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EMERGENCY AND CRITICAL CARE OF THE AVIAN PATIENT

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DISSERTAÇÃO DE MESTRADO EM MEDICINA VETERINÁRIA

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EMERGENCY AND CRITICAL CARE OF THE AVIAN PATIENT

Abstract

Increasing numbers of exotic animals are being kept as pets and owners want to receive the same high quality veterinary medical care as given to other animals. The field of emergency and critical care is rapidly developing so this dissertation focus on clinically relevant information, some new advances and their application to therapy.

In the first part of this work, the data gathered regarding all medical and surgical procedures performed during a four-month externship at Great Western Referrals Hospital, United Kingdom, under the scientific supervision of Dr. Neil Forbes is presented.

The second part is a description of the most common emergency presentations, effective diagnostic and therapeutic protocols, including the pathophysiology of shock and fluid therapy.

In the third part of this work, 12 clinical cases observed during the externship are presented. These cases were chosed due to their being representative of what the clinician may have to deal with in terms of avian emergency and critical care. For each case, clinical signs, diagnostic testing and treatment are described and discussed. The clinical presentation for each case is extremely diverse with inter- and intra- specific variations which is further complicated by the fact that most avian species mask signs of disease so owners are rarely aware of health problems that may occur.

One of the limitations of emergency avian procedures is the challenge to reach an adequate diagnosis and establish adequate treatment protocols for critical patients, for which time is crucial. Stabilization on initial presentation is more urgent than making a definitive diagnosis and supportive care can save more exotic animals than any other treatment.

Keywords: Avian, emergency, critical care, clinical cases, fluid therapy

EMERGÊNCIA E CUIDADOS INTENSIVOS EM AVES

Resumo

Cada vez mais animais exóticos são mantidos como animais de estimação e os seus donos desejam receber o mesmo nível de qualidade em termos de cuidados médico-veterinários que o prestado a outros animais. O ramo das emergências e cuidados intensivos está a desenvolver-se rapidamente sendo esta dissertação baseada em informação clinicamente relevante, avanços recentes na área e suas aplicações terapêuticas.

Na primeira parte deste trabalho é apresentada informação relativa a todos os procedimentos médicos e cirúrgicos realizados durante um estágio de quatro meses no hospital Great Western Referrals, Reino Unido, sob a supervisão científica do Dr. Neil Forbes.

A segunda parte contém uma descrição das apresentações clínicas de emergência mais comuns, protocolos de diagnóstico e terapia, incluindo patofisiologia do choque e fluidoterapia.

Na terceira parte deste trabalho são apresentados 12 casos clínicos observados durante o estágio. Estes casos foram escolhidos como sendo representativos do que o clínico poderá encontrar em termos de emergência e cuidados intensivos de aves.

Para cada caso, sinais clínicos, exames complementares e tratamento são descritos e discutidos. A apresentação clínica de cada caso é extremamente variada, com variações inter- e intra- específicas, sendo isto complicado pelo facto de que a maioria das espécies de aves escondem sinais de doença estando os seus donos raramente conscientes de problemas de saúde que possam ocorrer.

Uma das limitações dos procedimentos de emergência em aves é a dificuldade em estabelecer um diagnóstico adequado com o devido protocolo terapêutico em pacientes críticos para os quais o tempo é crucial. A estabilização inicial é mais urgente do que a elaboração de um diagnóstico definitivo e um tratamento de suporte adequado pode salvar mais animais exóticos do que qualquer outro tratamento.

Palavras-chave: Aves, emergência, cuidados intensivos, casos clínicos, fluidoterapia

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Abbreviations

2-Pam	2-pyridine aldoxime methyl chloride
ACE	Angiotensin converting enzyme
ACh	Acetylcholine
A&E	Avian and exotic
AGP	African grey parrot
APA	Air sac perfusion anaesthesia
ARF	Acute renal failure
AST	Aspartate aminotransferase
ATP	Adenosine tri-phosphate
BC	Biochemistry
BID	*Bis in die*
BMR	Basal metabolic rate
BP	Blood pressure
bpm	Beats per minute
BW	Body weight
CBC	Complete blood count
CK	Creatine kinase
CNS	Central nervous system
CO	Cardiac output
CPCR	Cardiopulmonary-cerebral resuscitation
CRT	Capillary refill time
CS	Clinical signs
CSF	Cerebral spinal fluid
CT	Computed tomography
ECG	Electrocardiogram
EDTA	Ethylenediamine tetraacetic acid
e.g.	*Exempli gratia*
EPH	Protein electrophoresis
ET	Endotracheal
et al.	*Et alia*
FB	Foreign body
g	Gram
G	Gauge
GA	General anaesthesia
GI	Gastro-intestinal
GWR	Great Western Referrals
h	Hour
HR	Heart rate
i.e.	*Id est*
IM	Intramuscular
IO	Intraosseous
IPPV	Intermittent positive pressure ventilation
IU	International units
IV	Intravenous
IZVG	International zoo vetgroup pathology
Kg	Kilogram
LRS	Lactated Ringer´s solution
MAC	Minimum anaesthetic concentration
MER	Maintenance energy requirement
min	Minute
mm	Millimeter
MM	Mucous membranes
MRI	Magnetic ressonance imaging
MRSA	Methicillin-resistant *Staphylococcus aureus*

nd	No date
No.	Number
NSAIDs	Nonsteroidal anti-inflammatory drugs
PBFD	Psittacine beak and feather disease
PCV	Packed cell volume
PDD	Proventricular dilatation disease
PDT	Photodynamic therapy
PDV	Pacheco´s disease virus
PE	Physical examination
PGE_2	Prostaglandin E_2
PGF2α	Prostaglandin factor 2 alpha
QID	*Quaque die*
RAAS	Renin-angiotensin-aldosterone system
RR	Respiratory rate
s	Second
SC	Subcutaneous
SCC	Squamous cell carcinoma
SCUD	Septicaemic cutaneous ulcerative disease
SID	*Semel in die*
SVR	Systemic vascular resistance
TID	*Ter in die*
TP	Total protein
UK	United Kingdom
US	Ultrasonography
WBC	White blood cells

Chapter 1. Externship report

The following report will describe the activities developed during a 4 month training externship in the field of exotic animal medicine and surgery as well as wildlife casualities. The externship took place at Great Western Referrals Hospital (GWR), Swindon, United Kingdom (UK), in the Avian and Exotic (A&E) department between the 20th of September 2010 and the 30th of January 2011 with a total duration of 800 hours, having Dr. Neil Forbes as my supervisor and Prof. Sandra Jesus as my coordinator.

During my externship I was enrolled in a training programme that covered nursing, hospitalisation, therapeutics, fluid and nutritional support of hospitalised patients. I performed procedures such as clinical pathology sample collection and testing, anaesthesia monitoring, diagnostic imaging and endoscopy. I also observed and assisted with consultations and surgical procedures. Not so frequently, I was involved in the small animal department for similar procedures with dogs and cats. I also accompanied Dr. Neil Forbes in some home visits to raptors and inspections to wildlife parks such as the Slimbridge Wetland Centre, Gloucestershire.

I had the chance of attending 6 CPD (Continuing Professional Development) courses on the subjects of "Update on rabbit medicine", "Veterinary Care of Backyard Poultry", "Avian cardiology", "Canine knees & hips", "The diagnosis & management of cognitive dysfunction" and "Wildlife casualities". I also participated in 2 practical trainning sessions performed by Dr. Neil Forbes to the veterinary staff of the A&E department on avian orthopaedic and soft tissue surgery and a day course designed for falconers on "Management of raptors for health and longevity" as well as some small animal meetings with specialists on dermatology, surgery and *diabetes mellitus*. I was also provided the oportunity to attend the 11th European Association of Avian Veterinarians (AAV) conference and 1st European College of Zoological Medicine (ECZM) meeting in Madrid, Spain, April 26-30, 2011.

GWR is recognized by the European College of Veterinary Surgeons (ECVS), European College of Avian Medicine and Surgery (ECAMS) and Royal College of Veterinary Surgeons (RCVS) as a veterinary training center for surgery and avian medicine.

GWR offers a small animal referral service in internal medicine and surgery and an avian and exotic service. GWR works mostly as a referral center so first opinion cases are not accepted in the small animal department (except for emergency cases). In the A&E department both referral and first opinion cases are accepted.

The hospital is open 24 hours a day, 7 days a week with an out of hours (OOH) service working with one veterinary nurse and one veterinary intern always available for emergencies and hospitalised patients.

The hospital building has 2 floors. The first floor comprises the small animal department, reception with a waiting room, 1 office, 4 consult rooms, an ultrasonography (US) room, a

pharmacy, a x-ray room and a small room for the x-ray developer. There are 3 hospitalisation wards for dogs, cats and infectious diseases, 2 surgical theatres (one for soft tissue surgery and the other for orthopaedic surgery), a laundry room, a sterilization room and a store house, known as Unit 14, for storage of medical and surgical material, 2 freezers and a fluoroscopy machine.

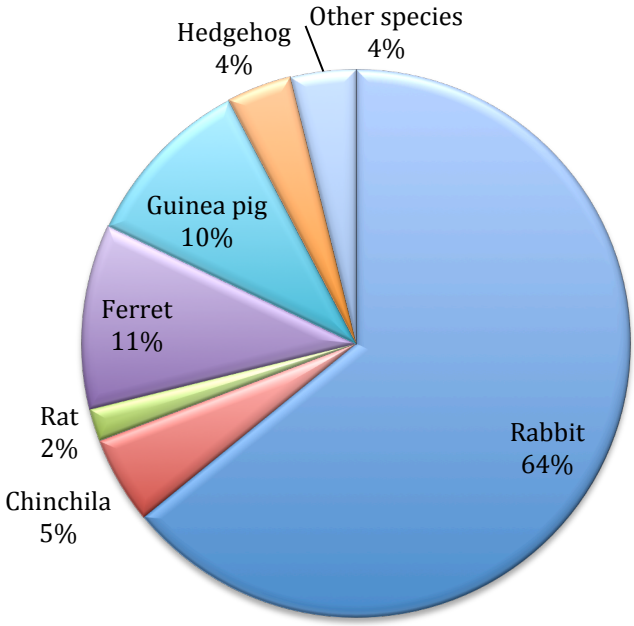
The second floor consists of the A&E department with a preparation room used for most procedures, one surgical theatre, a x-ray room and another room used for all the small mammal dental procedures or if the main room is occupied. There are also 3 hospitalisation wards, one isolation ward for birds where there are also reptile vivariums, another for psittacines and small mammals and other for raptors and ferrets. GWR has its own in-house laboratory also on the second floor where haematology, citology, parasitology, microbiology or biochemistry procedures are performed. There is also an autoclave room, staff room with kitchen, 2 bedrooms, an auditorium for conferences, 4 offices for the administrative, specialists, residents and common office for interns and externs with a veterinary library.

A dermatology specialist gives consultations in the hospital twice a week and a cardiology specialist gives consultations twice a month. Two times a week, there is a Magnetic Ressonance Imaging (MRI) mobile service operating at GWR.

The A&E veterinary staff consists of 1 specialist, 2 residents, 1 intern and 2 nurses.

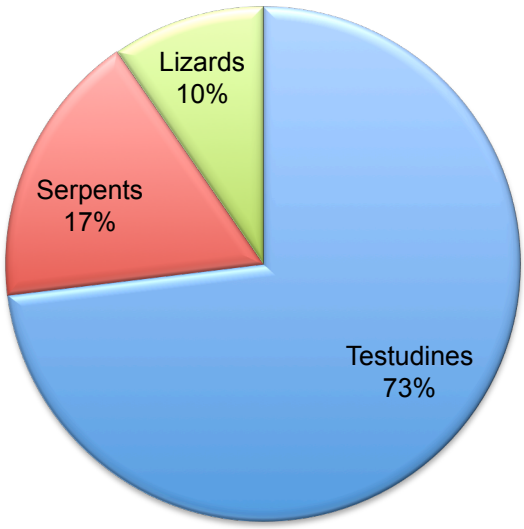
During my externship a total of 383 avian and exotic animals were observed including birds, mammals, reptiles, 3 Koi fish and 1 tarantula. A total of 156 mammals were seen, mostly rabbits (100) but also chinchilas (8), rats (3), ferrets (17), guinea pigs (16), hedgehogs (6) and other species including chipmunks (2), meerkats (2) and gerbils (2) (Graphic 1). Most common causes of mammal presentation included vaccination/health checks, dental disease, gut stasis, dermatology, referrals, ovariohysterectomy, castration and nail/hair trims.

Graphic 1: Mammal species seen at practice



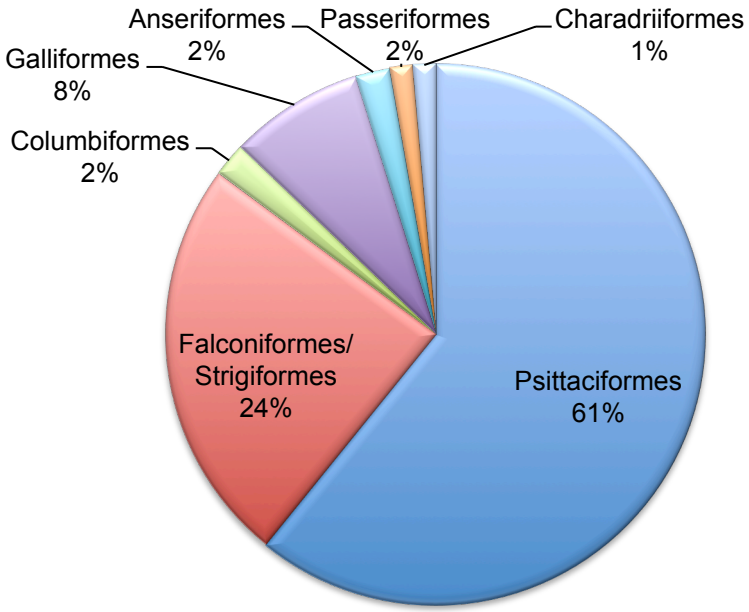
A total of 76 reptiles were seen including testudines (38), serpents (9) and lizards (29) (Graphic 2). Common causes of presentation included pre-hibernation checks, dewormings, nutritional problems or trauma.

Graphic 2: Reptiles seen at practice



A total of 148 birds were seen mostly Psittaciformes (90) and Falconiformes/Strigiformes (36) but also Anseriformes (3), Passeriformes (2), Galliformes (12), Columbiformes (3) and Charadriiformes (2) (Graphic 3). Most common causes of presentation included health checks, nail and beak trims, trauma, emergencies, gastrointestinal (GI) disease and referral cases.

Graphic 3: Avian orders seen at practice



Chapter 2. Literature review

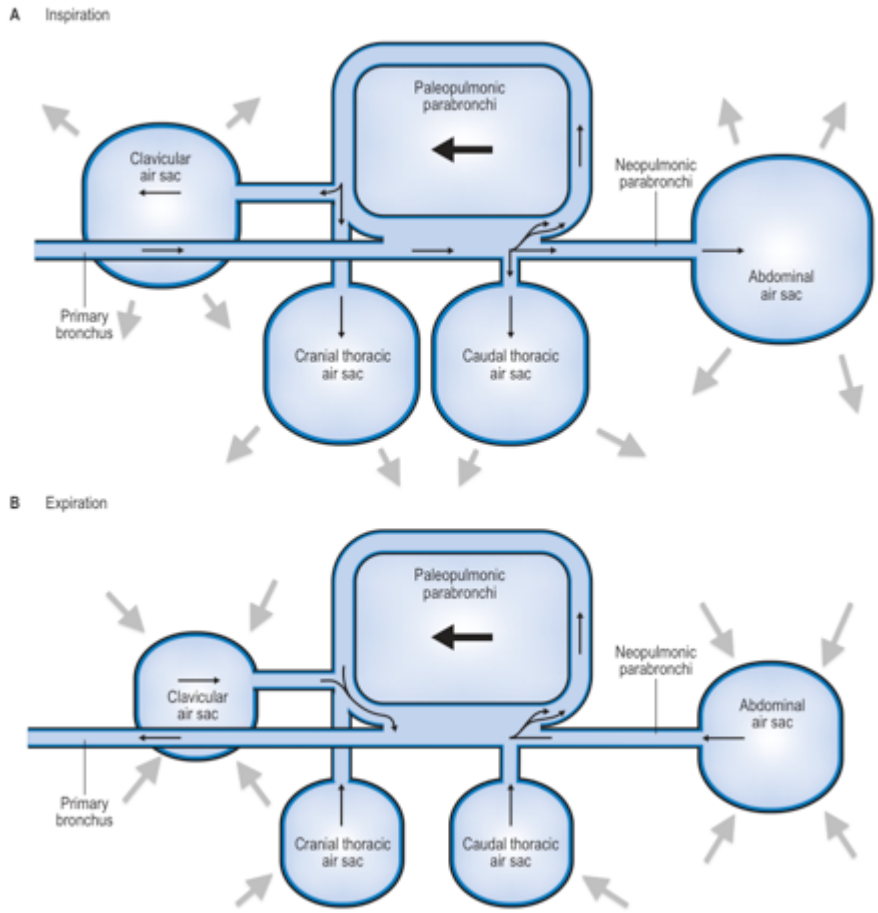
Increasing numbers of exotic animals are being kept as pets and owners expect to receive the same high quality medical care as given to dogs and cats (Lichtenberger, 2007a).
The veterinarian dealing with avian emergencies must be able to perform a fast and effective judgement of each case, knowing when it is appropriate to carry out a physical examination (PE) or when it is prudent to stabilise a critical patient first, and then be able to formulate a logical approach to diagnostics/treatments and anticipate potential complications (Harris, 2003).
A change of approach is necessary when treating exotic pets in general practice and value should be given to the bond between owner and pet, regardless of the species involved (Lichtenberger, 2007a).

2.1. Taxonomy, morphology and function

Among the *Animalia* kingdom, the largest class is *Aves* with many orders including Galliformes (e.g. turkey, chicken); Anseriformes (e.g. swan, duck); Psittaciformes (e.g. cockatiel, macaw); Strigiformes (e.g. owls); Columbiformes (e.g. dove, pigeon); Passeriformes (e.g. finch, canary) and others. There are over 9000 different species with a large variation in size (Sibley & Monroe, 1990).
Some particular considerations of morphology and function include a skeleton that reflects adaptations to flight with pneumatised bones reducing weight and trabeculae that maintain strength (Harcourt-Brown, 2005). Birds have a very high metabolic rate to permit a high level of activity and maintain a constant temperature (Hernandez-Divers, 2005). The average adult bird has a core body temperature of 38-42.5°C. Regulation of internal body temperature relies upon many factors, such as feather condition, adipose and muscle condition, hydration, food intake and respiration (Whittow, 1986 cited by Dorrestein, 2000).
The avian respiratory system is unique with the syrinx at the tracheal bifurcation involved in sound production and usually 9 air sacs: cervicocephalic (1-2), interclavicular (1), cranial thoracic (2), caudal thoracic (2) and abdominal (2) (Brown & Pilny, 2007). Air sacs are expansions of the bronchial wall, acting as bellows to the lungs and make up to 80% of the volume of the respiratory tract. Functions include cooling, reducing weight and aiding buoyancy in swimming birds, not being involved in gas exchange (Harcourt-Brown, 2005).
In the lungs, air flows through the paleopulmonic parabronchi**,** minimally anastomosing with uni-directional airflow and neopulmonic parabronchi, a meshwork of anastomosing parabronchi with bi-directional airflow. Airflow is crosscurrent to the blood flow in the blood capillaries, optimising oxygen (O_2) exchange. Air flows through the lung in the neoplumonic parabronchi to the caudal air sacs, from here to the paleopulmonic parabronchi (absorptive surface) and to the cranial air sacs, meaning that the air passes across the absorptive surface continually and unidirectionally (Fig. 1) (Macwhirter, 2000). The flow through air

capillaries within the lungs is a much more efficient gas exchange mechanism than the dead end alveoli found in mammalian lungs (Mitchell & Tully Jr., 2009).

Figure 1: Schematic representation of the air flow in the avian respiratory system. (A) Inspiration; (B) Expiration (Adapted from Longley, 2008).



Other particular features of avian morphology and function include: i) a heart proportionally larger and more efficient due to increased stroke volumes, decreased heart rate (HR) and increased cardiac output (CO) (Harcourt-Brown, 2005); ii) avian white blood cells (WBC) similar to mammalian WBC but avian neutrophil equivalents with heterophilic granules are termed heterophils (Fudge, 2000); iii) a gastro-intestinal (GI) system with a crop that carries out storage function, not involved in digestion (Macwhirter, 2000); iv) excretory system with trilobed retroperitoneal kidneys and ureters that feed directly into the urodeum of the cloaca, without a urinary bladder; v) nitrogenous waste excreted as non-toxic, semi-solid uric acid; vi) droppings passed as a mix of urine, urate and faeces (Harcourt-Brown, 2005); vii) blood flow through the kidney controlled by the renal portal valve and a complex system of shunts (King & McLelland, 1979 cited by Coles, 2007), and viii) a female reproductive system with (usually) a single functional left ovary and absent or poorly developed right ovary (Macwhirter, 2000).

Birds are oviparous and sexual dimorphism varies between groups. For sexing, the clinician can use rigid endoscopy under general anaesthesia (GA) for direct visualisation of the testes/ovary. DNA testing from feather pulp or blood is also used and since feather sampling is non-invasive, many laboratories offer testing directly to owners and breeders (Harcourt-Brown, 2005).

2.2. Emergency and critical care of the avian patient

2.2.1. Emergency stabilisation

2.2.1.1. Arrival of the avian patient and preliminary evaluation

When birds arrive to the veterinarian they are usually in 1 of 3 conditons: healthy, injured or ill. Injured birds need to be stabilised with supportive care before the injury can be successfully treated (Harris, 2003). It is part of a bird´s natural behaviour to evade predators by hiding illness, so owners frequently present them only when advanced clinical disease overcomes this evasion behaviour (Hernandez-Divers, 2005 and Matos & Morrisey, 2005).

Treatment and diagnosis must be performed in a step-wise fashion, with constant re-evaluation of the patient´s condition and ability to tolerate further procedures although a critically ill bird may not allow diagnostic tests to be performed because either the stress of sample collection or the time needed to process them is more than the patient can endure (Lightfoot, 2008).

Birds that display signs of illness for several days often compensate while those with acute serious clinical signs (CS) may be decompensated (Harris, 2003). History should include age, sex (if known), source of the bird, other pets in the household, diet (e.g. type, proportions, changes in food/water intake), exposure to trauma, toxins, droppings appearance, reproductive activity or other behavioural changes and environmental history (e.g. type of cage, ability to leave the cage unsupervised) (Echols, 2007).

After obtaining a brief history, the bird should be observed before handling it, if possible from a distance because the bird will often seem more alert and responsive when the clinician stands close giving an illusion of wellness. The cage should be examined for regurgitated material, blood/other discharges, faecal appearance and potential sources of toxins (Bowles, Lichtenberger & Lennox, 2007).

Before handling, general demeanor and overall appearance should be evaluated (e.g. fluffed feathers, unconscious, unable to perch, falling to one side, circling, wings hanging down, hanging onto perch/cage with the beak), respiration (Echols, 2007), droppings (i.e. if scant and green to black may suggest anorexia; many birds display stress-related polyuria; lime green urates, blood or no droppings) and any other gross abnormalities (Bowles et al., 2007).

Common CS of critical illness include tail bobbing, severe weakness, emaciation, changes of vocalisation and closed or slitty/"lemon" eyes (Echols, 2007).

The bird that brightens when approached may be stronger than one that continues to show significant signs of illness. After gathering all the information from history/observation the clinician can decide to: i) place the bird in a supportive intensive care chamber; ii) directly administer basic supportive treatment before proceeding, or iii) perform the physical examination with or without collecting diagnostic samples (Harris, 2003).

2.2.1.2. Physical examination

PE must be complete and thorough as in any other species. The crop should be palpated, and the choanal slit of the oropharynx checked for inflammatory debris, foreign material or blunted papillae. The beak is inspected for bleeding, asymmetry, altered keratin quality and fractures (Bowles et al., 2007). Perfusion status should be evaluated by determination of the capillary refill time (CRT) of the basilic vein and hydration by skin turgor. A CRT greater than 1-2s indicates more than 7% dehydration as normal veins are turgid and refill immediately (Echols, 2007). Hydration and temperature evaluation are major points on PE (Echols, 2007) and it can be presumed that a critical patient (due to illness, not trauma) is 10% dehydrated and probably hypothermic so dehydration and hypothermia should be treated before any further manipulations (Harris, 2003).

Auscultation is a challenge because of patient size, rigid lungs and unique respiratory system, but since respiratory sounds are minimal in birds, crackles, wheezes and clicks are usually abnormal. The coelomic space, between the ventral keel and pelvis, should be palpated. Overall body condition is evaluated by palpation of the pectoral muscle mass. The vent and cloaca are inspected for lesions (e.g. mass, swelling, prolapsed tissue) and the surrounding feathers should be clean (Bowles et al., 2007). Weight should be taken and the bird observed in an incubator after the PE to determine the impact of handling on overall condition. Sick birds should always be weighed in grams at least once daily, preferably at the same time of day, while hospitalised (Echols, 2007). In cases where PE is not recommended, a rapid examination is performed while transferring the bird from its cage to the incubator, palpating the pectoral muscle mass and coelomic space during transfer and observing the bird carefully after release into the incubator (Bowles et al., 2007).

2.2.1.3. Hospitalization

Heat, humidity and oxygen should be provided. Moist air appears to safely aid in correction of hypothermia by reducing evaporation from the extensive internal respiratory surface area, in addition, the oxygen drying tendency is nullified. The patient should be closely monitored while in the chamber in order to prevent overheating (Harris, 2003).

Some common avian infectious diseases include chlamydophilosis, Psittacine Beak and Feather Disease (PBFD), Proventricular Dilatation Disease (PDD), aspergillosis, tuberculosis or salmonellosis so multiple birds sharing the same airspace should be avoided to decrease

contamination across patients. Many zoonoses affect birds so adequate protective measures for staff should also be considered (Graham & Heatley, 2007).

2.2.1.4. Medical management

There is a distinction between a critically ill bird and a trauma patient because the first will invariably die without immediate medical attention, so the bird should be evaluated in order to know if injury management takes precedence over medical support (i.e. if haemorrhage is present it must be controlled, if an airway obstructed it must be cleared and simple injuries like lacerations or fractures should be managed only after the patient is assessed and stabilised) (Harris, 2003).

Before handling the bird, a plan should be created. Because the patient needing most support is the one least capable of tolerating stress a "conservative progression" approach is the best way to deal with it, one procedure at a time, prioritizing needs, then waiting for the clinical effect before proceeding. General techniques for supportive care can be employed on almost any critical patient without delay providing stabilisation (e.g. thermal support, fluid therapy, catheterisation, blood transfusion, nutritional support, pain relief) (Hess, 2002; Wilson, 2005).

2.2.2. Emergency procedures

2.2.2.1. Oxygen administration

Indications for O_2 therapy include respiratory/cardiac disease, shock, post-surgical recovery and stress. Critical patients may have a diminished CO and O_2 maximizes cardio-respiratory efficiency. For any dyspnoeic bird, O_2 is recommended and hyper-oxygenating the patient prior to handling may also decrease its risk. Some patient's don´t tolerate masks and if an oxygen cage is not available, most incubators, cages and aquariums can be modified so that supplemental O_2 can be provided into them. For most critical patients kept in a closed clear chamber, a 40-50% O_2 saturation level is recommended (Echols, 2007). Pure O_2 is acceptable for short-term use given via facemask or in an enclosed environment. Prolonged exposure to high O_2 levels was shown to be toxic in budgerigars (Wilson, 2005).

Low dose sedation may reduce some of the stress associated with upper airway obstruction (Graham, 2004). Restraint may increase the respiratory rate (RR) 1.5-2 times the resting rate, so it should be avoided, if possible, for dyspnoeic patients (Wilson, 2005).

2.2.2.2. Nebulization

Nebulizers convert liquids to aerosol particles appropriately sized for inhalation. They consist of a disposable/reusable nebulizer and a pressurized gas source and can be used to deliver moisture or topical medications to the mucosa of the respiratory system (Bowles et al., 2007) reducing the stress of handling (Harcourt-Brown, 2005).

The systemic drug dose is usually diluted in 5-10ml saline and delivered over a single 15-30 minute session. The most frequently used antibiotics for nebulization are aminoglycosides. Parenteral bronchodilators such as terbutaline have been empirically used in birds with lower airway disease via nebulization. Initially terbutaline (0.01mg/Kg) is given IM or SC while the bird is placed into an oxygen-enriched environment and then is nebulized (0.01mg/Kg diluted into 5-10ml of saline, TID) (Bowles et al., 2007).

2.2.2.3. Injection sites and sampling

Subcutaneous (SC), intramuscular (IM), intravenous (IV) or intraosseous (IO) routes can be used for injection and sampling of avian patients. SC sites include the featherless inguinal and/or axillary skin fold, taking care to aspirate prior to injection to confirm the needle has not penetrated the air sacs (Tully Jr. & Beaufrère, 2011). For IM administration the pectoral muscles are used while for IV administration, the right jugular vein, basilic vein or dorsal metatarsal vein in larger birds are the choice (Harcourt-Brown, 2005). For IO route the distal ulna or proximal tibiotarsus are the options (Tully Jr. & Beaufrère, 2011).

In birds, blood volume is around 10% bodyweight (BW) (Harcourt-Brown, 2005) and it will be safe to collect up to 1% of the BW from passerines and psittacines (Ritchie, Harrison & Harrison, 1994). In compromised birds, this should be reduced to 0.5% total BW. Samples for haematology and biochemistry (BC) are obtained before treatment for best diagnostic value, however, the patient´s needs must be prioritized (Graham & Heatley, 2007).

2.2.2.4. Intravenous catheterization

IV catheters can be placed in the right jugular, basilic or metatarsal veins. The jugular vein can be used in birds as small as 75g but it can be difficult to maintain the catheter in place (Bowles et al., 2007). There is no need for plucking at this site because there is an area of apterylae over it. A 20-22G catheter of sufficient length to reach the thoracic inlet can be used. The basilic vein (over the medial ulna) is easy to visualize and catheterise. Once the catheter is secured, the wing can be wrapped in a figure 8 bandage (Hess, 2002). The dorsal metatarsal vein (along the dorso-medial aspect of the tarsometatarsus) allows easy visualization of the catheter and the thick skin of the feet helps holding the catheter in place. This vein can be used in birds over 300g (Bowles et al., 2007).

2.2.2.5. Intraosseous catheterization

In many cases (small or hypovolaemic birds) IO catheterization may be preferable to IV catheterization, as the latter is often more technically difficult in terms of placement/maintenance and has a higher risk of significant blood loss if removed by the bird (Bowles et al., 2007). IO catheters can be placed in the distal ulna (larger birds) or proximal ulna, proximal tibiotarsus and lateral femur (young/smaller birds) (Tully Jr. & Beaufrère, 2011). Short spinal needles, 20-22G, can be used for larger birds and 22-25G for smaller

birds (Bowles et al., 2007). IO catheters require more maintenance than IV catheters and should be flushed daily to maintain patency (Tully Jr. & Beaufrère, 2011).

Birds with IO/IV catheters should be monitored daily for complications such as a cardiac murmur, local pain, swelling or heat that might suggest infection (Echols, 2007) and analgesia should be provided (Lichtenberger & Ko, 2007). All catheters should be removed after 48-72h even if no complications occur and a new catheter placed in a different site if needed (Echols, 2007).

2.2.3. Emergency avian surgery

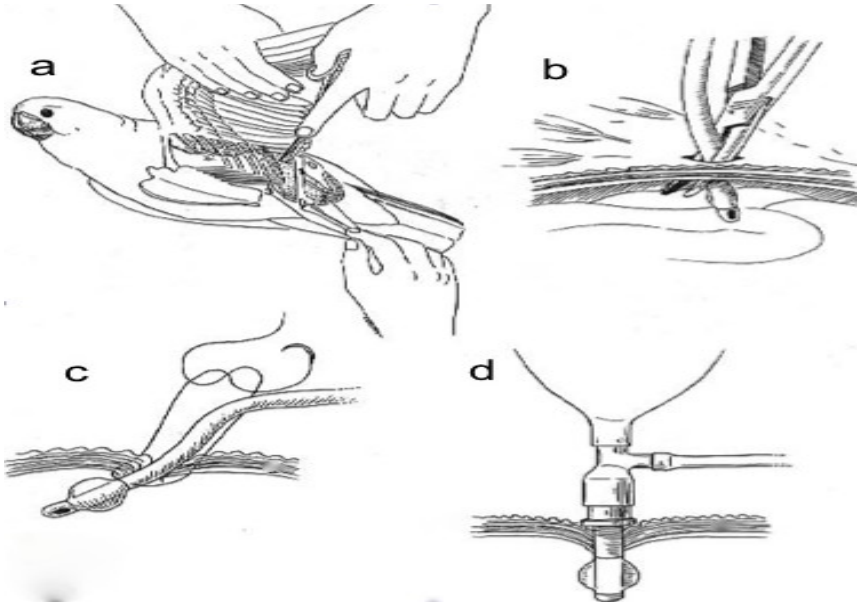
The actual procedures are too numerous to list here, however, the basics of emergency avian surgery techniques are discussed in this section. Microsurgery and magnification instruments are often required due to small patient size (N. A. Forbes, personal communication, December 21, 2010).

2.2.3.1. Air sac tube placement

The unique respiratory system of birds allows direct delivery of oxygen/anaesthetics through the air sacs, bypassing the trachea (Chavez & Echols, 2007). Placement of an air sac tube is an emergency technique that can be used if the oral cavity or trachea are occluded, causing acute respiratory distress (Brown & Pilny, 2007). Anaesthesia via air sac breathing tubes has been successfully used in patients as small as zebra finches (Nilson, Teramitsu & White, 2005). Sterile different sized air sac tubes should be available at all times. Commercial avian air sac cannulas exist but endotracheal (ET) tubes can be shortened for this purpose. Material should be rigid enough not to collapse but flexible enough to avoid damage of internal organs. For very small birds, 20 or 22G IV catheters can be used temporarily (Graham & Heatley, 2007). It is advised to use a tube 25% larger than the patient´s trachea, inserted to ⅓ of the width of the coelom at the point of insertion. The tube should have a length to suit patient size and multiple holes in the side as well as the end, with pre-placed sutures at the level of the coelomic wall (N. A. Forbes, personal communication, December 21, 2010). It is always recommended to radiograph patients prior to placement as presence of organomegaly or ascites can lead to complications (Graham & Heatley, 2007).

Air sac tubes can be left in place for up to 7 days, however they should be ideally removed within 4 days because of the risk of air sacculitis associated with prolonged tube placement (Brown & Pilny, 2007). Complications include coelomic organ damage, life threatening blood loss, air sac damage and SC emphysema (malpositioning, excessive perfusion volume, obstruction) (Korbel, 1999).

Figure 2: Placement of an air sac tube. Making a skin incision in the area of the sternal notch (a) then passing a pair of haemostats through the abdominal muscle (b) and suturing an ET tube to the body wall (c,d) (Adapted from Ritchie et al., 1994).



2.2.3.2. Oesophagostomy tube placement

Oesophageal tubes are used to bypass the upper GI tract, allow enteral medication and/or provide food to the crop/proventriculus of anorexic patients (Huynh & Forbes, 2011). The tube is measured from the midcervix (midneck) to the distal edge of the keel and cut on the proximal end to the measured point. The tube is then sutured into place, both at the cervical insertion site and on the skin of the dorsum. Liquid food can be given by a syringe, followed by flushing of the tube to maintain patency and hygiene, with minimal restraint (Chavez & Echols, 2007). The tube can remain in place for several days until the bird is able to feed sufficiently to maintain its weight without assistance. The sutures can be removed, the tube retracted and the wound left to heal by secondary intention (Echols, 2007). Complications include penetration of the oesophageal wall opposite to the incision site and oesophageal strictures (Ritchie et al., 1994).

2.2.3.3. Proventriculectomy

Proventriculectomy is used to access the proventriculus and ventriculus to remove foreign bodies (FB) or for surgical biopsy. The ventriculus is a muscular organ with a white tendinous lateral aspect (N. A. Forbes, personal communication, December 21, 2010).

Since birds have no mesentery, an enterotomy has a higher risk of postoperative peritonitis so the liver can provide some of the protective function of the absent mesentery (N. A. Forbes, personal communication, December 21, 2010). Exteriorized organs should be kept moist with warm saline but care should be taken to minimise fluid use as the air sacs are exposed (Chavez & Echols, 2007). Complications include contamination of the abdominal cavity with gastric contents so meticulous attention to proper closure is vital to prevent

leakage. A small, atraumatic needle should be used with a continuous suture pattern to provide the best seal. If the proventricular wall appears thin and friable, the potential for postoperative incisional leakage may warrant placement of a duodenal feeding tube that will allow enteral alimentation of the patient while bypassing the gastric incision (Ritchie et al., 1994).

2.2.3.4. Ingluviotomy

Ingluviotomy is indicated for retrieval of crop, proventricular or ventricular FB, placement of an ingluviotomy/proventriculotomy tube or rapidly emptying the crop (e.g. sour crop, ingluviolith) (N. A. Forbes, personal communication, December 21, 2010). Because of the ability of the ingluvies to stretch, the incision should be made only about half the size necessary to accomplish the procedure, however, having adequate exposure is more important than having a small incision, and retrieval of large FB through small ingluviotomy incisions should not be attempted to avoid serious tissue damage. Following the procedure, closure is done with 2 layers of continuous inversion sutures since this is less compromising on the size of the crop lumen. The crop may be inflated with saline or air to check for leakage prior to skin closure and avoid complications such as contamination of the abdominal cavity (Ritchie et al., 1994).

2.2.3.5. Tracheotomy and trachectomy

Tracheotomy is commonly indicated for treatment of syringeal aspergillomas or retrieval of a tracheal FB (N. A. Forbes, personal communication, December 21, 2010). Air sac tube placement for anaesthesia is recommended for both procedures (Harrison & Lightfoot, 2006). Trachectomy is used in cases of severe tracheal stenosis due to trauma, infection or stricture, when tracheal resection with removal of the affected tissue is performed (N. A. Forbes, personal communication, December 21, 2010). Not all species can tolerate a shortening of the tracheal length (Harrison & Lightfoot, 2006) and unless absolutely mandatory, the trachea should not be completely transected in order to maintain its alignment, reduce tension on the closure and prevent complete disruption of the blood supply (Ritchie et al., 1994). Postoperative care including analgesia, is best achieved with carprofen (4mg/Kg, IM), which may be augmented with butorphanol, although meloxicam (0.2-0.4mg/Kg) may be safely used. Fluid therapy, warmth, nutritional supplementation and good nursing are as important as the surgical skills (N. A. Forbes, personal communication, December 21, 2010).

Complications for both procedures include iatrogenic trauma during manipulation and stricture formation. Stay sutures placed around the tracheal rings adjacent to the incision site allow atraumatic manipulation of the trachea. When closing the incision, knots should be

placed external to the tracheal lumen because intraluminal granuloma formation at the suture site is common. This is a difficult procedure that should be only used as a life-saving technique, when all other methods have failed (Ritchie et al., 1994).

2.2.4. Diagnostic testing

Diagnostic testing techniques for avian emergency and critical care cases should include blood work, cytology, histology, microbiology, urinalysis, evaluation of faeces and crop specimens, nasal flush and endoscopy. Routine haematology and chemistry analysis should include a complete blood count (CBC) and at least aspartate aminotransferase (AST), creatine kinase (CK), glucose, uric acid, bile acids, serum calcium (Ca), phosphorus (P), albumin and electrolyte measurements. Packed cell volume (PCV) evaluates the presence of anaemia. A WBC count and blood smear should be performed. Other measurements include blood lead (Pb) level, plasma zinc (Zn) level and protein electrophoresis (EPH) (Bowles et al., 2007). Lichtenberger (2005) recommends for a critically ill bird, to collect no more than 0.4 to 0.5ml/100g BW. Imaging techniques include radiography, US, MRI and computed tomography (CT) scan. Fluoroscopy is used for GI motility studies or detection of metal FB without GA (Lichtenberger, 2005).

2.2.5. Frequently seen emergencies

Common emergency presentations of pet birds will be discussed on this topic. Knowledge of each disease, diagnostic tests and treatment protocols are essential to provide immediate and efficient/effective care to critical patients (Lightfoot, 2008).

2.2.5.1. Collapse/profound weakness

Stabilise the bird with O_2 therapy, IV/IO fluids containing dextrose and consider broad-spectrum bactericidal antibiotics such as enrofloxacin (7.5-15mg/Kg, IM/PO, q12h), cefotaxime (75-100mg/Kg, IM/IV, q4-8h), amikacin (hydrated birds, 10-15mg/Kg, IM/IV, q12h) or piperacillin (100-200mg/Kg, IM/IV, q6-8h) (Hess, 2002).

2.2.5.2. Acute respiratory distress

This is potentially life threathening and all patients should be gently handled to minimise stress and supplemental O_2 provided before handling (Echols, 2007). Some drugs can be administered to birds in respiratory distress before placement in an oxygen chamber, such as bronchial dilators (terbutaline 0.01mg/Kg, IM, q6-8h) (Plumb, 2005) and analgesics with antianxiety properties (butorphanol 1-2 mg/Kg, IM, q2-3h) (Echols, 2007).

It is important to differentiate decreased oxygenation from tracheal obstruction (which may require placement of an air-sac tube) from other conditions such as air sacculitis/pneumonia, sarcocystosis, internal haemorrhage, inhaled toxins, allergic pneumonitis, cardiac disease or abdominal masses (Lightfoot, 2008). History, auscultation and PE allow the clinician to reach

diagnosis (i.e. a bird from the genus *Amazona* with acute onset dyspnoea and abrupt loss of voice may have syringeal disease; a *Psittacus erithacus* fed only sunflower seeds may have hypovitaminosis A; and a Saker falcon - *Falco cherrug* - is likely to succumb to aspergillosis after stress) (Echols, 2007). Palpation of the abdomen allows detection of fluid, masses or displaced viscera. Auscultation of the lungs may not reveal subtle respiratory disease because of their rigidity but can help localizing and detecting abnormal sounds. Moist or harsh expiratory sounds are suggestive of bronchopulmonary disease while clicking and crackles indicate thickened air sacs touching (air sac disease). The heart should be auscultated for murmurs that may be evident with cardiac disease and an echocardiogram or US performed (Bowles et al., 2007).

Respiratory disease may affect the upper airways (sinus), large airways (glottis to syrinx), small airways (primary bronchi), parenchyma (lungs to air sacs) or intracoelomic cavity resulting in compression of the lungs (Echols, 2007).

2.2.5.3. Traumatic presentations

Traumatic injuries are common in the pet bird. The initial protocol for injured birds includes haemorrhage control, O_2, heat support, analgesics and parenteral antibiotics. External haemorrhage should be stopped immediately (Bowles et. al., 2007). The clinician should distinguish between haemorrhage from an external site and blood without active bleeding. Continued frank haemorrhage requires intervention while haemorrhage that has ceased is best left undisturbed. The color of the tissue in the choanal slit can be used to estimate anaemia. Hypovolaemia is the initial concern and rehydration should be performed rapidly. Moribund patients should have an IO/IV catheter placed, however, if the bird is still conscious this may be detrimental due to stress (Lightfoot, 2008). Warm SC fluids with hyaluronidase should be administered. Birds in septic shock or septicaemia from extensive wounds, should receive parenteral antibiotics (e.g. piperacillin, 100-200mg/Kg IM, IV q6-8h; trimethoprim-sulpha, 8-30 mg/Kg, IM, BID) (Hess, 2002 and Harrison & Lightfoot, 2006).

The goal of treatment is patient survival first and then accessing traumatized tissue. A bird that has been struggling for hours with a trapped wing may have a fractured bone but is in more danger of dying from stress related to prolonged struggling than complications from the fracture itself. Temporary stabilisation of the traumatized tissue is enough until the bird is stable. If the bleeding comes from the vent, the clinician should determine its origin (Lightfoot, 2008).

Birds with broken blood feathers often present as emergencies. The feather should be ligated or pulled at the base with a haemostat, extracting it from the follicle and pressure applied. In the case of large primary wing feathers, wing bones should be supported during extraction to avoid fractures. Haemostatic agents such as silver nitrate and styptic powder shouldn´t be applied on the feather follicle because they may damage it (Bowles et al., 2007) but can be used to stop the bleeding on broken nails (Hess, 2002).

The greater challenge is to recognize internal blood loss. The rate that blood is lost from circulation is the determining factor for mortality. Losing 20-25% of blood over minutes may be fatal while losing the same volume for hours is not. Discoloration or bruising of the abdominal wall, along the ventral midline and caudal abdomen, may suggest internal haemorrhage and radiology/endoscopy may help locating it (Jenkins, 2005).

Analgesia is necessary in birds that have suffered fractures or soft tissue trauma. Post-traumatic administration of nonsteroidal anti-inflammatory drugs (NSAIDs) may decrease soft tissue sensitization and reduce the dose of opioid drug required. Steroids are contra-indicated (Bowles et al, 2007).

2.2.5.3.1. Wounds (lacerations, bite wounds) and open fractures

The lesions should be flushed with chlorhexidine solution and parenteral β-lactams antibiotics (e.g. piperacillin 100-200mg/Kg, IM/IV, q6-8h), trimethoprim-sulpha or fluoroquinolones. Piperacillin/tazobactam or cefotaxime/amikacin are good choices and should be continued for a minimum of 5 days. Large recent wounds should be flushed with sterile saline solution and sutured partially closed and older lacerations flushed, debrided and bandaged with wet to dry bandages that should be changed twice daily. These wounds may be closed later when infection is minimized (Hess, 2002). Other option is to apply 50% dextrose, sugar and honey as a wound dressing in large contaminated wounds. These are inexpensive and readily available materials considered to be bactericidal because of their high osmolality and additionally provide the wound with moisture and a local nutrient source to promote granulation tissue. The high osmolality is also responsible for moving oedematous fluid out and drawing macrophages into the wound. The wound should be debrided and flushed with saline, then sugar/honey applied to it and covered by a wet-to-dry bandage. Some topical wound treatments use products that promote wound healing by stimulating the production of cytokines or growth factors within the wound (e.g. D-glucose, polysaccharide, hydrolyzed bovine collagen, porcine collagen products, tripeptide-copper complex) (Bowles et al., 2007).

Bite wounds from carnivores can lead to fatal septicaemia if not treated aggressively with systemic antibiotics (Jenkins, 2005) and silver sulfadiazine cream can be applied on contaminated wounds. Nonadherent bandaging materials are recommended to avoid disruption of the healing surface of the wound. Self-adherent bandages can tighten when wet, so the access of bandaged birds to open containers of water should be limited or monitored (Bowles et al., 2007).

In the case of self-mutilation, topical antibiotics such as silver sulfadiazine cream may be applied and the bird temporarily collared/bandaged with a moisture-permeable dressing while treating the underlying cause (Bowles et al., 2007). Ensure that the diet and home environment are appropriate. Psychotropic drugs (haloperidol, 0.2mg/Kg, q12h for birds

weighing less than 1Kg and 0.15mg/Kg, q12-24h for birds above 1Kg) may be used in cases of severe mutilation unresponsive to other therapies.

Diagnostic tests should include a CBC, plasma BC panel, EPH, faecal Gram´s stain and direct smear, polyoma and PBFD virus testing, radiographs and skin/feather biopsies (Hess, 2002).

2.2.5.3.2. External burn injuries

Burn injuries are classified by severity into superficial, partial thickness and full thickness burns.

For superficial burns only the epidermis is affected, with a transient erythema, desquamation and the site is very sensitive and hyperaesthetic. Such burns should be gently cleansed with sterile saline with 5% povidone iodine or chlorhexidine solution (Jenkins, 2005).

Partial thickness burns extend to the mid-dermis, loss of epidermis is complete, capillaries and venules in the dermis are dilated, congested and exude plasma (Bowles et al., 2007). The site may be painful but sensitivity is decreased (Jenkins, 2005).

Full thickness burns result in coagulation of epidermis/dermis so its no longer vital, a severe oedema of the subcutis develops because of the increased permeability of deep vessels and necrosis of the damaged tissues, resulting in dry, leathery eschar. Feathers and skin may be easily pulled or peeled, respectively (Jenkins, 2005).

Partial to full-thickness burns should be cleansed gently, debrided daily and treated topically with a water-soluble antibiotic dressing such as silver sulfadiazine. Due to associated pain, GA and analgesia should be provided (Bowles et al., 2007). If more than 50% of the body surface is involved (partial to full-thickness burns) prognosis is grave and euthanasia should be considered (Hess, 2002).

Diagnostics include radiography in case of exposure to smoke to evaluate pulmonary injury, and in severe/extensive burns haemogram and serum electrolyte testing are advised. Dyspnoeic birds often have laryngeal oedema and upper airway excretions so O_2 via an air sac tube may be provided. Systemic bactericidal antibiotics (e.g. Piperacillin) should be initiated in cases complicated by infection or treatments done outside a sterile environment. If the burn is recent, it should be treated with cold water or compresses for 20-30min (Bowles et al., 2007) to minimize coagulation and extent of burn depth by dissipating heat. Blood, protein loss and renal function should also be monitored (Hess, 2002).

Complications include circulatory collapse, decreased renal function (oliguria), renal failure, pneumonia, scarring/healing complications (especially in areas where tissue is mobile or under tension) and sepsis in the first 24-48h. Infection is a common cause of death for birds surviving the initial injury and can be caused by opportunistic bacteria (*Pseudomonas, Streptococcus, Proteus)* (Jenkins, 2005).

2.2.5.3.3. Fractures

Fractures alone are not usually life threatening. The bird should be evaluated for open or closed fractures and soft tissue trauma (Bowles et al., 2007) and the tissue immobilised. When splinting, the rule is to immobilise the joint above and below the fracture (Chavez & Echols, 2007). Bandages should be used to immobilise the fracture and supportive care provided until further fracture stabilisation (e.g. surgery, splinting) is attempted (Hess, 2002). It is preferable to let the bird stabilise than performing immediate surgery on a debilitated bird. Once stable, delays should be minimised to limit muscle contracture, which complicates surgical repair. Closed reduction and repair is associated with significantly less tissue damage (Bowles et al., 2007). Birds have little SC fat or other tissues, so immediate wound coverage decreases further damage and desiccation. If immediate wound closure is not possible, packing the wound with moistened sterile gauze sponges or a water-soluble lubricating gel (K-Y® Jelly, Johnson and Johnson Products) limits desiccation. The use of a water-based preparation is preferred and topical steroids avoided. A hydrogel containing acemannan (Carravet® Wound Dressing, Carrington Laboratories) stimulates healing and maintains moisture (Graham & Heatley, 2007). The selection of an appropriate technique for definitive repair will depend on several variables, including the size of the patient, the degree of postoperative return to function required, cost, the skill of the surgeon and concurrent medical conditions (Harrison & Lightfoot, 2006).

2.2.5.3.4. Ophthalmological injury

Ophthalmologic examination of anterior and posterior segments should be carried out in all trauma cases. Posterior segment examination should include evaluation of the pecten for signs of disruption. Retinal detachment and cataract formation are common sequelae of head trauma. Topical or systemic antibiotics are indicated for corneal ulceration or perforation. Topical ophthalmic NSAIDs can be used to treat uveitis in raptors without corneal laceration (Graham & Heatley, 2007).

2.2.5.3.5. Electrocution

It is not uncommon, especially in immature raptors, with a higher incidence during the winter. CS may not be evident for several days after injury. Consequences include cardiac arrest, pericardial effusion, neurogenic pulmonary oedema with acute respiratory distress and thermal burns, especially in the feet, head and carpi. Feather damage of the bars and barbules without damage to the rachis is a classic sign of electrocution. Injury can be so severe that euthanasia may be necessary (Graham & Heatley, 2007). Treatment is aimed at addressing the symptoms (Hess, 2002). Neurogenic pulmonary oedema may respond to a single dose (2mg/Kg) of furosemide (Lasix®, Sanofi Aventis, Paris, France) and supportive care with O_2 and sedation. This oedema is secondary to a capillary permeability-induced alveolar injury so continued administration of diuretics is not advised. The prognosis varies

from fair to grave depending on the extent and severity of the damage. Even if injuries are limited to the feathers, it may prevent releasing a wild bird from a moult cycle for up to 1 year (Graham & Heatley, 2007).

2.2.5.4. Egg binding (dystocia)

Cockatiels, budgerigars, lovebirds, finches and canaries are the most commonly and severely affected species, possibly because of their small size. It can occur from low Ca intake or overproduction of eggs. CS include acute depression, abdominal straining, blood-stained faeces, persistent tail wagging, wide stance, failure to perch, abdominal distension, dyspnoea and/or sudden death. If abdominal structures are compressed, circulatory disorders, lameness, paresis or paralysis can be present and pressure necrosis of the oviductal wall may occur (Bowles et al., 2007).

Presence of an egg is confirmed by radiography (with the bird standing awake on the radiographic cassette) unless the egg is low enough making palpation possible (Lightfoot, 2008). US can be useful and may identify soft-shelled eggs that are hard to see in radiographs. Blood work and serum BC can help determining secondary diseases (e.g. hypocalcaemia) (Stanford, 2005; Bowles et al., 2007).

The prognosis is guarded if the patient has paraparesis/paralysis, decreased BW, depression or labored breathing. Supportive care must be provided before extraction of the egg is attempted, including fluid therapy, injectable Ca gluconate (25-100mg/Kg, IM), adequate warmth/humidity and nutritional support (Lightfoot, 2008). If an infectious aetiology is suspected or the integrity of the oviduct compromised, broad-spectrum antibiotics are indicated. Analgesics are often required. Supportive care is usually enough for non-obstructive dystocia but care must be taken with monitoring the bird closely for any deterioration that may require further intervention (Bowles et al., 2007). If supportive care doesn´t lead to oviposition, further treatment with oxytocin and the avian equivalent, arginine vasotocin for uterine contraction, may be attempted (Lightfoot, 2008). Prostaglandin and hormonal therapy may be used to induce oviductal contraction which can result in extraction of the egg but only if: i) the contractility of the oviduct is sufficient; ii) the uterus is intact; iii) the egg is within the oviduct, and iv) there is no obstruction or egg adhered to the oviduct (Bowles et al., 2007). Oxytocin is more readibly available, but it does not dilate the uterovaginal sphincter, so oviduct rupture is possible (Lightfoot, 2008). $PGF_2\alpha$ and PGE_2 may also be administered. Neither $PGF_2\alpha$ or oxytocin relax the uterovaginal sphincter while inducing oviductal contractions, so the sphincter should be opened in order to avoid reverse peristalsis, severe pain and rupture of the uterus. It should also be noted that prostaglandin and hormonal therapy require exogenous Ca to be effective so supplemental Ca may be necessary prior to administration of these medications. PGE_2 gel can be applied topically to the uterovaginal sphincter (0.1ml/100g BW), causing relaxation of the sphincter and oviductal contractions while decreasing the incidence of systemic side effects. PGF2α is administered

parenterally, rather than locally increasing the risk of systemic side-effetcs (e.g. hypertension, bronchoconstriction, general smooth muscle stimulation) (Bowles et al., 2007).
If even after medical therapy the egg doesn't pass, GA and manual extraction may be necessary (Lightfoot, 2008).
Complications of these procedures include retroperistalsis of the egg out of the oviduct into an ectopic position within the ceolom, egg rupture, oviductal trauma/laceration, cloacal or oviductal prolapse and displacement of the egg or fragments out of the uterus and into the coelom which may cause an egg-related peritonitis (Bowles et al., 2007). If fertilisation has occurred and the egg is removed intact, it can be incubated. If any haemorrhage has occurred, antibiotics are indicated. Ovocentesis may aid in the passage of the egg, being performed through the cloaca if the egg is located distally or by lateral coeliotomy if more cranial (Lightfoot, 2008).
Radiography should be used to confirm that all the eggshell pieces have been expelled. It may be advised to flush the uterus postoviposition with saline, chlorhexidine or iodine to remove any shell fragments and decrease the incidence of metritis. In the case of ectopic eggs and dystocia or if after all the procedures described oviposition doesn´t occur salpingohysterectomy may be considered and secondary diseases corrected (Bowles et al., 2007).

2.2.5.5. Neurologic disease

2.2.5.5.1. Head trauma

If MRI and CT are not available, history and CS can guide diagnosis and treatment protocol. If there are no fractures, radiographs are usually unrewarding (Bowles et al., 2007). Birds should be placed in a dark, quiet area at approximately 24ºC (Hess, 2002). Analgesics are provided to restless birds. Seizures can be controlled with phenobarbital (2-7mg/Kg, PO, BID) or potassium bromide (25-75mg/Kg, PO, SID) (Carpenter, 2005 and Bowles et al., 2007). The mainstay of head trauma treatment is O_2 and maintenance of a normal blood pressure (BP). Systemic hypotension with intracranial hypertension (e.g. vascular bleed, mass) may worsen neurologic injuries and outcome (Bowles et al., 2007). If intracranial haemorrhage has occurred, fluids should be given judiciously at ½ to ⅔ the replacement volumes (Hess, 2002) (0.9% sodium chloride bolus, IO/IV, 10ml/Kg) until indirect systolic BP is 90-120mmHg. Mannitol (0.5mg/Kg, IV slowly) is used if there is no response to therapy and worsening of neurological status (i.e. stupor to comatose) (Bowles et al., 2007).

2.2.5.5.2. Seizures

Seizures can occur as a result of hypocalcaemia (e.g. *Psittacus erithacus* vitamin D3 deficiency), hypoglycaemia (e.g. neonates, raptors) (Hess, 2002), hypertension, toxin exposure, trauma or idiopathic epilepsy (e.g. *Agapornis roseicollis, Nymphicus hollandicus*) (Bowles et al., 2007). Work up includes PE and evaluation of blood glucose, Ca and

PCV/total protein (TP). When stable, further tests can be performed such as CBC and BC panel, radiographs and heavy metal screening (Hess, 2002). Indirect systolic BP should be measured with ideal levels being less than 200mmHg. Birds that present with *status epilepticus* should be given an injection of midazolam (0.25mg/Kg, IM) and the underlying cause adressed. If this is not effective the bird should be anaesthetised (birds will usually stop seizuring under GA) and phenobarbital administered IM every 15min at 4 mg/Kg for a maximum of 2 doses. The bird is then recovered in an incubator with the head elevated and phenobarbital continued at a daily oral maintenance dose of 2mg/Kg, PO for 12h after the phenobarbital load (Bowles et al., 2007). If seizures occur from hypoglycaemia, appropriate therapy includes the use of IV dextrose (50-100mg/Kg, IV slowly to effect) and diazepam (0.5-1.0mg/Kg, IV, IM, q8-12h). The cage should be padded and food and water available (Hess, 2002).

2.2.5.5.3. Heavy metal toxicity

Heavy metal toxicity it's usually caused by Pb or Zn. Birds may present with a history of investigating/ingesting foreign objects or no known exposure (Bowles et al., 2007). Sources of contamination include curtain weights, stained glass windows, coins, gunshot, galvanized cage wire, zipper teeth, staples, screws, nails, contaminated drinking water or paint/water pipes from old houses (Bowles et al., 2007 and Jenkins, 2005). Heavy metal toxicity is uncommon in passerines because of their limited capacity to destroy metal objects (Matos & Morrisey, 2005). *Amazona* spp. birds usually present with haematuria or haemoglobinemia. Haemoglobinuria presents as a classic "chocolate milk" to blood colored droppings (Bowles et al., 2007). Zn poisoning is now more commonly seen than Pb poisoning, which is more severe and likely to cause neurologic signs other than lethargy and sometimes haemoglubinuria but combination of both toxicoses is common (Jenkins, 2005).

Other less common metal toxicities include copper, iron, mercury (fish-eating birds) and arsenic (Graham & Heatley, 2007).

General illness with GI signs is the most common complaint with both toxicoses. Blood work may be normal or reveal moderate leukocytosis, mild to severe anaemia (Lightfoot, 2008) and erythrocytic ballooning (Bowles et al., 2007). Serum BC may show elevations of LDH, AST, CPK and uric acid. Blood Pb levels above 20µg/dL are suggestive of toxicity and greater than 50µg/dL are diagnostic. For Zn, diagnosis is made based on blood or tissue levels greater than 200µg and 75µg, respectively, but CS may not be noticed until levels of 1000µg/dL. Samples should be submitted in plastic containers as the rubber stoppers may leach Zn, giving a false low result (Jenkins, 2005).

Treatment is similar for both metals and includes fluid therapy, antiemetics, nutritional support, chelation and anticonvulsants if indicated. Radiography may reveal metallic objects in the GI tract or the metal may have already passed in the droppings (Bowles et al., 2007). Early administration of chelation therapy may be life-saving and consists of injectable Ca

EDTA (30-40mg/Kg, IM, q12h) and oral penicillamine can be started after 5 days at 30mg/Kg, PO, q12h for 14 days. Ca EDTA has the benefit of parenteral therapy for regurgitating patients or those that can´t absorb oral D-penicillamine effectively and in the early period when penicillamine is contra-indicated as it increases GI ion uptake (Bowles et al., 2007). Once the bird is stable, oral therapy should be continued at home with penicillamine (50mg/Kg, PO, q24h). Duration of treatment varies but shouldn´t exceed 2 weeks (renal toxicity) (Lightfoot, 2008). Barium sulfate or activated charcoal can help moving Pb particles out of the GI tract (Bowles et al., 2007) but larger Pb objects may be removed using a rigid or flexible endoscope after stabilisation. Surgical removal is only indicated as a last resort.

2.2.5.5.4. Hypocalcaemia syndrome

The hypocalcaemia syndrome is commonly seen in chronic egg layers. During egg formation, large quantities of circulating Ca are taken up by the shell gland (Bowles et al., 2007) depleting blood levels and bone stores. Poor nutrition is a major predisposing cause and CS include weakness, tremors and seizures with pathologic bone fractures often present (Jenkins, 2005). Ionized Ca levels less than 1mg/dL or total Ca less than 8mg/dL are often suggestive of hypocalcaemia. It is ideal to measure ionized Ca since hypoalbuminaemia can lower the total Ca falsely (Bowles et al., 2007). The birds should be given a proper diet and Ca and vitamin D_3 supplementation provided (Dorrestein, 2000). Birds require exogenous vitamin D_3 for a correct Ca metabolism (Bowles et al., 2007) which can be obtained by 11-30 minutes of exposure to direct sunlight each day or vitamin D_3 can be given parenterally: i) 3.300 IU/Kg (1000 IU/300g), IM, q7d as needed, or ii) 6.600 IU/Kg, IM, once. Care should be taken since hypervitaminosis D may occur with excessive use (Carpenter, 2005). Ca is given at 50-100mg/Kg, IM (Bowles et al., 2007).

An hypocalcaemic syndrome has been associated to *Psittacus erithacus*, apparently caused by an abnormal Ca and vitamin D_3 metabolism with CS varying from incoordination to *status epilepticus* so any *Psittacus erithacus* with seizures is suspect. In this case treatment as before plus diazepam (0.5-1.0mg/Kg IV, IM, q8-12h) to help control the seizures (Hess, 2002 and Jenkins, 2005).

2.2.5.6. Gastro-intestinal disease

2.2.5.6.1. Hypovolaemia/dehydration (from vomiting, regurgitation or severe diarrhoea)

Pathological vomiting or regurgitation can lead to life-threatening dehydration, starvation, electrolyte inbalances and aspiration pneumonia. Aetiologies include neonate crop stasis, ingluvitis, dietary indiscretion, infection, goitre in budgerigars (Matos & Morrisey, 2005), *Chlamydophila psittaci*, heavy metal or metabolic causes (e.g. sepsis, pancreatitis). Diagnostic testing includes CBC, plasma BC panel and EPH, oesophageal/crop culture and sensitivity and cytology, faecal Gram´s stain, radiographs, *Chlamydophila psittaci* and heavy

metal testing. Fluoroscopy or barium series can identify delayed gastric emptying time or obstructions (Bowles et al., 2007). Parenteral fluids with balanced electrolytes and an energy source should be given and the bird should be off food until vomiting ceases, then, nutritional support via feeding-tube should be provided (Hess, 2002). Appropriate antibiotics and/or antifungals are indicated when large numbers of Gram-negative bacteria or yeast are detected on cytology. Metoclopramide or cisapride improve crop motility and control regurgitation but shouldn´t be used in the case of obstruction (Bowles et al., 2007).

Severe diarrhoea can be caused by large or small bowel disease. Etiologies include diet change/indiscretion, protozoa (e.g. *Giardia, Cryptosporidium*), toxins, systemic disease, FB or neoplasia. True diarrhoea should be differentiated from watery stool due to polyuria. Blood in the stool can be caused by cloacal papillomas, haematuria (Zn/Pb), cloacal trauma and irritation (Bowles et al., 2007). Medications or stress can alter stool consistency. Diagnostic tests should include a faecal sample for Gram´s stain, cytology and culture of abnormal findings. Cloacal mucosa should be examined. Fluid therapy and treatment of underlying causes is necessary (Hess, 2002).

2.2.5.6.2. Crop burns

Crop burns are seen in young birds, caused by improperly heated or mixed hand-feeding formula. Some birds become ill from the tissue damage, develop endotoxaemia and die while others are asymptomatic even with a fistulated crop with clear demarcation between healthy and necrotic tissue (Lightfoot, 2008). If the bird is clinically ill, antibiotics should be started as well as fluid therapy, antifungal drugs and also gavage-feeding frequently in small volumes of a hand-feeding formula. The fistulated wound will generally heal within 7-14 days (Hess, 2002). It is not advised to perform surgery immediately after the burn has occurred, but to instead wait for necrotic tissue demarcation and for the area to start to granulate, providing a healthy tissue bed for surgical reconstruction, which will decrease the quantity of resected tissue (Lightfoot, 2008).

2.2.5.6.3. Gastro-intestinal foreign bodies

Larger psittacines can chew up and splinter wood, metal and bone. They may also play with cotton or wool attached to needles, and with large cactus houseplants. In waterfowl, impacted hooks and/or line may be found in the oesophagus. FB can become lodged in the tongue, oesophagus or crop. When feeding young birds, metal or plastic crop feeding tubes can be lost down the upper alimentary canal unless care is taken (Dorrestein, 2000 and Lightfoot, 2008).

In many cases, it will be known what type of FB has been swallowed. The bird may be presented trying to regurgitate the offending object, which can sometimes be palpated in the oesophagus or crop or demonstrated on radiography. In some cases, the FB can be gently removed out of the oesophagus with the bird conscious, however, forceps removal under

sedation or GA may be necessary (Dorrestein, 2000). GI foreign bodies can be identified and/or removed with the aid of an endoscope either PO or through an ingluviotomy incision. When an ingluviotomy is performed, the incision should be done in an avascular area on the left side of the crop (Lightfoot, 2008).

2.2.5.6.4. Acute poisonings

Common responsibles for acute poisonings in birds include insecticides (e.g. organophosphates, carbamates) and rodenticides (e.g. warfarin, bromadoline) (Graham & Heatley, 2007).

Insecticide intoxications occur after ingestion of contaminated food/water or secondary infections in the case of wild insectivorous species (Graham & Heatley, 2007). Pathology and CS result from the binding of the insecticide to, and inhibition of acetylcholinesterase (AChE), with accumulation of acetylcholine (ACh) in the ganglia and neuromuscular junctions. CS include anorexia, weakness, crop stasis, muscular twitching, ataxia, prolapsed nictitans, increased respiratory secretions, dyspnoea, bradychardia and death. Diagnosis is based on history, CS and response to therapy (Hess, 2002) with bradycardia non-responsive to atropine (0.02mg/Kg, IV) being suggestive. Definitive diagnosis is based on a cholinesterase assay from blood, plasma or serum, paired with an analogous subject. Pralidoxime chloride (2-Pam) is effective for cases presented soon after ingestion but care should be taken using 2-Pam with raptors since its toxic to these birds. In the case of carbamate toxicity the use of 2-Pam is contra-indicated (Jenkins, 2005).

Anticoagulant rodenticides include 1st (warfarin) and 2nd generation (brodifacoum, bromadoline) rodenticides. These agents are vitamin K antagonists that deplete and block the synthesis of prothrombin, acessory factors VII, IX and X. CS include depression, anorexia, feather follicle and SC haemorrhages (Coles, 2007). Once haemorrhage is noted, prognosis is guarded (Hess, 2002). Treatment involves vitamin K supplements and fresh whole blood transfusion for critical cases. Vitamin K1 is given IV until stabilisation then given SC/IM/PO daily (Coles, 2007). Bromethalin is not an anticoagulant but is a highly potent rodenticide that provides a lethal dose to rodents in a single feeding. It's pale, odourless and crystalline. It acts by uncoupling oxidative phosphorylation in the mitochondria of the central nervous system (CNS) leading to a decreased production of adenosine tri-phosphate (ATP). Lowers levels of ATP inhibit the activity of the Na/K ATPase leading to a subsequent build-up of cerebral spinal fluid (CSF) and vacuolization of myelin. The increased CSF leads to high intracranial pressure and damage to nerve axons, inhibiting neural transmission and leading to paralysis, convulsions and death (Jenkins, 2005). Venipuncture should be limited in cases of suspected rodenticide intoxication to limit blood loss (Graham & Heatley, 2007).

For any poisoning the main principle is to treat the patient, not the toxin. Patients should be stabilised and further exposure and absorption prevented/delayed. Soiled birds should be bathed, crops lavaged and absorbent/cathartic medications administered. Few toxins have

specific antagonists or antidotes available (Jenkins, 2005). Treatments that help removing the toxin (e.g. diuresis) can be performed (Bowles et al., 2007). For treatment for non-specific ingested toxins, GI protectant can be used such as slurry of fine activated charcoal with water as directed to 15ml (Jenkins, 2005). All raptors with suspected toxicosis should receive a complete necropsy and results reported to the wildlife authorities (Graham & Heatley, 2007).

2.2.6. Continued care

The goal of supportive therapy is to stabilise the patient until specific therapy can be established. Correcting fluid deficits and hypothermia is necessary and as the diagnosis is delineated, treatment becomes more focused (Harris, 2003). The aids of supportive care are heat, fluid administration, O_2 supplementation and gavage feeding. The optimum temperature for the ill bird is 29-30ºC. Heat sources should include a heating pad, clamp lamp, ceramic heat emitter or light bulb on one side of the cage avoiding direct contact of the heat source with the patient or putting the cage in a room with a space heater combined with thermometers to monitor cage temperature. Birds should be transported to the hospital in a warm enclosure (Harrison & Lightfoot, 2006).

When the patient starts to stabilise, routine-handling methods can be performed but supportive care should never be abruptly stopped (Bowles et al., 2007). Fluid therapy should be continued or hydration status monitored for 3-4 days, making sure deficits are fully corrected. Supplemental heat is usually unnecessary for adult birds once stable but O_2 should be continued. Calorific and nutritional support is also a major consideration once the bird is stable (Harris, 2003).

2.2.7. Fluid therapy

It is a clinical challenge when veterinarians have to administer critical care therapy to birds because of small size, physiologic diversity and lack of clinical data on response to therapy (Lichtenberger, 2007b). The thoughtful use of therapeutic procedures on a debilitated bird often correlates to the success or failure when treating that patient (Tully Jr. & Beaufrère, 2011).

2.2.7.1. Shock pathophysiology

Shock is defined as poor tissue perfusion from either low or unevenly distributed blood flow, resulting in an inadequate delivery of O_2 to the tissues (Lichtenberger, 2007b). There are many types of shock but this topic will focus on hypovolaemic shock, caused by decreased blood volume which can be absolute or relative (Lichtenberger, 2005). Absolute hypovolaemia occurs as a result of blood loss (e.g. arterial bleeding, coagulopathies, GI ulcers) while in relative hypovolaemia there is no direct blood loss from the intravascular space (e.g. severe dehydration from GI tract loss, loss of plasma in burns, extensive loss of

intravascular fluids). With significant loss of fluid and blood volume, blood return to the left side of the heart decreases, therefore, CO also decreases. With hypovolaemia greater than 30% blood/plasma volume, BP decreases below a mean arterial pressure of 60mmHg or a systolic pressure of less than 90mmHg and when this occurs, baroreceptors alert the vasomotor center in the *medulla oblongata* (Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011), inhibiting the vagal parasympathetic center and stimulating the sympathetic center, causing constriction of the peripheral vasculature and increased HR and strength of contraction. Circulating catecholamines increase through a humoral response, promoting the release of renin which stimulates activation of the renin-angiotensin-aldosterone system (RAAS) (Lichtengeberger, 2007b). Renin catalyses the transformation of angiotensin I to angiotensin II in the lungs, a strong vasoconstrictor that excites the adrenal gland to liberate aldosterone causing the reabsorption of sodium chloride. Aldosterone also acts on the hypothalamus causing the release of vasopressin. Receptors for vasopressin are located in the vascular smooth muscle and renal tissue, resulting in vasoconstriction and increased water reabsorption respectively, as a way to stabilise intravascular volume (Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011) leading to restoration of BP, increased cardiac performance and maximal venous return in case of blood loss.

According to Tully Jr. & Beaufrère (2011) hypovolaemic shock has 3 phases, as cited by Lichtenberger (2007b):

1. Early or compensatory phase: (...) as a result of the baro-receptor mediated release of catecholamines. Blood pressure increases because of the increase in cardiac output and systemic vascular resistance. (...) seen in birds with fluid loss less than 20% of their total body weight. Clinical signs in the bird include increase in heart rate, normal or increased blood pressure, and normal or increased flow (...). Volume replacement at this stage is usually associated with a good outcome.
2. Early decompensatory phase: (...) when fluid losses continue(...). Clinical signs (...) include hypothermia, cool limbs and skin, tachycardia, normal or decreased blood pressure, pale mucous membranes, prolonged capillary refill time and mental depression. This stage of shock is seen in birds with a blood volume fluid loss of greater than 25 to 30% of their total blood volume. Agressive fluid therapy (...) is required in this stage.
3. Decompensatory shock: When a large blood volume is lost (greater than 60% in avian species). The neuroendocrine responses to hypovolemia become ineffective and irreversible organ failure begins. (...) is the final common pathway to all forms of shock in all species.(...). The clinical signs are bradycardia with low cardiac output, severe hypotension, pale or cyanotic mucous membranes, absent capillary refill time, weak or absent pulses, hypothermia, oliguria to anuric renal failure, pulmonary oedema and stupor to comatose state. Cardiopulmonary arrest is common (Lichtenberger, 2007b, p.276-277).

2.2.7.2. The goals of fluid therapy

The goal of fluid therapy is to administer the least amount of fluids needed, while replacing fluid deficits, anticipating fluid loss, providing maintenance requirements, and correcting electrolyte and acid-base values. It is important to have a treatment goal, to continually assess clinical response to treatment, to intervine when necessary and to treat any underlying conditions (Tully Jr. & Beaufrère, 2011).

The fluid therapy plan typically has a resuscitation (correction of perfusion deficits), a rehydration (correction of interstitial deficits) and a maintenace phase. Intravascular volume must be replaced first and re-evaluation of hydration status after resuscitation is needed before continuing the rehydration phase to prevent volume overload. Interstitial deficits are associated with decreased skin turgor and dry mucous membranes (MM). Rehydration is accomplished using an isotonic replacement fluid; and for animals that are stable at a cardiovascular level, fluid deficits can be replaced in 12-24h and if rapidly lost in 1-4h (Lichtenberger, 2007b). Isotonic fluids are administered according to the patients estimated dehydration, maintenance needs and anticipated ongoing losses (Tully Jr. & Beaufrère, 2011). Maintenance requirements are higher than mammals in birds (high metabolic rate) (Lichtenberger, 2007b). Monitoring endpoints are still poorly understood but may include indirect arterial BP measurement, lactate or normal values for blood gases (Bowles et al., 2007).

2.2.7.3. Fluid therapy plan for the avian patient

After stabilisation, fluid therapy may be initiated (Tully Jr. & Beaufrère, 2011) and blood samples taken for PCV, TP and bicarbonate measurements. Monitoring urine output by checking the droppings for changes of quantity, color or consistency, and measuring glucose daily is a good starting point (Bowles et al., 2007).

Fluid therapy should be provided to all birds that are critically ill or injured, dehydrated, before or during surgery, if haematocrit greater than 55%, TP less than 2.0mg/dL or above 3.5mg/dL and blood glucose of less than 200mg/dL or more than 600mg/dL (Lichtenberger, 2007b). Normal saline or Lactated Ringer´s solution (LRS) can be administered SC/IV/IO/PO or through the cloaca, IO/IV cathethers are the preferable method for rapid rehydration with isotonic fluid solution given at ½ of fluid deficit for the first 12h as a bolus over 20min (Echols, 2007). SC fluid therapy is not an effective method of rapid restoration of circulatory fluid volume but adding hyaluronidase increases the absorption rate of the fluid into the circulatory system (Griffin & Snelling, 1998 cited by Tully Jr. & Beaufrère, 2011). SC fluid administration should not exceed 10ml/Kg/site (Dibartola & Bateman, 2006 cited by Tully Jr. & Beaufrère, 2011). SC fluid doses for some species are: budgerigar 1-2ml; cockatiel 3-8ml; amazon 15-20ml; african grey parrot (AGP) 15-20ml; macaw 20-35ml. Intraperitoneal fluids are not advised due to the risk of entering the air sac system (Harris, 2003 and Johnson-Delaney, 2008).

When determining dehydration, deficit percentage is calculated prior to replacement fluid volumes. Dehydration status can be estimated in psittacines by skin fold elasticity, corneal moisture and globe appearance (sunken eyes), MM (dry, pale), tachycardia/bradycardia, tachypnoea, cold extremities, weakness, reduced urine output, prolonged CRT, weak and thready pulse/hypothension, degree of loss of BW, altered mentation and PCV (Tully Jr. & Beaufrère, 2011). Dehydrated chicks have wrinkled and reddened skin, sunken face and prominent eyes (Clubb et al., 1992 cited by Tully Jr. & Beaufrère, 2011). These factors are subjective since they can also occur with decreased body fat or increased age. In most cases of severe trauma or disease a 5-10% dehydration status is estimated; 4-6% if there is increased skin tenting, dry oral MM, dullness to the skin and normal pulses; 10% if the animal presents with severe skin tenting, very dry MM, mild hypothermia, thick oral secretions, dry eyes; and more than 10% when presented with signs of hypovolaemic shock (e.g. profound weakness, tachycardia, collapse) (Lichtenberger, 2007b). To determine the volume of fluid required for rehydration the following formula should be used (Tully Jr. 2000 and Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011):

Volume (L) = hydration deficit x BW (Kg) x 1000 ml

(i.e. a 1Kg bird that is 10% dehydrated requires 0.1%x1x1000 ml or 100 ml of fluids for rehydration)

Dehydration deficits are added to daily maintenance fluid requirements and then an estimate of ongoing losses should be added. Maintenance requirements for birds are estimated to be 3-4ml/Kg/h (Lichtenberger, 2007b). The estimated deficit should be replaced over a 48-72h period (Tully Jr., 2000 cited by Tully Jr. & Beaufrère, 2011), 80% of the which in the first 24h. Usually acute losses are replaced over 6-8h and chronic losses over 12-24h (Stone & Redig, 1994 cited by Tully Jr. & Beaufrère, 2011). The recommended daily fluid maintenance formula for psittacine species is 50-100ml/Kg/day (Steinhort, 1999 cited by Tully Jr. & Beaufrère, 2011) and for neonates 50-150ml/Kg/day. Recent recommendations to compensate tissue fluid loss are crystalloid (10ml/Kg) and colloid (5ml/Kg) increments combined given in 1-2 bolus infusions to raise BP above 90 mmHg systolic (Tully Jr. , 2010 and Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011). Once the deficit is replaced and the bird eats and drinks normally for 2-3 days, maintenance therapy can be discontinued (Tully Jr. & Beaufrère, 2011). Enteral feeding amounts can be used as part of the maintenance requirements and should be included in the calculation of fluid volumes required (Lichtenberger, 2007b).

The approximate maximum initial IV bolus fluid doses for different species are: finch 0,5ml; budgerigar 1.0ml; cockatiel 2.0ml; conure 6.0ml; amazon 8.0ml; AGP 8.0ml; cockatoo 12-14.0ml; and macaw 12-14.0ml (Johnson-Delaney, 2008).

2.2.7.4. Types of fluids

When developing a fluid therapy protocol it is important to know which products to use, based on the knowledge of avian osmoregulation and response to the replacement fluids, differential diagnosis for the underlying conditions, identification of fluid deficits/electrolyte abnormalities and appropriate route of administration. Until now, fluid therapy has been administered to birds by extrapolation from other animals, however comparisons between plasma osmolality values in mammals and values reported for some psittacine species suggest that plasma osmolality is slightly higher in parrots than in mammals and species-specific differences exist (Tully Jr. & Beaufrère, 2011). The dynamics of body fluids between compartments and across membranes are based on the composition and osmotic pressure of each (Beaufrère et al., 2010 cited by Tully Jr. & Beaufrère, 2011), consequently, fluids with a slightly higher osmolarity than mammals, close to 300-320mOsm/L such as Normosol-R, Plasmalyte-R, Plasmalyte-A and NaCl 0.9% can be recommended in parrots when isotonic fluids are required (Tully Jr. & Beaufrère, 2011). Warmed fluids should be administered at 38-39ºC over 5-10min, for most patients and individual characteristics of fluids influence type and volume administered (Wilson,2005).

2.2.7.4.1. Crystalloids

Crystalloids are the mainstay of the rehydration and maintenance phases of fluid therapy containing sodium chloride and other solutes capable of distributing to all body fluid compartments (Lichtenberger, 2007b) because of their electrolyte concentrations similar to extracelullar fluid (Tully Jr. & Beaufrère, 2011). The most commonly used are 0.9% saline, LRS, Normosol-R or Plasmalyte-A (Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011). Hypertonic saline is a hyperosmolar crystalloid used for resuscitation of hypovolaemia administered as a 7.5% solution. Hyperosmolarity leads to rapid expansion of intravascular volume as it draws fluid from the interstitial and intracellular spaces into the intravascular space. Administer 5ml/Kg over 5-10min or 3-5ml of hetastarch in combination with 3-5 ml/Kg of 7.5% hypertonic saline (not mixed in the same syringe) each given over 10min. Only a limited portion of the volume deficit needs to be administered when using hypertonic saline.

Synthetic colloids provide a synergistic effect if added to hypertonic saline as the duration and extent of volume resuscitation is greater than with either agent alone, because 80% of the volume of crystalloid fluid infused re-equilibrates and leaves the intravascular space within 1h of administration (Lichtenberger, 2007b). Crystalloids expand the intravascular space but such effect is short-lived; crystalloids are interstitial hydrators not intravascular expanders. Buffered solutions containing lactate, gluconate or acetate are needed for resuscitation because of the metabolic acidosis but care should be taken in case of liver disease (Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011) because hepatic lactate conversion may be compromised (Echols, 2007). Fluids containing Ca shouldn´t be

administered in the same line as blood products with citrate anticoagulants because a precipitate may form (Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011).

Crystalloid fluids may lead to tissue oedema (Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011). Potential side effects of hypertonic saline include hypernatraemia, hyperchloraemia, hypokalaemia and dehydration. Cell dehydration is one of the feared complications of hypertonic saline resuscitation, so hypertonic saline should be avoided in dehydrated patients, because the extravascular fluid compartment is volume-depleted before therapy (Lichtenberger, 2007b).

When fluid therapy is prolonged or serum K levels are less than 4.0mEq/L, K may be added. KCl at a rate of 0.1-0.3mEq/Kg to a maximum dose of 11mEq/day at a rate of 0.5mEq/Kg/h can be added to the LRS. If the patient is hyperkalaemic (e.g. due to severe tissue injury, catabolic state, renal dysfunction), LRS is not contraindicated and Ca gluconate (0.5ml/Kg) facilitates movement of K across cell membranes (Hernandez & Aguilar, 1994 cited by Tully Jr. & Beaufrère, 2011).

2.2.7.4.2. Colloids

Colloids contain large molecular-weight substances generally not able to pass through capillary membranes (Tully Jr., 2000 & Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011), so they are considered intravascular volume expanders. There are 3 types of colloids: natural colloids (e.g. whole blood, plasma, albumin), synthetic colloids (e.g. Hetastarch) and hemoglobin-based O_2 carriers (e.g. Oxyglobin®, OPK Biotech) (Lichtenberger, 2007b). Natural colloids have limited availability/use in avian practice. The negative charge of hetastarch attracts Na and water while being maintained in the intravascular space thereby expanding volume of this area by 1.4 times the volume infused. Synthetic colloids are administered with isotonic crystalloids to reduce interstitial tissue depletion and dose of crystalloid administered (Tully Jr., 2000 & Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011 and Lichtenberger, 2007b) to only 40-60% of what would be needed if crystalloids were used alone (Lichtenberger, 2007b). Hetastarch is recommended for head injuries, pulmonary trauma and myocardial dysfunction. Hetastarch´s half life is 25h and oxyglobin 30-40h (Tully Jr., 2000 & Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011). In birds, oxyglobin has been infused as a rapid bolus at 5ml/Kg over few minutes (Lichtenberger, 2007b). Caution is advised when using synthetic colloids in patients with congestive heart failure or anuric/oliguric renal failure because they are excreted by the kidney (Joseph, 1998 cited by Tully Jr. & Beaufrère, 2011). The recommended dose of hetastarch is 10-15ml/Kg, IV for 1-4 treatments over a 24h period (Stone & Redig, 1994 cited by Tully Jr. & Beaufrère, 2011). The volume of crystalloid should be reduced by the volume of hetastarch used to prevent fluid overload (Joseph, 1998 cited by Tully Jr. & Beaufrère, 2011).

2.2.7.5. Glucocorticoids and sodium bicarbonate in shock

The use of glucocorticoids is controversial since they have not shown consistent efficacy in shock. Side effects outweigh their benefits in birds so they are not currently recommended.

When severe acidaemia resulting from lactic acidosis is present and aggressive measures to improve O_2 delivery and reverse tissue hypoxia have been introduced without improvement, crystalloid fluids with lactate, acetate and gluconate are used. They increase pH by increasing the alkalinity of extracellular fluid (Lichtenberger, 2007b). Sodium bicarbonate can be given at the dose rate of 1-2mEq/Kg over 30min as an infusion (Lichtenberger, 2004 cited by Tully Jr. & Beaufrère, 2011). Blood gas parameters should be monitored but since this is rarely possible in the avian patient, the use of sodium bicarbonate in shock is not recommended (Lichtenberger, 2007b).

2.2.8. Blood transfusion

Most animals can tolerate acute blood loss of 10-15% of their blood volume without a transfusion. Acute haemorrhage of more than 20% blood volume often requires transfusion after initial fluid resuscitation (Lichtenberger, 2007b). Recent studies (Shaw et al., 2009) recommend whole blood transfusions when PCV is less than 15% and if more than 40% of blood volume is lost. Regenerative or non-regenerative anaemia is diagnosed by measuring the PCV (Tully Jr. & Beaufrère, 2011) which after the transfusion should be maintained above 25%. Whole blood can be administered at 10-20ml/Kg, IV/IO. A cross-match is recommended (Morrisey, 2006 cited by Lichtenberger, 2007b) since fatal reactions have been described in birds given multiple heterologous blood transfusions (Lichtenberger, 2003 cited by Tully Jr. & Beaufrère, 2011). Although blood groups have not been studied in birds (Lichtenberger, 2007b) autologous and homologous blood transfusions are preferred (Wilson 2005; Finnegan et al., 1997 cited by Echols 2007). Blood should be collected from the donor by a jugular cathether or other accessible vein at a maximum volume of 1% BW and 1:6 to 1:9 anticoagulant added to blood such as heparin, acid-citrate dextrose (ACD), citrate-phosphate dextrose (CPD) or citrate-phosphate-dextrose-adenine 1 (CPDA1) avoiding citrate anticoagulants in hypocalcaemic patients. Transfusion should be performed within 4h of collection (Tully Jr. & Beaufrère, 2011). Blood should be warmed (42ºC) at least 15min before administration. The blood administration set should have a filter to remove most of the debris and blood administered by slow bolus or infusion with a syringe pump into an IO/IV catheter (jugular/basilic/metatarsal veins) (Lichtenberger, 2007b). Recommended blood volume replacement is 13-22ml/Kg delivery rate at 0.5ml/Kg for the first 20min. Recommended blood therapy involves administration of vitamin K, which improves clotting time, iron for formation of haemoglobin (contraindicated if infection or iron storage disease), supportive/fluid therapy and blood product administration (Shaw et al., 2009). Oxyglobin® (OPK Biotech, Massachusetts, USA) has the advantage over whole blood transfusions in

reducing disease transmission, also because a cross-matching is not needed and storage time is up to 3 years (Lichtenberger, 2007b). The patient should be monitored during and after blood transfusion and PCV evaluated 1-2h after the transfusion (Tully Jr. & Beaufrère, 2011). The relatively short live span of the RBCs (28-45 days) and presence of nucleus may account for a bird´s ability to mount a very early regenerative response (Lichtenberger, 2005). Iron dextran (except if bacterial infection or iron storage disease are concerns) and vitamic B complex (10mg/Kg) should also be considered for anaemic birds (Echols, 2007; Lichtenberger, 2007b).

2.2.9. Monitoring blood pressure

Systemic vascular resistance (SVR) and CO are responsible for BP and should be monitored. Although the "gold standard" is the direct method, arterial catheterization in birds is difficult and time-consuming, so indirect (non-invasive) methods are used more often (Echols, 2007; Lichtenberger, Lennox & Chavez, 2011).

The Doppler method uses ultrasonic waves to detect and make audible blood flow in an artery distal to the cuff. In birds, the Doppler cuff can be placed on the distal humerus or femur and the Doppler probe on the medial surface of the proximal ulna or tibiotarsus, basilic and metatarsal arteries, respectively (Lichtenberger, 2007b; Echols, 2007). The cuff bladder is inflated to a suprasystemic pressure with cut-off of the Doppler signal. The cuff is deflated with the first sound heard and marked as the systolic pressure. BP under GA varies from 90 to 140mmHg systolic. BP monitoring helps identifying cardiovascular problems and enables immediate corrections of hypotension (less than 90mmHg), hypovolaemia and prevention of cardiovascular collapse and death (Lichtenberger, 2007b). Reduction of arterial pressure leads to multiple organ failure and death if not treated (Echols, 2007). BP should be recorded every 5min, and if it decreases to less than 90mmHg systolic a bolus of crystalloids should be given at 10ml/Kg plus colloids at 5ml/Kg, repeating the procedure until pressure rises above 90mmHg (Lichtenberger, 2007b).

2.2.9.1. Hypertension

Severe hypertension is potentially life-threatening and considering the immediacy of reduction, it can be divided into hypertensive urgency and emergency. Hypertensive emergencies occur when hypertension is associated with organ damage, in this cases, BP must be immediately lowered to avoid further damage. Hypertensive urgencies on the other hand, include cases where there is no evidence of organ damage but the potential for such is elevated and BP can be lowered less rapidly over 24-48h (Lichtenberger et al., 2011a). Some authors (Bowles et al., 2007 and Lichtenberger et al., 2011a) have suggested that systolic Doppler BP higher than 200mmHg is an indicator of hypertension in birds. Hypertension tends to increase renal perfusion inducing a pressure natriuresis so patients with hypertensive crises are often hypovolaemic (Echols, 2007).

Hypertension is divided in primary and secondary with the former being unknown in birds and the latter due to renal failure, renal tumours, cardiac disease and possibly atherosclerosis. Signs of organ damage may be present in patients with severe hypertension and may include neurological signs (e.g. confusion, seizures), cardiovascular signs (e.g. ventricular hypertrophy, congestive heart failure) or renal system abnormalities (e.g. glomerulonephritis) (Echols, 2007 and Lichtenberger et al., 2011a).

The exact cause of hypertension in obese patients is not clear but possibly results from the effect of several organ impairments. As total body mass increases, blood flow increases to the expanding adipose tissue. Basal metabolic rate (BMR) and O_2 consumption increase stimulating an increase in CO, reduction in SVR and maintenance of a normal BP, however as obesity gets more severe, SVR and BP increase (Lichtenberger et al., 2011a).

Most common drugs used in hypertension include angiotensin converting enzyme (ACE) inhibitors such as benazepril that attenuate angiotensin mediated vasoconstriction and aldosterone release, decrease glomerular efferent arteriolar vasoconstriction, reduce protein loss and inhibit progression of glomerulosclerosis by lowering glomerular filtration pressure, with renoprotective benefits (Echols, 2007). Hypertension has been reported in a parakeet with a renal tumor. The bird was treated with benazepril (0.5mg/Kg, PO, BID) and its systolic BP dropped to 110-130mmHg within 1 week and the bird lived for 3 months, which is considered the average life span for birds with renal tumors (Lichtenberger, 2006 cited by Echols, 2007).

More studies are needed to evaluate anti-hypertensive drug usage but for now these are rarely described in birds. Ca channel blockers antagonise preglomerular vasoconstriction which theoretically shouldn't reduce glomerular hypertension, however, they appear to have additional renoprotective properties and may prevent renal injury (Echols, 2007).

2.2.10. Cardiopulmonary-cerebral resuscitation (CPCR)

The goal of CPCR is restoration of spontaneous circulation. Basic life support consists of the ABC approach (Airway, Breathing, Circulation). A crash cart should be readibly available with adequate supplies. Palpable pulses are not indicative of good organ perfusion during CPCR since they don´t relate to an adequate blood flow; in fact, end-tidal CO_2 and blood gas measurements provide more accurate assessment of organ perfusion. The excreted CO_2 in exhaled gas is a function of pulmonary blood flow, thus the level of CO_2 in exhaled gas changes in direct proportion to changes in CO (Lichtenberger, 2007b). If during CPCR there is an increase in end-tidal CO_2 not greater than 10mmHg after a resuscitation time of 15-20min, there is probably a good outcome. Monitoring venous blood gases is preferable to arterial blood gases, since the first represent oxygenation and acid-base status of the peripheral tissues. Arterial blood indicates respiratory alkalosis while venous blood shows metabolic acidosis during CPCR (Marino, 1997 cited by Lichtenberger, 2007b).

Coma persistent for more than 4h after CPCR carries a poor neurologic prognosis (Lichtenberger, 2007b). The brainstem reflex with the best predictive value in patients unconscious after CPCR is the pupillary light reflex and its absence for 1 or more days of coma indicates little to no chance for neurologic recovery. Care should be taken when assessing pupillary size after the administration of atropine or epinephrine for these drugs can produce mydriasis with no effect on the pupill response to light (Curro, 1998 cited by Lichtenberger, 2007b).

In birds, the prognosis for respiratory arrest caused by isoflurane overdose is good, however, cardiac arrest carries poor prognosis because it is impossible to perform direct compression of the heart due to the overlying sternum, and since birds don´t have a diaphragm, compressions are not effective to increase overall negative intrathoracic pressure (Lichtenberger, 2007b).

Birds under anaesthesia should be monitored using ECG and Doppler to determine rhythm and pulse quality, respectively. If cardiopulmonary arrest occurs, GA should be stopped immediately (Lichtenberger, 2007b). In a masked bird, intubation is warranted and one should start positive-pressure ventilation with 100% O_2 or place an air sac tube (Matos & Morrisey, 2005). Doxapram (0.2ml large bird; 0.1ml small bird, IM) should be administered to stimulate the respiratory center. It is common for birds to become bradycardic before cardiac arrest. Atropine is used to increase the HR (vagolytic effect). Epinephrine (0.01mg/Kg 1:10.000 solution = 0.1mg/ml) and atropine (0.02mg/Kg) can be given IV, IO or endotracheally (Lichtenberger, 2007b). Intracardiac injections are no longer advised because of the risk of lacerating coronary vessels (Costello, 2004 cited by Lichtenberger, 2007b). Fluids should be administered to hypovolaemic patients but avoided in euvolaemic patients (Lichtenberger, 2007b) (see annexes III and IV).

2.3. Pain management

Pain perception in birds is believed to be analogous to that of mammals, thus invasive and painful procedures should always be accompanied by appropriate analgesia (Lichtenberger & Ko, 2007). There are ongoing studies about opioid receptors in the brain and spinal cord of birds. It is believed that psittacines have primarily κ-receptors (Clyde & Paul-Murphy, 1999 cited by Lichtenberger & Ko, 2007), so, κ-receptor agonists (e.g. butorphanol), are used for avian analgesia. Behavioural changes in the bird´s posture, temperament or a decline in feeding should be assumed as pain (Machin, 2005 and Lichtenberger & Ko, 2007). Pain control involves drug administration but also physical, environmental and behavioural management. Reduction of fear and anxiety with anxiolytics, tranquilisers and muscle relaxants can also reduce muscle tension and CNS activity, decreasing pain perception. Nociceptive (pain) stimulation reaching the spinal cord, produces a state of neuron hyperexcitability known as central sensitisation. It is assumed that administering analgesics before surgery reduces pain magnitude experienced by an animal as a result of tissue

damage. A balanced approach to anaesthesia and treating postsurgical or injury pain is not only ethical but also promotes healing, reduces hospitalisation time and provides greater client satisfaction (Manchin, 2005).

Clinical studies demonstrate the benefits associated with the use of opioids, NSAIDs, α_2-agonists, ketamine and local anaesthetics. There is very few information in the scientific literature about pain and its treatment in birds, so knowledge on this area is limited. Butorphanol (1-3mg/Kg) is the most studied analgesic for use in birds being considered safe and effective (Machin, 2005 and Lichtenberger et al., 2011a).

Most birds are frequently only anaesthetised with inhaled anaesthetics, commonly isoflurane or sevoflurane. During anaesthesia, the CNS is depressed preventing perception of pain, but this depression does not provide analgesia (Manchin, 2005).

All inhaled anaesthetics can produce extreme sensitivity to pain (hyperalgesia) at very low concentrations (i.e. concentrations that would be obtained at some point during recovery from anaesthesia) by enhancing C-fiber (unmyelinated pain fiber) activity. The sometimes violent recoveries from inhalant anaesthesia in birds may be due in part to hyperalgesia produced by low concentrations of inhaled anaesthetics so appropriate perioperative analgesia may improve recovery in avian species (Machin, 2005).

An effective pain management plan should include drugs of different classes, each acting at a different step of the pathway, this is known as multimodal analgesia, which allows smaller doses of each drug to be used, because of synergistic or additive effects, reducing undesirable side effects from larger doses of individual drugs (Lichtenberger & Ko, 2007).

2.3.1. Opioids

In veterinary medicine, the most common opioids are fentanyl, hydromorphone, morphine, buprenorphine and butorphanol. Response to these varies with individual variability, breed, species and source of pain (Lichtenberger & Ko, 2007).

Unique distribution of opioid receptors in birds may be responsible for the differences in response (Wilson, 2005). In chickens, μ and κ opioid agonists decrease isoflurane requirement in a dose-dependent manner, however care should be taken when combining opioids with isoflurane because of the potential for respiratory depression (Machin, 2005). Lichtenberger et al. (2011b) demonstrated that butorphanol given by CRI effectively reduced isoflurane minimum anaesthetic concentration (MAC) in Umbrella cockatoos at a dosage of 3mg/Kg/h significantly reducing the response to painful stimuli. There are obviously, interspecific differences of the doses required for analgesia, related to differences in opioid receptor populations within the CNS and spinal cord, explaining why do not all birds respond to μ-agonists such as morphine, fentanyl and buprenorphine in the same manner as mammals.

Side effects (e.g. sedation, respiratory depression) can be reversed with naloxone or naltrexone which also terminates analgesia (Machin, 2005). Butorphanol may have delayed pathogenic effects on eagles and owls (Heatley, 2001 cited by Graham & Heatley, 2007). Buprenorphine has no analgesic effect on AGPs, Cockatoos or Amazons (Wilson, 2005).
A recent study involving pigeons showed that patients given tramadol had more resistance to pain than patients given butorphanol, demonstrating tramadol (2mg/Kg, IM) efficiency for pain management and a choice for pre-medication in pigeons, although more research needs to be done (Almeida & Henriques, 2011).
Intraoperative administration (20min before ending GA) of NSAIDS, such as meloxicam may decrease the tissue sensitization that occurs from surgical trauma and the dose of opioids required. They can be administered for pain relief (0.25-0.5mg/Kg, IM) if perfusion and kidney parameters are normal. NSAIDs are rarely used before long surgical procedures due to the risk of intraoperative bleeding and hypotension (Lichtenberger & Ko, 2007 and Lichtenberger et al., 2011a).

2.3.2. Local analgesia

Should be used with great care since birds are more sensitive than mammals to local anaesthetics. Lower doses in birds (2.7-3.3mg/Kg) produce toxic effects compared with higher doses (3.5-4.5mg/Kg) in dogs. The dosage of lidocaine should never exceed 4mg/Kg in birds, and lower dosages (not greater than 1-2mg/Kg) are recommended while dosages of bupivacaine should not exceed 2mg/Kg (Machin, 2005).
Local anaesthetic toxicity may be associated with ataxia, seizures, cardiac arrest, nystagmus, muscle tremors and hypotension (Machin, 2005). Lidocaine and bupivacaine can be safely/effectively used for local analgesia at 1mg/Kg of each, combined into a syringe and diluted with saline to the desired volume. Local analgesia can be used for incisional and regional/local blocks prior to surgeries or to facilitate painful procedures such as the placement of an IO catheter (Lichtenberger & Ko, 2007 and Lichtenberger et al., 2011a). The effects of lidocaine are not immediate so at least 10min should pass between its administration and the procedure. Pain upon injection can be reduced by warming the solution and/or adding 0.01ml sodium bicarbonate (Lichtenberger et al., 2011a).

2.4. Anaesthesia

Anaesthesia is defined by the American Society of Anesthesiologists (ASA) as a pharmacologically induced reversible state of amnesia, analgesia, loss of responsiveness and loss of skeletal muscle reflexes (Lennox, 2011).
There is no completely safe anaesthetic method for birds, only careful anaesthetists and there is always a risk whatever the species of bird, so an anaesthetic consent form should always be signed before any procedures are performed (Yaakov et al., 2006 cited by Coles, 2007). One of the most difficult situations that avian veterinarians face is the prospect of

placing their patients under GA and in the case of an emergency avian patient this is even more complicated (Mitchell & Tuly Jr., 2009) because these patients require extra care when anaesthetised (Longley, 2008). Problems encountered in avian anaesthesia include apnoea, hypoventilation, hypothermia and regurgitation (Edling, 2006 cited by Longley, 2008). Careful assessment and stabilisation of the patient, selection of anaesthetic protocol and attentive monitoring of the patient will reduce the likelihood of these problems occurring (Longley, 2008).

The avian patient must be assessed for its ability to withstand GA and the procedures that will take place under this form of anaesthesia. If the bird is not able to withstand the anaesthetic protocol, then it must be monitored for any adverse conditions, treated, and stabilized before going under GA. A “crash kit” should be assembled to manage emergency situations (Mitchell & Tuly Jr., 2009). Time is crucial when dealing with avian anaesthetic emergencies as respiratory arrest is often rapidly followed by cardiac arrest, so the patient will have a greater chance of survival if emergency equipment and drugs are close at hand should the need arise. If a patient is unwell prior to anaesthesia or a major procedure is to be performed, it is advisable to calculate emergency drug doses beforehand (Longley, 2008). If the patient is dehydrated or if there is a concern regarding hypoglycaemia during the procedure, IV or IO fluid therapy should be administered. Once it has been determined that the patient is in good enough condition to endure the procedure, induction should be performed (Mitchell & Tully Jr., 2009).

2.4.1. Induction and maintenance of anaesthesia

Induction can be done with volatile or injectable agents followed by intubation (Longley, 2008). Some authors (Lichtenberger et al., 2011a) have used metomidate by slow IV injection for induction in extremely critical patients where inhalant agents are considered excessively risky. Inhalant agents (e.g. isoflurane, sevoflurane) are commonly used. Injectable agents such as ketamine have been used but don´t provide ideal planes of anaesthesia or recoveries so are not advised for emergency and critical care patients (Lichtenberger et al., 2011a).

In the case of volatile agents, the patient should be preoxygenated prior to induction. After a few breaths in 100% O_2, via a facemask or induction chamber, the anaesthetic agent is switched to a high concentration. This usually results in rapid induction of anaesthesia with minimal stress for the patient (Edling, 2006 cited by Longley, 2008). If a gradual increase in concentration of anaesthetic agent is used, induction is slower and the patient may become stressed due to the prolonged restraint so this should be avoided (Edling, 2005 cited by Longley, 2008).

When the procedure takes less than 10 minutes or is non-invasive (e.g. blood collection, radiography), intubation is not necessary, but in other cases its crucial (Lichtenberger & Ko,

2007). Intubation allows the clinician to have more control over the bird's respiratory system, with airway patency assured and allowing ventilation to be peformed. There is also less risk of waste gas escaping into the environment compared to when using facemasks (Longley, 2008). It must be remembered that birds have complete tracheal rings so cuffed ET tubes should be avoided (Bennett, 2008).

Inhalant anaesthetic agents induce vasodilation leading to hypotension to which immediate attention is required. Anaesthetic depth should be assessed and anaesthetic dose reduced. If hypotension is caused by bradycardia, atropine should be administered. If the anaesthetic concentration cannot be reduced, a crystalloid bolus with a colloid should be administered, and repeated until systolic BP is greater than 90mmHg. The PCV and blood glucose concentration should also be adressed because hypoglycaemia and anaemia can lead to refractory hypotension and should be corrected (Lichtenberger & Ko, 2007).

2.4.2. Anaesthetic agents

2.4.2.1. Inhaled anaesthetics

Inhalant anaesthesia its currently the method of choice for almost all procedures involving birds requiring GA (Bennett, 2008). It allows rapid alterations of anaesthetic depth, rapid induction and recovery, and have fewer side effects that with an injectable route (Muir & Hubbell, 2000 cited by Longley, 2008). Non-rebreathing systems work well on birds with a recommended gas flow rate of at least 660ml/Kg/min, with rates of 1-3L/min (Bennett, 2008).

As birds do not have an alveolar lung, the term "minimum alveolar concentration" (MAC) is not appropriate. MAC in birds is, therefore, defined as the "minimum anaesthetic concentration" required to keep a bird from purposeful movement to a painful stimulus (Ludders et al., 1989 cited by Longley, 2008). MAC values will vary slightly between bird species, for example the MAC for isoflurane is 1.44% in cockatoos and 1.32% in ducks (Ludders & Mathews, 1996 and Ludders et al., 1990 cited by Longley, 2008).

All inhalants potentially increase intracranial pressure and produce dose-dependent decreases in cardiac performance. For hypovolaemic patients, lower concentrations are used to avoid significant cardiovascular destabilisation. Volatile anaesthetic agents may depress the $PaCO_2$ chemoreceptors, reducing the respiratory rate in the anaesthetised patient (Coles, 1997 cited by Longley, 2008). Respiration is therefore usually assisted either manually or using a mechanical ventilator to prevent hypercapnia (Forbes, 1999 cited by Longley, 2008).

The most common inhalant agents used for avian anaesthesia are halothane, isoflurane and sevoflurane (Lichtenberger & Ko, 2007). Isoflurane is the anaesthetic of choice, almost entirely excreted by the respiratory system with minimal systemic effects (Bennett, 2008), being preferred to sevoflurane if cost is an issue (Lichtenberger & Ko, 2007). Induction is quick (1-2min) at a concentration of 3-5% and maintenance 1.5-2%. Recovery is fast and

there seems to be a direct relationship between total anaesthetic time and recovery time. Most patients are standing safe in their cage within 5 minutes after anaesthesia is discontinued. HR may decrease but not as much as it does with halothane (Bennett, 2008). Respiratory depression may still be seen with isoflurane, in part due to the muscle relaxation caused by this agent (Straub et al., 2003 cited by Longley, 2008). Only 0.3% of isoflurane is metabolised in the liver, therefore, it is much safer for patients with hepatic dysfunction and for veterinary staff who may be exposed to leaked gases (Coles, 1997 cited by Longley, 2008).

Sevoflurane is less soluble and less potent than isoflurane (Longley, 2008). Lower solubility and MAC result in shorter induction and recovery time compared to isoflurane (Greenacre & Quandt, 1997 cited by Longley, 2008), so if a critical patient requires anaesthesia or a prolonged anaesthetic is envisaged, sevoflurane is considered a better option than other agents as the recovery will be faster and the birds are less ataxic after sevoflurane anaesthesia compared with isoflurane (Longley, 2008). Sevoflurane does not cause respiratory tract irritation and mask induction is, therefore, less stressful than with isoflurane (Edling, 2006 cited by Longley, 2008). A disadvantage is the higher current cost of sevoflurane over other agents. Sevoflurane also depresses plasma ionised Ca levels (Edling, 2005 and Stanford, 2003 cited by Longley, 2008).

Lower concentrations of volatile anaesthetic should be used in compromised patients, as they will be more susceptible to adverse effects. If sevoflurane is used, 4-5% should be administered to ill patients rather than the 8% used in 'healthy' birds (Helmer, 2004 cited by Longley, 2008). Neither drug provides analgesic properties during or after termination of anaesthesia (Lichtenberger & Ko, 2007).

2.4.2.2. Injectable anaesthetics

Injectable anaesthetics require little equipment, being convenient for short procedures but there are several problems associated with their use, such as the risk of overdosage. The weight of the patient should be accurately determined so appropriate doses are administered. Small volumes of drug are difficult to measure so dilution of the agent with sterile water for injection or saline and the use of insulin syringes may improve the accuracy of drug measurements (Longley, 2008).

One of the problems with this route is the difficulty to control depth and duration of anaesthesia once administered, so it is not recommended for long procedures or debilitated patients. Other problems include species and individual variability in response to agents, cardio-respiratory depression, slow induction and prolonged and/or traumatic recoveries (Ludders & Mathews, 1996 cited by Longley, 2008) so inhalational agents are much more reliable and safer to use. If isoflurane is available, inhalant anaesthesia is prefered for even short procedures (Bennett, 2008).

There are some exceptions where injectables may be used, such as in conjunction with

volatile anaesthetics, or where sedation or anaesthesia is required outwith the practice premises where equipment is stationed. Injectable drugs may be cheaper than volatile agents, so if cost is an issue they can also be used. Some agents are used in combination protocols to reduce the concentrations of volatile agent required, for example, analgesic agents that also produce sedation. In waterfowl injectable agents can be useful in induction, because of their breath holding "dive response", followed by intubation and maintenance of anaesthesia with a volatile agent (Longley, 2008).

Ketamine can be used in combination with other drugs. This agent will produce chemical restraint and moderate analgesia, but is unsuitable for major surgical procedures when used alone (Ludders et al., 1989 cited by Longley, 2008). The dose varies greatly between species, with higher doses required in smaller birds (Longley, 2008). Ketamine is usually administered IM, with anaesthesia occurring within 3–5min and persisting for approximately 35 minutes (Longley, 2008). Administration should be slow and incremental, as a rapid overdose may cause apnoea and/or cardiac arrest. When used in combination with other agents, repeating the ketamine dose will extend the period of anaesthesia and prolong the recovery period, which is not advised for emergency patients (Edling, 2005 cited by Longley, 2008). Ketamine may be given IM with medetomidine to induce anaesthesia in several species (Jalanka, 1991; Reither, 1993 and Scrollavezza et al., 1995 cited by Longley, 2008) at a dose of 25mg/Kg ketamine + 100 µg/Kg medetomidine for psittacine species (Carpenter, 2005). Medetomidine causes sedation, muscle relaxation, analgesia and has a wide safety margin. Side effects include bradycardia and peripheral vasoconstriction. Induction of anaesthesia occurs in 2–3min and anaesthesia can be maintained with low doses of volatile anaesthetic agents (Coles, 1997 cited by Longley, 2008).

2.4.3. Monitoring

The principles of GA for exotic species are the same as for dogs and cats: tidal volumes, O_2 requirements, CO_2 elimination, RR and body temperature must all be maintained under GA. Monitoring SpO_2 can be a challege in birds being capnography a reliable mean of monitoring these animals, measuring expired CO_2 levels (Simpson, nd and Longley, 2008). RR and character are the most reliable indicators of anaesthesia depth. Respiration becomes slower and shallower as the plane of anaesthesia deepens. Since apnoea results in death quickly, immediate measures should be taken to lighten anaesthesia and provide ventilation should the bird stop breathing on its own. HR should be monitored with a stethoscope or ECG monitor (Bennett, 2008).

Several reflexes can be used to monitor anaesthesia depth in birds. The palpebral reflex is present in very light planes, as is reflex movement in response to cere stimulation. The corneal reflex is the best to evaluate (i.e. movement of the 3rd eyelid in response to corneal stimulation) and the pedal reflex (i.e. withdrawal of the foot in response to toe stimulation)

remain longer and are slow but present at a surgical plane of anaesthesia (Flecknell, 1996 cited by Longley, 2008).

The most common problem in anaesthetised birds is hypothermia, since the high surface to volume ratio predisposes heat loss if not moving, therefore, birds should be kept warm during the procedure and recovery period avoiding alcohol in surgical preparation (Bennett, 2008).

Once the patient is asleep, the clinician may or may not choose to start with intermittent positive pressure ventilation (IPPV). Often IPPV is not used because of the extra effort required but the advantages (i.e. O_2 and anaesthetic agent adequate delivery and waste gases, particularly CO_2, reliably eliminated) usually outweigh such disadvantages. IPPV can be done manually but to avoid over/under-inflation an appropriate ventilator can be used. In order to use IPPV the bird must be intubated (Simpson, nd).

Parameters that should be routinely checked include HR, chest movements/RR, response to noxious stimuli and body temperature (Longley, 2008). With IPPV respiration is controlled so the only warning sign is HR. A Doppler monitor can aid in following pulse rate (Bennett, 2008). Variations in HR may indicate lightening/deepening of anaesthesia or pain response (Longley, 2008). The parameters mentioned above are not reliable guides for the adequacy of ventilation, therefore, additional monitoring techniques may need to be used, such as end tidal CO_2 and O_2 saturation, monitored using a capnograph and a pulse-oximeter, respectively. A capnograph can be used by means of a sidestream device with the advantage that the patient can still be monitored after extubation or with a facemask.

When using a capnograph, care must be taken to ensure the animal is not over-ventilated (falling End-Tidal CO_2 value) or under-ventilated (rising End-Tidal CO_2). End-Tidal values should be kept between 3.0-5.0% (23-38mmHg) to avoid respiratory alkalosis and acidosis respectively (Simpson, nd).

Monitoring devices that can be used to collect baseline data include a Doppler unit to monitor heart rate and strength of the heart beat, a respiratory monitor, an esophageal thermometer, and an ECG, if available. There are other monitoring units that provide additional vital information, but the basic monitoring items mentioned at the beginning of this paragraph are essential in most, if not all cases. Perivascular access should be achieved using the IV route (e.g., tarsometatarsal vein, jugular vein, or basilic vein) or IO route (e.g. distal ulna or proximal tibiotarsal bone) (Mitchell & Tully Jr., 2009)

2.4.4. Air sac perfusion anaesthesia (APA)

APA is used for oxygenation (e.g. dyspnoeic birds) and GA in cases where tracheal intubation would be difficult (e.g. tracheal trauma, collapse, obstruction) (Longley, 2008). It is conducted by retrograde perfusion of the lung-air sac system through a tube introduced into the left caudal thoracic air sac. The required puncture of the air sac is performed under conventional head chamber inhalation anaesthesia.

When changing to administration of gas via APA, the carrier gas-perfusion volume is reduced to 0.3 l/min/Kg BW to ensure a virtually physiological blood pH. Higher perfusion volumes will increase CO_2 washout causing hypocapnic alkalosis associated with cardiac arrythmias. After about 8-15s APA induces APA-specific reversible apnoea which is attributable to perfusion-induced, subphysiological $PaCO_2$ with loss of stimulation of the respiratory centre (Korbel, 1999). The isoflurane concentration is slightly higher than with conventional head chamber administration (Longley, 2008). APA is ended by interrupting the flow of isoflurane and nitrous oxide but the bird is perfused with pure O_2 for a further minutes to shorten the recovery phase by rapidly washing isoflurane out of the air sac system and preventing nitrous oxide diffusion hypoxia. Generally, spontaneous respiration starts 3-3.5 minutes after ending O_2 perfusion once physiological CO_2 partial pressure is restored and stimulates the respiratory centre (Korbel, 1999).

2.4.5. Recovery from anaesthesia

The bird shouldn´t be disconnected from the O_2 supply before there are signs of recovery with slight jaw movement. Soon movement of the wings and legs can be detected and the ET tube should be removed when movement of the beak takes place. During the whole recovery period the bird should be gently held upright, wrapped in a towel to prevent self-trauma (Coles, 2007 and Graham & Heatley, 2007). Because of their high metabolic rates, birds must recover and start eating quickly. Environment should be kept warm, with adequate heat sources, to prevent hypothermia (Longley, 2008).

2.5. Sedation

Sedation is a "drug induced depression of consciousness during which patients cannot be easily aroused, but respond purposefully following repeated or painful stimulation" (ASA, 2005 cited by Lennox, 2011).

The advantages of sedation are mostly related with patient safety (Lennox, 2011). Sedation could replace GA in cases where the latter is considered especially risky, such as emergency and critical care patients. Sedation can aid in physical restraint, reduce stress in hospitalised patients and even reduce patient memory of unpleasant procedures (Broadbelt, 2008 cited by Lennox, 2011). Disadvantages of sedation include incomplete elimination of movement, semi-awareness, lack of complete analgesia and drug related risks (Lennox, 2011).

The most common drugs used for sedation are midazolam and butorphanol. Butorphanol is an opioid analgesic actually considered the most useful agent of its class to use in avian medicine (Abou-Madi, 2001 cited by Lennox, 2011) and recent pharmacokinetics research demonstrated safety in selected psittacine species (Sanchez-Migallon et al., 2008 and Klaphake et al., 2006 cited by Lennox, 2011). Midazolam is a benzodiazepine sedative without analgesic effects, currently used as an alternative to GA in human medicine. It reduces anxiety and it has been determined to produce amnesia in humans and some

laboratory species (Reznick et al., 2000 and Isotobi et al., 2009 cited by Lennox, 2011). Lennox (2011) claims to use butorphanol (1-3mg/Kg) combined with midazolam (0.25-0.5mg/Kg) for sedation in avian patients with a variable response, ranging from profound to barely perceptible sedation.

The level of sedation is mainly affected by patient condition and demeanor with more profound effects seen in ill or calm birds, so dose modification is based on degree of debilitation. There were no recorded deaths directly associated with sedation, even for severely debilitated patients and although more research needs to be done, it can be assumed that the use of sedation does not appear to be linked with increased mortality. Some birds with respiratory distress display improved breathing patterns under GA that could be linked to the provision of O_2, however, sedated birds in respiratory distress also improve without O_2 delivery, suggesting the benefit is more likely to be caused from anxiety reduction (Lennox, 2011).

For diagnostic procedures, patient movement is obviously more reduced with GA than with sedation, however, calm handling and patience can result in production of high quality radiographs for patients in which GA is too risky (Lennox, 2011).

2.6. Nutrition

Critical patients suffering from anorexia, maldigestion and weight loss are in need of nutritional support. If possible, enteral feeding is prefered to parenteral feeding since the former decreases intestinal cellular death and subsequent bacterial translocation leading to sepsis (Lichtenberger, 2005 and Wilson, 2005). While mammals use carbohydrates as the immediate energy source, birds use lipids and unless being fasted for surgery, the hospitalised bird should always have food available (Wilson, 2005).

Nutritional requirements are based on age, species and underlying problems (Lichtenberger, 2005). As most pet birds are diurnal, eating may be stimulated by light so lights may be left on through the night for ill patients (Echols, 2007). If the bird is not eating at all, forced feeding may be necessary (Wilson, 2005 and Graham & Heatley, 2007). Birds can be nutritionally supplemented in many ways: by hand, syringe/tube feeding, IV parenteral nutrition or surgically placed feeding tubes. Recording the body weight daily until the bird is self feeding is paramount and then, at least weekly until clinically normal. Daily weighing and pectoral musculature evaluation are good indicators of the caloric status. Care should be taken on birds that are gaining weight while hospitalised but are not eliminating wastes for these birds often have GI stasis, renal failure or other conditions that may require intensive care (Echols, 2007).

Persistently regurgitating birds or birds with beak, oesophageal or crop injuries may need to have an oesophagostomy tube placed into the proventriculus until eating on its own (Lichtenberger, 2005).

All diets should be easy to administer, highly digestible and sufficiently energetic (Harris, 2003). The species natural diet (e.g. granivores, carnivores, herbivores) should always be kept in mind (Wilson, 2005). Calculation of enteral feeding requirements for birds should be based on the following equations (Sedgewick, 1988 cited by Wilson, 2005):

BMR (basal metabolic rate) (kcal/day) = $kW^{0.75}$
where k= Kcal/Kg constant (passerines = 129; non-passerines = 78) and W=weight in Kg.
and
MER (maintenance energy requirement) (kcal/day) = 1.5xBMR

In birds, the MER can then be adjusted for health status as follows (Quesenberry & Hillyer, 1994 cited by Tully Jr. et al., 2000): starvation (0.5-0.7); trauma (1.0-1.2); sepsis (1.2-1.5, most critical care patients are in this range); burns (1.5-2.0). If the energy content of the feeding formula is known, the daily caloric needs are divided by the calories per ml of formula to calculate the total volume of formula needed daily (Ritchie et al., 1994).

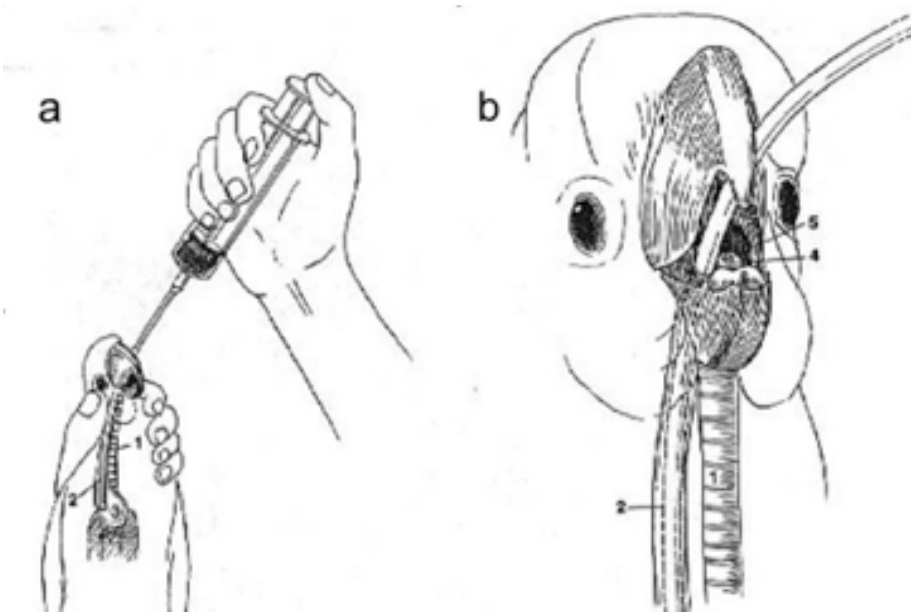
2.6.1. Gavage

Gavage is the most common method to provide nutrition to anorexic, non-regurgitating birds (Echols, 2007). If the bird is eating on its own, familiar foods should be offered and gavage reserved for supplementing calories (Johnson-Delaney, 2008). Many commercial formulas are available but a homemade formula can be made or hand-feeding formulas for neonates can be used (Lichtenberger, 2005). Formulas should be pre-warmed (38.3-40ºC) to promote normal digestion and decrease the incidence of crop stasis (Echols, 2007). One should begin with ½ the suggested volume and volumes increased as needed (Johnson-Delaney, 2008). The crop must be empty and other procedures should be performed before tube feeding to prevent regurgitation and aspiration (Hess, 2002 and Lichtenberger, 2005). Approximately 3-5% BW can be gavage fed at one time and 7-10% to birds that have not fledged (these have greater crop capacity) (Wilson, 2005).

Feeding should be done 3-4 times a day (Lichtenbeger, 2005). Approximate doses for gavaging birds are: budgerigar (0,5-1.0ml); cockatiel (1.0-2.5ml); amazon (5-10ml); macaw (10-20ml). Nutritional support cannot be overlooked because high metabolic avian patients will suffer negative nitrogen and calorific balance within 2-24h depending upon body size and diet (i.e. a 80g cockatiel shouldn't go more than 3-4h without eating, while an 800g hawk can go 24h) (Johnson-Delaney, 2008). A curved metal feeding needle with a balled tip is recommended. Rubber feeding tubes can also be used in non-psittacine species, ensuring the bird doesn´t bite and swallows the end of the tube (Fig. 3) (Lichtenberger, 2005 and Johnson-Delaney, 2008). If the formula backs up, one should stop, re-aspirate the excess and the bird let to rest. If the bird continues to vomit or regurgitate only warmed fluids should

be given and motility modifier medication considered (Johnson-Delaney, 2008). CS of aspiration include coughing, choking and gagging. Hydration should always be corrected prior to gavage feeding (Echols, 2007).

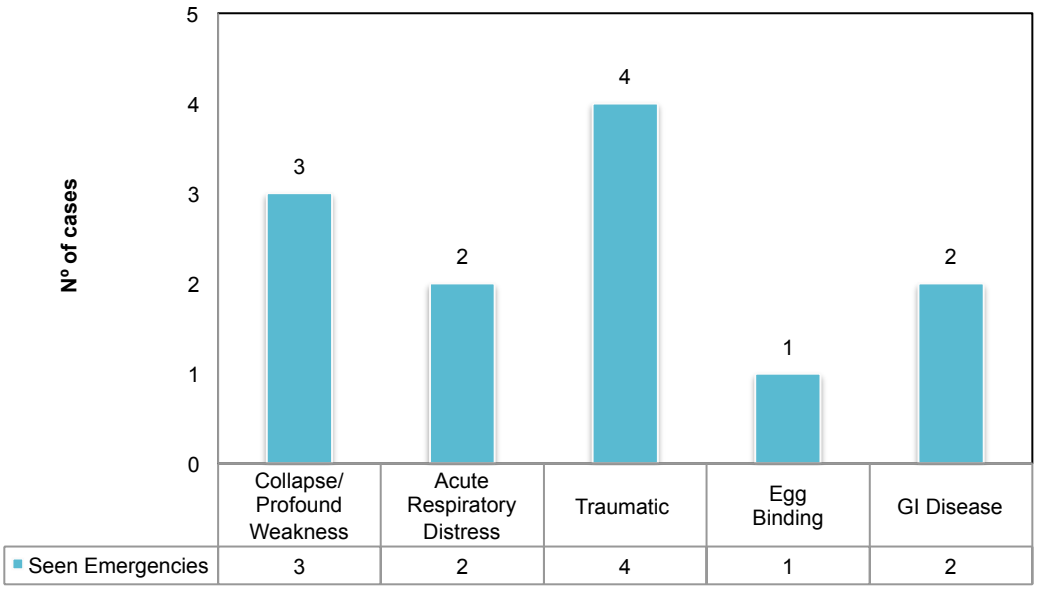
Figure 3: Tube-feeding. The bird is held in an upright position with the neck extended (a) and the tube passed through the left side of the oral cavity down to the oesophagus and into the right side of the pharyngeal cavity (b). 1)trachea 2)oesophagus 3)crop 4)laryngeal mound 5)Rima glottis (Adapted from Ritchie et al., 1994).



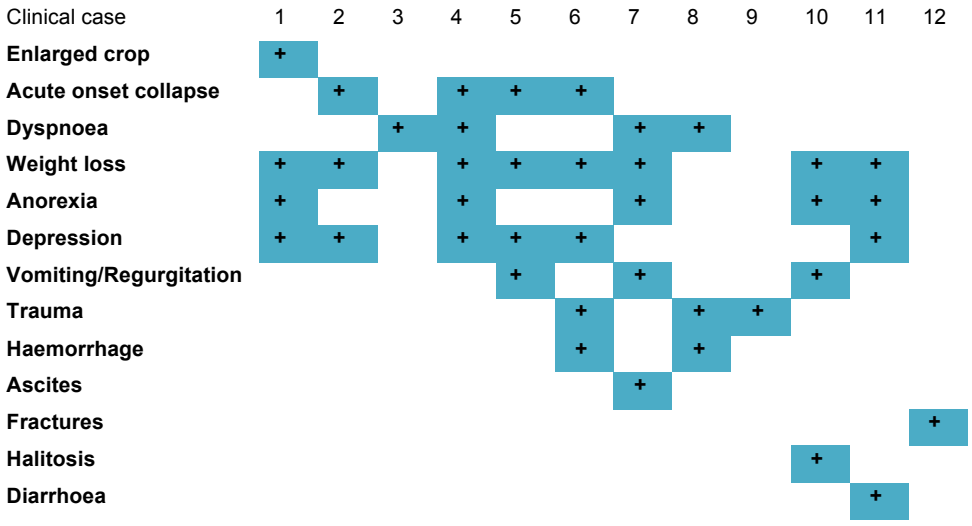
Chapter 3. Clinical Cases

A total of 12 avian clinical cases are presented, from which 7 birds are still alive and 5 birds died. Causes of initial presentation included collapse/profound weakness, acute respiratory distress, traumatic presentations, egg binding and GI disease as shown in graphic 4. CS included dyspnoea, enlarged crop, weight loss, anorexia, depression, vomiting/regurgitation, diarrhoea, trauma, haemorrhage, ascites, fractures, halitosis and acute onset collapse (Graphic 5 and 6).

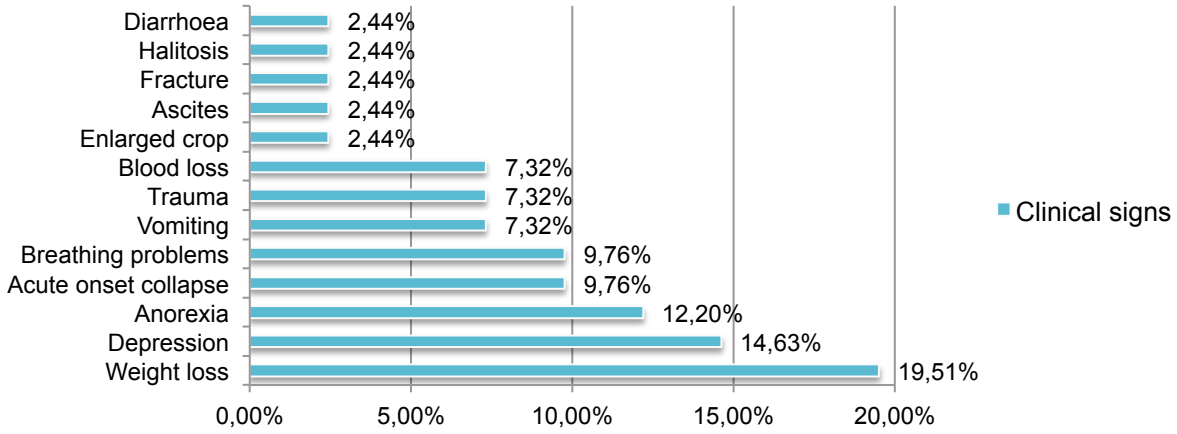
Graphic 4: Emergency presentations seen at practice



Graphic 5: Clinical signs observed



Graphic 6: Percentage of the different CS observed



Clinical case No.1

History and clinical signs

An 1 year old female chicken (*Gallus gallus domesticus*) was examined at GWR in October 2010. On palpation an enlarged crop was detected with what felt like some grass-like material inside it forming a bolus on the crop leading to the impaction.

Diagnosis and treatment

An ET tube was placed and with the bird anaesthetized in lateral recumbency, a probe was placed orally into the crop to elevate and delineate the position of the crop wall. The overlying skin was incised and the crop wall localized and isolated, prior to incision. An ingluviotomy was then performed and the crop emptied, flushed and closed with 2 layers of continuous inversion sutures and a separated skin closure (Fig. 4 and 5) . A "crop bra" was applied to provide bandage support for the healing crop. The bird was sent home on clavulanate-potentiated amoxycillin (*Synulox*® Bolus 500mg film-coated tablet**,** Pfizer, Amboise, France) at the dose of 500mg/l of drinking water for 10 days. After 2 weeks the chicken returned to remove the bandage and was healing nicely. There was less fluid felt on palpation of the crop. It was advised to rebandage the bird leaving the bandage for as long as the chicken was not pulling it off.

Figure 4: A) The anaesthetized bird in lateral recumbency under GA, positioning and surgical preparation of the site for ingluviotomy, note the enlarged crop (yellow circle) ; B) Emptying the crop (Original picture).

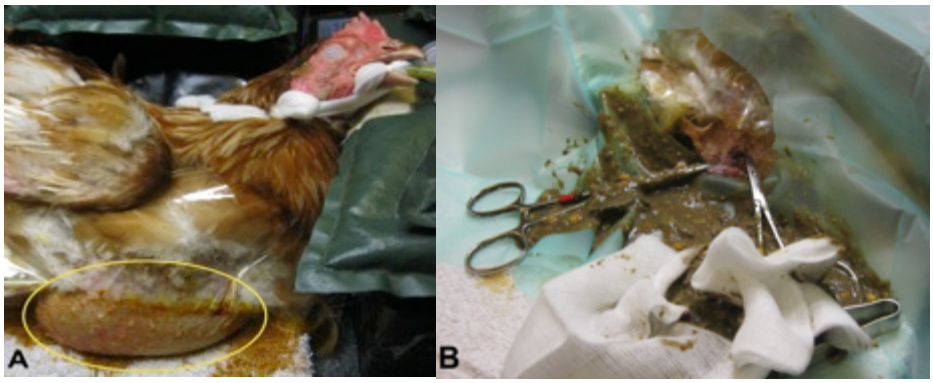
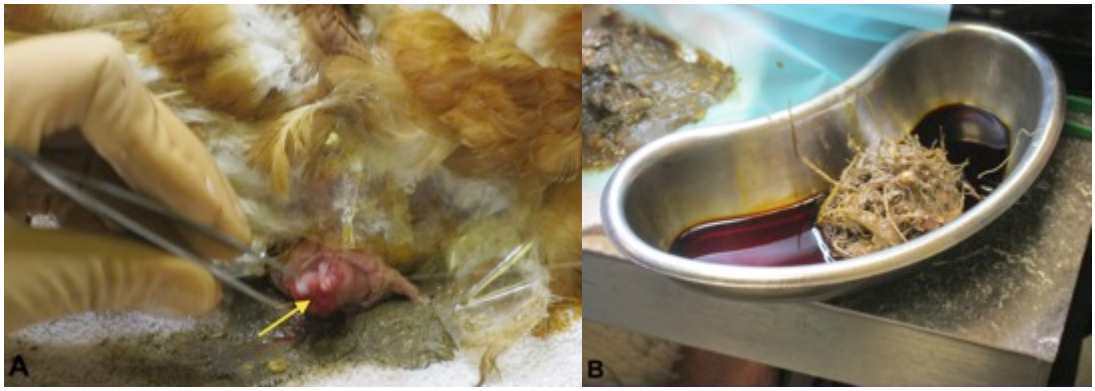


Figure 5: A) Suturing the crop wall (arrow); B) Material removed from the crop (Original picture).



Discussion

In the above mentioned case, crop impaction was present which is caused by hard, fibrous feed or litter, whose accumulation results in impaction. The retained content into the crop can lead to a putrefactive necrotic process, affecting the crop wall and the covering skin (Dinev, 2007). Crop impaction is occasionally seen in Galliformes and Anseriformes that have sudden access to an abundant supply of lush grasses and sprouted grains (MacNeil & Barnard, 1978 cited by Ritchie et al., 1994) and can occur in birds provided *ad libitum* grit. A less agressive treatment would involve softening the impacted material in the crop with warm water followed by massage of the crop. However, an ingluviotomy, as performed in this case, is generally the method of choice for removing impacted material. The crop usually heals without complications as it occurred in this case, although monitoring the chicken and its diet is always advised. It was explained to the owner that if the problem persisted, bandaging would not fix the problem but only give support, so considering welfare issues, euthanasia could be advised in the future if the problem persisted (Ritchie et al., 1994).

Clinical case No.2 (Moby)

History and clinical signs

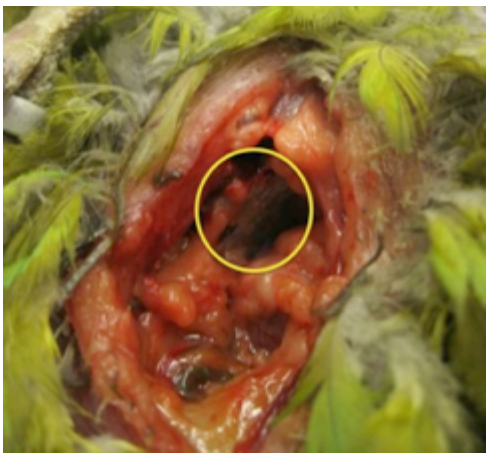
A 9 year old, male, Blue-fronted Amazon (*Amazona aestiva*) was examined at GWR in October 2010. The animal had no previous medical history and presented with signs of acute onset collapse. "Moby" had been apparently nesting over the previous days. The bird was in good body condition so the peracute onset was reasonable.

Diagnosis and treatment

An IV catheter was placed into the basilic vein and a blood sample was taken for haematology and BC. Results showed elevated uric acid and phosphate levels and leukocytosis of $100x10^9$/L (6-$13x10^9$/L) (Fudge, 2000), indicators of acute renal failure.

The diagnosis was bacterial nephritis resulting in acute renal failure with grave prognosis. Treatment plan included fluid therapy with 20% of an injectable solution of electrolytes, vitamins, amino-acids and dextrose (*Duphalyte®,* Fort Dodge Animal Health, Southampton, UK) + 80% Hartmann´s solution, pre warmed and mixed in the same syringe, IV, given at the dose of the estimated fluid deficits (10% BW) in ml plus maintenance (40ml/Kg/day). O_2 therapy was provided via a facemask, antibiotherapy with enrofloxacin (*Baytril*® 2,5% Injectable solution, Bayer Corp., Leverkusen, Germany) at the dose rate of 30mg/Kg, IM and nutritional support by gavaging *Harrisson´s*© Recovery Formula (Harrison's© Bird Foods, Tennessee, USA) at the dose of 5ml/100g BW, BID. "Moby" died 12h after admission. A *post mortem* examination, as required by the owner, was perfomed to determine the cause of death. *Post mortem* revealed an excessively obese bird with fungal colonies observed throughout the air sacs (Fig. 6) typical of a fungal air sacculitis.

Figure 6: Fungal colonies observed on *post-mortem* examination (circle) appearing macroscopically as white granulomatous foci, dry in texture and protruding out of the surface of the air sacs (Original picture).



Discussion

In this case both renal and respiratory systems were involved. Important laboratory findings included leukocytosis, hyperuricemia and elevated phosphorus levels. Uric acid is not a sensitive indicator of renal function and does not elevate until significant renal tubular damage has taken place. Causes of hyperuricemia include hypervitaminosis D, toxic nephropathy, bacterial nephritis and soft tissue gout. In this case, history and CS were suggestive of a bacterial nephritis leading to acute renal failure (ARF) although whole body radiographs, urinalysis, renal biopsy as well as a thorough review of all items ingested by the patient should have been performed for a more accurate diagnosis because uric acid is not a specific test for renal disease so it does not confirm the diagnosis (Fudge, 2000 and Campbell & Grant, 2010). Bacteria can infect the kidneys by ascending infections from the cloaca to the ureter or via the haematogenous route (Schmidt, Reavill & Phalen, 2003). The renal portal system of birds creates the potential for exposure of microbial or toxic agents from the alimentary tract to the kidneys (Lumeij, 2000). *Mycobacterium* and *Chlamydophila* infections can cause pathology of the kidneys but usually cause systemic disease (Schmidt et al., 2003) although there have been reports of *Chlamydophila* infections only affecting the kidneys in psittacines (Shivaprasad et al., 2002 cited by Burgos-Rodríguez, 2010). In a retrospective study, 50% of nephritis cases were associated with bacterial disease (Lumeij, 2000), for this reason antibiotics were prescribed. Amynoglycosides are nephrotoxic and should be avoided if renal disease is suspected (Burgos-Rodríguez, 2010).

Post-mortem examination revealed the presence of fungal colonies throughout the air sacs not affecting the kidneys. Although fungal infection affecting the kidneys can be associated with extension of a fungal air sacculitis (Schmidt et al., 2003) this didn´t seemed to be the case and if renal function is affected with a mycotic infection, the bird usually has CS of severe fungal disease (Phalen, 2000 cited by Burgos-Rodríguez, 2010), including weight loss, labored or open-mouth breathing, voice change or decreased vocalizations. The most common fungal diseases in companion birds are respiratory tract infections caused by *Aspergillus* spp.. Aspergillosis is an opportunistic infection that occurs in compromised hosts or when birds are exposed to large numbers of aerosolised spores. Risk factors include species predilection (Amazon parrots are included), exposure to spores (i.e. mouldy feed can increase spore exposure), immunosuppression, hypovitaminosis A and food aspiration. Marked leukocytosis can occur (Flammer, 2007). The bird was also fat and on a seed diet. Seed based diets are high-fat and low in vitamin A. An association between renal disease, high-fat diets and hypovitaminosis A can be made in psittacine birds. The main cause of death in this case was probably the ARF caused by a bacterial infection while the fungal infection was likely caused by the poor diet/husbandry on a compromised bird. The problems of seed diets were discussed with the owner and how to prevent nutritional deficiencies or aspergillosis by eliminating the seeds from the diet, or reducing the risk by checking the

seeds prior to feeding and getting human grade seed mixes while gradually switching the birds to a pellet-based diet suplemented with fresh fruits and vegetables with 50% of the diet being pellets, 25% fruits and vegetables, 15% nuts and 10% seeds (Burgos-Rodríguez, 2010).

Clinical case No.3

History and clinical signs

A Ferruginous Hawk (*Buteo regalis*) was examined at GWR on October 2010 with breathing problems. The bird was kept in an aviary for the last 12 months, started flying recently and had been very dyspnoeic, even at rest. Food and water intake were normal. On PE crackles were detected on auscultation of the cranial air sacs. A chaotic heart rhythm was suspected on cardiac auscultation but difficult to interpret due to high rate and vocalisations. Many lice were suggestive of poor preening.

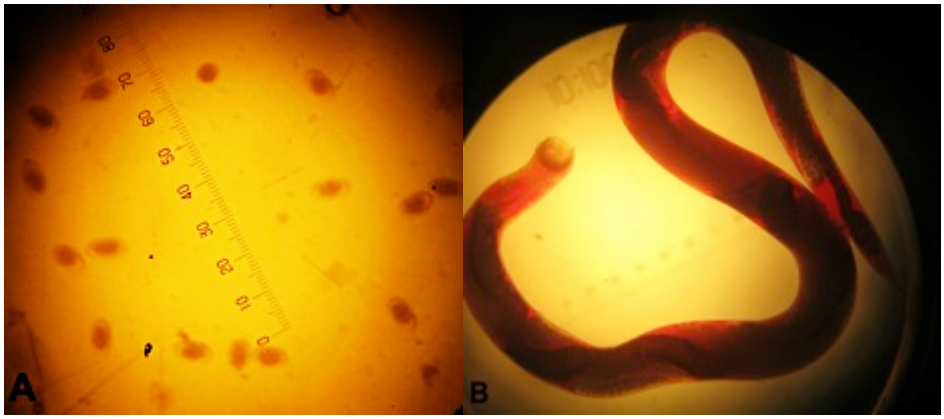
Diagnosis and treatment

On faecal examination small nematode eggs were observed in moderate numbers (Fig. 7A). On radiographs, increased artery opacities suggestive of mild atherosclerosis, a distended proventriculus and pulmonary opacities were detected. On tracheoscopy a single *Syngamus trachea* worm was removed and observed under microscope magnification (Fig. 7B). It wasn´t possible to reach the syrinx so further parasites could be present more distally. Large quantities of mucus were present in the trachea. On ECG, abnormal oscillations of trace when the bird was static on the table were consistent with atrial fibrillation. HR was 205 bpm. Differential diagnosis included *Syngamus* spp. infection, mild atherosclerosis and cardiac (conduction) disease.

Treatment plan included parasite therapy with febendazole (*Panacur*® 10% Oral Suspension, Intervet, Buckinghamshire, UK) at the dose rate of 25mg/Kg, SID, PO for 5 days and marbofloxacin (*Marbocyl*® P 20mg, Vétoquinol, Bicester, UK) at the dose rate of 10mg/Kg, BID, PO for 5 days to adress the tracheitis. Reassessment was scheduled in 2 weeks time to repeat the ECG, faecal wet mount and to perform an endoscopy to assess the lungs if not improving.

The bird presented 2 weeks later slowly improving. Comparing radiographs, in the recent ones there were less opacities detected on the air sacs, suggesting the air sacculitis was decreasing, WBC count was normal and no faecal parasites were found on the wet mount.

Figure 7: A) Nematode eggs after fecal wet mount under microscope magnification (x40); B) *Syngamus trachea* worm under optical microscope magnification (x10) (Original picture).



Discussion

CS from the above mentioned case were likely caused by the *Syngamus* infection with cardiac conductivity changes probably induced by isoflurane, stress or a primary cardiac pathology. *Syngamus trachea* is a common parasite particularly in buzzards and other scavengers. The life cycle of the parasite is either direct or a paratenic host may be present (e.g. snails, earthworms, slugs) (Forbes, 1996b). Passage via earthworms renders the parasite more infective to the main host (Morgan & Clapham, 1934 cited by Forbes, 1996b). After ingestion, the larvae pass via the bloodstream into the lungs within 6 hours and from there they pass into the trachea. Faecal ova may be present 17-21 days after ingestion of the parasite. Common CS include dyspnoea, neck stretching and coughing (Forbes, 1996b). A differential diagnosis should include aspergillosis and mycoplasmosis. Effective medication include the use of the benzimidazole group (Forbes & Altman, 2010), febendazole was used for this case, plus antibiotics. Only endoscopic evaluation, as performed, can reveal the presence of *Syngamus trachea* on the tracheal wall. The owner was advised to beware of possible reinfections from the environment and opt for concrete aviary floors to prevent mollusc and arthropod intermediate hosts (Forbes, 1996b) and slowly reduce the birds weight since atherosclerosis is associated with lipid deposition from high-fat and high-cholesterol diets. Obesity also predisposes for coronary heart disease, myocardial infarctation, heart failure, hypertension (Lichtenberger et al., 2011a) and may have been responsible for the mild atherosclerosis and heart conduction disease. Numerous arrhythmias have been described in birds and some have been associated with anaesthesia, hypothermia, hypoxia and drugs. Restraint of unanaesthetized birds may alter the HR and rhythm (Rosenthal et al., 1997 cited by Rembert, Smith, Strickland & Tully Jr., 2008), in fact, catecholamines sensitize cardiomyocytes to arrhythmias and have been implicated in arrhythmias seen during induction and recovery of bald eagles (*Haliaeetus leucocephalus*). Isoflurane, handling stress or both may have exacerbated an existing problem but because

of the challenges in properly examining avian patients for diagnosis of heart disease, this is likely to be underdiagnosed and underreported (Straub, Pees & Krautwald-Junghanns, 2001).

Clinical case No.4 (Elsie)

History and clinical signs

A 6 year old African Grey Parrot (*Psittacus erithacus*) was examined at GWR in October 2010. The owner contacted GWR via telephone reporting "Elsie" sitting on the cage floor looking very depressed, having deteriorated in the last 48 hours but eating and drinking. The owner was advised to bring "Elsie" to the hospital as soon as possible. "Elsie" had been with the owner for 6 years with no prior health problems and was tested negative for PBFD. It was kept with a clinically healthy Black Capped Conure (*Pyrrhura rupicola*). On examination "Elsie" was quiet, spending most of the time on the cage floor. Watery urine was observed. "Elsie" was thin with a body condition of 2/5, fluffed up but responsive.

Diagnosis and treatment

Radiographs showed a marked air sacculitis and a soft tissue mass in the mid-coelom. It was difficult to visualize other organ borders clearly due to air sac opacities. An IV catheter was placed into the basilic vein and a blood sample taken.

WBC count was $30x10^9$/L (6-$13x10^9$/L) (Fudge, 2000), blood smear showed a heterophil dominance, with more than 70% toxic heterophils. Uric acid measurements were high (584µmol/l) considering the normal range (117.8 - 648.3µmol/l) (Fudge, 2000). The recovery from GA was good. Differential diagnosis included chlamydophilosis, neoplasia, reproductive pathology, air sacculitis, septicaemia (though no bacteria were detected on haematology) and renal disease. Treatment plan included IV fluid therapy with 20% of an injectable solution of electrolytes, vitamins, amino-acids and dextrose (*Duphalyte®,* Fort Dodge Animal Health, Southampton, UK) + 80% Hartmann´s solution, pre warmed and mixed in the same syringe, given at the dose of the estimated fluid deficits (10% BW) in ml plus maintenance (40ml/Kg/day) and also meloxicam (*Metacam*® 5mg/ml Solution for Injection, Boehringer Ingelheim Vet-medica/Merial, Berkshire, UK) at a dose rate 0.1mg/Kg, IM, SID while waiting for further results. Endoscopy was scheduled for the next day for further investigation. On the next day a *Chlamydophila* test was performed using *Immunocomb*® (Biogal, Galed Laboratories, Kibbutz, Israel), a rapid EIA (Enzyme Immunoassay) for the detection of antibodies or antigens in serum or plasma and the result was negative.

On endoscopy the right side of the coelomic cavity was too opaque to see through the air sacs with diffuse opacities. The left side was also opaque but more discrete with small white masses adherent in multiple places. Two large white plaques were present with increased

vascularization. A mass visible on radiography appeared to be a large yellow plaque in the coelomic cavity, no soft tissue masses were evident. Samples were taken for cytology and culture. On the samples submitted it was possible to observe blue stained hyphae compatible with aspergillosis. Aspergillosis was diagnosed based on CS, haematologic results, radiographic abnormalities, endoscopic examination of the lower respiratory tract, cytologic examination of samples from the air sacs and fungal cultures.

"Elsie" started to slowly recover. Voriconazole (*VFEND*® 50mg film-coated tablets, Pfizer, Amboise, France) was administered PO and a liquid formulation of it was achieved by crushing a 50mg tablet into a fine powder, which was then mixed with 5ml of distilled water. A loading dose of 14mg/Kg, was administered BID for 3 days and then a maintenance dose of 10mg/Kg BID for the next 8 weeks. For the next 2 days "Elsie" was alert and responsive without signs of respiratory distress and went home on the 4th day.

One week later the owner contacted GWR via telephone to inform that "Elsie" was improving but for the last hour had been more quiet and in the corner of the cage. The owner was advised to continue to carefully monitor "Elsie" for the next hour and bring it back to GWR if any deterioration occurred or if not brighter. If coming in again the treatment plan would include hospitalisation for nebulisation and supportive care but "Elsie" improved and this was not necessary.

Discussion

Laboratory results from "Elsie" showed leukocytosis and heterophilia with toxic heterophils. Toxic heterophilia in avian patients indicates a severe inflammatory response (Campbell & Grant, 2010) being mostly associated with bacterial infections, including mycobacteriosis, but can occur with chlamydophilosis, fungal and some viral infections (Fudge, 2000). The greater the degree of toxicity and number of cells affected, the more severe the condition is, so the presence of numerous heterophils showing signs of marked toxicity (increased cytoplasmic basophlia, nuclear hypersegmentation, basophilic cytoplasmic granules) represents a severe condition and hence a poorer prognosis for survival (Forbes & Altman, 2010). In this case, the presence of more than 70% of toxic heterophils carried a poor prognosis. The uric acid levels and watery urine are suggestive of kidney involvement. The kidney is rarely involved in systemic and respiratory mycotic diseases (Phalen & Graham, 1990 cited by Fudge, 2000) but since lesions are commonly found in the air sacs that are in direct contact with the kidneys, extensive granulomas can cause a mass effect that may affect kidney function and local invasion into the kidney may occur. In an advanced state, there is dissemination of hyphae as embolus into the kidney and throughout the body, in this terminal phase of disease, renal function is compromised but there are other indicators that the bird has severe systemic disorder (Fudge, 2000). As for clinical case No.2 the same principles about aspergillosis apply, with African Grey Parrots also reported to be at risk. Aspergillosis was

diagnosed based on CS, haematologic results, radiographic abnormalities (air sacculitis and air sac granulomas), endoscopic examination of the lower respiratory tract, cytologic examination of samples from the air sacs and fungal cultures. CS combined with radiographic findings can establish a strong presumptive diagnosis but Flammer (2007) prefers to reach a diagnosis by culture of tracheal or air sac samples collected with an endoscope as it was performed with "Elsie". Cytological specimens can be stained with lactophenol cotton blue. On the samples submitted it was possible to observe blue stained hyphae compatible with aspergillosis. It is important to establish a confirmed diagnosis because treatment is expensive, prolonged and potentially toxic (Flammer, 2007). According to some authors (Di Somma, Bailey, Silvanose & Garcia-Martinez, 2007) this is a case of grade 3 aspergillosis (advanced) with large and coalescing air-sac granulomas (>10mm diameter) present, marked leukocytosis and heterophilia, general CS of malaise, easily detectable radiographic lesions in air sacs and culture and cytologic results positive for *Aspergillus* spp.

Optimal treatment regimens include debriding lesions, followed by topical therapy administered in conjunction with early, aggressive, systemic antifungal treatment. Topical therapy may be achieved by nebulization or surgical air sac flushing (Oglesbee, 1997 cited by Di Somma et al., 2007). Traditional treatment for aspergillosis infections in birds involves the use of amphotericin B, itraconazole, and terbinafine (Jones & Orosz, 2000). Voriconazole is a novel, broad-spectrum, triazole antifungal with good activity against *Aspergillus* spp. (Verweij & Denning, 1997), having some distinct benefits compared with traditional antifungal agents, such as reliable oral bioavailability and excellent activity against many *Aspergillus* spp. that are resistant to currently available therapy (Hoffman & Rathbun, 2002 and Greer, 2003 cited by Di Somma et al., 2007). Voriconazole is also used in patients who cannot tolerate other therapies. Most adverse events seen with voriconazole are similar to those seen with other triazole drugs and are not life threatening (Whelan, 2002 cited by Di Somma et al., 2007). Infection with *Aspergillus* spp. should be considered to occur secondary to an immunosuppressive event (Kunkle, 2003). Although there was no evidence of a primary disease that immunocompromised "Elsie", environmental factors may have caused physical stress that predisposed the bird to a systemic fungal infection (Di Somma et al., 2007).

Clinical case No.5 (Darcy)

History and clinical signs

A male Lilac-crowned Amazon (*Amazona finschi*) arrived at GWR on October 28th, 2010 with a history of being depressed and anorexic all day although brighter in the consult room. The owner refused to sign a consent form for procedures and opted to start a full work up the next day. PE was performed the next day with nothing abnormal detected on oral

examination or palpation, no nasal or ocular discharge, cloaca was clean but "Darcy" was very stressed when handled.

Diagnosis and treatment

Enrofloxacin (*Baytril*® Injectable Solution 2,5%, Bayer Corp., Leverkusen, Germany) at the dose rate of 30mg/Kg, IM was administered. The advised treatment plan consisted of silymarin (Tesco© Milk Thistle tablets, Dundee, UK) at a dose rate of 100mg/Kg, PO, BID, enrofloxacin (*Baytril*® Oral Solution 2,5%, Bayer Corp., Leverkusen, Germany) at the dose rate of 20mg/Kg, PO, SID, celecoxib (*Celebrex*® Caps 100mg, Pfizer, Amboise, France) at the dose rate of 10mg/Kg, PO, BID, fluid therapy with 20% of an injectable solution of electrolytes, vitamins, amino-acids and dextrose (*Duphalyte®,* Fort Dodge Animal Health, Southampton, UK) + 80% Hartmann´s solution, pre warmed and mixed in the same syringe, IV, given at the dose of the estimated fluid deficits (10% BW) in ml plus maintenance (40ml/Kg/day) plus ongoing losses and crop feeding but the owner declined and decided to take "Darcy" home that night.

On November 4th "Darcy" was back at GWR with an episode of vomiting the previous night and a change in voice. An IV catheter was placed into the basilic vein and a blood sample was taken. CBC showed a WBC count of 18x10^{9}/L (7-13 x10^{9}/L), PCV 30% (44-52%) (Fudge, 2000), raised AST but normal CK and low bile acids. On radiographs a dilated proventriculus and distended intestinal loops were observed, visible spleen, lungs and clear air sacs. Nothing abnormal was detected on tracheoscopy. A swab was taken from the crop for bacteriology. *Immunocomb*® (Biogal, Galed Laboratories, Kibbutz, Israel) test was negative for chlamydophilosis. The owner declined fluoroscopy and crop biopsy and opted for the initially advised medical treatment. Differential diagnosis included PDD, enteritis and hepatic disease. The next day "Darcy" lost weight so fluid and food volumes were increased and the treatment plan continued. The IV catheter was out so fluids were given SC into the inguinal and axillary regions at a maximum dose of 10ml/Kg/site. "Darcy" was brighter and perching.

On November 5th "Darcy" had a slight increase in weight during the day but still weighted less than on admission. Food and fluid volumes were increased and therapy continued. The bird was eating some fruit on its own. Sensitive results were still pendant.

On November 6th "Darcy" was eating vegetables, fruit, some seeds and *Harrisson´s*© Recovery Formula (Harrison's© Bird Foods, Tennessee, USA) at the dose of 5ml/100g BW, BID, with all the medications included in the morning having lost weight in the morning after not being fed throughout the night. Antibiotic was changed to carbenicillin (*Geocillin*® Roerig, Pfizer, France) at the dose rate of 100 mg/Kg, PO, BID as indicated by sensitivity results (culture was a *Pseudomonas* spp. colony sensitive only to β-lactam penicillins).

On November 7th "Darcy" was stable and eating on its own. "Darcy" was sent home on carbenicillin (*Geocillin*® Roerig, Pfizer, France) at the dose rate of 100 mg/Kg, PO, BID, celecoxib (*Celebrex*® Caps 100mg, Pfizer, Amboise, France) at the dose rate of 10mg/Kg, PO, BID and silymarin (Tesco© Milk Thistle tablets, Dundee, UK) at a dose rate of 100mg/Kg, PO, BID and if not improving, the owner was advised to bring it back for fluoroscopy to rule out PDD.

On November 10th "Darcy" was improving but had suddenly became lethargic even if much better than when it first came in to the hospital. The owner was not checking it´s weight but said "Darcy" was eating more. A recheck for "Darcy" was advised for as soon as possible.

On November 11th "Darcy" was back in the hospital and a CBC was performed with a WBC count of $100x10^9$/L ($7\text{-}13x10^9$/L) (Fudge, 2000). The next day a slight tracheal noise was detected and 1ml of cephalexin (*Ceporex*™ Oral Drops, Schering-Plough Animal Health, Buckighamshire, UK) was administered PO.

On November 13th "Darcy" was bright, eating some food on its own and maintaining weight. WBC count increased to $130x10^9$/L ($7\text{-}13x10^9$/L) (Fudge, 2000). The next day "Darcy" was stable, maintaining its weight and being crop fed 15ml of *Harrisson´s*© Recovery Formula (Harrison's© Bird Foods, Tennessee, USA) TID but not eating much on its own.

On November 15th a blood smear was performed and on examination, mostly heterophils were found. Radiographs were repeated to discard a potential mycotic infection, increased air sac opacities were observed. Concurrent antifungal therapy was started with voriconazole (*VFEND*® 50mg film-coated tablets, Pfizer, Amboise, France) at the dose rate of 10mg/Kg, PO, BID and sulfamethoxazole/trimethoprim (*Septrin*® Paediatric Suspension, GlaxonSmithKline, Middlesex, UK) at the dose rate of 8mg/Kg, IM, BID.

On November 16th "Darcy" was still not improving and weighted the same as the previous day. IV catheter was absent so fluids were administered SC as before to avoid repeating GA.

On November 17th "Darcy" was brighter, eating on its own and maintaining weight. Therapy was continued. The next day CBC was repeated with a WBC count of $56x10^9$/L ($7\text{-}13\ x10^9$/L) (Fudge, 2000). "Darcy" was improving so on November 20th crop feeding and fluid therapy were stopped to assess if still maintaining its weight. The next day "Darcy" maintained it´s weight overnight and was very bright. On November 22th "Darcy" gained some weight and another blood sample was taken with the WBC count now at $41x10^9$/L ($7\text{-}13\ x10^9$/L) (Fudge, 2000). "Darcy" was discharged and another blood sample scheduled to be repeated in 7-10 days. On December 12th "Darcy" was back seeming clinically better. A blood sample was taken with a WBC of $8{,}5\ x10^9$/L ($7\text{-}13\ x10^9$/L) and a PCV of 51% (44-52%) (Fudge, 2000).

Discussion

The plasma BC panel revealed a mildly elevated AST suggestive of hepatocellular disease, however, this enzyme is not specific for the liver (Campbell & Grant, 2010). More likely, and since this elevation on AST was noted after a course of IM enrofloxacin, muscle necrosis secondary to the injection could have been responsible for the AST elevation (Fudge, 2000). The absence of elevated CK activity after the injection can be explained by the relatively short half-life of CK compared to AST (Forbes & Altman, 2010). Other enzymes were normal and bile acid levels were low so hepatic disease should be excluded (Fudge, 2000). Mild leukocytosis supports an inflammatory leukogram and the decreased PCV reveals the presence of anaemia that could be related to GI malabsorption. Leukocytosis and heterophilia are present in some patients with PDD but are not a consistent finding and seem to be related to stress and/or to the existence of secondary infections (Gancz, Clubb & Shivaprasad, 2010). The most consistent finding in birds with PDD is a moderately to markedly distended proventriculus as it was observed. Other GI compartments that may be distended include the crop, ventriculus and small intestine but this is not specific for PDD. The owner refused to perform a crop biopsy so a definitive diagnosis could not be made. The NSAID celecoxib (*Celebrex*®, Pfizer, Amboise, France), was used to address pain and inflammation (Dalhausen, Aldred & Colaizzi, 2002 cited by Gancz et al., 2010). PDD is an infectious disease that causes inflammation of the central and peripheral nervous system as well as the digestive system so management of the disease involves prevention of transmission, reducing inflammation, aiding digestion and controlling secondary infections, which in many cases needs to be maintained for a long time. With prolonged therapy and control of secondary infections, birds that are diagnosed early can return to good physical condition (Gancz et al., 2010). The medical extract from the milk thistle plant (*Silybum marinum*) is silymarin which is thought to assist in the treatment of liver disease through its antioxidant properties, stabilization of cell membranes, regulation of cell permeability and promotion of DNA, RNA and protein synthesis. Side effects of silymarin are rarely reported, but include nausea and diarrhoea, which may explain the vomiting (Plumb, 2005). The severe leukocytosis that developed after the initial presentation and while treatment was being performed was likely caused from stress-related aspergillosis that commonly develops in immunocompromised psittacines exposed to physical stress, predisposing the bird to fungal infection (Di Somma et al., 2007). Because of the response to treatment and evolution of CS this was likely to be a case of enteritis complicated by stress-related aspergillosis. Although the bird seemed stable, close monitoring was recommended and the owner advised about the risks of PDD infection, which couldn´t be ruled out since a definitive diagnostic biopsy was not performed.

Clinical case No.6

History and clinical signs

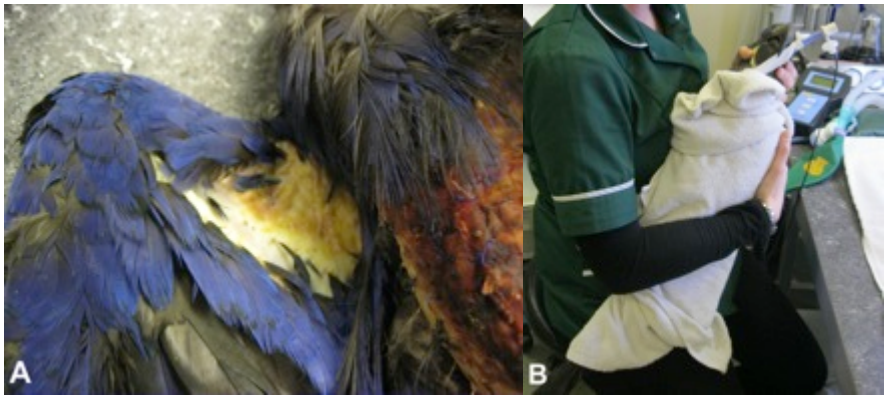
A 2 year old, male Hyacinth Macaw (*Anodorhynchus hyacinthinus*) was examined on 2nd December 2010 at GWR as an emergency. The bird was emaciated, with septicaemic cutaneous ulcerative disease (SCUD) posterior to self-trauma and blood loss most prominent on the pectoral region and ventral surface of the wings (Fig. 8 and 9A).

Diagnosis and treatment

An IV catheter was placed in the basilic vein and a blood sample taken for haematology and BC. Enrofloxacin (*Baytril*® Injectable Solution 2,5%, Bayer Corp., Leverkusen, Germany) at the dose rate of 15mg/Kg and carprofen (*Carprieve*® Small Animal Solution for Injection 5%, Norbrook, Newry, UK) at the dose rate of 5mg/Kg were administered IV. While radiographs were being taken the bird collapsed. Resuscitation was attempted and doxapram was administered (*Dopram*®-V Injectable, Boehringer Ingelheim, Ingelheim am Rhein, Germany) at the dose rate of 5mg/Kg, IV. When stable (Fig. 9B), the bird was put into a cage but after few minutes it collapsed again. Unfortunately, all efforts to resuscitate the bird for a second time failed and it died. Blood parameters revealed kidney failure which might have been responsible for the collapse and *Immunocomb*® (Biogal, Galed Laboratories, Kibbutz, Israel) result was positive for chlamydophilosis.

Figure 8: Placement of an oesophageal stetoscope into an *Anodorhynchus hyacinthinus* with SCUD lesions on the pectoral region and wings (Original picture).

Figure 9: A) Detail of the SCUD lesions of an *Anodorhynchus hyacinthinus*; B) Recovery from GA - the bird is held in a towel with the head elevated until awake (Original picture).



Discussion

The pattern of feather loss and skin lesions was suggestive that feather picking and self-mutilation were the cause of chronic skin disease caused by an opportunistic infection. Generally, feather picking is observed in companion birds kept in relative isolation without the benefit of social interaction with owners or other birds (Perry et al., 1991 cited by Peleteiro et al., 1998). The owner reported that this bird had a companion that died recently and since then a self-mutilation behaviour developed. Primary bacterial dermatitis is uncommon in birds, perhaps because of the bacteriostatic and mycostatic effects of epidermal lipids (Koski, 2002), however, inflamed and ulcerated skin has lost its protective barrier against colonization by opportunistic bacteria and yeasts. Secondary infection occurs early in the course of disease (Powers & Van Sant, 2006). Bacteria that have been cultured from avian skin wounds include species of *Staphylococcus*, *Streptococcus* and *Pseudomonas aeruginosa*. *Serratia*, *Nocardia*, *Mycobacterium*, and *Klebsiella* spp. have been associated with dermatitis and folliculitis of birds (Koski, 2002). *Staphylococcus* spp. are considered part of the normal cutaneous flora in birds so overinterpretation of their isolation by culture should be avoided (Koski, 2002) although in avian medicine, staphylococcal infections have been linked with ulcerative dermatitis and chronic feather destructive disorders in pet psittacines (Bauk, 1997 cited by Smith & Forbes, nd). Clinical examination of the bird, demonstrated markedly inflamed and exudative areas of skin of the chest and ventral aspects of both wings and the corresponding areas of body wall consistent with SCUD. Methicillin-resistant *Staphylococcus aureus* (MRSA) is becoming increasingly recognized as a pathogenic organism in veterinary medicine (Smith & Forbes, nd). Although no sensitivity or culture results were obtained there was a strong suspicion that this was the causative agent of the disease. MRSA infection should be considered in pet parrots with chronic history of feather plucking, self-mutilation or infection (May, 2006 by Smith & Forbes, nd). Isolation of MRSA from skin lesions of pet parrots shows that they are also at risk from MRSA infections as humans or other mammals, raising the suspicion that pet parrots may act as a reservoir for

zoonotic infections of susceptible humans. This condition warns that the use of antibiotics for extended periods without culture and sensitivity testing may lead to more widely resistant staphylococcal strains, for which there may be no effective antibiotic. Treatment options may be limited to euthanasia in severe cases (Smith & Forbes, nd). The ideal initial diagnostic evaluation should include a CBC, plasma BC analysis, impression smears of the lesions and microbiologic culture (Powers & Van Sant, 2006), unfortunately the sudden collapse of the bird while performing diagnostic tests left this evaluation incomplete. Blood parameters where consistent with kidney failure, which may have developed from the septicaemia or *Chlamydophila* infection. Macaws experience an incidence of renal mineralization associated with exclusive feeding of some pelleted diets and in fact, experts believe that these birds need a higher caloric density and lower quantity of vitamin D_3 in their feed compared to other parrots. Many birds with diet-induced renal mineralization die without obvious CS of renal disease although there may be a history of polydipsia/polyuria (Fudge, 2000), so this could also explain the kidney failure. More diagnostic tests should have been performed to determine the accurate cause of death but were denied by the owner.

Clinical case No.7 (Ekky)

History and clinical signs

On December 15th an 18 year old, female, European Eagle-owl (*Bubo bubo*) (Fig. 10) arrived at GWR with acute onset dyspnoea. The bird also presented with anorexia, lethargy, dehydration, weight loss, regurgitation, dyspnoea and poor feather quality. On PE trachea was normal and ascites was detected on palpation.

Figure 10: "Ekky" (*Bubo bubo*), hospitalised at GWR (Original picture).

Diagnosis and treatment

Under GA (Fig. 11), abdominocentesis was performed and 60ml of exudate drained. Heart size and liver parameters were normal but low albumin (6g/dL) (11-20g/dL) and total proteins (18g/L) (33-45.9g/L) (Fudge, 2000) were present, so a protein loosing nephropathy, since uric acid was marginally high, was suspected.

On December 21th "Ekky" vomited fluid and WBC count increased from 13x10^{9}/L to 20x10^{9}/L (10-25x10^{9}/L), PCV was 34% (30-50%) (Fudge, 2000). Albumin and total protein were still low. "Ekky" was hospitalised for monitoring and treatment.

Figure 11: Corneal reflex evaluation during GA (*Bubo bubo*) (Original picture).

The next day "Ekky" was bright but was not eating on its own so was given *a/d*® Canine/Feline Critical Care (Hill´s Prescription Diet®, Watford, UK) given by crop-tube at a dose of 5ml/Kg, every 2 hours, increasing to 10ml/Kg on at least 3-4 occasions. Clavulanate-potentiated amoxycillin (*Synulox*®**,** Pfizer, Amboise, France) at the dose rate of 150mg/Kg, PO, BID for 7 days was administered.

On December 23th "Ekky" had lost 50g and wasn´t eating by itself but was bright and had passed liquid faeces and urates. Started regurgitating after given 10ml *a/d*® Canine/Feline Critical Care (Hill´s Prescription Diet®, Watford, UK). Conservative care was continued and in the afternoon "Ekky" ate some pieces of skinned meat when offered without regurgitating and faeces were firmer.

On December 24th "Ekky" was alert and responsive. A blood sample was taken and WBC count was 15x10^{9}/L (10-25x10^{9}/L) (Fudge, 2000). "Ekky" was still not eating by itself but was taking meat when offered, regurgitated in the morning but was gaining weight since the previous day. A liver biopsy was performed and exudate was detected in the hepatic peritoneal space. Cytology of the exudate and liver revealed a primary infectious inflammatory reaction without indication of neoplasia. The plan was to maintain clavulanate-potentiated amoxycillin (*Synulox*®, Pfizer, Amboise, France) at the dose rate of 150mg/Kg, PO, BID for 3 days and retest the WBC count in 4 days, if less than 10x10^{9}/L (10-25x10^{9}/L), "Ekky" could be sent home with antibiotics. Histopathology results were still pending.

On December 25th "Ekky" was bright, eating offered meat and gained weight. On December 29th a blood sample was taken and WBC count was $10x10^9$/L ($10-25x10^9$/L) (Fudge, 2000). On December 31th bile acids increased from previous 37µmol/l to 111µmol/l (8-54µmol/L), AST from 640IU/l to 769IU/L (104-344IU/l) and LDH from 634IU/L to 795IU/l (142-383IU/L) (Fudge, 2000). "Ekky" was sent home on marbofloxacin (*Marbocyl*® P 20mg, Vétoquinol, Bicester, UK) at the dose rate of 10mg/Kg, BID, PO for 7 days.

On January 26th, 2011 "Ekky" was back at GWR. After being on marbofloxacin (*Marbocyl*® P 20mg, Vétoquinol, Bicester, UK) at the dose rate of 10mg/Kg, BID, PO for a brief period it became anorexic so medication was changed to cephalexin (*Ceporex*™ Oral Drops, Schering-Plough Animal Health, Buckighamshire, UK) at the dose rate of 50mg/Kg, PO, TID for 5 days. On the next day the owner contacted GWR via telephone to inform that "Ekky" was regurgitating so returned to GWR for supportive care and further diagnostics. Voriconazole (*VFEND*® 50mg film-coated tablets, Pfizer, Amboise, France) at the dose rate of 10mg/Kg, PO was given but the bird regurgitated it. A blood sample was taken and WBC count decreased from $20x10^9$/L to $18.5x10^9$/L but was still high ($10-25 x10^9$/L). PCV was 27% (30-50%) (Fudge, 2000). A dose of 1.42ml marbofloxacin (*Marbocyl*® SA injection, Vétoquinol, Bicester, UK) at the dose rate of 10mg/Kg, IM and 1.73ml of clavulanate-potentiated amoxycillin (*Synulox*®, Pfizer, Amboise, France) at the dose rate of 125mg/Kg, PO were administered.

On January 28th radiographs were taken to reassess and compare with the previous ones and there was liver enlargement, more prominent than previously, with no visible ascites. Liver biopsy results revealed a severe chronic-active hepatitis with fibrosis, biliary hyperplasia and nodular regeneration typical of a liver with "cirrhosis". It was also detected folliculostasis with necrosis and recent ovarian haemorrhage on the ovary.

The histology report (see annex II) was provided by INTERNATIONAL ZOO VETGROUP PATHOLOGY (IZVG Pathology), UK and was as follows:

> Liver: Normal architecture is largely replaced by irregular tracts of bridging fibrosis with hyperplastic sprouting bile ducts, isolated clusters of hepatocytes embedded in fibrous tissue, and occasional more distinct regenerative nodules. Within the fibrous tissue there are moderate multifocal to coalescent infiltrates of heterophils, lymphocytes, plasma cells and monocytes/macrophages. Small numbers of haemosiderin-laden macrophages are also present. The capsule is irregularly and markedly thickened in some areas.
>
> Ovary: An enlarged follicle contains large amounts of degenerate/necrotic eosinophilic debris with residual vitelline granules and focally extensive recent intra-follicular haemorrhage. The degenerate material is interspersed with lipid-laden foamy macrophages, and surrounded by a fibrous capsule of variable thickness that is continuous with fibrous tissue in the adjacent ovarian stroma.

Discussion

Hepatic disease is related to chronic conditions and is not a common emergency presentation but secondary effects of liver disease can create emergency presentations, such as the acute onset dyspnoea presented with this case. Hepatopathy is a non-specific disease condition with different aetiologies in birds such as drugs/chemicals (e.g. mebendazole, chemical poisons), infections (e.g. *Campylobacter jejuni*, reovirus, *Yersinia pseudotuberculosis*, *S. typhimurium*) or metabolic inbalances (e.g. *diabetes mellitus*, hypothyroidism) (Forbes, 1996a). Owl herpesvirus infection is seen in captive and wild Strigiformes in the UK and elsewhere, with affected owls seen having multiple caseous necrotic foci of the lining to the posterior palate and with ill birds invariably dying, so this differential diagnosis was excluded (Forbes & Harcourt-Brown, 1996). There aren´t pathognomonic CS of avian liver disease but some nonspecific signs associated with it were observed in this case, including anorexia, lethargy, weakness, dehydration, weight loss, regurgitation, vomiting, dyspnoea and poor feather quality (Jaensch, 2000). Hepatomegaly and ascites can be associated with liver disease and can result in coelomic distension (that may explain the vomiting/regurgitation) and respiratory compromise (Harrison & Lightfoot, 2006) due to compression of the abdominal air sacs, dorsal displacement of the proventriculus and caudodorsal displacement of the ventriculus (McMillan, 1994 cited by Grunkemeyer, 2010). Ascites resulting in coelomic distension and respiratory distress requires immediate coelomocentesis. Exudate was collected, being characterized by a high specific gravity (>1.020), a high protein content (>3g/dL) and the presence of inflammatory and mesothelial cells (Bowles et al., 2007). Anaemia can occur as a result of coagulopathies, hepatic trauma, haemochromatosis and bone marrow suppression caused by hepatic disease. Leukocytosis may be present with infectious or inflammatory liver disease, especially if caused by *Chlamydophila*, *Mycobacterium* or *Aspergillus* spp. (Campbell & Ellis, 2000 cited by Grunkemeyer, 2010). Hypoproteinemia can occur with hepatic failure since the liver is responsible for synthesizing plasma proteins. AST is sensitive to hepatic disease but is also present in muscle tissue so can be indicative of both liver or muscle damage (Harr, 2006 cited by Grunkemeyer, 2010). LDH is not specific or sensitive for liver damage but if elevated indicates recent tissue damage because of its short plasma half-life. If the liver is damaged, bile acids are not appropriately extracted, conjugated and secreted (Fudge, 2000) so the measurement of the bile acid concentration in plasma provides a sensitive and specific indication of hepatic function (Ritchie et al., 1994). During the aspiration of ascitic fluid from the coelomic cavity, the volume of fluid aspirated was restricted to only that required to relieve respiratory compromise since the removal of a large volume of ascitic fluid can result in severe protein loss (Grunkemeyer, 2010).

According to the histopathology report by result, diagnosis was a severe chronic-active hepatitis with fibrosis, biliary hyperplasia and nodular regeneration ("cirrhosis") of the liver

and a folliculostasis with necrosis and recent ovarian haemorrhage of the ovary. Results revealed non-specific changes and in this case, the chronicity of the lesion obscured the possible aetiology. Aflatoxin exposure is frequently speculated to be involved, as well as other bile-excreted toxins, whilst chronic infectious diseases including chlamydophilosis and some viral infections have been implicated in parrots. In this case there were no evidences to suggest either of the latter. The ovary included a necrotic follicle and recent haemorrhage without evidence of bacteria or significant heterophilic inflammation, which could have been caused by a sterile degenerative process in a yolk laden follicle that has failed to ovulate, seeming unlikely that this was the underlying cause for the hepatitis.

Possible causes for the changes observed in the reproductive tract could also correlate to infection of surrouding tissues, in this case, the liver (Hadley, 2010). It was not possible to determine the cause of the initial insult and no pathogenic organism could be demonstrated (Forbes, 1996a). Specific testing such as bacterial and fungal cultures, viral polymerase chain reactions and heavy metal blood levels should have been pursued to determine the inciting cause of the hepatic damage (Grunkemeyer, 2010). The treatment plan included both targeted and supportive therapies. Response to treatment was closely monitored by periodically repeating the tests that were used to diagnose the hepatic disease (Grunkemeyer, 2010). Bile acid evaluation may support diagnosis of liver disease, but absolute confirmation, including evaluation of type and extent of disease is only possible with liver biopsy (Bowles et al., 2007).

Clinical case No.8 (Sonny)

History and clinical signs

On November 14th, 2010 a 6 year old, male Galah (*Eolophus roseicapilla*) was examined at GWR. The owner noticed a swelling of the right leg around the bird´s ring for the past 4-5 days. "Sonny" was bright, perching and eating. The owner wanted to have the ring removed.

Diagnosis and treatment

"Sonny" was induced and maintained with isoflurane. An ET tube and IV catheter on the basilic vein were placed. The right intertarsal joint was extremely swollen and infected with the ring completely embedded in the skin. The ring was removed with a dental sore, the skin underneath completely exposed and the flap removed. The leg bled profusely so haemostatic sponges and radiosurgery were used to stop the bleeding without success. *Vicryl** 4/0 (Ethicon Inc., Auneau, France) suture material was applied to close the bleeding vessel. Haemostatic sponges and Co-Flex® (Andover Healthcare, Salisbury, USA) were applied to bandage the wound. The left intertarsal joint was swollen and hyperaemic as well. IV fluid therapy with 20% of an injectable solution of electrolytes, vitamins, amino-acids and

dextrose (*Duphalyte®,* Fort Dodge Animal Health, Southampton, UK) + 80% Hartmann´s solution, pre warmed and mixed in the same syringe, given at the dose of the estimated fluid deficits (10% BW) in ml plus maintenance (40ml/Kg/day) plus ongoing losses was provided. Marbofloxacin (*Marbocyl*® SA injection,Vétoquinol, Bicester, UK) at the dose rate of 10mg/Kg, IM, BID for 5 days, meloxicam (*Metacam*® 5mg/ml Solution for Injection, Boehringer Ingelheim Vet-medica/Merial, Berkshire, UK) at the dose rate 0.1mg/Kg, IM, SID and butorphanol tartrate (Torbugesic® Injection, Fort Dodge Animal Health, Southamptom, UK) at the dose rate of 1mg/Kg, IM, SID were administered.

The treatment plan was to continue with the marbofloxacin, meloxicam, butorphanol tartrate and fluid therapy. Crop feeding with *Harrisson´s*© Recovery Formula (Harrison's© Bird Foods, Tennessee, USA) at the dose of 5ml/100g BW, BID was started. In the afternoon the bird removed the IV catheter. On the next day "Sonny" was bright, perching, bearing weight on the affected leg and maintaining weight. Radiographs were taken under GA to rule out an underlying bone infection with no observed evidence of bone involvement. "Sonny" was sent home on marbofloxacin (*Marbocyl*® P 5mg, Vétoquinol, Bicester, UK) at the dose rate of 5mg/Kg, SID, PO and meloxicam (*Metacam*® Oral Suspension, Boehringer Ingelheim Vet-medica/Merial, Berkshire, UK) at the dose rate of 0.1 mg/Kg, PO, SID and scheduled to come back in a week time. On November 19th the leg was bleeding and "Sonny" was depressed at the bottom of the cage. Under GA, the right foot was checked having dried blood but no active haemorrhage. PCV was 31% (40-55%) (Fudge, 2000). The foot was cleaned and *Kaltostat*® (Convatec, Skillman, USA) and a bandage with *Co-Flex*® (Andover Healthcare, Salisbury, USA) were applied. SC fluids were given into the featherless inguinal and axillary regions at the maximum dose of 5ml/Kg/site. "Sonny" stayed in the hospital overnight for monitoring and a buster collar was applied (Fig. 12).

Figure 12: "Sonny" with a buster collar (Original picture).

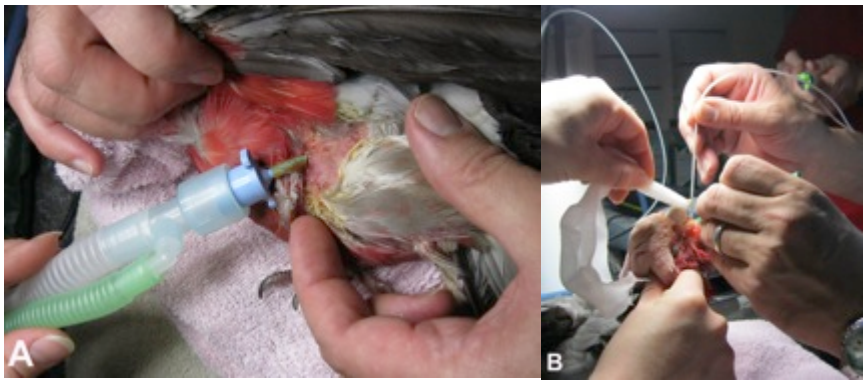
On the next day "Sonny" was anorexic but bright and not bleeding. On 22nd November "Sonny" was discharged with instructions to weight, hand feed and return for dressing

change in 3 days. On November 29th "Sonny" removed the collar and chewed its leg. On PE a large amount of blood and a small wound were detected. "Sonny" stayed in the hospital for the next week and was given SC fluids and crop feeding with *Harrisson´s*© Recovery Formula (Harrison's© Bird Foods, Tennessee, USA) at the dose of 5ml/100g BW, TID. The next day it had lost weight so *Harrisson´s*© Recovery Formula (Harrison's© Bird Foods, Tennessee, USA) and SC fluids were increased to QID but "Sonny" was not able to take more than 2-3ml of formula via the crop tube without regurgitating. The next day "Sonny" was doing well, maintained weight overnight with no supplemental feeding and the leg was looking good. On December 2nd it was bright, eating on it´s own and maintaining weight so was discharged. The next day "Sonny" was again sitting at the bottom of its cage and anorexic. On PE the foot was looking normal, the collar was adjusted and ½ of the foam part removed to make it smaller. The owner was advised to monitor the collar and ensure "Sonny" didn´t chew it off. On December 8th the owner brought "Sonny" to remove the collar. On PE, the left leg was hypertrophied with a possible xanthoma formation. The left foot was flaccid and with no withdrawal movements or sensitivity. The next day radiographs (lateral and dorsoplantar views) of the tarsus were taken to see if bone involvement was present and it was observed erosion of the articular surface of the intertarsal joint and widening of the intra-articular space with osteophyte formation on the caudal aspect. Small surgery of the foot was performed and a large plug of caseous material was removed from the tarsus. Recovery from GA was slow. Started oral marbofloxacin (*Marbocyl*® P 5mg, Vétoquinol, Bicester, UK) and meloxicam (*Metacam*® Oral Suspension, Boehringer Ingelheim Vet-medica/Merial, Berkshire, UK).

On Janurary 3rd, 2011 "Sonny" was hypersalivating. Mouth, oesophagus and crop were clear but there was an ulcer (3x8mm) on the left caudal side of the tongue.

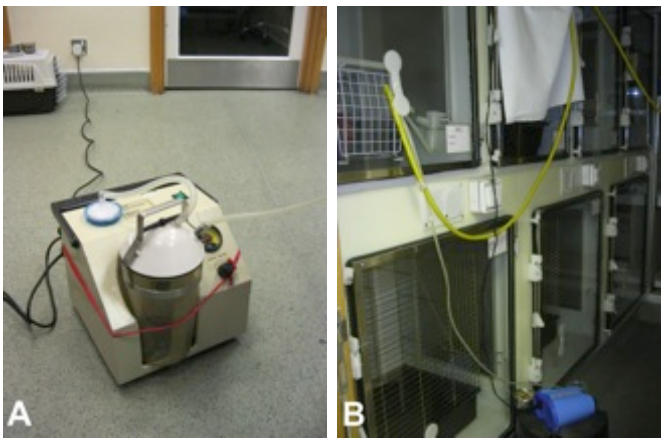
On January 12th during a recheck, "Sonny" seemed to be improving. Advised to continue on meloxicam for 3 weeks and then recheck. On the next day acute onset dyspnoea probably from aspiration after handling the previous day started which eased considerably after an air sac tube was placed (Fig. 13A).

Figure 13: A) Air sac tube placement to "Sonny"; B) Tracheoscopy and aspiration of exudate (Original picture).



The next day a tracheoscopy showed hard white exudate in the trachea, possibly a reaction posterior to aspiration, that was removed by suction (Fig. 13B). Trachea was inflammed but clear. On January 15th "Sonny" had lost some weight but was looking stable. While being medicated it became very dyspnoeic so was returned to its O_2 enriched cage (Fig.14B) until stable. Considered GA, scope and suction if not improving the next day.

Figure 14: A) Nebulizator pressurized gas source; B) Nebulization and O_2 (yellow cable) into patient´s cage (Original picture).



The next day "Sonny" gained some weight. The stress caused by the parenteral medications was considered so medication started to be given via a nebulizator into it´s cage (Fig. 14A) as well as *Harrisson´s*© Recovery Formula (Harrison's© Bird Foods, Tennessee, USA) at the dose of 5ml/100g BW, BID and 5ml SC fluids BID. In the afternoon "Sonny" was still dyspnoeic but not as stressed as before. On January 17th severe tracheal pathology was detected so a trachectomy was performed. Anaesthesia was performed via the air sac tube and "Sonny" was placed in dorsal recumbency with the head directed towards Dr. Neil Forbes. The front of the bird was elevated at 45º to the tail to aid visualization into the thorax (Fig. 15A). A skin incision was made adjacent to the thoracic inlet (Fig. 15B) and the crop identified, bluntly dissected and displaced to the right side. The interclavicular air sac was penetrated and the trachea elevated, then the *sterno-trachealis* muscle was transected. The superficial pectoral muscles were elevated and an osteotomy of the clavicle performed for additional access. Stay sutures were placed into the trachea. A trachectomy was performed by cutting through the ligaments between the cartilages of the affected tracheal segment and gently removing them (Fig. 16 and 17). Close apposition of cartilages following surgery was performed with suture material that elicited minimal tissue reaction (PDS*II, Ethicon, Auneau, France) to minimise intraluminal granuloma formation. Unfortunately "Sonny" died at the time of tracheal closure due to anaesthetic complications despite all the efforts to resuscitate it.

Figure 15: A) Surgical preparation and positioning of "Sonny"; B) Skin incision during trachectomy (Original picture).

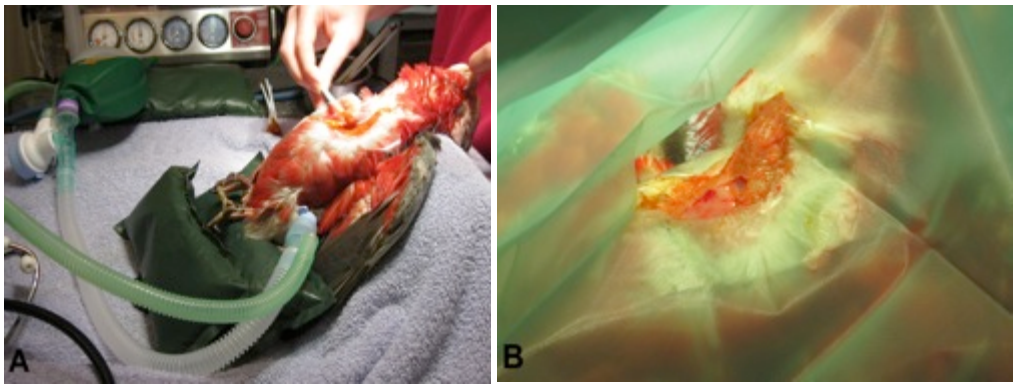


Figure 16: A) After removal of the affected traqueal segment; B) Lone Star Retractor System™ (CooperSurgical Inc., Staffordshire, UK) aiding in visualization and suturing the separated tracheal segments back together during surgery (Original picture).

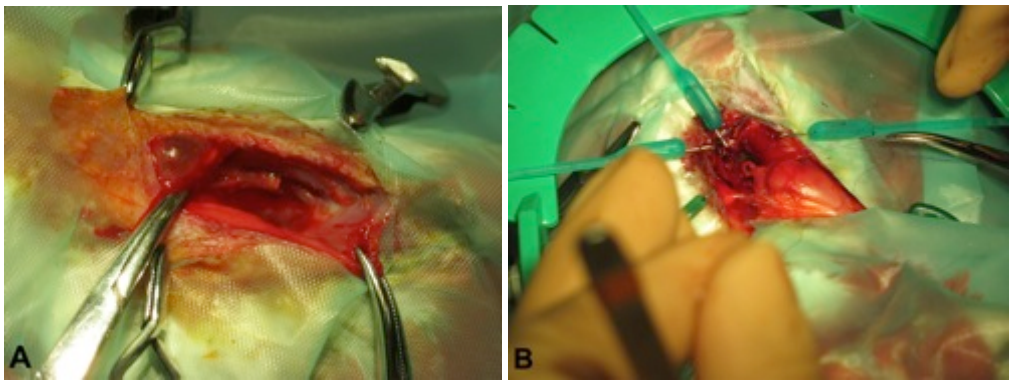
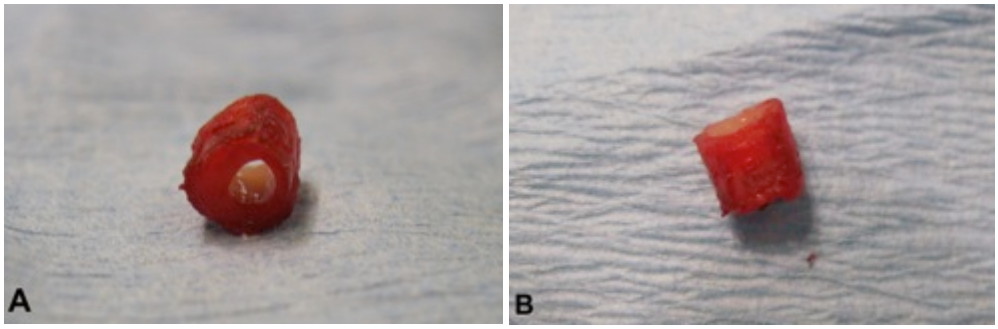


Figure 17: A) and B) Removed affected segment of the trachea with a decreased luminal diameter (Original picture).



Discussion

On initial presentation, this case was not an emergency situation but complications during treatment of the initial cause of presentation (traumatic) and the stress related to repeated handling and medical procedures may have been responsible for the final outcome of this case. Injuries from leg rings are common in birds and inappropriately sized rings may cause soft tissue swelling and vascular compromise to the distal leg and toes (Bowles et al., 2007). Wounds that are deep, extensive or obviously contaminated require parenteral antibiotic administration as well as topical debridement (Harrison & Lightfoot, 2006). Once an injury or associated problem with a ring is recognized, extreme caution should be exercised with ring removal to avoid additional injury to the bird. The owner should always be warned of potential risks to the bird whenever a band is removed, even when the procedure is elective and not associated with trauma. Complications include fractures, dislocations and lacerations. If a wound is already present, avascular necrosis may complicate the band removal procedure. Specific treatment options following ring removal were performed and involved wound debridement and cleansing, surgical closure and coverage with appropriate dressing and bandaging material (Ritchie et al., 1994). There are numerous blood vessels, both capillary and larger vessels, within the avian skin and haemorrhage can be a problem, as it was with this case. When possible, incisions should be made with a radio or laser surgical instrument and the scalpel should not be used at all for most types of avian surgery. If the above mentioned instruments are not available, an incision can be made by slightly tenting the skin, nicking with scissors and then bluntly dissect with the scissors after crushing the skin with artery forceps along the line of the intended incision (Coles, 2007). After the bleeding stopped, the animal was collared and the leg bandaged to prevent further damage (Hess, 2002). No bone involvement was present, which is important because osteomyelitis is associated with a guarded prognosis (Harrison & Lightfoot, 2006), and the wound was healing nicely until "Sonny" removed the collar. Another collar was applied and since then there were no more complications on the right foot. Almost 1 month after initial presentation the left foot became hypertrophied with what seemed like a xanthoma formation. Xanthomatosis results from the accumulation of lipid-laden macrophages, giant cells, free cholesterol and variable degrees of fibrosis. Xanthomas often occur at the distal wing but have been found in other locations where they are locally invasive and wide margins may be necessary to completely excise and prevent recurrence. Some birds may mutilate these lesions, causing ulceration and secondary infection. Elevated serum cholesterol, trauma and genetic predisposition of some species have been implicated in the formation of xanthomas. Dietary correction may be curative in some species and individuals, however, very large, painful, haemorrhagic or infected xanthomas often require surgical resection (Coles, 2007) which was the case. Masses may be removed with radiosurgery, taking care to avoid damage to blood supply (Harrison & Lightfoot, 2006). The site may be closed if there is

enough remaining tissue or allowed to heal by second intention and bandaged with a hydroactive dressing. If extensive subcutaneous tissue and bone are involved, amputation of the affected area may be necessary (Bennet & Harrison, 1994 cited by Harrison & Lightfoot, 2006) which wasn´t the case. Both feet were healing nicely and "Sonny" seemed to improve until it was back for a recheck on Janurary 12th. The next day "Sonny" arrived as an emergency at GWR with acute dyspnoea. Tracheal obstruction can usually be differentiated from lower or generalized disease or dyspnoea of non-respiratory origin by the sound of the respiration (tracheal noise) and the forward-leaning, neck extended posture, as it was assumed by "Sonny". Airway obstruction is characterized by stridor and dyspnoea exacerbated by exertion and intervention is necessary to relieve obstruction. In severe cases, immediate placement of an air sac tube, as it was performed, is indicated (Lightfoot, 2008). Trachectomy was attempted because severe tracheal stenosis was detected probably following trauma due to repeated handling, intubation or other procedures that might have damage the tracheal wall at some point. Membranous stenosis is seen in birds following suspected tracheal damage caused by intubation and ventilation. This should be kept in mind when a bird presents with evidence of airway obstruction and a history of recent anaesthesia (Graham, 2004). "Sonny" died from anaesthetic complications related with the patency of the air sac tube. The air sac tube should always be kept clean to maintain its patency (N. A. Forbes, personal communication, December 21, 2010) and avoid anaesthetic complications although such a surgery has always a high risk associated (Harrison & Lightfoot, 2006).

Clinical case No.9

History and clinical signs

On January, 2011 a Swan (*Cygnus olor*) was examined at GWR (Fig. 18A). The bird was picked up that day with a fish hook in the neck. The hook was removed but a lump remained.

Diagnosis and treatment

On fluoroscopy a fragment of the hook was detected. An IV catheter was placed in the dorsal metatarsal vein and under GA (Fig. 18B), surgery for extraction of the metal fragment was performed. The necrotic area and abscess were shelled out and removed (Fig. 19A and 20A). Closure was performed with intradermal sutures (Fig. 19B) and the neck was bandaged. A single injection of enrofloxacin (*Baytril*® 2,5% Injectable solution, Bayer Corp., Leverkusen, Germany) at the dose rate of 30mg/Kg, IM was given. The bird recovered well (Fig. 20B).

Figure 18: A) Injured *Cygnus olor*; B) GA (Original picture).

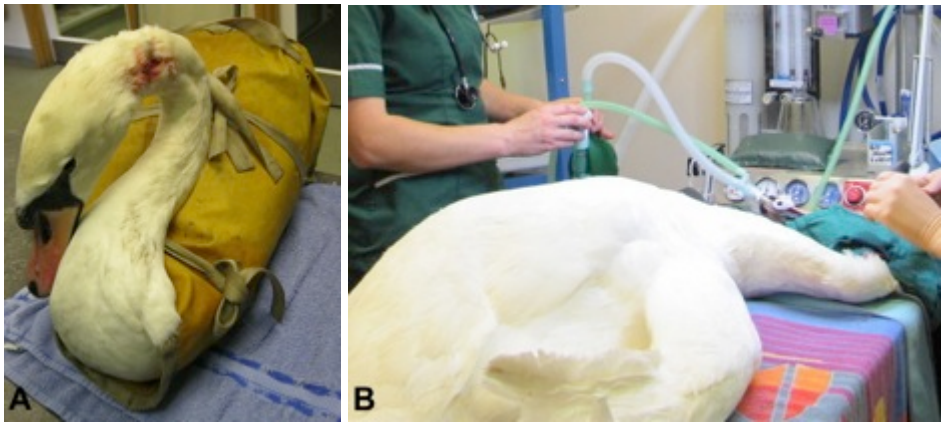


Figure 19: A) Abscess removal; B) Closure (Original picture).

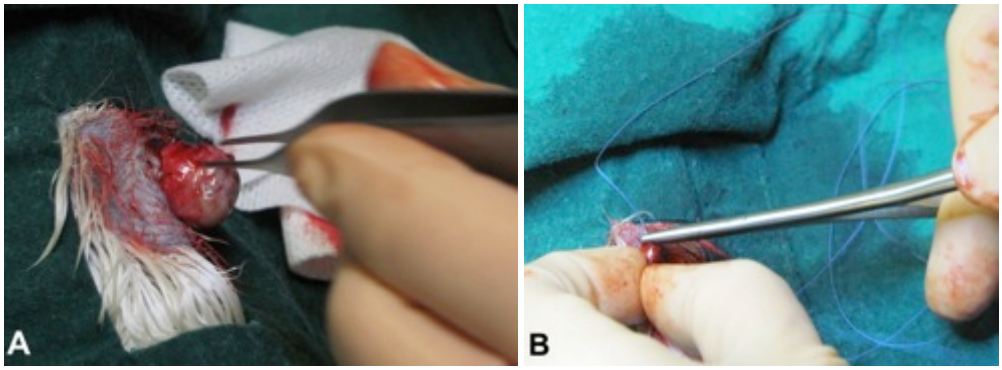
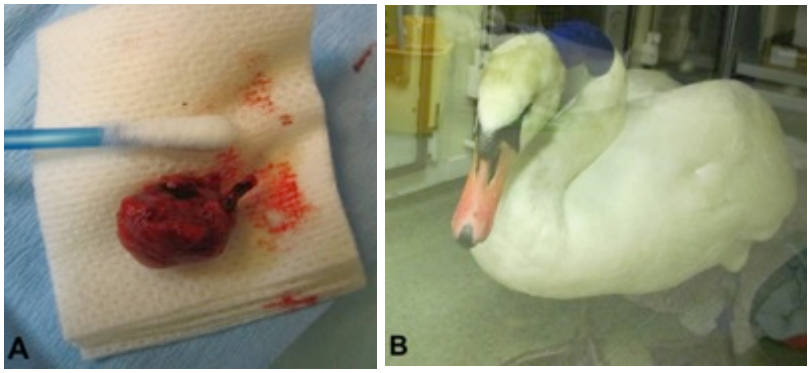


Figure 20: A) Removed abscess; B) Recovery (Original picture).



Discussion

Clinical case No.9 was a traumatic presentation due to a fishing hook. Wounds and other injuries may cause emergency presentations of birds. A thorough PE is essential to determine the extent of trauma and best approach for treatment. In such cases, the protocol should always be similar: prioritize therapy, cleanse wounds, examine wounds for any evidence of myiasis and stabilize fractures, if present, until patient stabilization allows surgical repair and more detailed treatment (Redig, 1996 and Graham & Heatley, 2007).

Since this was a wild bird, of a common species, the only aim was to return it to the wild with full function to survive (Forbes & Altman, 2010). Anseriformes, particularly swans, are prone to damage from fishing hooks and line. Hooks are commonly found embedded in the rhamphothecae, tongue, skin of the neck or the oesophagus. Some hooks are very small and may only be revealed by careful examination. If the hook cannot readily be dislodged, then, after location by palpation or radiography, it can either be removed surgically under local anaesthesia or left in place after cutting any attached line as short as possible (Waine, 1996). In this case fluoroscopy was used to detect the metal fragments in the cervical region of the swan, providing a faster and less stressful diagnostic imaging technique. A subcutaneous abscess formed after the injury. *Staphylococci* spp. are introduced through the skin from a puncture wound and form a caseous abscess, which cannot be drained. In the present case, the abscess was surgically removed, including the surrounding capsule because leaving the capsule could allow the abscess to reccur (Harcourt-Brown, 1996). The remaining space was obliterated with intradermal sutures. It should be noted that when removing the feathers of a wounded skin, it is better to cut them right next to the skin rather than attempt to pluck them since plucking feathers in such a situation can lead to tearing of the skin (Redig, 1996). The bird made a full recovery and by the time of removal of the bandage it was much brighter than on initial presentation and ready to be released.

Clinical case No.10 (Boyo)

History and clinical signs

On January, 2011 a 45 year old, male, Moluccan cockatoo (*Cacatua moluccensis*) was examined at GWR. "Boyo" presented sneezing with clear oral mucous discharge. "Boyo" had a history of coughing up water when drinking and now oral mucous discharge was present. The problem lasted for a month and was gradually worsening. The owner had noticed halitosis. "Boyo" lived with another parrot which had chlamydophilosis seven years ago but was treated with no recurrence. "Boyo" was previously on a seed diet but changed to a parrot mix of seeds and pellets, fresh fruit and vegetables. On PE "Boyo" was alert and responsive. Halitosis was obvious and a slight expiratory and inspiratory stridor (which was constant according to the owner) was noted.

Diagnosis and treatment

GA was performed for blood sampling and radiographs but prior to intubation, a mass that seemed like a massive aspergilloma was detected at the back of the glottis, not occluding the airway but occluding the pharynx by approximately 50-75% (Fig. 21). Initially "Boyo" was regurgitating so the mass was gently cleaned with a cotton bud but started to bleed. Samples

were taken for histopathology (Fig.22). "Boyo" was severely emaciated so it was suspected that it had not been able to swallow food due to the size of the mass.

Figure 21: A) Positioning for examination of the oral cavity; B) Mass (circle) at the back of the oral cavity (Original picture).

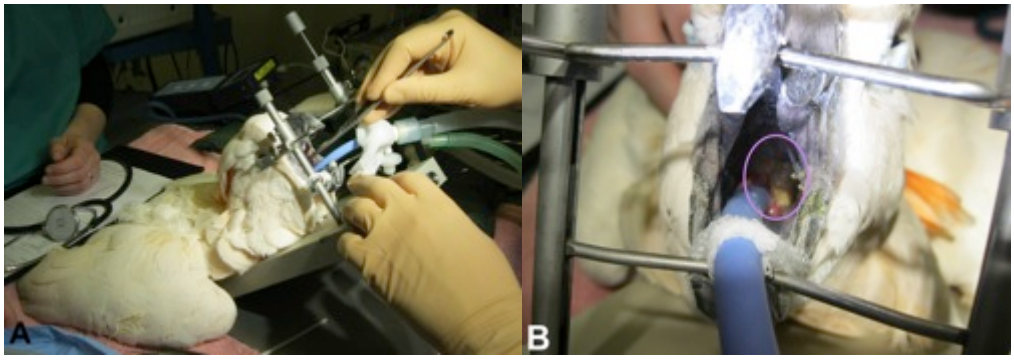
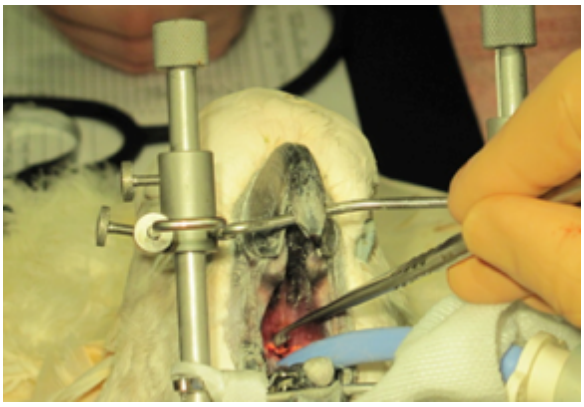


Figure 22: Removing a sample from the mass for histopathology (Original picture).



Differential diagnosis included aspergilloma, papilloma (possible to progress to malignant Squamous Cell Carcinoma - SCC), squamous hyperplasia or other neoplasias. SC fluids (10ml) were administered during GA. A blood sample was sent for *Chlamydophila* testing. *Chlamydophila* could not be ruled out and possible associated liver problems were present. "Boyo" was hospitalised. Laboratory results showed elevated uric acid levels possibly from dehydration despite a normal PCV. *Chlamydophila* testing with *Immunocomb*® (Biogal, Galed Laboratories, Kibbutz, Israel) revealed a high positive (5+) for chlamydophilosis. WBC count was $57.5x10^9$/L ($8-12x10^9$/L) (Fudge, 2000). Voriconazole (*VFEND*® 50mg film-coated tablets, Pfizer, Amboise, France) at the dose rate of 10mg/Kg, PO, BID and doxycycline (Vibramycin®, Pfizer, Amboise, France) at the dose rate of 25mg/Kg, PO, SID were started. During the next few days while in hospital "Boyo" was bright and eating, with minor respiratory sounds so was discharged. "Boyo" returned 3 days later depressed and wheezing. On PE the proximal oesophagus was inflammed, "Boyo" was anorexic, probably due to pain response. Euthanasia was performed after pathology report confirmed a SCC, ulcerated and inflamed. The diagnosis based on histopathology, was SCC ulcerated and

inflamed. The histology report (see annex I) was provided by IZVG Pathology, UK and was as follows:

> The 6 largest biopsy fragments are examined. All are similar. They comprise arborescent cords of poorly differentiated squamous epithelial cells, exhibiting partial central keratinisation and pearl formation in some cases. Cords are fragmented and numerous small clusters and individual cells bud into the intervening stroma. Cells exhibit marked anisocytosis and anisokaryosis, with nuclei having marginated chromatin and prominent nucleoli. Mitotic figures are frequent (approximately 4 per high power field). There is extensive surface necrosis, inflammation and colonisation by swarms of bacteria. Coagulative necrosis of the neoplastic cells themselves is also widespread. Neoplastic cells clusters are intimately associated with vascular channels in some biopsies (IZVG pathology, 2011).

Discussion

"Boyo" never had problems until one month before initial presentation. The halitosis and poor body condition were caused by a mass detected before intubation that was occupying a large portion of the pharynx, making it impossible for "Boyo" to eat adequately and causing it to regurgitate. Samples from the mass were taken and sent for histopathology evaluation. "Boyo" was positive for chlamydophilosis, which might have been transmitted from another parrot of the same household that had the disease years ago. The CS of avian chlamydophilosis range from asymptomatic carriers to severe disease and there are no pathognomonic signs (Flammer, 2007). "Boyo" was probably an asymptomatic carrier so doxycycline was prescribed. Voriconazole was initially prescribed while waiting for histopathology results since there was a suspicion that the mass could be an aspergilloma.

Necrosis and secondary bacterial infection were causing the halitosis. SCC are second to papillomas in frequency and may involve the oral cavity and tongue (Ritchie et al., 1994). The upper GI tract and skin are the 2 most common primary sites of SCC in pet birds, with this tumour most commonly diagnosed in cockatiels, Amazon parrots and budgerigars. These carcinomas appear as ulcerative-to-cauliflower-like, painful lesions or masses that are associated with inappetence, dysphagia, regurgitation, halitosis and frequent head shaking (Anderson & Steinberg, 1989; Chin & Barr, 1990 and Turrel, McMillan & Paul-Murphy, 1987 cited by Ricthie et al., 1994). Differential diagnosis for such lesions should include oral neoplasia, hypovitaminosis A, trauma, candidiasis or protozoal infection (trichomoniasis) (Ritchie et al., 1994). These tumours tend to be extremely locally aggressive and prone to recurrence, with few reports of metastasis. Complete excision is rarely accomplished (Harrison & Lightfoot, 2006). Radiation therapy has been attempted with some success, however, SCC appears to be an exceptionally radioresistant tumor and long-term control is rare. Anecdotal reports indicate that radioresistance may be even greater in birds than in mammals (Manucy, Bennet & Greenacre, 1998). Strontium therapy when tumor depth is not

a limiting factor has shown some promise in selected psittacine cases. Since distant metastasis is rare, chemotherapy is not commonly used. Photodynamic therapy (PDT) has been attempted in 2 reported cases. One case of a SCC in the beak of a hornbill showed a positive result in decreasing tumor size but failure to eliminate the neoplasia (Suedmeyer, 2001 cited by Harrison & Lightfoot, 2006). Another case demonstrated a positive response to PDT after each treatment, but treatments couldn´t be administered at regular intervals (Harrison & Lightfoot, 2006). According to the IZVG pathology report, the prognosis for SCC of the oral cavity is guarded, with 1 survey indicating that all of the birds with tumours of the beak, oral cavity or oesophagus ultimately died or were euthanased as a result of uncontrolled tumour growth or secondary infection. Considering the guarded prognosis and limited treatment options, "Boyo" was euthanized.

Clinical Case No.11 (Laura)

History and clinical signs

On January, 2011 a 17 year old, female parrot (*Psittacus erithacus*) was examined at GWR. "Laura" was egg binding, severely emaciated and had a greenish diarrhoea (Fig. 23A).

Diagnosis and treatment

Radiographs were taken (Fig. 24A) and revealed the presence of an egg and what seemed like an enlarged proventriculus so PDD was suspected. An IV catheter was placed into the basilic vein (Fig. 23B) and supportive care provided before extraction of the egg was attempted, including fluid therapy with 20% of an injectable solution of electrolytes, vitamins, amino-acids and dextrose (*Duphalyte®,* Fort Dodge Animal Health, Southampton, UK) + 80% Hartmann´s solution, pre warmed and mixed in the same syringe, IV, given at the dose of the estimated fluid deficits (10% BW) in ml plus maintenance (40ml/Kg/day) plus ongoing losses, injectable Ca gluconate (10%) at the dose rate of 100mg/Kg, IM and adequate warmth/humidity. Analgesia was provided with butorphanol tartrate (Torbugesic® Injection, Fort Dodge Animal Health, Southamptom, UK) at the dose rate of 1mg/Kg, IM. Faecal examination by wet mount was negative for parasites. After stabilization, intracloacal application of PGE_2 vaginal gel (Prepidil® Gel dinoprostone cervical gel, Pfizer, Amboise, France) at the dose rate of 0.02mg/Kg was performed. The gel was applied topically to the uterovaginal sphincter using a sterile, cotton-tiped applicator into the cloaca. After 2 hours, expulsion of the egg had not occurred so surgical intervention was necessary. The head was elevated to aid respiration and lubricants infused into the cloaca. Steady, unchanging digital pressure was applied between the end of the sternum and the cranial portion of the egg to cause its slow descent and mosquito forceps were carefully inserted into the uterine opening and gently spread to expedite dilation. This procedure was not successful so ovocentesis

was performed through the cloaca to aid in the passage of the egg. A 22G needle was attached to a 12ml syringe and directly inserted into the egg via a vent approach. The egg contents were aspirated (Fig. 24B), the egg imploded and was removed via the cloaca (Forbes & Altman, 2010).

Radiographs were taken to confirm that all the eggshell pieces were expelled. The uterus was then flushed with chlorhexidine to remove any shell fragments and decrease the incidence of metritis. *Immunocomb*® (Biogal, Galed Laboratories, Kibbutz, Israel) result was a weak positive for chlamydophilosis. The next day a blood sample was taken and PCV was 19% (45-53%) (Fudge, 2000) with a normal WBC count. The blood smear evaluation revealed a regenerative anaemia. A blood transfusion was performed from an healthy *Psittacus erithacus* from the hospital. By the end of the day "Laura" was not improving but was still responsive. While crop-fedding a swelling was palpable on the right side of the head under the beak with approximately 2-3cm diameter but medication was continued. During the night "Laura" was found dead on its cage with oral bloody discharge.

The next day a *post-mortem* examination was performed and many haemorrhages where detected, blood in the trachea, lungs and haemothorax (Fig. 25). There were also IM haemorrhages probably from the IM injections.

Figure 23: A) Diarrhoea of "Laura"; B) IV catheterization of the basilic vein (Original picture).

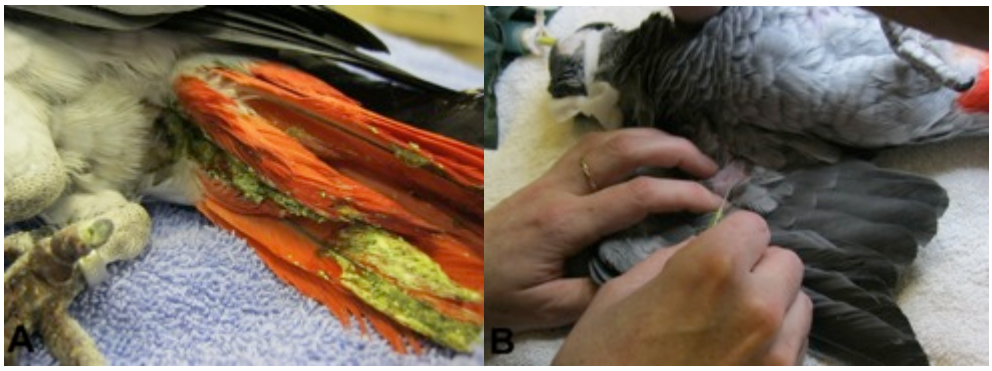


Figure 24: A) Positioning "Laura" for x-ray; B) Egg yolk removed by ovocentesis (Original picture).

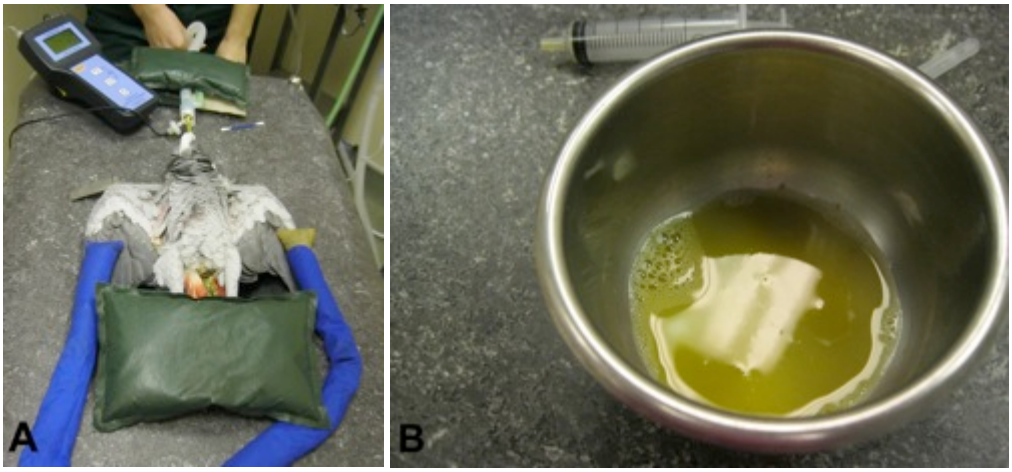
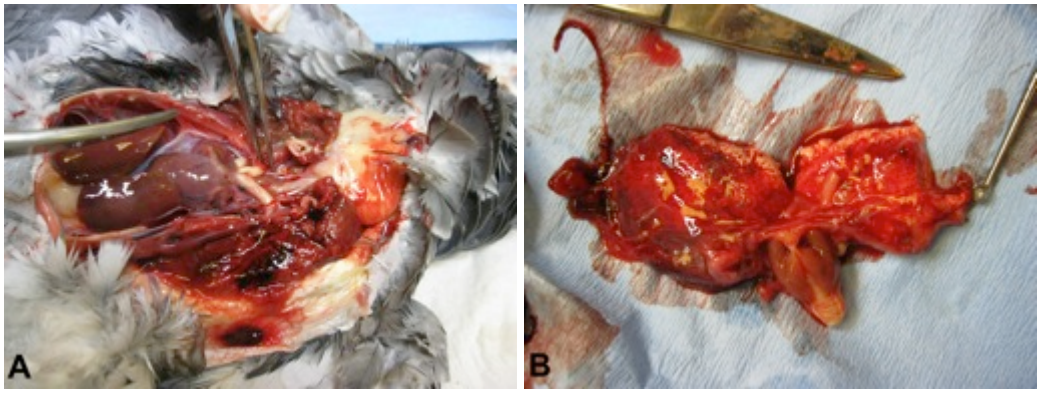
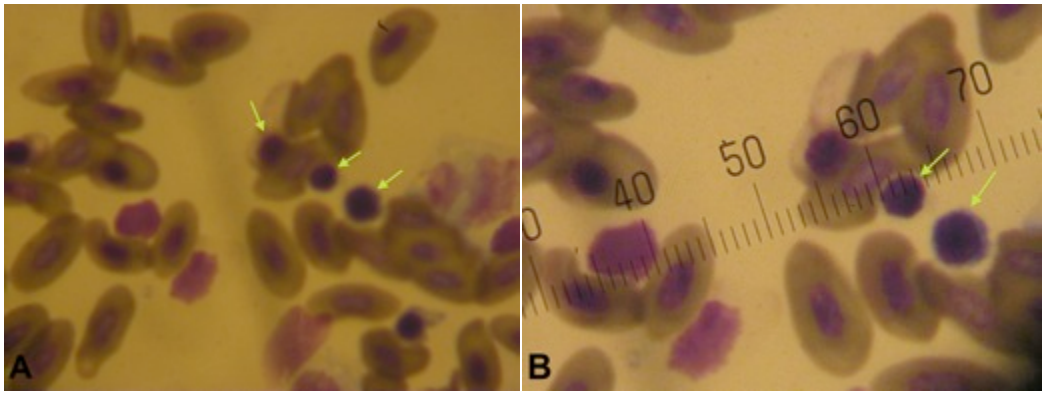


Figure: 25 A) and B) *Post-mortem* exam of "Laura" with multiple haemorrhagic foci (Original picture).



Impression smears were taken from the liver (which had an increased left lobe), spleen, lungs and from the contents of the proventriculus and cloaca. Proventriculus was not as dilated has expected with PDD so this was ruled out. The impression smears revealed inflammatory cells and increased number of thrombocytes (thrombocytosis) (Fig. 26). The swelling near the beak was dissected inside which there was a caseous pink-purple material that seemed old with no visible rupture from the oesophagus detected.

Figure 26: A) and B) Microscopic examination of the impression smears from "Laura" with an increased number of thrombocytes (arrows) under microscope magnification (x1000, oil immersion) (Original picture).



Discussion

Clinical case No.11 was a typical presentation of egg binding. "Laura" had paraparesis, decreased BW, depression and labored breathing, indicators of a poor prognosis. Radiographs were taken to determine the number of eggs present. One egg was detected and the proventriculus seemed enlarged so PDD was suspected (Campbell & Grant, 2010).

Initially, dystocias should be managed medically. Often dystocic birds are dehydrated, so stabilization and correction of any fluid deficits are extremely important, so IV fluids were given through an IV catheter (Clayton & Ritzman, 2006). Seed-eating, egg-laying females are often hypocalcaemic so parenteral calcium was administered (Campbell & Grant, 2010). PGE_2 vaginal gel acts similarly to arginine vasotocin, the hormone responsible for normal uterine contractions and relaxation of the uterovaginal sphincter in birds. Birds do not use the oxytocic system for uterine contractions so oxytocin should probably not be used in birds. Gloves should always be worn by humans handling this drug or treated birds to prevent contact with the skin or MM, particularly in women. Because this didn´t cause expulsion of the egg within 2h, surgical intervention was necessary. A 22G needle attached to a 12ml syringe was directly inserted into the egg via a vent approach and the egg contents were aspirated, the egg imploded and removed via the cloaca (Forbes & Altman, 2010).

Egg binding can be associated with chronic egg laying, oviductal muscle dysfunction secondary to excessive egg laying, hypocalcaemia, vitamin E and selenium deficiencies, malnutrition, obesity, inadequate exercise causing decreased muscle strenght, malformed eggs, mechanical tears or damage to the oviduct, oviductal or uterine bacterial infections, parasitic disease, ovarian or oviductal neoplasia and general systemic disease (Bowles, 2002 and Joyner, 1995 cited by Campell & Grant, 2010). Recommended management options were adopted and include supportive medical care, manual egg manipulation, medications to induce oviposition and ovocentesis. Endoscopy and abdominal surgery (Campell & Grant, 2010) may also be attempted if necessary. Supportive medical management is often successful in resolving cases of egg binding but if this does not result

in oviposition other options must be considered. Drugs may be used to induce oviposition such as PGE_2 gel, however, its use is contraindicated if the egg is adhered to the oviduct wall or if the egg´s passage is obstructed. Ovocentesis can be used to remove egg contents and allow collapse of the eggshell to facilitate passage. Ovocentesis is easily accomplished and is best done when the egg can be visualized from the vent, confirming its location in the oviduct as in this case. Oviduct biopsy was not performed because the tissue appeared normal, however, this doesn´t rule out disease and would be recommended in future cases (Clayton & Ritzman, 2006).

Diarrhoea is not a typical CS with egg binding. The differential diagnosis list for the emergency patient with diarrhoea includes gram-negative enteritis, hepatopathy, chlamydial infection and heavy metal toxicity (Campbell & Grant, 2010). Yellowish-to-greenish droppings are suggestive of liver involvement and the weak positive from the *Chlamydophila* test could explain the diarrhoea. Asymptomatic *Chlamydophila* spp. infections are characteristic in adult birds exposed to moderate numbers of a moderately virulent strain of *Chlamydophila*, these birds may shed the organism for several months while remaining asymptomatic until extreme environmental changes or concurrent infections activate persistent infections, resulting in the occurrence of clinical disease. In this case egg binding was the inciting factor (Ritchie et al., 1994). Clinical pathology findings associated with chlamydophilosis and consistent with this case include increased number of inflammatory cells with normal heterophils and anaemia (Mallison, 1989 cited by Ritchie et al., 1994). Gross lesions seen at *post-mortem* mainly involving the liver and respiratory system could also be explained by this condition. Acute lesions are characterized by hepatomegaly, fibrinous peritonitis, air sacculitis, perihepatitis, pericarditis, bronchopneumonia, enteritis and nephrosis. Splenomegaly wasn´t detected and may not occur with chlamydophilosis at all (Harrison & Lightfoot, 2006). Pacheco´s disease virus (PDV) can also cause diarrhoea. Biliverdin staining of liquified faeces is indicative of the severe liver necrosis caused by the virus. Many outbreaks are linked to a stressful event such as a change in the environment or the onset of breeding season. Stress factors are thought to induce recrudescence in asymptomatic carriers resulting in virus excretion (Ritchie et al., 1994), but *post-mortem* examination findings were not consistent with such a condition and although the liver was enlarged it was not as damaged as it was expected with PDV. Even after removal of the egg and supportive care " Laura" wasn´t improving and PCV was still low so a blood transfusion was performed. Anaemia can be caused by blood loss, heavy metal toxicosis, parasitic infection and chronic disease (Shaw, Tully Jr. & Nevarez, 2009). The blood smear evaluation revealed a regenerative anaemia that can be caused by haemorrhage or haemolysis (Tully Jr. & Beaufrère, 2011). A whole blood transfusion was performed. Crossmatching is often recommended before transfusion, however, it does not appear to be an accurate means of predicting transfusion reactions in avian species. Most birds have not received a previous blood transfusion, therefore, a single transfusion from a

donor bird can be given in an emergency situation. In this case a homologous transfusion (donor and recipient are the same species) was performed. When a blood transfusion from the same species of bird is not possible, blood from a donor of a closely related species may have the longest therapeutic effect (Shaw et al., 2009). Blood collection from donors must be performed aseptically (Lichtenberger, 2004 cited by Shaw et al., 2009). The jugular vein is the collection site of choice in most donor birds and a butterfly catheter is the preferred means of blood collection, such as it was performed in this case. Blood can also be collected from the basilic and dorsal metatarsal veins. Blood should be warmed before administration to prevent hypothermia (Uhl, Pacini & Kruskall, 1992) and can be administered through a catheter in the jugular, basilic or dorsal metatarsal vein (Shaw et al., 2009). In this case the basilic vein of the recipient was used. The total blood volume needed to raise avian PCV to a desired level has not been established and clinicians are often limited by the amount of blood that can be safely collected from the donor bird. A dose of 1% BW or 10% of blood volume has been suggested (Hoefer, 1992). Transfusion reactions that have been reported in birds include regurgitation, haemoglobinuria and death (Altman, 1982 cited by Shaw et al., 2009). In an emergency situation, complete screening of donors may not be practical because rapid on-site tests are not available and would delay the transfusion. Most commercial blood banks do not store avian products so veterinary hospitals may benefit from pre-screening avian donors (Shaw et al., 2009). After the transfusion "Laura" seemed to improve but during crop-feeding a mass was felt under the beak. On *post-mortem* it was found that the mass was isolated and had no relation with the oesophagus, since the initial concern was it´s rupture. Hypovitaminosis A can lead to squamous metaplasia of the oropharyngeal epithelium, particularly glandular epithelium, leading to plaque and granuloma formation. In psittacines this typically involves the submandibular or lingual salivary glands. Sometimes affected birds exhibit a subcutaneous swelling caudal to the mandible with affected birds typically being fed a seed-based diet as in this case (Hargis, 1991 cited by Harrison & Lightfoot, 2006). The impression smears revealed inflammatory cells and thrombocytosis. Coagulopathies are described for other companion animal species as causes of blood loss. There is currently no standardized testing for coagulopathy in birds, therefore, primary coagulopathies have not been reported in avian species but only secondary coagulopathies associated with toxins (Leighton, Peakall & Butler, 1983 and Katavolos et al., 2007 cited by Shaw et al., 2009). Avian thrombocytes play a primary role in haemostasis in a manner similar to mammalian platelets. They may also have a phagocytic function and participate in removing foreign material from the blood (Ritchie et al., 1994). The thrombocytosis detected was possibly a response to a bacterial infection or the result of excessive haemorrhage (Coles, 2007). Excessive heamorrhage was probably the cause of the severe anaemia but it was only detected after the bird was found dead on its cage with oral bloody discharge and during *post-mortem* exam where multiple haemorrhagic foci were detected. The haemorrhage could

have been caused either by a primary or secondary coagulopathy. The former, as mentioned above, was never reported in birds so a secondary coagulopathy as a result of the blood transfusion might have been the cause of death, leading to bleeding into potential spaces such as the peritoneum, pericardium, respiratory tract and GI tract. Clotting disorders are rare in birds making clinical research of haemostatic disorders difficult in avian species (Fudge, 2000).

Clinical case no.12

History and clinical signs

On January, 2011 a 5 year old, female Regent parrot (*Polytelis anthopeplus*) was examined at GWR. The bird had flown into a ceiling fan the previous night and was hurt on the right leg which had an outward deviation. Initially the bird seemed depressed but as time progressed started to be more alert, responsive and using the right leg. In the morning the parrot was sitting on its perch resting the affected leg but not using it and not putting weight on it.

Diagnosis and treatment

On PE a fracture of the tibiotarsus was detected, probably caused by the ceiling fan trauma, the owner was offered surgery or splint options and opted for splinting due to costs (Fig. 27A).

Figure 27: A) The bird´s leg after the traumatic event; B) Material used for splinting: 1 - Technovit®6091 (Electron Microscopy Sciences, Hatfield, UK); 2 - Mixing the liquid and solid part of Technovit®6091; 3- A common rubber tube and wood applicator (Original picture).

Figure 28: A) The splint after molding it to the bird´s leg and filling with Technovit®6091; B) Applying the splint (Original picture).

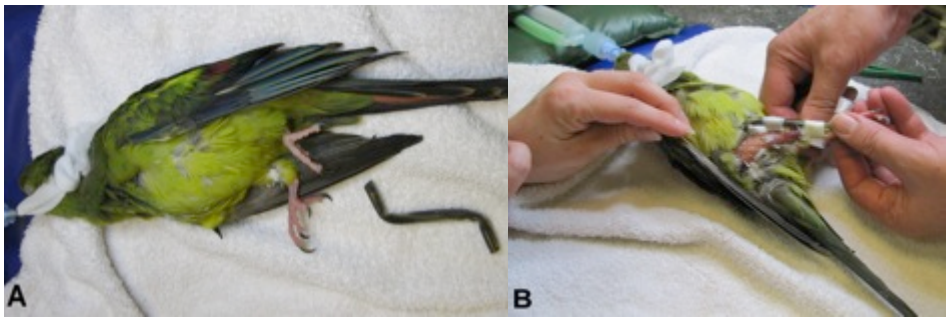
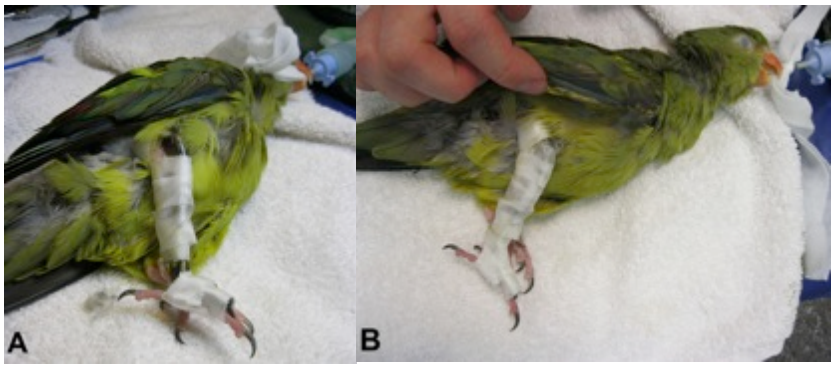


Figure 29: A) and B) Bandaging and splinting the leg (Original picture).



Under GA a splint was applied, made out of a rubber tube and a wood applicator which where filled with Technovit®6091 (Electron Microscopy Sciences, Hatfield, UK), a synthetic resin used for numerous treatments in veterinary medicine (Fig. 27B), and shaped to the bird´s leg, making the middle portion as long as the tibiotarsus, one edge as long as the tarsometatarsus and the other edge supporting the femur. The leg was held in a functional position, the intertarsal joint flexed in the normal standing position for the bird species until the filling material hardened (Fig. 28A). The splint was bandaged around the leg in a functional position (Fig. 28B and 29). Vetrap™ (3M Animal Care, Diegem, Belgium) was then wrapped around the bandage. The owner was advised to return in 10 days and then 24 days, however, only 2 days later the bird was back with blood seeping through its bandage. The bleeding started that morning but the blood was now dry, otherwise the bird was bright and starting to put pressure on the affected leg. The bird was rebandaged under GA. Enrofloxacin (*Baytril*® Oral Solution 2,5%, Bayer Corp., Leverkusen, Germany) at the dose rate of 15mg/Kg, PO, SID and meloxicam (*Metacam*® Oral Suspension, Boehringer Ingelheim Vet-medica/Merial, Berkshire, UK) at the dose rate of 0.1 mg/Kg, PO, SID were prescribed. After 3 weeks the bird returned to remove the bandage and was using the leg normally.

Discussion

Clinical case No.12 was a traumatic presentation. Fractures alone are not usually life threatening but stress associated to it can be in a debilitated bird (Bowles et al., 2007). Birds with fractures present as great challenges because avian bones have a wide medulla and thin cortex (minimal weight, maximal flight ability). Avian bones are brittle and surrounded by only a small amount of soft tissue, hence many fractures are compound and prone to dissecation. The approach to an avian orthopaedic case, requires initial consideration of some factors: is it a wild or a captive bird, is complete repair and return to normal function possible and what degree of functional disability is acceptable for the species and individual bird to live a compassionate future life (N. A. Forbes, personal communication, December 21, 2010). When repairing an avian fracture, the aims should be (in chronological order): (i) Treat contaminated or infected wounds; (ii) Preserve soft tissue, if necessary by applying splints or dressings. Special care is required to prevent dessication due to the fragility of avian skin and small soft tissue mass; (iii) Realign fractures or replace luxations; (iv) Rigidly stabilize the fracture site, preventing any movement or rotation while, if possible, maintaining joint function and movement during healing, and (v) return the limb to normal function with the aid of physiotherapy and controlled exercise (Forbes & Altman, 2010). After due consideration, if the case is to be treated rather than euthanased, the condition of the bird must be assessed. Many birds, which have undergone sufficient trauma to fracture bones will at the same time have suffered other considerable soft tissue damage. Pre-anaesthetic screens can be performed and supportive care administered if appropriate (N. A. Forbes, personal communication, December 21, 2010). Fluid inbalances must be corrected, the patient stabilised, analgesia and antibiosis administered, prior to surgery or splinting.

Methods of fracture repair include external coaption, internal fixation or external fixation. In this case, external coaption (splints, extension splints, bandages) was performed, which is frequently used for small or younger birds. Bone healing in birds, if bones are correctly aligned and opposed, is done by endosteal callous. If not rigidly fixed, periosteal callous will also form. Stable properly aligned fractures heal more rapidly than in mammals being fully stable in 3-4 weeks (N. A. Forbes, personal communication, December 21, 2010). In this case only the tibiotarsal bone was affected with a closed fracture and the owner opted for splinting (external coaption) and supportive care. Healing without internal fixation can lead to malunion and shortened bone (Myers, Bailey & Davidson, 2006). Also, with conservative treatment, problems with soft tissue healing may develop such as joint ankylosis, muscle atrophy, tendon contracture or entrapment of tendons or ligaments within the callous (Myers et al., 2006) but considering the location of the lesion and since it was a small psittacine, bone was too small for any orthopaedic repair. When splinting, the joint should be immobilised above and below the fracture for stabilisation. Tarsometatarsal fractures are easily diagnosed by palpation and can be supported with a tape splint in birds less than

300g. The pelvic limb was splinted in a normal perching flexed position with porous tape, creating enough stabilisation to allow healing (Chavez & Echols, 2007). The bandage had incorporated support material to properly immobilise the fracture. The bird came back few days later with blood on the bandage but no active haemorrhage was noted and the leg was rebandaged. The skin might have slough or the fracture might have open. The owner of the bird was advised to use smooth-sided cages without perches to prevent climbing during the healing period (Chavez & Echols, 2007) but no more problems were detected. Removal of the bandage was performed 3 weeks after the incident and care was taken during removal of the tape bandages to avoid damage to the skin (Chavez & Echols, 2007), the bird was using the leg normally.

Chapter 4. Conclusion

Increasing numbers of exotic animals are being kept as pets and owners want to receive the same high quality medical care as given to dogs and cats, so a basic knowledge of emergency and critical care of exotics should become a concern to emergency veterinarians.
The previously described cases try to serve as an example of what the clinician may have to deal with in terms of avian emergency and critical care seen at practice and what can be done considering diagnostic and treatment options for such cases. Emergency and critical care is an extensive field that can involve different organ systems hence diagnostic and treatment options can be a challenge to the clinician.
The previously described clinical cases involved the musculoskeletal, respiratory, GI, cardiovascular, reproductive and excretory systems with initial presentations varying from specific CS to profound weakness and collapse. Although each system should be addressed in a particular way, species and individual variability/susceptibility should be kept in mind because each case is unique and requires careful consideration.
An important principle to keep in mind, that increases significantly the survival rates of arriving emergency avian patients is that stabilization on initial presentation is more urgent than making a definitive diagnosis and in fact, oxygen, fluid therapy, heat support and analgesia save more exotics that any other drugs or treatments.

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Annexes

Annex I

IZVG Pathology

Telephone: 01535 692000 Fax: 01535 690433

11-0410

IZVG number:	11-0079	**Owner:**	Lowton
Vet submitting sample: N. Forbes, Great Western Referrals, Avian and Exotic Service, Unit 10, Berkshire House, County Park Business Park, Shrivenham Road, Swindon, SN1 2 NR, Tel: 01793 603800		**Animal ID:** **Species:**	Boyo Moluccan cockatoo Cacatua moluccensis
Date received:	19/01/2011	**Age:**	45 years
Date reported:	21/01/2011	**Sex:**	Male

Brief clinical summary:
Mass caudal to glottis.

Histology Report

Description:
The six largest biopsy fragments are examined. All are similar. They comprise arborescent cords of poorly differentiated squamous epithelial cells, exhibiting partial central keratinisation and pearl formation in some cases. Cords are fragmented and numerous small clusters and individual cells bud into the intervening stroma. Cells exhibit marked anisocytosis and anisokaryosis, with nuclei having marginated chromatin and prominent nucleoli. Mitotic figures are frequent (approximately 4 per high power field). There is extensive surface necrosis, inflammation and colonisation by swarms of bacteria. Coagulative necrosis of the neoplastic cells themselves is also widespread. Neoplastic cells clusters are intimately associated with vascular channels in some biopsies.

Diagnoses:
1. Squamous cell carcinoma ulcerated and inflamed.

Comments:
The upper gastrointestinal tract and skin are the two most common primary sites of squamous cell carcinoma in pet birds, with this tumour most commonly diagnosed in cockatiels, Amazon parrots and budgerigars. These tumours are locally aggressive and prone to recurrence, with few reports of metastasis. The prognosis for squamous cell carcinoma of the oral cavity is guarded, with one survey indicating that all of the birds with tumours of the beak, oral cavity or oesophagus ultimately died or were euthanased as a result of uncontrolled tumour growth or secondary infection.

Reavill DR. Pet bird oncology. Proceeding Association of Avian Veterinarians, Avian Specialty Advanced Programme, Orlando, Florida (2001) pages 29-43.

Pathologist: M.F. Stidworthy MA VetMB PhD FRCPath MRCVS 11-0410

Annex II

IZVG Pathology

Telephone: 01535 692000 Fax: 01535 690433

11-0410

IZVG number:	11-0410	**Owner:**	Rawlings
Vet submitting sample:		**Animal ID:**	Ekky
N. Forbes, Great Western Referrals, Avian and Exotic Service, Unit 10, Berkshire House, County Park Business Park, Shrivenham Road, Swindon, SN1 2 NR, Tel: 01793 603800		**Species:**	Eurasian eagle owl Bubo bubo
Date received:	25/03/2011	**Age:**	20 years
Date reported:	01/04/2011	**Sex:**	Female

Brief clinical summary:
Chronic hepatitis and leucocytosis. Follicles on ovary, one appeared necrotic. Previous IZVG ref: 10-1769

Histology Report

Description:
Liver: Normal architecture is largely replaced by irregular tracts of bridging fibrosis with hyperplastic sprouting bile ducts, isolated clusters of hepatocytes embedded in fibrous tissue, and occasional more distinct regenerative nodules. Within the fibrous tissue there are moderate multifocal to coalescent infiltrates of heterophils, lymphocytes, plasma cells and monocytes/macrophages. Small numbers of haemosiderin-laden macrophages are also present. The capsule is irregularly and markedly thickened in some areas.
Ovary: An enlarged follicle contains large amounts of degenerate/necrotic eosinophilic debris with residual vitelline granules and focally extensive recent intra-follicular haemorrhage. The degenerate material is interspersed with lipid-laden foamy macrophages, and surrounded by a fibrous capsule of variable thickness that is continuous with fibrous tissue in the adjacent ovarian stroma.

Diagnoses:
1. Severe chronic-active hepatitis with fibrosis, biliary hyperplasia and nodular regeneration ("cirrhosis"), liver.
2. Folliculostasis with necrosis and recent ovarian haemorrhage, ovary.

Comments:
The histological lesion in the liver is identical to that of chronic-active hepatitis as described primarily in psittacine birds. In this case, as in many others, the chronicity of the lesion obscures the likely aetiology. Aflatoxin exposure is frequently speculated to be involved, as well as other bile-excreted toxins, whilst chronic infectious diseases including chlamydiosis and some viral infections have been implicated in parrots. I cannot see any evidence to suggest either of the latter.
The ovary includes a necrotic follicle within which there is recent haemorrhage. There is no evidence of bacteria or significant heterophilic inflammation here, and this may be a sterile degenerative process in a yolk laden follicle that has failed to ovulate. It seems unlikely that this is the underlying cause for the hepatitis.

Pathologist: M.F. Stidworthy MA VetMB PhD FRCPath MRCVS 11-0410

Annex III

Emergency Drugs Dosage Chart

	Dopram inj 1ml/Kg IM IV IO CPR	Atropine inj 0.8ml/Kg IM IV IO IT CPR	Adrenaline 1ml/Kg IV IM IO IT CPR	Diazepam 0.1ml/Kg IV IM Seizures	Diazepam 0.2ml/Kg IV IM Seizures
100g	0.1ml	0.08ml	0.1ml	0.01ml	0.02ml
150g	0.15ml	0.12ml	0.15ml	0.015ml	0.03ml
200g	0.2ml	0.16ml	0.2ml	0.02ml	0.04ml
250g	0.25ml	0.2ml	0.25ml	0.025ml	0.05ml
300g	0.3ml	0.24ml	0.3ml	0.03ml	0.06ml
350g	0.35ml	0.28ml	0.35ml	0.035ml	0.07ml
400g	0.4ml	0.32ml	0.4ml	0.04ml	0.08ml
450g	0.45ml	0.36ml	0.45ml	0.045ml	0.09ml
500g	0.5ml	0.4ml	0.5ml	0.05ml	0.1ml
550g	0.55ml	0.44ml	0.55ml	0.055ml	0.11ml
600g	0.6ml	0.48ml	0.6ml	0.06ml	0.12ml
650g	0.65ml	0.52ml	0.65ml	0.065ml	0.13ml
700g	0.7ml	0.56ml	0.7ml	0.07ml	0.14ml
750g	0.75ml	0.6ml	0.75ml	0.075ml	0.15ml
800g	0.8ml	0.64ml	0.8ml	0.08ml	0.16ml
850g	0.85ml	0.68ml	0.85ml	0.085ml	0.17ml
900g	0.9ml	0.72ml	0.9ml	0.09ml	0.18ml
950g	0.95ml	0.76ml	0.95ml	0.095ml	0.19ml
1Kg	1ml	0.8ml	1ml	0.1ml	0.2ml

(Adapted from Carpenter, 2005)

Annex IV

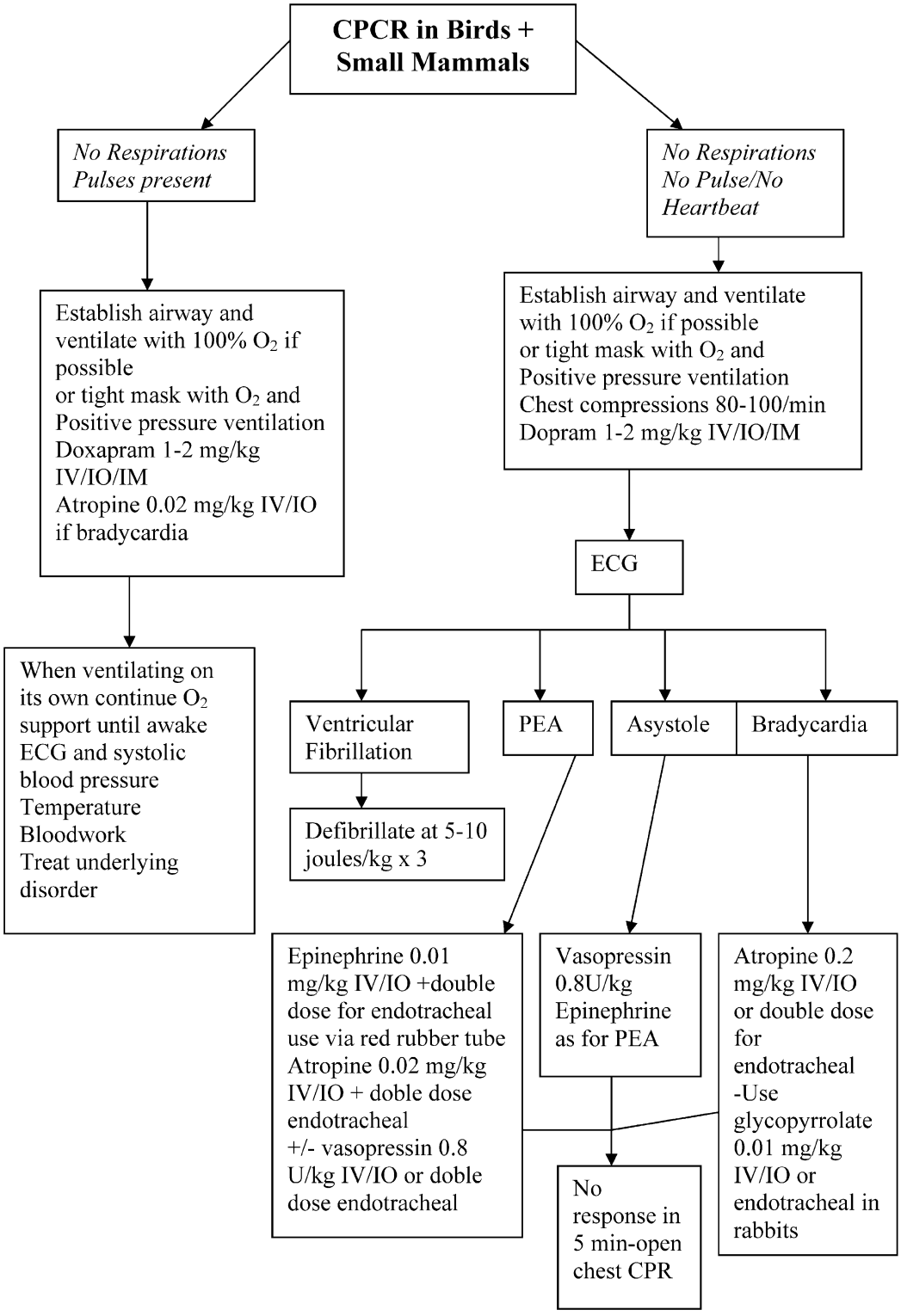

Fig. 1. Cardiopulmonary-cerebral resuscitation in birds and small mammals.

(Adapted from Lichtengerger, 2007b)