bone.

Myology

Because of the complexity of this system and the large number of muscles present on the chicken body, only those related to the wing and leg will be highlighted because they are anatomical landmarks for clinically relevant structures and procedures. The relevant muscles of the thoracic limb are latissimus dorsi, rhomboid,
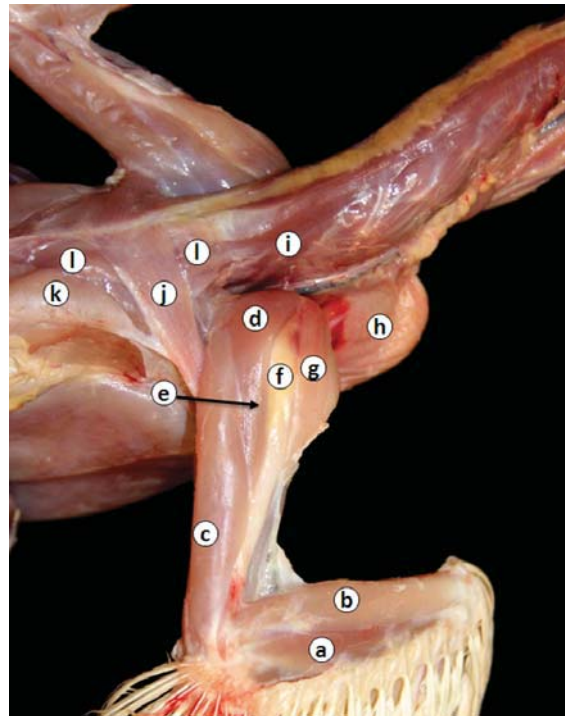
Figure 7.15 Dorsal aspect of the shoulder girdle of adult rooster. (a) extensor carpi (metacarpi) ulnaris; (b) extensor carpi (metacarpi) radialis; (c) triceps brachii; (d) deltoid major; (e) superficial coracoid; (f) humerus; (g) propatagial (deltoideus pars propatagialis; tensor propatagialis; pars longa/brevis); (h) crop (ingluvies); (i) longissimus dorsi (cervical portion); (j) scapulohumeralis; (k) latissimus dorsi; (l) tapezius.

deltoid, coracoid, propatagial, scapulohumeral, trapezius, extensor carpi ulnaris, extensor carpi radialis, biceps brachii, triceps brachii, pronator, digital muscles, flexor carpi ulnaris, and alular muscles (Figures 7.15–7.17).

In the pelvic limb, in order to find the sciatic (ischiadic) nerve the sartorius, quadriceps femoris, femrotibial, flexor crural medial, and lateral muscles should be identified (Figure 7.18) [8–10].

Skin and appendages

Generally, the skin of poultry is thin and loosely attached to the body. Apterylae, non-feathered skin, is covered with extremely thin keratinized stratified squamous epithelium. In the areas where feathers are present on the skin (pterylae) a similar type of thin but highly keratinized stratified squamous epithelium is present. However, the amount of keratin increases as it approaches the feathers.
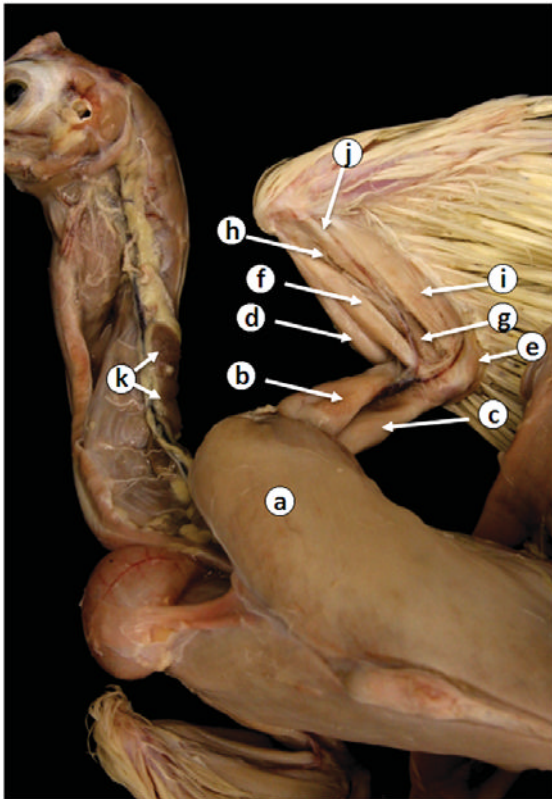
Figure 7.16   Ventral view of the muscles of the wing of an embalmed rooster. (a) thoracic pectoral (superficial); (b) biceps brachii; (c) triceps brachii (humeral head); (d) extensor carpi radialis; (e) expansor secondarum (dermoulnaris); (f) pronator superficialis (longus – deep et brevis – superficial); (g) deep pronator; (h) extensor indicis digital major (longus); (i) flexor carpi ulnais; (j) deep digital flexor (flexor digitorum sublimis); (k) thymus.

Figure 7.17   Ventral aspect of the left wing of adult rooster after complete removal of the feathers. (a) Skin of patagial fold and embedded within is it the propatagial ligament; (b) propatagium; (c) biceps brachii; (d) triceps brachii (humeral head); (e) basilic vein; (f) extensor carpi radialis (extensor metacarpi radialis); (g) superficial pronator; (h) deep pronator; (i) flexor carpi ulnaris; (j) deep digital flexor; (k) extensor indicis longus (extensor digiti longus majoris); (l) interosseous palmaris (ventralis); (m) abductor alulae (policis); (n) adductor alulae (policis).

The spur (metatarsal spur) is a horny structure present on the caudal surface of the leg of domestic fowl (Figure 7.19). It is well developed in males and may reach several inches in length, while it is less developed in female fowl and could be very small in size. The spur may need to be trimmed, especially in larger breeds, as it results in injury of the back of the female during mating. This is more obvious in the heavy breeds [11,12]. Chickens have four digits, or toes, numbering 1,2,3 and 4 medial to lateral, with 2,3,4 and 5 phalanges respectively.

The comb and other ornamental structures differ among different breeds of poultry and they are usually well developed in males (see Figures 7.4 and 7.10). These structures are routinely used to detect the general health condition of chickens. The process of

comb removal is called dubbing and if done is usually performed on day-old-chicks to avoid trauma from cannibalism or frostbite in cold climates. After caponization (castration) the comb shrinks in size, as its development is dependent on the androgen hormone level.

The ear lobes (ear flaps) are extensions of the skin with modified underlying tissues (see Figure 7.4). The surface of the ear lobes are covered with a few cell layers of stratified squamous epithelium with little keratinization.

Double folds of skin extend downwards from the head region creating the wattles, which have two surfaces - medial and lateral (see Figure 7.20). There are no feathers on the wattle in an adult chicken; however, with advancing age the folds will be drawn downwards carrying medially short feathers with it.

The uropygial gland, also known as the oil, preen, rump, caudal, or perunctum gland, is associated with the skin of the dorsal aspect of the caudal rump region. The gland has two lobes separated by an interlobular septum, which continues with the gland capsule. The gland duct from each primary cavity is carried through the papilla to its tip where it opens to the outside as the
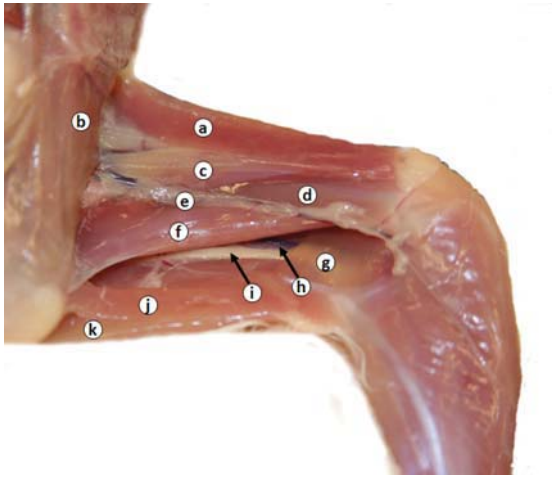
Figure 7.18 Medial aspect of the thigh of a chicken. (a) Cranial iliotibial (sartorius) m., (b) external abdominal oblique m., (c) quadriceps femoris/ambiens m., (d) internal femorotibial m., (e) circumflex femoral (femoral) artery, (f) adductor/ puboischiofemoral m., (g), accessory head of puboischiofemoral m., (h) ischiatic artery and vein, (i) Ischiatic nerve; (j) flexor crural medialis, (k) flexor crural lateralis.

Figure 7.20 External features showing the wing feathers on a four-week-old male chick. (a) Alulae; (b) primary feathers (I-X); (c) secondary feathers (XII-XVIII); (d) under cover for secondary feathers; (e) under wing feathers; (f) chest feathers.

Figure 7.19 Right metatarsal and digits of adult chicken. (a) claw dorsal plate; (b) claw ventral plate; (c) metatarsal fold; (d) metatarsal pad; (e) metatarsal scutes; (f) carpal scutes; (g) claw; (h) digital pad; D. spur; D1–4 digits.

uropygial duct. The gland often elevates the dorsal skin of the tail to produce the uropygial eminence.

The body of the chicken is covered with feathers with the exception of a few regions, depending on the species and breed (Figure 7.20). The main type of feather covering the body are called contour feathers or pinnae. In general, the feathers can be described as consisting of a shaft (quill) and the vane [13,14]. The shaft is composed of two portions, the calamus (proximal portion implanted in the skin) and the long solid segment above it called the rachis. On each side of the shaft, barbs and barbules are attached to form the vane (Figure 7.21).

Digestive system

The digestive system of the chicken consists of the beak, mouth, tongue, esophagus, crop, proventriculus, ventriculus, small intestine, and large intestine. The proctodeum is a portion of the cloaca that leads to the vent and is considered a continuation, or the end point, of the digestive system[15].

The *beak* is a highly keratinized structure covering the jaws (Figure 7.22). It is also known as the ramphotheca. It is pointed at its tip and composed of upper (superior, maxillary) and lower (inferior, mandibular) portions that meet at the angle of the mouth. The mandible forms the lower part of the beak. The base of the beak is sharply convex in the upper portion where it forms the operculum. The operculum covers the external nares in one-day-old chicks. The keratinized region of the beak is supported caudally by the jawbones. The outer dorsal surface of the beak is keratinized and the midline is called the culmen. The cutting edges of the beak are called tomia (singular tomium). When the mouth is closed, the two edges of the beak do not come
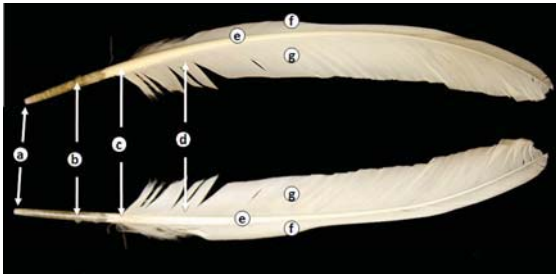
Figure 7.21 Adult male primary feather (remix). (a) Inferior umbilicus; (b) remnant of feather sheath; (c) calamus (quill); (d) barbs; (e) rachis; (f) anterior barbs; (g) posterior barbs.
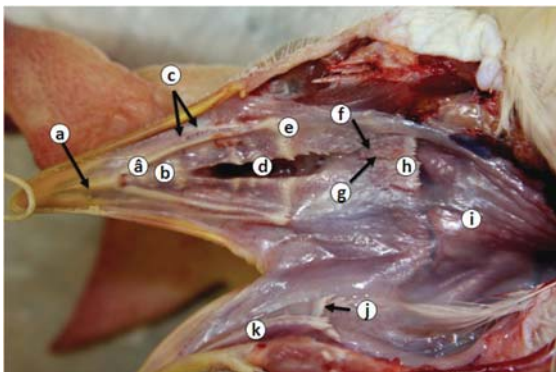
Figure 7.22 Roof of the mouth cavity of adult chicken. (a) median swelling; ā. Lateral palatine ridge; (b) palate; (c) opening of lateral palatine glands; (d) palatine (choanal) cleft; (e) Papillae; (f) medial palatine glands; (g) infundibular cleft; (h) pharyngeal papillae; (i) pharynx; (j) lingual papillae; (k) tongue.

together as the lower edge glides inside the edges of the upper beak.

A small protrusion (egg tooth) is found in one-day-old chicks at the surface of the upper beak. It is used to break the shell of the egg during hatching and is usually shed soon after. The process of cutting the beak is called de-beaking, and if done, is preferably performed on one-day-old chicks, as the beak is a highly innervated structure. One third of the distance from the tip of the beak is usually cut to prevent bleeding. De-beaking is routinely exercised in laying breeds to prevent cannibalism and picking of each other's feathers, especially under crowded conditions and if mineral deficiencies are present.

In the *mouth* (oral) cavity, the hard palate is incomplete and communication between oral and nasal cavities exists through a slit-like midline opening, the choana (see Figure 7.22). The surface of the hard palate has

several visible ridges with caudally oriented papillae. The cheeks are very much reduced and the floor of the mouth cavity has the median fold of the mucosal membrane connected to the free portion of the tongue and is called the lingual frenulum.

There are large numbers of salivary glands present in the roof and floor of the oral cavity, body of the tongue and pharyngeal wall. They usually open by several ducts into the oral and pharyngeal cavities (see Figure 7.22).

The *tongue* (glossa, lingua) is a pointed organ in the chicken and has a triangular shape in a cross section, adapting to the space of the lower beak where it lies. The tongue has a hyaline cartilage inside its rostral portion. This cartilage is an extension of the entoglossal portion of the hyoid apparatus that can be found in the caudal lingual segment or root. The tongue is covered by extremely thick stratified squamous epithelium on the dorsal surface and a much thinner and highly keratinized type of epithelium on the ventral surface.

The *pharynx* is a thickened muscular tube that connects the oral cavity to the digestive and respiratory systems. In the dorsal aspect of the pharynx the opening of the infundibular slit (Eustachian or auditory tubes openings) is present. There are rough caudally directed papillae similar to those described in the oral cavity. The caudal portion of the pharynx is connected to the esophagus. The floor of the pharynx is formed by the root of the tongue. The area of the junction between the pharynx and the esophagus is lined by a thick highly keratinized stratified squamous epithelium with papillae. Keratinization of the epithelium decreases, as well as the amount of glands, as the esophagus is approached. A characteristic smooth muscle layer of the lamina muscularis mucosae starts to appear at this junction, where lymphoid follicles or tonsils are also present.

The *esophagus* is a muscular tube connecting the pharynx to the stomach. It has two distinct parts, the cervical and the thoracic part. The cervical part connects the pharynx to the crop, while the thoracic portion connects the crop to the proventriculus [16]. The crop is relatively large in diameter and researchers consider it an embryonic dilation of the esophagus before it enters the thoracic cavity.

The cervical esophagus is shorter than the vertebral column of the neck. It starts dorsal to the larynx and trachea and, caudal to the fifth cervical vertebra, it lies on the right side of the neck. Close to the entrance into the thoracic cavity, the esophagus returns to the midline and enlarges ventrally to form the crop. The thoracic esophagus is much shorter than the cervical part, and extends dorsal to the trachea along the base of the heart.
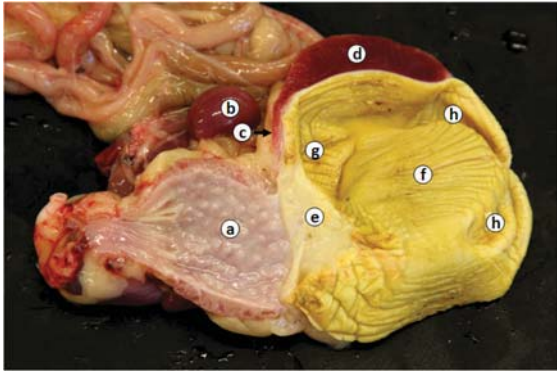
Figure 7.23 Chicken stomach interior structures. (a) proventriculus; (b) spleen; (c) thin muscle; (d) thick muscle; (e) intermediate zone (isthmus); (f) body (fundus); (g) cranial blind sac; (h) caudal blind sac.

The most caudal portion of the esophagus is reduced in diameter.

The crop (ingluvies) is a large thin-walled diverticulum, which can store food for a short period of time. It differs in shape and size among different breeds of poultry. In chicken, it is displaced towards the right side of the median plane, in front of the furcula, on the pectoral muscles. The crop is only covered by the skin, and can be palpated easily when it is full [17]. On the dorsal wall surface of the crop, there is a cleft or a channel (crop channel) and easily digestible food may pass through it directly to the proventriculus.

The crop is lined by stratified squamous epithelium, which contains mucus glands at the crop channel only. The so-called "crop milk" is produced by these glands.

The proventriculus (glandular stomach) is connected to the esophagus orally and to the muscular stomach (ventriculus, gizzard) aborally (Figure 7.23). It is a fusiform structure and has no separating point or clear sphincter with the esophagus. When the proventriculus is cut open, large numbers of papillae (openings of the glands) appear on the surface. The proventriculus has a typical tubular organ layer arrangement [16,17]. The mucosa is represented by folds of simple columnar epithelium. Loose connective tissue with lymphatic cells is present in the lamina propria. In several areas, these cells form well-defined lymphatic nodules extending in a few places into the tunica submucosa. Thin scattered layers of ill-defined lamina muscularis mucosa are present. Large compound tubular glands occupy the tunica mucosa almost completely [18,19] (Figure 7.24).

The intermediate zone is the area of connection between the proventriculus and the ventriculus (gizzard). This segment is relatively short and has the
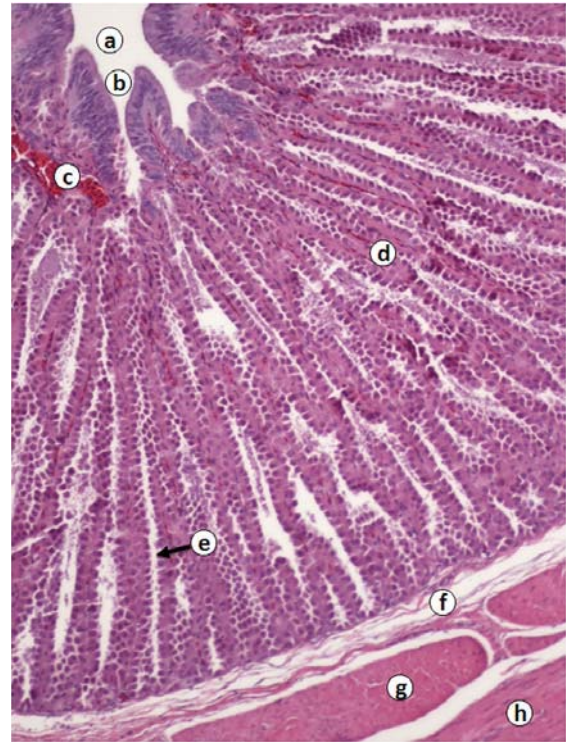
Figure 7.24 Cross section of proventriculus of an adult chicken. (a) Lumen of the gland; (b) simple columnar epithelium; (c) blood capillaries in the lamina propria; (d) body of the gland; (e) lumen of individual gland; (f) lamina propria; (g) muscularis mucosa; (h) inner muscular layer. Stained with H&E Mag.120 X.

characteristic features of the wall of the ventriculus in an unorganized way.

The ventriculus (muscular stomach, gizzard) is a muscular flattened structure connecting the proventriculus to the duodenum. It is composed of four definitive muscular compartments separated by constrictions and connected in the middle by a thick aponeurotic region. These compartments are named cranial dorsal, cranial ventral, caudal dorsal, and caudal ventral. The inner surface of the ventriculus is covered by a thick keratinized yellowish layer, which at necropsy or at butchering, can be stripped easily from the surface, as it is the secretion of the glands. The yellowish coloration results from the antiperistaltic movements of the small intestine, which brings contents from the initial portion of the duodenum. The epithelium has a thick layer on the surface called koilin that some authors call the horny layer of the stomach.

The *duodenum* is the first segment of the small intestine and it starts at the ventriculus and ends at the jejunum.
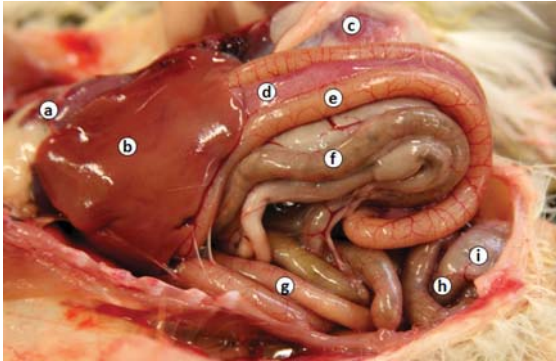
Figure 7.25 Visceral organs of adult chicken in situ. (a) heart; (b) liver; (c) gizzard (ventriculus); (d) pancreas; (e) duodenum; (f) cecum; (g) jejunum; (h) rectum (colorectum); (i) cloaca.

Figure 7.26 Digestive system of adult chicken, removed from the body to show different segments starting at the proventriculus. (a) proventriculus; (b) intermediate zone; (c) gizzard (ventriculus); (d) duodenum; (e) pancreas; (f) jejunum; (g) ileum; (h) cecum; (i) colorectum; (j) cloaca.

It forms a long loop of descending and ascending portions. These two portions are connected by a fold of peritoneum (inter-duodenal ligament) (Figure 7.25). The pancreas is lodged between these two portions and extends the entire length of the duodenal loop. The site of termination of the descending duodenum and the beginning of the jejunum is considered the area of opening of the pancreatic and bile ducts.

The *jejunum* forms loose coils and has a long mesentery (Figure 7.26). The Meckel's diverticulum (vitelline diverticulum) is a short blind remnant of the embryonic yolk sac and yolk stalk that is located on the surface of the jejunum (Figure 7.27).

The *ileum* is relatively short and it is the last segment of the small intestine. It is lodged between the two ceca

Figure 7.27 The Meckel's diverticulum (vitelline diverticulum) is a short blind remnant of the embryonic yolk sac and yolk stalk that is located on the surface of the jejunum.

and attached by a fold of peritoneum called the ileo-cecal ligament. The ileum has a relatively thicker wall when compared to the duodenum or jejunum.

The chicken has a pair of blind end sacs or ducts called ceca (see Figure 7.25). They are smaller in diameter at their origin and wider as they ascend into the blind end. The ceca are lined by highly folded simple columnar villated epithelium with relatively large numbers of goblet cells. Simple branched tubular glands extend down into the lamina propria.

The *colon* (colorectum) of the chicken is well delineated as it starts from the ileocecal junction and transitions to the *rectum*; therefore it is also known as the colorectum (Figure 7.28). It is connected distally to the cloaca. Generally, it is a short and straight tubular structure. The internal surface of the colon appears folded with villi and lined by simple columnar epithelium with large numbers of goblet cells.

The *cloaca* is the dilated end of the digestive and urogenital systems. It is composed of three compartments: The first (coprodeum) connects to the colorectum while the second (urodeum) is associated with the ureters and the genital system. The third compartment is the proctodeum which opens to the outside through the vent. A well-developed fold of mucous membrane separating the coprodeum from the urodeum is usually seen (coprourodeal fold). An incomplete circular fold separating the proctodeum from the urodeum is called proctourodeal fold. The inner surface of the cloaca is thrown into folds and lined by simple columnar epithelium with goblet cells.

The external caudal opening of the cloaca is the vent, which has dorsal and ventral labii and a labial
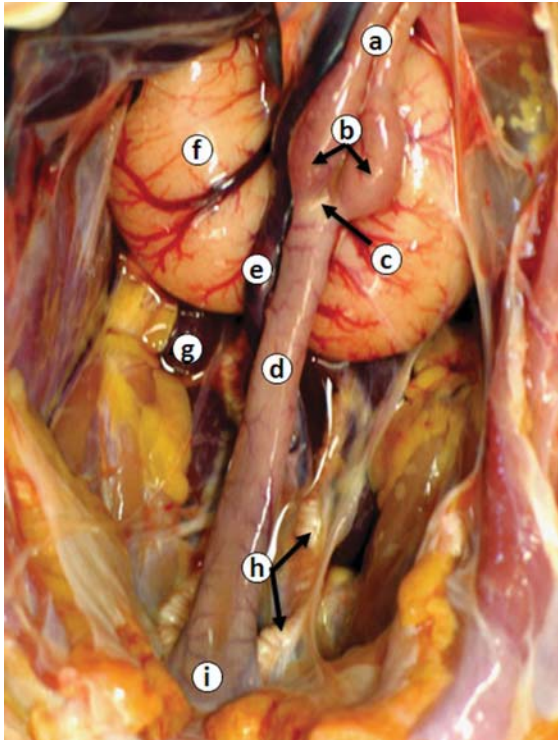
Figure 7.28 Dissected rooster showing testes and relationship to the distal segments of the intestinal tract. (a) Ceca; (b) cecal tonsils; (c) ileocecocolic junction; (d) colorectum; (e) common mesenteric vein; (f) testis; (g) kidney; (h) ductus deferens; (i) cloaca.

cleft between the two. An abrupt change in the type of epithelium is observed at the junction of the distal portion of the cloaca and the vent. The keratinization as well as the thickness of the stratified squamous epithelium decreases as it moves away from the vent to join the skin. In this area, it is composed of one to two cell layers with thin keratin covering. Herbst's corpuscles are present in this area and close to the feather follicles [19,20].

Liver (hepar, jecur)

The color of the liver is yellow in the one-day-old chick as it has pigments from the yolk lipids at the late stage of incubation. On the other hand, the liver in adult chickens varies in color depending on the nutritional status, general health condition and method of sacrifice [7]. The normal color is reddish brown, light brown, to yellow. The liver can be easily seen when the abdominal wall is cut open (see Figure 7.25). The liver is divided into two lobes with the right lobe larger than the left. The left lobe is clearly divided into two lobes (dorsal and ventral).

The caudal vena cava passes through the cranial region of the right lobe close to its dorsal edge. The fusiform gall bladder lies on the visceral surface of the right lobe. Each liver lobe is drained by a bile duct. The so-called hepatocystic duct drains bile from the right lobe to the gallbladder while the common hepatoenteric duct drains bile from both lobes to the duodenum [21,22]. Histologically, the liver lobules are not well delineated and each lobule is composed of hepatic cords arranged around a central vein. These cords are composed of double hepatocytes while other histological characteristics are similar to those found in mammals [18,19].

The hepatic portal vein is the functional circulation of the liver. It collects blood from the gastrointestinal tract with the exception of the caudal portion of the cloaca, the pancreas, the spleen, and the air sacs. The hepatic portal vein divides to enter the right and left lobes of the liver.

Pancreas

The pancreas is situated inside the loop of the duodenum, inside a fold of peritoneum called the duodenopancreatic fold. Dorsal and ventral lobes, as well as a small segment rich in islets of Langerhans lying close to the spleen known as the splenic lobe, can be identified. This latter lobe is very thin and embedded in the adipose tissue and may be difficult to visualize but can be identified microscopically. The pancreas has three ducts: two from the ventral and one from the dorsal lobe, with the splenic lobe having no separate excretory duct. The pancreatic and bile ducts open into the ascending part of the duodenum opposite the cranial part of the muscular stomach. The exocrine portion is composed of compound tubuloalveolar glands. The lobulations of the gland are not as clear as in mammals because of the low quantity of connective tissue. However, the lobation is obvious externally.

The endocrine portion of the pancreas is composed of islets of Langerhans. These islets are scattered circular structures in between the exocrine portion of different lobes. They are usually surrounded by a thin layer of connective tissue. The cells inside the islets are arranged in the form of branching cords separated by sinusoidal capillaries. Researchers describe two types of islets, alpha and beta. Alpha islets are larger and usually present inside the splenic lobe and at the site of the junction of the ventral and dorsal lobes. These islets are also called the dark islets because they stain with argyntaffine (argyrophilic staining) and are associated with glucagon production. These islets have alpha and delta cells. Beta islets are scattered randomly in all pancreatic lobes. They are smaller than alpha islets

and contain beta cells and a few delta cells. Beta islets are also called light islets because they do not take the argyrophilic stain and are associated with insulin production [23].

Respiratory system

The respiratory system consists of the nasal cavity, upper larynx (glottis), trachea, lower larynx (syrinx), lungs, and air sacs.

The nasal cavity has three conchae (rostral, middle, and caudal) and it opens posteriorly through the choanae to the pharynx. The choanae appear as a single slit in the roof of the mouth. The upper larynx (cranial larynx, glottis) is composed of cricoid and arytenoid cartilages only. It connects the pharynx to the trachea. The trachea is a long flexible tube made up of complete articulating cartilaginous rings (signet-shape) connected cranially with the upper larynx and distally with the lower larynx (syrinx). These rings tend to become ossified with age, especially at the distal part close to the syrinx [24,25]. They have narrow and wide portions to fit with adjacent rings to form a continuous tube of overlapping rings. No true trachealis muscle is present in the chicken but two well-developed skeletal muscles ascend on both sides of the trachea. Each tracheal ring has a thickened segment at the periphery and is extremely thin at both the dorsal and the ventral sides.

The syrinx is the organ of phonation in some species of birds and is considered a second larynx [24,26]. It is situated at the distal end of the trachea and at the beginning of the lungs. The syrinx is composed of several modified tracheal cartilages fused with membranes and muscles. The wedge-shaped cartilage is covered by a semilunar membrane, which extends distally and is called the inner tympanic membrane. The external tympanic membrane connects the middle group of cartilages to the caudal group.

The lungs (pulmo) of birds are located in the uppermost dorsal part of the coelomic cavity. They are pink in color and relatively small in size [26]. The lung is not lobated like other species, with the ribs deeply embedded in it (Figure 7.29). The primary bronchi bifurcate into secondary bronchi, and then these will divide to become the tertiary bronchi (para-bronchi) [26]. The smallest, terminal portion, known as air capillaries, are much smaller than mammalian alveoli.

The presence of air sacs in poultry is another unique characteristic feature that is not present in mammals [25]. Air sacs are connected with the lungs, where they develop at early stages of embryonic life. The air sacs are characterized by being easily expandable and having
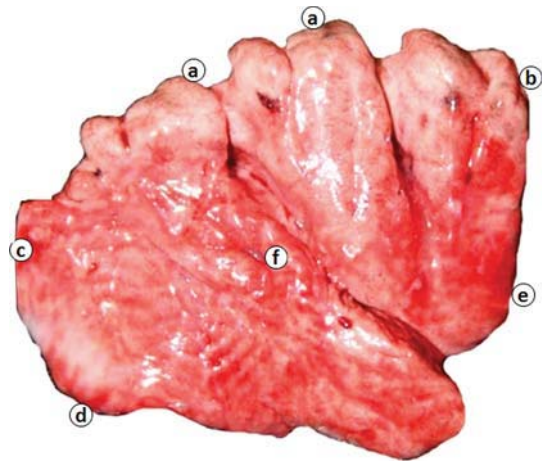
Figure 7.29 Lung-left dorsal view. (a) Costovertebral border; (b) Craniodorsal angle; (c) Caudodorsal angle; (d) Caudoventral angle; (e) Cranioventral angle; (f) Costal surface.

thin transparent walls. The outermost layer of the air sacs are covered by a serous membrane (simple squamous epithelium). The air sacs are poorly vascularized structures; therefore they are not involved in gaseous exchange. They perform other functions like lessen the weight of the body to facilitate flying, body temperature regulation, distribution of weight and balancing during flying, in addition to their role in phonation and air storage. They include the clavicular (unpaired sac), cervical, cranial thoracic, caudal thoracic, and abdominal (paired sacs).

Urinary system

The urinary system is composed of paired kidneys and paired ureters. The kidneys are symmetrically positioned occupying the depression inside the synsacrum (renal fossae)(Figure 7.30). Each kidney is divided into three distinct divisions: the cranial, middle, and caudal divisions, which are separated by the passage of the external iliac and the ischiatic arteries respectively [27].

On section, the kidney has a cortex and medulla. Three types of nephrons have been described, cortical with a relatively small glomerulus (mammalian type), medullary with large glomerulus (reptilian type), and intermediate (infrequently found). Microscopically, distinct lobulations inside the cortical region are seen following the branching of the duct system (Figure 7.31). The epithelial lining of the proximal tubule is simple cuboidal that can become pyramidal in shape. The lumen of the tubule is not always clear as a result of the presence of the brush border on
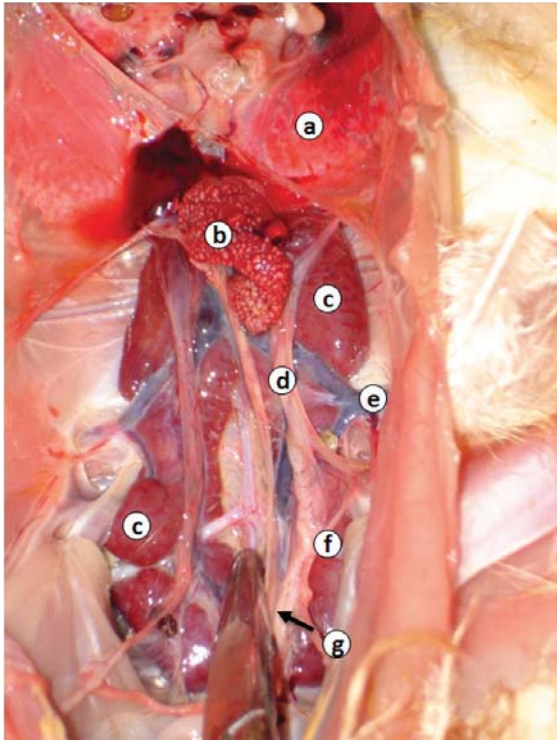
Figure 7.30 Dorsal view of the celomic cavity of young hen after removal of the gastrointestinal tract. (a) lung; (b) ovary; (c) cranial and middle lobes of the kidney; (d) left oviduct; (e) external iliac vein; (f) Internal iliac artery; (g) Uterus (left oviduct).

Figure 7.31 Micrograph of the Kidney of an adult chicken. (a) capsule; (b) cortex; (c) medulla; (d) renal corpuscles; (e) renal tubules. H&E stain Mag.150 X.

the surface. The distal tubule is lined by lower simple cuboidal epithelium with larger lumen and darker cytoplasm (basophilic).

The collecting duct system starts at the periphery of the cortex and passes through it towards the medulla. These ducts increase in diameter progressively until they terminate collectively as a series of large ducts ending in the ureter [23].

Each kidney is drained by a ureter, which passes caudally to open at the urodeum of the cloaca. The ureter can be divided into two portions, intrarenal and extrarenal. The ureter is a well-developed muscular duct, which is described to receive about 13–17 large ducts from the kidney. Histologically, the ureter is lined by transitional or pseudostratified columnar epithelium, having variable thickness from one region to another [28].

Renal portal system

Blood from the rectum, pelvis, and hind limbs is carried to the kidney through the caudal mesenteric, ischiatic, and external iliac veins. The blood enters a venous ring lying on the ventral surface of the kidneys. The venous ring has connections with the common iliac vein to join the caudal vena cava. Branches of the vein enter the kidney and form the peripheral interlobular veins, which discharge into the capillary network surrounding the nephrons. The flow of blood inside the caudal mesenteric vein is usually toward the kidneys.

Blood in the portal (renal) venous ring normally flows from the kidneys cranially into the caudal vena cava via the common iliac veins (under sympathetic influence). Alternatively, under parasympathetic influence, the flow can be diverted into the hepatic portal circulation via the caudal mesenteric vein. Within the lumen of the common iliac vein is the renal portal valve, which when open allows portal blood to enter the caudal vena cava (Figure 7.32). The renal portal system enhances renal tubular secretion and reabsorption and is especially important in the secretion of urates. The renal lobules drain to the central intralobular veins, the branches of which eventually discharge into the caudal and cranial renal veins [20,29].

Ideally parenteral medication administration should be confined to the cranial half of the body (pectoral muscles for intramuscular injections) just in case the renal portal system is causing blood to flow directly through the kidney to avoid potential renal damage from renal toxic medications.

Male genital system

The male genital system consists of paired testes, epididymis, ductus deferens, and a phallus.
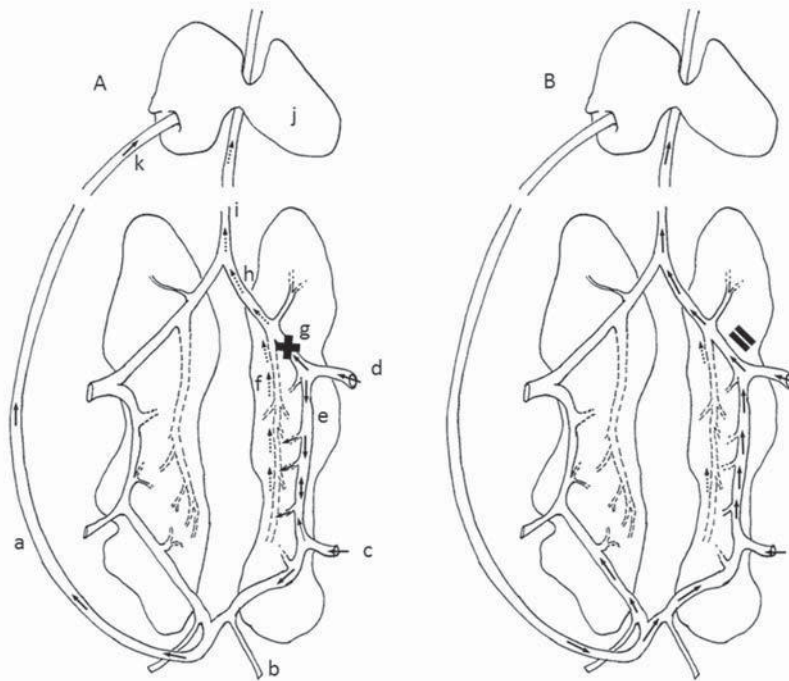
Figure 7.32 Drawing depicting the renal portal system blood flow under two different autonomic nervous system stimuli. A: under parasympathetic control, the valve (g) is closed (**X**) and the blood is diverted towards the kidneys as well as the caudal mesenteric vein to reach the hepatic portal vein. Branches of the caudal portal renal veins enter the kidney and form the peripheral interlobular veins, which discharge into the capillary network surrounding the nephrons. After being involved in renal tubular secretion and reabsorption, these vessels end up into the caudal renal veins that join the common iliac veins. B: under sympathetic control, the valve (g) is open (**II**) and the blood coming from the caudal mesenteric, ischiatic and external iliac veins flow towards the caudal vena cava via the common iliac veins.
A: blood flow under parasympathetic control; B: blood flow under sympathetic control; a: caudal mesenteric vein; b: internal iliac vein; c: ischiatic vein; d: external iliac vein; e: caudal portal renal vein; f: caudal renal vein; g: renal portal valve; h: common iliac vein; i: caudal vena cava; j: liver; k: hepatic portal vein.

The two testes (testicle, orchis) are situated symmetrically in the dorsal portion of the coelomic cavity between the lungs and the cranial lobe of the kidneys [20,29]. Each testis is bean-shaped and whitish yellow in color in the adult (Figure 7.33). The testes are covered by the abdominal air sacs, especially at their cranial portions. The seminiferous tubules are separated by extremely thin connective tissue, which contains some interstitial cells (Leydig, testosterone producing) and connective tissue.

The epididymis is much less convoluted compared to mammals. There are no clear demarcations between different segments of the epididymis as the efferent ductules open over all of its length.

The ductus deferens (vas deferens) has a small lumen, is white in color and relatively difficult to differentiate from the epididymis in its initial segment. Histologically it has a thick muscular wall. It courses towards the cloaca, lying next to the synsacrum and to the kidney. It ends at a small elevated papilla inside the urodeum. There are no accessory sex glands in chicken.

The phallus is the male copulatory organ and it is a non-protrusible intromittent structure. It has a spiral phallic sulcus where the ejaculate passes through. The phallus can be found on the ventral lip of the vent. It consists of a median white phallic body (few millimeters in diameter) and two lateral phallic bodies (2–4 mm) surrounded by two phallic folds.

Female genital system

The female chicken has a single left ovary because the right ovary is not fully developed and regresses early
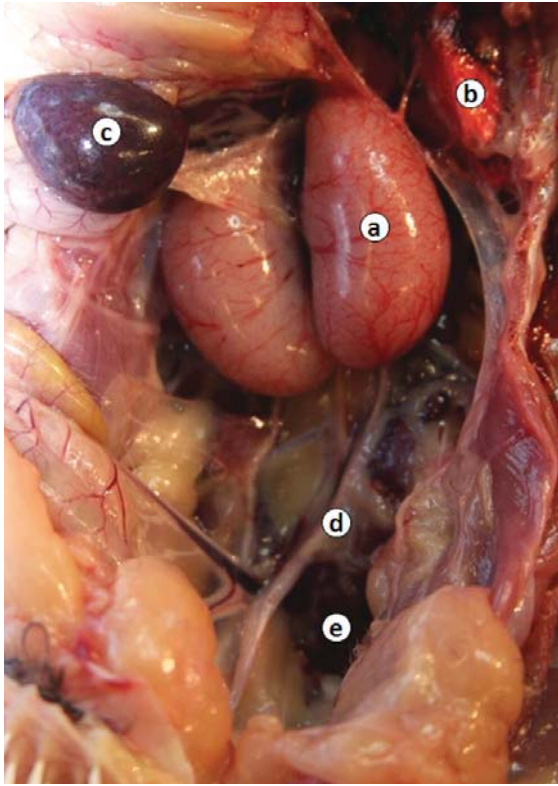
Figure 7.33 Exposed male genital system in a rooster after removal of the abdominal viscera. (a) testis; (b) lung; (c) spleen; (d) ductus deferens; (e) kidney.

Figure 7.34 Female genital system of adult hen. (a) fimbria; (b) infundibulum; (c) mesotubarium; (d) ampulla; (e) developing ova; (f) stigma; (g) spleen.
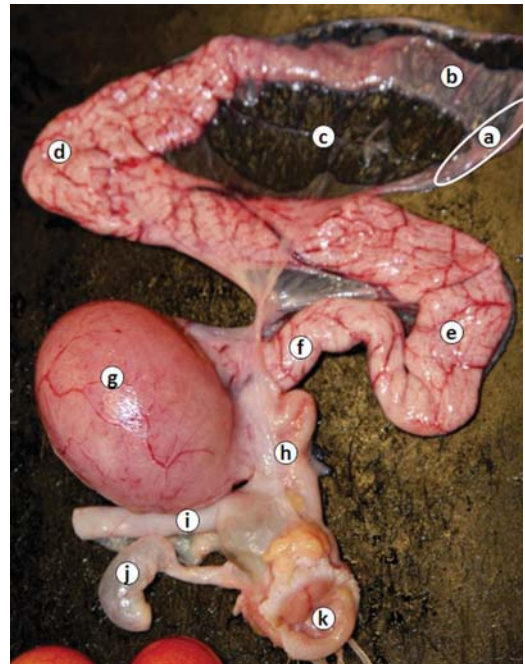
Figure 7.35 Female genital system of adult hen with an egg inside the uterus. (a) fimbria; (b) infundibulum; (c) mesotubarium; (d-e) ampulla; (f) isthmus; (g) uterus (egg shell gland); (h) vagina; (i) colorectum; (j) reminent of atretic (cystic)right oviduct; (k) cloaca.

during development. The remnant of the right genital organ in the adult hen is called the regressed right cystic oviduct (Figure 7.34 and Figure 7.35). The left ovary is found within the peritoneal (coelomic) cavity attached to the dorsal wall by the mesovarium. It can be described as active when it is composed of a number of follicles (yolks) at different stages of development and inactive when the follicles (yolks) are extremely small, white in color, and transparent. There is no corpus luteum described in the chicken ovary at any stage [20,29].

The oviduct is a muscular tube that conveys the ovum from the ovary to the cloaca (urodeum). It is attached to the body wall by a mesentery called the mesotubarium. The oviduct is composed of five segments:

1. *Infundibulum* The infundibulum length is close to nine centimeters in laying hens and is much shorter in non-laying hens. The wall is thin and the shape is like a funnel which collects the ova after being released from the ovary. The infundibulum is covered by ciliated low columnar epithelium with few goblet cells.

2. *Magnum* The magnum participates in the formation of the thin and thick albumin on the developing egg. The ova descend spirally inside the magnum. The spiral chalaza is formed in this segment. The surface

epithelium is ciliated simple columnar with large numbers of goblet cells. Well-developed branched tubular glands fill the propria-submucosa.

3. *Isthmus*   The isthmus is a short segment of the oviduct (about 8 cm). The boundary between the isthmus and magnum is sharply distinguished by a narrow band of tissue called zona translucent. The membranous isthmus forms the internal and external shells of the egg.

4. *Uterus (egg shell gland)*   The uterus functions in laying the shell on the outer shell membrane. It also works to deposit the pigment in certain species which produce colored shells. There is no distinct anatomical boundary between the isthmus and the uterus. The cranial part is short through which the egg passes rapidly. The major part is pouch-like and holds the egg during shell formation.

5. *Vagina*   The vagina is a short muscular tube, which extends from the sphincter of the uterus to the cloaca. It has a thick wall as a result of the presence of a thick muscular layer. The mucosa is composed of primary and secondary folds.

Central nervous system

The central nervous system is composed of brain and spinal cord surrounded by the meninges to protect and nourish. As in mammals, the meninges consist of the dura matter (outer), arachnoid and pia mater (inner) [30,31].

The brain can be divided into cerebrum, cerebellum, and the midbrain. The cerebrum is composed of two cerebral hemispheres separated by a median (longitudinal) groove or fissure. A small pointed olfactory bulb projects from the rostral end of the cerebrum (Figure 7.36). Caudally, the hemispheres extend to contact the well-developed optic lobes in bird. The cerebrum has the lateral ventricles inside the hemispheres and the third ventricle around the thalamus. The fourth ventricle is found in the hindbrain. The choroid plexuses produce and regulate the amount of the cerebrospinal fluid. These ventricles are connected with the central canal of the spinal cord. The outer most portion of the cerebrum is covered by the cortex. The cortex is composed of one or two layers of cells (neurons). Overall, the cortex is divided into three functional regions: The limbic cortex, general cortex, and true olfactory cortex.

The paired optic lobes comprise a relatively large proportion of the mesencephalon, projecting laterally on either side of the brain from the posterior ventral aspect of the forebrain. Within each optic lobe the greater part
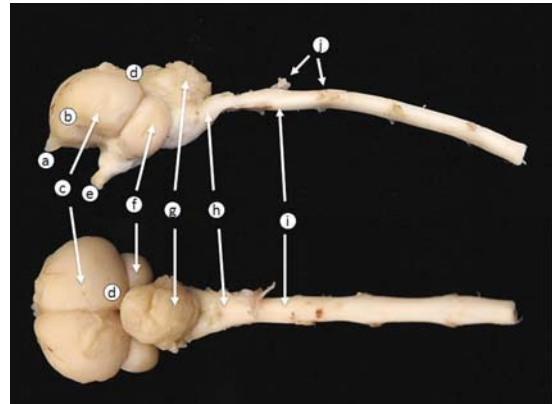
Figure 7.36   Central nervous system of adult chicken. (a) olfactory bulb; (b) cerebrum/frontal part; (c) cerebrum/parietal part; (d) occipital part (e) optic tract; (f) optic lobe (mesencephalon); (g) cerebellum; (h) medulla oblongata; (i) spinal cord; (j) cervical spinal nerves.

of the structure consists of a well-developed optic tectum or rostral colliculus.

The cerebellum attaches to the dorsal aspect of the medulla oblongata by rostral and caudal peduncles. The cerebellum can be divided into three main lobes, rostral, middle, and caudal, as a result of the presence of two fissures known as the fissura prima and fissura secunda (first and second fissures). Externally, the vermis can be observed with deep transverse sulci.

The pyramids and the decussation of the mammalian medulla are not seen in the chicken medulla oblongata. There is no obvious pons. Histologically, the arrangement of the nuclei of the cranial nerves inside the medulla shows similarity to the gray matter of the spinal cord.

The spinal cord extends along the spinal canal including the coccygeal (caudal) region; however, it decreases in diameter as it descends caudally. The conspicuous difference is the presence of the glycogen body, which is an elongated dilation at the lumbar region.

Endocrine system

The endocrine system includes glands and scattered glandular tissues [32]. These glands are:

1. Pituitary gland
2. Pineal gland
3. Thyroid glands
4. Parathyroid glands
5. Ultimobranchial gland (body)
6. Adrenal glands

Included within the endocrine system are the endocrine cells inside the pancreas (Islets of Langerhans), cells in the wall of the gastro-intestinal tract,

hypothalamus, testis (interstitial, Leydig cells), and ovary (thecal cells).

Pituitary gland (Hypophysis cerebri)

The pituitary is a small gland attached directly to ventral aspect of the brain stem, caudal to the optic chiasma. Histologically, there is no intermediate zone or region in the chicken pituitary gland. The glandular portion has two regions: Distal and tuberal; while the nervous portion divides into infundibular, median eminence, and nervous parts. The glandular portion has six types of cells with different staining affinity. Their function or secretions are similar to those of mammals.

Pineal gland (Epiphysis cerebri)

The pineal gland is a small bean-shaped structure that arises from the roof of the diencephalon. It is situated in the mid-line between the two hemispheres of the brain at the junction of the cerebrum with the cerebellum. It consists of solid group of cells subdivided into lobules by connective tissue. It is the neuroendocrine gland that secretes melatonin.

Thyroid gland

The thyroid gland is a paired gland, dark red in color, and situated at the vascular angle formed by the subclavian and common carotid arteries. The gland is oval-shaped in the chicken, but the size may differ according to the season, sexual hormones, nutrition, and age. The thyroid gland is enclosed in a thin capsule of collagen bundles with numerous elastic fibers. The gland parenchyma consists of roughly spherical follicles with little interstitial connective tissue between them (Figure 7.37). Each follicle is lined by a single layer of cells with different heights depending on the gland activity. Follicles from very active glands, as in young growing chickens, are lined by low columnar to cuboidal cells. Active follicles contain small amounts of colloid. In relatively quiescent glands, as in the normal laying hen, the follicles are large as a result of accumulation of colloid and the lining epithelium is reduced in height, which may reach flattened or almost squamous in appearance. No parafollicular cells are seen within the thyroid gland of chickens.

Parathyroid glands

The parathyroid glands are normally found caudal to the thyroid glands inside the thoracic part of the coelomic cavity. Accessory parathyroid tissue is frequently encountered in different locations. Nodules of functioning parathyroid cells are found within nearly all ultimobranchial bodies. As many as three nodules
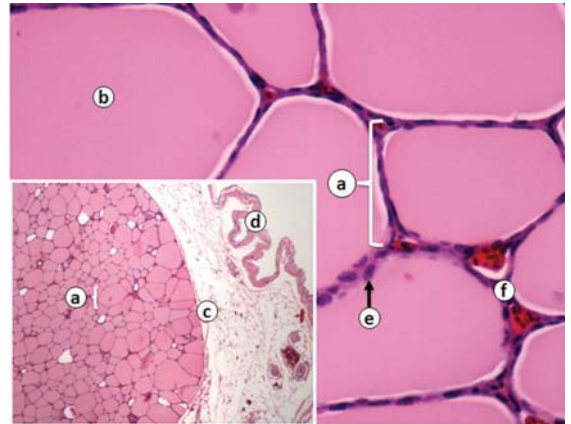
Figure 7.37 Thyroid gland of adult chicken. (a) thyroid follicle; (b) colloid; (c) thin connective tissue capsule; (d) lining epithelium of thoracic air sac; (e) follicular epithelium; (f) blood capillaries. H&E stain, Mag. 120 X, insert 80 X.

have been described in the literature. Parathormone secreted by the gland regulates calcium and phosphate metabolism.

Ultimobranchial glands (bodies)

The ultimobranchial bodies (postbranchial) arise as an L-shaped sac from the caudoventral face of the fourth pharyngeal pouch [33]. These bodies migrated with the thyroid to the entrance of the coelomic cavity in birds. The ultimobranchial bodies frequently enclose parathyroid tissue within them, and consist of strands of principal cells similar to mammalian C cells, as well as follicular structures formed by distinct endocrine cell types with larger granules. In mammals they become incorporated into the thyroid gland and differentiate into parafollicular cells (C cells) that secrete thyrocalcitonin hormone. This hormone acts to reduce concentrations of calcium in the blood.

Adrenal glands

The adrenal glands are situated on both sides of the abdominal (caudal part) aorta, close to the cranial end of the kidney. In the hen, the left gland is normally embedded within the ovarian stalk, and in the cock, the adrenals are closely associated with the anterior end of the epididymis [32]. The gland is usually yellow in color with the weight varying considerably according to breed, age, health and various environmental factors. There is no clear separation of cortex and medulla. The cortex is formed by columnar cells aligned in chords intermingled with the medullary cells which are

arranged as clumps of irregular masses forming a mesh-work. These cells are polygonal in shape and larger than the cortical cells. Cells have basophilic cytoplasm and a large, spherical, centrally located nucleus with diffuse chromatin. Secretions of both cortical and medullary tissues are similar to those in mammals.

Cardiovascular system (heart, cor)

The heart of chickens is composed of four typical chambers and is relatively large in size. It is dark red to bluish red in color and with a conical outline. It is surrounded by the pericardium [34]. The base of the heart is situated at the level of the second rib, while the apex is directed towards the sternum to reach the intercostal space between the fifth and the sixth ribs. The myocardium is relatively thick because of its high activity. The outermost layer of the pericardium is connected by a fibrous tissue to the hepatoperitoneal sac and the air sac in this region. It is further connected to the horizontal and oblique septum in addition to the sternum.

Lymphatic system

The normal lymphoid organs of the fowl are basically the spleen, thymus, bursa of Fabricius, and the mural lymphoid nodules. These small lymph nodules occur irregularly throughout the main lymphatic vessels of the neck, wing, and the hind limb. Apart from these organs, foci of lymphoid tissue of variable sizes are found in a large number of tissues and organs of the body.

Spleen

The spleen is spherical, dark-red to reddish brown in color, and lies on the dorsal right side of the junction between the proventriculus and the gizzard (Figure 7.28). Small accessory spleens are reported in the literature close to the celiac artery.

Thymus

The thymus extends from the pharyngeal region to the distal part of the neck, in young chicks. The gland is embedded inside the connective tissue under the skin close to the jugular vein. During its development, the thymus is divided into 6–8 lobes separated by connective tissue (see Figure 7.16). The thymus eventually regresses almost completely in the older bird.

Cloacal bursa (bursa of Fabricius)

The cloacal bursa is a unique structure to birds. It consists of a dorsal median diverticulum of the proctodeum. It is pear-shaped and it reaches its maximum size before the bird is fully mature [29,35]. In domestic fowl, this

size is attained at 4–12 weeks of age depending on the strain. The internal structure of the bursa consists of about 12 thick longitudinal folds and lymphoid tissues are found in each of these folds separated by collagen fibers (Figure 7.38). The internal lumen is lined by simple columnar epithelium. The cloacal bursa is the site of differentiation of immunologically competent bursal (B) lymphocytes. Involution of the bursa begins at about 2–3 months of age. However, remnants of the bursa persist for a relatively long time after involution.

Sensory organs
Eye

The three main tunics of the eye (fibrous, vascular, and nervous) are present in the chicken. The main differences from the mammalian eye will be described here. Between the anterior-most part of the eye (cornea) and the large globular posterior component (sclera) is an intermediate region occupied by the bony scleral ring [35–37]. The avian retina is avascular and relatively thick compared to that of mammals. Another characteristic feature of the chicken eye is the black trapezoid-shaped pecten oculi which projects from the linear optic disk into the vitreous body. For the purpose of lens and corneal accommodations the presence of striated ciliary muscles are described and sometimes they attach directly to the lens.

Ear

The pinna of the ear is absent and the entrance to the external acoustic meatus is 4–5 mm in diameter. It has glands and is covered externally by modified feathers.
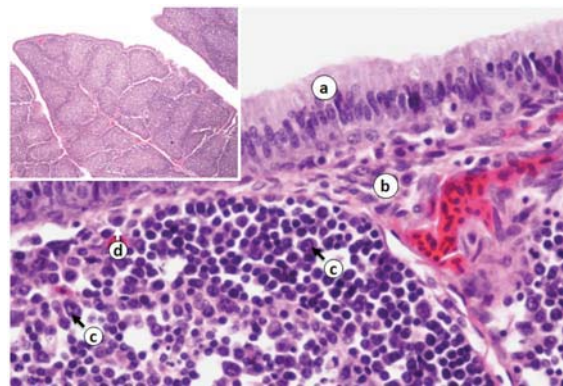
Figure 7.38 Cloacal Bursa (Bursa of Fabricius) of young chicken. (a) simple columnar epithelium; (b) lamina propria submucosa; (c) macrophage; (d) plasma cell; and darkly stained rounded nuclei represent lymphocytes predominate the bursa. H&E stain, Mag. 220 X, insert 100 X.

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CHAPTER 8

Biosecurity and Zoonotic Diseases

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Biosecurity

Biosecurity is an important part of any avian health management program. "Bio" means life and "security" implies "protection," so such a program is designed to protect life. In its simplest terms, it means keeping the infectious agents away from the poultry and keeping the poultry away from infectious agents and other hazards to health [1]. To minimize the occurrence and spread of disease, the following steps can be taken to reduce the interaction of poultry and infectious agents: i) a conscious examination of how infectious agents can be introduced to birds through humans, other poultry, food, water, infected equipment, and other animals such as pets and pests, and ii) implementation of a routine cleaning and disinfection program [1–3]. Minimizing the contact between poultry and infectious agents such as bacteria, viruses, fungi, and parasites can reduce the likelihood of a disease outbreak. Steps can also be taken to reduce the risk of disease and other health risks to humans and other animals, such as pets, that may encounter the poultry.

Informational resources are readily available for both poultry owners and veterinarians from a variety of sources. The United States Department of Agriculture Animal and Plant Health Inspection Service (USDA-APHIS) publishes a "Backyard Biosecurity 6 Ways to Prevent Poultry Diseases" poster and a "Backyard Biosecurity: Practices to Keep Your Birds Healthy" video, and provides a toll free help line (866-536-7593) [4,5] (Figure 8.1). Many agricultural college extension websites offer information on biosecurity and many aspects of care for the backyard flock [6–13].

Besides US governmental websites, other countries offer exceptional governmental websites with on-line manuals for poultry producers, backyard flock owners, and other domestic bird keepers [14,15]. Clear advantages of practicing biosecurity include having healthy birds, minimizing the potential for significant costs and loss of revenue, protecting human health, protecting future ability to move birds without restriction, protecting other industries such as feed suppliers, and protecting export markets. Table 8.1 provides some disease prevention tips that can be provided to backyard flock owners.

Methods used to reduce interactions between poultry and infectious agents

The following procedures can be used to reduce interactions between poultry and infectious agents, including minimizing human contact, establishing a visitor's policy, reducing exposure from contaminated food and water, reducing exposure from pests, and reducing exposure from new poultry introductions.

Minimizing human contact and establishing a visitor policy

In the commercial poultry industry, a visitor policy is necessary to restrict human access to poultry. Disease agents can be transmitted to poultry through fomites such as soiled soles of shoes, contaminated clothing and equipment, and even vehicles [1,14]. The rationale for this biosecurity measure is that visitors to commercial poultry farms have probably come in contact with other birds, and contaminated soil is a primary mode of transmission of many infectious agents. For backyard poultry flocks, people may be visiting a farm to purchase

Table 8.1 Tips for the backyard flock owner to prevent diseases

Recommendations	Recommendation rationale
1. Thorough cleaning and disinfection of the poultry house is an important factor in disease prevention.	This action keeps bacteria, viruses, fungi, and parasites from building up to levels that may cause disease outbreaks
2. It is not recommended that chickens, either young or old, be raised on old litter used by a previous flock of birds. Always add a new layer of bedding to the old litter if an entirely new bedding substrate is not possible. This top dressing should be 5–7 inches in depth.	Exposing birds to old litter is not recommended as the litter may have a build-up of disease agents to which the new flock has not been exposed. This can result in a disease outbreak.
3. Do not bring new chickens, especially adult birds from other flocks, and mix them immediately with your flock.	Chickens need a minimum two-week quarantine period in a separate house in order to be monitored for any diseases.
4. Do not permit visitors in your poultry house if they have had contact with other poultry. If you do have visitors, they should not be wearing clothes and shoes that have come into contact with other birds and/or their feces.	Visitors can transfer diseases through their clothing, shoes, and unwashed hands. If you have frequent visitors that have exposure to poultry, provide footbaths and/or boot covering at minimum.
5. Prevent other birds (e.g., sparrows, pigeons) from direct contacting with your chickens.	These free-living birds can carry diseases and parasites to the chickens. Don't place bird feeders in areas where your poultry can congregate. One common backyard situation is having a bird feeder and chickens will often congregate under the feeder to eat the fallen bird food.
6. Purchase feed from a reliable source; do not use old moldy feed.	For health and productivity, chickens require a nutritionally balanced feed. Ensure that the feed is stored properly in a rodent proof container and in a cool dry area so that there is not heat degradation of nutrients and there is no mold growth.
7. Vaccinations are important in disease prevention, if needed.	Backyard chickens should only be vaccinated if there is a confirmed disease on your site. Marek's disease is common, so purchase chicks that were vaccinated in-ovo or at 1 day of age.
8. Provide a well-ventilated but draft-free building with appropriate space available for the number of chickens housed.	This reduces ammonia build-up, stress, and pen-mate fighting.
9. Properly dispose of all dead birds and old litter.	This prevents flies and odor and reduces potential transmission of diseases. Flies can be carriers of disease from infected birds. Properly disposing of birds will reduce a potential source of odor and reduce a potential fly breeding source.
10. Keep all sick chickens separated from the rest of the flock.	Diseases can be spread through direct contact with infected birds. Isolate any sick chicken from the rest of the flock.
11. In the event of a disease outbreak in your flock, get an accurate diagnosis as soon as possible.	Since many diseases show similar clinical signs, it is advisable to get an accurate diagnosis before beginning treatment. Your veterinarian can help you with diagnostic procedures.
12. If the hobbyist also has pet birds of different species (e.g., parrots), extreme care must be exercised when undertaking routines between the different bird species.	Pet birds, like parrots, can pose a serious threat to chickens because they can harbor diseases that can be very devastating to a chicken flock, or vise versa.

Source: Adapted from Ebako and Morishita [14].

eggs or poultry for their own flocks, or simply visiting the flock. If these visitors have exposure/contact with other poultry and/or bird species, they should wear coveralls or alternative protective clothing, and plastic disposable boots [1,16]. Additionally, a disinfectant foot bath that visitors step into prior to visiting the birds should be available. Recommended classes of disinfectants used in footbaths include phenols, iodophores, hypochlorites, quaternary ammonium compounds, and oxidizing agents. It should be noted that the footbath is only effective if it is kept fresh, and if it is not routinely maintained it can be a source of infection [2]. A typical footbath is changed weekly, depending on frequency of use, and consists of a long handled scrub brush, a tray with short sides with fake grass or some other synthetic bristled mat in the bottom, and enough disinfectant to cover the entire sole of the shoe [17]. If there are no visitors that will have contact with the birds, there is a reduced need for protective clothing, disposable boots, and footbaths.

Disinfectants

Cleaning and disinfecting is one of the most important steps you can take in practicing backyard biosecurity. Below are some examples of disinfectants available on the market. Follow the directions on the label carefully for the best results.

- Thoroughly clean and scrub objects before applying disinfectants. Disinfectants cannot work on top of caked-on dirt and manure, so thoroughly wash surfaces before disinfecting.
- Apply disinfectants using brushes, sponges and spray units. Allow adequate contact time (follow manufacturer's instructions.)
- Dispose of used disinfectant according to local regulations.

Examples of Disinfectants

- Roccal®: Mix 1/2 fluid oz of Roccal per gallon of water.
- Nolvasan® (chlorhexidine diacetate 2 percent): Mix 3 fluid oz of Nolvasan per gallon of water.
- Household bleach (sodium hypochlorite 6 percent): Mix 3/4 cup of household bleach per gallon of water.
- Lysol® spray for footwear
- Purell® hand pump for hand disinfection

Note: Trade names used in this publication do not constitute an endorsement, guarantee, or warranty of these products. USDA bears no responsibility resulting from the use of the described products. These procedures are not guaranteed to prevent highly contagious diseases from affecting your birds; however, they will reduce the risks.

Why Be Concerned?

- Not only could an outbreak of a bird disease such as exotic Newcastle disease or highly pathogenic avian influenza harm or kill your birds, it could spread quickly and kill other nearby birds.
- Early detection and reporting is the most important step in eradicating a disease outbreak. Don't be afraid of "crying wolf." State and Federal veterinarians want to hear about sick and dying birds.
- There is no charge for USDA veterinarians to work with you to conduct a disease investigation.

BIOSECURITY FOR THE BIRDS

www.aphis.usda.gov/vs

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Backyard Biosecurity Practices To Keep Your Birds Healthy

LOOK for Signs.
Watch for signs of disease or unexpected deaths among your birds.

REPORT Sick Birds.
Don't wait—early detection and reporting can make a difference. If your birds are sick or dying, call the USDA Veterinary Services office at **1–866–536–7593** or your State Veterinarian or local cooperative extension office to find out why.

PRACTICE Backyard Biosecurity.
Restrict traffic onto and off your property. Disinfect shoes, clothes, and hands to prevent the potential spread of disease. You are the best protection your birds have.

Figure 8.1 The United States Department of Agriculture Animal and Plant Health Inspection Service (USDA-APHIS) publishes this poster titled "Backyard Biosecurity 6 Ways to Prevent Poultry Diseases" which members of the public can download for free from http://www.aphis.usda.gov/publications/animal_health/content/printable_version /6-StepPoster-English_Araboc.pdf (accessed 14 September 2013). The USDA-APHIS also has available an informational video on proper biosecurity practices called "Backyard Biosecurity: Practices to Keep Your Birds Healthy" which members of the public can download for free from http://www.aphis.usda.gov/animal_health/birdbiosecurity. (accessed 14 September 2013).

Backyard biosecurity means doing everything you can to protect your birds from disease. As a bird owner, keeping your birds healthy is a top priority. Your birds can become sick or die from exposure to just a few unseen bacteria, viruses, or parasites. In a single day, these germs can multiply and infect all your birds. However, by practicing backyard biosecurity, you can keep your birds healthy.

If you follow these basic tips and make them part of your routine, you decrease the risk of disease entering your flock and persisting in soil, droppings, and debris. Practicing biosecurity is an investment in the health of your birds.

Biosecurity Tips: 6 Ways To Prevent Poultry Disease

Keep Your Distance.
Restrict access to your property and your birds. Consider fencing off the area where your birds are to form a barrier between "clean" and "dirty" areas. The clean area is the immediate area surrounding your birds, and the dirty or buffer area must be considered to be infected with germs, even if the birds appear healthy and disease free.

Allow only people who take care of your birds to come into contact with them. Your caretakers should not attend bird shows or other events where birds are present. If visitors to your property want to see your birds, be sure they wash up first and clean their shoes. Better yet, keep clean boots for visitors to wear. If your visitors have birds of their own, **do not** let them near your birds at all.

Game birds and migratory waterfowl should not have contact with your flock because they can carry germs and diseases. If your birds are outdoors, try to keep them in a screened area.

Keep It Clean.
You wouldn't think of tracking dirt and disease into your house, where it could infect your family. Don't do that to your birds either! Germs can be picked up on shoes and clothing and moved from one area to another. To keep your birds "germ-free," keep a pair of shoes and a set of clothes to wear only around your birds. Many people keep these clean clothes in a covered pail at the entrance to their bird area. Or, clean and disinfect your shoes and launder your clothes before you check on or work with your birds.

Scrubbing your shoes with a long-handled scrub brush and disinfectant (see section on disinfectants) will remove droppings, mud, or debris. Clothes should be washed in a washing machine with laundry detergent.

Wash your hands thoroughly with soap, water, and a disinfectant before entering your bird area.

Keep cages, food, and water clean on a daily basis. Clean and disinfect equipment that comes in contact with your birds or their droppings. That includes tools such as feed scoops, shovels, rakes, and brooms. All manure must be removed before disinfectant can work, so clean surfaces with soap and water first. Properly dispose of dead birds by burial or incineration or take them to a landfill. Check on local ordinances for acceptable disposal methods.

Don't Haul Disease Home.
Car and truck tires, poultry cages, and equipment can all harbor "germs." If you travel to a location where other birds are present, or even to the feed store, be sure to clean and disinfect these items before you return to your property.

Taking some of your birds to a fair or exhibition? Keep those birds separated from the rest of your flock and watch them for at least 2 weeks after the event to ensure that they didn't pick up a disease.

New birds should be kept separate from your flock for at least 30 days before putting them with the rest of your birds. To prevent disease, it is best not to mix young and old birds or birds from different species or different sources.

Don't Borrow Disease From Your Neighbor.
Do not share birds, lawn and garden equipment, tools, or poultry supplies with your neighbors or other bird owners. If you do bring these items home, clean and disinfect them before they reach your property. And remember to clean and disinfect borrowed items before returning them. Never share items such as wooden pallets or cardboard egg cartons because they are porous and cannot be adequately cleaned and disinfected.

Know the Warning Signs of Infectious Bird Diseases.
Many bird diseases can be difficult to diagnose. The list below includes some of the things to look for that signal something might be wrong with your birds. Early detection of signs is very important to prevent the spread of disease.

- Sudden increase in bird deaths in your flock
- Sneezing, gasping for air, coughing, and nasal discharge
- Watery and green diarrhea
- Lack of energy and poor appetite
- Drop in egg production or soft- or thin-shelled misshapen eggs
- Swelling around the eyes, neck, and head
- Purple discoloration of the wattles, combs, and legs (AI)
- Tremors, drooping wings, circling, twisting of the head and neck, or lack of movement (END)

Report Sick Birds.
Do not wait to report unusual signs of disease or unexpected deaths among your birds. Call your agricultural extension agent, local veterinarian, the State Veterinarian, or U.S. Department of Agriculture (USDA) Veterinary Services office. USDA operates a toll-free hotline **(1–866–536–7593)** with veterinarians to help you. USDA wants to test sick birds to make sure they do not have a serious poultry disease. There is no charge for USDA veterinarians to work with you to conduct a disease investigation. Early reporting is important to protecting the health of your birds!

Call **1-866-536-7593** (toll-free) to report sick birds!

Figure 8.1 (*Continued*)

For veterinarians doing field calls to multiple backyard flocks, it is imperative that clean coveralls be used when visiting each site, and to have rubber boots that can be scrubbed and disinfected before and after each farm visit [18]. Additionally, vehicles should be parked at the edge of the property to reduce the likelihood of obtaining contaminated soil or feces in tires and wheel wells. Because Marek's disease, a common disease in chickens, can be spread from the feather dander of infected birds, wearing a hairnet may be necessary to minimize this risk if visiting multiple flocks on the same day [18].

Backyard flock owners should have a separate set of shoes or boots to wear while working with the flock. This reduces the likelihood of bringing contaminated feces or soil into the house. Appropriate hand washing after handling poultry is also advised [16].

Reducing exposure from contaminated feed and water

In the process used in commercial pelleted feed production, the temperature is elevated and the majority of infectious agents will be destroyed from the heat. However, feed can become contaminated during storage, often by rodent feces. Therefore, feed should be kept in rodent-proof containers and stored in a cool, out of direct sunlight, moisture-free environment to reduce nutrient degradation and mycotoxin production [1,14,16]. Additionally, appropriate containers should keep other animals, including pets such as dogs, from accessing feed.

A clean water source is necessary and daily cleaning of water containers will prevent build-up of organic debris and infectious agents [1,18]. Periodic disinfection of the water containers will also ensure that the flock's exposure to disease agents is minimized. While the water supply for many backyard flocks is city municipal sources and generally clean, some farms may use well water, which should be tested annually as run-off after heavy rains may contaminate nearby wells [18]. Local public health agencies, land-grant state universities, state diagnostic laboratories, local extension offices, or water municipalities can be contacted for information on how and where well water can be tested.

Reducing exposure from pests

Rodents, specifically rats and mice, can serve as a reservoir for several poultry diseases including *Salmonella,* and rodent control measures should be in place for any backyard flock. The presence of readily available feed can attract rodents. Hence, appropriate containers for feed, minimizing feed spills and rapidly cleaning feed spills is of utmost importance to controlling rodents on the premises [18]. If you can see signs of rodents, including feces, then this usually indicates a rodent problem [1].

Free-living birds can serve as reservoirs for many diseases that can be shared among avian species, including, but not limited to, avian influenza, Newcastle disease, avian cholera, salmonellosis, chlamydiosis, campylobacteriosis, and avian tuberculosis [19–21]. Internal parasites tend to be host specific so they may not be readily spread to poultry, but external parasites like mites have a wider host range and can be spread through contact [22–25]. Like rodents, free-living birds are attracted to accessible feed, and exposure to free-living birds can vary per flock situation [26]. Feeding the poultry at night and before they are released from the coop in the morning could reduce contact with free-living birds and their feces [18]. The poultry could still have free access during the day to graze. In addition, bird feeders (for free-living birds) should not be hung near poultry areas as this will likely increase contact between free-living birds and poultry. Poultry may congregate under the feeders where they can become exposed to free-living bird feces or discarded food [18].

Predators, such as raptors and raccoons, can also serve as a source of disease or trauma for backyard poultry. Poultry should be housed in predator-proof housing at night [18,27].

Reducing exposure from new poultry introductions

For many backyard flocks, the addition of new poultry to the existing flock is one of the most common ways infectious agents are introduced. Obtaining background information such as vaccination history, previous diseases experienced in the birds being introduced or their parents, and causes of previous morbidity and mortality, is important for the prevention of infectious agent introduction. New birds should have a minimum two week, preferably four week, quarantine period before introduction to the rest of the flock [1]. This allows time for underlying diseases to manifest clinical signs because many disease agents have approximately a two week incubation period, and it also allows for enough time to perform and receive serological testing and fecal examination results [1].

During quarantine, birds should have a physical examination performed to determine health status and appropriate vaccinations should be administered [28,29]. A prominent keel bone indicates a thin bird, either as a result of chronic disease and/or a poor nutritional state (see Chapter 7) [30]. A poor nutritional state can also

make a bird more prone to disease. Examine the ventral abdomen and the vent region of the bird as most lice and mite infestations can occur in these areas [24]. Clumps of lice eggs (nits) are found at the base of the feather shaft [24,25]. Brownish black debris along feathers are often indicative of mite infestation [24,25]. As a prevention, birds can be treated for external parasites at the start and end of the quarantine period before they are added to the existing flock (see Chapter 12).

Also, during quarantine, blood should be collected to perform serological tests to determine exposure to certain disease agents, such as *Mycoplasma gallisepticum* [28,29]. Fecal samples should be collected at the start of quarantine and 2 weeks later to ensure that birds are negative of parasites. If parasites are detected, the birds should be treated and retested during the quarantine period to evaluate the effectiveness of the treatment. The quarantine period will help prevent seeding of facilities with internal parasites that survive for prolonged periods in the soil [23].

Keeping poultry away from infectious agents

The following procedures can be used to minimize the introduction of poultry to areas where infectious agents are present, including management of sick birds, carcass management, and vaccination.

Management of sick birds

Despite the best management practices, sometimes birds will become sick. It is important that poultry owners know what is considered normal behavior for their flock, including appetite. If any signs of clinical disease, such as diarrhea or respiratory signs, are noted, the affected bird should be removed from the flock to minimize the exposure to the other birds. Owners must also recognize that poultry are flock animals and may get stressed if housed away from the rest of the flock [18]. While the sick bird is in isolation, provide extra warmth and ensure access to feed and water while collecting specimens for diagnosis [18].

Carcass management

Proper management of a carcass can provide answers as to disease etiology and prevent further spread to other birds. Carcasses should be submitted for necropsy as soon as possible, or immediately disposed of in an area where other poultry, predators, and pets will not have access [18]. Do not allow flies to lay eggs on a carcass that develop into maggots because under certain circumstances other flock members could acquire botulism through ingestion of the maggots [18].

Vaccination program

An appropriate vaccination program is necessary for all backyard poultry flocks [20,29]. Vaccination protocols should be based on what diseases are present in the geographic region and what diseases have previously been diagnosed in the flock [20]. Diagnostic laboratories, extension veterinarians, and National Poultry Improvement Plan (NPIP) personnel can often provide information on which diseases are present in a region. It is also important to know if the birds will be taken off the farm and exposed to other birds, including at poultry shows or state fairs [26]. Owners must be made aware of potential disease exposure that might occur when birds are exposed to other birds of mixed or unknown vaccination history; owners must be willing to weigh this risk with value of the birds [2,20].

Typically, backyard flocks are only vaccinated against Marek's disease, and this is performed at the hatchery on day one of age or in ovo (vaccinate the egg), but vaccines for other diseases can be given if there is risk (see Chapter 21). Other vaccines that are available include a combination Newcastle disease and infectious bronchitis vaccine, laryngotracheitis vaccine, avian encephalomyelitis vaccine, fowl pox vaccine, and a fowl cholera bacterin [31].

Maintaining adequate records

Maintaining adequate records of bird transactions and movements, such as the name and address of who sold or purchased the bird, and bird mortality records, is crucial if there is a disease outbreak and an epidemiological investigation takes place. If a pet parrot, canary, or other birds are on the premises of a disease outbreak, having good records documenting biosecurity measures could save your pets from culling.

Zoonotic diseases

Zoonotic diseases are infectious diseases that can be transmitted either directly or indirectly from animals to humans. An example of direct transmission would be *Salmonella* spreading via the fecal-oral route. An example of indirect transmission would be transmission of infectious agents via fomites such as shoes, clothing, equipment, and vehicles. Zoonotic diseases that can potentially be spread by backyard poultry includes, but is not limited to, salmonellosis, chlamydophilosis, mycobacteriosis, influenza, Newcastle disease, eastern and western equine encephalomyelitis, West Nile virus, campylobacteriosis, listeriosis, and cryptosporidiosis [32–34]. The prevalence of these diseases in backyard

poultry is unknown and risks to backyard poultry owners or others exposed to poultry is probably variable. However, veterinarians should be knowledgeable about these diseases and their implications in order to appropriately educate owners. Seemingly healthy birds can be reservoirs and transmit disease. Owners of sick birds should be encouraged to consult a veterinarian. In most states the following diseases/infectious agents are reportable: *Salmonella* gallinarium, *Salmonella* pullorum, eastern equine encephalomyelitis, West Nile virus, influenza and *Chlamydophila psittaci* (avian chlamydiosis). The National Poultry Improvement plan (NPIP) is a great resource and has been instrumental in controlling *Salmonella* Pullorum and other diseases [35]. The Centers for Disease Control and Prevention (CDC) is also a great resource for salmonellosis and other diseases [36]. General hygiene practices, such as hand washing after handling birds or their excrement, not eating or drinking around the birds or their environment, and wearing personal protective equipment, help to decrease exposure to zoonotic pathogens. Also, the CDC recommends that people who are immunosuppressed (elderly, children under 5 years of age, or those with HIV or receiving chemotherapy) avoid contact with chickens to prevent potential life-threatening disease such as salmonellosis. The CDC goes on to say veterinarians should educate owners of these risks, and in some cases, education can take place through a handout that the owner signs after reading. Figure 8.2 shows a poster available from the CDC regarding Salmonellosis and live poultry [37].

Salmonella
General information
Most of the estimated 1.4 million annual cases of human salmonellosis in the United States result from ingestion of contaminated food including eggs, but recently there has been an increase in cases resulting from direct exposure to live poultry [38].

Taxonomy
Salmonella is a Gram-negative bacterium in the Enterobacteriaceae family. It is distributed worldwide and most serovars infect animals as well as people [37]. As a result of recent DNA analysis of the bacteria in the *Salmonella* genus, two species have been designated, *S. enterica* and *S. bongori*. *S. enterica* is further subdivided into six subspecies, with the most frequent human pathogens being found within subspecies I, also known as subgroup 1, and designated, for example, as *S. enterica* subsp. *enterica*. Both the genus and species name are italicized and the serovar name begins with a capital

letter, for example: instead of *Salmonella typhi* the new nomenclature is *Salmonella enterica* serovar Typhi, abbreviated S. Typhi or ST.

There are over 60 serogroups (i.e., B and D), and over 2400 serotypes, many of which are serologically classified by the geographic location where the serotype in question was first isolated. *Salmonella* from groups B and D account for approximately two-thirds of all reported *Salmonella* infections and include the two most common serotypes, *Salmonella enterica* serotype Enteritidis (S. Enteritidis) and *Salmonella enterica* serotype Typhimurium (S. Typhimurium), which together account for half of all human infections in the United States [39].

There is great genetic variation within many *Salmonella* serotypes. Serotyping and phagetyping are now complemented by molecular subtyping techniques, such as pulse field gel electrophoresis, for epidemiological disease surveillance and outbreak investigations. An overview of *Salmonella* can be found at the Center for Disease Control's (CDC) website at http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salmonellosis_g.htm [40].

Reservoir and incidental hosts
Many species of wild and domestic animals, including poultry, can act as reservoirs for *Salmonella* bacteria. There are many reports of chicks and ducklings acting as a source of infection for humans, especially children. The CDC website has an informational page titled "Salmonella Infection (Salmonellosis) and Animals" which is available at http://www.cdc.gov/healthypets/diseases/salmonellosis.htm [41].

Pathogenesis
Exposure usually occurs by ingestion of the bacteria, which is shed in the feces of infected animals or fecal contaminated food or water. Relatively few bacteria can cause infection, but usually more than 1000 are needed. The incubation period is generally from 6–72 hours, but is usually between 12–36 hours. *Salmonella* naturally lives in the intestine of poultry and is considered normal flora in healthy, live poultry. Raw and undercooked foods, such as eggs, milk, and meat products, are a common source of infection, as well as cooked foods not maintained at an adequate temperature, or cross contamination with these products. Recently, it has become more common for live poultry to be a source of exposure to *Salmonella*. Anything that touches live poultry can also be contaminated with *Salmonella* including food and water dishes, pens, coops, plants and soil. To prevent exposure to *Salmonella*, wash hands with soap and warm water immediately after handling

Figure 8.2 This poster from the CDC regarding Salmonellosis and live poultry is available for download free to the public from http://www.cdc.gov/healthypets/resources/salmonella-baby-poultry.pdf. (accessed 8-16-14). This poster could accompany any newly acquired poultry to educate the new owner about Salmonella risks.

poultry or anything they touch, keep poultry and anything they touch outside the house, and follow other biosecurity recommendations discussed previously. The CDC further states: Do not eat or drink where the birds live or roam, do not wash their bowls in the kitchen sink, do not house poultry in bathrooms or keep where food is prepared, served or stored [41].

Epidemiology

S. Enteritidis was shown to survive in poultry litter and feed for 26 months after removal of an infected flock [42]. Increasing the pH of the soil by adding lime has been shown to decrease growth and survival of *Salmonella* bacteria [42]. Freezing will also decrease its survival, but some can still live to replicate when thawed. *Salmonella* is most susceptible to heating and drying and many common disinfectants including phenolic compounds, chlorine, and iodine based compounds [43].

Since the 1990s there have been 45 *Salmonella* outbreaks linked to live poultry (not eggs or raw chicken), including 1563 illnesses, 221 hospitalizations, and 5 deaths [44]. As backyard poultry are becoming more popular, the number of *Salmonella* outbreaks linked to live poultry has increased to eight outbreaks reported in 2012 [44]. One outbreak in 2013 involving backyard flocks affected 316 people, 37 states, and resulted in 51 hospitalizations [44]. This outbreak was caused by *S.* Typhimurium from a hatchery in New Mexico selling chicks to multiple agricultural feed stores. Fifty-nine percent of those taken ill were children 10 years old or younger. The New Mexico hatchery participated in the USDA's NPIP and was free of *Salmonella* Pullorum, but testing for other *Salmonella* organisms is currently not a part of that program. Another *Salmonella* outbreak in 2013 linked to live poultry (chicks) from a hatchery in Ohio involved 125 people in 26 states, and was associated with the following outbreak strains: *S.*Infantis, *S.*Lille, *S.*Newport, and *S.*Mbandaka [44]. Again, 41% of the ill were children <10 years old. There were reports of chicks being brought into the home and being kissed and cuddled. Outbreaks in 2012 involved *S.*Hadar, *S.*Montevideo, *S.*Infantis, *S.*Newport, and *S.*Lille. Outbreaks in 2011 involved chicks and ducklings with *S.*Altona and *S.*Johannesburg [44].

Avian chlamydiosis
General information

Although human exposure to *Chlamydophila psittaci* from poultry usually occurs at turkey slaughter plants, backyard chickens can carry the organism and infect humans. A complete description of *Chalmydophila psittaci* and the

disease in humans and animals can be found in the periodically updated Compendium of Measures to Control *Chlamydophila psittaci* Infections Among Humans (Psittacosis) and Pet Birds (Avian Chlamydiosis) (2010) by the National Association of State Public Health Veterinarians [45,46].

Taxonomy and nomenclature

The human disease psittacosis is also known as parrot fever, ornithosis, chlamydiosis, and chlamydophilosis. The term "psittacosis" refers to the disease in people originating from a parrot (a psittacine bird), whereas the term ornithosis refers to the disease in people originating from any species of bird. The term chlamydiosis is a more generic term and refers to infection in any animal or person with any organism in the *Chlamydia* or *Chlamydophila* genus. The term "avian chlamydiosis" is used to specify an infection with *Chlamydophila psittaci* in birds.

Note that *Chlamydophila psittaci* should not be confused with a related organism in humans, *Chlamydia trachomatis*, a sexually transmitted disease of people, or another related organism, *Chlamydophila pneumoniae*, a common mild respiratory pathogen of humans.

In 1999 changes in nomenclature occurred that reflected recent advances in DNA testing that revealed differences between organisms that were previously thought to be the same. What was previously classed as one genus, *Chlamydia*, is now described as two, *Chlamydia* and *Chlamydophila*. When reading any literature on chlamydial organisms prior to 1999, the reader may not be 100% certain as to which of the newly categorized organisms was being referred to in the document. The term *Chlamydophila psittaci* will be used throughout this document to refer to the organism formerly known as *Chlamydia psittaci* [47].

Epidemiology

The organism has been found in 130 species of birds worldwide and a variety of mammals, including humans, and is therefore a zoonotic disease. Birds are known to be a potential source of infection, and include domestic or wild pigeons, passerines (soft-billed birds), or poultry, although poultry do not usually exhibit overt illness with this disease.

The general prevalence of chlamydial infection in captive birds is thought to be less than 5%, and in wild birds less than 1%, but may increase dramatically in birds stressed by shipping, crowding, chilling, and breeding. Those at occupational risk for the disease include pet store employees, veterinarians, veterinary technicians, laboratory workers, workers in avian

quarantine stations, farmers, wildlife rehabilitators, zoo workers, and employees of poultry slaughtering and processing plants (usually involving turkeys).

Pathogenesis

The *Chlamydophila psittaci* organism is transmitted by either inhalation or ingestion of the spore-like elementary body phase of the organism. Shedding in birds can be activated by stress, such as shipping, crowding, chilling, and breeding. Those individuals that are immunosuppressed are more susceptible to the disease and its effects.

The organism *Chlamydophila psittaci* is relatively resistant, surviving in soil for 3 months or within bird droppings for up to 1 month. *Chlamydophila psittaci* is a dimorphic organism, meaning it exists in two different forms called phases, the infectious phase and the replicating phase. In general, the *C. psittaci* organism enters the host via inhalation or ingestion and replicates in the host's cells. The infectious form is small (0.2–0.6 μm) and is known as an elementary body. After the elementary body is inhaled or ingested it is endocytosed by a host cell where it transforms into a reticulate body. The reticulate body is larger (1.5 μm) than the elementary body and is the replicating, or metabolically active, form of the organism inside the host's cell. The reticulate body uses nutrients from the host cell and undergoes multiple rounds of binary division before releasing multiple elementary bodies from the cell when it ruptures. Two or more days pass between the time the host cell is infected and the elementary bodies are released. These infectious elementary bodies then infect other host cells or are released into the environment via feces, nasal secretions, sputum, blood, or infected tissues. The elementary body is metabolically inactive and resistant to environmental forces, so it can survive long enough to be inhaled or ingested by another host.

Persons performing a necropsy of any bird suspected of having avian chlamydiosis should wear gloves and wet the carcass with soapy water to decrease aerosolization or organisms, as well as practice Animal Biosafety Level 2 practices, which include containment equipment and facilities, and respiratory protection.

Clinical signs

Poultry, pigeons, and passerines seem to exhibit little if any clinical signs of disease while infected with the *Chlamydophila psittaci* organism, and therefore are sometimes referred to as asymptomatic carriers of the disease.

Diagnosis

There are many tests available for use in birds, including tests to detect antibodies in the serum (elementary body assay [EBA] and immunofluorescent antibody [IFA]), tests to detect antigen in the feces or blood (enzyme-linked immunofluorescent antibody assay [ELISA], and polymerase chain reaction [PCR]). It is best to perform a panel of tests including PCR of blood, PCR of feces, and IFA of serum. Alternatively, a fluorescent antibody [FA] test can be performed on tissue such as liver from a biopsy or necropsy and is available through most state diagnostic laboratories. For legal purposes, cell culture from the feces is the best test, but the organism does not consistently grow, and shedding of the organism in the feces is intermittent. There is also risk to personnel when the organism is grown in the laboratory. The most common situation with backyard poultry that results in diagnosis of avian chlamydiosis is that PCR is performed on a necropsy specimen that has died with a concurrent illness, such as mycoplasmosis.

Treatment

The treatment of birds should be supervised by a licensed veterinarian. The treatment of choice in birds is oral doxycycline, as it is absorbed better and eliminated more slowly than other tetracyclines (see Chapter 20 regarding egg laying or meat birds). Care should be taken to observe for signs of toxicosis while treating such as lethargy, anorexia, or biliverdinuria. If it occurs, the doxycycline should be discontinued and the bird offered supportive care until recovered, and then a lower dose can be attempted.

Prevention and control

People cleaning cages or handling infected birds should wear protective clothing, gloves, a disposable cap, and a respirator or appropriate mask. Accurate records should be maintained of all bird-related transactions to aid in identifying sources of infection and potentially exposed persons and should include date of purchase, species of bird, source of birds, leg band numbers, and describe any illness or deaths of birds. Avoid purchasing or selling sick birds and isolate newly acquired birds for at least 30 days. Test birds before they are to be boarded or sold on consignment.

Chlamydial organisms can be killed with most commonly used disinfectants including freshly prepared 1:32 dilution of household bleach solution (1/2 cup per gallon), 1% phenol compounds, or 1:1000 dilution of quaternary ammonium compounds [45]. Gloves and respirator should be worn in an avian outbreak in any

areas that have been exposed to the positive bird or its fecal matter. It is important to clean and remove all organic matter first and then disinfect. Chlamydiosis in humans is a Nationally Notifiable Disease. Avian chlamydiosis is reportable in most states: If a veterinarian diagnoses chlamydiosis in a bird, the case must be reported to the state veterinarian or public health department. Usually the public health department becomes involved if humans are affected.

Mycobacteriosis

Mycobacteriosis has been diagnosed in many species including Galliformes and Anseriformes. Domestic fowl are relatively resistant to infection, whereas psittacine birds seem to be highly susceptible.

Taxonomy

Mycobacteriosis in birds is generally caused by the acid fast bacteria *Mycobacterium avium* or *M. genevense*. The bacteria can survive in soil for several months [48].

Epidemiology and pathogenesis

Transmission takes place through inhalation or ingestion. Because of the long incubation period, it is sometimes difficult to determine the source of human or bird exposure. The reported prevalence of mycobacteriosis in wild populations varies between 4% and 40%. It has been reported that poultry, pheasants, and sparrows are highly susceptible, guinea fowl and domestic turkeys are less susceptible, domestic geese and ducks are moderately resistant, and the domestic pigeon is highly resistant to infection with *Mycobacteria* [48].

Clinical signs

Avian mycobacteriosis generally causes chronic weight loss and unthriftyness. Unlike humans, infection of the respiratory tract is uncommon in birds; most birds develop granulomatous masses of the liver and intestines.

Diagnosis

Radiographs are suggestive. Demonstration of acid fast organisms in feces is suggestive but not definitive, as other non-pathogenic acid fast organisms can be found in the feces. Demonstration of acid fast organisms in tissues is highly suggestive. *M. avian* is usually associated with large numbers of organisms in birds, whereas *M. tuberculosis* and *M. bovis* are not. The organism is difficult and dangerous to culture.

The intradermal tuberculin test used in many mammals is not reliable in most birds, but has been used with some success in the wattle of chickens [45]. The standard purified protein derivative (PPD) is injected intradermally (0.05–0.1 ml, 2000 IU) into one wattle of the chicken [45]. Heat, swelling (>5mm), and edema of the injection site 48 hours later is considered positive for infection with, or sensitization to, *M. avium* [48]. This tuberculin test is considered about 80% accurate in detecting infected birds compared to gross lesions, but birds in the advanced stages of disease may have no reaction [48]. If testing waterfowl, the whole blood agglutination test is considered better than the tuberculin test, but may give false positives [48].

Treatment

Treatment of birds with mycobacteriosis should be discouraged and euthanasia should be the first option discussed for numerous reasons including the following: The *Mycobacterium avium* organism is often resistant to antimycobacterial drugs (such as isoniazid, ethambutol, rifampicin, and pyrazinamide); long term (12–18 month) treatment can be excessively expensive; the organism has zoonotic potential; and there is a lack of pharmacokinetic or pharmacodynamic data regarding anti-mycobacterial drugs in birds.

Influenza A

Avian influenza is highly infectious and affects many species. The disease in birds is explained in detail in the Avian Influenza and Exotic Newcastle Disease chapter elsewhere in this book (see Chapter 9). The zoonotic capabilities of this virus are described below.

Taxonomy and nomenclature

Influenza virus is located in the orthomyxoviridae family. There are three types of influenza, types A, B, and C, but only type A is found in birds. There are influenza A subtypes based on surface proteins, "H" for hemagglutinin, and "N" for neuraminidase. There are 15 known H subtypes and 9 known N subtypes. All subtypes are found in birds, but only H1, H2, H3, and N1 and N2 are typically found in humans. All avian virulent strains to date have been H5 or H7 subtype, but most H5 or H7 isolates have been of low virulence.

History

In 1918–1919 there was an influenza A pandemic in people that was known as the Spanish flu. This outbreak was found to be H1N1 and may have killed 20–50 million people worldwide, 50% of them young and healthy adults. Greater than 500,000 deaths were attributed to the flu in the United States. In 1957–1958 the "Asian flu," H2N2, caused approximately 70,000 human deaths in the United States. In 1968–1969 the

"Hong Kong flu," H3N2, caused 34,000 human deaths in the United States and still circulates today. In 1961 influenza A H5N1 was first isolated from terns in South Africa. In 1997 influenza A was shown to pass from birds to humans in an outbreak in Hong Kong where 18 people that had contact with infected birds became sick and 6 died. This outbreak was controlled by killing 1.5 million chickens. In 1999 in Hong Kong, China, H9N2 was confirmed in two children, both of whom recovered. Both children had contact with chickens.

In 2003 influenza A H5N1 was isolated from two human cases in Hong Kong, one of whom died. Then the H5N1 subtype was confirmed in turkey poults in Cambodia, China (Hong Kong had a single positive peregrine falcon), Indonesia, Japan, Laos, South Korea, Thailand, and Vietnam. Human deaths from influenza A H5N1 were reported in Thailand and Vietnam after contact with infected birds or their excretions (nasal, saliva, or feces). This H5N1 subtype isolated from humans has been genetically sequenced and all genes were found to be of bird origin. It was also found to be resistant to two antiviral drugs, amantidine and rimantidine, but still sensitive to oseltamivir and zanamavir. To date, approximately 600 human cases have been reported with a 60% fatality rate. Also in 2003 influenza A H7N7 was isolated from poultry workers in the Netherlands. Over 80 cases were reported and one, a veterinarian, died. People in the Netherlands outbreak exhibited eye infections with some respiratory infections.

More recently, in the spring of 2013, a low pathogenic H7N9 influenza virus emerged in China and led to approximately 130 human cases with 31 human deaths. This virus was particularly interesting because it did not lead to morbidity or mortality in poultry.

Epidemiology and pathogenesis

Wild birds, especially waterfowl, are the natural hosts for influenza A and do not generally exhibit illness from infection. Domestic birds, particularly chickens, are very susceptible to high mortality with influenza A. Animal to human transmission has occurred but is unusual at the current time. The virus is currently not efficient at human to human transmission, but gene mutation is common and may lead to sustained human to human transmission in the future.

The World Health Organization (WHO), as well as other organizations, is monitoring for human gene development in order to stop the virus before it spreads by killing birds infected or exposed to pathogenic strains (H5 or H7).

The OIE is an international organization based in France. It was formed by 28 countries in 1928 and now comprises 165 countries. The OIE is a clearing house of reported cases of animal disease from each of the member countries and aids in the rapid response to multi-country outbreaks.

Diagnosis

Many tests are available including the Agar gel immunodiffusion (AGID) test, ELISA, and RT-PCR. Not all birds develop demonstrable antibodies on the ELISA. The hemagglutination and hemagglutination inhibition tests are also available. Birds with H5 or H7 are required to be depopulated.

Prevention and control

People cleaning cages, handling infected birds or slaughtering birds should wear protective equipment. Influenza is typically contracted through inhalation, so respiratory protection such as a mask is of utmost importance. Gloves and eye protection can also be beneficial. Areas where birds are housed or slaughtered should be regularly cleaned and disinfected. The virus can survive days to weeks in feces depending on environmental conditions. The virus is typically susceptible to most commonly used disinfectants.

Newcastle disease

Newcastle disease is caused by a paramyxovirus of which there are four pathotypes: Lentogenic, mesogenic, neurotropic velogenic, and viscerotropic velogenic [49]. The latter is also known as VVND or exotic Newcastle disease, which is a foreign animal disease in the United States. In people, Newcastle disease causes a mild, acute granular conjunctivitis, general malaise, and sinusitis that resolves within 7–20 days. Exposure usually takes place from affected chickens, but can also occur from the live vaccine. Please see Chapter 9 for further information on the disease in poultry.

Eastern and Western equine encephalomyelitis virus

Eastern equine encephalitis (EEE) and Western equine encephalitis (WEE) are both caused by togaviruses. Clinical disease of EEE is most common in birds that are not native to North America such as imported pheasants and cranes [50]. Clinical disease of WEE has been reported in pheasants, emus, chukars, English sparrows, chickens, and turkeys [50]. Chickens rarely develop disease from either of these viruses, and they do not typically develop high enough viremia to play a significant role in transmission of the virus via mosquitos. Therefore, chickens are often used as sentinels for these diseases.

Epidemiology

Many species of birds act as reservoirs for EEE and WEE. The virus is transmitted through a mosquito bite, or in pheasants it can be transmitted through the broken skin created by pecking [50].

The mosquitos known to transmit EEE virus in the United States and Canada include *Culiseta melanura*, *Aedes* spp., and *Coqiullettidia* spp. Interestingly, *Culiseta melanura* feeds mainly on birds and rarely on people, whereas *A. sollicitans* and *A. vexans* act as a bridge for the virus by feeding on both birds and people, but are less likely to be infected with the virus. The mosquitos known to transmit WEE virus include *Culex tarsalis* in the western United States and Canada and *Culiseta melanura* in the eastern United States.

People are an accidental host to the EEE or WEE viruses and mortality can reach 80% if infected with EEE virus and 5–15% if infected with WEE virus. During the 1962 outbreak in Saskatchewan, Canada, the WEE virus was isolated from 22% of the wild bird population.

Clinical signs, diagnosis, and treatment

Birds rarely show clinical signs, but ataxia, trembling, weakness, paralysis, and death can occur. People show varying degrees of neurological signs and death. Antemortem diagnosis is difficult, and histopathologic lesions of lymphoplasmacytic encephalitis are suggestive. There is no specific treatment for viral encephalitis other than supportive care.

West Nile virus
History

The causative organism is a flavivirus. West Nile virus (WNV) was endemic in other countries, but in the late 1990s it was found within the eastern United States. The virus has since become endemic in the continental United States.

Epidemiology

Crows, jays, raptors, ducks, and horses are susceptible species, whereas most poultry species are considered resistant to clinical disease. The WNV virus is spread by numerous species of mosquitos. If people or dogs are clinically affected, they are usually older or immunosuppressed.

Clinical signs, diagnosis, and treatment

Clinical signs range from none in resistant species, such as poultry, to neurologic signs (ataxia, circling, head tilt, and seizuring) and death in susceptible species. A CBC is usually normal or a lymphocytosis may be present.

A serum antibody test is available. Treatment consists of supportive care, and recently in people the use of alpha interferon has resulted in more successful case outcomes.

Prevention and control

There is no vaccine available for humans and mosquito control is the primary method used to prevent disease in people. There is a vaccine available for use in horses that is often used in at-risk birds.

Campylobacteriosis

Campylobacteriosis is a commonly reported food-borne illness in humans and is estimated to affect 1.3 million people annually in the United States (CDC) [51]. Transmission usually occurs through ingestion of contaminated food or water, but can also occur from live poultry [52]. *Campylobacter* bacteria are common in poultry but cause them no clinical disease. Wild birds can be a source of infection for backyard poultry and humans. One study evaluating 333 wild birds in the mid-Atlantic region of the United States detected *Campylobacter jejuni*, *C. coli*, or *C. lari* via multiplex PCR in six avian families with an overall prevalence of 7.2%. Crows (Corvidae) and gulls (Laridae) had the highest prevalence at 23% and 25% respectively [19]. Another study evaluating 318 fecal samples from urban resident Canada geese in North Carolina, United States, detected six different strains of *C. jejuni* including one strain (ST-4071) that has been associated with human illness [49]. Prevalence in Canada geese was 5% in 2008 and 16% in 2009 [53]. Biosecurity measures described above, including exclusion of wild birds and proper hygiene around poultry, will reduce exposure to *Campylobacter sp*.

Listeriosis

Sporadic outbreaks of listeriosis, caused by the Gram positive, non-spore forming bacteria *Listeria monocytogenes*, have been described in chickens, turkeys, ducks, geese, pigeons, canaries, parrots, and other birds [54,55]. Usually affected birds are young and show signs of torticollis with the encephalitic form or emaciation and diarrhea with the septicemic form [54]. The usual route of human exposure is through consumption of contaminated poultry, resulting in neurological signs. Direct handling of apparently healthy birds infected with the bacteria has been reported to cause conjunctivitis in humans [55]. Ruminants on the farm can be infected by poultry feces that contains the bacteria [54]. Presumptive diagnosis is based on gross and histopathological lesions, including inflammatory foci

in the brain, splenomegaly, multifocal hepatic necrosis, myocardial necrosis, and pericarditis, but definitive diagnosis is based on culture [54,55]. This organism is resistant to many commonly used antibiotics and therefore treatment is often unsuccessful in poultry.

Cryptosporidiosis

Currently, it is questionable whether contact with live poultry can act as a source of human cryptosporidiosis infection because *Cryptosporidium baleyi*, found in avian species, has not been found to infect animals other than birds. Further, *C. parvum*, found in mammals including humans, is not commonly seen in poultry [56]. Recent studies have suggested that because a *C. meleagridis* strain was shown to infect mice and therefore has the potential to be zoonotic from birds to humans [57, 58]. A recent study of 2579 fecal samples from 46 chicken farms and 8 Pekin duck farms in Henan Province, China, showed *C. bailei* was present on many farms, but more importantly that *C. meleagridis*, which has the potential to be zoonotic, was isolated via PCR from 31–120 day old chickens from 3 of the 46 chicken layer farms [53]. Another study using PCR found the prevalence of *C. meleagridis* in turkeys from 16 farms and chickens from 23 farms in Algeria was 44% and 29% respectively [59].

Hypersensitivity pneumonitis

Hypersensitivity pneumonitis is not an infectious disease but is an allergic inflammation of the lungs in humans in response to certain antigens, such as feather dander, droppings, or moldy hays. The disease is sometimes called farmer's lung, bird breeder's lung, pigeon breeder's lung, or poultry worker's lung, and can present in acute, sub-acute, or chronic forms and can lead to death [60]. Poultry workers have been shown to have a higher prevalence of toxic pneumonitis, airway inflammation, and chronic bronchitis compared to controls [61]. Appropriate ventilation, cleaning of facilities, and respiratory protection such as a mask can reduce the likelihood of developing this problem.

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Avian Influenza and Viscerotropic Velogenic (Exotic) Newcastle Disease

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Introduction

The following two diseases, avian influenza (AI) and exotic Newcastle disease (END), are unlikely to occur in a backyard flock, but nonetheless, the primary veterinarian should be familiar enough with the presentation and consequences of these diseases to be able to identify them when encountered and know what to do next [1–3].

Avian influenza

Avian influenza is also known as bird flu**,** fowl plague, fowl pest, and grippe.

Clinical history

The likelihood of AI occurring in backyard flocks is relatively low. The most likely premises are those that have waterfowl, access to wild migratory waterfowl, or flocks near bodies of water where migratory waterfowl congregate. Clinical history for flocks with AI depends on the pathogenicity of the infecting AI virus. There may be complaints of mild respiratory signs in the affected flock such as a snick, similar to a reverse sneeze, cough, tracheal rales, or rattling when breathing and other general signs of illness. With more pathogenic strains of AI virus, the owner may find a large amount of their flock dead with no previous clinical signs noticed.

Causative agent

AI is caused by type A influenza viruses that occur in the avian species. Humans are infected with types A, B, and C influenza viruses, with types A and B being most common. Influenza viruses belong to the family *Orthomyxoviridae*. Influenza virus genomes are composed of eight segments of RNA. Segmentation of the genome allows swapping of gene segments between viruses and thus dramatic shifts in antigenic make-up of the virus (referred to as antigenic shift) can occur over short periods of time. AI viruses are enveloped; therefore they are relatively easy to inactivate via such methods as soap and water, heat, sunlight, and most if not all disinfectants. On their envelopes are two glycoproteins that project from their surface and allow further classification of these viruses. One glycoprotein is a hemagglutinin and the other is a neuraminidase. The hemagglutinin is responsible for allowing the virus to attach to a host cell. The neuraminidase is responsible for allowing newly formed viruses, assembled in the host cell, to escape from the cell. So far, 16 different hemagglutinins and 9 different neuraminidases have been identified on influenza viruses. These proteins help identify the H (hemagglutinin) type and the N (neuraminidase) type of AI viruses. Each influenza virus has a single H type and a single N type and they can occur in any combination. There can be an H1N1, an H1N2, an H1N3, an H2N1, an H3N1, an H4N1, and so on, so theoretically there can be 144 different AI viruses. The AI virus that most people are aware of is the Asian strain of H5N1, which has killed lots of poultry worldwide, made people sick and caused some people to die. To date, the Asian strain of H5N1 has not been identified in North America. An H5N1 strain has been found multiple times during surveillance of North American migratory waterfowl but those viruses have not been classified as highly pathogenic or related to the Asian strain.

Strains of AI differ in their ability to cause illness and death in domestic poultry. In the past, strains have been classified by their ability to kill a specific number of experimentally infected embryos or 6-week-old chicks. Those producing few signs and lesions and little or no dead animals were classified as low pathogenic AI viruses, referred to as LPAI. Those producing dramatic lesions and many dead animals were classified as highly pathogenic AI viruses, referred to as HPAI. Historically, only H5 and H7 viruses have caused catastrophic death loss in domestic poultry worldwide and have been classified as highly pathogenic AI viruses. Not all H5 and H7 AI viruses are highly pathogenic BUT, all H5 and H7 AI viruses have the potential to become highly pathogenic over time, as they pass from bird to bird. More recently, these viruses have been evaluated molecularly. It was discovered that a specific area on the H protein may be used as a predictor of high pathogenicity. Classical methods are still the most reliable, in the author's opinion, because in 2004 an H5N2 AI virus from a field case in a commercial egg-laying chicken flock was molecularly predicted to be a highly pathogenic type but proved to be avirulent in experimental chickens and caused few clinical signs in the commercial flock.

AI viruses can spread from waterfowl to domestic poultry, from pigs (where it is referred to as swine influenza) to domestic poultry and more recently, as in the case of the pandemic H3N2, from humans to turkeys. H1N1 and H3N2 are typically found in swine populations, where they cause a respiratory disease of limited consequences. H1N1 and H3N2 cause little or no disease in chickens but cause commercial turkey breeder hens to stop laying eggs.

Clinical signs and lesions

AI viruses can infect any poultry at any age. They are thought to cycle in wild migratory waterfowl where they cause little or no disease. The problem occurs when they infect domestic poultry or a highly pathogenic strain develops. Clinical signs vary with the pathogenicity of the infecting AI virus. Signs of low pathogenic AI virus infection may consist of mild respiratory signs such as a snick, cough, tracheal rales, or rattling when breathing, occasionally diarrhea, with or without other general signs of illness such as inactivity, reluctance to move and a "puffed up" appearance caused by extension of the body feathers. Highly pathogenic viruses often cause rapid onset of illness with death occurring within <24 hours post infection. Clinical signs associated with highly pathogenic AI may also include central nervous system (CNS) signs such as tremors, torticollis and opisthotonus, and cessation of egg production but are often not noted because of the rapid onset of the disease followed by death.

Lesions of LPAI could be that of septicemia if AI is complicated by bacterial infection. LPAI may produce reddening of the tracheal mucosa with red, wet, heavy lungs. Lesions of HPAI reflect fulminant disease as seen in other catastrophic diseases such as viscerotrophic velogenic (exotic) Newcastle (vvND) and duck viral enteritis. It is impossible to differentiate HPAI from vvND by gross examination alone. Additional testing is required. HPAI causes edema, hemorrhage, and necrosis in skin and many visceral organs. Swelling of the face, neck, and feet may be present as well as swelling, hemorrhage, and cyanosis of the comb and wattle. Hemorrhages on the serosa and mucosal surface of the intestine are common. Hemorrhage in the proventriculus, ventriculus, and cecal tonsil may be present as well. The lungs are typically edematous and hemorrhagic.

Transmission route

The virus is most typically transmitted by aerosol route from respiratory tract secretions but the virus may also be transmitted by fecal/oral route and, as with other avian pathogens, through fomites.

Diagnostic tests

Diagnosis of AI must be confirmed via testing typically performed at diagnostic laboratories. Agar gel immunodiffusion (AGID) is typically used as a screening test because poultry are typically not vaccinated for AI. Currently, rapid virus identification tests are used in contrast to classical methods such as virus isolation. Certain antigen capture enzyme-linked immunosorbent assay (ELISA) test kits, similar to home pregnancy tests, have been proven effective in detecting AI virus from respiratory and cloacal swabs during acute infection. National Animal Health Laboratory Network (NAHLN) laboratories are equipped with real time polymerase chain reaction (rtPCR) for AI virus matrix and for H5 and H7 identification. Further classification should be performed by more classical methods at the National Veterinary Services Laboratory in Ames, Iowa.

Differential diagnosis

Viscerotropic velogenic (exotic) Newcastle disease, septicemic *Pasteurella multocida*, power failure in confined poultry, toxicant exposure and predation for HPAI, and other respiratory diseases for LPAI.

Prevention and control

Vaccination for AI is not routinely practiced in the United States and is controlled by government agencies. Breeder turkeys are routinely vaccinated with H1N1 and H3N2 to prevent the dramatic egg production drop experienced during infection.

Newcastle disease

Newcastle disease is also known as ranikhet, avian pneumoencephalitis and pseudo-fowl pest. Viscerotrophic velogenic Newcastle disease is also known as exotic Newcastle disease (END).

Clinical history

The likelihood of Newcastle disease (ND) occurring in backyard flocks is relatively low in most of the United States. California however, has had occasional outbreaks of viscerotrophic velogenic ND (vvND) in fighting chickens in the 1970s, 2002, and 2003. The majority of the 2003 outbreak occurred in small household flocks in large cities. Worldwide, this disease is probably the most common disease of small household and village flocks. Throughout the world, vvND is known to spread from village to village, town to town, island to island totally decimating those populations. Unlike the rest of the world, vvND is not endemic in the United States and is thus commonly referred to as exotic ND. Clinical history for flocks with ND, like AI, depend on the pathogenicity of the infecting ND virus. Presenting complaints are respiratory disease with mild respiratory signs such as a snick, cough, tracheal rales, or rattling when breathing and other general signs of illness in milder forms. Complaints of CNS manifestations, such as torticollis, paresis, and paralysis, may be seen in addition to respiratory signs in more pathogenic strains in contrast to lack thereof in AI. Like AI, the owner may find a large amount of their flock dead with no previous clinical signs with infections caused by the highly pathogenic strains of ND virus.

Causative agent

Newcastle disease, so named because one of the first outbreaks was identified in Newcastle-upon-Tyne in England, is caused by avian paramyxovirus serotype 1 (APMV-1). Like AI virus, APMV-1 is an enveloped RNA virus; however, its genome is not segmented. Similar to AI, APMV-1 viruses vary in the effects caused by their infection. They too are classified by their ability to kill experimentally infected embryos or 6-week-old chickens. The types of ND are referred to as lentogenic, mesogenic, or velogenic ND viruses. Lentogenic ND viruses are typically subclinical or cause mild respiratory signs. They are the most common strains of ND in the United States. Mesogenic ND viruses typically cause respiratory signs and occasional nervous system signs but with low mortality. These strains are occasionally found in the United States. Velogenic ND viruses cause high mortality often without previously noted clinical signs. Velogenic ND viruses are further classified as viscerotropic or neurotropic. Viscerotropic velogenic ND causes hemorrhagic gastrointestinal lesions, and neurotropic velogenic ND viruses cause high mortality typically after respiratory and nervous signs. Viscerotrophic velogenic ND has occasionally been found in the United States and is thought to have arrived in smuggled birds including pet birds. Neurotropic velogenic ND appears to cycle in flocks of cormorants found in some areas of the upper Great Lakes.

Clinical signs and lesions

As with AI, clinical signs depend on the pathogenicity of the infecting ND virus. Birds infected with lentogenic strains show mild or no respiratory signs, no CNS signs and no mortality. Reddening of the tracheal mucosa may be present in birds dying from other causes. Lesions of bacterial septicemia, such as fibrinous polyserositis and vasculitis, may be present in secondary bacterial infections. Birds infected with the mesogenic strains have moderate respiratory signs such as coughing, rattling when breathing, CNS signs, and general signs of illness such as inactivity, reluctance to move, and a "puffed up" appearance and there may be some mortality in the flock. Lesions with mesogenic strains consist of reddening of the trachea, and lungs may be red and moist. There typically are no gross CNS lesions. Lesions of secondary bacterial infection may be present. Birds infected with velogenic strains are often found dead without previous clinical signs. Lesions of velogenic ND typically consist of hemorrhages of the gastrointestinal tract including esophagus, proventriculus, small and large intestine, and cecal tonsils. There may be facial edema and hemorrhage of the conjunctiva.

Transmission route

The virus is typically transmitted by aerosol route from respiratory tract secretions but the virus may also be transmitted by fecal/oral route and, as with other avian pathogens, through fomites.

Diagnostic tests

Diagnosis of ND must be confirmed via testing, typically performed at diagnostic laboratories. National Animal

Health Laboratory Network (NAHLN) laboratories are equipped with real time polymerase chain reaction (rtPCR) for ND virus matrix detection and fusion gene detection to predict velogenic character. Further classification should be performed by more classical methods at the National Veterinary Services Laboratory in Ames, Iowa.

Differential diagnosis

Avian influenza, septicemic *Pasteurella multocida*, power failure in confined poultry, toxicant exposure and predation for vvND, Marek's disease and toxicant exposure for paresis and paralysis. Other respiratory diseases should be considered in the differential for infections by milder strains of ND.

Prevention and control

Vaccination for ND is not necessary in backyard flocks unless there is a known exposure in the area. There are many relatively inexpensive and effective modified live vaccines available for ND. ND vaccines typically include infectious bronchitis vaccine virus as well. Vaccines should be applied at least twice to develop effective immunity. Poultry vaccines are typically applied through mass vaccination procedures including spraying of the vaccine or adding it in the drinking water. In backyard situations, water vaccination would be the easiest, most practical method of application. The B1 strain of ND virus is usually given for the first vaccine because it is a milder strain and causes little vaccine reaction. The second (booster) vaccine usually consists of La Sota ND virus, which produces better immunity but may cause more vaccine reaction.

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Respiratory Disease

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Respiratory diseases common to backyard poultry

There are many respiratory diseases of poultry. Not all of them are covered in this chapter because the chance of them occurring in backyard poultry is highly unlikely. This chapter is divided into two parts. Respiratory diseases that are common in backyard poultry (along with their occurrence) and those respiratory diseases that are not common in backyard poultry but could occur. This chapter does not include all of the respiratory diseases of poultry, so for further information the reader is referred to a more definitive text such as *Diseases of Poultry*, D. E. Swayne editor (2013).

Non-specific respiratory disease (extremely common)

This condition is probably the most common respiratory disease of small flocks.

Clinical history

In the spring or fall of the year, when the weather is changing, the author receives many calls from small flock poultry owners concerned about their chickens rattling when they breathe. Snicking, which is similar to a reverse sneeze, sneezing and, rarely, coughing are also observed in some flocks. The flock continues to be active and feed and water intake remains normal.

Causative agent

There is no known specific cause for this condition. Typically, none of these birds ever make it to necropsy or have diagnostic work performed. Likely causes for this condition include changes in the birds' local environment, such as dust or high levels of ammonia, but more than likely there is a change in the resident bacterial flora of the respiratory tract. This observation is based upon response to treatment (see Section "Prevention and control").

Clinical signs and lesions

Typical clinical signs are described as all or some of the following: Tracheal rales, snicks, sneezes, and coughing. The differentiating feature of this syndrome as compared to infectious respiratory disease is that the affected birds continue to be alert, active, and continue to eat and drink.

Transmission route

No transmission routes are known.

Diagnostic tests

There is no known diagnostic test. A practitioner could proactively perform tracheal swabs of a normal flock and analyze the sample for the resident microflora including resident mycoplasmas. When or if the flock develops this syndrome, the tracheal swabs could be repeated and test results between the normal and affected flock compared.

Differential diagnosis

Mycoplasmosis.

Prevention and control

Maintain optimum environmental conditions. It is difficult to maintain proper humidity levels in manually-ventilated or naturally-ventilated poultry houses when there are huge temperature swings.

During the spring and fall, temperature swings can be as large as 40°F from morning to evening. This large temperature swing allows increases in poultry house humidity and ammonia levels because ventilation is typically set for the cool temperatures and not adjusted or adjusted rapidly enough to match the outside temperature. The only way to control poultry house moisture and litter moisture is through ventilation. Cooler air is brought into the house where it is warmed and picks up moisture. The warm moist air has to be removed from the poultry house to remove the moisture. Build-up of moisture in the litter allows release of ammonia from poultry manure. Therefore, proper ventilation during these periods is extremely critical.

If untreated, the respiratory signs usually subside in 7–10 days. If the flock is treated with antibiotics, typically a member of the tetracycline family, the respiratory signs usually subside 3 days after treatment is initiated.

Mycoplasmosis (very common)

There are many mycoplasmas that infect poultry [1]. Not all mycoplasmas cause disease and many are considered commensal organisms. Furthermore, not all mycoplasmas cause respiratory disease. Many mycoplasmas that cause overt diseases in other species, such as turkeys and peafowl, can be silent infections in chickens. In addition to spread from animal to animal, mycoplasmas are typically passed from the hen to the chick or poult through the egg. This phenomenon is known as transovarial transmission. The lack of overt clinical signs in chickens combined with transovarial transmission and the ease of spread from bird to bird perpetuates these infections in backyard poultry flocks.

Mycoplasmosis resulting from *Mycoplasma gallisepticum* (MG)
Clinical history
Clinical history varies with the type of poultry infected and whether or not the infection is complicated by other organisms. Uncomplicated infections with *Mycoplasma gallisepticum* (MG) in meat- and egg-type chickens are typically silent with no obvious clinical disease. Secondary infections with *Escherichia coli* (E. coli) typically causes elevated flock mortality. In broiler chickens, this condition is typically known as Chronic Respiratory Disease (CRD). In turkeys and peafowl (peacocks and peahens), infection with MG alone typically presents as dilated infraorbital sinuses and owners complain of birds with puffy or swollen faces. With secondary E. coli infections there is an elevated death loss within the

flock. Clinical history of mycoplasma infections often include the recent addition of new poultry or adding birds back to the flock after they have been to a fair or exhibition.

Clinical signs and lesions
In uncomplicated infections in broiler- and egg-type chickens there is a cough at most. There may be tracheal rales and/or snicks and sneezes. The lesion of uncomplicated MG presents as small to moderate amounts of yellow frothy material (proteinaceous exudate) within the air sacs; in other words, a mild airsacculitis. With secondary E. coli infections, overt illness results and birds may cough, have decreased activity, have their contour feathers extended so they have a "puffed up" appearance, and appear sleepy and reluctant to move. There may be elevated flock mortality. Gross lesions of CRD present as sheets of white to yellow material (fibrin) over the surfaces of the pericardial sac, liver, and air sacs (fibrinous polyserositis).

In turkeys and peafowl, where the disease is called infectious sinusitis, the birds' infraorbital sinuses are often greatly distended. Pushing on the swollen sinuses may cause thick mucus to flow into the bird's mouth through the choanal cleft. If the sinuses are lanced or opened at necropsy, thick stringy mucus exudes from the interior. With secondary bacterial infection, the character of the sinus exudate may change to more caseous.

Transmission route
Transovarial or aerosol are the transmission routes. Mycoplasmas are easily carried on peoples clothing and shoes. There have been cases reported of aerosol spread as far as one half mile from an infected premise.

Diagnostic tests
Culture and identification, PCR, and/or serology are used. Culture is the gold standard for this organism.

Differential diagnosis
Pathogenic E. coli, Fowl cholera, infectious bronchitis, *Mycoplasma synoviae* (MS), turkey viral rhinotracheitis, or swollen head syndrome.

Prevention and control
Only buy replacement birds from flocks that are known to be MG free. Establish a quarantine procedure for the flock, where birds that are purchased to add to the flock are kept in a separate area to ensure that they are free of disease. Quarantine procedures work to prevent not only mycoplasmas but many other diseases

as well. Many small flock owners inadvertently cause their flocks to become infected with MG when they put new birds in their flock immediately after buying birds at sales and swap meets, or someone gives them some "free" birds. Commercial vaccines are available; however, some of them (F-strain) can cause clinical disease in turkeys. Purchase only mycoplasma free poultry. Tylosin and tetracyclines have been effective at diminishing the effects of infection, although no antibiotic totally eliminates the organism. Clinical signs often reoccur after discontinuing antibiotic treatment.

Mycoplasmosis resulting from *Mycoplasma synoviae* (MS)
Clinical history
Chickens develop a snick, sneeze, and/or tracheal rales soon after being added to an existing flock of chickens. MS is uncommon to rare in backyard poultry of the Midwest. In general, almost all commercial egg laying chickens are infected with MS after being placed in the egg laying house. MS does not typically cause overt illness or production problems. The opposite is true in meat-type chickens and turkeys. When found in meat birds, multiple steps are taken to eliminate it from flocks because lameness, synovitis, and other production issues ensue.

Clinical signs and lesions
Clinical signs consist of a snick, sneeze, and/or tracheal rale. If an animal is sacrificed at this time, there may be a mild airsacculitis with a frothy exudate within the air sacs. In meat birds, swelling of the hock joints may be present.

Transmission route
This disease is transmitted by the aerosol and transovarial route.

Diagnostic tests
Culture and identification, PCR, and/or serology are used.

Differential diagnosis
Mycoplasma gallisepticum (MG), infectious bronchitis, or Newcastle disease.

Prevention and control
Practice good biosecurity and only purchase mycoplasma-free poultry. Tylosin and tetracyclines have been effective at diminishing the effects of infection, although no antibiotic totally eliminates the organism.

Infectious coryza (common in the southern United States and in California but rare in the Midwest)

Do not confuse this disease, infectious coryza, with the term "coryza" used through the years by poultry people to describe an upper respiratory infection. Infectious coryza is a defined disease phenomenon [2].

Clinical history
Acute death in a flock of chickens, pheasants or guinea fowl or the development of sick poultry with oculonasal discharge, facial edema, and/or swollen infraorbital sinuses (Figure 10.1).

Causative agent
Avibacterium (*Hemophilus*) *paragallinarum*.

Clinical signs and lesions
Acute death or the development of respiratory signs including sick poultry with oculonasal discharge, facial edema, and/or swollen infraorbital sinuses. Infraorbital sinuses may contain mucus or a hard yellow caseous material.

Transmission route
This disease is transmitted by aerosol, ingestion, and/or people's clothing. The source of the bacteria is typically thought to be chronically infected sick or asymptomatic carriers.

Diagnostic tests
Culture and identification are the tests used. To culture this organism, it is important to use the correct media or

Figure 10.1 Chicken with swollen infraorbital sinuses due to *Avibacterium (Hemophilus) paragallinarum*. (Source: Photograph courtesy of University of Tennessee pathology department website http://vetgrosspath.utk.edu.)

inform the laboratory that *A. paragallinarum* is suspected because it requires factor V for growth.

Differential diagnosis

Fowl cholera (*P. multocida*), secondary bacterial infections following infections by mycoplasmas, ornithobacteria infection or swollen head syndrome (extremely rare in the United States)

Prevention and control

Depopulation to remove any disease carriers is necessary. Cleaning and disinfection should be followed by a period, typically 3 weeks, where no poultry are allowed on the premise. This may also work to control mycoplasmosis. It is important to restock with *A. paragallinarum*-free stock. Treatment with sulfonamides, tetracycline, or erythromycin may be of some immediate help. See Chapter 16 on removing the caseous material from the infraorbital sinus. Vaccination may also be used as a preventative although serotype-specific (A, B, or C) vaccines should match the serotype of the infecting bacteria otherwise vaccine failure may be expected.

Fowl cholera (very common)

Clinical history

Fowl cholera, in its septicemic form, may be seen in small flocks as mortality events in chickens and turkeys. The septicemic form is more common in turkeys than in chickens. In chickens, where it causes accumulation of a hard inflammatory exudate leading to subcutaneous masses, swollen eyes, ears, or wattles, it is a chronic disease [3].

Causative agent

Pasteurella multocida.

Clinical signs and lesions

Clinical signs consist of death without premonitory signs or subcutaneous masses, swollen eyes, ears or wattles. Lesions of septicemia take the form of fibrin in multiple body cavities. Egg contents in the coelomic cavity of egg-laying birds that have been found dead has been described. Subcutaneous masses and swollen structures, on cross section, contain large amounts of yellow, easily-crumbled material. In turkeys, a fibrinous bronchopneumonia is commonly found on necropsy.

Transmission route

Pasteurella multocida is often introduced in a flock through bites from either cats or rats. Cats and rats carry *Pasteurella multocida* in their mouths as a commensal organism. Once in the flock, it can be spread through cannibalism and shed through bodily discharges.

Diagnostic tests

Culture and sensitivity tests are used.

Differential diagnosis

Septicemias caused by *Escherichia coli* or *Avibacterium paragallinarum*.

Prevention and control

Exclude cats and rats from poultry flocks. Commercial vaccines are available. It is important to determine the serotype of the infecting *Pasteurella multocida* because some vaccines are serotype specific. Tetracyclines and sulfa drugs should be used during disease breaks. Antibiotic treatment is futile in the chronic form of the disease.

Infectious laryngotracheitis (not uncommon)

Clinical history

Death loss, which can be dramatic, is often the first indication of infectious laryngotracheitis (ILT). The mortality rate may double each day the disease persists in a flock. Disease and mortality typically occur 7–10 days after returning from a poultry exhibit (county fairs, swap meets, breed shows, etc.) or after adding new birds to a flock. This disease is a disease of chickens but can also cause illness in flocks of peafowl and pheasants. Turkeys have only been infected experimentally. This disease is common in fairs and exhibitions where vaccinated and non-vaccinated birds are co-mingled.

Causative agent

Infectious laryngotracheitis, also referred to as LT or ILT, is caused by *Gallid herpesvirus* 1. Clinical disease can occur as a result of wild-type viruses or from modified live virus vaccines. Vaccine viruses can cause disease as it passes from bird to bird in a vaccinated flock, or from previously vaccinated birds when vaccine virus is shed and spread to non-vaccinated birds.

Clinical signs and lesions

Often the first clinical sign noticed by flock owners is mortality in their flock. The mortality rate in infected flocks can be explosive. Clinical signs can consist of death without any premonitory signs, coughing, head

shaking, and dyspnea, blood on the mouth, feathers and on chicken coop walls. Dyspnea typically consists of "pump handle" breathing. For example, when affected chickens inhale, they raise their head high. When they exhale, they lower their head. Thus, the movement mimics an old fashioned well pump handle. Lesions consist of blood alone, fibrin alone, or a mixture of blood and fibrin within the larynx and proximal one third of the trachea. Some cases of ILT may have conjunctivitis in addition to the tracheal lesions. In mild cases of ILT, a transmissible conjunctivitis may be the only presenting complaint.

Transmission route

Aerosol exposure is the most common route of infection. ILT virus is transmitted by infected birds, from previously infected birds that have recovered, or birds previously vaccinated with modified live vaccines. ILT is also known to be transmitted via fomites including clothing, shoes, and equipment.

Diagnostic tests

Tests include histopathology, virus isolation, and PCR.

Differential diagnosis

Viscerotrophic velogenic Newcastle disease, highly pathogenic avian influenza, septicemic pasteurellosis, acute infectious coryza, or infectious bronchitis.

Prevention and control

Do NOT use modified live ILT vaccines because vaccination can result in latently infected carrier birds [5]. If vaccines are warranted, only genetically modified pox or turkey herpes virus (Marek's) vaccines should be used. These vaccines contain only the protective portion of the ILT virus and not the whole virus. Genetically modified ILT vaccine is best used in chickens that are taken to poultry exhibits. Modified live ILT vaccines should not be used in backyard poultry. Biosecurity is also important in preventing ILT.

Respiratory diseases not common to backyard poultry

Infectious bronchitis (rarely seen)

Clinical history

Chickens show respiratory signs such as sneezing, snicking, tracheal rales, and/or cough [6]. Typically, affected chickens appear sick with reluctance to move and have a puffed up appearance. If they are laying eggs, the eggs may have a hard shell with a wrinkled appearance (Soft shells, shell-less eggs, and wrinkled eggs may be seen when birds are first coming into or going out of egg production).

Causative agent

Coronavirus.

Clinical signs and lesions

Sick chickens are reluctant to move, have a puffed up appearance, and may appear sleepy. Lesions of uncomplicated infectious bronchitis may be hyperemia of the caudal one third of the trachea. There may or may not be an increased amount of mucus within the trachea. Infectious bronchitis is often complicated by a secondary infection with E. coli. In these cases, lesions consist of bacterial septicemia with fibrin deposits over the pericardial sac, liver capsule, and within the body cavity.

Transmission route

Aerosol and/or oral routes.

Diagnostic tests

High titers in non-vaccinated birds, virus isolation, and PCR are used.

Differential diagnosis

Mycoplasmosis, mild strains of Newcastle disease, low pathogenic avian influenza, or infectious coryza.

Prevention and control

Once identified as a flock problem, future flocks should be vaccinated with a serotype that protects against the field strain. Tetracycline or sulfa drugs could be used in cases of suspected secondary bacterial infections.

Pox (diptherhetic, wet form) (uncommon)
Clinical history

Increased mortality in a flock of chickens. This disease appears to spread slowly through the flock. Birds may exhibit dyspnea prior to death.

Causative agent

Pox virus, most likely fowl pox. There are many different strains of pox viruses and they are typically named for the species that they infect naturally [6]. In poultry, fowl pox can infect both chickens and turkeys, while turkey pox only infects turkeys. In chickens, wet pox can occur with fowl pox virus alone but is often the result of a dual infection with infectious laryngotracheitis virus and fowl pox virus.

Figure 10.2   A 1-year-old Welsummer hen with fibrinonecrotic material involving the oropharynx and tongue consistent with the wet form of fowl pox. Eosinophilic intracytoplasmic inclusion bodies were demonstrated in the tissue of the tongue. (Source: Photograph courtesy of Cheryl Greenacre, University of Tennessee.)

Clinical signs and lesions

Dyspnea and increased flock mortality are the typical presenting clinical signs in wet pox. Wet pox causes lesions on the wet tissues (mucus membranes). It is called dry pox when it causes lesions on the dry skin, typically the non-feathered areas of the body. The lesions of wet pox as they relate to respiratory disease show as a polypoid mass at the opening of the larynx and proximal trachea. Birds die when this mass occludes the trachea, thus suffocating the chicken (Figures 10.2, 10.3).

Transmission route

Pox virus requires a break in the epithelium to infect an animal. This may occur as a result of dust or a different respiratory pathogen that disrupts the epithelium. Thus, during ILT infection, a break in the epithelium occurs and pox virus infects the epithelium at the break.

Figure 10.3   The same hen as in Figure 10.2 also had bubbly ocular discharge and severe dyspnea, probably due to concurrent bacterial infection (see video on website). (Source: Photograph courtesy of Cheryl Greenacre, University of Tennessee.)

Diagnostic tests

Histopathology, virus isolation, and PCR are used.

Differential diagnosis

Infectious laryngotracheitis.

Prevention and control

Vaccination and control of biting insects are important because pox virus can be spread from bird to bird by biting insects. The disease spread is usually slow enough that one can vaccinate in the face of an outbreak to prevent the spread of pox within the flock.

Gape worm (uncommon in poultry, common in game birds)
Clinical history

Increased flock mortality in young poultry. Game birds, such as pheasants, quail, and partridges, are most often infected. Any back yard poultry may be at risk for this infection; however, it has only been reported in chicken, turkeys, guinea fowl, pea fowl, and geese. Affected live birds may breathe with their mouths open. They may shake their heads or may drag their open mouth on the ground as they walk.

Causative agent

Syngamus trachea (see Chapter 6).

Clinical signs and lesions

Increased flock mortality in young birds. Open mouth breathing, head shaking, and/or walking with their open mouth on the ground. Lesions consist of a reddish

nematode parasite attached to the tracheal mucosa. Sites of attachment may consist of raised white nodules. The parasite may have a "Y" formation because the female parasite envelopes the male parasite as he is attached to the tracheal mucosa. In dead birds, the parasites may block the trachea (see Chapter 6).

Transmission route
The infection is spread directly from bird to bird via ingestion of embryonated ova or larva or indirectly through the ingestion of an earthworm or insects containing the larva.

Diagnostic tests
Gross lesions are diagnostic.

Differential diagnosis
Any respiratory pathogen causing open mouth breathing such as wet pox and infectious laryngotracheitis.

Prevention and control
Control of earthworms and other insect vectors is important in preventing the disease and its carry over between flocks. Treatment is off label. Thiabendazole, mebendazole, cambendazole, fenbendazole, and levamisole have shown effectiveness.

Aspergillosis (uncommon)
Clinical history
In young birds this disease is called brooder pneumonia because the disease occurs during the time that birds are being brooded and the source of infection may be the brooding environment, which is usually dark, warm and moist. Typically, there is an increased flock mortality within the first two weeks of life [7]. In adult birds, the disease usually presents as one or a few birds with chronic illness and weight loss followed by death. Geese and turkeys are most commonly affected. Ducks are also susceptible to infection.

Causative agent
Aspergillus fumigatus most commonly causes this disease, although other species may cause the same lesions.

Clinical signs and lesions
In young birds, clinical signs may be open-mouth breathing and/or an increased mortality rate. Lesions in young birds consist of yellow, seed-like granules of granulomatous inflammation within the lung parenchyma. In older birds, clinical signs consist of chronic illness with weight loss followed by death. Lesions in these birds typically consist of a fungal airsacculitis. The air sacs, most commonly the cranial thoracic air sacs next to the lungs, are filled with large amounts of yellow, easily-crumbled material. The surface may or may not have a gray fuzzy covering, which consists of fruiting bodies. In birds with acute death without premonitory signs, a plug of fibrin and fungal mycelia may be found blocking the tracheal bifurcation (syrinx).

Transmission route
Aerosol. In brooder pneumonia, birds can be infected while they are in the hatchery and an infected egg explodes, spraying its contents and fungal spores throughout the incubator or setter/hatcher, or they can be infected during the brooding phase. As stated above, the brooder is an ideal place for fungi to grow. The source of fungal spores in the brooder is hard wood shavings: It is thought that the mycelia and spores are picked up on the trees when they are being processed in the forest.

Diagnostic tests
Gross lesions provide a presumptive diagnosis which can be confirmed by histopathology. A tease prep, using lactophenol blue, may be performed on the fuzzy growth on the air sacs.

Differential diagnosis
Fowl cholera and E. coli septicemia.

Prevention and control
Frequent candling of eggs in incubators and setters is important to detect embryos that may have died as a result of fungal infection. In older turkeys, dust control is important in preventing this condition. Providing environmental conditions that are not warm and damp is important in preventing this condition in ducks and geese.

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Musculoskeletal Diseases

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Introduction

Causes of lameness in backyard poultry vary greatly and can be associated with abnormalities in any part of the rear limb, but can also be associated with disorders of the central or peripheral nervous system, the reproductive system or just from generalized illness (Figure 11.1). Diseases can include localized infection, inflammation, trauma, or generalized disease presenting as lameness. A thorough history and physical examination is necessary, including a neurological examination, to arrive at a diagnosis. Nerve blocks are helpful to localize a lesion, or in cases of multiple lesions, can help differentiate which lesion is causing more pain. One of the most helpful tools is radiography. The reader is referred to a general text on avian medicine for descriptions on performing and interpreting avian radiography in general [1].

Common causes of lameness in backyard poultry

Trauma

Trauma is a very common presenting complaint in backyard poultry, either as a result of predator attack (dog, raccoon, etc.), being kicked by a horse, being accidently stepped on by the owner, or getting caught in some part of the enclosure and struggling (Figure 11.2). Ducks can present for toe or foot trauma after being bitten by a snapping turtle (Figures 11.3, 11.4). Wound management is covered in the Dermatological Diseases chapter of this book.

Fracture repair

Radiographs should be taken before and after fracture repair and during the healing process to determine the type of repair needed, assess stability of the fracture after repair, assess the integrity of any bandage or hardware used, and to check for evidence of osteomyelitis (Figures 11.5–11.9). Frequent follow-up visits also allow for readjustments in the treatment plan.

Avian bones differ from mammalian bones in that they have a thin, brittle cortex, may be pneumatic (humerus and femur), and heal by endosteal, not necessarily periosteal, new bone growth. Generally, avian fractures heal faster than mammalian fractures at 2–3 weeks versus 4–6 weeks. The goal is to align the fracture as best as possible so that weight bearing can occur almost immediately so as not to cause excess stress and load on the unaffected limb. Pododermatitis needs to be prevented in the non-affected limb/foot while the affected limb heals (Fig. 11.10). This is best done by having the affected limb be usable as soon as possible, providing soft substrate, and watching for early signs of pododermatitis and taking steps to treat it early.

Ideally fractures need to have both rotational and bending forces controlled with the bone in as near to normal apposition as possible. External fixation pins, with or without an intramedullary (IM) pin tie-in, usually provide the best fixation for most fractures in birds and can also be partially destabilized part way through the healing process to strengthen the bone and promote maximal healing. Birds have thin, brittle cortices; therefore positive profile pins should be used for external fixation, rather than negative profile pins, in

Figure 11.1 A 2-year-old Speckled Sussex hen presented for sudden onset of reluctance to ambulate. Initially it was suspected that leg lameness was occurring but a thorough physical examination revealed an enlarged doughy coelomic cavity consistent with egg related peritonitis. Within one week of oral antibiotics (trimethoprim sulfamethoxizole) she was ambulating normally and continues to do well for over a year.

Figure 11.2 Adult Guinea fowl attacked by a dog presented with a de-gloving injury over the left leg extending into the inguinal area. Wounds in a high motion area such as the inguinal area require long term, sometimes months of, wound care management.

Figure 11.3 An adult wild Canada goose presented with trauma to the webbing of one foot due to suspected snapping turtle bite.

Figure 11.4 The goose in Figure 11.3 after surgery to close the skin wounds. Care was taken to separately suture the two layers of skin that make up the webbing of the foot.

order to provide greater purchase of the bone, increase stability, and lessen the chance of pin loosening.

An IM pin alone counteracts bending forces but not rotational forces; therefore a splint, cast, or a Robert-Jones bandage is applied in conjunction with an IM pin. The IM pin may pass through or near a joint; therefore a smooth pin without threads is used to cause the least amount of trauma.

The connecting bar used to connect the external fixator pins can be made of light-weight materials such

as polymethylmethacrolate (PMM), acrylic, plumbers putty, epoxy, or casting material. The freshly mixed soft PMM is usually syringed into a drinking straw or penrose tube that has been impaled on the

Figure 11.5 A Welsummer hen endotracheally intubated under isoflurane anesthesia for a radiograph.

Figure 11.7 Lateral radiograph of the left leg of a <1-year-old hen with an oblique distal tibiotarsal fracture that is close to but not involving the hock joint (tibiotarsal tarsometatarsal joint or intertarsal joint).

Figure 11.6 Ventrodorsal radiograph of the left leg of a <1-year-old hen with an oblique distal tibiotarsal fracture that is close to but not involving the hock joint (tibiotarsal tarsometatarsal joint or intertarsal joint).

external fixator pins and held in place until the PMM hardens.

The integrity of the splint is directly proportional to its ability to counteract rotational being kicked forces. Place the bandage in flexion in as close to a normal position as possible so the bird can have immediate use of the limb, less abnormal forces placed on nearby joints, and rest with the leg underneath the body rather than out to the side (Figure 11.11). External coaptation (splint or Robert-Jones bandage) is sometimes used alone, but it is not ideal because there is a risk of bending and rotational forces, causing less than ideal healing or maybe even a false joint or malunion healing.

Other methods, such as physical therapy, used in mammals to complement fracture repair can also be used in avian fracture repair (Figures 11.12, 11.13, and see website for video of chicken trained to go over a step).

Pododermatitis (or bumblefoot)

Pododermatitis or bumblefoot has many causes including excess weight bearing from obesity or unequal

Figure 11.8   Ventrodorsal radiograph of the left leg of the chicken in Figure 11.6 immediately after open reduction. Only an intramedullary pin (IM) and external coaptation were used to repair this fracture since the distal fragment was deemed too small to sustain one external fixation pin and the forces that would have been exerted upon it. An external fixator, IM pin tie-in, and external coaptation combined could also have been used. The redundant part of the pin has not yet been trimmed.

Figure 11.9   Lateral radiograph of the left leg of the chicken in Figure 11.6 immediately after open reduction. Only an intramedullary pin (IM) and external coaptation were used to repair this fracture since the distal fragment was deemed too small to sustain one external fixation pin and the forces that would have been exerted upon it. An external fixator, IM pin tie-in, and external coaptation combined could also have been used. The redundant part of the pin has not yet been trimmed.

weight bearing between the two feet as a result of lameness of one foot, causing less weight to be placed on that foot and more on the contralateral foot, or from abnormal abrasions of the plantar surface from inappropriate substrate (too sharp or rough, wire, etc.), decreased blood supply to the foot (sometimes from lack of exercise), trauma, or standing for prolonged periods of time, especially in ducks that are not provided with adequate swimming opportunities.

Pododermatitis is divided into varying grades depending on the literature source used, but generally includes mild, moderate, and severe grades with the severe grades including osteomyelitis (Figures 11.14–11.19).

Obtain a thorough history including environment and substrate. Perform a thorough physical examination and determine if any other factors are present that may be contributing to or causing the pododermatitis. Perform radiographs to determine whether osteomyelitis is present.

For mild cases of pododermatitis, changing to a softer substrate, exercise to increase blood supply to the foot, soaking the affected foot in warm water, and the use of keratin softeners may be all that is needed. The foot can also be soaked in a dilute chlorhexadine or iodine solution, but realize that chlorhexadine does not kill *Pseudomonas* spp. organisms. If there is a break in the skin, then soaking in a solution called Tricide-Neo ™ with an antibiotic can speed healing.

If the tissues of the foot are severely swollen then surgery may be indicated to remove pus or a large callous, but it must be performed under anesthesia with pain relievers administered. Be prepared for possible hemorrhage from the surgery site. If surgery is performed and the lesion is opened then an aerobic

Figure 11.10 A 7-month-old Pekin duck presented with a swollen right hock with evidence of pododermatitis in the left foot from excessive weight bearing in the left foot to compensate for lameness in the right hock. At necropsy the right hock was found to be septic.

Figure 11.11 Same chicken from Figure 11.6 showing external coaptation used in conjunction with the IM pin. Note the flexed usable position of the bandaged leg (Source: Photo courtesy of Dr. Katherine Baine.)

Figure 11.12 Same chicken as Figure 11.6 showing physical therapy in the form of gently extending the toes since later this bird had some contracture (Source: Photo courtesy of Dr. Katherine Baine.)

and anaerobic culture should be performed. Treatment can include parenteral antibiotics, wound management, and bandaging (Figures 11.20–11.28).

Septic joint

There are many possible causes of a septic joint, but the most common include *Staphylococcus aureus*, *Escherichia coli*, *Pasteurella multocida*, *Salmonella gallinarium*, *Mycoplasma synoviae*, and reoviruses [2]. The history may include an initial trauma, diarrhea, or respiratory disease that allowed the bacteria access to the joint either directly or through hematogenous spread. The bird usually presents with lameness either in one or multiple joints and an enlarged, warm joint (Figure 11.29). Aerobic and anaerobic culture, and maybe even Mycoplasma culture or serology, aids in the diagnosis and choice of antibiotic. Radiographs help determine the degree of osteomyelitis and the duration of antibiotic therapy needed. The antibiotic chosen depends mainly on the culture, but thought must be given to treating a chicken, which even if it is a pet is covered by laws regarding antibiotic use in a food animal (see Chapter 20).

Prognosis is fair to poor and in many cases long term therapy results in a joint with arthrodesis and limited

Figure 11.13 Same chicken as Figure 11.6 showing physical therapy in the form of training the chicken to step up to help extend the toes back to normal. See video on this book's accompanying website (Source: Photo courtesy of Dr. Katherine Baine.)

Figure 11.15 White leghorn chicken with mild pododermatitis. Note the 3×2 mm hard callous on the plantar surface of the metatarsal pad.

Figure 11.14 Normal foot of a white leghorn chicken. Note the clean, intact, textured surface.

range of motion, but enough for the bird to walk around without putting undue stress and weight on the unaffected limb.

Marek's disease

Marek's disease (MD or MDV) is very common and affects only chickens. The causative agent is an alpha herpesvirus, but there three serotypes, four pathotypes, and many different strains of varying pathogenicity [3]. The prototype virus, the one that is associated with virulence or pathogenicity, is technically serotype 1, also known as *Gallid herpesvirus* 2 [3]. Serotype 1 is further subdivided into four pathotypes depending on virulence (mild, virulent, very virulent, and very virulent plus) [3]. Marek's disease (MD) causes a variety of clinical signs that have been divided into distinct pathological syndromes, including the one most people think of when they hear the words Marek's disease: Fowl paralysis, but MD can also present as MD lymphoma, persistent neurological disease, skin leukosis, and ocular leukosis. Clinical signs of MD are generally seen in birds that are 10–20 weeks of age, but can be seen as young

Figure 11.16 White leghorn chicken with moderate pododermatitis. Note the 5 × 5 mm hard callous with associated 10 × 10 mm swelling on the plantar surface of the metatarsal pad.

Figure 11.17 White leghorn chicken with severe pododermatitis. Note the 20 × 20 mm hard callous with associated 30 × 30 mm swelling on the plantar surface of the metatarsal pad.

as 4 weeks of age. Be sure to obtain the exact age of the bird in weeks, as this helps to differentiate between MD and a very similar disease caused by avian leukosis virus.

MD is highly contagious and is transmitted horizontally either directly or indirectly by contact with virus via the airborne route. It can easily spread via fomites. Common sources are feathers, feather dander, secretions and droppings (litter). Once a bird is infected they shed the virus indefinitely. The cell free MD virus remains infectious for 4–8 months at room temperature and for at least 10 years at 4°C (39°F), which in a natural environment is practically indefinitely. Cleaning a plastic or metal cage of all debris and then disinfecting with a common disinfectant, such as a 1:10 dilution of household bleach, for 10 minutes, inactivates the organism [3].

Fowl paralysis (range paralysis)
Clinical signs of unilateral paresis or paralysis are seen 3 – 4 weeks post infection usually between the ages of 6–12 weeks but can be seen as young as 3–4 weeks of age and much older than 12 weeks. The typical stance

is one leg stretched forward and the other pointing back (Figure 11.30). The classic gross lesion that occurs with fowl paralysis is a unilateral enlargement of the sciatic plexus (the ischiadic nerve). A similar, but distinct, syndrome is called transient paralysis, which presents with a flaccid neck in young chickens. Sometimes birds that survive fowl paralysis go on to develop torticollis or nervous ticks and this is known as persistent neurological disease [3].

Marek's disease lymphoma (MD lymphoma)
Outward clinical signs of MD lymphoma are usually subtle, even with extensive neoplastic involvement, and include weight loss, pale comb, anorexia, and diarrhea. The whitish to grayish focal nodular tumors or diffuse infiltration of mononuclear cells can involve a variety of tissues as in avian leukosis virus (ALV) and include ovary, lung, heart, mesentery, kidney, liver, spleen, adrenal gland, pancreas, proventriculus, intestine, iris, skeletal muscle, and skin (Figures 11.31, 11.32). When mononuclear infiltrates are found in the iris, the affected iris turns a pale tan to gray color instead of the

Figure 11.18 Pekin duck with mild pododermatitis. Note the 1 × 1 mm erosion on the digital pad of the #4 toe.

Figure 11.19 Pekin duck with severe pododermatitis. Note the 2 3 × 3 mm erosions on the plantar surface (digital pads) of the #3 toe and the 10 × 10 mm hard callous on the metatarsal pad.

usual yellow and this is known as ocular lymphoma or "gray eye" (Figure 11.33). Skin lymphoma presents as multiple small (2 × 2 mm) nodules associated with a feather follicle, giving the skin a bumpy appearance. Organ distribution of MD lymphoma lesions is influenced by the genetic strain of the chicken and the strain of the virus.

Testing and prevention of MD

Testing for MD can be performed on 0.5 ml of serum using an inexpensive ELISA test, or PCR or virus isolation can be performed on fresh tissue. This author is most familiar with sending samples to the Poultry Diagnostic and Research Center at the University of Georgia in Athens, Georgia (http://www.vet.uga.edu/avian). Testing is helpful when trying to differentiate between MD lymphoma and ALV.

There is no treatment for MD. Prevention is implemented by administering a polyvalent vaccine in the egg or at 1 day of age. There are several types of vaccine commonly used against MD either individually or in combination and they include a low pathogenic serotype 1, a naturally avirulent Turkey Herpesvirus

(HVT), and serotype 2 virus [3]. When purchasing chicks from a hatchery, always pay the slightly higher cost for a pre-vaccinated chick. Backyard poultry owners can purchase commercially available MD vaccine but they are only available in 1000–2000 dose vials, which must be refrigerated or frozen prior to reconstitution and then used within one hour after reconstitution and the remainder discarded appropriately. Understand that no vaccine is 100% protective and even "vaccinated" chicks can develop the disease. The MD vaccines offer greater than 90% protection, which is considered very effective. Much research has gone into MD resistant strains of chickens, but even a "resistant" strain of chick should be vaccinated because even they can succumb to a virulent strain of MD [3]. Prior to the use of MD vaccines, mortality caused by MD was as high as 60% [3].

Avian leukosis virus (ALV) (including lymphoid leukosis)

Avian leukosis virus (ALV) is also known as the leukosis/sarcoma group of diseases and causes a variety

Figure 11.20 An adult Welsummer hen presented with an abscess ruptured through both the anterior and the plantar surface of the right foot between the third and fourth digit.

Figure 11.22 Foot of the adult Welsummer hen in Figure 11.20 after surgery to remove the abscess and debride the associated necrotic tissue. There was an expected amount of hemorrhage.

Figure 11.21 Another view of the adult Welsummer hen in Figure 11.20 that presented with an abscess ruptured through both the anterior and the plantar surface of the right foot between the third and fourth digit.

Figure 11.23 Foot of the adult Welsummer hen in Figure 11.20 after amikacin impregnated Gelfoam was placed in the lesion.

of tumors in chickens as a result of retroviruses [4]. Lymphoid leukosis (LL) is the most common form of this group of diseases, but many other neoplasia of chickens can also be caused by retroviruses including avian erythroblastosis, fibroma, fibrosarcoma, myoma, myxosarcoma, chondroma, osteoma, osteogenic sarcoma, squamous cell carcinoma, granulosa cell sarcoma, hemangioma, mesothelioma, meningioma, and glioma to name a few [4]. This retrovirus is transmitted both horizontally and vertically. LL typically develops in chickens between 14–40 weeks of age, but can occur later. Clinical signs of LL rarely develop before 14 weeks

of age, which helps differentiate it from MD lymphoma, which usually occurs in younger chickens.

After clinical signs develop, chickens usually die within weeks. The clinical signs are non-specific and include inaapetance, weakness, diarrhea, dehydration, and emaciation. A predominant physical examination finding is a firm coelomic enlargement. Diagnosis is based on serology using ELISA or virus isolation on fresh tissue [4]. At gross necropsy gray to white tumors are observed in the liver and other organs (Figure 11.34). The clinical signs and gross pathological lesions are sometimes difficult to differentiate from those of MD, but LL does not occur before 14 weeks of age, whereas MD usually occurs at 10–12 weeks of age.

Figure 11.24 Foot of the adult Welsummer hen in Figure 11.20 after a polyethylene foam pad was cut to accommodate all four toes and leave a hole over the lesion area so that it could heal with air exposure and no mechanical pressure.

Figure 11.26 Foot of the adult Welsummer hen in Figure 11.25 showing the final bandage with a layer of duct tape on the plantar surface.

Figure 11.25 Foot of the adult Welsummer hen in Figure 11.20 showing how the polyethylene foam pad was kept in place with a layer of cast padding, then wrap.

Figure 11.27 Welsummer hen in Figure 11.25 during soaking of foot in warm chlorhexadine solution at her two week recheck.

There is no treatment and currently no vaccine. The best prevention is to test and cull positive breeder birds.

Reproductive disease

Commonly, older hens with reproductive disease have coelomic distension either as a result of fluid or soft tissue/organ enlargement and they present with lameness resulting from weakness, illness, or simply as a result of an enlarged coelomic cavity that prevents normal ambulation. Perform a thorough physical examination and palpate the coelomic cavity. It should be soft in an egg-laying female, but not enlarged or fluctuant, nor enlarged and firm. If necessary perform radiographs to

confirm enlarged coelomic cavity or to determine its cause. If necessary perform coelomocentesis to determine possible causes of "ascites" or excess coelomic fluid (see Figure 11.1, (see video on accompanying website)).

Valgus deformities

There are many causes of valgus deformities in young chickens and the lateral deviation can be at the intertarsal joint, tibiotarsus, the hip, or a combination of the above. Dyschondroplasia specifically refers to an abnormal persisting accumulation of cartilage at the growth plate common in meat-type chickens, ducks, and turkeys and usually involves the tibiotarsus [5]. Broiler-type breeds commonly have a valgus deformity

Figure 11.28 Welsummer hen in Figure 11.25 2 weeks post-surgery and healing well.

Figure 11.29 Enlarged and warm right hock in a duck due to a septic joint. Note that holding the bird in this position allows easy size comparison between the two hock joints.

Figure 11.30 Two lethargic 10-week-old blue Orpington chicks showing the typical stance of Marek's disease with one leg positioned forwards and the other leg positioned backwards. The diagnosis was confirmed on necropsy with the typical lesion of unilateral sciatic (ischiadic) nerve enlargement. (Source: Photograph courtesy of El Morse.)

Figure 11.31 An 8-week-old chicken (pullet) with Marek's disease with diffuse hepatomegaly and multifocal nodular tumors in the liver. (Source: Photograph courtesy of Dr. Linden Craig, University of Tennessee, Department of Biological and Diagnostic Services.)

at the intertarsal joint and/or the tibiotarsus of unknown cause but it is probably a result of rapid growth [5]. Often treatment includes slowing down the growth of the bird with a lower protein diet, which can be done by adding corn scratch to the diet at no more than 25% of the diet. Splay leg, otherwise known as spraddle leg, is a lateral deviation at the hip that is usually associated with high humidity during incubation [5]. If caught early, lateral deviations of the leg can be bandaged to encourage the leg to a normal, usable position. Frequent, almost daily, bandage changes are necessary to keep up with the growth of the bird, deterioration of the bandage, and reassessing the changing position of the leg.

Perosis (slipped tendon)

Perosis, also known as "slipped tendon," refers to luxation of the gastrocnemius tendon forcing the affected leg into a valgus position with an enlarged hock (Figures 11.35 a–c). Perosis is caused by a deficiency of choline, manganese, or biotin. Supplementing with choline, manganese, and biotin, and suturing or tacking of the tendon sheath to keep the tendon in place have met with variable success [6]. Results of surgery are better if performed when young, early in the clinical signs, and the leg is bandaged in flexion after surgery so that the bird can immediately use the leg and place it under its body. Be careful not to bandage the leg in extension as this may create another deformity.

Figure 11.32 Same 8-week-old pullet as in Figure 11.28 that also had diffuse renalmegaly and multifocal nodular tumors in the kidney due to Marek's disease. (Source: Photograph courtesy of Dr. Linden Craig, University of Tennessee, Department of Biological and Diagnostic Services.)

Figure 11.34 Kidney of a >1-year-old hen with suspected avian leukosis virus (AVL) due to age of the bird and gross and microscopic evidence of multiple organ tumors consistent with AVL. Note the similarity to the kidneys shown in Figure 11.31 of the 8-week-old chick with Marek's disease lymphoma. (Source: Photograph courtesy of Dr. Danielle Reel, University of Tennessee, Department of Biological and Diagnostic Services.)

Figure 11.33 One year old male chicken (rooster) exhibiting the ocular form of Marek's disease with a light colored tan/gray iris due to mononuclear cellular infiltrates as shown here in the eye on the right. The eye on the left is a normal yellowish color. (Source: Photograph courtesy of Dr. Deb Miller, University of Tennessee, Department of Biological and Diagnostic Services.)

Because it occurs at such a young age and the bird is growing fast, frequent rechecks and bandage changes are necessary to provide the best bandage as the patient grows. Prognosis is usually poor.

Less common causes of lameness in backyard poultry

Mycoplasmosis

There are three main species of Mycoplasma that can infect poultry. *Mycoplasma gallisepticum* (MG) causes

respiratory disease in chickens, but an infectious sinusitis in turkeys. *Mycoplasma meleagridis* (MM) causes an air sacculitis and skeletal deformities in turkeys, and *Mycoplasma synoviae* (MS) causes air sacculitis, mild upper respiratory infection and synovitis /lameness in chickens and turkeys. *Mycoplasma gallisepticum* (MG) is seen in backyard flocks and is of concern because it can easily spread to nearby commercial flocks and cause economic devastation for such a flock. Most commercial flocks are MG free. To participate in the National Poultry Improvement Plan (NPIP) a flock needs to be MG free. Transmission takes place through fomites.

Clinical signs relating to lameness caused by MM in turkeys usually occur in 1–6-week-old poults and include bowing, shortening, and twisting of the tarsometatarsal bone and associated hock swelling [7]. Morbidity is about 5–10%, with more males being affected. The main route of transmission is vertical through the egg, but horizontal transmission can also occur either directly or indirectly. There are no vaccines for MM [7].

Clinical signs relating to lameness caused by MS in chickens or turkeys include an exudative synovitis, tenosynovitis, or bursitis of hock joints and foot pads [8] (Figure 11.36). Sometimes the sternal bursa or other joints are affected as well [8]. Morbidity is variable but is typically between 5–15% [8]. The main route of transmission is horizontal through the respiratory tract. A differential diagnosis is a septic joint caused by other bacteria, which tend to grow easily on aerobic and anaerobic culture (see Section "Septic joints"). You can

(a)

(b)

(c)

Figure 11.35 (a), (b), (c) Necropsy of a several week old duckling with perosis (slipped tendon) of the left hock. (a) Note the enlarged left hock with evidence of weight bearing on the hock because of no weight bearing on the foot. (b) Note the lateral deviation of the tendon so that it does not sit within the groove of the distal tibotarsus, and (c) also note the hyperemia and flattening of the condyle (the sutures seen were placed for practice of the surgical technique).

Figure 11.36 An 8-month-old white leghorn chicken with suspected *Mycoplasma synoviae* infection in the left intertarsal joint and the right hock. Note the swollen metatarsal area on the left foot compared to the right foot. This bird's right hock was very swollen (not visible in picture) and at necropsy was worse than the disease occurring in the left foot, hence the bird actually preferred to put its weight on the left leg rather than the right (see video on website).

ask the laboratory to attempt growth of *Mycoplasma* spp. on special media, but it grows slowly and in chronic infections the organism may no longer be present [8]. Radiographs may help because a septic joint resulting from the presence of other bacteria quickly progresses to include osteomyelitis, which is visible on radiographs, whereas MS tends not to do this.

The best prevention is to depopulate and repopulate with clean stock. Treatment can be attempted with antibiotics (tetracycline, spectinomycin, lincomycin, erythromycin, or tylosin), but birds remain carriers for life (see Chapter 20).

Gastrocnemius tendon rupture

Gastronemius tendon rupture is not common in backyard poultry but can occur in meat-type breeds of chickens, typically older than 12 weeks of age. One or both hocks can be affected and the bird presents

sitting on the affected hock or hocks with the toes pointing ventrally [5]. The loose end of the tendon can be palpated bunched up on the posterior surface of the leg cranial to the hock. There is a dark discoloration of hemorrhage under the skin in the affected area, or, if the rupture is over 3 days old, a green discoloration may be present because birds bruise green as a result of a lack of biliverdin reductase. Chronic lesions also have some degree of fibrous tissue. The cause is unknown, but may be related to infection with a reovirus, causing a tenosynovitis. There is no treatment currently described for this disease.

Vitamin E deficiency (encephalomalacia)

Vitamin E deficiency can cause encephalomalacia as well as other diseases such as exudative diathysis and nutritional myopathy in chicks. Encephalomalacia in chicks is characterized by ataxia or paresis but with rapid contraction and relaxation of the legs with forced movements, abnormal head and neck positions, and death [9]. Histopathological lesions include demyelination, neuronal degeneration, and marked hyperemia of meningeal, cerebral, and cerebellar vessels associated with ischemic necrosis [9]. Early treatment can quickly reverse signs, otherwise the prognosis is poor.

Vitamin B1 (thiamine) deficiency (star gazing)

Vitamin B1 (thiamine) deficiency causes "star gazing" in chicks associated with anorexia, weight loss, ruffled feathers, ataxia, ascending paralysis, and opisthotonos. The term "star gazing" describes the typical position, which consists of a drawn back head while sitting on the hocks with the legs flexed, and results from paralysis of the anterior muscles of the neck [9]. Chicks can develop clinical signs as early as 2 weeks of age on a deficient diet, whereas adults take a lot longer. Histopathologically, a myelin degeneration of multiple nerves is observed, as well as hypertrophy of the adrenal glands and edema of the skin. Early treatment with thiamine or vitamin B complex can quickly reverse signs, but chronic cases may be left with permanent damage despite treatment [9,10].

Vitamin B2 (riboflavin) deficiency (curled toe paralysis)

Vitamin B2 (riboflavin) deficiency causes "curled toe paralysis" in chicks, which causes them to sit on their hocks with their toe curled medially. Other signs include weakness, emaciation despite a good appetite, sitting on hocks, reluctance to walk, or walking on hocks and diarrhea. Chicks can develop clinical signs by 12

days of age on a deficient diet. Adults are less likely to show clinical signs. Histopathologically, a demyelinating peripheral neuritis is seen with edema of the ischiatic and brachial nerves. Early treatment with riboflavin or vitamin B complex can reverse signs, but chronic cases may be left with permanent damage despite treatment [9,10].

Botulism (limberneck)

Botulism in backyard poultry usually involves ducks and other waterfowl, but can also occur in other birds, including chickens, pheasants, and turkeys. It occurs as a result of ingestion of the type C exotoxin produced by the *Clostridium botulinum* bacteria often found in decaying meat and vegetation, or in the associated maggots [11]. Clinical signs consist of an ascending flaccid paralysis of skeletal muscle that eventually leads to death by respiratory paralysis or drowning from the inability to keep the head above water. Morbidity and mortality are related; the higher the dose the more acute and severe the signs [12]. A high dose can result in clinical signs within hours, whereas a low dose may be associated with paralysis signs in 1–2 days. If the case is not severe, spontaneous recovery can occur with supportive care. Confirming a diagnosis of botulism is difficult because there are no gross or histopathological signs, but the suspected ingested substance, crop contents, and GI contents can be analyzed for toxins [12]. Treatment with antitoxin may be considered in valuable individual birds because it has been shown to be effective in birds, but it only neutralized the free and extracellular bound toxin [12]. Preventing exposure to decaying meat and vegetation, removing cadavers promptly, and providing fresh food and water are the best preventative measures because both the bacteria and toxin are stable in the environment.

Avian encephalomyelitis (epidemic tremor)

Avian encephalomyelitis (AE) is a disease with worldwide distribution that may be seen occasionally in unvaccinated flocks of chickens, but natural infections have also been documented in pheasants, quail, and turkeys [13–16]. Avian encephalomyelitis is caused by a Hepatovirus in the Picornaviridae family and usually affects chickens at 1–3 weeks of age. Clinical signs include initial depression and then progressive ataxia with tremors of the head and neck, hence the lay name of "epidemic tremors." Birds exposed to the virus after 4 weeks of age are usually asymptomatic [13,14,16]. The morbidity in 1–2 weeks old chicks is about 40–60% with a usual mortality rate of 25%, although it can be as high as 50%. Birds that recover are immune but

have permanent ataxia, and some go on to develop lens opacity (cataracts) weeks later and may become blind [13,15,17].

Transmission is usually vertical from the hen to the egg, hence the importance of vaccinating breeder hens at about 14 weeks of age before they start to lay. Infected laying birds may experience a 5–10% decrease in egg production. Horizontal transmission can also occur via the fecal-oral route to propagate in the intestines of 1–3 weeks old chicks. Infected chicks can shed the virus for 5–21 days post-infection [16].

Gross pathology signs are minimal except for white nodules observed in the muscularis of the ventriculus as a result of massive lymphocytic infiltration [13,15].

Histopathologically, the changes that are strongly suggestive of AE are found in the brain/spinal cord and viscera and include a non-purulent encephalomyelitis with a severe perivascular infiltrate, a ganglionitis of the dorsal root ganglion, and microglioisis [13,15]. Aggregates of lymphocytes are also observed in the proventriculus, ventriculus, pancreas, and myocardium [13,15,16]. The peripheral nervous system is not involved as it is with MD, an important differential diagnosis. Other differential diagnoses include encephalomalacia resulting from vitamin E deficiency, toxins such as lead, Newcastle disease, and eastern equine encephalitis. Because the virus is non-enveloped it is extremely resistant in the environment, lasting months in the soil, and is easily spread by fomites. Antibody tests (ELISA, ID, VN) can be used to determine exposure. An ELISA can be run on 0.5–1.0 ml of serum at laboratories (http://gapoultrylab.org or http://www.usu.edu/uvdl/htm/services/avian-testing) or virus isolation can be performed on fresh brain tissue.

Note

1. Tricide-Neo is manufactured by Molecular Therapeutics, LLC, Athens, GA 30602.

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Dermatological Diseases

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Introduction

Dermatologic disease is sporadic in backyard poultry, and most commonly involves trauma and ectoparasitism. Other infectious skin diseases can occur, but are less frequently encountered in backyard flocks. Table 12.1 provides an overview of dermatologic diseases based on clinical presentation.

Infectious diseases

A number of infectious diseases specifically target the integument; in others, skin is secondarily affected. Most diseases listed here are included for completeness, but are seldom encountered in backyard poultry flocks. The incidence of many can be reduced with ideal husbandry, including proper nutrition, sanitation, and by avoiding overcrowding and exposure to predators.

With the exception of trauma and ectoparasitism, biopsy and histopathology greatly aid diagnosis. Diagnostic tests available for viral diseases of poultry include serology, PCR, and viral isolation. Culture and sensitivity help identification of bacterial pathogens.

Some vaccines are available in small quantities for viral diseases of backyard fowl. Many large poultry suppliers sell birds already vaccinated for diseases, in particular for Marek's disease. Vaccination of backyard poultry is discussed in another chapter.

Marek's disease

Marek's disease is an oncogenic cell-associated herpesviral-induced neoplastic disease that causes T-cell lymphoma in various tissues in chickens [1]. Unlike many other infectious diseases that are primarily caused by problems in production facilities, it is commonly encountered in backyard poultry flocks. The most common form of Marek's disease produces organ neoplasia and enlargement. A dermatological form of the disease produces reddened enlargement of feather follicles, which consist of aggregates of lymphocytes. Transmission occurs through shedding of secretions and feather follicle dander, and recent work has shown that viral particles can be found in skin epithelial cells as well [2]. Infected non-symptomatic carrier birds may shed the virus for life. Diagnosis of this cutaneous form takes place via biopsy and histopathology of affected feather follicles. As a result of the prevalence of Marek's disease in flocks, serology is likely to be less useful. Prevention is implemented through acquisition of vaccinated birds from reputable breeders. There is no known treatment for Marek's disease. Birds with confirmed disease should be isolated, and the premises thoroughly cleaned and disinfected although the organism can live for years in the environment.

Fowl pox (pox, avian pox)

Viral pox diseases affect nearly every species of bird. In poultry, chickens and turkeys can be affected [3]. Pox is caused by a large DNA poxvirus referred to specifically as fowl poxvirus and turkey poxvirus in these species. The virus produces typical round "pox-like" lesions of the integument, most commonly the unfeathered portions of the head or neck. Decreased weight gain and egg production often result. Lesions may also occur on the feet or vent. Respiratory pox infections can produce dyspnea and ocular or nasal discharge. Pox scabs are desquamated in the environment, and are infectious to other birds for many months. Transmission also occurs via cannibalism and vectors such as mosquitoes. Onset is gradual, and often not noticed until cutaneous lesions

Table 12.1 Diseases affecting the skin of poultry classified by clinical appearance. Note not all are commonly encountered in backyard poultry flocks

Lesion	Potential etiology
Round lesions of unfeathered portions of the head and neck, and sometimes feet and vent; often scabbed	Poxvirus
Masses associated with feather follicles	Marek's Disease; Bacterial folliculitis
Lesions of the feet, especially the ventral aspects	Footpad dermatitis; trauma
Yellow thickening of skin	Xanthomatosis
Feather loss and lesions of the back of the head and back	Rooster or cage mate trauma
Focal skin irritation/inflammation	Ectoparasites; trauma
Skin wounds, often necrotic	Gangrenous dermatitis, trauma
Lesions and irritation of vent in older birds	Cage mate trauma, Infectious bursal disease (IBD)
Lesions and irritation of the vent in young birds	
Pale discoloration of skin	Chicken anemia virus, other anemia
Lacerations and punctures; often of the head, neck and extremities	Predator trauma
Visible ectoparasites	Usually lice; consider ticks, fly larvae
Swollen inflamed wattles, neck and head, sometimes feet as well in visibly sick birds	Fowl Cholera

are detected. Diagnosis is confirmed via histopathology. Acquisition of birds from reputable breeders can prevent exposure to this disease. Pox vaccine is available and treatment is supportive. Affected birds should be isolated from the rest of the flock.

Chicken anemia virus (CAV, CIA, chicken infectious anemia, blue wing disease, anemia dermatitis syndrome)

While ubiquitous in production birds, CAV is uncommon in backyard flocks. This circovirus produces severe anemia in young chicks, which results in anorexia, lethargy, and pale tissues including the skin [4]. Hematocrit values typically range from 6–27%. Gangrenous dermatitis and a blue discoloration can be noted as well [5]. Adult infected birds do not develop disease, but infect the young via egg transmission. Transmission also occurs via the oral/fecal and possibly respiratory route. Diagnosis takes place via histopathology, serology,

and PCR. Treatment is supportive, and includes fluid therapy, blood transfusion in severely affected birds, and treatment of secondary gangrenous dermatitis. Purchasing birds from a reliable source can help prevent exposure to this disease. Vaccines are available for breeder flocks.

Infectious bursal disease (IBD, Gumboro disease)

This viral disease is included in diseases of the integument, as clinical signs include vent picking and trauma, along with trembling, ataxia, and diarrhea [6]. IBD is a highly contagious viral disease caused by a Birnavirus that primarily affects lymphoid tissue including the bursa of Fabricius. It is a disease of young chickens, most commonly 3–6 weeks old. The virus persists in the environment for months, is resistant to many disinfectants, and is therefore difficult to eradicate. Transmission occurs via exposure to virus in feces, feed, water, and is associated with fomites.

IBD is not commonly diagnosed in backyard flocks, and acquisition of birds from reliable sources can prevent exposure. Diagnosis occurs via histopathology and serology. Treatment is usually unrewarding, but improved husbandry may mitigate severity of the disease. Vaccines are available for breeder flocks.

Gangrenous dermatitis (necrotic dermatitis)

This term refers to several disease presentations characterized by sudden onset of cutaneous skin wounds and cellulitis, often over the wings, thighs, breast, and head. It is usually accompanied by septicemia and toxemia. Lesions are associated with a number of bacteria, including *Clostridia sp, Staphylococcus sp*, and *Escherichia coli.* Other factors include concurrent viral disease (in particular CAV and IBD in young birds), nutritional insufficiency, poor sanitation, cannibalism, or mechanical trauma. Large outbreaks in flocks are thought to result from immune deficiency, and may be associated with warm, humid conditions [5]. Recent outbreaks reported in the literature in broiler facilities have been linked to Clostridial infection and production of bacterial endotoxins. These birds often demonstrated antibody titers to other infectious diseases as well [7].

While outbreaks are unlikely in backyard flocks, individual birds are susceptible to the same syndrome when wounds are infected with bacteria.

A complete blood count can identify birds that are septicemic (leukocytosis, left shift, presence of heterophil toxicity). Many birds have anemia secondary to infection and inflammation, which can be severe.

Histopathology can identify necrosis, and culture of lesions may help identify specific bacterial agents. Therapy includes antimicrobials, ideally identified on culture and sensitivity, fluid support, blood transfusion in severely anemic birds, and eventual surgical debridement of wounds. Ideal husbandry and prevention of over-crowding can aid prevention. Vaccination of breeder birds against other viral diseases has been helpful as well.

Fowl cholera

This disease is caused by *Pasturella multocida*, and can produce inflammation and swelling of the face, wattles, neck, and footpads. Birds are generally sick and depressed as a result of septicemia. Caseous dermatitis and cellulitis are identified histopathologically. The most likely sources are chronically infected but asymptomatic birds, and rodents. Antibiotic therapy based on culture and sensitivity is ideal; however, sulfa drugs and penicillins are frequently listed as drugs of choice [8].

Ectoparasitic diseases

Many species of lice and mites infect birds [9]. Lice are generally species specific, but mites often are not.

External parasites such as mites and lice are common in poultry. Checking your flock periodically for external parasites and treating early helps prevent a larger flock outbreak.

Lice

Lice species vary in color, size, and preferred area of the body they infect. The entire life cycle occurs on the host, and transmission occurs via close contact with affected birds. More than 40 species of lice have been identified in domestic fowl. The more common include the body louse (*Menacanthus stramineus*), shaft louse (*Menopon gallinae*), and head louse (*Culclotogaster heterographa*) [10]. More than one species can be present on the bird at one time. The shaft louse lays its eggs on the shaft of the feather at the base (Figures 12.1a–b and 12.2) Lice may be clinically insignificant in older birds, but can increase in number and cause debilitation in younger or sick birds. Lice infestation appears to be worse in the fall and winter.

Clinical signs include hyperemia and irritation of the skin of affected birds with small scabs and clots. A moth-eaten appearance to the feathers may be seen. Nits (louse eggs) are laid in clumps at the base of feathers along the ventrum. The lice feed mainly on skin fragments and feather debris on the surface of

(a)

(b)

Figure 12.1 (a) Photograph of the common body louse of chickens, *Menacanthus stramineus*. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.) (b) Photograph of yellowish colored body lice on a white chicken. (Source: Photograph courtesy of Dr. Cheryl B. Greenacre.)

the skin, but can also feed on the blood inside blood (pin, quill) feathers, otherwise they do not suck blood, because they are chewing lice. They spend their entire life cycle on the chicken. Lice are easily seen with the naked eye and are yellowish in color and flat-bodied. Lice move fast in comparison to mites. Under the microscope their big head with chewing mouth parts can be seen.

Treatment options are variable. Ivermectin is commonly used as a lice treatment in poultry, with anecdotal reports of success, although there are no studies to support this. Follow label directions for withdrawal times in food-producing poultry. If selling the eggs commercially, the only permitted treatment is diatomaceous earth.

Figure 12.2 Photograph of the common shaft louse of chickens, *Menopen gallinae*. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.)

Figure 12.3 Photograph of *Dermanyssus gallinae*, the chicken mite, also known as the red chicken mite. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.)

Mites

Mites are much smaller than lice, and feed on blood, feathers, and skin. Appearance is variable. They are capable of infecting any avian host. Some mites spend their entire life cycles on the bird, but some do not. For this reason, treatment of mites involves treating the environment as well as the birds [10]. Some mites, including *Dermanyssus gallinae*, feed at night and hide in cracks and joints of the enclosure during the day; therefore diagnosis can be difficult. Some mites can survive in the environment for up to 30 weeks without food, making premise treatment critical for effective eradication. More commonly encountered mite species include the chicken mite (*Dermanyssus gallinae)*, the northern fowl mite (Ornithonyssus sylviarum*),* and the scaly leg mite *(Knemidokoptes mutans)* (Figures 12.3–12.6). While not specifically zoonotic, temporary infestation of humans may occur.

Ornithonyssus sylvarium (northern fowl mite, feather mite)

The northern fowl mite is the most common external parasite in poultry, especially in cool weather climates. This mite spends its entire life cycle (egg to larva to nymphal stage to adult), which can take as little as a week in ideal conditions, on the chicken. Clinical signs of this mite infestation include soiled feathers around the vent, tail, and rear legs. Mites are commonly first discovered or seen on eggs. Heavy infestations can cause decreased egg production. Barely seen with the naked eye, the adults are a dark red to black color and evidence of mites and eggs can be seen as a dark area at the base of feathers in the ventral regions (vent, ventral coelomic area, tail, ventral cervical area). Light colored birds may

Figure 12.4 Photograph of *Ornithonyssus sylviarum*, the northern fowl mite. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.)

have a darkening of the feathers from a build-up of mite feces. Diagnosis takes place based on typical clinical signs, seeing the mite grossly, or performing a tape prep of an affected area and examining for mites under the microscope. Mites can be transferred via fomites including crates, cages, clothing, and wild birds.

Dermanyssus gallinae (chicken mite, red mite, roost mite)

This mite feeds on poultry at night and then remains secluded during the day within the poultry house, making diagnosis difficult. This mite can live off the bird for 2–3 weeks. The life cycle can be completed in as little as 7–10 days with ideal conditions. Clinical signs include mild weight loss and decreased egg production.

Figure 12.5 Close up photograph of the mouth parts of *Ornithonyssus sylviarum* showing the difference from the mouth parts of *Dermanyssus* spp. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.)

Figure 12.6 Photograph of *Knemidokoptes* spp., the scaly leg mite. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.)

The mite is best seen by using a magnifying glass and inspecting the birds and house at night.

A recent study showed that mite populations were similar in hens that were raised either caged or free range [11]. Another study compared mite populations between hens that were caged, free range, and free range with access to dust boxes containing sand and either diatomaceous earth (DE), kaolin clay, or sulfur. All hens using dust boxes with any material showed a reduction in ectoparasites by 80–100% after one week when compared to the other two groups. Ectoparasite populations recovered when dust boxes containing DE or kaolin clay were removed; however, sulfur provided

a residual effect up to nine months post removal [11]. Provision of dust boxes may be a simple and effective method of ectoparasite control for backyard flocks.

Studies into the efficacy of various acaricides in the treatment of ectoparasites in chickens have revealed some information. Tetrachlorvinphos with dichlorvos was the most effective at treating chickens that were naturally infected with northern fowl mites, followed by Malathion dust and 10% garlic oil. Permethrin failed to reduce mite populations significantly [12].

Ivermectin is also commonly used to treat mite infestations in many species, including psittacine birds. Anecdotally, treatment in poultry appears to be effective, but only when combined with premises treatment for those mite species living for extended periods off the host.

Other ectoparasites identified in poultry include bedbugs (*Cimex lectularius*), chiggers (larval mites of *Neoschongastia ameri*cana) and sticktight fleas (*Echidnophaga gallinaceae*) (Figures 12.7–12.10). Recently, a henhouse was found to be infested with bed bugs and the hens were showing clinical signs of small, hard white welts on the skin which became inflamed and pruritic. The bed bugs live in the cracks of the henhouse and come out to feed on the chickens at night. Diagnosis is based on grossly identifying the reddish-brown oval to teardrop-shaped flattened bugs that are about $1/4$ to 5/8 inch in length.

Chiggers are most commonly encountered in turkeys in the southern states. Sticktight fleas cause skin irritation and possibly anemia.

Black flies (*Simuliidae*) can affect poultry (Figure 12.11). Large swarms of flies can produce anemia. Fowl ticks

Figure 12.7 Photograph of bedbugs, *Cimex lectularius*, found to infect chickens and their environment. The dorsal aspect is shown on the left; the ventral aspect is shown on the right. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.)

Figure 12.8 Close up photograph of the proboscis of a bedbug, *Cimex lectularius*. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.)

Figure 12.10 Photograph of the stick tight flea, *Echidnophaga gallinae*. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.

Figure 12.9 Photograph of the chiggers, the larval mite of *Neoschongastia americana*. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.)

Figure 12.11 Photograph of the black fly, *Simuliidae* spp. (Source: Photograph courtesy of Aly Chapman, University of Tennessee.)

include a number of species that affect a wide range of poultry, birds, and mammals. Some cause skin irritation and anemia [9].

Non-infectious diseases

Husbandry

Skin quality is affected by a number of husbandry factors, including diet and sanitation. Research has focused on the effect of bedding type, size, and moisture on the development of footpad dermatitis in broiler chickens [13]. Not surprisingly, bedding moisture increased the incidence of foot lesions, particularly in younger birds. Another study compared three bedding types:

Wheat straw, chopped wheat straw, and wood shavings. Parameters measured were weight gain, food intake, and incidence of footpad necrosis. Weight gain and low footpad dermatitis scores were improved when birds were kept on wood shavings [13]. For backyard flocks, clean dry bedding, preferably not wheat straw, along with other appropriate husbandry measures is likely to decrease incidence of disease as well.

Trauma

Skin trauma results most commonly from other poultry and predators, including dogs, cats, and wildlife such as raccoons, weasels, foxes, and larger birds of prey. Injuries range from mild to catastrophic. Hens

frequently present with missing feathers and abrasions of the back and head created by the rooster during mating, or by attacks from dominant hens (Figures 12.12–12.15). In contrast, predator wounds are usually more severe and located around the face and neck or extremities. Fly strike is common in debilitated poultry with open wounds or fecal accumulation near the vent. Both severe injuries and the presence of maggots are frequently missed, as feathers often cover them. Injuries to digits occur, and can be caused by punctures or entrapment.

Initial therapy of the severely ill bird is aimed towards emergency stabilization and correction of shock. Fluid therapy is similar to that described in other avian patients. Vascular access is best accomplished in poultry via an IV catheter placed in the basilic (ulnar) vein; an

Figure 12.12 Chicken with feather loss over the dorsum due to conspecific aggression. Feather loss with or without skin lesions behind the head and over the dorsum are often caused by other poultry, including other roosters or dominant hens.

Figure 12.13 Chicken with feather loss over the dorsal cervical area due to conspecific aggression.

Figure 12.14 Pekin duck with feather loss over the dorsal cervical area from conspecific aggression. This duck had a severe case of pododermatitis and could not compete well with the other ducks. (Source: Photograph courtesy of Dr. Cheryl B. Greenacre.)

Figure 12.15 The same Pekin duck as in Figure 12.3 showing primary and secondary wing feather loss from conspecific aggression. (Source: Photograph courtesy of Dr. Cheryl B. Greenacre.)

intraosseous catheter placed in the distal ulna or proximal tibiotarsus. Hypothermic patients are warmed externally and via infusion of warmed fluids. Antibiotic

therapy is important. Selection should be based on results of culture and sensitivity when available, and determined in part with legality and withdrawal times in mind (see Chapter 20). Unless not indicated based on culture and sensitivity, the author prefers the use of intravenous or intramuscular piperacillin (Zosyn) at 100 mg/kg IV q4–8h for severe bacterial infections with septicemia.

In general, pet poultry are good anesthetic and surgical candidates with impressive healing potential. The author has treated numerous cases of severe soft tissue and degloving injuries that have healed by second intention after weeks of supportive and wound care.

Sedation, anesthesia, and analgesia of avian species are well described, and the author has found that poultry do well with pre-anesthetic, induction, mainte-nance, and analgesic protocols described for psittacines. Many minor wounds can be addressed with sedation (the author recommends butorphanol, 2–3 mg/kg and midazolam: 0.5 mg/kg IM) with lidocaine 2 mg/kg as a local or regional block (Figures 12.16 and 12.17). Other surgical techniques are described in another chapter. As for any therapeutic agent, legal ramifications, including drug withdrawal times and appropriate drug use, must be kept in mind (Figure 12.18).

Wound management

If the wound results from predator attack then antibi-otics are needed to prevent sepsis. The bird may be in shock and supportive care including subcutaneous or intravenous fluids, warmth, quiet, and administration

Figure 12.16 Toe lesions are common in poultry, and may be caused by various traumatic episodes. This chicken's toe has a black necrotic area.

Figure 12.17 Toe shown in Figure 12.5 during instillation of a local analgesic.

Figure 12.18 Toe shown in Figure 12.5 after amputation, which was easily accomplished with sedation and local analgesia without general anesthesia.

of pain relievers is usually necessary. Cleaning and debridement of the wound may need to wait a few hours until the patient has been stabilized. If the wound is older than 3 days it shows some evidence of green bruising. Birds bruise green as a result of the lack of biliverdin reductase. If the wound punctures into the coelomic cavity there may be subcutaneous emphysema present. Some wounds are extensive and may take a few days to fully declare viability of tissue and may take months to fully heal especially if in an area of high movement such as the inguinal area. Various types of bandages and compounds have

been used, but the most important aspect of wound management is daily re-assessment of the wound. Pharmaceutical-grade honey works well for wound healing and can be used in egg laying birds, thus removing concerns about drug use in such birds. In general, chickens heal skin wounds very well given enough time.

Breast blister

These fluid-filled lesions of the sternal bursa are sometimes noted in the ventral aspect of the keel bone of large, heavy-bodied birds. They are thought to be related to repeated trauma, and may become secondarily infected [14]. Surgical excision is indicated for large and/or infected cysts. As large, heavy bodies meat breeds are more prone to breast blister and other debilitating degenerative diseases, the keeping of these breeds as pets should be discouraged.

Xanthomatosis

This disease features abnormal subcutaneous accumulation of intracellular cholesterol [15]. Lesions are usually firm and yellow, but in early stages may be soft with straw-colored fluid. In the past, xanthomatosis in production birds was thought to be associated with contamination of feed fat with hydrocarbons. However, xanthomatosis is associated with repeated trauma in other bird species, and may be seen in poultry as well. Diagnosis occurs via biopsy and histopathology. Treatment includes investigating and removing sources of trauma. Lesions are usually self-limiting, but surgical removal may be indicated in birds with larger debilitating lesions. In psittacines, xanthomas are highly vascular and must be removed with care; the same cautions are likely valid for poultry as well. Prevention measures include ideal husbandry and prevention of trauma.

Feather cysts

Dysplastic feather and follicles are occasionally encountered in turkeys. Cysts are firm and yellow (Tsang Long, personal communication).

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Reproductive Diseases

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Reproductive disease is very common in backyard chickens because they are usually egg layers and they typically live longer than the average commercial chicken.

Uterine prolapse/vent prolapse of egg layers

Clinical history

Layers of any age are susceptible and display blood-stained vent areas or eggs. Mortality may be seen.

Cause of condition

This condition is caused by cannibalistic behavior of penmates. Some strains of breeds of layers are more cannibalistic than others. High light intensity, non-beak trimmed hens, nutrient deficits, or stressful conditions such as crowding or inadequate nesting space can promote this problem.

Clinical signs and lesions

Lesions of a blood-tinged vent area, uterus prolapsed, and possibly tissues missing (consumption by penmates) are seen (Figure 13.1). Blood-tinged eggs can also be seen.

Transmission route

This condition is not infectious.

Diagnostic tests

Diagnosed by observations of lesions.

Differential diagnosis

Wounding or trauma to the oviduct by other means than pecking needs to be ruled out.

Prevention and control

Proper beak trimming helps. An adequate nutritional plan can reduce cannibalistic tendency. It also helps to reduce the light intensity, especially during the egg laying process. In commercial settings provide one nest box for every four birds. In the backyard setting this disease is not as common because the birds are not as crowded.

Oviduct impaction

Clinical history

Birds that have oviduct impaction have been in production before ceasing and becoming progressively lethargic, depressed, and lose weight. Depending on the amount of exudate in the oviduct, a duck-walking gait may be seen. This condition occurs most often in older layer birds.

Cause of condition

It is felt that the older birds' reproductive tracts weaken with age and allow a greater amount of bacteria into the oviduct by retrograde peristalsis. Also with age, the amount of time the oviduct is everted during oviposition is increased, allowing more time for exposure to bacteria. Generally, *E. coli* is felt to be the causative

Figure 13.1 Cannibalism/peckout of the vent showing the contributing factor of a sharp beak. Note the blood on the eggs and on the beak of the bird doing the pecking.

agent involved although other bacteria, such as *Staph. aureus, Klebsiella pneumoniae,* and *Salmonella spp.,* have been implicated.

Clinical signs and lesions

Depression, lethargy, and loss in body weight are clinical signs. Upon necropsy, one finds an extended oviduct filled with caseous material [1] (Figure 13.2). Exposure of the oviduct lumen to bacteria from retrograde peristalsis of exposed oviduct during oviposition is felt to be the mechanism of disease. Palpation or radiography of the abdomen can detect the caseous mass in the oviduct.

Transmission route

This condition is not infectious.

Figure 13.2 Necropsy of a chicken with oviduct impaction. Note the caseous debris in the oviduct.

Diagnostic tests

Necropsy lesions.

Differential diagnosis

There are many different causes for cessation of egg laying and all of these should be considered as differential diagnoses. There is a normal amount of this condition in all flocks and it is increasingly seen as the flock ages.

Prevention and control

For prevention, maintaining clean nesting conditions does indeed aid in reducing the degree of oviduct exposure to bacteria during oviposition. Therapy using antibiotics orally has not met with success. Surgery to remove the caseous mass has been performed successfully.

Egg bound, egg binding

Clinical history

The typical history with this condition is that the bird has stopped laying, is having difficulty walking, and an egg may be seen in the cloaca.

Cause of condition

The underlying causes and contributing factors can include an excessively large egg (double yolk, large eggs in older hens); low blood calcium (hypocalcemia); calcium tetany; trauma to the uterus, vagina, or vent resulting from pecking; obesity; or stimulation into production before the bird's pelvis has matured.

Clinical signs and lesions

The lesion is a shelled egg lodged in the uterus or vagina, which the hen is not able to lay [1] (Figure 13.3).

Transmission route

This condition is not infectious.

Diagnostic tests

Palpation or radiography can be used to diagnose this condition.

Differential diagnosis

This condition must be differentiated from other causes of cessation of egg laying.

Prevention and control

Prevention lies with avoiding the causes and contributing factors such as avoiding double yolks by using a

Figure 13.3 Necropsy of chicken with egg impaction and regression of the ovary.

standard lighting schedule, controlling obesity and excessively large eggs by routine body weight monitoring and controlling nutrient intake accordingly, feeding adequate calcium levels, and preventing cannibalism and wounding of the vent.

A veterinarian should undertake treatment of this condition. It may involve i) lubricating the canal and attempting to ease the egg out or, ii) imploding the egg by extracting the contents (ovocentesis) or, iii) surgery to perform a salpingohysterectomy. Parenteral calcium in the form of calcium gluconate intramuscularly initially, and then calcium glubionate or calcium carbonate orally should also be given.

Retained cystic right oviduct

Clinical history

Normally, no outward signs of disease are seen with this condition. The prevalence of this condition is fairly rare in commercial chickens, but is seen more often in backyard chickens.

Cause of condition

Two Muellerian ducts are present early in the developing bird embryo [1]. The left duct develops into the oviduct and the right duct regresses. Occasionally in chickens, the remnant of the right duct becomes dilated with an accumulation of watery fluid.

Clinical signs and lesions

No outward signs of a problem typify this condition until a necropsy is performed and the retained, cystic right oviduct is found. Upon necropsy, a large, fluid-filled sac is seen (Figure 13.4). This condition is seen at a higher incidence in some strains of layers than others.

Transmission route

This condition is not infectious.

Diagnostic tests

The condition is identified during postmortem examination. A radiograph and ultrasound would show a fluid-filled structure suggesting this disease.

Differential diagnosis

This condition is differentiated from ascites by the presence of the membranous wall of the remnant right oviduct containing the fluid rather than the fluid being free in the body cavity. Sometimes during coelmocentesis it is difficult to ascertain if the fluid being aspirated is loose in the coelmic cavity or is from inside of the cystic duct.

Prevention and control

This is an error in embryonic development and not preventable.

Figure 13.4 Persistent cystic right oviduct.

Egg yolk peritonitis (egg related peritonitis)

Clinical history

The yolk-laden ova on the ovary are delicate structures surrounded by a thin membrane called the vitelline membrane. Rough handling of pullets or hens in production, sudden excitement inducing vigorous activity, and so on may cause trauma to the body wall that ruptures one or more of the yolks on the ovary. The vitelline membrane may also become weak secondary to bacterial septicemia or systemic viral infections and rupture.

Causative agent

Free yolk is very irritating to the body cavity linings and induces a severe inflammatory response that results in peritonitis, usually without microbial infection.

Clinical signs and lesions

An affected hen may be depressed and off feed. When examined at surgery or at necropsy the coelomic area contains thick friable yellow exudate that is adhered to the serosal linings (Figure 13.5). If the hen has been off feed for some time, the ovary may show evidence of involution.

Transmission route

This is not initially an infectious disease. A secondary bacterial infection, usually with *E. coli*, may occur.

Diagnostic tests

The condition is usually diagnosed at necropsy. Radiographs may show multiple radiopaque densities in the

Figure 13.6 Carcinomatosis in an adult hen either of ovarian or pancreatic origin. Note the multiple masses that should not be confused with egg-related peritonitis. (Source: Photograph courtesy of University of Tennessee pathology department website http://vetgrosspath.utk.edu.)

intestinal peritoneal cavity area. Ultrasound may help to further identify these masses. Coelomocentesis may provide fluid that can be evaluated for bacteria, yolk material, and inflammatory cells.

Differential diagnosis

Airsacculitis caused by primary agents such as *Mycoplasma* sp., respiratory borne bacterial infections, respiratory viral infections (infectious bronchitis virus, PMV1, etc.), coccidiosis, and carcinomatosis (Figure 13.6).

Prevention and control

Hens that are producing eggs should be handled carefully. Precautions should be used when among the birds to prevent or minimize startling of the flock. Individual hens can be administered antibiotics and/or surgery can be performed to remove some of the irritating egg material with or without performing a salpingohysterectomy, but the prognosis is fair to poor depending on the severity.

Paratyphoid oophoritis

Clinical history

Infection with *Salmonella* sp. may involve the ovary and attached yolks. Inflammation of the ovary, when gravid, frequently leads to debilitation and death of the hen.

Figure 13.5 Yellow exudate being surgically removed from the coelomic cavity of a 1-year-old bantam silkie hen with egg yolk peritonitis. One soft-shelled egg and the collapsed remains of six other eggs were also removed. The hen continues to do well over a year after surgery. Photograph courtesy of Dr. Cheryl Greenacre.

Causative agent

Salmonella spp.

Clinical signs and lesions

The infection may cause debilitation and death of the hen. In the gross examination, the follicles of the ovary are covered by tan-white caseous exudate. The yolk material loses its normal translucence and the follicles shrink. The inflammation may spread to involve the entire coelomic area.

Transmission route

Infection with *Salmonella* sp. may occur vertically through the egg of latently infected hens. It may also occur horizontally through contact with or ingestion of contaminated materials such as fomites, feed, and rodents [2].

Diagnostic tests

Bacterial culture should be used to identify the causative agent. *Salmonella* group D isolates should be reported to your state veterinarian.

Differential diagnosis

Other bacterial infections caused by organisms such as *E. coli* and *Pasteurella multocida* can cause similar changes.

Prevention and control

Antibiotic therapy helps to control the spread of the infection in a flock. Replacement hens should be obtained from breeding flocks that have been tested and found negative for the presence of the group D *Salmonella* sp. Flocks monitored under the National Poultry Improvement Plan (NPIP) are free of this group of *Salmonella* sp.

Zoonotic potential

Many of the *Salmonella* sp. have the potential of causing egg contamination that may lead to human infection.

Decreased egg production/cessation of laying

Clinical history

With this condition, a noticeable drop in number of eggs collected from one day to the next is seen. This happens quite often in backyard flocks for a variety of reasons.

Cause(s) of condition

Numerous causes of reduced egg production are possible including infectious (Infectious bronchitis, *Mycoplasma gallisepticum*, egg drop syndrome, Newcastle disease, and avian influenza), nutritional (water deprivation, inadequate calcium, phosphorus, sodium, protein, or vitamin intake), and environmental (declining day length, excessive heat, excessive cold).

Clinical signs, lesions, and diagnostic tests

Variable depending on the cause.

Prevention and control

To prevent and control the possible infectious disease causes see each specific disease section, but in general it helps to provide adequate nutrient intakes at all times and phases of life to support normal egg laying and to make sure adequate water is available to the birds at all times. Provide a warming device for water in the cold months.

Feather loss

Clinical history

This condition affects laying hens, which lose varying degrees of feather cover over the laying period until they cease egg production and molt. The amount of feather loss involves the interaction of several factors.

Cause(s)

There are several factors that may influence the degree of feather loss in hens. Malnutrition is a leading cause and can include inadequate amino acid intake throughout lay, especially the sulfur-containing amino acids methionine and cysteine, inadequate sodium intake that can influence feather pecking activity and cannibalism, or inadequate vitamin or trace mineral intake, which leads to poor feather growth and quality. In commercial chickens the beak trimming quality can influence the degree of feather loss. Longer beaks yield more feather pecking. Stresses such as crowding (inadequate floor, perch, nest, feeder, or waterer space), group size and external parasite load, can also increase feather pecking activity of the flock and penmates. Other stresses include egg production and egg size level; for example, high egg production or larger than normal egg size reduces the amount of amino acids for feather growth and maintenance.

Table 13.1 Normal percentage of the body covered with feathers from point-of-lay through one lay cycle is outlined

Part of lay cycle/weeks of age	Percentage of body covered with feathers
Start of lay/18 weeks of age	100
30 weeks of age	95
40	90
50	85
60	80
70	75
80	70

Clinical Signs and lesions

Normal feather loss progression over the period of lay starts with neck feather loss and progresses to loss of feathers over the crop, the breast, and then the back. The normal percentage of the body covered with feathers from point-of-lay through one lay cycle is outlined in Table 13.1. Higher percentage feather losses resulting from the causative factors listed above can be seen.

Transmission route

This condition is not infectious.

Diagnostic tests

Physical examination findings.

Differential diagnosis

Varied.

Prevention and control

To avoid excessive feather losses, i) beak trim birds to avoid feather pecking/pulling, ii) feed a complete ration that fulfills the birds' needs for protein, energy, vitamins, and minerals, iii) avoid over-crowding and inadequate floor, perch, nest, feeder, or waterer space, and iv) control external parasites.

Lighting

Clinical history

This is a relatively common problem among backyard flock owners, especially newcomers.

Cause(s) of condition

Birds' reproductive systems are sensitive to lighting differences in day length and intensity. Increasing day length stimulates reproductive development and production, while a decreasing day length de-stimulates the reproductive system. In nature, the increasing springtime day length stimulates bird populations to reproduce when conditions become more favorable for hatchling survival, while the declining day lengths of fall and winter prevent birds from reproducing when conditions are not favorable. Lighting effects can come from either natural day length or artificial sources of light. Stimulation with light promotes the growth of the ovaries and oviduct and induces birds to lay. De-stimulation with decreased photoperiod or light intensity causes birds to cease egg production and begin molting.

Diagnostic tests

An array of serologic tests can be performed to determine whether one of the infectious agents is the cause of the egg production loss.

Differential diagnosis

Some infectious disease conditions, such as infectious bronchitis and *Mycoplasma gallisepticum*, can mimic loss of egg production in a subtle way with very few clinical signs.

Prevention and control

Obtain information on the sunrise and sunset times in your area to aid in setting time clocks. Utilize lighting program information from the various poultry breeder organizations.

Molting

Clinical history

Members of the flock are seen to be out of production, lose feathers, re-generate their feathering, and regain egg production.

Cause(s) of condition

Molting is a natural process whereby the laying hen rests her reproductive tract to renew and renovate the oviduct for another cycle of laying. This occurs in nature when day lengths decline in the fall and the ovary is de-stimulated.

Clinical signs and lesions

The molting layer loses almost all of its feathers during a molt in the following order: Neck, breast, body, wings, and tail. The primary wing feathers are lost first followed

by the secondary wing feathers. Some hens completely cease egg production, while others may continue to lay. As the replacement of feathers takes a lot of nutrients, egg production does not take place at a normal rate.

Diagnostic tests

A diagnosis of molting can be made if evaluations of management factors involving feed, lighting, air quality, water, and diseases that result in loss of egg production are ruled out.

Differential diagnosis

Lack of adequate nutrition, declining day length, poor air quality, excessive cold, excessive heat, lack of water, and a variety of disease agents (Newcastle, avian influenza, infectious bronchitis, etc.) that result in loss of egg production.

Prevention and control

Commercial egg producers and some backyard flock owners perform planned molts to synchronize egg production and improve egg quality and numbers. A planned molt consists of reducing the day length (reduced to 8 hours) and reducing nutrient intake (feeding 50 grams of growing-type ration per bird per day for example) to bring birds out of production then rest them. Once they have rested for about 3 weeks after ceasing egg production, the day length is increased to 14 hours and lay ration is full fed. Weekly day length increases of 30 minutes are then implemented until a total of 16 hours is reached.

Calcium depletion, calcium tetany, hypocalcemia, caged layer fatigue

Clinical history

Cage layer fatigue is a term used to describe leg weakness and acute deaths in chickens in cages, and is caused by inadequate calcium levels in the blood stream. Calcium is required for muscle function, bone formation, and egg shell formation. This condition may be seen even in floor birds under certain conditions. It is seen most often in young hens early in production.

Cause(s) of condition

Calcium depletion is rarely caused by feed formulation errors. It is more commonly associated with feed manufacturing errors such as ingredient separation during manufacture, delivery, or feeding. Insufficient calcium particle size to stay in the gut during the night may occur.

Inadequate feed intake may not support the level of egg production. This is a problem that may occur if the hens are fed a complete ration and allowed to forage. The ingestion of forage of inadequate nutrient composition dilutes the value of the desired ration and potentially leads to nutritional inadequacies. Vitamin D is required for absorption of calcium from the intestine.

Clinical signs and lesions

Affected hens are weak and unable to stand (Figure 13.7). Very few postmortem lesions may be evident. Hens that die may have a completely shelled egg in the shell gland. Dead hens may have soft bones. The ribs are most likely to display softness because they are thin structures and more susceptible to the effects of calcium loss. On a flock basis there may be a decline in egg numbers and egg shell quality may decrease. Bone deformities, such as a curved keel bone and bent ribs, develop in a hen with soft bones over time.

Diagnostic tests

Knowledge of the flock history, clinical signs, and postmortem findings are usually sufficient to diagnose the problem. Response to treatment helps confirm the diagnosis.

Differential diagnosis

Botulism.

Prevention and control

Calcium depletion is rarely caused by feed formulation errors. It is more commonly associated with feed manufacturing errors such as ingredient separation during manufacture, delivery, or feeding. Insufficient calcium particle size to stay in the gut during the night may occur. Large particle limestone or oyster cannot usually

Figure 13.7 Hen weak from hypocalcemia.

be used in pelleted feed or crumbles. Inadequate feed intake may not support the level of egg production. This is a problem that may occur if the hens are fed a complete ration and allowed to forage. The ingestion of forage of inadequate nutrient composition dilutes the value of the desired ration and potentially leads to nutritional inadequacies. It is important to feed a ration appropriate for the stage of production. Vitamin D is required for absorption of calcium from the intestine. A deficiency of vitamin D is uncommon in hens that are allowed access to sunlight. It should be noted, however, that vitamins may deteriorate over time. Feeds should be stored in cool dry conditions and used as soon as possible to insure freshness.

Broodiness

Clinical history
Broodiness denotes the behavior of the hen when she desires to sit on a nest.

Causative agent
This usually occurs after a group of eggs (clutch) has been laid.

Clinical signs and lesions
At this time the hen seeks dimly lit secluded areas where her other eggs are located. The bird becomes secretive and quiet.

Differential diagnosis
This behavior is rarely seen in breeds of chickens selected for high rates of egg production. Few of the species of game birds kept in captivity display broodiness. The instinct may, however, be very strong in some breeds of chickens and turkeys. This may be a desired quality if breeding your own chickens.

Prevention and control
Prevent access to dimly lit areas and hiding places.

Shell-less eggs

Clinical history
This condition is characterized by finding fully formed eggs in the nest area with no shell, only the shell membrane.

Cause(s) of condition
Certain diseases that affect the oviduct function such as infectious bronchitis, *Mycoplasma gallisepticum* (Mg), or Egg Drop Syndrome (EDS) resulting from adenovirus 127, results in numerous shell-less eggs. Nutrient deficits such as calcium, phosphorus, or vitamin D3 or excessive intakes of phosphorus or vitamin D3 may lead to an increase in the finding of shell-less eggs. A normal increase in shell-less eggs is seen in older layer flocks.

Clinical signs and lesions
Clinically, one finds an increased number of shell-less eggs.

Diagnostic tests
Various tests to rule out the possible etiologies.

Differential diagnosis
Other possible diseases that need to be ruled out are infectious bronchitis, *M. gallisepticum* infection, and EDS.

Prevention and control
Prevention starts with preventing those diseases that may cause shell-less eggs; IB, Mg, and EDS.

Maintaining adequate nutrition, especially for calcium and phosphorus, is also important.

Double yolking, double yolks

Clinical history
Normally seen in young flocks just starting into production. There can be a very high incidence in a flock with perhaps up to 20% of eggs affected.

Cause(s) of condition
Double yolking is caused by excessive stimulation with light, caused by either an excessive increase in day length or an excessive increase in light intensity. Excessive intake of the amino acid methionine can also induce this condition.

Clinical signs and lesions
Unusually large eggs are seen. When broken out, two yolks are found (Figure 13.8).

Transmission route
This condition is not contagious.

Figure 13.8 Example of a double-yolked egg.

Differential diagnosis
Excessive egg size with a single yolk.

Prevention and control
For prevention, use a controlled lighting program of both day length and intensity to prevent double-yolking. Removing the inciting cause(s), excess day length, excess light intensity, or excess methionine, may help reduce this problem.

Discolored yolks/blood spots/meat spots

Clinical history
In this condition, discolored yolks, bloodspots, meat spots, and other abnormalities of the internal contents of the egg occur without any indication of a problem with the flock.

Cause(s) of condition
Certain materials eaten by the bird may cause discolored yolks. For example, high copper levels in the soil caused discolored yolks that after rain allowed the hens to drink high levels of copper from the water puddles (Figure 13.9). Gossypol, a natural component of cotton-seed meal, can result in extreme discoloration of egg yolks as well as production loss.

Blood spots are the result of stress on the hen such as a thunderstorm or sudden excitement such as a dog attack. Inadequate vitamin K can also be a contributing factor. Blood spots are much more common in brown
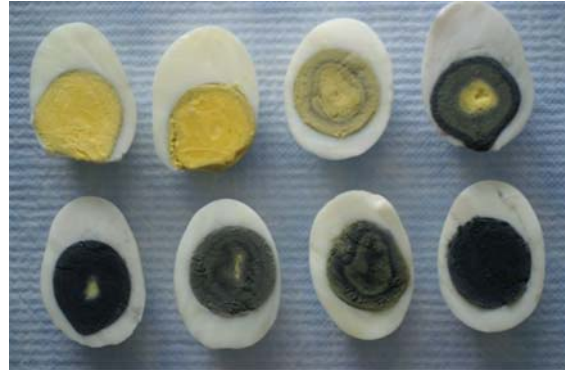
Figure 13.9 Discolored yolks from copper contamination of the soil.

egg layers than white egg layers. Some genetics companies have placed much more emphasis on eliminating blood spots from their lines of layers than others.

Meat spots are the result of a piece of tissue from the ovary or oviduct becoming incorporated into an egg during ovulation. The incidence is much higher in brown egg layers than white. Some genetics companies have put much more effort into reducing meat spots in their lines of birds than others.

Clinical signs and lesions
Normally, there are no outward signs of anything wrong with the flock.

Transmission route
Usually, the agent causing a yolk discoloration problem is ingested. Blood and meat spots are not transmittable.

Diagnostic tests
For yolk discoloration, chemical assays can be run on the yolk to determine the cause. For blood and meat spots, visual observation is diagnostic.

Prevention and control
For yolk discoloration, do not allow birds access to water puddles after a rain. Control the intake of feedstuffs to avoid possible causative agents. For blood spots, provide a nutritionally sound diet complete with adequate vitamin fortification. Avoid stressors that excite the flock. Use strains of layers with a low incidence of blood spots. Break out eggs in a bowl before use. For meat spots, use strains of layers with a low incidence of meat spots. Break out eggs in a bowl before use (see Chapter 17).

Zoonotic potential

Depending on the chemical causing the yolk discoloration, illness may be seen in the consumer of the eggs. There is no zoonotic potential with blood or meat spots.

Abnormally shaped eggs

Clinical history

Various shaped eggs are seen during the lay cycle of most flocks.

Causative agent

In many cases, it is not known what causes these abnormal shapes. In some cases the cause may be a change in lighting schedule that disrupts the ovulation pattern resulting in double ovulation with two eggs developing at one time. Two eggs side-by-side in the uterus results in a slab-sided egg. Infectious bronchitis or egg drop syndrome (EDS) virus infection causes a variety of misshapen eggs.

Clinical signs and lesions

Misshapen or "wrinkled" egg shells seen. Other clinical signs are usually not seen unless infectious bronchitis or EDS is involved, in which case respiratory signs and lesions along with egg production loss are seen.

Transmission route

If caused by infectious bronchitis, fomites can transfer the virus from one flock to another.

Diagnostic tests

Have a veterinary diagnostic lab perform tests for infectious bronchitis and EDS.

Prevention and control

Prevention of misshapen eggs may be accomplished by using a consistent lighting schedule and preventing infectious bronchitis.

Zoonotic potential

Normally not an infectious or toxic condition. IB or EDS are not zoonotic agents.

Shell color loss

Clinical history

Shell color loss is only noticeable in brown egg layer flocks. As flocks age, brown shell color loss occurs. Also, the different strains of layers have differing rates of lighter color eggs than others.

Cause(s) of condition

The protoporphyrin-IX pigment that is secreted from the epithelial cells lining the uterus during the 90 minutes just prior to oviposition is responsible for the brown eggshell color. Strain of bird, age of bird, and stress levels can affect the shade of brown of the eggshell. Nicarbazine, a coccidiostat, if fed to brown egg layers causes a temporary total loss of shell color while the material is present in the diet. The diseases EDS and IB also result in total shell color loss. The pigment loss recovers in the case of IB infection but may take up to 6 weeks.

Clinical signs and lesions

If Infectious Bronchitis (IB) is the cause then respiratory signs and egg production loss are seen, whereas with EDS and nicarbazine poisoning usually only egg production loss is seen.

Diagnostic tests

Virus isolation and serology are used to determine if IB or EDS are the cause of the loss of color. An assay of the feed can be performed to test for nicarbazine poisoning.

Prevention and control

Preventing shell color loss involves i) keeping stress level of the flock to a minimum, ii) controlling diseases such as EDS and IB through vaccination and biosecurity efforts, and iii) avoiding Nicarbazine contamination of feed.

Zoonotic potential

None of the causes have zoonotic potential.

Poor egg shell quality

Clinical history

Eggshell characteristics are affected by a variety of nutritional, infectious, and physical influences.

Cause(s) of condition

Factors that interfere with calcium utilization, such as inadequate mineral supplementation, inappropriate phosphorus levels in feed, inadequate vitamin A or D levels in feed, may lead to defects in the egg shell such as thinness. Roughness or shells with a "sandpaper" consistency may appear in flocks early or in the middle stages of production. This condition may be associated

with inadequate vitamin supplementation, especially vitamins A and D.

Body checks or cracks occurring in the eggshell while it is still developing in the shell maker gland can be made. The cracks are covered by secretion of additional shell material in the shell maker gland and appear as ridged areas over the original breakage sites. The shell is weak in these areas and more subject to breaking. This damage may occur after excessive vigorous activity in the flock.

Soft ends of the eggshell are occasionally seen early in production. The cause of this condition is not known. It may be associated with strain of chicken or nutritional factors.

Clinical signs and lesions

The problems can occur at any age and may not be associated with clinical signs in the hen unless associated with a systemic disease condition.

Diagnostic tests

Diagnosis is based on location and characteristics of the shell changes. Serological evaluation for certain viral diseases, such as infectious bronchitis and PMV1 infection, may help determine if a challenge from one of these agents may have occurred.

Prevention and control

Include animal movement, vaccines, and disinfectant use.

Worms in egg

Clinical history

The avian roundworm, *Ascaridia galli*, or tapeworm segments may occasionally be found by a consumer in an egg.

Causative agent

Intestinal parasitism of the hen.

Clinical signs and lesions

The hens are not usually sick.

Transmission route

It is believed that the worm migrates from the cloaca up the oviduct and becomes incorporated into the egg.

Diagnostic tests

The worms can be detected by candling the eggs.

Prevention and control

Hygromycin B is currently the only feed additive deworming compound approved for use in chickens that produce eggs for human consumption. It is not effective against tapeworms. No medications are available for treating tapeworms in chickens. If there is a concern, the eggs should be candled and any that contain the parasite discarded.

Zoonotic potential

Roundworms and tapeworms of chickens do not parasitize humans.

Egg drop syndrome (EDS)

Clinical history

As this disease is not known to be present in the United States or Canada, its prevalence is non-existent. As a result of its presence in Mexico however, one must be aware of its possible introduction from infected birds imported into the United States or Canada.

Causative agent

An adenovirus, EDS −76 or adenovirus 76 (not present in the United States or Canada), is the cause of this disorder.

Clinical signs and lesions

Clinically, a dramatic drop in egg production without clinical signs of illness characterizes this disease. Egg quality also declines dramatically with a loss of pigmentation and shell quality. A large number of shell-less eggs are seen. Pullets may be infected but do not show any clinical signs.

Transmission route

This virus may be transmitted vertically from infected parent stock to their progeny. The virus can then become active when the bird reaches sexual maturity. The virus can also be transmitted horizontally to different locations by fomites contaminated with the virus. The virus may be shed in the feces and enter through the oral route.

Diagnostic tests

For diagnosis, virus isolation from the uterus should be attempted. Hemagglutination inhibition tests can be used diagnostically. Serology tests such as the HI are available for use in countries where the virus is found.

Differential diagnosis

One must use the diagnostic laboratory to differentiate between diseases such as avian influenza, Newcastle disease, infectious bronchitis, and *Mycoplasma gallisepticum*.

Prevention and control

In countries where EDS is prevalent, an effective inactivated vaccine is available for use.

Zoonotic potential

There is no known human disease from the EDS virus.

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Gastrointestinal and Hepatic Diseases

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Gastrointestinal diseases are common in floor-raised backyard chickens. One way to clinically assess the gastrointestinal health of poultry is to observe their feces. Chickens have two different physical forms of feces. The feces of clinically healthy chickens are brown in color and are well formed in consistency [1]. There is often a white portion on the surface of the feces. This white portion, often referred to as the white cap, is not any excreta from the digestive system but rather is nitrogenous waste excreted as solid uric acid (Figure 14.1). The other excreta from clinically healthy chickens are the excreta from the ceca. These cecal droppings are loose and tenacious in physical form and are dark (Figure 14.1). While an occasional cecal dropping is normal, an increased observation of cecal droppings has been associated with stress [1]. The loose cecal droppings should be distinguished from diarrhea. Diarrhea is defined as an increased amount, increased frequency, and/or change in consistency of feces [1]. Depending on the cause of the diarrhea and the pathogenicity of the infectious agent involved, the color can vary from yellowish brown to bloody [1]. Hence, periodic monitoring of feces on the floor can help to detect early gastrointestinal disease and to determine the normal conditions in the flock.

Approaching the sick poultry patient

A physical examination is most important to access the general physical state of the patient and to determine the chronicity of the disease state. Emaciation can be evaluated by palpation of the breast muscles to determine if a prominent keel bone is present [2]. Assessment of feather quality also provides clues as to the nutrition provided to the flock [2]. Numerous feather lines, called stress bars, appear as clear areas that are visible transversely across the feather vane, and appear if the bird was provided inadequate nutrition on a long-term basis [2]. The presence of a pasty vent, fecal material adhered to the vent feathers, could indicate past and/or current diarrhea [2].

To evaluate the gastrointestinal health of poultry, the following samples should be collected and associated diagnostic tests should be performed:

1. Collect uncoagulated blood for hematology for general health assessment [2,3]
2. Collect serum for clinical biochemistry evaluation and serological monitoring of diseases [2,3]
3. Collect feces for evaluation of intestinal parasites and repeat collection at 4 week intervals [2]
4. If diarrhea is observed, the feces should also be cultured for bacteria such as *Salmonella* and should be collected at successive periods as shedding can be intermittent. [2] Feces can also be collected for detection of enteric viruses that can be performed at a state diagnostic laboratory, but for most backyard flocks this may be cost prohibitive for the owner unless there is mass mortality and
5. If a bird dies, it is important to perform a necropsy, especially if more than one bird has died [2]

While there are conditions, such as intestinal parasitism, that primarily affect the gastrointestinal tract, conditions of other systems, such as respiratory disease, might present with gastrointestinal signs (e.g., diarrhea). This chapter is organized to assist the veterinary practitioner to first look for clinical signs; followed by descriptions of diseases that can be considered by biopsy

Figure 14.1 Normal chicken feces generally consists of semisolid green to brown excreta admixed with a cap of white urates. Note the looser normal cecal dropping on the left.

or necropsy. While the primary purpose of backyard poultry medicine is to keep the individual bird alive, diagnostic tests on euthanized or sick birds may be indicated if the health of large numbers of birds is at stake.

Lesions of the oropharynx

Examination of the oral cavity should be performed during a physical examination [2]. The oral cavity can be observed by grasping the lower beak and drawing it ventrally while stabilizing the head. The mucosa should be pink and smooth. Diseases that can occur in backyard poultry include whitish yellow plaques in the mouth of birds [2]. The top five differential diagnoses for poultry include fowl pox, candidiasis, trichomoniasis, vitamin A deficiency, and aspergillosis [4]. One can gently scrape the mucosa to identify the causative agent via cytology, or biopsy of the lesions can identify fowl pox. If malnutrition is suspected, samples of the feed should be collected and submitted for vitamin analysis. Balanced commercial diets are readily available for backyard poultry.

Fowl pox

Fowl pox is a relatively slow-spreading viral disease of chickens and turkeys that is characterized by eruptions and scab-like lesions on the skin, combs, wattles, and inside the mouth, as well as diphtheritic or plaque-like lesions in the mouth, esophagus, and the upper part of the trachea. The causative agent is a poxvirus belonging to the *Poxviridae* family, subfamily *Chordopoxvirinae*, genus *Avipoxvirus*, species *Fowl pox virus*,

a large double-stranded DNA virus with a biconcave core. There are a number of strains within the group that differ in their specificity for and pathogenicity to various species of birds [5]. The disease spreads slowly in a flock with an incubation period of 4–10 days. Infection occurs to the injured or lacerated skin through mechanical transmission of the virus. Mosquitoes have been shown to spread the disease in chicken flocks. Flies can deposit the virus in the eye or in open wounds or lacerations. The mucosa of the upper respiratory tract and oropharynx are highly susceptible to the virus, and infection can occur in the absence of skin trauma or injury [6].

Clinical signs

Fowl pox has two forms; the cutaneous (dry) and membranous (wet) forms. In the cutaneous form, formation of nodules on the comb, wattle, eyelids, and other unfeathered areas of the body occurs. These nodules increase in size and can coalesce to form large brown to yellow scabs. These lesions eventually form scabs that dry up and drop off. The membranous form, referred to as wet pox, is characterized by raised fibrinous plaques or nodules on the mucous membranes of the oropharynx, esophagus, or upper part of the trachea (Figure 14.2). These lesions may coalesce to form an adherent membrane that covers the ulcerated areas. Lesions in the oropharynx often make it difficult for birds to eat or drink. High mortality resulting from suffocation can occur if the lesions occlude the upper trachea, particularly the glottis (tracheal plugs) [7].

Diagnosis

A presumptive diagnosis is based on the presence of scabs on the skin, comb, wattles, or other unfeathered areas of the body, or yellowish plaques on the

Figure 14.2 Raised friable plaques on the mucous membranes of oropharynx, and choana of a chicken with wet pox.

mucous membranes of the oropharynx or esophagus. A definitive diagnosis can be made by histopathological examination of the scabs, or by virus isolation on the chorioallantoic membrane of embryonated eggs (dropped CAM method) [6,7].

Treatment

There is no specific treatment that is effective against the poxvirus. Good management, including mosquito control, reduces stress in infected flocks. Vaccination is presently the only method of controlling fowl pox. There are two types of live virus vaccines used to immunize birds. Fowl pox vaccine of chick embryo origin is used to vaccinate birds of 4 weeks of age and older. Fowl pox vaccine of tissue culture origin is milder and can be used to vaccinate chicks as young as 1 day of age. The pigeon pox vaccine is mild and can be used in chickens of any age. Pox vaccines should be administered by the wing-web method in chickens. A thigh stick is usually used in turkeys because they tend to tuck their heads under their wings when resting and the face can come into contact with the vaccine strain and cause a vaccine reaction on the head. Cannibalism should also be controlled in a flock to reduce transmission of poxvirus [6,7].

Candida albicans infection (candidiasis)

Candida albicans is a mycotic infection affecting a wide variety of birds and occurs primarily in the upper digestive tract, especially the oropharynx and crop. This yeast infection is fairly common and is usually the result of long-term (1–2 weeks) administration of oral antibiotics. Candidiasis has also been referred to as crop mycosis, crop mold, and thrush. *Candida albicans* is yeast that forms pseudohyphae in tissues. This yeast is ubiquitous and overgrowth is usually controlled by normal bacterial microflora in the digestive tract. This condition is often associated with other diseases, usually those of the digestive or respiratory tract. In addition, long-term oral antibiotics, especially when administered in drinking water, can alter microflora of the upper digestive tract and promote yeast growth [8]. *Candida* is not transmitted from bird to bird and can often affect more than one bird if the flock has been treated with long-term oral antibiotics.

Clinical signs

There are no specific signs but birds may appear unthrifty. Lesions commonly occur in the crop, which has a fine, white pseudomembrane lining on its mucosal membrane, giving it a "Turkish towel" appearance (Figure 14.3). The oral cavity and/or esophagus

Figure 14.3 Turkey poult with moderate to severe crop infection caused by *Candida albicans*. Note the white pseudomembrane lining the open crop

can also be affected. The pseudomembrane is often friable and can be peeled off the mucosa. Other diseases may resemble candidiasis. The five top differentials for white patches/plaques in the oropharynx of birds are candidiasis, trichomoniasis, the wet form of fowl (avian) pox, aspergillosis, and vitamin A deficiency. A differential diagnoses for a roughened crop lining besides candidiasis is capillariasis.

Diagnosis/prevention/treatment

Diagnosis for candidiasis is usually made using gross and histopathological lesions (Figure 14.4), although fungal culture can also be considered. Candidiasis can be prevented by focusing on primary husbandry or infectious disease problems and avoiding unnecessary use of antibiotics, especially in young birds; copper sulfate or nystatin might be an effective treatment [8].

Lesions of the crop

Abnormalities in the crop that can be detected during a physical examination include crop enlargement. Gallinaceous birds that have recently eaten have a full crop, which feels doughy on palpation. This is normal and should disappear within a couple of hours. However, if the crop does not decrease in size, it could indicate crop stasis. A crop wash can be collected for cytological evaluation and culture, if needed. Radiographs can also be performed to determine whether the crop is enlarged by a space-occupying mass (e.g., neoplasm or foreign body). A bird with an enlarged crop should be treated
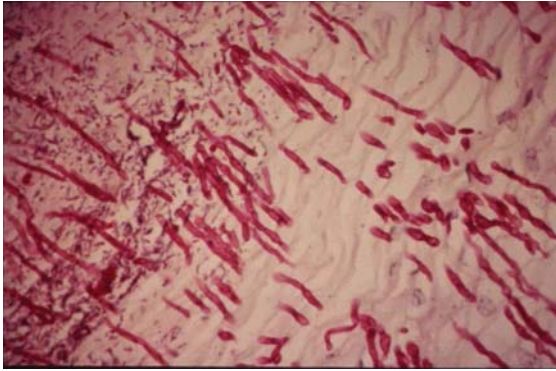
Figure 14.4 Candidiasis of chicken crop. In histologic section there is epithelial hyperplasia and the crop epithelium is infiltrated with linear pseudohyphae consistent with *C. albicans* (Periodic acid-Schiff stain).

gently because excessive handling and manipulation can cause aspiration pneumonia if there is excessive crop fluid. The crop can be enlarged as a result of excess fluid, from an impaction resulting from indigestible materials or tumor, or from ingestion of a foreign body. A fecal examination or crop wash cytology preparation may reveal the presence of crop worms.

Pendulous crop

The crop can become enlarged and filled with fluid. Crop washes do not recover any fungal elements, only foul smelling liquid. The exact cause of pendulous crops is not known and there may be a genetic component as it has often been observed in related birds. Surgical reduction can be attempted but its effectiveness has not been documented.

Crop impaction

Poultry have been known to ingest items out of curiosity or as a response to stress. Ingestion of poorly digestible items (e.g., grass, newspaper, sawdust shavings/wood chips, and feathers) has been known to cause crop impaction [9]. Impaction can also occur when poultry are exposed to new environmental substrates, especially floor substrates. Surgical removal of the impaction is necessary to alleviate the problem; however, the condition may not be recognized by the owner until the bird is near death.

Candidiasis

As described in the Oropharynx section, candidiasis is one of the most common crop diseases occurring for poultry.

Crop worms (capillariasis)

Although there are several nematode species that can be found in the crop, *Capillaria annulata* and *Capillaria contorta* are the most prevalent and are diagnosed most often in gamebirds (pheasants, partridges, and quail), but can also occur in turkeys. These nematodes are long and slender, and are often referred to as threadworms, and are less than 60mm long. These nematodes generally have a 30-day life cycle from ovum to adult. Adults embedded in the crop mucosa produce ova that are shed in the feces. The ovum must mature in the intermediate host (earthworm for *C. annulata*) or while free in the environment (*C. contorta*) [10].

Diagnosis

When embedded in the crop mucosa in large numbers, the affected birds can be depressed, weak, and emaciated. There can often be a history of sudden death without previous signs. Affected birds might gasp and appear as if they are having trouble breathing. With light infections, one may observe crop stasis or increased white fluid in the crop. In mild infections, the crop mucosa can be covered with a thin, white film; while in heavy infections, the entire crop wall can be thickened, fluid-filled, with a rough, irregular mucosa covered by a thick white, fibrinonecrotic exudate. The infection can extend beyond the crop into adjacent regions of the esophagus.

Treatment

The off-label medications fenbendazole or levamisole have been used but cannot be used if consumption of meat or eggs is intended. As infections are most severe in floor-raised birds, rotating or moving pens to decrease the build-up of ova in the soil is recommended. Moreover, the soil should be dry and well-drained to decrease the number of earthworms [11].

Lesions of the intestines

Intestinal disease is usually manifested as diarrhea or weight loss. Adherence of excessive feces to the vent feathers (pasty vent) indicates repeated bouts of diarrhea. In addition to the physical examination, the quality of the feces provides potential clues as to the causative agent. When diarrhea is present, it is important to consider the age of the affected bird and other clinical signs. In poultry, there are many diseases, while not primarily intestinal diseases, that can affect the gastrointestinal system to cause diarrhea. To work-up diarrhea cases, a fecal examination and

fecal bacterial culture are the primary diagnostic tools. Feces can be examined by zinc sulfate or sugar flotation to identify parasitic ova/oocysts. Fecal culture can determine whether *Salmonella* is present, especially in young birds, but culture may have to be performed over several sampling periods because *Salmonella* is shed intermittently. In addition, physical visualization of the feces can help with differential diagnoses. Blood analysis can identify potential blood loss if feces are not readily available. Serum should be collected to determine the presence of respiratory pathogens that can cause diarrhea.

Salmonella pullorum

Salmonella pullorum infection is an infectious, egg-transmitted disease affecting chicks and turkey poults. This disease is often associated with white diarrhea and high mortality in young birds, whereas adults are non-symptomatic carriers of infection. This disease has been known as bacillary white diarrhea or pullorum disease. *Salmonella pullorum* is a Gram-negative, rod-shaped bacterium that is usually poultry-specific and is closely related to *Salmonella gallinarum*, the causative agent of fowl typhoid. These two bacteria share surface antigens and the pullorum test can be used to identify reactors to both pullorum disease and fowl typhoid [12].

Young chicks and turkey poults are particularly affected and infection is often fatal. Older birds are more resistant and may not show clinical signs or may serve as inapparent carriers, but can transmit this infection through the egg to the hatchling [13]. Infected hatchlings can also transmit the infection horizontally to other birds in the hatcher. This disease is monitored through the National Poultry Improvement Plan (NPIP) in the United States. Many states provide training for flock owners or veterinarians to become certified blood testers for pullorum-typhoid.

Because this disease is primarily egg-transmitted, the concern for backyard flocks is that infected hens lay infected eggs, which hatch to produce infected chicks. Infected chicks that do not die can produce infected eggs at sexual maturity to repeat the cycle. Hence, this disease is of concern for flock owners who hatch their own breeding stock.

Peak mortality occurs about 2–3 weeks after hatching and can begin prior to 10 days of age.

Clinical signs

In severe cases, dead chicks can be found in the hatcher. Pasty white vents (cloaca) are noted in affected birds (Figure 14.5), which appear chilled and are reluctant

Figure 14.5 Turkey poults with vent feathers stained with feces.

to eat. Adult birds that are necropsied occasionally have misshapen ovaries, pericarditis, and peritonitis, but some have no lesions. Young birds may have no gross lesions if the infection is peracute. White to gray nodules in the heart, liver, cecum, and gizzard may be seen. Moreover, cecal plugs, firm, caseous, yellow cores found in the lumen of the cecum, are characteristic of this disease (Figure 14.6). Septic arthritis, including swollen hock and wing joints, has been noted in some birds; urates accumulate in the ureter as a result of dehydration. Birds may also have septicemia with hepatosplenomegaly (hepatitis).

Diagnosis

Bacterial culture of sick birds (definitive) and blood testing (whole blood plate test) in adult breeder birds are needed to identify reactors. The same killed Salmonella antigen is used to detect antibodies to both *Salmonella pullorum* and *Salmonella gallinarum* (fowl typhoid) in the whole blood plate test. A tube agglutination test is preferred for turkeys [12]. A differential diagnosis

Figure 14.6 Salmonellosis is often associated with formation of caseous cecal cores, particularly in young birds. Note that the open cecum contains an inflammatory core.

should include fowl typhoid; colibacillosis; chilling or overheating associated with white diarrhea; omphalitis (navel infection) caused by *Escherichia coli*, *Pseudomonas*, or *Staphylococcus*. To prevent this disease, birds should be purchased only from NPIP-approved hatcheries. This disease may be common in backyard flocks.

Treatment

This disease is reportable, so backyard flocks are euthanized, rather than treated, under the supervision of the state regulatory agency [12].

Salmonella gallinarum

Fowl typhoid, which is caused by a gram-negative, non-motile rod-shaped bacteria *Salmonella gallinarum*, is an infectious disease that primarily affects chickens and turkey. It has many features similar to pullorum disease as both of these bacteria share surface antigens and hence cross-agglutinate on the pullorum test [12]. This disease usually affects young adults or mature chickens, and is occasionally reported in chicks and poults. Ducks, geese, peacocks, pheasants, and turkeys are more resistant than chickens. Fowl typhoid usually affects chickens that are older than 12 weeks with reported losses of up to 50% [8,14].

Similarly to pullorum disease, fowl typhoid is egg-transmitted; however, it is most frequently transmitted horizontally between adults. As with other *Salmonella* infections, it can also be spread between houses by rats, wild birds, and humans, which serve as fomites. Adults have a higher mortality when compared with pullorum disease, which mostly causes mortality in young birds.

Clinical signs

If chicks and poults are infected, clinical signs include dead or dying birds in the hatcher with whitish, pasty vents; anorexia; and labored breathing. Growing and mature birds usually have an acute disease, with dead birds found on the nest or floor. Affected birds can also show a drop in feed consumption; depression with pale combs; high fever with associated open-mouth breathing; and greenish diarrhea. Lesions in chicks and poults are similar to pullorum disease. Adult birds may have bile-stained (bronze) livers, occasionally with necrotic foci, enlarged dark spleens, hepatosplenomegaly, and enteritis.

Diagnosis

Bacterial culture is definitive, and it is best to use a whole blood plate agglutination test to check for reactors to both pullorum disease and fowl typhoid [14]. To prevent this disease, purchase chicks and poults from NPIP-approved hatcheries. NPIP serologic monitoring of breeder flocks eliminates breeder sources of infection.

Treatment

As this is a reportable disease in the United States, reactor birds are reported to the state regulatory agency and culture-positive birds are euthanized, rather than treated.

Paratyphoid *Salmonella* Infection

Paratyphoid *Salmonella* infection is characterized by lesions of septicemia, and caused by one of over 2000 paratyphoid S*almonella* species, *Salmonellae,* which have a wide host range. Over 250 Salmonella species have been isolated from chickens. This is a disease that usually occurs in poultry and many may carry the bacterium but not demonstrate any clinical signs. The infection can be devastating in chicks, poults, and gamebirds of less than three weeks of age. Infected birds are intermittent fecal shedders [15]. Moreover, egg shells may become contaminated by these carrier birds; hatchery contamination can occur from infected hatchlings; and rats and mice can perpetuate this disease on the farm. One particular strain, *Salmonella enteritidis (SE)*, can be vertically transmitted from hen to egg and has been associated with foodborne illness in humans when contaminated raw eggs are pooled and improperly cooked [16]. Transmission of SE in feces can be enhanced when infected hens are stressed by molting, which at one time was induced by feed deprivation [15].

Clinical signs

While most birds do not display any clinical signs, affected birds are listless and huddle together. They have diarrhea and pasting of feces on the vent [17,18]. Diagnosis is made by culturing the agent from the intestines and organs such as the liver. It may be difficult to prevent this disease because a wide variety of animals can serve as carriers.

Infectious bursal disease

Infectious bursal disease, often referred to as IBD or Gumboro disease, is a highly contagious viral disease of 3 to 6-week-old chickens characterized by high mortality, anorexia, diarrhea, and depression. The virus has a preference for lymphoid tissue, primarily the bursa of Fabricius and may cause prolonged immunosuppression of chickens. The causative agent is a virus belonging to the genus Birnavirus, which replicates in B lymphocytes, is very stable, and persists for long periods in poultry houses, even when they have been thoroughly cleaned and disinfected. This disease spreads rapidly within a flock by direct contact, inhalation, or contaminated feed and water. The darkling beetle can also spread the virus [19].

Clinical signs

Clinical signs can be seen in 48–72 hours after infection. Initially, affected chicks appear depressed, have ruffled feathers, and a whitish or watery diarrhea may be present. As the disease progresses, anorexia, dehydration, trembling, and death can occur. Vent picking can be observed. In affected flocks, morbidity may reach 100% and mortality may vary from 0% to 30%. The subclinical form of IBD has no clinical signs, but immunosuppression occurs to make the birds more susceptible to other diseases, such as E. coli, coccidiosis, necrotic dermatitis, and necrotic enteritis. These birds may have poor responses to vaccination. Necropsy reveals dehydration; hemorrhages in the thigh and pectoral muscles; and a bursa of Fabricius that is swollen and hemorrhagic or edematous [20]. The bursa atrophies to approximately 1/3 of the original weight by day 8 post-infection. Kidneys can be swollen with urate retention in ureters.

Diagnosis

In acute IBD, a presumptive diagnosis can be made based on the lesions observed in the bursa of Fabricius and clinical signs that are typical of IBD. A positive diagnosis of IBD can be made by histological examination of the bursa or by virus isolation. The bursa and spleen are the tissues of choice for isolation of IBD virus. There is no specific treatment that changes the onset of immunosuppression after infection. When secondary infections are present, specific treatment for the secondary disease is suggested. As the IBD virus is very stable in the poultry house environment, good sanitation procedures are essential in helping to reduce exposure in subsequent flocks [19]. The best method to control IBD in chickens is by vaccination at 1 day of age and re-vaccinated at 7–14 days. IBD vaccination is rarely practiced in small chicken flocks.

Coccidiosis

Coccidiosis, which is caused by the protozoan parasite *Eimeria* spp., affects the intestinal tract of poultry. These parasites are species-specific so coccidia that infect chickens do not infect turkeys and vice versa. Most outbreaks involve infection with two or more species of Eimeria.

Infections are more common in floor-raised birds, but can occur in caged birds. The life cycle is initiated by ingestion of sporulated oocysts (eggs) and usually takes 4–6 days to complete. A single, mature oocyst (egg) contains four sporocysts, and each sporocyst contains two sporozoites (eight sporozoites in each oocyst), which can sporulate in less than 48 hours (under warm and moist conditions). These infected oocysts are then consumed by the bird [21].

Clinical signs

Affected birds display pale combs and wattles from blood loss in the intestines, ruffled feathers, depression, blood in droppings, and shivering. The mortality rate may increase, particularly in young birds. Decreased egg production can occur in adult birds. Each species affects a different part of the intestines, so lesions vary depending on the *Eimeria* involved. *Eimeria tenella* causes marked cecal hemorrhage and cecal cores in chickens.

Diagnosis

Diagnosis is made by observing gross lesions and fecal floatation, or by confirming the presence of intestinal sexual or asexual forms of coccidia by histopathology. Alternatively, segments of infected intestine can be opened and the mucosa can be gently scraped with a glass coverslip. A coverslip is then placed on the glass microscope slide and the oocysts can be observed using a 40X objective lens [22].

Treatment/prevention

To control coccidiosis in the flock, anticoccidial drugs that kill (coccidiocidal) or decrease the growth rate (coccidiostat) of coccidia should be used on a semi-annual

rotational basis to avoid resistance build-up in the parasite. In addition, live, attenuated coccidial vaccines containing up to six species of *Eimeria* have been used on farms with severe infections. Effective control includes killing oocysts in the environment or preventing contact with viable oocysts (using deep litter and salting the floor with 60–80 pounds of rock salt per 100 ft^2 before placing litter). Litter should not be recycled between flocks, or at least the top three inches of used litter should be removed and replaced [23] (see Chapter 6).

Hemorrhagic enteritis (HE)

Hemorrhagic enteritis is an acute disease of young turkeys of 4 weeks of age or older, and is characterized by depression, massive hemorrhage into the intestinal tract, and sudden death. Mortality is variable, but can be high. In the subclinical form, HE is characterized by immunosuppression and secondary bacterial infection, especially from *Escherichia coli*. HE is generally considered to be the most immunosuppressive viral infection of turkeys that is caused by a type II adenovirus, a double-stranded RNA virus with an icosahedral morphology [24]. Transmission of the virus occurs by ingestion of contaminated feces. Contaminated litter may infect subsequent flocks in the same house, as the disease recurs in houses in which it has occurred previously. Equipment and boots may carry infected fecal material from farm to farm. There is no evidence of egg transmission.

Clinical signs

Clinical signs are usually observed in affected turkeys of 6–12 weeks of age, but may occur as early as 4 weeks. In the classical form of the disease, HE is characterized by rapid onset with depression, bloody droppings, and death. All signs usually occur within 24 hours. Dark red to brownish blood is found on the skin and feathers around the vents of dead or dying birds. In the subclinical form of the disease, there are no clinical signs of HE, but viral–induced immunosuppression can promote secondary *E. coli* infection. At necropsy, the intestines are distended, dark in color, and full of red or brownish blood. Spleens of infected birds are characteristically enlarged, fragile, and mottled. In subclinical cases, mild enteritis may be present in addition to lesions of colibacillosis [25].

Diagnosis

A presumptive diagnosis can be made based on clinical signs and lesions. Confirmation of the diagnosis can be accomplished by submitting spleens for histopathology [26]. Serology may also help support the diagnosis.

Treatment/prevention

There is no satisfactory treatment of affected birds. Supportive care and good management helps to minimize losses. Convalescent antiserum given within 24 hours of the onset of signs may prevent heavy losses. Secondary *E. coli* infections may be treated with antibiotics. A good biosecurity program should include an all-in, all-out procedure and thorough cleaning and disinfection of the premises [25]. Vaccination with a live, avirulent HE vaccine has been effective in reducing the clinical signs that result from HE. A variety of HE vaccines are commercially available.

Roundworms (ascariasis)

Ascaridia galli, a nematode residing in the upper small intestine of chickens and turkeys, causes the common roundworm infection. Young birds, less than 3 months of age, are most susceptible to intestinal damage and light-weight egg breeds (e.g., Leghorns) are more susceptible to infection than heavy meat-type breeds (e.g., Plymouth Rocks). In caged birds, infection can occur from exposure to contaminated flies. The life cycle is direct and takes 30 days to complete [27]. The eggs contaminate the environment and can remain infective for 160 weeks on the ground.

Clinical signs

Parasitized birds can show depression, weight loss, diarrhea, and decreased growth. Lowered egg production can occur in cases of heavy infection [27]. Worms in the small intestine can occasionally cause death by blockage/impaction [28]. Gross lesions can include a reddened intestinal mucosa (enteritis). Thin birds show atrophy of the breast muscle and decreased body fat [2]. Ascarids can migrate to the oviduct and become incorporated in the egg prior to shell formation [28,29]. Necropsy reveals worms that are large, yellow-white, and 5–11 cm long. Fecal flotation needs to be performed using zinc sulfate or sodium nitrate solutions.

Diagnosis

Confinement and cage rearing has reduced problems with most intestinal parasites. Using deep litter (4–6 inches of wood shavings) reduces exposure to parasite eggs, and proper clean up between flocks reduces future infections [23]. It is important to evaluate new birds for such parasites before introducing them to the flock.

Treatment

Piperazine is the treatment of choice (see Chapter 6).

Cestode (tapeworm) infections

Tapeworms are usually of no clinical significance in poultry, although these parasites may have a minor effect on growth rates [30]. Seven species affect chickens and all require an intermediate host to complete the life cycle. *Choanotaenia infundibulum* and *Raillietina cesticillus* are the most commonly found tapeworms in caged pullets or layers because of consumption of an intermediate host (housefly or beetle). Gross lesions of heavy tapeworm burden are usually striking, but the infection likely has little clinical effect. There is no effective chemical treatment. Butynorate (Tinostat) is no longer available. Prevention should focus on control of intermediate host: Flies, beetles, and ants. Heavy tapeworm infections indicate that there is a need for fly or darkling beetle control on the premises [31].

Capillariaisis (*Capillaria obsignata*)

Capillaria obsignata, is a 0.5–1.8 cm long threadlike nematode that primarily affects the small intestine of chickens, with a direct life cycle of about 18 days. Eggs are infective for up to 102 weeks in the environment. Usually, young adults display signs, and the effects of the infection can diminish as the bird ages. Infected birds are often in poor condition and may have diarrhea, weight loss, pale combs and wattles, and decreased egg production [32]. "Platinum egg yolks," white egg yolk caused by a decreased absorption of vitamin A and carotenoids in the intestine, can occur. Unless there is a severe infection, mortality is minimal.

Adult worms partially burrow into the small intestines and cecum to cause hemorrhagic enteritis, and the intestinal wall may become thickened in severe cases. Affected layers can have decreased egg production and pale egg yolks.

Diagnosis

For heavy infections, diagnosis can be made by scraping the intestinal mucosa and placing it onto a glass slide for microscopic evaluation (Figure 14.7). For light to moderate infections, wash intestinal scrapings through a fine mesh screen (100 mesh) to observe the worms. Regular and proper sanitation should prevent infections.

Treatment

Birds that are in lay should be supplemented with vitamin A to maintain egg yolk color. Fenbendazole can be effective as treatment [33] (see Chapter 6).

Necrotic enteritis

Necrotic enteritis is a common enteritis of chickens that causes depression and sudden death. The disease
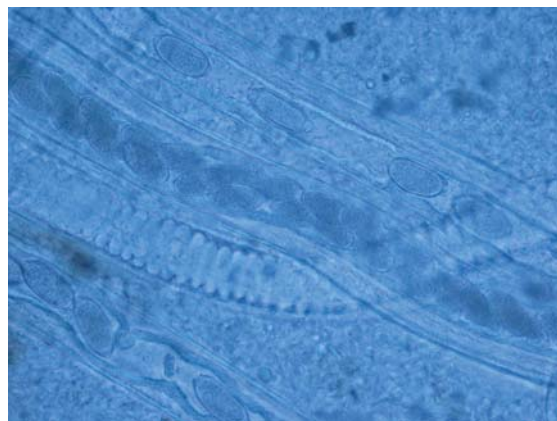
Figure 14.7   Mucosal scraping of the intestinal mucosa reveals *Capillaria* worms containing distinctive bioperculate ova.

is observed less often in turkeys and older chickens. Broiler chickens are most often affected. This disease often occurs concurrently, or following an outbreak of coccidiosis or occasionally ascariasis [34]. The etiologic agent is *Clostridium perfringens*, type A and C, which produce alpha and beta toxins that cause necrosis of the intestinal mucosa. This gram-positive bacillus requires anaerobic conditions for culture [18,35].

Clostridium is ubiquitous and infection is probably initiated by changes in intestinal pH, damage from coccidiosis, and gut stasis that promote conditions for growth of *Clostridium perfringens*. Necrotic enteritis has been associated with feed containing wheat, which possibly causes alterations of the intestinal pH [36]. This disease usually occurs when birds are around 3 weeks of age.

Although *Clostridium* may be found in the soil, its spores can reach high concentrations in litter that is not replaced periodically and infection can recur in contaminated houses [18].

Clinical signs

In affected birds, sudden death can occur, or birds can appear weak and hold their heads down. The small intestines are swollen and are filled with a tan to yellow, thick pseudomembrane ("turkish towel") (Figure 14.8a and b). Moreover, water in the crop and marked dehydration may occur in affected birds [36].

Diagnosis/prevention

Diagnosis can be made based on history (recent coccidiosis outbreak), gross lesions, and optional bacterial culture. To prevent future outbreaks litter should be changed periodically; a coccidial prevention program
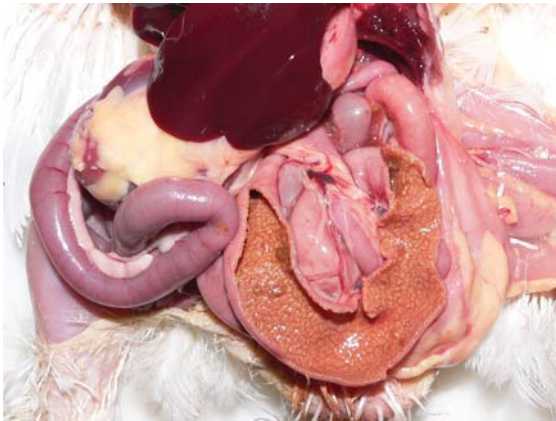
Figure 14.8 Necrotic enteritis in a three-week-old broiler chicken. The dilated small intestine is lined by a tan pseudomembrane of fibrinoecrotic exudate.

should be established; and wheat midlings should be avoided in the ration. Moreover, the birds should be assessed for infectious bursal disease that could immunocompromise the flock.

Treatment

The use of ionophore anticoccidials, such as monensin, has helped reduce the occurrence of necrotic enteritis. During outbreaks of necrotic enteritis, birds may be treated with a Gram-positive spectrum antibiotic [37].

Avian mycobacteriosis

Avian mycobacteriosis, also known as avian tuberculosis, avian TB, TB, or mycobacteriosis, is a chronic bacterial infection that forms visceral granulomas (nodules) in a variety of mature/adult birds, resulting in progressive wasting and death. The causative agent is *Mycobacterium avium*, subspecies *avium*, a nonmotile, nonspore-forming bacterial rod that stains acid-fast. It is highly resistant to pH, water, cold, and many disinfectants [38]. The granulomas (tubercles) of *M. avium* often develop in the intestinal tract. Tubercles rupture to release bacteria into intestinal lumen and subsequently into the feces. Infected feces contaminate feed, water, and litter [18]. *M. avium* is also zoonotic for humans [38]. Transmission takes place via ingestion of contaminated feed, water, and litter. Affected birds show progressive wasting with occasional diarrhea. However, birds can die suddenly without premonitory signs. On necropsy, birds appear light weight to emaciated, with marked atrophy of breast muscle and no internal body fat (Figure 14.9a). White to gray nodules

in the intestine, spleen, liver, and bone marrow may be present (Figure 14.9b) [39].

Diagnosis

A diagnosis can be made based on history, gross lesions, and histopathology. Histologic sections show

(a)
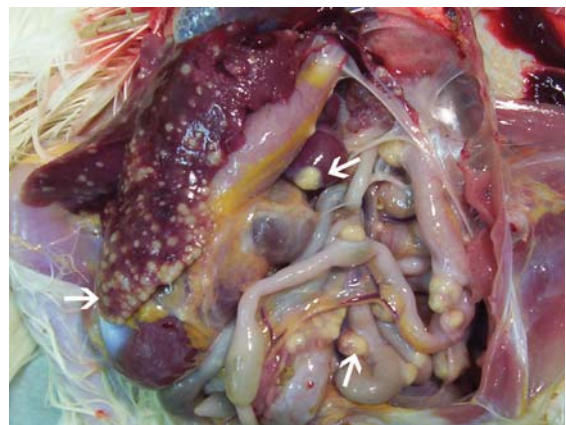
(b)

Figure 14.9 Aged laying hens with mycobacteriosis are often emaciated (a) with granulomas (arrows) (b) on liver, spleen and intestine

characteristic granulomatous inflammation with acid-fast bacterial rods in the center of the lesion. To prevent outbreaks, all-in, all-out breeder programs to eliminate infected birds are recommended. Thoroughly dry-clean and disinfect houses and equipment between flocks. Keep young and old birds separated and cull affected birds [8].

Avian chlamydophilosis

Avian chlamydophilosis, also referred to as psittacosis or ornithosis, is an acute to chronic infectious disease that can cause systemic, pulmonary, and enteric lesions. This agent is a public health concern as a zoonosis and is reportable in some states [40]. The causative agent, *Chlamydophila psittaci*, order Chlamydiales, is an obligate intracellular gram-negative bacterium that occurs in a wide variety of birds. Until recently, the microorganism had been referred to as Chlamydia.

Most outbreaks occur in young birds. This disease is extremely rare in poultry, and is only occasionally diagnosed in turkeys. Infected birds can act as carriers by intermittently shedding the agent in oculonasal secretions and feces. Young birds can contract infection by ingesting nasal secretions or fecal material.

Clinical signs

Clinical signs are often quite mild with low-grade respiratory signs or diarrhea. Turkeys may show signs of depression, weakness, anorexia, weight loss, nasal discharge, or marked yellowish green diarrhea. Necropsied birds may show fibrinous pneumonia, airsacculitis, hepatitis, pericarditis, peritonitis, and splenitis [41].

Diagnosis

Chlamydophilosis can be diagnosed by culture in embryonated chicken eggs, antigen capture assay on tracheal swab, or by performing a complete necropsy and histopathology examination. Macchiavello stain has been used to identify intracytoplasmic elementary (infectious) *Chlamydophila* bodies in impression smears of the lungs, spleen, and liver [42]. Other diseases to consider include *Mycoplasma gallisepticum*, Pasteurella multocida, avian influenza, and aspergillosis. There is no vaccine for chlamydiosis, so thorough cleaning and disinfection, as well as all-in, all-out management to prevent infection in young birds is recommended. *Chlamydophila* is prevalent in pigeons, so caution should be used if free-living or captive pigeons are present on the same farm (see Chapter 8).

Ulcerative enteritis

Although this is a common bacterial enteric disease of domestic bobwhite quail and other upland game birds, such as pheasants, chickens and turkeys can also be affected. The lesions are characterized by multifocal discoid ulcers in the small intestine and multifocal hepatic necrosis [43]. The causative agent is *Clostridium colinum*, a gram-positive, spore-forming bacterial rod, and is spread in the feces of infected birds [44]. *Clostridium colinum* is hardy and can persist in the soil or litter for several months. *Clostridium colinum* spreads rapidly from bird to bird and via flies that have been in contact with contaminated feces. The disease is rare in birds that are raised in cages.

Clinical signs

Affected quail are usually 6–10 weeks of age and have white, watery diarrhea with subsequent sudden death. Birds that do not die suddenly are depressed with closed eyes and ruffled feathers. Infected birds are thirsty and huddle around the drinkers. The course of disease lasts about 2 weeks and can result in nearly 100% mortality in bobwhite quail [11]. Lesions are prominent in most affected birds and include fluid-filled distended crops and deep punctate to discoid ulcers that are tan to gray in color in the small intestine (Figure 14.10). These ulcers often penetrate the entire wall of the intestine to result in peritonitis and adherence of intestinal loops. The liver may or may not contain pale foci of necrosis on the capsule and on cut surfaces. Birds that live for more than 10 days can become emaciated.

Diagnosis/treatment

Diagnosis is usually made using history and gross lesions. The organism has specific growth requirements and bacterial isolation is generally not needed for a diagnosis. Affected birds can be treated with bacitracin, the antibiotic of choice, in the water at 0.25–0.50 gm per gallon of water for 7–10 days. Penicillin and tetracyclines can also be effective [45]. Ulcerative enteritis rarely occurs in birds that are raised on wire. If free-range is desired and if this disease occurs on the farm, rotation of pens on a regular basis can reduce exposure to *Clostridium* spores.

Newcastle disease (ND)

Newcastle disease is an acute, rapid-spreading, contagious disease of birds of all ages characterized by lesions in the respiratory tract, visceral organs, and brain. It causes minor to severe mortality in susceptible flocks, depending on the pathogenicity of the virus. The causative agent belongs to the family *Paramyxoviridae*, subfamily *Paramyxovirinae*, genus *Avulavirus*, species

Figure 14.10 Four-week-old Bobwhite quail with ulcerative enteritis. Note the pale, necrotic foci in the small intestine and liver.

Newcastle disease virus, and is a negative sense ssRNA virus. The agent is a paramyxovirus, an enveloped single- stranded RNA virus with helical capsid symmetry (100–150 nm diameter). There are nine serogroups of avian paramyxovirus and Newcastle disease virus is PMV-1 [46]. ND viruses are classified according to their pathogenicity for chickens. Pathogenicity is determined by inoculating virus directly into the brain of 1-day-old chicks, intravenously into the 6-week-old chickens or by characterizing amino acid sequences in the fusion protein. The velogenic strains produce severe disease and high mortality in susceptible birds. The mesogenic strains cause respiratory disease or marked drop in egg production in field infections, but with lower mortality. The lentogenic strains (e.g., B-1 and LaSota) only produce a mild respiratory disease. They are commonly used for vaccine production. The lentogenic strains can cause a moderate respiratory disease in broilers and pullets, particularly if complicated by secondary E. coli infection. Additionally, chicken embryos that are inoculated via the allantoic sac with Newcastle disease virus have different mean death times depending on whether the virus is velogenic (death in less than 60 hours), mesogenic (60–90 hours), or lentogenic (greater than 90 hours to kill embryo) [46,47].

The virus is present in the discharges from the respiratory and intestinal tracts. Therefore, the infectious ND virus can be transmitted by aerosol droplets, contaminated feed and water, off-farm movement of poultry, and infected wild birds. The greatest potential for spread of Newcastle disease is via humans and contaminated equipment.

Clinical signs

Clinical signs vary with the age of the birds, strain of ND virus, the immune status of the birds, and the environmental conditions. In young birds that have little or no maternal antibodies, or haven't been vaccinated, the signs can be severe. Birds under stressful conditions are also more susceptible to severe clinical signs. The velogenic form spreads rapidly through a susceptible flock. Birds may be found dead without any signs. Initially, depressed birds are observed with increased respiration. There is progressive weakness and prostration. The birds develop a watery greenish diarrhea. A marked cough, gasping respiration, and nasal and eye discharge are often present. Comb and wattles may turn dark and bluish, and birds may develop swollen heads. Birds that survive the initial acute phase show involvement of the nervous system. Egg production drops sharply and deformed eggs may be present. Mortality is usually over 90% in a susceptible flock [47]. For the mesogenic form, the clinical signs are similar to the velogenic form, but less severe. Mortality may vary from 5–50%, depending on the age of birds and environmental conditions. Nervous signs may occur but are not common. The lentogenic form is characterized by mild respiratory signs and a sudden drop in egg production. The egg production returns to normal within a few weeks and birds completely recover from the disease. In young susceptible birds, severe respiratory disease can occur. Lesions found on necropsy vary depending on the strain of the infecting virus. With the velogenic strain, there are varying degrees of congestion and hemorrhages in visceral organs, including the proventriculus, ceca, and small intestines [48]. Chickens and turkeys that are infected while in lay usually have egg yolk in the abdominal cavity (egg yolk peritonitis). With the mesogenic form, hemorrhages may occur in the proventriculus and less commonly in the small intestines. There is clear fluid present in the nasal passages, larynx, and trachea. In the lentogenic form, no clinical signs or a mild tracheitis may be seen in early cases [49]. A presumptive diagnosis can be made based on the clinical signs, lesions, and

serological tests. A positive diagnosis of the causative virus can only be made by isolation and identification of the virus by embryonated egg inoculation. Specimens for attempting isolation of the virus should be selected from birds that show early clinical signs of the disease. Swabs should be taken from the trachea, cloaca, and brain [47].

Treatment

There is no effective treatment against the ND virus. Broad-spectrum antibiotics may help to prevent secondary bacterial infections. Good management practices to reduce any additional stress on the birds aids in recovery. Prevention of Newcastle disease involves a sound biosecurity program and an effective vaccination program. Keep unauthorized personnel out of the poultry area, and maintain a good clean out and sanitation procedure. Frequency and timing of Newcastle vaccination depends on the type of bird and the incidence of Newcastle disease in the area [50]. Chickens can be vaccinated with the Type B vaccine strain at 1 day, 14 days and 6 weeks of age. Laying chickens can be vaccinated with the LaSota strain at 13–16 weeks and then every 60 days during production. The Newcastle vaccine is usually administered in combination with the infectious bronchitis vaccine. Turkeys can be vaccinated with B, Type, B, Strain at 3 weeks and then revaccinated with the LaSota strain at 8 weeks (see Chapter 9 for more information).

Avian influenza

Avian influenza is an infectious respiratory disease of poultry, especially turkeys, and is characterized by respiratory symptoms, depression, and lowered feed and water consumption. In laying birds, there is a severe drop in egg production and hatchability. Avian influenza is a type A orthomyxovirus, an 80–120 nm diameter, icosahedral enveloped single-stranded RNA virus, that can infect a wide variety of birds, including most game birds. The virus can be inactivated in 3 hours at 56°C and 30 minutes at 60°C. In warm weather, the virus can survive for 35 days in water, soil, manure, and on contaminated equipment. In cold climates, the virus can survive for up to 3 months [51]. The virus is found most often in wild waterfowl and shore birds, which serve as natural reservoirs by carrying and transmitting the virus, usually without showing clinical signs, so exposure to backyard flocks with such free-living birds should be reduced [52]. The AI virus is rapidly destroyed by most commercial disinfectants. The virus exists in high pathogenic (HPAI, ability to cause severe disease) and low pathogenic (LPAI, mild

disease) forms that are categorized by both live bird inoculation and determination of amino acid sequences at the hinge region of the hemagglutinin molecule. Avian influenza viruses are also characterized by the glycoproteins attached to the surface of the viral envelope. One glycoprotein is hemagglutinin (H), of which 16 types can be encoded by the viral genome. A second glycoprotein is neuraminidase (N). The genome of the AI virus can encode for one of nine different N types. AI virus is primarily described by the H and N types on the virus surface. The AI virus has the ability to change form through antigenic drift (point mutation in the H or N) or antigenic shift (two or more viruses with differing H and N types sharing genomic segments to create a new virus with a novel combination of H and N expressed on the virus envelope). HPAI usually take the form of H5 and H7 [53,54]. The virus is transmitted by direct contact between infected and susceptible birds and indirect contact, including aerosol droplets or exposure to virus-contaminated boots, clothing, or equipment.

Clinical signs

Poultry that have been infected with low path AI (LPAI) can show decreased egg production, respiratory signs (coughing, sneezing), or no clinical signs at all. Secondary infections with *Escherichia coli* can increase the flock mortality. HPAI can cause rapid death without clinical signs, or signs can involve the respiratory (cough, sneeze), nervous (paralysis, ataxia), and digestive systems (diarrhea), and decreased egg production. Edema of the head and neck is commonly observed. Poultry that have been infected with LPAI may have no gross lesions or can have fibrinous exudates in the trachea, sinuses, air sacs, and conjunctiva. The oviduct can be inactive or shrunken. With HPAI infection, lesions are severe. The comb or wattle may be shrunken, ulcerated, or cyanotic (purple). Edema of the face and feet along with hemorrhages on the shanks are commonly observed. Hemorrhages or fibrinous exudates can cover the pericardial sac, mesentery, air sacs, abdominal fat, trachea, intestine, and oviduct. In addition, hemorrhage and necrosis can be observed in the cecal tonsils and proventricular glands [55].

Diagnosis

Diagnosis of LPAI is based on serology (agar gel immunodiffusion test) and virus isolation to differentiate the infection from other diseases such as colibacillosis, Newcastle disease, and infectious bronchitis. HPAI is diagnosed by observing the extreme clinical signs and must be differentiated from exotic Newcastle disease. Nine to ten-day embryonated chicken eggs are inoculated via

the allantoic sac to cultivate the virus [10]. Polymerase chain reaction analysis can also be performed on cloacal or oral swabs of ill or dead birds [54].

Treatment

There is no practical treatment for avian influenza virus infections except to prevent secondary bacterial infection. Antibiotic treatment has been used to reduce the effects of concurrent bacterial infections. As the disease is spread from infected bird to susceptible bird and by contaminated boots and equipment, strict biosecurity is important. In outbreaks that involve highly pathogenic subtypes, eradication programs are used to control the disease. In low pathogenic cases, use of a killed vaccine (autogenous) has been allowed. Random vaccination is not permitted by the United States Department of Agriculture (see Chapter 9 for more information).

Heterakis gallinarum (cecal worm)

Heterakis gallinarum, a cecal worm that is found in chickens, turkeys, and pheasants, can harbor the protozoan *Histomonas meleagridis*, the causative agent of blackhead in turkeys, in its eggs. Hence, chickens can serve as a possible source of infection for turkeys if the turkeys are raised in proximity to chickens. *Heterakis gallinarum* are thin, white, 0.5–1.5 cm long worms that can reach the infective stage at two weeks or less, depending on ambient temperature, and can remain infective for up to 230 weeks [56]. *Heterakis gallinarum* eggs are occasionally ingested by earthworms, which can also serve as a source of infection when ingested by poultry. Affected birds usually have no clinical signs other than the presence of worms in the ceca (Figure 14.11); however, in heavy infections *H. gallinarum* or related species can imbed in the cecal mucosa to form mural granulomas (Figure 14.12). Prevention is similar to that described for ascarids [8,57] (see Chapter 6 for more information).

Cloacal prolapse

The cloaca (vent) of the laying hen temporarily everts when an egg is laid. The cloaca can become permanently everted and inflamed if is traumatized by other hens (cannibalism, pecking, peckout, etc.) or if the egg being laid is particularly large relative to the cloacal lumen. Birds dying from peckout/prolapse show hemorrhage around the vent area and most of the intestinal tract can be absent as a result of removal by other birds. Factors affecting the severity and incidence of cloacal prolapse include strain of bird, the quality of beak trim, quality of ration, amount of floor, feeder, or drinker space, high light intensity, and large egg size. Young birds early in
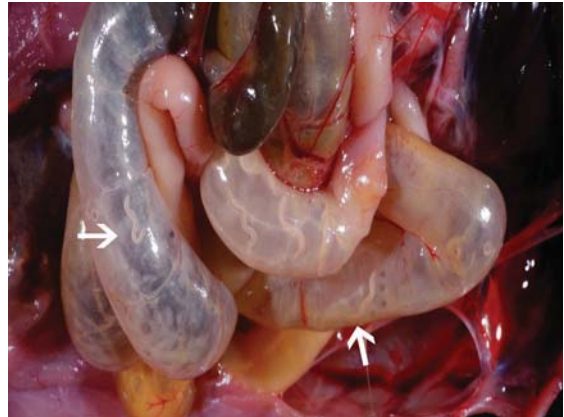
Figure 14.11 Open cecum of adult floor-raised hen distended with cecal worms (*Heterakis gallinarum* arrows).

Figure 14.12 Adult chicken with cecal worms (*Heterakis*). The nematode can occasionally invade the cecal wall to form inflammatory nodules.

lay are more susceptible to cloacal prolapse because the cloacal lumen has not become fully expanded to accept relatively large eggs. Additionally, vent trauma and cloacal prolapse in floor-raised chickens can be decreased by offering perches and obstacles to provide protection for hens, and by maintaining adequate nest: hen ratios (1 nest: 4 hens) to decrease fighting for nest space [8].

Diseases of the liver

Liver disease may be detected using biochemical tests. Physical examination can reveal an enlarged liver [58]. A normal-size liver should not be palpable past

the keel bone [2,58]. Palpation of part of the liver extending past the keel bone can indicate hepatomegaly [2]. Radiographs may also indicate liver enlargement. Marek's disease is a common cause of liver disease in some backyard flocks.

Other liver lesions include the masses seen in avian mycobacteriosis and should be the primary differential for nodular lesions in the liver along with colibacillosis. Crater-like lesions are unique with histomoniasis being one of the most common diseases.

Some peracute bacterial diseases, which cause septicemia, may cause a slight liver enlargement. Acute bacterial diseases can cause multifocal white spots in the liver with colibacillosis being the most common. While not as common, white spots on the liver can indicate larvae migrations in the liver from severe roundworm infections.

Perihepatitis, manifested as a white film around the liver, can be caused by bacterial diseases with chlamydiosis, salmonellosis, and colibacillosis being top differentials. These tissues should be cultured and also placed in formalin for histopathology.

Marek's disease (MD)

Marek's disease is a herpesvirus infection that causes lymphoma of T lymphocytes and is ubiquitous throughout the world. Tumors can occur in the nerves, ovaries, testes, viscera, eyes, muscles, and skin. Leg paralysis resulting from Marek's disease is often referred to as range paralysis. The disease is caused by a cell-associated herpesvirus (double-stranded DNA virus, hexagonal enveloped virus). There are three serotypes of the MD virus: serotype 1, the oncoviruses (tumor-causing); serotype 2, the non-oncogenic viruses; and serotype 3, the herpes virus turkey (HVT) [59]. The virus is intranuclear (cell-associated) and normally cannot live outside the host cell, as it is protected from the environment by the host epithelium. Infectious virus is only produced in the feather follicle epithelium and spreads by direct or indirect contact between birds. The infectious virus contaminates the premises through infected molted feathers and dander. Birds become infected when they inhale dust that contains the virus. Contaminated dust may remain infectious for several months. Many apparently normal birds are carriers and can transmit the infection. Some birds have been found to shed virus from skin for as long as eighteen months. Darkling beetles may also act as a mechanical vector [60].

Clinical signs

In acute outbreaks, birds become severely depressed, anorectic, and uncoordinated followed by unilateral or bilateral paralysis of legs and wings. Many birds become dehydrated, emaciated, and eventually die. The extremities affected include the legs, wings, and neck. In an infected flock, mortality gradually builds and generally persists for 4–10 weeks. Ocular Marek's disease is characterized by decreased pupil size and irregular diameter, and the iris becomes gray ("gray eye"). A number of factors influence the extent of losses in affected flocks, such as virus strain, dosage, route of exposure, and genetic resistance of the host. Immunosuppression can occur as a long-term effect. Gross lesions can usually be found in one or more peripheral nerves, particularly the sciatic and brachial nerves. Affected nerves are characterized by loss of cross-striations, gray or yellow discoloration, and may be swollen (Figure 14.13). Lymphoid tumors may be found in the gonads, heart, liver (Figures 14.14 and 14.15), lungs, kidneys, spleen, bursa, intestines, muscles, and skin. Skin lesions are not readily seen until feathers are removed, and the feather follicles may be enlarged and pale. The cloacal bursa is usually not involved.

Diagnosis

A presumptive diagnosis is made based on the presence of tumors and the observed paralysis; however, gross necropsy and histopathology are important to arrive at a definitive diagnosis and to differentiate Marek's disease from other forms of paralysis or recumbency [60].
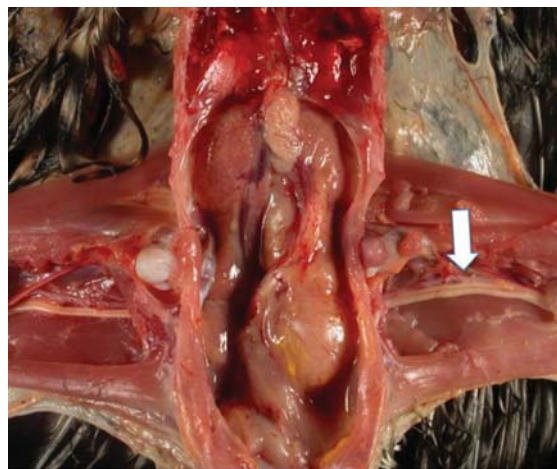
Figure 14.13 Eight-week old Barred Rock chicken with Marek's disease. Note the enlarged kidney (lymphoma). The right sciatic nerve is of normal thickness while the left sciatic nerve (arrow) is swollen, pale and has lost cross-striations.

Figure 14.14 Hepatic lymphoma (hepatomegaly) can be seen in Marek's disease.
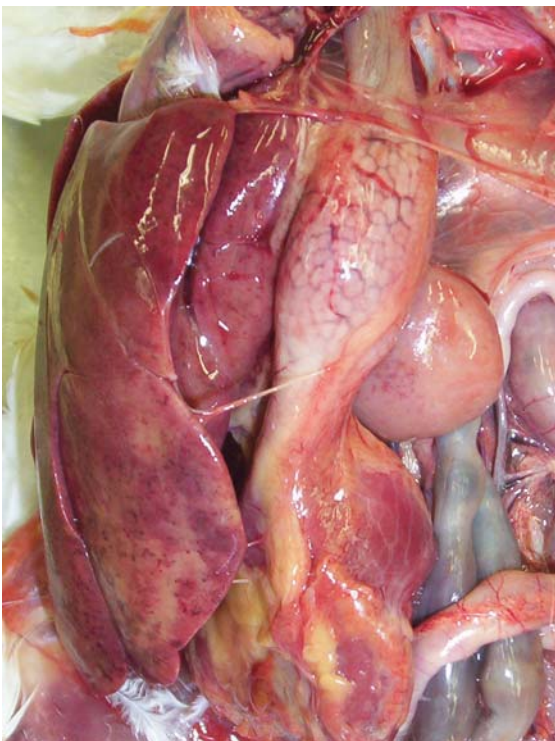
Figure 14.15 Nineteen-week-old pullet with hepatosplenomegaly caused by the lymphoid leukosis virus.

Treatment/prevention

There is no specific treatment for chickens with Marek's disease and the emphasis is on prevention. Vaccination against MD is effective in controlling the disease.

Marek's disease vaccine is usually administered on day 1. Three types of vaccines are commercially available: The HVT serotype 3, natural occurring avirulent isolates of serotype 2, and nononcogenic strains of serotype 1 (Rispens) [61]. For backyard flocks, the HVT vaccine is commonly used.

Lymphoid leukosis (LL)

Lymphoid leukosis is a viral disease of chickens that is characterized by the formation of tumors (lymphoma of B lymphocytes) in internal organs. Under natural situations, lesions are seen mainly in sexually mature birds because of the long incubation period (270 days) of the virus. The causative agent is an RNA virus (80–120 nm diameter) belonging to the avian type C oncoviruses [62].

The most common route of vertical transmission of the LL virus is from the infected oviduct to the progeny through the egg. Chicks infected through the egg are immunotolerant (serum antibody negative, viremia positive) and have a high incidence of tumors [63]. There is some horizontal transmission of the virus from bird to bird at a young age; these birds are only temporarily viremic and do develop antibody to the virus. Usually, only a small number of LL-infected birds develop lesions; the others remain as carriers and shedders [64]. LL is rarely seen in large-scale poultry production because of elimination of the oncovirus from primary breeder flocks.

Clinical signs

Clinical signs usually do not appear before 4 months of age and are nonspecific. Affected birds may appear pale, emaciated, and dehydrated. The comb may become shriveled, and occasionally cyanotic. There is a drop in egg production and loss of appetite. The abdomen is often enlarged and feathers are sometimes spotted with urates and bile. LL virus can also cause erythroblastosis (anemia, hepatosplenomegaly), myeloblastosis (bone marrow, leukemia, and hepatomegaly), and myelocytomatosis (deformation of flat bones of the skull and mandible). LL virus can also induce proliferation and activation of osteoblasts in bone; a disease known as osteopetrosis, characterized by formation of new bone on the periosteum and endosteum of long bones. These bones are heavier and thicker than normal. Lymphoma that is characterized by tumors or organ enlargement occurs in the organs, especially the liver and spleen (hepatosplenomegaly) (Figure 14.15). Tumors can also develop in the kidneys, lungs, ovaries, testicles, bursa of Fabricius, heart, and bone marrow [65]. Tumors vary in size and are soft, smooth, glistening, and gray to white.

Diagnosis

A presumptive diagnosis can be made based on the presence of tumors and the age of the birds. Usually with LL, lesions are seen in birds of 4 months and older, whereas, in birds affected with Marek's disease, lesions may appear as early as 4 weeks of age.

Diagnosis/treatment/prevention

A positive diagnosis requires histological examination. There is no effective treatment and no vaccine is available. It is helpful to cull all birds that are obviously affected. The best prevention method is the laboratory detection of infected breeders. Breeding leukosis-free offspring from leukosis-free breeders can eventually lead to eradication of the disease. An ELISA is available to test egg albumin or serum for the presence of avian leukosis antigen.

Colibacillosis

Colibacillosis is an infectious disease caused by the gram-negative rod *Escherichia coli* as the primary pathogen or as a secondary invader that causes septicemia, peritonitis, cellulitis, omphalitis, salpingitis, and airsacculitis. Colibacilosis, also referred to as *Escherichia coli* infection, coligranuloma, or colisepticemia, is caused by *Escherichia coli* that are serotypes 01, 02, or 078, but are also often untypeable. *Escherichia coli* is ubiquitous and is present in the intestines of birds and mammals [66]. It is disseminated in feces, and infections often result from management failures. Birds may be infected by direct contact with dirty litter and hatchers or contaminated egg shells.

Clinical signs

Affected birds usually display non-specific signs and include ill-thrift, ruffled feathers, enlarged and swollen navels (Figure 14.16), decreased appetite, depression, diarrhea, and pasting of feathers around the vent. Depending on the body system that is affected, there can be a variety of lesions, including airsacculitis, perihepatitis, and pericarditis, resulting from secondary invasion of *E.coli* into a primary subacute to chronic respiratory disease [67]. A white, friable material covers the air sacs, liver, and pericardial sac (Figure 14.17). The respiratory form of *E.coli* infection in juvenile birds is often preceded by *Mycoplasma*, Newcastle disease, or infectious bronchitis. Newly hatched birds have omphalitis (swollen, red, and crusted navels), which is caused by contamination of egg shells through a dirty setter, feces-covered eggs, or excessive moisture during storage of eggs. Cases of omphalitis resulting from colibacillosis should be differentiated from those caused by

(a)

(b)

Figure 14.16 a and b One-day-old chick with omphalitis (a) and yolk sacculitis (b) from colibacillosis.

other bacteria [68–70]. Birds may also have septicemia with hepatosplenomegaly (hepatitis), hemorrhages, and necrosis in affected organs [71]. Hepatitis and cecal cores were observed in turkeys with colibacillosis [72]. Septic birds can develop hypopyon (exudate within

Figure 14.17 Nine-week-old turkey hen with fibrinous pericarditis, perihepatitis, and airsacculitits resulting from colibacillosis.

Figure 14.18 Colibacillosis is the most common cause of salpingitis in laying hens. Note enlarged and exudate-filled oviduct (arrows).

the eye). Infected laying hens commonly develop salpingitis, in which the oviduct is filled with yellow, caseous exudate, with or without peritonitis [73,74] (Figure 14.18). Cellulitis ("scabby hip") with yellow exudate can accumulate underneath the skin of the hip, leg, and breast, particularly in broiler chickens [75].

Diagnosis/treatment

Histopathology, along with bacterial culture of affected organs, is required for diagnosis. Many *E. coli* strains are resistant to antibiotics, so a combination of treatments have been attempted for large flocks including: i) Combining 200 g Neomycin and 200 g Terramycin per ton of feed for 7 days, followed by 2 weeks of probiotic in the feed; ii) adding either household bleach (6–10 ounces per gallon stock metered at 1 ounce per gallon drinking water) or iodine disinfectant (8–12 ounces per gallon stock) for 10 days to reduce bacterial load

in the water; and iii) fog the house with a fine mist of VirconS (1% solution) or chlorine dioxide (0.5% with no activator) twice a day for 4–5 days in order to reduce aerosolized bacteria. To prevent outbreaks, a vigorous sanitation program in the breeder house, hatchery, and at the grow-out facility is recommended. Reducing aerosolized dust, routinely removing dead birds, and avoiding overcrowding in the poultry house are strategies to reduce outbreaks.

Visceral gout/urolithiasis

Gout/urolithiasis is a condition commonly seen in older layer flocks and is often related to kidney failure. On occasion, gout can be a very significant part of flock mortality, sometimes as high as 0.5% per week, but it is often associated with sporadic low-grade mortality. Kidneys can be damaged by low phosphorus diets, water deprivation, high vitamin D3 in the ration, or excessive calcium before sexual maturity (15–16 weeks) [76]. A nephrotropic infectious bronchitis (e.g., Australian T or Italian strain) can cause similar lesions. Losses resulting from gout tend to be chronic with the number of affected birds dependent on the way in
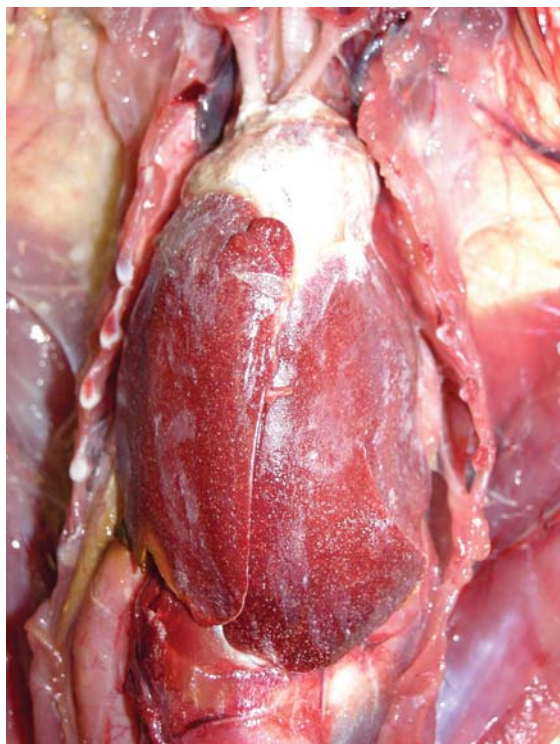
Figure 14.19 Visceral gout with c halk-like urate deposits on pericardial sac and liver capsule.

which the renal damage was induced in the flock. The strain of bird can also affect the severity and incidence of gout. Birds with gout usually show no clinical signs before death or are emaciated. The lesions of gout are associated with the accumulation of urates (uric acid is the primary nitrogenous excretory product of birds) on the surfaces of the internal organs (visceral gout) (Figure 14.19) as well as within joint spaces and along synovial membranes (articular gout). The urates are gritty and white as opposed to the inflammatory exudates that result from bacterial infections such as colibacillosis, which are yellow and friable [4]. Portions of kidney are atrophic or absent and contralateral portions are often swollen (compensatory hypertrophy) [77]. Birds can be treated, with varying success, by adding ammonium sulfate or ammonium chloride to the ration but these treatments do not cure gout and may cause wet droppings and deterioration of shell quality [78]. Gout can be prevented or minimized by providing proper calcium and phosphorus nutrition throughout the growing process (1% calcium and 0.50−0.45% available phosphorus), starting layer levels of calcium feeding at the proper time (one week prior

to first egg), and avoiding water deprivation at the housing [79].

Histomoniasis

Histomoniasis is a protozoal disease that affects the cecum and liver of gallinaceous birds and has also been referred to as blackhead, infectious enterohepatitis, and *Histomonas meleagridis* infection. The disease has been diagnosed in peafowl and turkeys, and occasionally in chickens [80]. The causative agent, *Histomonas meleagridis*, is a flagellated ameboid protozoan that replicates in the ameboid state in the cecum and liver. The infection is often carried in eggs of the cecal worm *Heterakis gallinae*, which are shed in feces and consumed by earthworms. The cecal worm or earthworms were often considered a required intermediate host, but recent research indicates that turkeys can transmit the protozoan directly from bird to bird, in the absence of cecal worms and earthworms, through the phenomenon of cloacal drinking, by which contaminated fecal material in litter is carried into the colon by rhythmic contractions of the cloaca or vent [81].

Clinical signs/diagnosis

Lesions are usually present in ceca at 8 days post-infection and liver lesions are present by 10 days. Birds that are affected can appear to die suddenly in good condition or exhibit progressive wasting. Fecal material can be yellow in color as well as containing flecks of blood. The birds are depressed, with closed eyes, huddling, and ruffled feathers. The ceca are often enlarged, pale, thick-walled, firm and contain abundant gray to tan, friable material (cecal cores) [82]. Peritonitis can occur if inflammation penetrates the cecal wall. The liver is enlarged and contains multifocal to coalescing circular, and occasionally concentric, dark red rings with a yellow center that resemble an archery target (hence the term "target lesion") (Figure 14.20). Some birds in early stages of infection have cecal lesions without liver lesions [82]. The classical gross lesions are often diagnostic. Additionally, histomonads in liver and cecum can be observed in tissue impressions and histologic sections (Figure 14.21).

Treatment

There is no effective treatment that is commercially available. The emphasis is on prevention. Few preventive medications are commercially available. Good sanitation is the key to prevention. Keep chickens and turkeys separated [19]. Regularly deworm turkey flocks to decrease the population of cecal worms. Birds should be raised on litter or sandy, dry soil to minimize

Figure 14.21 Wright-Giemsa-stained section of chicken liver reveals multiple round amoeba (arrows) consistent with *Histomonas meleagridis*.

Figure 14.20 Immature laying hen with *Histomonas meleagridis* infection with round, target-like, necrotic foci in liver and enlarged ceca filled with fibrinous to caseous material.

exposure to earthworms [23] (see Chapter 6 for more information).

Fatty liver syndrome

Fatty liver is caused by an imbalance of energy (positive energy gain) and protein intake. Fatty liver is observed most often in caged laying hens and occasionally in breeder turkey hens. Caged layers are particularly prone to fatty liver because of minimal exercise accompanied by high caloric intake [83,84]. Obese backyard laying hens are also susceptible to developing fatty livers. The liver is enlarged, pale orange, soft, friable, and easily fractured (Figure 14.22). Rupture of the fatty liver with hemorrhage into the abdominal cavity or around the liver capsule is a common cause of death in laying hens [8]. Treatment with choline chloride, vitamin K, biotin, and vitamin E in the feed for 2 weeks has been used to control mortality with varying results and is certainly not necessary in small flocks, in which the problem is excessive calorie intake. Prevention is effected by means of an adequate diet with proper energy and protein levels [83]. Similar changes in small flocks are

Figure 14.22 Obese hen with enlarged fatty liver that has ruptured, resulting in fatal pericapsular hemorrhage.

best prevented by monitoring body weights and feed intake, and by limiting access to high fat treats (e.g., egg noodles and cheese).

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Cardiovascular Diseases

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Diagnosing cardiovascular diseases in backyard poultry

General considerations

There are a number of differences between the avian and the mammalian heart, some of which have clinical implications in the pathophysiology and diagnosis of poultry cardiovascular diseases (Table 15.1) [1].

The avian cardiovascular system is highly efficient but commercial poultry have been intensely selected for increased growth and production, and the high energy and oxygen demand to even meet the basal metabolic requirements may be close to its physiological maximum [2–4]. Consequently, heavy meat-type galliformes (turkeys and broilers) do not thermoregulate well and readily experience periods of dyspnea and tachycardia with mild stress and exercise and are far more susceptible to cardiovascular diseases than other species and breeds. In fact, a high number of these kinds of birds have subclinical cardiac disease [3]. Furthermore, already compromised animals may collapse easily during examination and restraint. They may also be poor anesthetic candidates and preoxygenation and preliminary therapy (abdominocentesis and/or diuretics) are recommended before diagnostic procedures can be considered. In broilers and turkeys, cardiovascular diseases may be the most common cause of mortality [5]. In backyard poultry flocks raised for meat, cardiovascular diseases such as ascites syndrome may still be encountered in small flocks of broilers and other chicken breeds because most are genetically related to commercial broilers of the White Plymouth Rock breed.

In addition to general signs of disease, specific clinical signs of cardiovascular diseases include dyspnea, exercise intolerance, cyanosis or hypoperfusion (bluish or pale comb, wattles, ricti, or periorbital skin, and increased comb capillary or ulnar vein refilling time), ascites, syncope, collapse, and sudden death. Cardiac auscultation may reveal muffled heart sounds, murmurs, or rhythm abnormalities but is usually not as rewarding in birds as it is in mammals because of their rapid heart rate. Normal heart rates are listed in Table 15.2. Arterial catheterization for direct blood pressure measurement is challenging in poultry because of their relatively atrophied wings. However, indirect blood pressure measurement using a Doppler unit, while inaccurate in small to medium sized birds, seems more reliable in larger birds but may be closer to the mean rather than to the systolic arterial blood pressure [6]. Observing trends in indirect blood pressure may be useful.

Laboratory ancillary diagnostics

Apart from assessing the general health of the backyard poultry patient, clinical pathology tests may reveal specific changes associated with cardiovascular diseases. Erythrocytosis, described as a PCV greater than 35% (note: the PCV is lower in poultry than in other birds), may be caused by chronic hypoxia resulting from persistent ventilatory-perfusion mismatching (e.g., congenital cardiac malformation, ascites syndrome, pulmonary pathology) and increased oxygen demands (e.g., ascites syndrome). Leukocytosis may be seen in bacterial myocarditis and valvular endocarditis. Cardiovascular microfilariae may be observed on the blood smear. Poultry are large enough for arterial blood samples and blood gas analyses may help pinpoint

Table 15.1 Some avian cardiovascular anatomical and physiological peculiarities which differ from mammals. AV: atrioventricular

Avian cardiovascular system peculiarities
Muscular unicuspid right AV valve
No chordae tendinae in the right AV valve
Tricuspid (poorly defined) left AV valve
Muscular ring around aortic valve
Negative cardiac mean electrical axis (except broilers, Pekin ducks)
Ring of Purkinje fibers around aorta and right AV valve
Depolarization of epicardium precedes endocardium
Higher heart rate, arterial blood pressure, cardiac output
Larger heart
Smaller cardiac muscle fibers
Absence of T-tubules in cardiac myocytes
Absence of M-bands connecting myosin filaments
Ascending aorta on the right
Two cranial vena cava
Brachiocephalic arteries larger than aorta
Cartilage/ossification at base of aorta
No cerebral arterial circle of Willis
Renal portal system

an oxygenation problem. Myocardial damage can lead to a rise in CK (and cardiac CK isoenzyme) and cardiac troponin T (only 68% sequence homology with humans which may affect diagnostic tests accuracy). Electrolyte disorders (Ca, Mg, K, Na), hypoproteinemia, and hyperuricemia can also cause arrhythmia and cardiac diseases. Bile acids are frequently elevated with

hepatic congestion secondary to congestive heart failure. Lipoprotein abnormalities may also be diagnosed in conjunction with some degenerative lesions but have to be interpreted in the context of the egg-laying cycle and the genetic selection lines [7]. Finally, ascitic and effusion fluid should always be analyzed and can provide useful information. Cardiac-induced ascitic (coelomic) fluid is a pure or modified transudate, thus having low protein and cellular content and a low specific gravity. Blood culture may be valuable to isolate causative agents of cardiac bacterial infections and can be performed with only 0.5–2ml of blood.

Electrocardiography

An ECG is invaluable to investigate conduction disorders and arrhythmia. The avian ECG is typically obtained by placing two cranial electrodes on each propatagia and one (left) or two caudal electrodes on the knee web, just cranial to the legs, using needles or clips. Each lead evaluates the cardiac electrical activity on a different plane and a standard examination classically includes three bipolar leads (I, II, and III) and three augmented unipolar leads (aVR, aVL, aVF). Recordings need to be performed at a minimum speed of 100 mm/s to better assess the morphology of the QRS complexes. In contrast to mammals, the cardiac mean electrical axis is usually negative in birds, which gives negative QRS complexes on lead II (Figure 15.1). In broilers and Pekin ducks, however, the mean electrical axis is most commonly positive [8,9]. The mean electrical axis is affected by changes in heart position and relative
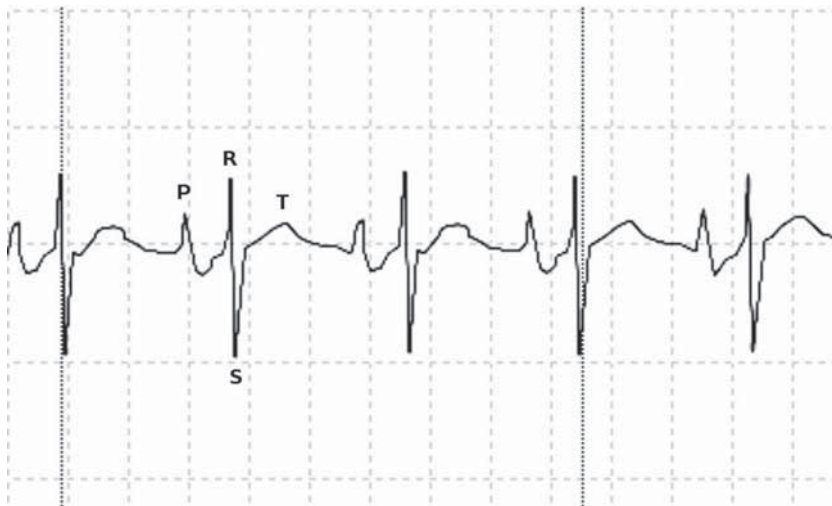
Figure 15.1 ECG from a normal chicken in Lead II. Note the typical negative QRS complex.

dilation of cardiac chambers and is one of the commonly modified parameters identified on the ECG with common poultry cardiac diseases. A T_a wave (auricular T wave related to atrial repolarization) is normal in some species [10]. The Q wave is absent in chickens, small in turkeys, and prominent in Pekin ducks [1,9,11,12]. In lead II, the QRS complex often shows a prominent S wave and a small R wave. This differs from mammals, in which the R wave is usually more prominent [13]. Normal variations of the QRS complex, however, have been documented in chickens, especially in broilers [8]. In addition, the P and T waves are often fused [13]. Measurements are normally performed on lead II and reference values have been determined for some poultry species (Table 15.2) [8,9,11,12,14–18]. Interpretation of the ECG should be methodical and include an evaluation of the heart rate, cardiac rhythm, mean electrical axis, and measurements. The reader is referred to more exhaustive references for further information on avian ECG [10]. Anesthesia and stress may induce alterations of the normal ECG such as AV blocks

and sinus tachycardia or bradycardia. Broilers have a normally high prevalence of arrhythmias especially under anesthesia [19]. Common ECG abnormalities in chickens include mean QRS axis deviation, ventricular premature contractions, and AV blocks.

Diagnostic imaging

Whole-body or thoracic radiographs (large galliforme and anseriforme species) are useful for a preliminary assessment of cardiovascular diseases. However, no reference intervals for cardiac radiographic measurements are available in commonly seen poultry species. Radiographic signs that may be observed in cardiovascular diseases include an enlarged cardiac silhouette (cardiomegaly or pericardial effusion), an enlarged hepatic silhouette (hepatic congestion), pulmonary edema, and a decrease in coelomic details and airsac space (ascites). Angiocardiography using radiography, fluoroscopy, or CT-scan may be of value to diagnose cardiomegaly or vascular abnormalities (e.g., aneurysms) and can be performed with an injection of 3 ml/kg of intravenous

Table 15.2 Published reference intervals for selected electrocardiographic parameters in different backyard poultry species on lead II

Species	Chicken [11] (white leghorn)	Chicken [8] (broilers)	Turkeys [12]	Chukar partridge [15]	Pekin duck [9]	Guineafowl [16]
N	72	300	50	10	50	8
Age	6 months	4–5 weeks	20 weeks	mature	12–18 months	6–12 months
Anesthesia	none	isoflurane	none	none	none	none
Heart rate (bpm)	180–340	270–450	146–266	200–435	200–360	300–376
Mean electrical axis (°)	negative −(91–120)	mainly positive 0–180	negative −(75–120)	negative −(60–139)	mainly positive −160−+95 (mean: +147)	negative −(12–108)
P duration	0.035–0.046		0.021–0.061	0.006–0.034	0.015–0.035	
R amplitude			−0.305–0.279			
PR interval	0.073–0.089		0.054–0.122		0.04–0.08	0.024–0.056
QRS duration	0.02–0.028	22.8–48.4	0.038–0.066	0.019–0.021	0.028–0.044	0.03–0.05
(Q)rS amplitude	females: 0.10–0.33 males: 0.795 (mean)	−0.96−+0.78	0.276–3.736	0.26–0.94	0.35–1.03	−0.17–0.39
ST interval	0.119–0.149			0.106–0.138		0.018–0.042
T amplitude	females: 0.03–0.19 males: 0.255 (mean)		0.1–0.65	0–0.38	0.04–0.40	0.15–0.41
QT interval			0.142–0.198	0.106–0.138	0.08–0.12	0.106–0.134
T duration	0.119–0.145				0.03–0.07	

Note: To obtain a 95% interval, all published results in the form of mean±SD were reported as mean±2SD and in the form of mean±sem were reported as mean±2sem√n, when only the range was published, it was reported as is. Values are in seconds for wave and intervals duration, and mV for amplitudes. n=number of birds examined.

contrast (e.g., iohexol) over 3 seconds [21]. Diagnostic imaging procedures should be started immediately or a few seconds after the injection to obtain high concentration of contrast in blood during radiographic exposure. Coelioscopy through the left or right thoracic approach, or interclavicular approach may be performed to identify pericardial diseases. However, endoscopic procedures are more challenging in chickens than in other birds as a result of the reduced airspace present, the heavy muscles, the degree of subcutaneous and abdominal fat, the size the liver, and the potential presence of a large oocyte on the left.

Echocardiography is the diagnostic imaging method of choice for assessing the cardiovascular function in birds and the procedure in chickens presents certain peculiarities. Cardiac chamber dimensions, myocardial contractility, and hemodynamic function can be evaluated in a non-invasive manner with a cardiac ultrasound examination, which does not require anesthesia in most cases. As in other birds, a 7.5 MHz or higher frequency probe with a microcurved or small straight transducer is appropriate in most cases. Sonographic windows are limited in birds because of the extensive airsac system surrounding cardiovascular structures, the fact that the heart lies in a ventral indentation of the keel bone, the high heart rate of birds, and the relatively small size of the imaged structures. While only the ventromedial transcoelomic sonographic approach can be performed in most raptors and parrots, an additional transcoelomic approach, the parasternal approach, can be used in galliformes either caudally to the ribs, because the ribs have limited caudal extension, or between the sternal ribs through the large window of the sternum present in poultry, but the optimal approach is not always consistent and depends on individuals, age, and breeds [20–22]. These two approaches allow a more complete echocardiographic examination in chickens than in most other avian species with the possibilities of performing unidimensional M-mode for assessing ventricular contractility (Figure 15.2), two-dimensional B-mode for evaluating chamber dimensions in longitudinal and transverse views, spectral Doppler for flow velocities, and color flow Doppler for the detection of valvular insufficiency. The most commonly used approach in chickens and turkeys is the parasternal approach. The probe is positioned either in front of the stifle joint on either side of the thorax with the bird standing (intercostal approach) or behind the pelvic limb and the last rib, again with the bird standing, and the probe angled cranially (Figure 15.3) [22–24]. For the ventromedial approach, the probe is placed on the midline behind the caudal border of the keel
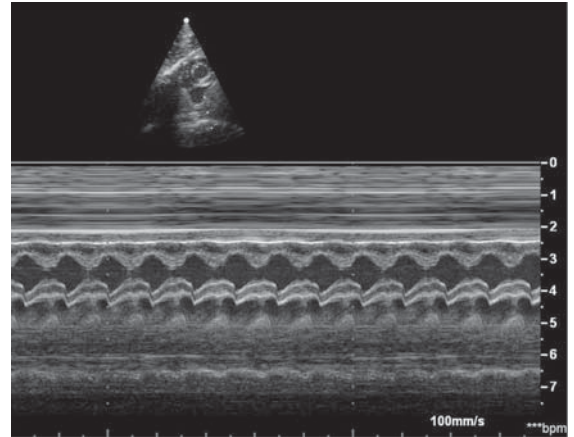
Figure 15.2 Echocardiogram in M-mode through a parasternal approach showing chicken left ventricle.

Figure 15.3 Performing an echocardiogram in a chicken showing the parasternal approach.

and angled cranially dorsal to the keel. The heart is imaged through the liver, which is used as an acoustic window [21]. Echocardiographic reference values have been produced through the parasternal approach and fast-growing chickens have smaller cardiac measurements than slow-growing chickens relative to their body weight (Table 15.3) [22,25,26]. It is noteworthy that most reference values have been obtained from birds younger than 2–3 months and limited information is available for older birds. Nevertheless, the evaluation of the relative sizes of the cardiac chamber and their functional assessment is probably more clinically useful than taking echocardiographic morphometric measurements, which appear to show low reliability in birds [27]. No standardized echocardiographic examination with a clear description of the different views

Table 15.3 Published reference intervals (in centimeters) for selected echocardiographic parameters in different backyard poultry species using a parasternal approach. LVDS: left ventricular diameter in systole; LVDD: left ventricular diameter in diastole; FS-LV: fractional shortening of left ventricle; RVDS: right ventricular diameter in systole; RVDD: right ventricular diameter in diastole; IVSD: interventricular septum width in diastole

Species	Broiler chicken [24,26]	Leghorn chicken [22,24]	Turkey [23]
N	30	5	34
Age	6 weeks	7 weeks	4 weeks
Anesthesia	none	none	none
LVDS	0.17–1.27	0.22–0.30	0.01–0.54
LVDD	0.81–1.25	0.63–0.71	0.44–1.05
FS-LV (%)		32.6–54	37–93
RVDS	0.06–0.50		
RVDD	0.00–0.97		
IVSD	0.25–0.69	0.17–0.35	0.14–0.38

Note: To obtain a 95% interval, all published results in the form of mean±SD were reported as mean±2SD and in the form of mean±sem were reported as mean±2sem$\sqrt{n}$, when only the range was published, it was reported as is. n=number of birds examined.

have been described in chickens but the classically obtained views include transverse views (short axis two-chamber views) at the level of the ventricles and a longitudinal view (long axis four-chamber view) through the parasternal approach, and a longitudinal horizontal (four-chamber) (Figure 15.4) and a vertical (two-chamber) (Figure 15.5) view through the ventro-median approach. The echocardiographic examination is more easily performed, and the views obtained are of better resolution, in birds with cardiac disease because the presence of coelomic fluid and hepatic congestion provides better acoustic windows. In anseriformes, the transcoelomic ventromedian approach is typically used [28]. Transesophageal echocardiography uses a small transducer at the tip of a long flexible tube and consists of imaging the heart from inside the proventriculus and esophagus [29]. The examination must be performed under anesthesia and has overall a superior resolution than transcoelomic techniques but the equipment is expensive and not widely available. Chickens, turkeys, and ducks are easily imaged using this technique as they are large birds.

Therapeutics

The use of drugs in poultry is controversial as only a few first-generation medications are legally approved and withdrawal times are not determined for many therapeutic agents. Furthermore, legislation may vary according to country. This is more of a problem for antimicrobials because of development and transfer of resistance among bacteria. Specific cardiac medications are probably less problematic and medical treatment

Figure 15.4 Echocardiogram of chicken showing the long axis view. LV, left ventricle; RV, right ventricle; LA, left atrium; RA, right atrium; arrow: right atrioventricular valve.

may not be practical. Valuable or companion poultry patients can be treated. Draining of ascitic or pericardial fluid is recommended to alleviate signs of dyspnea or cardiac tamponade. Drug dosages for selected cardio-vascular therapeutics in birds are shown in Table 15.4. Veterinarians should focus more on prevention, man-agement, and breed selection to limit cardiovascular diseases that are only prevalent in fast-growing and heavy birds. Educate owners regarding the importance of not breeding birds with genetic forms of cardiac disease.

Table 15.4 Cardiovascular therapeutics in poultry

Drug	Dose	Comments
Furosemide	0.5–1 mg/kg 0.015% in food	Loop diuretic
Spironolactone	Extrapolated dog dose	Diuretic, aldosterone receptor antagonist
Enalapril	1–5 mg/kg	Angiotensin conversion enzyme inhibitor
Digoxin	0.004–0.02 mg/kg	Digitalic, inotrope
Pimobendan	0.25–0.5 mg/kg	Phosphodiesterase inhibitor, inotrope
Atenolol	25 ppm in food	β-blocker
Propranolol	0.1–0.2 mg/kg	β-blocker
Lidocaine	1–6 mg/kg	Antiarrhytmic, short half-life
Atorvastatin	5–10 mg/kg	Statin, lipid lowering agent

Note: Dosages are based on pharmacodynamic studies (but not pharmacokinetic) in chickens and turkeys. Some dosages are empirical or based on other avian species. Therapeutic plasma levels monitoring is recommended for valuable or companion poultry birds.

Figure 15.5 Echocardiogram of chicken showing the short axis view. LV, left ventricle; RV and arrow, right ventricle.

Common cardiovascular diseases of backyard poultry

Dilated cardiomyopathy in turkeys
Clinical history
Also known as round heart disease or spontaneous cardiomyopathy of turkeys, it is most commonly encountered in 1–4-week-old turkeys and usually peaks at 2 weeks. The prevalence is typically 0.5–3% in commercial turkey flocks but can be higher [30]. Young fast-growing males are more susceptible.

Causative agent
The exact cause of the disease is unknown in turkeys. It has been demonstrated that some of these turkeys show an abnormal troponin T structure and dysregulation of some cardiac enzymatic pathways. Some toxic compounds such as furazolidone and antitrypsin may also have been implicated in some cases [30–32]. Genetic factors, previous myocarditis, hypoxia during incubation, and other environmental and dietary factors have also been proposed to play a role in the etiology [30,32,33].

Clinical signs and lesions
In many cases, affected birds die suddenly. They may also show abdominal distension resulting from ascites, respiratory signs, other signs of congestive heart failure, and non-specific signs such as listlessness, lethargy, ruffled feathers, and impaired growth. Gross lesions include cardiomegaly, which is caused by dilation of both ventricles, congested and edematous lungs, congested liver, hypertrophic left ventricle (in older animals), ascites, and hydropericardium [30]. Right ventricular dilation may be the only observable gross lesions in early cases. Histopathologic lesions include degeneration of myofibers with vacuolation, secondary endocardiosis, focal infiltration of lymphocytes, and secondary changes in the liver [30,34].

Transmission route
Not applicable.

Diagnostic tests
The combination of clinical signs, age, and fast-growing types of turkeys should raise a strong suspicion of dilated cardiomyopathy. Radiographs show an enlargement of the cardio or cardiohepatic silhouette and organomegaly or ascites as a result of passive congestion. On the ECG, the following changes, associated with dilation and hypertrophy of the ventricles, can be identified: Increased R wave amplitude, negative T wave, and rotation of the mean electrical axis [13,35]. Reports of the use of cardiac ultrasound have been limited in turkeys. Dilation of the cardiac chambers (more than double in chamber measurements), reduced ventricular fractional shortening (to 14%), pericardial effusion, and ascites can be expected on echocardiography [23].

Differential diagnosis
Other causes of congestive heart failure, congenital cardiac abnormalities, restrictive cardiomyopathy, valvular insufficiency.

Prevention and control
Slowing the growth rate of susceptible lines by dietary manipulation and avoiding hypoxic condition in hatchers may reduce the incidence of the disease [5,30].

Zoonotic potential
Not applicable.

Ascites (Coelomic fluid)/pulmonary hypertension syndrome in broilers
Clinical history
This syndrome is one of the most common causes of mortality in commercial flocks of broilers, with an average prevalence of approximately 4.7%, which can go as high as 15–20% in certain roaster chicken flocks [30,36,37]. The condition can be exacerbated by rearing at high altitude and additional genetic and environmental factors (intensive rearing, cold or hot temperature, activity, hypoxia during incubation, ventilation, electrolytes supplementation) may promote its occurrence [3]. Affected chickens are usually younger and males seem to be more susceptible. The disease is expected to be uncommon in backyard flocks but may occur if meat-type poultry are raised (broilers and related breeds).

Causative agent
This metabolic disease is associated with growth and production and has increased in frequency with selection for a higher and faster muscle mass production and different body conformation. The ascites is caused by right-heart congestive heart failure and valvular insufficiency. The physiopathogenesis is associated with an increased workload of the heart and oxygen demand coupled with an overall insufficient pulmonary capillary capacity and decreased respiratory efficiency in chickens compared to other birds. This quickly leads to pulmonary hypertension, which in turns leads to right ventricular hypertrophy and ultimately to dilation. With the dilatory changes affecting the right ventricle, the right atrioventricular valve, which extends from its wall, develops insufficiency, which in turns increases the preload and leads to systemic congestion and ascites by increased hydrostatic pressure. Some researchers also argue that left-heart dysfunction plays a major role in the pathogenesis and that chronic hypoxemia and pulmonary hypertension are secondary to chronic left heart failure. In addition, the elevated PCV triggered by the chronic hypoxia may increase blood viscosity and increase the resistance to the flow [3,4,33,37,38]. Hypoxia and increased metabolic rate are the two most important factors that influence the development of this condition [39].

Clinical signs and lesions
Birds present impaired growth and lethargy. Specific signs may include coelomic distension caused by ascites, dyspnea, cyanosis, pale comb, and acute death
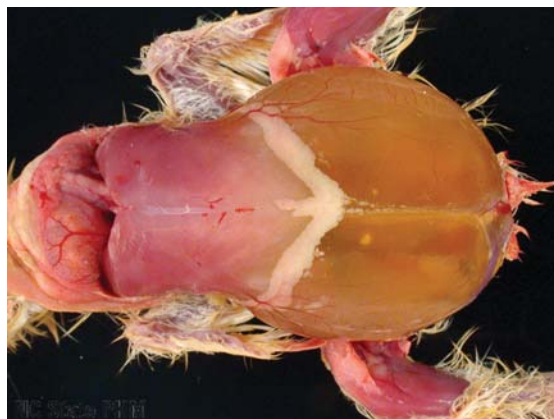
Figure 15.6 Gross necropsy of ascites syndrome in a broiler. (Source: Photo courtesy of Dr. Oscar J. Fletcher, North Carolina State University.)

(Figure 15.6). Some birds can die from pulmonary edema caused by pulmonary hypertension. Gross lesions may include dilated and hypertrophic right ventricle, ascites, pericardial effusion, pericarditis, organ congestion, dilation of pulmonary veins, and a fibrotic liver when chronic [30,33,36,37]. Ascitic (coelomic) fluid is typically clear but can also be cloudy with fibrin clots. In advanced cases, histologic cases other than associated with systemic congestion include myofiber degeneration with swelling, focal necrosis, edema, and fibrosis [34].

Transmission route
Not applicable.

Diagnostic tests
A loss of abdominal detail and an enlarged cardiac and/or cardiohepatic silhouette are present on radiographs. Coelomic fluid analysis is consistent with a pure or modified transudate but can still show clots of fibrin and proteins because of associated chronic coelomitis. On electrocardiography, the heart rate is usually decreased and there may be an increase in S-wave amplitude and ventricular fibrillation associated with right-heart dilation and a right deviation of the mean electrical axis (which becomes negative) [8,13,38,40]. These chickens consistently show an increased PCV and serum troponin T level is elevated (normal is <0.50 ng/ml, n=20, age 1–2 months) [38,41,42]. Echocardiographic findings include right and left atrioventricular valves regurgitation, reduction in fractional shortening of the left ventricle, dilation of cardiac chambers, and pericardial effusion [24–26].

Differential diagnosis
Other causes of ascites in chickens: Vascular damage (toxins, nutritional deficiencies), blockage of lymph drainage (ovarian adenocarcinoma), increased vascular pressure (portal hypertension resulting from advanced hepatic disease), hypoproteinemia, congenital abnormalities, sodium toxicosis, other causes of right-sided and bilateral cardiac failure (valvular endocarditis, congenital cardiac abnormality, atherosclerosis, toxic, dilated cardiomyopathy, restrictive cardiopathy). Other causes of coelomitis can induce production of coelomic fluids as well.

Prevention and control
Genetic factors increase bird susceptibility and selection against this syndrome may be useful [3]. The prevalence of the disease can be lowered by decreasing growth rate, restricting food intake, modifying food type (pelletized diet seems to increase feed efficiency and then metabolic demand), and minimizing sources of decreasing oxygen availability (low or high temperature, altitude [above 1500m is inappropriate for meat-type chickens], concomitant respiratory diseases such as fungal pneumonia) [30,36,37]. As a result of the low individual value of broilers, treatment has rarely been reported but, experimentally, furosemide has been shown to reduce mortality and L-arginine supplementation appears to reduce the incidence of the disease [43,44]. Treatment with β2-agonists may reduce the ventilatory-perfusion mismatch by inducing bronchodilation, hence reducing mortality [38]. The use of L-carnitine, antioxidants, and omega-3 fatty acids have been mentioned to reduce the incidence of ascites but scientific evidence is either lacking or inconclusive [3,45]. However, it must be kept in mind that pharmacologic interventions may have limited results as the disease is associated with high metabolic demand in birds with poor cardiovascular and respiratory efficiency.

Zoonotic potential
Not applicable.

EDITOR'S VIGNETTE (CBG)

An approximately 6 month old White Leghorn broiler chicken presented with a sudden onset of dyspnea, lethargy, and exercise intolerance. A physical examination identified a mildly thin bird, with severe coelomic distension due to coelomic fluid and a Grade III/VI heart murmur. Approximately 200 ml of coelomic fluid (about one fourth to one third of the fluid present) was removed by coelomocentesis. Dilated cardiomyopathy was diagnosed based on echocardiogram. Treatment with digoxin (this was before the advent of pimobendan), enalapril, and furosemide improved respirations and myocardial contractility, and decreased coelomic distension dramatically. Later, the bird required the addition of the potassium-sparing drug spironolactone. With periodic rechecks, including recheck echocardiograms, and minor adjustments in medications, this bird went on to live to eight years of age despite our initial grave prognosis. This outcome is not typical.

Aortic rupture/dissecting aneurysm in turkeys
Clinical history

The disease is mostly encountered in growing turkeys of 7–24 weeks of age and primarily affects males [30]. Mortality is approximately 1–2% in commercial turkey flocks but has been as high as 50% in the past.

Causative agent

As with most poultry cardiovascular diseases, the exact cause of aortic dissecting aneurysm is uncertain. Systemic hypertension (common in meat-type turkeys, especially young males), atherosclerosis, absence of *vasa vasorum* in the abdominal aorta, genetic factors, connective tissues disorders, peas in the ration (peas' toxin β-aminopropionitrile causes aortic rupture experimentally by interference with collagen formation), and dietary deficiencies, notably in copper (also demonstrated in ratites) may contribute to the pathogenesis [30,32,37]. Hypertension is thought to be the most significant risk factor.

Clinical signs and lesions

Turkeys usually die acutely from severe internal hemorrhage. On necropsy, the head, skin, and muscles appear anemic, blood may be noticed in the mouth, and massive hemorrhage, coagulated blood is found in the intestinoperitoneal cavity, and the aorta is torn longitudinally between the external iliac and ischiatic arteries [30,33]. Less commonly, the rupture may occur in the ascending and "thoracic aorta" with blood present around the heart (Figure 15.7). Histologically, there may be a separation of the tunica intima and media from the adventitia with the presence of folds, degenerative changes in the media, and inflammatory cells infiltration. Intimal thickening, a fibrous intimal plaque, and disintegration of elastic laminae may be observed at the rupture site [30].

Transmission route

Not applicable.

Diagnostic tests

Aneurysm, if large enough, should be expected to be identified on angiography but antemortem diagnosis has not been reported because of the low individual value of meat-type turkeys. Turkeys are large birds; therefore indirect blood measurement using a Doppler unit is expected to be more reliable than in smaller birds and may help pinpoint a hypertensive problem.
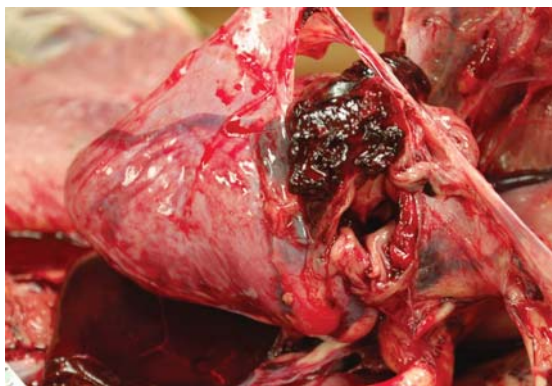
Figure 15.7 Gross necropsy of turkey with atrial rupture. (Source: Photo courtesy of Ms. Megan MacAlpine, University of Guelph.)

Differential diagnosis

Sudden death syndrome of turkeys, acute presentation of spontaneous dilated cardiomyopathy.

Prevention and control

Minimizing stress and excitement and slowing growth in susceptible birds may reduce the incidence of the disease. Treatments are uncommonly reported but reserpine, an antihypertensive drug, and propranolol may have favorable preventative effects in susceptible birds [46,47].

Zoonotic potential

Not applicable.

Sudden death syndrome of broilers
Clinical history

Broiler chickens of 1–8 weeks of age, mainly males (about 70% of cases), are affected. The prevalence depends on genetic, environmental, and dietary conditions and approximates to 0.5–4% [30,36,48].

Causative agent

The cause is unknown but is thought to be associated with ventricular fibrillation as a result of electrolytic or other metabolic imbalances.

Clinical signs and lesions

As the name implies, birds die acutely with no premonitory signs and exhibit a short violent wing-flapping seizure at time of death. The disease is also termed flip-over disease because dead birds are frequently found on their back. Birds are in good body condition at necropsy and no specific lesions are seen but edematous lungs, enlarged liver, renal hemorrhage, and contracted

heart ventricles are common findings. Histopathologic lesions are not specific [30,48].

Transmission route
Not applicable.

Diagnostic tests
As a result of the acuteness of the disease and the unknown etiology, the identification of birds at risk may not be possible. Birds dying of sudden death syndrome show ventricular fibrillation on electrocardiogram [13].

Differential diagnosis
Sudden death from pulmonary hypertension syndrome, rupture of right atrium, heart defects, and other non-cardiac causes (e.g., fatty liver hemorrhagic syndrome).

Prevention and control
Feed restriction and other nutritional modifications (mash, lower caloric density) may lower the incidence. Stress, bright light, and overcrowding should be limited [33,48].

Zoonotic potential
Not applicable.

Sudden death syndrome of turkeys
Clinical history
Heavy meat-type turkeys are mainly affected by this syndrome and almost exclusively males are susceptible. The disease is common in turkeys of 8–15 weeks of age. Mortality is usually around 0.8–1.8% but can reach 6–10% [30,33].

Causative agent
As in most poultry cardiovascular diseases, the etiology lies in the intense selection for meat production and the cardiovascular system of turkeys may not be able to meet the extra demand brought up by exercise, stress, and other environmental factors, leading to hypotension, lactic acidosis, circulatory shock, and death. Systemic hypertension and hypertrophic cardiomyopathy also appear to play a role in this syndrome [32,33].

Clinical signs and lesions
As in broilers, there is no premonitory signs and birds usually die following a brief wing-flapping period. Gross lesions are consistent with generalized passive congestion and perirenal hemorrhage (other name of the disease) and birds are generally in good condition. The left ventricle and interventricular septum are often hypertrophied. Contrary to a ruptured aneurysm, free blood is usually not found in the intestinoperitoneal cavity but the perirenal hemorrhage may be related to small aortic tears nonetheless. Microscopic lesions are not specific [30].

Transmission route
Not applicable.

Diagnostic tests
Because birds die acutely, antemortem diagnosis is rarely made. Cardiac ultrasound may be able to detect cardiac changes associated with hypertrophic cardiomyopathy. Indirect arterial blood pressure measurement may reveal increased blood pressure in susceptible males.

Differential diagnosis
Ruptured aortic aneurysm, ruptured atrium, acute presentation of dilated cardiomyopathy, non-cardiac causes of sudden death (e.g., obstructive pulmonary disease caused by aspergillosis).

Prevention and control
Avoid stress and excitement of susceptible male birds.

Zoonotic potential
Not applicable.

Less common cardiovascular diseases

Round heart disease in chickens
Clinical history
The condition is seen in mature chickens but the prevalence is extremely rare nowadays and the disease has not been seen for several decades [33].

Causative agent
A nutritional deficiency is thought to contribute to the disease but the etiology is unknown.

Clinical signs and lesions
Birds typically die acutely and clinical signs are rarely seen [49]. Gross lesions are characterized by an enlarged heart with hypertrophy of the left ventricle and yellowish color, ascites, and excess gelatinous fluid in the pericardial cavity [30,34,49] (Figure 15.8). Histologic lesions include primarily myofiber degeneration with swelling and vacuolar changes (lipidosis) [34].

Figure 15.8 Gross necropsy of chicken with dilated cardiomyopathy with multifocal myocarditis. This 7-year-old chicken also had concomitant marked bilateral cystic thyroid hyperplasia and hepatic sarcoma. (Source: Photo courtesy of Dr. Linden Craig, University of Tennessee.)

Transmission route
Not applicable.

Diagnostic tests
Published information is rare on the antemortem diagnosis of this disease but is presumed to be difficult because birds usually die abruptly without premonitory signs.

Differential diagnosis
Sudden death syndrome of broiler, ascites syndrome of broilers.

Prevention and control
Appropriate nutrition.

Zoonotic potential
Not applicable.

Infectious cardiopathies
Clinical history
All backyard poultry species and breeds are thought to be susceptible to infectious cardiopathies and the prevalence is generally accepted to be low. Some pathogens are more species specific than others (e.g., viruses in chickens).

Causative agent
A wide variety of pathogens have been reported to cause cardiac diseases (Table 15.5). Among bacteria, *Enterococcus, Staphylococcus, Pasteurella*, and *Erysipelothrix* are more commonly isolated from valvular endocarditis. *Escherichia coli* is frequently involved in pericarditis and myocarditis. Avian chlamydiosis is frequently associated with pericarditis and myocarditis in ducks and turkeys. Epicarditis is usually caused by *Salmonella* spp. or *E. coli* [10, 34 ,50]. Chronic bacterial infection in other organ systems (e.g., salpingitis and bumblefoot) can lead to bacteriemia and subsequent cardiac infection. Infectious agents colonizing cardiac tissues cause a destruction of valves and other cardiac structures, leading to valvular insufficiency and impaired cardiac function. Septic emboli are possible. Marek's disease virus (gallid herpesvirus 2) and avian retroviruses can induce lymphoid tumors in the heart or vascular tumors [34]. Marek's disease virus may also promote atherosclerosis in chickens. In addition, avian leucosis virus, avian influenza virus, and avian paramyxovirus 1 (Newcastle disease virus) can cause myocarditis in chickens and other avian species [51,52]. Fungal infections can reach the heart via emboli or by extension of adjacent airsacculitis lesions. Protozoan parasites mainly induce myocarditis as a result of the presence of cysts in myofibers. Nematodes (filarioid nematodes) and trematodes (schistosomes) localized in the vascular system are mainly found in wild galliformes and anseriformes but can be a concern to backyard poultry, and some species have been recovered in chickens and domestic ducks and geese [53,54].

Clinical signs and lesions
Clinical signs may not be specific but congestive heart failure or arrhythmia can be documented as a result of ensuing valvular insufficiency and myocardial involvement respectively. Birds are also frequently lethargic and may show dyspnea. Restrictive pericarditis can also lead to heart failure and ascites. Gross lesions may include pericardial exudate (Figure 15.9), granulomatous materials on the valves and on the endocardium (Figure 15.10), and lesions elsewhere in the coelom, such as perihepatitis and coelomitis.

Table 15.5 Infectious agents reported to cause cardiovascular lesions in poultry

Pericarditis/epicarditis	Myocarditis
Listeria monocytogenes	*Escherichia coli*
Riemerella anatipestifer (turkeys, ducks and other waterfowls)	*Salmonella* spp.
Chlamydophila psittaci (ducks, turkeys)	*Listeria monocytogenes*
Mycoplasma gallisepticum	*Pasteurella multocida*
Salmonella spp.	*Mycobacterium* spp.
Escherichia coli	*Aspergillus* spp.
Reovirus	West Nile virus
Pericardial effusion	Eastern equine encephalitis virus
Fowl adenovirus (serotype IV)	Avian leucosis virus
Reovirus	Parvovirus (geese and Muscovy ducks)
Endocarditis	Avian encephalomyelitis virus
Enterococcus spp.	Reovirus
Streptococcus spp.	Avian paramyxovirus 1 (Newcastle disease virus)
Staphylococcus spp.	Avian influenza
Pasteurella multocida	*Sarcocystis* spp.
Erysipelothrix rhusopathiae	*Leucocytozoon* spp.
Pseudomonas aeruginosa	*Toxoplasma gondii*
Escherichia coli	
Reovirus	
Intravascular/intracardiac parasites	**Cardiac neoplasias**
Splendidofilaria spp.	Marek's disease virus
Chandlerella spp.	Avian leukosis virus
Cardiofilaria spp.	Reticuloendotheliosis virus
Paronchocerca spp.	
Sarconema spp. (swans and geese)	
Schistosomes (geese)	

Figure 15.9 Valvular and mural endocarditis in a broiler, note the dilated right ventricle and thickened right ventricular wall. *Staphylococcus aureus* was recovered from the tissues. (Source: Photo courtesy of Ms. Megan MacAlpine, University of Guelph.)
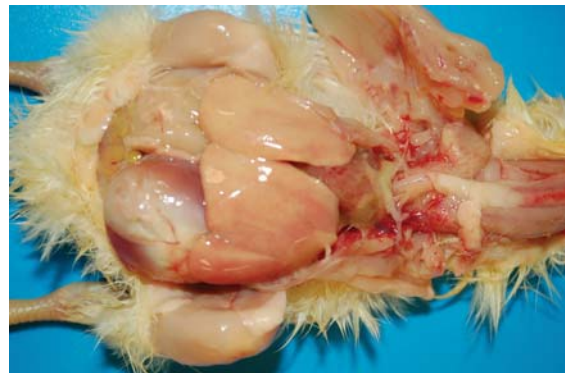
Figure 15.10 Bacterial fibrinous pericarditis and pale liver in young broiler, bacterial septicemia was present. (Source: Photo courtesy of Ms. Megan MacAlpine, University of Guelph.)

Transmission route

Bacterial and viral infectious agents, for the most part, do not have an intrinsic cardiac tropism and, for more specific information, the reader is invited to consult respective chapters on infectious diseases. Backyard flocks are naturally exposed to avian chlamydiosis through its wild bird reservoir (e.g., pigeons, gulls, egrets). The definitive host of *Sarcocystis falcatula* is the Virginia opossum, and cockroaches can serve as mechanical vectors. The definitive host of *Toxoplasma gondii* is the domestic cat. Some infectious pathogens are transmitted by arthropod vectors (*Leucocytozoon* by simuliidae, West Nile virus by *Culex*, and Eastern equine encephalitis by *Culisetta*, intra-cardiovascular parasites). Schistosomes penetrate hosts through the skin from a water environment.

Diagnostic tests

CBC may reveal leukocytosis. and biochemistry may reveal elevation in CK or troponin T in the case of myocardial involvement. Blood culture can be attempted to isolate a bacterial organism. Blood smear examination may identify microfilaria and blood stages of *Leucocytozoon* (gametocyte). Electrocardiography findings are not specific and may show augmented P, T, S, and/or R wave, prolonged segment intervals, axis deviation, ventricular premature contraction, ventricular tachycardia, and arrhythmias [10,13]. These changes are consistent with alterations of electrical conduction in the heart and myocardial ischemia. Valvular vegetation may be identified on a cardiac ultrasound examination and signs of congestive heart failure,

valvular regurgitation, and myocardial dysfunction may also be evident.

Differential diagnosis

Other infectious processes, other cardiac diseases and causes of congestive heart failure (ascites syndrome, turkey dilated cardiomyopathy). On necropsy, pericardial urate deposition can look very similar to pericarditis.

Prevention and control

Antimicrobials targeting the causative agents are indicated but most of these medications cannot be given to poultry from which eggs or meat are destined for human consumption because of an undetermined withdrawal period. Vaccines are available for selected bacterial and viral agents.

Zoonotic potential

Some bacterial pathogens causing cardiac lesions have zoonotic potentials (e.g., *E. coli*, avian chlamydiosis).

Nutritional and toxic cardiopathies
Clinical history

Younger poultry birds are more commonly affected by nutritional deficiencies and excess. Most non-nutritional cardiac toxicoses are of iatrogenic origin.

Causative agent

Some noncurrent antimicrobials used to cause cardiac diseases: Furazolidone (nitrofuran) causes dilated cardiomyopathy, and ionophores (e.g., monensin, salinomycin) cause myocardial necrosis. Doxorubicin and ethanol also have cardiac toxicities in poultry. Toxicities may also be caused by minerals in excess, such as silver, cobalt, selenium, lead, sodium, and potassium. Vitamin E/selenium deficiency may cause myofiber degeneration and Vitamin D3 toxicity leads to cardiac mineralization. Plant toxicities may be encountered with *Cassia*, *Crotalaria*, rapeseed meal (erucic acid, glucosinolate), and avocado (persin). Environmental toxins, such as chlorinated biphenyls and dioxin, have been involved in some poultry cases [5,34,55].

Clinical signs and lesions

Signs are usually non-specific. Furazolidone toxicity causes similar signs to dilated cardiomyopathy in turkeys and has been used in experimental induction of this disease. Sodium toxicity may induce ascites and edema similar to the ascites syndrome in chickens. Potassium deficiency and toxicity may lead to cardiac

arrhythmia. Chlorinated biphenyls, dioxin, and cresol may cause hydropericardium.

Transmission route

Not applicable.

Diagnostic tests

History and diet analysis are used for diagnosis. Myocardial lesions may lead to changes in the electrocardiogram and increase plasma muscle enzymes and troponin T. Dilated cardiac chambers, ascites, and hydropericardium may be detected on echocardiography.

Differential diagnosis

Furazolidone toxicity cannot be distinguished from dilated cardiomyopathy in turkeys, sodium toxicosis causes similar signs and lesions to ascites syndrome in chickens.

Prevention and control

Not applicable.

Zoonotic potential

Not applicable.

Atherosclerosis
Clinical history

Atherosclerosis mainly affects older birds and seems to be more common in male than in female poultry birds. Chickens, quails, and turkeys are susceptible and have been used as experimental models. Turkeys seem to be the most susceptible galliforme species to spontaneous atherosclerosis. Some lines are hypertensive and lesions have even been found in wild turkeys [56,57].

Causative agent

Atherosclerosis is a chronic inflammatory fibroproliferative vascular disease characterized by the buildup of atheromatous materials containing numerous compounds including inflammatory cells (mainly macrophages), lipid, calcium, and collagen in the luminal side of the arteries in response to multiple forms of endothelial injuries, which, in chickens, include Marek's disease (which also affects lipid metabolism) [30,33,58]. Risk factors have not been completely characterized in poultry but age, gender, specific breeds, inbreeding (some experimental lines of chickens and quails), and dietary factors may have a role in the pathogenesis. Hypertension may also be a risk factor in turkeys [57]. Cholesterol feeding induces atherosclerosis in poultry [59,60].

Clinical signs and lesions

Clinical signs are associated with arterial stenosis, myocardial infarction, aneurysm, and thromboemboli. However, they are uncommonly recognized in spontaneous disease and birds may die acutely or never show clinical manifestation. In some cases, atherosclerotic lesions may predispose turkeys to aneurysm and aortic rupture but the two conditions seem to be different clinical entities overall. Atherosclerotic lesions develop mainly in the coronary arteries, aorta, and brachiocephalic trunks in poultry. Advanced atherosclerotic lesions are composed of a necrotic and lipid core (atheroma) covered by a fibrous cap (fibroatheroma). Spontaneous lesions are rarely advanced in chickens [33,59].

Transmission route

Not applicable.

Diagnostic tests

Dyslipidemia may be a risk factor for the development of atheromatous plaques, such as an increase in cholesterol and LDL. Chickens have a normally high cholesterol level. Some lines of chicken are deficient in HDL but this does not necessarily correlate with more severe lesions [61]. Atherosclerotic lesions are usually not detectable in birds using current imaging modalities except when calcified, but may promote cardiac dysfunction and congestive heart failure.

Differential diagnosis

Aortic rupture/aneurysm in turkeys, vasculitis, fibromuscular dysplasia.

Prevention and control

Limiting risk factors by increasing activities and decreasing fat in the diet. Omega 3 fatty acids may be useful dietary supplements [62].

Zoonotic potential

Not applicable.

Acknowledgments

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Soft Tissue Surgery

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Introduction

In contrast to production practice, pet poultry and waterfowl commonly require surgical correction of various disorders. The popularity of backyard poultry and waterfowl, which are sometimes kept as indoor companions, means that more requests than ever are being made for advanced veterinary care. Clinicians treating these animals should at least be aware that surgical options for a large variety of soft tissue disorders are available. While not exhaustive, this chapter provides clinicians with information on a large variety of soft tissue surgical procedures that may be encountered in poultry and waterfowl.

A basic understanding of general surgical principles is necessary prior to performing avian surgery. Although there are many anatomical and physiological differences between birds and mammals, surgical techniques are very similar. As a result of small patient size and anatomical differences (avian air sacs for example), microsurgical instrumentation with magnification and focused light is often necessary for efficient bird surgery. For larger poultry and waterfowl, standard surgical instruments are often used. Because of physiologic variations (compared to mammals, birds exchange oxygen on inspiration and expiration and can frequently go into cardiac arrest following relatively brief apnea), anesthetic techniques in avian species are very different and are discussed elsewhere in the literature.

Halsted listed several principles that hold very true to maximize avian surgical success and are listed as follows: [1]

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
7. Obliteration of dead space

These principles are simple enough, but are important to understand and practice during all avian surgical procedures.

These principles are simple enough, but are important to understand and practice during all avian surgical procedures. Other than a brief mention, the following are not covered in this chapter but are essential to patient surgical success: Pre-surgical patient evaluation, appropriate anesthetic techniques, perioperative support (including fluid therapy, thermal support, antimicrobials and pain control as needed), and longer term pain management (Figures 16.1 a,b,c,d). The reader is encouraged to pursue education related to these topics prior to performing bird surgery.

If you are interested in avian surgery, actively pursue continuing education. One of the best continuous education courses is available at one's own hospital in the form of a necropsy. If permitted by the caretakers, perform as many necropsies on animals as possible to gain experience and exposure to avian anatomy, tissue handling, and instrument use. Also attend continuing education courses that teach avian medicine and surgery. Publications in refereed journals that focus on avian topics provide numerous well-referenced papers on surgical techniques, in addition to medical topics. Some of these journals are referenced herein.

Also familiarize yourself with the numerous potential surgical "tools." These "tools" include radiosurgery, microsurgical instruments, endoscopes, high powered microsurgical loupes with light, operating microscopes,

(a)

(b)

(c)

(d)
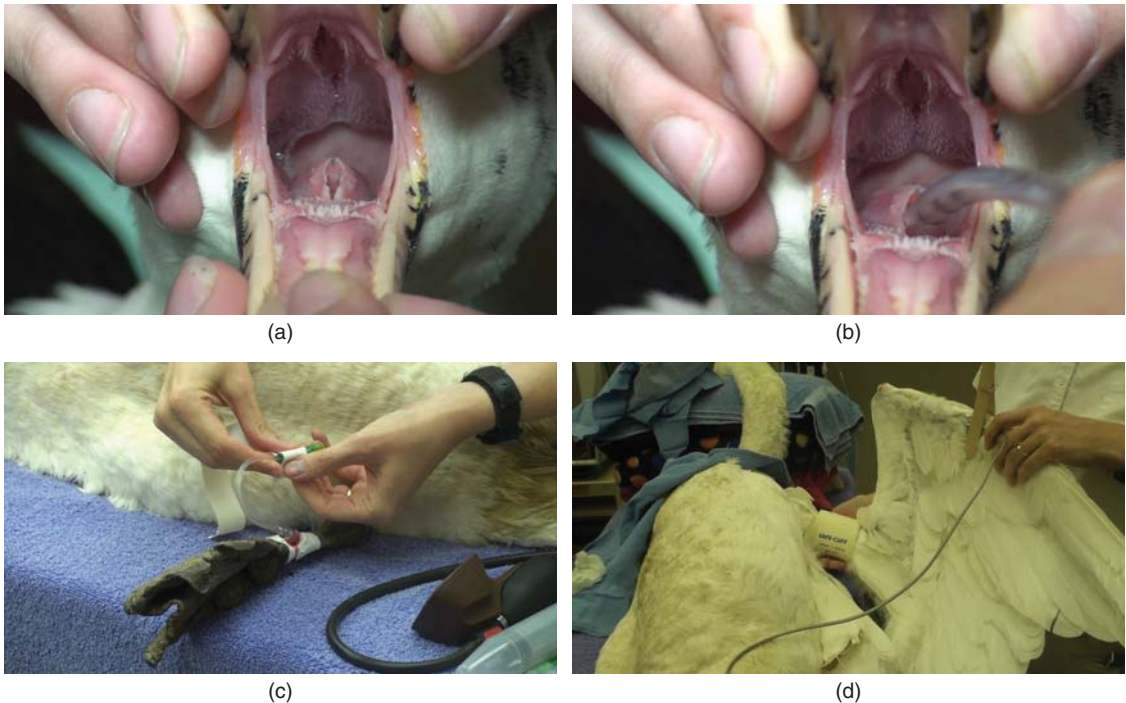
Figure 16.1 Poultry and waterfowl deserve perioperative supportive measures as with other animals. Perioperative support includes, but is not limited to, (a–b) intubation as with this Pekin duck (*Anas platyrhynchos*), (c) intravenous fluid support and (d) indirect blood pressure evaluation as with this mute swan (*Cygnus olor*) and peri-operative pain relief, thermal, hydration and antimicrobial management as dictated by the needs of the patient.

laser units, and other items that have become commonplace with avian surgery. Advanced diagnostics, including digital radiographs, ultrasound, high resolution computed tomography (CT), and magnetic resonance imaging (MRI), may be used to help better define the scope of the disease being addressed and to better guide surgery. Consult with surgical instrument companies, colleagues, and avian continuing education resources to stay up to date.

Throughout this chapter, case report examples are used to demonstrate certain points. Some of these case reports involve non-poultry or waterfowl birds and are intended to give the reader a greater depth and understanding of potential surgical techniques that may be used and complications that may be encountered.

Suture material

Suture material has been poorly studied in living avian species. There are, however, numerous studies describing various suture materials and patterns, mostly regarding poultry tendons, conducted on deceased animals. In one study, nylon, polyglyconate, or polybutester was used for long digital flexor tenorrhapy in live chickens. The trial was carried out to 8 weeks before euthanasia. The results showed that all three suture materials had insignificant differences in maximum load to failure, scar maturity, and tissue reactivity at 4 and 8 weeks [2]. Various chicken organs have been used as models of human tissues. Again, most of these studies are focused on deceased animals and offer little long term outcome information as to tensile strength, tissue reactivity, infection rates, and other clinical factors that determine the efficacy of a given suture material or pattern in living avian tissue.

Chromic catgut, polyglactin 910, polydioxanone (PDS), monofilament nylon, and monofilament stainless steel have been evaluated in rock doves (*Columba livia*) [3]. The authors concluded that PDS was slowly absorbed, strong, and caused minimal tissue reaction making it most suitable for closing body wall incisions [3].

In a separate study of polygalactin-910, chromic catgut, and PDS used in cloacopexy surgery in pigeons, the authors concluded that inflammation and fibrosis were most prominent with polygalactin-910 [4]. Because of the degree of inflammation and fibrosis,

the authors felt that polygalactin-910 would be more appropriate for cloacopexy as a means to promote adhesion formation at the surgical site [4]. Based on clinical experience and limited studies, PDS is used as the author's primary monofilament, absorbable suture in bird surgeries and is implied throughout this chapter when no specific details are given.

The upper respiratory tract and trachea

Infraorbital sinus surgery

Diseases of the sinus, especially related to infections, trauma, and cancer, are occasionally noted in waterfowl and poultry. Some of these require surgical intervention. The classic but non-specific signs of sinus disease include swelling of infraorbital sinuses, ocular and nare discharge, and head shaking (Figure 16.2). Solid masses, such as with cancer and walled off granulomas, may present with infraorbital sinus swelling only (Figure 16.3).

Especially among Galliformes, there are a number of viral and bacterial diseases that result in significant upper respiratory infections and may be reportable. These diseases include, but are not limited to, *Mycoplasma gallisepticum*, Newcastle disease virus and infectious bronchitis virus. Especially in flock situations, birds showing upper respiratory disease are significant for more than just the individual (Figures 16.4a,b,c). Properly characterizing and addressing infectious sinusitis in poultry species is important for more than just the individual and is discussed in the Respiratory Diseases chapter of this book.

Because of the cavernous anatomy of the infraorbital sinus and its many diverticuli and chambers, fluid, soft tissue, and inflammatory debris can build up unnoticed. Once the debris fills one portion of the infraorbital sinus it either spills over into an adjacent chamber or diverticulum (which may continue to go unnoticed) or reaches a point where a swelling is evident externally. Occulonasal discharge, conjunctivitis, and/or head shaking may or may not be present before a physical swelling is noticed by the owner or attending veterinarian. By the time a bird is identified as having a sinus mass or supportive clinical signs, it may have advanced disease that affects many regions of the infraorbital sinus(es).

Diagnostics such as skull radiography, choanal or nares inspection via magnified light or endoscopy, CT, MRI, infectious disease testing (PCR, culture and sensitivity, etc.) and sinus aspiration (with or without flushing small amounts of sterile saline), and cytology can be used to better characterize the cause and distribution

Figure 16.2   Typical signs of avian sinus infections include a distended infraorbital sinus, ocular and nare discharge and head shaking which were all present in this chicken.

Figure 16.3   Birds with non-infectious sinus disease often display a mass or distension of the sinus with minimal or no ocular and nare discharge as with this post-biopsy white Chinese goose (*Anser cygnoides*) with a sarcoma originating from the left infraorbital sinus.

of the swelling (Figure 16.5). While early cases of infectious sinusitis (before the debris becomes caseated) may respond to appropriate antibiotic therapy, most cases involve at least partially solidified masses that require surgical removal.

Unless guided differently by advanced diagnostics, incisions are best made directly over swollen sinus tissue and away from the eye (Figures 16.6a,b). Skin overlying inflamed sinus tissue is often very vascular and hemostasis is generally needed. Conversely, once inside the sinus the tissue is generally poorly vascular unless a mass is attached to the sinus wall or surrounding bone and muscle as sometimes occurs with cancer. The beak may be opened to increase potential sinus space to improve visualization.

(a)

(b)

(c)
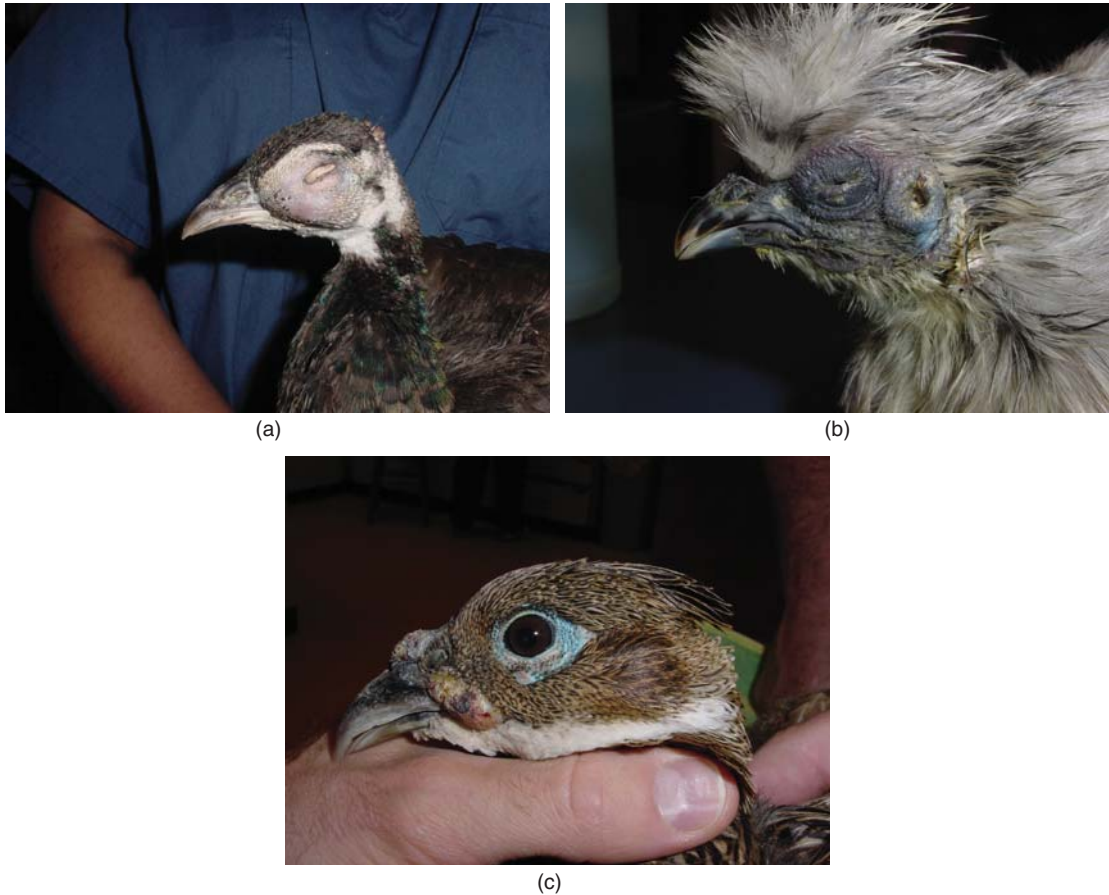
Figure 16.4 Poultry species with sinusitis or solid sinus masses should be evaluated for infectious disease that may have flock and/or regulatory importance such as (a) with *Mycoplasma gallisepticum* noted in this Indian peacock (*Pavo cristatus*), (b) lymphoma due to avian sarcoma leukosis virus in this chicken and (c) pox virus infection in this Impeyan pheasant (*Lophophorus impejanus*).

Magnification with light is essential to adequately inspect the infraorbital sinus. Microsurgical and miniaturized instruments are also beneficial when retrieving debris from deep recesses within the sinus (Figures 16.7a,b,c,d). The normal sinus space should be clear with no visible debris or discharge. Any discharge, foreign bodies, or debris are removed. Solid tissue may come out as an impression mold of the sinus space. Neoplastic tissue may form extension to and through the sinus spaces requiring either partial removal or more radical resection of surrounding tissues. Submit collected tissue for culture and sensitivity, other infectious disease testing, and/or histopathological evaluation if not previously undertaken.

The sinus space can hold a surprising amount of material and every attempt to remove debris should
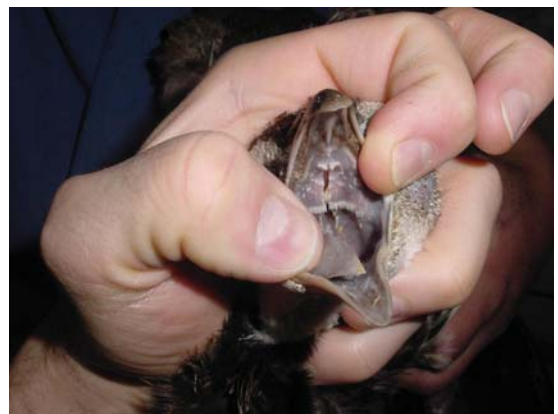
Figure 16.5 Indian peacock (*Pavo cristatus*) with a choanal granuloma associated with *Mycoplasma gallisepticum* sinusitis.

(a)

(b)

Figure 16.6 (a) A chicken with bacterial sinusitus presents with mild infraorbital sinus swelling. (b) The area is prepped for surgery and the incision is made over the swollen sinus (rostral diverticulum of the infraorbital sinus in this case).

(a)

(b)

(c)

(d)
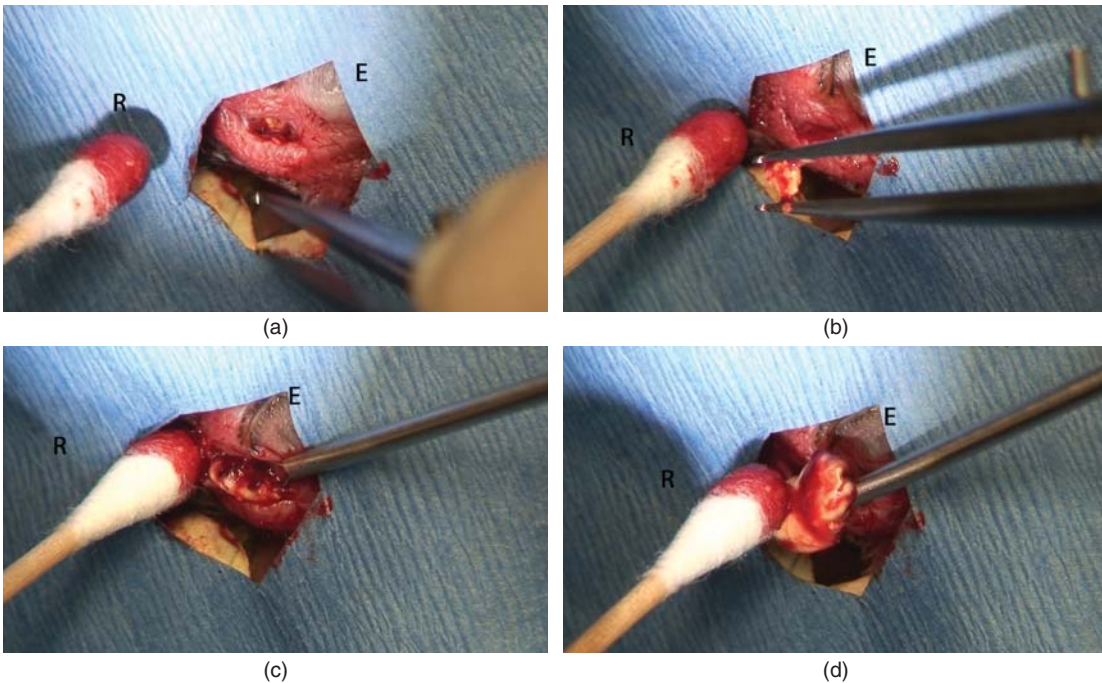
Figure 16.7 (a) The same chicken as in Figure 16.6 is covered with a drape and the rostral diverticulum of the infraorbital sinus has been incised and granulomatous tissue can be seen deep within the sinus. (b) Microsurgical ring tipped forceps are used to retrieve a small granuloma. (c and d) A small ear loop curette is used to retrieve a large granuloma from deep within the sinus. R, Rostral, E, Eye.

be made. Additionally, invasive material may extend into the beak diverticuli and opposite side of the head. Occasionally multiple surgical entries are required to reach visible debris. Trephination, as described for psittacines, into the beak or skull is rarely needed for addressing sinus disease in poultry and waterfowl but may be considered if necessary [5].

Once the bulk of the fluid, debris, and abnormal tissue is removed, the surgical site can be left open to drain (if infectious disease is suspected) or closed if the sinus was clean, such as with an excised encapsulated mass (Figure 16.8). Post-operative care may include daily or twice-daily flushing the open wound with an appropriate antiseptic solution (such as dilute chlorhexidine) until the wound closes. Use caution with any flush solution that is highly tissue-reactive, such as hydrogen peroxide, as this may cause more inflammation and inflammatory fluid and debris accumulation.

Figure 16.8 Post sinusotomy in the chicken featured in Figures 16.6 and 16.7, the wound is left open to heal by second intention.

Open sinusotomy wounds generally heal rapidly and make flushing difficult within 3–5 days. If appropriate, sinusotomy closure is standard and any sutures placed can generally be removed in 10–14 days. Systemic antibiotics and analgesics are given as needed.

Tracheal surgery

Numerous cases describing tracheal surgery in birds have been reported [6–13]. Tracheal surgery in poultry and waterfowl is sometimes required to resolve traumatic injuries, avulsions, intraluminal foreign bodies, parasites, or other mass obstructions and strictures. As with sinusitis, some tracheitis cases in poultry and waterfowl are caused by, or associated with, infectious diseases that affect other animals (Figure 16.9).

Partial tracheotomies can be used to access the tracheal lumen to remove foreign bodies that are non-retrievable via less invasive techniques (endoscopy, suction, etc.) [12]. Palpation, tracheal endoscopy, radiography, and/or transillumination can be used to identify the general location of the tracheal lesion. Endoscopic intraluminal tracheal examination is preferred as the other methods are less dependable to both identify and determine the extent of tracheal luminal lesions [14]. An air sac breathing canula is often required during tracheal surgery to provide adequate ventilation and gas anesthetic delivery. If concerned about distal migration of the foreign body, temporarily place a 25 gauge needle (or two) through the center of the trachea just distal to the object.

With the patient in dorsal recumbency, the ventral neck skin is incised over the general location of the foreign body. The trachea is readily found beneath the skin. Perform a transverse tracheotomy just proximal to the foreign body on the ventral half of the trachea between the tracheal rings. Handle the tissue with care and make precise incisions so as to limit trauma to the
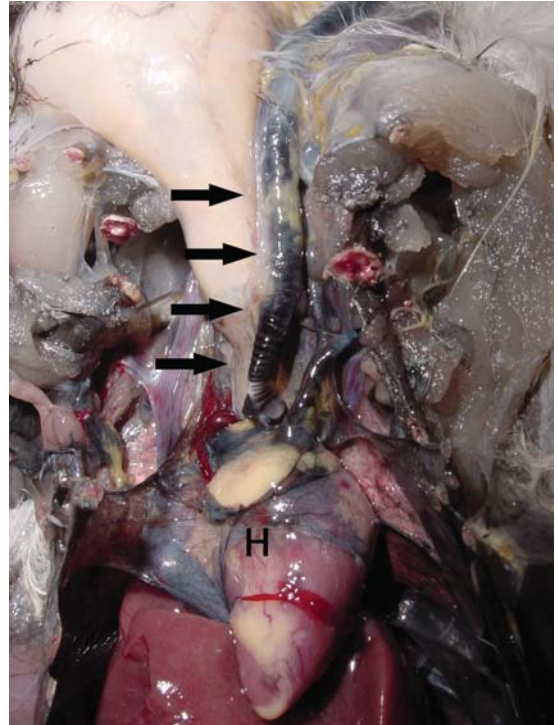
Figure 16.9 Inflammatory tracheal disease, such as hemorrhagic tracheitis (arrows) associated with avian sarcoma leukosis virus infection in this chicken, may be infectious and have implications that affect multiple animals. A definitive diagnosis of such cases should be determined. H, Heart.

trachea. If possible, avoid cutting the recurrent laryngeal nerves running alongside the trachea and preserve adhering and local blood vessels. Stay sutures may help to both retract the trachea out of the incision and guide closure. Retrieve the foreign body, being careful not to damage the tracheal luminal tissue. Pre-place small simple interrupted sutures (4-0' and smaller depending on the size of the bird) along the incised trachea and bring tissue edges back to normal apposition [9]. Tie knots on the serosal tracheal surface. Generally, at least one tracheal ring is incorporated on each side of the incision. Subcutaneous and skin closure is routine.

The crop may need to be reflected laterally and the thoracic inlet approached if the foreign body is located distally within the trachea. Incise the skin over the thoracic inlet and distal crop [15]. Bluntly dissect the skin from the crop. Bluntly dissect the crop from the surrounding tissues, keeping local blood vessels intact. Once freed, the crop can be reflected to the bird's left. The distal trachea and sternotracheal muscles (which

traverse obliquely and are attached proximally at the caudal lateral distal trachea) are identified. Transect the sternotracheal muscles near the tracheal attachments and coagulate or ligate bleeding vessels within the muscle bellies. Bluntly dissect the interclavicular air sac. Using a small blunt hook, latch the distal syrinx at the tracheal bifurcation and gently retract cranially. As a note, some ducks (especially *Anas* genus) have a well-developed assymetrical osseous bulla at the distal end of the trachea. This structure is easily seen radiographically and should not be misinterpreted as an abnormality (Figures 16.10a,b**)**. This normal structure should be carefully preserved. A tracheotomy is performed as above.

Tracheal resection and anastomosis is reserved for tracheal necrosis, avulsions, severe trauma, neoplasia, tracheal collapse, tracheal strictures, and other fixed obstructive masses that cannot be resolved non-invasively and significantly affect respiration. As a note, some tracheal strictures can be adequately treated with endoscopic debulking. One common reported cause of tracheal stricture is the result of trauma from endotracheal intubation [8,9]. The cause of the stricture may be a result of chemical disinfectant residues on improperly cleaned tubes, inflated cuffed endotracheal tubes, and/or simple trauma resulting from tube movement, advancing the tube distally into the narrowing trachea or other undue pressure within the tracheal lumen resulting in mucosal inflammation and subsequent fibrosis [9]. These iatrogenic tracheal strictures typically result in severe dyspnea 1 to 3 weeks after the offending intubation. Birds may exhibit dyspnea once 50% or more of the tracheal lumen is blocked.

Tracheal collapse has been reported in waterfowl following animal bite trauma to the bird's neck [10,11]. Tracheal resection and anastomosis with good outcomes were reported [10,11]. Similarly, an acute, 4 cm displaced tracheal avulsion was reported in a mallard duck (*Anas platyrhynchos*) with severe rapidly progressive dyspnea presumably resulting from some type of trauma [13]. The trachea was successfully anastomosed restoring respiration 30 minutes following surgery. The bird was found dead 3 weeks later and the cause of death was not determined [13]. While only reported in one ostrich, a 2.5 cm section of 0.75 cm wide polypropylene rings constructed from a 12 ml syringe case was used to support six collapsed tracheal rings that were transected as a result of blunt trauma and neck laceration [16]. The polyprolyene rings were sutured to the tracheal cartilages and submucosa (without perforating the lumen) using 2-0' PDS. Surgery was successful and the ostrich was doing well at least 6 months post-surgery [16].

The approaches are as described above. The damaged tissue is resected leaving as much viable trachea as possible in an effort to reduce tension along the anastomosis site. Either the tracheal rings are bisected ("split ring technique") or the annular ligaments between the rings are cut [11]. The split ring technique allows for better anatomical alignment during the anastomosis, however both methods have been successfully used with birds [11]. As with a partial tracheotomy, pre-placing sutures helps with tracheal anastomosis.
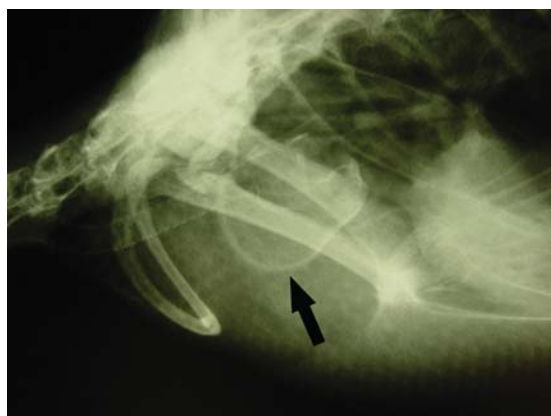
(a)

(b)

Figure 16.10 The osseous syringeal bulla (arrow) is a normal structure found at the distal end of the trachea in male ducks of some species and can be seen (a) on radiographs, as with this lesser scaup (*Aythya affinis*), during surgery of the distal trachea or (b) at necropsy as with this cinnamon teal (*Anas cyanoptera*).

Both simple interrupted and near-near-far-far patterns have been successfully used with tracheal anastomoses in birds [7–11].

With all invasive tracheal procedures, granulation tissue formation and subsequent additional luminal stenosis may be seen within 5-14 days of surgery [10]. Anti-inflammatory medications and often antibiotics and antifungals are indicated perioperatively as the trachea is not sterile [9]. Patients should be rested for at least 2–3 weeks following tracheal surgery with regular follow-ups to check for the presence of stricture formation.

Celiotomy approaches

A celiotomy is used to access the coelom in birds. The specific approach is determined by access needed, surgeon preference, and individual bird anatomy and physical condition as each entry point has distinct advantages and disadvantages. The approaches generally require the bird to be in dorsal or lateral recumbency. If ascites or significant organomegaly is present, elevating the proximal half of the body may help reduce pressure on the heart, lungs, and more cranial air sacs, improving ventilation. Regardless, most birds do not ventilate well while in dorsal or lateral recumbency and positive pressure ventilation is often required throughout the procedure (even if the bird appears to respire normally).

Left lateral celiotomy

The left lateral celiotomy provides good exposure to the proventriculus, ventriculus, spleen, colon, left male and female reproductive tracts, hepatic lobe, lung, heart apex, kidney, and ureter [17]. Place the anesthetized patient in right lateral recumbency with the wings pulled dorsally, right leg caudally and left leg cranially. In some cases, the left leg is best pulled caudally, especially when a more cranial approach to the lateral coelom is required. Tape the extremities in place with masking tape, Durapore™ (3M, St Paul, MN), or any other tape that is easily removed. Make a longitudinal incision from cranial to caudal in the left paralumbar fossa. The incision may extend from the cranial extent of the pubis to the uncinate process of the last rib. If needed, the incision can be further extended cranially by incising through the last rib(s) at the costochondral junction(s). Use radiosurgery, laser, sutures, or simple hemostasis to control hemorrhage.

Once through the skin, bluntly dissect through the lateral coelomic muscles including the external oblique, internal oblique, and transversus abdominus mm. It is best to dissect the muscles in the direction of their fibers to reduce excessive tearing. At this point, the abdominal air sac is visible dorsally. The air sac is commonly punctured to approach more dorsal structures but is preserved if possible. Palpebral, Gelpi or similar retractors are very useful to better expose the underlying structures. The muscles are closed individually, if clearly defined, via a simple interrupted pattern. Thin, stretched, or indistinguishable muscles may be closed in one layer. Skin may be closed via multiple patterns (simple interrupted, simple continuous, everting, Ford interlocking, etc.) and is based on surgeon preference. Monitor for subcutaneous emphysema and air leakage through the skin incision. Re-suture as needed to reduce emphysema and stop air movement through the skin incision.

Right lateral celiotomy

A right lateral celiotomy provides good exposure to the duodenum and pancreas, right male and female (if present) reproductive tracts, lung, heart apex, kidney, ureter, and hepatic lobe. This approach is far less commonly performed given the more frequent need to access the ventriculus and female reproductive tract via a left lateral celiotomy. The approach is otherwise reversed from a left lateral celiotomy and closure is routine.

Ventral celiotomy

A ventral midline, transverse, or combination celiotomy is used to expose the middle and/or both sides of the coelomic cavity gaining access to the liver, intestines, pancreas, kidneys, ureters, cloaca, and oviduct. The testes and ovaries can also be accessed via a ventral approach but this does require manipulating surrounding tissues to improve exposure. The incision is made on the ventral midline from just caudal to the sternum extending caudally to the interpubic space. The supraduodenal loop (ileum) lies relatively ventral along the midline of the caudal coelom and can be easily transected if not careful. For this reason, the midline incision should be made as cranial as possible unless the caudal ventral coelom must be explored as with some cloacal surgeries. After the skin incision is made, the linea alba is tented upward and carefully transected, taking care not to damage underlying organs. The air sacs are preserved using ventral approaches.

The transverse and combination ventral celiotomy can be used to increase exposure to the coelomic cavity in birds. A transverse incision is made just caudal to the sternum. If needed, a ventral midline incision is used in conjunction with the transverse incision ("T" incision)

to increase exposure. Alternatively, a transverse incision can be made on the left or right half of the midline, combined with a ventral midline incision, creating an "L" incision. The "T" or "L" incision is only made if increased exposure is needed. As discussed above, underlying structures should be carefully avoided when incising through the underlying coelomic wall.

The linea alba and other transected muscle is closed in a simple interrupted pattern. As found in some overweight poultry and waterfowl, the subcutaneous tissue may need to be closed (commonly a simply continuous pattern). Skin closure is routine.

The gastrointestinal tract

The gastrointestinal tract, from oral cavity to vent, may require surgical corrective procedures in pet poultry and waterfowl. The beak, while technically the starting point of the gastrointestinal tract, is highly specialized and variable between species. Surgery of the beak, most commonly trauma related, will not be covered here. Additionally, "debeaking" procedures performed in some production facilities as a behavior management practice are not recommended for pet animals.

The approaches and closure methods for the avian gastrointestinal tract have not been critically evaluated, controlled and reported in scientific journals as most such surgeries are based on anecdotal experience [18]. While there is a belief that avian gastrointestinal tract surgery carries an "unacceptably high incidence of post-operative complications," the author feels these procedures can be performed safely and effectively in many circumstances [18].

Because of the potential for leakage of food or intestinal contents and dehiscence, careful attention should be paid to aseptic technique and meticulous closure. Antibiotics may be required for many gastrointestinal surgeries and should be determined based on the surgeon's evaluation.

Oral cavity surgery

Diseases of the oral cavity are infrequently reported in poultry and waterfowl. Most simple trauma and infections can be medically managed. Larger lacerations and removal of small masses may require surgical debridement or excision and closure. Occasionally, oral parasites such as flukes may be found in waterfowl and can usually be mechanically removed. Disruptions of the hyoid apparatus may result in difficulty swallowing, breathing, and vocalization [19]. A fractured ceratobranchial bone (of the hyoid) resulted in difficulty swallowing,

localized soft tissue swelling, and ipsilateral epiphora in a black African goose Except for intermittent epiphora, all clinical signs resolved with antibiotics and supportive therapy [19].

Oral masses, especially neoplastic, may interfere with breathing and deglutition and should always be investigated. Oral squamous cell carcinoma has been reported in several chickens and, as with other neoplasms, should be considered along with abscesses, granulomas, and cysts when oropharyngeal masses are noted [20]. Oral pox lesions (see Figure 16.4c) rarely require surgical removal and are usually treated conservatively. Successful treatment of oral neoplasia in poultry and waterfowl has not been described likely because reported cases describe large masses that either resulted in euthanasia or are found on necropsy. If small, oral masses may be removed. Otherwise, biopsy oral masses in at least two locations if possible. Abscesses, granulomas, and cysts may be drained or debrided. Consider more radical removal, cryotherapy, radiation, chemotherapy, or other modalities for neoplastic lesions.

Sublingual entrapments (impactions) are occasionally seen in herbivorous waterfowl and some require surgical correction. Common presenting signs include difficulty swallowing, ventral intermandibular swelling, and, rarely, debilitation with chronic and severe impactions (Figure 16.11). Although risk factors have only been suggested, ingesting dry fibrous foods may be a major predisposition to sublinqual impactions [21]. With impactions, the food accumulates lateral to the frenulum beneath the tongue. In early cases, the beak can be opened and the material simply pulled out. Brown recommends preventing birds from grazing for 7–10 days following removal of simple impactions [21]. This time away from coarser food hopefully gives the sublinqual "pouch" time to return to normal size.

With chronic cases, an intermandibular pocket forms, requiring removal of the food and surgical resection of the stretched tissue (Figures 16.12 a,b,c). The bulk of the impaction can be removed per os. If the mass is large and chronic, it may cause local oral mucosal necrosis and subcutaneous food invasion. A ventral intermandibular approach is made and all remaining food and necrotic tissue is removed. With or without subcutaneous food invasion, the excess sublingual "pouch" is resected on either side of the frenulum (one or both sides may be stretched) and sutured closed in a simple interrupted or continuous pattern. The goal is to decrease the potential space to prevent additional impactions. If the subcutaneous tissue is infected and cannot be completely removed, marsupialize the ventral intermandibular skin and allow healing by second

Figure 16.11 Sublingual food intrapment and impactions (arrow) are occasionally seen in herbivorous waterfowl, such as this mute swan (*Cygnus olor*).

intention. Otherwise, the author tacks the partially resected oral mucosa to the subcutaneous tissue and overlying skin, to further reduce any potential space. Skin closure is routine. Antibiotics may be required.

Post-surgery, dry fibrous foods are avoided. Options for feeding include access to natural pond grasses (swans), access to green watered grass (geese), short cut commercially available grasses (using a food processor), or pellets. The latter two are intended to be short term options. The owners should periodically inspect the oral cavity for signs of impaction for up to 3 weeks post-surgery and when dry and coarse food sources may increase risk of re-impaction.

Esophageal surgery

There are few discussions on esophageal surgical procedures in birds. Most center on pharyngeal lacerations, crop burns (that extend up into the cervical esophagus), esophageal foreign bodies, and simple trauma. For the most part, the avian cervical esophagus is expansible and is easily closed in a simple interrupted or continuous inverting pattern. The overlying skin may be closed in a separate layer or with the esophagus incorporated into the closure. One more common reason for esophageal surgery is to place an esophagostomy feeding tube.

Mostafa *et al.* describe laceration of the upper third of the esophagus in a male ostrich (*Struthio camelus*) [22].
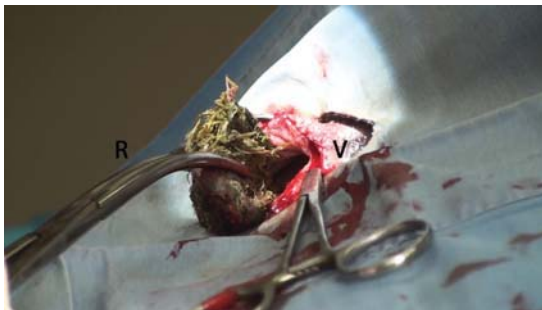
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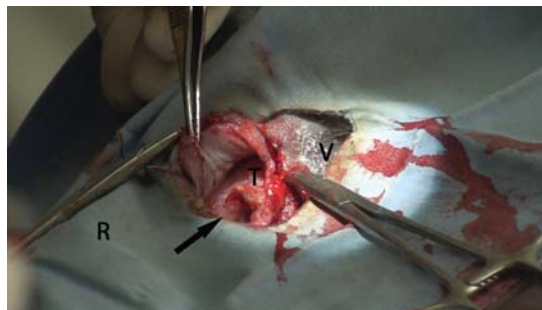
(c)

Figure 16.12 Using a ventral approach to the caudal intermandibular space of the mute swan in Figure 16.11, (a) the impacted food mass is being retracted from the ventral oral cavity out the incision until (b) the entire mass is removed (c) once the mass and remaining debris are removed, the ventral oral cavity and frenulum (arrow) are visible. R, rostral, T, Tongue Base, V, Ventral.

A 10 cm skin laceration resulted in underlying damage to the cervical esophagus. The esophagus was closed in a simple continuous pattern using 3-O' polyglycolic acid. The skin was closed using nonabsorbable suture material in a simple interrupted pattern. The wound healed uneventfully and sutures were removed 10 days after surgery [22].

An esophagotomy was successfully performed on a Canada goose (*Branta canadensis*) to remove an esophageal impaction consisting of legumes, grass, and fishing line [23]. The esophageal incision was closed with 4-O' polyglactin 910 in a simple interrupted pattern and then oversewn in a simple continuous pattern and skin closed in a simple interrupted pattern. The authors noted that if an esophageal foreign body is stuck and cannot be retrieved per os, it should be lubricated and pushed into the crop (if possible) for an ingluviotomy [23].

Crop surgery

Ingluviotomy may be indicated for determination of crop masses (neoplasia, food impaction, and foreign bodies), repair damaged tissue (especially from predator bite wounds and infection,) and to gain access to the thoracic esophagus, proventriculus, and ventriculus via intraluminal endoscopy. As a note, some chickens infected with Marek's disease virus have crop distension and should be considered as a differential as the infection may have implications for other birds (Figure 16.13).

Incise the skin over the bird's right side or middle of the crop near the thoracic inlet. Bluntly separate the skin and crop until you can pull the crop partly out of

Figure 16.13 As shown here, chickens infected with Marek's disease virus may develop a distended crop due to lymphoma of the innervating ingluvial nerves.

the incision. Remove abnormal tissue, masses, impacted food, or foreign bodies if present. A two-layer closure works best with crop incisions. The first layer is closed with an inverting suture. One author notes that the skin and crop should not be closed together as a single layer as this may increase the risk of dehiscence [15]. However, the author closes the skin and crop together as the second layer and has not noted problems in clinical cases. Realize that the crop does have peristaltic movements separate from the overlying skin and closing the two together may affect motility.

The same approach is used to perform thoracic esophageal, proventricular, and ventricular endoscopy. One report noted using endoscopy and an endoscopic wire basket retrieval device to snare a ventricular foreign body (rubber tube) via an ingluviotomy in a gray parrot (*Psittacus erithacus*) [24]. Another report described use of multiple ingluviotomies and ventricular endoscopic retrievals to remove artificial grass fibers from a gyr falcon (*Falco rusticolus*) [25]. As a result of the density of the artificial grass mass, only small pieces of the mass could be removed during each surgery (which totaled five). Once the bulk of the mass was removed, the bird was offered feathered quail acting as casting material The bird casted up the remaining foreign material 2 days later [25]. The same principles of ingluviotomy and endoscopy apply to poultry and waterfowl.

Crop repair is most often indicated following trauma. After the traumatic incident and prior to surgery, wait until the margins of the necrotic tissue are clearly visible (usually 4-7 days after burn trauma). Acute crop, non-thermal, trauma can usually be immediately surgically addressed. Remove all necrotic tissue and close as described above. The crop has an incredible ability to stretch and even large crop resections seem to be well tolerated by most birds. Subsequent feedings obviously need to be reduced depending on the post-operative size of the crop. Rarely, a proventricular feeding tube may be required if crop resection is extensive and this area needs to be bypassed during the healing process.

While a total ingluviectomy would be rare, it can be performed if the crop must be resected as in the case of severe trauma, cancer limited to the ingluvies, necrosis, and infection. A resection and anastomosis is performed with an attempt to limit as much tension as possible on the cervical-thoracic esophageal suture line. One study showed that using a stent (straight macaroni) to guide esophageal closure dramatically reduced six-week post-operative mortality (due to surgical site stricture) from 50% to 0–3% in cropectomized chickens [26]. The anastomosis site may be closed in two layers

(simple interrupted followed by continuous inverting) or as best determined by the surgeon. Skin closure is routine. Interestingly, some cropectomized birds may regenerate a new "out-pouching" soon after removal of the crop [26].

One obvious complication of ingluviectomy is reduced feeding post-operatively. One serious potential cause of reduced feed intake (aside from the absent crop) is incision site stricture, which may be reduced by a stent as described above. However, studied laying chickens showed lowered serum calcium, egg specific gravity, and overall egg production post-ingluviectomy compared to non-ingluviectomized controls. The authors postulated that the crop serves as an important storage depot of feed providing nutrients necessarily for egg shell quality during non-feeding periods [27]. Ready access to food, and possibly calcium supplementation, should be considered if a laying hen is ingluviectomized.

Proventricular and ventricular surgery

The "stomach" of birds consists, from orad to aborad, of the proventriculus, isthmus, and ventriculus respectively. In general, the proventriculus and isthmus are soft muscular organs and the ventriculus is composed of strong contractile muscles and a tough inner lining (koilin layer) designed to crush food, sometimes with the aid of ingested stones. The proventriculus secretes digestive enzymes that help prepare the food for mechanical digestion in the venticulus. The isthmus is the short region between the proventriculus and the ventriculus.

Birds such as granivores, which eat a coarse diet, have well-developed paired thick and thin muscles with a thick koilin layer on the mucosal surface similar to galliformes [28]. Birds such as planktivores, pisciovores (fish), and carnivores, which consume a soft diet, have a relatively thin-walled ventriculus and koilin layer [28]. Depending on the type of waterfowl and its diet, the ventriculus may range from poorly- to well-developed.

Proventriculotomy and ventriculotomy are reserved primarily for the removal of foreign bodies that have not been eliminated via conservative therapy or are non-retrievable using endoscopy or other less invasive techniques. Although not reported in poultry or waterfowl, ventricular diverticula were found in parakeet auklets (*Aethia psittacula*) that were kept on loose stone substrate. It was postulated that stones left in the soft ventriculus of these fish, krill, and copepod-eating birds could result in diverticula formation and ventriculotomy should be considered if the foreign bodies are present in this species [28]. However, most reported cases involve gastrointestinal impactions in ratites, but have also

been described in kiwis (*Apteryx australis*), umbrella cockatoos (*Cacatua alba*), Micronesian kingfishers (*Halcyon cinnamomina cinnamomina*), and sarus cranes (*Grus antigone*) [29–32]. Ventricular foreign bodies and subsequent obstruction and perforation have been reported as an important cause of mortality in bustards [33]. Food, fiber, and sand proventicular impactions are reported in waterfowl with lead toxicosis [28]. While seemingly absent from the refereed literature, poultry and waterfowl also develop proventricular and ventricular foreign bodies that require surgical extraction.

The same approaches to the proventriculus and ventriculus are also used to obtain biopsies (neoplasia, etc.), address perforating ulcers and diverticula and to explore the serosal surface of the proventriculus, isthmus, and ventriculus. Prior to surgery, conservative therapy using bulking agents, fluid therapy, and basic support should be attempted.

The ventriculus consists of two opposing muscle pairs: The cranial and caudal thin muscles and the lateral and medial thick muscles [34]. The alternating contractions of the thin muscles, duodenum, thick muscles, and proventriculus make up the gastroduodenal motility sequence in poultry [28]. Contrast fluoroscopy can be used to view organ shape and ventricular contraction sequence [28].

The myenteric nerves cover the entire surface of the thin ventricular muscles and isthmus. Studies of domestic fowl have shown that in order for proper gastroduodenal motility to occur, the myenteric plexus associated with the isthmus must remain intact. It is also suspected that initiation and regulation of the thick muscles also act via the nerves covering the isthmus. Specifically, isthmus denervation reduces the frequency of duodenal and muscular stomach contractions by 50% and abolishes glandular stomach contractions (in turkeys) [35]. The nerves encircling the isthmus do not appear important in regulating thin muscle contractions [34]. These findings support the need for atraumatic and precise surgery when incising the isthmus as discussed below.

For adult birds undergoing proventriculotomy or ventriculotomy, fast the patient for at least 12 hours to help "clean" the gastrointestinal tract. If possible, use handfeeding formula 1–2 days prior to surgery, as these easily digestible foods tend to leave little residue in the ventriculus. Discontinue feeding of formula food 6–12 hours prior to surgery. Pre- and post-operative antibiotics should be considered as with other animals undergoing enterotomies.

A left lateral or ventral midline combined with transverse celiotomy may be used to approach the

ventriculus. If the ventriculus is displaced medially (as supported by contrast study radiographs), the ventral midline approach is more appropriate. Some surgeons prefer a ventral midline approach to enter the ventriculus through the caudoventral sac (see below). The proventriculus and isthmus are approached via a left lateral celiotomy.

With either a lateral or ventral approach, place stay sutures in the white tendinous portion of the ventriculus to help retract the organ(s) out of the coelomic cavity and improve exposure [36]. As a result of its location, the proventriculus cannot be exteriorized but visualization is improved by retracting the ventriculus. It is best to pack moist sponges around the retracted organs to help prevent coelomic contamination.

Via a left lateral celiotomy, incise into the relatively avascular isthmus and extend the incision orad into the proventriculus or aborad to the ventriculus as needed. At this point, both the proventriculus and ventriculus can be explored. As a result of the massive mobile muscular tunic and high tensile strain on the tendinous centers, the ventriculus does not have a good site for incisional entry [18]. Additionally, an endoscope may be introduced to improve visualization and help retrieve foreign bodies when present. The caudal thoracic (cranial coelomic) esophagus can be partially evaluated via this approach. Additional ribs may need to be transected to better view the lower esophagus. Irrigation and suction are often needed – be careful not to contaminate the coelomic cavity. Use fine monofilament, absorbable suture in a simple continuous pattern to close the wound. If possible, oversew with a continuous inverting pattern. Meticulous closure is required to help prevent dehiscence.

Using the ventral midline approach, the ventriculus may also be approached via the caudoventral sac [36]. The ventriculus has two blind sacs (craniodorsal and caudoventral) covered with relatively thin muscles. The ventriculus is slightly rotated clockwise to help expose the caudoventral sac. Incise through the muscle fibers to enter the ventricular lumen. Again, use meticulous closure. This tissue does not invert, so use interrupted sutures placed close together. In a study of Coturnix quail undergoing caudoventral sac ventriculotomy, ventricular mucosal healing was not complete until 21 days post-surgery [18].

While collagen patches have been suggested in mammals to help intestinal wounds heal, they may be detrimental to birds. Porcine submucosal collagen patches placed over the serosal surface of the ventricular suture line in Cortunix quail that underwent ventriculotomies resulted in a statistically significant increase in gross or microscopic perforations [18]. The authors of the study suggested that the collagen patch generated a lymphocytic xenograft rejection response [18].

In one study of proventriculotomies performed in ostriches, 6 of 18 died immediately post-operatively and 4 of the 12 surviving birds died within 30 days of the procedure [30]. The authors noted that many of the birds were debilitated prior to surgery and recommended an esophagotomy be performed in all young or debilitated birds at the same time as surgery to provide post-operative nutrition as many birds are anorectic for several days after surgery. The authors also made note that no adverse sequelae were noted from the esophagotomy [30]. In a separate report involving phytobezoars in three Micronesian kingfishers (*Halcyon cinnamomina*), the authors noted that one bird died during ventriculotomy via a ventral midline approach [37]. The other two birds were treated medically. When medical therapy failed to resolve the phytobezoar in one bird, ventricular endoscopy was unsuccessful and the bird died during preparation for ventriculotomy. Although the success rate was poor in this group, the authors recommended brief medical management followed by surgical extraction in non-resolving cases [37]. Despite these reported surgical mortalities, the author feels that proventriculotomy and ventriculotomy can be safely performed in poultry and waterfowl and should be considered if conservative measures fail.

Lower intestinal surgery

With the exception of research studies on cecal surgery and colostomies in poultry, lower intestinal surgery is rarely reported in avian medicine. However, both metallic and non-metallic intestinal foreign bodies are described in multiple bird species [38]. Non-metallic lower gastrointestinal foreign bodies are most frequently linear, occasionally form a nidus or enterolith, and are diagnosed based on palpation or necropsy. Radiographs, with or without barium or iodine (especially if gastrointestinal perforation is suspected) and ultrasound may also aid in diagnosis. One case report describes a 14-month-old female Eclectus parrot (*Eclectus roratus*) with a mineralized intestinal foreign body. The foreign body and proximal part of the duodenum were removed and the bird recovered uneventfully. The details of the actual surgery were not included other than that the foreign body was brittle upon removal and had a central fiber-like structure [38]. One paper briefly notes that an ostrich died of complications associated with small intestinal resection and anastomosis performed because of a perforating intestinal foreign body. The same ostrich underwent a proventriculotomy

2 months previously [30]. In a separate ostrich case, a 7-and-a-half-month-old castrated male died suddenly 4 months after castration. A segment of intestines was herniated and entrapped within the right pulmonary ostium, was dilated and underwent ischemic necrosis. The cause of the entrapment was believed to be disruption of the air sac walls separating the right caudal thoracic and abdominal air sacs. Eventually, the intestine found its way through the opening, resulting in the herniation. The author noted that care should be taken to not disrupt this air sac integrity (at least in ostriches) [39].

Intestinal resection, repair and anastomosis are delicate procedures in birds. Use microsurgical instruments to remove necrotic or damaged bowel and spare healthy tissue and the surrounding vascular supply. Use 6-0′ to 10-0′ absorbable monofilament suture on $\frac{1}{4}$ circle atraumatic needles for intestinal anastomosis and enterotomy closures [15]. Six to eight simple interrupted sutures are often necessary for end to end anastomosis. Enterotomy closures should be performed so as to limit intestinal stricture.

Duodenal feeding tubes may be needed to bypass a diseased proventriculus, ventriculus, or other upper intestinal area. Via a midline or transverse celiotomy, an indwelling jugular catheter >1/3 of the diameter of the small intestine is placed through the left coelomic wall and into the descending duodenal loop [15]. Advance the catheter through the descending and ascending duodenal loop and remove the catheter's needle. Use 5-0′ monofilament suture to attach the duodenum to the coelomic body wall and provide a tight seal. Test the catheter patency and duodenostomy seal by injecting sterile saline and then close the body wall. Secure the external portion of the catheter using monofilament suture. Coil the external catheter and secure it to the bird's leg and wing. Divide the liquid diet into small frequent feedings. Flush the catheter with warm isotonic fluids before and after each use to prevent catheter obstruction. Closely monitor the incision site and patient for signs of coelomitis, leakage, and catheter damage. When complete, cut the sutures and pull the catheter, leaving the incision to heal by second intention [15]. The intestine is at least temporarily adhered to the body wall.

Duodenal aspiration may be helpful in identifying occult parasitic (*Giardia* spp. and other protozoa) and *Mycobacteria* spp. infections and small intestinal bacterial overgrowth [40] (Figure 16.14). Via a ventral midline surgical approach, the duodenal loop is isolated [41]. Using a 25 gauge or smaller needle, aspirate the duodenal contents for culture and cytology. Additionally, use
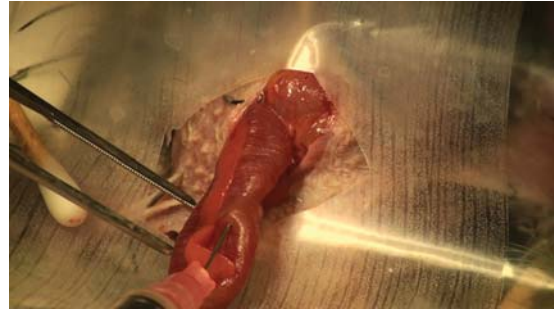
Figure 16.14 Duodenal aspiration using a 25 gauge needle in a blue headed pionus (*Pionus menstruus*).

another needle with the bevel side up to aspirate the mucosal surface of the duodenum. Oftentimes, occult mycobacterial organisms can be recovered cytologically by aspirating affected thickened duodenal mucosa. Closure is standard and the collected samples should be processed and evaluated as soon as possible.

During percutaneous implosion and subsequent collapse of a soft-shelled egg in an eclectus parrot (*Eclectus roratus solomonensis*), a tear in the cloacal mucosa developed that required closure and ultimately a duodenal serosal patch [42]. The iatrogenic 5 mm cloacal tear, located between the opening into corprodeum and uterine opening into the urodeum, was approached via a ventral midline cloacotomy and sutured closed. The bird did not produce feces for over 36 hours after surgery. A barium series supported a terminal colonic-rectal obstruction. Ventral midline coeliotomy revealed that the entire intestinal tract was severely distended and suture material surrounded the distal colon near its junction with the cloaca (causing the obstruction). Upon manipulation, the colon ruptured in two places. The fecal material was removed and the colonic defects were closed with 5-0′ polydioxanone in a simple interrupted pattern. The serosa of the adjacent duodenum was sutured circumferentially over the repaired colonic defects using 8-0′ nylon in a simple interrupted pattern at 2 mm intervals without penetrating the lumen of the colon or duodenum. The sutures were placed approximately 2 mm from the sutured colonic defects. The sutures placed during the original surgery were removed, allowing feces to enter the cloaca. The cloacal wall defect was closed with 5-0′ polydioxanone in a simple continuous pattern. A salpingohysterectomy was also performed. The bird recovered uneventfully and was followed up to 3 years post-surgery and reported to be recovering well [42].

Cecal surgery has been described but only under experimental conditions and cecectomized or cecal

ligated chickens, ducks, geese, and turkeys are used in digesta analysis and other studies [43–47]. In many avian species, ureteral urine flows aborad (via peristalsis) into the colon and ceca where water absorption occurs. At least in chickens and turkeys, when the ceca are ligated, total water excretion is increased [45–48]. Cecostomized chickens show increased water intake and reduced transit time of digesta in the ceca [49]. Cecal ligation alters nitrogen metabolism in adult and young chickens [46,47]. Also, both cecal ligation and colostomy in turkeys significantly alters cecal (colostomy only), ileal, and rectal motility, possibly as a result of changes in lower intestinal water content [45]. Current studies show the importance of avian ceca in water balance, and other metabolic functions, and should be considered if this organ is surgically manipulated.

While described primarily for research purposes, cecectomy is a fairly simple procedure. Clinically, cecectomy would be indicated to address diseases that could not be medically managed such as cancer, necrosis, and so on. The bird is placed in right lateral recumbency with the left leg pulled cranially for a paralumbar approach [44]. A 3–4 cm paralumbar fossa incision is made, being careful to avoid penetrating the air sacs, and the ceca are retracted. Each cecum is then separated from the ileum via blunt dissection of the ileocecal ligament. Vessels running within the ligament are ligated. Ligate each ceca 1.0 cm distal to the ileal junction with absorbable suture. Remove the ceca. Carefully lavage the coelom and cecal stumps to ensure no free ingesta is present. Closure is routine [44].

Colostomy, while not reported in clinical cases, has been frequently described in chickens and turkeys for experimental purposes (in studies that require separation of urine from feces) [50,51]. This procedure might conceivably be used as a temporary or permanent solution to address distal colonic and/or proximal cloacal disease (such as cancer) where the affected tissue must be removed and a continuous colonic-cloacal segment would not be possible.

The technique in chickens starts with exteriorizing the colon via a distal ventral midline incision and milking the feces from the distal colon into the cloaca and/or proximally. Next, transect the distal colon approximately 1.5–2.0 cm proximal to the cloaca and ligate both ends of the colon. Atruamatic intestinal forceps can be used to gently clamp the distal colon during manipulation instead of ligation. Using 4-0' silk, ligate the seromuscular coat of the colon to the peritoneal tissue, lateral to the cloaca and vent, at three points in a triangular shape. Ligate the distal-most aspect of the colonic mesentery to prevent bleeding out of the stoma. Next, place three sutures to form a triangle with the seromuscular aspect of the proximal transected colonic segment and the skin, again lateral to the cloaca and vent. Last, suture the everted colonic mucosal tissue (remove colon ligation sutures if placed) to the skin in a simple interrupted pattern to complete the stoma. The authors recommended antibiotics for 3–5 days post-surgery and frequent monitoring of the stoma for closure, infection, or other complications and showed that chickens did quite well for months following this procedure [50]. A similar procedure, with some variation, has been reported to be successful in turkeys [51].

The cloaca of birds consists of three main compartments (from orad to aborad); the coprodeum, the urodeum, and proctodeum. The distal colon enters the coprodeum which is separated from the urodeum via a coprourodeal fold. The ureters enter the urodeum through a sphincter muscle and an opening that is covered by transitional epithelium preventing ureteral reflux [52]. Further, the urodeum is separated from the proctodeum via the uroproctodeal fold. Feces combine with urine and urates form the complete dropping, which is eliminated through the proctodeum and out of the external vent.

The most common cloacal abnormalities seen in Galliformes and waterfowl result from trauma (animal attacks, dystocia, "vent pecking," etc.), but infections, cancer, and other disorders may be encountered. When performing cloacal surgery, careful attention must be paid to the normal anatomic features, especially the colon-coprodeal and ureter-urodeal junctions. The goal of cloacal surgery is to restore as much of the normal anatomy and function as possible.

A 7-year-old female umbrella cockatoo (*Cacatua alba*) was evaluated after an incisional cloacopexy that incorporated the pubis [53]. The bird had a chronic history of cloacal prolapse. Six days post-cloacopexy, the bird's coelomic cavity was explored because of a recent history of anorexia, regurgitation, elevated creatine kinase, hyperuricemia, and decreased intracoelomic detail on screening radiographs. Midline celiotomy revealed yellow serous fluid throughout the coelom, a 2–3 cm section of colon trapped between the cloaca and body wall and adhesions between the colon and cloacopexy site. Adhesions were removed revealing 2 mm colonic and cloacal tears, which were repaired with 4-0'PDS in a simple interrupted inverting pattern. Cloacopexy sutures were removed to further free the entrapped colon. The bird passed feces the next day but died 3 days post-surgery. Upon necropsy, an

adhesion incorporating the cloaca, colon, and body wall at the level of the caudal margin of the keel blocking the passage of fecal material was found. The gastrointestinal tract was distended, with greenish fluid proximal to the adhesion. The authors noted that this and another bird (sulfur-crested cockatoo - *Cacatua galerita*) had a segment of large intestine trapped in the potential space between the cloacopexy sites and ventral body wall ultimately leading to the death of both [53].

A 2 cm diameter cloacalith was found and subsequently removed from within the coprodeum of a 4-year-old blue-fronted Amazon parrot (*Amazona aestiva*) [54]. The parrot was evaluated for acute onset of respiratory noises and straining. A cloacal mass was palpable on physical examination and saline infusion cloacoscopy was used to visualize the mass. The cloacolith was fragmented using 3-Fr biopsy forceps and larger pieces lavaged out. The remaining small pieces of the cloacolith passed shortly after recovery from anesthesia. Stone analysis revealed that the cloacolith was composed of 100% urates. The bird was found to be normal at 1 week and 9 months post-surgery. The cause of the cloacolith was not determined [54].

An infiltrative lipoma of the cloacal serosa was successfully removed from a 14-year-old blue-crowned conure (*Aratinga acuticaudata*) with a 3 week history of straining and vocalizing during defecation [55]. Physical examination revealed a 2.5 cm soft tissue swelling on the mid-caudoventral coelom. Ventral midline celiotomy was used to identify a subcutaneous soft tissue mass extending through the body wall musculature into the coloem and adhered to the cloacal serosa. The mass was causing the cloaca to deviate caudally and ventrally. The mass was removed via blunt dissection without penetrating the cloaca. Histopathologic evaluation determined the mass was an infiltrative lipoma with adipose tissue at the surgical margins. The bird was clinically normal with no evidence of tumor recurrence 1 and 7 months post-surgery [55].

Ventplasty

Ventplasty is reserved for chronic cloacal prolapse. In the author's experience, chronic cloacal prolapses are most commonly associated with prolonged egg laying. However, intestinal parasites, chronic masturbation (ducks), coelomic masses, and more may all result in a cloacal prolapse. The cause of the cloacal prolapse should be determined and resolved if possible. If the prolapse is chronic, the cloacal muscles and supporting structures may be permanently stretched and non-functional. The

goal of ventplasty is to reduce the vent size such that cloacal prolapse does not recur. It should be understood that ventplasty will likely fail if the underlying cause of the prolapse is not resolved and the bird continues to strain post-operatively.

The extent of the dilated vent determines how much tissue must be resected. For mild to moderate distension, usually one section of the vent is resected. For more severe distension, two areas of vent resection may be required. The basic incision is the same, but one versus two resections is based on surgeon preference in relation to the animal's needs. Pre- and post-operative antibiotics should be considered, based on culture and sensitivity results of a cloacal swab or cloacal tissue culture.

Prior to making the incision(s), estimate how much tissue needs to be resected in order to make a normal vent diameter. Triangular incisions work best with the "base" of the triangle on the leading edge of the vent and the "point" away from the vent. A single incision works best over the cranial ventral side of the vent, while two opposing incisions can be performed at the right and left lateral sides.

Once the resection site(s) is(are) determined, excise the desired triangular area(s) taking epidermis and dermis. Save excised tissue in formalin if needed. If the sphincter and transverse cloacal muscles are visible, spare these muscles. The dermis can usually be bluntly resected from the underlying muscular and submucosal tissue layers. When apposed, the new epidermal edges should form the desired vent diameter. If needed, more epidermal/dermal tissue is removed.

With the appropriate "new" vent margins, close the surgery site. First close the submucosa with the dermis. Place simple interrupted absorbable sutures medial (which represents the new vent wall) to lateral for all tissue layers. Next, close the dermis in the same fashion. Finally, the overlying epidermis is closed. The distal cloacal mucosa should extend distally to the vent epithelial margins without additional measures. If not, simply suture the mucosa in place as needed. The end result should be one suture line extending cranially (single vent resection) or one suture line extending laterally on the left and right sides of the vent (double vent resection). The new vent diameter should be just large enough to allow passage of droppings. Use lubricated cotton-tipped applicators to test the patency of the vent. Sutures are absorbable but can be removed in 2 weeks if needed.

If the patient is female, egg laying must be controlled either via a salpingohysterecomy, behaviorally, and/or chemically. Otherwise dystocia or rupture of the ventplasty sutures may result.

The liver

Liver surgery is generally limited to partial hepatectomy to remove solitary masses and liver biopsy. Numerous non-invasive diagnostics, such as serum biochemistries, radiographs, high detail CT, and MRI, can be performed to help determine if liver surgery is necessary.

Liver biopsy is a fairly common procedure and is very useful in determining hepatic pathology. Liver biopsy is obviously indicated when hepatic disease is suspected, but is also used to evaluate environmental toxins and in determining response to therapy. A thrombocyte estimate and capillary clot time (<5 minutes is normal) can be performed prior to surgery [56]. With that stated, avian platelets can only be estimated as they tend to clump in birds [57]. If a coagulopathy is suspected, give vitamin K_1 (0.2–2.5 mg/kg IM) 24–48 hours pre-operatively [57]. If ascites is present, as much fluid as possible should be drained via coelomocentesis prior to surgery.

Minimally invasive endoscopic and ultrasound-guided and blind percutaneous biopsies are also described [56,58,59]. One study showed that ultrasound guided liver biopsies resulted in 96.7% and 63.3% recovery of hepatic tissue in pigeons and quail (*Corturnix coturnix*) respectively [57]. While only a small amount of liver tissue was recovered using a tru-cut biopsy needle and biopsy aid in the study, the authors noted the sample size was sufficient for histopathological evaluation. In the study, one of 19 quails died under anesthesia as a result of hemopericardium. While no pigeons died during the procedure, 6 of 15 necropsied pigeons (40%) had right liver lobe hematomas 1 week post-surgery. The authors concluded that "ultrasound guided liver biopsy without a biopsy aid [such as endoscopy] is too risky considering the size of the avian liver." [57]

A cranial ventral midline coelomic (just caudal to the sternum) approach works well for most hepatic surgeries and is the author's preferred method over endoscopic liver sampling. Incise through the mid-line skin and linea alba to gain access to the cranial coelom and ventral hepatic peritoneal cavities. With hepatomegaly, the liver is readily visible and the right lobe is usually larger. With microhepatica, the liver is tucked under the sternum. Use cup-end biopsy forceps or curved hemostat to collect as large a piece of liver as possible without undue risk of hemorrhage. As an example, 3 X 10 mm liver biopsies (mean biopsy sample was 62.4 mg) were safely collected from 36 captive and 157 free-ranging harlequin ducks (*Histrionicus histrionicus*) [60]. In another study, 0.5 g and 1.2 g (6% and 18% hepatectomies respectively) liver biopsies were safely collected from a total of 16 galahs (*Eolophus roseicapillus*) [61].

Typically, the edge of the liver is biopsied using either instrument, while the cup-end forceps are more appropriate for selecting specific lesions and with microhepatica. When biopsying the liver's edge, bleeding is often minimal and sutures are rarely required. If hemorrhage is persistent, use hemostats to clamp on the bleeding area until hemostasis is established. Absorbable gelatin foam (Gelfoam®, Pharmacia and Upjohn Company, Kalamazoo, Michigan) may also be placed along the cut liver's edge to further reduce bleeding. If possible, collect extra tissue for culture and electron microscopy. Close the muscle and skin layers as with other coelomic surgeries.

Although complications such as uncontrolled hemorrhage, perforation of intestines and other underlying organs, and introduction of ascitic fluid into the air sacs are reported, these problems are fairly uncommon with the coelomic approach discussed above [56]. Even with severe liver disease, complications such as clinically evident coagulopathies are uncommon in the author's experience.

Selected laboratory values will likely change following a liver biopsy. In pigeons and quails undergoing ultrasound-guided tru-cut liver biopsies, aspartate aminotransferase (AST), creatinine kinase (CK), lactate dehydrogenase (LDH), alkaline phosphatase (ALP), total protein (TP), and albumin were measured before and 1 week after surgery. In pigeons, the AST and albumin both significantly increased post-surgically while only AST increased in the quails [57]. In a study of mixed wild raptors, "liver and kidney" values increased within 5 days after liver biopsy [62]. With the exception of mildly elevated alanine aminotransferase immediately following (18% liver weight) biopsies, galahs that underwent 6% and 18% hepatectomies had normal serum bile acids and elevated AST, CK, and ALP values that were statistically no different from sham operated birds immediately following, 4 and 7 days post-surgery. This last report suggests that these "liver enzymes" elevated as a result of celiotomy and not liver trauma [61].

In chickens, if both bile ducts are ligated, severe fibrosing cholehepatitis results within 28 days [63]. The typical lesions that result from extrahepatic bile duct ligation in poultry include cholestasis, fibrosis, proliferated biliary ductules, and increased Ito (fat storing) cells within the liver [64]. While not jaundiced, chickens with both bile ducts ligated also developed intensely yellow-stained droppings 6–7 days post-surgery [63]. Bile duct ligation results in jaundiced skin, diarrhea,

low serum testosterone, and atrophic and sclerotic testes 10 weeks post-surgery in 1-year-old chickens likely as a result of the hepatic fibrosis and obstructive cholestasis [65].

The left and right bile ducts are located on the caudal visceral surface of the respective liver lobe and typically unite on the right hepatic lobe then branch (hepatocystic duct) to enter the gall bladder (if present) or the duodenum (common hepatoenteric duct) [66]. Bile ducts are easily avoided during liver biopsy but should be considered a potential issue with extensive hepatectomies, cholecystectomy, distorted anatomy (especially with neoplasia), and proximal duodenal surgery.

In one study involving eight Pekin ducks infected with duck hepatitis B virus that underwent serial surgical liver biopsies at 4–5 week intervals (34 surgical procedures total), there was only one perioperative death, with no evidence of wound complications or intra-abdominal sepsis [67]. Seven of 157 (4.5%) free-ranging, and 0 of 36 captive, harlequin ducks died during recovery from anesthesia following liver biopsy and radio transmitter implantation. It was determined that none of the deaths were attributable to the liver biopsies [60]. With a little experience, surgical liver biopsies can be easily and safely performed in poultry, waterfowl, and other birds.

The pancreas

Birds, and their pancreas, seem to tolerate pancreatic surgery well. Following 99% pancreatectomy in chickens, the splenic pancreatic lobe undergoes a rapid enlargement (400% increase) over 16 days [68]. Partially depancreatomized chickens, with splenic lobe intact, also seem to maintain metabolic parameters remarkably well, although a post-surgical transitory hyperglycemia may be noted. One conclusion drawn is that the avian splenic lobe appears to be "extremely competent following removal of the major avian pancreatic lobes in adjusting to the demands placed on it for adequate nutrient absorption and distribution." [68]

Total pancreatectomy is fatal, but subtotal pancreatectomy (leaving the splenic lobe intact) results in transient "diabetes" that resolves in 12 days in Peking ducks [69]. Based on the author's experience and published studies, pancreatic biopsies and partial debulking is well tolerated in birds.

Pancreatic duct ligation results in severe damage to the pancreas [70]. Most of the pancreas lies within the duodenal loop and has 1–3 draining ducts that enter the terminal duodenum in close proximity to the bile and hepatic ducts. The potential complications of bile duct ligation are listed above. Pancreatic duct ligation results in atrophic pancreatic acini and interstitial fibrosis in chicks. Pancreatic duct obstruction has been a proposed cause of stunting syndrome in chickens [70].

A high grade pancreatic exocrine adenocarcinoma was removed from a 5-year-old male cockatiel (*Nymphicus hollandicus*) via celiotomy [71]. The report describes a "large, firm, white multinodular pedunculated mass (2.5 cm in diameter) that originated between the distal portion of the pancreas and ascending loop of the duodenum." The authors also reported they removed the distal tip of the pancreas adjacent to the mass at the same time. Neoplastic cells were surgically evident at the biopsy margins. Six weeks after surgery, the bird was recovering well and celecoxib (10 mg/kg PO SID) was administered for 3 months. One hundred and forty-two days post-surgery the bird presented with dyspnea and died during diagnostic sample collection. The bird had diffuse metastatic pancreatic adenocarcinoma. Of note, the bird had acute diffuse renal tubular necrosis (possibly as a result of the celecoxib) [71].

Pancreatic biopsy

Pancreatic biopsy is indicated when pancreatic disease, such as pancreatitis and neoplasia, is suspected and accurate diagnosis is needed for individual case management (Figure 16.15). A cranial ventral midline approach is used similar as with liver biopsy. The dorsal and ventral pancreatic lobes rest between the ascending and descending duodenal loop. The duodenum is located to the right of midline and is often
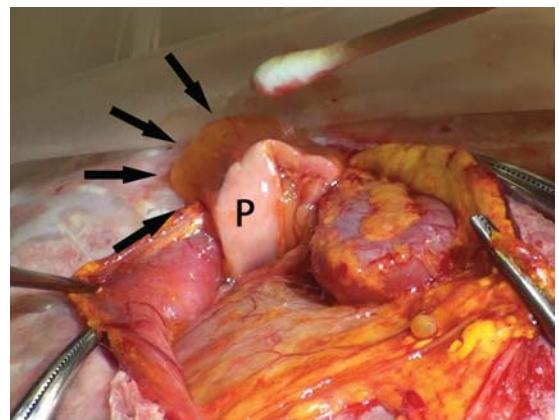
Figure 16.15 Diseases of the pancreas (P), such as these benign cysts (outlined by arrows) in a Pekin duck (*Anas platyrhynchos*) with sterile coelomitis, may require biopsy or partial pancreatectomy.

covered by a thin coelomic membrane. Incise through the thin membrane and gently retract the duodenal loop. After examining the pancreas and duodenum for gross abnormalities, select the distal (free) end of the dorsal pancreatic lobe (unless another site is clearly abnormal). Using hemostats, clamp the pancreas just distal to its distal-most vessel coming off the duodenum. Remove the distal pancreatic fragment and submit for histopathologic evaluation. Usually, a 3–8 mm section of pancreas is harvested. Remove the hemostats, but re-apply if bleeding occurs. Sutures to control hemostasis are rarely indicated. Close the coelom in standard fashion.

The urinary tract

Renal surgery

Because of the dorsal coelomic location within the renal fossae and complex vascular system, kidney surgery is often limited to focal procedures such as biopsy and superficial mass removal. The close associations with the lumbar and sacral plexuses and extensive vascular network surrounding the kidneys lead to the high probability of significant hemorrhage and possible neurologic damage expected during surgery.

Most cases of avian renal disease can be managed conservatively. However, some require additional diagnostics to better guide diagnosis and therapy. The only means to definitively diagnose avian renal disease and specific pathologic patterns is with a kidney biopsy and histopathologic evaluation. A renal biopsy is most frequently performed during endoscopic examination of the coelomic cavity and specifically, kidneys. However, a renal biopsy can be easily performed using 5-French cup biopsy forceps during exploratory coeliotomy.

Using a left lateral paralumbar fossa (most common with endoscopy) or ventral midline approach (celiotomy), the kidney is identified dorsal to the abdominal air sac. A right lateral approach may be used if disease is suspected to be limited to that side. The kidney is visualized and examined as much as possible. A small incision is made through the abdominal air sac and any other overlying membranes to expose the serosal surface of the kidney. Generally one to three 5-French cup biopsy samples are collected from the cranial renal division in an effort to avoid the large vessels coursing through and around the kidneys. The middle renal division may also be biopsied especially if the cranial division is atrophied or covered by vascular tissue (such as with an active ovary or neoplasia) (Figure 16.16). Once the tissue is collected, the site
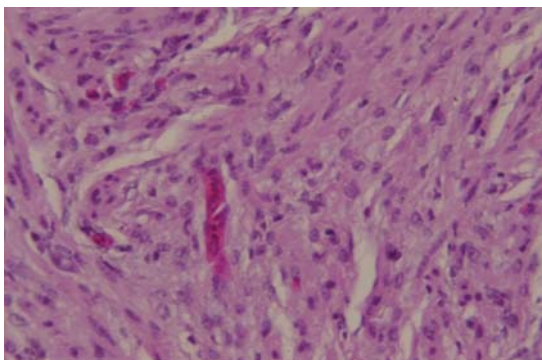
Figure 16.16 Renal neoplasia, such as this hematoxylin and eosin stained histopathologic section of an undifferentiated sarcoma in a Toulouse goose (*Anser anser domesticus*), may significantly alter the appearance of renal tissue during exploratory surgery.

is monitored for excessive bleeding. Direct pressure using a cotton tip applicator or hemostatic agent (such as Gelfoam) may be used if needed. Kidney biopsy samples are immediately placed in formalin and body wall closure is standard.

In a study of 89 free-living birds of prey, 126 endoscopic renal biopsy samples (2 biopsies from 37 birds) using 1.8 mm biopsy cup forceps were taken [72]. Post-biopsy hemorrhage averaged 67 seconds (10–172 seconds). The average biopsy was 2.2 mm long, 1.3 wide, and 1.0 mm deep. All samples contained proximal and distal tubuli and 1–89 glomeruli, with most having 25–29 gomeruli per histologic slide. Of 126 samples, 113 could be evaluated well or very well. Sixty-six samples revealed lesions including subcapsular bleeding (19/66), inflammation (16/66), cell casts (12/66), periodic acid Schiff positive reactions (8/66), and protein casts (6/66). Correlation between endoscopically visible change and histologic disease was 76.1% (96/126). The cranial division was considered the best site to collect biopsy samples because of its size and visibility. The authors noted it was possible to obtain specimens from the middle and caudal renal divisions in larger birds [72].

A separate study examined the effects of intramuscular meloxicam on kidney tissue in Japanese quail (*Coturnix japonica*). Fifteen birds underwent 5-french endoscopic biopsy cup biopsies from two sites in the cranial division of the left kidney with minimal complications [73]. In the author's experience, one to three 3- or 5-French cup biopsies can be safely collected from the cranial and/or middle renal divisions in birds.

Renal histologic lesions are rarely pathognomonic for a specific disease process, as many different diseases

cause similar kidney changes. The author encourages veterinarians to work with a pathologist who is familiar with normal and abnormal avian histology. Often, it is the pathologist's interpretation of a renal biopsy combined with the attending veterinarian's case familiarity that enables both parties to make a definitive diagnosis or build a reasonable differential diagnoses list compatible with the kidney lesions noted.

Urolithiasis and ureteral obstructive disease

Urolithiasis refers to the "formation of large urate 'stones' in the ureters," is primarily seen in pullets and caged laying hens and can result in increased mortality and decreased egg production [74]. Urolithiasis appears to be a primarily poultry disorder but has rarely been described in other avian species.

Birds that are affected with uroliths may show no or vague clinical signs. In the author's experience, many are initially suspected radiographically by finding small radio-dense objects in the dorsal caudal coelom with no associated physical or other laboratory abnormalities. Some birds may exhibit excessive straining when producing a dropping. This is also consistent with egg laying, diarrhea, intra-coelomic masses, and more. A contrast pyelogram may be used to further support ureteral obstruction. Ultimately, the stone needs to be visualized with endoscopy or celiotomy or, less commonly, with advanced high resolution CT.

Common intracoelomic findings include a dilated ureter obstructed with one or more urate stones (that may be visible on radiographs), atrophic ispsilateral renal tissue, and a normal to hypertrophic (compensatory) contralateral kidney. Renal histologic lesions noted with urolithiasis have included glomerular nephritis, tubular nephrosis, ureteritis, and interstitial mononuclear infiltrates. In birds, ureteral obstruction (as may occur with ureteroliths, cloacal masses, urodeal fold thickening, etc.) may cause a post-obstructive form of renal disease.

Based on studies in chickens, it may take significant renal loss before uric acid levels become persistently elevated. Uric acid values were elevated within 1–2 days of ligating one ureter at its junction with the cloaca and the other, along with caudal renal vein occlusion, at the midpoint of the opposite kidney in chickens. However, the uric acid values returned to normal within 12–14 days after surgery, which was attributed to the hypertrophy of the unobstructed remaining kidney tissue [75]. With the exception of a small island of tissue adjacent to the left adrenal gland, both kidneys atrophied significantly cranial to the ligated ureters. In studied chickens,

the total kidney weights of birds with urolithiasis do not differ significantly to those without uroliths [75]. This demonstrates the tremendous compensatory capacity of the healthy remaining kidney tissue and may explain why urolithiasis-affected birds seem otherwise normal.

Simple ligation of a bird's ureter results in ipsilateral renal atrophy and this result is similarly expected with urolithiasis. Naturally occurring ureteroliths in chickens are known to contain uric acid, urates, calcium, and ammonia.

The cause of urolithiasis in poultry flocks is not completely understood. However, coronavirus-associated nephritis in pheasants can induce interstitial nephritis, ureteral impaction, tubular dilatation, and subsequent visceral gout. In addition to infectious bronchitis virus infection (IBV, a coronavirus), other proposed causes of urolithiasis in poultry include water deprivation, excess dietary calcium, and nutritional electrolyte imbalances. By adding additional phosphorous, changing the form of calcium from small particle size to flakes and modifying the IBV vaccination protocol, investigators have been able to significantly reduce the incidence of urolithiasis in a previously affected layer flock. However, it has not been determined which management change results in the beneficial effect.

Treatment of urolithiasis in birds is rare. A 21-year-old male double-yellow headed Amazon parrot (*Amazona ochracephala*) with a history of lifelong straining to void and chronic intermittent vomiting for a "few years" was diagnosed with septic ureterolithiasis [76]. Dorsocaudal coelomic radio-dense opacities were found on screening whole body radiographs. Urolithiasis was diagnosed via exploratory coeliotomy. Multiple surgeries were required to remove the stones. A kidney biopsy was not collected and a relationship to renal disease could not be made. The ureteroliths were composed of "monosodium uric acid crystals and proteinaceous material mixed randomly or forming irregular laminae." Although the bird had dry flaky skin, a urate pasted vent, dull feathers, and heterophilic (28,840 cells/µl) leukocytosis (32,000 cells/µl), the authors concluded that the clinical signs associated with ureterolithiasis in this bird were non-specific and may result in delayed diagnosis with other birds. The cause was not determined [76].

For those birds in which the urolithiasis appears to be causing pain, renal compromise or other associated problems, surgical removal may be the best option. While lithotripsy has been reported to manage renal stones in a Magellanic penguin (*Spheniscus magellanicus*), there are no other such reports for managing uroliths in birds [77].

The patient is placed in dorsal recumbency and a ventral midline incision is made. The intestines are gently moved medially or laterally in effort to visualize both kidneys. The ureters are located on the ventral medial surface of the kidneys. Affected ureters are often significantly dilated, making visualization easier. If the ureters are clear, the stone may be visualized. Otherwise, follow the dilated ureter distally until the obstruction can be felt or seen. A small and precise longitudinal incision is made on the ventral surface of the dilated ureter over the obstructed area. Remove the stone(s) and any debris present. Using a lacrimal duct cannula, small ball-tipped or standard red rubber feeding tube, or an appropriately sized IV catheter without the stylet, flush the ureter proximally and distally to ensure patency. Close the ureterotomy site with fine (5-0' to 8-0') absorbable suture material in a simple interrupted pattern being careful to minimally reduce the lumen size. Any fluid and debris leaked from the surgery site should be removed prior to closure. Coelomic closure is routine.

Female reproductive tract surgery

Reproductive tract disease is very common in poultry and waterfowl - especially in female birds. These domesticated birds have been selectively bred, in many cases, over centuries and have a high reproductive drive. This translates into prolonged egg laying seasons and complications resulting from this physically and energetically demanding process. In addition, the physiologic changes associated with egg laying (such as weight gain, medullary hyperostosis, etc.) can result in unwanted consequences (fatty liver disease, fractures, etc.) when the process becomes more continuous with shortened rest periods.

Emergency surgery of the avian reproductive tract is rarely indicated. However, pre-surgical conditioning is recommended for all stable birds with reproductive tract diseases to improve the chance of a positive surgical outcome. To correct obesity and any nutritional imbalances, and possibly reduce reproductive drive, the diet is modified to reduce caloric intake and increase foraging opportunities. For pet poultry and waterfowl, this often means reducing diets high in simple sugars (such as corn and flour-based foods and treats) and allowing the birds to forage for food naturally outside (if possible). Caloric restriction may also be considered, especially if the hen is overweight. Pre-surgical conditioning may occur over several weeks to months and depends on the problem(s) and health status of the bird.

The drive to produce eggs is very strong in poultry and domestic waterfowl (especially ducks). However, attempts are still made to reduce reproductive stimulation in hens with reproductive disease. With poultry, affected hens may need to be removed from the presence of a rooster and separated from other actively laying hens until the reproductive disease is resolved. If possible, place the hen with other non-cycling birds or non-predatory animals. With waterfowl, the problem is more commonly associated with owners petting, stroking, and cuddling the pet. This activity should be discouraged. However, the owner can still interact with the bird - just with minimal handling. Separating a waterfowl hen from her bonded mate may result in significant stress. Unmated ducks, which are being kept with other egg laying ducks, may be placed with non-cycling birds or non-predatory animals as with chickens. Decreasing the photoperiod to 8–10 hours of light a day may be beneficial. As noted above, getting hens engaged in other activities such as foraging, swimming (waterfowl), and trick training (best with ducks) can help minimize reproductive stimulation (Figure 16.17a,b,c,d,e,f,g).

Leuprolide acetate (Lupron Depot, TAP Pharmaceuticals, Inc., Deerfield, IL, USA) has been used clinically to suppress reproductive activity in many birds. Lueprolide acetate depot is a long-acting GnRH analog that (in women) results in an initial stimulation followed by prolonged suppression of pituitary gonadotropins. Repeated monthly injections are intended to result in receptor down-regulation and decreased secretion of gonadal steroid hormones. From the author's experiences and reported information, recommended doses vary but have been safely used from 100 µg/kg up to 1000 µg/kg IM q 14–28 days to help suppress reproductive activity in birds. Attia *et al.* showed that a single IM injection of leuprolide acetate, providing 10 µg/kg BW per day for 30 days in broiler hens, caused a marked reduction in egg production [78]. The authors also reported a linear decline in oviduct, but not ovary, weight with an increasing dose of leuprolide acetate [78].

When extrapolated to other bird species, these findings suggest that leuprolide acetate may decrease egg production and have value to decrease oviduct size in preparation for salpingohysterectomy. Per the author's experience, some reproductively active female waterfowl and poultry can override the effects of leuprolide acetate with continued stimulation. For this reason, GnRH agonists are rarely used as a sole "treatment" to stop egg laying or prepare for surgery. Behavior and dietary modification are often combined with GnRH

Figure 16.17 A mixed breed domestic duck (*Anas platyrhynchos*) is clicker trained to perform productive, and discourage reproductive, behaviors. The duck twirls while following the owner's clicker (right hand). Then the duck continues to follow the clicker while walking 2 planks of wood. Finally, the duck is rewarded with a treat. Clicker training and numerous other methods can be used to allow owners to positively interact with their birds and encourage productive activity without reinforcing reproductive behaviors.

agonists to prepare reproductively active birds for surgery.

Deslorelin (Suprelorin®, Peptech Animal Health/ Virbac, Australia) implants have also been recommended for the same purpose but are typically implanted every 3–12 months as needed. Noonan *et al.* studied the effects of 4.7 mg and 9.4 mg deslorelin subcutaneous implants (versus placebo) in 2-year-old egg laying chickens over a 1 year period of time [79]. One hundred percent of deslorelin-implanted birds stopped laying eggs and had an ultrasound-determined "inactive ovary" by 2 weeks post-implantation. All placebo birds continued egg laying. Egg laying in the deslorelin groups was suppressed for a mean of 180 days (range 125–237) and 319 days (range 229–357 [with two birds still suppressed beyond this time]) with the 4.7 mg and 9.4 mg implants respectively [79].

In contrast to chickens, Japanese quail decreased egg laying for only 70 days when given 4.7 mg deslorelin acetate implants [80]. Additionally, of the 10 experimental group quail, only six ceased egg production. The other four birds, and the control group animals, continued to lay throughout the 180-day study. Interestingly, several of the experimental birds laid eggs with atypical color patterns 2 days after receiving the implant [80]. Although only representing two species, these studies demonstrate the potential wide variation of effects of deslorelin acetate on egg laying suppression in poultry species.

Both of these GnRH agonists, over time, pose significant expenditure outlay, and should not be considered a "first choice" maintenance treatment modality for controlling reproductive activity, unless all environmental, nutritional, and behavioral factors involved have been evaluated and deficits addressed. The author uses leuprolide acetate or deslorelin acetate as a means to help "condition" the bird in preparation for surgery. The goal of GnRH agonist use is to help reduce reproductive activity and subsequently reproductive organ size and vasculature.

The author strongly feels that environmental, dietary, and behavioral modifications are often needed for long term successful management of reproductive tract diseases in hens - even after surgery. Occasionally, long term GnRH agonist use is needed, especially if the owners are non-compliant with other recommended modifications or it is used as a form of chemotherapy for some reproductive tract neoplasia (not well founded at the time of writing). Even if the oviduct and most of the ovary are surgically removed, the reproductive drive remains high in domestic poultry and waterfowl. Continued stimulation can result in internal ovulation and

Figure 16.18   The proximal most portion of the oviduct, or infundibulum featured in this chicken during celiotomy, serves to engulf or "catch" the mature follicle as it is released from the ovary.

other problems. These issues should be discussed with owners prior to considering surgery.

Anatomy of the avian oviduct

The oviduct, or salpinx, develops from the left Mullerian duct and can be divided into 5 regions. The cranial-most region is the infundibulum, which is the site of fertilization and engulfs the ovulated ovum (Figure 16.18). The ovum next moves into the largest region, the magnum, which produces albumin that surrounds the developing egg. The inner and outer shell membranes are then formed in the isthmus. The egg is then "plumped" with water and solutes, calcified to form a shell and pigments deposited during the prolonged stay in the shell gland or "uterus." The shell gland transfers the complete egg through the uterovaginal sphincter into the vagina. The uterovaginal area contains sperm-storage tubules allowing many species to store viable spermatozoa for prolonged periods of time (>21 days in turkey hens) [81]. The vagina terminates at the cloaca and coordinates with the shell gland to ultimately expel the egg.

In adults, there is normally a left ovary and oviduct as any embryological right tissue typically regresses. However, there are numerous reports of right ovaries and/or oviducts in poultry, birds of prey, and parrots. There is even a double oviduct line of Rhode Island Red chickens that commonly have right oviducts [82]. While some eggs may be produced in a right oviduct, these are rarely fully functional [82].

The oviduct is suspended within the coelomic cavity via a dorsal and ventral ligament. Blood is supplied to the oviduct by the cranial, middle, and caudal oviductal arteries running in the dorsal mesentery. Only generalizations can be made as the origins of each vessel vary between species. The cranial oviductal artery arises from the left cranial renal artery, aorta, or external iliac artery. The middle oviductal artery comes from the left ischiadic

artery or its branch, the medial renal artery. The caudal oviductal artery arises from the left internal iliac artery or the pudendal artery. The veins draining the cranial oviduct empty into the caudal vena cava (via the common iliac vein), while those draining the caudal oviduct enter the renal portal or hepatic systems.

Diseases of the oviduct

Oviductal disorders may be incidental findings or clinically relevant and are surgically addressed as needed. Birds with oviductal disease may present with non-specific clinical signs. The most commonly recognized abnormalities with oviductal disease are related to a space occupying coelomic mass, including compression of surrounding organs, coelomic distension, coelomitis, and ascites. Generally, abnormal oviductal tissue is removed at the time of exploratory surgery.

Common congenital defects that are recognized in birds include a right oviduct that ranges from rudimentary, discontinuous, and atretic up to full size and functional, of which many are cystic (Figure 16.19). The author has noted a direct correlation between the presence of right oviducts and non-specific reproductive tract problems in hens (including cystic ovarian follicles, excessive egg laying, etc.) However, the cause and effect, if any, of this relationship are not clear. Cystic oviductal tissue can be substantial, may be associated with cancer and is always removed by the author when identified. Persistent right oviducts typically have a limited blood supply but are removed in a similar fashion to the more normal left counterpart.

Ectopic ovulation occurs when the infundibulum fails to engulf an ovum or fails to retain the ovum because of oviductal rupture or reverse peristalsis. The ectopic ovum is often found in various stages of development from yolk to a shelled egg. Potential causes include infundibulum failure from oviductal fat, trauma or disease, exuberant reverse peristalsis,

Figure 16.19 A right oviduct from a chicken is exteriorized out the ventral midline incision during exploratory celiotomy.

and oviductal disease. Ectopic ovulation is thought to occur frequently and has been reported in 28.6% of necropsied birds from nine orders [83]. The author has also seen ectopic ovulation associated with a persistent right oviduct in several avian species. Ectopic ovulation of yolk commonly results in mild, self-resolving, sterile yolk coelomitis and requires no or minimal supportive therapy (fluid therapy, anti-inflammatories, etc.)

Partially and completely shelled ectopic eggs result when a developing egg enters the coelomic cavity through an oviductal rupture or via reverse peristalsis from oviductal or even cloacal disease. Any disruption in the oviduct function, such as cloacal or oviductal masses (including egg binding, impactions and neoplasia), salpingitis, cystic hyperplasia, and oddly shaped or large eggs, can potentially result in ectopic eggs. A large ectopic egg can cause a penguin-like stance in small birds and is often associated with ascites and varying degrees of depression. Ectopic eggs can often rest unnoticed within the coelom in larger hens. Diagnosis can often be suspected using radiography, ultrasonography, and sometimes endoscopy (depending on how much debris is in the coelom), but celiotomy is often required for definitive diagnosis. Ectopic eggs should always be considered when conservative therapy for egg binding fails. Partially and fully formed ectopic eggs should be surgically removed after stabilizing the patient and determining the underlying cause(s).

Severe sterile and life-threatening septic egg yolk coelomitis may result from ectopic ovulation or eggs, systemic sepsis, and oophoritis. Acute egg yolk coelomitis may result in significant depression, anorexia, ascites, and, rarely, respiratory distress and death. Depending on the degree of inflammation associated with egg yolk coelomitis, coelomic adhesions may result and may be found days to more than a year after the episode during coeliotomy. Coliforms such as *E. coli*, *Yersinia pseudotuberculosis* and *Staphylococcus* spp. are commonly identified in septic yolk coelomitis [84]. *Salmonella sp.* may also be found with septic oophoritis and should be considered with bacterial coelomitis. Coelomocentesis and cytologic fluid analysis and culture are used for definitive diagnosis. Treatment of severe egg yolk coelomitis, especially when associated with bacteria, includes aggressive supportive care, antimicrobials, identifying and resolving causative factors if possible, and occasionally may require coeliotomy to remove infected tissue.

Egg binding and dystocia

Egg binding and dystocia are commonly described problems in pet bird medicine. However, these are

uncommonly seen in poultry and waterfowl except with small birds. Oviposition is the expulsion of the egg from the oviduct and is conducted by vigorous contraction of the oviductal muscles and peristalsis of the vagina. Egg binding is simply defined as prolonged oviposition (egg is arrested in oviduct longer than normal for the given species), while dystocia implies that the developing egg is within the distal oviduct either obstructing the cloaca or prolapsed through the oviduct-cloacal opening. Dystocia is often more advanced than egg binding alone, has many potential causes, and is commonly associated with functional (malformed eggs, cloacal masses, and obesity), metabolic (calcium imbalance and nutritional deficiencies), environmental (temperature changes, lack of exercise, and other stressors), and hereditary diseases.

Most cases of egg binding and dystocia are managed medically and are discussed elsewhere. Surgical intervention (primarily exploratory celiotomy) is rarely required in poultry and waterfowl.

Oviduct cystic hyperplasia

Cystic oviductal hyperplasia or dilatation has been reported in many bird species, including poultry. Although little etiologic information is forwarded, cysts may occur secondary to improper formation of the oviduct. Hyperplastic oviducts are often thickened with white to beige masses and distended with brown or white mucoid fluid. Affected birds often show no signs (and the disease is discovered incidentally) or occasionally show signs typical of reproductive tract disease (especially if the oviduct is significantly enlarged). Antimicrobials may be tried if organisms are recovered from aspirated samples, otherwise salpingohysterectomy is indicated.

Oviduct impaction

An impacted oviduct is usually distended and simply contains caseated material and misshapen, ruptured, soft-shelled partially or fully formed eggs. Potential causes include excess mucin and albumin secretion secondary to inspissated egg material and cystic hyperplasia. Salpingitis is often found concomitantly, especially in older birds. Metritis, salpingitis, egg binding, dystocia, and neoplasia commonly precede oviductal impactions. Oviductal impactions are described in many bird species and, in the author's experience, are common in prolific egg layers (Figures 16.20a,b).

Typical of most reproductive tract diseases, vague clinical signs with or without coelomic swelling and ascites are common with oviductal impaction (Figures 16.21a,b). Affected birds may show persistent "broodiness" with recent cessation of egg laying. Definitive diagnosis is made at celiotomy or sometimes via ultrasound and endoscopy with aspiration of the oviductal contents. Chronic oviductal impactions may be found incidentally during exploratory celiotomy and are often associated with a history of sudden cessation of egg laying several months or years prior to presentation. Acute impactions may be treated by salpingotomy, culture and appropriate antibiotic use, and oviductal flushing, while severe or chronic diseases are best treated with salpingohysterectomy.

Oviduct prolapse

Powerful coelomic contractions combined with the process of oviposition can result in oviductal prolapse, which is often secondary to dystocia. A temporary prolapse is normal immediately after laying an egg (oviposition). Predisposing factors may include large or abnormally shaped eggs, general debilitation, malnutrition, systemic illness, disease of the oviduct, and, sometimes, normal egg laying. In turkeys that are selected for high meat yield, decreased vaginal collagen has been associated with uterine prolapse [85,86]. The uterus is most commonly prolapsed but the vagina and other portions of the oviduct may also prolapse. The cloaca, and rarely colon, may also prolapse and should be distinguished from the oviduct.

Because the exposed tissue can rapidly become devitalized and infected, aggressive treatment with warm saline flushes, antibiotics, and replacement of the prolapsed oviduct is warranted. If the prolapsed oviduct is edematous, topical dextrose, dimethyl sulfoxide (DMSO), and/or steroids may be needed to reduce the swelling. If an egg is present in the prolapsed or oviductal tissue, ovocentesis and implosion of the egg are often needed to reduce associated pressure and aid in egg shell removal. It is best to only aspirate the egg by inserting the needle directly through the shell and not through the oviductal tissue, which can easily tear and potentially lead to problems later. After stabilizing the bird, remove the egg medically if possible and replace the prolapsed tissue. Two transcloacal sutures may be required to prevent the immediate recurrence of prolapsed tissue.

Salpingohysterectomy is indicated when the oviduct is necrotic and/or the egg (or its fragmented shell) cannot be removed medically or pass on its own. If an oviductal torsion is present distal to the egg (within the oviduct), attempting to force deliver the egg often results in further damage. Oviductal torsion, neoplasia, adhesions, and other anatomic disorders should be
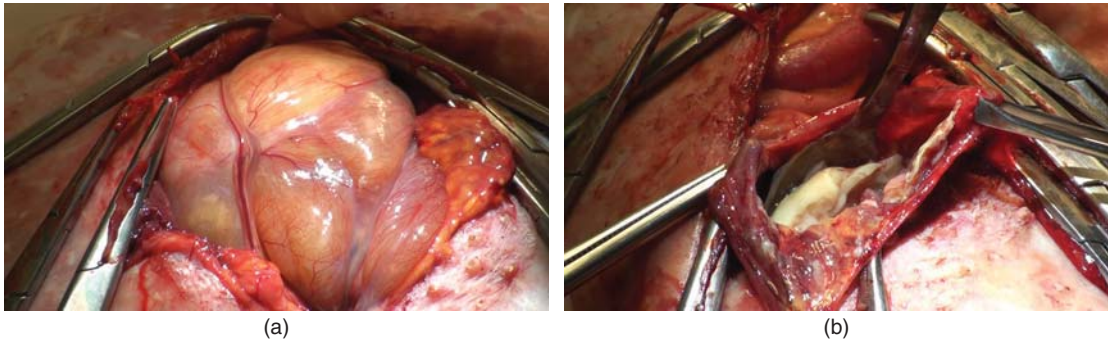
Figure 16.20 A Pekin duck (*Anas platyrhnchos*) has a severely impacted oviduct and sterile salpingitis. While the mass was hot to the touch, no organisms could be found on culture or cytologic and histopathologic evaluation. (a) The distended and vascular oviduct completely fills the ventral midline incision obscuring view of all other coelomic tissue. (b) Because it was too large to exteriorize, the oviduct was incised and fluid and debris were removed (a sterile spoon shown here is scooping out solid granulomatous debris) to better facilitate salpingohysterectomy.

Figure 16.21 (a) The caudal and ventral coelom of this mixed breed domestic duck (*Anas platyrhynchos*) is severely distended. (b) Once the feathers have been plucked and the site surgically prepared, the ventral coelom of this chicken is noticeably distended. Both birds had severe oviductal disease and represent the common but non-specific coelomic distension common with reproductive tract disorders.

considered if a bound egg cannot be delivered without forceful techniques, and surgical options should be pursued.

Oviduct torsion

Oviductal torsion has been infrequently reported in birds. Torsion of the oviduct may occur following a tear of the dorsal, and possibly ventral, oviductal ligament(s) or be associated with oviductal cysts [87,88]. In four parrots, all birds presented with signs of egg binding and/or general lethargy and had a history of previously laying "many eggs" prior to the oviductal torsion. One thin cockatiel presented with lethargy, depression, and coelomic distension and died despite emergency therapy. Of the three other parrots, two cockatiels were treated with salpingohysterectomy and one eclectus parrot (*Eclectus roratus vosmaeri*) was treated with a salpingotomy, egg removal, torsion correction, and subsequent closure of the oviductal ligament tear. All birds recovered uneventfully from surgery. The eclectus successfully laid normal clutches after surgery [88]. An 11-month-old chicken was found to have a 360 degree oviductal torsion and cyst twisted around the dorsal ligament on necropsy. Severe oviductal congestion, hyperemia, devitalization, and dilatation were noted [87].

Salpingitis and metritis

Salpingitis, inflammation of the oviduct, is common in birds (Figure 16.22). In poultry, salpingitis has been listed as the most prevalent form of reproductive tract disease [89]. *E. coli* infections are fairly common in poultry and can cause salpingitis, but *Streptococcus* sp.,

Figure 16.22 At surgery, bacterial salpingitis and an impacted oviduct was found in a chicken. The bacteria were observed microscopically but the organism was not cultured and speciated. Photograph courtesy of Dr. Cheryl Greenacre.

Figure 16.23 An oviductal mass (focal adenocarcinoma) is found on the magnum region of the oviduct in this chicken and is an indication for salpingohysterectomy.

Mycoplasma gallisepticum, *Acinetobacter* sp., *Corynebacterium* sp., *Salmonella* sp., and *Pasteurella multocida* have all been implicated from various species. Some ground-nesting species, such as Anseriformes and emus, may develop non-lactose fermenting, gram negative (*Pseudomonas aeruginosa*, *Proteus mirabilis*, *P. vulgaris*) salpingitis [90]. Non-infectious salpingitis can also be seen, especially with chronic sterile oviductal impactions, and is fairly common per the author's observations.

Metritis is inflammation within the shell gland portion of the oviduct and may result from or cause egg binding, chronic oviductal impaction and rupture, coelomitis, and septicemia. *Prosthogonimus ovatus* and other related trematodes (flukes) can inhabit the oviduct of poultry and waterfowl and result in salpingitis with heavy infestations [91]. Other infectious agents ascending from the vagina or cloaca or systemic infections can also cause salpingitis. Specifically in poultry, vent cannibalism has been implicated as a precursor to salpingitis [89] (see Chapter 13).

Birds with non-septic salpingitis or metritis often show vague signs of illness, while septic birds are usually clinically ill. Egg shell deformities and embryonic and neonatal infections are often secondary to metritis. Definitive diagnosis is made at celiotomy or endoscopy with aspiration of oviductal fluid for cytologic and microbiologic analysis or, if the oviduct has no liquid contents, biopsy with culture. Use antibiotics based on culture and sensitivity results. If trying to spare the oviduct, repeated endoscopic evaluation, direct and indirect oviductal flushing, and long-term antimicrobials are recommended. Salpingohysterectomy is indicated for most cases.
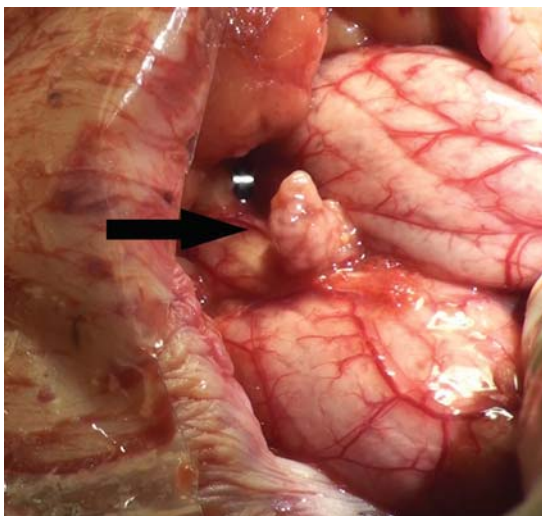
Salpingohysterectomy

Salpingohysterectomy is the surgical removal of the oviduct, infundibulum to uterus, and is indicated for chronic egg laying and any oviduct disease that cannot be medically managed (Figure 16.23). Every attempt should be made to understand the bird's overall health status prior to surgery, as the patient should ideally be stable. Although rare compared to sterile inflammation, birds with septic yolk peritonitis generally carry a poor prognosis. Patients with underlying health problems, such as various lung, liver, and kidney diseases, can also complicate surgery. Otherwise, healthy salpingohysterectomy candidates typically do well with the procedure.

Oviductal hypertrophy occurs secondary to elevated estrogen levels during sexual activity and can take up most of the left side of the intestinal-peritoneal portion of the coelomic cavity. This oviductal hypertrophy includes increased vascularity and risk of bleeding during surgery [92]. If the patient is stable, time permits, and increased reproductive tract vascularity is suspected, the author conditions the bird prior to surgery as described previously in this chapter.

In the author's experience, a left lateral approach offers the best exposure to the left female avian reproductive tract. However, a ventral midline approach is better for exploratory celiotomy - especially when the degree of coelomic disease and/or presence of right-sided reproductive tract components are unknown.

Perform a left lateral celiotomy. After incising through the left abdominal air sac, the ovary and oviduct are readily visible. Gently retract the cranial oviduct (infundibulum area) out of the incision and ligate using a surgical clip or cauterize suspensory ligament vessels as needed. The closer the bird is to laying, the larger the vessels present. Depending on the size, the cranial, middle, and/or caudal oviductal artery(ies) may need to be ligated with a surgical clip or cauterized. Once visualized, a surgical clip is placed at the base of the oviduct just proximal to its junction with the cloaca. Suture material can be used in larger poultry and waterfowl. Excise the oviduct.

When performing a ventral midline celiotomy, the air sacs do not need to be breached. A careful evaluation of the caudal coelom is made and right oviductal tissue, in addition to the more normal left, is identified and removed as described above. With a little more difficulty, right oviductal tissue can also be accessed from a left lateral approach.

Well-developed preovulatory follicles (F1 and F2 +/- F3 and F4) may pose a risk for intra-coelomic ovulation and can usually be easily removed. Use cotton tip applicators to rotate the follicle in one direction continuously until it separates from its pedicle (Figures 16.24a,b). This may require 15–30 full rotations until the follicle is free. Once free, simply remove the follicle. If concerned about a well-developed vascular pedicle, use surgical clips and then excise the follicle. One study in domestic chickens demonstrated a pause in laying that increased with the number of follicles removed compared to sham-operated hens [93].

Cystic follicles should either be aspirated (drained) or ideally removed. If a follicle is accidentally incised, yolk leaks into the coelom. Simply "mop up" excess yolk and other fluid if present. Collect culture and samples for histopathologic evaluation as needed.

Endoscopic salpingohysterectomy of juvenile cockatiels has been described and may potentially be applied to young poultry or waterfowl [94]. A left lateral coelomic endoscopic approach (left leg pulled caudally) was performed on 3–to–11-month-old cockatiels. Once visualized, the supporting ligament of the infundibulum was carefully pulled laterally toward the coelomic entry site using flexible endoscopic grasping forceps (Karl Storz Veterinary Endoscopy, Inc., Goleta, CA, USA). This action broke down the supporting structures (ventral and dorsal suspensory ligaments of the cranial oviduct and uterus) and separated the oviduct from the overlying kidney, caudal vena cava, and left ureter. Next, a cotton-tipped applicator was placed in the cloaca and was used to better visualize the cloacal-uteral junction and ensure the oviduct was "peeled" from the surrounding tissues. The oviduct was exteriorized and then crushed and cut with microsurgical forceps and scissors respectively, at the point of exit from the coelomic cavity, just cranial to the uterovaginal sphincter. The endoscope was replaced to check for hemorrhage and closure was routine [94].

Multiport endoscopic salpingohysterectomy has also been performed and studied in 14 white Carneau pigeons (*Columba livia*) [95]. Mean surgery time was 34 minutes. While the procedures were generally considered effective (complete removal of the oviduct) and safe, minor complications were noted. Mild damage or hematoma formation was found post-surgery in 28% of test subjects. Also, one bird had remnant distal oviductal tissue post-surgery. No ovarian follicles were removed during endosurgery. Ovarian activity, including pre-ovulatory follicles, was noted up to 90 days post-surgery in some of the birds. The authors reported

(a)   (b)
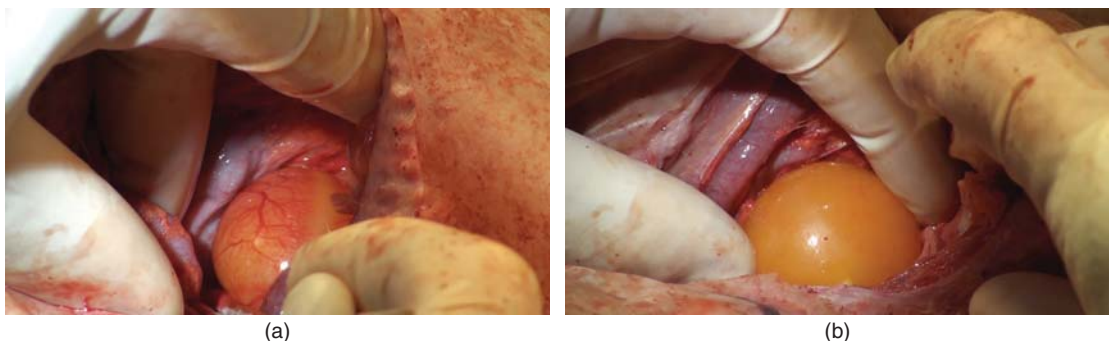
Figure 16.24 (a) An active follicle in this Pekin duck (*Anas platyrhynchos*) is being rotated with the aid of a cotton tipped applicator. (b) After 15–30 full rotations, the blood supply to the follicle diminishes (seen here) and the follicle can be simply pulled out with minimal risk of hemorrhage.

that standard salpingohysterectomy (via celiotomy) and endosurgical salpingohysterectomy can result in serious yolk coelomitis in ducks and quail, and chickens respectively [95]. Details of a "standard salpingohysterectomy" were not given. As explained above, the author recommends removing active ovarian follicles when performing salpingohysterectomy.

Endoscopic salpingohysterectomy has several distinct limitations and benefits. As indicated in the first study, this procedure was acceptable in the juvenile birds because of a poorly developed blood supply of the oviduct, and that if attempted in mature, egg producing cockatiels, it may result in fatal hemorrhage [94]. Additionally, this procedure required an endoscope and two surgeons. The author has worked with parrots that were endoscopically "salpingohysterectomized" only to find incomplete oviductal removal and subsequent active tissue remnants that resulted in various forms of reproductive-related coelomic disease. All birds required exploratory celiotomy to correct the problems.

On the positive side, a properly performed endoscopic procedure results in minimal hemorrhage, can be performed safely, and offers an option for juvenile salpingohysterectomy. Endoscopic salpingohysterectomy does not preclude behavioral and dietary management, as these birds can still internally ovulate and develop ovarian disease if reproductively stimulated. However, this is an option to consider for juvenile birds, especially domestic ducks, which have a high risk of chronic egg laying and no owner requirement for egg production.

Caesarian section and reproductive tract sparing

Caesarian section is indicated when the bird's reproductive capabilities need to be spared and is typically limited to egg binding with an otherwise normal, or minimally diseased, oviduct. Depending on the location of the egg, a caudal left lateral or ventral midline approach is used. The oviduct should be incised directly over the bound egg and away from prominent blood vessels. After removing the egg, inspect the oviduct for other abnormalities and collect biopsies and cultures as needed. Close the oviduct in a single simple interrupted or continuous layer using fine (4-0' or smaller) absorbable suture material. Coelomic closure is standard. The author recommends resting the hen from reproductive stimuli for at least 2–4 weeks as dictated by culture and/or histopathologic results.

Anatomy of the avian ovary

A right and left ovary and oviduct are present in the embryologic stages of all chicks, but the right half regresses as a result of the action of Mullerian inhibiting substance prior to hatching [81]. Although a persistent right oviduct with or without a functional right ovary is present in some birds, most birds only have a left female reproductive system. The brown kiwi is an exception and normally has a functional left and right ovary. About 480,000 oocytes develop by hatching in the chicken. Of these, about 2000 can be seen as a mass of small ova and approximately 500 reach maturity and ovulate within the lifespan of domestic species and even fewer mature in wild species. By 2 and a half years of age, chicken hens ovulate approximately 500 times, which is equivalent to a woman entering menopause [96]. Ovarian follicles are arranged hierarchically. The largest follicle (F1) ovulates on the next day, the second largest (F2) the following day, and so on (Figure 16.25).

The ovary is attached to the cranial renal division and dorsal body wall by the mesovarian ligament and receives its blood supply from the ovarian artery, which originates off the left cranial renal artery or directly off the aorta. Baumel notes that accessory ovarian arteries may also arise from other adjacent arteries [97]. The ovarian artery further divides into many branches, with the greatest blood flow directed to any large preovulatory follicles that are present. Ovarian veins unite into main anterior and posterior veins that drain into the overlying vena cava. As more specifically described by Baumel, multiple left ovarian veins may exist and drain into the cranial oviductal vein, which then enters the common iliac vein and finally the caudal vena cava [97].

The author has noted that the cranial oviductal vein is too short or poorly developed to recognize grossly. Instead, multiple short veins seem to enter the common

Figure 16.25 Active F1–F5 follicles were removed from a Pekin duck (*Anas platyrhynchos*) with salpingitis. The F1 follicle is the largest.

iliac vein over the length of its contact with the dorsum (base) of the ovary. This, in part, makes ovariectomy in adult birds difficult, as there is not a single artery and vein to ligate.

Surgery of avian ovary
Partial and "complete" ovariectomy

Ovariectomy in hens is a challenging and often high-risk procedure. Ovariectomy has been used in many poultry studies and mention of this procedure can be found throughout the literature [98–102]. Unfortunately, most papers poorly describe the specific details of ovariectomy or its complications. In one chicken study, it was noted that ovariectomized birds "lost considerably more blood than sham-operated hens." [102] Terada *et al*. described ovariectomy by "destroying ovarian tissue by local application of small pieces of dry ice." [101]

Although it has been stated that the short stalk of the cranial renal artery or proximity to the aorta are what make ovariectomy difficult, the author suggests that the intimate and lengthy attachment to the overlying common iliac vein is what makes this procedure risky [80,92]. As mentioned above, multiple small ovarian veins often connect directly into the common iliac vein. It is often venous, and not arterial, bleeding from a lacerated common iliac vein that usually causes life-threatening hemorrhage during ovariectomy. As with the oviduct, the ovary can dramatically change in size and vascularity with reproductive activity. As with salpingohysterectomy, the bird is ideally conditioned (described earlier) to reduce ovarian vascularity. Some diseases requiring ovariectomy do not allow attending clinicians the time in which to "condition" the avian patient prior to surgery.

Ovariectomy is reserved for ovarian diseases such as cancer, chronic recurring cysts, persistent follicular activity, oophoritis, and other diseases that cannot be managed medically and are life-threatening without further treatment. A true complete ovariectomy is very difficult to achieve in adult birds. Most "ovariectomies" are partial with the goal to debulk abnormal tissue.

A cranial left lateral celiotomy often provides the best exposure to the left ovary. However, a ventral midline approach can also be used successfully. It is important to clean the surgical field of fluid and debris to best visualize the ovary and its vasculature. Surrounding organs may need to be gently pushed aside using moistened cotton-tip applicators or other non-traumatic instruments.

The first step of ovariectomy is to debulk its mass. The goal of this first step is to be able to visualize the ovarian attachment to the overlying common iliac vein and any other vessels present. If the ovary is inactive or juvenile, very little debulking is needed. If present, remove large preovulatory follicles as discussed under "Salpingohysterectomy." Aspirate and drain any cystic follicles that are present being careful not to spill contents into the coelomic cavity, especially if there is concern of oophoritis. When aspirating follicles, guide a small gauge (23–25 g) needle into the most visibly avascular portion (stigma) and aspirate contents. Butterfly catheters are useful for this procedure. Using this aspiration technique, a significant amount of an active and/or cystic follicle can be removed improving visualization of and around the ovary. As a note, blood-filled follicles may represent previously ruptured blood vessels from an invasive mass and warrants caution when attempting debulking.

Once the fluid component is minimized, progressively clamp or surgically clip the ovarian mass closer to its dorsal base. When used properly, angled Debakey neonatal vascular clamps are atraumatic, rest in the surgical site without obstructing view, and seem to provide some hemostasis to the ovarian mass. Once a section of the mass is surgically clipped or clamped, surgically excise or cauterize and remove the ventral-most ovarian segment. Reassess the mass and move the clamp (or place new surgical clips) closer to the ovarian base and repeat the excision process. This process is repeated until the surrounding vasculature is identified and the course of the common iliac vein can be seen.

Once the mass has been debulked, several options exist for complete or partial ovariectomy. Altman reports using an electrocautery ball electrode to coagulate ovarian follicles in immature females [103]. The same procedure can result in ovarian regeneration and subsequent ovulatory activity in mature hens [103]. The author has noted that some juvenile bird ovaries can be gently "peeled" in toto from caudal to cranial off its dorsal attachments with no or minimal bleeding. In these cases, the caudal edge of the ovary is grasped with angled hemostats and pulled in a cranial direction with a clear separation, and minimal effort, from the dorsally located common iliac vein. If attempting this procedure, stop if any resistance is noted to prevent tearing the overlying vein.

Another technique with juvenile or sufficiently debulked ovaries is to place surgical clips in the potential space between the dorsal ovarian base and the common iliac vein. Gently lift the caudal pole of the ovary and place a small to medium surgical clip from caudal to cranial across the ovarian vascular supply. Although difficult without good exposure, a last surgical

clip can be placed from cranial to caudal in the same manner in an attempt to ligate the more cranially located ovarian artery. This is generally only possible via a cranial left lateral approach. With the blood supply adequately clamped, the ovary can be gently shaved off with precise radiosurgery using an Ellman B "loop" series or blade electrode (Ellman International, Inc., New York, NY, USA), precise cold excision or left to die without a blood supply. Altman describes this method as "a difficult, high-risk procedure" but the author has successfully performed ovariectomies in adult hens using this technique [103]. Obvious complications include hemorrhage when trying to remove the surgically clipped ovary and inadequate, blind placement of the surgical clips.

The author has used another approach when the ovarian attachment to the overlying common iliac vein is indistinguishable or there is erosion into the overlying vessel and the entire ovary must be removed for the bird's survival (as with otherwise untreatable cancer). Debulk the ovarian mass as described above. Once clearly identified, using a surgical clip, ligate the common iliac vein just caudal to the ovary and cranial to its junction with the caudal renal vein. Next, using a surgical clip, ligate the common iliac vein just cranial to the ovary and caudal to its junction with the caudal vena cava. If performed properly, the ovarian artery and common iliac veins are effectively clamped, allowing one to carefully dissect the entire ovary from the overlying vessel(s). If necessary, the ventral wall of the common iliac vessel can be safely removed. There is real potential of damaging the left adrenal gland, significantly altering blood flow through the renal portal system and the cranial renal division, and causing physical damage to the overlying kidney and lumbar and/or sacral nerve plexus(es). The author has noted that once the common iliac vein is ligated, the cranial renal division rapidly changes colors but returns to normal within a few minutes.

As has been shown in young chickens and Japanese quail, transplanted ovarian tissue (from other birds of the same species) may grow and become functional [104,105]. These studies were conducted in young (1-day-old chickens and 1-week-old quail) birds and transplantation was more often successful when immunosuppressive therapy was given [104,105]. However, these studies support the concern that dislodged ovarian tissue may remain viable, implant, and become functional if left in the coelom - especially as an autologous "transplant." While this statement has not been proven in adult birds, the author recommends

Figure 16.26 The oviduct and multiple active ovarian follicles were removed in a domestic duck (*Anas platyrhynchos*). Courtesy of Dr. Brian Speer.

removing any free ovarian tissue that becomes dislodged during surgery.

With all of the above described ovariectomy procedures, it should be understood that none have been satisfactorily studied in pet birds and that each carries a significant risk to the patient. Partial ovariectomy (at least active follicle removal) is commonly performed alongside salpingohysterectomy (Figure 16.26). With each procedure, closure is routine.

Diseases of the avian ovary
Cystic ovarian disease

Although the cause is often unknown, cystic ovarian disease has been reported in numerous bird species [90,106]. Cystic ovaries are sometimes secondary to neoplasia. Depending on their size, ovarian cysts may be found incidentally if small or may cause coelomic distension when large and/or numerous and can be associated with ascites. Large or numerous cysts can often be diagnosed non-invasively using ultrasound. Cysts can be treated by ultrasound-guided transcoelomic aspiration or more directly via celiotomy or endoscopy. If collected, evaluate the fluid for evidence of infection, neoplasia, or other abnormalities. Severe cystic disease

may require partial or complete ovariectomy and should include biopsy for histopathological evaluation. Leuprolide acetate has also been suggested to reduce or resolve ovarian cysts in birds. Deslorelin has also been recommended anecdotally. However, in the author's experience, aspiration or physical removal is the only means to remove ovarian cysts.

Oophoritis

Ovarian infections can be life-threatening and are often associated with septicemia especially in poultry. *Salmonella pullorum* is the etiologic agent of pullorum disease of poultry and most frequently affects the ovary [107]. Clinically affected birds usually show more severe, but non-specific, signs of illness and if not treated quickly septic coelomitis and death may result. Abnormally shaped or colored follicles that are identified during celiotomy or endoscopy should be either removed (celiotomy) or carefully aspirated for cytologic and microbiologic analysis and broad spectrum antibiotics should be initiated pending culture results. An injection needle with Teflon guide (Karl Storz Veterinary Endoscopy America, Goleta, CA) is particularly useful as an endoscopic means to aspirate ovarian follicles. If possible, completely drain the abscessed follicle(s) being careful not to contaminate the coelom. Partial or complete ovariectomy may be required for chronically infected and caseated follicles.

Reproductive tract neoplasia (ovary and oviduct)

Ovarian cancer is reported with some frequency in birds and can be associated with egg retention, ascites, cystic ovarian disease, medullary hyperostosis, coelomic hernias, oviductal impaction, and general malaise [90,108]. One study noted that 38% of USDA Inspection Service mature fowl condemnation is the result of neoplastic disease, most of which are from the genital tract. Fredrickson states "there is indeed a unique propensity for hens (poultry) to develop cancer of the reproductive system in the almost total absence of tumors at other sites." [109] Ansenberger *et al.* report that the incidence of ovarian cancer in 2.5–3.5-year-old hens is 4–20% [96]. Granulosa cell tumors and ovarian adenocarcinomas are most frequently reported but carcinomas, leiomyosarcomas/leiomyomas, adenomas, teratomas, dysgerminomas, fibrosarcomas, lipomas, and lymphomatosis have all been identified in bird ovaries [90,91,109,110]. Oviductal tumors are less common than ovarian neoplasia and include adenocarcinomas/adenomas, adenomatous hyperplasia, carcinoma, and carcinomatosis [108,110].

Granulosa cell tumors and possibly other reproductive tract neoplasms may be functional and cause increased plasma hormone levels [109].

Polyostotic (medullary) hyperostosis may also result in a paraneoplastic syndrome with functional ovarian and oviductal neoplasms [108,110]. Interestingly, one study found that hyperestrogenism did not cause polysostotic hyperostosis in several species of birds with various neoplastic and non-neoplastic reproductive tract diseases [111]. Nevertheless, the author has frequently observed significant medullary hyperostosis in many waterfowl and poultry with reproductive tract diseases including cancer.

Clinical signs of ovarian and oviductal cancer vary and are non-specific for most reproductive tract diseases, including coelomic swelling, dyspnea, ascites, poor or altered reproductive performance, and lethargy. If a mass compresses the overlying lumbar or sacral nerve plexus, lameness (usually left sided) may be seen. Diagnosis can be further supported using radiography, ultrasonography, CT, MRI, exploratory celiotomy, endoscopy, and biopsy. Once a definitive diagnosis is made, options for therapy include chemotherapy, radiation therapy, and partial or complete ovariectomy. All carry a guarded to poor prognosis unless the neoplastic tissue is completely removed.

Anatomy of the avian testicle

Avian male reproductive anatomy of most birds consists of three main gross structures: The testes, epididymis and ductus deferens. Some birds also possess a phallus (discussed below). The paired testes are located ventral to their respective left or right cranial renal division. The mesorchium connects the testes to the dorsal body wall. The left testicle is typically larger than the right in most young birds, but this relationship can change as the bird ages. In seasonal breeders, such as some passerines, the testes can increase 300–500 times in size and should not be interpreted as neoplasia. Large active testes can also be readily evident radiographically (Figure 16.27). In addition to size, the color of the testicles can also change with fluctuating hormone levels, ranging from black in the sexually immature or inactive cockatoos to white or yellow in the chicken.

The epididymis is located at the testicular hilus, or dorsomedial aspect of the testes. The ductus deferens continue from the epididymis as highly convoluted tubes running lateral to and alongside the ureters and then terminate at the urodeum as a papillae ventral to the ureteral ostium.

The testicular artery arises from the cranial renal artery and provides most of the arterial blood supply to the

Figure 16.27 Lateral radiographic view of a crested duck (*Anas platyrhynchos*) with normal but enlarged testes. The approximate silhouette of the superimposed left and right testes is outlined.

testes. An accessory testicular artery may arise directly from the aorta. The venous drainage is returned either directly to the caudal vena cava or forms a common stem with the adrenal veins. Kremer and Budras found that two testicular veins empty directly into the caudal vena cava of Pekin drakes (*Anas platyrhynchos*) [112]. Given the diversity within the class Aves, it is likely that multiple variations of the testicular vasculature exist.

Although most birds lack a copulatory organ, some birds possess a non-protrusible (Galliformes) or protrusible (ratites and Anseriformes) phallus. Domestic chickens and turkeys have a non-intromittent phallus, consisting of a median and two lateral phallic bodies, on the floor of the lip of the vent. Lymphatic flow through the phallic bodies and their laterally associated lymphatic folds result in tumescence. Because the lymphatic folds and lateral phallic bodies accumulate more fluid than the median body, the phallus everts during tumescence producing a groove for semen to travel. Semen is deposited when the phallus contacts the everted oviduct opening in the hen.

Surgery of the male avian reproductive system
Castration

Clinical avian castration is infrequently discussed, especially in comparison to salpingohysterectomy, suggesting that male reproductive tract diseases are relatively uncommon. Although caponization is common in the poultry industry (performed between 1–2 weeks or up to 6 weeks of age), routine castration is rare in pet birds. As a result, there is little information

regarding the behavior and physiologic altering effects of castration in pet birds.

It is known that caponized chickens (capons) have increased coelomic fat weight, total hepatic lipid content and saturated fatty acid percentage compared to intact birds [113]. The medical consequences, if any, of this body change are not known.

Castrated Gambel's (*Callipepla gambelii*) and scaled (*C. Squamata*) quail have reduced or eliminated courtship behaviors and lower rates of male-male threats. However, the castrates maintained ornate plumage, exhibited overt aggression and frequently won contests when actually engaged [114]. Yearling European starlings (*Sturnus vulgaris*) that were castrated when non-reproductively active were shown to be significantly more aggressive than non-castrated controls [115]. The authors concluded that "nonreproductive aggression in yearling male starlings is independent of gonadal sex steroids and suggests it even increases following castration." [115]

These limited results suggest that persistent "male" behaviors are either already learned at the time of castration, result from hormones other than testosterone or another source of testicular hormones is still present post-castration. It is known that some species have an appendix epididymis extending from the epididymis into the adrenal gland that may secrete androgens following castration. The author has performed castration in roosters in an effort to stop crowing. While castrated roosters did exhibit reduced crowing behavior, it did not stop. The author concluded that castration was not appropriate or effective to eliminate crowing behavior in roosters.

Until further studies are available, castration should be used judiciously as a method for altering avian behaviors, especially in adult birds and should always be considered secondary to more conservative methods of behavior management. However, castration has real benefit with testicular cancer, abscesses/granulomas, cysts, and other conditions that may not respond to medical management alone.

Several methods of castration have been forwarded and include simple extraction (caponization), laser ablation, intracapsular suction, en bloc surgical excision, and endoscopic orchidectomy. Even with early age caponization, testicular regrowth is well documented. This supports the need for complete testicular removal, which is why the author prefers en bloc surgical excision.

Caponization is typically performed in young male chickens to create meat that is believed to be more tender, juicier, and tastier than that of an intact rooster

[116]. Heavy chicken breeds are caponized at 2–4 weeks, while the procedure is performed on some slow-growing meat-type birds after 6 weeks old. As the bird ages, the tunica albugenea of the testes becomes hard, making caponization more difficult and time consuming. The procedure is typically performed without anesthesia with the bird held or strapped to a table. A sterile preparation is given to the appropriate side and an incision is made between the last two ribs through the lateral body wall, which is then spread with a "spreader." If performed correctly, specialized caponizing forceps are used to enter the incision; delicately hold the entire testes and pull with a twisting motion until the testicle is free and removed. The wound is disinfected and left to close by second intention. Incompletely caponized birds may regrow the testes and the birds tend to develop secondary sex characteristics, unlike true capons [116]. A similar technique using standard curved forceps is described in 9–10-week-old Japanese quail (*Coturnix coturnix japonica*) [117].

Use a cranial left lateral approach or ventral midline incision with transverse flap to evaluate the testes. As a result of the cranial location, the lateral celiotomy is often extended cranially by cutting the last two ribs to improve exposure to the testes. With a left lateral approach, puncture through the caudal thoracic and/or the abdominal air sac(s) to expose the left testis. The right testis may be exposed through the same incision by cutting through the midline junction of the corresponding air sacs or the process may be repeated with a right lateral celiotomy. With gentle traction, pull the testis ventrally and surgically clip the dorsal blood supply. Use of a right angle surgical clip often makes the approach easier. If two can be placed, then incise between the surgical clips and remove the testis. Otherwise, use electrocautery to carefully free the testis from the surgical clip and vascular cord. The cautery should destroy any remaining testicular cells that are attached to the surgical clip but be careful not to damage the overlying blood vessels, kidney, or adrenal gland.

Alternatively, if the testicular blood supply is small, a hemostat can be temporarily used in place of a surgical clip and the testis pulled free. Leave the hemostat on the vascular stump for 1–2 minutes prior to release. Use direct pressure hemostasis as needed. Diode laser excision can also be used through this approach and may be performed without the need for direct hemostasis. Closure is routine.

Multiport endoscopic orchidectomy has been described in Carneau pigeons [95]. While the details of the procedure are described in the paper, endoscopic orchidectomy produced good results in 10 of 11 pigeons with a mean surgical time of 39 minutes. Mild hemorrhage and partial necrosis of the cranial renal pole was noted in 27% of the tested birds and represented the most common complication of surgery. The one surgical failure (regrowth of the testes) was considered to be a result of surgeon inexperience. When performed using appropriate equipment and techniques, the authors concluded that endoscopic orchidectomy was successful and safe in pigeons [95]. The technique could potentially be used in poultry and waterfowl. As is expected and mentioned in the study, large testes are more difficult to remove endoscopically. In the author's experience, orchidectomy is more often needed for clinically abnormal (cancer, cysts, etc.) and often large testes that require celiotomy.

Vasectomy

Vasectomy is useful to produce "teaser males" and aids in population control. It has been described in small passerines and budgerigars. In anesthetized budgerigars, a 3 mm incision, 7 mm lateral to the cloacal sphincter (vent), was used for the initial approach [118]. Careful dissection was made through the coelomic musculature and fat. An operating microscope was used to find and aid in the removal of a 5 mm section of the vas deferens. Only the skin incision was closed. The authors recommended performing left and right vasectomy 2 weeks apart. Two of 12 birds died post-operatively and one was found to have pre-existing disease. The only other complications were post-operative tenesmus for 2 days and accumulation of droppings around the vent in 3 of the remaining 10 birds. The procedure was successful (no semen upon collection attempts) in 9 of the 10 surviving birds [118].

Anesthetized Bengalese (*Lonchura striata*) and zebra (*Taeniopygia guttata*) finches have been vasectomized similarly to the procedure described above [119]. In the anesthetized finches, a 3 mm incision 5 mm lateral to the cloaca was made using an operating microscope. The muscle and fat were incised to locate the seminal glomera (glomus). It was noted that the seminal glomera of the Bengalese finch was "obvious and highly accessible," and that of the zebra finch was "less obvious and in some cases difficult to locate." The vas deferens was carefully separated from the ureter and "one or more pieces" were removed with no ligature. The skin was closed. The authors performed single (14 days apart) and bilateral vasectomies successfully. The procedure was successful in 12 of 12 Bengalese, and 14 of 15 zebra, finches [119].

In larger species, the vas deferens zig zags lateral to the ureter and can be transected endoscopically

or via celiotomy. A left, and sometimes right, lateral coelomic approach is(are) used. The ureter is avoided to prevent damage. In roosters vasectomized just distal to the epididymus, spermatogenesis ceased within 5–7 days [120].

The author prefers endoscopic vasectomy in large birds - most commonly as a means of population control in gallinaceous birds. As described by Samour, the ductus deferens is identified endoscopically (as if evaluating the kidney) and grasped just distal to the epididymis and approximately 5–8 mm is removed with simple traction [121].

At this location, the ductus deferens is usually not closely associated with the ureter. Depending on the species, both ductus deferens may be approached through one endoscopic portal. Alternatively, two endoscopic entry points (left and right) can be used. Vasectomy does not stop courtship and copulation [121].

Diseases of the male avian reproductive system
Orchitis

Inflammation of the testicle, or orchitis, usually results from bacterial infections and may originate from septicemia, renal obstruction, cloacitis, or even prolapsed or ulcerated phalli. Affected birds may show signs of septicemia. However, the author has seen cases of focal orchitis with no associated clinical signs or reduced fertility only. Orchitis may be diagnosed via cytology and/or microbiologic analysis via aspiration through endoscopy or celiotomy when the whole testicle appears abnormal, or biopsy when focal lesions are seen.

Initial treatment for bacterial orchitis should include antibiotics based on culture and sensitivity results. If a focal granulomatous lesion is seen and appropriate antimicrobials have proven ineffective, the testicle can be partially ablated. Clamp with hemostats or surgically clip the testicular tissue dorsal (towards the blood supply) to the lesion(s) and remove using cold excision, laser, or electrocautery. Avian testicular tissue has great regenerative capabilities and may redevelop following partial ablation. En bloc surgical removal of the affected testicle is indicated for diffuse, non-medically-responsive orchitis.

Testicular neoplasia

Avian testicular neoplasia most commonly includes sertoli and interstitial cell tumors, seminomas, teratomas, and lymphoproliferative diseases. Sertoli cell tumors seem to be the more prevalent testicular neoplasm in birds. Reported neoplasms of the epididymis and ductus deferens include leiomyosarcoma and carcinoma.

Chronic weight loss, coelomic swelling, and unilateral paresis are most commonly associated with testicular cancer. Surgical removal of the affected testicle is the treatment of choice and carries a good prognosis as long as metastasis is not present. As noted in the literature, and in the author's experience, many testicular tumors are cystic. Cystic testicular masses can be aspirated and drained during surgery to reduce their mass and facilitate removal. Some testicular tumors may metastasize as has been reported in a guinea fowl (*Numida meleagris*) with malignant seminoma [122].

Cystic testicular disease

Non-neoplastic cystic testicular disease is very infrequently reported and its significance is unknown. Cystic dilatation of the seminiferous tubules (and testes), has been produced in fowl that have been fed a diet high in sodium [123]. Cystic testicles have also been noted in chickens that have been fed egg albumen as a source of protein [123]. Dilatation of the seminiferous tubules, but not gross cystic testicular change, has been noted in roosters affected with epididymal cysts and stones of unknown origin [120]. As mentioned above, consider cancer first when cystic testicles are found. Cystic testicles should be drained, biopsied, and, ideally, removed.

Disorders of the phallus

Male waterfowl have a protrusible phallus, which is highly variable and particularly long and corkscrewed in the Muscovy duck (*Cairina moschata*). Partial and complete phallic prolapses are possible in waterfowl with large phalli and are usually secondary to trauma, local infection, and masturbation. Over-exuberant vent sexing and mating, *Neisseria* spp. (suspected to be sexually transmitted in geese and the cause of "goose gonorrhea") and contamination have all been implicated as causes of phallic infections. A prolapsed phallus may become enlarged, ulcerated, and/or necrotic, which compounds the problem (Figures 16.28a,b). Frostbite and resultant necrotizing dermatitis of a prolapsed phallus has been noted in ostriches [124]. Birds with severe prolapse and infection may be significantly depressed and often lose interest in copulation.

Clean exposed phalli and carefully debride abnormal tissue prior to replacement. Topical antibiotic creams, DMSO, and systemic antibiotics may be beneficial, and their use is based on clinical findings. The cloaca may need partial closure (via transcloacal sutures) to prevent recurring prolapses. If the prolapse is prolonged and will not stay when replaced, use 4-0' monofilament absorbable suture to gently tack the phallus to its resting

Figure 16.28 A Prolapsed phallus of a domestic duck (*Anas platyrhynchos*) is readily apparent. (a) The distal end of the phallus is necrotic requiring amputation. (b) Examine the full extent of the exteriorized phallus for additional lesions prior to considering simple replacement versus amputation. (Source: Courtesy of Dr. Laura Wade.)

Figure 16.29 The phallus of the same duck in Figure 16.28 is ligated at its proximal base. The hemostat is distal to the ligature and will be the site of amputation. (Source: Courtesy of Dr. Laura Wade.)

position within the cloacal mucosa. Severely necrotic phalli often need surgical debridement (Figure 16.29). Using absorbable suture in an encircling pattern, ligate the phallus proximal to necrotic tissue. It is best if there is a clear demarcation from healthy tissue. Amputate the tissue distal to the ligature ensuring that all necrotic tissue is removed.

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Diagnostics, Drugs, and Vaccines

Egg Diagnostics

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The most common reason for backyard flock owners to seek assistance on egg diagnostics is the egg's failure to hatch to a viable chick. Another reason could be that the egg appears unaesthetic and is deemed inedible by the flock owner.

Failure to hatch

The egg can fail to hatch as a result of internal factors, such as poor hen nutrition, or external factors, such as incorrect incubation temperature and humidity [1]. In these cases, the egg is fertile but is unable to hatch as a result of internal or external factors. Hence, the first step in investigating why an egg does not hatch is determining if it was a fertile egg to begin with.

Egg formation

Formation of an egg has been extensively documented and in-depth reviews for further reading include King and McLelland (1984), Burley and Vadehra (1989), or Whittow (2000) [2–4]. The egg is a complete source of nutrients for the developing chick. It takes approximately 24–28 hours to produce an egg. In the chicken, egg production is initiated when a mature ovum is released into the infundibulum, the first portion of the hen's oviduct [5] (Figure 17.1a). This process starts at the ovary. The mature avian ovary is composed of finger-like stalked projections or follicles (Figure 17.1b), each one of which has a single layer of granulosa cells surrounding the primary oocyte. The structure of the ovary resembles a bunch of grapes, with each individual grape being a follicle. The primary oocyte of the chicken can expand its cytoplasm considerably, reaching approximately 30 mm diameter, to accumulate the proteins and lipids that form the yolk. The surrounding stromal tissues of the follicles, the well vascularized theca interna and theca externa, rearrange towards the time of ovulation to leave a vascular-free area called the stigma (Figure 17.1b). The stigma ruptures during ovulation and the oocyte with the surrounding granulose cells leaves the follicle. If some vessels are still present and rupture at the time of ovulation, blood spots can be observed on the yolk. The remaining thecal cells of the follicle do not transform into a corpus hemorrhagicum or corpus luteum as in mammals.

In the process of forming an egg, the egg spends approximately 15–30 minutes in the infundibulum (Figure 17.1a), a short period of time to synthesize and incorporate a significant number of layers. External layers of the yolk membrane and the chalaziferous layer are produced primarily in the caudal portion of the infundibulum. In addition, the ovum is fertilized in this portion of the oviduct if viable sperm is present in the sperm glands.

After leaving the infundibulum, the ovum then enters the portion of the oviduct known as the magnum (Figure 17.1a), which has an abundance of mucus-secreting glandular cells. The transit time in the magnum is about 3 hours. During this time, approximately 50% of the total albumen is produced and appears homogeneous and dense in nature, although dense and thin albumen is also formed. Hence, differentiation between dense (thick) and thin albumen results from the addition of water occurring after the egg leaves
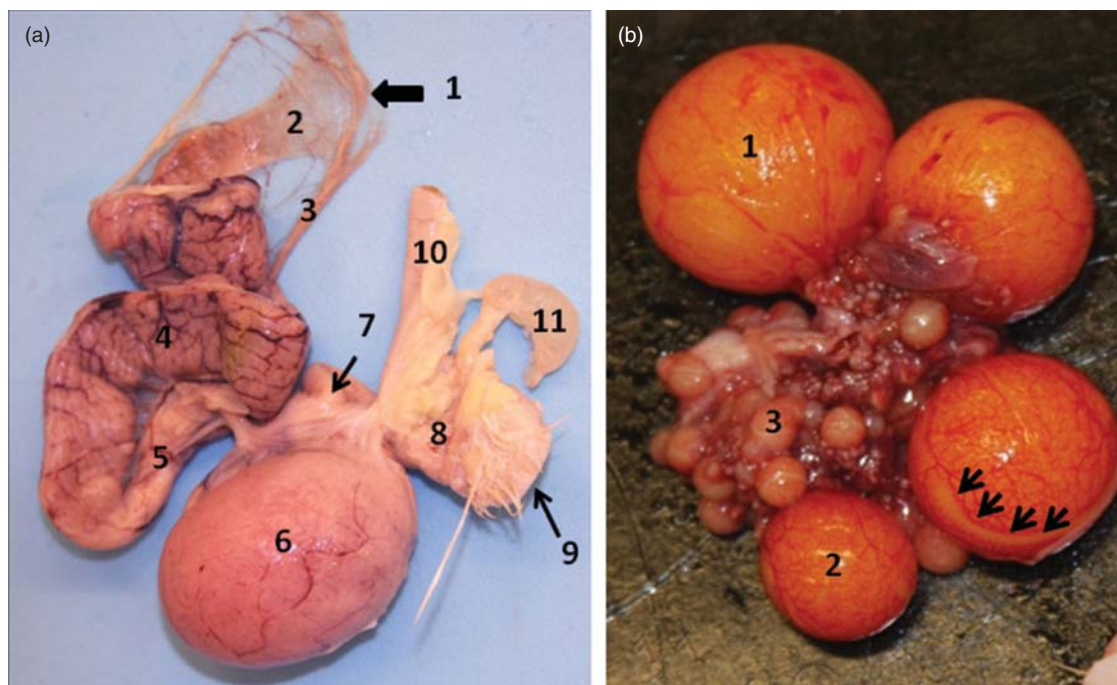
Figure 17.1 (a) Adult hen's reproductive tract: 1. infundibular openning (ostium abdominale); 2. infundibulum; 3. ventral suspensory ligament; 4. magnum; 5. isthmus; 6. uterus (shell gland) containing a calcified egg; 7. vagina; 8. cloaca; 9. vent; 10. colorectum; 11. regressed right ovary. (b). Adult hen's ovary with follicles at different stages of development (1: mature - 3: immature). Notice the area (arrows) without vascular blood supply corresponding to the stigma. Pictures courtesy of Drs. Khamas and Rutllant.

the magnum. The thick portion is added first and is the closest to the yolk [5]. It provides most of the protein for the developing embryo. In addition, white rope-like structures known as chalazae form at both poles of the egg (Figure 17.2). Diseases such as infectious bronchitis can make the dense albumen thin. In chickens that are affected, the thick egg white becomes thin and runny, so the lack of a thick egg white is noticeable [6–8].

The main function of the next segment of the oviduct, the isthmus (Figure 17.1a), is the synthesis and addition of the shell membranes. Before the shell membranes are completed more water is added "plumping" the already existing albumen and further differentiating the two albumen types, thick and thin. The whole process takes approximately 1–2 hours to be completed.

The egg remains in the uterus, where the eggshell is formed, for most of the time (around 20 hours) (Figure 17.1a). During the first 3–5 hours, the organic matrix is formed and in the following 15–16 hour phase, calcium is deposited to form the inorganic substance consisting of more than 95% of crystallized calcium carbonate. The most common source of the calcium for eggshell production is intestinal absorption

when dietary calcium levels are adequate. Alternative sources may come from mobilization of bone calcium stores and renal reabsorption. If there is inadequate calcium in the diet, hens may lay shell-less eggs or no eggs. Often, hens that are unable to obtain calcium from the diet extract calcium from their own bones, leading to disorders such as cage layer fatigue or osteomalacia. The color of the egg shell depends on the breed of chicken. It is interesting to note that, in general, chickens with red ear-lobes tend to lay brown eggs and those with white ear-lobes tend to lay white eggs [5]. The exceptions are those breeds such as the Araucana that lay greenish colored eggs. The eggshell coloration depends on the presence of pigments (brown-red) such as porphyrins, a byproduct of hemoglobin.

Finally, the egg's transit through the vagina takes seconds to a few minutes and the secretions produced in this segment of the oviduct may contribute to the formation of the cuticle. The cuticle can be easily rubbed off, so gentle egg handling is necessary to avoid its removal. Finally, the egg passes through the cloaca and is laid (Figure 17.1a).
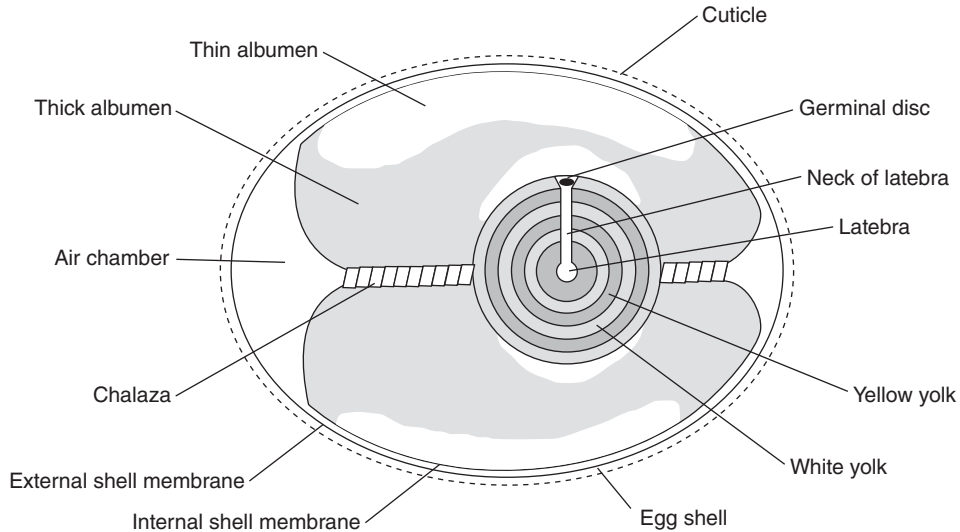
Figure 17.2 Diagram of a hen's egg in a longitudinal section depicting the thick and thin albumen surrounding the yolk, with the chalzae rope-like structures at both ends of the yolk.

Normal egg anatomy

The fully formed chicken egg consists of four main parts: *Germinal disc, yolk, albumen with chalaza, and shell* [9–11]. The germinal disk (also known as the blastodisc, or, if fertilized, the blastoderm) is a circular structure of approximately 3–4 millimeters in diameter located on the surface to the yolk and is white-gray in color (Figures 17.2 and 17.3a–b). It contains the remnant of the oocyte nucleus while the cytoplasm is an extremely thin layer that covers the rest of the surface of the yolk. The yolk is suspended in the center of the egg by the chalaza. The yolk (vitellus) consists mainly of lipoproteins and phosphoproteins arranged in concentric layers (Figure 17.2). Depending on the content of protein and lipids, it can be distinguished into alternating yellow and white layers of the yolk, but cannot be visually observed. The inner-most central nucleus of white vitellus is known as the latebra and is connected to the germinal disk through the neck of the latebra. The main function of the yolk is to nurture the developing embryo.

The yolk membrane is the thinnest of all egg layers but could present the principal barrier for fertilization. It is composed of four different layers that can only be distinguished with electron microscopy: i) plasma membrane (plasmalemma) of the oocyte; ii) perivitelline membrane or lamina (also known as inner layer of vitelline membrane); iii) continuous membrane or lamina; and iv) extravitelline membrane or lamina (also
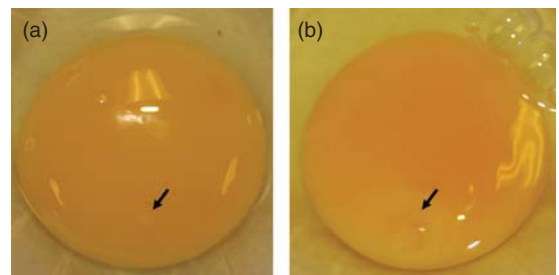
Figure 17.3 (a) Egg yolks from non-fertilized and (b) fertilized eggs. The arrows show the germinal disc or blastodisc (a) and blastoderm (b).

known as the outer layer of vitelline membrane) [12]. The first two layers are produced by the oocyte and the granulosa cells while still in the ovary as a follicle. The last two layers are produced as the eggs pass through the infundibulum. The yolk membrane is the barrier between the yolk and the albumen but allows for the movement of water and electrolytes.

The albumen (or egg white) is the main component surrounding the yolk. Although it is described as having less structure than the yolk, two different regions can be identified; thick (dense) and thin (liquid) albumen (Figure 17.2), depending on the proportion of water and protein (ovomucin) [2]. Thick albumen has a higher quantity of ovomucin than thin albumen and consequently has more internal structure. The chalazae are parts of the dense albumen that fix the yolk to

the egg poles. They are made of twisted strands of ovomucin fibers arranged in a spiral as a result of the rotation of the egg while it descends in the oviduct. The thin albumen contains mucin with less fibrous arrangement, and consequently less structural scaffold, giving an appearance of higher fluidity. The inner layer of the thin albumen is attached to the yolk and the outer layer is in contact with the shell membrane, but only at the egg poles. The main functions of the albumen are to mechanically protect the embryo with a soft environment and nurture the embryo with a good source of protein.

The shell membranes are two thin, pliable but strong membranes, composed mainly of several layers of protein fibers (Figure 17.2). It is only at the egg poles that the inner shell membrane is fused with the dense albumen. In the other regions of the egg, the inner shell membrane is in contact with the thin albumen. The inner shell membrane is firmly attached to the outer shell membrane, which in turn is also tightly attached to the eggshell. A few minutes after the egg is laid, and as it cools down, the internal and external membranes detach at the blunt pole creating the air cell. During the process of embryo development, the head of the embryo lies adjacent to this air cell (Figure 17.2). Being aware of egg anatomy will assist in egg culture techniques.

Finally, the eggshell is mainly composed of two basic parts: The organic matrix and the inorganic interstitial substance, which is composed of inorganic salts. Both parts are interrelated and intermingled. The organic matrix is the primary biological layer, and is composed of a meshwork of fine fibers of different arrangements infiltrated with calcite crystals. The eggshell has microscopic pores all over the surface, which open into pore canals that end at the level of the outer surface of the outer shell membrane. This passageway from external surface to shell membrane is the reason why egg membranes may need to be cultured, as it may provide clues as to the hygienic condition of egg management. These pores are covered and plugged by the cuticle, the outermost organic layer of the egg (Figure 17.2). The cuticle is an extremely thin proteinaceous layer, which is permeable to gases [13]. It is generally recommended to only lightly brush off any organic debris from eggs, rather than washing them, to prevent damage to the cuticle. If eggs are to be cleaned then it is recommended to use water that is warmer, specifically 20°F (11°C) warmer, than the egg so that contaminants are not drawn into the egg via vacuum action through the pores in the shell [14].

Fertile and non-fertile eggs

From external appearances, one cannot tell if an egg is infertile or fertile. Chickens lay eggs whether they are fertile or infertile. The only way to determine whether a newly hatched egg is fertile is to crack it open. An infertile egg only has a white-gray dot on the surface of the egg yolk (Figure 17.3a). This area is called the blastoderm. A fertile egg has a white donut-like structure with a white-gray dot in the center on the yolk surface (Figure 17.3a). This structure is the blastoderm, the developing chick. Fertility rates are normally deemed to be the number of fertile eggs laid over a period of time when compared to all eggs laid during that same time period. It is not practical to determine the fertility rate of backyard poultry, as the only way to determine this is to open eggs up. This practice contradicts the backyard owner's goal of hatching new chicks [15]. Hence, for veterinarians faced with questions of hen fertility, the best recommendation is to incubate all potentially hatchable eggs. If fertility determination is needed, flock owners need to know that hatching eggs need to be sacrificed and opened.

Maximizing egg fertility

Backyard flock owners may want to develop their own genetic lines or breeds and they can use artificial insemination to breed their chickens. As mentioned previously, chickens have the ability to store viable sperm in the sperm glands that line the infundibulum. While sperm can live for prolonged periods in this gland, specifically 7–14 days in chickens and 40–50 days in turkeys, the probability of hens laying fertile eggs declines after 5–7 days [16]. To guarantee a high percentage of fertile eggs, it would be better to allow hens and roosters to mate naturally. This is more commonly seen in backyard flocks in which the sexes are mixed. The ideal ratio of roosters to hens is one rooster to 5–7 hens [15]. This ensures a high rate of fertile eggs. Although this is an ideal ratio in a backyard flock, one other factor should come into play: Whether the flock has a lone rooster. Flocks that have a single rooster have more behavioral problems. A single rooster in a flock tends to be more aggressive to its human caretakers and this may be a problem for owners with young children, as the rooster may attack its human caretakers [15]. Hence, in small flock situations where fertile eggs are needed, it is highly recommended that two roosters are added to the flock with some additional hens so that the roosters can establish a pecking

order of their own rather than with their human care-takers.

Hatching fertile eggs

If potentially fertile eggs are laid, the next step is to incubate the eggs. Eggs are unique in that they can be laid fertile and the developing chick can remain in suspended animation until incubation, during which time the developing chick can continue its development. Once development starts, it must continue until hatching or the developing embryo will die. The only time that chick development can be delayed is the point at which the fertile egg is laid but has not yet been incubated [15]. Poultry breeders have utilized this unique feature of chick development to hold newly hatched eggs under refrigerated temperatures until a batch is laid, at which time all eggs are incubated to ensure synchronous hatching of a large batch of chicks. If you are storing hatching eggs, they should be held at 55–65°F (13–18°C) and at a humidity ranging from 80–90% with the large end of the egg facing upwards. Hatchability will decrease if eggs are stored longer than 7 days [15]. Depending on the type of incubator and the number of eggs, if the goal is to hatch as many as possible, then the eggs should probably be incubated soon after laying. When eggs are placed in an incubator, it is ideal to mark the date of lay or the expected day of hatch in pencil on the egg shell to ensure that it is incubated for the appropriate time period for that species [15].

Incubation of eggs

When incubating hatching eggs, four factors need to be in alignment in order to successfully hatch a chick. These four factors include temperature, humidity, ventilation, and egg position [15]. The temperature for incubation can vary depending on the type of incubator: "Still" air versus "forced" air. A "still" air incubator does not have an internal fan to circulate the air; therefore a temperature of 102°F (39°C) is required. A "forced" air incubator contains an internal fan to circulate the air and the ideal temperature is 99.5°F (37.5°C). In either type of incubator, the temperature should be constant and uniform. Humidity at 60% for the first 18 days of life is ideal, increasing to 70% for the last three days of hatch [17,18]. Humidity is very important because if there is not enough humidity, chicks can become entrapped in the dehydrated shell

Table 17.1 Incubation times of common poultry species

Species	Incubation period
Chickens	21 Days
Turkeys	26–28 Days
Pheasant	22–23 Days
Quail	23–24 Days
Peafowl	27–28 Days
Guinea Fowl	26–30 Days
Duck	28 Days
Muscovy Duck	33–35 Days
Goose	29–31 Days
Swan	42 Days

Source: Hayes, C. [17], Schwartz, D.L. [18].

membranes and cannot hatch out of the egg, or if they are able to hatch they are very exhausted and stressed. Ventilation is required as the embryo develops because oxygen is needed for the developing chick. For this factor, it is recommended that flock owners follow the manufacturers' recommendation for the particular incubator used. Eggs are usually rotated at least five times a day to prevent the yolk from sticking to the shell and hindering chick development. The incubation time varies per poultry species (Table 17.1) [17,18]. It should be noted that waterfowl require a higher level of humidity compared to chickens.

Chick embryo development

Normal chicken development inside the egg is a complex process that needs to be understood in order to identify possible causes of malformations, lack of development, or embryo mortality during incubation. Historically, the chicken embryo was one of the first embryos studied and described because the eggs were easily available and the incubation conditions were not difficult to mimic [19]. By cutting a small window in the egg shell and covering it with glass, the formation of an embryo could be directly observed. Under normal conditions, it takes 21–22 days to develop a live chick (1 day in the oviduct and 20–21 days in the nest or incubator). The first step occurs in the infundibulum with the fertilization of the ovum or germinal disk (haploid) by the sperm (haploid), which forms the zygote (single diploid cell) [20]. The zygote undergoes a series of cell divisions at the level of the isthmus and becomes the blastoderm or embryo. The process of cell division or embryonic

development is temporarily interrupted during the laying process and is resumed to completion once the temperature increases again during natural brooding or artificial incubation. When the temperature of the egg is below 68°F (20°C), the embryo becomes quiescent and development stops. However, if temperature reaches 68°F (20°C) or above, embryonic activity starts again. The best time to store viable fertilized eggs that are placed in cool storage below 68°F (20°C) is as soon as possible after collection. Once in the incubator, the temperature must be controlled within very close parameters (optimal temperature 99.5°F [37.5°C]) and oscillations should be avoided for normal embryo development and maximum viability results.

All the cells in the blastoderm divide in a single monolayer on top of the yolk and create a central zone known as the *area pellucida* (clear area) and a marginal area known as the *area opaca*. The cells in the *area pellucida* divide faster, creating a superimposed number of layers that become the ectoderm (outer or upper-most), mesoderm (middle), and endoderm (deeper or inner-most) [21,22]. The major body parts that are formed from these three initial embryonic layers are

1. **ectoderm:** Nervous system, epidermis and its derivates (feathers, beak, claws) and some of the skeletal and connective tissue of the head;

2. **mesoderm:** Muscles and skeletal tissues, reproductive, urinary and circulatory (heart and vessels) systems;

3. **endoderm:** Lining of digestive tract and associated organs (liver and pancreas) and respiratory system.

While the chick embryo is growing, four associated membranes can be distinguished (Figure 17.4) [21,22]. Although they are similar to those described in mammals, the chick embryo must develop independently and outside of the hen's body, which makes these membranes especially important for accessing the nutrients present in the egg and acting on essential living functions (respiration, excretion, and mechanical protection). These four membranes and their major functions are

1. **yolk sac:** Although initially the embryo is a flat structure, it progressively folds cranio-caudally and laterally, leaving an aperture at the ventral aspect of the coelomic cavity and creating the yolk stalk. The yolk sac is the membranous sac that is attached to the embryo at the yolk stalk and mainly provides nourishment. After Day 6 of incubation, the yolk sac surrounds all of the yolk and is the site of primary vessel growth, blood cell formation, and germ cell differentiation before these cells migrate into their respective organs. Around Day 19 of incubation, the yolk sac is drawn into the abdominal/ceolomic
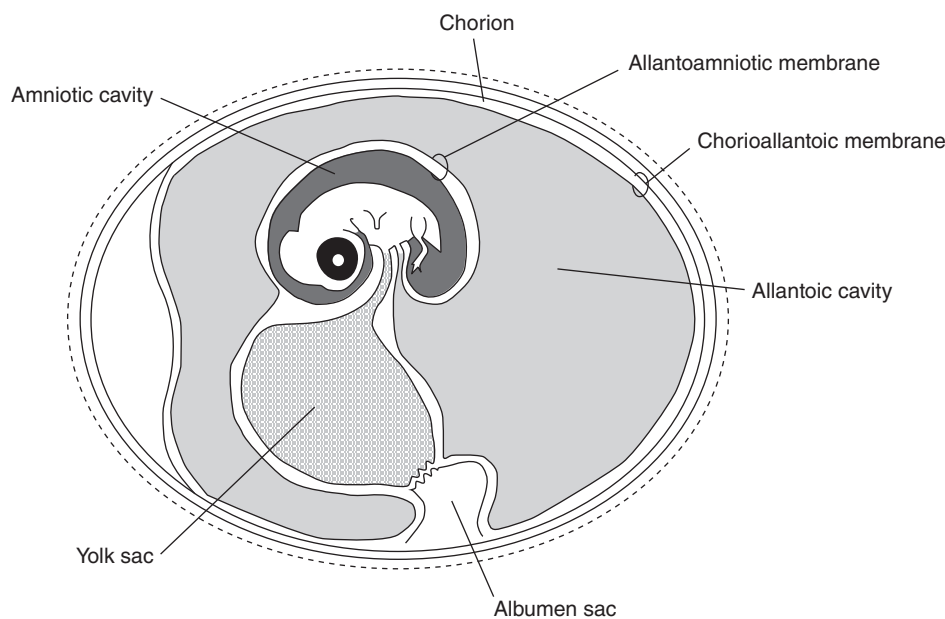
Figure 17.4 Diagram of a chick embryo in a longitudinal section showing the different membranes and sacs formed around the embryo for protection, nourishment, and basic life functions.

cavity. Sometimes, the yolk sac may not be fully regressed and a remnant of the yolk stalk can be found at Meckel's diverticulum (see Chapter 7);

2. **amnion:** When the most peripheral membranes in the egg (future chorion) grow dorsally as two different folds (one cranial and one caudal), the embryo sinks and allow the folds to join dorsally, covering and completely surrounding the embryo and thus forming the amnion and the amniotic cavity. This cavity is filled with watery fluid (amniotic fluid) in which the embryo floats and is protected from external forces or impacts;

3. **allantois:** The allantois arises during Day 3 of incubation as an outgrowth of the hindgut, which passes through the umbilicus close to the yolk stalk. It grows rapidly and occupies the space between the amnion and the chorion by Day 10 of incubation. Vessels from and to the heart (umbilical or allantoic arteries and veins) rapidly colonize this membrane and occupy the space between the allantois and the chorion. When the chorioallantoic membrane is completely apposed to the shell membrane, the exchange of respiratory gases can occur (respiratory function). The allantoic sac also has an important excretory function in collecting urinary and digestive waste that precipitates as uric acid components. As uric acid is a solid nitrogenous waste product, it does not diffuse across the allantoic membrane, which allows it to remain in close proximity without toxic effects;

4. **Chorion:** The chorion is the most peripheral membrane and fuses externally with the inner shell membrane and internally with the allantois. As mentioned previously, the chorion, together with the allantois (chorioallantoic membrane), serves to mediate gas and water exchange.

In order to identify the developmental stage of chick embryos, a simplified table describing the formation of the major organs or structures is presented in Table 17.2 [19,23].

During this process the position of the chick changes progressively so that the anterior part of the body lies towards the large end of the egg by day 14, the head is covered by the right wing by day 18, and the feet are in contact with the head when the egg is ready to hatch (see video on website showing chick hatching).

Monitoring chick development

As the chick develops in the egg, development can be monitored by using the process known as candling.

Table 17.2 Simplified table describing the developmental stage of chick embryos and the formation of major organs or structures

Day	Organs or anatomical structures present
0	Small germinal disc not fertilized
1	Blastoderm, embryonic tissue
2	Formation of blood vessels on top of yolk
3	Leg and wing buds, heart formation and beats can be observed
4	Formation of eyeball (pigmented area)
5	Identification of limb joints (elbow and knee), digits and toes
6	Beak and feather tracts
7	Web between toes and, egg tooth
8	Initial appearance of feathers, nictitating membrane
9	Phalanges in toes
10	Primordium of comb
11	Allantois has the maximum size, embryo looks like a chick
12	First complete feathers
13	Claws
14	Whole body covered by feathers
15	Vitellus (yolk) shrinks
16	Egg white disappear
17	Urates appear
18	Total growth near completion
19	Yolk sac attached to body cavity
20	No presence or minimal yolk sac
21	Newly hatched chick

Sources: Hamburger, V. and Hamilton, H. [19], Warin, S. [23].

Anyone who incubates eggs should have a candler, and these are available for purchase through poultry supply companies. Candling is the use of a strong external light source to view the developing egg through its thin shell. Dark-shelled eggs or the thicker eggs of waterfowl are not candled as easily as those of chickens. While candling is useful for monitoring chick development, its frequent use can also disrupt chick development if performed excessively or if eggs are handled roughly. As the chick develops, the branching of pulsating blood vessels can be noted through the egg shell. When the egg is candled, visible chicks have blood vessels that show disappearance of the branching of the blood vessels and a noticeable static blood ring if there is early mortality in the incubation period.

If there is no branching of blood vessels after a few days of incubation, this indicates that the egg may have been infertile or that there was early embryonic mortality. Most mortality in hatching eggs occurs in the early

or late period of incubation. If there are no signs of life in the egg, then egg diagnostics could be employed to help determine the cause of death. Eggs should immediately be removed if there is no branching of blood vessels or if a static blood ring is present after 5–7 days of incubation.

Egg diagnostics

In the early stages of incubation, the primary diagnostic tools used are egg visualization, egg pathology, and egg culture [24,25]. Hatching eggs that do not show visible signs of life should be cracked open and emptied into a sterilized glass petri dish or bowl to observe for the remnants of the blastoderm or blastodisc. Depending on the degree of work-up required by the owner, this area can be clipped out of the yolk with sterile ophthalmic surgical scissors and forceps and placed in formalin for further histologic examination to determine if it was fertile. In addition, as bacteria play an important role in egg infections, the egg can also be cultured and plated in blood agar and MacConkey's media to check for aerobic bacteria [24,25]. Some of the common isolated bacteria that are found in eggs and ill chicks are *Escherichia coli*, *Klebsiella* species, *Staphylococcus aureus*, *Steptococcus* species, *Pseudomonas aeruginosa*, *Salmonella* species, and *Proteus mirabilis* [6,24,25].

Bacterial culture of eggs

There are many sites where eggs can be cultured, depending on the questions that need to be answered. These include the egg roll, egg membranes, and yolk [24,25]. Positive determination of bacterial presence in the egg usually indicates problems with nest sanitation, egg storage, hen infection, and/or hatchery sanitation [24,25]. These bacteria, which can result in a decrease in hatchability, can also cause weak chicks to hatch with subsequent omphalitis (yolk sac infection) [6,24,25]. The basic tools needed to perform egg culture include an incubator, and bacterial media such as sheep blood agar and MacConkey's media plates. These two solid plate media are the basic culture medium necessary for egg microbiology techniques, as the major bacteria associated with egg infections can be isolated from these media. These media should be incubated at 98.5°F (37°C) in air for 24 hours. If there is no growth, then these media plates should be incubated for an additional 24 hours.

Egg roll culture

On occasion, the external surface of the egg needs to be cultured to determine the bacterial load to which it is exposed in the nest environment. For this procedure, with sterile gloves pick the newly hatched egg from its nest environment and gently roll the surface of the egg on two blood agar and two MacConkey's media agar plates. The egg can then be placed in a sterile Whirlpak bag and incubated in brain-heart infusion broth for 18 hours [24,25]. After incubation, the suspension should be centrifuged and the resulting pellet should be plated on blood agar and MacConkey's media. This will determine if there are nest management sanitation problems. Nest material should be frequently cleaned to ensure that there is minimal fecal debris on the surface of hatching eggs. Bacteria enter the egg through the egg shell pores, so a clean nest environment and timely collection of eggs minimizes bacterial contamination.

Egg membrane culture

It is sometimes necessary for the shell membranes to be cultured, especially when there is suspected contamination resulting from improper storage and handling of the egg [24,25]. When an egg is laid, sometimes it can be stored at varying temperatures and this may cause sweating/condensation on the surface of the egg. The moisture on the surface of the egg can facilitate bacteria to enter the egg via the egg shell pores. The shell membranes can serve as a partial mechanical barrier to hinder bacteria from reaching the nutrient-rich yolk. To determine bacterial presence at the level of the shell membrane, take an egg carton, or a similar alternative container, and place the egg that is to be cultured in one of the egg slots to hold the egg upright with the large end up. Be careful not to come into contact with the egg carton as it is not sterile. Pour 70% alcohol over the top of the egg and let it air dry. Using a sterile forcep, gently break and remove the shell. This provides access to the air cell. Take a sterile cotton-tipped swab that has been moistened with brain-heart infusion broth and place it in the air cell. Gently work the swab between the layers of shell membrane so that at least the top half of the egg is swabbed. You can pick up the egg from the egg carton if it facilitates culturing. Place the swab in a tube of brain-heart infusion broth and incubate for 18 hours at 98.5°F (37°C) in air. At this point, Salmonella-selective media can also be used if needed. After incubation, plate on blood agar and MacConkey's media for 48 hours and incubate at 98.5°F (37°C) in air. The presence of bacteria can usually be noted after 24 hours of incubation.

Egg yolk culture

Culturing the egg yolk is one of the most important diagnostic tools in egg diagnostics [24,25]. The yolk is nutrient rich and can enhance and support bacterial growth. The presence of bacteria in this region of the egg indicates contamination from the external outer shell to the yolk or may indicate infection from the hen. Because the yolk is nutrient rich, bacteria, upon reaching the yolk, can multiply rapidly; therefore the timing of infection can be estimated. If there are bacteria present as a result of unsanitary nesting or egg storage conditions, early infection would probably have occurred, with early embryonic mortality. Bacterial infection of the yolk at hatching usually results in a weak chick that has omphalitis. This would most likely occur as a result of contamination caused by unsanitary and improper hatching conditions. Yolk that is contaminated with bacteria is often dark and sometimes brown in color [24,25]. There may be an odor to the egg. Sometimes both the egg white and yolk are cultured together as a pooled sample. However, the egg white has antibacterial properties and this may hinder the culture of low numbers of bacteria unless they are placed in an enrichment broth, such as brain-heart infusion broth, rather than plating directly on bacterial media plates.

Exploding eggs

Eggs have been reported to explode in incubators. The exploding eggs are usually the result of an infected egg. The egg yolk can become contaminated with bacteria and provides a good nutrient source for them. As the bacteria multiply, they also produce gas and this causes the egg to explode in the incubator. *Pseudomonas aeruginosa* is the most common bacteria that is associated with exploding eggs [6,24,25]. Unfortunately, if there are other eggs within the incubator, an exploding egg can spread its contaminated debris onto other eggs and contaminate them. This can lead to more exploding eggs. Eggs can become contaminated when the bacteria from the contaminated debris enters a new egg via the eggshell pores. Under the appropriate conditions, additional eggs can become infected. If an exploding egg occurs, be aware that additional eggs may explode and the chicks, if they hatch, may not flourish. They may potentially have omphalitis. The incubators should be thoroughly disinfected after an exploding egg incident, based on recommended disinfectant protocols, as soon as feasibly possible [26–28].

Egg breakouts

An egg breakout can be performed on eggs that do not hatch. An egg breakout provides clues as to why the developing chick did not successfully hatch into a viable chick. As mentioned previously, there internal and external factors can influence the hatch of a viable chick. Internal factors are inherent to the developing chick and include nutrition of the hen. For example, if the hens are not provided with adequate nutrition, the hatching chick is weak. A lack of vitamin E can result in a weak hatching muscle, which results in the chick being unable to use its neck (hatching) muscle to break open the shell. The inability to break open the shell results in a dead-in-shell chick. External factors affect a chick from hatching, given it is a fully capable chick. These external factors include temperature, humidity, and ventilation of the incubator environment [29,30]. For example, if humidity is too low the shell membranes dry out. The chick may have pipped (broken through the shell membrane and shell), but because of the low humidity, the shell membranes become dried out. These dried out membranes then harden and become adhered to the chick. The chick is not able to break free and uses its energy in attempting to do so. If it becomes exhausted, the chick will die in its shell. Table 17.3 provides information about egg breakout and its possible causes (Table 17.3). It should be noted that even under normal conditions not all eggs will hatch. Normally, for any given batch that is incubated, 2–4% may be

Table 17.3 Common egg breakout findings and potential internal or external causes

Egg breakout finding	Potential causes
Cracks in Shell	Improper or Rough Handling of Eggs
Moldy Contents	Hatchery Sanitation Issues
abnormal body parts (brain, eye, beak); hemorrhage	too high incubation temperatures
chicks with red hocks	hen nutrition or high incubation temperatures
chicks that have pipped but dead-in-shell and shell membranes not dried out	hen nutrition or humidity too high during the incubation period
chicks that have pipped but dead-in-shell and shell membranes dried out	too low humidity
unhealed navels	bacterial contamination

Sources: Morishita, T.Y. [15], Ande, T.B. and Wilson, H.R. [29], Stephenson, A.B. [30].

infertile; 2–3% die early in the incubation period (up to day 7); 1% die during the middle phase of incubation (Day 7–14); and 3–4% die during the late phase of incubation (Day 14–21). All dead-in-shell chicks can be placed in formalin and submitted for necropsy.

Unaesthetic eggs

The other reason why flock owners ask for egg diagnostics is that the egg may be deemed inedible because it appears unaesthetic and unappetizing.

Abnormalities of the yolk
Color
Yolk color is influenced by diet. Hens that are fed commercial diets tend to have pale yellow yolks when compared to the darker yellowish orange yolks from hens that are allowed access to grass [15]. Some backyard flock owners have been known to feed marigold flower petals, which are naturally high in carotenoids, to impart a darker orange color to their hens' egg yolks [15].

Taste
Yolk flavor can be influenced by the hen's diet. While not commonly fed to hens, fish by-products can impart a fishy flavor to the eggs [15].

Blood Spots
On occasion, fresh blood can be found on the egg yolk's surface (Figure 17.5). A common misconception is that it may be a developing embryo. The red blood is often referred to as blood spots and represents the rupture of ovarian blood vessels when the yolk was released from the ovaries. Some hens are more nervous than others and blood spots are noted in some genetic lines [15]. The blood should not be part of embryonic development unless the egg was previously incubated. However, eggs destined for human consumption should be collected immediately after they are laid rather than incubated.

Meat spots
The other debris that can be found in the egg white takes the form of light beige debris, often referred to as meat spots (Figure 17.5). Meat spots are the sloughed inner lining of the oviduct and are usually noted in hens that have been laying eggs for a long period of time without a molt. It is more often observed in hens that have not had a natural molt [15]. Molting is a period of time during which the hen needs to rest its reproductive system. The oviduct regresses until the hen begins a new lay cycle.
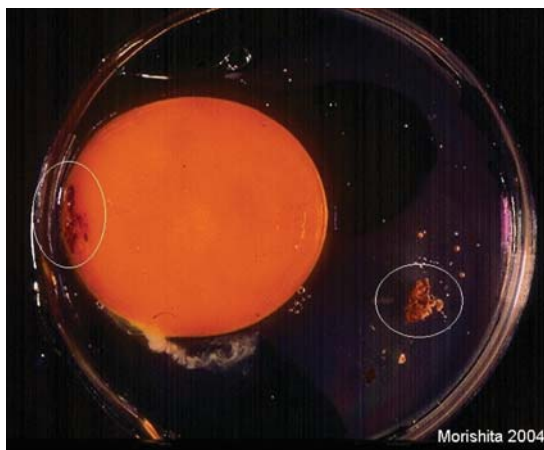
Figure 17.5 An egg with both blood spots (left circle) and meat spots (right circle).

Thin egg white
When an egg is cracked out, the yolk is surrounded by an inner thick egg white (albumen) and an outer thin albumen. Infectious bronchitis is a disease that can affect the magnum and can result in eggs of poor internal egg quality, which have runny egg whites [6–8,31].

Worms in eggs
In hens with severe roundworm infections, there can be occasions when a worm becomes encased in the egg. This can occur when an adult worm exits the gastrointestinal tract and happens to migrate up the reproductive tract. If it migrates up to the magnum, it is incorporated within the egg [32].

Ruptured shell membranes
On occasion, the outer shell membrane can break or form incompletely, leaving an opening in the calcified shell from which the disrupted shell membranes form a tuft [33].

Misshapen eggs
Besides affecting internal egg quality, infectious bronchitis can also impact external egg quality. The infectious bronchitis virus can affect the uterus (shell gland), resulting in eggs that are wrinkled (Figure 17.6) [6,7].

Yolkless and double-yolked eggs
On occasion, in hens that are beginning to lay, there is an incoordination within the reproductive tract. A yolk is released, but some egg white is made, moves down the reproductive tract, and become encased in shell membranes and a calcified shell. These eggs are normal in
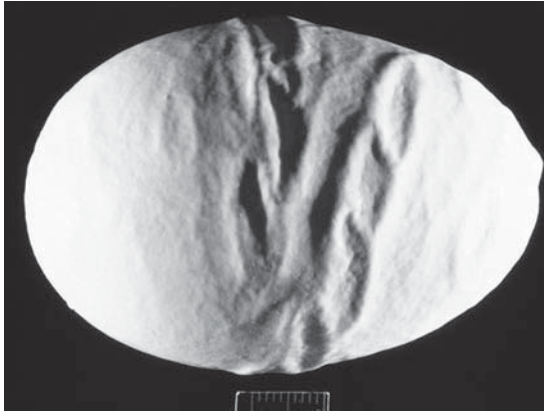
Figure 17.6 Wrinkled egg from a laying hen with infectious bronchitis.

appearance and are usually less than one half the size of a normal egg.

Double-yolked eggs are formed when two yolks are released at the same time and become encased within the egg. Some genetic lines of chickens have a tendency to form double-yolked eggs. Double-yolked eggs can be 1.5 times larger than normal eggs.

In conclusion, egg laying complaints usually result in eggs not hatching out or the production of abnormal eggs. It is important to identify the causes of these abnormal hatches or abnormal eggs, so that any future problems can be prevented for the flock owner.

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Diagnostic Lab Sampling

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Introduction

Veterinarians are educated and trained to diagnose and treat animal diseases. Many practicing veterinarians do not specialize in poultry medicine/diagnostics. However, for veterinarians with an interest in treating backyard poultry, the advantages of incorporating these patients into the practice are numerous. With only minimal equipment additions and continuing education in the field of poultry, the practitioner can increase patient diversity and practice volume and gain an introduction to the challenge of poultry diagnostics and therapeutics.

Diseases that affect poultry have a wide range of overlapping clinical signs and visible lesions. Most veterinarians should be able to quickly diagnose most common avian problems and send appropriate tissue samples to the diagnostic laboratory for further testing if necessary. This is especially important when a foreign or notifiable disease, such as avian influenza, Newcastle disease, or infectious laryngotracheitis, is suspected. In this chapter, where and how to obtain appropriate diagnostic samples is discussed. Additionally, guidelines on how to best collect and preserve samples and considerations for sending samples to an outside laboratory are provided.

History

Clinical examination is a key part of diagnosis of diseases in birds. Even before a bird is handled for examination, there are important prerequisites. The first of these is to ensure one has as complete a history as possible. Good background information increases the chances of an accurate and rapid diagnosis. At the clinic, veterinarians are normally confronted with the individual bird, but a visit to inspect the premises where a flock is kept may be helpful and is sometimes necessary.

The collection of clinical history is very similar to the gathering of information in general veterinary practice. However, clinical signs in mammals are more obvious to owners than those of birds. Therefore, careful, methodical, and logical questioning is essential when dealing with birds. History should include information not only about the bird(s) itself but also about the environment in which the bird lives and the management to which the flock is subjected.

First, it is important to collect basic information regarding the owner and the patient, such as owner's name, address, and contact information, patient's name or other identification (e.g., leg band), species, breed, gender, age, source, and duration of ownership. It is also pertinent to ask the reason for the visit. The patient may be submitted for routine health assessment, inspection of the flock (e.g., for export purposes), or a medical condition.

Start gathering clinical information with general questions, proceeding to more specific ones. A thorough history frequently provides clues that may identify risk factors that are important in diagnosing and resolving a patient's problems. Important aspects to consider during the gathering of the clinical history include general clinical details, housing, and feeding, vaccinations, and medications if any (Table 18.1).

Table 18.1 Important aspects to consider during clinical history collection

History of the problem

Where possible list dates of onset and/or duration.

- General abnormalities – sudden death, morbidity (the number of clinically sick birds), droopiness, depression, lack of appetite, ruffled or missing feathers, abnormal color of wattles and combs, dehydration, loss of feathers, etc.
- Respiratory system – sound of fluid mucus in the airways (rales), gasping, coughing, swelling of areas around eyes, inflamed sinuses, watery eyes, nasal exudates, etc.
- Digestive system – loose droppings, diarrhea, abnormal color of feces, big belly, etc.
- Nervous system – head shaking, neck twisting, abnormal extension of legs, circling, etc.
- Skin and musculoskeletal system – scratches, abnormal discoloration, lumps, lameness, scaly legs, twisted legs, abnormal back curvature, etc.
- Reproductive system – drop in egg production, poor egg quality: Thin shell, abnormal shape, color, and size, etc.

Flock description and history

- Size of the flock/Number of birds at risk.
- Number (or %) sick.
- Number (or %) dead (distinguish natural deaths vs. culls), in the last week and last 3 months.
- New bird arrivals? Where did they come from; their medication/vaccination history?
- Are there other species on the farm? How much contact is there with these other species and the birds?
- Have the birds been to a show or race recently?
- Have they been moved from one barn/loft to another recently?
- Have they had normal molting and brooding behavior?

Management practices – feed and water

- What type of feed – any recent changes in feed or feed supplier?
- Are there any feed additives? Do you give treats (i.e., scratch, garden vegetables, kitchen scraps)?
- What is the source of water (city, well, surface, cistern, etc.) and any recent changes in the source?
- What watering system is used (i.e., trough, bell, nipple, etc.)? Any changes in the watering system (i.e. from troughs to nipple drinkers)?
- Is the drinking water treated or has the treatment changed? (i.e., filtered, chlorinated, etc.).
- Any water additives used (i.e., apple cider vinegar, vitamin packs, antibiotics, etc.)?

Management practices – housing

- Access to outside.
- Access to open water.
- Access to wildlife (mainly wild bird populations).

Table 18.1 (*Continued*)

History of the problem

- Cage or housing system.
- Litter/bedding materials, (type of bedding, changes, source).
- Other: Ventilation problems, weather or temperature changes, abnormal noise, electrical surges, blackout, recent use of insecticides and/or herbicides, etc.

Source: adapted from Hunter *et al.* [5].

Sample collection

A variety of samples may be collected, including blood, swabs, and tissues samples. Collected samples may be used for a variety of analyses. The tests to be carried out may dictate how the sample is taken, preserved, transported, and processed (Table 18.2). It is therefore important to plan carefully and to make sure that appropriate materials are available when taking samples.

The following rules apply generally to samples for clinical investigation:

1. As a general rule, be prepared to take blood, swabs, and other samples from every case. Have tubes, syringes, bottles, slides, and so on ready before clinical examination starts
2. Use the best-quality equipment, as poor samples can yield erroneous results. Swabs and reagent should be stored properly and used before the expiration date
3. Follow standard techniques when sampling poultry and ensure that this is performed efficiently and humanely
4. Ensure that all samples taken are properly labeled and recorded
5. Monitor the bird carefully following sampling. This is not just good practice, but it may provide further information on the condition of the bird
6. Be aware of the possible risks to human health when taking samples and follow appropriate guidelines. Do not expose staff or owners to hazards unnecessarily.

Collection of blood samples

The total blood volume of a bird is approximately 10% of its body weight, and ideally, for phlebotomy purposes, no more than 10% of the blood volume (or 1% of the body weight) should be removed from healthy adult chickens. If the bird is unhealthy, young, or elderly then even less should be removed. For example, the blood volume of a 3 kg bird (3 x 0.1) is 30 ml, and if it is healthy then a maximum of (30 ml x 0.1) 3.0 ml can be removed. It is possible to run a full hematology

Table 18.2 Guidelines for sample collection and transportation

Sample type	Test type	Suspected condition	Medium	Transportation
Blood	Hematology		Blood collection tube with anticoagulant (i.e., EDTA, heparin)	Chill
	Biochemistry		Blood collection tube with anticoagulant (i.e., EDTA, heparin)	Chill
	Serology	AI, NDV, IBV, Mycoplasma (MG, MS, MM), *Salmonella* Pullorum and S. gallinarum, etc.	Blood collection tube with or without anticoagulant	Chill
	Microbiologic evaluation*	Septic bacteria (i.e., Pasteurella)	Swab in Aimes or Stuart medium	Chill
	PCR	Marek's disease	FTA card[††]	Room temp.
	Toxicology	Heavy metal exposure	Blood collection tube with non-EDTA anticoagulant	Chill
		Trace minerals	Plasma or serum (without RBC)	Chill
Oropharyngeal, sinus, and trachea	Microbiologic evaluation	Mycoplasma sp., coryza	Swab in Aimes or Stuart medium	Chill
	PCR	AI, NDV, IBV, ILT, Mycoplasma	Polyester swab in BHI	Chill
			FTA card	Room temp.
	Virus isolation	AI, NDV, IBV, ILT, pox	Swab in BHI or VTM	Freeze
Fecal	Microbiologic evaluation	Salmonella	Fresh feces in sterile container or swab in Aimes or Stuart medium	Chill
	Parasite	oocysts and eggs	Fresh feces in sterile container	Room temp.
			Slide smear (heat or acetone fixed)	Room temp.
Tissue/carcass	Gross evaluation[†]		Sealed container	Chill
	Histologic evaluation[†]	Infectious and non-infectious conditions	Formalin fixed	Room temp.
			Slide smear (heat or acetone fixed)[†††]	Room temp.
	Microbiologic evaluation	Aerobic and anaerobic microorganisms	Fresh tissue in sterile container or swab in Aimes or Stuart medium	Chill
	PCR	Various viruses and bacteria	Sterile container	Chill
			FTA card	Room temp.
	Virus isolation	Various viruses	BHI or VTM	Freeze
	Toxicology	Various	Consult with lab	Consult with lab

*Microbiologic evaluation includes bacteria and fungus isolation.

[†]Gross and histologic evaluation may be used for the identification of infectious diseases, as well as nutritional, toxic, and neoplastic problems.

[††]FTA cards are used for identification only. Samples submitted on FTA cards cannot be used for isolation of pathogens.

[†††]Special stains such as gram, acid fast, or Giemsa.

profile on 0.3 ml of blood. On the other hand, 0.5–1 ml of whole blood is needed for each immunologic test (on average 0.3 ml of serum or plasma is required per test). Blood collection tubes should only be $^1/_2$–3/4 full.

Blood or serum samples can yield a surprising amount of valuable information. Techniques that can be carried out on blood include hematology, biochemistry, and immunology. Immunology or serology can test for antibodies to various viruses and bacteria, and are very important tests as they can be quick and economical. Blood can also be used to perform microbiology, parasitology, toxicology, and molecular studies.

The site for collection may depend on the age of the bird, species, and competency of the blood collector. The jugular vein is commonly used on day-old or very young birds (Figure 18.1a) but can be used in birds of any age. The wing or brachial vein is the most commonly used to draw blood in poultry (Figure 18.1b). It is also used for collecting blood from small poultry breeds, such as quail, pigeons, or bantam (dwarf) chickens. The metatarsal vein is the vein of choice when collecting blood from waterfowl (Figure 18.1c). Blood can also be collected from occipital venous sinus and heart, but these techniques should be reserved for birds that are used in research, commercial poultry facilities, or prior to euthanasia and, ideally, with the bird under anesthesia. These sites may cause severe injuries or even kill the bird if performed improperly. Vacuum tubes apply too much pressure on the vein, causing collapse, and are not recommended.

Blood samples for hematologic or biochemistry analyses are collected in tubes containing anticoagulant. EDTA and heparin are most common. Because anticoagulants may cause artifacts on the blood cells,

it is recommended to prepare a blood film and to submit this along with the blood sample. The standard two-slide wedge technique works well with avian blood (Figure 18.2).

Blood samples for immunologic tests are collected aseptically in sterile blood collection tubes without anticoagulants, separator tubes, or other non-EDTA/heparin tubes. For maximum serum yield, do not fill the tubes to more than two-thirds of their capacity, and lay the tubes containing freshly drawn blood on their sides. Once the blood has clotted, the serum can be processed from the clot and sent to the laboratory, or the tubes containing clotted blood can be sent to the laboratory. Submit a minimum of 0.3 ml (300 µl) of serum per test, or 1–3 ml of clotted blood, depending on the number of tests requested. Refrigerate the serum or clotted blood until shipment. Whole blood that is kept refrigerated may be used for immunologic testing within 5 days of collection. If testing cannot be performed in that time, separate the serum/plasma and freeze it. Do not freeze serum or clotted blood.

Live bird sampling

Swabs, aspirates, skin scrapings, and biopsies can yield valuable information that may assist in diagnosis. Examples of sites in birds from which swabs can be taken include the oropharynx and cloaca. Aspirates can be taken from joints (synovial fluid) and purulent exudates or transudates in the body cavity. Individual ectoparasites and feathers that contain eggs can be collected in a sealed container and submitted for identification. The region around the cloaca is the best place to examine the bird for mites. Any sample collection from a live bird should be performed after the bird has

(a)   (b)   (c)
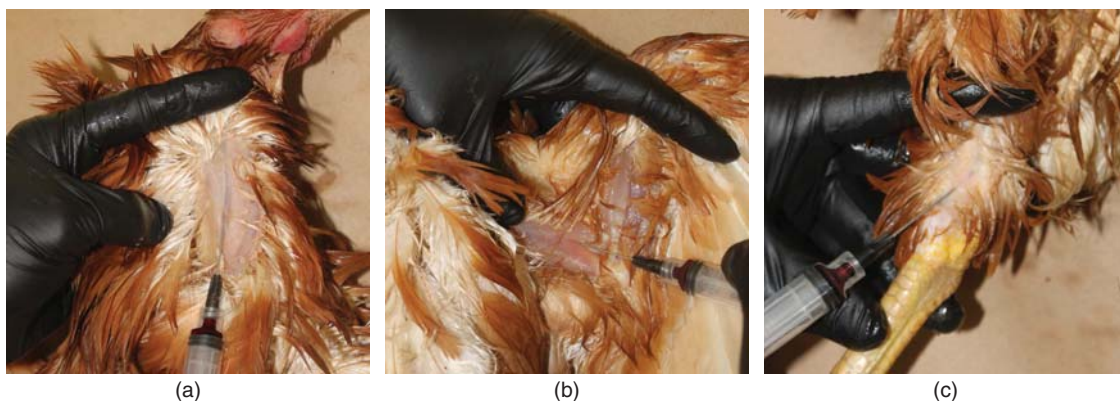
Figure 18.1 Demonstration of phlebotomy sites used in chickens. (a) – Blood can be drawn from the jugular vein of chickens of any age and is especially good for small poultry breeds such as quail. (b) – Blood being drawn from the wing or brachial vein (also known as the basilic or cutaneous ulnar vein). (c) – Blood being drawn from the medial metatarsal vein.
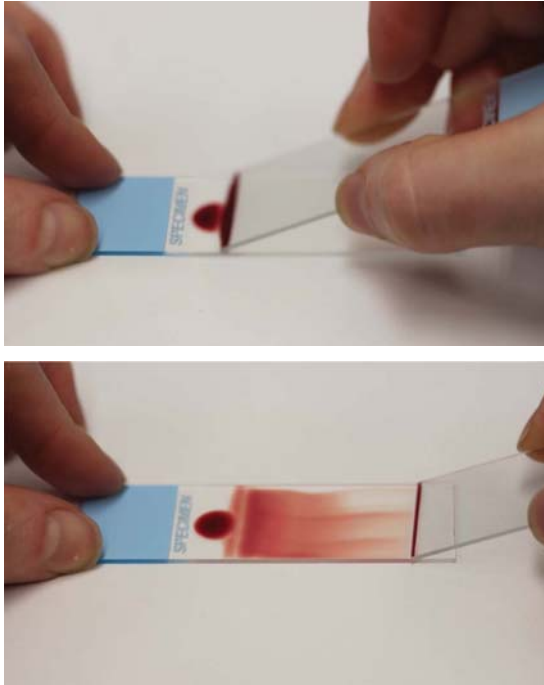
Figure 18.2 Preparing a blood film using avian blood. The standard wedge technique can be used.

Figure 18.3 Demonstration of swabbing the choanal cleft of a chicken.

been fully evaluated. For identification, these parasites should be fixed and stored in 70% alcohol.

Swabs from the oropharynx and cloaca often provide good samples for detection of respiratory viruses (such as avian influenza, Newcastle, or infectious bronchitis), and bacteria including *Mycoplasma* spp. Label containers holding the samples with the animal ID, date of collection, and body region from which the samples are taken.

There are a variety of swabs and media available. In general, avoid using calcium alginate swabs, cotton tips swabs, and swabs mounted on a wooden shaft. Chemicals on these swabs can interfere with some tests. It is also important to select the appropriate transport media for the test. Transport tubes containing Aimes and Stuart media are good not only for bacteria and *Mycoplasma* isolation, but also for virus isolation. Media that contain antibiotics may be good for virus isolation, but they are not suitable for bacteria culture. For anaerobic isolation, Cary-Blair medium is recommended.

Oropharyngeal swabs

The bird should be held securely to prevent stress and injury. It may be tucked under the arm with the ventral side facing up. Use one hand to open the beak (a finger may be inserted in the side to hold the beak open). Use the other hand for sample collection by swabbing the mucosa around the oropharynx and the choanal cleft (Figure 18.3). The goal of swabbing is to collect as much mucus as possible. Avoid blood in the swab, as this interferes with some tests.

Cloaca swabs

The bird should be held securely to prevent stress and injury. Gently lift the tail feathers of the bird with one hand and then insert the swab in the cloaca. Shake off excess fecal material to prevent bacterial contamination.

Postmortem sampling

Practitioners may choose to perform their own necropsy evaluation and then submit tissue samples to the lab. Before necropsy starts, soak the bird's feathers with soapy water to prevent aerosol build-up. If the necropsy

Figure 18.4 List of items recommended to be included in a necropsy field kit: Cooler/container, disposable gloves, pen (waterproof), submission forms, gauze, 70% alcohol, syringes (1, 3, and 6 ml), needles (25, 22, and 21 g), blood collection tubes, dry swabs, tubes with transport media, swabs with media, disposable cotton applicators, sterile scissors, sterile forceps, sterile scalpel blades (and handle), disinfected knife, disinfected shears (or large scissors), 10% formalin, clear sealable plastic bags, magnifying lens, camera/video.

is performed on the premises in which the birds are housed, the examination should be performed away from the pen to reduce the risk of spreading infection. The selected location should also have easy access to water for cleaning and disinfection after the necropsy is completed. Figure 18.4 provides a list of items that are recommended for inclusion in a necropsy field kit.

In sick flocks, it is important to select birds with typical clinical signs. If the main problem is increased mortality without any other sign, choose birds that have died recently for the necropsy examination. It is important to examine *all* organs, with or without gross lesions. In most cases, gross examination does not provide full diagnosis and additional samples need to be tested to confirm a diagnosis and rule out other possible problems. Bird samples that can be submitted to the lab include blood, tissues, and swabs. Environmental samples can be also submitted. If a nutritional problem is suspected, also submit feed and water samples for analysis.

Tissue swab or tissue samples that are submitted for microbiologic evaluation must be collected and transported in a sterile container. Depending on the sample size, media may be needed to keep the sample moist. As indicated above (see Section "Live bird sampling"), Aimes and Stuart media are good not only for bacteria and *Mycoplasma* isolation, but also for virus isolation. Broth that contains antibiotics may be used to preserve samples in which virus isolation is desired, but cannot be used for bacterial isolation.

If samples are submitted for molecular testing, several poultry labs accept not only tissue samples, preserved in standard transport media, but also tissue impressions, scrapings, or swab samples made on FTA™ cards (Fisher Scientific, US, 1-800-766-7000) (Figure 18.5). The FTA™ card allows for easier shipment because there is no required transport media or special preservation (i.e., chill or frozen) of the sample. Additionally, because the pathogen is inactivated on the card, it eliminates the transportation of potentially harmful pathogens or hazardous materials (i.e., phenol). Nucleotide sequencing of PCR products allows for the characterization of the bacteria or viruses detected. On the other hand, if isolation of a specific pathogen is desired (for instance for antibiotic sensitivity testing) submission of a fresh sample or swab in the appropriate transport media is required.

When tissues are submitted for histologic evaluation, the selected tissue sample needs to be converted from a three-dimensional tissue into a stained section, approximately 4 mm thick, adhered to a glass slide that can be examined under the microscope. As with any other sample, it is important to collect a representative tissue sample and use appropriate fixation. The aim of fixation is to maintain fresh tissue in a state that stabilizes its architecture and chemical components in a form that enables it to be processed for histological staining and long-term preservation. Buffered 4–10% formaldehyde solutions are best. Because formaldehyde penetrates tissue at a rate of about 5 mm per 24 hours, it is important to avoid submitting samples that are too big. In general, the volume of fixative should be at least 10 times the volume of the piece of tissue. It is best to add the tissue to the fixative to avoid one surface of the sample adhering to the wall of the container. For larger samples, or multiple tissues following postmortem examination, the volume of fixative required may be too great to send through the mail. The excess fixative should be poured off before submission and the tissues should be sent to the laboratory moist with a small amount of fixative in a sealed container.

If whole birds are submitted to a diagnostic laboratory for evaluation, live and freshly deceased birds should be submitted. In most cases, live birds would have to be hand delivered, as most commercial couriers only accept carcasses. In order to slow down decomposition of dead birds, wet all the feathers on the body with cool soapy water. Place the carcass in a sealed bag and refrigerate as soon as possible. Do not freeze the carcasses unless they are going to be delivered more than 5 days after death. Freezing produces some artifacts, but a decomposed carcass is worse.

Figure 18.5 If samples are submitted for molecular testing, tissue impressions, scrapings, or swab samples can be made on FTA^{TM} cards (Fisher Scientific, US, 1-800-766-7000) as shown without the need for transport media or special preservation of the sample.

Submitting samples

The decision regarding which tests to perform in-house and which to send to other laboratories depends on several factors: Speed of desired results; effect of results on therapeutics decisions; staff ability to perform tests accurately and proficiently; equipment sensitivity and suitability for sample volume; consultation; and trouble shooting. While it is frequently convenient for results of a test to be available during the patient's visit, the results have to be accurate and reliable, as well as cost-effective.

Considerations for choosing an outside laboratory include experience in poultry diagnostics; types of services and tests available; sensitivity and specificity of the tests offered; policies regarding lab supplies and transport media; mailers, billing and invoice policies; turnaround time; and method of reporting (telephone, fax, computer, or mail).

When submitting samples, select specimens and/or freshly dead carcasses that are representative of the problem. Whenever possible, make sure that they have not been treated with antibiotics. Call ahead so that the veterinarian or laboratory knows that you will be submitting samples and to determine the information that is required, so that your submission can be analyzed as quickly as possible.

Obtaining useful/accurate results from the diagnostic laboratory requires good samples and a complete history. It is important to become familiar with laboratory submission and shipment protocol and methods of reporting results. Submitted samples should always be clearly identified and accompanied by a completed submission form that indicates the tests requested, a brief history of clinical signs, differential or tentative diagnoses, and medications being used. A summary of management practices and vaccinations are also helpful. Most diagnostic laboratory submission forms require standard information such as the species, breed, age, sex, weight of the bird(s), flock statistics, and relevant bird/flock history. It is advisable to keep appropriate transport media and shipping containers in the hospital.

Packing and shipping samples

When shipping samples it is important to maintain the integrity of the specimen, prevent leaking, and avoid

Figure 18.6 The laboratory submission form is included in the box with the sample, but it is a good idea to place the paperwork in a sealable plastic bag to prevent the paper from getting wet and becoming contaminated.

cross contamination or misidentification of samples. The following tips will hopefully help limit any damage to samples during transportation to the lab.

If delivering carcasses, prepare the body as described above in the section on postmortem sampling. When ready to ship, place the bagged bird and a few ice packs (e.g., blue ice) within a second plastic bag and seal. Place the bundle in an inexpensive, leak-proof styrafoam cooler, such as those found at a grocery store, and then place in a cardboard box or a cardboard box with additional loosely wadded paper for extra insulation.

If delivering tissues or other samples, place each individual sample in a bag or container that is leak-proof and secure properly. It is good practice to use a double-sealed container or bag within a sturdy cardboard box or padded envelope for shipping. If the sample needs to be refrigerated or frozen, include the appropriate frozen freezer packs to preserve sample integrity during transport.

Remember to complete the lab submission form and include it in the box (Figure 18.6). It is a good idea to place the paperwork in a Ziploc bag to prevent the paper from getting wet and becoming contaminated. Seal the box appropriately and mail or ship the package overnight. If the samples are shipped using a commercial carrier, it is necessary to pack samples in compliance with local postal regulations. Furthermore, if the samples need to be transported refrigerated or frozen, unless you have made previous arrangement with the lab, avoid shipping on Friday, Saturday, or

immediately before a holiday to ensure prompt delivery before the coolant is exhausted.

Conclusions

1. Provide a complete and detailed history of the problem
2. When birds are sick be sure to carefully select representative samples for testing
3. Preserve samples in the appropriate medium for the requested test
4. Identify samples properly and avoid cross contamination
5. Follow local postal regulations for packing when shipping samples using a commercial carrier.

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Interpretation of Laboratory Results and Values

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Introduction

Accurate diagnosis of disease in birds, including poultry, depends upon a series of carefully carried out investigations. Meticulous evaluation of the clinical history and examination of the bird should aid the collection of appropriate samples to carry out a variety of analyses. In the previous chapter, collection and handling of samples was reviewed. In this chapter, an overview of the diagnostic testing (serology, microbiology, histopathology, and molecular biology) that is available and information gained from testing, including normal hematologic and biochemical parameters are discussed.

Hematology

Hematology is the discipline of medical science that studies the blood and blood-forming tissues. Hematology assays seldom provide etiologic diagnosis, but they remain an important tool to evaluate the health in individuals, to monitor the response and progress of therapeutic regimens, and to offer prognosis.

Although chickens have been used as research animal models for establishing normal parameters for other avian species, little information has been published on hematology of domestic poultry in clinical settings. Backyard poultry has become more popular in recent years and visits to veterinarians are increasing. Most of the current information about routine hematologic parameters is extracted from clinical values that have

been established for psittacines. Serology is still the predominant method of disease monitoring in commercial poultry, and examination of blood smears and blood chemistry is rarely performed.

Hematocrit or packed cell volume (PCV)

The PCV is a quick assay that serves to evaluate the hemoglobin concentration and cell count. The PCV is obtained by centrifuging a microhematocrit tube full of blood at 12,000G for five minutes. In general, the average value for poultry is around 40%, but in younger birds the PCV is lower (25–35%) compared to adults (30–45%). Many standard textbooks on avian medicine, such as those edited by Samour (2006), Campbell (2005), and Ritchie *et al.* (1994), provide PCV values for specific poultry species, including chickens, quail, pheasants, and ducks.

Blood smears

Blood smears can provide information about cell morphology, differential white cell count, and blood parasites. A variety of stains can be used to evaluate the air-dried blood or methanol-fixed films. Slides can be stained with Wright's and Giemsa stains or Diff-Quick kit. Avian white cells are more difficult to find than the corresponding mammalian cells. One reason is that avian red cells and thrombocytes are nucleated. In addition, the avian leucocytes are scattered throughout the slide and are not aggregated in the margins of the slide as in mammals. Blood smears are not recommended for manual counting of cells because

they are often inaccurate. However, blood smears can be used if no other systems are available.

Hematogram

Evaluation of the hematogram involves counting the various blood cells as well as cytological evaluation of the cells. Normal blood values of chickens and turkeys are presented in Table 19.1. The processing of avian hematology samples can be performed using automatic analytical systems. However, these systems are useful to differentiate between heterophils and lymphocytes, but are not so useful for segregating all cell types. Otherwise, most laboratories use a manual method, such as the Natt and Herrick method or the Eosinophilic Pipette Method, to differentiate white blood cells.

Erythrocytes are elliptic and large (Figure 19.1). They also have an elongated nucleus. The erythrocyte life span is 20–35 days. The total red blood cell count is not only an important value in hematology, but is also essential for the estimation of the mean corpuscular volume (MCV) and the mean corpuscular hemoglobin (MCH). Many labs today use automatic systems, rather than manual methods. As with PCV, younger birds have lower total erythrocyte count and MCH than adult birds. There are also variations between males and females. As with other bird species, MCV lies between 121–200 fL. Anemia causes changes in these values.

Causes of anemia in poultry are summarized in Table 19.2. Acute blood loss (hemorrhagic and

Figure 19.1 Wright's stain of red blood cells from a 2-year-old female white leghorn chicken with egg-related peritonitis. Note that the cell in the center is a polychromatophil (young RBC) with bluer cytoplasm and plumper, less dense, nucleus than the mature RBCs. It is acceptable to see up to 8% polychromasia in a normal sample. (Thank you to Dr. Jennifer Scruggs for assistance in producing this picture.)

hemolytic anemia) is usually characterized by normal cell morphology, but the PCV and Hb are reduced if the blood sample is taken immediately after the acute event. However, if the sample is taken even a few hours after the loss of blood has occurred the MCV will be increased. In regenerative anemia, where the hemopoietic tissue is trying to replace the depleted erythrocytes, the morphology is characterized by polychromasia of erythrocytes. Polychromatic cells are precursors of mature red blood cells. Sometimes in regenerative anemia, an increase and/or variation of red blood size is also observed. In chronic non-regenerative anemia, the cell morphology may be normocytic or microcytic (MCV value may be normal or depressed) and the reticulocytes are depressed or absent. In most birds, up to 8% polychromasia is acceptable because of the relatively short erythrocyte lifespan and high turnover.

The leucocyte numerical value is variable, even in birds of the same species. Therefore it is more helpful to compare leucocyte counts over time. In general, white blood cell counts vary from 10–45 x 10^3/μL. *Leucopenia* is an overall decrease in the number of all types of circulating white blood cells. It usually occurs with an overwhelming bacterial infection or immunosuppressive diseases. Leucocytosis is an overall increase in the number of white blood cells. This could be a result of infection (bacteria, fungi, or parasite), trauma (resulting in massive tissue necrosis), or neoplasia (with extensive tissue necrosis or because of lymphoid leucosis complex).

Heterophils are the counterpart of the neutrophils that are found in mammalian species. They have a segmented nucleus and cytoplasmic granules that are elongated (fusiform shaped) and highly eosinophilic in chickens and turkeys (Figure 19.2). Heterophils from poultry show increased phagocytic activity and killing activity compared to macrophages. A relative increase of heterophils is suggestive of an acute infection (bacteria or fungal), acute tissue damage, myeloid leukemia, and so on. Coccidiosis and *Escherichia coli* septicemia are examples of infectious diseases in poultry that are associated with an increase of heterophils. A decrease of heterophils may be caused by bone marrow damage, viremia, and aleukemic leukemia.

Lymphocytes are the predominant leukocyte in the peripheral blood of chickens and turkeys. Lymphocytes may be small or medium. Small lymphocytes are round with a round nucleus, high nuclear:cytoplasmic ratio, and a small amount of basophilic cytoplasm (Figure 19.3). Medium lymphocytes are more abundant, may be difficult to differentiate from monocytes, and may be increased in some infectious and metabolic

Table 19.1 Hematologic values for the domestic chicken (*Gallus gallus domesticius*), turkey (*Meleagris gallopavo*), quail (*Coturnix* spp.), and ring-necked pheasant (*Phasianus colchicus*)

Analyte (abbreviation)	Units, SI [conventional]	Chicken	Turkey	Quail	Ring-necked pheasant
White blood cell count (WBC)	x 10^9/L = [x 10^3/µl]	9-32[17] 12-30[16]	16-25.5[17] 10.3-46.5[16]	12.5-24.6[16]	18-39[16]
Heterophils absolute	x 10^9/L = [x 10^3/µl]	3-6[16]	4-27.6[16]		
Heterophils	Proportion of 1.0 [%]	0.15-0.5 [15-50][17]	0.29-0.52 [29-52][17]	0.25-0.50 [25-50][17]	0.12-0.30 [12-30][17]
Lymphocytes absolute	x 10^9/L = [x 10^3/µl]	7-17.5[16]	4.2-34.3[16]		
Lymphocytes	Proportion of 1.0 [%]	0.29-0.84 [29-84][17]	0.35-0.48 [35-48][17]	0.50-0.70 [50-70][17]	0.63-0.83 [63-83][17]
Monocytes absolute	x 10^9/L = [x 10^3/µl]	0.15-2[16]	0-3.9[16]		
Monocytes	Proportion of 1.0 [%]	0.001-0.07 [0.1-7][17]	0.03-0.1 [3–10][17]	0.005-0.038 [0.5-3.8][17]	0.02-0.09 [2–9][17]
Eosinophils absolute	x 10^9/L = [x 10^3/µl]	0-1[16]	0-1[16]		
Eosinophils	Proportion of 1.0 [%]	0-0.16 [0-16][17]	0-0.05 [0-5][17]	0-0.15 [0-15][17]	0[17]
Basophils absolute	x 10^9/L = [x 10^3/µl]	Rare[16]	0-2[16]		
Basophils	Proportion of 1.0 [%]	0-0.08 [0-8][17]	0.01-0.09 [1–9][17]	0-0.0015 [0-0.15][17]	0-0.03 [0-3][17]
Hematocrit (packed cell volume=PCV)	Proportion of 1.0 [%]	22-55[17] 22-35[16]	30.4-45.6[17] 31-42[16]	30-45.1[17]	-
Hemoglobin	g/L [g/dl]	70-186[17] [7-18.6][17] 70-130[16] [7–13][16]	88-134[17] [8.8-13.4][17] 103-152[16] [10.3-15.2][16]	107-143[17] [10.7-14.3][17]	80-112[17] 8.0-11.2][17]
Red Blood Cell (RBC) Count, (Erythrocytes)	x 10^{12}/L = [x 10^6/µl]	1.3-4.4[17] 2.5-3.5[16]	1.74-3.7[17] 2.3-3.8[16]	4-5.2[17]	1.2-3.5[17]
Reticulocytes	Proportion of 1.0 [%]	0-0.6[16]	-	-	-
Mean Corpuscular Volume (MCV)	µm³ = fL	100-139[17] 90-140[16]	112-168[17] 129[16]	60-100[17]	-
Mean Corpuscular Hemoglobin (MCH)	pg/cell	25-48[17] 33-47[16]	32-49.3[17] 42.9[16]	23-35[17]	-
Mean Corpuscular Hemoglobin Concentration (MCHC)	g/dl	20-34[17] 26-35[16]	23.2-35.3[17] 29.6[16]	28-38.5[17]	-

Sources: Wakenell, Patricia, S. [16], Hawkins, M.G., Barron, H.W., Speer, B.L., Pollack, C. and Carpenter, J.W. [17].

Table 19.2 Causes of anemia in poultry

Type of anemia	Causes
Hemorrhagic	Hemorrhage: Trauma, cannibalism, aortic rupture, fatty liver hemorrhagic syndrome
	Parasitic: Ticks, mites, helminths, coccidiosis
Hemolytic	Parasites: Hemoprotozoa
	Bacterial infections: Salmonellosis, spirochetosis
	Toxic: Aflatoxin, lead (acute), copper, dimethyl disulfide, phenylhydrazine
Nutritional	Minerals: Iron deficiency (ochratoxin-induced), copper deficiency
	Vitamins: Pyridoxine, folic acid
Anaplastic/ pancytopenia	Viral infections: Chicken anemia virus, infectious bursal disease, adenovirus, retrovirus, Marek's disease virus
	Toxic: Sulfonamides, lead (chronic), trichothecene mycotoxin, penicillum citrium, irradiation
Inherited	
Undetermined	

Figure 19.3 Wright's stain of two lymphocytes from a 2-year-old female white leghorn chicken with egg-related peritonitis. Note the scant cytoplasm and pseudopodia. (Thank you to Dr. Jennifer Scruggs for assistance in producing this picture.)
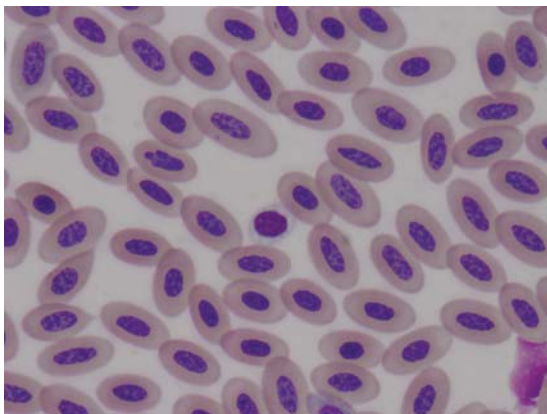
Figure 19.4 Wright's stain of thrombocyte from a 2-year-old female white leghorn chicken with egg-related peritonitis. Note the scant clear cytoplasm and small size of the cell compared to the RBCs. (Thank you to Dr. Jennifer Scruggs for assistance in producing this picture.)

Figure 19.2 Wright's stain of two heterophils from a 2-year-old female white leghorn chicken with egg-related peritonitis. Note the rod-shaped pink (eosinophilic) granules. (Thank you to Dr. Jennifer Scruggs for assistance in producing this picture.)

diseases, as well as in lymphoid leucosis; while they are decreased in cases of stress, uremia, and immunosuppressive conditions such as Marek's disease, chicken infectious anemia, and infectious bursal disease.

Thrombocytes are also nucleated (Figure 19.4). They may be confused with small lymphocytes. Thrombocytes participate in hemostasis and also have phagocytic properties. Thrombocytosis or increase of thrombocytes is observed in response to bacterial infection and as a result of excessive hemorrhage.

Monocytes are the largest of the leucocytes and are macrophages. As mentioned above, they need to be differentiated from lymphocytes. Monocytes are round cells, and usually have indented nuclei and abundant pale cytoplasm, which contain fine azurophilic granules (Figure 19.5). An increase of these cells or monocytosis usually indicates a chronic bacterial infection or tissue necrosis. It is seen in cases of Chlamydiosis and Mycobacteriosis.
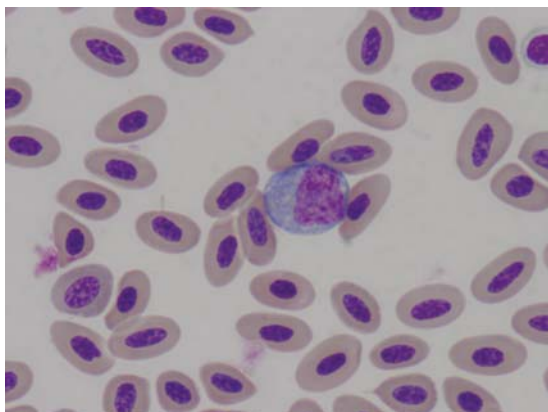
Figure 19.5 Wright's stain of a monocyte from a 2-year-old female white leghorn chicken with egg-related peritonitis. Note the relatively large size of this cell and the abundant light blue foamy cytoplasm, and a nucleus that is less dense than that of a lymphocyte. (Thank you to Dr. Jennifer Scruggs for assistance in producing this picture.)

Figure 19.6 Wright's stain of a basophil and a heterophil from a 2-year-old female white leghorn chicken with egg-related peritonitis. Note that the basophil has dark purple cytoplasmic granules. (Thank you to Dr. Jennifer Scruggs for assistance in producing this picture.)

Eosinophils have irregular shape and their granules are round. The function of *eosinophils* in birds is unclear; therefore an increase of eosinophils may not necessarily be an indication of a parasitic infection. In raptors, eosinophils can increase in response to trauma. It is believed that basophils have the same function in birds as in mammals, and they are involved in early acute inflammatory reaction and in reaction to neoplasms with significant tissue necrosis.

Basophils are round cells with deeply basophilic granules in the cytoplasm (Figure 19.6). Basophils are one of the first leucocytes to enter tissue as part of an early inflammatory response.

Blood chemistry

In many species, blood clinical chemistries are essential for medical assessment and diagnosis. For some diseases, they are essential for the diagnosis and treatment of affected birds. On the other hand, the results of the biochemical analysis in poultry should be taken as a rough guide to a final diagnosis. Blood clinical chemistries have rarely been used to assess diseases in poultry. There is only limited information regarding chemistry in poultry, as these species have been traditionally considered food animals and diagnosis has been attained through other means, such as serology investigation and necropsy. Other factors that contribute to the lack of established standard values for poultry is the variability among breeds or strains of birds, wide variation in the physiological conditions (i.e., age, gender, egg laying, molting), and environmental differences (i.e., husbandry, nutrition).

In recent years progress has been made in the knowledge of standard blood chemical values in poultry thanks to the use of handheld analyzers such as i-STAT® (Table 19.3). This analyzer allows blood analysis directly in the field, and concerns regarding sample handling and transportation, which could alter blood gasses, potassium, or ionized calcium concentrations, are minimized.

Blood plasma proteins participate in the maintenance of colloid osmotic pressure, gluconeogenesis, transport of minerals and hormones, and production of enzymes and immunoglobins. Because plasma/serum proteins have numerous roles in the physiology of birds, they are a significant indicator of the condition of animals' health. Total plasma protein is probably the most used. As with most other avian species, the total value is 3–5 g/dl and concentration of proteins is significantly lower in young animals than in adults. Serum protein increases with prior egg laying and with age. As the liver produces most of the serum proteins, a reduction in total serum protein may be one indicator of liver disease. Other possible causes of reduced serum protein are anemia, malnutrition, malabsorption (secondary to gastrointestinal disease), intestinal parasitism, glomerulonephritis, severe trauma, prolonged stress, and heavy metal poisoning. On the other hand, hyperproteinemia

Table 19.3 Blood biochemical and gas values for the domestic chicken (*Gallus gallus domesticius*), turkey (*Meleagris gallopavo*), quail (*Coturnix* spp.), and ring-necked pheasant (Phasianus colchicus)

Analyte (abbreviation)	Units, SI [conventional]	Chicken	Turkey	Quail	Ring-necked pheasant
Calcium	mmol/L [mg/dL]	3.3-5.9[17] [13.2-23.7][17]	2.9-9.7[17] [11.7-38.7][17]	-	-
Ionized calcium	mmol/L [mg/dL]	1.20-1.73[16] [4.8-6.9][16]			
Phosphorus	mmol/L [mg/dL]	2-2.5[17] 1.3-1.8[17] [6.2-7.9][17] [4.1-5.7][16]	1.7-2.3[17] [5.4-7.1][17]	-	-
Sodium	mmol/L = [mEq/L]	131-171[17] 141.6-152.6[16]	149-155[17]	180[17]	-
Potassium	mmol/L = [mEq/L]	3-7.3[17]	6-6.4[17]	1.4[17]	-
Bicarbonate	mmol/L = [mEq/L]	18.9-30.3[16]			
pH		7.28-7.57[16]	-	-	-
Carbon dioxide partial pressure	mmHg	25.9-49.5[16]			
Oxygen partial pressure	mmHg	32.0-60.5[16]			
Carbon dioxide	mmol/L = [mEq/L]	19.9-31.5[16]			
Base excess	mmol/L	6.8-7.2[16]			
Oxygen saturation	%	70.6-93.3[16]			
Creatinine	µmol/L [mg/dL]	80-160[17] [0.9-1.8][17]	71-80[17] [0.8-0.9][17]	4.4[17] [0.05][17]	-
Uric acid	µmol/L [mg/dL]	149-483[17] [2.5-8.1][17]	203-310[17] [3.4-5.2][17]	322-328[17] [5.4-5.5][17]	334-357[17] [5.6-6][17]
Glucose	mmol/L [mg/dL]	12.63-16.69[17] [227-300][17] 11.53-14.51[16] [207.2-260.7][16]	15.30-23.65[17] [275-425][17]	14.41-17.36[17] [259-312][17]	-
Cholesterol	mmol/L [mg/dL]	2.3-5.5[17] [86-211][17]	2.1-3.8[17] [81-129][17]	-	-
ALT	U/L = [IU/L]	-	-	6.5-9.6[17]	-
AST	U/L = [IU/L]	-	-	402-422[17]	-
GGT	U/L = [IU/L]			1.7-1.9[17]	
Total protein	g/L [g/dL]	33-55[17] [3.3-5.5][17]	49-76[17] [4.9-7.6][17]	34-36[17] [3.4-3.6][17]	45-51[17] [4.5-5.1][17]
Globulin	g/L [g/dL]	15-41[17] [1.5-4.1][17]	17-19[17] [1.7-1.9][17]	-	19-21[17] [1.9-2.1][17]
Albumin	g/L [g/dL]	13-28[17] [1.3-2.8][17]	30-59[17] [3-5.9][17]	13-15[17] [1.3-1.5][17]	26-27[17] [2.6-2.7][17]

Sources: Wakenell, Patricia .S. [16], Hawkins, M.G., Barron, H.W., Speer, B.L., Pollack, C. and Carpenter, J.W. [17].

is encountered in cases of dehydration (PCV is also elevated), shock, or acute infection.

To evaluate the health status of birds, in addition to the total concentration of total plasma proteins, it is important to determine the concentration of individual fractions. Albumins serve as a source of amino acids during insufficient intake of food, and participate in transporting fatty acids, minerals, vitamins, and thyroid hormones. Inflammatory processes induce an increase of globulins and a decrease of albumin.

Serologic investigation

The laboratory examination of serum sample can be a valuable aid for assessing if a bird has been exposed to a specific pathogen. Serology is most powerful and accurate when used in association with other sources of information (i.e., clinical signs, production data, vaccination, necropsy findings, etc.) When poultry are moved between states or out of the country, serologic testing may be required to demonstrate that birds have not been exposed to certain diseases. There are a variety of serological tests available, such as enzyme-linked immunosorbent assay (ELISA), agar gel immune diffusion (AGID), agglutination test, hemagglutination inhibition (HI), and complement fixation (Table 19.4).

It is essential to know the uses and limitations of this procedure to ensure that maximum benefit is realized. Most serological tests have the following limitations:

1. Most tests only evaluate circulating antibodies, and take no account of mucosal antibody or cell mediated immunity. Serology testing has little value for diseases in which protection depends on cellular immunity (such as infectious laryngotracheitis or pox virus infection), rather than antibody production

2. Lack of antibodies against a particular pathogen may be interpreted as the lack of exposure to such a pathogen. However, in acute infections, antibodies may not be measurable at the time of testing. Birds remain negative for at least 4 days after infection. For clinical diagnostic situations, paired serum testing is critical. Paired serum involves testing of serum samples collected two weeks apart, during the acute and convalescent periods of a disease. By using a paired serum sample the bird can be shown to be actively responding to the infection immunologically by a rising titer. On the other hand, if titers plateau or decrease it is generally an indication that no recent exposure has occurred. The antibodies persist in the circulating blood of the bird for months, even after the pathogen is no longer present

3. Antibodies to antigenically related agents might cause confusion as a result of cross-reactions

4. Serologic assays cannot "type" the immune responses against specific variants, such as detection of the H or N group of avian influenza or the strain of infectious bronchitis

5. There is an inherent risk of false positive and false negative reactions.

An agglutination test (Figure 19.7) is probably the simplest of the serologic tests. It is a qualitative method and cannot be automated. This assay is inexpensive. It is commonly used for the detection of antibodies of *Salmonella* Pullorum (pullorum disease), *S*. Gallinarum (fowl typhoid), *Mycoplasma gallisepticum* (MG), and *M. synoviae* (MS). However, because false positive reactions may occur, it is only used as a screening tool. Positive results need to be confirmed with other serologic tests.

The HI test is considered the gold standard for serology assays. It is a quantitative assay, because HI is highly specific and consequently requires specific reagents for each antigen or antiserum tested. There are only a few false positive reactions. It requires homologous red blood cells for the test at hand. It is used for determination of the avian influenza subtype (Hemagglutining and Neuraminidase groups), infectious bronchitis serotype (i.e., Arkansas, Connecticut, Delaware, and Massachusetts), and confirmation of MG and MS. In general, titers >1:8 are suggestive of previous exposure.

ELISA (Figure 19.8) is currently the preferred serologic testing method in commercial poultry. ELISA is a qualitative method that is easily automated and is more sensitive than other assays. It measures IgY (equivalent to the IgG antibodies in mammals) (Table 19.5), Because it can be automated, large numbers of samples can be processed in a single day to detect and measure
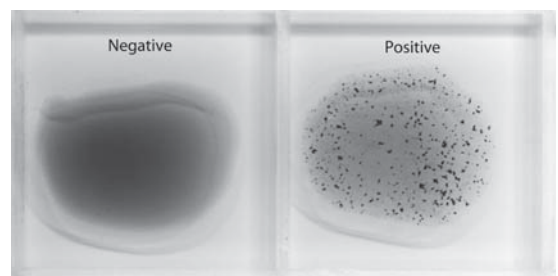
Figure 19.7 Agglutination test. The interaction of particulate antigens with antibodies leads to agglutination reactions. In a positive agglutination reaction (right) sufficient antibodies are present in the serum to link the antigens together, forming clumps of antigen-antibody complexes.
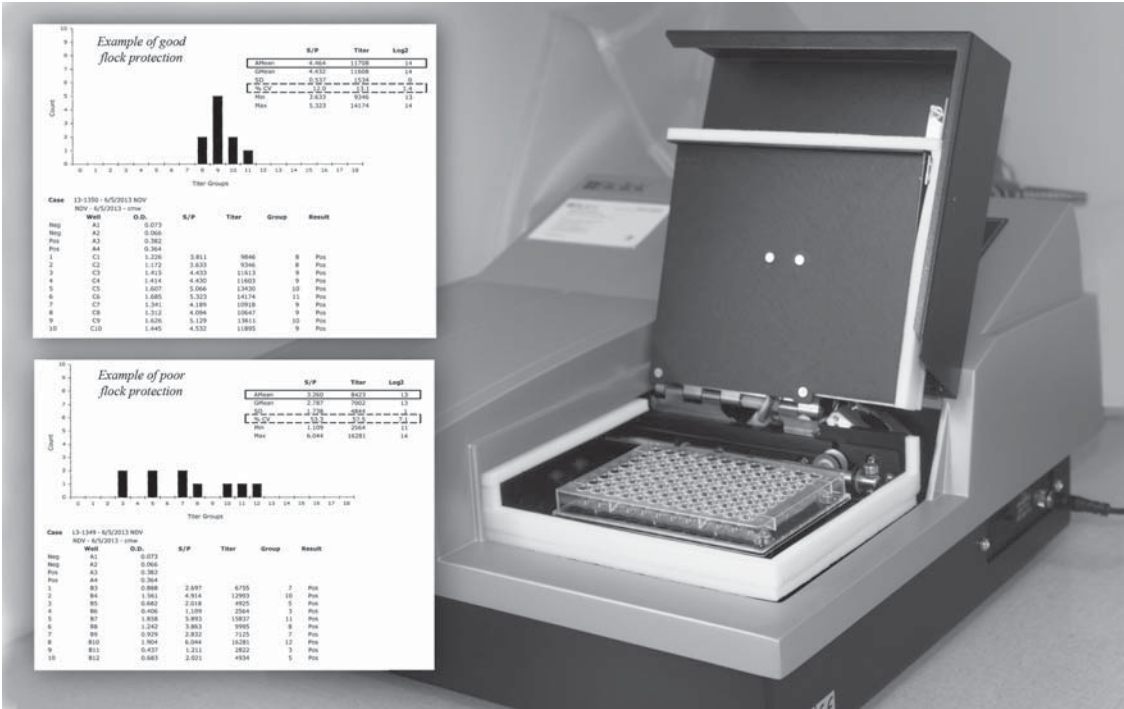
Figure 19.8 Enzyme-linked immunosorbent assay (ELISA) equipment and examples of good (top) and poor (bottom) results in a poultry flock. The mean titer of the tested birds within a flock indicates the strength of the antibody response of a flock. It basically provides a measure of the immune response of the flock. The coefficient of variation, or CV%, provides an indication of mean titer response variability of a flock. The lower the %CV, the more uniform the distribution of titers and the better the vaccination. For most diseases, the %CV after a correctly applied vaccination should be less than 40%. A CV% greater than 60% indicates uneven immune status of the flock or poor vaccination.

Table 19.4 Selected serologic immunoassays available for poultry

Disease	Serologic test	Comments
Avian Influenza	ELISA	Chickens and turkeys only. If positive, confirm with AGID.
	AGID	All species. If positive perform HI.
	HI	H and N groups determination
Newcastle disease	ELISA	Chickens and turkeys only
	HI	All species
Infectious bronchitis	ELISA	Chickens only
Mycoplasma	ELISA (MG, MS)	Chickens and turkeys only. If positive confirm with HI
	Agglutination (MG, MS)	All species. If positive confirm with HI
	HI (MG, MS, MM)	All species
Salmonella Pullorum/thyphoid	Agglutination	If positive confirm with microtiter
	Microtiter	
Reovirus (viral arthritis)	ELISA	Chickens only
Infectious bursal disease	ELISA	Chickens only
Avian encephalomyelitis	ELISA	Chickens only

antibodies against a variety of poultry pathogens. It is known to lead to false positive reactions occasionally; consequently positive results may need confirmation by different methods. Finally, ELISA is species specific and most of the assays have been validated for chickens and turkeys. ELISA cannot be performed to measure antibodies in pigeons, waterfowl, and game fowl. Other types of assays can also be sensitive, specific, and possibly less costly, but they often require more complex procedures and intensive labor.

The AGID test (Figure 19.9) is based on the passive diffusion of soluble antigens and/or antibodies toward each other, leading to their precipitation in a gel matrix. It is commonly used for the detection of avian influenza antibodies. It is a semi-quantitative method and cannot be automated. In addition, it is difficult to interpret; this is especially true of weak positive sera.

Histology

Samples that are taken for histopathology can provide diagnostic results that are timely and relatively inexpensive for the clinicians when performed properly. Histologic examination allows differentiation between infectious and non-infectious conditions. It can also narrow down whether an infectious problem is caused by bacteria, virus, fungus, or parasite, as well as if a non-infectious condition may be caused by a neoplasia, nutritional deficiency, or toxicity. Histology is an invaluable tool for diagnosing such common diseases in backyard chickens as Marek's disease and ovarian carcinoma. Special stains performed on the sections, such as Gram, Giemsa, PAS, GMS, Acid Fast, Tri-chrome, Warthin Starry, Von Kossa, Brown and Hopps, and Perl's Iron, can further identify or narrow down the possible causes of the disease. Specific pathogens, such as Avian paramyxovirus, Avian Influenza, Infectious bronchitis, and *Mycoplasma gallisepticum*, can be identified in the tissues by using immunohistochemistry. But these tests are not available in most laboratories. Formalin-fixed and paraffin-embedded samples can also be cut into sections for molecular testing, from cases where fresh samples are not sent, cannot be shipped, or are not available, to identify potential pathogens.

Microbiological investigations

Poultry become infected with multiple microorganisms from contact with wild birds, rodents, humans, and the environment. Birds that are newly introduced
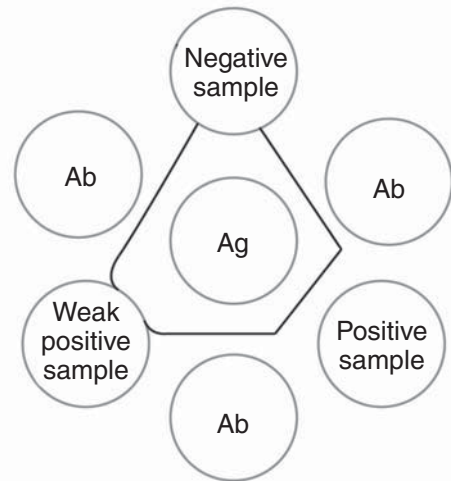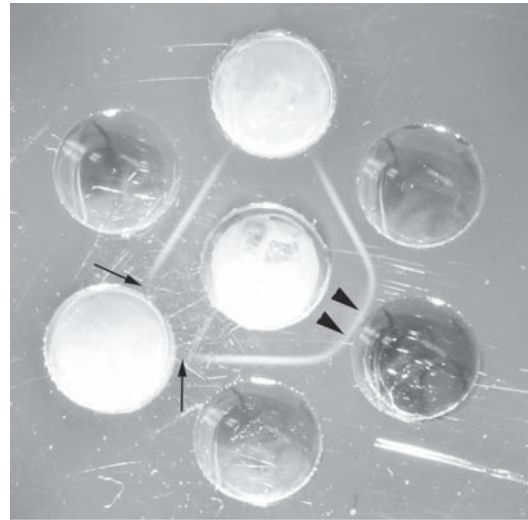
Figure 19.9 Agar gel immune-diffusion (AGID) is the passive diffusion of soluble antigens and/or antibodies toward each other leading to their precipitation in a gel matrix. There is slight bending of the lines associated with the weak positive sample (arrows), while in strong positives the precipitation line is clearly formed between the antigen and positive sample wells (arrowheads). Ab, antibody; Ag, antigen.

in the aviary can bring a disease such as infectious laryngotrachetis or mycoplasma. The primary goal of microbiologic testing is the detection of bacteria, viruses, and fungi that are possibly involved in a disease process.

The harvesting of the microorganism from a particular site is easier than determining whether the finding is significant. In reality, microbiologic testing can be misused as an indication of avian health. It is very easy to make a quick decision and conclude that a particular

Table 19.5 Guidelines for interpretation of ELISA titers in poultry. In general, titer group 0 is interpreted as negative (not exposed to pathogen) and titer groups >1 are positive (exposed or vaccinated)

Doubling dilutions	Log titers	Interpretation	
1	0	Negative or no exposure	Negative or no exposure
2	1	No immunity	Maternal Immunity (*up to 4 weeks of age*)
			Live Prime Vaccine
4	2		
8	3	Poor immunity	
16	4		
32	5		2nd Live Prime Vaccine
64	6	Protection Against mortality	
128	7		
256	8		
512	9		Prime Plus Killed Vaccine
1024	10	Field Challenge	
2048	11		

microorganism is the sole or primary cause of the disease process. The best way to interpret microbiology findings is to closely examine the patient, even before attempting to identify a possible pathogen. Then the clinician should establish if the bacteria, viruses, or fungi are actually involved in the clinical condition. Additionally, the clinician needs to determine whether the microorganisms that are detected are the primary cause of the disease or are secondary to another condition (i.e., malnutrition, contamination of the water source, immunodepression).

Bacteria culture

The harvesting of bacteria from a particular site and their subsequent identification and testing for antibiotic susceptibility is a relatively easy process; more difficult is determining the significance of the findings. In reality, bacterial culture/sensitivity testing may be misused as an indicator of avian health. The best way to interpret bacterial findings is to closely examine the patient, even before attempting to identify a possible pathogen. The first question to be addressed is whether or not there are any visible clinical signs of infection.

A bacterial culture from a perfectly normal oropharynx may be irrelevant, regardless of the bacteria that might be isolated. It should be noted that a wide variety of bacteria are normal commensals of the gut of birds. Apparently undesirable bacteria may actually be harmless, or they may be present secondary to another condition (i.e., malnutrition, contamination of the water source). For instance, *Escherichia coli* is a normal inhabitant of the gut in poultry and *Salmonella* sp. may not cause enteritis in mature poultry. However,

these bacteria may be pathogenic if the bird is stressed. Furthermore, when taking samples from postmortem specimens, one should take into account that cultures obtained from specimens that have been dead for more than 24 hours may not be representative. Some organisms, such as *Proteus* sp., present in the gut, may rapidly invade other organs after death, and may overgrow other pathogens on a culture plate. Only when a direct specific disease condition can be directly linked to a possible pathogen should a culture be considered significant. Rarely should a patient be treated with antibiotics merely because of the presence of suspect bacteria.

The laboratory may not be able to recover certain bacteria, even if present, or if the patient has been treated with antibiotics. Antibiotics in the sample may inhibit the growth of bacteria. Furthermore, bacteria may not be isolated if the specimen is not cultured in the proper media and under optimal environmental conditions. Bacterial cultures are routinely set on standard aerobic media, such as streaking of the sample onto Blood and McConkey agars and incubating at 37°C for 24 hours. However, some bacteria may require special conditions in order to grow in the laboratory. Failure to use the appropriate culture medium or environment may result in no recovery of the desired microorganism. A few important bacterial groups that may be found in poultry samples, do not grow in standard media and/or conditions, and require special handling to be isolated include Avibacterium, Campylobacter, Clostridium, and Mycoplasma. When submitting to a laboratory for bacteria isolation, the submission form should include a

list of suspected conditions or pathogens to avoid lack of isolation.

Antibiotic sensitivity testing (aka Kirby Bauer testing or disk diffusion antibiotic sensitivity testing) may be performed from a particular bacterial isolate. Because poultry have been traditionally considered a food animal, there are very few approved antibiotics. A list of currently approved antibiotics for poultry and their withdrawal time is available online at http://www.farad.org/members/index.html. Practitioners for back yard poultry flocks must be cautious when prescribing antibiotics to these birds. Many people who keep poultry in their backyard consume their eggs. Some of these drugs may pass through the egg, and may cause the development of antibiotic-resistant bacteria, or induce allergic reactions, if the eggs containing antibiotics are consumed. Please refer to the chapter on appropriate drug use in this book.

Parasite examination

Parasites that affect poultry range from single-celled protozoans to multicellular helminthes and arthropods. Parasitic life cycles may be direct or indirect. Parasites also may invade various organs. It is important to stress that identifying a parasite (or parasite egg) does not always mean clinical disease. Many parasites coexist with their hosts without causing pathologic changes. For instance, poultry older than four months of age have usually built up immunity to coccidia; therefore detection of a few coccidial oocysts in their feces may not be relevant.

Poultry may become infected with these parasites from multiple sources, that is, contaminated equipment, humans, or the feces of other animals. Parasitic infections may be diagnosed through examining samples from living animals or through necropsy of affected individuals or representatives of the flock.

In living animals, the most common diagnostic samples are feces, blood, and skin/feathers for the detection of parasites, eggs, or intermediate life forms. From dead birds, investigation can be performed from gross and histopathologic observations. It is a good idea to consult with a specialist who can help to classify the parasite.

Fecal samples should be collected and examined on a routine basis. Standard flotation and centrifugation concentrating techniques are usually performed from fecal samples to look for protozoan (Cryptosporidium, coccidia, and giardia) and nematode eggs (Table 19.6). Identification of ova or oocytes can be performed by flotation by examining fresh feces (Figure 19.10). Please refer to the Parasitology chapter for further detail.

A direct smear is best for detecting motile protozoa (i.e., *Coclosoma*, *Giardia*, *Trichomonas*). Samples are not

Table 19.6 Fecal flotation protocol

Preparation of flotation medium
Heat 355 ml (1.5 cups) tap water and add 454 g (1 lb) sugar while stirring.
Continue stirring the solution on low heat until sugar is dissolved.
The solution will last about three weeks.

Sample preparation
Transfer approximately one gram of feces into a vacutainer tube.
    Note: If needed, filter sample out coarse material using a funnel covered with the gauze.
Fill tube half way with the sugar solution and vortex. Continue filling the tube until a slight positive meniscus is formed.
Let rest for 20 minutes.
Then float a cover slip over the tube, making contact with the sample.
Examine microscopically for ova, cysts and oocytes.

Interpretation
Positive: Presence of ova, cysts, and/or oocytes and identified to genus. *For coccidia it can be qualified as high: >50, moderate: 20–50, low: <20 oocytes per field at a 100x.*
Negative: No ova, cyst or oocysts observed.

diagnostic if they are more than 15 minutes old. Feces or oral scrapings are mixed with lactate Ringer's solution or physiologic saline solution, rather than tap water. Examine the sample under the microscope and look for flagellar movement. To confirm the morphology of the parasites, a slide with the sample can be fixed in polyvinyl alcohol and stained with trichrome.

Blood films are used to detect hematozoa. Some examples of the parasites found in poultry are Plasmodium (mostly in pigeons) and Leucozytozoon (occasionally seen in turkeys). Giemsa or Wright's stains provide excellent results. It is also possible to use the Diff-Quick kit to stain the blood film.

Fungal culture

The primary fungi of concern in poultry are *Candida albicans* and *Aspergillus fumigatus*. When isolated, their significance must be interpreted in light of clinical signs, hematology data, and so on. As it occurs with other microorganisms, their presence alone does not confirm disease.

Viral culture

Viruses can be cultured from a variety of tissues in chick embryos and living cells. Embryos from specific-pathogen-free dams should be used for virus isolation. Samples may be from ante- and post-mortem
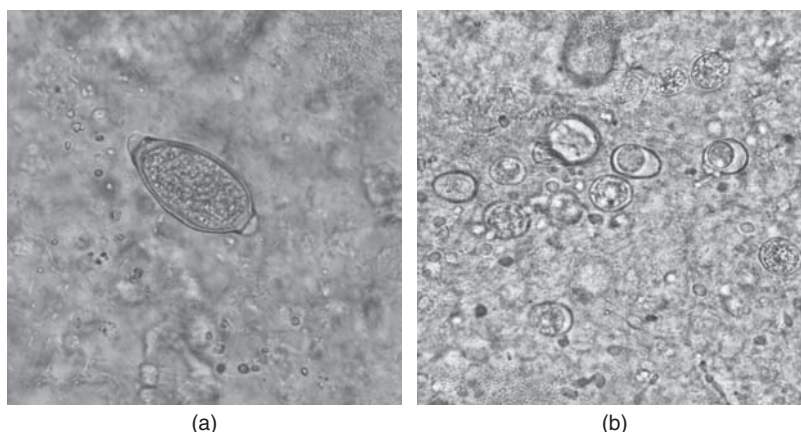
Figure 19.10 Example of parasite eggs found in fecal samples of poultry: (a) *Capillaria* sp. form a pigeon fecal sample. *Note* the bi-polar poles. (b) Coccidia (*Eimeria* sp.) from a chicken fecal sample. *Note* that a couple of stages (oocyst and schizoonts) are present.

cases. Virus isolation is currently offered in a few laboratories. The test is time consuming, it may take weeks to get results back, and it is expensive. It may require inoculation of embryos at different ages or routes and the use of multiple cell cultures. However, there are always emerging viral diseases and virus isolation always plays a role in the investigation of poultry illness. If a suspected foreign animal disease or a disease that would be costly to the poultry industry is suspected, such as END(exotic Newcastle's disease), AI (avian influenza), mycoplasma, or ILT (infectious laryngotracheitis), please contact your state veterinarian and state laboratory regarding testing, which is often available at no or minimal cost. Please refer to the AI chapter for further detail on this important pathogen.

Molecular testing

Diagnostic microbiology has been transformed with the use of molecular technology. Molecular techniques have become a crucial tool for identifying microorganisms that are important in animal production and health. Molecular techniques can complement the work of immunologists, microbiologists, and veterinarians. The incorporation of molecular techniques has been of great importance in the identification and characterization of many viruses, including avian influenza and velogenic Newcastle.

Essentially, molecular techniques are based on a polymerase chain reaction (PCR), a method for detection of specific DNA or RNA (Figure 19.11). There are many modifications of the PCR method, that is, conventional PCR, multiplex PCR (simultaneous amplification of

several DNA sequences), nested PCR, semiquantitative PCR, reverse transciptase PCR or RT-PCR (for the amplification of RNA), and real time PCR (aka quantitative PCR).

As these techniques develop, the number of tests available for the identification of pathogens, including viruses, bacteria, parasites, and fungi in any samples (i.e., blood, feces, exudates, tissues) increases. PCR has advantages as a diagnostic tool in conventional microbiology, particularly in the detection of slow-growing or difficult to cultivate microorganisms, or under special situations in which conventional methods are expensive or hazardous. As a result of the stability of DNA, nucleic-acid based detection methods can be also used when inhibitory substances, such as antibiotics or formalin, are present. In summary, PCR is sensitive, specific, and can provide quick results.

However, it is important to point out that molecular techniques should be used as another tool for diagnosis and the results from these tests should be interpreted in the context of the other findings, that is, clinical signs, serology, and pathology. PCRs only indicate the presence of DNA or RNA of a potentially infective microorganism; they do not give any indication of the activity of the infection.

Conclusions

1. An accurate diagnosis in poultry is usually obtained after a series of evaluations of multiple analyses. No one test is more important than another
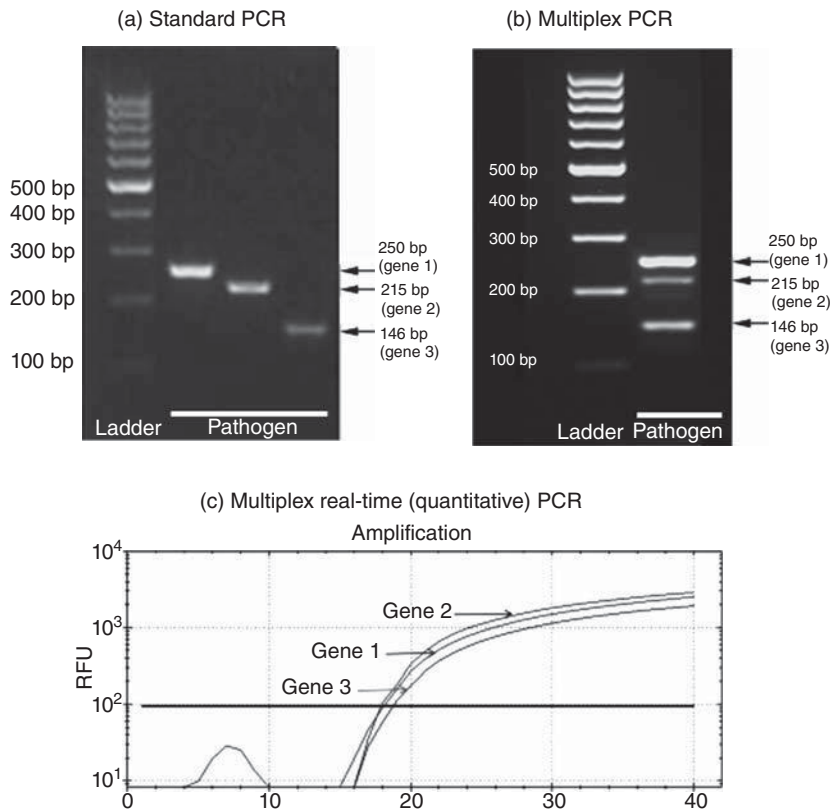
Figure 19.11 Different outputs for a given pathogen using different PCR methods. (a) For standard PCR a single genetic marker is tested per reaction. It needs three reactions (lanes) to identify the three markers. (b) For multiplex PCR multiple, the three genetic markers are tested in a single reaction (lane). (c) In a multiplex real time PCR, the presence of the three genetic markers is also tested in a single reaction, in addition the concentration of the pathogen can be calculated. In (a) and (b) only positive (presence) or negative (absence) results are provided.

2. Although chickens have been extensively used in research settings, there is limited published information on hematology of domestic poultry in clinical settings. However, because of technical advances and the interest in backyard poultry as pets the situation is changing

3. Traditionally, serology has been the preferred method of monitoring health and disease in poultry species

4. Histologic examination allows morphologic examination of the tissues and allows for the differentiation of infectious from non-infectious disorders

5. Microbiologic testing is used for the detection of bacteria, viruses, and fungi. The role a microorganism plays on a particular condition should be evaluated in the context of other diagnostic findings, that is, clinical signs, serology, and pathology.

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Regulatory Considerations for Medication Use in Poultry

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Abbreviations

AMDUCA Animal Medicinal Drug Use Clarification Act
ANADA Abbreviated New Animal Drug Application
ANDA Abbreviated New Drug Application
AOAC Association of Official Analytical Chemists
AVMA American Veterinary Medical Association
CFR Code of Federal Regulations
CPG Compliance Policy Guide
CVM Center for Veterinary Medicine
ELDU Extra-Label Drug Use
EPA Environmental Protection Agency
FAO Food and Agriculture Organization
FARAD Food Animal Residue Avoidance and Depletion
FDA US Food and Drug Administration
FDA-CVM US Food and Drug Administration Center for Veterinary Medicine
FIFRA Federal Insecticide, Fungicide and Rodenticide Act
FSIS Food Safety and Inspection Service
GFI Guidance for Industry
JECFA Joint FAO/WHO Expert Committee on Food Additives
MRL Maximum Residue Limit
MUADP Minor Use Animal Drug Program
MUMs Minor Use/Minor Species Animal Health Act
NADA New Animal Drug Application
NDA New Drug Application
NRP National Residue Program
ONADE Office of New Animal Drug Evaluation
PAMTA Preservation of Antibiotics for Medical Treatment Act
US United States

USDA United States Department of Agriculture
VCPR Veterinarian Client Patient Relationship
VFD Veterinary Feed Directive
WDI Withdrawal Interval
WDT Withdrawal Time
WHO World Health Organization

Introduction/overview

Medicating poultry is a worldwide practice regardless of whether it applies to an individual bird or a large commercial flock. However, medication use in poultry has both rewards and challenges. It prevents or treats illness, thus producing healthier animals. In addition, flocks with multiple birds have the advantage of increased production. However, consumers, regulators, and legislators are increasingly concerned about direct and indirect hazards of drug use and potential impacts on public health. For example, when medications are administered to poultry, drug residues can be present within edible products if an insufficient drug withdrawal time is not observed. In order to help minimize drug residues in poultry food products, it is important that all veterinarians, whether they treat individual birds or advise large-scale operations, are educated and inform their clients about how to minimize drug residues in the human food chain.

Veterinarians who treat individual or small numbers of poultry patients face several challenges. Many of the approved products for poultry are formulated for large-scale commercial operations, because only a few poultry products are approved for laying hens, and

pet owners are often willing to treat individual birds with complex treatment regimens that lack approved withdrawal times for poultry. This chapter is designed to provide veterinarians with information that will help them navigate the legal and logistical hoops of medication use in poultry, provide resources for finding withdrawal times for drugs approved for poultry, provide guidance for estimating withdrawal intervals when drugs are used in an extra-label manner, and highlight guidance recommendations regarding prudent use of antibiotics. The information presented in this chapter is the authors' interpretation of the legislation, guidelines, and literature; however, the regulatory agencies have the ultimate authority and should be contacted with any questions. For most of the chapter, the resources and legislative/legal information are focused on the United States, however, information relative to other worldwide geographic areas is briefly highlighted.

Definition of poultry

In the United States, the Food, Drug, and Cosmetics Act defines the term "poultry" to include birds that are deemed to be major animal species (chickens and turkeys). The US Center of Veterinary Medicine (CVM), a branch of the Food and Drug Administration (FDA), has a list of definitions (Table 20.1) for poultry species and classes of chickens and turkeys [1]. In a broader sense, the term "poultry" also encompasses avian species that are "minor" animal species. The Code of Federal Regulations (CFR) defines "minor" animal species by exclusion, meaning that the "major" animal species have been identified and all others are deemed "minor." Major animal species are dogs, cats, horses, cattle, swine, chickens, and turkeys. Therefore, examples of "minor" poultry would include ducks, geese, game birds, pigeons, and so on. For the purposes of this chapter, "poultry" is deemed to refer to any avian species that has the potential for its meat, eggs, offal (entrails and internal organs of an animal used as food), by-products (i.e., feathers), or manure to directly or indirectly enter or influence any portion of the human food chain.

Drug administration, on-label and extra-label drug use in poultry

In poultry medicine, medications can be administered to an individual bird or an entire flock depending on numbers of birds needing treatment, disease, and/or overall management practices. The types of medications commonly administered to poultry include antibiotics, anticoccidials, and antiparasitics. Similar to any other animal species that are in need of medications, administration routes can be oral or parenteral. Because they require lower labor efforts, oral routes of drug administration for birds commonly include medicated water or feed regardless of whether or not the flock is small or large. Parenteral administration routes for medications are typically reserved for individual patients that might have a severe illness and have a high likelihood of not entering the human food chain because parenteral administration routes have a higher likelihood of resulting in drug residues. When medicating poultry, practitioners should consider their unique gastrointestinal anatomy, physiology, and drug elimination processes that differ from mammals [2]. Practitioners treating poultry in the United States can use FDA-approved veterinary products according to the label or in an extra-label manner. Use of medications according to the label directions is termed "on-label drug use," meaning that all of the drug label specifications (animal species and class, administration route, dose, dosing frequency/interval, indication, limitations and withdrawal time) are fulfilled and the FDA-approved withdrawal time is observed. The FDA-approved withdrawal time (WDT) is the amount of time that must be observed after the last dose is administered and before the meat, eggs, or offal that is intended for human consumption can enter the food chain.

In contrast to on-label drug use, medications for poultry in the United States can also be prescribed by a veterinarian for "extra-label drug use." Extra-label drug use (ELDU) occurs when the animal species/class, administration route, dose, dosing frequency/interval, or indication differs from the FDA-approved label. In the United States, ELDU was legalized with the passage of the Animal Medicinal Drug Use Clarification Act of 1994. When veterinary products are used in an extra-label manner, a withdrawal "interval" must be estimated based on scientific evidence and must be extended beyond the FDA-approved WDT regardless of the dose, route, or indication. It should be mentioned that legal ELDU does not include the use of prohibited drugs (such as fluroquinilones in chickens). More details on the prohibition of certain drugs and drug classes in food-producing species will be discussed later in this chapter.

Definition of residues

The word "residue" is defined in many ways by the literature and worldwide regulatory agencies. For the

Table 20.1 United States of America Food and Drug Administration's definitions of species and classes of chickens and turkeys

Species	Class	Definition
Chickens	Egg	From in ovo until hatching.
	Laying Hens (or layers)	Hens that produce eggs for human consumption.
	Chicks	Chickens from day of hatch until they are able to survive in ambient temperature (no longer brooded).
	Broiler Chickens (or fryers or frying chickens)	Meat-type chickens normally grown to a market age of 35 to 49 days and market weights between approximately 4 and 7 lb (1.80 and 3.2 kg).
	Roasters (or roasting chickens)	Meat-type chickens grown to market weights between approximately 6 and 9 lb (2.7 and 4.1 kg).
	Replacement Chickens	Chickens intended to become laying hens or breeding chickens.
	Breeding Chickens	Sexually mature male and female chickens of any type intended for the production of fertile eggs; the eggs are not intended for human consumption.
Turkeys	Egg	From in ovo until hatching.
	Laying Hens (or layers)	Hens that produce eggs for human consumption.
	Poults	Turkeys from day of hatch until they are able to survive in ambient temperature (no longer brooded).
	Growing Turkeys	Turkeys grown for meat purposes to a market age of approximately 17 (female) or 22 (male) weeks; may be further divided into heavy or light turkey strains.
	Finishing Turkeys	Turkeys intended for meat production during the last 2 to 4 weeks of growth.
	Replacement Turkeys	Turkeys intended to become laying hens or breeding turkeys.
	Breeding Turkeys	Sexually mature male or female turkeys intended to produce fertile eggs; their eggs are not intended for human food use.

Source: US Food and Drug Administration: Animal & Veterinary [1].

purposes of this chapter, a residue is deemed to be either the parent compound or metabolite of a parent compound that may accumulate, deposit, or otherwise be stored within the cells, tissues, organs, or edible products (e.g., milk, eggs) of an animal following its use to prevent, control, or treat animal disease or to enhance production [3]. According to the FDA, a residue is any compound that is present in edible tissues of the target animal that results from the use of the sponsored compound, including the sponsored compound, its metabolites, and any other substances formed in or on food because of the sponsored compound's use. (21 CFR 500.82). The main focus of this chapter is on drug residues; however, residues can also originate from pesticides [4], biotoxins, heavy metals, radionuclides, and so on. In general, residues that accumulate in food products can be problematic from a human health standpoint and should be considered when estimating how long to withhold poultry meat, eggs, and/or offal before they enter the human food chain.

Human health hazards of drug residues

The human health hazards of drug residues can be classified as direct or indirect impacts [5]. Direct impacts are those that result more immediately and include toxic reactions that impact consumers directly, such as the clenbutarol exposure of 135 residents of Spain in 1990; and similarly, 15 residents of Italy in 1997 that consumed contaminated beef [6,7]. Other examples of direct impacts include allergic/hypersensitivity reactions or bone marrow toxicity. Indirect impacts usually have negative effects over a longer time period and include carcinogenicity, mutagenicity, reproductive disorders, immunopathological effects, and transfer of antibiotic-resistant bacteria to the human population. The US FDA-CVM assesses the risks of veterinary drugs on public health prior to granting approval [8–11]. However, when drugs are used in an extra-label manner, these risk assessments have not been performed

by a regulatory agency. A review of how risk assessment principles can be applied to evaluate the human health risks posed by different classes of drugs used in extra-label manner has been published [9].

Regulatory monitoring of drug residues in animal products

Monitoring of drug residues in food-producing animals has been previously described [13]. In addition, regulatory systems for Europe, Australia, Canada, and Japan have also been reviewed [14].

In the United States, the primary mission of the National Residue Program (NRP) is to verify control of animal drug residues, pesticides, environmental contaminants, and any other chemical hazards in or on meat, poultry, or egg products. The principal agencies that work together to achieve the mission of the NRP include the Food Safety and Inspection Service (FSIS), the Environmental Protection Agency (EPA) and the FDA. Through the Federal Food, Drug and Cosmetic Act, the FDA is given the authority to establish drug tolerances (maximum permissible concentrations) that are published in Title 21 of the CFR. Title 21 also contains tolerances set by the FDA for heavy metals, industrial chemicals, and pesticides that are no longer approved for use. The EPA is provided a similar responsibility for pesticide tolerances under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA). The tolerances for pesticides are published in Title 40 of the CFR.

The United States Department of Agriculture (USDA) oversees FSIS, an agency that is responsible for the analytical testing program for residues in domestic and imported meat, poultry, and egg products. In particular, the National Residue Program Sampling Plan, known as the Blue Book, is developed yearly and made publicly available, whereas the Red Book contains NRP drug residue testing results from previous years. Both the Blue and Red Books are available online by linking to their URLs, which are listed in Table 20.2.

Through the NRP, poultry and egg products are tested in federally inspected establishments according to the sampling plan that is listed in the Blue Book. An inspector may also sample products that, according to his/her professional judgment, warrant testing and include analysis for approved and unapproved drugs, pesticides, hormones, and environmental contaminants. Overall, the number of violative residues found in poultry and poultry products from large commercial operations are relatively low [15,16].

In July 2012, FSIS announced that it was restructuring the way in which the NRP sampling plan will be scheduled [17]. In particular, the number of overall samples per production class will be reduced with the adoption of new analytical methods that allow samples to be analyzed for more chemical compounds than was previously possible.

In contrast to federally inspected large-scale commercial operations are the mid-size, smaller scale, and individual producers that are involved with local, farmers', or flea market sales. To the knowledge of the authors of this chapter, for these operations there is minimal regulatory oversight; therefore, it is critical that the advising veterinarians take responsibility and educate their clients regarding best practices for avoiding drug residues.

Legislation, regulations, and programs related to drug use and drug residues in poultry species

Minor use/minor species (MUMs) Animal Health Act

The MUMs Animal Health Act was passed in 2004. This legislation added new options for approving limited-use drugs and provided a new mechanism to legally market some unapproved products. The intention of this legislation was to increase the number of FDA approvals for minor food animal species (i.e., game birds such as ducks or quail) and to provide sponsors with incentives to seek label claims for veterinary products that would have "minor uses" in major species of animals (for poultry these would include chickens and turkeys). "Minor use" is determined by frequency of use and geography. More specifically, minor use means that a drug can be used in a major species for an indication that occurs infrequently and in a small number of animals, or in a limited geographical area and in only a small number of animals annually.

The MUMs Animal Health Act modifies the Federal Food, Drug and Cosmetic Act to include conditional approval, designation, and indexing for veterinary drugs used in minor animal species [18]. Conditional approval and designation provide incentives to drug sponsors with the ultimate goal of drug approval. Drugs that are conditionally approved have shown a reasonable expectation of effectiveness, but the sponsor is granted up to 5 years to provide all the necessary data proving effectiveness. Conditionally approved drugs cannot be used in an extra-label manner. Designation provides incentives for approvals, including grants to

Table 20.2 World-wide web URL links for on-line resources that provide veterinary drug and/or poultry specific information

Website	Description	URL website address
Drugs @ FDA	FDA's searchable database of approved human drugs	http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm
Animal Drugs @ FDA	FDA's searchable database of approved animal drugs and tolerances	http://www.accessdata.fda.gov/scripts/animaldrugsatfda/
Vetgram	FARAD's searchable drug database for food animal drugs and tolerances	http://www.farad.org/vetgram/
Bayer Animal Health	Searchable US compendium of veterinary products database; use charts tab for summarized label and WDT information	http://bayerdvm.com/ or http://bayerall.naccvp.com/ and http://bayerall.naccvp.com/?m=chartindex_main&type=wt
Canadian Compendium of Veterinary Products presented by Bio Agri Mix Total Solutions	Searchable Canadian compendium of veterinary products database; use charts tab for summarized label and WDT information	http://bam.naccvp.com/?u=country&p=msds and http://bam.naccvp.com/?m=chartindex_main&type=wt
Drugs.com	Searchable database of veterinary drugs; can search by animal groups; also lists Canadian veterinary drugs	http://www.drugs.com/vet/
CABI's Animal Health and Production Compendium	List of international animal drug databases	http://www.cabi.org/ahpc/drug-databases
Australian Pesticides and Veterinary Medicines Authority	Pubcris is a searchable database of registered pesticides and veterinary drugs.	http://services.apvma.gov.au/PubcrisWebClient/welcome.do
National Office of Animal Health	UK site with searchable drug compendium of approved drugs	http://www.noahcompendium.co.uk/Compendium/Overview/
Health Products Regulatory Authority	Searchable compendium of approved drugs in Ireland	http://www.hpra.ie/
European Medicines Agency Database	Public database providing on-line access to information about human and veterinary medicines available to EU citizens	http://www.eudrapharm.eu/eudrapharm/
USDA Foreign Agricultural Service	Veterinary drug and pesticide tolerance and MRL searchable database	http://www.fas.usda.gov/maximum-residue-limits-mrl-database
FAO/WHO Food Standards Codex alimentarius	Definitions of terms used in discussing veterinary drug residues in food animals	http://www.codexalimentarius.net/vetdrugs/data/reference/glossary.html
Joint FAO/WHO Expert Committee on Food Additives (JECFA)	Description of JECFA and links to publications	http://www.fao.org/food/food-safety-quality/scientific-advice/jecfa/en/
JECFA "Residues of some veterinary drugs in foods and animals"	Searchable database of maximum residue levels for veterinary drugs as recommended by JECFA that includes pharmacokinetic data summaries	http://www.fao.org/food/food-safety-quality/scientific-advice/jecfa/jecfa-vetdrugs/en/
Codex alimentarius international food standards	Homepage for codex alimentarius	http://www.codexalimentarius.org/about-codex/en/
European Medicines Agency European Public MRL Assessment Report	Summary report on the established MRL and the data supporting the MRL	http://www.ema.europa.eu/ema/index.jsp?curl=pages/medicines/landing/vet_mrl_search.jsp&mid=WC0b01ac058008d7ad
USDA/FSIS science chemistry page	Includes FSIS sampling plan (Blue Book) and results (Red Book)	http://www.fsis.usda.gov/wps/portal/fsis/topics/data-collection-and-reports/chemistry/residue-chemistry
USDA Food Safety and Inspection Service (FSIS)	Homepage with contact information and links to resources	http://www.fsis.usda.gov/

(continued)

Table 20.2 (*Continued*)

Website	Description	URL website address
FDA Center for Veterinary Medicine	Homepage with contact information and links to resources	http://www.fda.gov/AnimalVeterinary/default.htm 240-276-9300 AskCVM@fda.hhs.gov
FDA-CVM Compliance Policy Guide	AMDUCA	http://www.fda.gov/AnimalVeterinary/GuidanceComplianceEnforcement/ActsRulesRegulations/ucm085377.htm
AVMA: Animal Medicinal Drug Use Clarification Act (AMDUCA)	Reviews components of AMDUCA and includes list of prohibited drugs	https://www.avma.org/KB/Resources/Reference/Pages/AMDUCA.aspx
AVMA ELDU algorithm	AVMA's guide to ELDU under AMDUCA	http://www.avma.org/reference/amduca/amduca1.asp
AVMA: VCPR Reference Guide	AVMA's definition of a valid VCPR	https://www.avma.org/KB/Resources/Reference/Pages/VCPR.aspx
FDA-CVM Compliance Policy Guide	Extra-label use of medicated feeds for minor species	http://www.fda.gov/ICECI/ComplianceManuals/CompliancePolicyGuidanceManual/UCM074659 and http://www.fda.gov/animalveterinary/newsevents/cvmupdates/ucm048037.htm
FARAD	Restricted and prohibited drugs in food animals	http://www.farad.org/eldu/prohibit.asp
FARAD regulatory information	Details of the published law on compounding and AMDUCA	http://farad.org/amduca/amduca_law.asp
AVMA Compounding	AVMA brochure on veterinary compounding	https://ebusiness.avma.org/ProductCatalog/Product.aspx?ID=155
FDA-CVM Compliance Policy Guide	Compounding of Drugs for Use in Animals	http://www.fda.gov/ICECI/ComplianceManuals/CompliancePolicyGuidanceManual/ucm074656.htm http://www.fda.gov/ICECI/ComplianceManuals/CompliancePolicyGuidanceManual/ucm074656.htm
Society of Veterinary Hospital Pharmacists statement on compounding	Position statement on the compounding of drugs for use in animals	http://svhp.org/position-statements/
FARAD	Site where ELDU withdrawal requests may be made online and other resources for food animal veterinarians	http://farad.org/
Canadian gFARAD	Canadian program providing withdrawal recommendations following extra-label drug use.	http://www.cgfarad.usask.ca/home.html
National Pesticide Information Center	NPIC provides objective, science-based information about pesticides and pesticide-related topics	http://npic.orst.edu/
US Environmental Protection Agency (EPA)	Home page for EPA; pesticide information	http://www.epa.gov/
Centers for Disease Control and Prevention	CDC's Food Safety Program	http://www.cdc.gov/foodsafety/vitalsigns.html
Minor Use Animal Drug Program (MUADP)	Searchable database for drugs approved in minor food animal species. MUADP project requests can be submitted via this web site.	http://www.nrsp7.org/
American Association of Veterinary Laboratory Diagnosticians	Listing of AAVLD labs in USA by state	https://aavld.memberclicks.net/accredited-laboratories
US Food and Drug Administration: Animal & Veterinary. *CVM Guidance For Industry #152*	Appendix A identifies antibiotics deemed medically important by FDA and affected by Guidance #213	http://www.fda.gov/AnimalVeterinary/GuidanceComplianceEnforcement/GuidanceforIndustry/ucm042450.htm

Table 20.2 (*Continued*)

Website	Description	URL website address
Australian Pesticides and Veterinary Medicines Authority Guideline 31- Residues in Poultry Tissues and Eggs	Guideline describing the conduct and reporting of residue trials in poultry in Australia	http://www.apvma.gov.au/publications/guidelines/
Alabama Cooperative Extension	Downloadable publication on pest management in poultry	https://store.aces.edu/ItemDetail.aspx?ProductID=13589
Poultry Science Association	Resources for poultry veterinarians	http://www.poultryscience.org/index.asp
Chicken Farmers of Canada	Organization representing the chicken industry in Canada	http://chicken.ca/
Poultry Med	International website that provides information pertinent to poultry veterinarians including drug and residue information	http://www.poultrymed.com/Poultry/index.asp
National Chicken Council	A national, non-profit trade association representing the US chicken industry	http://www.nationalchickencouncil.org/
FARAD Digests	FARAD digests are publications providing guidance specific to drug use in food-producing animals. A digest on drug use in game birds is available through this URL.	http://www.farad.org/publications/digests.asp

support the studies required for approval, and up to seven years exclusive marketing rights by the sponsor. MUMs legislation also allows for medications to be categorized as "indexed" drugs. Indexed drugs have not undergone a formal drug approval process but the designation allows drug companies to market and sell these medications for selected populations. Indexing is intended for drug treatment of diagnosed conditions in minor non-food producing animal species, specifically targeting laboratory animals and zoological collection specimens. The purpose of indexing is to make products available that cannot meet requirements of the drug approval process as a result of a limited animal population and a wide variety of species. Indexed drugs may not be legally used in an extra-label manner in food-producing animals. More details regarding MUMS legislation have been previously described [19].

Minor Use Animal Drug Program (MUADP)

The Minor Use Animal Drug Program (also known as National Research Support Project-7) is a congressionally funded multi-institutional collaborative research program administered by the United States Department of Agriculture. The mission of MUADP is to identify animal drug needs for minor species and minor uses in major species, to generate and disseminate data for safe and effective therapeutic applications in minor species, and to facilitate FDA/CVM approvals for drugs that are

identified as a priority for a minor species or minor use in a major species. To accomplish these goals, NRSP-7 functions through the coordination of efforts among animal producers, pharmaceutical manufacturers, FDA/CVM, USDA/Cooperative State Research, Education, and Extension Service, universities, State Agricultural Experiment Stations and veterinary medical colleges throughout the country. The MUADP home web page (Table 20.2) lists FDA-approved veterinary products that have been approved for minor food-producing animal species including various poultry. In addition, via the MUADP web site, individuals can fill out a request form asking for programmatic consideration to seek FDA approval for a drug that is intended for use in a minor species or for a drug that would have a minor use in a major species. The process for selecting drugs that the MUADP will pursue for FDA approval is represented in a schematic on the web site. For a number of reasons, including manufacturer interest, known side effects of the drug, importance of the disease being treated, and the targeted animal species for treatment, some proposals have higher priority than others. Funds are limited, so the program must select projects carefully.

It should be noted that the Minor Use Animal Drug Program is a cooperative research program and is different from the MUMs congressional act. However, the MUMs legislation benefits MUADP by including a provision for competitive grants to help support

Table 20.3 Requirements of the Animal Medicinal Drug Use Clarification Act (AMDUCA) of 1994

Requirement	Explanation
Therapeutic Purpose	Extra-label drug use can only occur for therapeutic purposes when an animal's health is suffering or threatened. Extra-label drug use for reproductive purposes, growth promotion and efficiency is not allowable under AMDUCA.
No Effective Labeled Drugs	ELDU should not occur unless FDA approved drugs as labeled are clinically ineffective for their intended use.
VCPR	A valid veterinarian-client-patient relationship (VCPR) must exist. Table 20.2 includes the website address to AVMA's definition of a valid VCPR.
Veterinarian's Supervision	ELDU is permitted only under the supervision of a veterinarian. This includes the extra-label use of over-the-counter medications. Also, any over-the-counter product that is compounded in veterinary medicine is deemed a prescription drug and may only be used under veterinarian supervision.
FDA Approved Drugs	ELDU is permitted using only FDA-approved animal and human drugs. When using medications extra-label, medications approved for other food animal species should be used before medications approved only for non-food animal species which should be used preferentially over drugs approved for humans only. Using bulk chemical or active pharmaceutical ingredient (API) is not allowed as they are not FDA-approved.
Not in Feed	Extra-label use of an approved animal drug or human drug or feed additive in or on an animal feed is prohibited. Also, using combinations of medicated feed or feed additives not approved to be used together is not considered AMDUCA compliant. Extra-label drug use in water is permitted.
No Residues	ELDU must not result in residues.
Additional Food Animal Requirements	Make a careful diagnosis and evaluation of the conditions for which the drug is to be used. Establish a substantially extended withdrawal period supported by scientific information. Institute procedures to assure that the identity of the treated animal or animals is carefully maintained. If the individual animal cannot be identified for the extended withdrawal time, then the extended withdrawal time must be applied to the entire group. Take appropriate measures to assure that assigned timeframes for withdrawal are met and no illegal drug residues occur in any food-producing animal subjected to extra-label treatment.

Source: [21 CFR530.3 (a)].

studies that are necessary to seek label claims for minor species and minor uses in a major animal species.

The Animal Medicinal Drug Use Clarification Act (AMDUCA) of 1994

The Animal Medicinal Drug Use Clarification Act (AMDUCA) of 1994 made extra-label drug use of FDA-approved medications by veterinarians in the United States legal. "Extra-label use" is defined in the CFR as: "Actual use or intended use of a drug in an animal in a manner that is not in accordance with the approved labeling. This includes, but is not limited to, use in species not listed in the labeling, use for indications (disease and other conditions) not listed in the labeling, use at dosage levels, frequencies, or routes of administration other than those stated in the

labeling, and deviation from labeled withdrawal time based on these different uses." [21CFR530.3 (a)]

The American Veterinary Medical Association (AVMA) has developed an algorithm that helps veterinarians to determine whether the intended ELDU is legal according to AMDUCA. The web site address for the algorithm is included in Table 20.2.

The requirements of AMDUCA are listed in Table 20.3. Key points within AMDUCA include (i) ELDU can only occur on the order of a veterinarian within the context of a veterinarian-client-patient relationship (VCPR); (ii) ELDU must be limited to a therapeutic purpose to treat a sick or dying animal; (iii) ELDU in food-producing animals requires that no violative residues occur; (iv) Certain compounds are prohibited from ELDU. The AVMA has defined what constitutes a legal veterinarian-client-patient relationship and the website address for this definition is included in

Table 20.2. AMDUCA stipulates that ELDU only applies to FDA-approved human and veterinary medications. It does not legalize the extra-label use of EPA regulated pesticides or USDA regulated biologics. For example, fipronil cannot be administered to a chicken for diminishing ectoparasitism, because this product is deemed an insecticide and is regulated by EPA and not FDA. As all FDA-approved veterinary products are assigned either a new animal drug application (NADA) or an abbreviated new animal drug applications (ANADA) number, if a veterinary product does not have an NADA or ANADA number its use is most likely not AMDUCA-compliant in an extra-label manner. In addition, drugs that are approved for humans have new drug application (NDA) numbers for pioneer drugs or abbreviated new drug application (ANDA) numbers for generic medications. Approval status of both animal and human drugs can be found on the FDA-CVM and FDA websites respectively. The web site URLs are included in Table 20.2.

Compliance Policy Guide (CPG) 615.115 extra-label use of medicated feeds for minor species

Extra-label use of medicated feed or drugs used extra-label in or on feed is prohibited by AMDUCA. However, for some minor food-producing species, especially poultry, medicating via the feed might be essential. In many circumstances, medicating poultry may not be practical via water or other drug administration routes. Therefore, the CPG 615.115 was created. This CPG allows veterinarians to prescribe FDA-approved medicated feeds to be fed to minor food-producing animals in an extra-label manner. As the FDA considers chickens and turkeys to be major animal species, the CPG does not apply to them. However, for the "minor" poultry species, such as ducks, geese, pheasants, and quail, medicated feeds that are approved for major species (turkeys or chickens) can be used for the minor poultry species under the supervision of a veterinarian. In other words, the CPG does not make it legal to use medicated feed for these minor poultry species. However, the CPG does give field investigators "regulatory discretion," and if all of the CPG requirements are followed correctly, field investigators do not have to take any action against the producer or veterinarian.

When medicated feeds are being used under the auspices of the CPG, the other requirements of AMDUCA should be met. Additional stipulations for the ELDU of medicated feed in minor species include:

1. A written recommendation that includes the medical rationale and dated within 3 months prior to use is required. The producer and veterinarian must keep copies of the written recommendation that would be available in the case of a FDA inspection.
2. A medicated feed to be used in a minor food-producing species must be approved in a major food-producing species.
3. No changes in the formulation of the FDA-approved medicated feed may be made. For example, the percentage of protein in a medicated feed that is approved for chickens cannot be changed to meet the nutritional requirements of pheasants. The medicated feed that is approved for chickens must keep the original chicken label and the feed must be used "as is" for the minor species.

Prohibited drugs/prohibited drug use in poultry

In the United States, the FDA has established a list of drugs or drug classes that are either completely prohibited or prohibited from ELDU in food-producing animals. Other countries have their own lists of drugs of high regulatory concern. Table 20.4 lists the veterinary products that the FDA-CVM prohibits from use in poultry or drug classes that the FDA-CVM prohibits from extra-label use in poultry in the United States. A complete listing of the FDA-CVM prohibited drugs for all animal species can also be found at web site links referenced in Table 20.2. It is advised that the FDA prohibited drug list be checked on a regular basis for updates as it is subject to change. If any of these prohibited drugs are mistakenly used in poultry, then the affected animal and its by-products (i.e., eggs or poultry litter used as feed for other food-producing animals) should never be allowed to enter the human food chain, and ideally the animal should be isolated from other birds that are used as food producers for humans.

In the United States, any animal that has the potential to enter the human food chain or directly or indirectly impact public health should not be administered a prohibited drug. More specifically, in poultry medicine, regardless of whether a bird is in a social (i.e., a companion or "pet chicken") or production (i.e., small flock of backyard laying chickens) setting, it should not be administered FDA-prohibited medication. The FDA has established this prohibited drug list as a measure to protect public health. Examples highlighting how poultry might directly or indirectly impact public health include eggs from backyard chickens that are given to neighbors and/or sold at local farmer's markets; the feces of a companion chicken that might be co-mingled with excrement from other birds being used to produce eggs for human consumption; or chicken feces that

Table 20.4 United States of America Food and Drug Administration-Center of Veterinary Medicine prohibited drugs that have relevance to poultry and reported adverse reaction(s) in humans

Prohibited drug	Adverse reaction(s) reported in humans
Chloramphenicol	Idiosyncratic, non-dose dependent, irreversible, aplastic anemia
Clenbuterol	B-adrenergic toxicities
Diethylstilbestrol (DES)	Reproductive tract abnormalities and tumors in female offspring, Infertility
Nitroimidazoles such as metronidazole	Potential for carcinogenesis
Nitrofurans- including topical applications	Potential for carcinogenesis
Fluoroquinolones such as enrofloxacin can be used on label only	Potential to cause development of resistant human pathogens
Glycopeptides such as vancomycin	Potential to cause development of resistant human pathogens
Gentian Violet - prohibited from use in feed	Human food safety has not been assessed
Antivirals (adamantine and neuramidase inhibitors) in poultry	Potential to cause development of resistant human pathogens
Cephalosporins, not including cephapirin, must be used on label in cattle, swine, chickens and turkeys. They may be used extra-label only in the above species to treat a disease indication not labeled.	Potential to cause development of resistant human pathogens

Source: Davis, J.L., Smith, G.W., Baynes, R.E., *et al*. [20].

might be fed to cattle intended for human consumption [21]. In all these cases, there are potential direct or indirect impacts on public health. Therefore, even if an owner thinks that a companion pet chicken would never be used for human consumption, it should not be administered an FDA-prohibited drug as there is no guarantee that the chicken would remain in a companion status and/or there are other ways that that bird could indirectly impact human health.

There are three prohibitions proclaimed by the FDA that impact poultry: The prohibition of extra-label use of cephalosporins, the prohibition of fluoroquinolones, and the prohibition of anti-viral medications.

Prohibition of extra-label drug use of cephalosporins in major food-producing animals

On 5 April 2012, a prohibition of the extra-label use of cephalosporins in major food-producing animal species, including chickens and turkeys, took effect. The FDA enacted this prohibition because of concerns for increasing bacterial resistance to cephalosporins, many of which are used for treating humans. At the time that this chapter was authored, there was only one cephalosporin that was approved for poultry, ceftiofur sodium. Ceftiofur sodium is labeled for use in day-old chicks and turkey poults for control of early mortality associated with *E. coli* infections. From a legal standpoint, ceftiofur sodium can only be used in an extra-label manner in the aforementioned species/class for a different indication. In other words, the label dose, administration route, treatment duration, and species (in this case, day old chicks and turkey poults) must all be on-label. *In ovo* administration would be deemed prohibited. At the time that this chapter was authored, the minor food-producing poultry species (ducks, geese, etc.), were excluded from this ban; thus cephalosporins may continue to be used responsibly in an extra-label manner.

Prohibition of fluoroquinolones

In the early 1990s, there was a rapid increase in fluoroquinolone-resistant *Campylobacter* spp., a known contributor to foodborne illness in humans, that was associated with increased use of fluoroquinolones in poultry [22]. As a result, the extra-label use of fluoroquinolones was banned in the United States in 1997. At that time, sarafloxacin and enrofloxacin were approved for use in poultry. Even with this prohibition, increased fluoroquinolone-resistant *Campylobacter* spp. in poultry were linked with resistant infections in humans, leading to the voluntary withdrawal of sarafloxacin products in 2001. In 2005, the FDA approval for enrofloxacin was withdrawn [23]. At the time that this chapter was written, the use of any fluoroquinolones in US poultry is prohibited.

Prohibition of specific antiviral medications

In 1999, avian influenza entered the limelight in the United States as a deadly zoonotic disease on a global scale. Given the serious nature of avian influenza infection, the antivirals rimantadine, amantadine, oseltamivir, and zanamivir were prohibited from use in poultry in the United States in order to preserve their effectiveness for treatment of human beings. It has been reported that countries that have previously allowed the use of these medications in poultry have observed development of drug resistance [24,25].

Compounding of medications for poultry

Compounding is the term used for combining, mixing, or altering ingredients to create a medication that is tailored to the needs of an individual patient. It involves making a new drug for which safety and efficacy have not been demonstrated with the kind of data that FDA requires for new drug approval. In virtually all cases, FDA regards compounded drugs as unapproved new drugs [26].

According to the CFR, a veterinarian may consider using a compounded product in poultry "when there is no approved new animal or approved new human drug that, when used as labeled or in conformity with criteria established in this part, will, in the available dosage form and concentration, appropriately treat the condition diagnosed." [21CFR530.13]

Given that poultry are deemed food-producing animals, there are specific requirements for legal use of a compounded product in poultry (and any other food-producing animal) [21CFR530.13]. These requirements are listed in Table 20.5.

Using compounded medications is deemed to be ELDU of an approved animal or human drug. As compounding falls under AMDUCA, the requirements for ELDU and compounded medications under AMDUCA are listed in Table 20.3.

Often, bulk chemicals or active pharmaceutical ingredients (API) are used in commercial compounding. This is not deemed legal under AMDUCA for food animal species because this chemical is not an FDA-approved drug. Defined in 21 CFR 207.3, "bulk drug substance means any substance that is represented for use in a drug and that, when used in the manufacturing, processing, or packaging of a drug, becomes an active ingredient or a finished dosage form of the drug, but the term does not include intermediates used in the synthesis of such substances." In other words, by bulk chemical, we mean the drug in powdered chemical form, which is often used for research purposes and may not be of pharmaceutical grade. Compounding

Table 20.5 Requirements for legal use of compounded products in food-producing animals

All requirements for ELDU under AMDUCA are met

An approved animal drug should be used for compounding before a human drug

Compounding is performed by the veterinarian or pharmacist within their scope of practice

Adequate procedures are followed to ensure the safety and effectiveness of the compounded product

Scale of compounding is in line with the need for the product and is for a particular patient. Compounding in anticipation of receiving prescriptions, except in limited quantities, is illegal. The compounding of large quantities can fall under "manufacturing" and thus the compounded product would be deemed a drug in need of FDA approval. Also, compounding for third parties to resell or selling it at wholesale to another individual or entity for resale is illegal. So, it would not be legal for a compounding pharmacy to make a product for a veterinarian to keep on his truck to sell to dairies.

All state laws relating to compounding are followed

with bulk chemicals can be less expensive than using an FDA-approved medication. However, according to AMDUCA, ELDU (in this case compounding) is legal for therapeutic purposes only; therefore, cost is not an acceptable reason for compounding.

Despite the prohibition of bulk chemicals for compounding, there are some exceptions where the FDA has stated that, in most circumstances, they likely will not pursue regulatory enforcement if food-producing animals, including poultry, are treated with the bulk chemicals listed below:

1. ammonium molybdate
2. ammonium tetrathiomolybdate
3. ferric ferrocyanide
4. methylene blue
5. picrotoxin
6. pilocarpine
7. sodium nitrite
8. sodium thiosulfate
9. tannic acid

However, animals that are treated with any bulk chemical from the above list may require a prolonged withdrawal time, which should be taken into consideration when deciding whether to treat or not.

In order to be AMDUCA compliant, when compounded products or medications are used in poultry in an extra-label manner, the information listed in Tables 20.6 and 20.7 needs to be documented in the patient's medical records and on the prescription label respectively. These records must be kept for 2 years

Table 20.6 Required Information to be included in the patient's medical record with extra-label drug use or compounded medication use

Identity of the animals, either as individuals or a group
Animal species
Number of animals treated
Condition being treated
Established name of the drug and active ingredient
Dosage prescribed or used
Duration of treatment
Specified withdrawal, withholding, or discard time(s), if applicable, for meat, eggs, or animal-derived food

Table 20.7 Required information to be included on the prescription label with extra-label drug use or compounded medication use

Name and address of the prescribing veterinarian
Established name of the drug or each ingredient
Any specified directions for use including the class/species or identification of the animal or herd, flock, pen, lot, or other group; dose, frequency, and route of administration; and the duration of therapy
Any cautionary statements
The veterinarian's specified withdrawal, withholding, or discard time for meat, eggs, or any other food that might be derived from the treated animal or animals

and be accessible to FDA inspectors, so that they can estimate the risk to public health.

There can be some liability associated with the use of compounded medications, as they generally do not undergo the same quality assurance testing as commercially manufactured medications. In addition, AMDUCA requires that there be sufficient scientific data to estimate a withdrawal interval. In general, scientific pharmacokinetic data for compounded medication use in food-producing animals is limited, so estimating a withdrawal interval can be difficult. Recommending a withdrawal interval to the client is the legal responsibility of the veterinarian. It is particularly important that the veterinarian be aware of this, especially if a pharmacy is performing the drug compounding, as the pharmacy may not be aware of all the legal ramifications. If there is insufficient data to estimate a withdrawal interval, then the veterinarian must assure that the animal and its products never enter the human food chain.

It is also important to remember that medications can only be compounded for an individual patient with whom the veterinarian has a valid VCPR.

Overall, the compounding of medications for poultry and other food-producing animals should be rarely used. When treating an animal whose tissues or products have the potential to enter the human food chain, it is important to remember that food safety and public health come first. Additional resources regarding compounding and a link to AVMA's brochure on veterinary compounding and choosing a compounding pharmacy are listed in Table 20.2.

Future legislation/fda-cvm guidance documents impacting poultry

In April 2012, the FDA-CVM released the final version of Guidance for Industry (GFI) #209, "The judicious use of medically important antimicrobial drugs in food-producing animals." [27] This document summarizes the agency's findings that have suggested that food-producing animals that have been treated with antimicrobials affect the bacterial populations of humans who consume them. Based on the interpretations of these findings, the FDA has determined that use of antimicrobials that are important for therapeutic use in humans in food-producing animals should be limited to therapies necessary to maintain animal health only. In addition, the FDA has determined that there should be veterinary oversight for any use of medically important antimicrobials in food-producing animals to ensure the judicious use of these important medications. Medically important antimicrobial drugs as identified by the FDA are listed in Appendix A of FDA-CVM's Guidance For Industry #152. The web URL for GFI #152 can be found in Table 20.2 of this chapter.

Guidance for Industry #213, "New animal drugs and new animal drug combination products, administered in or on medicated feed or drinking water of food-producing animals: recommendations for drug sponsors for voluntarily aligning product use conditions with GFI #209" goes further [28]. Released only as a draft guidance at the time of press, this document alludes to coming changes that will involve the withdrawal of marketed feed or water antibiotics, deemed medically important in the treatment of humans, that are currently labeled for production purposes, including weight gain and feed efficiency. Any remaining over-the-counter medicated feed or water treatments that contain medically important antimicrobials with therapeutic indications, including treatment, control, and prevention of specific diseases need to be reclassified as prescription drugs or veterinary feed directive (VFD). The guidance outlines the steps involved for manufacturers of the current over-the-counter medicated feeds or drinking water products to seek approvals for

therapeutic claims, making them VFDs or prescription drugs. There are currently no VFDs that are labeled for use in poultry.

Proposed legislation that will have an impact on medication use in veterinary medicine if passed into law includes the Preservation of Antibiotics for Medical Treatment Act (PAMTA). This legislation proposes to preserve the effectiveness of antimicrobials for use in treating human diseases by phasing out what the bill calls "non-therapeutic uses" in food-producing animals. If passed, this legislation will ban any use of medically important antimicrobials as a feed or water additive for an animal in the absence of any clinical sign of disease for the purposes of growth promotion, feed efficiency, weight gain, routine disease prevention, or other routine purpose.

The bill would also require the withdrawal of antibiotics that are used for "non-therapeutic" reasons in food-producing animals unless it could be proven that they pose no harm to human health, a difficult task to prove.

Considerations for avoiding residues in poultry products intended for human consumption

General recommendations

Some general recommendations to help avoid residues include the following:

- All waterers should be thoroughly rinsed and cleaned once the course of medication is complete
- All equipment used for mixing and storing medicated feed and feeders should be cleaned once treatment is complete
- If the label WDT requires it, all birds should be switched to a non-medicated feed to allow residues to deplete prior to slaughter. Medicated feeds are a common source of drug residues if the proper WDT is not observed
- Calculations of dosages for medicated water and feeds should be carefully performed and even double checked by a second party in cases where large numbers of birds are to be treated. A list of conversions commonly used in poultry production has been previously published [29]
- Bedding litter should be changed after completing treatment as any remaining litter may serve as a source of drug exposure to both birds and humans from feces or dropped feed [30]. This could contribute to antimicrobial resistance and possible residues. Nicarbazin has been shown to be stable in litter for

prolonged periods and cause persistent residues in the feces and eggs of hens that are kept on unchanged litter after treatment [31]

- If the units for the withdrawal times are days, a full 24-hour period per day, beginning from the last treatment/dose, must be observed. For example, if the withdrawal time is 2 days, 48 hours must pass after the last treatment/dose before an animal can be slaughtered
- Proper records and identification of treated animals must be maintained. Inadequate treatment records or failure to identify treated animals lead to insufficient withdrawal times and violative residues
- When using rodenticides, bait stations that are inaccessible to birds should be used and dead rodents should be promptly disposed of
- Label instructions and withdrawal times should be followed when using pesticides and insecticides. Consult the EPA web site for guidance (Table 20.2).

When using medicated water to treat poultry there are some factors that may affect treatment efficacy. For example, serum concentrations of medications can vary considerably because of differences in water uptake by individual birds. Factors affecting water intake include environmental temperature, feed quality and amount of feed ingested, species, age, health, and circadian rhythm and accessibility [2]. Table 20.8 lists some factors known to affect water consumption in poultry. Tables 20.8 and 20.9 list estimated water and feed consumption rates that may be used as a guide when determining treatment regimens for poultry [32].

Characteristics of the water itself, including hardness and pH, should be evaluated when administering medications in water. Tetracyclines form less soluble complexes if the water is hard, reducing bioavailability in the treated birds. Additionally, many drugs have only a certain pH range at which they are stable for any length of

Table 20.8 Factors increasing and decreasing water uptake in poultry

Factors increasing water consumption	Factors decreasing water consumption
Soybean meal diet	High energy diets
High fiber diet	Nighttime
High environmental temperature	Sickness
Dawn and Dusk	Poor palatability
Age	Decreased feed intake
Electrolytes	
Increased feed intake	

Source: Vermeulen, B., De Backer, P. and, Remon, J.P. [2].

Table 20.9 Estimated water consumptions for chickens

Hens-nonlaying	5.0 gallons/100 birds	19 L/100 birds
Hens-laying	5-7.5 gallons/100 birds	19-28 L/100 birds
Chickens 4 weeks	2.0 gallons/100 birds	7.6 L/100 birds
Chickens 8 weeks	4.1 gallons/100 birds	15.5 L/100 birds
Chickens 12 weeks	5.5 gallons/100 birds	21 L/100 birds

Source: Buck, B.B. [32].

time. Therefore, the pH of the water is also an important consideration when determining a drug regimen.

In order to increase the likelihood of achieving therapeutic dosages, a fresh solution of drug should be prepared daily. The solution should be mixed thoroughly and checked to ensure that the drug goes into and remains in solution.

On-label drug use: the gold standard for minimizing drug residues

The ideal scenario for minimizing drug residues in poultry products is to use an approved product according to the label directions and comply with the label withdrawal time as established by the regulatory agency. Tables with summarized withdrawal times for poultry drugs approved in the United States and Canada can be found on the Compendium of Veterinary Products Web Site (Table 20.2). In the United States, a variety of web sites also provide the ability to search for approved poultry products (Table 20.2); however, it is the veterinarian's ultimate responsibility to consult the product information contained on the product label or package insert. Lists of US veterinary products that are approved for game birds have been published [33], but the information should be compared to the FDA-approved veterinary drug web site because the approvals might have changed since the publication date.

In the United States, the Office of New Animal Drug Evaluation (ONADE) within the FDA-CVM is responsible for reviewing and approving new drug applications. For a drug to be approved, the drug sponsor must demonstrate that the drug is safe in the intended species and is effective for the indication or disease condition that is being treated using the labeled dose and administration route. The sponsor must also demonstrate that the drug is safe for the person administering it, that use of the drug will not harm the environment, and that the drug can be consistently manufactured to standards of strength and purity. If the drug is to be labeled for food-producing animals, human food safety is also a major component of the approval process. In other words, for the product to gain approval there must be reasonable certainty of no harm occurring to human health from the ingestion of foodstuffs from food-producing animals that are treated with the drug.

Part of the approval process is to estimate a safe time for which the meat, organs, by-products, or eggs must be withheld before entering the human food chain. This withdrawal time is dependent on the maximum concentration of the drug or its metabolite allowed in the edible tissue that has the longest elimination. In the United States, this is known as tolerance, whereas in Europe and other parts of the world this concentration is known as the maximum residue limit (MRL). Calculations of tolerances or MRLs have been previously described [34,35].

When withdrawal times are established by the FDA, they are calculated so that 99% of the animals are below the tolerance when the drug is used according to label directions. This is based on data provided by the sponsor (i.e., a pharmaceutical company or any other entity applying for drug approval in healthy animals). Keep in mind that animals that are systemically compromised, suffering from liver or renal dysfunction, or a septic illness, may take longer to eliminate the drug and require an extended withdrawal time even when label directions are followed.

Other countries have their own approval processes that are not detailed in this chapter; however, for reference purposes a summary of approved poultry medications and their withdrawal times for the United Kingdom and Australia, respectively, can be found on this book's accompanying website. It is important to remember that this information is dynamic, thus veterinarians should always consult the product label or package insert to ensure accuracy (Table 20.2).

Extra-label drug use: Strategies for minimizing drug residues

In the United States, AMDUCA stipulates that it is the legal responsibility of the prescribing veterinarian to make a withdrawal recommendation based on scientific evidence when drugs are used in an extra-label manner. In addition, ELDU must be assigned a substantially "extended" withdrawal time. This "extended" withdrawal time(s) is/are referred to as withdrawal interval(s) or WDI(s) in this chapter. The WDI must be longer than the FDA-approved WDT, regardless of the dose, duration/frequency of treatment, or dosing frequency, even if any of these dosing factors are less than those listed on the FDA-approved label. For example, if a hypothetical drug label dosage is 10 mg/kg, the corresponding FDA-approved WDT is 5 days, and the drug is

administered in an extra-label manner at 5 mg/kg, then the WDI must be longer than 5 days.

A US program that provides advice regarding on-label and extra-label drug use and provides WDIs based on scientific data is the Food Animal Residue Avoidance and Depletion Program (FARAD). FARAD was previously known as the Food Animal Residue Avoidance Databank, but because the program serves many more functions besides data banking, the program's name was changed in 2011. A similar program exists in Canada (Canadian gFARAD), but they are a separate entity from the US program. Any comments in this chapter regarding FARAD are specific to the US program.

US FARAD is a national, congressionally-funded, USDA-administered, cooperative program, with a primary mission to prevent or mitigate illegal residues of drugs, pesticides, and other chemicals in foods of animal origin. FARAD collects, analyzes, and evaluates scientific data to provide withdrawal intervals when drugs are used in an extra-label manner. FARAD also advises on pesticide exposure and accidental contaminations (biotoxins, heavy metals, radionuclides, mistaken feeding of a batch of higher than label dose-medicated feed to a flock of chickens where there was an error in the dose calculation, etc.)

When veterinarians prescribe drugs according to the label or in an extra-label manner, they can contact FARAD for WDTs or WDI recommendations, respectively, via the telephone hotline (1-888-873-2723) or submit withdrawal requests online at www.farad.org. Withdrawal recommendations are provided to the prescribing veterinarian on a case-by-case basis. FARAD does not recommend generation of WDI "lists," as these lists can become outdated as a result of new information in the literature changing a WDI, tolerances being changed, or FDA-approved WDTs being modified.

Strategies and techniques for estimating WDIs when drugs are used in an extra-label manner in livestock have been previously published [36,37]. Additionally, factors or information that FARAD takes into consideration for estimating a WDI for poultry and other food-producing animals when they are treated with veterinary products in an extra-label manner have been previously published [38] and include those listed in the following sub-sections.

General factors impacting pharmacokinetic parameters and drug residues

When recommending a WDI after ELDU or a contamination incident, FARAD takes into account conditions that might impact drug absorption and elimination. Some treatment conditions include dose, duration of treatment, and administration route.

Egg withdrawal recommendations can be difficult to estimate, as the variables involved in residue deposition in eggs have not been fully elucidated. Systemic administration of medications generally results in exposure of the ovary, follicle, and oviduct potentially leading to egg residues. It has been reported that with some medications, egg white concentrations mirror plasma concentrations, with higher doses resulting in higher residue concentrations [39]. Imaging studies have found that during yolk formation, drugs are incorporated into the yolk through daily layering of yolk material. Consequently, drug exposure in early stages of yolk development results in drug residues in the inner rings of yolk, while exposure in the later stages of development results in residues in the outer portion of the yolk [40,41]. This means that even drugs with short elimination times may still cause detectable egg residues for prolonged periods as a result of exposure of early stage egg yolks [40,42]. Physical-chemical properties of the drug itself, including lipophilicity, hydrophilicity, protein binding, pKa, drug dose, and treatment length influences the extent of drug transfer to the yolk or albumin [39,43]. Two excellent review articles addressing the issues of modeling drug residues in edible poultry tissues and eggs have been published [44,45] and a literature review of scientific studies with egg residue data is also available [46].

Physiological factors/compromised health conditions impacting pharmacokinetic parameters and drug elimination

Drug clearance can be affected by the clinical condition of the patient receiving the treatment. Dehydration might impact how the drug is absorbed, especially if the drug is administered subcutaneously. If the drug has an extended absorption time, then this could lengthen the elimination time. Another important factor to consider when estimating a WDI is the overall function of the slowest organ to eliminate the drug. Most antibiotics are excreted via the kidney. Any clinical condition affecting the kidney could also impact drug elimination. For example, renal failure could result in prolonged drug elimination of most antibiotics including beta-lactams. Liver failure affects drugs that require hepatic activation, undergo biotransformation, or are affected by hypoproteinemia (i.e., highly protein bound drugs). As a result, avian-unique characteristics in drug metabolism and clearance should always be considered [2]. Similar to mammals, gastrointestinal

diseases in birds may limit drug absorption because of altered intestinal absorption [2].

Pharmacokinetic parameters: Drug residue serum, tissue or egg data from published studies

FARAD commonly uses published data (especially time versus concentration data) to calculate pharmacokinetic parameters that are subsequently used for recommending WDIs following ELDU. The principal pharmacokinetic parameters of a veterinary drug that are useful for predicting the concentrations of residues after a drug has been administered have been described [47]. In order for FARAD to deem published data to be useful, the time versus concentration data must derive from live animal studies, all of the dosing information must be provided, and the matrix, that is, serum, plasma, tissue, egg, and so on, that is analyzed must be clearly identified. Tissue or egg concentration data are more helpful than serum or plasma data as they represent the edible products that would be consumed by humans. In addition, plasma or serum data may or may not reflect residue concentrations in the tissues or eggs [40,42,48,49]. In some published studies, authors report that residues were still detectable on the last sampling day, thus FARAD is conservative when estimating a withdrawal recommendation, especially if no tolerance exists. Even if residues were not detected on a sampling day post-treatment, FARAD would compare the assay's limit of detection with the tolerance for the drug, poultry species, and matrix (i.e., tissue type, egg component, serum or plasma, etc.) If the assay's limit of detection is higher than the tolerance, then violative residues could still be present in the edible poultry products. According to AMDUCA, following ELDU in the United States, if there is no approved tolerance, no residues (i.e., the tolerance would be deemed to be 0) should be detected in products intended for human consumption. Therefore, if the analytical method is extremely sensitive, an extended WDI is necessary.

Established tolerance or MRL

Another factor that FARAD takes into account when recommending a WDI, is whether a tolerance has been established for the marker residue and matrix of interest for the bird species that was treated. A marker residue is the residue the concentration of which maintains a known relationship to the concentration of total residue in an edible tissue [50]. When the concentration of the marker residue is below the tolerance in the target tissue, the total residues in all the edible tissues are less than their respective safe concentrations [34].

In certain countries, MRLs are the focus. The USDA Foreign Agricultural Service MRL database, is a searchable international database for pesticide and veterinary drug MRLs for various commodities and markets. Worldwide web site URLs for on-line resources listing tolerances or MRLs can be found in Table 20.2. The Food and Agriculture Organization (FAO) and World Health Organization (WHO) have a joint program known as Codex Alimentarius Commission that publishes a collection of international food standards and guidance documents. The Codex Committee on Residues of Veterinary Drugs in Foods (CCRVDF) is responsible for establishing MRLs for veterinary drugs in foods and related details. Efforts related to international harmonization of MRLs are of interest to countries participating in trade of food animal products and to pharmaceutical companies that wish to market their products in multiple countries [51].

If a drug is not approved in a species, then a tolerance probably does not exist. If no tolerance exists, any detectable residue would be deemed violative following ELDU. Therefore, the WDI needs to be long enough to allow for residue depletion below the limit of detection for analytical methods used by regulatory authorities. This concept can result in a significantly extended WDI recommendation by FARAD in some cases.

Analytical testing method and limits of detection

Limits of detection for analytical methods that are used to measure residues are an important consideration when FARAD estimates WDIs, especially when there is no established tolerance. As mentioned previously, the WDI may need to be extended to achieve residue concentrations below the detection capability of the analytical method. FSIS publishes the results of the National Residue Program on a yearly basis known as the Red Book, which describes the analytical methods used by FSIS and their limits of detection (Table 20.2).

Foreign drug approval data

In some cases, a drug might lack US FDA approval, but be approved in another country. In these circumstances, FARAD may use foreign withdrawal times as a guide for recommending a WDI after ELDU. However, one should be mindful that the withdrawal time or withdrawal period for the drug with an approved foreign label would be based on an MRL that was established by that country, while there would be no FDA-approved tolerance for the drug in the United States. Therefore, when foreign labels are used to estimate withdrawal

interval recommendations following ELDU in the United States, the WDI must always be conservatively longer to allow the drug residues to deplete below the FSIS analytical methods' limit of detection. Table 20.2 includes a listing of international drug databases that have foreign-approved drug label information, including dose and withdrawal times.

Testing for drug residues

The analytical methods for detecting drug residues have a wide spectrum of needs when it comes to equipment, personnel expertise, reagents, and so on. Some analytical methods are cost-efficient, do not require highly skilled personnel, have simple equipment needs, and offer advantages of rapid testing. The opposite end of the spectrum includes sophisticated expensive techniques that are labor- and equipment-intensive. Sample preparation and drug residue analysis in poultry products have been previously described [52].

If a practitioner or producer/owner wants to confirm that poultry products (i.e., meat or eggs) are at or below the tolerance, they can consider submitting a limited number of flock-representative birds for drug residue testing. Age range, disease status, and gender representation are just a few things to consider when choosing birds to be representative of the flock. Samples or carcasses can be submitted to a commercial laboratory for residue testing. Another option for sample submission would be a state veterinary diagnostic laboratory. The web site URL for state veterinary laboratories in the United States is listed under AAVLD in Table 20.2.

Commercial rapid tests for drug residue testing in poultry are very limited in availability compared to those available for other food-producing animals. The commercial rapid tests for drug residue screening are typically either FDA or Association of Official Analytical Chemists (AOAC) approved. These rapid tests are evaluated for their ability to detect the targeted analyte in a matrix from the animal species from which the samples originated. An example of a manufacturer of rapid "commercial" chicken-side tests is Charm Sciences (charmsciences.com). Similar to recommendations made previously for other analytical tests, the limit of detection for the rapid tests should be at or below the tolerances in order to ensure that the poultry product can enter the human food chain.

Pharmacovigilence: Guidelines for prudent antibiotic drug use when medicating poultry

It is of upmost importance for veterinarians to use careful consideration when selecting antimicrobial therapy for poultry because of ongoing concerns regarding antimicrobial use in food-producing animals, the potential for microbial resistance, and growing concerns for protecting human public health. In some cases, especially with extremely resistant organisms, a veterinarian might have to advise as to whether or not treatment is appropriate or if euthanasia should be considered. This is especially important in households with immunocompromised individuals. In general, prolonged use of antimicrobials in a poultry population should be avoided to prevent the formation of antimicrobial-resistant reservoirs within the birds' normal bacterial flora.

When choosing an antibiotic, one with a narrow spectrum should be selected over a broad-spectrum agent when possible. Use of a broad-spectrum antibiotic can put selective pressure on non-target bacteria, thus increasing the likelihood of the development of resistance.

Guidelines for prudent antimicrobial use when treating poultry include

1. All antimicrobials (including over-the-counter medications) should be used under the supervision of a veterinarian
2. Good husbandry practices, including good hygiene, preventative strategies such as vaccination, probiotics, nutrition [53], and routine health monitoring, should be used to reduce the need for antimicrobials
3. Extra-label use of antimicrobials should be the exception (not the rule) and should only be performed

Table 20.10 Estimated feed consumption rates for chickens

Chicken body weight (lbs)	Chicken body weight (kgs)	Weight of food eaten per day expressed as percentage body weight
0.5	0.23	14
1.0	0.45	11.4
1.5	0.68	9.7
3.5	1.59	6.7
5.5	2.5	5.0

Source: Buck, B.B. [32].

Table 20.11 Health Canada's categorization of antimicrobial drugs based on importance in human medicine

Category I: Very high importance

These antimicrobials are deemed to be of very high importance in human medicine as they meet the criteria of being essential for the treatment of serious bacterial infections and limited or no availability of alternative antimicrobials for effective treatment in case of emergence of resistance to these agents. Examples include:
Carbapenems
Cephalosporins- 3rd and 4th generation
Fluoroquinolones
Glycopeptides
Glycylcyclines
Ketolides
Lipopeptides
Monobactams
Nitroimidazoles (metronidazole)
Oxazolidinones
Penicillin-β-lactamase inhibitor combinations
Polymyxins (colistin)
Therapeutic agents for tuberculosis (e.g. ethambutol, isoniazid, pyrazinamide and rifampin)

Category II: High importance

Antimicrobials in this category consist of those that can be used to treat a variety of infections including serious infections and for which alternatives are generally available. Bacteria that are resistant to drugs of this category are generally susceptible to Category I drugs, which could be used as alternatives. Examples include:
Aminoglycosides (except topical agents)
Cephalosporins- 1st and 2nd generations (including cephamycins)
Fusidic acid
Lincosamides
Macrolides
Penicillins
Quinolones (except fluoroquinolones)
Streptogramins
Trimethoprim/sulfamethoxazole

Category III: Medium importance

Antimicrobials in this category are used for treatment of bacterial infections for which alternatives are generally available. Infections caused by bacteria that are resistant to these drugs can, in general, be treated by Category II or I antimicrobials. Examples include:
Aminocyclitols
Aminoglycosides (topical agents)
Bacitracins
Fosfomycin
Nitrofurans
Phenicols
Sulphonamides
Tetracyclines

Table 20.11 (*Continued*)

Trimethoprim

Category IV: Low importance

Antimicrobials in this category are currently not used in human medicine. Examples include:
Flavophospholipols
Ionophores

Source: Abbas, R.Z., Colwell, D.D. and Gilleard J. [54].

under the supervision of a veterinarian and in compliance with stipulations set forth by AMDUCA

4. Antimicrobial therapy should be administered over as short a treatment period as possible at therapeutic doses to ill or at risk birds only
5. Culture and sensitivity results should be used when possible to guide antimicrobial selection
6. Records should be kept of all antimicrobial administration and may be used to evaluate efficacy and treatment protocols
7. Immunocompetent statuses of humans in direct or indirect contact with the medicated avian patient should be taken into account, especially when targeting highly resistant organisms. In some cases, if there are immunocompromised humans that will be in contact with the bird, a decision as to whether or not to treat the bird may be necessary. In some cases, euthanasia of the bird might need to be discussed.

Antimicrobials that are deemed less important for treating serious infections in humans should be used before more important antimicrobials are used. Canada Health has classified antimicrobials based on their importance for use in humans and the necessity of preserving their effectiveness [54]. These rankings are listed in Tables 20.10 and 20.11.

Editor's note: Abbreviated formulary

A complete listing of all medications that are approved for use in chickens and other backyard poultry is beyond the scope or size of this book. The FARAD website (under VetGram) lists over 600 approved water and feed medicated additives for chickens and turkeys. Each of these listed medications has specific guidelines on how it is used in an approved manner including such specifics as the type, size, and age of the bird, dose, formulation, duration, and indications for use. In an effort to provide user friendly and practical information,

an abbreviated formulary is provided, but these are just a few examples of medications commonly used in the United States Table 20.12. Vetgram is a user-friendly resource that has sorting capabilities and withdrawal time tables for FDA-approved veterinary products that are available in the United States for poultry and can be found on the FARAD website (http://farad.org). The US Food and Drug Administration (FDA) Center for Veterinary Medicine also has a searchable database called "Animal Drugs@FDA" and can be found on the FDA web site (http://www.fda.gov/). The Compendium of Veterinary Products also provides a list of medications, and details of their withdrawal times for chickens and turkeys and can be found at http://bayerall.naccvp.com. The Minor Use Animal Drug Program also has a database on their web site (http://www.nrsp7.org/) that allows individuals to search for approved drugs for different animal categories and has an avian-specific section. In general, it is important to note that there are very few FDA-approved medications that can be administered to a chicken that lays eggs for human consumption. For commercial layers, it is important for the FDA-approved withdrawal time to equal 0 days, thus there are limited numbers of FDA-approved medications. The website that accompanies this book provides a list of medications and their withdrawal times for Australia and the United Kingdom. The resource table (Table 20.1) lists URLs for approved drugs in other countries. To date, a single global database for approved animal drugs does not exist.

Pharmacokinetic and pharmacodynamic studies on various medications used in poultry are available in the literature, but the doses involved may be one-time doses, studied in healthy young animals or a single species of bird, or a dose or medication that is prohibited, deemed extra-label drug use, or does not pertain to the patient at hand for some other reason. An excellent source of doses for various medications for poultry, including references to research published in the literature, is available in the avian chapter of the "Exotic Animal Formulary," 4th edition, by James W. Carpenter. It includes individual doses, with references to research or anecdotal information, for over 50 antimicrobial medications for ring-necked pheasants, chickens, waterfowl, poultry, Galliformes, quail, and peafowl [55].

Table 20.12 Abbreviated formulary.

Abbreviated formulary for backyard poultry listing specific examples of commonly used medications and the approved method of their use according to the FARAD website accessed on 2-1-13.

Meat, meat withdrawal time, C, chicken, T, turkey, water additives are to be made fresh daily, used as the sole source of drinking water, dose, amount of medication added to drinking water to attain a final concentration, gal, gallon, g, grams, d, days, BW, body weight, IC, Infectious Coryza, FC, Fowl Cholera, PD, Pullorum disease, MG, Mycoplasma gallisepticum, MS, Mycoplasma synoviae, MM, Mycoplasma meleagridis, DW, drinking water, TE, Transmissible Enteritis (also known as Coronavirus enteritis), CRD, Chronic respiratory disease.

Medication	Indication	Meat	Dose	Directions
Sulfadi-methoxine, soluble powder, water additive, 94.6 g/packet	Anticoccidial, also used for Fowl Cholera and Infectious Coryza	C, 5d T, 5d	C, 1.875g/gal (0.05%) x 6 consecutive days T-0.938 g/gal (0.025%) x 6 consecutive days	C, for broilers and replacement only, not for egg laying hens, do not administer to chickens over 16 weeks of age T-do not administer to turkeys over 24 weeks of age
Sulfamethazine sodium, 12.5% solution, water additive	C, Anticoccidial (*E. tenella, E. necatrix*), IC, acute FC, and PD T, Anticoccidial	C, 10d T, 10d	C, 61-89g/lb BW/day T-53-130mg/lb BW/day	C, chickens, not laying hens, for IC medicate for 2 consecutive days, for PD or acute FC medicate 6 consecutive days, for coccidia use dose given 2 consecutive days and then use half that dose for 4 days T, for coccidia use dose given 2 consecutive days and then use half that dose for 4 days

Table 20.12 (*Continued*)

Medication	Indication	Meat	Dose	Directions
Oxytetracycline HCl, 50 mg/ml, SQ injection	C and T, Air sacculitis or sinusitis caused by MG or *E.coli*, FC, MS	C-5d T-5d	**Chickens:** 1d–2wks (6.25 mg/bird/day diluted 1 part medicine to 3 parts sterile water), 2–4 wks (12.5 mg/bird/day diluted 1 part medicine to 3 parts sterile water), 4–8 weeks(25 mg/bird/day undiluted), 8 wks or broilers and light pullets (5 mg/bird/day undiluted), Mature (100 mg/bird/day) **Turkeys:** 1d–2wks (6.25 mg/bird/day diluted 1 part medicine to 3 parts sterile water), 2–4 wks(12.5 mg/bird/day diluted 1 part medicine to 3 parts sterile water), 4–6 wks (50 mg/bird/day), 6–9 wks (100 mg/bird/day), 9–12 wks (150mg/bird/day), 12+wks (200 mg/birs/day)	C, chickens, not for laying hens, do not administer to laying hens unless eggs are used for hatching only T, turkeys not laying eggs for human consumption C and T: treatment should continue for 24–48 hours past remission of disease symptoms but not to exceed a total of 4 consecutive days T, 0.25–0.5 ml can be injected directly into each swollen sinus q5–7days concurrently with SQ injection for the treatment of infectious sinusitis caused by MG
Spectinomycin dihyrochloride pentahydrate, 100 mg/ml solution, SQ injection	C, to control mortality and decrease severity of infections caused by MS, *salmonella typhimurium* and *S. infantis, E.coli* T-prevent mortality associated with Arizona group infection, chronic respiratory disease with *E.coli* or MM	C, 0d T, 0d	C, 2.5–5.0 mg/chick T, 1–2 mg/poult at nape of neck	C, 1–3-day-old baby chicks T, 1–3-day-old poults
Spectinomycin dihydrochloride pentahydrate plus lincomycin HCl monohydrate, soluble powder, water additive, (each 75 gram packet contains: 33.3 mg spectinomycin and 16.7 mg lincomycin), (each 375 gram packet contains 166.5 mg spectinomycin and 83.5 lincomycin)	C, air sacculitis from MS, MG or chronic respiratory disease from MG or *E.coli*	C, 0d	C, 2 gm of total antibiotic/gal DW	C, up to 7 days of age

Table 20.12 (*Continued*)

Medication	Indication	Meat	Dose	Directions
Tylosin tartarate 100 g tylsin/bottle, water additive	C, broilers: aids in the treatment of chronic respiratory disease from MG, can be given during vaccination or other times of stress T, for weight gain, better feed efficiency, infectious sinusitis caused by MG	C-1d T-5d	C, 2 gm/gal DW (providing a dose to the chicken of about 50 mg/lb BW/day) T, 2 gm/gal DW (providing a dose to the chicken of about 60 mg/lb BW/day)	C-chickens, not egg laying hens, use 1–5 days T-use 2–5 days
Tetracycline soluble powder, water additive, 25 g tetracycline HCl/lb of medication as typically sold.	C, Chronic respiratory disease caused by MG, *E.coli*, Infectious sinusitis caused by MS T, for infectious sinusitis caused by MS, for the control of complications from bacteria associated with Bluecomb infections (TE)	C-4d T-4d	C, for CRD: 400-800mg/gal of DW x 7–14 days; for IS: 200–400 mg//gal DW x 7–14 days T-for IS: 400mg/gal DW x 7–14 days	C, chickens, not laying hens T, turkeys, not laying hens
Amprolium 20%, soluble powder, water additive	Anticoccidial medication	C-0d T-0d	C and T-0.012% in DW x 3–5 days (if severe can use 0.024% in DW) then continue with 0.006% in DW an additional 1–2 wks	C and T-ALL use classes, for use in growing chickens and laying hens
Dipiperazine sulfate plus piperazine HCl, oral formulation generally sold in 17 g, 34 g, or 230 g packets	Antihelminthic against roundworms (Ascarid spp.)	C-14d T-14d	C-<6wks of age 50mg/bird; >6wks of age 100mg/bird; flock treatment: 0.2%-0.4% in feed or 0.1-0.2% in DW T-<12wks 100 mg/bird; >12wks 200 mg/bird	C-chickens, not laying hens, treat only 1–2 days T-ALL use classes, treat one day

Conclusion

Veterinarians treating individual birds or poultry flocks have the challenging responsibility of protecting human public health while simultaneously ensuring avian health. It was the intent of the authors of this chapter to provide a comprehensive review of medication use in poultry and approaches for judicious and responsible drug use, because veterinarians are professionals who are well-suited to the task of educating owners and producers. To aid in this endeavor, readers were provided with information regarding US legislation affecting medication use in poultry, guidance recommendations regarding legal and prudent on-label and extra-label drug use in poultry, and approaches for establishing withdrawal intervals when drugs are used in an extra-label manner. In addition, resources listing withdrawal times for approved poultry drugs in the United States and other countries were provided. After reading this chapter, veterinarians will hopefully be

more informed about how to better serve their clients and patients while still helping to protect the human food chain.

Acknowledgements

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Vaccination of Poultry

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Vaccines that are available for commercial poultry are rarely used in backyard poultry. Nonetheless, a description of the available vaccines is presented here for reference and understanding. Chicks for backyard use should be purchased previously vaccinated for Marek's disease *in ovo* (in the egg) or at one day of age [20,24]. Vaccination for other agents might be indicated if a small flock incurs an abnormally severe or yearly challenge with an infectious agent such as infectious laryngotracheitis or fowl pox. Live vaccines should be applied judiciously in small flocks, especially if the flock are not to remain closed. Transporting vaccinated birds to exhibitions can allow for transmission of live vaccine agent to non-vaccinated birds (e.g., Newcastle disease virus, infectious laryngotracheitis virus, Mycoplasma gallisepticum). For this reason, application of live vaccines to birds that intermittently leave the flock for exhibition purposes is discouraged.

Poultry vaccines are used to immunize birds against three groups of agents: Viruses, bacteria, and parasites. Vaccines are applied in several forms: Inactivated (killed), live, or live recombinant vaccines [4,6]. Routes of administration of vaccines vary and include injection, *in ovo* (in egg) injection, drinking water, spray, wing web, and eye drop. The choice of route of administration depends on various factors such as the type of animal (broilers vs. turkeys vs. commercial layers), age of animal (1-day-old vs. 8 weeks), disease for the vaccination (Marek's disease or Newcastle disease), type of vaccine (Infectious laryngotracheitis – tissue culture vs. chick embryo origin, or genetically engineered/recombinant), and the labor involved in administering the vaccine (availability and cost of labor).

Inactivated (killed) vaccines

Inactivated (killed) vaccines usually apply to either viruses or bacteria**.** Killed vaccines usually result in high, long lasting, and uniform immunity. Killed antigen (the water phase) is encapsulated in oil or aluminum hydroxide, and is therefore known as a water in oil emulsion. Inactivated vaccines are administered by either intramuscular (IM) or subcutaneous (SQ) injection [23].

Advantages of inactivated (killed) vaccines

Killed vaccines should have fewer systemic reactions than other forms of vaccine because there is no live agent. Fewer revaccinations are needed with the use of inactivated vaccines (more direct hits). There is no risk of the antigen spreading to other birds, and long term immune response is generated; therefore it is usually used in breeders. Inactivated vaccines reduce the risk, compared to live vaccines, of interference when using multiple antigen combinations. There are a number of ways to properly administer an injection with a killed vaccine; as long as a full dose of the vaccine gets under the skin (not on the feathers) or in the muscle (not in the abdominal cavity, blood vessels, or bones) it should be effective [3,16].

Disadvantages of an inactivated (killed) vaccine

Disadvantages of an inactivated vaccine include the need to handle the birds (labor cost increased), adjuvant in the vaccine can adulterate tissue or leave residual oil, contaminated needles can transmit bacterial infection, and the vaccine can cause an exaggerated tissue reaction (usually caused by the adjuvant) [15,23].

Routes of administration of inactivated (killed) vaccines

Intramuscular injection

Intramuscular injections of inactivated vaccines are usually administered in the leg, breast, or possibly wing of turkey breeders. Injection in the leg is usually only recommended for commercial layers and is administered by injecting towards the head of the bird on the outside of the leg [9,15,21]. This should help to avoid nerves and major blood vessels. The breast (pectoral muscle) injection is administered in the thickest portion of the breast. Angled injection towards the head is recommended. The breast should be the easiest target area to hit, with very few misses; however, the breast should be injected at an angle to the skin surface to prevent direct injection into the thoracic cavity (Figure 21.1). Spent hen processors do not like to find residual oil in the white breast muscle. This residual oil from the vaccine can show up as dark spots in the white meat after cooking. The wing injection, which can be performed on turkey breeders, is administered to the ventral side of the wing [11,15].

Subcutaneous injection

Subcutaneous (SQ) injections are used for both viral and bacterial killed vaccines. The preferred route for SQ injections is the neck in case there are tissue reactions [9,22]. Injection should take place in the lower third of the neck where there is room for skin to expand. Avoid the esophagus or crop. If injection is placed too rostrad on the neck adverse swelling of the neck and head may occur [12,15,23]. Both subcutaneous and intramuscular injections of killed vaccines over the breast muscle should be avoided in meat-type birds to prevent adulteration of meat that is to be consumed (Figure 21.2).
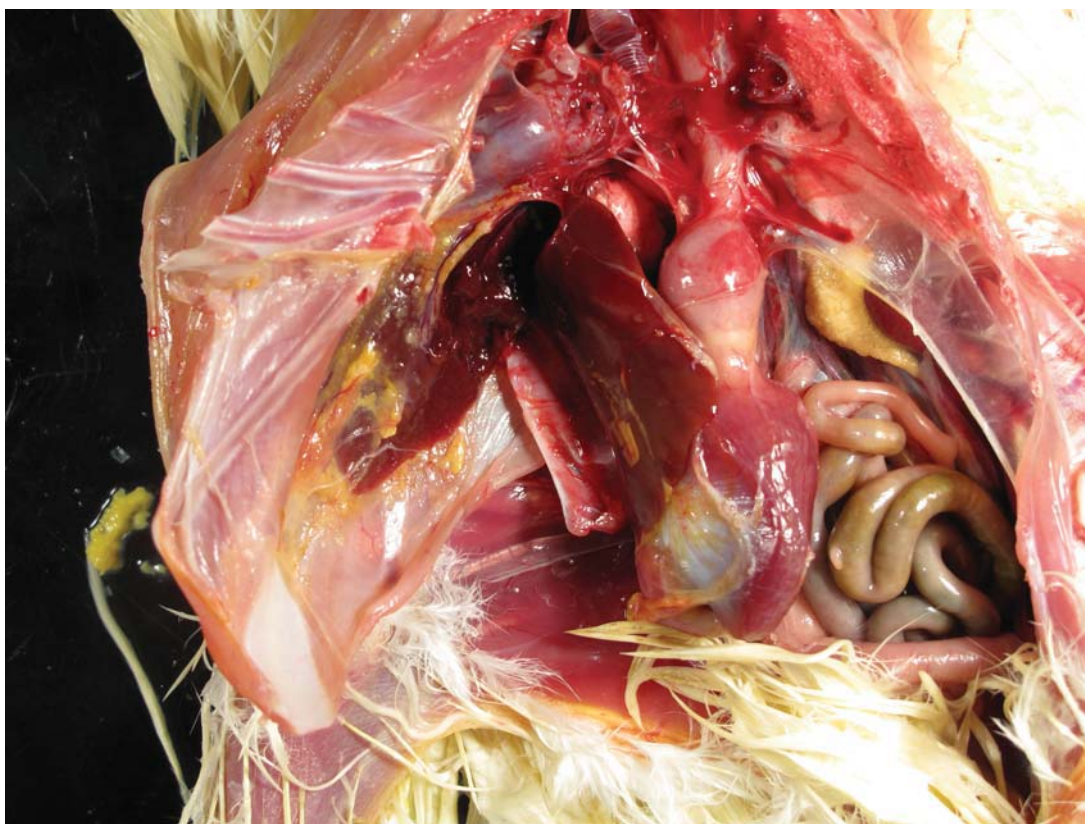
Figure 21.1 Layer pullet. Killed vaccine adjuvant and associated inflammatory reaction around the liver resulted from vaccination needle penetrating both the breast muscle and thorax, depositing vaccine directly into coelomic cavity.
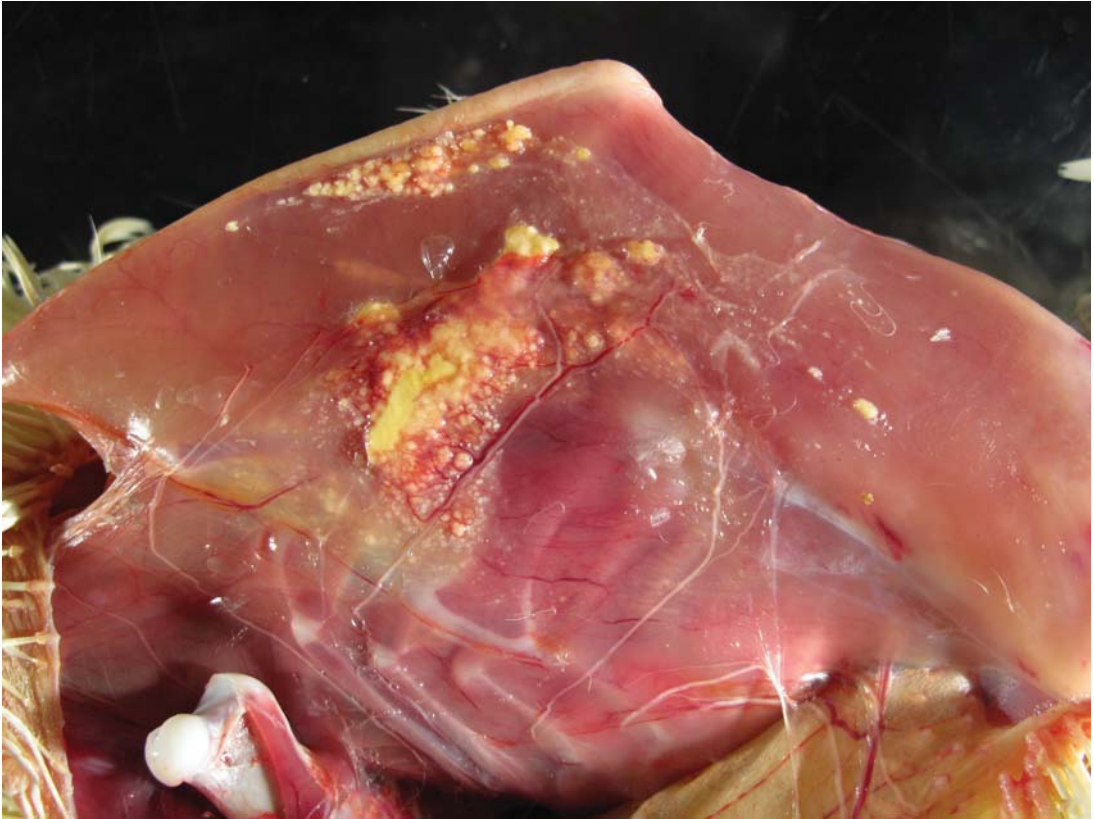
Figure 21.2 Killed vaccine injected into breast muscle or adjacent subcutis can result in adulterated tissue that is unfit for consumption.

Directions for injection

Always warm the inactivated vaccine to at least room temperature or preferably to 90°F prior to use. Cold vaccine can irritate the birds (make them uncomfortable) and does not flow well (low viscosity) through the needle. This creates more effort for injection and can increase wear on the syringe. Usually 1/4-1/2 inch needle length is used. Needle guards can be used to protect the vaccinator, but this may require a longer needle. Change needles at least every 1000 birds in commercial settings [12,23].

Live vaccines

Most vaccines, particularly viruses, are administered as live agents because of the advantages, but procedures for administration must be closely followed to ensure that birds are adequately exposed and the live agent is not inactivated during administration.

Advantages of live vaccines

Live vaccines are usually easier to apply compared to killed vaccines; there is often a faster application time; they are superior in inducing mucosal immunity compared to inactivated vaccines; they can be stored for longer periods (freeze dried) than killed emulsion vaccines; and no skin or muscle reactions occur [14,25].

Disadvantages of live vaccines

Live vaccines can induce adverse reactions in the respiratory tract; they require rapid application (careful attention to time is required); they have a limited half-life for microorganisms in suspension; and they induce a shorter humoral immune response compared to killed vaccines [1,14,23].

Routes of administration for live vaccines
Injection

Injection into the cervical or pipping muscle is used for Marek's Disease vaccination in 1-day-old chicks as well as recombinant Newcastle disease vaccines in 1-day-old turkeys [8,17,19]. This practice for Marek's disease vaccination has largely been replaced in large chicken hatcheries by *in ovo* injection, but is still used in small hatcheries. Bacterial contamination of the injection needle can cause localized infection and death in young birds (Figure 21.3).

In ovo

In ovo vaccines are used for vaccinating with Marek's disease and possibly IBD, coccidiosis, and some recombinant vaccines in embryos before hatch at transfer time from the incubator to the hatcher. For optimal performance, *in ovo* inoculation must be performed between 18 and 19 days of incubation either via the amniotic or the intraembryonic route. This greatly reduces labor costs in the hatchery, but must be closely monitored to avoid contamination [24].

Eye drop and nasal

Eye drop and nasal vaccines can be used for infectious laryngotracheitis virus and Mycoplasma gallisepticum (ts-11) vaccination. A blue dye mixed with the vaccine greatly aids in checking vaccination procedures. The back of the bird's tongue is stained blue for a short time after vaccination, so check for the staining within 10 minutes of vaccination. If the vial cannot be used

Figure 21.3 Three-day-old turkey poult that received a cervical muscle injection of recombinant Newcastle disease vaccine through a needle contaminated with Enterococcus sp. at one day of age resulting in muscle necrosis and death.

in one hour, discard the remaining vaccine and mix a fresh bottle. Better yet, mix a 1000-dose bottle and split between two people on the vaccination crew to ensure fresh vaccine is being used at all times [3,7,13].

Wing web vaccine

The wing web (patagium) vaccine is used for fowl pox, avian encephalomyelitis virus, Pasteurella multocida (fowl cholera), and chicken anemia virus (CAV). A blue dye and red dye are used to monitor application as the birds are being vaccinated. Two colors should be used when performing two separate wing web vaccinations - pox and Pasteurella multocida. Take care not to wipe off the vaccine on the feathers prior to piercing the wing web. Immediately after applying the vaccine, the wing web should show dye between the layers of the skin. The wing web vaccinations should be administered in the center of the wing web (patagium). Successful application of pox or cholera to the skin results in localized skin inflammation ("reactions" or "takes") [2,13]. These should be checked 7–10 days after vaccination, looking for a scab or thickening of the wing web. Less than 90% takes may require revaccination of the flock. Because of the larger takes from *Pasteurella multocida* vaccine it is critical to inject or stab in the center of the wing web. Keep an accurate and detailed record of who was applying the vaccine in case trouble shooting is necessary [12,16].

Drinking water

Drinking water vaccines are used for Newcastle disease virus, infectious bronchitis virus, Infectious bursal disease virus, infectious laryngogtracheitis virus, avian encephalomyelitis virus, and others [5]. Water vaccination appears easy - mix up the vaccine and run it through the proportioner - and might be less labor intensive than spray or injection, but there are challenges to proper administration. Consider the following points: What is the capacity of the water lines? What is the daily water consumption of the flock? Do all the birds drink when you are vaccinating? Is the water being treated with sanitizers or antibiotics? Have you applied a stabilizer to protect the vaccine in the drinking water?

The following are recommended procedures for drinking water vaccination: (i) Clean the water lines before vaccination; (ii) Birds should be water deprived ("water starved") for about 2 hours prior to vaccinating and the lights should be turned off [10].; (iii) Fill the water lines with vaccine, approved dye and vaccine stabilizer either while the lines are raised above the birds or before the lights are turned on; (iv) Make sure all water lines contain vaccine by observing the dye at the end of the water lines. Consider allowing at least an extra 15 seconds of vaccine water to flow through to make sure there is adequate vaccine in the entire line; (v) Lower the water lines to bird level or turn on the lights; (vi) The remaining vaccine stock solution should last for at least 30 minutes after the birds begin to drink; (vii) Running automatic feeders during vaccination can increase water consumption [3,10,27].

Spray vaccination

Spray vaccination includes coarse spray, fine spray, and very fine spray or aerosol and can also be used for most of the drinking water vaccines. A coarse spray is defined as 100 microns or larger. A medium spray is 50–100 microns, and a fine spray is <50 microns. Important factors to consider are the size of droplet that should be used for each vaccine. Finer droplets penetrate deeper into the respiratory tract to increase the chance of vaccine reaction. You should be familiar with the type of sprayer that you plan to use and clean it thoroughly after each use. There are many models of commercial sprayers, with a variety of features, from which to choose. Other factors to consider include the volume of water to spray on the birds and how long these droplets are to remain suspended in the air. Automated ventilation (fans and blowers) should remain off during spray vaccination to prevent vaccine being drawn to the outside of the house, but should be immediately turned back on after administration to prevent overheating of the flock [25,27].

Spray vaccination is an excellent method for mass administration of vaccine. Distilled water is recommended. A strong local immunity can be elicited if the spray reaches the mucous membranes, but these tissues also show more reaction if spray droplets travel too deep in the respiratory tract [18,22]. The depth of penetration of the spray depends upon the size of the droplets. Evaporation can change a coarse spray into a fine spray. Low spray pressure usually produces a coarse spray and high pressure produces a fine spray. The use of water-sensitive paper can show the droplet size and where the spray is actually going inside the house [25,26]. It is best to spray during the cooler portion of the day because house fans are turned off while the spray is applied to the birds. Birds that are overheated pant and can inhale a coarse droplet even deeper into the respiratory tract to cause a harsh reaction. Dim or shut off the lights to calm the birds when spraying.

Coarse spray (100–200 microns and larger) is used for initial vaccinations and when low reaction is desired.

This spray falls out of the air very quickly, having the appearance of a "wet fog." [25] Birds shake their heads to indicate that the spray is getting into the eyes; therefore, you can directly observe whether the birds are being vaccinated. The spray should be directed at or slightly above the head of the birds. Some vaccines, such as IBD and Salmonella, should only be administered by coarse spray when sprayed. 100-micron droplets fall 10 feet in 11 seconds [26].

Medium spray (about 50–100 microns) is used for revaccination of Newcastle disease and infectious bronchitis viruses. Direct the spray slightly above the head of the birds to allow them to breathe the spray in. Fifty-micron droplets fall 10 feet in 40 seconds and have the appearance of "misty rain." [21,25,26]

Fine spray (20–50 microns or smaller) can also be used for revaccination of Newcastle disease and infectious bronchitis viruses, but there is a greater risk of reaction. It can be used for initial vaccination of Mycoplasma gallisepticum (Mycovac-L). With this very small droplet size the person applying the vaccine should slow the walking pace to allow the vaccine to reach all birds [25,27]. Ten-micron droplets fall 10 feet in 1020 seconds (17 minutes) [26]. Use of medium to fine spray vaccine

Figure 21.4 Bantam chick received a fine droplet aerosol of LaSota strain of Newcastle disease, resulting in exaggerated inflammatory response ("vaccine reaction") observed as white bubbles within the tracheal lumen.

in susceptible birds can promote excessive vaccine reactions as a result of the virus extending deeper into the respiratory tract (Figure 21.4).

For examples of vaccination programs for chickens see Table 21.1 for Layers, Table 21.2 for Broiler Breeders, and Table 21.3 for Broilers.

Table 21.1 Vaccination program for layers

Vaccine	Age	Comment	Administration
Marek's Disease	1 day	HVT, SB-1, Rispens	Subcutaneous injection
Infectious Bronchitis	1 day	Mass-Conn,	Coarse spray (100 micron)
Newcastle disease	1 day	B1-B1	Coarse spray (100 micron)
Infectious Bursal Dz	14 days	Intermediate strain	Water or coarse spray
Infectious Bronchitis	14 days	Mass-Conn,	Coarse spray (100 micron)
Newcastle disease	14 days	B1-B1	Coarse spray (100 micron)
Infectious Bursal Dz	28 days	Intermediate strain	Water or coarse spray
Infectious Bronchitis	28 days	Mass-Conn,	Coarse spray (100 micron)
Newcastle disease	28 days	B1-B1	Coarse spray (100 micron)
Infectious Bronchitis	6 weeks	Mass-Conn,	Medium spray (50 micron)
Newcastle disease	6 weeks	Lasota	Medium spray (50 micron)
Infectious Laryngo-	7–8 weeks	Eye drop/drinking water tracheitis	
M. gallisepticum	10 weeks	F strain or 6/85	Fine spray (20 micron)
Infectious Bronchitis	12 weeks	Holland	Fine spray (20 micron)
Newcastle disease	12 weeks	Lasota	Fine spray (20 micron)
Poxvirus	12 weeks	Fowl/Quail Pox Combo	Wing web stick
Av. Encephalomyelitis	12 weeks	Combined with pox	Wing web stick
Infectious coryza	12 weeks	In problem flocks	Subcutaneous injection
Infectious Bronchitis	Every 8 weeks	Mass-Conn,	Medium spray
Newcastle disease	Every 8 weeks	B1-B1	Medium spray

Table 21.2 Vaccination program for broiler breeders

Vaccine	Age	Comment	Administration
Marek's Disease	1 day	HVT, SB-1, Rispens	Subcutaneous injection
Infectious Bronchitis	1 day	Mass-Conn,	Coarse spray (100 micron)
Newcastle disease	1 day	B1-B1	Coarse spray (100 micron)
Reovirus	7 days		Subcutaneous injection
Infectious Bursal Dz	14 days	Intermediate classic	Water or coarse spray
Infectious Bronchitis	14 days	Mass-Conn,	Coarse spray (100 micron)
Newcastle disease	14 days	B1-B1	Coarse spray (100 micron)
Infectious Bursal Dz	28 days	Intermediate classic	Water or coarse spray
Infectious Bronchitis	35 days	Mass-Conn,	Coarse spray (100 micron)
Newcastle disease	35 days	B1-B1	Coarse spray (100 micron)
Reovirus	6 weeks	Live or inactivated	Subcutaneous injection
Infectious Laryngo-	7–8 weeks	In problem regions	Eye drop or drinking water tracheitis
Infectious Bursal Dz	8 weeks	Classic + variant	Water or coarse spray
Poxvirus	12 weeks	Fowl/Quail Pox Combo	Wing web stick
Av. Encephalomyelitis	12 weeks	Combined with pox	Wing web stick
Infectious Bursal Dz	12 weeks	Inactivated	Subcutaneous injection
Reovirus	12 weeks	Inactivated	Subcutaneous injection
Fowl Cholera	12 weeks	Inactivated	Subcutaneous injection
Infectious Bronchitis	13 weeks	Holland	Water or spray
Newcastle disease	13 weeks	Lasota	Water or spray
Fowl Cholera	18 weeks	Inactivated	Subcutaneous injection

Table 21.3 Vaccination program for broilers

Vaccine	Age	Comment	Administration
Marek's Disease	−3 to 1 day	HVT, SB-1, Rispens	SubQ injection or *in ovo*
Infectious Bursal Dz	−3 to 1 day	Variant strain	SubQ injection or *in ovo*
Infectious Bronchitis	1 day	Mild Mass-Conn,	Coarse spray (100 micron)
Newcastle disease	1 day	B1-B1	Coarse spray (100 micron)
Infectious Bursal Dz	7 days	Classic/Variant strain	Water or coarse spray
Infectious Bronchitis	14 days	Mass-Conn,	Coarse spray (100 micron)
Newcastle disease	14 days	B1-B1	Coarse spray (100 micron)

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