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Chapter 27

Sphenisciformes, Gaviiformes, Podicipediformes, Procellariiformes, and Pelecaniformes

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INTRODUCTION

The avian order Aequorlitorithes consists of shorebirds, flamingos, grebes, gulls, tropicbirds, penguins, and other waterbirds. Five groups of this order are represented here: Sphenisciformes, Pelecaniformes, Gaviiformes, Podicipediformes, and Procellariiformes.

The Sphenisciformes (penguins) are pelagic birds with a lifespan of 25–40 years in captivity. The 6 genera and 18 currently recognized species range from cold tolerant species populating Antarctica and sub-Antarctic areas to temperate species living near the equator (Miller and Fowler, 2015). Sexual maturity in these generally monogamous birds usually occurs around 3–5 years of age.

Pelecaniformes include pelicans (Pelecanidae), cormorants, and shags (Phalacrocoracidae), anhingas (darters; Anhingidae), boobies and gannets (Sulidae), frigate birds (Fregatidae), and tropicbirds (Phaethontidae). Shoebills and hamerkops are included under current classifications. These birds are long-lived (40 years for captive pelicans, 30 years for free-ranging frigate birds). All feed on fish, aquatic vertebrates, and invertebrates. Monogamous mating is typical. Apart from tropicbirds, eggs are incubated using foot webs, limiting clutch size to one or two eggs. Neonates are precocial. They breed near freshwater but migrate and overwinter in marine environments where they may congregate.

Approximately 22 species of Podicipediformes exist worldwide. Like loons they are underwater hunters but they are more varied in size. Grebes inhabit freshwater wetlands but may overwinter in marine environments, congregating in large numbers as they migrate. Neonates are precocial. Molting occurs away from breeding grounds. Mostly they are solitary or live in small groups.

The Gaviiformes order comprises five species, distributed worldwide, known as loons in North America and as divers in the Old World. They are heavy, long bodied birds adapted for underwater hunting. Neonates are precocial. They breed near freshwater but migrate and overwinter in marine environments where they may congregate.

The order Procellariiformes includes four families: albatrosses and mollymawks (Diomedeidae), petrels and shearwaters (Procellariidae), storm petrels (Hydrobatidae), and diving petrels (Pelecanoididae). Although found worldwide, Procellariiformes predominantly inhabit the southern hemisphere. There is considerable size variation between the smallest storm petrels (adults 20 g) to the albatrosses (adults exceeding 10 kg). They are long lived: up to 20 years for the smaller petrels, 50–70 years for albatrosses. All Procellariiformes are pelagic, accomplished long-distance soaring fliers that spend months at sea feeding on fish, larger invertebrates, phytoplankton, and krill. Landfall is infrequent and occurs mainly for breeding and nesting. This is typically colonial on remote islands, on the ground or in burrows. Young are altricial to semialtricial with well-developed thermoregulatory capacity. Introduced predators, island habitat destruction, and intensive fishing pose threats. Many species, particularly albatross, are threatened or at risk of extinction.

A more detailed overview of the above groups is available (Padilla, 2014).

UNIQUE FEATURES

Sphenisciform feathers are short, dense, and highly water proof. Penguins lack a crop; the stomach is disproportionately large facilitating consumption of whole fish.
Sphenisciform bones are dense and apneumatic. Salt glands are well-developed (Fig. 27.1) but atrophy in captive fresh water environments without salt supplementation. Prior to molt, penguins gain more than 25% of their body weight, which is then lost again during the process. This is pertinent to body condition scoring. An insulating subcutaneous fat layer in penguins far exceeds that of other birds and resembles the blubber layer of seals (Miller and Fowler, 2015; Watson, 1883).

Typical features of most Pelecaniformes include the totipalmate foot, presence of a gular pouch, highly reduced tongue and hyoid structures (gular fluttering during open-mouthed breathing enables evaporative cooling), lack of a brood patch, vestigial intestinal ceca, and a bilobed uropygial gland. Skeletal pneumatization varies. Among diving species, anhingas have apneumatic postcranial skeletons, and cormorants are heavy-boned with pneumatization of only the humeri or caudal cervical vertebrae. In contrast, pelicans, being soaring fliers, have extensive skeletal pneumaticity, except for the femur. In pelicans, gannets and boobies, diverticula extend from the air sacs to form a substantial cranoventral subcutaneous air cushion system, which should not be misdiagnosed as pathological subcutaneous emphysema (Fig. 27.2). This anatomical feature is thought to provide cushioning for water entry during diving from height. As a diving adaptation, many Pelecaniformes have reduced (e.g., frigatebirds, pelicans) or obliterated (e.g., gannets, anhingas) external nostrils, and breathing (and access to the olfactory system) are via the open mouth or jugal operculum at the commissure of the bill (e.g., boobies, cormorants). Male frigatebirds have neck skin pouches with bare overlying skin that is inflated for display.

Gaviiformes and Podicipediformes are adapted to swimming and diving and have predominantly nonpneumatized bones and reduced air sacs (cervical sacs are absent or vestigial in most loons and grebes). Both groups have moderately sized, paired intestinal ceca, and grebes have particularly large uropygial glands. A unique feature of grebes is autophagy of feathers that are compacted into gas tric pellets and regularly expelled with indigestible debris (Fig. 27.3). This slows gastric emptying time and contributes to parasite control.

The Procellariiformes have external tubed nares and a highly developed sense of smell. Except for diving petrels, they produce stomach oil, a complex residue of neutral dietary lipids that is used in self-defense and by chicks and adults as an energy-rich food source.

**NON-INFECTIOUS DISEASES**

**Nutritional**

Nutritional deficiencies are uncommon in captive penguins and associated with feeding poor quality, improperly handled, or inadequately supplemented fish. Poor parental care or miscalculation of the mineral formulation may result in nutritional metabolic bone disease. Affected chicks fail to thrive and develop folding fractures of the tibiotarsus, rib fractures, and luxation of the gastrocnemius tendon. Radiographic reduction in bone density is apparent. There are occasional reports of rickets in zoo-captive double-crested cormorant chicks and fibrous osteodystrophy in free-ranging royal northern albatross chicks receiving supplemental hand-feeding on their nests. Deterioration of fat soluble vitamin D during frozen storage, feeding of fillets rather than whole fish, and peroxidation of stomach oil used as a supplement were contributing factors. In penguins, histologic findings include marked diffuse osteopenia predominantly affecting long bones (thin, irregular trabeculae, closely-spaced reversal lines, thin surface seams of osteoid) (Adkesson and Langan, 2007). Osteopenia and fibrous osteodystrophy predominate in the other species,
with rachitic elongation and widening of the metaphyseal cartilage in cormorants. Parathyroid glands may be grossly normal but histologically abnormal and hypertrophied with chief cell vacuolation.

Thiamine deficiency occurs in captive piscivores fed nonsupplemented diets. Torticollis, incoordination and “stargazing” are observed in affected little penguins. Histological features have not been described but polioencephalomalacia is reported from other susceptible bird species (Ladds, 2009). Vitamin A deficiency leading to periocular gland squamous metaplasia occurs infrequently in Sphenisciformes.

Hypovitaminosis E following feeding of frozen-thawed fish with inadequate dietary supplementation is reported in zoo-captive pelicans, cormorants, and rarely penguins. Dietary antagonism by excess vitamin A may sometimes play a role. Birds present dead or with lethargy, weakness and cutaneous edema, heterophilic leukocytosis, and elevated plasma creatinine levels. Necropsy findings include pale and streaked striated musculature, pericardial effusion, necrotic adipose tissue, and subcutaneous hemorrhage in the absence of trauma (Fig. 27.4A). Polyphasic multifocal rhabdomyolysis, myocardial necrosis, and necrotizing pansteatitis are histological features (Fig. 27.4B). Hypervitaminosis E, due to excess dietary supplementation, caused severe coagulopathy with extensive subcutaneous, muscular, and intracelomic hemorrhage in the absence of concurrent myopathy in a group of pink-backed pelicans (Nichols et al., 1989).

Starvation is a significant problem in free-ranging penguins, particularly chicks and juveniles, and can result in mass mortality events. Free-ranging Procellariiformes, including shearwaters and prions of all ages, frequently suffer mass mortality events (“wrecks”) of hundreds to thousands of birds. These events occur predominantly during migration and are related to adverse weather and/or inadequate food. Most affected birds are emaciated juveniles with reduced bodyweight compared to birds surviving migration, pectoral muscle atrophy, an empty stomach, complete fat exhaustion with serous atrophy, and multiorgan atrophy. Investigations exclude infectious disease, heavy metals, or persistent organic pollutants but sepsis and dehydration sometimes play a role. Mass downing of migrating, free-ranging eared grebes is seen periodically during the fall in North America; affected birds are typically adult birds in good body condition (Roberts et al., 2014). Events are associated with adverse weather or poor visibility. This species requires water for landing and take-off; downed birds may die on impact or remain trapped on the ground and subsequently die. Winter mortality of free-ranging common loons is seen periodically in Florida (United States) between December and April. Common findings include emaciation with pectoral atrophy, fat exhaustion, and hemorrhagic enteritis. Synergistic etiologies include inadequate food supply, the high energetic cost of migration and molt, poor weather and parasitism. Mass mortalities that sometimes involve thousands of birds also occur from idiopathic emaciation in free-ranging nesting American white pelican breeding colonies. Starvation and hypothermia due to adverse weather are implicated.

Metabolic

Neuronal storage disease consistent with sphingolipidosis occurred in five related adult female captive Humboldt penguins. Clinical signs included slowly progressive lethargy, weakness, dysphagia, and ataxia (Wuenschmann et al., 2006). Fine clear vacuolation within the perikarya of swollen neurons and margination of the Nissl substance was the histological hallmark. Axons contained fine clear vacuoles and infrequent spheroids. Multilayered concentric lamellar bodies resembling stored gangliosides were identified by transmission electron microscopy (Fig. 27.6). High cumulative doses of chloroquine, a lysosomotropic amine used for antimalarial treatment may have induced disease, raising concerns over long-term high dose administration.

Toxic

Dose dependent voriconazole toxicity occurs in multiple penguin species following antifungal treatment (Hyatt
et al., 2015). No specific gross lesions are identified. Central nervous system myelinic edema and acute pulmonary congestion are suggestive histopathologic findings but death is primarily due to unsuccessful treatment of the fungal infection. Hepatotoxicity, as described in other avian species, is not a feature in penguins with voriconazole toxicity; however, hepatotoxicity resulting in anorexia, weight loss, and hepatic swelling, discoloration, and necrosis can occur in captive king penguins treated with itraconazole (R. Pizzi, personal communication).

Penguins and American white pelicans are susceptible to fenbendazole toxicity. Intoxication of African penguins was accidentally induced by feeding fish dosed with 66–77 mg/kg of fenbendazole (S. Redrobe, personal communication). It causes death 5–9 days post-treatment. Necropsy findings include empty gastrointestinal tracts, stomatitis (pelicans), and inconsistent hepatic, renal, or splenic enlargement. Intestinal crypt cell necrosis is seen histologically, with villous architectural collapse, superficial mucosal necrosis, and secondary bacterial colonization (Fig. 27.5). Necrotic or regenerating epithelial cells line remaining crypts. Intestinal lesions mirror fenbendazole toxicity in other avian species including pigeons, storks, and vultures. Nonspecific radiomimetic bone marrow suppression and splenic lymphoid depletion are additional features.

Toad poisoning may occur in penguins living in temperate climates, particularly when toads are migrating. Affected penguins die suddenly, in good condition and without clinical symptoms. Ingested toads are found in the upper gastrointestinal tract together with large quantities of mucus (sialorrhea). Biogenic amines and steroid derivatives present in bufonid toad venom act similarly to digitalis and inhibit the sodium-potassium pump in cardiac myocytes. Histological abnormalities are not observed.

Many reports documenting toxins involve surveillance detection of tissue contaminants in free-ranging species, particularly persistent organic pollutants and heavy metals. Disease and/or pathology are frequently absent but it has been suggested that such pollutants may contribute to hepatopathy in penguins. High levels of mercury and cadmium have been associated with proximal renal tubular epithelial necrosis in Manx shearwaters and northern fulmars. Organochlorines caused reproductive failure, including extinctions, in brown pelican colonies during the 1960s.
Residues of 1,1-dichloro-2,2-bis (p-chlorophenyl) ethylene (DDE) caused thinning of brown pelican eggsheels leading to fragility and clutch failure. Regulations restricting the use of DDE in the 1970s have resulted in a decrease in environmental residues and restoration of normal shell thickness and reproductive success (Blus et al., 1977).

Free-ranging pelicans, cormorants, and particularly loons are susceptible to lead intoxication, mainly by ingestion of fishing tackle and ammunition. Similar items and coins may be ingested by penguins. Lead paint is a reported cause of intoxication in Laysan albatross chicks. Clinical signs include inability to fly, drooped wing posture, and bright green diarrhea. Lead in the gastrointestinal tract can be identified radiographically. Necropsy lesions can be absent or include pectoral atrophy, fat depletion, esophageal/proven-tricular impaction, ventricular erosions, gallbladder distension, and lead objects in gastric content. Histologic lesions may include rare acid-fast intranuclear inclusions in renal tubular epithelial cells and hepatocytes, and Wallerian degeneration of peripheral nerves. Bench top analysis of tissue fluids shows good correlation with liver lead concentrations in loons (Kornetsky et al., 2013). Lead tissue concentrations and their clinical relevance in birds are listed in Table 27.1A.

**Type C botulism** causes large die-offs of free-ranging American white pelicans (and smaller numbers of brown pelicans) in southern California (United States). Piscivores (common loons, red-throated loons, and horned grebes) are more typically affected by type E botulism. Ingestion of fish is implicated, rather than the carcass-maggot cycle associated with type C botulism in waterfowl. Outbreaks in loons typically occur in the fall (September) while pelican outbreaks may occur earlier in the summer. Elsewhere, pelicans, cormorants, grebes, and occasionally northern fulmar and Laysan albatross have contracted type C botulism. Botulinum toxins inhibit neurotransmission by blocking secretion of acetylcholine from peripheral cholinergic nerve terminals in the motor and autonomic nervous systems. Progressive paresis and paralysis lead to inability to fly, paddling wing movements across water, and paralysis of the nictitating membranes and the neck (limber neck). Diagnosis is based on history, clinical signs, absence of other causes of paresis/paralysis on gross, and histologic examination (e.g., trauma, inflammation), and laboratory confirmation of botulinum toxin in blood or tissue. Mouse bioassay remains the gold standard for diagnosis but antigen-capture ELISA testing of blood and serum for type C toxin has been used successfully in birds.

Aquatic birds feeding predominantly on fish and invertebrates are susceptible to *algal biotoxins* generated during harmful algal blooms. Pathobiology and epizootiology have been reviewed in detail elsewhere (Landsberg et al., 2007). Marine dinoflagellates and diatoms produce neurotoxic saxitoxins, brevetoxins, and domoic acid; freshwater cyanobacteria produce neurotoxic saxitoxins and anatoxins, hepatotoxic microcystins, and dermatotoxics. Exposure is through bioaccumulation in prey species or drinking contaminated freshwater. Coastal piscivores hunting pelagic prey, such as cormorants and shags, are particularly at risk. Neurotoxins cause signs including lethargy, disorientation, paresis, paralysis, tremors and muscle fasciculations, pupillary constriction, torticollis, loss of righting reflexes, seizures, and death by respiratory failure or drowning. Vomiting, diarrhea, excess oronasal and glandular discharges, dyspnea, and tachycardia may be seen. There are no specific gross or histologic lesions but birds may be either emaciated or in good condition with stomachs full of prey. Concurrent, nonspecific findings include intestinal hemorrhage, catarhal enteritis, hemorrhage and necrosis of striated muscle. *Microcystins* are the most common form of cyanobacterial toxicity; affected birds may be jaundiced with gross and histological evidence of massive hepatic necrosis and hemorrhage.

Algal and other toxins affecting these orders are listed in Table 27.1B.

### CONGENITAL/GENETIC

**Congenital defects** are uncommonly recorded in free-ranging or captive penguins. It is likely that congenital and genetic abnormalities in free-ranging populations of all bird species are overlooked, since long-term survival to allow identification of affected birds is unlikely. Low levels of beak abnormalities, carpal rotation, and hypopigmentation have been described in free-ranging double-crested cormorant chicks. Occasional case reports describe congenital cardiac anomalies in penguins, among which

| Lead Tissue Concentrations (Liver, Kidney) | Clinical Significance |
|------------------------------------------|-----------------------|
| <2 mg kg⁻¹ ww                            | Background level       |
| 2–6 mg kg⁻¹ ww                           | Subclinical poisoning  |
| 6–15 mg kg⁻¹ ww                          | Clinical poisoning     |
| >15 mg kg⁻¹ ww                           | Severe clinical poisoning and death |
| 27 and 107 mg kg⁻¹ ww                     | Chronic lead poisoning |
| Toxin                        | Captive/Free-Ranging | Toxic Effect                  | Species                                                                 | References                                                                 |
|-----------------------------|----------------------|-------------------------------|-------------------------------------------------------------------------|---------------------------------------------------------------------------|
| Anatoxin                    | F                    | Neurotoxin                    | Loons                                                                   | Reviewed in Landsberg et al. (2007)                                        |
| Brevetoxin                  | F                    | Neurotoxin, hemolytic          | Yellow-eyed penguins, Brandt’s cormorants, double-crested cormorants, pelagic cormorants, frigatebirds, pelicans | Gill and Darby (1993); Reviewed in Landsberg et al. (2007)                |
| *Clostridium botulinum*     | F                    | Neuromuscular                 | American white pelican, pelicans, cormorants, grebes, northern fulmar, Laysan albatross | Rocke and Bollinger (2007); Rocke et al. (2004, 2005)                     |
| type C                      |                      |                               |                                                                          |                                                                           |
| *Clostridium botulinum*     | F                    | Neuromuscular                 | Common loons, red-throated loons, horned grebes                         | Rocke and Bollinger, (2007)                                               |
| type E                      |                      |                               |                                                                          |                                                                           |
| Domoic acid                 | F                    | Neurotoxin                    | Penguins, Brown pelican, Brandt’s cormorants, double-crested cormorants, pelagic cormorant | Broadbent (2009); Reviewed in Landsberg et al. (2007)                     |
| Lead                        | F                    | Neurotoxin, emaciation syndrome, nephrotoxicity | Potentially all species, particularly common loons, pelicans, cormorants, Laysan albatross | Friend and Franson (1999); Pokras et al. (2009); Sider et al. (2003); Sileo and Fefer (1987); Stone and Okoniewski (2001) |
| Mercury                     | F                    | Neurotoxin, emaciation syndrome, impaired reproduction, nephrotoxicity | Common and red-throated loons, brown and American white pelicans, gannets, double-crested cormorants, fulmars, shearwaters | Friend and Franson (1999); Nicholson and Osborn (1983)                    |
| Microcystin                 | F                    | Hepatotoxic                   | Great crested grebes                                                   | Reviewed in Landsberg et al. (2007)                                        |
| Oil                         | F                    | Loss of waterproofing and insulation, gastroenteritis, teratogenicity/embryotoxicity, immunosuppression, endocrine, renal, hepatic and hematological abnormalities, impaired osmoregulation | All, particularly penguins, grebes, loons, pelicans, cormorants          | Balseiro et al. (2005); Butler et al. (1986); Crawford et al. (2000); Friend and Franson (1999); Fry et al. (1986); Leighton (1985,1986); Velando et al. (2005); Wolfaardt et al. (2009) |
| Organochlorine toxicity     | F                    | Neurotoxin, reproduction (thinned egg-shells) | Australian pelicans (dieldrin), pelicans, cormorants (DDT), pelicans, cormorants (cyclodiene) | Friend and Franson (1999)                                                 |
| Organophosphates/carbamates | F                    | Neurotoxin                    | Rare                                                                    | Friend and Franson (1999)                                                 |
| Polychlorinated bisphenyls | F | Behavioral, reproductive, cardiovascular (?) | Double crested cormorants, American white pelicans | Friend and Franson (1999) |
|---------------------------|---|----------------------------------|--------------------------------|---------------------------|
| Saxitoxin                 | F | Neurotoxin                        | Pacific loon, black-footed albatross, sooty shearwater, European shag, northern gannet, northern fulmar, great cormorant | Reviewed in Landsberg et al. (2007) |

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Septal defects appear to be most common. Ventricular septal defect (VSD) consistent with Eisenmenger VSD was reported in a 45 day old female Humboldt penguin (Laughlin et al., 2016). Ventricular septal defects have also been reported in a juvenile rockhopper penguin, a captive macaroni penguin and a 27-day-old Adélie penguin (Fig. 27.7). Two cases of atrial septal defects occurred in gentoo penguin chicks with cardiomegaly (J. St. Leger and R. Pizzi, personal communications).

Two cases of unilateral micromelia are described in free-ranging juvenile little penguins (Raidal et al., 2006), and an outbreak of craniofacial deformity occurred in free-ranging yellow-eyed penguin chicks in New Zealand (Buckle et al., 2014). Malformed beaks and the absence of flippers have been observed in little penguins (Reilly and Balmford, 1975). These examples raise suspicion of teratogenic exposures in breeding colonies. Congenital hemivertebrae with spinal cord compression were diagnosed in a 10-week-old kyphotic African penguin (Bradford et al., 2008).

AGE-RELATED/DEGENERATIVE

Degenerative joint disease is uncommon in free-ranging and captive penguins. Among adults, 16% of rockhopper, 4% of gentoo, and 2% of king penguins examined at a zoo in the United Kingdom had osteoarthritic changes, most commonly affecting the coxofemoral and stifle joints (R. Pizzi, personal communication). Bilateral degenerative joint disease was diagnosed in a free-ranging, juvenile, yellow-eyed penguin with abnormal stance and decreased mobility (Buckle and Alley, 2011). Necropsy identified bilaterally distended, thickened coxofemoral joints with increased laxity, and small, roughened and angular femoral heads (Fig. 27.8). Histologically, the left femoral articular cartilage and subchondral bone were absent. The remaining femoral head consisted of trabecular bone overlain by fibrin and granulation tissue. Evidence of infection was absent. Osteonecrosis and osteochondrosis with subsequent bilateral, aseptic degenerative changes of the femoral head was the suspected pathogenesis.

The importance of atherosclerosis varies within captive penguin collections. In one review a 2% incidence with no associated mortality was noted while in another, 37% of penguins had atherosclerosis, predominantly as an incidental finding. Adélie and emperor penguins were most commonly affected. The reported prevalence may be influenced by the age of captive populations, species, dietary or climatic differences, and specific interests of the examining pathologist.

Despite their longevity, age-related/degenerative problems in zoo-captive Pelecaniformes are poorly documented. They include arthritis, arteriosclerosis, and hyperkeratosis of the tibiotarsus and tarsometatarsus in shoebills. Catarracts are the most commonly reported age related ocular pathology in penguins, and recent evidence suggests that cataracts and vitreal degeneration may be under-recognized in zoo-captive adult pelicans (Bliss et al., 2015; O’Connell et al., 2017). Reports are scant in free-ranging or captive populations of Procellariiformes, Gaviiformes, or Podicipediformes. Most aged birds probably die at sea rendering them unavailable for examination.
INFLAMMATORY NON-INFECTIONOUS

Due to their curiosity, penguins, particularly juveniles and nesting females are prone to investigate and ingest novel items. These may include nesting and plant material, such as sticks and stones or bristles from cleaning utensils, coins, fence clips, nails, lead pellets, molted tail feather shafts, enrichment items, and items introduced into enclosures by members of the public (Fig. 27.9). Gastrointestinal foreign bodies are frequent in captive and free-ranging penguins, and cause significant morbidity and even death. Ingested objects are often retained in the stomach. Radiographically, it can falsely appears that objects are located in the distal intestine near the cloaca. Dehydration, lethargy, weakness, anorexia, celomic distension, weight loss, regurgitation, vomiting, ill thrift, diarrhea, scant feces, and extended molt are presentations of acute and chronic gastric impaction or obstruction. Foreign bodies are readily identified at necropsy. Associated pathology ranges from mild mucosal irritation and ulceration to transmural foreign body penetration/perforation with localized to diffuse celomitis and bacterial sepsis. Gastric impaction with feathers has been reported in penguin chicks and phytobezoar formation may lead to chronic obstruction in adults. Sporadic cases of gastrointestinal impaction and perforation due to foreign body ingestion are seen in zoo birds, particularly pelicans, and in free-ranging birds that tackle oversized or toxic prey items (e.g., puffer fish).

Plastic foreign bodies are commonly found in the gastrointestinal tracts of beached, bycaught, and free-ranging pelagic species trapped for research purposes worldwide, particularly Procellariiformes. Plastic ingestion leads to lower fledging weight in Laysan albatross chicks but does not appear to affect chick survival. Environmental plastic debris kills by entrapment and physical entanglement but evidence for detrimental effects of ingested plastics on digestion or body condition, including persistent organic pollutant accumulation, is surprisingly weak (Herzke et al., 2016). Alternative causes of death should be sought despite the presence of ingested plastic.

The underlying causes of pododermatitis (bumblefoot) in birds are multiple. Predisposing factors in captive penguins include sedentary behavior, decreased swimming and prolonged standing on abrasive, hard, moist, or fecally contaminated flooring. Similar considerations apply to pododermatitis in pelicans and shoebills. The primary lesion is ischemic pressure necrosis of soft tissues of the foot, or an initial puncture wound that compromises perfusion of the dermis and leads to erosion and ulcer formation. Affected animals present with abnormal gait, increased resting behavior, and discomfort on foot palpation. In nontraumatic lesions, unilateral or bilateral swelling and increased redness of one or multiple areas of the footpads followed by epithelial thinning, ulceration, hemorrhage, and scab or granulation tissue formation are seen on the plantar surfaces of the feet (Fig. 27.10). Histological findings include skin ulceration, laminated parakeratosis, serocellular crusting with active inflammation, and variable bacterial colonization. Adjacent epidermis is hyperkeratotic and hyperplastic with irregular acanthosis or atrophic thinning. Early cases have mild pleocellular perivascular dermatitis. Advanced cases exhibit severe perivascular inflammation, edema, and collagen degeneration resulting in necrotizing vasculitis. Inflammation may be diffuse and extends deeply into subjacent tissues. Secondary, opportunistic bacterial infection occurs frequently with Escherichia coli, Proteus mirabilis and Staphylococcus, Enterococcus, Pseudomonas, and Clostridium species, or invading fungi. Untreated lesions can evoke soft tissue mineralization and ascending necrotizing osteomyelitis and/or tenosynovitis, and may trigger the
Like other aquatic species, free-ranging aquatic bird species can be entrapped and drowned in marine bird species and fishing gear, including nylon drift nets, gill nets, and baited hooks. Closer to shore, nets catch pursuit-diving species including cormorants, loons, grebes, and penguins. Pelagic long-line fishing contributes to declines in albatrosses, petrels, boobies, and frigate birds. Most drowning cases occur in the absence of underlying disease. In contrast to birds stranding or affected by pollution, drowned birds have well-developed muscles, good nutritional condition (including subcutaneous fat), and their stomachs frequently contain recently ingested fish. The air sacs or distal trachea may contain clear watery fluid. The lungs are often congested and edematous, exuding white frothy supernatant-rich fluid from cut surfaces. The heart and major veins, including the dural venous sinuses, are grossly distended (Simpson and Fisher, 2017). Histologic lesions are not pathognomonic but aquatic environmental debris in severely edematous lungs is supportive.

**Neoplastic**

The reported overall prevalence of neoplasms in captive penguin species is low. Malignant melanomas are reported with the highest frequency, occurring in macaroni, Humboldt, and rockhopper penguins. Predilection sites are the skin of the foot or hock, and subcutis and subjacent muscle near the beak and oral cavity. Humboldt penguins may have a unique presentation, with variably pigmented, cornified lesions in the inguinal area. Histologically, neoplastic cells are pleomorphic and heavily pigmented; greater than 50% contain melanin granules. Junctional activity is prominent. Surface ulceration correlates with systemic spread and metastases occur to the liver, lung, adrenal gland, brain, and bone. Other prognostic factors include gross surface dimensions, mitotic index, and depth of neoplastic cell invasion. Amelanotic melanomas have not been reported and diagnosis is based on typical morphological findings. PNL-2 can be used for immunohistochemical confirmation; S-100, Melan-A, and Ki67 cannot. Average survival is 7 months. The pathogenesis underlying the high prevalence of melanocytic neoplasms in captive penguins is unknown. Advanced age is most probable. There is no evidence to suggest involvement of ultraviolet light or chlorine exposure, viral induction, or genetic predisposition (Duncan et al., 2014).

Other reported neoplasms include, in descending frequency, adenocarcinoma, squamous cell carcinoma, malignant lymphoma, cholangiocarcinoma, and single cases of orbital round cell tumor and anaplastic sarcoma. Adenocarcinomas primarily affect either compartment of the stomach with a few cases of reported intestinal and urogenital origin. Gastric adenocarcinomas are firm, pale masses that expand the gastric wall (Yonemaru et al., 2004). Transmural growth results in serosal adhesions. Gastrointestinal adenocarcinomas are histologically typical (Fig. 27.11). Apparent predilection for the proventricular/ventricular junction can

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**FIGURE 27.10** Severe chronic pododermatitis (bumble foot) in an adult Macaroni penguin. Focally extensive chronic ulceration with exuberant granulation tissue formation, urate contamination and marginal dermal hyperplasia overlies the joint. (Photo Courtesy of J. St. Leger, SeaWorld Parks & Entertainment)

development of systemic amyloidosis. Introduced foreign material incites granulomatous foreign body reactions with giant cell formation. Prevention is paramount, focusing on environmental enrichment, encouragement of swimming, and avoidance of hard, rough, wet, or contaminated surfaces.

The specific etiology of uropygial gland infections in penguins is unknown. However, infection has not been recorded in free-ranging penguins, suggesting a multifactorial process related to captive environments. Predisposing factors include sedentary habit and reduced swimming activity, poor plumage, failure to preen, irregular molt, and nutritional deficiencies. Glands are enlarged and swollen and contain purulent or caseous material. Cultures often yield mixed bacteria. *Candida* is commonly isolated, even following antifungal treatment. Rupture and secondary septicemia may develop. Histology may suggest underlying vitamin A deficiency but supplementation does not resolve the condition. Early diagnosis and treatment may prevent impaction. Once preen gland infection has been established in a bird, future episodes are likely (Schneider et al., 2014).

Trauma due to predatory attack is a significant cause of morbidity and mortality in free-ranging penguins and many of the colonial nesting seabirds, particularly ground-nesting or burrowing island-dwelling Procellariiforme species. Introduction of feral rodents and predators, such as cats or primates, can threaten extinction.
render it difficult to differentiate origin based on histomorphological features. Cytoplasmic positivity with alcian blue and periodic acid-Schiff is helpful in confirming ventricular origin and positive galactose oxidase-Schiff staining can confirm proventricular origin. Ventricular adenocarcinomas may metastasize to pancreas and intestines.

**Uropygial gland squamous cell carcinoma** should be considered an underlying problem in cases of chronic, recurrent disease of the uropygial gland. Chronic inflammation may predispose to neoplastic transformation, warranting aggressive early treatment. Underlying hypovitaminosis A should be excluded. Behavior is locally invasive with infrequent metastasis.

Despite their longevity, neoplasia is rarely documented in free-ranging or zoo-captive species in other groups. In captive pelicans, squamous cell carcinomas and chondrosarcomas of the beak are most common. Additional neoplasms are listed in Table 27.2.

**INFECTIOUS DISEASES**

In many of the free-ranging pelagic species in the order Aequorlitornithes, in which sampling may be infrequent, relatively little is currently known about the prevalence and significance of infectious disease and associated pathology. A recent review of the situation in Procellariiformes is indicative of the wide range of sources and limits of methodology that form the body of current knowledge (Uhart et al., 2017).

**Viruses**

**Avipoxvirus** infections have been documented in African, Humboldt, gentoo, rockhopper, and Magellanic penguins, but all species are probably susceptible. The disease affects both captive and free-ranging penguin populations and is more common in chicks than adults, suggesting adults have acquired immunity or more robust immune function. Increasing prevalence often coincides with weather-induced increases in vector populations but transmission also occurs by direct contact with abraded skin, conjunctiva, mucous membrane, contaminated air-borne particles or infected tissues. At least two divergent Avipoxvirus strains (Chordopoxvirinae subfamily) affect penguins (Offerman et al., 2014). Outbreaks manifest in cutaneous and diphtheric forms. Affected birds are often emaciated. Cutaneous poxvirus infection induces typical raised and ulcerated wart-like papules and nodules in feathered and unfeathered skin around the eyelids, beak, neck, flippers, cloaca, and feet. Glossal lesions may develop. Ocular pathology resulting in blindness occurs in Magellanic penguin chicks (Kane et al., 2012). Histological findings are characteristic, with epidermal hyperplasia and ulceration, eosinophilic intracytoplasmic inclusions consistent with Bollinger bodies, and dermatitis. Although cutaneous poxvirus infection is rarely fatal, debilitation and secondary bacterial or fungal infections are common and can cause mortality. The rarer diphtheritic form is more frequently fatal, and manifests as raised, yellow plaques in the oral cavity, throat, esophagus, trachea, and lungs. Infection in these locations can cause dyspnea or dysphagia. The lungs are often congested or hemorrhagic, and there may be spleno- and cardiomegaly. Histological findings include bronchopneumonia, nonsuppurative hepatitis, splenic necrosis, lymphopenia, and lymphocytolysis as well as necrotizing enteritis, esophagitis, and airsacculitis. Typical cytoplasmic inclusions are seen in affected epithelial cells (Fig. 27.13). Concurrent infections with common pathogens (e.g. Aspergillus) worsen the clinical course.

Avian poxvirus infections occur sporadically in Pelecaniformes (red-tailed and white-tailed tropicbirds, pink-backed pelican) and in Procellariiformes (southern giant petrel, Manx, flesh-footed, wedge-tailed and Audobon’s shearwaters, shy albatross, Laysan albatross), but reports are rare or absent in Gaviiformes and Podicipediformes. Most infections are cutaneous, affect fledgling birds on the nest, and result in proliferative and ulcerative lesions located on facial skin, beak, and legs. Diphtheritic lesions are documented in white-tailed tropicbirds and Laysan albatross. Prevalence in Laysan albatross fledglings can reach 88% in epizootic years of high rainfall (Young and VanderWerf, 2008). Despite a range of severity (including proliferative lesions covering eyes and deforming the bill and skull) most chicks recover and overall fledging rate is...
not affected (Fig. 27.12). Footweb lesions that are distinct from puffinosis have been attributed to poxvirus in juvenile and adult Manx shearwaters.

**Herpesvirus-like infection** is described in African penguins. Associated lesions resemble those of infectious laryngotracheitis (*Gallid herpesvirus 1* syn: *Avian herpesvirus 1*). Debilitation and respiratory distress were clinical features in two adults and a chick from Baltimore zoo (*Kincaid et al., 1988*). Histologically, inflammation and syncytial cells with Type A intranuclear inclusions were seen in respiratory epithelium. Findings bear some similarities to a reported outbreak of herpesvirus-like infection in eight African penguins in a rehabilitation center (*Parsons et al., 2015*). Affected chicks presented with poor weight gain, airsacculitis, and congested, edematous, and firm lungs. Tracheitis with epithelial necrosis, herpetic intranuclear inclusion bodies, and syncytial cell formation were characteristic histological features. Herpes-like virus particles were evident on transmission electron microscopy in both studies, but virus isolation and molecular testing were nonconfirmatory. *Herpesvirus*
infection has recently been demonstrated in cases of penguin diphtheria (see section Notable Diseases of Undetermined Etiology: Penguin diphtheria). Gaviid herpesvirus 1 has been isolated from ulcerative tracheitis lesions in common loons. An alphaherpesvirus was isolated from magnificent frigatebirds with crusting nodular proliferative hyperkeratotic lesions of the facial skin and beak, with evidence of ballooning epidermal degeneration.

A number of arboviruses affect birds, but West Nile Virus (WNV) and Eastern equine encephalitis (EEE) infections are most relevant to penguins. Sphenisciformes experience high rates of morbidity and mortality during outbreaks in both free-ranging and zoo settings. Disease spreads via biting insects, usually mosquitoes, and wild birds act as a virus reservoir.

West Nile Virus (WNV) is a flavivirus and OIE-listed pathogen. WNV was introduced into North America in 1999 and caused fatal infections in many species of birds including Sphenisciformes, Gaviiformes, Podicipediformes, and Pelecaniformes. Occasional cormorant fatalities are reported from Europe, where infection is considered enzootic. WNV is shed in respiratory secretions, and since avian to human transmission has been documented, should be treated as a zoonosis. Infection and deaths have been reported in Humboldt and African penguins, common loons, pie-billed grebes, Clark’s grebes, eared grebes, American white and brown pelicans, double-crested, and Guanay cormorants. Major die-offs have occurred in eared grebes and pelicans, and WNV was implicated in at least 11 mortality events resulting in the deaths of over 9000 American white pelicans assessed by the US Geologic Survey, National Wildlife Center prior to 2003. In penguins, clinical signs, when observed, include anorexia, weakness, abnormal behavior, vomiting, and dyspnea from excessive tracheal or pulmonary mucus secretion. No typical gross lesions have been described. Histological findings are limited to multifocal lymphoplasmacytic meningoencephalitis with mild gliosis and satellitosis, primarily affecting the cerebellum. Immunohistochemistry identifies frequent virus-positive Purkinje cells, neurons, granular and glial cells (Fig. 27.14) (D. McAloose, personal communication). In pelicans and cormorants, neurological signs including head tilt, ataxia, and disorientation are common. Brain hemorrhage may be apparent. Histological lesions in cormorants include meningeal or neuropil hemorrhage; neuronal vacuolation; lymphohistiocytic myocarditis, mineralization, and hemorrhage; splenomegaly with lymphoid depletion, fibrinous splenitis, and hemorrhage; renal and pancreatic lymphohistiocytic infiltrates; hepatocyte necrosis and intestinal crypt cell necrosis (Steele et al., 2000). Encephalitis or meningoencephalitis and myocardial necrosis or myocarditis are the dominant lesions in American white pelicans; encephalitis and myocarditis in eared grebes.

Eastern equine encephalitis (EEE) is an alphavirus. Clinical signs in African penguins include anorexia, antisocial behavior, mild lethargy, intermittent vomiting (Tuttle et al., 2005). Severely affected animals become recumbent and secondary infections may develop. Clinical
signs start to resolve 14 days after onset, but euthanasia may be warranted if animals fail to improve. Gross findings are minimal. Histologic examination reveals severe, chronic, multifocal, lymphoplasmacytic encephalitis with prominent perivascular cuffing. Lesions are notable in the cerebellum where mineralization of dendritic processes, focal infiltrates of mononuclear and glial cells, and loss of Purkinje cells are detected in folia; vacuolation, axonal degeneration, demyelination, and gitter cells are seen in cerebellar white matter. Mild necrotizing and histiocytic myocarditis occurs. Antibodies to EEE have been detected in pelicans, but clinical disease has not been identified.

Recurrent high mortality events in double-crested cormorants, attributable to virulent Newcastle Disease Virus (NDV); Avian paramyxovirus-1, an OIE-listed pathogen, have occurred in breeding colonies in Canada and the United States since 1992 (White et al., 2015). Mortality can exceed 90% of juveniles in a colony. Spillover events in white pelicans are occasionally confirmed based on histology and virus isolation, and large scale mortalities of American white pelicans (greater than 5000 birds) have occurred concurrently with confirmed outbreaks in double-crested cormorants. Evidence for a genuine role for NDV in mortalities in other species, such as common loons and eared grebes is weak. Paramyxovirus seropositivity is widespread in free-ranging penguins but disease is rare. The major clinical sign in cormorants is inability to fly, often with unilateral wing or leg paralysis. Pelicans present weak and unable to fly. Specific gross lesions are absent in pelicans or cormorants. Multifocal lymphoplasmacytic encephalitis and/ or myelitis are found histologically. Lesions can be mild, focal, and include perivascular lymphocytic cuffing, focal grey matter gliosis with loss of Purkinje cells in the cerebel-

lum, and white matter vacuolation in the cerebrum (Wobeser et al., 1993). Foci of lymphocytic infiltration are seen inconsistently in other organs including the pancreas and liver.

**Avian influenza viruses** (OIE-listed pathogen) are common in free-living birds including penguins. Serology or PCR-testing identify sporadic evidence of avian influenza exposure or infection in Pelecaniformes (cormorants) and Procellariiformes, but deaths are rare. Two spot-billed pelicans died during an outbreak of highly pathogenic, H5N1, avian influenza in Cambodia in late 2003. Mortalities associated with highly pathogenic H5N1 strains have also been recorded in eared and great crested grebes in Europe. Low-pathogenic avian influenza infects North American red-necked grebes (six different strains), and grebes in Siberia (mainly H5 and H7).

An overview of viruses is presented in Table 27.3.

**Bacteria**

As in all vertebrate species, the intestinal flora in healthy free-ranging penguins contains a variety of commensal bacteria, including diverse Gram-negative and Gram-positive aerobes and anaerobes. In chinstrap penguins, differences exist in the bacterial composition between juvenile and adult animals. When studied, the fecal flora of Pelecaniformes and Procellariiformes is also found to be mixed (Gram-negative bacilli, *Enterococcus, Staphylococcus*). Antibiotic resistance including extended spectrum β-lactamase (ESBL) strains is present in isolates obtained from shearwaters and boobies living even in remote locations, such as the Easter Island (Ardiles-Villegas et al., 2011).

Enteritis due to *Escherichia coli* and *Klebsiella* species occurs in captive penguins but *Salmonella* and *Clostridium* species are the most significant enteric pathogens. Outbreaks of *Clostridium enterotoxemia* have caused peracute and acute deaths in a variety of penguin species (Greenwood, 2000). Birds are found dead in excellent body condition or may present with acute depression and brown, foul smelling diarrhea. *Salmonella* (*S. typhimurium* and *S. anatis*) may cause clinical disease in penguins, and some infected birds are asymptomatic carriers. Mixed *Salmonella* serotypes, including antibiotic resistant strains, are carried by double-crested cormorants and common loons, occasionally contributing to overwintering mortality in loons. Occasional isolations are reported from grebes and albatrosses. Penguins may carry *Campylobacter jejuni* but clinical disease is not reported.

Confirmed disease due to *Chlamydophila* is rare in free-ranging penguins despite widespread seropositivity. One outbreak occurred in a captive colony of Magellanic penguins. Affected birds were inappetant, lethargic, and produced light green urates. Necropsy revealed hepatomegaly and splenomegaly. Histologic lesions included necrotizing...
### TABLE 27.3 Overview of Viral Pathogens Reported in Sphenisciformes, Gaviiformes, Podicipediformes, Procellariiformes, and Pelecaniformes

| Virus                                      | Rare | Occasional | Common | Captive/Free-Ranging | Vector | Comment                                                                 | Species Recorded                | Source/References                                                                 | OIE |
|--------------------------------------------|------|-------------|--------|-----------------------|--------|-------------------------------------------------------------------------|---------------------------------|----------------------------------------------------------------------------------|-----|
| Avian influenza virus                      | x    |             |        | F                     |        | Serum antibodies, PCR, culture                                           | Adelie penguin                  | Clarke and Knowles (1993); Hurt et al. (2014)                                   | OIE |
| Avian poxvirus                             |      |             |        | x                     | C/F    | Cutaneous, diphtheroid form, serum antibodies                             | Penguin (Magellanic, African, Humboldt, little) | Kane et al. (2012); Landowska-Plazewska and Plazewski (1968); Miller and Fowler (2015); Niemeyer et al. (2013); Offerman et al. (2014) |     |
| Birnavirus and reovirus                    | x    |             |        |                       | C      | Uncertain significance                                                   | Penguin (African, macaroni)     | Gough et al. (2002)                                                             |     |
| Eastern Equine Encephalitis virus (Alphavirus) | x    |             |        |                       | C      | Mosquito                                                                | Viral encephalitis              | Miller and Fowler (2015); Tuttle et al. (2005)                                 |     |
| Herpesvirus/Herpes-like virus              | x    |             |        |                       | C/F    | Respiratory tract                                                        | African penguin                 | Clarke and Knowles (1993); Parsons et al. (2015)                               |     |
| Newcastle disease virus/Avian paramyxoviruses | x    |             |        |                       | C/F    | Serum antibodies, cloacal swab (no clinical disease), pathogenic strains | Penguin (Adelie, royal, African, little, king) | Clarke and Knowles (1993); Haddas et al. (2014); Ladds (2009); Morgan et al. (1985); Thomazelli et al. (2010) | OIE |
| “Puffinosis-like” virus                    | x    |             |        | F                     |        | Blisters on feet, ataxia, death                                          | Gentoo penguin                  | Clarke and Knowles (1993)                                                      |     |
| Saumarez reef virus/Other flaviviruses     | x    |             |        | F                     | Ticks  | Serum antibodies, death upon inoculation                                 | Little penguin                  | Morgan et al. (1985)                                                          |     |
| Siadenovirus                               | x    |             |        |                       | F      | Opportunistic PCR (NCD)                                                 | Penguin (chinstrap, gentoo)     | Lee et al. (2016)                                                             |     |
| West Nile virus (Flavivirus)               |      |             |        | x                     | C      | Mosquito                                                                | Anorexia, vomiting, dyspnea     | Humboldt penguin                                                               |     |

(Continued)
| Virus                                        | Rare | Occasional | Common | Captive/Free-Ranging | Vector | Comment                                      | Species Recorded                                                                 | Source/References                                                        | OIE          |
|----------------------------------------------|------|------------|--------|----------------------|--------|----------------------------------------------|--------------------------------------------------------------------------------|--------------------------------------------------------------------------|--------------|
| Gaviiformes, Podicipediformes, Procellariiformes, Pelecaniformes |      |            |        |                      |        |                                              |                                                                               |                                                                          |              |
| Adenovirus                                   |      |            | x      | F                    |        | Serology                                     | Probably many species, sooty shearwater                                     | Miller and Fowler (2015); Labrín (2010)                                   |              |
| Avian influenza virus                         | x    |            | x      | F                    |        | Isolation occasional, serology common        | Arctic and red-throated loons, pied-billed grebe, great crested grebe, wedge tailed shearwater, great cormorant | Barral et al. (2008); Breed et al. (2010); Desvaux et al. (2009); Friend and Franson (1999); Ladds (2009); Lebarbenchon et al. (2015); Miller and Fowler (2015); Savchenko et al. (2015); Thomas et al. (2007) | OIE          |
| Avian poxvirus                                | x    |            | F      |                      |        | Especially island populations, rehabilitation| Manx shearwater, Laysan albatross, giant petrel, shy albatross, loons       | Australian Wildlife Health Network (2012); Gottdenker et al. (2008); Pesaro et al. (2009); Shearn-Bochsler et al. (2008); Wingate et al. (1980); Young and VanderWerf (2008) |              |
| Delta coronavirus                             | x    |            | F      |                      |        |                                              | Great cormorant                                                           | Chu et al. (2011)                                                        |              |
| Gaviid herpesvirus-1                          |      |            | F      |                      |        |                                              | Common loons                                                              | Quesada et al. (2011)                                                    |              |
| Herpesvirus                                   | x    |            | F      |                      |        |                                              | Little pied cormorant, magnificent frigatebird                            | de Thoisy et al. (2009); French et al. (1973)                              |              |
| Infectious bursal disease virus               | x    |            | F      |                      |        | Serology                                     | Shearwaters (sooty, short- and wedge-tailed)                               | Labrín (2010); Ladds (2009)                                              |              |
| Newcastle disease virus/Avian paramyxovirus   | x    |            | F      |                      |        |                                              | Great cormorants, shags, double-crested cormorants, American white pelican | Rocke et al. (2005); Thomas et al. (2007); White et al. (2015); Wobeser et al. (1993) | OIE          |
| “Puffinosis virus” (Type 2 coronavirus)       | x    |            | F      |                      |        |                                              | Manx shearwaters                                                          | Nuttall and Harrap (1982)                                                 |              |
| Species Recorded | Source/References |
|------------------|-------------------|
| Brown pelican, double-crested cormorant, anhinga | Spalding et al. (1994) |
| Frigatebird seropositive, no disease | Brown pelican, double-crested cormorant, anhinga |
| Penguins, cormorants, pelicans | Ip et al. (2014); Jaeger et al. (2016); Komar (2003); Rocke et al. (2005); Sovada et al. (2008); Sovada et al. (2013); Steele et al. (2000); Thomas et al. (2007) |
| West Nile virus (Flavivirus) | x |

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hepatitis, splenitis, and vasculitis (Jencek et al., 2012). Nearly 90% of birds in the colony developed severe keratoconjunctivitis with serous to purulent conjunctival exudate, chemosis, and variable corneal edema. Historically, human psittacosis (Chlamydia psittaci) on the Faroe Islands was associated with processing of northern fulmar chicks for human consumption. A recent study suggests that the prevalence of infection in juvenile fulmars in the Faroes remains at approximately 10% (Herrmann et al., 2006). Both northern gannets and short-tailed shearwaters are asymptomatic carriers of Chlamydophila.

Septicemic outbreaks of avian cholera (Pasteurella multocida), leading to significant mortality events in free-ranging birds, are reported in rockhopper and Adélie penguins, brown and great white pelicans, double-crested and Cape cormorants, common loons, pie-billed, horned, eared, and Western grebes. A single case is reported in a free-ranging southern giant petrel. Gross and histological findings are typical of Pasteurella multocida septicaemia with hemorrhage, intestinal hyperemia, hepato- and splenomegaly, and necrotizing hepatitis and splenitis containing intralysosomal Gram-negative coccobacilli. Other reported bacterial causes of mass mortality events in eared grebes include Erysipelothrix rhusiopathiae and Streptococcus zooepidemicus.

In penguins, Plesiomonas shigelloides and Corynebacterium amycolatum have been frequently isolated from cases of necrotizing gastroenteritis and diphtheritic stomatitis cases, respectively. The significance is uncertain. Polymicrobial and viral etiologies have recently been proposed as the cause of penguin diphtheria (see section Notable Diseases of Undetermined Etiology: Penguin diphtheria).

Mycobacteriosis is encountered rarely in free-ranging birds and more commonly in zoo collections. Mycobacterium avium complex species (M. avium sub. avium and M. avium sub. intracellulare) and M. genavense are most frequently implicated. Sporadic infections have been recorded in African and little penguins and Pelecaniformes, and probably occur in other species (Krause et al., 2015). Birds present emaciated, with typical necrogranulomas containing intrahistiocytic acid-fast bacilli in multiple organs (particularly liver, spleen, lungs). Rapid diagnosis by impression smear cytology can be confirmed by histology, mycobacterial culture, and PCR.

An overview of bacterial and fungal pathogens is provided in Table 27.4.

**Fungi**

Aspergillosis is by far the most important fungal disease and most common cause of death in penguins under human management (Denk and Stidworthy, 2016). It is rare in free-ranging populations. The causative agent is typically A. fumigatus; A. flavus occurs sporadically. Acute and chronic aspergillosis has been described. Acute cases associated with high spore inoculum and/or severe immunosuppression progress to death within 24–48 h. More commonly, aspergillosis has a prolonged course over weeks or months. Predisposing factors in rehabilitation settings and captivity include high environmental spore loads, poor ventilation, high ammonia levels, physiologic, and social stressors, inadequate nutrition, temperature stress, age (see below) and underlying disease. Acute and chronic aspergillosis differ grossly but may occur in the same bird. Acute aspergillosis targets the lungs, which are dark red, edematous, and contain multiple, small, white to tan disseminated foci on serosal and parenchymal cut surfaces. The more common chronic presentations are disseminated and incite variably severe air sac thickening with fungal plaques in interclavicular, thoracic, and abdominal air sacs (Fig. 27.15A). Plaques may appear blue- or olive-green, brown or black, and velvety if conidia develop. Compact mycelial masses (aspergillomas) may form in respiratory cavities. Pulmonary parenchyma is replaced by disseminated pearl-like pale white to yellow granulomas (Fig. 27.15B). Granulomas and granulomatous inflammation with fungal growth may extend along the celomic serosa and invade the liver, kidney, and adrenal glands. Histologically, visceral lesions have multifocal necrosis that surrounds large numbers of radially arranged, lightly basophilic to transparent, 3–6 µm, regularly septate, thin-walled fungal hyphae with dichotomous, acute angled branching accompanied by marked heterophilic infiltration. Necrotic cores are surrounded by dense infiltrates of macrophages, multinucleated giant cells, heterophils, and sparser lymphocytes and plasma cells. Air sacs are markedly thickened and expanded by similar inflammatory infiltrates, and fibrosis may be evident. Mats of fungal hyphae and conidia frequently replace air sac lining (Fig. 27.15C). Dual infection with avian malaria should be considered when splenomegaly and hepatomegaly are observed.

Adult birds are considered more resistant than chicks to aspergillosis, yet are more commonly affected in captivity. Maternal antibodies may provide transient protection but the role of humoral immunity is unclear. Long-term treatment is required and may be unsuccessful if begun late in the disease course. Antifungal drug resistance is increasingly recognized and newer antifungal drugs may be associated with toxicity (Hyatt et al., 2015) (see above).

Aspergillosis causes deaths in free-ranging common loons and American white pelicans. Nutritional and migratory stress and high spore loads in wetland habitat are probably contributory. Sporadic aspergillosis-associated mortalities occur in free-ranging nestling pelicans, zoo-captive and free-ranging adult pelicans, and occasionally in free-ranging gannets and albatross. Captive shoebills are highly susceptible. Although primarily a respiratory disease, a range of nonspecific clinical presentations can also be seen.
### TABLE 27.4 Overview of Bacterial and Fungal Pathogens Reported in Sphenisciformes, Gaviiformes, Podicipediformes, Procellariiformes, and Pelecaniformes

| Microorganism | Rare | Occasional | Common | Captive/Free-Ranging | Comment | Species Recorded | Source/References |
|---------------|------|------------|--------|----------------------|---------|------------------|------------------|
| **Bacterial** |      |            |        |                      |         |                  |                  |
| Aegyptianella | x    |            |        |                      |         | Penguin (African) | Clarke and Knowles (1993) |
| Bacillus species | x    |            |        | Feces               |         | Penguin (rockhopper, royal, gentoo) | Clarke and Knowles (1993) |
| Bacterial enteritis (E. coli, Proteus, Salmonella, Campylobacter) | x |            |        |                      |         | Pelican, cormorant | Miller and Fowler (2015) |
| Borrelia burgdorferi (P), Borrelas garinii (colonial seabirds) | x | F         |        | Serum antibodies (P), marine enzootic cycle in colonial seabirds via tick Ixodes uriae, no disease |         | Penguin (king), colonial seabirds | Schramm et al. (2014); Thomas et al. (2007) |
| Campylobacter species | x (P) | x (MW) | F (P) | Feces               |         | Penguin (macaroni), MW | Broman et al. (2000); Ladds (2009) |
| Chlamydophila species | x |            |        | Serum antibodies, sepsis, asymptomatic carriage in northern gannets, short-tailed shearwaters |         | Penguin (Adelie, emperor, rockhopper, royal, gentoo, Magellanic), fulmars, short-tailed shearwaters, Australian pelican, pelicans | Aaziz et al. (2015); Clarke and Knowles (1993); Herrmann et al. (2006); Jencek et al. (2012); Ladds (2009); Miller and Fowler (2015); Mykytowycz et al. (1955); Thomas et al. (2007) |
| Clostridium enteritis | x (Pe) | x (P) | C (P) | Enterotoxaemia, necrotizing enteritis (P) |         | Penguins (king, macaroni, gentoo), brown pelican | Greenwood (2000); Hines and Dickerson (1993); Miller and Fowler (2015); Penrith et al. (1994) |
| Clostridium species | x |            |        |                      |         | Australian pelican | Ladds (2009) |
| Corynebacterium amycolatum | x |            | W      | Penguin diphtheria |         | Yellow-eyed penguin | Alley et al. (2016) |
| Edwardsiella tarda/ species | x |            | C (P) | Enteritis, sepsis |         | Penguin (rockhopper, king), pelican (Australian, brown) | Clarke and Knowles (1993); Ladds (2009); Nimmervoll et al. (2011) |
| Erysipelothrix rhusiopathiae/species | x | x ? YNA | C/F | Sepsis |         | Penguin (African, little), great cormorant, pelican (brown, American white), common loon, eared grebes, yellow-nosed albatross (YNA) | Boerner et al. (2004); Clarke and Knowles (1993); Friend and Franson (1999); Jensen and Cotter (1976); Ladds (2009); Thomas et al. (2007) |

(Continued)
TABLE 27.4 Overview of bacterial and fungal pathogens reported in Sphenisciformes, Gaviiformes, Podicipediformes, Procellariiformes, and Pelecaniformes (cont.)

| Microorganism | Rare | Occasional | Common | Captive/Free-Ranging | Comment | Species Recorded | Source/References |
|---------------|------|------------|--------|----------------------|---------|------------------|------------------|
| Mycobacterium species | x    | x          | C/F    | M. avium complex, M. genavense in penguins | Penguin (little, African), pelicans, potentially all but sporadic | Brouwer et al. (1994); Clarke and Knowles (1993); Friend and Franson (1999); Keymer et al. (1982); Krause et al. (2015); Ladds (2009); Napier et al. (2009), Thomas et al. (2007) |
| Mycoplasma species |      |            | C      | Isolated from lung/liver | Humboldt penguin | Nicholas et al. (2004) |
| Nocardia asteroides |      |            | F      | Albatross especially Laysan albatross chicks | | Miller and Fowler (2015) |
| Pasteurella multocida (avian cholera) | x    |          | F      | Hemorrhages, GIT hyperemia, hepato- and splenomegaly with necrosis | Penguin (Adelie, African, rockhopper), pelicans, cormorants, Cape cormorants, southern giant petrel, yellow-nosed and sooty albatross, grebes, loons | Botzler (1991); Crawford et al. (1992); De Lisle et al. (1990); Friend and Franson (1999); Ladds (2009); Leotta et al. (2003); Leotta et al. (2006); Miller and Fowler (2015); Thomas et al. (2007) |
| Plesiomonas shigelloides | x (Pe) | x (P) | C (P) | Sepsis, penguin diphtheria | Penguin (Humboldt, king, African, Magellanic, rockhopper, gentoo, macaroni, Little), pelicans | Clarke and Knowles (1993); Denk and Stidworthy (2016); Ladds (2009); Nimrnervoll et al. (2011) |
| Pseudomonas pseudomallei/aeruginosa | x    |            | C      | Sepsis, tracheitis | Penguin (macaroni, Humboldt), shy albatross | Clarke and Knowles (1993); Ladds (2009); Widmer et al. (2016) |
| Salmonella species | x (C) | x (F) | C/F | Important in the wild | Penguin (Adelie, African, gentoo), loons, eared grebes, cormorants | Clarke and Knowles (1993); Ladds (2009); Miller and Fowler (2015); Thomas et al. (2007); White et al. (1976); White and Forrester (1979) |
| Staphylococcus aureus | x    |            | F      | Foot lesions secondary to pox | Shy albatross | Ladds (2009) |
| Streptococcus zooepidemicus | x    |            | F      | Mass die-off | Eared grebes | Jensen (1979) |
| Fungal | | | | Respiratory and disseminated mycosis | Penguins, common loons, American white pelicans, Dalmatian pelican, shoe-bills, Cape gannet, gray-headed albatross | Alexander (1991); Brouwer et al. (1994); Daoust et al. (1998); Denk and Stidworthy (2016); Eulenberger (1995); Forrester et al. (1997); Franson and Cliplef (1992); Hatt et al. (2003); Pyrovetsi and Papazahariadou (1995); Rocke et al. (2005); Sidor et al. (2003); Stone and Okoniewski (2001); Tham et al. (1974); Uys et al. (1966); White et al. (1976) |
| Species Recorded | Ranging | Trichophyton species | F | Dermatophytosis | Loons | Friend and Franson (1999) |
|------------------|---------|---------------------|---|----------------|-------|--------------------------|
| P = Penguin      |         |                     | F |                |       |                          |
| Pe = Pelecaniformes |       |                     | F |                |       |                          |
| Pro = Procellariiformes | |           | F |                |       |                          |
| MW = Migratory waterfowl | |         | F |                |       |                          |

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Parasites

A wide range of metazoan and protozoan parasites are reported in free-ranging populations of these species groups. These are summarized in Table 27.5. This list should not be considered comprehensive. Parasite burdens may contribute to morbidity and mortality, and act as vectors for other disease agents, but in many cases, parasites are incidental. Occasional case reports of parasite-related mortality are found in zoo-captive populations.

Protozoa

Avian malaria is one of the most significant parasitic disease in penguins, with mortality rates as high as 50%–80% (Grilo et al., 2016). Disease is common in captive penguin colonies and rehabilitation centers, and also occurs in the wild. Fatal infection is most commonly associated with Plasmodium relictum or P. elongatum, but cases involving P. cathemerium, P. juxtanucleare, P. tejerai, P. nucleophilum, and P. unalis have been recorded (Clarke and Kerry, 1993). The Plasmodium subgenus Haemamoeba, particularly P. relictum, appears more pathogenic to penguins than other subgenera.

Avian malaria is markedly seasonal and dependent upon the availability of mosquito vector populations. Culex species are most likely to be significant in transmission and local wild avifauna serves as the reservoir host. Sporozoites in infected saliva of female mosquitoes are injected into a host and enter reticuloendothelial cells where they develop into first generation exoerythrocytic meronts that undergo asexual multiplication and form merozoites. Host cell rupture releases merozoites into the blood and results in additional infection of reticuloendothelial cells (undergoing new cycles of exoerythrocytic merogony) or infection of erythrocytes (proceeding to erythrocytic merogony or gametogony). Within erythrocytes, merozoites develop into trophozoites, which further differentiate into either erythrocytic meronts or gametocytes (macrogametocytes or microgametocytes). Gametocytes remain inside erythrocytes until ingestion by an insect host in which sporogony takes place.

The cause of the extreme susceptibility of penguins to avian malaria is unclear. Disease risk is highest in chicks, juveniles and naive adults, and in animals with outdoor access. Stressors (molt, chick rearing, or poor husbandry) increase mortality. Clinical signs range from asymptomatic acute death to lethargy, anorexia, depression, vomiting, dyspnea, pale mucous membranes (anemia), and behavioral separation. Severe forms induce neurological signs including motor incoordination, seizures, and paralysis. Antemortem diagnosis is difficult as animals frequently succumb rapidly without detectable blood parasitemia. Organisms can be seen on blood smears but are often not seen in fulminating cases. A serological test has been validated for African
### TABLE 27.5 Parasites Reported in Sphenisciformes, Gaviiformes, Podicipediformes, Procellariiformes, and Pelecaniformes

| Organism | Captive/Free-Ranging | Tissue | Species Recorded | Source/References |
|----------|-----------------------|--------|------------------|------------------|
| **Metazoan** |                       |        |                  |                  |
| **Cestodes** |                      |        |                  |                  |
| *Armadoskrjabinia* species | F | Intestines | Pelicans, common loons | Kinsella and Forrester (1999); Ladds (2009) |
| *Clinostomum* species | F | Intestine | Pelicans, cormorants, African darter | Locke et al. (2015); Overstreet and Curran (2005); Ukoli (1968) |
| *Microsomacanthus pseudorostrellatus* | F | Intestines | Common loons | Kinsella and Forrester (1999) |
| *Parochites zederi* | | Intestinal diverticula | Penguin (rockhopper, Adelie, emperor) | Atkinson et al. (2008); Clarke and Knowles, (1993) |
| *Tetrabothrius* species | F | Intestine | Penguin (little, Magellanic, emperor, king, gentoo), pelicans, Kerguelen and imperial shags, boobies, loons, grebes, waved albatross, Antarctic petrels, greater, Manx and short-tailed shearwaters | Clarke and Knowles (1993); de Melo et al. (2012); Dronen et al. (2003); Dyer et al. (2002); Fonteneau and Cook (2013); Foster et al. (1996); Friend and Franson (1999); Hoberg (1987); Jiménez-Uzcátegui et al. (2015); Kinsella and Forrester (1999); Ladds (2009); Miller and Fowler (2015); Rubio-Godoy et al. (2011); Storer (2000, 2002) |
| **Nematodes** |                       |        |                  |                  |
| *Acuaria* (Dyspharynx) species; *Paracuaria* species; *Ingliseria* species; *Syncuarria* species; *Streptocara* species | F | Proventriculus | Pelicans, cormorants, Kerguelen shags, loons | Dronen et al. (2003); Fonteneau and Cook (2013); Kinsella and Forrester (1999); Ladds (2009); Overstreet and Curran (2005); Wong and Anderson (1987) |
| *Amidostomum/Epomidiostomum* species | F | Ventriculus | Great cormorant, pelican, grebes | Friend and Franson (1999); Ladds (2009) |
| *Anisakis* species | F | Proventriculus | Rockhopper penguin, pelicans, northern fulmar, greater shearwater | Clarke and Knowles (1993); Dronen et al. (2003); Miller and Fowler (2015); Nemeth et al. (2012); Riley (1972) |
| *Capillaria* species | F | Intestine | Pelicans, great cormorant, loons | Dronen et al. (2003); Ladds (2009); Overstreet and Curran (2005); Storer (2002) |
| *Contracaecum* species (multiple species) | C/F | GIT | Probably most species; Penguin (rockhopper, gentoo, macaroni, yellow-eyed, little, emperor, Magellanic), pelicans, cormorants and shags, gannets, boobies, darters, grebes, albatrosses and mollymawks, shearwaters | Campos et al. (2013); Clarke and Knowles (1993); de Melo et al. (2012); Dronen et al. (2003); Dyer et al. (2002); Fonteneau and Cook (2013); Jiménez-Uzcátegui et al. (2015); Ladds (2009); Overstreet and Curran (2005); Rubio-Godoy et al. (2011); Weekes (1982) |
| *Cosmocephalus obvelatus* | C/F | Esophagus, proventriculus | Rockhopper penguin, pelicans, loons | Clarke and Knowles (1993); Dronen et al. (2003); Ladds (2009); Storer (2002) |
| *Dirofilaria* /species | | Heart, Lung | Penguin (little, Humboldt) | Clarke and Knowles (1993); Sano et al. (2005) |

(Continued)
### TABLE 27.5 Parasites Reported in Sphenisciformes, Gaviiformes, Podicipediformes, Procellariiformes, and Pelecaniformes (cont.)

| Organism                                      | Captive/Free-Ranging | Tissue      | Species Recorded                                      | Source/References                                                                 |
|-----------------------------------------------|-----------------------|-------------|-------------------------------------------------------|----------------------------------------------------------------------------------|
| *Eustrongylides tubifex/species*              |                       | GIT         | African penguin, grebes, loons, cormorants and shags   | Atkinson et al. (2008); Dronen et al. (2003); Dyer et al. (2002); Friend and Franson (1999); Ladds (2009); Miller and Fowler (2015); Overstreet and Curran (2005); Weekes (1982) |
| Microfilarial nematodes                       | F                     | Blood, skin | Galapagos penguins, Galapagos flightless cormorants, African darter, common loon, red-necked grebe | Ashford et al. (1976); Bartlett and Anderson (1987); Merkel et al. (2007); Storer (2002) |
| *Stegophorus macronectes/diomedae*           |                       | GIT         | Penguin (Adelie, rockhopper, gentoo, macaroni), greater shearwaters | Clarke and Knowles (1993); Foster et al. (1996) |
| Stomachus species                             |                       | Stomach     | Penguins (gentoo, royal), albatrosses                  | Clarke and Knowles (1993); Weekes (1982) |
| *Syngamus trachea*                            |                       | Trachea     | Pelecaniformes                                        | Yamaguti (1961) |
| Tetrameres species; Microte-trameres species  | F                     | Proventriculus | Rockhopper penguin, pelicans, boobies, shy albatross, grebes | Clarke and Knowles (1993); Dronen et al. (2003); Ladds (2009); Overstreet and Curran (2005); Rubio-Godoy et al. (2011) |
| Trematodes                                    |                       |             |                                                       |                                                                                   |
| *Cardiocephaloides physalis*                  |                       | Intestine   | Penguin (Magellanic, Humboldt, African)                | Clarke and Knowles (1993) |
| Cryptocotyle species                          | F                     | Intestine   | Common loons                                         | Daoust et al. (1998) |
| Digenean trematodes (multiple species)        | F                     | Intestines  | Pelicans                                             | Dronen et al. (2003); Overstreet and Curran (2005) |
| Echinostomatidae                              |                       | Kidney      | Little penguin, pelicans, cormorants, loons, grebes   | Abro et al. (2016); Clarke and Knowles (1993); Dronen et al. (2003); Overstreet and Curran (2005); Storer (2000, 2002); Zamparo et al. (2005) |
| Galactosomum species                          |                       | Intestine, Liver | Little penguin, pelicans, boobies                       | Clarke and Knowles (1993); Dronen et al. (2003); Dyer et al. (2002); Overstreet and Curran (2005); Rubio-Godoy et al. (2011) |
| *Mawsonotrema eudyptulae*                     |                       | Bile duct, liver | Little penguin                                       | Clarke and Knowles (1993) |
| Mesostephanus species                         | F                     | Intestines  | Pelicans                                             | Dronen et al. (2003); Dyer et al. (2002); Overstreet and Curran (2005) |
| Phagicola species                             | F                     | Intestines  | Pelicans, African darter                              | Dronen et al. (2003); Overstreet and Curran (2005); Ukoli (1968) |
| Renal flukes/Re nicola species                |                       | Liver, kidney | Little penguin, pelicans, cormorants, boobies, loons, northern fulmars, shearwaters | Clarke and Knowles (1993); Dronen et al. (2003); Kinsella and Forrester (1999); Ladds (2009); Overstreet and Curran (2005); Riley and Owen (1972); Rubio-Godoy et al. (2011); Storer (2002) |
| Ribeiroia species                             | F                     | Esophagus, proventriculus | Pelicans                                             | Dyer et al. (2002); Ladds (2009); Overstreet and Curran (2005) |
| Organism                                      | Captive/Free-Ranging | Tissue                                      | Species Recorded                                                                 | Source/References                                                                 |
|-----------------------------------------------|----------------------|---------------------------------------------|-----------------------------------------------------------------------------------|----------------------------------------------------------------------------------|
| Schistosomes                                  | F                    | Intestines, mesenteric artery               | Pelicans, loons, grebes                                                          | Dronen et al. (2003); Ladds (2009); Overstreet and Curran (2005); Storer (2000, 2002) |
| *Acanthocephalans*                           |                      |                                             |                                                                                   |                                                                                  |
| Acanthocephalan species                       | F                    | Intestine                                   | Probably many species, pelicans, loons, grebes                                   | Dronen et al. (2003); Friend and Franson (1999); McDonald (1988); Overstreet and Curran (2005); Storer (2000, 2002) |
| Corynosoma species                            |                      | Intestine                                   | Little penguin, Kerguelen shags                                                  | Clarke and Knowles (1993); Fonteneau and Cook (2013)                            |
| *Protozoan*                                   |                      |                                             |                                                                                   |                                                                                  |
| Babesia species                               |                      | Blood (no clinical disease), may aggravate malaria | Penguin (chinstrap, little, African)                                             | Clarke and Knowles (1993); Montero et al. (2016)                                |
| Coecidia species/Eimeria gaviae/E. serventyi/E. pelecani | F                  | Intestine, kidney                           | Probably many species, penguins (little, chinstrap, gentoo), European shag, cormorants, common loons, short-tailed shearwater, brown pelicans | Abollo et al. (2005); Clarke and Knowles (1993); Dronen et al. (2003); Friend and Franson (1999); Golemansky (2002); Ladds, (2009) |
| Cryptosporidium species                       | F                    | Intestine                                   | Northern gannet                                                                  | Reboredo-Fernandez et al. (2015)                                                |
| Encephalitozoon cuniculi                      | F                    | Feces                                       | Great cormorant, great crested grebe                                             | Malčeková et al. (2013)                                                         |
| Hemoproteus species                           | F                    | Erythrocytes, liver, spleen, heart, lung    | Little penguin, frigatebirds, Australasian gannet, boobies                       | Cannell et al. (2013); Harvey and Alley (2008); Lee-Cruz et al. (2016); Levin et al. (2014); Merino et al. (2012); Work and Rameyer (1999) |
| Hepatozoon species                            | F                    | Blood                                       | Albatrosses, storm petrels                                                       | Harvey and Alley (2008); Ladds (2009); Merino et al. (2012)                     |
| *Leucocytozoon tawaki*                        | C/F                  | Blood                                       | Penguin (Fiordland, African, yellow-eyed, macaroni)                              | Allison et al. (1978); Argilla et al. (2013); Clarke and Knowles (1993); Pierce et al. (2005) |
| *Leucocytozoon ugwidi*                        | F                    | Blood                                       | Cape cormorant                                                                  | Parsons et al. (2010)                                                           |
| Plasmodium species (penguins: *P. reticulum, P. elongatum, P. cathelemur, P. juatanucrale, P. tajerae, P. nuclephilum, and P. unalis*) | C/F                  | Feces                                       | Penguin (king, African, Humboldt, little, rockhopper, Fiordland, chinstrap, macaroni, yellow-eyed, Magellanic), thin-billed prion, great white and pink-backed pelicans | Clarke and Knowles (1993); Gimba et al. (2014); Grilo et al. (2016); Quillfeldt et al. (2010) |
| Sarcocystis species                           | F                    | Kidney, small intestine, brain, muscle      | Penguins (gentoo, chinstrap), Australian pelican, northern gannet (encephalitis), little pied cormorants, hoary-headed grebes | Clarke and Knowles (1993); Friend and Franson (1999); Ladds (2009); Munday et al. (1979); Spalding et al. (2002) |

(Continued)
| Organism                      | Captive/Free-Ranging | Tissue               | Species Recorded                                                                 | Source/References                                                                 |
|-------------------------------|----------------------|----------------------|----------------------------------------------------------------------------------|-----------------------------------------------------------------------------------|
| Tetratrichomonas gallinarum   | C                    | Liver, spleen         | American white pelican                                                            | Burns et al. (2013)                                                              |
| Toxoplasma gondii             | C/F                  | Blood, liver, spleen, lung, intestine (pneumonia, hepatitis, myocarditis, encephalitis—red-footed booby) | Penguin (little, African, Humboldt), red-footed booby, masked boobies (seropositive) | Atkinson et al. (2008); Clarke and Knowles (1993); Gennari et al. (2016); Ploeg et al. (2011); Work et al. (2002) |
| Trypanosoma eudyptulæ/species |                      | Blood                | Little penguin, little pied cormorant                                             | Adlard et al. (2004); Clarke and Knowles, (1993)                                   |

**Arthropods**

**Ticks**

| Ixodes species (I. kohlsi, I. uriae, I. eudyptidus, I. percavatus, I. signatus) | F | Skin | Penguins (little, rockhopper, royal, king, macaroni, Magellanic, emperor, gentoo, Adelie), Pelecaniformes, Procellariiformes | Clarke and Knowles (1993); Ladds (2009); Muñoz-Leal and González-Acuña (2015); Vander Velde and Vander Velde (2013) |
|-----------------------------------------------------------------------------|---|------|-----------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------|
| Ornithodoros species (O. carpensis, O. spheniscus, O. yunkai)              |   | Skin | Penguin (little, African, Humboldt, Galapagos)                                                                       | Clarke and Knowles (1993)                                                                                                           |

**Biting and chewing lice**

| Austrogoniodes species (A. antarcticus, A. breviceps, A. bicornutus, A. concli, A. cristata, A. struthus, A. mawsoni, A. grossitti, A. hamiltoni, A. macquariensis, A. waterstonii) |   | Skin | Penguin (Adelie, king, macaroni, rockhopper, Fiordland, Snare’s, erect-crested, royal, emperor, gentoo, chinstrap) | Clarke and Knowles (1993) |

| Chewing lice species (Phthiraptera)                                         |   | Skin | Dalmatian and great white pelicans, albatrosses and petrels, sooty and short-tailed shearwaters, northern fulmars | Box and Meathrel (2011); Girisgin et al. (2013); Jensen et al. (1999); Mallory et al. (2006); Valim et al. (2006) |
| Mallophaga species                                                          |   | Skin | Pelecaniformes, Procellariiformes                                                                                   | Dronen et al. (2003); Martín (1990); Price and Clay (1972)                                                                       |
| Nesiotinus demerus                                                           |   | Skin | King penguin                                                                                                       | Clarke and Knowles (1993)                                                                                                           |

**Fleas**

| Listronius robertsianus                                                    |   | Magellanic penguin                                                 | Clarke and Knowles (1993)                                                                                                           |
| Parapsyllus species (P. longicorns, P. australiacus, P. heardi, P. jacksoni, P. taylori, P. magellanicus heardi, P. magellanicus) |   | Penguin (little, rockhopper, gentoo, macaroni, Magellanic, yellow-eyed)                                             | Clarke and Knowles (1993)                                                                                                           |
| Mites                                      | Captive/Free-ranging | Tissue     | Species Recorded                          | Source/References                        |
|-------------------------------------------|----------------------|------------|-------------------------------------------|------------------------------------------|
| *Epidermoptid mange* (Myialges nudus)     | F                    | Skin       | Laysan albatross                          | Gilardi et al. (2001)                     |
|                                            |                      |            |                                            |                                          |
| *Hypopial mites* (Phalacrodecetes species, others) | F                    | Subcutaneous | Pelicans, pelagic cormorants, shags, gannets, frigatebirds | Dyer et al. (2002); Fain and Laurence (1974); Pence and Courteny (1973); Pence and Newman (1997) |
|                                            |                      |            |                                            |                                          |
| *Ingrassia eudyptula*                     | F                    | Skin       | Little penguin                            | Mironov and Proctor (2008)               |
|                                            |                      |            |                                            |                                          |
| *Laminosioptes species*                   | F                    | Subcutaneous | European shag                             | V. Simpson, personal communication       |
|                                            |                      |            |                                            |                                          |
| *Megalloptes species*                     | F                    | Skin       | Pelicans                                  | Mironov and Pérez (2000)                 |
|                                            |                      |            |                                            |                                          |
| Miscellaneous                             |                      |            |                                            |                                          |
| *Hippoboscid flies*                       | F                    | Skin       | Frigatebirds                               | Levin and Parker (2014)                  |
|                                            |                      |            |                                            |                                          |
| *Nasal leeches (Theromyzon)*              | F                    | Nasal      | Grebes, loons                             | Friend and Franson (1999)                |

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penguins and PCR-detection of malarial organisms in blood is available but may fail to identify low-level parasitemia or mixed infections.

Common necropsy findings include splenomegaly, hepatomegaly, pulmonary edema, and hydropericardium with granulocytic interstitial pneumonia, mononuclear hepatitis, granulocytic splenitis, and myocarditis. Vascular occlusion and rupture due to swollen, meront-filled endothelial cells results in tissue hypoxia and necrosis. Extramedullary hematopoiesis and hemosiderosis are seen in the spleen and liver, and deposition of birefringent hemozoin granules (malaria pigment) may occur. Exoerythrocytic meronts are most frequently detected in macrophages and endothelial cells, especially in lungs, heart, liver, and spleen, and may be most obvious on cytological impression smears (Fig. 27.16). Endothelial changes (necrosis, regeneration) are present in many cases and are consistent with rupture of endothelial schizonts. In prolonged cases, erythrocytic stages are detected and enable differentiation of *Plasmodium* species from morphologically similar *Haemoproteus* species. Death is the culmination of circulatory shock from respiratory insufficiency, cardiac tamponade due to pericardial effusion, and massive release of inflammatory mediators (cytokine storms). Aspergillosis, enteritis, septicemia, chlamydiosis, *Avipoxivirus* and *West Nile Virus*, enteric and visceral helminths, amyloidosis, and cholestasis have all been recorded as concurrent conditions, accounting for variability in clinical presentation.

**Renal coccidiosis** due to *Eimeria* species may be encountered as an incidental finding in loons (*Eimeria gaviae*), European shags and Australasian gannets. In double-crested cormorants, renal coccidia (*Eimeria auritusi*) are common in healthy birds, but coccidia-associated mortality events have occurred on breeding grounds or wintering areas. Severe renal coccidiosis with *Eimeria serventyi* (*Limey disease*) affects short-tailed shearwater (Tasmanian mutton bird) nestlings (Munday et al., 1971). Affected birds are malodorous, emaciated with yellow discoloration of fat and muscle and have cloacal impact/vent caking with white urates and distal intestinal distension and hemorrhage. Kidneys are enlarged and contain disseminated, multifocal to coalescing white foci and chalky urate deposits. Gametocytes and oocysts are found in collecting duct and intrarenal ureteric epithelia and lumina where they are associated with heterophilic to lymphoplasmacytic inflammation.

**Miscellaneous**

A disease complex termed **penguin diphtheria** (also referred to as **necrotizing gastroenteritis** or **diphtheritic stomatitis**) is recognized in wild and captive penguins in New Zealand and Europe. It is currently among the most important causes of death in captive juveniles. Despite extensive research, a definitive etiology is currently not established (Alley et al., 2016; Denk and Stidworthy, 2016). Proposed causes include underlying viruses, polymicrobial infection, protozoal (trichomoniasis) or fungal infections, and environmental factors including mucosal injury due to foreign body trauma or biotoxins. However, the latter appear unlikely across captive and wild scenarios. Environmental hygiene may be important and prophylactic antibiotic treatment appears to limit the incidence in some collections. A novel avian **alpha herpesvirus**, preliminarily designated *Spheniscid herpesvirus 1*, has recently been isolated and characterized in Humboldt and African penguins with typical lesions (Pfaff et al., 2017). Further studies are underway to assay historic case material for the presence of the virus.

**FIGURE 27.16** Avian malaria in Humboldt penguins. Cytological examination of impression smears of lung, liver, and heart blood is a useful diagnostic tool. (A) Lung impression smear. Exoerythrocytic meront among red blood and epithelial cells. Meront has an elongated form consistent with an intraendothelial location. (B) Plasmodial hepatitis. Intracellular exoerythrocytic meronts are present among hepatocellular populations. Accompanying deposition of hemozoin granules (malaria pigment) and mononuclear infiltrates are evident. Hemozoin crystals are birefringent under polarized light.
Affected chicks present between 1 and 4 weeks of age with sudden death or nonspecific debilitation and anorexia. Once initiated, cases occur in clusters, with seasonality related to breeding. Necropsy findings include thick, yellow diphtheritic membranes on the hard palate, dorsal, and rostral surfaces of the tongue, buccal mucosa, and beak commissures that overlie mucosal ulceration (Fig. 27.17A). Lesions may extend into the esophagus and gastrointestinal tract. Cases with unremarkable oral mucosa but multifocal diphtheroid necrotizing enterocolitis along the length of the intestinal tract occur. Reduced intestinal content indicates anorexia. Histologically, severe diphtheroid necrotizing stomatitis, and/or esophagitis and/or enterocolitis predominate (Fig. 27.17). Thick serocellular crusts cover ulcerated mucosal and proprial surfaces. Extensive pleocellular inflammation extends into the submucosa where it is accompanied by granulation tissue as disease progresses. Heavy colonization by mixed pleomorphic bacteria is common. Milder lesions have mucosal hyperkeratosis, hyperplasia and mucosal edema, intra-cytoplasmic vacuolation, and separation of the basement membrane. Intranuclear eosinophilic inclusion bodies are occasionally seen in remaining oral mucosal epithelial cells. Secondary bacterial pneumonia may occur. Bacterial cultures yield mixed nonspecific growth, but *Plesiomonas shigelloides* and *Corynebacterium amycolatum* are disproportionately frequent. The significance of these widespread aquatic environmental organisms is uncertain. *Clostridium* species are occasionally detected. Survivors may develop chronic tracheal infections or diskitis but this is rare.

**Penguin feather loss disorder** is a syndrome characterized by aberrant premature loss of feathers, associated with retarded growth rates and increased mortality. First identified in 2006, it has subsequently affected free-ranging and captive African, Magellanic, rockhopper, and Adélie penguins (Grimaldi et al., 2015). Aberrant feather loss exposes bare patches among normal feathered skin, ranging from a few centimeters in diameter to complete feather loss prior to usual molt. Retarded growth rates and increased mortality have been associated with the syndrome. Survivors remain featherless for several weeks but those that reach fledging regrow normal, complete juvenile plumage. The etiology remains unproven but a viral etiology has been suggested.
Puffinosis is an enigmatic syndrome that causes annual seasonal mortality (late August/September) in free-ranging juvenile Manx shearwaters on Skomer and Skokholm Islands in Wales. Mortalities follow episodes of foot web blistering (Fig. 27.18), leg paralysis, locking of the tibiotarsalsotarsal joint, and occasionally conjunctivitis. There are no systemic lesions. An etiology remains unproven despite isolation (via mouse inoculation) of a type 2 coronavirus from blood, vesicle fluid, and Neotrombicula mites removed from the birds (Nuttall and Harrap, 1982). Similar blistering foot lesions have been described in European storm petrels, Leach’s storm petrels and northern fulmars from Scotland, California brown pelicans in the United States (J. St. Leger, personal communication) and in gentoo penguins with multiple ulcers on the dorsal surfaces of both feet.
27.e1 Fenbendazole toxicity, African penguin, intestine. Intestine, diffuse mucosal necrosis, effacing superficial lamina propria, with massive accumulations of fibrin and proteinaceous fluid, interspersed with red blood cells, viable and degenerate heterophils and numerous massive coalescent bacterial colonies. Residual mucosa displays widespread crypt epithelial necrosis accompanied by crypt dilation by necrotic cellular debris, and by regenerative epithelial cells exhibiting marked anisocytosis, anisokaryosis, karyomegaly and prominent nucleoli. eSlide: VM05065

27.e2 Penguin diphtheria, African penguin, pharynx. Pharynx, severe coalescent mucosal epithelial necrosis with replacement by accumulations of viable and degenerate heterophils, fibrin, and clusters of red cells admixed with large colonies of basophilic bacteria. Epithelium adjacent to ulcerations exhibits mild hyperplasia. Eosinophilic intranuclear inclusion bodies are evident within epithelial cells, especially adjacent to areas of ulceration. Heterophils admixed with macrophages and fewer lymphoplasmacytic cells extend into submucosa. eSlide: VM05066

27.e3 Aspergillosis, Humboldt penguin, air sac. Air sac, thickened and expanded by a superficial layer of viable and degenerate heterophils admixed with large numbers of radially arranged, fungal hyphae that are regularly septate, thin- and parallel-walled, with acute angle dichotomous branching (consistent with Aspergillus species.). Superficial conidial forms also present. Underlying the heterophilic debris, there are band-like accumulations of lymphocytes, plasma cells and macrophages, admixed with oedema fluid and fewer heterophils. eSlide: VM05063

27.e4 Aspergillosis, Humboldt penguin, air sac. PAS. Air sac, thickened and expanded by a superficial layer of viable and degenerate heterophils admixed with large numbers of radially arranged, PAS positive fungal hyphae that are regularly septate, thin- and parallel-walled, with acute angle dichotomous branching (consistent with Aspergillus species.). eSlide: VM05064

27.e5 Avian malaria, Macaroni penguin, liver. Liver, mild to moderate multifocal portal, perivascular and parenchymal infiltrates of mixed lymphohistiocytic cells and extramedullary haematopoietic cells. Frequent macrophages laden with abundant brown granular pigment, widespread Kupffer cell pigmentation. Small numbers of plasmodial schizonts distend cells, frequently those located along sinusoids. Hepatocytes have pale variably vacuolated cytoplasm with frequent intracanalicular cholestasis. Occasional drop out of individual hepatocytes. eSlide: VM05132
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