Common Diseases of Urban Wildlife

BIRDS

PART 2

The Australian Registry of Wildlife Health

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.

TARONGA ZOO | WESTERN PLAINS ZOO
Zoological Parks Board of New South Wales

1.1 Fungal Disease

1.1.1 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*.

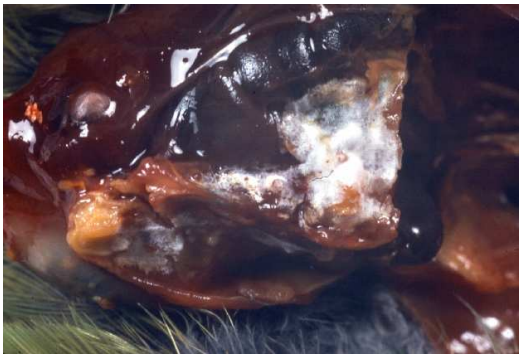
Birds with mycotic lesions in their respiratory tracts may exhibit a variety of clinical signs. Initial changes may reflect a subtle change in the voice, weakness, weight loss, or oculonasal discharge. Infection may become dormant, yet will most often progress to depression, emaciation, coughing or sneezing,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

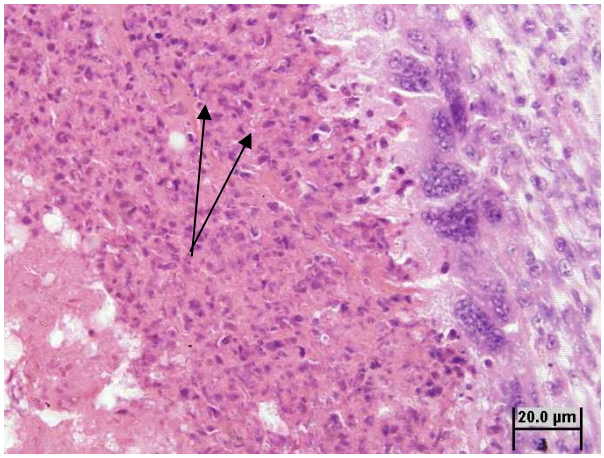
A. fumigatus may cause focal infections of the upper respiratory tract, in the sinuses or trachea. More often aspergillosis is associated with lesions within the pulmonary parenchyma and air sacs. Regardless of the location of the lesion, mycotic plaques are formed that have a white to yellow capsule, and may have a green centre. This green coloration represents the presence of conidiophores, or fruiting bodies.

Lung granulomas, Aspergillosis

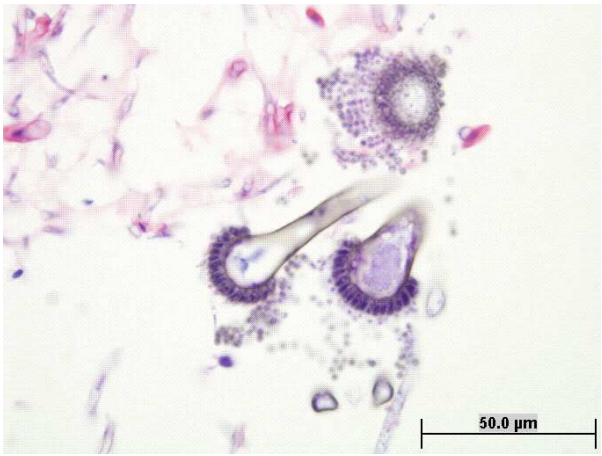


Air sac lined with fungal elements, aspergillosis.

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.



Granulomatous inflammation with giant cells, *Aspergillus* infection.



Conidiophores with associated spores, *A fumiogatus*. Note also hyphae (closed arrows)

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.

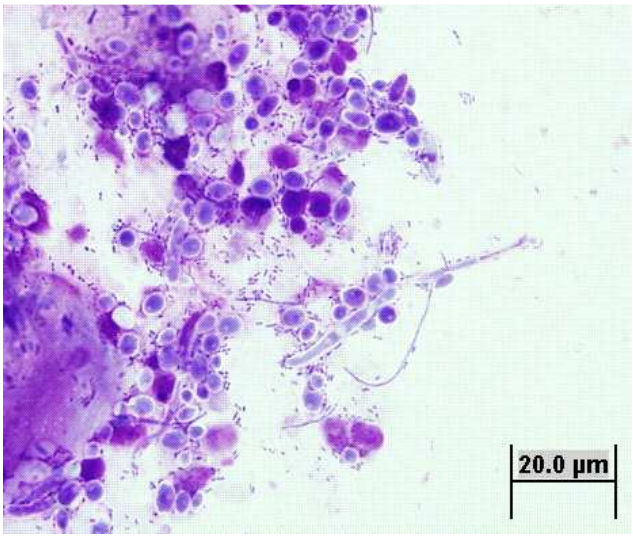
Prevention

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 are often treated with antifungal agents on a prophylactic basis.

1.1.2 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.



Budding yeasts and hyphae. *Candida albicans*

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.

1.1.3 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.

1.2 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.

1.2.1 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_3, 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.

Soft beak, osteodystrophy

1.2.2 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 *u*g thiamine per kilojoule of energy (Pass, 1993). Insects normally supply both amino acids and thiamine (4 *u*g thiamine per gram of insect). Research has demonstrated that red wattlebirds require 200 kilojoules of energy per day and 20 *u*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.

1.3 Toxicity

1.3.1 Botulism

Aetiology

Botulism is a paralytic disease of birds resulting from ingestion of bacterial toxins. *Clostridium bolutinum* 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

Paresis of the neck. Botulism, Pacific Black Duck

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 carcases 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 carcases should also be regularly removed to prevent the build up of flies and maggots in the environment.

1.3.2 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).

	Mean Tissue Concentrations (mg/kg wet weight)

Analyte	Liver (*n* = 8)	Brain (mg/kg) (*n* = 8)
oxychlordane	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, opishotonus, 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 oganochlorine compounds in the environment may result in toxicity in wildlife for years to come.

1.3.3 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.

1.3.4 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.

1.3.5 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^{o}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.

1.4 Traumatic Injury

1.4.1 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.

1.4.2 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.

1.4.3 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.

1.5 Diseases of Uncertain Aetiology

1.5.1 Encephalomyelitis of Rainbow Lorikeets

Aetiology

"Clenched claw syndrome", rainbow lorikeet

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 aetiologic 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 aetiologic 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

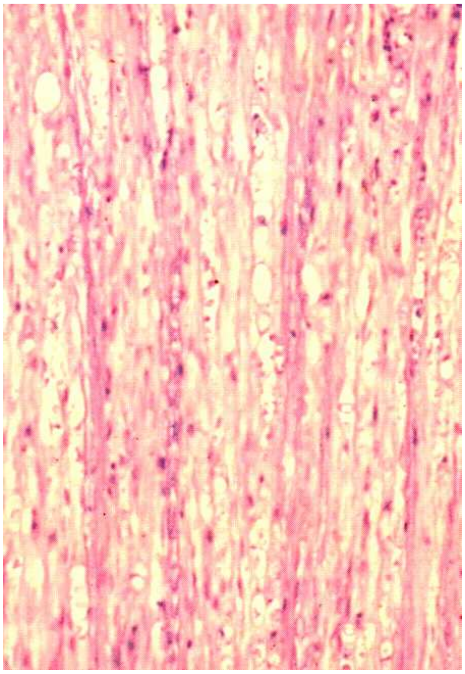
Lorikeets with encephalomyelitis present with clenched feet, which is the natural resting position of the avian foot. Upper motor neuron damage results in a bird assuming a clench claw posture. Histopathologic examination of the nervous system is required to rule out traumatic injury and reveal non-suppurative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

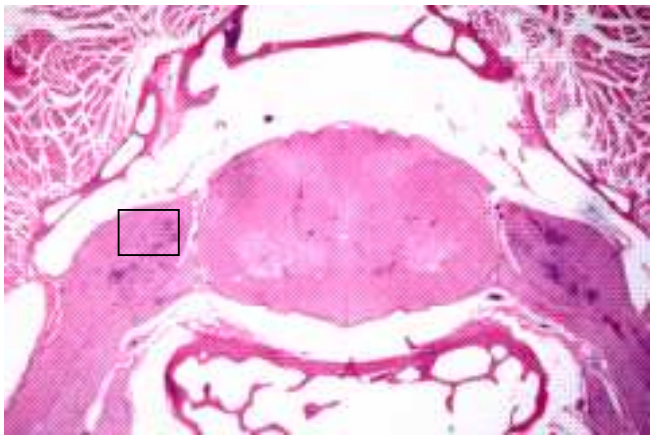
Pathology

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

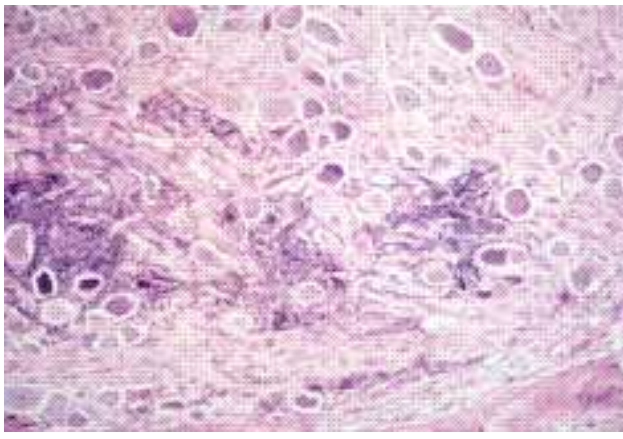
Histologic examinations reveal non-suppurative inflammation within the cerebellar white matter, caudal brainstem and spinal cord. There are multifocal perivascular cellular cuffs composed of 2 - 3 cell layers of lymphocytes, plasma cells and macrophages. The endothelium of these blood vessels often appears 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).



Wallerian degeneration, spinal cord. Note axonal vacuolation and digestion chambers containing phagocytic cells.



Inflammatory infiltrates spinal cord and nerve roots



Inflammatory infiltrate, spinal nerve root ganglion.(see inset left)

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.

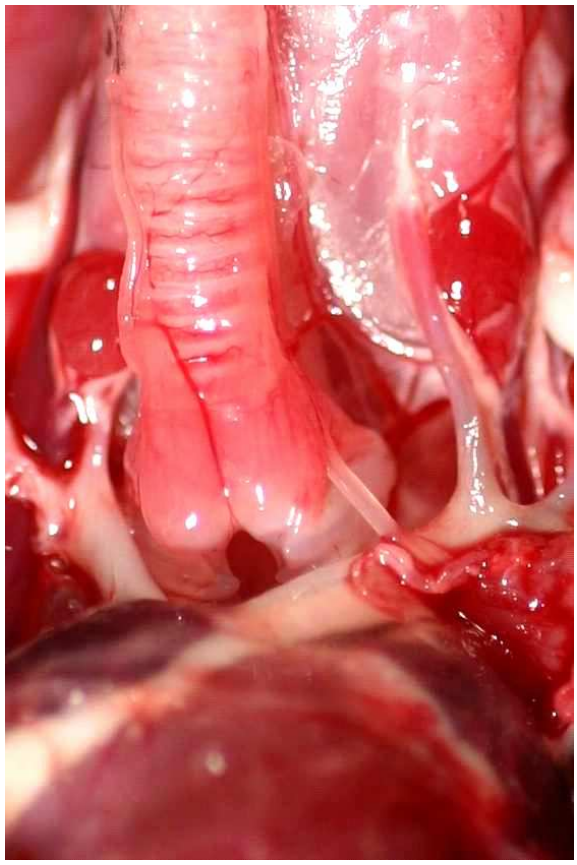
Treatment

There is no known treatment for encephalomyelitis in rainbow lorikeets.

1.5.2 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.

Abnormal feathering, soft beak, Pied Currawong.


Parathyroid hyperplasia (arrow), metabolic bone disease, Pied Currawong. (T = thyroid)

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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