Fluid therapy in cats can feel like a minefield, they have different requirements from dogs but are more prone to over-perfusion. Natasha Hetzel, Senior Clinical Scholar at Bristol University discusses these concerns and offers a practical guide to fluid therapy in the cat. Fluid therapy can be life saving but is not benign. It is most commonly administered for the treatment of dehydration and shock, to facilitate diuresis (e.g. in acute renal failure) and to correct acid/base or electrolyte disturbances.

**ROUTES OF FLUID ADMINISTRATION**

**Intravenous**

In the emergency feline patient, fluid therapy is usually administered via the intravenous route. The cephalic vein is most commonly utilised but in hypovolaemic patients, or when multiple intravenous catheters have been placed, access may be difficult. The medial saphenous vein runs on the medial aspect of the tibia and is a useful alternative in such patients (Figure 1A and 1B). The jugular vein may also be used in emergency situations. Whilst for long term use, a central line must be placed, an intravenous catheter can be placed temporarily in the jugular vein to facilitate fluid resuscitation. This can be secured in place with butterfly tapes and sutures with a light dressing placed around the neck. Strict asepsis should be observed when placing any intravenous catheter but this is of particular importance when using the jugular vein.

**Intraosseous**

If intravenous access cannot be obtained, intraosseous fluids can be administered either via a commercially available intraosseous needle or a 20 gauge spinal needle. This technique is particularly useful in neonatal patients. Fluid is readily absorbed from the intraosseous space and is not reportedly painful in human patients. The proximal humerus or trochanteric fossa of the femur are suitable sites.

**Subcutaneous/Intraperitoneal**

Due to the vasoconstriction that occurs in shock, these routes are not suitable for volume resuscitation.

How to Establish Intraosseous Access

1. After aseptic preparation of the site, a stab incision should be made into the skin and the needle is then advanced, parallel to the bone, into the medullary cavity using a firm, rotary pressure.
2. When in position, movement of the needle will result in movement of the limb.
3. Gentle aspiration of bone marrow confirms correct placement.
4. The needle should then be flushed with heparinised saline and secured with sutures.
5. Fluids can then be administered via a normal giving set. The site should be closely monitored for dislodgement of the needle and extravasation of fluids into the surrounding tissues. Infection is also a potential complication as in any intravenous catheter but osteomyelitis is unlikely if the site is kept clean.
EQUIPMENT
Cats are much more prone to overinfusion than dogs due to their small size. The rate of fluid administration can be controlled simply by counting and adjusting the drip rate manually. However this method is prone to variation in flow rate due to occlusion of the catheter, often as a result of the patient’s limb position. A paediatric giving set provides 60 drops per ml (rather than the 20 drops per ml of a normal giving set). This allows for more accurate attainment of lower fluid rates. A paediatric burette includes a chamber from which the fluid is administered to the patient. This provides a very cheap method of physically limiting the volume of fluid that can be infused thus reducing the risk of accidental overinfusion.

Figure 2: Drip pump and syringe driver.
An infusion pump or syringe driver (Figure 2) allows for much more accurate control of the rate of fluid administration and will alert the clinician or nurse to occlusion. Pumps and drivers also allow for accurate administration of fluid boluses and continued rate infusions of drugs. They are increasingly available in small animal practice and can be obtained relatively cheaply.

FLUID DISTRIBUTION IN THE BODY
Water can be considered to exist in compartments within the body as shown in Figure 3. Water moves freely between these compartments but remains in approximately the ratios shown. This concept is important in understanding the administration of fluid therapy and the physiological effects of infused solutions. It is particularly relevant for solutions that cause redistribution of water such as colloids and hypertonic saline.

TYPES OF FLUID
The main categories of fluid are crystalloid, colloid, haemoglobin-based oxygen-carrying solutions (e.g. Oxyglobin) and blood products. The latter two are discussed later in this issue in the treatment of anaemia.

CRYSTALLOID FLUIDS
Crystalloid fluids are the mainstay of fluid therapy. They are composed mainly of water with a sodium or glucose base and additional electrolytes or buffers.

REPLACEMENT SOLUTIONS
Replacement solutions are used to replace water and electrolyte deficits and have a similar osmolality, sodium and potassium content as extracellular fluid. Examples of such fluids are sodium chloride (0.9%) and lactated Ringers (Hartmanns). These solutions are used to replace lost water and/or electrolytes such as occurs in vomiting, diarrhoea, third space loss (for example into the gut in the case of intestinal obstruction), polyuria or pyrexia.

NaCl 0.9%
NaCl 0.9% contains a higher level of sodium and chloride than plasma, but no potassium. It is an acidic solution and will have an acidifying effect when infused as haemodilution effectively decreases serum bicarbonate and there is also decreased resorption and increased excretion of bicarbonate by the kidneys.

NaCl 0.9% is contraindicated in the hyperkalaemic patient such as the cat with urethral obstruction. However these patients often also have a metabolic acidosis and an acidifying solution may worsen the hyperkalaemia as H+ ions are moved into the cells in exchange for potassium ions.

Hartmanns Solution
Hartmanns solution, also known as lactated Ringers, contains sodium and chloride at lower levels than are found in the plasma as well as a small amount of potassium, calcium and magnesium. This fluid is hypotonic and is, as such, contraindicated in cerebral oedema (more fluid will move into the brain from the relatively hypotonic blood). The inclusion of lactate in this fluid makes it alkalinising, as lactate is converted to bicarbonate by the liver in a process that consumes hydrogen ions. This makes it an ideal fluid in cases of metabolic acidosis such as occurs in hypovolaemic patients, those with renal disease, vomiting with intestinal losses and diarrhoea. There is some debate about the use of this fluid in patients with reduced hepatic function. However, accumulation of lactate in this situation is probably more of a theoretical risk. Hartmanns is also indicated in patients with normal acid-base status. The amount of potassium in this solution is low and the effects of diuresis mean that this fluid is not contraindicated in the hyperkalaemic patient given that the alkalinising effect may be beneficial. Hartmanns is contraindicated in the hypercalcaemic patient due to its calcium content. It cannot be administered in conjunction with blood products as the calcium can overcome chelation by the citrate anticoagulant and cause agglutination. Hartmanns is also incompatible with bicarbonate, potassium phosphate, magnesium sulphate and many other drugs.

Ringers Solution
This fluid contains a slightly lower level of sodium and chloride than NaCl 0.9% but also contains potassium and calcium. It is an acidic fluid and thus may be useful in cases of metabolic alkalosis but, as for Hartmanns, cannot be administered with blood or many additives.

MAINTENANCE SOLUTIONS
Maintenance solutions are designed to meet the ongoing maintenance needs of a
Hypertonic saline has been suggested as the resuscitation fluid of choice in patients with traumatic brain injury and raised intracranial pressure. The osmotic effect of this solution draws water out of the brain parenchyma into the intravascular space reducing cerebral oedema. For the reasons described above there is a concurrent improvement in haemodynamic status. The method of administration of hypertonic saline is discussed below in the treatment of shock.

**HALF-STRENGTH SALINE (0.45%)**

Half-strength saline is mainly used in the management of hyponatraemic patients. In such cases it is vital that the serum sodium level is not increased too quickly (<0.5mmol/kg/h). If sodium levels change very quickly there is an osmotic effect of water moving from normal brain parenchyma into the intravascular space resulting in dehydration of the brain tissue. This can result in neurological signs. The use of half strength saline in conjunction with saline 0.9% allows the sodium content of the infused solution to be varied, thus controlling the rate of increase in serum sodium concentration.

Half-strength saline may also be utilised in dehydrated patients with cardiac disease where high sodium levels are a concern. Due to the lack of potassium in this fluid, serum potassium should be closely monitored and supplemented as required.

**COLLOIDS**

**Figure 5: Colloid solutions, gelatin, pentastarch and tetrastarch.**

Synthetic colloid solutions are isotonic crystalloid solutions to which large molecules have been added. These large molecules remain in the intravascular space much longer than crystalloid solutions. They increase serum oncotic pressure, and as a result, draw water from the interstitial space into the intravascular space. They therefore increase plasma volume to a greater extent than the volume infused and can thus be given in much lower volumes than crystalloid solutions. They are used in the treatment of hypovolaemia and to provide oncotic support in hypoalbuminaemic patients. The advantages of colloids over crystalloids are the longer intravascular effect, smaller volume requirement to achieve the same degree of plasma volume expansion and decreased risk of tissue oedema. The main disadvantages are allergic reaction, possible renal impairment, coagulopathies and greater cost.

Colloid solutions are classified as synthetic (starches, gelatins and dextrans) or natural (human albumin solutions and plasma).

**Synthetic Colloids**

The advantages and disadvantages of the various colloids depends on many factors and the reader is referred to more advanced texts on the physiochemical properties of colloids for a detailed explanation of the summary given below.

The starches or hydroxyethyl starch (HES) solutions are modified amylopectin molecules. Three different types of HES are available: Hetastarch (eloHaes 6%), pentastarch (Pentastarch 6% and HAES-steril 10%) and tetrastarch (Voluven).

Hetastarch is not currently widely available. It has the longest duration of effect but is more likely to cause coagulopathy and renal impairment. Pentastarch and tetrastarch have a similar duration of effect but pentastarch is a better plasma volume expander than hetastarch or tetrastarch.

Dextran (Dextran 40 and Dextran 70) are composed of naturally occurring glucose polymers. They produce a rapid but temporary plasma volume expansion (half the volume infused is extravasated within three hours). They may cause coagulopathies and renal impairment. Gelatins are derived from bovine collagen that has been alkali treated to increase the molecule size. Urea linked gelatine (Haemacel; Intervet) and succinylated gelatine (Gelofusine; Braun) are both licensed for veterinary use. Gelatins cause marked plasma volume expansion but remain in the intravascular space for a very short period of time (two to three hours). Gelatins are the most likely of the synthetic colloids to cause anaphylactic reactions. They have a limited effect on clotting.

On balance, pentastarch or tetrastarch probably represent the best compromise between duration of effect and plasma volume expansion. They are also less likely to cause allergic reactions and have limited effects on the renal system and coagulation.

**Natural Colloids**

Fresh frozen plasma (FFP) is obtained by centrifugation of whole blood. The plasma is frozen within six hours of collection. Canine FFP is commercially available from the Pet Blood Bank but this product is not currently available for cats.

Human albumin solution (HAS) is a controversial treatment for hypoalbuminaemic patients with concurrent hypovolaemia. It is not widely available, is very expensive and carries the...
risk of anaphylaxis in cats. There is one veterinary report of HAS use that included cats. The majority of work in this field has been conducted in human patients and there is still much debate about HAS use. Further work is required and risk benefit analysis regarding the use of HSA must be applied to each case. Due to availability its use is currently limited to referral institutions.

**FLUID ADMINISTRATION**

The first step in deciding a rational plan for fluid therapy is determining the reason for administering fluid therapy. In the emergency patient a basic clinical assessment of the cardiovascular, respiratory and neurological systems should be performed paying attention to volume and hydration status. It is vital that concurrent disease that may affect the patient's ability to cope with an increase in circulatory volume is also detected. The renal and cardiovascular systems are of particular importance in this regard. The feline patient is extremely sensitive to volume overload and thus fluids must be administered incrementally with close monitoring of fluid resuscitation to end points such as improvement or normalisation of heart rate, pulse quality, mucous membrane colour and mental status.

<table>
<thead>
<tr>
<th>Level of Dehydration</th>
<th>Clinical Signs</th>
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<tbody>
<tr>
<td>&lt;5%</td>
<td>Not detectable</td>
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<tr>
<td>5-8%</td>
<td>Subtle loss of skin elasticity</td>
</tr>
<tr>
<td>6-10%</td>
<td>Definite delay in return of skin to normal position</td>
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<tr>
<td></td>
<td>Eyes may appear slightly sunken</td>
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<tr>
<td></td>
<td>Mucous membranes may appear tacky</td>
</tr>
<tr>
<td>10-12%</td>
<td>Severe skin tenting</td>
</tr>
<tr>
<td></td>
<td>Eyes sunken</td>
</tr>
<tr>
<td></td>
<td>Mucous membranes tacky</td>
</tr>
<tr>
<td>12-15%</td>
<td>Severe skin tenting, sunken eyes and tacky mucous</td>
</tr>
<tr>
<td></td>
<td>membranes +/- signs of shock ; tachycardia/bradycardia</td>
</tr>
<tr>
<td></td>
<td>hypotension, poor pulse quality, pale mucous</td>
</tr>
<tr>
<td></td>
<td>membranes, prolonged CRT, weakness/collapse</td>
</tr>
</tbody>
</table>

**DEHYDRATION**

Dehydration occurs when there is an inadequate intake or excessive loss of water (and electrolytes) from the body. Common causes are vomiting, diarrhoea, anorexia and chronic renal failure. Estimating dehydration is difficult as it relies heavily on subjective assessment of the mucous membranes, capillary refill time and skin tenting. Assessment of skin in the axilla rather than over the “scruff” may be less prone to the effects of subcutaneous fat which can affect skin elasticity. However, it should be remembered that skin elasticity is affected by age and concurrent disease. More objective parameters such as PCV, TP, urea and urine specific gravity are valuable in assessing dehydration and can also be used to monitor the effects of fluid therapy. Figure 7 summarises estimation of the degree of dehydration.

**SHOCK**

Shock is defined as inadequate cellular energy production and usually results from inadequate tissue perfusion due to decreased blood volume or redistributed blood flow. The three main categories of shock are hypovolaemic, distributive and cardiogenic (Figure 9).
FLUID PLAN FOR DEHYDRATION

Crystalloid fluid (Hartmanns or NaCl 0.9%) should be administered. The indications for each fluid are discussed above. The volume to be given is calculated as shown below and is outlined for a 5kg cat in Figure 8.

1. The estimated dehydration is used to calculate the fluid deficit which should be replaced over 24 hours if cardiovascular parameters are stable. Fluid deficit (l) = Body weight (kg) x % dehydration/100.
2. Add in daily maintenance requirement. This approximates to 50ml/kg/day or 2ml/kg/h.
3. Maintenance fluid requirement (ml) = 30 x body weight (kg) + 70
4. The total volume from 1, 2 and 3 should then be administered over 24 hours.
5. The response of the patient to the fluid administration should be monitored closely so that the fluid plan can be increased or decreased as required.
6. Maintenance requirements should be provided using NacI 0.9% or Hartmanns at a rate of 1-2ml/kg/h.

Figure 10: Shock fluid dosages

Hypovolaemic shock is a decrease in circulating blood volume due to haemorrhage or loss of body fluids as occurs in severe vomiting, diarrhoea or polyuria.

Distibutive shock occurs due to a decrease in vascular tone caused by sepsis or systemic inflammatory response syndrome (SIRS). There is therefore no reduction in blood volume but rather a redistribution caused by this lack of vascular tone.

Cardiogenic shock occurs when there is adequate intravascular volume but a failure of the heart to work as a pump to move blood around the body. The resultant lack of tissue perfusion results in cardiogenic shock.

Clinical Signs of Shock

The physiological response to a decrease in cardiac output as occurs in shock is an increase in sympathetic activity to cause tachycardia, vasoconstriction and increased cardiac contractility. However in the cat, the heart rate in the shock patient is very variable and, whilst tachycardia may be seen, cats typically present with bradycardia as well as pale mucous membranes, weak pulses, cool extremities, hypothermia and generalised weakness or collapse.

Treatment of Shock

Fluid therapy is the treatment for both hypovolaemic and distributive shock but is detrimental in cardiogenic shock where an increase in the blood volume clearly presents more of a challenge to an ailing pump. The treatment of cardiogenic shock centres initially on the use of a frusemide to treat congestive heart failure and then on specific treatment of the underlying cardiac disease.

Fluid Plan for Hypovolaemic/Distributive Shock

Shock can be effectively treated with crystalloid or colloid solutions or a combination of both. Many studies have compared the effects of different strategies but it is likely that an equivalent outcome can be achieved if any solution is used appropriately and titrated to the same end points. (Figure 10).

- The golden rules are to administer fluid in increments and to monitor response.
- Fluid therapy can thus be titrated to meet the patient’s requirement and avoid overperfusion.
- Fluid therapy should be continued until end points are achieved.
- The end points are improvement or normalisation of mental status, mucous membrane colour, capillary refill time, pulse rate and blood pressure.
- Signs of over infusion include tachypnoea, crackles on thoracic auscultation and serous nasal discharge.

References available on request.

![Dorbene](https://example.com/dorbene.png)

**Dorbene**

**medetomidine hydrochloride**

On occasion, reference may be made to drugs which are not licensed for use in animals. The Editor does not take any responsibility for the safety and efficacy of such products. Any persons using these products do so entirely at their own risk.

The European Society of Feline Medicine is changing its name and widening its scope because it has been attracting interest, membership and strategic partnerships with individuals and groups way beyond the confines of Europe. ESFM is morphing to ISFM – the ‘European’ becoming ‘International’. Along with the change in name, FAB is undertaking a review of its activities so that those who are veterinary-oriented will all come under the ISFM branding. Information and resources that practitioners can use with clients both in and beyond the consulting room (including client information sheets) will come under the ‘fabcats’ branding. Having just two visible brands will help to clarify the organisation’s activities.

Members of ISFM will receive the monthly Journal of Feline Medicine and Surgery which now incorporates six clinical practice issues. It is a must for any veterinary team which sees cats regularly in practice. ISFM will also develop international guidelines and policies on important and relevant feline issues and collaborate with veterinary cat groups worldwide, where possible through ISFM National Partnerships, to share information, expertise and knowledge of ‘best practice.

For further information on ISFM go to [www.isfm.net](http://www.isfm.net) and for client-information sheets to [www.fabcats.org](http://www.fabcats.org).
Bertie, a 2 year old male neutered domestic shorthair, presented as an emergency to the Small Animal Surgery Department at Langford Veterinary Services following a road traffic accident, during which he had sustained multiple severe and life-threatening injuries.

**Initial Presentation:** Upon arrival at the hospital, Bertie was very painful (diffuse pain, difficult to localise) and was mouth breathing. Increased respiratory effort and absent lung sounds on auscultation bilaterally were documented.

**Initial Management:** An intravenous catheter was placed. Given the suspicion of a pneumothorax and how painful the cat was methadone was administered intravenously and 400ml of air drained by thoracocentesis, following which he appeared much more settled and comfortable.

**Ongoing Management:** Over the next hour, Bertie was given IV fluids and serum biochemistry and haematology were assessed, which showed moderate anaemia but were otherwise within normal limits. Full clinical examination revealed that he was particularly painful over his pelvis and was not able to stand on his hindlimbs, suggestive of multiple fractures, but the remainder of the physical and neurological examination was unremarkable.

**Figure 1:** Right lateral thoracic radiograph showing severe pneumothorax and ventral disruption of the border of the diaphragm, which was suspicious of a rupture.

Unfortunately, Bertie’s respiratory effort started to increase again and thoracic radiography revealed that the pneumothorax was recurring and was also suspicious of a diaphragmatic rupture (**figure 1**), which was confirmed by ultrasonography.

**Surgical Treatment:** Surgery was performed immediately and a ventral midline laparotomy showed a circumferential tear of the ventral diaphragm (**figure 2**), which was repaired using 2-0 PDS in a simple continuous pattern.

**Figure 2:** Intraoperative photograph showing the diaphragmatic tear

Further exploration of the abdomen showed major retroperitoneal haemorrhage and prevented inspection of the ureters, so an intravenous urogram and retrograde positive contrast urethrocystogram were performed, which were normal. Postoperative radiography of the thorax showed improvement of the pneumothorax and adequate placement of the chest drain.

**Recovery Period:** Bertie recovered from anaesthesia, but developed severe hypotension and tachycardia which was temporarily responsive to boluses of intravenous crystalloids. Abdominal ultrasonography was unremarkable, but thoracic ultrasonography showed a moderate volume of mixed echogenicity fluid and this was found to be consistent with haemorrhage. Because the respiratory rate and effort remained normal, the chest drain was not used to drain the haemorrhage and Bertie received a whole blood transfusion, following which he appeared much brighter and was able to maintain an appropriate heart rate and blood pressure.

**Ongoing Management:** After several days in ICU, Bertie was very bright, his anaemia had improved and his coagulation profiles had normalised. He was then taken to theatre to repair his orthopaedic injuries (**figure 3**), namely the left sacroiliac fracture-separation and the right ilial fracture. These were repaired with a 2.4mm 22mm lag screw and a 5 hole 2.4mm dynamic compression plate respectively (**figure 4**). Bertie recovered from his second surgery uneventfully and was discharged from hospital two days later (8 days after his original admission). He is to receive 6 weeks strict cage rest, after which he can walk around the house for a further four weeks before finally being let outside (and away from any more roads)!

**Discussion:** Bertie is an excellent example of a cat with multiple severe injuries following a road traffic accident and demonstrates the importance of a thorough clinical examination and prioritising life threatening injuries above orthopaedic injuries. Clients should be made aware that staged procedures may be required and that multiple shorter surgeries may be preferable.
The adage that “cats are not small dogs” is particularly pertinent when assessing the feline emergency patient presenting with anaemia, typically on a Friday night! The owner of an anaemic dog will usually notice lethargy and depression at an earlier stage than the owner of an anaemic cat, because of differences in activity levels and behaviour between the species. Further information on the causes and investigation of anaemia is beyond the scope of this article and a full explanation can be found in The Feline Update Spring 2008.

**Therapeutic principles:**

Initial requirements for patient stabilisation are dependent on the rapidity of onset type and underlying cause of the anaemia. Cats in peracute collapse require more immediate, intensive and aggressive stabilisation. In contrast cats with chronic anaemia are frequently cardio-vascularly stable (based on heart rate, pulse quality, respiratory rate and systolic blood pressure and general demeanour) despite their low PCV and aggressive treatment may not be required. Patients with evidence of blood loss or hypovolaemia will benefit from immediate IV fluid therapy to ensure organ perfusion. Initially this can be administered as crystalloid therapy (see front page article) although crystalloids will redistribute rapidly (within 30 minutes) and some patients may require additional products such as synthetic colloids or haemoglobin-based oxygen carrying solutions.

**Monitoring systolic blood pressure is important when managing these patients as an indication of ongoing blood loss and the success of resuscitation.**

Synthetic colloids hold fluid within the vascular space for longer than crystalloid fluids. They may be used in combination with crystalloids to maintain adequate plasma volume expansion. Caution should be used when administering these products to patients with a suspected coagulopathy as their affect on coagulation is unpredictable in critically ill patients.

**Blood transfusions**

Blood transfusions in cats are associated with a sharp intake of breath by some clinicians. With a sensible approach and careful monitoring of both donor and recipient cats, blood transfusions can provide life saving treatment for the anaemic feline patient. They are indicated in cases of severe anaemia, where the anaemia is associated with clinical signs. Blood transfusions provide oxygen carrying capacity, coagulation factors and will have a colloidal effect although provide very few platelets.

**There is no specific cut off point in haematological parameters indicating the need for a blood transfusion. A decision to transfuse should only be made if the patient is demonstrating signs of cardio-respiratory compromise such as weakness, bounding peripheral pulses, hypotension, tachycardia (and in cats sometimes bradycardia) and tachypnoea/dyspnoea.**

The potential concerns associated with blood transfusions in cats stem from the cat’s blood type system (AB system). Within this system there are three blood types: type A, type B, and type AB. In contrast to dogs, cats have naturally occurring antibodies against the blood type they are lacking. Type B cats often possess high anti-A allo-antibodies and a small volume of type A blood administered to a type B cat can result in a severe and fatal transfusion reaction. Certain breeds of pedigree cat are more likely to have particular blood types but this cannot always be predicted. Most non-pedigree cats are type A, but type B and AB moggies are encountered with increasing frequency. The prevalence of type B is high in breeds such as the British Shorthair, Ragdoll, Birman and Rex amongst others.

**Blood typing is essential and no cat should be transfused without prior blood typing.**

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**Characteristics of a Suitable Blood Donor**

- Healthy
- Large cats (>4kg) but not obese
- Calm temperament
- Age 1-8 years
- Ideally indoor only (not always possible)
- Fully vaccinated
- Blood typed (VITAL)
- FeLV, FIV and Haemoplasma spp negative (PCR)
- Ideally normal echocardiogram (particularly breeds at risk of cardiac disease e.g. Maine Coon)
- PCV >35% (and normal haematology and biochemistry)
- Normal blood pressure

Occult cardiac disease remains a concern and cannot be excluded without echocardiography. A list of healthy and willing donors should be kept by the practice to be called in an emergency allowing advanced blood typing to be performed.

**Collection of blood**

Collection of blood from the donor cat should be performed in a quiet, stress free environment. The donor cat should be weighed and a PCV checked prior to donation. 5 x 10ml syringes should be prepared with anti-coagulant (1ml citrate phosphate dextrose acid (CPDA) or acid citrate dextrose (ACD) per 9 mls of blood). The anticoagulant can be taken out of human blood collection bags used for canine blood donation. In most cases the donor will need to be sedated. A combination of midazolam (0.25 mg/kg) and ketamine (5mg/kg) can be administered intramuscularly. Agents resulting in hypotension should be avoided. An IV catheter should be placed to facilitate administration of crystalloids post donation and in case of emergencies. Blood is obtained from the jugular vein by restraining the cat in ventral recumbency with their neck elongated and their nose pointing towards the floor with the person restraining the cat raising the vein at the base of the neck. Be careful to ensure a patent airway at all times. The jugular vein should be prepared using aseptic technique and a 21G needle or 19G butterfly catheter with a 3 way tap attached is inserted into the vein. Up to 20% of blood volume (total blood volume = approximately 66ml/kg) can be collected as long as IV crystalloid replacement fluids are administered. For example a 4kg cat can donate 30ml blood = 20% blood volume. Blood collection is easiest as a three man procedure. Owners of the donor cat should be signed to facilitate the donation. A full examination of the donor cat should be completed to ensure mixing. Twice to three times the volume of blood removed from the donor should then be replaced with IV crystalloids over 30-40 minutes.
DIABETIC KETOSIS (DKA)

From a fluid therapy perspective it is important to know the following:

- **Sodium levels** – most patients are hyponatraemic due to excess urinary sodium loss caused by osmotic diuresis.
- **Potassium levels** – usually DKA patients have a marked whole body potassium deficiency due to shifts of potassium from cells into ECF and urinary potassium loss.
- **Phosphate levels** – phosphate also shifts from the cells into the ECF and there is enhanced urinary loss so there is often whole body depletion of this electrolyte as well.
- **Acid base status** – a metabolic acidosis results from accumulation of ketones (high anion gap acidosis).
- **Blood glucose levels** – on average 25mmol/l but can range from 10mmol/l to over 50mmol/l.

**TREATMENT**

Fluid therapy – 0.9% saline is an appropriate starting fluid in the hyponatraemic patient. Calculate the fluid rate as below.

% dehydration x body weight (kgs) = volume to be replaced (litres) over 24 hours
+ maintenance fluid requirements = approximately 50mls/kg/day
+ Ongoing losses (estimate of volume loss in vomitus, diarrhoea)

NB: Requirements will increase as a result of polyuria caused by ketonuria/glucosuria.

**Potassium supplementation** – cats with DKA have whole body deficits in potassium and once treatment is initiated there is often a dramatic decrease in serum levels. Supplement based on serum potassium levels which should be regularly assessed. Potassium chloride can be added to 0.9% saline. Hartmann’s or Ringer’s solution, see first article (‘Fluid therapy in the Emergency Feline Patient’ and the BSAVA formulary) for guide to potassium supplementation.

**Guide to potassium supplementation**

<table>
<thead>
<tr>
<th>Serum potassium (mmol/l)</th>
<th>Amount of potassium (mmol) to add to 500ml of NaCl 0.9%</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2</td>
<td>40</td>
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<tr>
<td>2-2.5</td>
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<td>3.5-5.5</td>
<td>10</td>
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</table>

To make a 2.5% dextrose solution, add 50mls of 50% dextrose to a litre bag of crystalloids such as Hartmann’s or sodium chloride. Pre-constituted (‘ready made’) 5% dextrose solutions will perpetuate electrolyte abnormalities so making up a solution as above provides more balanced electrolyte concentrations.

**Acid base status** - is helped by correcting dehydration and restoring water and electrolyte levels. Insulin therapy has to be instigated to stop the formation of ketones and resolve the ketoadidosis. Bicarbonate therapy to correct the acidosis can have dangerous complications and is usually reserved for patients with plasma bicarbonate levels <12mEq/l or total venous CO2 concentrations of <12mmol/l. If the levels are not measurable it should only be given if the animal is dangerously acidic (pH <7.0) and close monitoring is available.
ACUTE RENAL FAILURE (ARF)

**GOALS OF TREATMENT:**
1. Correction of fluid deficits and ongoing fluid therapy
2. Restore renal perfusion and urine output
3. Correction of acid-base and electrolyte abnormalities

1 Fluid therapy is the mainstay of the treatment of ARF. Most cats will be hypovolaemic on presentation and a bolus (10-20 ml/kg) of crystalloid administered over 10-20 minutes should be given to restore renal perfusion and repeated as indicated by regular reassessment. Following correction of hypovolaemia fluid requirements for the first 24 hours should be calculated according to dehydration and ongoing losses (see treatment of DKA). Remember that once urine output is restored the polyuric phase will result in large fluid losses. Conversely over perfusion is a risk if the patient is oliguric/anuric.

2 Some patients may be oliguric or even anuric with ARF. Urine production should be measured via placement of a urinary catheter and closed collection system, which will also allow matching of fluid input to output during the polyuric phase. In some cases correction of hypovolaemia will restore urine output. A fluid challenge can be administered (if possible monitoring central venous pressure, or as a minimum blood pressure and for signs of over perfusion; including regular thoracic auscultation for pulmonary oedema) with a crystalloid bolus of 5-10 ml/kg over 20 minutes. If output remains inadequate (i.e. <0.5 ml/kg/hr) then diuretics (furosemide initially dose 2 mg/kg IV) should be administered. A second bolus can be given after 30 minutes if oliguria persists. A continuous rate infusion of furosemide (1 mg/kg/hr) could also be used; evidence in dogs suggests an increased diuretic effect via this route. Mannitol is an alternative diuretic with additional effects of increasing renal blood flow. A dose of 0.25-0.5 g/kg is administered intravenously over 5-10 minutes.

A response would be expected within 20-30 minutes and the patient should be monitored for over hydration. Mannitol should not be used if the cat is hyperosmolar, as often occurs in ethylene glycol toxicity. Low doses of dopamine (0.5-3 µg/kg/minute) may also be used, particularly if hypotension remains present despite correction of volume deficits.

**DIURETICS SHOULD ONLY BE ADMINISTERED ONCE VOLUME DEFICITS ARE CORRECTED.**

3 Serum potassium may be high in oliguric/anuric patients but may then be lost excessively in polyuric states. Hyperkalaemia may resolve with volume expansion and restoration of urine production. If, as a result of hyperkalaemia, significant ECG abnormalities are observed then administer IV calcium gluconate (10% solution, 0.5-1.0 ml/kg) whilst monitoring the ECG. This will not lower potassium but will stabilise cardiac membranes. Other treatments to lower potassium include glucose, regular insulin and bicarbonate. Hypokalaemia can occur during the polyuric phase and should be corrected accordingly.

A metabolic acidosis is common. Addressing the hydration status and electrolytes will help. Addition of bicarbonate should be reserved for extreme acidosis (pH<7.0) and ideally only with blood gas analysis available. Tapering of parental fluid therapy can be attempted once the azotaemia has stabilised for 2-3 days and there is an improvement in the clinical status. Successful withdrawal of parental therapy relies on the cat eating and drinking voluntarily and also a reduction in ongoing losses through vomiting and diarrhoea. In general, a reduction of 25% of parenteral fluid volume daily for 2-3 days can be attempted and monitoring of urea and creatinine levels should be performed.

Additional treatments include antimetics and gastroprotectants. Attention to nutrition is required with assisted feeding (e.g. naso-oesophageal tube) if anorexia persists.

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**Introducing the New FAB Scholar at Bristol University:**

**Lara Boland**

Lara graduated from Sydney University in 2004 and is originally from Canberra, Australia. After completing internships at both the Animal Referral Hospital in Sydney and the University of Bristol, Lara is looking forward to moving back to the UK to start an FAB sponsored residency in feline medicine.

Lara has always been fascinated by all things feline and completed the Australian College of Veterinary Scientists membership examinations in feline medicine in 2008. She is particularly interested in feline endocrine diseases, urinary tract diseases and emergency medicine.

Lara has been adopted by three cats, Munchkin, Squeak and Wednesday; all rescue moggies from veterinary hospitals at which she has worked.
In this issue of Feline Update, Christina Maunder, Senior Clinical Training Scholar at Langford, discusses an interesting emergency case she saw recently. Pumpkin, a 4 yr old, previously healthy, male neutered DSH presented with a 24 hour history of vomiting, anorexia and depression. On presentation the cat was profoundly depressed and poorly responsive with reduced peripheral pulse quality and a small, empty bladder.

Bloods were taken (abnormal results in bold).

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>MEASUREMENT</th>
<th>REFERENCE RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea</td>
<td>36.5</td>
<td>6.5-10.5 mmol/l</td>
</tr>
<tr>
<td>Creatinine</td>
<td>420</td>
<td>133-175 µmol/l</td>
</tr>
<tr>
<td>Total protein</td>
<td>81</td>
<td>77-91 g/l</td>
</tr>
<tr>
<td>Albumin</td>
<td>34</td>
<td>24-35 g/l</td>
</tr>
<tr>
<td>Globulin</td>
<td>47</td>
<td>21-51 g/l</td>
</tr>
<tr>
<td>ALT</td>
<td>34</td>
<td>15-45 IU/l</td>
</tr>
<tr>
<td>ALKP</td>
<td>42</td>
<td>15-60 IU/l</td>
</tr>
<tr>
<td>Sodium</td>
<td>156</td>
<td>149-157 mmol/l</td>
</tr>
<tr>
<td>Potassium</td>
<td>6.2</td>
<td>4-5 mmol/l</td>
</tr>
<tr>
<td>Calcium</td>
<td>2.0</td>
<td>2.3-2.5 mmol/l</td>
</tr>
<tr>
<td>Phosphate</td>
<td>1.95</td>
<td>0.95-1.55 mmol/l</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>12.4</td>
<td>8-15 g/dl</td>
</tr>
<tr>
<td>Haematocrit</td>
<td>40.3</td>
<td>25-45%</td>
</tr>
<tr>
<td>RBC</td>
<td>7.31</td>
<td>5.5-10 x 10¹²/l</td>
</tr>
<tr>
<td>Platelets</td>
<td>325</td>
<td>200-700 x 10⁹/l</td>
</tr>
<tr>
<td>WBC</td>
<td>5.11</td>
<td>4.9-19 x 10⁹/l</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>4.08</td>
<td>2.4-12.5 x 10⁹/l</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>0.62</td>
<td>1.4-6 x 10⁹/l</td>
</tr>
<tr>
<td>Monocytes</td>
<td>0.08</td>
<td>0.1-0.7 x 10⁹/l</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0.25</td>
<td>0.1-1.6 x 10⁹/l</td>
</tr>
<tr>
<td>Basophils</td>
<td>0.08</td>
<td>0-0.1 x 10⁹/l</td>
</tr>
</tbody>
</table>

• 1. Discuss the changes seen on the laboratory results.
• 2. What further tests might you perform?

• 1. The biochemical changes include hyperphosphataemia and azotaemia, and are consistent with acute renal failure (ARF). The hyperkalaemia is suggestive of oliguria or anuria. There are no changes in red blood cell parameters and the history is of acute disease but an acute decompensation of chronic kidney disease (CKD) remains possible. The urea and creatinine are both elevated suggesting a pre-renal, renal or post-renal cause. There is a mild total hypocalcaemia which warrants further investigation and preferably confirmation via measurement of ionised calcium. The lymphopenia may reflect underlying disease and stress, and the mild monocytopenia was not considered significant.

Differential diagnoses for azotaemia

PRE-RENAL: Poor renal perfusion (shock, dehydration, haemorrhage, heart failure)

RENAL: Nephrotoxicosis (e.g. ethylene glycol, lily poisoning, NSAID, aminoglycoside, grapes/raisins)

Renal ischaemia (e.g. reduced cardiac output, hypovolaemia, renal vascular thrombosis)

Renal parenchymal disease (e.g. pyelonephritis, FIP, FeLV, glomerulonephritis, amyloidosis, neoplasia e.g. renal lymphoma)

POST-RENAL: Urethral or ureteral obstruction; bladder, urethral or ureteral rupture

• 2. It is important to obtain a urine sample before treatment to assess the specific gravity and cytology. Ideally a sample should also be collected by cystocentesis where possible for culture and sensitivity. The azotaemia should be interpreted in light of the urine specific gravity. With pre-renal and post-renal azotaemia the specific gravity would be expected to be high (>1.035) unless there was concurrent disease present that altered renal concentrating ability. Inadequately concentrated urine (SG 1.008-1.029) suggests a primary renal disease is present. Cytology may also demonstrate the presence of red blood cells, white blood cells or casts. Urinalysis should also be performed to measure protein levels (always quantify with a urine protein : creatinine ratio) and glucose. Further investigation including measurement of ionised calcium and assessment of acid-base status are also indicated in the current case, as well as measurement of blood pressure given the poor peripheral pulse quality.

A urinary catheter was placed to allow measurement of urine output and a urine sample obtained. Urine was examined under the microscope (figure 1). The urine specific gravity was 1.012.

Figure 1: Urine cytology (x500 magnification). Picture: Kathleen Tennant.

3. What conclusions can be made from the urinalysis?

• There is crystalluria present which consists of calcium oxalate monohydrate crystals. These have pointed to rounded
extremities, variable size and are 6 sided clear prisms. They have a 2-dimensional appearance. They can be mistaken for struvite (which have a more 3 dimensional appearance, figure 2).

Figure 2: Struvite crystals. Picture: Kathleen Tennant

Calcium oxalate dihydrate crystals have an appearance typical of a mail envelope or Maltese cross (figure 3).

Figure 3: Calcium oxalate monohydrate crystals. Picture: Kathleen Tennant.

The presence of calcium oxalate crystals and azotaemia is suspicious of ethylene glycol (anti-freeze) toxicity. Calcium oxalate monohydrate crystals can also be seen in hypercalcaemic states and oxalate toxicity. The urine specific gravity is consistent with isosmolaruria which indicates renal insufficiency and significant loss of functional nephrons. The cat’s acid base status was established and ionised calcium measured.

4. Comment on the i-STAT results (below).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Result</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ionised calcium (mmol/l)</td>
<td>0.90</td>
<td>1.2 - 1.4</td>
</tr>
<tr>
<td>pH</td>
<td>7.20</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>HCO3 (mEq/l)</td>
<td>16</td>
<td>20-24</td>
</tr>
<tr>
<td>pCO2 (mmHg)</td>
<td>30</td>
<td>30-45</td>
</tr>
<tr>
<td>Anion Gap (mEq/l)</td>
<td>25</td>
<td>&lt;10</td>
</tr>
</tbody>
</table>

There is an ionised hypocalcaemia. Causes include hypoparathyroidism (most commonly iatrogenic), CKD, ARF (particularly caused by ethylene glycol), acute pancreatitis, peri-partum hypocalcaemia and hypovitaminosis D. There is a moderate metabolic acidosis with respiratory compensation. The severely elevated anion gap indicates additional unmeasured anions in the circulation which can include lactate, ketones, metabolites of ethylene glycol.

5. What is the most likely diagnosis in this case?

- The combination of ARF, an increased anion gap acidosis and hypocalcaemia is consistent with ethylene glycol toxicity causing ARF. In the first 48 hours of intoxication, hepatic metabolism of ethylene glycol results in formation of glycoaldehyde, glycolic acid, glyoxalic acid, and oxalic acid. These metabolites cause a metabolic acidosis and a pH <7.4. The i-Stat measurements document an elevated anion gap due to the presence of these unmeasured anions and the hypocalcaemia results from the complexing of oxalate with calcium, and the deposition of these complexes in the renal tubules results in ARF.

6. What further diagnostic tests could be performed to attempt to confirm the diagnosis?

- An ethylene glycol test kit exists in the US but this is not commercially available in the UK. This test only measures the levels of ethylene glycol before it is metabolised (which may take only 12 hours). The toxic levels in cats may also be below the level the test kit can detect giving a false negative result.
- Some commercial antifreeze solutions have fluorescein dye added to help detect leaks in the coolant system. Fluorescence may be detected in the oral cavity, vomitus or urine by Wood’s lamp but this is not a reliable test as the brand of antifreeze is usually not known and fluorescein may not be present.
- Abdominal ultrasound – marked hyperechogenicity of the renal cortices is suggestive of ethylene glycol toxicity although hyperechogenicity is common in both acute and chronic renal diseases of other causes.

7. What treatment would you instigate?

- Cats are rarely observed ingesting the ethylene glycol so gastric decontamination (induction of emesis, gastric lavage) and activated charcoal are not beneficial. Decontamination is necessary within an hour of ingestion because absorption of ethylene glycol is very rapid and peak plasma levels are achieved within 3 hours. Also given the cat’s reduced mentation/depression emesis would be associated with a significant risk of aspiration.
- Successful treatment of ethylene glycol intoxication must begin before toxic metabolites are generated and therefore before the development of ARF. Unfortunately most cats are only presented when ARF has already developed. Ethanol is used, as this is substituted for ethylene glycol as a preferred substrate for alcohol dehydrogenase. Ethanol treatment must be administered within 8-12 hours of ingestion in order to be effective. Once azotaemia has developed the ethanol is unlikely to be successful and administration may complicate management. A 20% medical ethanol solution is used and 5mls/kg administered intravenously over 15 minutes. This can be repeated every 6 hours for 5 treatments, then every 8 hours for 4 treatments.
- Alternatively constant rate infusions have been administered for up to 72 hours (1.25mls/hour). In an emergency if medical ethanol is not available then plain vodka has been used with the dose adjusted for the alcohol percentage of the preparation. Oral administration of alcohol has been used but gastric irritation can result in vomiting and in depressed patient aspiration is a significant risk. Ethanol is a central nervous system depressant so will necessitate hospitalisation and monitoring of respiratory and cardiac depression. Peritoneal dialysis is available at some centres. If oliguria or anuria are present the prognosis is grave. Please see ‘Top Tips’ on page 9 for further information on the treatment of ARF.

Outcome:
Unfortunately Pumpkin presented in severe acute renal failure and produced no urine despite adequate fluid therapy and aggressive diuresis. Euthanasia was elected and post-mortem examination confirmed the diagnosis of ARF due to ethylene glycol toxicity. The Feline Advisory Bureau has recently produced a ‘fax back’ sheet for veterinary surgeons with practical advice on the diagnosis and treatment of ethylene glycol toxicity. See www.fabcats.org for more information and report all suspected poisonings to the VPIS on 020 7188 0200.
Administration of a blood transfusion
Blood products should be administered into the cephalic vein (intra-osseous administration can be used in young kittens, or if peripheral access not available). ALWAYS using a dedicated blood giving set with an appropriate filter to reduce aggregation and prevent microthrombi formation. The filters in standard fluid therapy giving sets are not suitable. Blood products should not be administered at the same time as fluids containing calcium (Hartmann’s) or glucose. The amount to be transfused depends on the level of anaemia and generally 2 ml of whole blood will increase the PCV by 1%.

A formula to calculate volume of blood to give is:

\[
\text{Volume to be transfused} = 66 \times \text{Weight of patient (kg)} \times (\text{target PCV (usually 20%)} - \text{patient PCV})
\]

PCV of donor

Speed of administration
The speed of administration should be determined by the condition of the patient. A balance must be found between the risk of a transfusion reaction with rapid administration and bacterial contamination/blood deterioration if not administered within 4 hours of collection. An initial rate of 0.5 ml/kg/hour for the first 5 to 15 minutes is appropriate and the patient should be monitored constantly for signs of a transfusion reaction (see Box). The rate can then be slowly increased up to 10 ml/kg/hour hypovolaemic patients can receive up to 20 ml/kg/hr with close monitoring.

Plotted a graph (i.e. using an anaesthesia monitoring form and recording temperature, pulse and respiration initially every 5 minutes) allows early identification of a reaction. Should the patient develop mild signs of a transfusion reaction (e.g. mild increase in temperature, one episode of vomiting) then the transfusion rate should be reduced and close monitoring continued. If marked clinical signs develop the transfusion should be stopped and the blood replaced with a crystalloid solution. Antiinflammatory doses of corticosteroids may be administered in conjunction with blood transfusion in general practice. See practical guide for vets and nurses on obtaining and administrating a blood transfusion. See any correspondence should be addressed to Samantha Taylor or Matthew Rowe BSc (Hons) at Pfizer Animal Health, Walton Oaks, Walton On The Hill, Dorking Road, Tadworth, Surrey, KT20 7NS.

Informed consent should always be obtained from the owner prior to administration. Although HBOC solutions are expensive, blood collection with appropriate donor assessment and recipient monitoring may actually be more costly.

Signs of a transfusion reaction
- Tachycardia
- Tachypnoea
- Hypotension
- Vomiting/hypersalivation
- Urticaria (facial swelling)
- Pyrexia
- Vocalisation and distress
- Haemoglobinuria

Indications for use of HBOC solutions in cats
- Euvolaemic anaemia (haemolysis or failure of production) - the rates of infusion should be kept low to avoid volume overload but Oxyglobin can provide oxygen carrying support whilst treatment is administered or the cause of the anaemia investigated
- Hypovolaemic anaemia (blood loss) - the colloidal support and oxygen carrying capacity can be life saving. Higher rates can be used as indicated by clinical examination, response to infusion and ongoing blood loss
- To stabilise an anaemic patient prior to/ during a procedure e.g. bone marrow biopsy, without interfering with results
- In patients where a blood transfusion is not possible i.e. no available donors, unusual blood groups (type B cats)

Considerations regarding administration of Oxyglobin to cats
- Potential side effects – volume overload is a significant risk, given the colloidal effect of Oxyglobin, therefore infusion rates should be kept low (0.5-2ml/kg hour) and cats monitored for signs of over-perfusion
- Considering the colloidal effect, HBOC solutions should not be used in patients with cardiac disease, pulmonary parenchymal disease, acute renal failure or cerebral oedema
- Although no cross-matching is required an immunological reaction to bovine haemoglobin can occur and patients should be monitored accordingly
- Changes in mucous membrane colour will occur with a brownish tinge and later a yellow-orange colour, urine may also become discoloured
- Use of Oxyglobin will interfere with laboratory testing including biochemical colormetric assays and haematological variables such as MCH and MCHC
- After infusion the PCV is often reduced through haemodilution, but an approximation of PCV can be made using the formula: PCV (%) = Hb (g/dl) x 3

Hypovolaemic anaemia (blood loss) – the transfusion should be stopped and the blood replaced with a crystalloid solution. Antiinflammatory doses of corticosteroids may be administered in conjunction with blood transfusion in general practice. There is often debate associated with administration of HBOC solutions versus whole blood transfusions in cats. HBOC solutions (e.g. Oxyglobin®, Dechra Pharmaceuticals, figure 3) increase plasma and total haemoglobin concentration and therefore increase the oxygen carrying capacity. They do not provide any plasma constituents such as clotting factors, platelets or other plasma proteins. HBOC solutions are not licensed for use in cats however they are very useful given the difficulties associated with blood transfusion in general practice.

In cats, rates suggested for canine patients are too rapid and will frequently result in signs of volume overload. We typically use a more conservative rate of 0.5-2 ml/kg/hr with total doses of 5-10ml/kg administered over 6-12 hours or longer. A bolus dose of 0.2-1ml/kg over 5-10 minutes can be given in an emergency in a hypovolaemic patient. The decision to continue...