Disease Conditions and Clinical Signs of Pet Birds

Rose Ann M. Fiskett, VMD, Dipl ABVP (Avian), and Drury R. Reavill, DVM, Dipl ABVP (Avian), Dipl ACVP

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Affiliation: From Potomac Valley Veterinary Hospital, 9553 Braddock Road, Fairfax, VA 22032 USA (Fiskett) and Zoo/Exotic Pathology Service, 2825 KOVR Drive, West Sacramento, CA 95605 USA (Reavill).

Abstract: Disease conditions and clinical signs of many companion birds are presented, including African grey parrots, Amazon parrots, budgerigars, canaries and finches, cockatiels, cockatoos, conures, lovebirds, macaws, and Quaker parrots. Infectious diseases, neoplasia, reproductive disorders, and metabolic and nutritional disorders common to each class of bird are reviewed.

African Grey Parrots

Infectious diseases

Adenovirus infection is more frequently recognized as sporadic outbreaks in small psittacine birds (cockatiels [*Nymphicus hollandicus*] and lovebirds [*Agapornis* species]) and African species (African grey [*Psittacus erithacus*] and Senegal parrots [*Poicephalus senegalus*]). There is a variable species susceptibility to adenoviral infections. Lesions include extensive necrosis of the liver, spleen, pancreas, and intestines with the typical intranuclear inclusions. The virus is spread by horizontal transmission; the incubation period is unknown. Diagnosis is difficult in the live bird; electron microscopy is necessary to demonstrate the virus in feces or pharyngeal secretions.

Circovirus infection in African grey parrots is characterized by significant immunosuppression; the progressive feather lesions that develop in other species are rarely seen. The infections are associated with pneumonia, enteritis, and death. There is a severe leukopenia and non-regenerative anemia. Virus is shed in the feces, crop secretions, and feather dust. The minimum incubation period is reported as 21–25 days with a maximum period of several years.[1] Several tests are available to detect this virus in exposed psittacine birds. In a review of case submissions, the African grey parrot is the most common species diagnosed with circovirus infections, followed by lovebirds and Eclectus parrots (*Eclectus roratus*) (D. R. R., oral communication, March 2004).

Cutaneous papillomas are virally induced warty lesions that occur on the skin of the face and feet. One report describes multiple cutaneous papillomas on the face of an adult male Timneh (*Psittacus erithacus timneh*).[2] Proliferative skin lesions were found over the head, affecting the palpebrae, the cutaneous areas at the angles of the upper and lower beaks, and the skin contiguous with the lower beak. Lesions tested positive by immunostaining with papilloma virus group-specific antibodies. No treatment was described; the lesions progressed. Similar virally induced lesions have been reported on the legs of European chaffinches (*Fringilla coelebs*) and bramlings (*Fringilla montifringilla*).[1] Both cases of cutaneous papillomas identified by one author (D. R. R.) were around the eye and beak on African grey parrots.

Proventricular dilatation disease (PDD) in African grey parrots presents most commonly as a central nervous system disease. (See discussion under Macaws.)

Salmonella typhimurium infection can result in high mortality of young birds, lories, and African grey parrots.[3] It is considered a primary pathogen; some serotypes can penetrate the mucosal barrier. Noninvasive serotypes may result in the carrier state. Transmission is by ingestion of contaminated food or water or by direct contact of aerosolized fecal or feather dust. Feces of chronically infected carrier birds are one of the most common sources of infection. The disease course in birds is dependent on the number of organisms, their serotype, and the age, species, and condition of the host. It ranges from a peracute, acute, chronic, to subclinical infection. Clinical signs include depression, lethargy, anorexia, weight loss, diarrhea, pasty vents, lameness, abscess formation, convulsions, poor hatching or excessive fledgling mortality, dehydration and crop stasis, meningitis, osteoarthritis, and sudden death. The classic gross lesions are hepatomegaly, splenomegaly, pneumonia, and a catarrhal to hemorrhagic enteritis. Diagnosis is made by fecal or affected organ culture. Antemortem diagnosis by fecal cultures is difficult because of the intermittent shedding. Therapy is based on antimicrobial sensitivity and the condition of the bird. Treatment periods can be long (3–8 weeks) and may not eliminate the organism. Control of this highly contagious disease is dependent on strict isolation, vector control, and appropriate disinfection.

Fungal pneumonia caused by *Aspergillus* species or other fungal organisms is a common finding in young African grey parrots infected with circovirus. Fungal pneumonia and granulomas can suggest immunosuppression as well as support exposure to an overwhelming number of fungal spores.[3] Environmental evaluation is necessary to identify a potential source of fungal spore formation (eg, wood shavings, corncob bedding, and seed hulls). Review of the recent medical history may identify use of immunosuppressive drugs, chronic antibacterial therapy, neoplastic conditions, severe malnutrition, or underlying infectious disease agents. Antemortem diagnosis is challenging; poor therapeutic response is common.

Metabolic and nutritional disorders

Atherosclerosis is a proliferative lesion of the tunica media and tunica intima of elastic and muscular arteries. Atherosclerotic plaques cause abnormal vascular flow and loss of endothelial integrity, with decreased vessel elasticity and variable degrees of vessel narrowing. These changes in vessel walls can initiate thrombosis. In birds, lesions are primarily in the aorta and brachiocephalic arteries. In psittacine birds, atherosclerosis can be a primary cause of death, which may occur without premonitory clinical signs. Birds may develop dyspnea, muscle wasting, progressive paresis of the legs, and seizure-like activity. Based on a submission review (D. R. R., oral communication, March 2004), atherosclerosis is most common in African grey parrots, lovebirds, Amazon parrots (*Amazona* species), and cockatiels. The average age range is 10–15 years.

Hypocalcemia syndrome in African grey parrots has been characterized by decreased levels of blood calcium associated with tetany or seizures.[4] The exact etiology of this condition is unknown but may be related to nutritionally poor diets. Glucocorticiod therapy will also decrease the total calcium concentration as will hypoalbuminemia, hypovitaminosis D3, hypoparathyroidism, or renal hypoparathyroidism. Total calcium is representative of the active ionized calcium, protein bound calcium, and calcium chelated with anions. In African grey parrots, a significant correlation has been found between total calcium and albumin concentration in the plasma. Interpret calcium along with albumin concentrations; bound calcium is biologically inactive.

Some birds, especially African grey parrots, develop massive keratin rhinoliths that distort the nares and nasal sinus. These are commonly due to hypovitaminosis A and secondary infections. Pet birds that are on a primary seed or cereal grain diet or have intestinal mucosal lesions that interfere with conversion of carotenoids to vitamin A may develop vitamin A deficiency. Vitamin A deficiency results in epithelial squamous metaplasia, which is manifested as hyperkeratosis of oral cavity, conjunctiva, nasal lacrimal duct, upper alimentary tract, and respiratory tract. In large parrots, the epithelial changes will appear when vitamin A concentrations in the liver decrease below 50 IU/g.[5] Keratinizing epithelium will block the ducts of submucosal mucus glands, causing the glands to

enlarge with secondary infections that produce large keratin granulomas within the nasal or oral cavity. The squamous metaplasia also alters the mucosal defenses, predisposing the bird to fungal and bacterial infections, primarily of the respiratory tract.

Neoplasia

Malignant melanomas are typically located on the face and beak of African grey parrots.

Gross lesions appear as brown-black masses with irregular borders that infiltrate into the surrounding tissues. These can look similar to cutaneous hemangiomas and hemangiosarcomas. One author (D. R. R.) has seen 2 cases of malignant melanomas developing on the head in African grey parrots and one on the beak of a budgerigar (*Melopsittacus undulatus*). All tumors recurred after surgical removal.

African grey parrots uncommonly develop lipomas, gastric carcinomas, fibrosarcomas, bile duct carcinomas, or xanthomas compared to other psittacine birds.

Parasites

Sarcocystis is a protozoal infection that results in high morbidity and mortality of Australian, Asian, and African origin psittacine birds and occasionally exotic columbiformes (pigeons and doves). In a review of cases (D. R. R., oral communication, March 2004), lories and lorikeets are the most common species affected by sarcocystis, followed by African grey parrots, cockatoos, and Eclectus parrots. American or Neotropical (Mexico, South, and Central America) psittacine birds are relatively resistant to the disease as adults. Transmission is by the definitive host, opossums, and by mechanical vectors such as cockroaches. The normal intermediate hosts are cowbirds and grackles. Death occurs rapidly due to early parasite schizogony (asexual reproduction) in the lung, which leads to fatal respiratory collapse. Severe pulmonary congestion and hemorrhage may be the only lesion recognized. The drug pyrimethamine may be of benefit in some cases with early diagnosis or high suspicion. Control of the opossum and cockroach population is important.

Other conditions

Automutilation of the sternum in psittacine birds (split sternum) generally occurs over the cranial portion of the sternum, along the point of the keel, and may appear as an open wound of the skin and muscle. Continued self-trauma can lead to tissue necrosis and infection, which can extend into the deeper muscles and bone of the keel. A mixed bacterial population (*Streptococcus* species, *Staphylococcus aureus, E coli, Pseudomonas* species, and *Klebsiella* species) and occasionally fungi can be isolated from these wounds. The pruritis typical of these lesions may be due to the secondary infections. Improper wing trims in heavy-bodied birds (African grey parrots and Amazon parrots) resulting in a traumatic landing on the sternum is suspected to be one cause. In flighted birds the reason for self-mutilation is unknown.

Choanal atresia is described in African grey parrots. A persistent membrane or bony plate at the palate of the nasal cavity results in a closed choanal slit. This blocks the normal drainage of nasal secretions into the oral cavity. Young birds present with a chronic nasal or ocular discharge and in some cases, the infraorbital sinuses will be distended with clear secretions. Surgical correction is recommended.

Constricted toe syndrome can be caused by fibers, scabs, or necrotic tissue and may result in avascular necrosis of the distal toe. In juvenile and nestling macaws (*Ara* species), African grey parrots, Eclectus parrots, cockatoos, and conures, the development of digital necrosis occurs without an identifiable constricting foreign material.

Proposed causes include low humidity, egg-related strictures, ergot-like intoxication, or digit fractures. This deformity consists of an annular ring of constriction that eventually causes swelling and necrosis of the distal segment of the toe. If identified early, some resolve with increased environmental humidity, hot moist compresses, massage therapy, and surgical correction. Fibrous annular rings are incised and accumulated serum and tissue debris gently debrided. The toe should be soaked in a warm, dilute, povidone-iodine solution and an antibiotic ointment applied. DMSO may reduce inflammation. If the distal segment is severely swollen or necrotic it should be surgically removed. Toe constrictions can often be prevented by keeping susceptible species on non-desiccating surfaces and increasing brooder humidity.[6]

Feather picking occurs quite commonly in African grey parrots. On histologic examination, there may be no lesions or a perivascular dermatitis. Perivascular dermatitis is suspected to be an allergic skin disease, although confirmation can be difficult. Gross changes possibly associated with allergic skin disease include feather loss, reddening of the skin, and occasionally surface exudates. Some of the gross lesions may be secondary to self-trauma. As yet, there are still an insufficient number of cases with follow-up information to better define this disease. A diagnosis of psychogenic self-mutilation is made when no organic cause can be identified and the feather follicles and skin have no histologic lesions.[7] This aberrant behavior is the commonly believed cause for many feather-picking birds. It is attributed to boredom, loss of a mate (human or feathered), dietary changes, environmental maladaptation, exposure to cigarette smoke, or intolerance to new stimuli. This condition looks like perivascular dermatitis with missing and frayed feathers, reddened skin, ulcers, and pruritis.

"Hematuria" syndrome in neonates

Reddish urine and urates have been described in juvenile African grey parrots and some Amazon and Pionus parrots (*Pionus species*). It can be distinguished from hematuria by a fecal occult blood test. It occurs sporadically with several hand-feeding formulas and can be more pronounced on certain bedding materials (paper towels). This condition is not associated with pathology or other clinical signs.[4]

Amazon Parrots

Infectious diseases

Mycobacteria infection in many species including birds, generally results in a chronic wasting disease. From case submissions (D. R. R., oral communication, March 2004), Amazon parrots, brotogerids, Pionus, and finches/canaries are most commonly infected. The lesions range from diffuse granulomatous inflammation to multiple granulomas that are easily confused with tumor masses. Mycobacterium has a zoonotic potential and is very stable in the environment. Diagnosis can be difficult, as Amazon parrots do not consistently develop an abnormal leukogram. Biopsy or aspiration, cytology, and acid fast stains can be used to evaluate masses or intestinal thickenings.

Chlamydophila psittaci is an energy dependent obligate intracellular parasite that is a zoonotic disease agent. Amazon parrots typically present with the classic disease. The clinical signs include sinusitis, dyspnea, conjunctivitis, polyuria, diarrhea, lethargy, anorexia, yellow to dark green droppings, poor feathering, and chronic weight loss. Rarely central nervous system signs such as opisthotonos, tremors, and convulsive movements can develop. Hematology, radiography, serum biochemistries, DNA PCR probes, antigen capture tests, antibody tests, cytology, and culture are all procedures that can provide a clinical diagnosis. *No* antemortem test will certify a bird is free of *Chlamydiophila*. Postmortem findings can include hepatomegaly, splenomegaly, and cloudy air sacs. Aerosol inhalation and ingestion are the primary routes of transmission. The incubation period of *Chlamydophila psittaci* ranges from 5 days to several weeks.

Psittacid herpesviruses [PsHV] are a heterogeneous group of avian herpesviruses.[5] Serologically 3–5 serotypes are recognized. One serotype and its corresponding genotype are most commonly associated with an acute fatal disease (Pacheco's disease) seen in many species of parrots. There is variable species susceptibility to this viral infection. Amazon parrots, cockatoos, cockatiels, and macaws are the most common species diagnosed with a herpesvirus infection (consistent with Pacheco's disease) identified in a recent survey (D. R. R., oral communication, March 2004). Susceptible birds usually die with no or few clinical signs (depression, anorexia, fluffed, intermittent diarrhea ± hemorrhagic, polyuria/polydipsia, convulsions, and biliverdin staining of feces and urates). Ingestion, inhalation, and conjunctival exposure have experimentally been shown to result in infection and disease. The incubation period ranges from 3 to 14 days. Survivors in an outbreak should be considered latently infected. Carrier birds shed the virus in the feces and pharyngeal secretions. PsHVs are maintained in nature in birds that are persistently infected with the virus. Amazon parrots and certain species of conures and macaws have been shown to continuously shed virus in oral secretions and feces.

Extra-hepatic lesions occur with a fair degree of frequency in PsHV disease. The herpesvirus outbreaks that primarily target the respiratory tract (Amazon tracheitis-like virus) have been recognized in Amazon parrots, brown-throated conures, Bourke's parakeets (*Neopsephotus bourkii*), whiskered lorikeets (*Oreopsittacus arfaki*), and rosellas (*Platycercus* species). The tracheal mucosa and lungs appear edematous and congested. The Amazon tracheitis virus is believed to be a variant of the infectious laryngotracheitis virus.

Amazona (Neotropical) pox enjoys a wide host range of South American psittacine birds. The disease may present in either the dry (cutaneous) or wet (diphtheroid) forms. The cutaneous form involves the nonfeathered areas of skin where papules or raised scab-like lesions develop around the eyes, beak, nares, tibiotarsus, and feet. The lesions will eventually desquamate, usually without leaving a scar. The wet form affects the mucous membranes and generally results in a high mortality rate. The lesion is a depigmented, raised plaque covered by a diphtheritic membrane on conjunctiva, oral membranes, trachea, and bronchi. Transmission is by fomites, arthropod vectors, and direct contact. The diagnosis is usually based on the histopathology of the affected tissues and identification of hyperplastic and hypertrophic epithelial cells with large eosinophilic intracytoplasmic inclusions. A vaccine is available for some species.

Hepatic lipidosis (fatty liver syndrome) is the excessive accumulation of lipid within hepatocytes that can result in hepatic dysfunction. In a recent survey (D. R. R., oral communication, March 2004), fatty liver syndrome was most common in cockatoos, cockatiels, Amazons, macaws, Eclectus parrots, African grey parrots, budgerigars, lories, and Quaker parrots (*Mysiopsitta monachus*). The etiology of the lesion in young birds appears to be diets rich in fats. In adult birds, protein malnutrition, high-fat diets, and obesity appear to be contributing factors. Fatty liver syndrome is also generally associated with renal lesions, heart disease such as atherosclerosis, lung lesions, and non-specific gastroenteritis.

Neoplasia

Cholangiocarcinoma (bile duct carcinoma) is an aggressive locally invasive liver tumor that can metastasize. Recent reports have suggested a relationship between papillomatosis and bile duct carcinoma. However, given the frequency of each disease in some psittacine birds (ie, Amazon parrots), this possible relationship needs further evaluation. One author (D. R. R.) has seen this tumor in Amazon parrots with an average age at presentation of 12 years (the youngest was 3 years of age and the oldest was 17 years of age). A smaller number of macaws and rarely budgerigars, cockatoos, conures, lovebirds, grey cheek parrots (*Brotogeris pyrrhopterus*), parrotlets (*Forpus species*), and Quaker parrots are recognized in a recent review (D. R. R., oral communication, March 2004). Birds generally present acutely with non-specific clinical signs (cloacal prolapse, weight loss, anorexia, acute trembling, coma, and seizures) or are found dead. On gross examination, the liver is either enlarged or has

multiple white to tan, semi-firm nodules. Metastases are unusual but have been found in the lung and pancreas. Antemortem diagnosis is difficult; chemotherapy may be tried.[8]

Granular cell tumors are rare in any species. The cells of origin are suspected to arise from multiple embryonic tissues. This type of tumor has been reported on an adult cockatiel and on adult Amazon parrots.[8] The tumors are commonly found within the dermis on the head. Differential diagnosis includes mycobacterial dermatitis. These tumors seem to be benign and complete surgical removal should be curative.

Lipomas are benign lumps of fat that are most common in Amazon parrots, budgerigars, Quaker parrots, rose-breasted cockatoos (Galahs) (*Cacatua roseicapilla*), and cockatiels. These soft, pale yellow, encapsulated, lobulated masses in the subcutis are most frequently found over the sternum, abdomen, and inner thighs. If traumatized, they may become inflamed and necrotic. Obese birds can develop fat pads in the typical locations for lipomas, so it is possible that some masses are hyperplastic adipose tissue and not lipomas. There is a possible relationship with abnormal thyroid gland function and the development of multiple lipomas. Therapy includes caloric reduction, diet change, and surgical removal when there is rapid tumor growth, increased vascularity of the tumor, or ulceration of the overlying skin. Recurrence is common.

Multicentric lymphosarcoma (malignant lymphoma) is the most common lymphoid neoplasia in psittacine birds and passerine birds. One study reports an age range from 5 months to 30 years, with an average of 8 years at the time of diagnosis.[8] Clinical signs associated with lymphosarcoma include periorbital or cutaneous swelling, depression, anorexia, weight loss, paresis, lameness, abdominal swelling, diarrhea, blindness, scant droppings, dyspnea, polydipsia, regurgitation, feather loss, and folliculitis. Anemia (PCV<35%) is common in birds. A leukemic blood profile is uncommon in psittacine birds with lymphoid neoplasia. This disease can present with both diffuse and nodular involvement. Organs and tissues that are typically infiltrated include the liver, spleen, kidneys, skin, bone, gastrointestinal tract, thyroid gland, oviduct, lungs, sinus, thymus, testes, brain, mesentery, trachea, fat, periorbital muscles, and pancreas. The liver, spleen, and kidneys are most frequently affected and generally appear enlarged and pale. Other diseases that may grossly resemble visceral lymphosarcoma include amyloidosis, fatty liver syndrome, atoxoplasmosis (in mynahs and canaries), hepatitis, systemic mycobacteriosis, and other forms of neoplasia. Some patterns of presentation noted include African grey parrots with periorbital masses, Amazon parrots with choanal masses, and umbrella cockatoos (*Cacatua alba*) with multiple nodular cutaneous masses. Diagnosis is made by aspiration cytology or biopsy of the affected tissue and, if indicated, bone marrow evaluation. In both the literature and in one author's experience (D. R. R.), cutaneous presentations have a more pleomorphic cell population containing large lymphoblasts, small well-differentiated lymphocytes, and scattered plasma cells. Treatment has included prednisolone, orthovoltage X-ray, vincristine sulfate, chlorambucil, cyclophosphamide, doxorubicin, and L-asparaginase; however, few birds have responded favorably.[8]

Other conditions

Amazon foot skin necrosis occurs in Amazon parrots of all ages (primarily yellow-naped Amazon parrots [*Amazona ochrocephala*]). Clinical lesions appear as irregular patterns of erythema or brown to black discoloration on the unfeathered skin of legs and feet. The birds act very pruritic and will attack the area of the lesions. Auto-mutilation is common. Etiology is unknown; treatment consists of preventing further mutilation while permitting the lesions to heal. Some birds have recurring episodes.

Additional syndromes

These conditions have been commonly identified in this species but more detailed description is provided under the species in parentheses following the condition: fungal pneumonia (African grey parrots), automutilation of

sternum (African grey parrots), xanthoma (Quaker parrots), Sertoli cell tumors (budgerigars), papillomatosis (macaws), atherosclerosis (African grey parrots), and chronic liver disease (cockatiels)

Budgerigars

Infectious diseases

Megabacteria (avian gastric yeast) are large, gram-positive and PAS (periodic acid-Schiff) positive yeast. In adult birds, weight loss, and depression mark the infection ("going light"). Megabacteria has been identified in budgerigars, cockatiels, finches, canaries (*Serinus canarius*), lovebirds, neophema parakeets, rosellas (*Platycercus species*), a lesser sulfur-crested cockatoo (*Cacatua sulphurea*), and chickens. In budgerigars and canaries, it may be a component of the normal upper alimentary tract flora. The organism inhabits the lower portion of the proventriculus within the superficial mucosal glands at the isthmus. The mode of transmission is unknown. The organism may be detected with multiple Gram stains of the feces and fecal smears of infected birds. Budgerigars may have non-specific hematologic changes including anemia, leukocytosis, heterophilia, monocytosis, lymphocytosis, basophilia, and thrombocytosis. Amphotericin B may be an effective therapy.

Adenovirus infection of the kidney is seen in a variety of psittacine birds, including budgerigars, cockatiels, lovebirds, and parrotlets. Grossly there may be some nonspecific renal enlargement. In some birds, particularly budgerigars and cockatiels, the only histologic lesion may be a few widely scattered large intranuclear inclusion bodies in tubular epithelial cells. Transmission is horizontal; incubation period is unknown. Diagnosis in the living bird is difficult and generally requires electron microscopy to demonstrate the virus in feces or pharyngeal secretions.

Polyomavirus infections have been associated with decreased hatchability and embryonic death. Clinical disease induced by polyomavirus in budgerigars appears to depend on the age and condition of the bird when exposed. Neonates from infected flocks may develop normally for 10–15 days and then suddenly die.[1] Mortality can range from 30–100% of hatchlings with higher rates in bird less than 15 days old. Clinical signs include anorexia, crop stasis, depression, abdominal distention, subcutaneous hemorrhage, tremors of head and neck, ataxia, and reduced formation of down and contour feathers. Birds often die acutely with the crop and gastrointestinal tract full of food. Gross lesions include hydropericardium, cardiomegaly, myocardial hemorrhage, hepatomegaly (yellow white foci), intestinal hemorrhage, renal swelling, pallor, or congestion (white foci), and petechiation. Surviving birds may have symmetrical feather abnormalities, dystrophic primary and tail feathers, decreased down feathers, and lack filoplumes on the head and neck; these lesions resolve after several months. Virus is excreted in feather dander and droppings and the disease spreads both horizontally and vertically. Virus persists in the kidneys of carrier birds and is excreted under stress. Environmental contamination can persist. Diagnosis is best accomplished by DNA-PCR probes on samples from birds or the environment.

Neoplasia

Fibrosarcomas are tumors that originate from fibrous connective tissue and occur on the limbs, face, beak, syrinx, liver, small intestine, cloaca, spleen, air sacs, and lungs. One author (D. R. R.) sees these most commonly in budgerigars, Quaker parrots, lovebirds, and cockatiels. Fibrosarcomas are firm masses with irregular and indistinct borders. When fibrosarcomas involve the skin, there is frequent ulceration, with the skin firmly attached to the mass. Fibrosarcomas are locally invasive, rarely metastasize, and have a moderate to high potential for recurrence, giving them a guarded prognosis.

Gastric carcinomas and adenocarcinomas are reported more frequently in *Brotogeris* species, budgerigars, lovebirds, cockatiels, as well as Amazon parrots, cockatoos, African grays, macaws, and lories. These tumors produce a thickening and irregularity of the proventricular and/or ventricular wall with a variable amount of hemorrhage and necrosis. There may be ulceration and perforation. Gastric adenocarcinoma can penetrate through the muscular tunics to adjacent organs as well as metastasize. Sites of metastasis include the pancreas and lungs. Clinical signs reported include weight loss, weakness, inability to perch, head tilt, melena, undigested seeds in the feces, beak overgrowth, and polyuria. Although this tumor is rarely diagnosed before death, a positive fecal occult blood test may suggest this diagnosis. Common hematology changes are anemia and hypoproteinemia. Radiographs may reveal fluid and gas in the intestinal tract and a mass lesion. Contrast radiography may help delineate a thickening of the proventriculus, incomplete filling of the proventriculus and ventriculus, and an irregular distention. The differential diagnoses for the clinical signs and findings include megabacteriosis, squamous cell carcinoma, papillomatosis, and mycobacteriosis. The definitive diagnosis is frequently obtained postmortem; therapeutic intervention has not been reported.

Renal tumors in pet birds include embryonal nephromas, adenocarcinomas, carcinomas, and adenomas. All are reported more frequently in young to middle-aged, male budgerigars. The tumors have a 3 week to 6 month course. Common clinical signs are weight loss, vomiting or regurgitation, and a unilateral lameness. Gonadal tumors can also present with lameness, but not as frequently as the renal tumors. Most of the tumors are found in the cranial division, and they grow by expansion and infiltration. The paresis is believed to be due to pressure on the lumbar plexus. Radiographically, there can be abdominal enlargement, and the ventriculus will be displaced ventrally against the body wall. Treatment options include surgical removal, although this is a difficult procedure with a high mortality. Methylprednisolone has been used as a palliative therapy. They are rarely reported to metastasize and do not appear to compromise renal function.

Sertoli cell tumor, a neoplasm of sustentacular or "nurse" cells of the testis, is a fairly common testicular tumor of birds. This tumor is most frequently reported in budgerigars where it is associated with hyperostosis, changes in the hemogram, and hyperestrogenism that can result in a color change of the cere (blue to brown). In other birds (cockatiels, Amazon parrots, finches, chickens, pigeons, ducks, and quails), the most common clinical sign is of coelomic cavity distention and respiratory compromise. Metastases are rarely described.

Nutritional and metabolic disorders

Goiter results from an iodine deficiency. The thyroid glands are located in the thoracic inlet and normally are not palpable. With goiter, the enlarging glands result in clinical signs of a loud, wheezing respiration with neck extension. Crop dilation and vomiting may occur if the goiter obstructs the outlet to the crop. Therapy includes a drop of iodine orally each day; injectable iodine, and dexamethasone may be necessary.[6] Long term, the bird should be converted over to a formulated diet. Budgerigars with thyroid tumors may have clinical signs identical to those seen with goiter. Differentiation is determined by treatment response.

Parasites

Trichomoniasis is common in Australia and has been identified in the United States. It involves the crop and gastrointestinal tract. Vomiting, crop distention, obstruction, and wasting may occur.

Knemidokoptes pilae is an infestation commonly recognized on budgerigars and Canaries. Lesions are raised yellowish, honey-combed encrustations found on unfeathered skin around the beak, cere, vent, legs, and feet. In severe infestations, feathered portions of the skin may be involved. Chronic infections may result in deformed growth of beak and nails. The mites are transmitted directly from bird to bird, although primary transmission may occur in the nest to featherless offspring. Infestations in budgerigars can be asymptomatic for over a year. Severe infestations may be associated with decreased immune function.

Toxins

The best documented reports of airborne pulmonary toxins are of those caused by inhalation of pyrolysis products produced from overheated polytetrafluoroethylene (PTFE)-coated cooking pans, stove tops, and coated heat lamps. At 530°F (280°C), pyrolysis occurs and PTFE is degraded, releasing irritant particles and acidic gases. Other airborne toxins are aerosol sprays, cooking gas, carbon monoxide, tobacco smoke, fumes from burned foods, and cooking oils. Although the specific toxin is unknown, the operation of self-cleaning ovens has resulted in acute deaths. Compounds emitted from burned foods and other materials may be toxic. The most common clinical finding is sudden death due to acute pulmonary edema, hemorrhage, and shock. The birds consistently have severely congested lungs. Minimal exposure can result in somnolence, dyspnea, wheezing, in-coordination, weakness, respiratory distress, and terminal convulsions. Treatment includes fresh air, steroids, broad-spectrum antibiotics, fluids and warmth to prevent shock, pulmonary edema, and bronchopneumonia. One author (D. R. R.) sees the lesions of airborne toxicity most commonly in budgerigars, cockatiels, conures, and lovebirds.

Unknown etiology

Brown hypertrophy of the cere occurs in older female budgerigars. The cornified layer of the cere undergoes extensive hyperplasia. In some birds, the growth has the appearance of a horn. It is considered a normal change in older hens.

Additional syndromes

These conditions have been commonly identified in this species but more detailed description is provided under the species in parentheses following the condition: *Microsporidia* (lovebirds), yeast infection (cockatiels), chronic liver disease (cockatiels), articular or synovial gout (cockatiels), hepatic lipidosis (Amazon parrots), persistent egg laying (cockatiels), lipoma (Amazon parrots), lymphosarcoma (Amazon parrots), myelolipoma (lovebirds), and xanthoma (Quaker parrots).

Canaries and Finches

Infectious diseases

There is some controversy as to whether bacteria and other microorganisms should be found in stained fecal smears from normal canaries and finches; low levels of gram-positive rods or cocci may be normal. Little or no bacterial growth occurs on routine aerobic microbiological cultures taken from passerine birds. The general principles for treating and controlling bacterial infections in passerine birds are similar to those discussed in psittacine birds.

Endoventricular mycosis results from yeast organisms proliferating within the koilin of the ventriculus in finch and finch-like birds. The clinical signs range from unexpected death to weight loss and passing intact seeds in the droppings. Although antibiotic therapy is typically associated with secondary yeast infections, this is not a frequently reported occurrence with endoventricular mycosis. Various conditions such as recent shipping, crowded housing, reproductive activities, and mixed species aviaries are commonly reported as recent stresses. Diagnosis and therapy in the live bird has not reported, as all cases have been necropsy findings.

Paramyxoviruses may be harbored by many finches and canaries. Both type 1 and type 2 infections have been identified in weaver finches. In type 1 infections, clinical signs include conjunctivitis, pseudomembrane formation

in the larynx, and death. Neurologic signs are rare. Canaries rarely develop clinical signs; infected birds should be considered asymptomatic carriers. Because species susceptibility varies, mortality patterns in an aviary may be sporadic and an infectious agent may not be considered as the cause. Type 2 infections occur commonly in African weaver finches and they are considered carriers of this virus. Many infected birds are asymptomatic but others may die following a period of emaciation and pneumonia. Type 3 infections have been identified from a variety of passerine birds including canaries, Gouldian finches and weaver finches. Infection is generally associated with an overall poor condition and central nervous system signs (tremor, paralysis, or torticollis).[1]

Polyomavirus has been sporadically characterized in canaries and others finches where it results in variable morbidity and mortality of young nestling birds. The mortality declines as the birds mature. Gross lesions are rarely recognized and the histologic lesions may consist only of intranuclear viral inclusions and mild lesions in the heart. Excellent husbandry and control of underlying diseases will reduce losses from this viral infection.

Pox virus (see discussion under Amazon parrots) in finches and canaries may produce lesions on the feet and legs, in oral the cavity, and the upper respiratory tract. In a flock outbreak, it generally presents as a fulminating infection with pneumonia. As the outbreak progresses, the typical cutaneous manifestations become more prominent. With the septicemic form, there is an acute onset of ruffled plumage, somnolence, cyanosis, and anorexia. Most birds die within 3 days of developing clinical signs, although the clinical signs can last for months in some birds.

Neoplasia

Lymphosarcoma (malignant lymphoma) occurs more often in canaries with an increased prevalence in males (more frequently presented). Canaries usually have abdominal enlargement, failure to sing, and dyspnea. There may be a leukocytosis and lymphocytosis. Differential diagnosis of visceral lymphosarcoma includes amyloidosis, fatty liver syndrome, atoxoplasmosis (in mynahs and canaries), hepatitis, systemic mycobacteriosis, and other forms of neoplasia. Diagnosis of lymphosarcoma relies on aspiration cytology or biopsy of the affected areas or organs and, if indicated, bone marrow evaluation.

Parasites

In the canary, coccidia within the intestinal mucosal epithelium may represent either *Isospora canaria* or *Atoxoplasma serini*. *Isospora canaria* is strictly an intestinal coccidia and severe infestations have been associated with very young or immune compromised birds. Coccidia may have a direct life cycle or an indirect life cycle. Diagnosis of these parasites is made by examination of a fecal flotation or direct fecal smears, however, oocyst shedding is intermittent so a negative fecal examination is not diagnostic. Atoxoplasma is a common parasitic disease diagnosed in the subadult canary. This host specific protozoan undergoes a sexual phase in mononuclear blood cells. It is transmitted to other birds through ingestion of fecal oocysts. Treatment for either of these parasites with sulfa drugs will only reduce the number of infective oocysts, and has no effect on intracellular stages.

Cochlosoma species are flagellates that inhabit the gastrointestinal tract of some finches. Bengalese finches may be inapparent carriers of this organism; when they are used to foster species of Australian finches (such as Gouldian finches), they may pass the organism on to juveniles, causing high mortality in nestlings. Clinical signs include debilitation, dehydration, and passing whole seeds in the droppings. Necropsy yields an intestine filled with a yellow suspension or whole, undigested seeds. Affected birds are usually 6–12 weeks of age. Identification can be made at necropsy by direct wet preparations of fresh, warm droppings or intestinal contents. *Cochlosoma* has 6 anterior flagella with a helicoidal, anterior ventral sucker. Treatment is with ronidazole or dimetridazole. Water containers should be disinfected and rinsed; the aviary should be kept clean and dry.

Cryptosporidia are intracellular protozoans that parasitize the apical portions of vertebrate occulorespiratory, gastrointestinal, and genitourinary epithelium. Although they are generally opportunistic and secondary invaders, they have been reported as primary pathogens producing respiratory and/or intestinal disease in birds. In cockatiels, lovebirds, and exotic finches, they appear responsible for debilitating diarrhea. Transmission is by ingestion of infective sporulated oocysts.

Sternostoma tracheacolum is the most common respiratory tract mite of canaries and Gouldian finches. This mite is presumed to have a direct life cycle, from the parents to the chicks. The infestations are generally more severe in juvenile birds. The clinical signs include a "clicking," sneeze or cough, dyspnea, voice changes or loss, generalized debilitation, and sudden death from suffocation. Visualization of mites may be possible with a bright light source as tiny, dark, moving, pinhead-sized spots in the trachea.

Toxins

Canaries and finches are particularly susceptible to inhalant toxins because they breathe more air per gram of body weight than larger birds; they also have a highly efficient gas exchange system. Carbon monoxide exposure from any source (car exhaust, gas furnace leaks, and kerosene stoves) can be rapidly fatal. (See discussion under budgerigars.)

Unknown etiology

Amyloid is an insoluble pathologic proteinaceous substance, deposited between cells in various tissues and organs of the body. This amorphous, eosinophilic hyaline extracellular substance encroaches on and results in pressure atrophy of adjacent cells. Systemic amyloidosis can be classified as primary (AL or amyloid light chain), secondary (AA or amyloid associated), or familial. Secondary amyloidosis is most commonly recognized in animals and has been characterized as a reaction to diverse inflammatory stimuli. Primary amyloidosis is less frequently observed and is the result of plasma cell dyscrasia, including both lymphoid and plasma cell neoplasms. Systemic amyloidosis has been reported in finches (especially Gouldian finches), captive domestic and wild Anseriformes especially of Anatidae family (swans, geese, and ducks), and in gallinaceous birds (domestic fowl and turkeys). Affected birds may be found dead. The predisposing factors include stress (environmental or social), inadequate nutrition, and diseases such as endocarditis, mycobacterial infections, staphylococcal infection secondary to fight wounds, and chronic septic arthritis. Antemortem diagnosis of systemic amyloidosis is difficult and specific treatment in any animal species has not been described. At necropsy, the liver and kidneys may appear grossly normal or pale and yellow; on histologic examination they may be severely affected.

Feather follicle cysts (*hypopteranosis cystica*) are hereditary, but the mode of inheritance is not simple; other factors may play a role in the development of the condition. The possibility of a vertically transmitted viral infection causing folliculitis with secondary cyst formation has been suggested. Norwich, crested, crest-bred, and dimorphic new color canaries have been genetically selected to produce an extra down-type feather (double buff) that predisposes them to cyst formation. The pectoral and scapular feather tracts are a frequent site of multiple cysts; the entire tract may be involved. The cysts appear as hard, yellow nodules from the curling trapped feathers. Badly affected birds have irregularly directed feathers all over their bodies. The texture of the material within the cyst will vary depending on the stage of molt. Actively growing feather cysts will have vascular walls and contain blood and gelatinous material. Mature cysts will contain drier keratinous material, and the cyst wall may be more expansive, thickened, and reduced in vascularity. Medical treatment for feather cysts is generally unrewarding. Once mature, the material can be expressed from small cysts but the problem will recur with the subsequent molt. Surgical options for feather cysts include excision of individual cysts, removal of complete feather tracts, or lancing and curetting individual cysts. Excision will remove the affected follicle and may be useful for solitary cysts, particularly those located on the body. Birds with feather cysts should not be used for breeding.

Additional syndromes

These conditions have been commonly identified in this species but more detailed description is provided under the species in parentheses following the condition: cutaneous papilloma (African grey parrots), megabacteria (budgerigars), mycobacteria, (Amazon parrots), and knemidokoptic mange (budgerigars).

Cockatiels

Infectious diseases

Chlamydophila infections occur commonly in cockatiels. Diagnosis can be made by sampling pharyngeal or ocular swabs using DNA-PCR technology, cytology, serology, culture of the organism, antigen detection systems, and antibody detection. These infections cause a variable basophilia and monocytosis. Leukocytosis is more common in acute infections; elevations in AST and bile acids also occur. Clinical signs can vary and include progressive emaciation, greenish diarrhea, occasional conjunctivitis, and high levels of urates in the droppings. CNS signs include an incapacitating flaccid paresis and paralysis. Clinical signs are often subtle and may be overlooked. Cockatiels commonly develop subclinical infections or have a mild form of the disease that presents as a chronic conjunctivitis and sinusitis. (See discussion under Amazon parrots for additional information.)

Bacterial infections include *E coli, Klebsiella, Pseudomonas, Staphylococcus* and occasionally *Salmonella*. Mycobacterial infections are uncommon but typically present as a single granuloma of the ocular adnexa.[9]

Yeast infections occur more commonly in malnourished cockatiels. Severe lesions are probably associated with immunosuppression. Alterations in normal bacterial flora, the integrity of the alimentary mucosa, or immunocompetence can permit an overgrowth (and possible dissemination) of resident yeast (most likely *Candida albicans*). Tissue invasion, which occurs with colonization and penetration of a disrupted epithelial surface, is indicated by the presence of mycelial forms. Factors predisposing avian species to infection include prolonged antibiotic therapy; hypovitaminoses (especially vitamin A); feeding spoiled, stale, or sour foods; a stressful environment with moist floors, dirt nests, and fecal contamination; malnutrition; and co-existing bacterial or viral infections. In hand-fed cockatiels, overfilling the crop or feeding formula at an improper temperature can predispose to crop stasis and fungal overgrowth. Cockatiels, budgies, and lovebirds appear to be highly susceptible, making yeast infections appear to be a primary disease in these species.[3] This disease is more prevalent in young birds, and may progress to a fatal systemic disease. Diagnosis is by clinical signs, history, lesions, and laboratory samples. Culture alone will not diagnose a yeast infection. Therapy must address the underlying conditions leading to infection.

Spirochetosis has been identified in young cockatiels. Infected birds present with pharyngitis.[10] This organism can be identified wet mount smears of the choanal area and can occur in large numbers on the mucosa. Diagnosis is based on pharyngeal Gram stain; the organism is difficult to culture. Histopathology demonstrates a mild inflammatory reaction in the nasal sinus, but not in the trachea. Treatment is with tylosin.

Viral infections most commonly seen in cockatiels include persistent polyomavirus infection (see discussion under Macaws and Budgerigars), psittacine herpesvirus (Pacheco's disease–see discussion under Amazon parrots), adenovirus (see discussion under budgerigars), proventricular dilatation disease (PDD) (see discussion under macaws), and paramyxovirus.

Cockatiel "lock jaw" syndrome results from extensive skeletal muscle fibrosis and inflammation in the muscles of the jaw which presents clinically as "lock jaw" (temporomandibular rigidity). This syndrome, with its distinctive

clinical sign of the inability to open the beak, also includes rhinitis, sinusitis, and moderate to high mortality of affected birds. *Bordatella avium* and *Enterococcus* have been identified as the causative agents of this disease.[10] Associated superinfection with other gram-negative bacteria or yeast is a common finding. Once clinical signs develop, treatment is usually unrewarding.

Neoplasia

Undifferentiated pulmonary tumors of cockatiels are massive, discrete, infiltrative masses in the thorax. They have been recognized in cockatiels ranging from 1 to 10 years in age. When identified early, the tumor appears to arise from mediastinal tissues. Lesions consist of large, firm, white to gray masses that replace areas of the lung. Tumors are so aggressive that they may invade vertebra or extends into the thoracic inlet, causing collapse of the intraclavicular air sac and compression of the trachea. Histologically, sheets of closely placed cells with regions differentiating toward fibroblastic, adipocytic, or chondroblastic cell types have been identified. Many cells have prominent karyomegaly and contain a pale intranuclear inclusion. Electron microscopy suggests a virus that is morphologically consistent with polyomavirus within the nucleus.[8] Antemortem diagnosis is rare and therapy has not been attempted.

Nutritional and metabolic disorders

Articular or synovial gout will present as white swellings over joints, most typically of the feet. It is common in cockatiels and budgerigars. The cause is not completely understood but many of the birds have underlying renal disease. The clinical signs include a shifting leg lameness, inability to bend the toes, and nonspecific feather picking in some species. Aspiration and exfoliative cytology is used to differentiate from granulomas. Therapy is only palliative. Allopurinol has been used as it interferes with the production of uric acid; it has no effect on the deposits in tissues. Surgical removal is controversial; it is not possible to remove all the deposits and profuse bleeding occurs. Aggressive treatment of the underlying kidney disease with fluid therapy, essential fatty acids, and non-steroidal anti-inflammatories is essential.

Chronic liver disease includes the lesions of hepatic fibrosis, bile duct reduplication, and aggregates of granulocytic extramedullary hematopoiesis. The etiology is unknown. These lesions may be the result of previous hepatic insults resulting in parenchymal necrosis (infectious/inflammatory hepatitis, severe vacuolar hepatopathy or fatty liver, chronic congestion from heart disease, or exposure to hepatotoxins). The extramedullary hematopoiesis is a non-specific response to an increased need for bone marrow elements. Hepatic fibrosis seems to be a progressive lesion. The diagnosis is most commonly made at postmortem examination, although the changes are usually diffuse and an antemortem biopsy should be diagnostic. One author (D. R. R.) sees chronic liver disease most commonly in cockatiels and Amazon parrots, with fewer cases in budgerigars, macaws, and cockatoos. Nutritional counseling, antibiotics, lactulose, and nutriceuticals are used in hepatic disease management.

Dietary induced renal mineralization occurs in some cockatiels fed an all-pelleted formulated diet. These birds develop renal mineralization and die. Levels greater than 1% calcium and >2000 IU/kg vitamin D3 are most often associated with these lesions.[10] Glomerulopathies may respond to essential fatty acids (omega 3) and non steroidal anti-inflammatory therapy.

Parasites

Giardiasis (in the United States) was reported 20 years ago as causing pruritis and feather picking in cockatiels. The organism most often identified is probably not G*iardia* but *Hexamita/Spironucleus*. *Trichomonas* and *Cryptosporidia* infections are occasionally diagnosed. Ascaridiasis is a problem in Florida and in Australia.[6]

Reproductive disorders

Excessive egg production is a fairly common problem in single cockatiels. Many of these birds are fed a seed millet and vegetable diet. Managing these cases consists of behavioral counseling (of the owner), light manipulation, leuprolide acetate (Lupron Depot, TAP Pharmaceuticals, Inc, Deerfield, IL, USA), and removing anything the bird perceives as a mate or love object and nesting site.

Egg-binding occurs when an improperly formed (shell-less or prickly shell) egg sticks in the oviduct, the egg is too big to pass, or is breach in presentation. Pressure on the sciatic nerves from the egg can result in paresis; the bird presents depressed and unable to stand. Case management includes radiographs, warmth, fluids, injectable calcium, and vitamin D_3. Some practitioners use prostaglandin F2 alpha or oxytocin. Uterine rupture is possible if the egg will not move as the uterus contracts around it. If the egg does not pass, the bird is anesthetized with isoflurane and after ovocentesis, the egg is removed via the cloaca. If the deflated egg does not pass, then surgical exploratory and removal is indicated. Some of these eggs are actually free in the abdomen, along with the expelled egg contents. Postoperative management includes vitamin D_3 injections, leuprolide, dexamethasone, oral calcium, and antibiotics, along with nutritional and behavioral counseling.

Egg-related coelomitis usually presents with abdominal ascites; occasionally, neurologic signs may be the presenting complaint. There may or may not be a recent history of egg production. Egg material that is free in the coelom incites an inflammatory response. Aspiration cytology reveals a fatty background with a granulocytic or macrophagic infiltrate. Culture may yield cocci or coliforms. Pancreatitis is most common in the reproductively active female; it is usually induced by yolk coelomitis. Temporary diabetes mellitus can occur with plasma glucose levels range between 600–1200 mg/dl. Treatment includes fluid removal, furosemide, and antibiotics for 5–7 days. Pancreatitis usually resolves with medical treatment. While some cases of yolk peritonitis can be resolved with medical therapy alone, radiographs and surgical debridement of the remaining egg material or retained egg, and salpingohysterectomy, may be necessary. Monitoring hemograms will determine therapeutic success.

Toxins

Lead ingestion can occur from nibbling on curtain weights, stained glass art, solder, costume jewelry, wine wrappers, and antiques. Clinical signs include somnolence, ataxia, torticollis, regurgitation and, occasionally, tan urates. Laboratory diagnosis includes erythrocytic ballooning and a responsive anemia on the blood smear, and normal or elevated blood lead levels.

Zinc toxicosis occurs when particulate zinc in ingested. Clinical presentation includes somnolence, marked polyuria, and regurgitation. Anemia is usually not a clinical finding but marked hyperamylasemia can occur. Treatment for both heavy metal toxicities is best accomplished initially with parenteral calcium disodium ethylene diamine tetracetate (CaEDTA) and then with one of the oral chelating agents.

Other conditions

Tail avulsions occur in recently wing-clipped fledglings. The bird falls and the tail is hyperextended. The muscles on the ventral tail base are ripped off the pygostyle on the ventral surface just caudal to the vent. A prolapsed cloaca has a similar clinical appearance. Treatment includes surgical debridement and closure, confinement until healed, and systemic antibiotics.

Millet seed aspiration presents as acute respiratory distress. Placement of an air sac breathing tube and removal by flushing or aspiration can be attempted. Cytology of the flush can help differentiate between millet (normal flush) inhalation and pathologic condition.[10]

Additional syndromes

These conditions have been commonly identified in this species but more detailed description is provided under the species in parentheses following the condition: atherosclerosis (African grey parrots), fibrosarcoma (budgerigars), hepatic lipidosis (Amazon parrots), lipoma (Amazon parrots), lymphosarcoma (Amazon parrots), malassezia (conures), megabacteria (budgerigars), myelolipoma (lovebirds), airborne toxins (budgerigars), and xanthoma (Quaker parrots).

Cockatoos

Infectious diseases

Circovirus infection usually causes beak lesions. (See discussion under lovebirds and African grey parrots.)

Cutaneous herpesvirus lesions can resemble dermal papillomas. These are warty or plaque-like areas of skin thickening and horny layer proliferation occurring on the foot, toes, legs of macaws (*Ara species*), and cockatoos. Nodules are proliferative and may be solitary or multiple, especially in cockatoos. The lesion in macaws is typically a roughening of the skin and/or a flat, raised plaque. De-pigmentation of the diseased tissue is common. Electron microscopy of the lesions occasionally demonstrates viral particles suggestive of a cytomegalic herpesvirus. Mode of transmission is unknown, but is assumed to be by direct contact; however, the virus does not appear to be highly contagious. Treatment attempts include grinding the lesions and acyclovir cream.

Feather picking and mutilation

(See also discussion under African grey parrots.) Feather picking occurs in captivity and this behavior has multiple precipitating factors. Damage occurs to the feathers and skin the bird can reach; companion birds may also be picked around the head and neck. Head feathers are normal, although crested birds may chew on their crests. After a period of time, the picking becomes an obsessive behavior and continues long after the inciting cause is gone. Follicular damage and atrophy results in cessation of feather growth; chronic picking results in abnormal feather development and molt and many advance to self-mutilation. The mutilation can lead to a chronic pruritic ulcerative dermatitis. Internal lesions or infections may be precipitating factors. Pruritis in humans has been associated with hepatopathy; many birds seem to have signs associated with chronic undiagnosed liver disease.

Psychologic feather picking is a diagnosis of elimination. A thorough physical examination, complete blood count (CBC), serum chemistries, feather pulp culture and cytology, skin lesion culture and cytology, radiographs, endoscopic and direct microscopic examination, and biopsies of affected feathers (preferably blood feathers, including the surrounding skin) may yield a reason for the feather destruction.

Management of the feather picking bird is complex. Identification and correction of underlying medical issues may alleviate the behavior. Nutritional aberrations must be identified and corrected. Identification of any psychological and environmental factors is more difficult; a site visit may be necessary. Several central nervous system drug regimens have been suggested. Successful therapy results in a decrease of destructive behavior, not necessarily its cessation.

Additional syndromes

These conditions have been commonly identified in this species but more detailed description is provided under the species in parentheses following the condition: constricted toe syndrome (African grey parrots), hepatic lipidosis (Amazon parrots), lipoma (Amazon parrots), polyomavirus (macaws), proventricular dilatation disease (macaws), and sarcocystis infection (African grey parrots).

Conures

Infectious diseases

Malassezia yeast organisms that have been identified in the keratin of feather follicle and epithelium seem to be a significant finding in pet birds. Although these lipophilic yeast are part of the normal cutaneous microflora of most warm-blooded vertebrates, their proliferation is recognized as a significant finding in seborrheic dermatitis of domestic canines. The pathogenesis of the dermatitis is unknown but probably reflects an abnormal microenvironment of the skin permitting the growth of the yeast. Birds with *Malassezia* dermatitis also have a leukocytosis and many are on marginal diets (deficient in vitamin A and other imbalances).[7] Underlying hypersensitivity disorders are also suspected. One author (D. R. R.) sees *Malassezia* dermatopathy uncommonly but conures are the most frequent bird species, with fewer cases in cockatiels, African grey parrots, Eclectus parrots, Amazon parrots, lovebirds, and macaws.

Unknown etiology

Bleeding syndrome in conures can be managed with appropriate dietary adjustments. While the cause is unknown, it may be related to decreased levels of vitamin K, calcium, and other unknown dietary components. Conures may have a higher requirement for vitamin K. Treatment includes injectable vitamin K_1, vitamin D_3, calcium, and antibiotics. This syndrome may be associated with erythemic myelosis (unregulated production of erythrocyte precursors). Severe bleeding episodes can cause an intense intramedullary erythropoiesis, especially with recycled iron. Histologic examination reveals acute and chronic hemorrhages within various tissues in conjunction with erythrocyte proliferation in the bone marrow, hepatic sinusoids, and splenic red pulp.

Additional syndromes

These conditions have been commonly identified in this species but more detailed description is provided under the species in parentheses following the condition: atherosclerosis (African grey parrots), herpesvirus (Amazon parrots), polyomavirus (macaws), proventricular dilatation disease (macaws), airborne toxins (budgerigars), and constricted toe syndrome (African grey parrots).

Lovebirds

Infectious diseases

Circovirus infection (psittacine beak and feather disease) in lovebirds has 2 clinical presentations. The first is a peracute to acute infection associated with pneumonia, enteritis, feather lesions, and death. The second or chronic form is characterized by progressive feather lesions and occasionally, beak pathology. Virus is shed in the feces, crop secretions, and feather dust. The minimum incubation period is reported as 21–25 days with a maximum period of up to several years. Diagnosis is made by DNA-PCR probes and histopathology. On histologic examination, one author (D. R. R.) commonly sees circovirus intracytoplasmic inclusions within abnormal feathers on lovebirds.

Agapornis (lovebird) pox produces lesions in the oral and nasal cavities, palpebrae, axilla, shoulder, or abdomen. The lesions are dark, discolored, very pruritic areas of skin. Secondary bacterial infections are common. (See discussion under Amazon parrots.)

Neoplasia

Myelolipomas are benign neoplasms in the cutaneous tissues and internal organs of birds. They arise from extra marrow hematopoietic cells and adipose tissue. The tumors are highly vascular and behave like lipomas with a slowly progressive growth. Problems arise when they become excessively large and interfere with physical movement or when the expansive growth results in functional changes of adjacent organs due to tissue compression. The majority of cases in psittacine birds have been described in lovebirds, budgerigars, and cockatiels. Excessive hemorrhage can occur during surgical removal.

Parasites

Microsporidia infections target the liver, kidney, spleen, intestine, and less commonly the eye. Only a single species, *Encephalitozoon hellem*, is documented to infect birds and this same organism can infect immunocompromised humans. Both asymptomatic and clinical infections occur in budgerigars and lovebirds and other species. Immunosuppression is a precipitating cause. Infection occurs most commonly in young lovebirds and budgies (less than 1 year). Infected birds die; survivors are stunted, unthrifty, and have diarrhea. Gross necropsy findings include pasted vents, pale voluminous feces, watery intestinal contents, and undigested seeds in the feces. Microsporidia keratitis presents with a history of a chronic conjunctivitis that is refractory to traditional antimicrobial treatment. Organisms are more common on the cornea than in the adjacent inflamed conjunctiva. Diagnosis in the live bird is made by staining conjunctival smears with calcofluor MR2 stain and viewing the specific organisms under ultraviolet light. The transmission is probably by ingestion of contaminated material. The spores are highly resistant in the environment and can remain infective for months.[5]

Unknown etiology

Polyfolliculitis syndrome is a chronic condition that results in multiple small feather cysts in lovebirds, budgerigars, parrotlets, and cockatiels. Lesions are commonly located around the base of the tail, rump, and mid to upper neck. Clinical appearance is of more than 1 quill projecting from a feather follicle. A definitive cause has yet to be determined, although diseases that affect feather development should be considered (ie, trauma or viral infections).

Additional syndromes

These conditions have been commonly identified in this species but more detailed description is provided under the species in parentheses following the condition: adenovirus (budgerigars), airborne toxins (budgerigars), fibrosarcoma (budgerigars), lymphosarcoma (Amazon parrots), yeast infection (cockatiels), megabacteria (budgerigars), and atherosclerosis (African grey parrots).

Macaws

Infectious diseases

Papillomatosis is characterized by proliferative, grey-white growths on mucous membranes. Recent research links these tumors to a herpes virus etiology. In psittacine birds, association between papillomatosis and bile duct and pancreatic duct tumors is suggested. Reported sites of occurrence include the oropharynx, choanal cleft, conjunctiva, larynx, esophagus, crop, proventriculus, ventriculus, nasal mucosa, nasolacrimal duct, bile ducts, pancreatic ducts, and cloaca. Papillomatosis primarily affects New World psittacine birds especially macaws (the most common), Amazon parrots, and hawk-headed parrots (*Deroptyus accipitrinus accipitrinus*). Regression

and recurrence is cyclic. Noted clinical signs include bleeding from the vent, lethargy, weight loss, cloacal prolapse, and agitation. Anemia is a frequent finding. Cloacal papilloma should be differentiated from cloacal carcinoma, which is infiltrative, aggressive, and typically has areas of necrosis and hemorrhage. Surgical removal of large masses may relieve some clinical signs although recurrence is common.

Polyomavirus causes organ necrosis and basophilic karyomegaly in many tissues. This virus is capable of causing disease in all psittacine species, however; nestling and juvenile birds are the most susceptible to disease. Infection results in nonspecific signs of illness including anorexia, crop stasis, depression, spontaneous cutaneous and feather hemorrhage, paresis, ataxia, and sporadic death. The virus is excreted in feather dander and droppings. Horizontal transmission is the major method of infection. Vertical transmission may also occur. Adults may also be affected. Infection persists in the kidneys of carrier birds and virus is excreted intermittently during times of stress. The virus is infective in contaminated environments. Assays using DNA-PCR probe technology can be used to diagnosis exposed birds and the environment.

Proventricular dilatation disease (PDD) is defined by lymphoplasmacytic inflammatory infiltrates within the peripheral and/or central nervous system. The clinical signs may vary between psittacine species but generally include depression, weight loss, constant or intermittent regurgitation, passage of undigested seed in the feces, ataxia, abnormal head movements, seizures, and proprioceptive or motor deficits. There is variable distention or dilation of the gastrointestinal tract, most commonly noted in the proventriculus. A viral etiology is associated with PDD; however, the mode of transmission, incubation, and disease development is unknown. One author (D. R. R.) identifies PDD most commonly in macaws (the youngest at 4 months), African grey parrots, conures, cockatoos, and Eclectus parrots.

Nutritional and metabolic disorders

Vitamin D_3 toxicity usually results from unnecessary supplementation. The blue and gold and hyacinth macaw is prone to the development of hypervitaminosis D_3. Increased vitamin D_3 supplementation may result in crop stasis, increased serum uric acid levels and the presence of articular gout, and regurgitation after feeding. Widespread soft tissue calcification can occur.

Lateral beak deviation (scissors beak) is most often diagnosed in macaws. The cause is unknown and may include low or unbalanced calcium in the diet, viral diseases, trauma, improper handfeeding techniques, and alterations in the mandibular occlusal surface, If recognized early, the problem may be corrected by trimming the lower beak, reshaping the occlusal surface of the rhamphotheca, and therapeutic massage. If that is not successful, a ramp built from dental acrylic over a stainless steel mesh can be attached to the lower beak to apply pressure to correct the upper beak. Careful monitoring is necessary so as not to overcorrect the beak.

Macaw thyroid hyperplasia (hyperplastic goiter) has been recognized in macaws and particularly blue and gold macaws. The cause of the condition was not determined with certainty in most cases. Possible causes include iodine deficiency, goitrogenic substances, and hereditary biosynthetic defects. Clinical signs in affected birds were either nonspecific or related to the other disease conditions present.[5]

Unknown etiology

Chronic obstructive pulmonary disease (macaw pulmonary hypersensitivity) is generally associated with polycythemia and appears to be most common in the blue and gold macaw (*Ara ararauna*). Early diagnosis is difficult due to the reserve capacity of avian lungs and relative inactivity of captive macaws. The lung lesions are generally advanced when polycythemia occurs. Signs may include dyspnea, tachypnea, and radiographic changes in the lungs. The hematocrit is often > 60%. A leukocytosis may be present if there is a secondary infection. The

prominent lesion is atrial smooth muscle hypertrophy and some atrial loss due to fusion and epithelial bridging. Occasional proliferation of parabronchial lymphoid tissue and lymphoid nodule formation has been identified. Many of these macaws have been kept in aviaries with poor ventilation or exposed to the feather dander of cockatoos, cockatiels, or African grey parrots. Therapy is generally symptomatic that includes placing bird in better ventilated areas, treating for secondary infections, and possibly the use of anti-inflammatory drugs.

Additional syndromes

These conditions have been commonly identified in this species but more detailed description is provided under the species in parentheses following the condition: constricted toe syndrome (African grey parrots), cholangiocarcinoma (Amazon parrots), lymphosarcoma (Amazon parrots), hepatic lipidosis (Amazon parrots), fungal pneumonia (African grey parrots), cutaneous herpes (cockatoos), and herpesvirus (Amazon parrots).

Quaker Parrots

Nutritional and metabolic disorders

Pancreatic necrosis is most commonly recognized in Quaker parrots. Many of these birds die suddenly without significant clinical signs. Gross lesions include a firm, pale pancreas, variable hemorrhage, and adjacent fat necrosis characterized by firm yellow-white foci. The exact cause is unknown; high-fat diets have been implicated.

Unknown etiology

Xanthomas are not neoplastic, although they are locally invasive mass lesions. They are most commonly found over the ventral abdomen, thighs, hips, wings, eyelids, and around the face; they are rarely described within internal organs. These masses are composed of foamy macrophages and cholesterol clefts, which produce a thickened and dimpled skin that is yellow to orange in color. Lesions may be pruritic and are often associated with other pathology, such as lipomas, hernias, and sites of chronic irritation. They have been reported in psittacine birds (particularly Quaker parrots, female budgerigars, cockatiels, and Amazon parrots) and gallinaceous birds. Therapy includes surgical resection, although with diffuse tissue involvement there may be insufficient normal skin to close the defect. A guarded prognosis is given for xanthomas covering large areas or having indistinct borders.

Additional syndromes

These conditions have been commonly identified in this species but more detailed description is provided under the species in parentheses following the condition: fibrosarcoma (budgerigars), hepatic lipidosis (Amazon parrots).

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