CHAPTER

33

BACTERIA

Helga Gerlach

Scientific principles of bacteriology are universal; however, in application, bacteria can adapt to their avian hosts, altering the form and pathogenicity of well known bacterial species. Avian bacteriology is further complicated by the fact that bacteria that are as yet taxonomically undescribed can be isolated from a variety of avian species. Some of these bacterial strains have been erroneously classified as taxons (eg, *Pasteurella haemolytica*, *Alcaligenes faecalis*). In general, bacterial adaption to an avian host minimizes cross-species transmission from birds to mammals. Non-host-adapted transmission usually requires large numbers of organisms, repeated exposures, specific susceptibility or immunosuppression. Some host-adapted strains may form biovars that are specific to one avian species (or a few closely related species) as compared to the dominant species.

Example of Bacterial Taxonomy:

Genus	*Salmonella*
Species	*S. typhimurium*
Subspecies	*S. typhimurium* var *copenhagen*
Biovar	Pigeon strains, chicken strains

Bacterial infections may be primary or secondary. This differentiation is important for the evaluation of a disease process. After becoming established, many secondary invaders are able to maintain a disease process independent of other infectious agents or predisposing conditions. Laboratory examinations (biochemical or serologic) are rarely of any help in differentiating primary from secondary invaders. The companion bird clinician must determine the importance of bacterial isolation for a specific bird species and a specific disease process. Analogous conclusions drawn from poultry literature may not be valid, and experimental infections are frequently not possible. Some specific points may guide the clinician in interpreting bacterial culture results (Table 33.1).

TABLE 33.1 Guides to Interpretation of Bacterial Culture Results[27]

- Isolation of an organism in an almost pure culture (approximately 80% of the colonies present) may indicate that the bacteria is a component in the disease process.
- Isolating large numbers of bacteria in almost pure culture from the heart tissue is suggestive of bacteremia, and the isolated agent should be considered part of the disease process.
- Isolating a bacteria that is part of the autochthonous flora may indicate that it is functioning as an opportunistic (secondary) pathogen.
- If the isolated organism has pathogenicity markers, it is probably involved in the disease process. It is not possible to determine if the bacteria is a primary or secondary pathogen.
- Isolating bacteria from parenchyma with pathologic or histopathologic lesions suggests that the agent contributed to the disease process.
- Isolating a bacterium without identifying other microorganisms (virus, chlamydia, other bacteria, fungi, protozoa) suggests that the agent is a primary pathogen. Finding virus or chlamydia suggests that the bacterium may be a secondary pathogen. Isolation of bacteria from a bird with fungi and protozoa suggests that the bacterium is a primary pathogen.
- Obtaining mixed cultures or identifying individuals within a given flock with different bacterial isolates suggests secondary infections are occurring.
- Isolating small to moderate numbers of bacteria from the liver or kidney can be "normal," because birds have hepatic and renal portal circulations and lack lymph nodes that filter blood before it drains into the liver and kidney. Because lymph follicles are distributed throughout these organs, defense responses actually occur within the parenchyma and not externally, as in mammals. These organs should not be expected to be sterile, but should be expected to contain autochthonous flora. The number of organisms isolated at necropsy depends on the time of death and the method of handling the body (eg, storage, preservation).

Because the isolation of "unusual" bacteria can be expected from avian samples, the clinician can enhance results by providing the laboratory with a thorough anamnesis, exact species of the bird in question, and as far as possible, the names of particular bacteria that may be suspected in the case. This latter point is especially important if special media or environmental conditions are needed for bacterial isolation. In addition, with some organisms, special transport media and shipping methods (eg, on ice) may be necessary to preserve the organism.

Blood cultures are considered a definitive diagnostic tool in humans and some mammals. In these species, samples are, as a rule, taken during a period of fever. A febrile period is difficult to determine in a bird and is generally considered of minimal importance. In birds, organs as well as blood are not necessarily sterile, although the number of bacteria is extremely low. Generally, the isolation of small quantities of autochthonous flora is considered normal, and only the isolation of primary pathogens is really helpful. Because whole blood has bacteriostatic and bactericidal properties, blood culture samples must be transferred immediately after collection to attenuant containing nutrients and one of the artificial heparinoids, for instance Na-polyanetholsulfate. Commercially available blood culture flasks are also useful.

Gram-negative Bacteria of Clinical Significance

"Intestinal bacteria" are considered to be those species that can colonize the intestinal tract. As a group, intestinal bacteria can be part of the normal flora or pathogenic organisms that are not routinely found in the GI tract; in some cases, normal flora can become secondary pathogens. When a bacterium leaves the mucosal surface and penetrates the intestinal wall, it then can induce systemic disease, including septicemia and death. The Enterobacteriaceae are considered the most important avian intestinal pathogens, but other groups, such as *Aeromonas*, *Pseudomonas*, *Alcaligenes*, *Bordetella* spp. and related organisms, as well as *Vibrio* and *Campylobacter*, may also colonize the gastrointestinal tract.

Enterobacteriaceae

The members of the Enterobacteriaceae family typically grow well on commonly used media. Enterobacteriaceae are divided into genera based on specific biochemical and serologic characteristics. Many species are further divided into biotypes and serotypes. In these species, complete identification requires differentiation between the O, K and (in motile species) H antigens. Serologic differentiation between the genera is often difficult since group-specific antigens (lipopolysaccharides) can cross-react.

Enterobacteriaceae are able to propagate in the environment if they are in the proper conditions. Enterobacteriaceae are ubiquitous and considered to be part of the autochthonous intestinal flora in many mammals, including humans and some species of birds (Table 33.2).

TABLE 33.2 Birds in which Enterobacteriaceae are not normal

- Psittaciformes (parrots and parakeets)
- Fringillidae (finches)
- Ploceidae (weaver finches)
- Astrildae (waxbills)
- Accipitriformes (hawks, vultures)
- Falconiformes (falcons)
- Strigiformes (owls)
- Gruiformes (cranes)
- Otididae (bustards)
- Sphenisciformes (penguins)
- Ciconiiformes (storks, ibises)
- Tetraoninae (grouse)
- Musophagiformes (turacos)
- Trochiliformes (hummingbirds)

Isolation of Enterobacteriaceae from the respiratory or reproductive tracts is abnormal. This group of bacteria can colonize most avian tissues, where it is frequently considered as a secondary pathogen. In some cases, Enterobacteriaceae can function as primary pathogens. Substantial differences exist in the virulence of the various Enterobacteriaceae and in the host response to infections. The genera *Shigella* and *Edwardsiella* are normally not cultured from birds (the latter rarely from pigeons). The genera *Enterobacter*, *Hafnia*, *Serratia* and *Proteus* are of a low pathogenicity. The isolation of *Enterobacter agglomerans* (a plant pathogen) in avian feces can indicate the consumption of seeds that contain more than 10^6 bacteria/g of food. This bacterium is common in decaying plant matter; foods containing the bacterium in high numbers should be considered toxic. *Serratia marcescens* is increasingly found in large parrots with chronic debilitating diseases. Predisposing factors seem to include previous antibiotic treatment and immunosuppression.

Escherichia (E.)

E. coli is the most commonly encountered member of this genus; in many avian species it is considered to be a more important pathogen than salmonella (see Table 33.6). This genus contains a number of species that may be motile or nonmotile, encapsulated or nonencapsulated. Classification of the strains of *E. coli* that infect birds has been difficult. The serologic and virulence factors used to classify the *E. coli* strains that infect humans and other mammals do not accurately predict which *E. coli* strains will be pathogenic in birds. Although each serovar of *E. coli* includes both virulent and avirulent strains, there seems to be a slightly higher frequency of virulent strains within the serovars 01, 02 and 078. In experimental transmission studies, all lysine decarboxylase-negative *E. coli* strains have been found to be virulent in birds. Unfortunately, many of the lysine decarboxylase-positive strains are also virulent.

Pathogenesis

The pathogenesis of *E. coli* infections in birds is poorly defined. Mammalian strains produce large quantities of exotoxins that cause many of the clinical and pathologic changes associated with infection. Except for the presence of enterotoxins, avian *E. coli* strains appear to produce few exotoxins. These enterotoxins cause diarrhea by inducing hypersecretion of fluids into the intestinal lumen. Endotoxins may cause hypersensitivity angiitis followed by septicemia and death.

Clinical Disease and Pathology

The clinical signs associated with primary or secondary infections are thought to be governed by the portal of entry to the avian host.

Colisepticemia is characterized by an acute onset of lethargy, anorexia, ruffled plumage, diarrhea and polyuria. *E. coli* septicemia usually involves the kidneys, although clinical signs of renal involvement may or may not be present. CNS involvement is rare. Ocular lesions occasionally occur and include exudation of fibrin into the anterior eye chamber or uveitis. Serofibrinous arthritis can occur as a sequela in some infected birds. Fibrinous polyserositis, the severity of which depends on the chronicity of the infection, may be noted at necropsy. Catarrhal enteritis is common but nonspecific. The most consistent histologic lesion is serofibrinous inflammation with plasma cell infiltration in the liver and kidneys (Figure 33.1).

Localized enteritis caused by *E. coli* is a result of enterotoxin production, which induces an increased secretion of fluids. The resulting diarrhea causes a substantial loss of electrolytes and proteins and induces dehydration and cachexia. Some strains of *E. coli* are capable of colonizing and destroying the intestinal epithelium. These strains typically induce a pseudomembranous or ulcerative enteritis. Clinically infected birds die peracutely or develop nonspecific signs associated with enteritis. Infections with these strains are usually diagnosed on postmortem examination.

Coligranulomatosis (Hjaerre's disease) is particularly common in Phasianiformes including chickens, turkeys, peafowl, partridges and capercaillie. Mucoid (eg, encapsulated) *E. coli* strains, mainly of serovars 08, 09 and 016, are the usual etiologic agents. Coligranulomas are thought to occur when other agents damage the intestinal mucosa and allow a secondary infection with specific *E. coli* serovars. It is currently

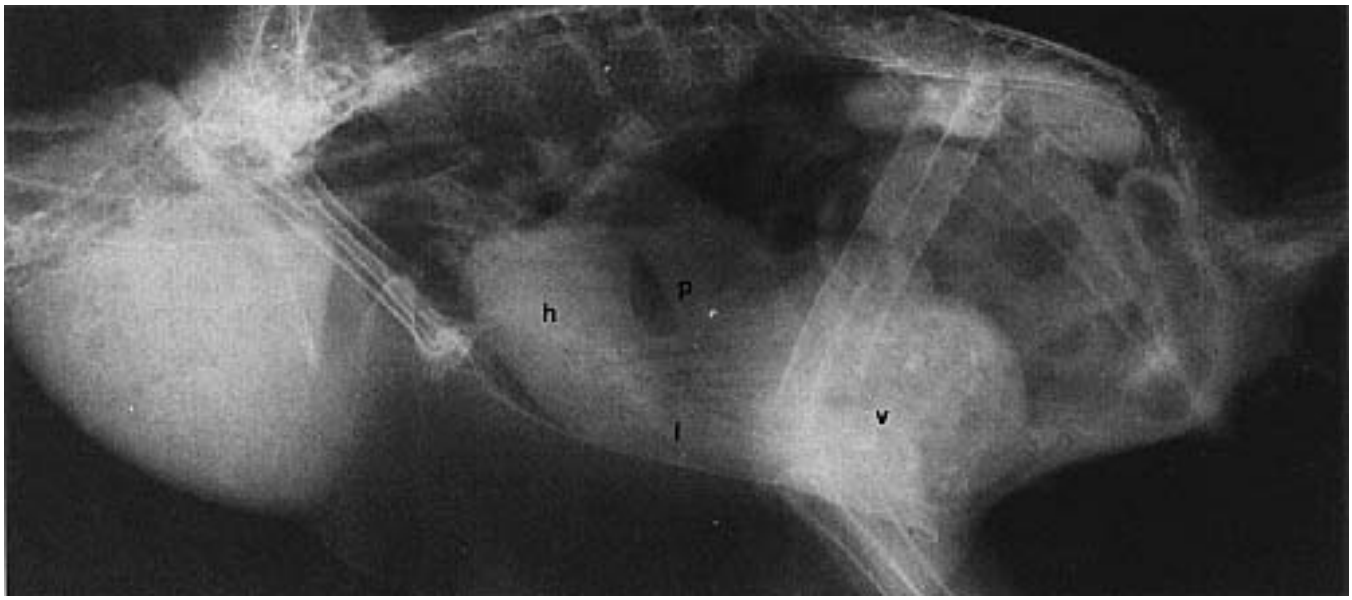


FIG 33.1 A seven-year-old male Amazon parrot was presented for weakness and diarrhea. Abnormal clinical pathology findings included PCV=22, WBC=33,000, SGOT=476. A Gram's stain of the excrement revealed 70% gram-negative rods. Radiographs indicated ileus (gaseous distension of the bowel), a full crop and microhepatia. Dried excrement is visible pericloacally. The dilated bowel loops are displacing the proventriculus (p) cranially and the ventriculus (v), liver (l) and heart (h) ventrally.

thought that galactans found in the *E. coli* capsule stimulate the granulomatous reaction.

Affected birds develop diarrhea, polyuria and chronic weight loss. Granulomatous dermatitis is occasionally noted. Grayish foci of varying sizes in the liver, intestinal subserosa and spleen or kidney are typical findings at necropsy. These granulomas are distinct from those induced by avian tuberculosis because they lack an opening into the intestinal lumen. The center of the foci may be mineralized. Histologic changes are characterized by multinucleated giant cells and a few heterophils around a central necrotic region. Acid-fast staining should be used to rule out mycobacteriosis.

E. coli can cause a primary rhinitis but is generally a sequela to infections elsewhere in the body. Air sac lesions can be severe and may extend to the peritoneum, causing a fibrinous polyserositis. Except for in geese, *E. coli* pneumonia is rare. This is due to the special anatomy of the avian lung. When pneumonia occurs, it is most common in young chicks that have inhaled a high number of virulent *E. coli* with contaminated dust. Affected birds are usually dyspneic and cyanotic. In contrast to mammals, pneumonia is not associated with prominent respiratory sounds.

Hens can develop *E. coli* infections characterized by fibrinous salpingitis or oophoritis originating from organisms that ascend from the cloaca or by imprint metastases from infected air sacs. Infections are usually chronic in nature, with untreated birds eventually dying from salpingoperitonitis. Genital tract infections in males are less common, but when they do occur they usually result in orchitis and permanent sterility.

Secondary colonization of joints and bone marrow can occur following *E. coli* septicemia. These lesions are rare, but when the do occur they are most frequent in nestlings and fledglings. Some of the finch species seem to be particularly susceptible. Bone marrow infections appear to be very painful, and affected birds are usually reluctant to move.

Diagnosis

Polyserositis and granuloma formation are lesions suggestive of *E. coli* infections. Specific diagnosis requires culturing the organism from infected tissues. Serotyping is of academic importance.

Treatment

The ability of a selected drug to penetrate target tissues or granulomas must be considered (see Table 33.7). Oral antibiotics may be effective in treating *E. coli* infections limited to the intestinal mucosa, but parenteral antibiotics are necessary for treating most *E. coli* infections. In addition to antibiotics, therapeutic considerations should also include administration of avian lactobacilli in an effort to lower the intestinal

tract pH and help establish a proper autochthonous flora. Mammalian strains of lactobacillus can be effective in changing the intestinal pH but require the administration of large quantities of product over a three- to four-week period. Lactulose may also be helpful in lowering the intestinal pH. Providing a nutritional diet is important in improving gastrointestinal physiology in malnourished birds.

Salmonella (S.)

The genus *Salmonella* includes approximately 2000 species divided into five subgenera. Subgenus I is the most important in birds. Subgenus III (*S. arizonae*, *Arizona hinshawii*) has occasionally been reported in birds, particularly those that are in contact with reptiles. Most strains are motile and grow on common media. The subgenera are determined by specific biochemical profiles, and species are differentiated serologically (O, K [Vi] and H antigens). Lysotyping is used for further characterization at the research level. Propagation can occur outside the host if the correct ambient temperatures and proper nutrients are available.

Most vertebrates can be infected with some *Salmonella* spp. However, the host susceptibility and development of carrier states vary widely among species. Free-ranging birds can be subclinical carriers and serve as a reservoir for the aviary. In addition to free-ranging birds, rats, flies and other vermin may also serve as vectors of salmonella. Avian species without ceca or with involuted ceca appear to be more susceptible to salmonella infections than birds with fully functioning ceca. Bacteroides and *Spherophorus* spp. are considered autochthonous cecal flora, and these gram-negative anaerobes may function as natural antagonists for *Salmonella* spp. The incidence of various *S.* spp. seems to vary with geographic location and the types of food consumed, particularly the proteinaceous component. Imported birds (and other animals) may serve as reservoirs for nonindigenous *S.* spp. that can cause devastating outbreaks. Some salmonella strains are host-adapted (eg, *S. gallinarum-pullorum* to chickens, *S. typhimurium var. copenhagen* [in two biovars] to either pigeons [malonate-negative] or European finches [malonate-positive]).

Transmission

Salmonella enters the host principally through the oral route. Contaminated dust from feces or feathers may be involved in aerogenic spread in some cases. Egg transmission can occur with fully walled and L-form salmonella. It is most common with adapted strains but is possible with any *S.* sp. Experimental studies indicate that patent infections in embryos occur with fewer than ten bacteria. These infected

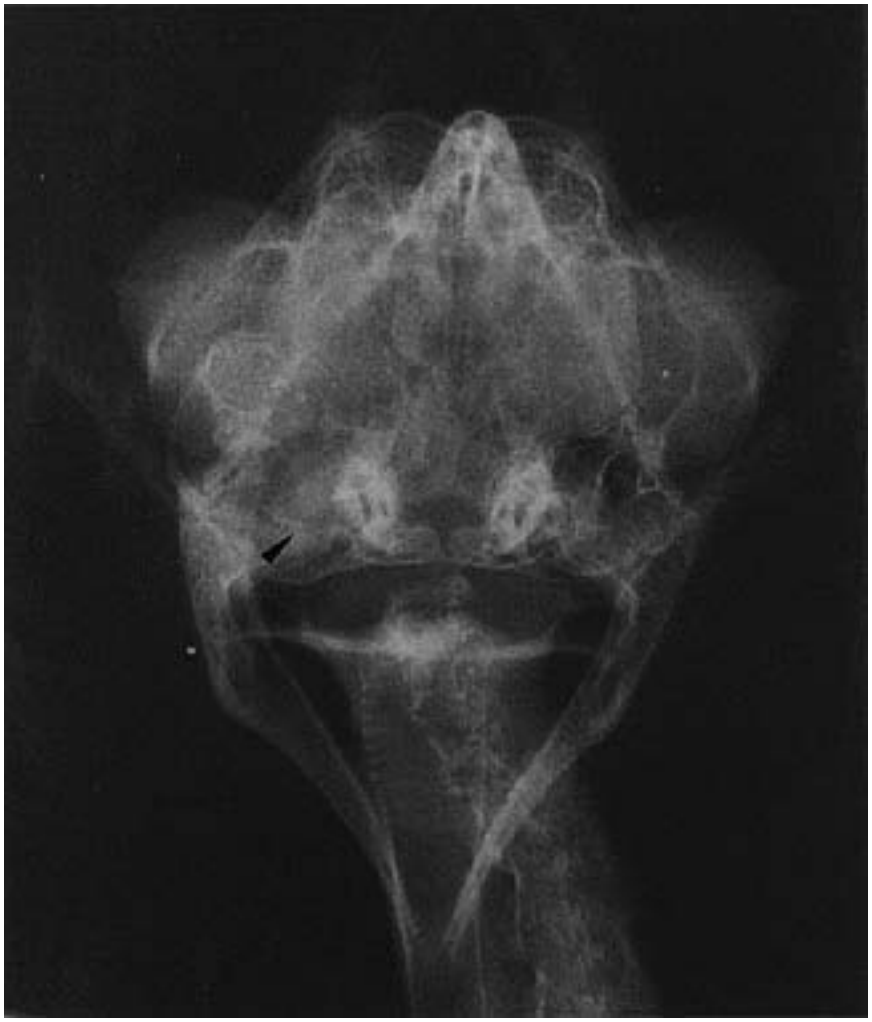


FIG 33.2 An adult Barn Owl from a zoological collection had a three-year history of intermittent depression and anorexia. The bird was presented with severe right-sided head tilt and vertical-to-rotatory nystagmus. The external ear canal was hyperemic, and the tympanic membrane was opaque and edematous. Radiographs indicated a soft tissue density in the right tympanic bullae suggesting an internal ear infection. Auditory evoke potentials indicated lesions in the peripheral and central auditory pathways. *Klebsiella* sp. and *Pseudomonas* sp. were recovered from the infratrochlear area and brainstem at necropsy.

chicks then hatch and spread salmonella by direct contact throughout a nursery. If an embryo has more than ten bacteria, it usually dies before hatching.[47] In freshly hatched chicks, salmonella often serves as the first bacterium to colonize the intestines. This organism becomes host-adapted during the egg incubation period, and the hatched chick can then serve as a subclinical carrier. In some cases, the salmonella infection can eventually induce a septicemia and death. Subclinical carriers allow an infective cycle to occur in the absence of other vectors. Vertical infections may also occur if infected hens feed their young contaminated crop contents.

Pathogenesis

One of the characteristics of the group Enterobacteriaceae is that they all produce endotoxins. Salmonella is no exception, and some cases of food poisoning are linked to this bacterium. Indirect death through endotoxin contamination of food is rare in birds; most avian salmonella problems are associated with direct infections. Interestingly, both virulent and nonvirulent strains of a given *Salmonella* sp. can exist simultaneously in a host. Virulent strains are those that can penetrate an intact intestinal mucosa, and nonvirulent strains are those that require a mucosal lesion to enter a host. Nonvirulent strains often colonize the gut, resulting in asymptomatic infections and intermittent shedding. Once virulent or nonvirulent strains have passed the mucosal barriers, they induce a septicemia that results in an immune response or colonization in tissues and eventual death of the bird. In some cases, *Salmonella* sp. may cause chronic infections that are characterized by intermittent septicemia and clinical signs. Recurrent infections usually result in progressive organ involvement; the CNS and joints are frequently end-stage sites of infection.

TABLE 33.3 Percentage of Psittaciformes Shedding Gram-negative Bacteria and Yeast

	E. coli	Enterobacter	Klebsiella	Pseudomonas	Yeast
Bare-eyed Cockatoo	44	0	0	13	6
Citron-crested Cockatoo	30	0	0	0	0
Major Mitchell's Cockatoo	27	20	0	0	13
Moluccan Cockatoo	81	13	0	0	13
Sulphur-crested Cockatoo	44	0	0	0	11
Triton Cockatoo	84	8	3	0	0
Red-vented Cockatoo	78	0	0	0	0
Umbrella Cockatoo	56	24	4	0	15
Rose-breasted Cockatoo	16	2	0	0	0
African Grey Parrot	17	0	0	0	0
Eclectus Parrot	10	0	0	0	10
Blue-crowned Amazon Parrot	0	0	0	0	0
Blue-fronted Amazon Parrot	20	0	0	0	0
Yellow-headed Amazon Parrot	12	0	0	0	0
Yellow-naped Amazon Parrot	38	0	0	0	6
Blue and Gold Macaw	23	0	0	8	8
Buffon's Macaw	23	0	0	8	8
Green-winged Macaw	26	3	0	3	3
Military Macaw	20	4	0	0	16
Red-fronted Macaw	7	7	0	0	0
Scarlet Macaw	19	2	0	0	0
Hyacinth Macaw	6	0	0	0	11

Incidence (in percent) of the isolation of gram-negative bacteria and yeast from the cloaca of a group of psittacine birds with no observed clinical abnormalities. Gram-positive bacteria were isolated from 91% of the 506 cloacal samples. It is not unusual to find transient populations of gram-negative bacteria in the cloaca of asymptomatic birds. However, the presence of these bacteria in the gastrointestinal tract can cause problems if a bird is stressed. The isolation of gram-negative bacteria from clinically asymptomatic psittacine birds warrants a close examination of management practices. Adapted from Flammer K: Avian Dis 32: 79-83, 1988.

Incubation

Incubation periods vary with the type of salmonella infection. Presumably, these differences are dependent on the strain of infecting salmonella, the route of infection and the condition of the host. In acute diseases, incubation periods are typically three to five days. With egg transmission the incubation period is shorter, generally considered to be two days. Subclinical carriers can have prolonged incubation periods.

Clinical Disease and Pathology

Acute diseases are characterized by nonspecific signs including lethargy, anorexia, polydipsia (sometimes followed by polyuria) and diarrhea. In subacute to chronic cases, CNS signs, arthritis (particularly in pigeons), dyspnea and indications of liver, spleen, kidney or heart damage are common. With high-dose infections, conjunctivitis, iridocyclitis and panophthalmia may occur.

Some individual avian species have unique clinical presentations. Outbreaks in lories (Loriidae) and penguins (particularly Jackass Penguins) are associated with peracute diseases and high flock mortality. African Grey Parrots are also very susceptible, but typically develop a more chronic disease exhibiting phlegmon, granulomatous dermatitis, arthritis and tenovaginitis (Figure 33.3). Respiratory signs with myocardial lesions are common in tangares, quetzals, Red-headed Barbets, terns and House Sparrows. Nonspecific CNS signs are common in

geese and ducks. Some infected ducks will swim with an inverted keel (keel disease) just prior to death. Subacute salmonellosis in many finches (Fringillidae) is characterized by granulomatous ingluvitis that may be confused with candida infections. Granulomatous dermatitis has been reported in several species and is thought to be induced by mosquitoes or other biting insects.

Subgenus III strains are considered less virulent than those of subgenus I; however, the clinical lesions induced by this subgenus are indistinguishable. Ocular lesions appear to be more frequent with subgenus III, and turkeys, ducks, parrots and canaries are particularly susceptible.

FIG 33.3 A three-month-old pigeon was presented with depression, anorexia, diarrhea and reluctance to move. An open ulcer was present on the bird's hock. *Salmonella* sp. was isolated from the bird's feces and the bird responded to treatment with antibiotics (courtesy of Louise Bauck).

Postmortem lesions include dehydration, degeneration or necrosis of skeletal musculature, gastroenteritis (occasionally with ulcers and granulomas), enlargement of the liver and spleen (with or without disseminated small whitish foci), bile congestion and nephropathy. Chronic infections usually cause pericarditis or epicarditis fibrinosa, granuloma formation in the liver, spleen and kidney, and degeneration or inflammation of the ovary or testis. Fibrin filling the cecal lumen is a common finding.

Histopathologic changes are nonspecific with purulent inflammation in the parenchymal organs. Granulomas are common with chronic infections. Purulent leptomeningitis and exudate formation in the subarachnoidal spaces are usually noted in birds with CNS signs.

Diagnosis

A confirmed diagnosis requires isolation and identification of the *Salmonella* species. Serologic evaluation of a flock can be used only if the precise species is known; however, chronically infected subclinical birds are frequently serologically negative. The same is true for birds infected with L-forms. The development of a serologic response requires penetration of the intestinal mucosa, and most subclinical carriers have infections limited to the intestinal lumen.

Treatment

Whether or not to treat salmonella infections in companion birds is controversial. The author believes that clinically affected birds and companion birds that are identified as carriers should be treated because of public health hazards. Therapy should include appropriate antibiotics (based on sensitivity) and lactobacillus products. In general, the frequently encountered salmonella strains are sensitive to commonly available antibiotics, but some strains from free-ranging birds (particularly from seagulls) demonstrate varying degrees of antimicrobial resistance. CNS signs and chronic infections tend to be refractory to therapy. Flock management of salmonella should concentrate on preventing egg transmission by identifying and removing subclinically infected breeders. Treating birds that have egg-derived infections is extremely difficult. Host-adapted strains of salmonella seem to cycle in periods of approximately three-month intervals. Cycles of egg transmission can best be broken by collecting eggs for future breeding stock four weeks after treating the parent stock. Newly hatched chicks from these birds should be cultured (fecal swabs) at hatching, and infected birds should be treated immediately.

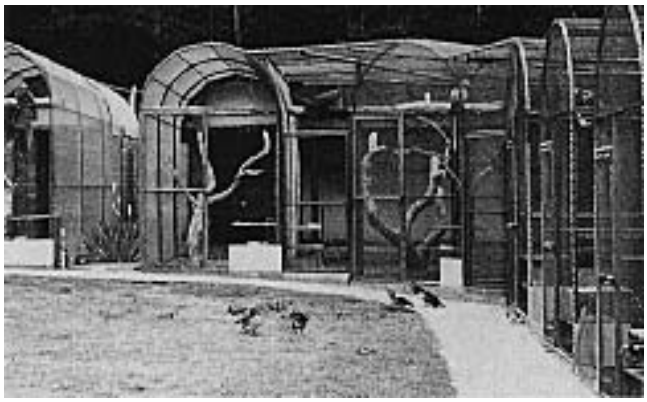


FIG 33.4 Aviary hygiene is important in preventing a bird's exposure to many infectious agents including bacteria. Walk-in type enclosures have several disadvantages when compared to drop-through type enclosures. The birds in walk-in enclosures can fly to the floor where pathogens can accumulate in excrement and food waste. Birds are more likely to come in contact with discharge from flies or rodents that have ready access to organic waste on the floor of the enclosure. Additionally, caretakers can act as mechanical vectors for the transmission of pathogens as they walk from one enclosure into the next.

Treatment of L-forms can be attempted with clindamycin (100 mg/kg body weight) or a combination of erythromycin and ampicillin (both components at the full dose).

Control

Proper hygiene is the best tool for preventing salmonella outbreaks. The effective control of flies, rodents and other vermin is essential (Figure 33.4). Regular cleaning and disinfection of the aviary and nursery, along with proper storage of food, are all important in preventing salmonellosis. Several *Salmonella* spp. vaccines have been evaluated experimentally, but none have proven to be effective.

Companion bird strains of *Salmonella* are not considered important human pathogens in healthy individuals, but can cause problems in infants, geriatric patients or those with immunosuppressive diseases. Humans carrying salmonellosis can infect their companion birds. Such human-to-animal interactions have been shown to occur with African Grey Parrots, Amazon parrots, cockatoos and macaws.

Citrobacter (C.)

The three species of *Citrobacter* (*C. freundii*, *C. amalonaticus* and *C. diversus*) are less commonly encountered than other members of the Enterobacteriaceae. *C. freundii* appears to be the most pathogenic of the group. *C. diversus* is rare in birds. When using serologic assays it is important to know that *C. freundii* antibodies often cross-react with those to *S. typhimurium*.

Citrobacter spp. cause serious secondary infections in weaver finches and waxbills. A rapid bacteremia followed by acute death occurs when the organism penetrates the intestinal mucosa. Ostriches, particularly chicks and young birds, also appear to be very susceptible to *Citrobacter* spp. Infected birds of any species may die without any clinical signs, or they can exhibit a brief period of depression and diarrhea prior to death. Postmortem changes indicate septicemia (petechiation of the heart, musculature and parenchyma). Surviving birds frequently become carriers. *C. amalonaticus* is frequently recovered from the intestinal tract of normal Psittaciformes. Intestinal infections would indicate that a disturbance has occurred in the autochthonous flora. A definitive diagnosis requires culturing the organism from affected tissues. The rule-out list is the same as for *Salmonella* spp. Therapeutic decisions should be based on appropriate culture and sensitivity. Neomycin delivered by gavage is often effective in clearing intestinal infections. In flock outbreaks, the same drug administered in the drinking water may be helpful in controlling infections.

There have been no reported cases of citrobacter infections in humans derived from exposure to infected birds.

Klebsiella (K.)

K. pneumoniae and *K. oxytoca* are frequently recovered from birds in which they can function as primary pathogens, particularly in weaver finches, or they can be involved as opportunists in immunosuppressed or stressed patients. These organisms are nonmotile Enterobacteriaceae, and most members of the genus are encapsulated. The mucoid capsule provides them with substantial protection from environmental extremes and many disinfectants. Heat and drying are the best methods of killing *Klebsiella* spp.

Specific information on the transmission, pathogenesis and incubation period for *Klebsiella* spp. in birds is not available. The *Klebsiella* capsule provides a barrier to protect the organism from cellular immunity. However, the capsule is also highly antigenic and stimulates a protective humoral immune response. *Klebsiella* spp. bacteremia usually results in the colonization of the kidney, causing renal failure. In chronic infections, the lungs may also be involved. In many bird groups (eg, pigeons, weaver finches,

goldfinches and siskins, geese, birds of prey, Amazons and African Grey Parrots), infections are not detected until late in the disease process when respiratory signs occur. Encephalomyelitis is occasionally noted in terminal cases. While systemic klebsiella infections are most common, local infections involving the sinuses, skin, oral cavity and crop may also occur, particularly in Psittaciformes. The diagnosis is made by isolation and identification of the organism. The rule-out list is the same as with salmonella.

Yersinia (Y.)

The genus *Yersinia* currently consists of eleven species.[1] Unlike other Enterobacteriaceae, which are strictly rod-shaped, *Yersinia* spp. form ovoid-to-coccoid rods that replicate in the environment at extremely low temperatures (+4°C) if provided the proper sources of organic nitrogen. Because the organism can grow effectively at low temperatures, infections are particularly common during the winter months.

Y. pseudotuberculosis appears to be the most important avian pathogen. *Y. intermedia*, *Y. frederiksenii* and *Y. kristensenii* are frequently isolated from various avian species, but their pathogenicity remains undetermined.

When grown at 20-28°C, *Y. pseudotuberculosis* is motile; when grown at higher temperatures it is nonmotile. Six serovars have been distinguished. Serovar 1 is most frequently isolated from birds. *Y. pseudotuberculosis* has been recovered in an L-form from free-ranging urban pigeons.

Transmission

Y. pseudotuberculosis is thought to be indigenous to northern and middle Europe. The occurrence of this bacterium in other parts of the world including Canada, the United States, Africa and Australia is thought to have arisen from the movement of European birds and rodents to other suitable geographic locations. An unknown percentage of the free-ranging birds in Europe are considered asymptomatic carriers.

Y. pseudotuberculosis infects a wide range of hosts, including many bird species and various mammals, particularly rodents and including humans. Toucans, toucanets, aracaris, barbets and turacos appear to be extremely susceptible.

Clinical Disease and Pathology

Y. pseudotuberculosis may be associated with peracute, acute or chronic clinical disease. Peracute death without clinical signs is common in infected Piciformes and Musophagidae. Clinical signs associated with acute disease include lethargy, dehydration, diarrhea and dyspnea. Emaciation, wasting and flaccid paresis or paralysis are common with subacute or chronic cases. Birds with a wasting syndrome appear similar to animals infected with tuberculosis. Infected ducks frequently develop tarsal joint swelling. Canaries may be severely dyspneic prior to death.

Gross changes associated with peracute infections include swelling of the liver and spleen and bloody-to-fibrinous exudate into the body cavity. Submiliary-to-miliary, sharply demarcated grayish foci within the liver, lungs, spleen and kidneys are common with the acute course. Chronic infections are characterized by granuloma formation in organs and the skeletal musculature. Ascites and osteomyelitis may or may not be present. Ulcers in the proventriculus, ventriculus and duodenum may occur in infected canaries. Tarsitis chronica deformans with caseous exudate in the joint cavity is frequently seen in ducks.

Coagulation necrosis and thrombophlebitis are the common histologic changes. In acute and chronic cases, inflammatory cells infiltrate the necrotic areas and eventually induce granulomas.

Diagnosis

The histopathologic changes, along with the identification of gram-negative coccoid rods, are suggestive of *Y.* spp. infections. Definitive diagnosis requires isolating the organism from affected tissues. Placing contaminated samples in a cool environment for two weeks may help in recovering *Y.* spp. Isolating avian strains of yersinia appears to be more difficult than isolating mammalian strains. Because there are no demonstrable biochemical or serologic differences, it has been assumed that avian strains are more difficult to grow due to different nutritional requirements. The most consistent isolation results have been obtained by placing fecal or organic material in heart-infusion broth with 5% glucose and storing this material in a refrigerator for two weeks. The material is then inoculated on blood agar plates with 0.2% Tween 80 and 50 ppm tellurite and incubated at 37°C for two days. *Yersinia* causes a reduction of the tellurite, turning the colonies black.[25]

Treatment

Birds with the peracute to acute forms of yersiniosis usually die before therapy can be instigated. Parenteral drug administration is required if therapy has any chance of being successful. Treating chronic cases is difficult because granulomas prevent antibiotics from reaching the yersinia organisms nestled in the center of necrotic debris. Flock outbreaks can be prevented by treating clinically unaffected animals and applying strict sanitary measures.

Control

In non-European countries where *Y. pseudotuberculosis* is not endemic, repeated culture of feces during the quarantine period should be used to prevent infected birds from entering the country. In endemic areas, rodents and free-ranging birds can serve as reservoirs, and flock control depends on preventing these animals from contaminating feed supplies and keeping them out of the aviary. Several experimental vaccines for *Y. pseudotuberculosis* have proven to be ineffective. Feeding infected mice to toucans could serve as a source of infection.

Y. enterocolitica is principally a human pathogen. Gulls, herons, birds of prey, crows, blackbirds and European robins that inhabit areas contaminated with human sewage are frequently infected. Young children of elementary school age are particularly susceptible to infections.

Pseudomonas (Ps.) and *Aeromonas (Ae.)*

These two gram-negative rods are taxonomically unrelated but nevertheless have characteristics that make it best from a clinical perspective to discuss them together. Both of these genera contain numerous species, but only *Ps. aeruginosa* and *Ae. hydrophila* are common avian pathogens. Both bacteria are frequently found in aquatic environments and can propagate in cool water (20°C or lower). Both bacteria will grow on common media and induce β-hemolysis on blood plates. These hemolysins are potent toxins and are capable of damaging many cells in addition to erythrocytes. *Ps. aeruginosa* produces a blue-green diffusible pigment and has a sweetish odor. *Ae. hydrophila* causes the typical bad smell of the proteinolytic organisms and may be confused with *E. coli* on Endo- or MacConkey plates. These bacterial genera are further divided into several serovars and biovars. There have been insufficient studies to divide the avian species in a similar fashion.

Both of these bacteria infect many mammals, including humans, as well as most of the birds that have been tested. Free-ranging waterfowl appear to be particularly susceptible, but any free-ranging avian species that contacts contaminated food or water is probably susceptible. Of the commonly maintained zoo birds, penguins are very susceptible.

Pathogenesis

Some strains of *Pseudomonas* and *Aeromonas* produce a number of extracellular toxins, including hemolysins, elastase, protease and lecithinase, that cause cellular damage resulting in edema, hemorrhage and tissue necrosis. Avian strains of *Ae. hydrophila* that are acetoin-producing (Voges-Proskauer-positive) are considered to be highly toxic.[6] Both species are principally secondary invaders. However, the toxins secreted by these organisms can be life-threatening once colonization of the host occurs. *Ps. aeruginosa* is resistant to many commonly used antibiotics and can cause secondary superinfections in patients being treated for other bacterial infections.

Clinical Disease and Pathology

Virulent strains of these bacteria can cause a septicemia that induces diarrhea, dehydration and dyspnea followed by acute death. Infected skin lesions are edematous or necrotizing. Localized infections may occur in the upper respiratory tract, causing rhinitis, sinusitis and laryngitis. Hemorrhages and coalescent necrosis in the liver, spleen and kidney are the most common postmortem findings (see Figure 33.2). Catarrhal to hemorrhagic enteritis with edema and fibrinous inflammation of the serosal membranes may also be noted. Histologic changes associated with infections include severe inflammatory reactions involving the venous and arterial walls. Bacteria are often identifiable within the lumina. The formation of thrombi, hemorrhage and necrosis of the infected vessels are the results.

Diagnosis and Control

The causative agent should be isolated and identified. Aviary outbreaks of pseudomonas are most common when organic material contaminates the water supply, allowing a proliferation of the organisms in the drinking water (Table 33.3). Routine cleaning of food and water containers, along with any external water pipes, is an important control measure. Incubator contamination can be prevented by periodically cleaning the water reservoir. Waterfowl are particularly susceptible to infections when water temperatures are above 20°C, allowing rapid proliferation of

Ae. hydrophila. Removing waterfowl from ponds during these periods is a good control measure.

Alcaligenes (Ac.) and *Bordetella (Bo.)*

The genera *Alcaligenes* and *Bordetella* are taxonomically related. Both genera are widely spread in the environment. Alcaligenes is found mainly in aquatic environments. *Ac. faecalis*, *Bo. avium* and *Bo. bronchiseptica* infect a wide variety of birds in many orders. Psittaciformes and turkeys as well as many finches seem to be particularly susceptible to these bacteria.

There are no details on the pathogenicity of these genera in birds. Alcaligenes and bordetella are opportunistic pathogens that potentiate viral and other bacterial infections. *Bordetella avium*, a more recently recognized member of the genus, seems to preferentially bind to the ciliated epithelial cells of the upper respiratory tract.

In turkeys, combined infections of *Bo. avium* and the turkey rhinotracheitis virus cause the clinical and pathologic signs of rhinotracheitis. In other avian species, clinical signs are uncommon and, if present at all, are nonspecific. At necropsy, tracheitis, bronchopneumonia and air sacculitis are common findings with subacute to chronic courses of bordetella, whereas alcaligenes infections are characterized by coalescent liver necrosis in addition to respiratory disease.

Diagnosis

A confirmatory diagnosis requires isolation and identification of the causative agent. Serologic flock diagnosis by means of the slide agglutination test or antibody titration by the Gruber-Widal method is possible although no commercial antigens are available.

Campylobacter (C.)

Campylobacter spp. from birds have been classified as: *C. jejuni*, the most frequent, and probably also the most pathogenic; *C. coli*, which is considered to be apathogenic, but is frequently confused with *C. jejuni*; and *C. laridis*, which is isolated from gulls, whose pathogenicity is still not definitely known.[36]

C. jejuni has incorrectly been labeled as *Vibrio* (usually *V. metschnikovii*). This error has serious consequences because *Vibrio cholerae*, serotype 01 and noncholera *Vibrio* can be recovered from healthy birds.

C. jejuni may appear in different forms including a short comma, s-shaped, long spiral or coccoid form. The latter is usually an indication of degeneration. Colony formation takes 72 to 96 hours at 37-42°C in a microaerobic environment. Blood agar or selective media are best for isolation. *C. jejuni* is relatively unstable in the environment (surviving less than one week).

There are at least 50 serovars of *C. jejuni*.[42] While the possibility of birds serving as a source of infection for mammals and vice versa has been discussed, this transmission potential has been insufficiently studied.

The host spectrum is large, and includes chickens, turkeys, pheasants, crows, gulls, ducks, geese, pigeons, shorebirds, Pekin Nightingale, Nandu and the Great Bustard. *C. jejuni* has recently been reported in passerine birds, particularly in tropical finches (Estrildidae) and, to a lesser degree, in canaries.[20] Details of occurrence and pathogenicity in many avian species have been reported.[51] Psittaciformes are susceptible, but few documented infections have been reported.

Pathogenesis

C. jejuni can be isolated from the intestinal tract of clinically affected and asymptomatic birds. Experimental infections generally cause hepatitis. Factors that determine if an infected bird becomes clinically affected have not been established. Clinical disease is common in birds with parasitic infections (coccidia and nematodes), and these agents have been suggested as predisposing factors.

Clinical Disease and Pathology

Clinical signs are generally associated with subacute to chronic hepatitis and include lethargy, anorexia, diarrhea (frequently with yellowish stained feces) and emaciation. Transmission takes several weeks through the flock, but spontaneous recovery and relapses do occur. High mortality has been noted in finches, especially among fledglings.[20] Sudden death as a result of liver rupture is possible. Heterophilia and thrombocytophilia are the most consistent changes in the CBC.

At necropsy the liver is enlarged, pale or greenish in color and is congested, with or without hemorrhage. Perivascular infiltrates make the lobules appear more prominent. Coalescing necrotic hepatitis is a

common histologic finding. Catarrhal enteritis (also hemorrhagic enteritis in pheasants, turkeys, Tengmalm's Owl and coot) has been described. In gulls, erosion of the ventriculus has been reported.

Diagnosis

Diagnosis requires isolation of *C. jejuni* from affected tissues. Phase-contrast microscopy of bile to demonstrate suggestive organisms may provide a tentative diagnosis. Fecal samples can be used for culture in live birds. A transport medium is necessary to ensure survival of the organism.

Treatment and Control

There are discrepancies between the antibiograms and clinical recovery. Erythromycin or tetracyclines, dehydro- or streptomycin (never in Psittaciformes) or furane derivatives (not in waterfowl) can be tried. Diseases frequently recur despite therapy. Thorough cleaning and disinfecting of the aviary may help prevent reinfection. Dogs can be a reservoir for human infections, but it is not known if they can transmit the organisms to birds. Dogs should not be allowed direct access to birds.

Vibrio (V.)

The genus *Vibrio* comprises numerous species that are not easy to differentiate. *V. cholerae* is of utmost importance as a zoonotic organism. In birds, especially in gulls, *V. cholerae* (serovar 01) is demonstrable in the intestinal tract, although no clinical or pathologic signs have been described. Numerous other *V.* spp. are collectively designated NAG (= non-agglutinable), because they do not agglutinate with human cholera antiserum. NAG strains can be isolated from the feces of many bird species, particularly waterfowl (vibrio is generally found in aquatic habitats).[4] NAG vibrios have not been documented as a cause of clinical disease in any avian species. Because some NAG strains can cause a mild intestinal disease in humans, an analysis of avian strains in the vicinity of human cases might be advisable.

Spirochaetaceae

Borrelia (*Bor.*) *anserina* (syn. *Spirochaeta gallinarum*) is a gram-negative, helical motile organism that stains with Giemsa. A granular form of the organism may occur in ticks and the blood of the birds that have recovered from a disease. The host spectrum includes geese and ducks, turkeys, chickens, pheasants, grouse, partridges, pigeons, crows, magpies, House Sparrows, starlings and African Grey Parrots.

Transmission

The main vectors for transmission are ticks, in which the organism can be passed transovarially and survive for over a year. Mosquitoes and other biting insects play a minor role in transmission. Transmission from bird to bird by excreta is of minor importance epizootiologically.

Pathogenesis

Young chicks (one to three weeks of age) are particularly susceptible. Adults may also be infected. *Bor.* is in the peripheral blood from the fourth to the ninth day post-infection and remains in the blood approximately seven days. The organism can then be found in parenchymal organs for another 30 days. Death may occur from embolism due to agglutinating borrelias. A strain-specific immunity develops in survivors. Incubation periods are four to eight days depending on the species of ticks.

Clinical Disease and Pathology

Acute cases are characterized by a high fever (bacteria generally cause a low body temperature), anorexia, depression (droopy, cyanotic heads), yellowish diarrhea, lethargy, ataxia and paralysis. Morbidity is high, and mortality may range from 10 to 100% depending upon the susceptibility of the host. Spontaneous recovery may occur around the sixth day post-infection. Chronic disease is characterized by anemia, paralysis and dyspnea.

The albumin fraction in the serum decreases to 37% and an increase of the aspartate aminotransferase is accompanied by a decrease of the alkaline phosphatase, the total lipids and the cholesterol.

At necropsy, a mottled, severely enlarged liver is characteristic except in pheasants. In these birds the spleen may be small or normal in size.[58] The liver shows hemorrhages and necrotic foci. Mucoid hemorrhagic enteritis, serofibrinous pericarditis and swollen kidneys may also be seen. Histology displays multiple necrotic foci without inflammatory reactions. *Bor.* can be demonstrated by argentation.

Diagnosis

Blood smears stained with Giemsa or examined by darkfield microscopy are useful for diagnosis. Culturing *Bor.* sp. is very difficult. Antibodies (agglutination, fluorescence techniques, immunodiffusion) can be demonstrated from the 4th to the 30th day post-infection.

- ***Treponema (T.) spp.:*** *Treponema* spp. are helical motile organisms that are much smaller than *Spirochaeta* spp. An unclassified *Treponema* sp. (0.5 μm wide with 13 to 15 flagella)[12] is the cause of a watery, intermittent typhlitis in chickens. The organism is antigenically related to but distinct from *T. hyodysenteriae*. Chickens are the only defined host and lose weight in response to a malabsorption syndrome.[12,22] Histopathology shows an increased number of goblet cells in the cecal mucosa, focal epithelial desquamation and many *Treponema* organisms in the small fissures of the epithelium. The incubation period is one to seven weeks dependent on the infective dose. Culture is possible on spectinomycin blood agar in an anaerobic atmosphere. Fluorescent antibodies designed for *T. hyodysenteriae* can be used to demonstrate the organisms in affected tissue.

- ***Spirochaetaceae Undifferentiated:*** A non-classified spirochete from choanal and tracheal mucus of a cockatiel has been described. The organisms were 0.3 to 0.4 X 10 to 15 μm in size and occurred in large numbers on the mucosa. Two other cockatiels have been found to harbor similar organisms in the respiratory passage. Histopathology showed a mild inflammatory reaction in the nasal sinus, but not in the trachea. In the lower parts of the respiratory tract, the organisms could not be demonstrated using argentation. The significance of these findings is unknown. Interference with the ciliary activity of the respiratory mucosa is conceivable.[65]

  Spirochetes were demonstrated in a pharyngeal swab of a cockatiel that was depressed and sneezing. The bird had spent ten minutes with another cockatiel that was showing similar clinical signs ten days before being presented for evaluation.

Pasteurella (P.)

The family of Pasteurellaceae currently includes the genera *Pasteurella*, *Actinobacillus* and *Haemophilus*.[49] All three genera can be pathogens in birds.

Pasteurella characteristically exhibit bipolar staining in tissue smears or from first culture passages when fixed in methanol and stained with methylene blue. There are presently eleven species within this genus, but others will undoubtedly be added. *P. multocida*, which causes fowl cholera, and *P. gallinarum* are two of the most commonly encountered species. The latter is usually considered a secondary pathogen. Some of the *Pasteurella* organisms isolated from waterfowl, pigeons and Psittaciformes have not been taxonomically classified. Some isolates designated *P. haemolytica* (gram-negative, polymorphic rods) are improperly classified and do not belong in this genus. The characteristics of these strains resemble those of the genus *Actinobacillus*. Reports on isolation of *P. pneumotropica* from birds are questionable. This species appears to be host-specific and is found in rats or other rodents. Similarly, *P. ureae* is believed to be host-specific for humans.

Pasteurella has been associated with disease in Phasianiformes, Anatiformes, Psittaciformes, Columbiformes and Passeriformes. There is variance in species susceptibility. The clinical presentations and pathomorphologic changes are similar to those described for yersiniosis. Propagation outside the host can occur but requires very specific conditions of temperature, relative humidity and pH. Such conditions may occur in large bodies of water, and in these situations *Pasteurella* spp. can survive for long periods. Epornitics are most common in the northern hemisphere from November to December. Outbreaks in tropical climates peak with seasonal highs in ambient temperature and humidity.

- ***P. multocida:*** The etiologic agent of fowl cholera, the species is divided into strains based on 16 serologically distinct endotoxins and 4 capsular polysaccharides. Serotypes 1 and 3 and capsule types A and D are most commonly isolated from birds. Three subspecies, *P. multocida (m.) multocida*, *P. m. gallinarum* and *P. m. septica*, have been distinguished based on differences in virulence. The latter is considered to be the most virulent.

- ***P. pneumotropica*** is indigenous in rodents and occasionally causes disease in aviary birds and pigeons. Infected birds develop pneumonia and may exhibit dyspnea shortly before death. Detailed information on the pathogenesis and clinical progression of *P. pneumotropica* in companion birds has not been reported.

- ***P. gallinarum*** appears to have a similar host spectrum as *P. multocida*; however, *P. gallinarum* is thought to be much less pathogenic. If the organism is able to colonize the respiratory mucosa, it can induce conjunctivitis and respiratory signs including coryza, rales and dyspnea. *P. gallinarum* has been isolated from the choanae and nostrils of Psittaciformes with concomitant *Aspergillus* spp. infections in the lungs and air sacs. Aspergillosis is one of the triggering factors that allows this secondary pathogen to overcome host defenses. Postmortem findings associated with *P. gallinarum* include catarrhal to

FIG 33.5 The hallmark sign of bacterial septicemia is depression. Birds that are bitten or scratched by cats frequently develop *P. multocida* bacteremia. Affected birds may appear normal immediately after the injury occurs, and become rapidly depressed and die 12 to 24 hours later. Any carnivore-related injury in a bird is a critical emergency.

fibrinous inflammation of the upper respiratory tract, pneumonia and air sacculitis.

Transmission

Pasteurella infections in birds principally occur in the respiratory tract. Asymptomatic carriers harbor the bacteria in the nasal cavities, sinuses and choanae. Transmission can occur through direct contact with contaminated aerosols or through mechanical vectors such as blood-sucking mites. Infected rodents and free-ranging birds are considered important reservoirs. *Pasteurella* is shed almost exclusively from the upper respiratory tract. Shedding in the feces is rare, and egg transmission has not been documented. *P. multocida* is a common inhabitant of the oral cavity of some carnivores, particularly cats, and septicemic infections can occur through bite wounds. Cats should always be considered to be carriers of *P. multocida*, and any bird that has been mouthed by a cat should be treated with antibiotics immediately (Figure 33.5).[40]

Pathogenesis

Virulent strains of *P. multocida* cause an acute septicemia and death. Less virulent strains result in bacteriemia and colonization of the lungs, liver, kidneys, spleen and heart. Weakly virulent strains generally cause a chronic respiratory disease. The endotoxins produced by *Pasteurella* damage blood vessels, causing edema, hemorrhage and coagulation necrosis, particularly in the liver. Diseases by less virulent strains usually occur in stressed or immunocompromised hosts.

Clinical Disease and Pathology

Acute forms are characterized by cyanosis, dyspnea and diarrhea followed by death. Excess mucus may be present around the nostrils or beak. Birds that survive acute disease often develop respiratory rales, sinusitis, conjunctivitis or swelling of the sinus infraorbitalis. Arthritis and CNS signs have been reported in some chronic cases. Granulomatous dermatitis has been noted in raptors, owls and pigeons.

Postmortem findings with acute disease may be absent or limited to petechiae or ecchymoses of the parenchymal organs. Prolonged cases are characterized by exudative serositis (mainly white, in contrast to yellow with *E. coli*) and the formation of necrotic foci in infected organs. Catarrhal to fibrinous rhinitis, necrotic pneumonia, sinusitis, blepharoconjunctivitis and tracheitis are common with chronic courses. Following bacteremia, *Pasteurella* may colonize numerous tissues, resulting in arthritis, osteomyelitis, otitis media and granulomatous dermatitis. Granulomas may also be noted in the liver and spleen. Some strains will colonize the air cells of the cranial bones causing fibrinous exudate. In waterfowl, a diphtheroid enteritis may be observed. Histologic changes are nonspecific.

Diagnosis

The isolation of the causative agent is necessary. The occurrence of *Pasteurella* spp. in a flock can be determined through serology using immunodiffusion or indirect hemagglutination tests. Unfortunately, commercial antigens for these tests are not available. Serotyping and differentiation of the subspecies require specialized testing.

Treatment

Septicemic birds rarely survive, even when treated intensively. Parenteral administration of broad-spectrum, long-acting sulfonamides can be tried. The combined use of antibiotics and hyperimmune serum has proven to be beneficial. Treating birds with chronic forms is very difficult because of the irreversible damage that occurs to parenchymal organs.

Control

Preventing rodents and free-ranging birds from entering the aviary is important in preventing infec-

tions. Vaccines for *P. multocida* are commercially available, but their effectiveness is poor. Failures associated with the vaccine occur because of the numerous different serotypes and the fact that endotoxins are more immunogenic than the bacterial capsule. The production of vaccines from strains that persist in an aviary may provide successful long-term control.

Actinobacillus (At.)

The genus *Actinobacillus* consists of a group of organisms provisionally differentiated into more than 20 biovars, some of which have a rather high host specificity. The taxonomic reclassification of the Pasteurellaceae is intertwined with the genus *Actinobacillus*. Because of these classification revisions, even the actinobacilli that are pathogenic in birds have not been named. The situation has been complicated in recent years because many new strains have been isolated from Psittaciformes, Columbiformes, Anatiformes and Fringillidae. One biovar has been referenced in the literature as *P. haemolytica* syn. *At. salpingitidis*. However, this classification is not valid. The knowledge of the biology and pathogenicity of the members of the genus is limited.

Actinobacilli are polymorphic rods that may exhibit bipolar staining similar to Pasteurellae. Most strains grow only on blood agar plates or media containing serum. Some strains, particularly of *At. salpingitidis*, hemolyze avian and bovine erythrocytes or produce exotoxins that are capable of causing arteritis. Some strains are considered to be primary pathogens, but the majority of this genus is comprised of opportunistic organisms. There are no simple laboratory tests for differentiation between primary and secondary invaders.

There is little information on routes of transmission. It has been proven that egg transmission of some strains occurs in chickens and geese. Incubation periods in the avian host are not known.

Clinical Disease and Pathology

Many infected birds die acutely. Birds with a more chronic course typically develop joint lesions. Species-specific strains that infect geese morphologically resemble *P. influenzae*. This organism has been referenced as a cause of chronic disease in the gosling,[26] characterized by emaciation, failure to thrive, poor feed conversion and arthritis. Egg transmission may cause reduced hatchability. Asymptomatic infections are thought to occur in adult breeders, with clinical signs developing in goslings each season. At necropsy, liver necrosis, salpingitis, peritonitis, endocarditis valvularis and fibrinous arthritis are typical lesions. In young geese, polyserositis and arthritis are prominent.

Diagnosis and Treatment

Isolation and identification of the causative agent is necessary for diagnosis.

E. coli and other bacteria, particularly anatipestifer infections in waterfowl, have to be considered as causative agents for the salpingitis, peritonitis and polyserositis.

Tetracyclines and chloramphenicol are indicated for initial therapy. Sensitivities for many strains are difficult to interpret because of oversized inhibition zones. In young geese, antibiotics must be given during the first week of life or lesions become too extensive to be reversed.

Haemophilus (H.)

The haemophilus strains that infect companion birds have not been properly classified. Chickens are considered to be the only definitive host of *H. paragallinarum*, which is the agent of coryza contagiosa gallinarum. Experimentally, Columbiformes and Anatiformes are resistant to infection. Several *Haemophilus* spp., including *H. avium* and *H. paravium*, can be isolated from birds with coryza; however, their involvement in the disease process is questionable. They probably serve as secondary invaders that sustain upper and sometimes lower respiratory tract disease.[11,35]

Pathogenesis

Details on the pathogenesis of *Haemophilus* spp. strains that infect Psittaciformes and Columbiformes are scarce. *H. paragallinarum* is known to produce a number of cellular toxins, including neuraminidase, nitratase and catalase. Birds do not appear to develop an immune response following infection, and relapses are common.

Clinical Disease and Pathology

Haemophilus infections generally cause a rhinitis that results in a serous-to-mucoid or even fibrinous exudate. Conjunctivitis and sinusitis may also occur. The most common postmortem finding is catarrhal-to-fibrinous rhinitis. Bronchopneumonia and air sacculitis are frequently described but are usually the result of concomitant infections (virus, other bacte-

ria, *Candida* spp.). For those lesions supposedly caused by *Haemophilus* spp. apart from *H. paragallinarum* see Table 33.4.[32,35] Histopathology reveals uncharacteristic lesions.

TABLE 33.4 Clinical Disease Caused by Haemophilus in Avian Species

Blue Crane	Pneumonia (together with staphylococci) and necrosis of liver tissue
Pigeon	Rhinitis
African Grey Parrot	Pneumonia, air sacculitis
Plum-headed Parakeet	Sinusitis, swelling of the liver together with E. coli or related organisms
Eastern Rosella	Sinusitis, swelling of the liver together with E. coli or related organisms
Budgerigar	Rhinitis (possibly together with Pasteurella or E. coli)
Muscovy Duck	Rhinitis, sinusitis
Andean Goose	Rhinitis, hemorrhage, jejunitis
Turkey	Sinusitis, air sacculitis
Golden Pheasant	Sinusitis, diphtheroid surface lining of the beak cavity
Siamese Fireback	Necrosis of the lung tissue.

Other Gram-negative Rods

New Duck Disease (Duck Septicemia)

The etiologic agent of duck septicemia has been suspected to be *Pfeifferella, Pasteurella* or *Moraxella anatipestifer*. The causative organism has recently been placed in the genus *Cytophaga*, which is a semiaerobic, nonmotile rod.[49] The genus contains at least 19 serovars; serovars 1-3 are most common in Europe; serovars 1, 2 and 5 are most common in the United States; serovar 3 is frequently isolated in Australia. This organism is known to infect ducks and geese, free-ranging waterfowl, turkeys, pheasants and Psittaciformes. There is no information on its virulence or ability to survive in the environment.

Experimental disease does not occur following oral administration, and the respiratory tract is thought to be the primary portal of entrance to the host. Egg transmission resulting in high morbidity and mortality of ducklings is a substantial factor for the flock.

The pathogenicity of cytophaga is undetermined. Undoubtedly, there are strain differences in virulence, and the condition of the host must also play a role. The incubation period in ducks ranges from three to ten days.

Clinical Disease and Pathology

Infected ducklings (two weeks of age) usually die peracutely. Mortality rates in this age group can reach 75% of exposed young. Acute disease develops in older birds and is characterized by sinusitis, conjunctivitis, coughing and diarrhea, followed in two days by tremors, ataxia and convulsions. Survivors are stunted and fail to grow. Fibrinopurulent polyserositis is the characteristic postmortem finding. Other changes include lung congestion, hepatomegaly, splenomegaly, pericarditis and perihepatitis. Cytophaga-induced spondylitis with compression of the spinal cord was reported in turkeys.[10] Diffuse fibrinous meningitis with lymphocytic infiltration around the meningeal blood vessels was a characteristic histologic lesion. There was exudate formation within the ventricles as well as proliferation of microglia in the subpial and periventricular system.

Diagnosis

The occurrence of polyserositis is highly suggestive of an infection. Isolation and identification of the causative agent is necessary in all other cases. An ELISA can be used to survey a flock for serologic response.

Tularemia

Tularemia is caused by *Francisella tularensis*, a motile, short rod, 0.2 X 0.3-0.7 μm in size. Isolates are reported occasionally, mainly from birds that inhabit the northern and subarctic regions of the northern hemisphere, such as the Common Pheasant, Waxwing, Ural Owl, Rough-legged Hawk and Common Raven. Rodents are considered to be the primary reservoir. Nothing is known about the clinical signs. The pathology resembles that of *Pasteurella* or *Yersinia* infections. *Francisella* previously has been classified with these two genera. The organism is considered to be a zoonotic agent.

Acinetobacter calcoaceticus (An.)

This organism forms either cocci (fresh culture) or rods. It grows on commonly used media, even on some selective media for Enterobacteriaceae. The host spectrum is wide, and many avian orders can harbor the organism in the respiratory or intestinal tracts. Egg transmission is possible in many avian species. No reports conclusively describe consistently

occurring lesions in any avian species. Therefore, it is assumed that the organism has a low pathogenicity, and infections indicate a compromised host.

Gram-positive Bacteria of Clinical Significance

Staphylococcus (S.)

Staphylococcus infections can induce sporadic or enzootic disease in many avian species. Clinical manifestations may include acute septicemia or subacute-to-chronic arthritis, osteomyelitis and osteitis. Less common clinical problems include vesicular dermatitis or omphalitis. Staphylococci, particularly *S. aureus*, can function as primary pathogens or may complicate other infections as secondary invaders.

Isolation of the staphylococci is relatively simple using common media and growth conditions. Taxonomy literature[50] currently lists 21 *Staphylococcus* spp., 14 of which can be found in birds and are given here in order of decreasing frequency. *S. xylosis* is considered almost apathogenic. *S. sciuri* and *S. lentus* (the latter a former biovar of *S. sciuri*) have some pathogenicity markers. *S. aureus* includes more virulent strains than any other species. These four *Staphylococcus* species are further divided into several biovars. Because some of these biovars are recovered only in a limited number of closely related bird species, they may represent bacterial organisms that have adapted to specific hosts. Other less commonly isolated species include *S. intermedius*, *S. hyicus*, *S. cohnii*, *S. saprophyticus*, *S. haemolyticus*, *S. warneri*, *S. hominis*, *S. epidermidis*, *S. gallinarum* and *S. capitis*. *S. epidermis* was frequently cited in earlier literature but, using current diagnostic tools, is today rarely found in birds. The isolation of these less common species frequently depends on the internal or external environment of the patient, and limited information is available concerning their pathogenicity.

Staphylococci have several pathogenicity markers including production of the clumping factor, hemolysins, DNase, phosphatase, protein A and leukocidine. The presence of the clumping factor seems to correlate closely with pathogenicity in avian patients. Other pathogenicity markers have not been studied sufficiently to determine their importance in birds. Staphylococcus protein A, which is found in the bacterial cell wall, is capable of binding to the Fc-fragment of immunoglobulin, thereby inhibiting phagocytosis of the organism. The correlation between protein A production and the virulence of avian *Staphylococcus* strains is poorly documented.

Members of the genus *Staphylococcus* (apart from *S. aureus*) are commonly recovered from many avian species and are considered part of the autochthonous flora. When present in diseased tissue, they are generally considered to be secondary invaders. Because *S. aureus* includes the most virulent strains, the following discussion applies mainly to this species.

S. aureus

Avian strains of virulent *S. aureus* are relatively species-specific and rarely induce disease in mammals. The organism is found in abundant quantities in air and dust. Isolation of the organism can frequently be accomplished from the skin and the mucosa of the respiratory or digestive tract of clinically normal birds (Figure 33.6). *S. aureus*, like other *Staphylococcus* species, is relatively stable in the environment and can remain infectious for long peri-

FIG 33.6 A five-year-old Amazon parrot was presented with an acute onset of picking at the feet and legs, which caused hyperemia and scab formation. This syndrome, called Amazon foot necrosis, has been reported in *Amazona* spp., and *Staphylococcus* spp. are frequently isolated from the lesions. However, staphylococci are part of the autochthonous flora and are probably not the primary cause of this problem. This affected Amazon parrot belonged to a client who smoked, and when the owner started washing her hands after smoking (presumably to remove nicotine sulfate, a potent toxin), the foot and leg lesions resolved. The client eventually stopped smoking and the bird had no further episodes of Amazon foot necrosis.

ods of time outside the host. Given proper conditions, the organism can propagate in an external environment. Like many bacteria, *Staphylococcus* can also develop resistance to disinfectants following continuous exposure, and frequent changing of disinfectants is required to prevent the development of resistant strains.

Techniques for differentiating between avian and mammalian strains of *S. aureus* remain unsatisfactory. Evaluating the type of lesions caused by experimental subcutaneous infection of *S. aureus* in birds may prove valuable in differentiating between avian and mammalian strains.

Pathogenesis

The importance of *Staphylococcus* infections in birds is clinically underestimated. Individual strains may cause clinical problems in one bird while being considered normal autochthonous flora in another. Lipoteichoic acid, a major component of the staphylococcal wall, is instrumental in the specific capacity of this organism to bind to host cell receptors, particularly in the respiratory system. Such binding is a precondition for colonization and subsequent infection. However, avirulent strains can also bind to the same receptors and may compete for receptor sites, preventing colonization of virulent strains. This process is called "bacterial interference." Suitable strains of *Staphylococcus* have been used as prophylactic tools, especially in poults. Some of these apathogenic strains also produce bacteriocin, which can inhibit the growth of a variety of bacteria.

Although endogenous infections can be primary, they are frequently secondary to respiratory tract colonization and progress to septicemia. If an infected bird survives the acute septicemic stage of the disease, it will typically develop localized changes. Focal lesions occur as a result of thrombi formation in the arterioles and capillaries, which leads to ischemic necrosis. Clinically, these necrotic areas are frequently localized at the tips of the extremities and in the skin. In addition, infarction can also cause necrotic lesions in internal organs, particularly the liver and kidneys, which are more difficult to discern clinically. The central nervous system (CNS) is another site prone to *Staphylococcus*-induced lesions. Postsepticemic development of arthritis, tenovaginitis, osteitis and osteomyelitis followed by chronic skeletal changes are considered together as one disease process (Figure 33.7). These problems usually occur after a four- to seven-day period of septicemia.

Exogenous infections usually result in localized skin disease, although subsequent septicemia can occur in some cases. Birds, unlike mammals, are generally resistant to wound infection. To become established, exogenous bacteria typically require epithelium damaged by other infectious agents (particularly clostridia or poxvirus), immunosuppression (including immunosuppressive viruses such as retroviruses or reovirus), environmental stressors or prolonged application of an antibiotic.

Localized problems associated with *Staphylococcus* spp. can also result from a delayed hypersensitivity reaction. This type of response is considered to be one of the major factors in treating staphylococcus-related bumblefoot.

Clinical Disease and Pathology

Staphylococcus can induce a wide range of clinical and pathologic lesions, including high embryonic mortality, yolk sac or umbilical inflammation, septicemia, arthritis-synovitis, osteomyelitis, vesicular dermatitis, gangrenous dermatitis and bumblefoot.

Endogenous infections usually cause internal lesions, while exogenous infections frequently result in dermatitis and bumblefoot. Staphylococcal lesions in the umbilical region are typically either dry and brownish or smudgy, reddish and edematous. Clinical problems are most common in newly hatched chicks (up to ten days of age), in which the yolk sac in the body cavity is not absorbed normally, with possible decomposition of its contents.

Staphylococcus septicemia may be characterized by nonspecific clinical signs including lethargy, anorexia, a kyphotic posture, ruffled plumage and sudden death. The acute occurrence of necrosis to the distal digits or adnexa of the head and neck is suggestive of a thrombi-inducing infection, which can be a sequela to staphylococcus septicemia. During the initial phases of the ischemic process, the involved digits may be swollen, congested and painful, and many affected birds exhibit lameness. The acute onset of tremors, opisthotonos and torticollis can often be linked to staphylococcus-induced necrosis in the CNS. Gross lesions associated with staphylococcus septicemia include petechiae and ecchymoses of internal organs. Chronic infections may result in endocarditis valvularis. Histologic changes vary with the clinical course of disease but typically consist of a heterophilic and granulomatous response.

Arthritis-synovitis, characterized by the formation of serofibrinous or fibrinous inflammation of the

FIG 33.7 *Staphylococcus* spp. can cause osteomyelitis either secondary to septicemia or following an injury that allows colonization of the bone. Chronic osteomyelitis, as demonstrated in this radiograph, typically requires surgery to remove necrotic tissue and long-term antibiotic therapy, preferably with clindamycin because of its high affinity to bone and bone marrow.

synovial membranes of tendon sheaths and articular bursae, is frequently noted with staphylococcal infections in gallinaceous species. Any joint may be involved but there appears to be a predilection for colonization of the tarsal and metatarsal joints. Following antibiotic therapy, staphylococcus may be present in its unstable L-form, which is difficult to treat.

In immature birds with active growth plates, *Staphylococcus* frequently localizes in the epiphyseal area with secondary invasion of the bone marrow, resulting in osteomyelitis. Endogenous osteomyelitis is considered to be impossible after consolidation of the growth plate.[46] Infection of the growth plates often leads to chronic skeletal abnormalities. Infections are frequently localized to the proximal epiphyses of the femur, tibiotarsus, tarsometatarsus and fifth to seventh thoracic vertebrae. Vertebral injury may lead to clinical changes described as "kinky back" (Figure 33.8). Swelling and colliquation associated with the infection cause deformation of the vertebral spongiosa, which may lead to narrowing of the vertebral foramina and compression of the spinal cord.

Staphylococcus-induced vesicular dermatitis is characterized by the formation of vesicles containing yellowish exudate that form brownish to blackish crusts following rupture. Concomitant infection with poxvirus (or other immunosuppressive agents) may be involved in the disease process. Histologic evaluation of biopsy samples is required to confirm an underlying poxvirus.

Staphylococcus-induced gangrenous dermatitis is initially recognized by the occurrence of subcutaneous edema and hemorrhage followed by inflammation of the skin. Affected skin is typically blackish and smudgy and feather loss is common. *Clostridium perfringens* or another *Clostridium* sp. is a common secondary invader. Both *Staphylococcus* and *Clostridium* require a triggering factor (often damaged epithelium) to enter the tissue. Gangrenous dermatitis is rare in most bird species.

Advanced bumblefoot is a necrotizing abscess on the plantar surface of the foot. Depending on the location and chronicity of the abscess, infection may or may not extend to neighboring joints, tendon sheaths and bones. The condition is frequently described in raptors but may occur in other avian species. The precise pathogenesis of bumblefoot is undetermined (see Chapter 16). Although staphylococci are frequently isolated from these lesions, they are by no means the only bacteria that can be recovered from diseased tissue. Systemic infections that result in other le-

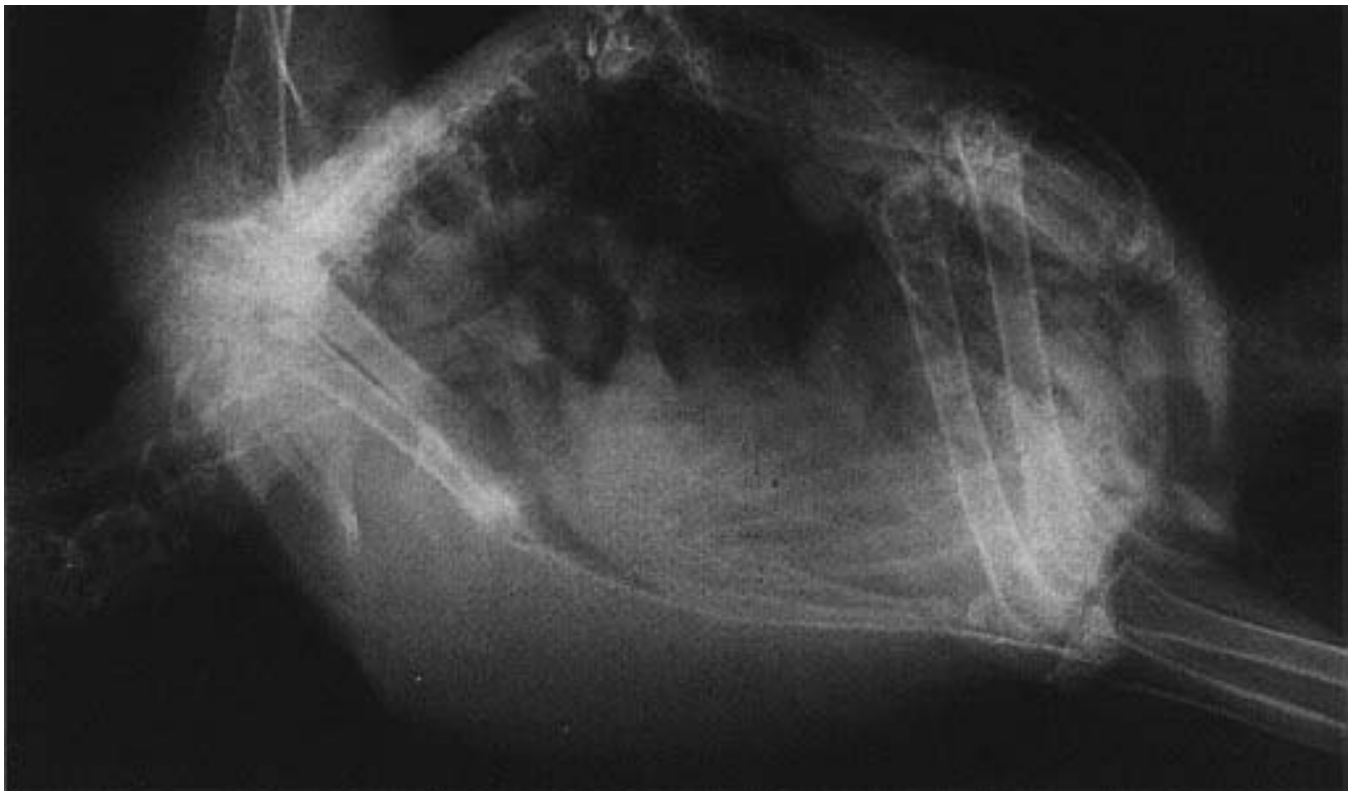


FIG 33.8 An eight-week-old African Grey Parrot was presented for an inability to stand or ambulate properly. On physical examination, the bird was BAR, in excellent weight (325 grams) and had a palpable spinal deformity. Radiographs indicated scoliosis. Bacterial infections including *Staphylococcus* spp. can cause spinal deformities. In this bird, the WBC was normal and the etiology of the problem was undetermined. Congenital abnormalities appear to be particularly common in African Grey Parrots and may have been the cause of this scoliosis.

sions or death can occur secondary to bumblefoot caused by virulent strains of *S. aureus*.

Diagnosis

Staphylococcal colonies, like *Micrococcus* spp., have opaque pigments (from white to yellow). Clumping factor-positive strains are likely to be virulent, and require an aggressive therapy based on antibiotic sensitivities.

Serologic diagnostic techniques using agglutinins, antihemolysins or antitoxins are of little value in diagnosing staphylococcosis. Cross-agglutinins, particularly against *Salmonella gallinarum-pullorum*, frequently result in false-positive reactions.

Streptococcus (Sc.) and *Enterococcus (Ec.)*

Streptococci and enterococci consist of numerous species that readily grow on most commonly used media. Differentiation between the species is based upon morphologic, biochemical and serologic characteristics. These organisms are ubiquitous (mainly in dust and air), and some strains can survive for long periods in the environment. They are sensitive to most commonly used disinfectants.

Sc. and *Ec.* are considered part of the autochthonous flora of the skin and the mucosal surfaces of the digestive, respiratory and reproductive tracts. *Sc.* and *Ec.* transition from normal flora to disease-inducing agents depends on the functional state of host defense systems. Predisposing factors to disease include immunosuppression, concomitant infections and exposure to a variety of toxins and pathogenicity factors that may be produced by some strains of *Sc.* and *Ec.*

The ß-hemolyzing, pyogenic streptococci, frequently found in mammals, are rare in birds. In comparison, α-hemolyzing streptococci are quite common. It has been suggested that most of the latter species should be included into the newly established genus *Enterococcus*.[45,54] Enterococci are less fastidious than streptococci, and many will grow on selective media used for isolating Enterobacteriaceae. Numerous species

of streptococci and enterococci have been isolated from birds.[15,16,17,29]

References to the pathogenicity of several *Sc.* spp. vary. Variance in the observed behavior of these opportunistic organisms may be a result of the effects induced by concomitant viral or chlamydial infections, the lack of experimental infections and confusion in taxonomy or nomenclature. Irrespective of predisposing factors, many *Sc.* and *Ec.* strains produce compounds that facilitate an infection (M-protein, capsules) or produce extracellular substances that inhibit the host defense system (hemolysin S, streptolysin S, hyaluronic acid, streptokinase, DNase, NADase and esterases) or inhibit other competing bacteria (bacteriocins).

Vertical transmission (including L-forms) can be a cause of early embryonic death and post-hatching developmental problems (Figure 33.9). Infections in hatchlings are usually associated with omphalogenic postnatal septicemia.

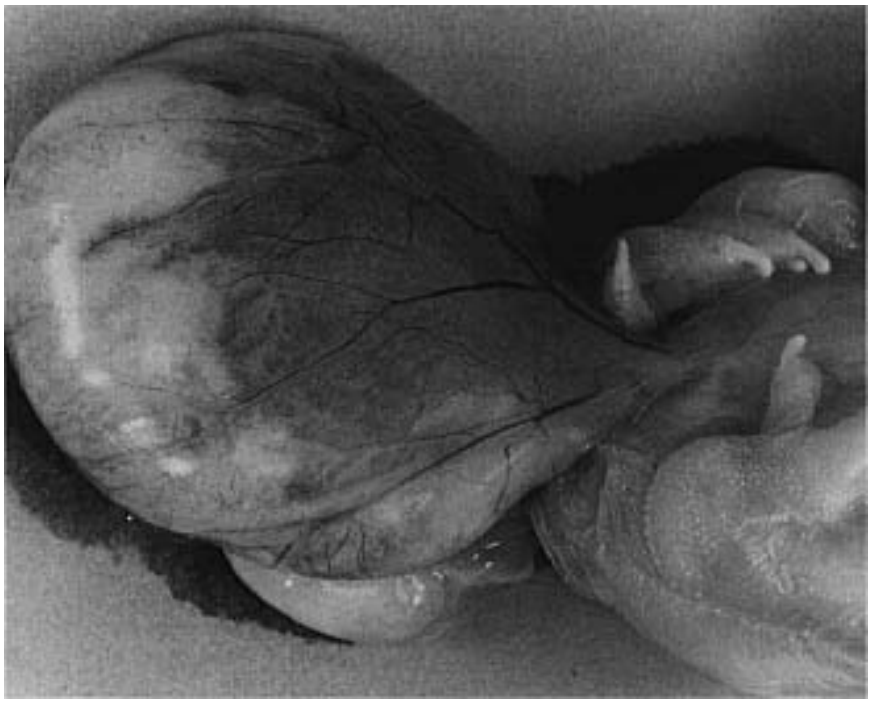


FIG 33.9 Bacterial infections (*Streptococcus*, *Staphylococcus*, *Enterococcus* and *Salmonella* spp.) of the egg can cause embryonal death, post-hatching developmental problems and yolk sacculitis.

Pathogenesis

The rarely occurring *Sc.* spp. group C can cause a septicemia followed by an embolic-thrombotic systemic disease and death, usually in adult birds. Following infection, hematogenic and intrahepatic spreading results in colonization in almost all organs if the host survives (probably agent- and host-specific). In experimental infections, a persistent bacteremia has been described, the duration of which can range from weeks to months. In addition to the pathogenicity factors already mentioned, *Sc.* group C may also produce reagins that govern the host reactions. Genetic and environmental factors as well as concurrent viral infections or immunosuppressions may predispose mainly adult birds to this rare infection.

Little information is available on the pathogenesis of *Sc. pneumoniae* or *Sc. pleomorphus* lesions in birds. *Sc. bovis* has been described as a pathogen in pigeons[17] and ducks;[52] however, the pathogenesis of these infections is unclear.

Enterococci outside of the digestive tract can cause necrotizing inflammatory lesions in infected organs. However, the pathogenicity of *Ec.* spp. is generally low. Experimental infections indicate that disease induction requires predisposing immunosuppressive factors. As a rule, natural infections occur by the oral route and are most common in postnatal and growing birds.

Ec. faecalis can induce an acute septicemic or a subacute-chronic disease. The acute form predominates in young birds, and survivors often develop endocarditis. Details on pathogenesis are known only in gallinaceous species. *Sc. faecalis* is thought to play a central role in the malabsorption syndrome described in chickens.

Egg transmission of *Ec. faecalis* (also as an L-form) is associated with an acute post-hatching septicemia. The subclinically infected yolk sac is thought to be the source of disease in the hatchling. *Ec. faecalis* is not considered autochthonous flora of canaries and may cause a primary respiratory disease in this species.[18] Subacute-to-chronic tracheitis with dyspnea and rales are considered natural components of the disease. *Ec. cecorum* has not been associated with disease in experimental infections.[14] *Ec. columbae* is mainly found in pigeons and constitutes a component of the autochthonous intestinal flora.

Clinical Disease

The clinical diseases caused by pyogenic streptococci and other streptococci and enterococci are relatively similar. Clinical presentation can be peracute to chronic, with birds surviving six to eight weeks in the chronic form. Omphalitis in recent hatchlings is typical with egg transmission or infections obtained from the hatchery.

An Amazon parrot with a slight nasal discharge was found to have a *Sc.* group G infection that was causing chronic respiratory disease in the children of the house. Presumably, the bird was infected by the children.

Septicemia may lead to a peracute apoplectiform death or severe depression followed by death in two to three days. Other signs such as diarrhea, dyspnea, paresis, conjunctivitis and sinusitis (Japanese Quail) may develop.

Chronic disease is typified by inflammation of joints, tendon sheaths and adnexa of the head. Fibrinous joint lesions, with or without abscess formation, can occur several months after the initial infection. Birds that survive systemic infections may develop cardiac valve insufficiency secondary to endocarditis. This condition is difficult to diagnose and often presents as chronic dyspnea.

Sc. pyogenes has been associated with bacteremia in the Humboldt Penguin, White Pelican, and several Psittaciformes, Anatiformes and Phasianiformes. It has not been clearly defined whether the bacteremia in these cases was a result of true group A streptococci, or whether the inciting strain was *Sc. pyogenes animalis* (classified in group C). Group C *Streptococcus* has been associated with pneumonia secondary to bacteremia in a variety of avian species. In the ostrich, *Sc.* group C cases are characterized by akinesia, anorexia and dysphagia. This organism together with *Corynebacterium pyogenes* can induce diphtheroid lesions in the mucosa of the beak cavity and the crop that can result in similar clinical signs.[32]

Sc. bovis infections in pigeons have been associated with clinical changes ranging from peracute death to chronic lameness (myositis) and arthritis.

Ec. group D is frequently implicated as a cause of pneumonia in various bird species, particularly Passeriformes, and primarily infects young birds. In canaries, *Ec. faecalis* can cause a tracheitis and chronic respiratory disease that manifests clinically as changes in the voice (more "sparrow-like") or complete voice loss. Tracheal mites (*Sternostoma tracheacolum*) can cause similar clinical signs in this species. Epidemiologically, the infection spreads slowly through the whole flock. Some affected birds develop dyspnea and die, while others may temporarily recover. Relapses are common in recovered birds.

Pathology

Gross lesions in birds that die acutely with bacteremia include subepicardial and myocardial hemorrhages and serofibrinous polyserositis. The subcutaneous tissues, serosal membranes and pericardium may be congested, and the spleen is frequently hyperplastic. Lung congestion, pneumonia or petechiation of the laryngeal and tracheal mucosa may be noted in some species. The liver may be slightly swollen and exhibit a greenish discoloration. Catarrhal enteritis, skeletal muscle hemorrhages and swollen kidneys may also be observed. Muscle necrosis and purulent myositis are frequently described in pigeons with *Sc. bovis* infections.

Pathologic changes in chronic cases are characterized by arthritis with a light, mucoid exudate and the formation of coagulated or dried exudates in the body cavity. Cauliflower-like or granulomatous atrioventricular inflammation may develop, particularly on the left cardiac valve. Pyogenic streptococci may cause chronic peritonitis, salpingitis and oophoritis.

Histologically, focal necrosis is common in the liver. This lesion is thought to be caused by bacterial endotoxins or the formation of thrombi in the bile ducts. Heterophilic infiltrates are most common in acute lesions. Granuloma formation (without a capsule and consisting of epithelioid cells and multinucleated giant cells), particularly in the spleen and heart, is more common in chronic cases. Purulent meningitis has been described in some cases. Endocarditis and cardiac infarct may occur secondary to embolic-thrombotic incidents. Localized lymph follicles may be described as hyperplastic.

Diagnosis

In acute cases, samples for culture taken from the liver, heart and brain are most diagnostic. Isolation from the brain indicates that the strain recovered is actively involved in the disease process. A triggering factor might have been necessary to enable the organism to cause septicemia. Special media are necessary for culturing L-forms.

FIG 33.10 White streaking is evident in the pectoral musculature of an Amazon parrot that was being treated with IM enrofloxacin.

Treatment and Control

Aggressive treatment with parenterally administered antibiotics is the recommended therapy. Pyogenic streptococci are generally sensitive to penicillins, erythromycin, tylosin, spectinomycin, clindamycin and pleuromutilin. Enterococci have varying antimicrobial sensitivities. Chronic joint and tendon sheath infections are difficult to resolve and may require a combination of surgery, joint lavage and prolonged antibiotic therapy. Joint lavage appears to be more successful in birds than in mammals because iatrogenic secondary infections are less likely. Streptococci in synovial membranes are frequently in their L- or protoplastic form. Treatment in these cases consists of ampicillin and erythromycin or enrofloxacin (Figure 33.10).

Mycobacterium (M.)

Colony morphology of pathogenic mycobacterium may be smooth or rough and change through successive *in vitro* subcultures. Most strains are nonphotochromogenic and may become yellow with age. Some strains are scotochromogenic and have bright yellow pigments.

All bird species that have been experimentally exposed have been found to be susceptible to *M. avium*. Lesions are typified by large numbers of bacteria in infected tissues. This is in contrast to *M. tuberculosis*, which is typically found in relatively small numbers in infected tissue. *M. avium* is highly resistant to environmental extremes and can survive in the cage or aviary environment for periods ranging from months to years. Shedding from an infected host occurs primarily in the feces and urine, causing contamination of the soil or water supplies within the aviary. Mycobacterium has been found to remain infectious in soil for up to seven years. For disinfection, only compounds tested against mycobacterium are recommended.

- ***M. avium and M. intracellulare:*** DNA cleavage techniques (restriction fragment length polymorphism and pulsed-field gel electrophoresis) have shown that *M. avium* consists of three clusters on the subspecies level as follows:[62]

M. avium subsp. avium: This subspecies is ubiquitous in the environment and is the agent most commonly associated with avian mycobacteriosis. The organism also has a widely documented mammalian host range including cattle, sheep, goats, pigs, cats, kangaroos and humans. In humans, adults typically develop respiratory infections, children usually develop submandibular adenopathies, and disseminated infections are common in patients with immunosuppressive diseases (eg, AIDS). Strains experimentally caused paratuberculosis in cattle and tuberculosis in fowl (Table 33.5).

M. avium subsp. paratuberculosis: This subspecies of *M. avium* requires mycobactin for *in vitro* culture and has not been isolated from the environment. This subspecies is considered to cause paratuberculosis in ruminants and has been implicated as a cause of Crohn's disease in humans.

M. avium subsp. silvaticum: Like *M. avium subsp. paratuberculosis*, these strains also require mycobactin for primary cultivation and have not been recovered from the environment. Most strains have been

isolated from Wood Pigeons and free-ranging ruminants. The agent causes paratuberculosis in mammals and mycobacteriosis as well as paratuberculosis in birds.

M. intracellulare is designated as a distinct species and is considered less pathogenic to birds than *M. avium* (whether justified or not). Morphologic, biochemical and serologic differentiation between *M. avium* and *M. intracellulare* are relatively difficult. These two species are routinely grouped together into the *M. avium-intracellulare* (MAI) complex. MAI complex strains are serologically distinct and have been divided into serovars.[64] *M. avium* is divided into serovars 1 to 11; *M. intracellulare* into three subspecies (subspecies 1: serovar 12 to 17, 19 to 28; subspecies 2: serovar 7; subspecies 3: serovar 18). Serovar 1 is most prevalent and pathogenic in the United States, while serovar 2 is most common in Europe. To date, serovar 3 has mainly been isolated in Europe.

TABLE 33.5 Zoonotic Potential of Bacteria From Companion Birds

Bacteria	Zoonotic Potential
Actinobacillus	None reported
Alcaligenes	None reported
Bordetella	None reported
Campylobacter	Undetermined, possible *C. laridis* – diarrhea in children
Clostridium	Negligible
E. coli	Possible Prevention – good hygiene
Erysipelothrix	Persistent dermatitis Avoid contact with infected birds
Haemophilus	None reported
Listeria	Conjunctivitis when infected from birds
Klebsiella	Theoretically possible None reported Humans – Friedländers pneumonia
Megabacterium	Avian-specific
Mycobacterium	Possible Immunosuppressed humans
Pasteurella	Rare human cases
Pseudomonas	Possible Prevention – good hygiene
Salmonella	Negligible (see text)
Staphylococcus aureus	Negligible Avian-adapted strains
Streptococcus/ Enterococcus	Negligible Birds may be infected by humans
Francisella tularensis	Possible/unlikely from birds
Vibrio	Mild enteritis
Yersinia pseudotuberculosis	High potential Transmission documented Humans – difficult to treat

Serovar 8 has been isolated worldwide and is probably the most frequent serovar reported. Birds living in aquatic environments are particularly susceptible to infections, which can be subclinical. Recently, serovars 25 and 27 have been isolated from sick birds. In addition, *M. avium* strains have been recovered that do not belong to serovars 1 to 28. These newly isolated strains vary serologically, have a broad host spectrum and are considered as virulent as serovar 2.[38]

Transmission

Avian mycobacteriosis primarily involves the alimentary tract. Transmission occurs mainly through contaminated feces, although aerogenic routes of transmission are possible. Arthropods can serve as mechanical vectors of *M. intracellulare* and *M. avium subsp. avium*. Egg transmission can occur but is epornitically unimportant because *M. avium* bacteremia causes an immediate cessation of egg production. Mycobacteria may persist in contaminated soil, litter and, less frequently, feed. Birds of prey can be secondarily infected while consuming infected quarry. The incidence of mycobacteriosis in free-ranging birds is estimated to be less than 1%.[59]

Pathogenesis

The main portal of entrance in birds is the intestinal tract, which typically results in a visceral infection. Initial colonization occurs in the intestinal wall. Subclinical bacteremia occurs early with subsequent spread to the liver through the portal circulation during the infectious process (relatively low numbers of organisms in the blood). The lack of lymph nodes allows unabated hematogenous spread within the host. The lungs can be secondarily infected during bacteremia. Avian mycobacteria are removed from the endothelium of the vessels by reticuloendothelial cells, mainly in the liver, spleen and bone marrow. Locally, avian mycobacteria induce cellular reactions governed by the cell-mediated immune system. In contrast to this typical *M. avium* infection, Columbiformes, Anseriformes and some weaver finches of the genus *Textor* develop lesions only within the lungs (Figure 33.11). An acute bacteremia is frequently observed in cranes (Gruiformes), the Hermit Ibis, Columbiformes and some Passeriformes. Tubercles that form at the site of infection in the intestinal wall commonly remain open to the intestinal lumen. This allows for constant shedding of *M. avium* into the feces (open infection). In birds, three different types of lesions can be recognized although the pathogenesis has not been clarified: 1) classical form with tubercules in many organs; 2) paratuberculous form with typical lesions in the intestinal tract (prone to

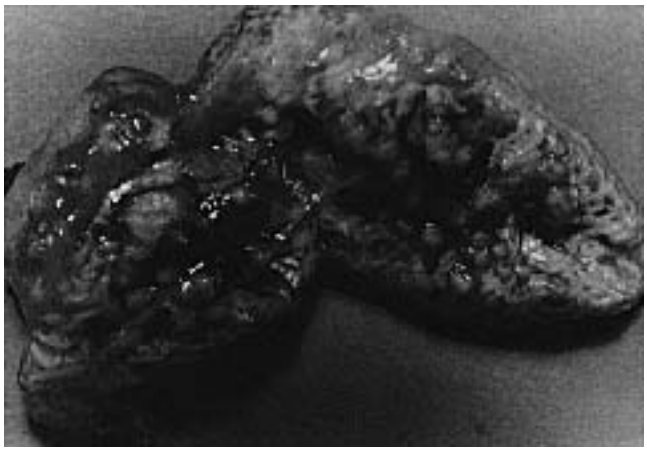


FIG 33.11 In Psittaciformes, mycobacteriotic lesions are generally not limited to the intestinal tract. In Columbiformes and some other species, atypical granulomas may form in the lungs. *Mycobacterium avium* was recovered from the lungs of this pigeon that was presented for severe emaciation and severe dyspnea. Radiographs indicated soft tissue masses in the lungs. Abnormal clinical pathology lesions included WBC=54,000 and PCV=19.

develop this form of myobacteriosis are *Amazona, Pionus, Brotogeris, Psittacula* species and the Horned Parakeet); 3) non-tuberculous form, which may be difficult to recognize at necropsy (many Psittaciformes).

Clinical Disease

In some bird species the clinical course is atypical, and acid-fast rods have been detected more or less accidentally. This is particularly the case with small Passeriformes, especially the Hooded Siskin.[19] Clinical signs associated with mycobacteriosis are highly variable. Adult birds usually develop a chronic wasting disease associated with a good appetite, recurrent diarrhea, polyuria, anemia and dull plumage. Immature individuals frequently develop subclinical conditions. Intermittent switching lameness may occur as a result of painful lesions in the bone marrow. Arthritis, mainly of the carpometacarpal and the elbow joints or tubercle formation of the muscles of the thigh or shank can be seen occasionally. These clinical changes are particularly common in Falconiformes and Accipitriformes. Skin over the affected joint is often thickened and ulcerated. Tubercle formation in the skin is rare, but when it is present, pinpoint to pigeon egg-sized nodules filled with yellow fibrinous material may be noted. Granulomas may be seen within the conjunctival sac, at the angle of the beak, around the external auditory canal and in the oropharynx. Mycobacteriosis should be suspected when tumor-like lesions recur after surgery. Greater Rhea frequently develop granulomas in the upper pharynx. In the Goshawk, loss of balance, convulsions and necrosis of the base of the tongue have been observed.[43] Clinical signs associated with colonization of the lung are rare. However, the peafowl may develop respiratory sounds caused by granuloma formation within the trachea.

Pathology

The pathology associated with *M. avium* infections varies widely, probably based on the species of bird infected and the serovar of the bacterium. Specific relationships between avian hosts and individual serovars have not been defined. The presence of miliary to greater-than-pea-sized nodules in the wall of the intestinal tract and in the liver, spleen and bone marrow are characteristic of *M. avium* infections. These typical lesions have been described in Falconiformes, Accipitriformes, Strigiformes, Phasianiformes, Charadriiformes, Ciconiiformes, Cuculiformes, Piciformes and Ralliformes. Granuloma formation can occur in any organ but is generally localized to the intestinal tract and reticuloendothelial organs. The nodules are frequently necrotic in the center and in chronic cases may be calcified. In contrast, Columbiformes, Anatiformes, Passeriformes and most of the Psittaciformes do not form typical granulomas. Acid-fast rods are found distributed throughout the parenchyma of infected organs. An infected liver or spleen may be only swollen or may show necrotic foci or even general induration. In pigeons, liver lesions may resemble *Trichomonas* abscesses. In pelicans, greasy tumor-like swellings like those seen in leukosis may be observed. The lungs, particularly of geese, weaver finches (genera *Queleopsis*, *Quelea* and *Euplectes*) and some *Amazona* spp., develop necrotizing or ulcerating lesions. Paratuberculous lesions are characterized by the occurrence of clubbed villa containing acid-fast rods in the intestinal mucosa. Lesions may also occur in glands of Lieberkühn, with characteristic proliferation of their epithelial cells.

Histopathologic identification of foci of single or confluent epithelioid cells in affected organs is suggestive of an *M. avium* infection. Parenchymal lesions generally consist of epithelioid cells or multinucleated giant cells (mostly the foreign body type, only rarely the Langhans type) and occasionally contain lymphocytes and plasma cells. Acid-fast rods in varying quantities can be demonstrated in the affected epithelioid or multinucleated giant cells. Acid-fast rods may also be noted in tissues in the absence of cellular reactions. Some infected birds will have cell-free acid-fast rods in the proventriculus or jejunal villi without an inflammatory response. It has been

postulated that the acid-fast rods may actually be contained within lymphatic vessels in the villi. *M. avium* infections frequently induce depletion of lymphocytes in the spleen (particularly the white pulp) and a proliferation of macrophage or reticular cell types in the same tissue. Depletion of the splenic lymphocytes and lymph follicles may induce an immunosuppression.

Diagnosis

The demonstration of acid-fast rods in tissues or on cytologic preparations is suggestive of mycobacteriosis. False-negative staining can occur by not obtaining an adequate sample. The demonstration of acid-fast rods in the feces has been suggested as a useful diagnostic tool in subclinical birds. Mucus present in the feces can interfere with test results, and samples should be processed with one of the sputum solvents used in human medicine before staining. The most consistent results can be obtained by centrifuging the feces and then spreading the surface of the pellet on a slide for staining. This test is relatively insensitive and requires the presence of approximately 10^4 bacteria/g of feces to be positive. The clinician must differentiate between pathogenic and nonpathogenic strains of mycobacteria, both of which may be present in the feces. In general, nonpathogenic strains are wider and are not granular. Demonstrating acid-fast organisms in the stool is not diagnostic for a mycobacterial-induced disease.

Culture is required to make a distinct diagnosis. Some strains of *M. avium-intracellulare* require mycobactin and will not grow on egg medium. *M. avium* subsp. *paratuberculosis* and subsp. *silvaticum* can be separated by responses to six parameters: subsp. *paratuberculosis* has a mycobactin requirement, grows on egg medium, tolerates cycloserine (50 μg/ml), is stimulated by pyruvate but not by pH 5.5, and has no alkaline phosphatase. The subsp. *silvaticum* has the opposite characteristics.[62]

Unfortunately, most of the mycobacterial strains from Psittaciformes have not been cultured. The future availability of species-specific antibodies will help in delineating infections. Endoscopy (with biopsies) can be used for diagnosis in cases of advanced classical tuberculosis. Radiographs may indicate granulomas in respiratory tissues in some cases. Biopsy is required to differentiate between mycobacterial and fungal granulomas, which radiographically appear similar.

Several indirect tests have been discussed for diagnosing *Mycobacterium*. The tuberculin test (allergenic test) and the slide agglutination test (serologic test) have both been used in birds with some success. The tuberculin test is frequently associated with false-negative results, particularly in early and late stages of the disease and is no longer recommended. The slide agglutination test requires fresh plasma or serum and is evaluated against a bank of antigens for the different serovars; there are cross-reactions between the different serovars. Unfortunately, only serovar 2 antigen is commercially available. To estimate the probability of an acute disease process, serotitration (using the Gruber-Widal scheme) is possible. Only titers greater than 1:64 are considered positive. Psittaciformes may exhibit a cyclic reduction in titer and mycobacterial excretion, which may lead to an incorrect suspicion that natural healing or a successful therapy has occurred. An ELISA system has been tested to distinguish between *M. avium* serovars but has been hampered by a high degree of cross-reactivity.

Treatment and Control

Several treatment modalities have been discussed for birds with *M. avium* infections. However, treating infected birds is not recommended because:

- All *M. avium* isolates that have been tested are totally resistant to the antituberculous drugs routinely used in humans. Recent information revealed that ethambutol, while ineffective, does change the cellular wall of *M. avium* in a manner that allows other tuberculostatics to enter the organism.[21] However, successful therapy is then dependent on pharmacokinetic conditions and requires a combination of drugs to be available at the same time, at the correct concentration and at the correct anatomic location. The pharmacokinetic data necessary to ensure that these parameters are met are not available for a single avian species.
- *M. avium* infections are considered to be "open," allowing infected birds to continuously shed large numbers of organisms into the environment.
- There is potential danger to man, and there is no appropriate method of treatment for infected humans.

Birds that are definitively diagnosed (biopsy of affected tissue with histopathology and culture) with *M. avium* or *M. intracellulare* infection should be euthanatized. Contact birds should be removed from the contaminated area, quarantined for two years and tested every six to 12 weeks to determine if they

are reacting. Birds that remain negative (also not shedding the agent with the feces) and are in good physical condition following the quarantine procedure can be considered free of the disease. There are currently no absolute means of control. The chances of introducing *M. avium*-positive birds to the flock can be reduced by performing serologic or repeated fecal examinations of all new additions during the quarantine period.

The transmission of *M. avium* to humans is possible. However, transmission is probably dependent on inherent resistance, the immune status of the person in question, the frequency of exposure and the number of bacteria per exposure.

- ***M. tuberculosis:*** *M. tuberculosis* lesions in Psittaciformes are possible but are extremely rare. When present, they are generally characterized by the formation of benign localized granulomas of the dermis, frequently around the cere or nares as well as the retroorbital tissue.[63] Birds affected are usually pets with very close human contact and, as such, serve as sentinels for patent tuberculosis in the owners. The affected dermis looks granulomatous and may even ulcerate. The swelling of the retrobulbar tissue causes a protrusion of the eye (exophthalmos). Diagnosis can be made by histology or culture of biopsies. Infected birds should be euthanatized.
- ***M. bovis:*** *M. bovis* was associated with a generalized infection in an Amazon parrot. This animal developed a nontubercular lesion similar to those seen in Psittaciformes infected with *M. avium intracellulare*. The strain in question was sensitive to all common tuberculostatic drugs.

Erysipelothrix (E.)

Erysipelothrix rhusiopathiae can induce an acute-to-subacute septicemic disease. Infections are most commonly discussed in ducks and geese but can occasionally occur in other avian species including Psittaciformes.[55,32] The eleven serotypes of *E. rhusiopathiae* are ubiquitous in the environment and can propagate outside the host. Survivability in moist soil and in the water of shallow lakes and ponds, even salt and seawater, is particularly high. Infections in birds occur most frequently in the late fall, winter and early spring (northern hemisphere). Rodents, pigs and raw fish have all been implicated as reservoirs for the bacterium.

Pathogenesis

E. rhusiopathiae infections are most common in waterfowl and fish-eating birds during cold weather when food is scarce and energy requirements are high. Concomitant infections and inadequate hygiene seem to be precipitating factors of natural diseases. Most affected birds die during the bacteremic phase of an infection. Those birds that survive the acute disease frequently have secondary dermatitis and arthritis caused by hypersensitivity reactions.

Clinical Disease and Pathology

E. rhusiopathiae usually causes peracute death. If clinical signs occur, they may include lethargy, weakness, anorexia and hyperemia or bruising of the featherless, nonpigmented skin. Greenish discolored droppings, dyspnea and nasal discharge have been reported in some cases. In the Marabou Stork, infections have been characterized by inflammation and necrosis of the cutaneous adnexa of the neck.

Petechiae in the subcutis, musculature and intestinal mucosa are common gross lesions in diseased birds. The liver and spleen are friable and discolored (red to black). Histologically, these organs are degenerated and necrotic. Chronic *E. rhusiopathiae* cases are rare but have been reported in geese and turkeys. Clinical changes associated with this form of disease include thickened, leather-like skin, serofibrinous arthritis or valvular endocarditis. Histologic changes in these tissues include thrombi and degeneration of vascular walls.

Diagnosis

Diagnosis is confirmed by isolating *E. rhusiopathiae*. The best samples for isolation (if the tissues are fresh) are liver or spleen. In severely autolytic cases, bone marrow samples may be the most diagnostic. As in most bacterial diseases, cell-mediated immunity is more important in resolving infections than the development of humoral antibodies. Thus, serologic methods of diagnosis are of little value.

E. rhusiopathiae adjuvanted bacterins have been used to control flock outbreaks; however, vaccination may sensitize the birds and potentiate chronic disease. Flock control can best be implemented through sound aviary hygiene and rodent control.

Listeria (L.)

Listeria monocytogenes causes β-hemolysis on blood agar. The motility of listeria is dependent on the ambient temperature to which it is exposed. *Listeria*

spp. may infect a number of avian species, including Psittaciformes.[32] Canaries appear to be more susceptible to infections than other birds.

L. monocytogenes is a ubiquitous organism that is frequently found in areas. The bacteria is environmentally stable and can propagate outside the host.[56]

Pathogenesis

The role of *L. monocytogenes* as a primary pathogen is controversial; however, there is no question that ingested bacteria can lead to a latent or abortive infection. Acute disease is characterized by bacteremia progressing to death within one to two days. The subacute and chronic forms of the disease involve reactions of the cell-mediated immune system. Intracellular bacteria like *L. monocytogenes* typically induce cell-mediated immunity.

FIG 33.12 Encephalitis caused by several bacterial pathogens including *E. coli, Listeria, Salmonella, Staphylococcus* and *Klebsiella* spp. can cause neurologic signs characterized by torticollis, tremor, ataxia, depression, paresis or paralysis.

Clinical Disease and Pathology

Clinical disease is usually associated with sporadic deaths in a collection. Epornitics can develop in canaries and related birds that are maintained in dense populations. Chronic infections can induce lesions in the heart, liver and, rarely, the brain. If clinical signs are noted, they are generally associated with CNS signs and include blindness, torticollis, tremor, stupor and paresis or paralysis (Figure 33.12).[41] Subacute-to-chronic cases usually cause a severe monocytosis (10 to 12 times normal).

The presence of serofibrinous pericarditis and myocardial necrosis is considered suggestive of *Listeria*. There are usually no gross lesions evident in the brain. There may be no lesions in birds that die acutely, or there can be a few petechiae present.

Histologically, infections are characterized by degenerative lesions, without a cellular response, in the heart and liver. Brain tissue is usually normal, even in cases in which CNS signs are present and the organism can be recovered from the cerebrum.

Diagnosis

A confirmed diagnosis requires the isolation of *L. monocytogenes* from affected tissues. Appropriate transport media are necessary for proper shipment of samples. Listeria isolates must be speciated, because *L. ivanovii*, *L. innocua* and *L. seeligeri* are commonly recovered from birds.[8] There is little information on the pathogenicity of these *Listeria* spp. although *L. innocua* is considered apathogenic.[37] Latent infections limit the diagnostic value of serologic tests (slide agglutination).

Clostridium (Cl.)

The genus *Clostridium* includes a group of ubiquitous bacteria that are considered to be autochthonous flora in raptors and in birds with well developed ceca, including Phasianiformes (gallinaceous birds) and Anseriformes. In birds in which the cecum is small or absent, clostridium is rarely isolated from the intestinal tract and when it is, it is often considered to be transitory.

Pathogenesis

Clostridium spp. produce more potent toxins than any other bacterial genus. The pathogenesis of *Cl.* spp. toxin-induced damage in a bird remains poorly understood except for *Cl. botulinum*. This *Cl.* sp. principally causes an alimentary intoxication. The ability of clostridia to colonize the intestines appears to depend on reduced gastrointestinal motility. Peristaltic abnormalities can occur as a result of enteritis, low levels of dietary fiber, administration of some

medications, or during some viral infections (particularly reovirus). Experimental reproduction of disease by using a culture alone is usually not possible, and clostridial organisms are considered to be opportunistic pathogens. Following colonization, pathogenic clostridia produce exotoxins, which then induce clinical lesions or death.

It is best to discuss clostridial infections by grouping them under clinical signs because a clostridial species can cause differing clinical signs, and various clostridial species can cause similar-appearing diseases.

Necrotic or Ulcerative Enteritis

Clostridia-induced enteritis can occur in many avian species. Flock outbreaks are most commonly associated with Phasianiformes, especially those within the subfamilies Tetraoninae (grouse) and Odontophorinae (New World quail) or captive and free-ranging lorikeets.[44] Ulcerative gastritis, not enteritis, is the most common form of clostridial infection reported in the ostrich.[60] Clostridial enteritis in game birds (Tetraoninae) is usually associated with *Cl. perfringens* type A. On rare occasions, types B, C or D may be the etiologic agent. *Cl. perfringens* type E is vary rare in Phasianiformes. Ulcerative enteritis in the Bobwhite Quail is usually caused by *Cl. colinum* (species *incertae sedis*). This organism is also thought to cause necrotic enteritis in the other Odontophorinae, grouse, chicken, turkey and domesticated pigeon (Köhler, personal communication).

Necrotic enteritis usually occurs in young birds after the second post-hatching week. Adult birds are more resistant. In the acute form of the disease, clinical changes include diarrhea (with or without blood) and polydipsia, followed by death within a few hours. Birds with chronic lesions exhibit retarded growth and weight loss before dying.

Pathologic changes include diffuse or focal hyperemia of the mucosa, which develops into necrotic areas or ulcers. These lesions are most common in the upper jejunum. Lesions start as pinpoint foci and progress to include a necrotic center with a wall and a reddish halo. Ulcers may coalesce and perforate the intestinal wall. Swelling and necrosis of a grayish liver, spleen and kidney are common.

Identification of characteristic lesions and isolation of the organism are confirmatory. If *Cl. perfringens* is the etiologic agent, a diagnosis can be achieved by demonstrating toxins in the serum, intestinal contents and liver homogenates (sterilized by filtration). The differential diagnosis includes salmonellosis, enterotoxic *E. coli* and drug overdoses. Newcastle disease virus can induce ulcers called "boutons" that resemble those induced by *Clostridium*.

Gangrenous Dermatitis

Gangrenous dermatitis can be caused by *Cl. perfringens* type A, *Cl. septicum*, or *Cl. novyi*. These organisms can directly colonize damaged skin. Microscopic epithelial lesions caused by abrasions, avipoxvirus or staphylococci can become secondarily infected with *Clostridium* spp. The patient's immune status may also play a role in the overall pathogenesis.

The sudden occurrence of regional feather loss with a blue-red or almost black skin discoloration is a characteristic lesion. Affected skin may also be edematous and painful as a result of gas accumulation in the tissue. Sick animals typically develop toxemia and die within 24 hours. With screamers (genus *Chauna*), their corneous-lined bony wing spurs can cause prick-like skin injuries predisposing them to clostridial diseases.

The occurrence of emphysema, edema and hemorrhages (with or without necrosis) in the subcutis, skeletal musculature and myocardium are characteristic necropsy findings. A confirmed diagnosis requires isolation of *Cl.* spp. from affected tissues. *Aeromonas hydrophila*, staphylococci and avipoxvirus can induce pathologic changes similar to those caused by *Cl.* spp. Rapid production and systemic release of toxins usually prevents successful therapy.

Botulism (syn. Limberneck)

Cl. botulinum neurotoxins are typically ingested in contaminated foods. Rarely, clinical disease may result from primary colonization of the alimentary tract. *Cl. botulinum* has been found to produce six thermolabile exotoxins (designated A to F). Types A and C are predominantly involved in inducing pathologic changes (occasionally also type E). High concentrations of clostridium toxins are common in decaying meat and vegetation. Fly larvae (maggots) that feed on decaying material are resistant to the toxins but can serve as a source of intoxication for those species that eat maggots. With a few logical exceptions, most birds are probably susceptible to *Cl. botulinum* toxins. Free-ranging waterfowl are particularly susceptible, and enzootic intoxications are common following periods of drought or flooding. Vultures seem to be resistant to the toxins (by a still

unknown mechanism), and some raptors (that eat carrion) have a reduced susceptibility.

Foods contaminated with *Cl. botulinum* toxins have no particular change in taste or smell and are thus difficult to detect. Toxins enter the body through the intestinal wall, move to the ends of the autonomic and somatic efferent nerves and then ascend into the spinal cord. Once in the spinal cord, toxins selectively and irreversibly bind to the neuromuscular junctions and block the production of acetylcholine. Death is due to respiratory paralysis. Toxins also damage vascular endothelium, resulting in edema and petechiation.

Flaccid paralysis of the skeletal musculature (including the tongue) is the characteristic clinical change. Bulbar paralysis, feather loss and diarrhea may be noted in some birds. Birds with substantial clinical signs usually die, although a few can spontaneously recover. Recovery is more likely with type A rather than type C toxin. Petechial hemorrhage of the cerebellum and focal necrosis and hemorrhage of the central lobe of the cerebrum are indicative of *Cl. botulinum*, but there are no pathognomonic lesions.

Confirming a diagnosis requires using mouse animal models to demonstrate the presence of toxins in serum, filtered liver or kidney from an affected animal. Feed or water (sewage, mud) extracts from a pond also provide diagnostic samples. *Cl. botulinum* toxins are heat-sensitive and degrade at room temperature. Materials to be tested should be preserved by deep-freezing (-20°C).

Intoxication with some algae, eg, red tide (see Chapter 46), may cause clinical signs similar to those caused by *Cl. botulinum*. Affected birds usually have access to muddy, drying bodies of water. *Aeromonas hydrophila* can cause the acute death of large numbers of waterfowl that consume contaminated water. A rapid death, typically in the summer, is characteristic, but some infected birds can develop signs that mimic those of *Cl.* sp. toxin ingestion. *A. hydrophila* and *Cl. botulinum* may both cause epornitics of acute death in connection with contaminated water.

Flock problems can be prevented by reducing contact with decaying foods, removing cadavers, providing toxin-free feed (especially for insect eaters) and regulating the water level and temperature in waterfowl collections (see Chapter 46). A commercial type C antitoxin is available for mink and has proven to be effective in birds. One-half of the dose recommended for mink should be used in birds.

Tetanus

A few cases of *Cl. tetani* intoxication in birds have been reported in older literature. There have been no cases of tetanus reported in birds using more confirmatory diagnostic tests. Generally, birds are considered to be highly resistant to *Cl. tetani*; cases reported in older literature may have been incorrectly diagnosed.

Other Gram-positive Rods

Bacillus spp., *Corynebacterium* spp., *Streptomyces* spp. and *Lactobacillus* spp. are commonly recovered from avian-derived samples. Because these organisms are frequently isolated from clinically normal birds, they are considered to be components of the autochthonous flora. Megabacterium is considered to be a pathogenic organism, although little information is currently available on its involvement in avian disease. *Bacillus* spp. isolated from birds are difficult to differentiate, and most have not been described taxonomically. *Bacillus anthracis* has not been associated with clinical disease in birds, presumably because their high body temperature inhibits the production of the pathogenic toxins. Vultures, and to a lesser extent raptors, are known to be mechanical vectors.

None of the *Corynebacterium* (*Co.*) spp. that have been isolated from birds have been found to be pathogenic. Ostriches have been reported to have concomitant *Co. pyogenes* and *Sc.* group C infections.[32] Antibodies to *Corynebacterium* spp. and *Mycobacterium avium-intracellulare* can cross-react, making differentiation between these organisms difficult.

Streptomyces spp. can occasionally be isolated from the avian respiratory system. The pathogenicity of the organism is questionable. Some reports describe nocardia in birds. These cases were described based on bacterial morphology and not on analysis of wall components, which is necessary to confirm nocardia in infected tissues.

Lactobacillus spp. can be identified on the mucosa of the intestinal, respiratory and reproductive tracts of many avian species. In birds that do not possess Enterobacteriaceae as a normal component of the gut flora, lactobacillus seems to play an important role in inhibiting colonization of Enterobacteriaceae (although probably not in many species of grouse). Supplementing with lactobacillus has been discussed as a method of inducing a natural competitive inhibition

TABLE 33.6 Survey of Clinically Important Bacteria

Bacteria	Characteristics	Incubation	Transmission	Disinfectants/Stability
Alcaligenes/ Bordetella	Motile 0.3-0.5 x 1-2 μm	Turkeys – 5-7 days Others – unknown	Alc. ingestion Shed in feces Egg transmission Bord. respiratory tract, feces Egg transmission	Resistant to drying Environmentally stable (Particularly low temps)
Campylobacter	Gram-negative Motile rod 0.2-0.5 x 1.5-5 μm	5 to 15 days	Ingestion Feces	Survives 1 week out of host Most disinfectants effective
Clostridium	Gram-negative Anaerobic Spore-forming rod	Experimental – hrs to 3 days Natural – unknown	Ingestion Wound infection	Resistant in environment Spores – survive years Most disinfectants ineffective Autoclaving/flaming effective
Erysipelothrix	Gram-positive Nonmotile rods 0.2-0.4 x 1-2.5 μm	Turkeys, ducks – 1.8 days Other birds – unknown	Ingestion Egg transmission (stork)	Most disinfectants effective
E. coli	Gram-negative 0.5 x 3 μm Motile or nonmotile rod	Experimental – 24 to 48 hrs	Ingestion – mainly Aerogenic – dust Egg transmission (normal & L-forms)	Most disinfectants effective
Haemophilus	Gram-negative 0.3-0.6 x 1-3 μm Coccoid-to-pleomorphic rods	*H. paragallinarum* 1-5 days	Respiratory tract Asymptomatic carriers	Survives a few days Most disinfectants effective
Listeria	Gram-positive 0.4 x 0.5-2 μm Motile rod	Unknown	Ingestion Common in aquatic environments Reptiles, amphibians, snails natural reservoirs	Most disinfectants effective
Mycobacterium	Gram-positive Acid-fast Granulated short-to-long rods	Weeks to years Exposure and immune system dictate	Feces, urine Aerogenic possible (rare) Arthropods-mechanical vectors	Persists in contaminated soil or litter for years
Pasteurella	Gram-negative Nonmotile Ovoid-to-coccoid rod	Vary with species Cat bite – 6-48 hrs	Upper respiratory tract Fecal shedding rare See text	Not stable to stable depending on conditions Most disinfectants effective
Pseudomonas Aeromonas	Gram-negative 1-3 x 0.3-0.6 μm	Specifics unknown Pseud — few hrs Aero — 1-3 days	Ingestion Proliferates in H_20 Secondary in open wounds	Pseudo: resistant to most disinfectants – steam, boiling H_20 effective Aero: most disinfectants effective
Salmonella	Gram-negative 0.5 x 3 μm Motile, rarely nonmotile rod	Varies with type Acute 3-5 days Egg — 2 days	Ingestion Egg transmission Normal and L-forms	Dried feces stable – 8 months to 2 years Water stable – 3 weeks Most disinfectants effective >60°C kills most strains
Staphylococcus	Gram-positive 0.8 x 1.0 μm Nonmotile Spherical coccoid	Unknown Few hrs to few months Latent infections	Vertical, horizontal Normal & L-forms Chronic carriers	Environmentally stable Most disinfectants ineffective
Streptococcus Enterococcus	Gram-positive <2 μm Coccoid-to-ovoid pairs or chains	Mammals – days to weeks Birds – unknown	Horizontal, vertical Shed in feces Aerogenic Percutaneous (skin lesion)	Probably no growth in environment Sc spp – short-lived Ec spp – long-lived
Yersinia	0.4 - 0.8 x 0.8 - 1 μm Motile or nonmotile Ovoid-to-coccoid rods	Days to 2-3 weeks	Ingestion Carrier birds & rodents Direct contact Fecal contaminated food or water	Most disinfectants effective

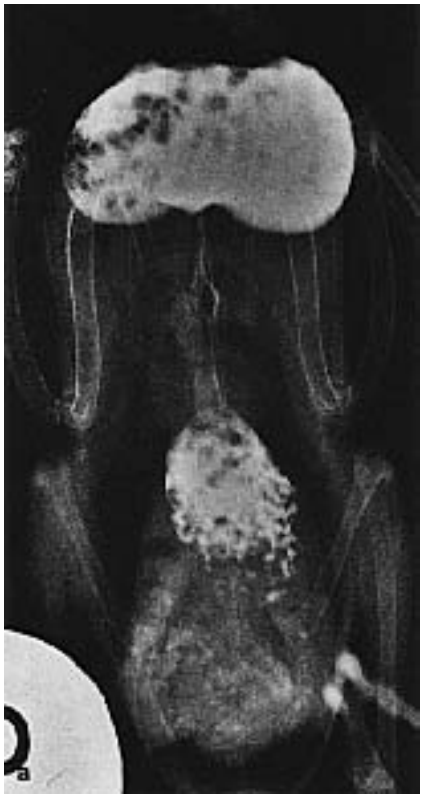
TABLE 33.7 Bacterial Control and Therapeutics

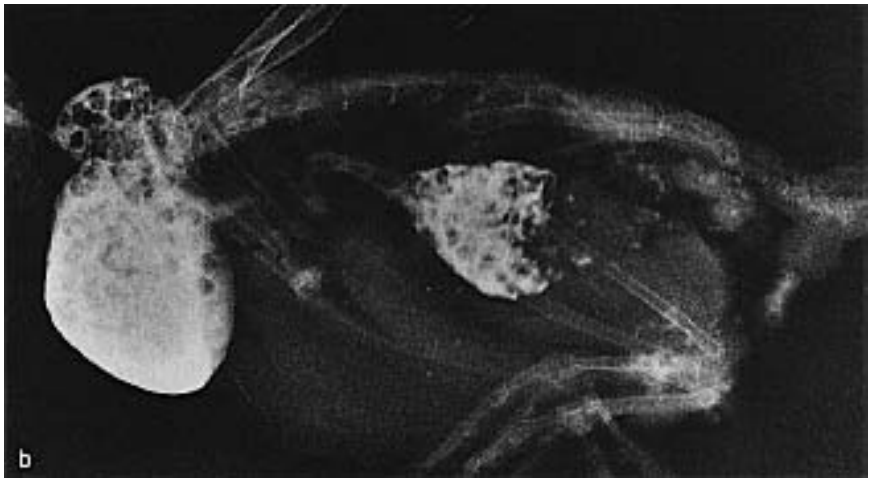
Bacteria	Treatment/Control
Actinobacillus	Tetracyclines/chloramphenicol See text
Aeromonas	Most antibiotics See text
Alcaligenes/Bordetella	Antibiotic resistance common Rx based on sensitivities
Borrelia	Tylosin/Spectinomycin Control ticks Strain-specific vaccines
Campylobacter	Erythromycin/tetracyclines Streptomycin, non-psittacines Furane derivatives in non-waterfowl (See text)
Clostridium perfringens	Clindamycin/spiramycin Laxatives – Remove unabsorbed toxins Wound irrigation Control – Vaccination (toxoid) Zinc bacitracin (200 ppm) (Bobwhite Quail in food) Prevent skin wounds
Clostridium botulinum	Toxoid vaccine, antitoxin Laxatives – remove unabsorbed toxins Guanidine – (15 to 30 mg/kg) May bind neurotoxin Prevent food contamination Activated charcoal See text
Cytophaga	Parenteral antibiotics Treat soon after hatching Bacterins provide sero-specific protection
Escherichia coli	Rx based on sensitivity See text
Erysipelothrix	Parenteral penicillin/doxycycline Surgically remove affected skin Hyperimmune serum See text
Haemophilus	Most antibiotics Sulfonamides – drug of choice Sinusitis – surgical drainage Flushing Topical vitamin A Asymptomatic carriers Detect by culturing sinuses Polyvalent vaccine – poor efficacy
Klebsiella	Resistant to many antibiotics Rx based on sensitivity Enrofloxacin initially
Listeria	Tetracyclines CNS signs – poor prognosis Hygiene critical
Megabacterium	No effective therapy Resistant to all tested antibiotics Control – unknown Acidifying water with HCl
Mycobacterium	Not recommended See text
Pasteurella	Parenteral antibiotics See text Control – Prevent rodents/free-ranging birds Vaccines – poor efficacy
Pseudomonas	Resistant to many antibiotics Rx based on sensitivity Enrofloxacin initially Systemic infections usually fatal See text
Salmonella	See text
Staphyloccocus	Resistant to many antibiotics Semisynthetic penicillins pending sensitivity Bumblefoot – Debride, antibiotics Heparin – prevents fibrin
Streptococcus Enterococcus	Parenteral antibiotics See text Control – hygiene Stimulates minimal immune response
Yersinia	See text

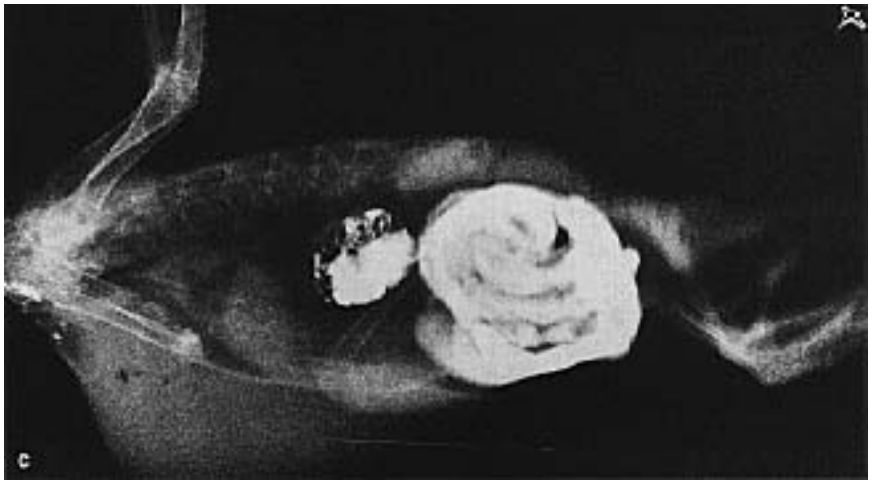
Treatment of all bacterial infections should be based on specific antibiotic sensitivity. Supportive care in the form of fluids and enteral feeding is frequently necessary. Correcting predisposing factors that cause immunosuppression in the host or allow exposure to an organism are critical control methods. Preventing exposure to rodents, insects and free-ranging birds can reduce bacterial exposure. Hygiene is always important, particularly in neonates, to prevent reinfection.

of gram-negative and other pathogenic bacteria. However, there are strong indications that many groups of birds have specific lactobacilli that can effectively colonize the gut. Strains derived from soured milk do not colonize the avian gut and must be given daily for two to four weeks in order to lower the gastrointestinal tract pH. This drop in pH will favor the colonization of autochthonous microorganisms. Inhibitory expulsion itself takes from four to six weeks, provided no serious triggering factors interfere.[23]

Isolation of megabacteria is difficult, and biochemical descriptions that would allow appropriate taxonomic classification have not been performed. This organism has a unique morphology and is a large (1 x 90 μm) gram-positive rod.[13] Successful culture requires the use of Merck's MRS medium; however, not all megabacterial strains have been found to grow on this medium. The colonies are rough and measure 3 to 4 millimeters in diameter with a dented margin. Development requires 48 hours in a moist chamber. Subcultures are progressively more difficult and the organism may stop growing in successive passages.[28]







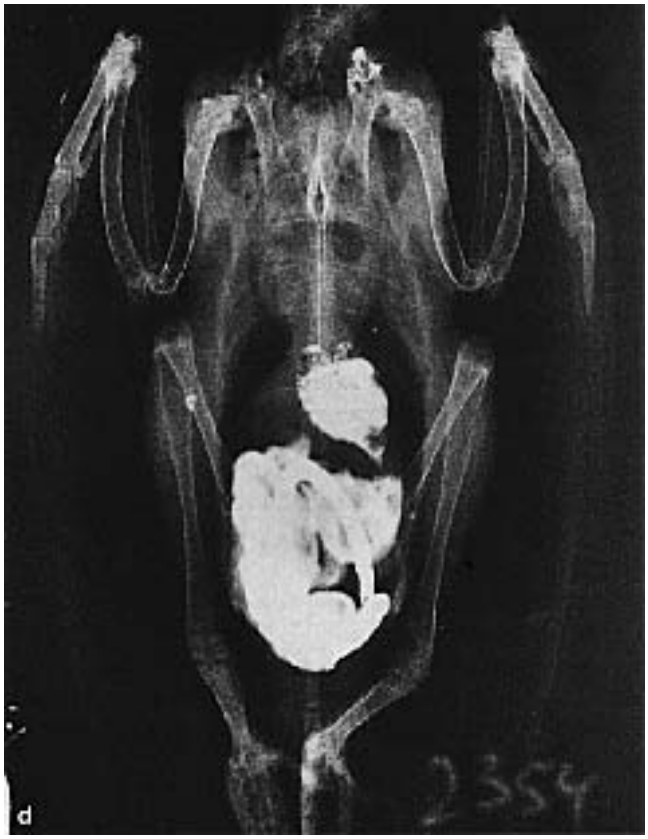


FIG 33.13 A group of budgerigars was presented for occasional melena and chronic weight loss over a prolonged period. Large (1 x 90 μm) gram-positive rods (suggestive of megabacteria) could be detected in the feces. Radiographs in various affected birds indicated **a,b)** proventricular dilatation and **c,d)** filling defects and ulceration in the proventriculus. At necropsy, the filling defects were found to be globules of mucin that had a propensity to accumulate at the isthmus. It should be noted that the presence of a dilated proventriculus is not diagnostic for neuropathic gastric dilatation (courtesy of Nina Ungerechts).

TABLE 33.8 Differential Diagnoses of Bacterial Infections

Arthritis or Synovitis
- Staphyloccoccus
- *Actinobacillus* sp.
- *E. coli*
- *E. rhusopathiae* (ducks and goose)
- *Mycobacterium avium*
- *Mycoplasma* spp.
- *Pasteurella multocida*
- *Salmonella* spp.

CNS Signs
- Listeriosis
- Chlamydiosis
- *E. coli*
- *Klebsiella pneumoniae*
- *Listeria monocytogenes*
- *Salmonella* spp.

Dermatitis
- *Pseudomonas/Aeromonas* spp.
- *Clostridium* spp.
- *Staphylococcus* spp.

Enteritis
- *E. coli*
- *Aeromonas* sp.
- Most enteric organisms
- *Pseudomonas* spp.
- *Salmonella* spp.
- *E. rhusiopathiae*
- Chlamydiosis
- *Listeria* sp.
- *Pasteurella* spp.

Hepatitis
- Most bacteria that cause septicemia
- Campylobacter
- *Pasteurella* spp.
- Chlamydiosis
- *Salmonella* spp.

Pseudomonas enteritis
- *Clostridium* spp.

Respiratory Disease
- Alicalgenes
- Enterobacteriaceae
- *Pasteurella* spp.
- Cytophaga (duck septicemia)
- *Pasteurella* spp.
- Haemophilus
- Chlamydiosis (eg, psittacines, Columbiformes)
- *Salmonella* spp.

Septicemia
- *E. coli*
- Many bacteria
- *Listeria* spp.
- *E. rhusiopathiae*
- *Pasteurella* spp.
- *Salmonella* spp.
- *Yersinia pseudotuberculosis*
- Pseudomonas/Aeromonas
- Most Enterobacteriacae
- Staphylococcus (mimics numerous other infectious agents)
- Streptococcus/Enterococcus
- Borrelliosis (high tick areas)

Morphologically similar strains of megabacterium that were considered normal components of the budgerigar proventricular flora have been described.[53]

Some researchers believe that megabacterium is the causative agent of progressive weight loss ("going light syndrome") in budgerigars. The name "going light syndrome" should provisionally be replaced by megabacteriosis, because weight loss is a clinical sign of a variety of chronic diseases. Experimental infections with pure cultures of megabacterium induce disease only in English standard budgerigars and not in the normal breed.[28] These findings suggest that birds vary in susceptibility to the organism, and other factors are involved in the pathogenesis. Spontaneous recovery was common in experimental cases. The host spectrum includes canaries[34] and related finches, cockatiels, lovebirds, chickens and young (3-week-old) ostriches (Hüchzermeier, unpublished).

Clinically infected birds develop chronic emaciation over a 12- to 18-month period that may or may not involve intermittent periods of recovery. Severely affected birds may pass digested blood in the feces. Contrast radiography typically indicates a sand-glass-like retraction between the proventriculus and ventriculus (Figure 33.13). This finding is considered highly suggestive of megabacteriosis.[31] Megabacterium is shed in the feces and can be detected by gram-stained samples from severely sick birds.

At necropsy, a proventriculitis or proventricular ulcer with or without hemorrhages can be observed. Lesions are most common in the pars intermedia gastris. The organisms lie densely together in the necrotic tissue foci. There is usually little inflammatory cellular reaction associated with the organism, which can be seen readily at low magnification from proventricular scrapings. Impression smears from the liver and spleen may be useful in detecting the bacteria, which can be encapsulated in the tissues. There is no treatment because of resistance to all antibiotics commonly used.

References and Suggested Reading

1. **Aleksic S, et al:** Mikrobiologie und Epidemiologie der Yersiniosen. Immun & Infekt 18:178-185, 1990.
2. **Backstrand JM, et al:** Survival of *Pasteurella multocida* in soil and water in an area where avian cholera is enzootic. J Wildl Dis 22:257-259, 1986.
3. **Bergey's Manual of Systematic Bacteriology:** Vol 1. Baltimore, London, Los Angeles, Sydney, William and Wilkins, 1984.
4. **Bisgaard M, et al:** Isolation, characterization and public health aspects of *Vibrio cholera* NAG isolated from Danish duck farm. Avian Pathol 19:271-276, 1975.
5. **Böttger EC:** Systematik, Differenzierung und Nachweis von bakteriellen Infektionserregern - die Familie Mycobacteriaceae. Immun Inf 19:143-152, 1991.
6. **Braumiller W:** Zur Charakterisierung von Hämolysinen aus aviären *Aeromonas-hydrophila*-Stämmen. Vet Dis, München, 1991.
7. **Bredy JP, et al:** The effects of six environmental variables on *Pasteurella multocida* populations in water. J Wildl Dis 25:232-239, 1989.
8. **Bühler-Nickel E:** Untersuchungen zum Isolierungsverfahren, Vorkommen und zur Differenzierung von Listerien beim Geflügel. DVG-Tagung Fachgruppe Bakteriologie, 1990, pp 74-86.
9. **Calnek BW, et al (ed):** Diseases of Poultry. Wolfe Publishing Ltd, 1991.
10. **Cooper GL, et al:** Spondylitis associated with *Pasteurella anatipestifer* infection in turkeys. Proc 41th West Poult Dis Conf, 1991, p 54.
11. **Crosta L, et al:** Oral treatment with clindamycin in racing pigeons. Proc Euro Assoc Avian Vet, 1991, pp 293-296.
12. **Davelaar FG, et al:** Infectious typhlitis in chickens caused by spirochetes. Avian Pathol 15:247-258, 1986.
13. **Davis RW, et al:** A preliminary study of thin bird disease. Proc Electron Micro Soc America, 1981, pp 602-603.
14. **Devriese LA, et al:** *Streptococcus cecorum*, a new species isolated from chickens. Int J Syst Bacteriol 33:772-776, 1983.
15. **Devriese LA, et al:** Streptococcal infections in pigeons. Proc 2nd Europe Symp Avian Med Surg, 1989, pp 113-117.
16. **Devriese LA, et al:** *Enterococcus columbae*, a species from pigeon intestines. FEMS Microbiology Letters 71:247-252, 1990.
17. **Devriese LA, et al:** *Streptococcus bovis* infections in pigeons. Avian Pathol 19:429-434, 1990.
18. **Devriese LA, et al:** Tracheo-bronchitis in canaries: A respiratory condition caused by *Enterococcus faecalis*. J Assoc Avian Vet 4:113-116, 1990.
19. **Dorrestein GM, et al:** Diseases of passerines, especially canaries and finches. Proc Assoc Avian Vet, 1983, p 62.
20. **Dorrestein GM, et al:** Campylobacter infections in cage birds: clinical, pathological and bacteriological aspects. Proc Joint Meeting Vet Pathol, Utrecht, 1984.
21. **Dortbudak, et al:** *In vitro* activity of clarithromycin against *M. avium*. 12th Conf Europ Soc Mycobac, Prague, 1991.
22. **Dwars FG, et al:** Incidence of spirochetal infections in cases of intestinal disorder in chickens. Avian Pathol 18:591-595, 1989.
23. **Eichinger E:** Lactobacillen zur Bekämpfung von Enterobacteriaceen im Darm von Psittaciformes. DVG VII. Tagung Vogelkrankheiten, München, 1990, pp 231-234.
24. **Forster F, et al:** Mycobacteria in psittaciformes. Proc 1st Intl Conf Zool & Avian Med 39-56, 1987.
25. **Gerlach H:** Diagnose der Pseudotuberkulose bei lebenden Vögeln. DVG-Tagung Krankht. der Vögel, München, 1979, pp 96-101.
26. **Gerlach H:** Infection with the so-called *Haemophilus septicaemia anseris* in geese. Proc VIIth Intl Cong WVPA, 1981, p 80.
27. **Gerlach H:** Bacterial diseases. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, London, WB Saunders Co, 1986, p 434.
28. **Gerlach H:** Going light in budgerigars. Proc Assoc Avian Vet, 1986, pp 247-250.
29. **Gratzl-Köhler:** Spezielle Pathologie und Therapie der Geflügelkrankheiten. Stuttgart, Ferdinand Enke Verlag, 1968, p 548.
30. **Grebe HH, et al:** Vorkommen von Bakterien der Gattung Haemophilus bei verschiedenen Vogelarten. Zbl Vet Med B 22:749-757, 1975.
31. **Grimm F, et al:** Die röntgenologische Darstellung von gastrointestinalen Läsionen beim Wellensittich verursacht durch Megabakterien. DVG VII. Tagung Vogelkrankheiten München, 1990, pp 321-325.
32. **Gylstorff I:** *In* Gylstorff I, Grimm F: Vogelkrankheiten. Stuttgart, Verlag Eugen Ulmer, 1987, p 316.
33. **Heesemann J:** Enteropathogene Yersinien: Pathogenitätsfaktoren und neue diagnostische Methoden. Immun & Infekt 18:186-191, 1990.
34. **Herck van H, et al:** A bacterial proventriculitis in canaries (*Serinus canaria*). Avian Pathol 13:561-572, 1984.
35. **Hinz K-H:** Über einige bakteriell bedingte Infektionen bei Zierund Wildvögeln. Collegium veterinarium 15:9-12, 1984.
36. **Karmali MA, et al:** Taxonomy of the genus *Campylobacter*. *In* Butzler PJ (Hrsg) (ed): Campylobacter infection in man and animal. Boca Raton, CRC Press Inc, 1984, pp 1-20.
37. **Knorz W, et al:** Zur Pathogenität von Listerien. Immune Infect 14:76-80, 1986.
38. **Koppers N, et al:** Aviäre Mykobakteriose durch bisher nicht identifizierbare Stämme. J Vet Med B 38:3-10, 1991.
39. **Koppers N, et al:** Mykobakteriose bei Waldrappen (*Geronticus eremita*). Berl Münch Tierärztl Wschr 104:57-62, 1991.
40. **Korbel R:** Epizootiologie, Klinik und Therapie der *Pasteurella- multocida*-Infektion beim Vogelpatienten nach Katzenbiß. Tierärztl Prax 18:365-376, 1990.
41. **Korbel R:** Ocular manifestations of systemic diseases in birds. Proc Europ Assoc Avian Vet, 1991, pp 157-167.
42. **Lauwers S et al:** Campylobacter serotyping and epidemiology. Lancet 158-159, 1981.
43. **Lumeij JT, et al:** Observations on tuberculosis in raptors. *In* Cooper JE, Greenwood A (eds): Recent Advances in the Study of Raptor Diseases. Keighley, England, Chiron Publications Ltd, 1980, pp 137-139.
44. **McOrist S, Keece RL:** Clostridial enteritis in free-living lorikeets, (*Trichoglossus* spp.). Avian Pathol 21:503-507, 1992.
45. **Mundt JO:** Enterococci. *In*: Bergey's Manual of Systematic Bacteriology Vol 2, Baltimore, London, Los Angeles, Sydney, Williams and Wilkins, 1986.
46. **Nairn ME:** Bacterial osteomyelitis and synovitis of the turkey. Avian Dis 17:504-517, 1973.
47. **Ochsenhirt B:** Lokalisation von *Salmonella typhimurium* im Hühnerembryo nach experimenteller Eiinfektion. Diss med vet, München, 1974.
48. **Paterson JS, et al:** A method for the recovery of *Pasteurella pseudotuberculosis* from faeces. J Path Bact 85:241-242, 1963.
49. **Piechulla K, et al:** Phenotypic and genetic relationships of so-called *Moraxella* (*Pasteurella*) *anatipestifer* to the Flavobacterium/Cytophaga group. Vet Microbiol 11:261-270, 1986.
50. **Pulverer G, et al:** Coagulase-negative staphylococci. Zbl Bakt Hyg A(264):1-28, 1987.
51. **Riedel B, et al:** Untersuchungen über das Vorkommen von Campylobacter im Kot von im menschlichen Wohnbereich gehaltenen Vögeln. Berl Münch Tierärztl Wschr 100:52-59, 1987.
52. **Rinaldi, et al:** Episodi di streptococcosi dell'anatra. Atti della Societa Italiana della Scienze Veterinarie 24:665-666, 1970.
53. **Scanlan CM, et al:** Characterization of a gram-positive bacterium from the proventriculus of budgerigars (*Melopsittacus undulatus*). Avian Dis 34:779-786, 1990.
54. **Schleifer KH:** Transfer of *Streptococcus faecalis* and *Streptococcus faecium* to the genus *Enterococcus nom. rev.* as *Enterococcus faecalis comb. nov.* and *Enterococcus faecium comb. nov.* Int J Syst Bacteriol 43:31-34, 1984.
55. **Schröder H-D:** Beitrag zu den Infektionskrankheiten der Zoo- und Wildvögel. IV. DVG-Tagung Vogelkrht, München, 1985, pp 173-174.
56. **Sinn LC:** Listeriosis in birds. Proc Assoc Avian Vet, 1988, pp 189-190.
57. **Skirrow MB:** Campylobacter enteritis: A "new" disease. Br Med J 2:9-11, 1977.
58. **Small PJ:** Spirochetosis on a California pheasant ranch: A case report. Proc 33rd West Poult Dis Conf, 1984, p 31-33.
59. **Smit EK, et al:** Avian tuberculosis in wild birds in the Netherlands. J Wildlife Dis 23:485-487, 1987.
60. **Smit JA, et al:** Necrotic enteritis and colitis in ratite birds. Proc 40th West Poult Dis Conf, 1991, pp 258-260.
61. **Stoll L:** Kälteanreicherung von Yersinia pseudotuberculosis zum Nachweis aus Untersuchungsmaterial. (Vortrag, 1966) ref Zbl Hyg Bakt I ref 206:534, 1967.
62. **Thorel M-F, et al:** Numerical taxonomy of mycobactin-dependent mycobacteria, emended description of *Mycobacterium avium*, and description of *Mycobacterium avium subsp. avium subsp. nov.*, *Mycobacterium avium subsp. paratuberculosis subsp. nov.*, and *Mycobacterium avium subsp. silvaticum subsp. nov.* Int J Syst Bacteriol 40:254-260, 1990.
63. **Woerpel RW, et al:** Retro-orbital *Mycobycterium tuberculosis* infection in a yellow-naped Amazon parrot (*Amazona ochrocephala auropalliata*). Proc Assoc Avian Vet, 1983, pp 71-76.
64. **Wolinsky E, et al:** Proposed numbering scheme for mycobacterial serotypes by agglutination. Int J Syst Bacteriol 23:182-183, 1973.
65. **Woods LW, et al:** Summary and review of diagnostic observations of pet/exotic bird cases at the veterinary laboratory services in Petaluma. Proc 36th West Poult Dis Conf, 1987, pp 124-127.