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Everything You Need to Know about Birds (Not Chickens!) Mini Series

Session Three: Medicine and Surgery of Avian Species

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Session three

Common medical conditions of birds

There are a number of common medical problems presented in clinical practice. It is best these are grouped according to clinical presentation and major system involved and the most relevant diseases discussed.

Respiratory tract disease.

Dyspnoeic birds are commonly presented. Not all dyspnoeic birds suffer from respiratory tract disease. Egg binding, coelomic fluid, organomegaly, cardiovascular disease, lead poisioning and goitre (in budgerigars) can lead to signs of dyspnoea.

Upper respiratory tract disease is common. The infraorbital sinus occupies much of a birds skull and has many diverticula. Primary and secondary infections are common. Swelling of the sinus due to discharge and infection can lead to inflammation of the conjunctiva and periorbital swelling due to an extension called the infraorbital diverticulum which lies close to the orbit. This can lead to a serous discharge which can progress to a mucopurulent discharge. Infection can become inspissated and encapsulated. Infections such as *Aeromonas, Mycoplasma, Pseudomonas*, Mycobacterium, *Aspergillus, Candida* and *Chlamydophila* are common. *Mycoplasma* and *Chlamydophila* common in cockatiels. Poor nutrition such as vitamin A deficiency. Treatment can include sinus flushes, nebulisation and surgical debridement. Sterile abscess can also occur sublingually due to hypovitaminosis A.

Rhinoliths can develop as a result of sinusitis or rhinitis. Pre disposing factors to the development include any irritants of the respiratory epithelium such as vitamin A deficiency and tobacco smoke. A large rhinolith can destroy soft tissue and lead to bone lysis and formation of the sinus passages. Infection can involve bacteria and fungi (*Aspergillus spp*.), and there may be an associated Chlamydophila or Mycoplasma infection. In budgerigars, infection can be secondary to hyperkeratosis associated with *Cnemidocoptes sp*. mite infections. Treatment involves mass removal, culture and sensitivity then flushing with an appropriate antibiotic diluted in sterile saline. Small masses can be removed conscious but bleeding may be a complication. Reoccurrence is frequent if there has been damage to the nasal passage architecture. Frequent flushing after removal and correcting underlying predisposing factors are important. Educating owners about diet and inhaled irritants is important.

The trachea is wide and long but undergoes a 30% narrowing at the thoracic inlet and is ofsiginifcance when performing tracheoscopy. In some species (herons, swans) the trachea channels subcutaneously before entering the thoracic inlet. The tracheal rings are complete in birds. The syrinx is a series of modified cartilages and membranes capable of vibration and usually found at the tracheobronchial bifurcation. The syrinx is analogous to a mammal's larynx. Male ducks have an asymmetrical, ossified syryngeal bullae which should not be confused with a neoplastic mass.

Tracheal inflammation is unusual in the commonly seen bird species in general practice. Mycotic tracheitis is seen and typically causes syringeal obstruction. A change in or loss of voice is almost pathognomic for this condition. The pathogenesis for this is acute and granuloma formation develops over 12 days. An air sac tube should be placed urgently and the upper and lower respiratory tract evaluated endoscopically. Granulomas can be removed by biopsy or suction under direct visualisation with an endoscope or broken up with the hope they will pass through the bronchi and into the caudal thoracic air sac. Some infections can occur in the bronchi and lead to obstruction and severe respiratory distress. CT is the only way to diagnose bronchial obstructions in birds and they are typically fatal as the bird fails to respond to therapy after removal of the air sac tube.

Tracheal obstruction can occur after feeding and typically presents with a choking bird. These cases are usually dead by the time they reach the vets, but once again an air sac tube is required as a priority. Tracheal Obstruction is occasionally seen in Cockatiels due to seed inhalation. Tracheal endoscopy and suction is required to facilitate removal of the obstruction. A catheter can be used to facilitate this.

Tracheal strictures are also possible and occur after intubation in particular species. Blue and gold macaws, scarlet macaws and owls seem overly represented to the extent that some authors avoid tracheal intubation of blue and gold macaws whenever possible, preferring mask induction or air sac ventilation. Strictures occur at the end of the tracheal tube and have been linked to chlorhexidine.

Parasitic obstruction and inflammation of trachea can be seen in wildlife casualties, passerines and raptors. This is due to gapeworm (*Syngamus trachea*) which can be identified on endoscopic examination. The air sac mite (*Sternostoma tracheacolum*) is common in canaries and gouldian finches. Air sac mites can be seen on transillumination of the passerines trachea or on endoscopic examination. Treatment is oral, subcutaneous or “spot on” ivermectin as well as husbandry changes to break the parasite life cycle.

The lungs are rigid and small and only minimally expansile. Pneumonia can be seen due to toxins, allergic, inflammatory or infectious disease. Inhaled toxins can lead to peracute respiratory disease and haemorrhage. This typically occurs around the ostia but can spread over the whole of the lung field. On endoscopic examination it is possible to identify congestion, fibrosis, abscesses (typically granulomas visible on the surface) or pigmentation due to smoke inhalation (anthracosis). Steroidal therapy may be considered in a low dose if the clinical history supports exposure to a toxin or an asthmatic response (typically blue and gold macaws). Lung biopsies can be taken endoscopically.

Bird’s do not have a diaphragm but have a series of airsacs. They act like a bellows to move air through the lung and are not a site of gaseous exchange. Within the airsacs gasflow is unidirectional from caudal to cranial, and continous rather than tidal. This leads to a greater efficiency of oxygen exchange than occurs in the mammalian lung. Air sacculitis is very commonly seen and can be identified subclinically on endoscopic examination or can be clinically significant with severe disease. This may be associated with pneumonia and upper respiratory tract disease. Air sacculitis can occur with *Chlamydophila psittaci*, *Mycoplasma spp* and *Aspergillus spp* infections. Other bacteria less commonly cause air sacculitis and pneumonia. Air sacculitis is also commonly seen in birds exposed to respiratory irritants. Radiology, culture, serology and faecal tests (*Chlamydophila* PCR) can be used to differentiate the cause. Rigid laparoscopy can be used to visualise and biopsy focal lesions as well as offering a means of direct treatment. Treatment also involves systemic antibiotic therapy in addition to nebulisation. Inection is often seen first in the caudal air sacs or on the ostia due to the pattern of gas flow through the respiratory system.

Aspergillus can lead to clinical signs of any of these conditions. It is a fungal disease that can affect any bird, with psittacines, raptors, penguins and waterfowl being particularly susceptible. *Aspergillus fumigatus* is the commonest isolate. These fungi are ubiquitous in the environment and can even be found in the lungs of healthy birds. Aspergillosis is non contagious, as infection is usually via inhalation of spores from the environment.

Damp litter (especially straw), dusty conditions, mouldy feed and poor ventilation predispose as they provide ideal conditions for fungal proliferation. Birds with a competent immune system normally do not develop disease but birds that are immunocompromised due to poor nutrition, transport, antibiotic therapy, age, captivity or other stressors are predisposed. Certain species of raptor that are not traditionally exposed to high levels are predisposed and these include golden eagles (*Aquila chrysaetos*), goshawks (*Accipiter gentilis*), gyrfalcons (*Falco rusticolus*) and snowy owls (*Nyctea scadiaca*) for example. Handling wild birds may be enough to precipitate disease with sea birds and swans being most commonly diagnosed with the disease. Some wildlife centres routinely screen seabirds for *Aspergillus* by endoscopy upon arrival. The disease can be acute and multisystemic but most often is chronic with primary respiratory involvement. Thus it is always worth checking for predisposing factors leading to the disease. In young parrots for example screening for PBFD is of paramount importance.

Clinical signs are referable to the location of lesions and associated pathology. The lower and or the upper respiratory tract are often involved.

The diagnosis of aspergillus rests on confirmation of fungal hyphae on cytology, histopathology or a successful culture. Cytology slides should be stained with lactophenol blue and reveal branched (at 45 degrees) septate hyphae 2.5 – 4.5 µm in diameter. Fruiting bodies can also be seen.

Many diagnoses are made clinically based on a typical endoscopic appearance (fungal colonies are typically grey white and in cases with fulminating disease fruiting bodies will be seen). Fulmininating disease is typically seen in those species not naturally exposed to aspergillus and immunocompromised birds. However radiography, blood haematology and biochemistry can be used to guide the severity of disease when endoscopic exsamination is not possible (you should therefore refer the case).

Heterophilic leucocytosis, often with a monocytosis and lymphopaenia with toxic changes to heterophils are often noted. A non-regenerative anemia due to chronic inflammation is often present as is hypoalbuminemia and hypergammaglobulinemia. This is not typical for aspergillus and can be seen with Chlamydophila or Mycobacterial infection for example. Serology is not useful in psittacines as weakly positive titres occur in clinically normal birds making interpreting the significance of a positive test difficult. In raptors aspergillus antibody titres have only a moderate predictive value in disease diagnosis but have been used to document response to treatment.

Radiography is often only useful where disease is advanced, and granulomas are evident. Air sacculitis may appear radiographically as thickend air sac walls. Asymmetry, hyperinflation or consolidation of the air sacs may be evident.

A nodular air sacculitis with focal air sac densities is often seen and occurs primarily in the abdominal and, less often, thoracic air sacs.

Treatment of aspergillus may be rewarding depending on the case. Syringeal or upper respiratory forms carry a better prognosis. Birds with fulminating disease will die. Birds with advanced granulomas in the coelomic cavity will also die but may survive treatment over a few months. Milder cases, particularly in psittacines, should be treated and can go on to lead acceptable lives. Raptors may have performance compromised.

Treatment options include:

Amphotericin B can be used either by direct application onto the syrinx or injection into a sinus or via an endoscope directly onto a lesion. Care has to be taken as it is irritant. Nebulisation is also performed in many cases but this is more likely to be with other agents. Intravenous thereapy is rarely performed as it is nephrotoxic. Resistant to this agent is quite high.

Itraconazole is commonly used as an oral therapy for birds, It takes three days to reach therapeutic levels and so other therapies are indicated alongside this. There are anecdotal reports of toxicity in grey parrots and the dose used should be halved or the drug avoided altogether. This is available as a liquid preparation. Newer agents such as voriconazole have less resistance but are generally cost prohibitive.

Terbinafine is used as an alternative in grey parrots and can be given alongside itraconazole. It can also be nebulised. Tablets require crushing and dilution.

F:10® disinfectant, this is an antifungal and antibacterial disinfectant used for nebulisation at 1:250 dilution for 4 - 12 weeks or the rest of the bird life.. Can also be used to reduce the number of spores in the environment and many keepers fog their buildings (and vets should fog our and consult rooms).

Chlamydophila psittaci is best known for affecting parrots and parrotlike birds but can affect other avian species (such as pigeons and waterfowl) as well as being passed to mammals and humans. It is known to lay people as chlamydiosis, ornithosis, psittacosis and parrot fever. It is shed in body secretions (faecal, nasal and ocular fluids). It is an obligate intracellular parasite with a biphasic developmental cycle. Elementary bodies are the infective stage and are shed in secretions and faeces while reticulate bodies reproduce by binary fission within the host’s cells. Chlamydophila organisms survive in the environment for several weeks and may be distributed by dust. The infection is mainly airborne; with the pathogen first reproducing in the respiratory tract.

Clinical signs seen include conjunctivitis (uni or bilateral), nasal discharge, ocular swelling, sneezing, depression, ruffled feathers, weight loss, inappetence, bright green diarrhoea, feather colouration changes, occasionally nervous signs, or even sudden death. Asymptomatic carriers are common, particularly cockatiels, budgerigars and pigeons. Pigeons and finches may get conjunctivitis alone. In cockatiels can be associated with *Mycoplasma* as well with a marked hyperplastic response (especially white or albinos). The severity of clinical signs depends on the virulence of the strain & immunocompetance of the bird and is a common secondary pathogen in cases of PBFD.

The diagnosis of psittacosis can be difficult. A heparin blood sample can be taken for antibody titre using in house immunocomb testing kits or sent to an external laboratory. A faecal PCR is considered the gold standard on pooled faeces taken over 3 – 5 days. However shedding may be intermittent and false negative occur. Equally a positive result may indicate the bird is infected but it may not be clinically ill with *Chlamydophila* at this stage. Birds treated with antibiotics (such as fluoroquinolones or tetracyclines) prior to collection with potentially have false negative results as the treatment will stop shedding within 48 hours. In house testing with a test kit based on the immunochromatographic principle to detect antigen is possible but the PCR is preferred.

Prognosis and severity of infection is important and blood work may demonstrate elevations in AST and there is often leucocytosis and monocytosis. Radiography may show signs of hepatosplenomegaly and air sacculitis. Endoscopic examination will allow a direct evaluation of these organs.

On post mortem examination impression smears of organs stained with machiavelos stain may reveal intracytoplasmic inclusion bodies. Culture- is considered the gold standard but is not very practical and requires very careful sample handling.

Treatment of birds generally involves doxycycline for 6 weeks either once a week by injection or orally. Fluoroquinolones also have some activity. Critically ill birds will require supportive care as well.

All birds require follow-up testing to confirm no reactivation of the disease. Even a bird that tests negative for *Chlamydophila* after treatment is not considered clear of infection, as a negative test can only indicate there is no organisms shed at the point of testing. To minimise this risk faecal samples should be collected after two weeks have passed since any drugs given have been excreted. In the case of injectable doxycycline this is three weeks later.

Once the infection is over many birds are susceptible to re-infection or re activation of the organism.

Inhaled toxicity or asthmatic episodes are also commonly seen, primarily in birds kept in the house. The most common cause of toxicity is Teflon from non stick frying pans which release toxins when heated. Other common toxins are found in aerosols, plug-in air fresheners and smoke. Asthmatic type responses are typically seen in some psittacine species such as Blue and Gold Macaws (*Ara ararauna*).

The symptoms seen with these cases are an acute dyspnoea and collapse. The client should take the bird outside into fresh air or to increase the ventilation in the room. Once acute signs have settled the bird should be brought to the surgery. Emergency treatment involves oxygenation and dexamethasone administered either via nebulisation (2mg in 10ml saline) or injection (2mg/kg). Once stable a standard diagnostic work up is indicated. Coelioscopic examination can reveal haemorrhage around the ostia and lung itself with some air sac inflammation. Lung biopsy can yield histopathological changes consistant with acute inflammation.

Skin diseases

Avian skin is very thin and inelastic compared to mammalian skin. It has a limited nerve and blood supply and tears very easily, but does not tend to bleed excessively when cut. However significant blood loss is possible in birds that self traumatise.

Feathers function not only to facilitate flight but also insulate, waterproof and protect the bird. Courtship, camouflage and display are other functions. Feathers are keratinized epidermis derived from follicles in the dermis. They are set in feather tracts known as pterylae. There are various types of feathers with different appearances and functions including contour, down, powder downs and filoplumes. The basic structure of a flight feather is a hollow shaft (rachis) and base (calamus). The feather vane is comprised of filaments called barbs which extend on either side of the rachis. The barbs are zippered together by rows of small hooks called barbules. Molting is the the replacement of old feathers with new and in many species (except ducks), is a continous process. Nutrition, temperature, photoperiod and reproduction can all influence molting. Growing feathers are called “blood feathers” and have a softer proximal shaft. It usually appears darker than a fully grown feather as it contains blood. Do not cut a blood feather as they will bleed. Cut, plucked or damaged feathers do not regrow until the feather shaft is lost in the molt Cauterizing a bleeding feather follicle can destroy the dermal papillae permanently preventing feather growth.

Skin diseases are also commonly presented and include feather plucking and self trauma. These can be due to primary skin disease or behavioural or underlying conditions. In many passerines allopreening is commonly encountered.

Psittacine beak and feather disease is an important viral infection of parrotlike birds. It is caused by a DNA circovirus that can remain stable in the environment for up to a year. There are a number of strains seen PBFD I is seen in many species but PBFD II is found in lorikeets. The virus is shed in feather dander and faeces and is transmitted by inhalation or ingestion. Vertical transmission is also possible. The virus favours rapidly dividing cells and the clinical signs are related to its effects on dividing tissues and the targeted systems are the epithelial cells, the gastrointestinal tract and the immune system. Birds that are exposed to the virus can develop a wide variety of clinical signs and have a varying incubation period. Latent infections are also possible.

In the chronic or classical form birds develop lesions between six months and three years of age. Rapidly growing feathers affected first (e.g. the powder down feathers). Feathers grow abnormally, are dysplastic with retained sheaths, haemorrhage can occur within the pulp cavity, feather shafts can fracture and eventually complete feather loss can occur. There can also be hyperkeratosis, overgrowth or fracture of the beak, oral ulceration. Some texts report the beak as being shiny or glossy as there is no powder down. Secondary infections are common as the disease is immunosuppressive. Typical examples include aspergillus and chlamydophila.

The acute form affects young birds during first feather formation and is characterised by depression +/- diarrhoea followed by the rapid development of dystrophic feathers. These birds can also develop anaemia and a leukopaenia.

The peracute form commonly seen in cockatoo and African grey parrot nestlings. This typically occurs by sixteen weeks of age and in many cases within a month of purchase of a young parrot. This form is characterised by severe leukopaenia (on a blood smear you do not see hardly any white blood cells), depression, regurgitation and death, before feather lesions develop. Occasionally fret marks on the feathers may be seen. Often the only sign is sudden death. Birds can also suffer paresis from internal secondary pathogens.

The strain affecting lorikeets appears to be less pathogenic and recovery is possible.

Budgerigars commonly carry PBFD and juveniles can exhibit normal feathering except for complete absence of primary and secondary flight feathers or become severely alopecic. Mild cases survive and can re grow feathers at the first moult (12 weeks old). The same signs occur with polyoma virus infection in budgerigars although PBFD is reported to be far more common. Polyoma is more likely to lead to nestling death with leucopaenia, anaemia and widespread haemorrhages.

The diagnosis is based on a PCR for the virus. This can be taken from pulpy feathers which can be gently plucked in a consultation (typically the keel feathers are used) or from a blood sample. It is present in the leucocytes and severely leucopaenic birds may have a false negative blood test. These tests are available commercially and some birds may have a negative test report sent to the new owners. Sadly the bird may have been exposed to birds which are PBFD positive after this test result. If you are suspicious but have a negative test then a bone marrow biopsy can be taken for PCR. If the bird has died then the bursa of fabricus in the cloaca or a skin biopsy may reveal the basophillc intracytoplasmic inclusions or lymphoid depletion typical of this infection. Any suspect carcases should be kept frozen as a PCR can be performed on frozen tissue. Positive birds that are healthy should be quarantined and re tested in 90 days to see if they have cleared the virus.

Treatment is supportive care including treatment for secondary pathogens such as aspergillus and chlamydophila. Many authors nebulise these birds anyway as a precaution. Some work using avian interferon has lead to an increased survival rate but the product is not available commercially. Mammalian interferon is of no use.

Control is through proper quarantine with PPE and fogging. Screen all birds prior to entry to a collection or if a pet bird once it is at home. Birds should be contained in separate air spaces to limit disease transmission.

Polyoma virus is caused by a DNA papovavirus. It affects all psittacines and passerines. Typically budgerigars are infected. The virus is shed in the faeces, feather dust and secretions. Transmission is via inhalation or ingestion. Vertical transmission is also possible.

Clinical signs in budgerigars include neonatal death (can have abdominal distension, subcutaneous haemorrhage and reduced down and contour feathers). If neonates survive greater than 15 days they will lose tail and flight feathers and are known as ‘French molters’. PBFD can cause similar signs and is more likely. If they survive the feather loss resolves after several months. Recovered birds are carriers and shed when stressed. In other psittacines sudden death occurs or death after depression, weight loss, diarrhoea, regurgitation, subcutaneous haemorrhage, dyspnoea and polyuria. A chronic form manifests as weight loss, intermittent anorexia, polyuria, recurrent infections, poor feather formation and neurological disease. It is unknown but suspected that persistent infections occur in large psittacines similar to the disease in budgerigars. Typically polyoma virus is more unusual in other psittacines and typically very young birds only. In passerines acute death in fledglings and adults can occur. Beak abnormalities and feather dystrophies may also be present. The psittacine PCR does not work in finches.

The diagnosis is made by PCR and to some extent clinically. On post mortem examination there is hepatic necrosis, bursal lymphoid depletion, membranous glomerulopathy, basophilic intranuclear and intracytoplasmic inclusion bodies in feather follicles and renal tissue.

Control is dependant of good quarantine with closed flocks. In budgerigars the recommendation is to stop breeding and clean out facilities for 3 – 6 months. In other psittacines identifiying shedders and removing them may be economically feasible. There is a vaccine in the USA.

Psittacines of low economic value should be housed in a separate air space away from more valuable tested (and hence negative) parrots.

Pox viruses can also lead to skin disease. These are the largest DNA viruses with 10 species and many variants reported in over 60 bird species (does not infect mammals). Raptors and pigeons are commonly affected. The virus is environmentally resistant and excreted in saliva, nasal, lacrimal secretions, and sometimes faeces. Transmission is via mosquitos (mechanical vector), direct contact, fomites, inhalation, ingestion, and wounds. Infection is common in lovebirds, canaries pigeons, new world parrots and raptors. Birds that recover do not carry the virus. There are three forms and the wet and dry forms can occur together:

The cutaneous form is called dry pox and causes nodular lesions around the eyes, beak and feet with self trauma making the lesions worse. The scabs fall off after 4 weeks.

The diptheritic form is called wet pox and causes lesions on mucous membranes of the oropharynx that affect feeding and breathing with URT signs.

The septicaemic form is common in canaries and sparrows. This is rapidly fatal causing pneumonia with or without diptheritic enteritis.

The diagnosis is by the clinical signs, history of exposure, biopsy/cytology (eosinophilic cytoplasmic inclusion bodies [Bollinger bodies] in epithelial cells are found). The dry form can be confused with ectoparasites and trauma and the wet form with trichomoniasis, candidasis, vitamin A deficiency, aspergillosis and pigeon and falcon herpesviruses. It is believed that ectoparasites are linked to disease leading to skin trauma and allowing entry of the virus.

Treatment involves nonspecific supportive care with antibiotics, nutritional support, and ocular lubricants. DO NOT remove scabs as this can exacerbate the lesions. They will fall off on their own after 2-8 weeks, A vaccine is available outside the UK and has been imported on occasion by veterinarians. Affected birds should not be vaccinated. In some species such as lovebirds the infection is generally self limiting.

Control includes elimination of ectoparasites and disinfection.

Pododermatitis (Bumblefoot) is also a commonly seen condition. It occurs in raptors, waterfowl and occasionally in psttacines. The main predisposing factors are inappropriate perching (too wide/narrow, incorrect surface), a lack of exercise and nutritional imbalances, particularly vitamin A deficiency.

Day old chicks fed to raptors can be high in cholesterol and low in calcium. Lesions often become infected with Staphylococcal sp. and other bacteria leading to abscesses and tracking infections within the tendons.

Treatment depends on the severity and duration of the condition. The owner should be warned that treatment could be prolonged (usually months). Husbandry needs to be improved. For raptors covering perches with Astroturf helps to alleviate pressure on specific areas. A cooled iron can be used to melt the tips if the Astroturf is too rigid. The bird should be kept at its flying weight and allowed exercise daily.

Feet should be radiographed to determine if there is any bone or tendon involvement.

For mild cases medical therapy may be sufficient and includes topical treatments such as preparation H which contains a number of moisturisers.

For more severe cases aggressive surgical therapy is required. Lesions should be flushed and debrided and then the feet should be dressed with either ball or doughnut bandages. Cultures should be taken when possible any systemic antibiotics provided for at least a two week period.

Feather plucking and self trauma are commonly seen in pet parrots and occasionally in Harris Hawks due to boredom. These cases require a complete clinical history to be undertaken followed by a thorough diagnostic work up to eliminate and underlying disease conditions that could lead to these clinical signs. There are a number of causes for poor quality feathers including poor nutrition, husbandry, viral infections such as PBFD or polyoma virus, hypothyroidism, systemic disease, bacterial or fungal folliculitis, liver disease, air sacculitis, coelomic masses, ectoparasites, reproductive disease and boredom or neurosis.

An accurate history of where the plucking or damage initially presented (for example over a painful area or around the vent, legs and rump which may suggest sexual frustration) and if the owner is witnessing it (attention seeking) or if it is independent of the owners presence. Feather plucking birds generally fall into two categories: Those that are ill and those plucking for psychological reasons.

Standard tests used to differentiate these include include examining the bird as a whole for general illness - blood work, radiography and endoscopic examinination. This is then followed by specific investigation of skin or feather disease - feather microscopy, skin biopsies, skin impression smear cytology and bacterial and fungal culture of feathers. Further diagnostic tests include faecal examination, PCR for chlamydophila, PBFD and polyoma based on the presenting signs and the results of the initial diagnostic sortie. If these tests are negative then a diagnosis of a behavioural problem can be made. Feather plucking can be difficult to resolve.

General advice can be given to all cases and should include

- Improve plane of nutrition necessary for feather regrowth (especially protein, calcium, energy).
- Allow exercise out of the cage, give branches to gnaw, increase the attention paid to the bird by a number of individuals.
- Provide a hide so bird has privacy. A nest box or hammock can be used but this may stimulate reproductive activity. A sheltered area with a perch (but no solid base) may be a better alternative.
- Encourage daily or frequent bathing. If the bird wants to cooperate, take it into the shower with you. There are perches available that use suction cups to attach to shower interiors. Many birds love to shower with the human. Otherwise offer the bird a bowl of water or mist the bird from a hand held sprayer. Many enjoy being sprayed from above (a hosepipe is great during the summer).
- Maintain a 12 hour light: 12 hour dark cycle. The bird requires sufficient time to sleep.
- Provide artificial UV-b lighting.
- Provide some background noise such as the radio or televison.
- Place the answering machine close to the bird then you can call and leave a message for the bird.
- Keep the number of toys low but rotate them frequently, changing the selection regularly. Also provide one off chewable toys. E.g. a whole orange, paper towel roll, ends torn off computer paper, leaves and cardboard boxes.

If there are severely traumatised feathers then these should be pulled out under anaesthesia using a pair of haemostats. This is painful and you should provide analgesia. In addition a number of feathers are due to grow in at the same time and this may stimulate an excessive preening response and analgesia at this time is also indicated. Re growth should be complete within eight weeks.

Many cases do have a reproductive or behavioural component, but underlying conditions that have been identified should be treated. Behavioural feather plucking is an exaggerated form of a parrot's normal preening behaviour. It may be initiated by a complex interaction of factors involving the household environment, the human inhabitants and the avian patient. Few generalizations can be made regarding treatment as parrots are individuals and behavioural modifications that may work for one case may prove detrimental for other cases.

Elizabethan collars should only be used as a last resort or in cases where significant self trauma is likely. Allow the bird to adapt to it in hospital before being discharged (usually 48 hours). This allows assessment of follicular activity if there is still doubt with a diagnosis of behavioural feather plucking often obtained by exclusion.

The collar is removed when normal feathers have regrown (about 2 months). DO NOT BE TEMPTED TO MAKE THE COLLAR A PERMANENT SOLUTION.

Generally behavioural modification and medical therapy are continued while the collar is in place. Psychotropic agents used in birds include anxiolytics (busipirone hydrochloride), tricyclic antidepressants (amitriptyline, clomipramine) and dopamine antagonist tranquillizers (haloperidol, doxepin). These drugs are used in cases of stereotypic behaviour and self mutilation, with haloperidol possibly being the most commonly prescribed. These drugs are mostly human formulations and are an off label usage. Their safety, efficacy and pharmacologic effects in avian species are poorly documented.

Other skin and feather diseases which can be seen independently of feather plucking include traumatic wounds. The skin of birds is easily torn and there is minimal underlying subcutaneous tissue for support. Areas can easily become devitalised. These should be cleaned and flushed as any other wound and sutured where possible or left to granulate.

Skin parasites are also seen such as *Cnemidocoptes pilae* typically in budgerigars. Hyperkeratosis, crusting and scaling on cere, beak and feet occur. The diagnosis is by cytology of crusting lesions. Treat with ivermectin or fipronil and supportive care.

Cloacal papillomatosis can be seen commonly in blue fronted amazons. Close examination of the vent is required. Endoscopic examination of the cloaca with saline insufflation is an ideal way to visualise these.

Herpesvirus can cause dry proliferative lesions of the feet in cockatoos.

Feather cysts can occur due to follicle damage or feather structure. There are breed predispositions in canaries and budgerigars. These arise due to failure of feather to exit follicle leading to cyst formation. Treatment is surgical excision.

Xanthomas are seen most commonly in budgerigars and cockatiels. These are orange yellow thickened dermal masses filled with cholesterol. They cause a chronic inflammation and will contain many lipid laden macrophages. They can be excised but the skin is very friable and often it may be best to leave alone and concentrate on dietary modification.

Neoplasia is also commonly seen most notable are lipomas in budgerigars. These are typically seen onbirds fed a high fat seed based diet. Other types seen include haemangioma, lymphosarcoma, squamous cell carcinoma, fibrosarcoma and liposarcoma.

Constricted Toe Syndrome- Constriction of the toe is common in juvenile amazon parrots and grey parrots with an unknown aetiology. Low humidity levels may be involved. An annular band of tissue causes digit swelling and eventually necrosis.

The band should be debrided and necrotic tissue removed. Closure using simple interrupted sutures placed to allow for any swelling is ideal. If closure is not possible the wound should be treated with hydroactive (e.g. orobase/duoderm) dressings and allowed to heal by second intention.

Diseases that affect the gastrointestinal tract

Sour-crop is an emergency in any species of bird. It is most commonly seen in raptors and juvenile parrots, though any bird can be affected. The crop is the first part of the digestive system and it is effectively a storage chamber that allows the rapid collection of food that can then be digested when the bird has moved to a safe roosting position. If the crop becomes impacted or does not process its contents to the next part of the digestive system then these contents will start to ferment. The toxins produced during fermentation are toxic and if left untreated, the bird will die. When the crop is full it is a prominent feature in the neck of the bird and as such, a crop stasis is usually recognised by the owner and treatment is sought quickly. Birds require fluid therapy and the crop needs to be emptied. This should be performed under anaesthesia. The bird should be intubated and any contents of the crop milked out. Haemostats can be used to empty any remaining contents. Flushing the crop with a crop tube and warmed saline is advisable at the end of liberate any small particles of food left.

Antibiotic cover should be provided with antiinflammatories if the bird has become septic. Gastrointestinal stimulating drugs can help speed recovery by encouraging crop turn over, although their use is based on personal preference. Metoclopramide 0.5mg/kg is given by injection. Feeding should initially be by proventricular feeds to ensure that they are digested immediately. This should initially be critcal care formula from vetark. Once faeces have been passed and the crop has turned over, hills ad diet can be offered once again by proventricular feeding. Once this has passed through the bird can be offered prey items without castings and subsequent to this whole prey can be offered. It wil take 36 – 48 hours for most birds to be back on their usual diet. Food should only be offered if the bird is keen for food.

Megabacteria (Avian Gastric Yeast) are large yeasts, *Macrorhabdus ornithogaster* that morphologically resemble large gram positive rods. They require microaerophilic conditions for growth. The disease is most often seen in canaries, finches budgerigars, lovebirds, cockatiels, small parrots and occasionally in larger psittacines. Megabacteria cause proventricular and ventricular disease in a variety of psittacine and other avian species. Clinical signs include wasting, lethargy and passage of undigested food. Radiography may show a dilated proventriculus with an hour-glass constriction between proventriculus and ventriculus. Necropsy lesions include proventricular ulceration and dilatation, thickening of the proventricular wall with mucus production, and softening of the koilin layer of the ventriculus. Diagnosis is by demonstration of organisms on gram stain or wet mounts. *Macrorhabdus ornithogaster* is a long, straight, narrow rod that is 3 to 4 µm wide and 20 to 80 µm long.

It will occasionally branch, but this is rare. They are gram-positive but sometimes will not stain evenly. It is responsive to treatment with amphotericin B. A crop wash or faecal sample can reveal organisms. This is a very common problem in wasting canaries and budgerigars. Oral amphotericin is unavailable in the UK in a suitable formulation and an SIC is required to obtain an oral formulation legally from Australia.

Candida albicans is a yeast that is a normal part of the avian gastrointestinal flora. Disease is seen with the indiscriminate or prolonged use of antibiotics or in young birds with poorly developed immune systems. Hand reared birds fed improperly prepared rearing formulas are also at risk. The crop is the main site of infection but the proventriculus and ventriculus can also be involved. Clinical signs include regurgitation and vomiting, a distended slow emptying crop, beak necrosis and white/cream plaques in the oral cavity. Rarely the infection will spread systemically. The diagnosis is by cytology or culture of lesions. Treatment is by the direct application of nystatin. This is only active in the gastrointestinal tract.

Trichomoniasis is caused by a flagellate protozoan spread via contaminated water or food (feeding raptors infected pigeons). White plaques form on the pharynx, oesophagous and proventriculus. Falcons, pigeons and budgerigars are commonly affected. Diagnosis is by wet mount of swabbed lesions from a live bird or within 30 minutes of death. A crop wash may also reveal the organisms. Treatment is with metronidazole or carnidazole orally. It is an emerging wildlife pathogen with collard doves, finches and predatory birds including owls increasingly infected.

Mycobacteriosis is a differential for many presenting signs, but is unusual. *M. avium* and *M. genavense* are the most common isolates in psittacines and both cause a chronic wasting disease. Infection starts in the alimentary tract and spreads to other organs, including bone and skin. The disease is marked by granuloma formation, but a diffuse form is common in canaries and finches. Falconiformes and waterfowl are also quite susceptible. Diagnosis can be difficult but acid fast stains of faecal material may identify an infected bird but intermittent shedding makes this method relatively insensitive. Acid fast staining of granulomas or liver biopsies can assist with diagnosis. DNA PCR testing of faecal samples has a sensitivity of 77.8%. Culture is the gold standard but takes weeks. Radiographs, haematology, endoscopic biopsy and cytology may assist in diagnosing. Intradermal skin testing, as used in the poultry industry is not effective. Treatment is not usually attempted due to the zoonotic implications and long treatment regimes (> year). Transmission of *M. avium* from birds to humans is considered a rare occurrence with most human infections originating from environmental sources. *M. bovis* and *M. tuberculosis* are occasionally diagnosed infecting birds with humans acting as the source of transmission.

Pachecos disease is caused by a herpesvirus that has a narrow and well adapted host range that typically is thought to include new world psittacines, especially conures. The latter can be persistently infected without developing clinical signs.

Acute mortality occurs when the virus infects a bird outside its normal host range (often old world parrots) or when a bird is immunocompromised. Respiratory and faecal oral transmission is common with fomites being particularly implicated in spreading the virus within collections. The virus causes rapid and extensive liver, kidney and splenic necrosis and can be presumptively diagnosed by identifying eosinophilic intranuclear inclusions in necrotic viscera. Identifying carriers is difficult as antibody titres are inconsistent so serology is not useful. Flock treatment with antivirals such as acyclovir will reduce transmission. A vaccine is available in the USA but is not protective against all strains and must be boostered annually. Vaccine reactions are characterized by granuloma formation have been reported so its use is controversial and only recommended for birds at high risk of exposure. Good quarantine is the best preventative measure.

Proventricular Dilatation Disease (PDD) has recently been linked to infection with a Bornavirus in macaws. PDD affects psittacines, finches and pigeons. Whether the causative agent is the same in all species is still to be investigated. Clinical signs include regurgitation, weight loss, altered GIT motility, passing whole seeds in the faeces, head shaking, seizures and ataxia. Secondary infections are also common and the disease course usually lasts weeks but infected birds may survive for years. The mode of transmission is unknown, but is assumed to be the oral faecal route. The pathogenesis is associated with a lymphoplasmacytic ganglioneuritis or the digestive tract and, less commonly, encephalomyelitis. Presumptive diagnosis is via the clinical signs and radiographic evidence of proventricular, ventricular and intestinal enlargement. Physical examination may reveal an enlarged coelomic cavity with a ‘doughy’ feel. Fluroscopy demonstrates a protracted transit time. Coelioscopic examination can be used to confirm proventricular (or intestinal loop) size.

Common species affected include, macaws, cockatoos and grey parrots. Amazon parrots are more resistant. Birds can demonstrate clinical signs from as young as 10 weeks to 17 years of age. It can take up to seven years for clinical signs to develop after exposure.

Confirmation is by biopsy or histopathology of the proventriculus or ventriculus demonstrating a lymphoplasmacytic ganglioneuritis, or with CNS involvement, an encephalomyelitis. However this is a risky procedure in the live patient and generally a crop biopsy is taken as a safer alternative. This should be centred on a blood vessel (we are aiming for the associated nerve), however as the gastrointestinal tract is segmentally affected (some birds may only have a section of the small intestine affected for example, then false negatives do occur. Up to 76% of cases can be detected by a crop biopsy.

Treatment is not curative but palliative and includes the use of antiemetics and celecoxib. Celecoxib is a Cox-2 NSAID, has been used to improve clinical signs by decreasing the inflammatory reaction around affected nerves. A dose of 10 mg/kg is given orally daily for 6 – 24 weeks. Improving the digestability of the diet can also help increase the longevity of patients.

Prevention is possible primarily by protracted quarantine. Some breeders will house disease free sentinel species in the quarantine. New arrivals will be introduced and held in quarantine for a variable period. Many keepers will breed with the stock here and once happy transfer them to the main collection. Ideally quarantine should last for 7 years. A detailed physical examination, radiography, coelioscopy and crop biopsy should also be considered with highly valuable collections.

Lead Poisoning is a common condition seen in waterfowl, some raptors and psittacines. Common sources include lead shot in raptor prey items, lead fishing weights, paint, putty, solder, and lead batteries. Small amounts of lead are absorbed from the GIT and stored in bones and soft tissue. It is excreted slowly over months by the kidneys. Clinical signs include depression, weakness, vomiting, polydipsia, polyuria and death. Diagnosis is by clinical signs, radiography and whole blood lead analysis. Levels > 0.2 ppm are suggestive and > 0.5 ppm diagnostic of lead toxicity. On post mortem examination of wild waterfowl with chronic toxicity, the vent may be stained green, the bird will be emaciated and in some instances the crop will be impacted. Treatment should be to maintain hydration status, provide supportive care (heat, oxygenation and a non stressful environment) and prevent convulsions (diazepam) if present. Chelation therapy with injectable CaEDTA or oral penicillamine until signs have resolved. Standard courses of CaEDTA are five days then stop for five days and repeat the course. The CaEDTA only chelates blood heavy metals. Lead in muscle does not cause toxicity. The lack of radiodense FB in the gastrointestinal tract does not rule out heavy metal toxicity.

Zinc Poisoning is occasionally seen and is associated with new wire cages that are coated with zinc powder. Always let cages weather for 1-2 weeks or wash them with dilute acetic acid before use. Clinical signs include lethargy, weight loss and in acute cases ataxia, recumbency, convulsions and diarrhoea. Treat the same as lead poisoning cases.

The liver is an important organ and functions include synthesis of proteins, clotting factors and bile acids. The avian liver is large and bilobed. Hepatic dysfunction occurs after severe injury or repeated insults. There is however considerable reserve and regenerative capacity possible. Focal lesions may therefore be present without obvious specific clinical signs. Chronic injury may lead to fibrosis and scarring.

Liver disease can occour due to a variety of diseases either secondary to systemic disease (such as *chlamydophila* or patcheco viral infections) or as a primary problem (such as hepatic lipidosis or haemosiderosis).

Non specific clinical signs seen include, anorexia, lethargy, weight loss, dysponea, diarrhoea, polyuria and polydipsia. More specific clinical signs seen include coelomic distension, coelomic ascites, coagulopathies, melena, abnormal beak, nail and feather growth. In severe cases suffering from hepatic encephalopathy neurological signs may be seen. The main bile pigment is biliverdin (green).

Thus if liver disease becomes severe the build up of bile pigments leads to a yellowing or green colouration to the urates and faeces usually combined with some polyuria. Some authors consider this to be pathgnomic for severe liver disease. It is possible that severe haemolysis could lead to similar signs due to overloading the system with haemoglobin from red cell lysis.

The diagnosis of liver disease rests on a number of procedures. Clinical presentation may be suggestive and lead to specific tests being performed. Blood biochemistry and haematology may demonstrate an inflammatory response or elevations in AST/CK or bile acids. Bile acids are elevated in 74% of cases of liver disease. 25% of these cases will have AST and CK withing reference values as AST is an acute enzyme and chornic disease is common. The remaining 20% are likely fibrotic livers.

Reference values for bile acids vary depending on species, but generally over 70 - 90 umol/L is considered diagnostic. Many birds with significant disease have elevations 3 – 4 times the reference range. SPE may also be useful in the diagnosis of hepatic disease.

Radiography can show hepatomegaly, microhepatica or coelomic ascites. There is a great variation in liver size between birds and in some cases doing a GI barium study can help outline the full extent of liver size.

Endoscopic examination will reveal the gross appearance of the liver and endoscopic biopsy is possible. It is best to wait until the radiographs have been assessed as liver puncture is a possible complication when a bird is suffering from hepatomegaly.

A ventral midline incision can be made for a surgical biopsy.

Chlamydophila is a common psittacine pathogen and ruling this out is wise. This can be performed either by collecting faeces over a three to five day period for a PCR or a blood antibody test. Fatty liver can be presumptively diagnosed based on clinical history and an endoscopic examination, although a liver biopsy is indicated for a diagnosis. Other liver diseases seen include, hepatic haematoma (due to hepatomegaly and trauma through the body wall), amyloidosis, iron storage disease, hepatotoxins, or other infectious agents such as mycobacteria, ascending bacterial infections or pachecos disease.

Hepatic lipidosis occurs when the rate of triglyceride accumulation exceeds the rate of degredation or their use in lipoproteins. It can occur as a consequence to a number of conditions such as excessive fat intake, increased mobilisation at times of increased demand (such as starvation) or endocrine problems. Hepatotoxins can impair metabolism and also lead to fatty liver. Deficiencies in particular amino acids (biotin, choline and methionine) can lead to fatty liver in humans. In juvenile birds it can be seen on high fat diets with birds grossly overweight for their age.

In older birds those on a high fat diet, obese birds, birds with high blood cholesterol and females with ovarian disorders. Lipaemia may interfere with blood biochemical analysis. This can still be present after starvation. Measurement of blood cholesterol is indicated and high and low density lipoproteins may help in the evaluation of fatty liver in the future.

Treatment of liver disease involves genralised hepatic supportive measures and specific treatments (or presumptive treatments) depending on the level of diagnostics.

General treatment of all liver disease may include: -

- Nutritional and fluid support. Many of these cases will be anorexic and dehydrated. Caution is to be advised in cases with severe hypoalbuminaemia.
- Anabolic steroids such as nandrolone at 5 mg/kg IM.
- Lactulose can be used to bind up any toxins and is used particularly for hepatic encephalopathy. It acts by reducing gastrointestinal pH and NH_3 migrates from the blood into the bowel and converted to NH_{4+} and expelled.
- If severe coelomic ascites is present then the fluid can be drained to ease respiration. However this is a significant loss of protein to the bird. Frusemide may also be helpful in reducing fluid retention.
- Clotting problems may also occur in severe disease. Normal clotting times are not available but the clinician should look out for excessive bruising (from venepuncture), melena and petechiae. In many cases surgical biopsy is undertaken without assessment of the birds clotting ability if there are no clinical signs present.
- H2 blockers may also be indicated including rantidine.
- Hepatoprotectants can be used and include milk thistle, S adenosylmethionine (SAMe) or vitamin E (AViX sunshine factor has high levels) which have antioxidant properties.

Specific treatment may include doxycycline or colchicines.

Reproductive Tract Disorders

Bird's have intracoelomic testes which vastly increase in size during the breeding season. Only the left ovary is functional in almost all species of birds. The ovary lies caudal to the left adrenal gland and at the cranial tip of the kidney. When quiescent it appears as a small grapelike cluster of follicles. Some owners may not be aware that a male is not necessary for a bird to lay non fertile eggs. Fertile eggs can be laid weeks after a male is removed as sperm is stored at the uterovaginal junction. Oviposition is controlled by vasotocin and prostaglandins.

Excessive egg laying is especially common in pet cockatiels. Excessive egg laying is caused by removal of eggs or stimulation of breeding behaviour by the presence of another bird, inanimate object (cage furniture, toy) or a person with whom the bird has bonded.

Do not remove laid eggs, encourage the hen to brood them. Long term, remove nest boxes and items that may stimulate laying, artificially reduce daylength to eight hours by covering the cage and reduce calorific intake (only if in optimal condition). Deslorelin implants can be used to inhibit ovulation. Injectible leuprolide acetate, a gonadotrophin releasing hormone has been used once every two to three weeks, for three to seven weeks, to reduce or completely inhibit egg laying. If unsuccessful salpingohysterectomy provides a permanent solution, but ovarian activity is likely to continue.

Egg binding is a common condition seen in cage birds. The aetiology is often multifactorial and complex and includes uterine inertia due to excessive laying, oversized or misshapen eggs, inappropriate nesting site, calcium/vitamin D 3 deficiencies, oversized or misshapen eggs, metritis and obesity. Clinical signs include depression, lethargy, tail bobbing, dyspnoea, anorexia, swollen abdomen, tenesmus and weakness. Some birds may be presented in a collapsed state on the bottom of the cage unable to stand or perch. Pressure on the ischiatic nerves to the legs can lead to paresis and paralysis of the legs. This may be unilateral. Persistant egg laying birds are frequently presented but any female can be presented and these are typically on a poor diet without UV exposure. Egg retention is more common in smaller birds such as cockatiels, lovebirds and budgerigars but can present in any species. Persistant egg layers sequentially deplete energy and calcium reserves leading to hypocalcaemia which can lead to fitting and death if untreated. Egg stasis is a critical emergency and develops quickly in 24 – 48 hours. A bird with a longer history than this will not have egg stasis.

The diagnosis is easy as the lower coelom may be swollen and an egg may be palpable or it is easily seen on radiography or ultrasound.

Stabilization can be performed by providing heat, humidity, parenteral 10% calcium borogluconate (100 mg/kg every six hours) and oxygen as necessary. Depending on the birds condition, radiography and bloodwork may be performed. In an ideal situation the birds ionised calcium level should be quantified prior to supplemental injectable calcium.

Oxytocin therapy can be tried but is not naturally produced by birds. If the egg has not been passed in a few hours, application of prostaglandin E2 gel (0.1ml/100g) or prostaglandin F2α has been used to increase uterine contractions and relax the uterovaginal sphincter if these are available. There is a risk of oviduct rupture with these treatments.

If unsuccessful the egg can be removed by physical manipulation. Sterile lubricant can be applied into the cloaca and the vagina dilated while applying gentle pressure to the body wall to encourage expulsion of the egg from the oviduct. If this fails ovocentesis with a large bore needle via the cloaca (if the egg can be seen), or through the coelomic wall may be successful. The collapsed egg will usually be passed over the next day or so. No pieces of shell should be left in the oviduct.

Flushing using an endoscope is useful to achieve this and provide visualisation. However it is possible the bird will subsequently pass the shell uneventfully, but there is a risk of oviductal damage.

These procedures are best performed under anaesthesia. These cases are an emergency. More invasive surgery may be required to extract the egg including laparotomy. Once the egg is removed, consider hormonal therapy such as deslorelin, luprorelin, cabergoline or chorionic gonadotrophin to suppress egg laying, supplemental calcium, UV-b lighting, photoperiod reduction, dietary change and behavioural modification.

Oviductal prolapse is also a potential complication of egg binding. If the tissue is viable, clean and replace using a lubricated cotton bud under anaesthesia. Try to ensure correct placement of the tissue. Apply a pure string suture to maintain once reduced. More severe cases will require a surgical approach.

Egg yolk peritonitis is due to ectopic ovulation but may also be seen with salpingitis, metritis, neoplasia, or a ruptured oviduct. These can be sterile or septic. Some birds respond to medical management while others will require removal of yolk material and extensive lavage. If chronic, coelomic enlargement, dyspnoea, tail bobbing, depression and lethargy may be seen. Chronic egg yolk peritonitis is the commonest fatal obstetrical condition seen in birds. The diagnosis can be made by aspiration and cytology of coelomic fluid or endoscopic examination.

Renal diseases of birds

Avian kidneys are split into three lobes and lie retroperitoneally in the fossae below the synsacrum. Spinal nerves run through kidney parenchyma and the ureters can be seen coursing across the surface of the kidneys. Given the location of the spinal nerves many renal conditions leading to swelling or nephritis can manifest as lameness by mechanically impinging on the spinal nerves.

The kidneys roles include water conservation, vitamin D production and toxin elimination. Birds can concentrate their urine but the ability is limited. In periods of dehydration one way of limiting water excretion is to limit glomerular filtration (by blood flow). Water resorption also occurs through the colon (and caecae) and reverse peristaltic waves facilitate this. The main nitrogenous waste compound is uric acid which requires minimal amounts of water to excrete. Uric acid is cleared by tubular secretion. This precipitates out on standing into urine and solid chalky white urates (usually > 60% of the waste is urates).

Birds are very prone to dehydration and in severe cases these changes can lead to the development of articular and/or visceral gout, where uric acid precipitates in the tissues.

Reduced blood flow to the renal tissue and increased solute concentration in the filtrate can also lead to renal pathology. High purine based protein diets can also predispose an animal to gout as blood uric acid can elevate four fold above reference rnages after a protein meal. This care is to be advised when dehydrated birds are presented to the clinic.

Decreased uric acid secretion as a result of reduced renal clearance can result from dehydration (reducing blood flow) or a number of pathologies (limiting elimination) and as a result uric acid levels build up above reference ranges. In order for this to occur damage to the renal tissue may already be severe.

Visceral gout results when uric acid is deposited on visceral organs. The pericardium, liver and spleen are commonly seen sites. This occurs as a white coating of the organs. Articular gout occurs when uric acid is deposited in the joints. Gross swellings occour in the feet at the metatarsophalangeal joints and these appear creamy white and are painful. Generally either visceral or articular gout occurs.

Articular gout is identified by aspirating the tissue (which is gritty) and examination microscopically in a dark field. The crystals are needle shaped. The murexide test (mix with nitric acid, flame dry, cool, ammonia added then goes mauve if urates present) can be used but is generally not required.

Many cases of renal disease are identified at a late stage and usually because there is elevated blood uric acid, articular gout is evident or severe renal pathology is identifed endoscopically.

Renal biopsy is an important tool to obtain a histological diagnosis but in many cases the underlying insult may have long since passed and the histological diagnosis is not usually typical for any specific underlying disease process or cause. Histological diagnoses include glomerulopathies, nephritis or nephrosis. Glomerulopathies are usually immune complex mediated and can be seen with polyomavirus infection. Nephritis can be due to bacteria and can represent an ascending infection, typically with gram negative aerobic bacteria. Biopsies can be submitted for culture in these cases. Parasitic diseases such as coccidiosis, microsporidiosis and cryptosporidiosis can also be diagnosed histologically. Nephrosis is a degenerative change in the kidney without any associated inflammation. This can be the end stage of many insults including toxins such as mycotoxins or lead. Nutritional diseases can also claed to renal pathology, such as vitamin D toxicity (leading to nephrocalcinosis), hypovitaminosis A (other signs occur earlier but metaplasia of the ducts can occur leading to ureteral obstruction and gout) or fatty liver and kidney disease in some species. Neoplasia or amyloidosis are other histological diagnoses.

A thorough history and physical examination may yield information useful in the diagnosis of renal disease. Howver it is most likely that this will only come to light after renal disease is suspected or has been definitively diagnosed. The clinical signs of renal disease are non specific and include generalised weakness, debility and dehydration. However if articular gout is identified then current or previous renal disease and pathology will have occurred.

Diagnostic tests performed should include a full blood count, particulary for signs of dehydration, anaemia and uric acid levels. A relative heterophilia is also possible. Birds with articular gout should have samples aspirated for cytology. Urinalysis is generally not performed in birds as it is mixed with cloacal and colonic contents. Radiography will be helpful if there is renomegaly or calcification of tissues, but is typically non diagnostic. Coelioscopic examination is indicated to obtain a direct visualisation of the kidneys and biopsy is indiciated, if pathology is suspected based on elevated uric acid, gout tophi in the joints or on the appearance of the organ.

Given the methods of diagnosis typically employed and the stage at which the diagnosis is made, treatment of many cases of renal disease follows a similar pattern.

Diuresis and fluid therapy is an important part of treatment. Providing fluid therapy is important initially and throughout any attempts to diurese the bird. Frusemide can be given at 1 mg/kg BID. However, providing copious amounts of parenteral and enteral fluids serves the same purpose. As a general rule of thumb providing twice maintenance levels is appropriate for a bird with suspected renal pathology. Antibacterial therapy may be indicated but is rarely provided routinely by this author, unless endoscopic examination demonstrates a requirement.

Allopurinol is a xanthine oxidase inhibitor and is used to reduce the production of uric acid by competing with xanthine and hypoxanthine. The result is elevations in these products which are excreted instead. Toxicity is possible and a dose of 10 mg/kg SID PO is appropriate. Toxic doses may be as low as 50 mg/kg. Once rehydrated the normal diet should be provided. Protein restriction may be indicated and pyramidine based sources should be sought. Surgically removing gout tophi and the administration of low dose NSAIDs is controversial.

Common behavioural problems of pet birds

Providing the correct environment to stimulate pet psittacines is important and the first step when dealing with birds with behavioural problems is to thoroughly evaluate the history. The second step is to improve the husbandry and instigate some basic training. The third stage is to thoroughly evaluate the bird to rule out other underlying disease or predisposing factors (such as reproductive activity). Then, finally, specific action may need to be taken depending on the particular vice being considered.

Psittacines can form inappropriate bonds with their owners and clinical signs seen may include reproductive activity, feather plucking and dominant behaviour to everyone apart from the birds ‘mate’. It is important to understand how birds communicate with each other and what the underlying meaning of a behaviour is.

Parrots communicate in many ways. Their group cohesion involves dominant and submissive signals, threats, and occasional squabbles. Parrots also communicate with affection, forming lasting bonds and alliances with other flock members. Parrot owners must learn to use these friendly interactions in order to become full-fledged flock members.

When two parrots meet, the first interactions are vocal; therefore talking to birds is important; it lets the bird know you are communicating with it and keeps the bird in touch when you are out of sight. Your voice also conveys pleasure and displeasure. Singing to a bird is a friendly form of communication. Some of the more noisy types such as Amazons and some cockatoos like noise and can enjoy music and loud singing. Some birds dislike loud noises, so be aware of the birds reaction to noise. Don’t throw your arms around when you talk to birds; this is often a dominant and frightening gesture.

Talking and repeating phrases you want the bird to learn will enhance communication. By imitating you, the bird is “speaking your language” and letting you know it wants to communicate. Be careful what you teach the bird to say; chances are it will repeat it forever and the last thing you need is for a bird to have picked up inappropriate phrases during its stay at the vets.

Mutual preening is one of the steps toward bonding. If your bird wants to groom owner’s hair and is gentle, it will be bonding with them. Birds like jewellery, however, and they will try to pick up neck chains, often pinching the skin at the same time, or they will bite rings. Owners should remove jewellery during these sessions, especially earrings or piercing jewellery.

In turn, they must play gently with the bird’s feathers, pulling a little as if cleaning them of dirt specks. This is something that birds find enjoyable and it tells them you are a friendly being. Birds that are moulting may be a little tender around new feather growth, but appreciate assistance in removing the quill sheath. This is performed once the feather is more than halfway grown out. Using your thumb and forefinger, gently roll the feather until the sheath breaks up. If the bird seems sensitive around a certain area, then don’t groom that area.

Cuddling is an interaction generally reserved by birds for their closest “friends”. Many birds will allow holding and cuddling, especially cockatoos, which may find even strangers worthy of a little cuddle. This however can be interpreted as quite an ‘intimate’ behaviour. Small birds such as budgies or lovebirds may like to snuggle into a shirt pocket or hide under long hair and once again this may be in part ‘nesting’ or ‘intimate’ behaviour.

Treats or food are generally used for social bonding between paired birds. Birds that are nesting will feed each other by regurgitation, and both parents will regurgitate to feed their young.

Treats given by hand will sometimes help break the ice between owner and bird. We would suggest that birds not be allowed to take food from an owner's mouth. Bonding should be on the "friendship" level, not on the pair bonding level.

Many times a bird bonds so completely to a human that it becomes sexually aroused. The bird sees its human companion as a mate and wishes to start a family, masturbating on the human, or becoming too "clingy". This behaviour is a problem, but mostly it is embarrassing, requiring that the bird be distracted from its intense effort to mate. Some birds, especially the males, may begin to "protect" the mate from other family members, biting both the mate and the intruders. In this case also, prevention is the key. Early socialisation to many family members will deflect the attention from one person to many. Later, if the bird does select one family member as its mate, other family members will still be able to handle and care for the bird.

In all cases, care must be taken not to sexually encourage the bird. Keep grooming to neutral areas and stay away from the legs, inside legs, lower stomach and lower back. If a bird begins to masturbate on the owner, put it back in the cage and walk away. Refrain from hand feeding, put all foods in a dish.

Sexual interest should be discouraged as soon as is evident. It is very hard for a bird to sexually bond with a human and then be shunned by that same person.

If a bird becomes so sexually aggressive that it is no longer an enjoyable companion, it may be time to consider obtaining it an appropriate mate. Feather plucking is a common sequel in a bird that is sexually frustrated and the diagnosis is made based on clinical history and coelioscopic examination. Behavioural modification and horrmone therapy are used to try to switch of your birds reproductive activity.

Other behavioural problems seen in birds include dominant behaviour and although birds do not necessarily want to be highest in the pecking order they will take the job if it is offered. A bird actually requires a stable position within its perceived family. To understand this, both dominant and submissive behaviours must be understood.

Dominant individuals send out signals that they are in charge. Birds that have assumed high ranking may:

- Try to look as large as possible and stand up on the perch.
- Seek the highest perch.

- Fail to come when called.
- Evade capture.
- Become aggressive when handled such as biting individuals in the family group.
- Actually attack owners by ‘rushing up to them’ or flying at their heads.
- Flapping their wings.

Many birds will have been dominat for some time and it is the extreme cases that typically present for ‘help’.

Once dominant behaviour is recognised in the bird, there are steps that can be taken to regain the alpha position in the “flock”. This applies to all family members as pair bonding to one individual can lead to dominant behaviour being displayed to all other members of the family but not the birds ‘mate’.

Dominant signals can easily be adapted for use by the owners to indicate ranking withing the group.

Dominant signals that the owner can use:

- Do not cower away and stand up when talking to the bird.
- Ensure the bird is always below head height even when the owner is sitting down.
- Show no hesitation or fear. Such as offering a firm, steady hand as a perch.
- Speak as though expecting to be answered, not pleading or cajoling.
- Teach basic commands and reinforce them frequently.
- Correct inappropriate responses, by withholding postitive experiences.

It is important for an owner to avoid displaying submissive behaviours as well.

A signal is submissive when:

- The bird’s eyes are above the handlers mouth.
- The handler averts his/her eyes.
- The handler holds his/her hands behind the back (folded wings).
- The tone of voice is soft and cajoling (baby talk).

Dealing with a dominant bird

Height, as mentioned, is one of the easiest methods used to establish a leadership attitude. Most birds will exhibit an immediate change when placed on a lower level. Certain positions are inherently submissive. Encouraging a bird to lie on its back in your hand is another submissive act.

Touch can be used to convey dominance. By gently handling the beak, wings and feet, areas often vigorously defended by some birds, dominance and trust can be developed. Commands should be used and rehearsed frequently to maintain the flock hierarchy. Each bird should follow the “up”, “down”, or “stay” commands, plus any others the bird has learned. Dominance must be communicated to the bird consistently by use of eye contact, tone of voice, and other posture or attitude means.

Corrections are used when the bird fails to follow commands, bites, or exhibits other unacceptable behaviours. For simple failure to respond to commands, I use several “step up” commands in a row: this is similar to getting 50 push-ups from a drill sergeant. You will have to determine how many to repeat before it becomes unpleasant for the bird. Following a command on cue (e.g. step up) should not be perceived as punishment; going back to a basic drill should be more like going back to kindergarten.

For bites, the “dump” or “wobble” can be used, or in California, the “earthquake”. This is achieved by wobbling, or shaking the arm, or hand either mildly or vigorously, when the bird reaches to bite or actually bites. Grabbing the beak may only anger the bird. Likewise, hitting the beak is discouraged. Using a combination of tone of voice, facial expression, and mild reprimand will usually get the point across. Most important, be consistent. If the bird is unmanageable and simply not cooperating, a time out may be in order. (Place the bird in a quiet place alone for about ten minutes). Then try again. Some people will also use a water jet as a form of distraction without a direct association with the owner. Birds can learn to stop the behaviour by simply being shown the water pistol. However both of these should be used with caution if at all as they are forms of negative reinforcement and withholding positive reinforcers is a better route.

In many cases isolation of the bird is a useful way to communicate your displeasure at behavioural traits. In many texts this is known asa ‘time out’. In many circumstances, if the bird is out of the cage for example and is being aggressive then catching the bird to place it in its cage and cover it is confrontational. A more appropriate response would be to ‘flounce’ out of the room. The owner should return after a short period of time when the bird has been quiet and reinstigate interaction. Always begin any session cheerfully, even those resumed after a deserved time out.

Displaying aggression to birds will not work, if the bird fears for its life, then its trust will be lost forever.

Some aggression that is directed to owners may actually be a fear response from a bird that has not been used to being handled and cornering such birds and forcing them to engage in social activities will not help. This is one of the reasons wing clipping can have a long term detrimental effect on birds.

New and strange experiences are always something to fear. Fear and panic are easy to recognise: recoiling in fear, panic screaming, or running away. If a bird hangs onto the back wall of the cage, flies or runs away if uncaged, flips on its back and fights with its feet, these are all signs of fear. Some birds show only mistrust by constantly watching people. A fearful bird may bite, but usually only if cornered. Birds prefer to flee. Wing clipping only increases the fear in a bird by limiting its ability to escape and is not advised.

Prevention is more effective than treatment. Keep a fearful bird near the family group or centre of traffic. Slowly expose the bird to the source of its fear until it becomes tolerant of the feared item or person.

Training exercises that owners can implement to reduce the risk of a dominant bird

- Step down.
- Hooding.
- Carrier.
- Towel.
- Wing touching and handling: Gently handle the wings and look at the feathers.
- Foot touching: Gently touch the toes. If the bird is standing on your hand, caress the toes with your thumb.
- Repeat and practice the handling exercises 15-20 minutes each day.

Screaming is another common complaint, both from bird owners themselves and from their neighbours. Companion birds that scream outdoors may lead to the local authority being called in due to noise pollution. This may mean the bird is forced to live inside or be removed. Equally if the noise becomes intolerable to the birds owners then it may need rehoming.

The normal vocalisation of parrots can be loud, but this is not a behavioural disorder. Attempting to curtail all screaming may be psychologically harmful to the bird, but then a normal bird does not scream all of the time. There is usually more social noise in the early morning and early evening, when birds normally gather in the trees to socialise. This can be a happy time for the bird. Playing loud music may make the bird happy (someone else is joining in) and make the owner happy (the music drowns the screaming). Screaming at other times of the day or night may be attributed to various causes, including loneliness or a strong pair bond; the bird may be calling for its human companion and in many cases the owner reinforces this by appearing.

Another reason could be a painful focus. A thorough physical examination is important to rule these out. Other causes are vermin (a mouse running through the room will frighten a bird and cause it to scream), and a pet snake is a real reason to scream. Sometimes the family dog or cat will set a bird screaming.

Screaming in alarm is a natural reaction of birds and is not a behaviour problem. One of the primary causes of problem screaming is that the bird has learned, if it screams, it will get attention.

Training the bird and the owner is required. If the bird is seeking attention, then the attention should be withheld. Cover the bird, turn out the light and shut the door, or just ignore the bird. If the screaming continues, then flounce out of the room in disgust. When the screaming stops, open up the room or uncover the bird, and talk to it very softly. You want it to know that its soft voice attracts you, not the loud one. This should be repeated as often as required. Usually 10 – 20 minutes 'time out' is sufficient, any longer and the association will be lost.

Keeping in touch with your bird when out of the room by calling its name, or by talking to it in a gentle singing voice, is also helpful. The bird may begin to call back in a pleasant voice, and the owner can answer it. This will teach the bird that if it wants the owner, there is a way it can call that they will respond to, and that screaming chases them away.

Neurological diseases

These are commolnly presented and a number have been covered already in passing. Signs seen may include fitting, paresis, paralysis or neurological or crainial nerve deficits.

Fitting can occur for a number of reasons some of which may be clearly evident from the clinical history such as trauma. But hypoglycaemia, hypocalcaemia, toxicity (inhaled or ingested), or viral infections are also prevelent. For many birds heavy metal toxicity, hypocalcaemia and hypoglycaemia are the main three differentials thus should therapy be required, prior to a diagnosis, then presumptive treatment for these could be given as part of the emergency stabilisation offered to a bird.

Given these differnentials then the following protocol is suitable for neurological cases but in fitting birds control of the immediate clinical signs may override the need for a diagnosis.

Birds that are seizuing should be placed in a warm, dark and quiet box with no perches or bars so they remain on the ground. In severe cases antiepileptics are indicated and diazepam can be administered at the rate of 0.5 mg/kg.

If possible intravenous access or intraosseous access is advised. For this brief anaesthesia is required but allows the clinican to obtain samples prior to treatment. A blood profile should be taken for both haematology and biochemisty. Radiography may also be useful to assess for radiodense foreign bodies or proventricular enlargement. Once IV or IO access is obtained then presumptive therapy can be given. This should include diazepam, if indicated, calcium gluconate at 100 – 200 mg/kg, glucose saline and calcium edatate if heavy metal toxicity is suspected on radiographic examination.

Hypoglycaemia is commonly seen in a malnourished bird or in certain raptor species (Goshawks for example) where hypoglycaemia has been seen to cause seizures. Typically these are stressy birds that have got a little low in body condition. Glucose saline can be provided along with Critical Care Formula® for hypoglycaemic patients.

Hypocalcaemia occurs in birds on a poor diet and not exposed to UV-b light. Many pet psittacines will develop a subclinical hypocalcaemia around 0.8 mmol/L or greater under these situations. However Grey parrots (*Psittacus e.erithicus*) are particularly prone and onced the ionised calcium drops in the region of 0.5 mmol/L then marked clinical signs can be seen which usually prompts the owner to seek veterinary attention. Early clinical signs include; weakness, falling off perches and 'star-gazing', if allowed to progress this condition will lead to fitting and if untreated death. Calcium gluconate 100 – 200 mg/kg every six hours, or ideally based on blood ionised calcium levels, can be given in hypocalcameic birds. UV-b light should be provided immediately and oral calcium and vitamin D supplementation when the bird is capable of taking oral medications and fluids. Ideally a blood ionised calcium should be taken prior to therapy to confirm the diagnosis and in severe cases give consideration to an anaesthetic to take a blood profile, place and IVor IO catheter and administer therapy via this route. Further diagnostics can be taken subsequently.

Fitting can be seen as a result of heavy metal toxicity. Lead poisoning is seen primarily in waterfowl, raptors and parrot species. Clinical signs include weight loss, difficulty digesting food, an 's' shaped kinked neck in swans, green droppings with faecal staining around the vent, blood in droppings, lethargy, fitting and death. Calcium EDTA can be diluted 1:1 with saline and given at a dose of 35mg/kg twice a day. This is potentially nephrotoxic and the bird should be well hydrated prior to use. It can also be added to an intravenous infusion. Alternatively or in addition penicillamine at 55mg/kg orally once a day can be used. Given the concentration of calcium Ecan be diluted 1the solution diluting down with saline is of paramount importance. Zinc poisoning is commonly seen in parrots. Often cages and toys are galvanised with zinc and over time as the bird uses its beak to move around the cage or playing with the toy, ingested metal will build up, clinical signs are as for lead poisoning. This diagnosis usually rests on three elements, clinical history of injestion, a radiodense foreign body or particles in the gastrointestinal tract or based on blood lead or zinc levels.

Viral Infection – Certain viral infections will cause seizures, Paramyxovirus (Newcastle's Disease) for example.

If the convulsions cannot be controlled then diazepam is a useful agent in birds. This can be given IM or IV at a dose of 0.5mg/kg.

Avian ophthalmology

Avian sight is well developed and is important in essential behaviours such as hunting, identification of ripe fruits, mating and flying. Despite massive species diversity the basic avian eye remains remarkably consistent and with the exception of several anatomical features peculiar to birds it differs little from the mammalian eye.

The visual capabilities of birds outstrips all others and their visual acuity is 2 - 8 times mammals. Their flicker fusion frequency is 160 frames per second compared to 60 in man. They have the ability to rapidly accommodate in order to deal with changes from air to aqueous media when diving for example. They have a high number of cones and no tapetum lucidum. In wild life casualty raptors 67% has intraocular lesions and only 15% were in the anterior segment. This is due to the high speeds at which raptors may collide. Detached or torn retinas are common injuries in wild owls and render the bird non releasable.

It is thus essential that we are able to identify ocular disease early and accurately to enable the best chance of maintaining this highly important sense. The orbits take up a large percentage of the skull (15% of the head in birds compared to 1% in man). A bird's eye movements are independent of each other and there is no consensual light reflex.

Scleral ossicles are a ring of overlapping bones around the eye that serve to facilitate accommodation and provide stability for the large globe (well developed in raptors). The extraocular muscles of birds are small and they see by rotating their head on a very flexible neck. Eye rotation is limited to 5 degrees.

The pecten is a black vascular structure that extends from the optic disk and nourishes the avascular retina. It can be a veil, cone or a vane pattern.

Examination from a distance particularly for the ability to fixate and track objects, head carriage, pupil size and equality, eye lid carriage and blink rate are all essential parts of the ophthalmic examination.

Examination of the avian eye is easy due to the large globe and more easily manipulable eye lids. However the small pupil size and the striated iris muscle makes full examination of the fundus more challenging than in other groups of animals. Iris muscles are striated so parasympatholytics such as atropine or tropicamide and sympathomimetics such as phenylephrine or adrenaline are ineffective in dilating the pupil. Keeping the bird in a darkened room and using minimal illumination may facilitate the examination and avoid the need for skeletal muscle blocking agents or anaesthesia. Air sac anaesthesia can also facilitate ocular examination. Vercuronium at 0.2mg/kg can be given intramuscularly in addition to anaesthesia. Intraocular injection of d-Tubocurarine has been suggested for those birds where post anaesthesia mydriasis is indicated.

Topical neuromuscular blocking agents have been used with varying species dependent success. The most readily available is Vecuronium bromide (Norcuron) which is available as a reconstitutable powder. Multiple applications of 4mg/ml Vecuronium at 5 minute intervals alone or in combination with phenylephrine provides dilation in some species at 40mins. The effect is very species dependant and systemic side-effects are of concern in small birds and with bilateral application (up to 25% of small Psitticine birds can show mild systemic side-effects).

The corneal reflex can be elicited using a damp cotton bud and used to protrude the nictitans and evaluate it for mobility and pathology. The third eyelid function is important to maintain due to poor lid mobility in many species. Tear production can be assessed using cut down shirmer tear test strips. Normal birds have values of 5mm in small birds and 8 mm per minute in larger birds.

The intraocular pressure of birds is typically 5 – 16 mm Hg with nocturnal species having significantly lower values.

Ocular ultrasound can also be performed in birds.

Congenital abnormalities are rare in birds. Blepharitis is common due to a variety of causes including pox, ammonia toxicity, Cnemidocoptes and hypovitaminosis A. Conjunctival disease is commonly associated with corneal disease and dust, smoke plug in air fresheners and ammonia can predispose. Pathogens involved in conjunctivitis include *Chlamydophila, Haemophilus* (cockatiels), *Mycoplasma* (finches particularly), *Pseudomonas, Staphylocouccs, Pasteurella, Citrobacter, E. coli, Klebsiella* and *Salmonella*. Other agents include Cryptosporidia, Mycobacterial, viral (herpes viruses) and fungal agents. A conjunctival biopsy can help to differentiate these. Traumatic keratitis is common and corneal ulceration is frequently found. Thus fluorescein should be routinely instilled during the examination. Superficial ulcers can be managed with ocular drops but deeper ulcers are a serious problem. Avian corneas are thin and birds are poor candidates for conjunctival flaps. Use of topical EDTA or serum from the bird can help to reduce collaginases. Temporary tarsoraphy or third eyelid flap possible but care re corneal ulceration due to sutures (do not place them all the way through) and the integrity of the marginal plica must be maintained.

Intraocular pathology is common. Hyphaemia, pectin injuries and retinal detachment common after trauma. Topical NSAIDs such as flurbiprofen required.

Anterior uveitis and hypopyon commonly seen. Can be due to systemic disease or perforation. Topical steroids used with caution and systemic NSAIDs.

Death and euthanasia of birds

As clinical techniques improve and drug availability increases, more avian patients survive procedures and there is an increased requirement for offering euthanasia of birds with disease that is too severe for treatment. Euthanasia may also be required in cases of behavioural problems or as part of health screening of a population of birds. Pet owners will usually want to be present for this procedure and euthanasia by intravenous pentobarbitone after induction with gaseous anaesthetic is the recommended technique. Intracoelomic injections are very unpredictable and often lead to considerable distress in the birds together with damaging tissues for any post mortem procedures. Intracardiac injections are reliable but practically difficult in all but the smallest psittacine bird. The heart rate should be checked after administering the pentobarbitone.

Post mortem examination

This is important to diagnose why the bird has died, but also to diagnose what significant diseases the bird may be carrying. This is of increased importance when considering flock management or zoonotic diseases. Dead birds should be kept in the fridge for 24 hours maximum prior to post mortem examination. In many cases euthanasia of a very sick bird is indicated to obtain fresh samples. You should always wear a mask and use a fume cupboard where possible. Feather samples should be taken prior to wetting the carcase with dilute chlorhexidine. The entire carcase should be examined and samples taken for analysis. Generally histopathology of multiple internal organs (everything) should be taken, but also include the vent (to check the bursa) and the central nervous system. Cultures, parasitology and cytology may be indicated for particular pathogens based on presentation and species. Take the opportunity to screen for common conditions. Freeze the carcase and await results. A frozen carcase can be used for virology, toxicology or PCR analysis.

Technique:

Many techniques have been described but the important point is to develop a consistent and systematic approach. Here is one that describes how to get to the “display stage” when tissue sampling can occur:

1. Place the cadaver in dorsal recumbency

2. With an inverted scalpel blade make a midline skin nick over the pectoral muscles and extend this in both directions from the vent to the base of the bill

3. Gently use your fingers or a scalpel handle to separate the very friable skin from underlying tissues. If the bird is dehydrated this may require blunt dissection.

4. With an inverted scalpel blade and lifting with forceps, make a shallow transverse incision just below the caudal edge of the pectoral muscles and sternum and extend this laterally.

5. Make a midline incision from the transverse incision caudally to the vent

6. Applying gentle pressure to the sternum lift the breast plate and cut the ribs, coracoid, clavicles and dissect attachments at the thoracic inlet to remove the sternum and ventral rib cage (this may require shears or rongeurs). This is the most delicate part of the dissection as accidental incision of the brachycephalic arteries in freshly dead birds leads to blood contamination of the lungs.

7. Expose the trachea, oesophagous and cervical structures and cut either side of the mandible to expose the oral cavity.

8. The bird is now at the “display stage” and tissue sampling can commence after thorough examination of the organs in situ.

Avian Drug Dosages and Therapeutic Regimes

Drug	**Dose**
Antibiotics	
Amoxycillin Clavulanate	125mg/kg PO BID
Doxycycline	25-50mg/kg IM q5-7d for 45d (for treatment of Chlamydophila infections), 200mg/15ml saline nebulisation
Enrofloxacin	5-15mg/kg SC, IM, PO BID, 10mg/ml saline nebulisation. (enrofloxacin is very alkaline and thus painful- avoid repeated IM injections & consider dilution with saline); Nebulised at 10mg per ml of saline
Antifungals	
Itraconazole	10mg/kg PO BID (African Greys: 2.5 mg/kg PO SID, deaths have been reported in this species)
Amphotericin B	1.5 mg/kg IV q 8 hrs for 7 days for aspergillosis in most species; 1 mg/kg IT (intratracheally) q 12 hrs for 12 days in raptors, psittacines with syringeal aspergillomas; 100 mg/kg q 12 hrs for 10-30 days by crop gavage to treat megabacteriosis in budgerigars; nebulisation 1mg per ml of saline for 15 min BID, TID for 5-7d, repeat every other week.
Nystatin	300,000 IU/kg PO q 12 hrs for 7-14 days. Must have direct contact with oral lesions to treat candidiasis

Terbinafine	10-15 mg/kg PO q 12-24 hrs for aspergillosis
F10 disinfectant	Dilute 1:250 with sterile saline to use for nebulization for 20 minutes every 8 hrs
Antiparasitics	
Fenbendazole	50 mg/kg PO SID for 3 d or 25-50 mg/kg PO once and repeat in 14 days. Toxicities reported in a number of species. Avoid using during molting as can cause feather abnormalities
Ivermectin	0.2 mg/kg PO, IM, SC, topically, repeat in 14 days
Praziquantel	5-10 mg/kg PO repeated in 14 days for treatment of cestodes. Toxic to finches.
Tolutrazil	7 mg/kg PO q 24 hrs for 3days (for treating coccidiosis)
Metronidazole	30mg/kg PO BID for 10 d (budgerigars 40 mg/kg)
Carnidazole	20-30 mg/kg PO once for treatment of *Trichomonas*; 30-50 mg/kg PO repeated in 14 days to treat *Giardia* in cockatiels
Analgesics	
Butorphanol	0.5- 4mg/kg IM every 4 hours (higher doses may cause sedation, recumbency)
Carprofen	1-2 mg/kg IM, IV, SC q 12-24 hrs (may go up to 4 mg/kg)
Ketoprofen	1-2mg/kg IM, SC 24-hrly
Meloxicam	0.1-0.2 mg/kg PO, IM, SC q 24 hrs
Psychotropic drugs	
amitriptyline	1-5 mg/kg PO q 12-24 hrs for a minimum of 30 days
Buspirone HCl	0.5 mg/kg PO q 12 hrs
Clomipramine	0.5-1.0 mg/kg PO q 12-24 hrs (start low and gradually increase the dose over 4-5 days)
Haloperidol	1 - 2 mg/kg IM every three weeks. 0.1-0.4 mg/kg PO q 24 hrs (great individual variation in effect- increase dose in small increments to achieve effect without excessive sedation)
Hormones	
Leuprolide acetate (depot preperation)	0.25-0.75 mg/kg IM monthly for at least three treatments to suppress egg laying.
Deslorelin implant 4.7mg	Placed intramuscularly based on clinical effect (10 months or so) into pectoral muscle.
Chelating Agents	
Calcium EDTA	30 mg/kg SC q 12 hrs for 5 days then off for 4 days then repeat as needed

Penicillamine	30-55 mg/kg PO q 12 hrs x 7-14 days
GIT Motility Modifiers	
cimetidine	5 mg/kg PO q 8-12 hrs
metoclopramide	0.5 mg/kg q 8-12 hrs
Miscellaneous Drugs	
Calcium Gluconate (10%)	50-100mg/kg IM, SC, IV once for acute hypocalcaemia (give slowly, especially if IV- dilute 1:1 with sterile water or saline for all routes. Can repeat as needed but at a lower dose IM, SC once stable- 5-10 mg/kg)
Lactulose	150-650 mg/kg PO q 8-12 hrs
Frusemide	0.15-2mg/kh SC, IM, PO q12-24hrs
Celecoxib	10 mg/kg PO q 24 hrs for 6-24 weeks. For treatment of clinical proventricular dilatation syndrome