Emergency and Critical Care of the Avian Patient

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Summary

Many people are of the opinion that birds die easily. There are three main reasons for this belief. Firstly, many bird owners are ignorant of the clinical signs of illness in their pets. Secondly, birds tend to exhibit a "preservation reflex"- compensating for and disguising any abnormalities to avoid attracting attention from predators and flock mates. Often, then, a bird is only noticed as sick when it has reached a stage of decompensation in a chronic disease. Lastly, while the field of small animal emergency medicine and critical care is burgeoning, the equivalent in avian medicine is restricted by a lack of research.

The aim of this paper is two-fold. The Emergency Medicine section is designed to give a logical outline of procedures that can be used by the practitioner presented with an avian emergency. It covers most of the therapeutic techniques utilised to stabilise a bird until a definitive diagnosis can be reached. The Critical Care section details monitoring methods for assessment of the critical avian patient, and describes selected techniques employed in this field.

A **Introduction**

Although the ownership of birds as companion animals is increasing, it is fair to say that many people have not been closely associated with avian species, and do not have a well-developed appreciation of normal avian behaviour. This leaves them unable to recognise abnormal behaviour, and subtle clinical signs of disease.

Complicating matters is the "preservation reflex" shown by many birds, which suppresses the clinical manifestation of disease until a stage of decompensation is reached.

These two factors lead to the presentation of many chronic diseases in birds as emergency conditions. To exacerbate the situation, although small animal emergency and critical care is becoming a recognised specialty area, avian emergency and critical care still suffers from a lack of publicity and a relative deficit in research.

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This paper attempts to give an overview of the current body of knowledge in the field of avian emergency and critical care.

The first section - emergency medicine - will discuss the initial assessment and systematic stabilisation of the emergency avian patient. Attention will then be turned to the collection of common diagnostic samples, before the several supportive therapies are considered.

The critical care section deals with care of the avian patient after emergency stabilisation is achieved. It is designed to provide a guide to selected therapies in long-term supportive care, and will discuss techniques used to monitor the patient's progress.

B **Emergency Care**

1 **Initial Assessment**

This section will cover the usual procedure for assessment of an ill bird. If a patient is presented with an immediately life-threatening condition, the complete physical examination would be delayed until the bird was stable enough to allow time to continue with a thorough history, environmental assessment and physical examination. Procedures for emergency stabilisation are covered in section B2. If possible, the client should be instructed to bring the bird in its usual cage. Although the cage should not be cleaned prior to coming in, the water dish and any moving toys should be removed (Cannon, 1994).

While the bird is settling down, the history should be taken. The information gathered should include the bird's environment (aviary versus caged, description of conditions, periods of free flight, any recent changes), the signalment (age, sex, species), duration of ownership and source, diet (current and any recent changes), any new introductions, behaviour (usual and any changes), history of illness (current, other illnesses, any other birds or family members ill) and any medication provided (Cannon, 1994).

Throughout the history-taking, the bird can be assessed surreptitiously. Demeanour, activity, posture, obvious physical abnormalities and respiratory rate and depth can be assessed, and a subjective estimation of the bird's weight can be made. Allowance should be made for the "preservation reflex" (Cannon, 1994).

After taking the history the bird's immediate environment should be examined for hygiene, type of foods and/or supplements being offered and any discharges from the bird (vomitus etc). The number of droppings should be estimated, and the colour, consistency and amount of undigested material

compared with normality for that species. The faecal (usually solid), urates (solid) and urine (liquid) components of the dropping should be evaluated. A transient polyuria due to the stress of transport can be expected (Cannon, 1994).

Following this, physical examination can proceed if the bird appears capable of surviving the stress, otherwise oxygen should be provided until the bird is more stable (Jenkins, 1987). The delivery of oxygen will be discussed in section B.2.a.

It is imperative to complete the physical examination quickly, but thoroughly, to minimise the stress to the patient. To this end, all anticipated equipment (eg. medication, swabs etc) should be assembled prior to handling the bird. Birds may lose up to four degrees Celsius during a clinical examination (Verkest, 1994b).

Cannon (1994) recommends beginning physical examination with the head. Eyes, ears, nares and adjacent feathers are inspected for symmetry and evidence of discharges. Erosions, malformation or bruising of the beak should be noted. The mouth should be opened, and the tongue, choana, choanal papillae and pharynx surveyed. The entire ventral portion of the bird is now palpated, beginning with the crop. This procedure should not cause pain in the normal bird. Extra care should be taken if the abdomen is enlarged. A prominent keel indicates poor nutrition/chronic disease. In the abdomen the gizzard may be felt, as can an enlarged liver. The vent is surveyed for swellings, soiling or crusting. Feathers are examined, and flight feathers can be transilluminated. Alcohol can be sprayed on skin to aid visualisation. The wings should be palpated, and each joint should be assessed for range of motion. The legs are examined similarly, but with attention paid to the strength of grip of each toe, length of claws and the plantar aspect of each foot. Lastly, the respiratory system and heart should be auscultated (Cannon, 1994).

Although many workers routinely assume 10% dehydration for all ill birds, it is important to know common parameters used' for assessment of hydration. Moderate dehydration is evidenced by smaller than normal diameters, and filling times greater than 1 to 2 seconds in the ulnar artery and vein, "tenting" of the skin over the dorsum of the tarsometatarsus, dry mucous membranes and decreased sliding of the skin over the sternum (Redig, 1984; Abou-Madi and Kollias, 1992; A. Gallagher, pers. comm.). These signs, along with decreased skin turgor, sunken eyes and thick, stringy mucous in the caudal pharynx, signify severe dehydration, as can central nervous system depression, lethargy and weakness (Kaufman, 1992; Abou-Madi and Kollias, 1992).

Objective confirmation of dehydration is done via laboratory testing. This will be discussed in section **B.4.a.i.**

Common sample required for diagnosis are listed in section B.3. These, along with a pre-treatment weight can be taken now. If at any time, the bird appears unable to tolerate the stress of handling, it should be returned to its cage, oxygenated and allowed to recover. Section B.2 covers patient stabilisation by system. Once examination is complete, the bird is returned to its cage, and its tolerance to the procedure assessed (Cannon, 1994; Quesenberry and Hillyer, 1989).

2 First Aid and Stabilisation

A complete discussion of each emergency condition that occurs in birds is beyond the scope of this paper. It will endeavour to cover, however, the major first aid treatments involved with each body system. Once the patient is sufficiently stabilised, specific treatment required for the presenting condition can occur.

a Respiratory

Dyspnoea can be due to primary respiratory disease or extra-respiratory disease. The latter category causes respiratory distress by interfering with the normal air flow, or limiting expansion of lungs and air sacs. Regardless of the cause, the immediate objective is to relieve the dyspnoea enough to allow sufficient blood oxygenation. Such techniques will be discussed in this section. The technique of cardiopulmonary rescucitation will be covered in the Cardiovascular section (B.2.b).

Any bird that presents with rapid or difficult breathing should be placed on supplemental oxygen (Quesenberry and Hillyer, 1994). Small oxygen cages are commonly used, but can take some time to reach an adequate oxygen concentration. Flow-by oxygen directed at the nares, or into the mouth, can be useful (in small mammals, flow rates of up to 5-10L/min are used) (Crowe, 1995a). In a comatose or anaesthetised bird, endotracheal intubation (with an uncuffed tube) or a face mask can be used, but this may be too stressful on a conscious bird (Philip, 1981).

Transtracheal illumination will localise some tracheal foreign objects or obstructions (Ritchie, 1990). In any case of tracheal obstruction, abdominal air sac cannulation can be used. Rosskopf and Woerpel (1990) describe an emergency procedure for use in cyanotic patients. The bird is grasped, its abdomen is quickly cleaned with alcohol, a skin incision is made with a scalpel blade, and mosquito forceps are used to thrust into the abdominal air sac. The opening is maintained with the forceps until the patient is stable enough to have a tube (any small plastic or rubber catheter) sutured in place

with non-absorbable sutures. This is usually performed on the left abdomen, immediately caudal to the last rib, although any site overlying an air sac could be used. Interclavicular air sac cannulation has been used in raptors (Redig, 1992).

Dyspnoeic birds may benefit from diazepam or a light diazepam/ketamine combination to decrease their anxiety (Quesenberry and Hillyer, 1989).

If respiratory distress is suspected due to the extension of a wound into an air sac, the site should be covered, and supplemental oxygen provided (Kaufman, 1992). Some workers advocate using water-soluble cream underneath the bandage to provide a better seal (Jenkins, 1987).

Flushing of the sinuses with normal (0.9%) saline and/or antibiotic can assist breathing in patients with upper respiratory tract infections. By removing debris from the sinus and nares, it allows the bird to breathe more easily, whilst speeding the resolution of the problem (Quesenberry and Hillyer, 1994). The solution should be administered gradually via alternate nostrils with the head down. Swabs for gram staining and culture should be taken before treatment (Vogelnest, 1994). Enrofloxacin at 15mg/kg twice daily is one antibiotic recommended for this procedure (Rosskopf and Woerpel, 1991).

Ascites induced dyspnoea can be lessened by abdominocentesis performed with a 21-25 gauge needle on the ventral midline, just caudal to the tip of the sternum. Care must be taken to avoid an iatrogenic hypoproteinaemia from excessive loss of ascitic fluid (Ritchie, 1990).

b Cardiovascular

Goals for emergency management of the cardiovascular system are cessation of bleeding, ensuring sufficient blood volume for tissue perfusion and ensuring cardiac function. Unfortunately, avian cardiology is in its infancy, so emergency treatment of impaired cardiac function is probably limited to a single intramuscular dose of frusemide at 0.05mg/300g (Quesenberry and Hillyer, 1994; Clubb, 1986).

Bleeding can be controlled with direct pressure, electrocautery, vessel ligation, styptics (eg silver nitrate or ferrous sulphate) or thromboplastin (Kaufman, 1992). Blood feathers are a common cause of blood loss. These can result from excessively severe feather clipping or trauma to new feathers. Treatment involves removal of the feather by gently pulling in the direction of feather growth. Any further bleeding can be controlled as described above (Quesenberry and Hillyer, 1994).

In birds with suspected coagulopathies, vitamin K may be given at 0.1 ml/100g bodyweight (Rosskopf and Woerpel, 1987; Rosskopf, Woerpel, Albright, Shindo and Kibota-Sharp, 1991).

Unlike mammals, restoration of blood volume can revive birds suffering from acute blood loss, even in the terminal stages. In response to blood loss, or dehydration, birds change pre- and post-capillary pressure, so that hydrostatic pressure is decreased. This causes fluid to be drawn from the interstitial space into the vascular compartment. This is in part replaced by intracellular fluid. Birds' compensatory mechanisms act to maintain blood volume rather than blood pressure. Restoration of blood volume can lead to rescucitation even at the terminal stages (Wyse and Nickerson, 1971; Sturkie, 1986a). Fluid therapy and blood transfusions will be discussed in detail in section 4.a.

To rescucitate the avian patient, an airway must first be established (see B.2.a). Positive pressure ventilation should occur once every four to five seconds. Once this is begun, a check should be done for heart beat and peripheral pulse. If none is detected, cardiac massage via rapid, firm sternal compression at 30-60 cycles per minutes should be performed. If required, adrenaline (mammalian dose rates range between 0.1-0.5 ml of 1:100 solution) and/or doxapram (0.007mg/g) may be given via intratracheal, intracardiac, or intraosseous routes if the peripheral circulation is not accessible (Quesenberry and Hillyer, 1994; Harrison, 1986b; Clubb, 1986).

c Neurologic

The major neurologic emergencies are central nervous system trauma and seizures.

Recent cranial and spinal trauma can be treated with corticosteroids. Methyl prednisolone sodium succinate (MPSS) improves recovery in humans and cats following spinal injuries, while dexamethasone showed no benefit over a placebo in improving neurologic signs. The optimal dose of MPSS was 30mg/kg in cats and mice. Prednisolone sodium succinate is equally effective, but half as potent when given to mice 5 minutes post concussive head trauma (Brown and Hall, 1992). Mannitol (no dose rate given) or frusemide can be tried if birds with head trauma do not respond to initial therapy with corticosteroids (Quesenberry and Hillyer, 1994). It should be noted that mannitol is contraindicated in cases of central nervous system haemorrhage (Jenkins, 1987). Care must be taken with fluid therapy to avoid iatrogenic cerebral oedema (Quesenberry and Hillyer, 1994).

Seizuring in birds can be caused by many conditions. Intramuscular or intravenous diazepam (0.001mg/g) can be used to stop seizures (Pasco, Hickman and Levine, 1983). Other treatments may include administration of

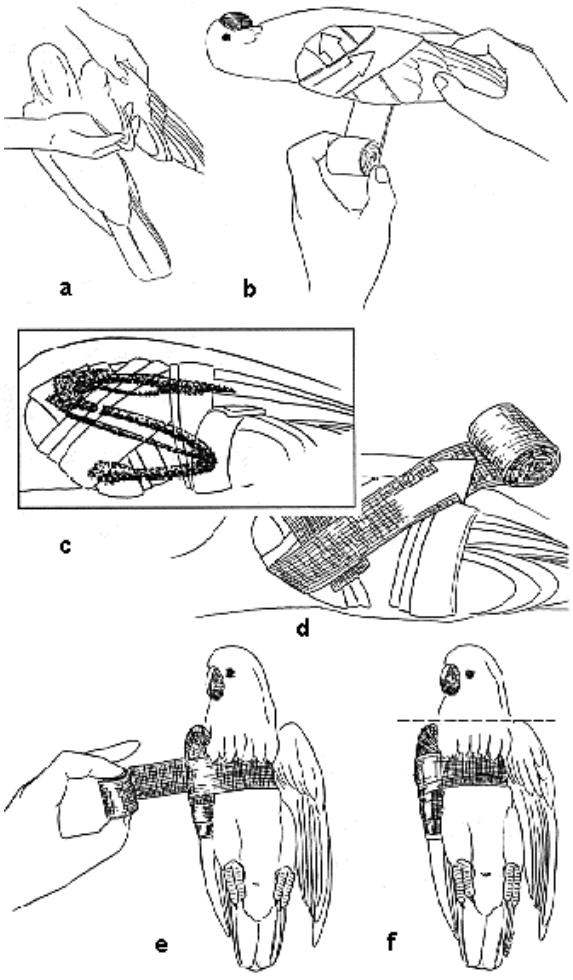
dextrose, calcium gluconate (50-100mg/kg) or calcium EDTA (0.035mg/g intramuscularly every 8-12 hours for 5-10 days). These are treatments for seizures caused by hypoglycaemia, hypocalcaemia and lead poisoning, respectively (Quesenberry and Hillyer, 1994; Clubb, 1986).

d **Musculoskeletal**

The main category of musculoskeletal injuries that require emergency stabilisation are fractures. Non-surgical approaches to fracture immobilisation are useful for stabilisation, but can be used as the primary treatment in many instances. As in other species, external coaptation devices must immobilise the joints above and below the fracture.

Wing fractures distal to the elbow, and elbow or carpal luxations may be secured with figure-of-eight bandages (see Diagram 1). The wing should be bandaged in a normal flexed position. Those fractures proximal to the elbow may be immobilised with wing-body wraps. This is a continuation of t he figure-of-eight bandage around the body wall. Care should be taken to avoid compromisation of the bird's respiration by overly tight bandages. Self adhesive rope is preferred, as it will not damage a patient's feathers (Degernes, 1994).

Figure 2:
Taken from Quesenberry, KE and Hillyer, EV (1994). Supportive care and emergency therapy, In Avian Medicine: Principles and Practice, Eds. Ritchie, BW, Harrison, GJ and Harrison, LR. Wingers Pblishing Inc, Lake Worth, p 429



The upper two pictures show a figure-of eight wing bandage being applied, while the lower two demonstrate how this technique can be modified to form a wing-body wrap. The middle pictures demonstrate the wing position for bandaging.

Fractures of the tarsometatarsus and distal one-third of the tibiotarsus can be stabilised with either Schroeder-Thomas splints or Robert-Jones bandages. When using a Robert-Jones bandage to treat a tarsometatarsus fracture, a ball bandage should be used to immobilise the foot. This can be used alone to treat toe fractures (Degernes, 1994).

Leg fractures proximal to the distal one-third of the tibiotarsus cannot be treated by external coaptation (Degernes, 1994).

e Other Systems

Management of wounds to the integument is much the same as in mammalian medicine. After lavage and debridement, the wound may be repaired surgically if less than eight hours old, or not heavily contaminated. Seriously contaminated wounds, or those older than eight hours can be left to heal by second intention (Degernes, 1994). If a cat attack has occurred, then prophylactic antibiotics are essential as *Pasteurella multocida* infections are a common sequela (Kaufman, 1992). Non-water soluble medication cause loss of insulation due to their effect on feathers, so should be avoided (Degernes, 1994).

Initial, symptomatic management of gastrointestinal emergencies may include fluid therapy, antibiotics and possibly corticosteroids. Oral aminoglycosides can be useful, as their action is local with minimal absorption. The long-term use of corticosteroids is discussed in Section 4.b.i. Once stable, the bird may undergo surgery, or other treatment dictated by the primary cause of disease. Suspected liver disease can be treated with antibiotics, oral lactulose and parenteral vitamin K (Quesenberry and Hillyer, 1994).

In the urogenital system, the main emergencies that require significantly different treatment to mammals are egg-binding and egg peritonitis. Emergency supportive treatment for the former includes fluids, cloacal lubrication, intramuscular calcium gluconate(0.5-1.0 ml/kg bodyweight), vitamin A and vitamin D3 (1000 and 100 units per 100 grams, respectively) and supplemental heat. Supportive therapy for egg peritonitis requires parenteral fluids and broad-spectrum antibiotics. Two to five days of low-dose corticosteroid therapy may be beneficial (Ritchie, 1990; Huff, 1993; Quesenberry and Hillyer, 1994).

3 Samples Required for Diagnosis

A pretreatment blood sample should be taken, and evaluated for haematocrit and total plasma protein. A blood smear should be made to enable estimation of white cell count and differential, red blood cell morphology and haemoparasite detection (Jenkins, 1987; Cannon, 1994).

Blood can be collected from the jugular vein, a peripheral vein or by clipping a toenail. Jugular venipuncture is done with a 22- to 28-gauge needle. Pressure must to be maintained for a short period to avoid haematoma formation. In pigeons and doves, a 25-gauge needle is often used in the medial metatarsal vein, due to the minimal haematoma formation and easy access to this site. Venipuncture of the cutaneous ulnar vein is done in birds weighing over 300-400 g, and requires prolonged pressure (at least 30 seconds) to avoid haematoma development, and is more difficult to access. Saphenous veins are often used in long-legged birds. Toenail clipping can cause tissue fluid contamination of the sample due to the crushing action used for collection, while milking the toe can cause changes in the cell populations. It

is painful for the bird and can only provide small volumes of blood (Hoefer, 1992).

Faecal analysis via wet smear, gram stain and faecal flotation is useful, as is swabbing or aspirating the crop in conditions of upper gastrointestinal disease (Quesenberry and Hillyer, 1989).

Cannon (1991a) recommends cloacal and choanal swabs for gram stain and possibly culture and sensitivity.

4 Supportive Therapy

a **Fluid Therapy**

i *Assessment of Hydration and Estimation of Volume Required*

A small blood sample is used to define dehydration objectively. Subjective parameters were discussed in section B.2.

Martin and Kollias (1989) found that the most useful parameters for assessment of dehydration in water-deprived pigeons were serum osmolality and total solids. Baseline values for total solids varied greatly between individuals, whereas osmolality is reasonably constant. Total solids may be low in a starving bird, regardless of hydration status (Kaufman, 1992). Elevations of plasma urea, and plasma creatinine (compared to both baseline and reference values) have been shown to be good indicators of dehydration (Lumeij, 1987).

Although useful in mammals, the packed cell volume (PCV) is not a good indicator of dehydration in healthy pigeons, and can be deceptive, as birds often have an anaemia associated with chronic disease that may mask any rise in PCV due to dehydration (Hoefer, 1992; Martin, Palmore and Kollias, 1987).

The aim of fluid therapy is to increase tissue perfusion, correct existing electrolyte and water imbalances and meet daily fluid requirements (Orosz, 1992). The fluid deficit can be calculated as follows:

$$\textit{normal body weight for the species (grams)} \times \textit{percentage dehydration (decimal)} = \textit{estimated fluid deficit (ml)}$$

Many workers routinely assume 10% dehydration in their ill patients. Half of the deficit should be administered in the first 12-24 hours, with the remainder being administered over the next 48 hours.

Maintenance fluids should be given for each 24 hour period (Quesenberry and Hillyer, 1994). All fluids should be warmed to 38-39°C (Abou-Madi and Kollias, 1992).

The average bird has daily fluid requirements of 40-60 ml/kg/day (Abou-Madi and Kollias, 1992). Paediatric patients require two 'to three times the maintenance volume per kilogram of adult birds (Bauck and Kupersmith, 1991).

ii Routes of Administration

Regardless of the route of administration, all fluids should be warmed. Verkest (1994b) showed that even warmed saline infusions have a large effect on core body temperature.

Mild cases of dehydration can be treated with oral or subcutaneous fluids (Abou-Madi and Kollias, 1992). In pigeons, oral administration of 5% dextrose appears to be the treatment of choice. Contraindications for oral administration include Seizuring, comatose or regurgitating patients, birds in shock or those with gastrointestinal status (Quesenberry and Hillyer, 1994).

Some workers use Gatorade (Gatorade Co, Chicago, IL) for oral correction of fluid and electrolyte imbalances. Pysillium mixed with the oral fluids may increase fluid absorption from intestinal villi (Quesenberry and Hillyer, 1994). Normal crop emptying time is 3 to 4 hours, although this will decrease if fluids instead of solids are administered. Any delays should be investigated (Abou-Madi and Kollias, 1992).

Whether oral or subcutaneous routes are used, fluid administration should be repeated in 60-90 minutes, as blood parameters return to baseline at this time in pigeons (Martin and Kollias, 1989).

Sites of subcutaneous administration include the patagium (wing web), intrascapular areas and the skin fold between the medial past of the proximal inner thigh (Abou-Madi and Kollias, 1992). Only isotonic fluids can be administered by this route as hypertonic fluid causes further dehydration of the vascular compartment (Martin and Kollias, 1989). Care must be taken to avoid the air sacs when injecting fluids. It is recommended to use small needles (25- to 27-gauge), limit the volume to 5-10 ml/kg/site and to have only one puncture hole per administration site. Absorption of fluid is limited in hypothermic or shocked patients due to peripheral vasoconstriction (Abou-Madi and Kollias, 1992). Ventral abdominal oedema may occur as a sequela, and

indicates the need to decrease or cease administration by this route (Quesenberry and Hillyer, 1994).

Intraperitoneal injections of isotonic fluids can be given, but carry the risk of organ laceration. Position of air sacs must be taken into consideration. This route should not be used with peritonitis, abdominal masses, coelomic effusions or hypotension (Abou-Madi and Kollias, 1992).

More severe dehydration, or shock, must be treated via direct access to the vascular compartment. Venipuncture sites were described in section B.3.

Fluid boluses can be administered through capped maintained catheters or through butterfly needles. Slow boluses of 10-25 ml/kg can be administered over five to seven minutes. Bolus injections can be repeated every 3-4 hours for the first 12 hours, then every 8 hours for the next 48 hours, then twice daily thereafter (Harrison, 1986). Repeat administration of large volumes should induce a diuresis within 24 hours (Redig, 1984).

Transient bradycardia is a usual response to bolus administration. Tachypnoea, cardiac dysrhythmias, agitation and collapse can indicate fluid overload, although these side-effects occur rarely (Abou-Madi and Kollias, 1992).

Some authors state that serial use of veins without intravenous catheterisation is possible with atraumatic and aseptic technique (Redig, 1984), although others disagree.

Intravenous catheterisation is most commonly performed on the jugular or cutaneous ulnar veins. Over-the-needle catheters, butterfly needles or human preemie catheters can be used (Abou-Madi and Kollias, 1992; Johnson-Delaney, 1993). Maintenance of these catheters will be discussed in Critical Care. Infusions of 10 ml/kg/hr can be used for the first two hours, then reduced to 5-8 ml/kg/hr is used to avoid fluid overload (Abou-Madi and Kollias, 1992).

Intraosseus catheterisation can be used in patients with inaccessible veins, and is sometimes better tolerated than intravenous catheterisation. The distal end of the ulna is most commonly used.

Chemical restraint may be required, although is unnecessary in many sick birds. The feathers are removed from the carpus and the area is prepared aseptically. Twenty to twenty-two-gauge spinal needles are

used in larger species, while 25- to 30- gauge hypodermic needles are used in smaller patients. The needle is placed parallel to the median plane of the bone at the distal end of the ulna, and is advanced by applying fu-m pressure with slight rotation. The cannula should penetrate past the midpoint of the ulna. After placement, aspiration via the cannula should produce a small amount of bone marrow. The cannula should be flushed with heparinised saline, and secured by suturing a butterfly strip to the skin. Antibiotic ointment is placed around the hub, which is padded with a gauze patch. Tissue adhesive can be used instead of sutures. The wing is placed in a figure-8 bandage, and should incorporate 1-2 loops of extension robe (Ritchie, Otto, Latimer and Crowe, 1990; Lamberski and Daniel, 1992).

Cannulas can also be placed in the tibial crest, and passed distally. Light bandages or lateral splints are used to secure the cannula (Quesenberry and Hillyer, 1994).

Many drugs and fluids can be infused via this method. In one study, more than half of the dose administered was outside the ulna within 30 seconds. Solutions must be administered slowly to avoid retrograde flow into the subcutaneous space. Rates of 10 ml/hr have been administered using an infusion pump (Lamberski and Daniel, 1992).

iii Selection of Fluids

Shock in mammals can be treated with several different fluid regimes. Crystalloid solutions at a rate of 40-90 ml/kg/hr are often used, however, only 20% of the volume initially infused remains in the vascular compartment after 30-60 minutes (Crowe and Devey, 1995).

Hypertonic saline (7.5%) can be given at a rate of 3-4 ml/kg as a bolus. Some workers advocate using combinations of solutions, eg colloids and hypertonic saline (Firth, 1995).

Colloids may be used, at a maximal rate of 20 ml/kg. Due to endothelial impermeability to these molecules, fluids are drawn from the interstitial compartment by increased vascular osmotic pressure. The action can last from hours to days (Firth, 1995a). Rarely, complement activation can occur (Abou-Madi and Kollias, 1992).

There is little information available on the most effective fluids for the treating birds in shock. There is, as yet, no model available to reproduce the syndrome of shock in birds. Although fluid infusion has been shown to be effective in reviving birds suffering from acute blood loss, little research has been done on which fluid may be the most

effective (Verkest, 1994b). Acidosis, which occurs in shocked mammals, appears not to occur in birds (A. Gallagher, pers. comm.)

Solutions used to replace lost fluids include lactated Ringers solution CLRS), normal saline (NS), dextrose and dextrose and saline. Solutions used to replace lost fluid volume are not suitable for long-term maintenance, as different electrolytes are required for replacement as compared to maintenance (Quesenberry and Hillyer, 1994).

Crowe (1995b) advocates the addition of 3g amino acid/100 ml and 3-5g glucose/100 ml when giving intravenous fluids to mammals. The rationale is to help prevent catabolism, which provides amino acids for gluconeogenesis and to assist maintenance of body proteins.

LRS is isotonic, contains balanced electrolytes for fluid replacement, and administration of this alone can correct mild acidosis. LRS can worsen a pre-existing hyperkalaemia or hypematraemia (Abou-Madi and Kollias, 1992). If used as a maintenance fluid, 0.1-0.3mEq/kg potassium must be added daily (Redig, 1984). Sodium bicarbonate should not be added (Abou-Madi and Kollias, 1992).

When used as a replacement solution, potassium chloride must also be added to NS (0.9%NaC1). NS causes restoration of sodium concentrations, and excretion of potassium. It does not contain balanced electrolytes, and is unsuitable for maintenance (Abou-Madi and Kollias, 1992).

Dextrose at concentrations of five, ten or fifty percent is often used to correct hypoglycaemia (defined by blood glucose concentrations less than 200mg/dL) (Kaufman, 1992). Initially, one ml/kg of 50% dextrose is given intravenously, and the patient's blood glucose is monitored. Following this, oral administration of dextrose can begin, to assist further correction and prevent a recurrence of hypoglycaemia. Ten or fifty percent solutions can be used orally or intravenously. Five percent or less solutions are hypotonic, and solutions less than 2.5% should be used for subcutaneous injection. Dextrose promotes cellular acidosis, so should be used guardedly in acidotic patients (Abou-Madi and Kollias, 1992).

Hyperuricaemic and azotaemic patients can be treated with 0.45% NaC1 and 2.5% dextrose. This is isotonic. Half dextrose with half LRS gives both electrolytes and dextrose, and promotes diuresis. It is slightly hypotonic. Five percent dextrose with NS is hypertonic, and can cause cellular dehydration, diuresis and hypovolaemia. It should be

used with careful consideration (Abou-Madi and Kollias, 1992).

Mammals in shock often experience a mild acidosis, but this appears not to occur in birds. Trauma can cause a mild acidosis in birds, due to muscle release (A. Gallagher, per. comm.) With mild acidosis, the simple administration of fluids will often lead to a correction, but bicarbonate may need to be used with more severe conditions. Ideally, blood gas analysis would be used to determine the degree of acidosis, and the amount of bicarbonate required could be calculated as follows:

$$base\ deficit \times 0.3 \times weight(kg) = net\ bicarbonate\ required\ (mEq)$$

Without access to blood gas analysis, the degree of metabolic acid-base disturbance can be "guesstimated" by subtracting the expected species plasma bicarbonate concentration from the actual plasma bicarbonate concentrations. It gives no indication of the respiratory components of acidosis or alkalosis (Redig, 1984). One third to one half of the required amount of bicarbonate should be administered over the first hour of fluid therapy, then a second analysis should be performed. The remaining deficit, if required, can be administered over the next 24 hours. Sodium bicarbonate (8.4%) should always be diluted before intravenous administration (Abou-Madi and Kollias, 1992).

Alternatively, an empiric dose of 0.5-1.0mEq/kg sodium bicarbonate, diluted in 10-20 ml/kg fluid can be administered subcutaneously or intraperitoneally at 15-30 minute intervals, to a maximum of 4mEq/kg (Redig, 1984).

Whichever method used, care must be taken to avoid too rapid administration, which can lead to a paradoxical cerebrospinal fluid acidosis, or overdosing the patient, which will cause a metabolic alkalosis (Abou-Madi and Kollias, 1992).

Hyperkalaemia may be treated with calcium gluconate at 0.5 ml/kg (Cannon, 1991c).

iv Use of Blood Products

Indications for the transfusion of whole blood include: clinical signs of anaemia (tachypnoea, tachycardia, weakness, pallor), PCV less than 15-20% or acute blood loss greater than 25% of the total blood volume. (Hoefer, 1992). The average haematocrit for most mature birds is 40-60%, with younger birds having slightly lower values (Sturkie, 1986). However, the responses of the anaemic or acutely

haemorrhaged avian patient to fluid and parenteral iron dextran therapy are usually so successful that blood transfusions are not commonly performed (Bos, Todd, Tell, Ramsay and Gowler, 1990). One study conducted in healthy domestic pigeons showed that a 70% blood loss was treated most effectively with fluid treatment, not heterologous or homologous blood transfusion (Bos, Todd, Tell, Ramsay and Fowler, 1990). Quesenberry and Hillyer (1994) found the most clinical benefit from blood transfusions in birds suffering from chronic anaemia. Normocytic, normochromic, non-regenerative anaemias are typical of chronic disease, resulting from decreased erythrocyte production. Some nutritional deficiencies can cause similar anaemias. (Hoefor, 1992).

Although the reports on interspecific blood transfusions are conflicting, most workers appear to have adopted similar protocols. Abou-Madi and Kollias (1992) recommend use of donors that are (in order of preference) closely related, same species, then phylogenetically related species to the recipient.

Psittacines will generally tolerate a single infusion of chicken or pigeon blood (Harrison, 1986a). Donor birds are usually larger psittacines or pigeons. Screening of donor birds involves a full blood health profile, tests for blood parasites (eg. Plasmodium, Haemoproteus, Leucocytozoan and microfilariae), Chlamydia psittici, psittacine feather and beak disease, herpesvirus and papovavirus (Abou-Madi and Kollias, 1992; Hoefer, 1992).

Collection of blood from the donor is best done via jugular venipuncture. Approximately 10% of the total blood volume, or 1% body weight can safely be taken from healthy birds. Quesenberry and Hillyer (1989) calculate the volume required for the recipient as follows:

$$ml\ needed = 2.2 \times recipient\ weight(kg) \times normal\ PCV \times (PCV\ desired - PCV\ recipient)$$

PCV donor

The donor should receive 10-30 ml/kg intravenous fluid, intramuscular vitamin B (0.025- 0.25 ml/kg) and iron dextran injections (10mg/kg) (Kaufman, 1992; Pasco et al, 1984).

To prevent coagulation, acid citrate dextrose or citrate phosphate dextrose can be used at 0.1 ml/l ml whole blood, but could cause problems with small or

hypocalcaemic recipients. Sodium heparin is an alternative, either at 0.6 ml of 125 unit/ ml solution per ml of whole blood or as a thin coating on the inside of the syringe used for collection. Care should be taken to avoid heparinisation of the recipient. Heparin will not preserve erythrocytes, so blood treated in this fashion must be used immediately. (Hoefer, 1992; Abou-Madi and Kollias, 1992).

If the blood is to be stored, erythrocytes must be separated from plasma quickly, as they absorb plasma potassium (Sturkie, 1986a).

Ideally, major and minor crossmatching procedures would be performed, but this happens rarely. Altman (1986) stated that crossmatching of unwashed red cells and sera was not accurate in determining compatibility, but Hoefer (1992) states that using washed red blood cells is accurate.

Prior to administration, the use of either dexamethasone sodium phosphate (4-6mg/kg) or prednisolone sodium succinate (10mg/kg) is advised to minimise the risk of a transfusion reaction. Transfusion reactions can involve urticaria, fever, haemolysis, haemoglobinuria and death. Erythrocyte and haemoglobin casts can lodge in the renal tubules (Allman, 1983). In raptors, antibody titres peak four days after sensitisation to sheep red blood cells, and remain increased for seven days. They return to normal levels by three weeks. Single heterologous transfusions seems to be safe.. Multiple heterologous transfusions can cause fatal reactions, especially if administered less than 10 days after the initial transfusion (Altman, 1983)

The transfusion is administered intravenously or intraosseously. Procedures for both are as indicated in the fluid therapy section (4.a). Intravenously, the blood is given as a slow bolus over a 1 to 3 minute time. The intraosseous route is indicated on small patients or those with collapsed or inaccessible veins. The rate of infusion is slower than for intravenous administration. (Hoefer, 1992).

Little research has been done on the use of blood products (fresh plasma, fresh frozen plasma, coagulation factors etc) in avian medicine.

b **Drugs**

I ***Corticosteroids***

The use of corticosteroids to treat neurologic injuries has already been described. These drugs are often used in the treatment of shock, although complications include immunosuppression and delayed wound healing, bleeding and ulceration of the gastrointestinal tract and adrenal suppression. Of these, immunosuppression can occur with a single dose, while the others occur with more chronic administration (Quesenberry and Hillyer, 1994). As mentioned previously, there is no effective model for reproducing shock in birds. Therefore, the use of

corticosteroids as a treatment lacks supportive experimental evidence (Verkest, 1994b).

Rosskopf and Woerpel (1987) use dexamethasone at 2-8mg/kg bodyweight, while Jenkins (1987) uses prednisolone sodium succinate at 40mg/kg or dexamethasone sodium phosphate at 4.4mg/kg.

Red-tailed hawks and Barred Owls given intravenous or intramuscular dexamethasone at a rate of 3mg/kg, had peak plasma concentrations within 15 minutes, although intravenous administration gave a higher peak concentration (Burns, Baker and Birrenkott, 1992). Serum half life and corticosterone suppression varied with species (serum half life was 53.3 minutes in Red-tailed Hawks, 37.5 minutes in Barred Owls and 36 minutes in male broiler chickens), while plasma corticosterone concentrations were suppressed for 18 hours in hawks, and 24 hours in owls.

Use of steroids for longer than one week would seem to potentiate secondary bacterial and fungal infections, indicating birds' susceptibility to the immunosuppressive effects of steroids (Quesenberry and Hillyer, 1994).

ii ***Antibiotics***

Rosskopf and Woerpel (1987) recommend the use of piperacillin and amikacin in septic patients pending culture and sensitivity results. Piperacillin is effective against a variety of gram-negative, gram-positive and anaerobic bacteria, m ~ d is given at 100-200mg/kg bodyweight every 12 hours via intramuscular injection. Amikacin sulfate is given at 10-20mg/kg every 12-24 hours via intramuscular injection. Both are bactericidal.

Other antibiotics commonly used include cefotaxime, enrofloxacin, trimethoprim-sulphonamide and doxcycline (Quesenberry and Hillyer, 1994).

A 26-30-gauge needle is used to minimise muscle damage, and the injection is given in the pectoral muscles (Quesenberry and Hillyer, 1994).

Other clinical syndromes can be treated similarly - using arbitrary antibiotics until the culture and sensitivity results return.

iii ***Analgesics***

Of the non-steroidal anti-inflammatory class of drugs, only acetylsalicylic acid and flunixin have been described for use in birds. A 5 grain tablet of acetylsalicylic acid is dissolved in 250 ml drinking water.

Flunixin meglumine is given by intramuscular administration at a dose of 1-10mg/kg. The injection can be repeated. It is recommended as an analgesic, anti-inflammatory and antipyretic (Clubb, 1986).

Butorphanol tartrate used at 3-4mg/kg in English parakeets was not found to depress heart rate or respiratory rate, but did cause vomition/regurgitation when used at a rate of 10mg/kg. Almost half of the birds showed mild motor deficits, and some still vocalised after administration of an intramuscular ketamine injection (Bauck, 1990).

Codeine and morphine have been investigated as analgesics in the turkey and chicken, but there is much controversy surrounding the experiment design and interpretation of results (Bauck, 1990).

The use of metomidate has also been described (Ryder-Davies, 1973). Xylazine has been used, mainly in combination with ketamine as a parenteral anaesthetic regime (Harrison, 1986b).

Local analgesic agents, such as lignocaine, can be used in birds (Hall and Clarke, 1983). It is important to monitor the total dose, as it is easy to give a gross overdose, especially in small patients. Very dilute solutions (0.25-0.5% lignocaine) should be used, as birds may be more sensitive to local analgesics than mammals of equivalent body weight.

iv ***Other***

As a treatment for mild anaemias, iron dextran at 10mg/kg can be given intramuscularly and repeated in 1-2 weeks if necessary (Abou-Macli and Kollias, 1992). Quesenberry and Hillyer (1989) recommend administration of iron dextran again in 4-5 days, as well as daily supplementation of B complex vitamins.

Birds with suspected nutritional deficiencies or liver disease can benefit from vitamins A, E and D3 (at dose rates of 50-100 units/kg, 10000units/kg and 1000 units/kg, respectively). B complex vitamins with or without thiamine can be used in animals with neurologic signs, or inappetence (Orosz, 1992).

Drugs requiring hepatic metabolism should be used with care in patients with suspected liver disease (Quesenberry and Hillyer, 1989).

Endotoxic shock can be treated with glucocorticoids to decrease the endotoxic effects, and antibiotics (Ritchie, 1990). Nerotect (Sirevex, Kenmore, QLD) can also be useful (A. Gallagher, pers. comm.).

c **Environment**

i ***Heat***

Many authors recommend enclosures which are warm (29-30 degrees Celsius for adults, and 30-34 degrees Celsius for neonates), with the correct humidity (approximately 70%), for avian patients (Quesenberry and Hillyer, 1994; Harrison, 1986). This can be provided with heating pads, hot water bottles wrapped in cloth, non-blowing electric heaters, hot lamps or brooders. There are many varieties of commercial brooders and hospital cages available. Care must be taken to avoid overheating which can cause vasodilation and shock (Ritchie, 1990). Hyperthermia manifests as panting with wings held away from the body (Quesenberry and Hillyer, 1994). Body temperature should be taken before heat therapy and monitored periodically (Verkest, 1994b).

There must be a heat increment across the enclosure, so the bird can position itself in its zone of comfort. Vogelnest (1994) believes that warming is only indicated for fluffed up and depressed birds. Room temperature will suffice for other birds.

Jenkins (1987) suggests a combination of external and internal warming, by warming fluids for intravenous administration to 30-38°C, and warming the bird's environment to 24-27°C. After tissue perfusion is somewhat restored, the environment may be further warmed to 27-30°C.

ii ***Housing***

Birds with leg fractures or those too weak to perch can be placed in enclosures with thick paper or padding on the floor (Quesenberry and Hillyer, 1994). Birds can use their beaks to hold themselves upright if horizontal bars are provided (Quesenberry and Hillyer, 1989).

The avian patient should be housed in quiet and darkened surrounds, to minimise stress. Access to food and water should be maintained, unless contraindicated by the bird's condition.

C Critical Care

1 Monitoring the Critical Patient

Daily weight monitoring is a good means of determining the bird's shams. If on fluid therapy, there should be a daily net gain, with a small weight loss between each administration. An absence of loss between treatments can signal sequestration of fluid (Cannon, 1991b).

Parameters to be monitored with intravenous fluid therapy include daily weights, daily fluid inputs and outputs, PCV, total protein (TP) and complete blood counts and electrolytes (Bond, Downs and Wolf, 1993). Blood must be collected a site other than that used for fluid administration. Capillary refill time should also be noted (Redig, 1984).

It is not practical to measure central venous pressure with indwelling catheters in birds, but an estimate can be obtained by noting the degree of distension of peripheral veins, colour of mucous membranes, capillary refill time and limb temperature. Some authors advocate cutting a toenail and observing the blood flow, but this may not be indicated for daily monitoring (Jenkins, 1987). Blood pressure has been measured in some avian species. An indirect method utilises a cuff on the thigh (it has been tried on chickens). The appropriate cuff size is important (Sturkie, 1986b). Presumably, this technique could be modified with the use of a Doppler machine.

Haematocrit and total solids provide valuable information about hydration status and anaemia. It is recommended to check these parameters 6-12 hours after initial treatment (except on extremely small patients), and then daily until the bird no longer requires critical care (Jenkins, 1987).

Size, consistency and number of droppings should be noted daily, and the presence of uric acid in the faeces gives an assessment of urine production (Jenkins, 1987).

2 Supportive Therapy

Supportive therapy for the critically ill patient is a difficult topic to cover thoroughly. Each patient requires a customised critical care protocol, to allow the primary disease condition to be treated, and also cover the secondary or supportive procedures required. This section will deal with some common critical care practices. It is meant as a guide to critical care, not as a reference text.

a **Oxygen Therapy and Nebulisation**

Although a patient may need supplemental oxygen, 100% oxygen supplied for greater than 12 hours can cause anorexia, lethargy and respiratory distress in canaries and budgerigars. Death can occur with exposures greater than 3-4 days (Stauber, Krinke, Greene and Wilderson, 1991). Therefore, the oxygen content supplied to a bird can be increased compared to room air, but the bird should be observed for signs of oxygen toxicity. Warming and humidification of oxygen is important to avoid chilling and dehydration of the patient (Quesenberry and Hillyer, 1994).

Air sac cannulas may be left in place for up to 14 days, although 7 days is usually the upper limit (Rosskopf and Woerpel, 1990). Although these usually remain patent, regular checks for patency should occur. Some workers use shortened endotracheal robes, and inflate the cuff inside the abdomen to prevent slippage (Quesenberry and Hillyer, 1994).

Nebulisation can be useful with bacterial or fungal infections of the upper respiratory tract. Ultrasonic nebulisers will produce particle sizes small enough to penetrate a bird's respiratory system, but other nebulisers may not. Equipment must be sterilisable between birds. Most of the antibiotics formulated for intravenous use can be used in nebulisation. Cefotaxime (100mg in saline) or piperacillin (100mg in saline) is used by Quesenberry and Hillyer (1994) in treating unidentified bacterial air sacculitis. Two to four sessions per day of 10-30 minutes nebulisation for 5 - 7 days is a protocol used (Spink, 1986). Mucolytic agents are irritant, and should be avoided (Toms, 1989).

b Fluid Therapy and Maintenance of Intravenous Access

Fluid therapy should be reduced to maintenance levels after rehydration of the patient has occurred.

The calculation for maintenance requirements is described in section 4.a.i.

Maintenance of access to the vascular system can be difficult once a bird begins to recover. Abou-Madi and Kollias (1992) advocate a technique involving over the needle catheters or butterfly needles sutured, tissue-glued or taped to the surrounding skin, and covered with a bandage such as Coflex (Andover, Salisbury, MA, USA). Bond, Downs and Wolf (1993) describe fixing the ulnar vein with a tongue depressor that extends three centimetres beyond the end of the catheter. Proximal and distal ends of the tongue depressor are. then incorporated into a wing bandage for stabilisation. A Coflex collar has

been suggested for use when the jugular vein is catheterised. Johnson-Delaney (1993) describes the use of human preemie intravenous catheters in birds.

The catheter should be periodically flushed with saline. Heparin should be used with caution to avoid patient heparinisation, especially with small birds (Quesenberry and Hillyer, 1994).

With aseptic technique, an intravenous catheter may be left in place for 2-3 days (Abou-Madi and Kollias, 1992). Some workers maintain the catheter for 4-7 days (Bond et al, 1993). Complications can include thrombosis, cellulitis and loss of patency. Some patients will not tolerate the catheter, and others may remove it as they begin to recover (Bond et al, 1993).

With twice daily flushing with 10 units/ml heparinised saline, an intraosseous catheter can be maintained for 72 hours (Ritchie, 1990). These catheters can be attached to spring-loaded infusion pumps for continual medication (A. Gallagher, pers. comm.). After some days, a painful response may be demonstrated following infusion of fluid. Local oedema or extravasation of fluid could cause this (Quesenberry and Hillyer, 1994).

A new technique described by Harvey-Clark (1990) involves the use of vascular access devices. These can be radiolucent or radiopaque. A catheter is introduced to the central venous circulation using a skin incision and venotomy, under general anaesthesia. Access to the catheter is achieved by using a non-coring Huber-point needle to enter the subcutaneous reservoir. This is attached to the catheter with a loop to allow for neck movement. It takes 10-15 minutes to implant surgically, and can be maintained up to 12 weeks, and accessed up to 300 times. It has been used successfully in a clinical case on an Auklet, and also in an experiment in providing total parenteral nutrition to two geese. It needs to be flushed regularly, or to have a saline or heparin lock. The catheter is secured with sutures above and below the retention ring, and the reservoir is secured to muscle fascia. It must, however, be surgically removed, and the jugular must be closed with a suture. The removal can be done under local or general anaesthesia.

It may be better to give periodic oral fluid therapy once the bird has improved, until it is drinking adequately alone.

c **Nutrition**

The caloric demand of a sick animal is increased, so nutritional support should be provided (Ritchie, 1990). Withholding food will further increase catabolism. The basal metabolic rate of birds (in kcal/day) can be estimated by the formula:

$$\mathbf{BMR = K(W_{kg}^{0.75})}$$

where for passerine birds K = 129; Non-passerine birds K = 78 (Sedgewick, Pokras and Kaufman, 1990).

"K" is a constant, and varies between the species of birds. Maintenance energy requirements (MER) incorporate the BMR with the addition of energy required for normal physical activity and gastrointestinal function. Depending on the type of activity and time of year, the MER can exceed the BMR by 1.3-7.2 times in wild passerines (Whirtow, 1986). A caged bird can be estimated to have a MER of approximately 1.5 times BMR. Stress, disease, growth and reproduction cause a multiplication of the MER, and the some factors has been calculated by Quesenberry (1992) in the following table:

TABLE 1: Adjustments to Maintenance for Stress (as multiples of MER)

Starvation	0.5-0.7
Elective Surgery	1.0-1.2
Mild Trauma	1.0-1.2
Severe Trauma	1.1-2.0
Growth	1.5-3.0
Sepsis	1.2-1.5
Burns	1.2-2.0
Head Injuries	1.0-2.0

In a supplied diet, crude protein, crude fat and carbohydrates can be expected to provide 4.3, 9.3 and 4.1 kcal/g, respectively. It is suggested to have 3-7 g protein as essential amino acids, 3-7 g fat as essential fatty acids and 10-13 g carbohydrate per 100 kcal daily energy cost for adults. The rates recommended for infants are 5-g, 5-9g and 3-6g, respectively (Quesenberry, Mauldin and Hillyer, 1992)

Either enteral or parenteral regimes may be used. In mammals, the gastrointestinal tract receives up to one third of its nutrients from food passing through its lumen. Crowe (1995b) recommends providing enteral nutrition unless there is a specific contraindication related to primary gastrointestinal disease.

Enteral nutrition can be provided per os, pharyngostomy robe or duodenal catheter.

If the bird will consume food voluntarily, and can digest it successfully, then that is the least stressful and more efficient route. If the bird is not eating sufficient amounts to maintain itself, force-feeding must be done with either stainless steel feeding needles ("crop needles") or robber feeding catheters.

The implement is passed from the oral commissure into the oesophagus, with the bird's neck extended.

The crop must be palpated for evidence of the tube to ensure it has not entered the trachea. Once certain, the a small amount of formula can be injected. If no respiratory distress occurs (that is, the needle is not situated in the trachea), then the remainder of the formula can be introduced (Quesenberry and Hillyer, 1994). If regurgitation occurs, the bird should be released immediately (Ritchie, 1990).

If the crop or upper gastrointestinal system can not process food, it may be directly placed into the proventriculus with a pharyngostomy robe. A tube is passed into the oesophagus at the base of the mandible, and positioned with the end in the proventriculus. It is then sutured in place. Cellulitis often occurs, but usually resolves upon removal (Quesenberry and Hillyer, 1994).

Another alternative is to place a small needle catheter some distance (6-8cm) into the proximal duodenum, under general anaesthesia, and to pass it through the lower abdominal wall. Sutures are placed to secure the duodenum to the abdominal wall, and to secure the catheter to the skin. The catheter end, and any excess, is secured to the bird's dorsal aspect with tape or sutures. Either small, frequent feeds (up to 12-24 per day), or an infusion pump are necessary with this method. In a study, liquid diet was administered to pigeons via this method for 14 days without adverse effects. It is unsuitable for use in small birds, due to the difficulty of surgical access, and the inability to infuse fluids through a very small diameter tube (Goring, Goldman, Kaufman, Roberts, Quesenberry and Kollias, 1986). Clinically, this method has been successfully used for up to 6 days (Quesenberry and Hillyer, 1994).

Products for enteral nutrition should be of high biological value, low residue and lactose free (Huff, 1993). Products can require little or no digestion (monomeric diets) or some digestion (meal replacement formulations) (Quesenberry and Hillyer, 1994). Initially, the gruel fed should be low protein, high carbohydrate (Ritchie, 1990; Orosz, 1992). Abou-Madi and Kollias (1992) advise tube feeding dilutions of Emeraid (Lafeber, Odell, IL) or human low-residue diets to ameliorate this situation. The strength is increased over 12 hours, and gradually more protein is added, unless liver

disease is suspected (Orosz, 1992). Feeds should be given in small amounts, frequently. Hypertonic products can cause osmotically induced diarrhoea (Goring et al, 1986). Products with a high caloric density (2.0kcal/ml) can provide the required daily energy in two to four feeds per day. It is important to monitor hydration status in birds given calorie-dense formulas (Quesenberry and Hillyer, 1994). Formula should be gently heated before administration (care should be exercised with microwave heated foods), and can be stored for 2-3 days in a refrigerator (Quesenberry and Hillyet, 1994). Some commercial products commonly used include PolyAid (Vetalarm, Wagga Wagga, NSW), Sustagen (Mead-Johnson, Crowes Nest, NSW), Roudybush (Roudybush, Australia), Hills A/D (Cenvet, Sydney, NSW) and Complan (Farex, Noble Park, Vic).

As the bird recovers, blended food (baby food, dog food, canned food) is added in increasing amounts. Finally, the bird is placed on hospital diet, and is weaned off robe feeding (Abou-Madi and Kollias).

If the formula curdles or sludges in the crop, which can occur with ingluvitis, gastrointestinal stasis, or powdered products mixed with insufficient mounts of water, the crop should be flushed with warm water and gently massaged to break the curdled portion apart and allow aspiration and removal (Quesenberry and Hillyer, 1994).

Although some workers advocate withholding food for 24-48 hours in severely debilitated or cachectic birds, Crowe (1995b) believes that providing small amounts of nutrition to the gastrointestinal mucosa (microenteral nutrition) can assist prevention of stress ulcers in the stomach, decrease bacterial translocation and diminish the increase in metabolism that occurs with illness to a certain extent.

Once the bird is capable of eating alone, a wide variety of appealing food should be presented. Examples include millet sprays, warmed food, seed soaked in warm water shortly before offering and fruit and vegetables (Cannon, 1991b; Quesenberry and Hillyer, 1989). Care must be taken to offer food appropriate to the species (A. Gallagher, pers. comm).

Parenteral nutrition can be total or partial. Partial parenteral nutrition has been documented in small animals, but not in birds. It involves the feeding of a solution composed of 3% amino acids, 3% glucose or glycerol and maintenance water and electrolytes. Being isotonic, this solution can be given via a peripheral or a central vein. It is used to prevent the breakdown of muscle protein into amino acids that are converted to glucose when the body is fasted. It can satisfy, at best, 50-75% of the animal's requirements, so is not suitable for long-term use (Crowe, 1995b).

In dogs, a 20% lipid solution can provide 50-60% of caloric requirements, with the remainder supplied by a 50% dextrose solution (Labato, 1992). The 1.5-6g/kg/d protein required is supplied by amino acid supplementation.

Total parenteral nutrition should be started on approximately 50% calculated energy requirements, and increased to 100% over 2-3 days. Blood glucose should be monitored (Crowe, 1990).

Complications of parenteral nutrition can include any of those possible from catheter usage, plus some metabolic abnormalities (hypophosphataemia, changes in blood potassium, hyperglycaemia and liver dysfunction) (Quesenberry, 1992). Continuous infusion is recommended, to dilute the hypertonic solution.

Experimentally, vascular access devices have been used to provide TPN to two geese (Harvey-Clark, 1990). Four daily infusions of 20-30 minutes at a rate of 5ml/minute were given. One goose died, seemingly from a *Staphylococcus aureus* infection in the vascular access device. The second goose was maintained for the full four days with no clinical abnormalities. Both showed haematologic changes after each treatment of TPN, although this could be ascribed to the infusion of large volumes of hypertonic, acidic solutions over a short time period. It has also been used in one clinical case involving a Cassins auklet (Harvey-Clark, 1990).

Another study has been done using a VAD to administer TPN to pigeons. Four daily infusions given over 5 days resulted in only mild clinical changes, including weight loss, regurgitation, transient hyperglycaemia, polyuria and glycosuria and tachycardia (Degernes, Davidson, Kolmstetter, Flammer and Munger, 1992).

Intraosseous cannulas have also been suggested for use with TPN (Quesenberry and Hillyet, 1994).

In the study by Harvey-Clark (1990), a 10% amino acid solution (giving 100mg protein/ml), a 20% lipid emulsion (giving 2 kcal/ml) and a 50% dextrose solution (giving 1.7kcal/ml) were mixed and placed in an aseptically emptied, 1L bag of 5% dextrose. The ingredients are added amino acid first, then dextrose solution. Mixing is effected by inversion. Lastly, the lipid solution is added slowly over a 2 minute period. The bag should be stored in the refrigerator and used within 24 hours.

Whichever route of nutrition provided, daily weighing provides the most accurate means of determining the adequacy of treatment (Quesenberry and Hillyer, 1994).

D Environment

As mentioned above, birds should not be subjected to environmental stress. the critical care patient should be kept in a quiet room, and at a suitable ambient temperature (Quesenbcrry and Hillyer, 1994). Whirtow (1986) states that a lower ambient temperature will decrease the efficiency of food assimilation. Some workers recommend 12 hours of darkened environment for the bird each 24 hour period, to allow it to rest (Pasco, Hickman and Levine, 1983).

Birds with locomotive disability or neurologic deficits should be placed in well padded cages without perches. Horizontal bars on the cage walls, or items placed within the cage allow the bird to use its beak for balance (Quesenberry and Hillyer, 1989).

Comatose or semi-comatose patients should be turned frequently, and kept on soft surfaces. The eyes should be lubricated if required, to prevent exposure keratitis. The cloaca should be emptied manually if the tone is impaired. This can be done via abdominal pressure or with a cottonbud (Quesenberry and Hillyer, 1989).

Of course, food and water should be available as discussed above.

D Discussion

As is obvious from the number of times mammalian values or protocols have been included, there is a definite lack of even basic data about birds.

It is obvious that simple extrapolation from mammalian medicine will not result in optimal treatment of the avian patient. This is the case in routine medicine and surgery, but becomes crucial when dealing with life-and-death decisions in emergency and critical care. A prime example of this need is the use of bicarbonate to treat the "acidosis" caused by shock.

Part of the problem is due to the enormous variation between birds - once values have been determined for one group, they are not necessarily transferable to another group of birds. Much replication in research is then required to establish an "avian protocol". For instance, much of the nutritional information about birds is based upon the requirements of commercial poultry. These birds have been highly selected for food conversion and are probably the least representative group to use, yet those are the most comprehensive values available.

The final challenge of avian critical and emergency care is the special behavioural needs of the patients. Once a bird begins to recover, it can seem

to do everything in its power to oppose the clinician in their efforts. The optimal cage environments for convalescing birds may need to be researched.

Avian cardiology is a field in its infancy (like many avian fields). There appears to be little information on the treatment of non-critical problems, let alone critical ones, and more research is obviously required. Similarly, there is still no consensus about the treatment of central nervous system trauma in mammals, let alone birds.

Fluid therapy has been shown to be beneficial to the avian patient, although it remains to be seen which solutions are superior. Avian analgesia is still in its infancy, with only a few papers published.

One would expect, however, the adverse effects of stress to be as detrimental to birds, with their propensity to stress in strange situations, as they are to mammals. On the other hand, avian pain is difficult to monitor due to the "preservation reflex".

The field of antibiotics used in birds appears to growing rapidly. Pharmacokinetic studies have been conducted, and many papers exist about this subject.

E Conclusion

This work should function as a guide to current theories in emergency and critical care for the avian patient. It will need updating as more experimental and anecdotal evidence comes to light. One of the important points to be gained is that much more research is needed for this area to flourish.

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Shock and Compensatory Mechanisms Clinical Considerations

Kurt Verkest[1]

A. Summary

This essay summarises the evidence that birds experience a syndrome similar to mammalian shock, discusses the compensatory mechanisms that birds have available for dealing with shock, and fin ~ y outlines some of the implications of these mechanisms on the formulation of a rational diagnosis of and treatment for shock. A detailed explication of mammalian shock and compensatory mechanisms will not be provided due to space restrictions except where the analogous avian mechanism has not been sufficiently characterised.

B. The Nature of Shock

Shock is defined as a state in which there is insufficient tissue perfusion. Affected tissues will experience decreased oxygenation and reduced clearance of metabolic wastes. Shock can arise due to an absolute decrease in blood volume (hypovolaemic shock), an expansion of the vascular space without an increase in blood volume (effective hypovolaemic Shock) or an ineffectively pumping heart (cardiogenic shock). Hypovolaemic shock can be caused by haemorrhage, water deprivation or any other form of dehydration. Effective hypovolaemic shock can be caused by sepsis, endotoxaemia, anaphylaxis and fear. Cardiac failure can cause cardiogenic shock (Bednarski, 1989).

Regardless of the initiating cause, the consequences of shock are directly related to inadequate tissue perfusion, which affects different tissues to a greater or lesser extent, depending on the duration and severity of the shock, the level of compensation, the metabolic requirements of the tissue and the preferential status of the organ.

Inadequate perfusion of the kidneys (prerenal renal failure) will initially result in oliguria and azotaemia without renal cell injury. In the more advanced stages of shock, renal tubular cells will die and slough off, leading to irreversible organ damage unless the tubules can be repopulated. Acidosis is a feature of all forms of established shock. It arises when hypoxic tissues undergo anaerobic glycolysis, producing lactic acid in the presence of reduced lactate clearance and impaired renal acid/base regulating mechanisms (Cotran, Kumar and Robbins, 1989).

C. Compensatory Mechanisms

There is a number of compensatory mechanisms which support the cardiovascular system in the

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short to medium term during shock. In mammals they are well characterised; their primary function is to maintain tissue perfusion. This is achieved by maintenance of arterial blood pressure and/or maintenance of plasma volume.

The sympathetic nervous system is the first to respond to shock. The baroreceptor reflex, and later the central nervous system ischaemic response elicit an increased sympathetic tone, manifested both by increased sympathetic nervous outflow and catecholamine release.

This results in vasoconstriction, increased heart rate, increased cardiac contractility and, as a result, increased arterial blood pressure. Vasoconstriction is manifested to differing extents in different tissues, leading to changes in blood flow distribution. The heart and brain are preferentially supplied. Venous return and blood pressure are also maintained by blood vessel contraction in response to reduced filling pressure.

Angiotensin (AII) and vasopressin (antidiuretic hormone, ADH) promote arterial constriction and increased renal reabsorption of sodium (AII) and water (ADH). The result is vastly decreased renal water loss (with consequent oliguria) and maintenance of plasma and extracellular fluid volume. Finally, water is drawn from the interstitium and the gut to maintain plasma and total body water volume, and thirst and salt-desire are felt (Guyton, 1991).

D. Shock in Birds

In birds, the causes and consequences of shock are poorly understood. The available information covers three causes of shock: dehydration, haemorrhage and endotoxicosis. The relevance of these studies to clinical practice remains to be established, but they could conceivably model dehydration and inanition, which are common features of disease (Phillip, 1981); trauma, which commonly involves haemorrhage; and gastrointestinal disease and sepsis; which can result in endotoxaemia (Wessels et al, 1987; Miyagawa, Numata and Mkuuchi, 1991).

a. Dehydration

Lumeij (1987) documented the existence of prerenal renal failure (the manifestation of shock on the kidneys) by observing that water-deprived pigeons exhibited a 6.5 to 15-fold increase in plasma urea concentration, while creatinine and uric acid levels rose 1.2 to 1.5-fold and 1.4 to 2-fold respectively. This indicates that renal arterial blood supply is decreased during dehydration, while excretion of uric acid continues by virtue of the presence of a renal portal blood supply.

A subsequent study with pigeons, deprived of drinking water for up to 48 hours showed that packed cell volume (PCV) and serum sodium levels did not change despite 10% dehydration. Serum potassium levels also did not change, but this is most likely because avian red blood cells absorb potassium (Lumeij, 1985), and serum is therefore not an appropriate sample with which to estimate potassium status. In the same study plasma total solids and plasma osmolality rose (Martin and Kollias, 1989).

This is at variance to studies in starlings, which did show significant elevations of plasma sodium, chloride and phosphorus concentrations as well as osmolality (Roberts and Dantzler, 1989). Similarly, plasma osmolality rose in Japanese quail deprived of