

# Common Diseases of Urban Wildlife

## BIRDS

### MISSION STATEMENT:

The Australian Registry of Wildlife Health is committed to contributing to the preservation of Australia's biodiversity through increased understanding of the interactions among animals, the environment, and disease causing agents.

## 1 Common Diseases of Birds

### 1.1 Introduction

A wide variety of diseases have been documented within free ranging Australian birds. The following segment focuses on those diseases that occur often within a particular species or taxonomic group of birds.

### 1.2 Parasitic Disease

For additional information see also: (Bain and Mawson, 1981).

#### 1.2.1 *Cheilospirura gymnorhina*

*Cheilospirura gymnorhina* is also referred to as the throat worm of juvenile magpies. The same parasite, or a similar parasite, occurs in the oral cavity and pharynx of currawongs, butcherbirds, magpie larks, and black-faced cuckoo shrikes. *C. gymnorhina* burrows its head into the mucosa of the oral cavity and pharynx. The host then responds by creating a fibrous nodule around the parasite. Although small numbers of parasites result in self-limiting infections, large numbers can impair prehension of food, or partially obstruct the glottis. Treatment trials using a variety of anti-helminthic agents have not been successful in eliminating the parasites (Larry Vogelnest, personal communication). Repeated manual removal of the parasites with haemostats is recommended and this is assisted by application of moxidectin directly to the nematodes. Euthanasia may be the most humane option for severely debilitated young birds with heavy burdens of *C. gymnorhina*.

#### 1.2.2 *Syngamus trachea*

*Syngamus trachea* is commonly found in the trachea of a variety of birds. Occasionally magpies that die suddenly are found to have complete tracheal obstruction with masses of these parasites.

#### 1.2.3 Trichomoniasis

Oral trichomoniasis has been observed in debilitated free ranging birds, but is most common in captive wildlife that are undergoing treatment for various injuries. Birds of prey, columbiforms, little penguins, and psittacines are sporadically affected by trichomoniasis. Trichomonads are common commensal agents within the avian alimentary tract. Trichomonads are ovoid protozoa that have four anterior flagella and an undulating membrane. These organisms are spread through either direct or indirect contact. The factors that predispose a bird to develop trichomoniasis are unknown.

Caseous oral plaques are created when the organisms cause tissue necrosis. Lesions are often subject to secondary bacterial infections. A diagnosis of trichomoniasis is best made by examining a wet mount preparation of the caseous debris. Flagellates can be seen moving within the wet preparations under light microscopy. The organisms are much more difficult (often impossible) to see within cytologic and histologic preparations of affected tissues.

Treatment of trichomoniasis includes debridement of the caseous plaques, supportive care and administration of antiprotozoal agents.

#### 1.2.4 *Haemoproteus*

Megaloschizonts of *Haemoproteus* cause clinically significant myopathy in pied currawongs within the Sydney region of New South Wales. This organism was initially reported as *Leucocytozoon* sp. *Haemoproteus* spp. infection occurred in juvenile, sub-adult and adult birds of both sexes, at any time of year, but it has not been seen in recent years. Infection in some birds may have been incidental; however, heavy parasite burdens in some birds resulted in lethargy, weakness and debility. If the breast feathers are parted, pale oval foci were evident throughout the pectoral musculature in affected birds. There is no known treatment for this protozoal infection and birds often die shortly after initial examination. The lifecycle of this protozoal agent is unknown.

Upon post mortem examination of affected birds, discrete, pale oval foci measuring up to 1.5 cm long and 0.5 cm wide are scattered throughout the skeletal muscles, tongue, myocardium and ventricular muscularis externa. Histopathologic examination demonstrates that pale foci consist of central megaloschizonts, surrounded by necrotic muscle and an intense inflammatory response. Haemorrhage, necrosis, and inflammation are most severe around ruptured megaloschizonts. Pigmented oval *Haemoproteus* gamonts may or may not be evident within circulating erythrocytes of affected birds.

#### 1.2.5 *Toxoplasma gondii*

Toxoplasmosis is a potentially fatal disease in native birds (Hartley and Dubey 1990, ARWH). Birds with toxoplasmosis are depressed, fluffed, or are found dead. Gross post mortem findings consist of pulmonary oedema, pulmonary congestion, and pale foci within the liver, spleen and intestinal mucosa.

Histologic examination reveals pulmonary oedema and congestion, fibrin within the distal airways, and non-suppurative inflammation or necrosis within the liver, spleen, brain, skeletal muscle, ventriculus, adrenal gland, and intestine. Numerous protozoa, morphologically consistent with *T. gondii* may be observed in the interstitium of the lung or within foci of necrosis in other tissues. Definitive diagnosis of *T. gondii* protozoal encephalitis has been established in some native birds using immunohistochemistry (Hartley and Dubey, 1991).

Protozoal cysts resembling those of *T. gondii* are observed in the absence of inflammation during routine histologic examination of nervous tissue of a variety of native birds, especially the tawny frogmouth. These cysts are consistent morphologically with *T. gondii* and they appear to be a common incidental finding (ARWH).

#### 1.2.6 Other Parasites

##### **Ectoparasites**

Birds can be parasitised by ticks. *Ixodes holocyclus*, the paralysis tick, is found occasionally on birds. Anecdotal reports of tick paralysis in birds have been documented. Three species of the genus *Ornithodoros* are known in Australia, *O. macmillani* on wild birds, *O. capensis* on sea birds and *O.gurneyi* primarily on kangaroos (David Spratt, personal communication).

Lice commonly infest wild birds, but rarely cause disease, such as anaemia. Heavy infestations may be treated with topical antiparasitic powders.

*Cnemidocoptes* sp. infestations are capable of causing severe debility in pied currawongs. Infestations are associated with significant epidermal hyperplasia, primarily involving the skin of the toes. Mites can be demonstrated by microscopic examination of scrapings of the thickened skin. Oral or subcutaneous administration of ivermectin-like drugs will control *Cnemidocoptes* sp. infestation, but more severe infestations are often associated with foot deformation and general debility, which may not be amenable to treatment.

Hippobosca are a genus of fly that occurs commonly within the plumage of birds of prey, especially the tawny frogmouth. Hippoboscid flies can bite and are capable of acting as a vector in the transmission of

disease. These flies will infest and bite humans, but do not seem to remain on human hosts for prolonged periods.

*Sternostoma tracheacolum* is the tracheal and air sac mite of Gouldian finches. Heavy burdens of these mites are capable of causing coughing, sneezing, and open mouth breathing. Disease associated with this parasite is most common in captive finches. Ivermectin-like drugs can be used to treat affected birds.

### **Trematodes**

*Australobilharzia* spp. are schistosome parasites that live in the blood vessels of water birds. These parasites are most commonly reported in silver gulls. The parasites have an affinity for the blood vessels of the gastrointestinal tract and kidney. The presence of adult trematodes and trematode eggs may result in the formation of multiple granulomas within the intestine and liver. *Australobilharzia* sp. infection, however, is most often an incidental finding in birds. The cercaria of *Australobilharzia* sp. penetrate the skin of humans and are the cause of “swimmer’s itch”.

*Mawsontrema eudyptulae* is a trematode that lives in the bile ducts of little penguins. Infection with this parasite may be incidental, or may be associated with liver enlargement, necrosis, and possibly haemorrhage from the liver (Harrigan 1992, Norman et al., 1992). For the population of little penguins in Bass Strait in southeastern Australia to remain stable, the scale of annual, natural mortality is astonishing and has been estimated at 100,000 birds (Norman et al., 1992). In addition, large numbers of immature birds also perish and many become beach washed (loc. cit.). In these so-called “wrecks”, adult mortality generally occurs in winter due mainly to starvation. Wrecks involving immature birds occur mainly in late summer and early autumn and are directly or indirectly the result of parasitic infection (Obendorf and McColl, 1980; Harrigan, 1992).

### **Nematodes**

Gastrointestinal nematodiasis is usually an incidental finding in wild birds. Parasite burdens in wild birds are usually mild. Captive birds, however, may experience excessive parasite burdens that can contribute to debility.

*Capillaria* spp. are often evident within serpiginous tracts created as the nematode burrows through the mucosa and lamina propria of the oesophagus, proventriculus and liver. Capillariasis occurs primarily in captive birds. Infected birds may suffer from extensive hyperplasia of the oesophageal mucosa and

marked inflammation surrounding the parasites in the mucosa and lamina propria. Emaciation and dehydration can result from these infections. *Capillaria* sp. infections in wild birds are primarily an incidental finding during post mortem examination.

*Contracaecum* spp. are nematodes that parasitise the oesophagus and proventriculus of piscivorous birds. Small numbers of parasites pose no threat to the host. There are reports of large parasite burdens of *C. spiculigerum* being associated with proventricular ulceration, haemorrhage, emaciation and death in little penguins in Victoria (loc. cit.).

*Dispharynx nasuta* is a nematode parasite that burrows into the mucosa of the proventriculus in a variety of birds. Infection with this parasite can result in either granulomatous inflammation or the development of fibrous nodules surrounding the parasites within the wall of the proventriculus. *Echinuria uncinata* is a parasite that is capable of causing similar lesions in waterfowl.

*Serratospiculum anaculata* resides within the airsacs of birds of prey, and infection is most common in falcons. Two species are known in Australia: *S. guttatum* from *Falco longipennis* and *F. peregrinus* and *S. tendo* from *F. peregrinus*. Overseas, *S. anaculata* occurs primarily as an unapparent infection. Clinical respiratory disease has been described in Australian birds infected with *S. anaculata* that are subject to stress or concurrent disease. Diagnosis of *S. anaculata* requires endoscopic examination of the air sacs. Ivermectin-like drugs can be used to treat affected birds.

Microfilariae are occasionally found during examination of peripheral blood smears of wild birds. Adult filarial nematodes may reside within the air sacs, coelomic cavity, subcutaneous tissues, heart, greater vessels, or lungs. Infection is diagnosed during microscopic examination of peripheral blood smears or buffy coat smears. Microfilariae are transmitted by haematophagous arthropods. Microfilarial infections are incidental to the host.

*Oxyspirirura* spp. are nematode parasites that can be found within the conjunctiva and nictitating membrane of a number of species. Infection with this parasite is usually asymptomatic, but may be associated with conjunctivitis in a small proportion of birds (Pass, 1993).

*Angiostrongylus cantonensis*, the rat lungworm has been found to neurological dysfunction associated with eosinophilic or non-suppurative encephalomyelitis in yellow-tailed black cockatoos, and more commonly in tawny frogmouths. Infection in tawny frogmouths is now a very common occurrence and seems to have a seasonal prevalence (Monks et al., in press, Montali et al., 2004, Prociv, 1999). Birds become infected with the parasite by eating snails and slugs, the intermediate host. Diagnosis of the infection can be very difficult, since birds do not usually develop eosinophilia. Cerebrospinal fluid taps collected from infected animals are also often non-suppurative rather than eosinophilic, making it difficult to differentiate angiostrongylosis from viral or protozoal infection. Treatment of the infection in birds is also difficult. The parasite's cuticle retains many antigens and killing the worms can result in release of antigens with subsequent severe host immune response.

### **Cestodes**

Birds are parasitised by many species of cestode. None of these is considered to be highly pathogenic in free-living birds. It is possible, however, that large burdens of cestodes will add to the debility of a captive or compromised bird. If necessary, cestodiasis is treated with praziquantel. The treatment is usually repeated 10 days after the first dosage.

### **Protozoa**

A wide variety of protozoa have been reported within the gastrointestinal tract, cardiovascular system, musculature and renal tissues of free-flying birds. The following discussions regarding protozoa are limited to those protozoal infections known to be clinically significant.

*Spiroucleus* (formerly Hexamita) -like organisms have been associated with numerous outbreaks and individual cases of emaciation, diarrhoea and fatal enteritis in Australian king parrots (Philbey et al., 2002, Vogelnest, 1994). Similar parasites have been identified in an emaciated, wild sulphur crested cockatoo from western NSW (ARWH). These birds become emaciated and have very thin walled intestinal tracts, often filled with fetid brown fluid. The intestinal tissues of affected birds seem to decompose very rapidly making it very difficult to identify organisms within tissues on histologic examination. Saline wet-mount preparations can be used to demonstrate the organism within the gastrointestinal tract during gross post mortem examination.

Gastrointestinal *Giardia* spp. infections have been documented in a variety of wild and aviary birds in Australia. *Giardia* spp. have been recovered from the intestinal lumen of straw-necked ibis in Western Australia, and a sulphur-crested cockatoo in Victoria (Foreshaw et al., 1992, Gallagher et al., 1995). Giardiasis in captive young budgerigars can result in decreased growth rates, dehydration, and diarrhoea (Filippich, 1998). Diagnosis of giardiasis is based upon direct microscopic examination of faeces or intestinal content. *Giardia* sp. trophozoites are pear shaped, binucleate, and have eight flagella. A cyst form, with four nuclei is occasionally shed in the faeces. Treatment of budgerigars with metronidazole decreased shedding of these protozoa in the faeces (Filippich, 1998). Treatment for giardiasis is the same as for trichomoniasis. Careful attention to hygiene will prevent clinical infection in most captive birds.

*Cryptosporidia* spp. have been observed within the intestinal brush border of wild Pacific black ducks, a red-tailed black cockatoo, an Australian magpie, and a rock parrot (ARWH). The significance of this parasite as an avian pathogen is poorly understood.

Eimerian and isosporan coccidial oocysts are commonly identified within the faeces of healthy captive and free-flying birds. Coccidiosis may cause necrotising enteritis in young captive birds of a variety of species. Disease associated with coccidial infection in free ranging birds is rare. When large numbers of faecal oocysts accompany diarrhoea, treatment of coccidiosis is advisable.

Renal coccidiosis is a common incidental finding within little penguins, Australian gannets, and short-tailed shearwater (also known as the Tasmanian mutton bird). Limey disease is the term used to describe clinically apparent renal coccidiosis in nestling short-tailed shearwater. Chicks with limey disease are thin and have urate and faecal soiling of the pericloacal feathers (Munday et al., 1971). Renal enlargement and multiple pale foci throughout the kidney are evident on gross post mortem examination. The ureter and cloaca may also be distended with urates. Microscopic examination of the affected renal tissue reveals inflammation within the interstitium surrounding the large collecting ducts. Coccidial oocysts are often evident within multiloculated granulomas within the collecting duct mucosa and in the surrounding interstitium.

*Caryospora* spp are coccidian parasites that can be found within the intestinal lamina propria and mucosa of carnivorous birds of prey, but these are generally incidental findings. *Caryospora* spp. can be found infecting reptiles, birds and rodents and can have a single host, or two host (predator-prey) lifecycle. The

intestinal forms of *Caryospora* spp. are characterised by a single sporocyst containing eight elliptical sporozoites.

*Haemoproteus*, *Leucocytozoon*, *Plasmodium*, *Atoxoplasma*, and a *Babesia*-like organism are genera of the family Plasmodiidae that are commonly found within the peripheral blood of wild Australian birds. Each of these organisms is arthropod borne. A bird may be infected with two or more of these organisms concurrently without any clinical signs. Young or debilitated birds may develop anaemia, anorexia, and depression as a result of large parasite burdens.

Trypanosomes are occasionally found within the peripheral blood of native birds. These parasites are extracellular flagellates that are transmitted by biting midges. Trypanosomes are reported most commonly in little penguins, and they do not appear to be pathogenic.

Systemic coccidiosis associated with *Lankesterella* spp. and *Isospora serin* have been identified within circulating monocytes of the brown treecreeper, house sparrows, and aviary birds within NSW.

O'Donoghue and Adlard (2000) in the "Catalogue of Protozoan Parasites Recorded in Australia" employ the generic name *Lankesterella* rather than *Atoxoplasma*. Of Australian *Lankesterella* spp, one species occurs in tree-creepers and one in white eyes and sparrows (David Spratt, personal communication).

These systemic coccidian parasites undergo sexual reproduction (gametogony) within the mucosa of the gastrointestinal tract, and asexual reproduction (schizogony) within extra-intestinal tissues such as the liver and spleen. Sporozoites of this organism are evidently transported among these sites within mononuclear cells. Sporozoites are round basophilic organisms that have a small basophilic nucleus. These sporozoites are visible, usually as individual organisms, within the cytoplasm of mononuclear cells, which have an indented nucleus.

A *Babesia*-like piroplasm is occasionally evident within the erythrocytes of little penguins. The organism is most likely transmitted by ticks, and infection has been associated with regenerative anaemia (ARWH).

### **1.3 Bacterial Disease**

Sporadic outbreaks of mortality in native birds have been attributed to infection with *E. coli*, *Salmonella* spp., *Pasteurella* spp., *Mycobacterium* spp., *Erysipelothrix rhusiopathiae*, *Listeria monocytogenes*

*Streptococcus* spp., *Staphylococcus aureus*, *Haemophilus* spp., *Mycoplasma* spp., and *Clostridium* spp (ARWH). Ideally treatment of bacterial infection is based upon isolation of the organism within lesions, and antimicrobial sensitivity testing.

### 1.3.1 Yersiniosis

*Yersinia pseudotuberculosis* infections can result in either acute enteritis and septicaemia, or multisystemic abscesses.

### 1.3.2 Necrotic Enteritis

#### **Aetiology**

Wild rainbow lorikeets, scaly-breasted lorikeets, and king parrots in coastal eastern Australia are seasonally affected with necrotising enteritis. A variety of organisms, primarily coliforms, have been isolated within the necrotic intestinal tissue. *Clostridium perfringens* and *E. coli*, however, are most commonly isolated within the intestine and other tissues of birds with necrotic enteritis.

Carbohydrate overload has been suggested as a means of causing intestinal overgrowth with *Clostridium perfringens*, and subsequent necrotic enteritis (Pass, 1993, McOrist and Reece, 1992). Numerous artificial feeding stations are established for lorikeets in urban areas. Unfortunately, many of these stations provide only sugar water. The presence of an underlying viral infection in birds suffering from necrotic enteritis, however, has not been thoroughly investigated.

Necrotic enteritis occurs in male and female birds, juvenile animals and adults. Free-ranging birds are most commonly diagnosed with necrotic enteritis; however, the disease has been observed in captive lorikeets. Necrotic enteritis is most often observed in July and August (ARWH).

An investigation into the occurrence of necrotic enteritis identified 58 dead rainbow lorikeets, red-collared lorikeets, and scaly-breasted lorikeets originating from 18 different flocks in eastern Australia over a ten year period (McOrist and Reece, 1992). *Cl. perfringens* was isolated from the intestinal tissues of many birds, and beta toxin was demonstrated within the bacterial colonies and within intestinal content using gas liquid chromatography (McOrist and Reece, 1992).

In 1998, eight birds were presented to the ARWH with necrotic enteritis. *E. coli* was isolated in pure culture from the necrotic segments of intestine, and in the lung and liver of seven of these birds. These

tissues were also submitted for anaerobic culture; however, *Cl. perfringens* was not identified. Thus, necrotic enteritis may be caused by a variety of bacteria and its pathogenesis may be multifactorial.

### **Clinical Signs**

Birds with necrotic enteritis exhibit a variety of clinical signs. Most of these birds are in good body condition, but are weak, depressed, dehydrated, regurgitate clear fluid, and have soiled vent feathers as a result of watery diarrhoea. The bird's abdomen may be palpably distended. Alternatively, birds with necrotic enteritis are found dead or moribund. The species affected by necrotic enteritis normally have wet faeces, and the detection of diarrhoea may be difficult.

### **Pathology**

During the gross post mortem examination of these birds, the intestinal tract is distended by gas or reddish-brown fluid. A diphtheritic membrane coats the mucosa, or the mucosa is found to be friable and haemorrhagic. Microscopic examination of affected segments of intestine reveals the following lesions: mucosal to transmural necrosis, intense mononuclear cell infiltration, oedema and congestion throughout the lamina propria and submucosa, and colonies of bacteria scattered throughout a superficial layer of necrotic debris and fibrinous exudate.

### **Diagnosis**

Necrotic enteritis is identified based upon the clinical signs and microbial culture of faeces. Many birds with necrotic enteritis are found dead. Post mortem examination and microbial culture of segments of intestine are used to establish a diagnosis.

### **Treatment**

Although sensitivity testing of the *E. coli* isolated within the intestine of birds with necrotic enteritis indicates that the organism is sensitive to a variety of commonly used antibiotics, treatment of these birds is rarely successful. Presumably, the birds are suffering from either enterotoxaemia, or bacteraemia by the time they demonstrate clinical signs.

#### 1.3.3 Chlamydiosis

### **Aetiology**

*Chlamydophila psittaci* is a bacterium of the family Chlamydiaceae. These bacteria are obligate intracellular parasites that are capable of causing severe disease in free-living birds, aviary birds and humans.

Outside of the body *C. psittaci* take the form of elementary bodies, which have a rigid cell wall. Elementary bodies are weakly gram-negative, non-motile bacteria, which are phagocytosed by host cells. Once enveloped within a phagosome, elementary bodies expand to become reticulate bodies, which have a more flexible cell wall and are capable of growth and multiplication. After a period of division, these revert to elementary bodies, which are released with the death of the host cell.

*C. psittaci* is endemic throughout Australia. It is a notifiable disease. Psittacine and columbiform birds are most susceptible to *C. psittaci* infection. Chlamydiosis is a common disease of lorikeets, cockatoos, budgerigars, rosellas, and aviary psittacines. *C. psittaci* is transmitted either through the faecal-oral route or through respiratory secretions. Elementary bodies may remain infective within dried faeces for several months. Chlamydiosis should be considered among the differential diagnoses in any emaciated wild bird, and barrier methods should be employed to prevent potential spread of infection to other wildlife or humans.

### **Clinical Signs**

Birds with active chlamydiosis may exhibit a broad range of symptoms associated with either acute or chronic disease. Many birds will function as asymptomatic carriers of the organism, while others may suffer severe or fatal infection. Chlamydiosis is most often manifested as respiratory or gastrointestinal illness. Clinical signs associated with *C. psittaci* infection include: weight loss, depression, lethargy, anorexia, diarrhoea, bile stained faeces, ocular or nasal discharge, and dyspnoea.

### **Pathology**

Post mortem findings can be highly variable in birds suffering from chlamydiosis. Some birds may die acutely with very few morphologic lesions, while some will merely have splenomegaly and hepatomegaly, and others may have fibrinous air sacculitis, pericarditis and enteritis.

### **Diagnosis**

Definitive diagnosis of chlamydiosis relies upon isolation of the organism within cell culture or embryonated chicken eggs. Marked leucocytosis, monocytosis, and an elevated AST may be suggestive of *C. psittaci* infection, however, there is significant species and individual variability in the haemogram of birds with chlamydiosis. Antigen can be detected within conjunctival, nasal, or faecal swabs using antigen capture ELISA tests or direct immunofluorescence testing. Diagnostic tests based upon antigen capture are highly sensitive, but may not be highly specific. Some gram negative bacteria will cross react with the antibody used in the ELISA test, thus, conjunctival and choanal swabs will provide far fewer false positive reactions compared with faecal swabs. ELISA based antigen capture test kits are commercially available for in-house identification of *Chlamydia* sp. antigen (Clearview® test kits). These kits are marketed for the detection of human *C. trachomatis* within urine samples, but they are effective in the identification of *C. psittaci*.

Post mortem diagnosis of chlamydiosis is usually based on finding multisystemic histiocytic inflammation on histologic examination and identification of the organism within lesions. Impression smears of spleen, lung, and liver can be stained using modified Machiavello's staining protocols. This protocol can also be used to identify the organism within paraffin embedded tissue. Fresh tissues, such as liver, spleen and lung may be submitted to a microbiology laboratory for culture, or swabs from fresh tissues can be tested with antigen capture ELISA tests or PCR. Immunohistochemical demonstration of the organism is possible in fixed tissues.

## **1.4 Viral Disease**

### **1.4.1 Psittacine Beak & Feather Disease (Pbfd)**

#### **Aetiology**

Psittacine beak and feather disease is a common disease in wild and aviary psittacines throughout Australia. The disease is caused by psittacine circovirus, and is manifested by lesions in the feathers, beak and occasionally the claws.

Psittacine circovirus is an icosahedral, non-enveloped virus, which has a single, round strand of DNA. Presence of the virus can be demonstrated with feather epithelium, follicular epithelium, macrophages within the feather pulp and dermis, macrophages in the bursa of Fabricius, Kupffer cells in the liver, and within faeces. Psittacine circovirus has an affinity for epithelial cells and lymphoid cells.

## **Clinical Signs**

Clinical signs of infection with psittacine circovirus are highly variable depending on the age and species of the bird, and the quantity of virus in the infective exposure. The progression of disease is also highly variable, ranging from acute to chronic. Young birds most often exhibit the acute form of infection. Clinical signs of acute psittacine circovirus infection include diarrhoea, weight loss, anorexia, depression and either death or residual feather damage. The chronic form of psittacine circovirus infection in cockatoos begins with loss of the powder keratin in the plumage, and the production of dystrophic down feathers over the hips. Powder down feathers become short and lose the plumaceous barbs. The loss of powder down feathers results in a dull and dirty look of contour and flight feathers, and imparts a glossy black appearance to the beak.

When damaged by psittacine circovirus, the beak may become elongated, softened, broken, cracked, or it may have uneven wear. These changes are most commonly seen in cockatoos in the late stages of infection. If the germinal epithelium of the beak is exposed by fractures or cracks in the keratin the bird will often stop eating due to pain.

Young lorikeets of the genus *Trichoglossus* that are infected with psittacine circovirus will often present with the last two to four primary feathers of the wings missing. If pulled from their follicles, the calamus of the remaining tail feathers and flight feathers will often exhibit characteristic morphologic lesions. These lorikeets are called “runners” since they are unable to fly, yet healthy enough to forage and run on the ground. Young lorikeets are identified by their dark brown beaks. Occasionally, these young lorikeets will have a blotchy yellow pattern on the tail feathers that are usually green. Beak lesions rarely occur in lorikeets infected with psittacine circovirus.

Feathers damaged by psittacine circovirus are curled, clubbed, easily broken, or they have retained feather sheaths, haemorrhages within the calamus (shaft), or annular constrictions of the calamus. Replacement feathers grow slowly, or fail to regrow.

## **Pathology**

Histologic lesions associated with psittacine circovirus infection occur primarily in the growing feather, but may also be evident within the follicle. Necrosis occurs in the germinal layer of the follicular and

feather epithelium, and basophilic cytoplasmic inclusion bodies may be evident within the epithelium, and in reticuloendothelial cells in the dermis, feather pulp, and bursa of Fabricius.

Psittacine circovirus infects the thymus and bursa of Fabricius and is associated with lymphoid necrosis and premature atrophy of these tissues. Birds with psittacine beak and feather disease often succumb to secondary viral, bacterial or fungal infections.

### **Diagnosis**

The presumptive diagnosis of psittacine beak and feather disease is based upon gross and microscopic lesions in the feather and feather follicle.

Psittacine circovirus is very difficult to isolate in culture. Definitive diagnosis of infection with this virus can be established through serological testing, which is available through commercial laboratories. Haemagglutination inhibition (HI) testing detects antibodies to psittacine circovirus in blood, serum and yolk, while haemagglutination (HA) testing detects the virus in faecal samples or feathers. Birds that suffer from severe psittacine beak and feather disease may not mount an effective immune response to the virus and their titres measured via HI tests may not be elevated.

Elevated HI titres merely indicate that antibodies have been formed in response to exposure to psittacine circovirus. Birds with either the acute or chronic form of circovirus infection often have low titres. Budgerigars, lorikeets, and king parrots, however, usually have high psittacine circovirus titres and continue to shed the virus.

Immunohistochemistry, PCR, and DNA *in-situ* hybridisation tests for psittacine circovirus are available overseas.

### **Treatment**

Some species of psittacine can spontaneously recover from the acute form of beak and feather disease. Rainbow lorikeets, budgerigars, eclectus parrots and king parrots may recover from this infection with only mild residual feather changes. Birds with the chronic form of psittacine circovirus infection rarely recover. The cause of death most often relates to secondary infection with other viral, bacterial or fungal agents as a result of immunosuppression.

There is no known cure for psittacine beak and feather disease. Nursing care to keep the bird warm and eating will prolong the life of cockatoos. Lorikeets may spontaneously recover from psittacine beak and feather disease, but can shed the virus for a prolonged period.

### **Prevention**

Since there is no effective treatment for birds suffering from psittacine beak and feather disease, controlling the spread of the virus relies on strict hygiene and euthanasia of affected birds. Birds that have clinically apparent psittacine beak and feather disease can shed psittacine circovirus for extended periods, functioning as a source of infection for other birds. If euthanasia is not an option, these birds should be maintained under strict quarantine. Viracidal disinfectants used to kill parvovirus should inactivate psittacine circovirus. A killed vaccine for the prevention of psittacine beak and feather disease is currently under investigation.

### 1.4.2 Poxvirus

#### **Aetiology**

Australian magpies, native pigeons and raptors are occasionally clinically affected by poxvirus infection.

Poxvirus is a member of the genus *Avipox*, which has a worldwide distribution. Poxvirus is shed in saliva, nasal secretions, faeces and wound exudates or scabs. The virus is transmitted primarily by haematophagous arthropods, such as mosquitoes; however, other vectors and fighting can also result in transmission. Infection results in viraemia and then localisation within the skin or mucosa.

#### **Clinical signs**

Clinical signs of poxvirus infection vary from blistering and small nodules in the skin to large dermal nodules with markedly hyperplastic epithelium, which may have foci of ulceration. These lesions primarily occur on the skin of the feet, legs and head, and around the eyes, mouth and cloaca. Secondary bacterial infection is a common finding in poxvirus lesions. Some birds will recover spontaneously, while others will become debilitated due to difficulty walking or obtaining food.

#### **Pathology**

The microscopic lesions associated with poxvirus infection include marked epidermal thickening. Hyperplastic epithelial cells may contain cytoplasmic vacuoles that house large, eosinophilic inclusion bodies. These inclusion bodies are called Bollinger bodies.

### **Diagnosis**

A diagnosis of poxvirus infection is based on finding the characteristic intracytoplasmic eosinophilic inclusion bodies within epithelial cells upon microscopic examination of formalin fixed tissue sections. Some diagnosticians can use Diff Quik® stained scrapings of the proliferative lesions to identify intracellular inclusions, but this is very difficult. Alternatively, biopsies of the proliferative wounds can be submitted for electron microscopy to look for viral particles.

### **Treatment**

Many birds will respond to cage rest and nursing care. Surgical debulking of large lesions may provide relief for some birds. Nutritional support and prevention of secondary infections will aid in the recovery of many birds suffering from poxvirus infection.

#### **1.4.3 Adenovirus**

Adenovirus associated hepatic necrosis is a fairly common finding in tawny frogmouths along coastal eastern Australia. Tawny frogmouths with adenovirus hepatitis may have evidence of traumatic injury, or may be depressed and weak. Diagnosis of adenovirus infection is based on post mortem examination. The liver is mottled, friable, enlarged and has rounded margins upon gross post mortem examination. Foci of acute hepatocellular necrosis are scattered throughout the parenchyma of the liver on histological examination. Individual hepatocytes at the margins of the necrotic foci will have peripheralisation of nuclear chromatin and contain intranuclear eosinophilic to basophilic inclusion bodies (Reece et al., 1985). Inclusion body hepatitis associated with adenovirus infection has also been described in a cockatiel (Scott et al., 1986).

#### **1.4.4 Other Viruses**

An enterovirus-like agent has been identified within the faeces and enterocytes of galahs and sulphur-crested cockatoos that were depressed, anorexic, and had profuse, green, mucoid diarrhoea. Over a period of approximately three weeks affected birds became dehydrated, emaciated and died. Dilation of the intestinal tract with mucoid fluid, gas, and a thickened mucosa were evident during gross post mortem examination. Histological examination of affected segments of intestine demonstrated atrophy of villi,

and hyperplasia of the crypts of Lieberkuhn. Concurrent psittacine circovirus infection was evident within some of the ill birds (Wylie and Pass, 1989).

## **1.5 Fungal Disease**

### **1.5.1 Aspergillosis**

#### **Aetiology**

A variety of fungi are capable of causing respiratory infections in birds, however, *Aspergillus fumigatus* is the organism most commonly isolated within mycotic lesions. *A. fumigatus* is considered to be an opportunistic pathogen that causes disease in birds that are otherwise debilitated by bacterial, viral, nutritional, traumatic, or toxic disease.

Aspergillosis is primarily a disease of captive birds, however, wild birds may also contract this fungal infection. Spores of *A. fumigatus* are common within the environment. Captive birds may be exposed to spores that occur within food, bedding or nesting material.

#### **Clinical Signs**

Wild rainbow lorikeets that are infected with psittacine circovirus are commonly presented to wildlife care centres with extensive mycotic pneumonia. Birds of prey, aquatic birds, and some passerine birds seem to be particularly susceptible to infection with *A. fumigatus*.

Birds with mycotic lesions in their respiratory tracts may exhibit a variety of clinical signs. Initial changes may reflect a subtle change in the voice, weakness, weight loss, or oculonasal discharge. Infection may become dormant, yet will most often progress to depression, emaciation, coughing or sneezing, and dyspnoea.

Birds with clinical aspergillosis will have a marked leucocytosis. Heterophilia occurs in the early stages of disease, and monocytosis reflects more chronic infection. Active fungal infection may result in toxic change within heterophils

#### **Pathology**

*A. fumigatus* may cause focal infections of the upper respiratory tract, in the sinuses or trachea. More often aspergillosis is associated with lesions within the pulmonary parenchyma and air sacs. Regardless

of the location of the lesion, mycotic plaques are formed that have a white to yellow capsule, and may have a green centre. This green coloration represents the presence of conidiophores, or fruiting bodies.

The histological appearance of aspergillosis is quite variable. Early lesions most often reflect focal granulomatous inflammation. Fungi often have an affinity towards blood vessels. *Aspergillus* sp. may spread from the primary site of infection into blood vessels resulting in vasculitis with widespread coagulation necrosis of adjacent tissues. Ischaemic necrosis associated with mycotic vasculitis most commonly occurs in the pulmonary parenchyma, but is also evident within the liver of some birds.

### **Diagnosis**

Definitive diagnosis of aspergillosis is dependent upon microbial culture and isolation of the organism within lesions. Clinical signs and the presence of leucocytosis may also be suggestive of aspergillosis. Endoscopic examinations are used to visualise fungal plaques within the air sac, lung or trachea. Cytologic examination of oculonasal discharges, or infraorbital lavage fluid may be useful in the diagnosis of mycotic sinusitis. Serology for the detection of aspergillosis is available at commercial veterinary laboratories. Radiographic examination may reveal multifocal densities within the lungs, trachea, or air sacs that reflect the presence of fungal plaques.

### **Prevention**

The prevention of aspergillosis in captive birds is based upon strict standards of hygiene. Bedding materials used for the care of birds should be clean and dry. Straw, hay, and other agricultural products should not be used as bedding or nesting material for birds. Bird food should be properly stored and regularly examined for the presence of fungi. Any husbandry technique that minimises the stress experienced by captive birds is likely to reduce the risk of aspergillosis. Some species are known to be particularly susceptible to aspergillosis. These birds may be often treated with antifungal agents on a prophylactic basis.

#### **1.5.2 Candidiasis**

*Candida* spp are yeasts that are commensal within the upper gastrointestinal tract of a variety of birds. Disease associated with an overabundance of this organism occurs most commonly in young, hand-raised birds. Candidiasis in young birds is associated with poor hygiene, and inappropriate hand rearing formulas. Adult birds that are administered broad-spectrum antibiotics orally or those birds that have

concurrent systemic illness are also susceptible to candidiasis. Occasional feather loss and hyperkeratosis in captive native birds have been associated with external candidiasis, but most of these birds also have yeast overgrowth within their gastrointestinal tracts (ARWH).

Overgrowth of *Candida* sp. within the oral cavity, oesophagus, proventriculus and ventriculus results in weight loss, depression, anorexia, crops stasis, regurgitation, and diarrhoea. Oral infections may result in visible white plaques along the mucosa.

Diagnosis of candidiasis relies upon cytologic examination of smears made from oral lesions. Wet preparations of faeces may be examined microscopically for the presence of yeast. Gram stains and Diff Quik® stains are useful to illustrate the presence of yeast within smears. *Candida* spp. are commensal within the gastrointestinal tract, and scattered yeast cells within tissue smears or faeces are not unusual. The presence of large numbers of budding yeast, and pseudohyphae reflect active infection with *Candida* sp. *Candida* sp can also be identified through standard fungal culture techniques.

### 1.5.3 Mycotoxins

Mycotoxins are toxic products that can be produced by a wide variety of fungal species. Exposure to mycotoxins occurs when birds ingest mouldy grain. The clinical signs of mycotoxicosis will depend upon the type of mycotoxin ingested. Diagnosis of mycotoxin exposure is difficult since few laboratories will isolate mycotoxic fungi and quantify the toxins.

## 1.6 Nutritional Disease

Malnutrition, other than emaciation, is rare in free ranging wildlife. When malnutrition does occur in free ranging wildlife, it is primarily the result of inappropriate supplemental feeding by humans.

### 1.6.1 Nutritional Osteodystrophy

Nutritional osteodystrophy occurs most commonly in young birds that are hand raised with inappropriate diets. Free ranging magpies and kookaburra in Perth, Melbourne and Sydney are often provided with supplemental feeds consisting of lean meat. Lean meat contains scant calcium and abundant phosphorus, which can trigger nutritional osteodystrophy. Nutritional osteodystrophy occurs as a result of prolonged feeding of diets either deficient in calcium or vitamin D<sub>3</sub>, or diets that have a high concentration of

phosphorus. The clinical syndrome associated with nutritional osteodystrophy is called rickets in growing birds, and osteodystrophy in older birds. The primary lesion in animals suffering from rickets is failure to properly mineralise cartilaginous bone models. This lesion result in stunting, curved long bones, folding fractures of the long bones, a soft beak and poor feather growth. Gross post mortem examination may also reveal hypertrophy of the parathyroid glands as a result of hyperphosphataemia. Treatment of advanced osteodystrophy bears a poor prognosis. If birds are presented with early lesions, such as a soft beak, and do not have any fractures, treatment through calcium supplementation and an appropriate diet may be successful.

### 1.6.2 Thiamine Deficiency of Red Wattlebirds

Red wattlebirds wintering in Melbourne have historically been reported to suffer thiamine deficiency manifested by opisthotonus, convulsions, muscle spasms, and wing flapping. Some birds will respond to injections of thiamine.

Thiamine deficiency in red wattlebirds was thought to occur as a result of altered migration patterns. These birds ceased migrating north for winter due to the presence of flowering ornamental shrubs planted in suburban gardens. Red wattlebirds are both nectivorous and insectivorous; however, they obtain most of their thiamine from insects. Nectar contains only 0.01 to 0.02  $\mu\text{g}$  thiamine per kilojoule of energy (Pass, 1993). Insects normally supply both amino acids and thiamine (4  $\mu\text{g}$  thiamine per gram of insect). Research has demonstrated that red wattlebirds require 200 kilojoules of energy per day and 20  $\mu\text{g}$  of thiamine per day (Paton et al., 1983). Thus, a red wattlebird can obtain its daily thiamine requirements by eating as few as five large insects. Large insects are not available in Melbourne in winter, and a red wattlebird would have to ingest up to 500 small insects to obtain their daily thiamine requirement. Since only 9% of the red wattlebird's foraging time is invested in searching for insects, it is very difficult for these birds to ensure adequate thiamine intake in winter (Paton et al., 1983). Rehabilitation centres in Victoria report that they have not seen this condition in many years.

Piscivorous birds maintained in captivity can also develop neurological signs of thiamine deficiency due to the presence of thiaminase in fish. Captive seabirds are usually provided with oral thiamine supplementation.

## 1.7 Toxicity

### 1.7.1 Botulism

#### Aetiology

Botulism is a paralytic disease of birds resulting from ingestion of bacterial toxins. *Clostridium botulinum* is a gram positive, spore-forming bacterium that is capable of forming seven types of botulinum toxin.

Sporadic outbreaks of botulism occur in both urban and rural environments. Significant risk factors associated with botulism toxicity include:

- an abundance of bacterial spores in the environment due to previous botulism outbreaks,
- birds that utilise the margins of waterways,
- decomposing vegetation, especially at the water margin,
- intermittent flushing of fertiliser run off or sewage into the waterway, which results in surges in plant growth and subsequent plant death,
- elevated water temperature due to increased environmental temperature and shallow waterways,
- stagnant water, and
- an abundance of flies.

These conditions can also promote the growth of blue green algae, which can produce toxins. Blue green algae toxicosis should be considered among the differential diagnosis for botulism.

Maggots are relatively resistant to the toxic effects of botulinum toxic. Thus, maggots are an important source of toxicosis as they may contain large concentrations of botulinum. Ingesting as few as two maggots that contain botulinum can kill a bird.

#### Clinical Signs

Birds suffering from botulism will have paresis or paralysis of the legs, wings, and neck. The first sign of intoxication is usually a drooping head. Impaired vision, difficulty in opening eyelids, difficulty swallowing and paralysis of the third eyelid rapidly follow. Once the legs have become paralysed, the bird may attempt to locomote by weakly flapping its wings. Birds with botulism may succumb to drowning, predation, or they asphyxiate due to paralysis of the respiratory musculature.

## **Pathogenesis**

Type C toxin is most commonly associated with avian botulism. Botulinum toxin inhibits the release of acetylcholine at the motor end plates resulting in peripheral neuropathy.

Clostridial spores persist in the environment and are very resistant to heat and desiccation. Botulinum toxin is only created during multiplication of the vegetative form of the bacterium. The optimum microenvironment for the growth of *Cl. botulinum* includes an anaerobic environment, pH ranging between 5.7 and 6.2, high temperatures, and a protein source. Rotting vegetation and carcasses of birds provide ideal conditions for the growth of *Cl. botulinum* and production of botulinum.

## **Pathology**

Gross and histological examination of birds that have died of botulism are unrewarding. Occasionally maggots are evident within the oesophagus or proventriculus.

## **Diagnosis**

A presumptive diagnosis of botulism is based upon the clinical signs exhibited and lack of significant lesions on post mortem examination. Definitive diagnosis of botulism requires identifying the toxin within serum or gastrointestinal contents. Mouse inoculation has been used to illustrate the presence of botulinum toxin, however, ELISA tests are now available for types C and D toxins. These ELISA tests are more sensitive, specific and humane than mouse inoculation. ELISA tests can also be conducted in large numbers in the field in the face of an outbreak.

## **Treatment**

Symptomatic treatment and supportive care are the primary means of treating botulism. Cathartic agents may aid in flushing the source of toxin from the gastrointestinal tract. Botulinum antitoxin has been recommended in the literature; however, it is not commercially available.

## **Prevention**

The prevention of botulism in urban environments depends upon management of ponds. Ornamental ponds should be designed to incorporate aeration, water circulation, steep sides and sufficient depth to keep water temperature stable. The layout should prevent water that is rich in organic material from flowing into the pond. Decomposing vegetation and other organic matter should be regularly removed

from any pond. Animal carcasses should also be regularly removed to prevent the build up of flies and maggots in the environment.

### 1.7.2 Organochlorine toxicity in Tawny Frogmouths

#### **Aetiology**

Tawny frogmouths along the east coast of Australia are found moribund, weak, vocalising or convulsing during late winter and early spring of each year.

Toxicity was suspected as the cause of this syndrome, since birds consistently died without evidence of infectious, inflammatory, or parasitic disease. The first report of possible organochlorine (OC) toxicity in a tawny frogmouth was published in 1981. A tawny frogmouth originating from Victoria was found to have elevated concentrations of lindane, heptachlor, and alpha-benzene-heptachlor in the brain (Fleay, 1981).

A prospective investigation into tawny frogmouth mortality was undertaken in 1994 in a collaborative effort between W.I.R.E.S. and Veterinary Pathology Services. This investigation revealed elevated concentrations of four OC compounds within liver and brain samples collected from tawny frogmouths that died suddenly in 1994 (Charles, 1995). The tissue concentrations of these compounds were sufficient to explain the clinical neurological signs and mortality when compared with concentrations required to cause disease experimentally in domestic animals.

#### **Organochlorine residues in the liver and brain of tawny frogmouths** (Charles, 1995).

| <b>Analyte</b>         | <b>Mean Tissue Concentrations (mg/kg wet weight)</b> |                              |
|------------------------|--|------------------------------|
|                        | <b>Liver (n = 8)</b>                                 | <b>Brain (mg/kg) (n = 8)</b> |
| oxychlorane            | 1.1 (0.3 - 1.4)                                      | 1.0 (.35 - 2.3)              |
| heptachlor-epoxide     | 15.2 (7.1 - 39.0)                                    | 11.1 (5.5 - 21.0)            |
| DDE                    | 1.4 (0.1 - 4.8)                                      | 5.6 (0.14 - 3.0)             |
| Dieldrin               | 7.1 (1.1 - 23.0)                                     | 5.5 (1.0 - 7.1)              |
| Total OC Concentration | 24.7 (13.1 - 65.8)                                   | 18.0 (10.85 - 28.97)         |

Chlorinated hydrocarbons have been used primarily as insecticides. Although OC use was tightly regulated as long ago as 1987, the use of certain OC has been permitted for termite eradication. The widespread use of heptachlor, aldrin, and dieldrin for domestic cockroach and termite control has most likely provided a persistent environmental source of OC for tawny frogmouths (Charles, 1995).

### **Clinical Signs**

Tawny frogmouths with OC toxicity abandon their nocturnal habits and are active during the day. Most of these birds are found weak, and unable to fly. Upon examination, leg extension, head tilt, opisthotonus, dilated pupils, blindness, droopy or closed eyes are noted. Tawny frogmouths with organochlorine toxicity may be hyperexcitable or may have central nervous system depression. Hyperexcitable birds convulse in response to stimuli, vocalise repeatedly, and undertake repetitive jerky motions. Birds with central nervous system depression are recumbent with a dazed appearance, or they are moribund.

### **Pathogenesis**

The pathogenesis of OC toxicity is poorly understood and can vary depending upon the exact compound involved. The presence of two or more OC within tissues may exert a synergistic effect. Some OC compounds, however, are more toxic than others, and metabolites can be more toxic than the parent compound (heptachlor-epoxide is 10 times more toxic than its parent, heptachlor). The clinical effects of toxicity also vary depending upon host factors such as age, sex, metabolism, and patterns of anorexia.

Central nervous system effects of OC are suspected to primarily occur as a result of altered axonal impulse transmission. Organochlorine compounds are highly soluble in lipids. Slow metabolism and excretion of these chemicals occurs during the normal turnover of lipid rich tissues. Organochlorines are persistent both in the environment and in tissues. Due to their persistent nature, OC compounds biomagnify within the food chain. Outbreaks of OC toxicosis may be associated with acute exposure to the chemicals, or may be due to catabolism of tissues that have stored a large OC concentration through repetitive low concentration exposure.

Epidemiological data suggests that tawny frogmouths are exposed to toxic concentrations of OC compounds over time, and that acute toxicity develops as a result of mobilisation of lipids in times of low food availability. Organochlorine chemicals enter the body through inhalation, percutaneous absorption, and ingestion.

Non-lethal effects of OC exposure in avian species can include: induction of hepatic microsomal mixed function oxidase enzymes, which may result in increased metabolism of exogenous toxins, and endogenous steroid hormones, altered ATPase driven calcium transport, which causes thinning of egg shells, deformity of chicks exposed to high concentrations of OC sequestered within yolk, altered behaviour patterns.

### **Pathology**

The gross and histologic examination of birds suffering from OC toxicity demonstrates few significant findings. Non-specific findings upon gross post mortem of tawny frogmouths with OC toxicity include emaciation, microcardia, microhepatica and generalised pallor. Pulmonary, hepatic, renal, and pancreatic congestion, and hepatic and pancreatic atrophy are noted in experimentally induced OC toxicity in other avian species.

### **Diagnosis**

Diagnosis of OC toxicity is based upon determining the concentration of organochlorine compounds within the tissues. Liver, adipose tissue, and brain are collected into aluminium foil during post mortem examination. These samples may be frozen prior to submission to a toxicology laboratory.

### **Treatment**

There is no specific treatment for OC toxicity. Seizures may be controlled with intravenous or intramuscular diazepam, however, even birds that are sedated for prolonged periods often do not recover (Charles, 1995). Atropine may be administered, in low doses, to reduce the effects of excessive stimulation of the parasympathetic nervous system (Blus et al., 1996). Even with intensive supportive care, the prognosis for tawny frogmouths with OC toxicity remains poor.

### **Prevention**

Despite recent legislation regulating the use of these chemicals, the persistent nature of organochlorine compounds in the environment may result in toxicity in wildlife for years to come.

### 1.7.3 Oil Toxicity

Oil spills occur in coastal environments as a result of marine pollution. Small inland oil spills are not as visually dramatic as coastal spills, but they occur with greater frequency. Oil reaches inland waterways after vehicle accidents that release petrochemicals into stormwater drains, and when petrochemicals are accidentally or maliciously dumped into streams and creeks. The products that reach waterways are diverse, ranging from vegetable oil to heavy bunker fuel.

Sea birds and shore birds are commonly affected by oil spilled into waterways since they live at the water's surface where oil accumulates. The toxic effects of oil are as diverse as the products spilled. The external effects of oil exposure may include irritation of mucous membranes and displacement of air from the porous structure of the feather. This alteration in feather structure leads to altered function, such as loss of insulating properties, buoyancy, and flight. Internal effects of oil exposure include aspiration pneumonia, inflammation of the gastrointestinal tract, altered activity of hepatic microsomal enzyme systems and haemolysis. Exposure to oil can also result in altered reproductive behaviour and physiology.

The initial goals of treating oiled birds are to prevent further ingestion of oil during preening, and to medically stabilise the animal through supportive care. Small ponchos can be created to cover the bird's plumage to prevent preening. The eyes, feet and oral mucosa are flushed with saline to remove irritating toxins. Ophthalmic ointments may be applied to soothe inflamed conjunctiva. Supportive care includes rehydration, cathartic agents or other medications that reduce gastrointestinal irritation, and nutritional support. Removing oil from the plumage can be a tedious task, and should not be undertaken until the bird is medically stable.

### 1.7.4 Lead Toxicity

Wild birds are exposed to lead in the form of fishing sinkers and ammunition. Waterfowl ingest the lead pellets that accumulate on the soft mud bottom of waterways. Lead that is lodged within muscle is not a source of lead poisoning. Birds that ingest lead shot embedded in the tissues of their prey, however, may suffer lead poisoning. Captive birds that lick recently galvanised wire mesh may be exposed to toxic concentrations of lead or zinc.

Clinical signs of lead poisoning include a depression, weakness, regurgitation, vomiting, diarrhoea, droopy wings, tremors, and convulsion. Lead poisoning is diagnosed when blood lead concentrations are

elevated. Radiographic examination of the bird may reveal the presence of radio-dense particles within the gastrointestinal tract.

Treatment of lead poisoning includes removing lead from the gastrointestinal tract using cathartic agents. Chelating agents, such as calcium disodium edetate, bind to lead within the blood stream and aid in its elimination.

Refer to section 6.5.1 for further information regarding lead poisoning.

### 1.7.5 Organophosphate Toxicity

#### **Aetiology**

Organophosphate and carbamate compounds are contained within insecticides, herbicides, and fungicides. A broad range of species is susceptible to the toxic effects of these compounds. Birds and bats can be exposed to organophosphates when they eat contaminated insects or vegetation, and when they fly through aerosolised chemicals during application. Unfortunately, access to these organophosphate compounds also occurs through malicious poisoning.

#### **Clinical Signs**

Birds that are exposed to organophosphate compounds may be found salivating, dyspnoeic, ataxic, with tremors, convulsing, paralysed, regurgitating, and with diarrhoea. Many animals subject to organophosphate toxicity are found dead. Death most often occurs as a result of paralysis of the respiratory muscles and ischaemia.

The effect of exposure to lower concentrations of organophosphates and carbamates is not certain. Reproductive success may be altered due to changes in physiology and behaviour subsequent to exposure to these compounds. Birds experimentally exposed to organophosphates in their food lay fewer eggs, abandon their nests, and have altered feeding and activity patterns (Fairbrother, 1996).

#### **Pathogenesis**

Organophosphates and carbamates inhibit the activity of cholinesterase. Acetylcholine released into the inter-neuronal space during impulse transmission is degraded by cholinesterases that, thus, stop signal transmission. Inhibition of cholinesterase activity results in continuous firing of neurons. Clinical signs

of organophosphate and carbamate toxicity occur as a result of over stimulation of the parasympathetic nervous system, skeletal muscles, and, to a lesser degree, the central nervous system.

Organophosphates and carbamates compete for binding sites on the cholinesterase molecule. Organophosphates bind to the enzyme in a reversible fashion, whereas carbamates are irreversibly bound.

### **Pathology**

The gross and histologic examination of birds suffering from organophosphate and carbamate toxicity is usually unremarkable. Occasionally a bird will have evidence of diarrhoea, salivation or increased respiratory secretions.

### **Diagnosis**

When birds are found dead in good body condition and significant lesions are not evident on gross post mortem examination, toxicity should be suspected. A diagnosis of organophosphate or carbamate toxicity is established through measurement of cholinesterase activity in the blood or brain, or analysis of ingesta for organophosphate and carbamate compounds and metabolites using high pressure liquid chromatography. Samples of the brain are wrapped in aluminium foil and may be frozen prior to submission to a laboratory. Samples of brain and ingesta may be stored for up to six months at 80°C prior to submission to a laboratory.

### **Treatment**

Atropine is administered to treat intoxication with anticholinesterase compounds. If the bird is not cyanotic, 2-PAM can be administered. 2-PAM will not reverse the effects of carbamate toxicity.

### **Prevention**

Prevention of organophosphate toxicity relies upon judicious use and storage of the chemical agents.

## **1.8 Traumatic Injury**

### **1.8.1 Skeletal Injury**

Fractures of long bones are commonly encountered in injured birds. The prognosis of return to full function should be carefully considered prior to attempting fracture management. A thorough physical and radiographic examination will assist in the identification of other injuries, such as joint damage, and

soft tissue injuries that may have an impact on the bird's overall prognosis. Evaluation of the blood and nervous supply distal to the fracture, and evaluation for potential underlying causes such as bone infection or metabolic bone disease, is imperative prior to mounting any attempts at fracture repair.

Luxations and subluxations are difficult to manage in birds. Bandaging techniques to stabilise the joint also contribute to joint stiffness.

Blunt trauma to the chest can result in a transverse fracture of the keel. The irregular fragments of the fractured keel must be forced into the coelomic cavity at the time of trauma, since many of these birds sustain extensive myocardial contusions or hepatic rupture and haemorrhage.

Fractures that occur along the pectoral girdle can be very difficult to palpate, and the bird may only have a droopy wing. Coracoid fractures occasionally have sharp fracture fragments that lacerate the brachiocephalic trunk or the cardiac musculature, resulting in death due to massive haemorrhage. Coracoid fractures should be stabilised as quickly as possible. Radiographic examination of these birds is indicated to assess the full extent of tissue damage. A figure eight bandage and cage rest may result in satisfactory repair of non-displaced fractures. Birds of prey, however, may require surgical correction of fractured coracoid bones through intra-medullary pinning to regain sufficient flight to be releasable.

Humeral fractures are most often repaired with retrograde intramedullary pinning procedures and post surgical figure-eight bandages that are secured with a body wrap.

Radial and ulnar fractures frequently occur in birds subject to trauma. If one bone is intact, and the fracture is non-displaced, conservative management with a figure-eight bandage is often sufficient for bone repair and return to flight. If both the radius and ulna are fractured, the ulna should be treated with either intramedullary pinning and a figure of eight bandage, or external fixation.

Fractures of the carpus and phalanges are associated with extensive soft tissue injury. These fractures often result from high-energy trauma, which produces highly comminuted fractures. The prognosis for return to flight is poor when fractures occur within or distal to the carpus.

Femoral fractures are most often repaired with intramedullary pinning procedures; however, cage rest may be sufficient for return of function when the fracture is non-displaced.

Splints or other external fixation techniques are primarily used to stabilise tibiotarsal and tarsometatarsal fractures. These bones rely heavily upon their medullary blood supply. Intramedullary pinning or KE pins that interfere with the blood supply of these bones may result in ischaemic necrosis.

Fractured toes may be amputated if the wounds are severe. Alternatively the foot may be bandaged. The contralateral foot must be monitored closely for bumblefoot, due to increased weight bearing.

Spinal fractures and luxations may result in paresis, paralysis and an inability to void the cloaca. Intensive nursing care is required to support these birds, and the prognosis for recovery is guarded when birds have significant neurological deficits. Spinal luxations and fractures most often occur at the junction of the thoracic and lumbar vertebrae. It has been proposed that the thorax is very rigid, and thoracic trauma results in damage at the first flexible vertebral junction (Bill Hartley, personal communication).

Beak injuries occur occasionally in wild birds. The prognosis for these birds depends upon the degree of damage. If only the tip of the beak is injured, bleeding may be stopped with cautery and the rough edges trimmed. Trimming the opposing beak may aid the bird in prehension of food. Surgical glue, bone cement, fibreglass, or cerclage wire may be used to construct beak prosthetics. Prosthetic devices often require routine monitoring and intermittent replacement and should not be placed on birds to be released. When extensive beak damage occurs in a wild bird, the bird is unlikely to return to a releasable state.

### 1.8.2 Soft Tissue Injury

Uncomplicated soft tissue injuries in birds heal relatively rapidly due to effective contraction and epithelialisation. Soft tissue wounds may be left open and a sliding grafting procedure undertaken once there is a healthy granulation bed.

**Scalping injuries** occur in wild and captive female birds as a result of intraspecific aggression. These injuries can expose a large portion of the cranium. Successful management of scalping injuries most

commonly relies upon initial debridement, and wound lavage, followed by a period of open wound management.

**Exertional myopathy** is occasionally reported in birds subjected to exertion through chase, fear or isometric forces during restraint. Lameness, shifting leg lameness, weakness, and recumbency are clinical signs associated with avian exertional myopathy. Elevated serum concentrations of AST, CK and LDH may occur in affected birds. Recommended treatment regimes for exertional myopathy consist of fluid therapy, glucocorticoid administrations to stabilise membranes, and administration of vitamin E and selenium. Diazepam, at a dose of 0.5 to 1.0 mg/kg IM or IV, may aid in relaxation and increase perfusion of injured tissue. Prevention of exertional myopathy is dependent upon minimising stressful stimuli and proper care when capturing and restraining birds.

**Pododermatitis**, or bumblefoot, is a common injury of birds of prey, waterfowl, and pelagic birds in captivity or rehabilitation care. This injury is often attributed to poor hygiene or inappropriate substrates and perches within the bird's enclosure. Calluses, or nodules of hyperplastic epidermis, along the plantar surfaces of the feet are the earliest clinical signs of pododermatitis. These lesions often go unnoticed and progress to ulceration and infection. Chronic ulceration and secondary bacterial invasion may then lead to infectious tenosynovitis.

Treatment of pododermatitis focuses on improved hygiene and perching surfaces. Soft tissue wound management, including wound cleansing, flushing and bandaging, and sometimes surgical debridement are used to accelerate healing of the lesions.

**Bite wounds** inflicted by cats often inject large quantities of bacteria deep into the tissues. Septicaemia, caused by *P. multocida*, is a common sequela of cat bite wounds. Parenteral antibiotics and thorough wound management are indicated for bite wounds. The prognosis for recovery from bite wounds is guarded, even when the wounds do not appear to be extensive. Euthanasia should be considered when bite wounds are extensive or the bird is already debilitated.

### 1.8.3 Central Nervous System Injury

Cranial trauma commonly occurs when birds fly into stationary objects. Blood tinged mucous within the oral cavity, periocular contusions and hyphaema, are often associated with cranial injury. Anisocoria,

nystagmus, ataxia, head tilt, tremor, and paresis may also indicate central nervous system trauma. A thorough neurological examination should be conducted, and the presence of deep pain perception evaluated in order to formulate a prognosis for each bird.

## **1.9 Diseases of Uncertain Aetiology**

### **1.9.1 Encephalomyelitis of Rainbow Lorikeets**

#### **Aetiology**

A neurologic syndrome in rainbow lorikeets occurs primarily within Sydney and the Central Coast of New South Wales, but has also been observed in Queensland and Victoria. This syndrome is known as encephalomyelitis of rainbow lorikeets, and clench claw syndrome. A variety of injuries or infections may result in the clinical signs of clenched claws; thus, the term encephalomyelitis will be used in this discussion.

Sub-adult and adult birds are affected by encephalomyelitis and the disease may occur at any time of year. The aetiological agent responsible for encephalomyelitis has not yet been discovered, despite reports dating back to 1986 suggesting a viral aetiology (McOrist and Penny, 1986). Psittacine circovirus, and Newcastle's disease virus have been proposed as potential aetiological agents. Immunohistochemistry upon paraffin embedded nervous tissues from 10 rainbow lorikeets with encephalomyelitis did not indicate the presence of Newcastle disease virus (ARWH).

#### **Clinical Signs**

Lorikeets with encephalomyelitis present with clenched feet, which is the natural resting position of the avian foot. Upper motor neuron damage results in a bird assuming a clench claw posture. Histopathologic examination of the nervous system is required to rule out traumatic injury and reveal non-suppurative leptomeningitis consistent with encephalomyelitis syndrome.

Additional clinical signs associated with encephalomyelitis in rainbow lorikeets include progressive paralysis of the legs and body. Affected birds are bright and alert, but may have a head tilt, unusually worn beak, or intention tremors. The bird's body condition may be good or the bird may be emaciated and dehydrated. Young lorikeets, with dark brown beaks, are most often diagnosed with this syndrome.

#### **Pathology**

Birds with encephalomyelitis do not have significant lesions evident upon gross post mortem examination.

Histologic examinations reveal non-suppurative inflammation within the cerebellar white matter, caudal brainstem and spinal cord. There are multifocal perivascular cellular cuffs composed of 2 - 3 cell layers of lymphocytes, plasma cells and macrophages. The endothelium of these blood vessels often appears plump or proliferative. Foci of mononuclear cell infiltration are often visible within the spinal sensory and motor nerve rootlets. Some birds also have foci of neuronal necrosis, astrocytosis, vacuolation of the neuropil, and nerve cell necrosis within the hind brain or spinal cord. Wallerian degeneration is frequently observed in the spinal cord and peripheral nerves of birds suffering from encephalomyelitis (Pass, 1993, ARWH). Recently, several rainbow lorikeets with encephalomyelitis have had concurrent fibrinous pericarditis, primarily within the epicardium surrounding the greater vessels (ARWH).

Many of the young rainbow lorikeets that suffer from encephalomyelitis virus have concurrent psittacine circovirus infection, diagnosed through the presence of characteristic intracytoplasmic inclusion bodies in reticuloendothelial cells within the bursa of Fabricius (ARWH). No thorough epidemiological study has been conducted, however.

### **Diagnosis**

The diagnosis of avian encephalomyelitis relies upon the presence of clinical signs relating to altered central nervous system function, in conjunction with histologic evidence of non-suppurative encephalomyelitis. Differential diagnoses for encephalomyelitis include traumatic injury and focal symmetrical poliomyelomalacia.

### **Treatment**

There is no known treatment for encephalomyelitis in rainbow lorikeets.

#### **1.9.2 Magpies and Currawongs**

A syndrome of poor body condition and very poor feathering is emerging in young magpies and currawongs. Down feathers are often retained after growth of primary and secondary feathers, and those feathers that do emerge show severe stress-banding. In some birds it is associated with rickets. These birds are usually heavily parasitised by a wide variety of pathogenic organisms, for example, birds have several of the following: pox virus lesions, *Xenocordon* species burrowing through the oral mucosa, pox

lesions and ventriculus invaded by yeast and fungal hyphae, a heavy burden of intestinal coccidia, and a heavy burden of cryptosporidia within the mucosa of the bursa of Fabricius, Leucocytozoon infection. Ectoparasites are often present on the feathers and Cnemidocoptes infestation is evident on the skin on the feet. The cause of the poor body condition, bone and feather abnormalities in these birds is suspected to be a poor absorption of nutrients associated with these infections.

## 2 Animals mentioned in text

### 2.1 Aves

Little penguin (*Eudyptula minor*)

Short-tailed shearwater (*Puffinus tenuirostris*)

Australian gannet (*Morus serrator*)

Straw-necked ibis (*Threskiornis spinicollis*)

Pacific black duck (*Anas superciliosa*)

Silver gull (*Larus novaehollandiae*)

Sulphur-crested cockatoo (*Cacatus galerita*)

Galah (*Cacatua roseicapilla*)

Red-tailed black cockatoo (*Calyptorhynchus magnificus*)

Yellow-tailed black cockatoo (*Calyptorhynchus funereus*)

Rainbow lorikeet (*Trichoglossus haematodus*)

Scaly-breasted lorikeet (*Trichoglossus chlorolepidotus*)

Red-collared lorikeet (*Trichoglossus haematodus rubritorquis*)

Australian king parrot (*Alisterus scapularis*)

Cockatiel (*Nymphicus hollandicus*)

Budgerigar (*Melopsittacus undulatus*)

Laughing kookaburra (*Dacelo novaeguineae*)

Tawny frogmouth (*Podargus strigoides*)

Black-faced cuckoo-shrike (*Coracina novaehollandiae*)

Brown treecreeper (*Climacteris picumnus*)

Red wattlebird (*Anthochaera carunculata*)

Gouldian finch (*Erythrura gouldiae*)

House sparrow (*Passer domesticus*)

Magpie-lark (*Grallina cyanoleuca*)

Pied butcherbird (*Cracticus nigrogularis*)

Pied currawong (*Strepera graculina*)

Australian magpie (*Gymnorhina tibicen*)

Long-billed corella (*Cacatua tenuirostris*)

Peregrine falcon (*Falco peregrinus*)

Australian hobby (*Falco longipennis*)

Barking owl (*Ninox connivens*)

Rock parrot (*Neophema petrophila*)

Shy albatross (*Diomedea cauta*)

Blue-faced parrot finch (*Erythrura trichroa*)

White-eyes (*Zosteropidae*)

Eclectus parrot (*Eclectus roratus*)

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