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RESPIRATORY DISEASES OF GALLINACEOUS BIRDS

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The veterinary practitioner can be requested to provide service to the owners of gallinaceous birds under many different circumstances. A large commercial flock of meat- or egg-producing birds, birds from smaller flocks belonging to independent producers of poultry and eggs, the backyard poultry flock, a hobby flock of poultry or show birds intended for 4-H or Future Farmers of America (FFA), or even the occasional pet or game chicken can be the patient. Owners of flocks of gamebirds or ornamental fowl, including quail, partridge, pheasants, and guinea or peafowl, also can present birds for diagnosis and treatment.

Most large producers of commercial poultry have access to veterinary service provided by specialists in poultry medicine. The approach to commercial poultry practice is generally one of population medicine at the flock level. The main emphasis of this article is on respiratory medicine practiced at the level of the small flock and possibly the individual bird.

Respiratory diseases constitute a significant proportion of all disease conditions in both commercial and small backyard flocks.[90, 105, 108] Serologic evidence of infection with respiratory disease agents in backyard poultry located close to commercial flocks suggests the potential for disease transmission among such flocks.[83]

The same respiratory diseases affect birds in both commercial and backyard flocks. Because of the intensive confinement systems, high population density, and management procedures used for commercial poultry operations, complicated infections caused by multiple causal agents are more commonly observed than are simple infections.[70] Backyard poultry and gamebirds are more likely to have respiratory conditions arising from a single cause.[57, 118]

The potential origins of respiratory conditions in gallinaceous birds are quite varied. Infections with viruses, mycoplasmas, chlamydia and other bacte-

From the Texas Veterinary Medical Diagnostic Laboratory, College Station, Texas

ria, fungi, helminths, and protozoa, as well as injury caused by toxins, gaseous irritants, and nutritional deficiency, all can result in respiratory disease.

CLINICAL SIGNS AND GROSS LESIONS

The clinical signs of respiratory disease in gallinaceous birds include the production of nasal exudate and ocular discharge, distention of the infraorbital sinuses, rales, coughing, sneezing or dyspnea.[62] Similar signs can result from either infectious or noninfectious insults to the respiratory system. Clinical examination of the individual patient helps to determine which specimens to collect and how to evaluate the specimens, and can suggest the most likely diagnosis. Establishing a specific diagnosis often requires additional diagnostic tests because the clinical signs of many respiratory conditions are similar.

Nasal exudation is a common clinical sign in most upper respiratory conditions but is most severe in bacterial infections, such as infectious coryza *(Hemophilus paragallinarum)* and fowl cholera *(Pasteurella multocida)* in chickens. The discharge varies from serous to mucoid. This exudate can be associated with infraorbital sinusitis and is sometimes seen with conjunctivitis and ocular discharge.

Oculonasal discharge, conjunctivitis, and swelling of the infraorbital sinuses can be observed with viral infections such as Newcastle disease (ND), infectious bronchitis (IB), infectious laryngotracheitis (ILT), avian influenza (AI), and fowl pox. Other causes for these signs include mycoplasmosis, *Bordetella avium* infection in turkeys, cryptosporidiosis, chlamydiosis, aspergillosis, vitamin A deficiency, and high levels of environmental ammonia.

The most common cause of swelling of the infraorbital sinuses in gallinaceous birds is mycoplasma infection. This lesion is very prominent in turkeys, in which *Mycoplasma gallisepticum* (MG) infection is referred to as *infectious sinusitis.* Pheasants, partridges, peafowl, bobwhite, and coturnix quail also react in a manner similar to turkeys.[35, 79, 106] Facial swelling is a less prominent feature of MG infection in chickens but occasionally is observed in affected birds.[96] The causal agent of infectious synovitis, *Mycoplasma synoviae* (MS), generally results in lameness and swollen joints in chickens and turkeys. It has also been reported to cause sinusitis in turkeys, guinea fowl, and partridges.[78, 98, 101, 110]

Infectious coryza, caused by the bacterium *Hemophilus paragallinarum,* does cause marked sinus swelling in chickens. Usually, this disease is found only in chickens, but it has been reported in coturnix quail in Australia.[104] The nasal exudate initially is serous but becomes mucoid and occasionally caseous as the infection becomes more chronic. The change in consistency of the exudate is not specific for infectious coryza and is more indicative of chronicity than of any specific cause. Many cases of infectious coryza are complicated by underlying MG infection.

Keratoconjunctivitis with epiphora resembling ocular exudation often is due to exposure to high levels of environmental ammonia. This toxic gas causes thickening and roughening of the cornea, accompanied by edema and swelling of the conjunctiva. This condition is the result of excessive ammonia production in poultry houses with wet litter and poor ventilation.[139] A visit to the farm to observe and smell the environment of the birds is valuable to confirm the diagnosis. Ammonia exposure also damages the respiratory membranes and predisposes to infection with various respiratory pathogens.[7, 94]

Another noninfectious cause of keratoconjunctivitis and sinus swelling is vitamin A deficiency. Early in the condition, watery discharge from the eyes and

nares occurs. The eyelids may stick together, and eventually the conjunctival sacs fill with caseous exudate. Similar exudate progresses from serous to caseous in the nasal cavity and infraorbital sinuses, causing distention. Vitamin A deficiency results in squamous metaplasia of mucous membranes and mucous glands, which results in distention of the glands, a condition that then resembles small pustules in the oral cavity and the esophagus. These gross lesions allow differentiation of vitamin A deficiency from infectious causes of sinus swelling and conjunctivitis. Severe deficiency can cause epithelial changes in other systems, including the ureter. Subsequent obstruction of the ureter results in gout secondary to renal failure. Such deficiency usually is associated only with a history of feeding rations consisting mostly of unsupplemented milo, wheat, or corn.[15]

Vitamin A is necessary not only for the maintenance of mucous membranes and epithelial surfaces (which provide a first line of defense against infections), but also to serve a regulatory role in the immune response.[10, 67] Birds with vitamin A deficiency are unable to mount an adequate immune response to infection with respiratory agents or to respond to vaccination against respiratory disease agents.[53]

Abnormal respiratory noise and dyspnea generally are associated with disease of the larynx, trachea, and pulmonary bronchi.[62] Excessive mucus can be clinically observed in the choanal cleft, glottis, and trachea. If the tissues are severely damaged, the exudate can be turbid owing to the presence of sloughed epithelium, inflammatory cells, and hemorrhage.

The oral cavity sometimes has raised yellow plaques in cases of the diphtheritic or wet form of avian pox virus infection. Similar lesions can form in the larynx, resulting in occlusion of the glottis, and suffocation. Some birds in a flock affected by the wet form of fowl pox also exhibit the more typical proliferative pox lesions on the unfeathered portions of the face, beak, nares, comb, wattles, legs, and feet. Both the cutaneous and diphtheritic forms of avian pox virus infection occur in quail, pheasant, and peafowl, although less frequently than in chickens and turkeys.[2, 5, 99]

Atypical presentation of ILT can resemble wet pox with ulceration and crusting of the laryngeal mucosa around the glottis.[37] This type of ILT infection is associated with a chronic low level increase in flock mortality rates. Combined avian pox and ILT infections also have been reported.[48, 135]

The most typical form of ILT is an acute infection, resulting in necrohemorrhagic tracheitis with clinical signs of severe dyspnea, squealing noises, and expectoration of bloody exudate. This form is generally accompanied by a high mortality rate. Dead birds in such cases have frank hemorrhage or caseonecrotic exudate within the tracheal lumen. Pheasants and peafowl are susceptible to ILT and have clinical signs and gross lesions similar to those found in chickens.[39]

When poultry flocks suffer high mortality rates with the postmortem finding of blood-tinged tracheal exudate, the differential diagnosis should also include exotic ND as well as highly pathogenic avian influenza (HPAI) infection. These other two diseases are characterized by much more generalized vascular and multisystemic lesions.[1, 31, 65] Sporadic outbreaks of exotic ND and HPAI accentuate the importance of considering these conditions in the differential diagnosis of respiratory disease and of obtaining a rapid diagnosis.[24, 40] Newcastle disease virus (NDV) and avian influenza virus (AIV) both have an extremely wide host range and affect many avian species, but ILT affects only chickens, pheasants, and peafowl.[3, 11, 46] All three of these conditions generally are considered to be reportable diseases in most states when the diagnosis is confirmed.

In the less virulent forms of ND caused by endemic lentogenic strains of

the virus, and disease caused by low pathogenic strains of AIV or ILT virus, tracheal lesions are usually limited to the presence of excessive mucus on the luminal surface.[25, 76] Similar catarrhal tracheitis is observed with infections caused by infectious bronchitis virus (IBV) in chickens, quail bronchitis in quail, and *Bordetella avium* infection in turkeys.

Young chickens with IB may exhibit moist rales and severe dyspnea as a result of caseous plugs in the trachea and bronchi. Mortality results from suffocation. The typical form of the disease in older chickens is a mild respiratory infection with lesions limited to mild catarrhal tracheitis. Adult hens can have a concurrent decline in egg production, with reduced egg quality characterized by thin albumen and misshapen, pale eggshells.[29] Similar reproductive effects are also associated with most other viral respiratory infections in adult birds. Chickens generally are considered to be the only species affected by IBV, which is a coronavirus, however there is a single report of isolation of IBV from pheasants.[129]

A high mortality rate is a characteristic of quail bronchitis in quail chicks less than 4 weeks of age. Chicks have clinical signs of coughing, sneezing, and conjunctivitis, and try to huddle near a heat source. At necropsy, gross lesions include excessive mucoid tracheal exudate with extension into the anterior air sacs, consolidation of the hilar region of the lungs, and occasionally pale foci of necrosis in the liver.[61, 107] The disease is caused by an adenovirus that is closely related to CELO virus from chickens.[44] The disease was reported in adult coturnix quail, in which it resulted in reduced egg production and soft-shelled eggs with reduced shell pigmentation. Mortality was negligible, and gross lesions consisted of conjunctivitis, sinusitis, tracheitis, and pulmonary congestion.[32] Similar reproductive effects in adult bobwhite quail were associated with inclusion body salpingitis caused by a serogroup I avian adenovirus.[77] Related serogroup I avian adenoviruses tend to cause subclinical respiratory disease in other species of gallinaceous birds and can be isolated in cases of severe disease when other pathogens are also present.[84]

Bordetella avium infection is also called *turkey coryza* and is primarily a disease of turkeys, although the organism has been isolated from chickens with respiratory disease.[121] Turkey poults have copious oculonasal discharge, with dark staining around the nares, eyes, and sometimes over the wings. Submandibular edema also sometimes occurs. Serous to mucoid exudate is present in the nasal sinuses and trachea. The tracheal rings become soft, and the trachea becomes dorsoventrally compressed, resulting in altered vocalization.[9, 124]

Rare cases of mycotic tracheitis caused by *Aspergillus fumigatus* and *A. flavus* have been described in chickens.[17, 36, 122] In these cases, the diagnosis was made postmortem. Grossly, the tracheas had white-to-yellow nodular or plaque-like lesions and sometimes had caseous plugs of exudate that occluded the lumen. Fungus was cultured from the tracheal lesions. This type of lesion has been described in pet birds, in which the diagnosis was made by endoscopy and radiography in live birds.[85, 132]

Parasitic tracheitis can be caused by infestation by *Syngamus trachea* as well as by infection by *Cryptosporidia* sp. Syngamiasis can result in significant losses of young poultry and gamebirds. The gapeworm, *Syngamus trachea,* has been reported to affect chickens, turkeys, geese, guinea fowl, pheasants, peafowl, and quail.[114] The bright-red attached male and female worms form a Y-shaped configuration that can be observed within the glottis of a live bird by transillumination.[14] The parasites are also easily identified at necropsy. Nodules can be present in the wall of the trachea at points of attachment of the female worm.

The pathogenicity of cryptosporidia as a primary respiratory pathogen is

uncertain and it is often diagnosed in combination with other respiratory conditions, in which it has been shown to increase the severity of disease.[22, 56] Cryptosporidia can cause sinusitis in turkeys that clinically resembles MG infection.[54] Varying combinations of conjunctivitis, rhinitis, sinusitis, tracheitis, and pneumonia have been associated with cryptosporidial infection in chickens, pheasants, quail, peafowl, and partridges.[42, 81, 103, 123, 133, 138] Cryptosporidia also has been reported to cause severe enteric disease in quail.[59, 112]

Pneumonia and airsacculitis in gallinaceous birds is generally a postmortem diagnosis. Gross lesions in the lungs, ranging from congestion and edema to interstitial pneumonia, to necrotizing hemorrhagic pneumonia, have been attributed to various respiratory pathogens. Most pneumonias are the result of a bacterial infection or a primary viral infection that has been complicated with a secondary bacterial infection.

Pulmonary edema, congestion, and mild interstitial pneumonia can be observed in primary viral infections caused by AIV and infectious laryngotracheitis virus (ILTV).[49, 128] Severe pulmonary edema and congestion resulting in dyspnea and death are lesions of marble spleen disease of pheasants, which is caused by a group II avian adenovirus. Affected birds also have marked splenomegaly.[50] A disease caused by an adenovirus with clinical signs and lesions similar to marble spleen disease has been described in guinea fowl.[38]

The lungs from birds exposed to the fumes of overheated polytetrafluoroethylene (the synthetic polymer used in Teflon) are dark, red, wet, and heavy.[137] This condition has been reported most often in pet birds, but it has been suspected in cases of acute death in chickens brooded under Teflon-coated heat lamps.

Marked cyanosis and dyspnea are clinical signs of broiler chickens with the ascites syndrome, a metabolic condition associated with rapid growth, high-density rations, and cold stress. Affected birds have severe pulmonary congestion and edema. The respiratory lesions are secondary to congestive heart failure, and the hearts of these birds have right ventricular enlargement accompanied by hydropericardium and severe ascites. The livers are often shrunken and firm with an irregular surface.[63] Ascites syndrome is a common diagnosis in broiler chickens raised for 4-H and FFA shows.

Necrotizing pneumonia is a feature of velogenic ND and HPAI but not in the more common respiratory conditions caused by the less pathogenic strains of these two viruses. Both velogenic ND and HPAI are characterized by systemic vascular lesions, with widespread edema, hemorrhage, and necrosis, usually with severe lesions in the gastrointestinal tract, accompanied by high mortality rates.[1, 31, 40, 65, 131]

Fowl cholera, caused by *Pasteurella multocida*, causes diffuse consolidation of the lungs owing to flooding of the respiratory lobules by fibrin, hemorrhage, necrotic debris, and inflammatory exudate.[51] The pleural surface is often covered by a thick layer of fibrin. The lung lesion is especially prominent in turkeys. Chickens and gamebirds with pasteurellosis often develop an acute septicemia with swollen viscera and disseminated vascular lesions, but usually these birds have much less evident lung involvement than turkeys.[19, 55, 60, 109] Pneumonia caused by respiratory disease with acute secondary *E. coli* infections is similar in appearance to that caused by fowl cholera.

Chronic bacterial and fungal infections in birds elicit granuloma formation that progresses from an initial heterophilic response to a lesion consisting of macrophages, multinucleated giant cells, lymphocytes, and plasma cells.[88] Avian lungs respond to chronic inflammation as other tissues do, with formation of discrete granulomas.

In young birds, multiple pale nodules throughout the lung can represent granulomas caused by fungi, most commonly *Aspergillus fumigatus*. The infection can originate from fungal contamination of the hatching eggs. The disease often is spread during hatching, and flocks can exhibit high mortality rates in the first week of life. This condition is known as *brooder pneumonia*. The presenting signs of aspergillosis can be limited to keratoconjunctivitis with periorbital swelling and caseous exudate within the conjunctival sac, especially when birds are infected in the poultry house.[18, 89] In turkeys with milder forms of aspergillosis, small granulomas sometimes form within the pulmonary secondary bronchi and can be detected by palpating the lungs after they have been removed from the carcass at necropsy.

Rarely, pulmonary nodules are caused by *Salmonella pullorum*, but these usually are accompanied by similar lesions in the heart and other viscera, as well as lesions of purulent arthritis.[127] Pullorum disease is now a rare diagnosis owing to efforts to eradicate the disease from commercial poultry. It still occurs in backyard flocks with some frequency and potentially can be found in commercial birds.[47, 115] Chickens and turkeys are the most common hosts of *S. pullorum*, but natural infections have been reported in pheasants, quail, and guinea fowl, usually as a result of exposure to infected chickens.[127] Pullorum disease is a reportable disease. Chronic *E. coli* infections can also result in pulmonary granulomas, but such lesions are more likely found in older birds.

Pulmonary involvement is not a significant feature of avian tuberculosis but does occurs occasionally. More often, granulomas are found in the liver, spleen, and along the intestine. The granulomas (tubercles) are white to yellow and vary in size from 1 mm to several centimeters. Chickens with tuberculosis are usually emaciated.[93]

Nodular lesions in the lungs of chickens also can represent neoplasms. The most likely neoplasms are lymphoid tumors caused by Marek's disease virus or avian leukosis virus. Another type of virally induced neoplasm in chicken lungs is a myeloid tumor, caused by some strains of avian leukosis virus. In many instances, virally induced tumors are also found in other tissues of the affected bird. Primary respiratory tumors and metastatic tumors to the lung are extremely rare in birds.

Airsacculitis is a common feature of avian respiratory disease. Normal air sacs are thin and transparent. Depending on the nature of the insult and the chronicity of the condition, gross lesions in the air sac consist of cloudiness, thickening, and accumulation of exudate. Birds with a primary viral infection have scant amounts of frothy serous exudate in the air sac. Bacterial airsacculitis is characterized by thick deposits of fibrinous exudate. Chronic air sac infections caused by uncomplicated MG infections can have a beaded appearance owing to discrete lymphoid follicles in the stroma of the air sacs.[75] Fungal airsacculitis, usually caused by *Aspergillus* sp., causes plaque-like and nodular granulomatous lesions in the air sacs. These lesions tend to coalesce and often are green to black on the surface owing to the presence of the fungal spores.[111]

The causal agents enumerated earlier can cause signs of respiratory disease and gross lesions affecting parts of both the upper and lower respiratory system. Secondary *E. coli* infections often complicate viral and mycoplasmal respiratory infections in chickens and turkeys. The resultant disease is usually called *chronic respiratory disease* (CRD) by poultry producers. The gross lesions of such combined infections are much more severe and include fibrinous airsacculitis, pneumonia, pericarditis, peritonitis, and perihepatitis. In most cases, MG is the mycoplasmal agent associated with CRD. Some strains of MS have a tropism for

the respiratory system and are more prone to cause airsacculitis than synovitis, especially when there is a concurrent infection with either NDV or IBV.[69, 71]

As with CRD, combined viral and bacterial infections in other species can result in a confusing clinical picture of combined lesions of conjunctivitis, sinusitis, tracheitis, pneumonia, or airsacculitis. Infection with AIV complicated by a secondary bacterial infection results in such extensive lesions in pheasants and quail.[28, 43]

The gross lesions in turkeys with chlamydiosis are similar to those of CRD in chickens.[6] Turkeys are the species of commercially raised gallinaceous birds most commonly affected by *Chlamydia psittaci* infection, however this is only a sporadic problem. Chlamydiosis in commercial chickens is rare.[16] Chlamydiosis is more likely to occur in free-ranging poultry because of potential exposure from wild birds. Recognition of this disease is also important because of its zoonotic potential.

DIAGNOSIS

Few if any clinical signs or gross lesions of avian respiratory disease can be considered specific or pathognomonic. Because clinical signs are so similar for the various respiratory conditions of poultry, the practitioner must formulate a broad differential diagnosis and then employ a varied selection of diagnostic methods to rule in and rule out the potential conditions. Demonstration of a causal agent consistent with the clinical disease and observed lesions is the goal of diagnostic testing. Once established, the diagnosis determines the appropriate therapeutic and control measures.

In many instances, laboratory assistance is required to establish a specific etiologic diagnosis. Careful collection of information and specimens, preservation of the specimens, and timely delivery to a diagnostic facility are essential to establishing a rapid diagnosis.

The first step in veterinary disease diagnosis, including conditions of the avian respiratory tract, is to obtain a complete clinical history. Pertinent facts relating to signalment, flock management, nutrition, and environment, as well as farm history of disease, can be extremely valuable in establishing a specific diagnosis.[52] The age of the affected birds, species present within a flock, type of feeds and housing provided, feed consumption and production rates, vaccination history, source of birds, duration of illness, morbidity and mortality rates, flock treatments, clinical signs observed, and any necropsy findings are all important factors that contribute to establishing a differential diagnosis.

When individual birds are presented for diagnosis and treatment, nondestructive techniques used in pet bird medicine must be employed.[92] In a typical practice setting, the basic avian physical examination can be augmented by cytology, culture, and sometimes radiology.

Examination of wet smears or stained cytologic preparations of nasal or sinus exudate or conjunctival scrapings sometimes assists in identifying a specific etiologic agent. Intranuclear inclusions can be demonstrated in conjunctival scrapings from birds with ILT.[8] Vacuolated intracytoplasmic inclusions within degenerating epithelium of the conjunctiva can be demonstrated with Giemsa stain and are suggestive of avian poxvirus infection.[13] Cryptosporidia can be demonstrated with quick stains more easily than by examination of an unstained wet smear.[73, 102] Quik and Giminez stains can demonstrate intracytoplasmic chlamydial organisms within macrophages and epithelial cells in oculonasal exudate.[26]

Gram's stains of nasal and sinus exudates from birds with bacterial infections can suggest a particular etiology. Gram-negative, bipolar staining rods that are pleomorphic and sometimes form filaments are suggestive of *Hemophilus paragallinarum,* the cause of infectious coryza in chickens. More uniform, gram-negative, bipolar staining rods suggests *Pasteurella multocida,* which can cause sinusitis in chronic localized infections. The bipolar staining characteristic is well demonstrated by the Giemsa staining technique.[20] Sinus exudate should always be collected aseptically and submitted for culture of the causative organism to confirm the diagnosis and provide the opportunity for antibiotic sensitivity testing. Cytologic examination of tracheal exudates is of similar diagnostic value to examination of exudate from the nares and sinuses.

If syngamiasis is suspected, examination of a fecal flotation specimen for parasite eggs is appropriate. Characteristic eggs measure approximately 45 × 90 μm and have a thickened operculum at either pole. Eggs can be detected in fecal specimens from birds that have been infected for at least 2 weeks. The eggs must be differentiated from those of *Capillaria* sp.

Clinical evaluation of the lower respiratory system in live birds is possible and involves auscultation, cytology, hematology, endoscopy, and radiology, as applied in pet bird medicine. Auscultation is less valuable in avian patients than in mammals for evaluation of respiratory disease. Abnormal crackles or clicking noises can indicate airsac disease.[33] Hematology can be helpful in differentiating between viral and bacterial causes of respiratory disease, leukocytosis suggesting bacteria and leukopenia suggesting a viral etiology.[33] Endoscopic examination of the trachea, lungs, and airsacs can aid in diagnosis. Endoscopically guided collection of specimens for culture and cytology is also possible.[132] Radiographic evaluation can localize and demonstrate the distribution of lesions in lungs and airsacs.[95]

Laboratory tests used to differentiate respiratory diseases in poultry include serology, bacterial and fungal culture, virus isolation, and cytology. These services are generally available from either a full-service veterinary diagnostic laboratory or a specialized poultry diagnostic laboratory. An understanding of the specific disease conditions, their causes, and available diagnostic tests are required to ensure proper sample collection and test selection.

A variety of serologic tests are available for the common respiratory diseases of poultry. The availability of particular tests varies among different laboratories. Serologic tests are used to determine if exposure to an agent has occurred or to monitor response to vaccination, but serologic diagnosis is possible when acute and convalescent serologic titers are compared. When evaluating a flock of birds using serology, sera should be collected from a sampling of 20 birds.[134]

Blood should be collected from the cutaneous ulnar vein and placed in a sterile glass clot tube. A blood sample of 1 mL to 2 mL is adequate. For the best yield of serum, tubes should not be filled to more than one fourth of their capacity to maximize the surface area of the clot. The blood tubes should be stored horizontally for several hours at room temperature, or preferably incubated at 37°C. The tubes then should be refrigerated overnight and the serum poured into vials for shipment.

Exudates collected on culturettes from the conjunctival sac, nares, choanal cleft, and trachea, as well as aspirates from the sinuses, should be submitted for isolation of bacteria, viruses, and mycoplasmas. Systemic viral infections with AIV and NDV can result in shedding of the virus in feces, making collection of swabs from the cloaca a valuable diagnostic specimen. Cloacal swabs are also valuable for isolation of chlamydia. Taking multiple swabs of the same site should be done if possible so multiple tests can be performed. The choanal cleft

has been demonstrated as a superior site over the trachea for isolation of mycoplasma.[23]

Swabs and exudates can also be used for laboratory antigen detection systems, which are more rapid and sometimes more sensitive than isolation. Increasing use of antigen capture and polymerase chain reaction (PCR)–based tests allows rapid diagnosis of mycoplasmosis, avian influenza, chlamydiosis, and possible other diseases.[58, 74, 126]

In commercial poultry flocks or in small flocks in which mortality is the complaint, necropsy is the most efficient manner to collect diagnostic specimens. In such cases, examination of dead birds is more likely to result in a diagnosis than examination of live birds sacrificed for necropsy.

Postmortem examination of birds should be complete, systematic, and consistent. All organ systems should be examined thoroughly. Abnormalities should be noted as part of the medical history. The examination should be performed using aseptic technique so that collected specimens are not contaminated.

To maximize the diagnostic value of any necropsy, specimens of all organ systems should be collected. Fresh tissues should be saved for microbiologic examination, and fixed tissues should be preserved for histopathologic examination. Collection, handling, and proper shipment of the samples is critical to the success of any diagnostic effort.

Specimens for bacterial and viral isolation should be placed into sterile petri dishes or plastic bags. Tissue specimens for bacterial culture should be large enough to allow surface decontamination by searing. They should be measure 1 cm to 2 cm in all dimensions if possible. Intestinal specimens should be packaged separately. Fresh tissues submitted for bacteriology or virology should be chilled with cold packs but not frozen. Freezing is especially harmful if virus isolation is to be performed. If mycoplasma isolation is desired test, and the tissues cannot be processed within 24 hours, freezing of specimens is required.[68] Submission of a complete set of tissues is always best; however, those tissues absolutely necessary in cases of respiratory disease include sinus, trachea, lung, and airsac. Tissue from the cecal tonsils also should be collected for both bacterial culture and virus isolation. Whereas respiratory viruses are most easily isolated from the upper respiratory tissues during the acute phase of disease, the cecal tonsils provide a better sample for virus isolation in chronic disease.[34, 82]

Tissues submitted for histopathology should be no greater than 0.5-cm thick and fixed in ten times their volume of 10% neutral buffered formalin. Histopathology is a valuable diagnostic tool but should not be relied on as the sole technique for diagnosis. Because of the often stereotypical response to injury of which any particular tissue is capable, a specific etiologic diagnosis may not be possible based on histopathology alone.[64] Microscopic lesions are more often suggestive of a specific type of injury than a specific etiologic agent and provide a basis for selection of other specific laboratory tests such as virology, bacteriology, or toxicology. There are, however, certain instances in which histopathology can be diagnostic, especially when inclusion bodies indicative of ILT, fowl pox, or QBV can be demonstrated.

All specimens for laboratory submission should be properly labeled and accompanied by a complete clinical history. Shipment to the laboratory should be expedited by overnight delivery whenever fresh tissues are being submitted for microbiologic examination. Tissues should be prechilled before shipment. Specimens should be packed in nonbreakable, leakproof containers. Fresh tissue specimens should be shipped in insulated containers along with at least an equal volume of cold packs. Shipment should be planned so that delivery is on a weekday.[136]

As an alternative to performing a necropsy and collecting all specimens for diagnosis, whole carcasses and possibly live birds to be sacrificed can be sent to a diagnostic laboratory. Proximity to a laboratory, personal experience and preference, time available, cost, and other factors determine when such submissions are most appropriate.

TREATMENT

Once a presumptive diagnosis has been established and efforts to obtain a definitive diagnosis are under way, some form of treatment is usually appropriate in cases of respiratory disease. Appropriate medical treatment of gallinaceous birds varies in accordance with the intended purpose of the particular birds and the availability of approved drugs for the species involved. Available treatments for those gallinaceous birds produced for food or egg production are dictated by the Federal Food, Drug, and Cosmetic Act. The Animal Medicinal Drug Use Clarification Act allows extralabel drug prescription by veterinarians for food-producing animals, within specific guidelines. Of special importance when treating birds intended for use as food is observation of a sufficient withdrawal time to avoid drug residues.[113]

Those respiratory diseases for which medical treatment is most beneficial include mycoplasmosis, fowl cholera, infectious coryza, and colibacillosis. As with treatment of all bacterial diseases, antibiotic sensitivity testing following isolation of the causative agent is recommended prior to selection of any therapeutic agent.

The mycoplasmal infections of gallinaceous birds are generally responsive to treatment with tylosin, oxytetracycline, and chlortetracycline. In commercial poultry, feed medication with either of the tetracyclines at 200 g/ton for several days is a common practice. Water-soluble tetracyclines are also available. Subcutaneous injection of tylosin at 3 to 5 mg/lb body weight or administration in the drinking water at 2 g/gallon for 3 to 5 days is also recommended.[75] Antibiotic treatment, although palliative, does not eliminate infection with the avian mycoplasmas.

Most isolates of *Pasteurella multocida* from poultry are susceptible to the tetracyclines.[30] Injectable oxytetracycline has been successful in controlling experimentally induced fowl cholera in turkeys.[125] The most commonly used treatments for fowl cholera in commercial turkeys include chlortetracycline administered in feed or water and sulfadimethoxine in the water.[91]

Infectious coryza is treated with erythromycin, oxytetracycline, and various sulfonamides. The sulfonamides cannot be used in birds producing eggs for human consumption. Recurrence of disease when treatment is discontinued is common with infectious coryza and fowl cholera.

Colibacillosis in commercial poultry is often refractory to treatment due to antibiotic resistance. Antibiotic sensitivity testing is especially important in selecting a treatment for this disease. Recently, water-soluble sarafloxacin and enrofloxacin have been approved for treatment of colibacillosis in chickens and turkeys. Use of these drugs is by prescription only, and extralabel use is not allowed in food animals. The potential for development of resistance even to these drugs by *E. coli* has been demonstrated.[86]

Syngamiasis in pheasants can be treated with thiabendazole at 454 g/ton in the feed for 14 days. Levamisole administered in the drinking water at 1 g/gallon of drinking water has been used to treat syngamiasis in poultry and gamebirds. This treatment should be repeated in 7 to 14 days.[117]

Some of the diseases discussed have no effective treatments available. Bordetellosis in turkeys does not respond significantly to antibiotic treatment. Control of bordetellosis depends on sanitation methods following removal of the affected flock. If diagnosed, pullorum disease should not be treated because eradication of this disease is desired. There is no effective therapy for cryptosporidiosis. Depopulation followed by cleaning and disinfection with a 50% bleach solution has been recommended for control.[13] Likewise, no effective treatment is available for aspergillosis, and prevention is the preferred control method.

No practical drug therapy is available for the viral respiratory diseases of gallinaceous birds. Owing to the slow rate of spread of both fowl pox infection and ILT, vaccination in the face of an outbreak can reduce the severity of disease in a flock. In all respiratory diseases, the environment of the birds should be optimized with adequate ventilation and additional heat if needed.

Specific recommendations for treatment of diseases of quail and pheasants have been reviewed. Treatments for respiratory as well as other types of conditions were included.[97, 117, 119]

Prevention and Control

The responsibilities of veterinarians specializing in poultry medicine go beyond diagnosis and treatment of disease. Poultry veterinarians and veterinary practitioners serving owners of gamebirds and backyard poultry should endeavor to provide a comprehensive plan for prevention of disease and promotion of flock health.

Such a program must be multifaceted and designed to keep the flock productive and free of disease.[80] Environmental, nutritional, and management factors must be considered as being equally important as specific disease conditions, so that knowledge of the husbandry of the types of birds involved is essential.

Confinement is a basic requirement of any poultry flock, and the type of housing and confinement provided has an enormous effect on disease exposure. Confinement of the flock excludes potential carriers of disease, such as wild birds and rodents. Waterfowl have been implicated in outbreaks of AI and NDV in commercial poultry.[4, 66, 87] House finches have been shown to be potential sources of MG infection in chickens.[130] Rats from poultry farms were carriers of *P. multocida* and can be sources of fowl cholera.[41] Provision of adequate shelter and confinement protects the flock from extreme weather conditions and predators and also reduces exposure to parasitic conditions.

Proper nutrition of the poultry flock is important for growth, reproduction and good health. The type of ration supplied should be appropriate to the species and age of the birds in the flock because nutritional requirements differ among types of birds and change with their maturity. Multiple nutrients are involved with the ability of birds to resist disease and the development of their immune system.[67] The method and length of feed storage as well as feeding practices can influence flock health and should be considered under the broad category of nutrition with regard to disease control. Allowing feed to become wet can promote mold growth and potentially lead to aspergillosis or problems associated with mycotoxin production. Feeds stored at high temperatures or for excessively long periods lose significant amounts of vitamin activity.

Many potential health problems can be avoided by starting with quality replacement stock. The National Poultry Improvement Plan (NPIP) provides guidelines for poultry breeders to produce birds that are free of several hatchery-

disseminated diseases, including pullorum disease, MG, and MS. Because these conditions are vertically transmitted, obtaining stock from NPIP-approved sources eliminates introduction of these diseases to an operation. Birds can be purchased from participating breeders who are certified by the NPIP as free from infection with these agents. NPIP publishes an annual directory of participants handling chickens, turkeys, waterfowl, exhibition poultry, and gamebirds.

Biosecurity is best described as that component of the poultry health program that is concerned with prevention of introduction of potential pathogens to the poultry operation. A flock of birds can be directly exposed to respiratory pathogens as well as other types of pathogens by introduction of new birds to the flock, or indirectly by exposure to contaminated clothing, footwear, equipment, or feed. Isolation of the flock is the best method of preventing exposure to disease from an outside source. Visitors should always be discouraged from entering a poultry or gamebird facility. If visitation is necessary, clean protective clothing, including coveralls, boots, and hats, should be provided.

A flock management system practiced in commercial poultry operations that has a significant role in disease control is the application of all-in/all-out flock placement. This limits the flock to a single age and prevents transmission of disease organisms from older, subclinically infected birds to younger, more susceptible birds. Where this type of management cannot be practiced, the different age groups should be isolated physically. There should be different caretakers for different age groups of birds, or the traffic patterns should be such that people move from the younger birds to the older birds. Where new stock must be added to existing flocks, some provision for quarantine for at least 1 month must be made, but even this may not prevent introduction of disease. Chickens that have recovered from ILT have been shown to carry the virus for up to 16 months, and latent infections can be reactivated by stress.[11, 12]

The importance of single-age poultry units is illustrated by one of the recommended control methods for quail bronchitis. In farms with quail bronchitis, hatching operations should be interrupted, and new susceptible birds should not be introduced for a period of at least 2 weeks beyond clinical evidence of the disease in the flock. Because quail bronchitis affects birds younger than 4 weeks of age most severely, such a break-in introduction of new birds should reduce the length of the outbreak.[107]

Just as the separation of different age groups of birds is important, different species of birds should be reared separately. The clinical manifestations and susceptibility to different diseases can vary greatly with the species of bird involved. Whereas chickens often show few signs of infection with MG, turkeys and game birds tend to have a much more serious disease as a result of mycoplasma infection. Both domestic and wild ducks can have subclinical infections with AIV, but much more serious disease can result in gallinaceous birds.

Sanitation is a basic component of any biosecurity program. Important hygienic measures include insect and rodent control, cleaning and disinfection, and proper disposal of dead birds. Careful handling of hatching eggs and proper sanitation of incubation and hatching equipment is essential to reducing the incidence of brooder pneumonia and other hatchery-disseminated diseases.

Of those respiratory diseases discussed above, fowl pox, ND, and avian influenza implicate insects as agents of spread. Mosquitoes act as a vector for pox virus and arboviruses, such as eastern equine encephalitis virus.[13] Flying insects have been suspected as possible sources of infection in outbreaks of ND and HPAI.[4] Ticks have been reported to spread fowl cholera. Chemical control of insect pests should be conducted with attention to avoidance of potential residues and safety considerations for the poultry flock and personnel.

Rodent control is important in regard to controlling fowl cholera as well as other types of infection, especially salmonellosis in poultry flocks. Careful use of rodenticides must be practiced to eliminate risks of accidental exposure to the flock and other nontarget species. Proper design of facilities helps to exclude rodents and wild birds from flocks.

Cleaning and disinfection of poultry facilities should be routine practice between flocks of birds and is practiced most effectively in conjunction with all-in/all-out flock placement. Complete physical cleaning to remove organic material prior to application of disinfectants is imperative. An extended period between removal of the old flock, decontamination of the environment, and placement of new birds helps to reduce early exposure of the new flock to pathogenic organisms. Complete cleanout and disinfection is especially important following any significant disease outbreak.

All poultry operations occasionally have birds that die even in the absence of a major flock disease problem. Prompt and proper disposal of diseased carcasses is necessary to prevent spread of potential disease organisms. Carcasses should be disposed of by incineration, burying, or composting (where allowed by local environmental regulations). Removal of carcasses from other birds prevents cannibalism or contamination of the environment. Similarly, removal and humane euthanasia of moribund birds should also be an element of flock sanitation. In those instances in which significant mortality in a flock indicates a current disease problem, carcasses should be collected for diagnostic examination.

Management practices to reduce the stress level of birds are an important element of disease prevention strategies. Optimal environmental conditions are particularly important in preventing respiratory diseases. Suboptimal ventilation in poultry housing can result in high ammonia levels or excessive dust. Temperature control, watering system management, litter moisture, and ventilation must be in balance to provide the best environment for poultry.

The incidence of respiratory disease in commercial poultry is higher in winter and is associated with decreased ventilation rates. Wintertime ventilation is often reduced as a means to maintain temperature but results in decreased air quality. Production of ammonia increases with litter moisture and fecal content.[27] Adequate ventilation removes moisture as well as ammonia produced by the microbial breakdown of poultry wastes. Wet litter also can result from leaking or improperly managed drinking systems. Other disease conditions such as coccidiosis can be exacerbated by wet litter.

High dust levels in poultry houses result in higher levels of airborne pathogens. Viral, bacterial, and fungal agents carried on inhaled dust particles can serve as a means of spread of respiratory disease.[116]

The quality and type of litter materials is a factor in the incidence of aspergillosis. Moldy litter sources can result in high mortality rates because of aspergillosis.[45] Pine shavings are the preferred type of litter, as opposed to hardwood shavings, sawdust, hay, or other materials that may have a higher level of mold spore contamination.[100] Contaminated feed also is a potential source of aspergillus to poultry operations.

Environmental stresses affecting poultry include extreme conditions of temperature, light, and availability of feed and water, as well as social and behavioral stresses resulting from high population density. Disease organisms and toxins can also serve as potential stressors. Both acute and chronic stress has been shown to reduce growth in young birds and reproductive capacity of adults. Humoral and cell-mediated immunity are suppressed by various forms

of stress.[120] Management practices that provide an environment that minimizes stress to the birds should be part of any flock health program.

A vaccination program can be a useful adjunct to a flock health plan. Such a program must be customized for each flock with consideration of the species of birds, age groups, and disease risks present. Commercial poultry flocks have somewhat standardized vaccine programs, but even in these flocks there is considerable variation owing to the types of vaccines available, methods of application, and the philosophy of the managers.

Vaccination in a well-managed, isolated flock maintained with a high level of biosecurity is sometimes unnecessary. In flocks of birds that potentially could be exposed to other birds (e.g., exhibition poultry), application of some vaccines is needed.

Live attenuated and inactivated vaccines and bacterins are available for most of the respiratory disease agents affecting chickens and turkeys. Some of these products, such as those for ILT and MG, are closely controlled by state regulatory agencies. The use of attenuated live vaccines entails some degree of risk to members of the flock to be vaccinated, especially flocks with mixed ages. Also, nearby flocks can be inadvertently exposed to vaccines, so that such products should be used carefully and then only when necessary in backyard flocks. Routine vaccination with inactivated vaccines against some agents in adult breeding stock is safe and can result in improved productivity and decreased disease incidence over time. Any vaccination program for gallinaceous birds should be designed with the help of a specialized poultry veterinarian or poultry extension specialist, to satisfy the specific needs of the flock and locale.[72]

When one is presented with gallinaceous birds having signs of respiratory disease, the practitioner is obligated to establish a definitive diagnosis. Owing to the similarity of a variety of such conditions in different species, accurate diagnosis can require laboratory assistance. After providing appropriate therapy, the practitioner should assist his or her client in developing a flock management plan to reduce the incidence of future respiratory disease problems.

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