Journal of Feline Medicine and Surgery (2013) **15**, 189–199

CLINICAL REVIEW

THE FLAT CAT
2. The emergency database and management of common metabolic abnormalities

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Aim: Detailed information regarding the causes and treatment of acute collapse in the cat can be difficult to locate in a single published source. This two-part review aims to provide a logical approach to the clinical assessment and stabilisation of the critically ill collapsed cat.

Practical relevance: Laboratory evaluation, in the form of an emergency database, is an important part of the initial assessment of a collapsed patient and should be considered in conjunction with physical abnormalities.

Clinical challenges: Rapid identification and correction of life-threatening metabolic abnormalities, including hypoglycaemia, hypocalcaemia and hyperkalaemia, is essential in stabilising this group of patients. Clinicians often lack confidence if they are not dealing with these problems regularly.

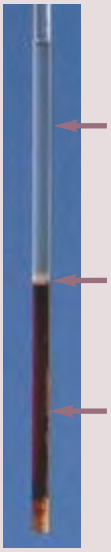
Audience: The information provided in this article will be of use to any veterinarian working with feline patients and particularly those dealing with emergencies on a regular basis.

Evidence base: There is an extensive body of published literature, both original studies and textbook chapters, pertaining to the causes and treatment of the important metabolic abnormalities covered in this article. The authors draw on information from original articles, reviews and their clinical experience to provide simple but detailed practical information to guide interpretation of the emergency database and its application to therapy in the emergency setting.

The emergency database – what you really need to know in the first 30 mins

An emergency database should comprise a series of laboratory tests, the results of which will influence your immediate stabilisation of the patient. Often, further laboratory testing is subsequently performed. An emergency database could be as limited as a packed cell volume (PCV) and total solids (TS) from a microhaematocrit centrifuge and a refractometer, dependent on the equipment available within the practice. A suggested 'essential' database is shown below. Assessment of blood gases, lactate, ionised calcium and magnesium is helpful but often unavailable in first opinion practice.

Suggested 'essential' emergency database



Microhaematocrit tube

- PCV measured on microhaematocrit tube
  - observe serum/plasma colour: pink = haemoglobinaemia; yellow = hyperbilirubinaemia
  - observe size of the buffy coat
- Total solids measurement on refractometer
  - estimate of plasma proteins
- Blood smear
  - check for platelets, erythrocyte morphology, white blood cells, parasites
- Urea
- Glucose
- Sodium, potassium (chloride)
- Calcium
- Blood gases (venous sample is suitable for assessment of acid–base status)
  - pH, bicarbonate, PCO_2, PO_2, base excess, electrolytes
- Lactate

Part 1
Part 1, which reviews a logical approach to the challenging presentation of the collapsed cat, appears on pages 175–188 of this issue of *J Feline Med Surg* and at DOI: 10.1177/1098612X13477538

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DOI: 10.1177/1098612X13477539

Abnormalities on the emergency database

The more common abnormalities identified on an emergency database are highlighted, with differential diagnoses, in the boxes below and on page 191. In all cases, investigations will be required to determine the underlying cause of the laboratory abnormality. However, immediate management of some abnormalities including hypoglycaemia, hyperkalaemia, hypo/hypernatraemia, hypocalcaemia and anaemia may be necessary to stabilise the cat and prevent further detrimental effects.

Differential diagnoses for common abnormalities on the emergency database

Hypoglycaemia
- Sepsis
- Hepatic dysfunction (acquired/congenital)
- Iatrogenic; eg, insulin overdose, remission of diabetes mellitus during insulin therapy
- Neonatal
- Starvation and severe malnutrition
- Insulinoma
- Paraneoplasia
- Hypoadrenocorticism
- Erythrocytosis and leukocytosis

Hyperglycaemia
- Physiological – 'stress'
- Diabetes mellitus (DM)
- Diabetic ketoacidosis (DKA)
- Hyperglycaemic hyperosmolar syndrome
- Iatrogenic; eg, supplemented fluids
- Pancreatitis (decreased insulin production and antagonism)
- Drug or toxin effect – glucocorticoids, progestagens, ethylene glycol
- Acromegaly
- Hyperadrenocorticism

Hypokalaemia
- Anorexia
- Vomiting or diarrhoea
- Iatrogenic; eg, post-frusemide, post-insulin treatment in DKA
- Hyperaldosteronism
- Diuresis
- Kidney disease
- Hypokalaemic myopathy in Burmese cats
- Metabolic alkalosis
- Refeeding syndrome
- Chronic liver disease

Hyperkalaemia
- Post-renal urinary tract obstruction (urethral or ureteral)
- Ruptured bladder
- Acute kidney injury – anuric, oliguric
- Muscle trauma; eg, post-seizures, trauma
- Iatrogenic; eg, excessive supplementation of IV fluids, ACE inhibitors, NSAIDs, spironolactone
- Haemolysis
- Metabolic acidosis
- Hypoadrenocorticism
- Peritoneal or pericardial effusion
- Repeat drainage of chylothorax
- Gastrointestinal (GI) disease; eg, ruptured duodenum
- Spurious; eg, sample contamination with EDTA

Hyponatraemia
- Na^+ loss > H_2O loss:
  - GI disease (vomiting, diarrhoea)
  - Renal loss
  - Third space loss
  - Severe burns
  - Hypoadrenocorticism
  - Diuresis
- H_2O excess:
  - Congestive heart failure
  - Nephrotic syndrome
  - Hepatic failure
  - Excess hypotonic fluid administration; eg, 0.18% glucose saline
- Compartmental shifts of Na^+ or H_2O
  - Hyperglycaemia (DM)
  - Mannitol administration
  - Acute muscle damage
  - Uroperitoneum

Hypernatraemia
- H_2O deficit (Na^+ free fluid loss or reduced intake)
  - Adipisa, hypodipsia
  - Diabetes insipidus
  - Renal or GI water loss
- Hypotonic fluid loss
  - GI loss – vomiting or diarrhoea
  - Renal loss; eg, osmotic diuresis in DM, glucose-containing IV fluid therapy
  - Third space loss
- Na^+ excess
  - Hypertonic saline
  - Na^+ bicarbonate administration
  - Hyperaldosteronism
  - Hyperadrenocorticism

continued on page 191

Hypoglycaemia

Hypoglycaemia is most commonly associated with sepsis, iatrogenic overdose of insulin or spontaneous remission of diabetes during insulin treatment. Clinical signs associated with hypoglycaemia reflect the constant requirement of the central nervous system for glucose. Signs are often vague such as weakness and lethargy. However, more overt neurological signs may develop including ataxia, tremors, seizures and coma. The severity of signs associated with chronic hypoglycaemia is usually milder, due to adaptive mechanisms.

Differential diagnoses for common abnormalities on the emergency database

Hypomagnesaemia

- Inadequate nutritional intake, anorexia
- Administration of Mg^{2+}-depleted IV fluids or parenteral nutrition
- GI tract losses
  - Inflammatory bowel disease (IBD)
  - Lymphangiectasia
  - Cholestatic liver disease
- Renal losses
  - Glomerular disease
  - Tubular disease
- DKA
- Drug effect; eg, diuretics, amphotericin B, digitalis
- Lactation
- Shift from extra→ intracellular: glucose, insulin, amino acid administration
- Chelation
  - Increased catecholamines; eg, sepsis/shock, trauma
  - Large volume blood transfusion
- Sequestration in pancreatitis
- Primary hyperparathyroidism
- Hyperthyroidism

Hypocalcaemia

- Hypoparathyroidism; eg, post-bilateral thyroidectomy
- Hypoalbuminaemia (total hypocalcaemia; ionised calcium levels may be within reference interval)
- Acute pancreatitis
- Sepsis
- Alkalosis
- Ethylene glycol toxicity
- Iatrogenic; eg, excess phosphate administration in IV fluid therapy, bicarbonate administration, bisphosphonates
- Spurious; eg, sample contamination with EDTA
- Eclampsia
- Dietary deficiency, vitamin D, excess phosphate
- Chronic kidney disease
- Intestinal malabsorption, protein-losing enteropathy
- Hypomagnesaemia

Anaemia

- Regenerative – haemorrhage: External or internal; eg, trauma, bleeding disorder (rodenticide intoxication, disseminated intravascular coagulation, thrombocytopenia), neoplasia
- Regenerative – haemolysis:
  - Immune-mediated – primary autoimmune, secondary to neoplasia, infection (eg, *Haemoplasma* species), drugs, inflammatory process
  - Pyruvate kinase deficiency
  - Oxidative damage (Heinz body formation) – heavy metals, onion, garlic, paracetamol toxicity
- Non-regenerative:
  - Anaemia of chronic disease (usually mild–moderate; eg, PCV ≥14%)[1,2]
  - Chronic kidney disease
  - Bone marrow neoplasia
  - Myelophthisis
  - Feline leukaemia virus infection
  - Pure red cell aplasia
  - Aplastic anaemia

Hyperbilirubinaemia

- Prehepatic due to haemolysis
- Intrahepatic
  - Hepatic lipidosis
  - Cholangitis (neutrophilic, lymphocytic)
  - Primary or metastatic neoplasia
  - Toxicity (eg, diazepam)
  - Feline infectious peritonitis
  - Sepsis
- Posthepatic
  - Choleliths
  - Cholangitis
  - Biliary neoplasia
  - Pancreatitis
  - Pancreatic neoplasia
  - Biliary tract rupture
  - Obstruction of the duodenal papilla (inflammation, neoplasia, foreign body)

Hypoalbuminaemia

- Decreased synthesis; eg, malnutrition, malabsorption, hepatic failure, portosystemic shunt
- Renal loss – protein-losing nephropathy
  - Glomerulonephritis
  - Amyloidosis
- GI loss – protein-losing enteropathy (globulins may be reduced also)
  - IBD
  - Neoplasia (eg, lymphoma, mast cell tumour)
  - Lymphangiectasia
- Chronic effusion
- Negative acute phase inflammatory response
- Burns
- Haemorrhage and external blood loss

continued from page 190

Treatment

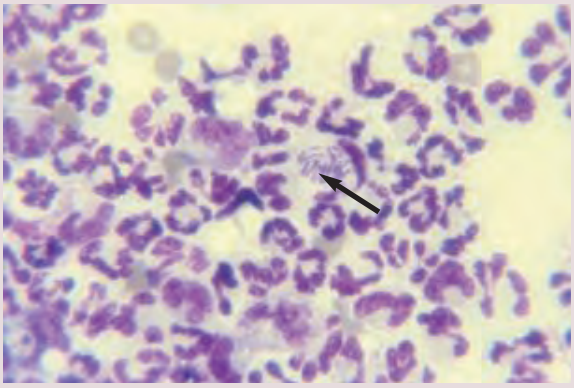
- Administer glucose as a bolus intravenously (IV) 0.5 g/kg (ie, give 2 ml/kg of a 25% dextrose solution slowly IV over 5–10 mins; make a 25% solution by diluting 50% dextrose in an equal volume of saline for administration).
- Follow with a 2.5% constant rate infusion (CRI) (25 ml of 50% dextrose in 475 ml of isotonic fluid); the rate of infusion can be adjusted to titrate the level of supplementation in response to serial assessment of blood glucose levels, aiming to maintain normoglycaemia.
- If the dextrose concentration required is >5% for CRI, then a central route of administration is advocated to avoid the development of thrombophlebitis.
- Glucagon infusions are occasionally used for refractory hypoglycaemia, particularly in patients that have received an overdose of insulin or have an insulin-secreting tumour.[7,8]

Is there sepsis?

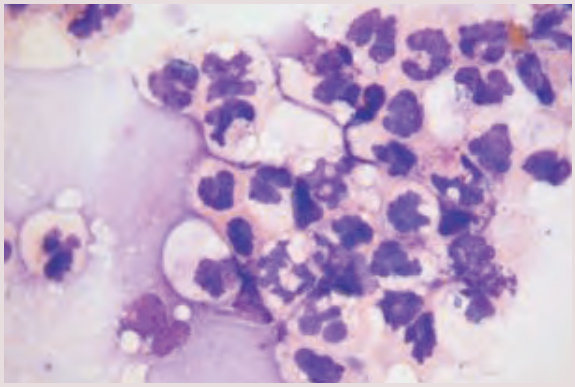
The clinical picture of sepsis is often more subtle in cats compared with dogs, and may lack the typical hyperdynamic features of tachycardia, fever and hyperaemic mucous membranes. Physical abnormalities characteristic of severe sepsis include lethargy, pallor, diffuse abdominal pain, tachypnoea, bradycardia, hypothermia and weak peripheral pulses.[3]

Clinicopathological abnormalities that may be seen on the emergency database include hypoglycaemia, hypoproteinaemia, hyperbilirubinaemia, hypocalcaemia, hyponatraemia, hypochloraemia, thrombocytopenia, anaemia and band neutrophilia;[3,4] unexpected hypoglycaemia should warrant careful examination of the patient for a focus of infection.

Effusions, even of small volume, should be sampled and analysed if sepsis is suspected. Features of septic effusions (exudates) include high protein levels (>30 g/l) and high cellularity, classically with a predominance of degenerate neutrophils ('ragged' nuclei, hypersegmented nuclei, vacuolated cytoplasm) and intracellular bacteria. However, pretreatment with antibiotics may mean intracellular bacteria are not seen and neutrophils appear non-degenerate (see images below). It is helpful to evaluate effusion fluid and serum glucose and lactate simultaneously and to compare values; high lactate and low glucose in the effusion fluid are highly suggestive of sepsis.[5,6]



Septic peritoneal effusion showing a predominance of non-degenerate neutrophils with intracellular rods (arrow). Modified Wright's stain x1000. *Courtesy of Dr K Papasouliotis, University of Bristol*



Septic peritoneal effusion showing degenerate neutrophils, vacuolated cytoplasm and hypersegmented swollen nuclei. Modified Wright's stain x1000. *Courtesy of Dr K Papasouliotis, University of Bristol*

Hyperglycaemia

Hyperglycaemia is most commonly associated with diabetes mellitus or diabetic ketoacidosis (DKA), or develops as a physiological 'stress' response; the last requires no specific treatment. Complications of diabetes include DKA and hyperglycaemic hyperosmolar syndrome (HHS) (where glucose is >30 mmol/l but there are no ketones). HHS can cause neurological signs (eg, circling, altered mentation and seizures) and a more gradual reduction of glucose is required to prevent cerebral oedema.[9] In all cases where insulin therapy is indicated, fluid resuscitation should be performed before administering insulin; crystalloid administration will reduce glucose levels to some degree by dilution and increasing the renal excretion of glucose.

Hyperkalaemia

Hyperkalaemia is a commonly encountered metabolic abnormality, most often associated with acute kidney injury, urethral obstruction or urinary tract trauma. The most significant clinical signs are due to effects on the cardiac conduction system, typically manifesting when potassium levels are >7–8 mmol/l. The following electrocardiographic changes occur sequentially with increasing levels of potassium:

- Peaked T waves;
- Reduced R wave amplitude, prolonged PR interval and reduced QT interval;
- Reduced P wave amplitude, followed by widening; P waves subsequently become absent ('atrial standstill');
- Widening of the QRS complex and bradycardia;
- Ventricular fibrillation, asystole and cardiac arrest.

It is important to note, however, that the effect of hyperkalaemia on the heart will be dependent on other factors, too, such as the presence of hypocalcaemia and acid–base abnormalities, meaning that there is no set 'cut-off' point at which certain changes will be seen on the electrocardiogram (ECG).[10]

Abnormalities in potassium, calcium, sodium and magnesium will interfere with function of the neuromuscular and cardiovascular systems by virtue of their effect on cell membrane potentials.

Treatment

- Ideally an ECG is recorded to obtain a baseline rhythm, which can be monitored with treatment. If electrocardiography is not available, the heart and pulse rate should be closely monitored for the development of bradycardia and arrhythmias. All drugs that increase potassium levels should be discontinued (eg, ACE inhibitors, spironolactone and NSAIDs).
- Begin IV fluid therapy using 0.9% NaCl or lactated Ringer's. If K^+ >6 mmol/l, administer an initial 5 ml/kg bolus over 10 mins; repeat according to response, monitoring urine output and for signs of volume overload. In most cases fluid therapy will significantly reduce potassium levels by addressing hypovolaemia, increasing renal perfusion for excretion of potassium and by dilution. Fluid therapy is the primary method of reducing hyperkalaemia.
- Address the underlying cause where possible; for example, establish urine outflow in the case of urethral obstruction. Sedation or anaesthesia should not be performed until potassium levels have reduced and the ECG shows a sinus rhythm again. Careful cystocentesis may be required initially for stabilisation of cats with urethral obstruction. Use of a 23 G needle attached to an extension set, three-way tap and syringe is less traumatic (Figure 1), and gentle handling of the bladder is important to minimise the risk of inducing a vasovagal event.

'Blocked cats': fluid choice and hyperkalaemia

There is no evidence that 0.9% sodium chloride alters the rate of reduction of serum potassium more quickly when compared with lactated Ringer's in cases of urethral obstruction. However, lactated Ringer's has the benefit of reversing metabolic acidosis, which is invariably present.[11]

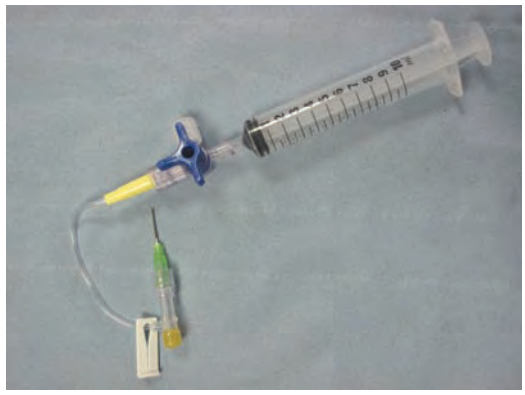


Figure 1 Cystocentesis may be necessary to stabilise a cat with a urethral obstruction before sedation or anaesthesia can be safely performed. Use of a needle attached to a T-port, three-way tap and syringe is less traumatic and enables drainage of larger volumes of urine compared with a needle attached to a syringe

Urinalysis

Assessment of urine provides useful baseline information and, ideally, the urine sample should be collected before fluid therapy is initiated. Urine specific gravity provides information about hydration status and renal function, and should be interpreted along with packed cell volume and total protein for assessment of hydration, and urea and creatinine for assessment of renal function. Assessment for proteinuria and quantification via the urine protein:creatinine ratio can provide useful information; if abnormal, consider investigating whether protein loss is pre-glomerular (haemoglobinaemia, myeloma), glomerular (and non-glomerular renal) or post-glomerular (infection/inflammation of the lower urinary or genital tract).

Ideally, urine should be sampled before fluid therapy is initiated.

The urine sediment should be examined under a microscope for crystals, cells and microorganisms. Identification of pyuria, haematuria, proteinuria and bacteriuria is consistent with a urinary tract infection and bacterial culture and sensitivity testing should be performed. Immunocompromised patients (eg, those receiving steroids or chemotherapy) may not develop an active sediment despite having a urinary tract infection. It is recommended that urine is submitted for bacterial culture if a source of sepsis/systemic inflammatory response syndrome is being searched for. Calcium monohydrate oxalate crystals may be present following ethylene glycol intoxication and have a 'picket fence' appearance (Figure 2).

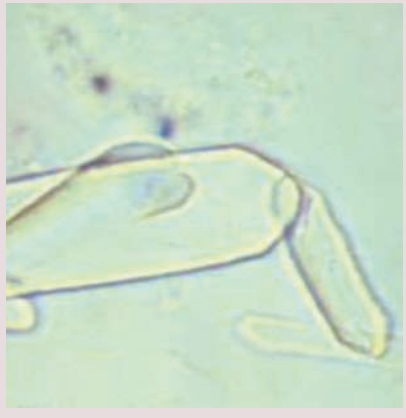


Figure 2 Calcium monohydrate oxalate crystals identified in a cat following ethylene glycol toxicity. Both the monohydrate and dihydrate form of the crystals may be seen; however, the former are considered more specific.[12] Crystals may not be identified if presentation following intoxication is delayed. *Courtesy of Dr K Papasouliotis, University of Bristol, Erasmus-Socrates Project*

Glucose ± insulin therapy for severe hyperkalaemia

First-choice treatment

Glucose can be administered alone as a 0.5 g/kg bolus (ie, give 2 ml/kg of a 25% dextrose solution slowly IV over 5–10 mins; make a 25% solution by diluting 50% dextrose in an equal volume of saline for administration). Follow with a 2.5% CRI (25 ml of 50% dextrose in 475 ml isotonic fluid) until potassium levels reduce. This approach relies on the effect of endogenous insulin secretion following glucose administration.

Second-choice treatment

Regular (soluble) insulin (0.25–0.5 IU/kg) can be given with glucose. Initially, administer a bolus of 1–2 g glucose per unit of insulin (the 25% dextrose solution described above contains 0.25 g glucose/ml). Follow with glucose supplemented in lactated Ringer's or saline as a 2.5% or 5% CRI, as required, to maintain glucose levels >3.5 mmol/l. Glucose must be given with insulin to prevent hypoglycaemia. The onset of action is usually 30 mins. Glucose levels need to be monitored for 12–24 h following insulin administration.

- If severe hyperkalaemia (K^+ >7–8 mmol/l) and/or ECG abnormalities persist, administer calcium gluconate: 50–100 mg/kg (0.5–1 ml/kg of a 10% solution) IV slowly over 10–20 mins; the rate of administration should be reduced if the heart rate slows. Calcium gluconate is cardioprotective by increasing the myocardial membrane threshold potential; it does not reduce potassium levels and is therefore an adjunct treatment only. Effects last for around 30–60 mins.
- If severe hyperkalaemia and/or ECG abnormalities persist despite aggressive fluid therapy and calcium gluconate, glucose ± insulin can be given to drive potassium intracellularly (see box).
- Rarely sodium bicarbonate is used when there are persistent severe ECG abnormalities despite aggressive fluid therapy, and there is a significant metabolic acidosis (eg, pH <7.1). Administer 1–3 mmol/kg IV over 20–30 mins (1 mmol/ml in an 8.4% sodium bicarbonate solution), ensuring that a dose of 4 mmol/kg is not exceeded. The effects may last several hours. Sodium bicarbonate also causes potassium to move intracellularly; this treatment should only be given when acid–base and calcium levels can be measured. Sodium bicarbonate is contraindicated if there is hypocalcaemia or alkalosis. There is an associated risk of causing paradoxical cerebral acidosis (if given too fast) and hypocalcaemia.

Hypokalaemia

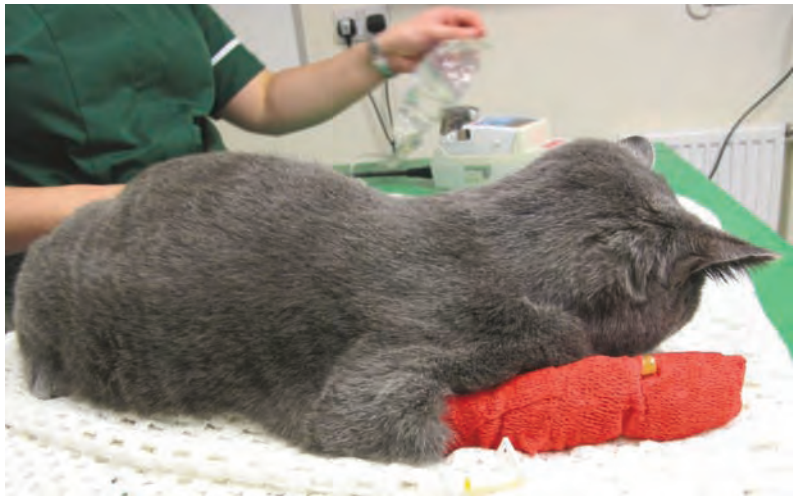
The most overt sign of hypokalaemia is muscular weakness. The effects on skeletal muscles may manifest as cervical ventroflexion (Figure 3) and a stilted gait; smooth muscle effects produce gastric atony and ileus.[13] In severe hypokalaemia, respiratory muscle function may be affected. Hypokalaemia predisposes to cardiac arrhythmias and may also impair renal tubular function (causing polyuria and polydipsia). Rapid correction of hypokalaemia is rarely required, however; potassium chloride can be supplemented in replacement IV fluids following fluid resuscitation, ensuring that the rate of delivery does not exceed 0.5 mEq/kg/h.

Hypokalaemia that is refractory to supplementation may be due to hypomagnesaemia.[14]

Hypomagnesaemia

Hypomagnesaemia can be clinically silent and is often only recognised in cats that have hypokalaemia that is refractory to treatment.[14] If clinical signs are seen, they tend to comprise cardiac arrhythmias or non-specific neuromuscular signs. Patients may also be hypocalcaemic.[15] Usually total serum magnesium concentration is measured; however, as the majority of magnesium is intracellular this result may not reflect total body levels. A low total magnesium in a patient at risk of deficiency warrants supplementation. Ionised magnesium is believed to more accurately reflect the active component, but this measurement is not readily available.

Figure 3 Cervical ventroflexion in a severely hypokalaemic collapsed cat



Mild deficiency may resolve with treatment of the underlying disorder. Supplementation should be considered if total magnesium is <0.6 mmol/l, if clinical signs are apparent or if there is concurrent hypokalaemia, hypocalcaemia or hyponatraemia. Magnesium is renally excreted and thus the dose should be halved if the patient is azotaemic. If the ECG is abnormal before magnesium supplementation then the ECG should be carefully monitored during supplementation.

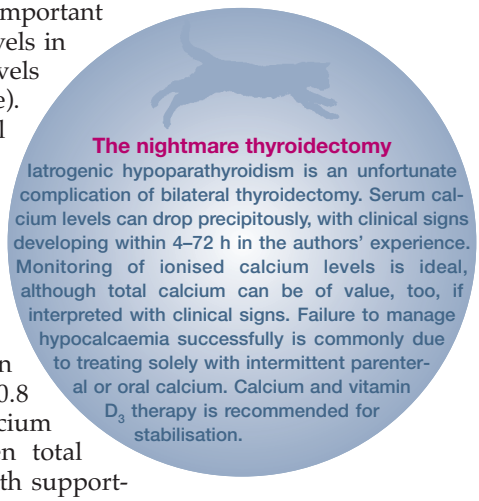
Magnesium sulphate can be administered at 0.5–1 mEq/kg/day by CRI in normal saline or dextrose in water. The dose can be reduced to 0.3–0.5 mEq/kg/day over the following days. Calcium should be monitored during treatment, since chelation of calcium with sulphate may occur; magnesium chloride should be used if hypocalcaemia is also present.

Side-effects of treatment include hypotension and development of atrioventricular or bundle branch blocks; these are more common with bolus administration than with an infusion.[15] Oversupplementation can be avoided by monitoring serum magnesium and making dose adjustments in patients with renal impairment.

Hypocalcaemia

Common causes of hypocalcaemia are iatrogenic hypoparathyroidism post-thyroidectomy, sepsis and ethylene glycol intoxication. The pathogenesis in sepsis is poorly understood.[16] Clinical signs include muscle weakness, fasciculations or tremors, stiffness or tetanic seizures, tachypnoea and pyrexia.[17] Often the first signs may be a change in behaviour with agitation, vocalisation and facial irritation (frequent pawing at the face). Cardiovascular signs include tachyarrhythmia, prolongation of the QT interval and hypotension.

Biochemistry analysers usually measure the total calcium level, which is the sum of ionised, protein- and anion-bound calcium; the most important fraction is the ionised form, which is considered to be the physiologically active component. Many hand-held analysers now measure ionised calcium (eg, i-STAT machine, www.woodley.co.uk). It is important to consider total calcium levels in respect of serum protein levels (and acid–base if available). Hypoalbuminaemia will reduce the total calcium level; however, ionised levels may be maintained within the reference interval.



The nightmare thyroidectomy

Iatrogenic hypoparathyroidism is an unfortunate complication of bilateral thyroidectomy. Serum calcium levels can drop precipitously, with clinical signs developing within 4–72 h in the authors' experience. Monitoring of ionised calcium levels is ideal, although total calcium can be of value, too, if interpreted with clinical signs. Failure to manage hypocalcaemia successfully is commonly due to treating solely with intermittent parenteral or oral calcium. Calcium and vitamin D_3 therapy is recommended for stabilisation.

Treatment

Treatment is indicated when ionised calcium is <0.8 mmol/l; or, if ionised calcium cannot be measured, when total calcium is <1.5 mmol/l, with supporting clinical signs.

- Administer calcium gluconate: 50–100 mg/kg (0.5–1 ml/kg of a 10% solution) IV slowly over 10–20 mins while monitoring the heart rate and rhythm on an ECG or by auscultation; the rate of administration should be reduced if the heart rate slows. Effects are usually seen within 30 mins.
- A common reason for failing to stabilise hypocalcaemia is administering calcium as bolus doses only (IV or subcutaneously). If the underlying condition cannot be quickly resolved, follow with a CRI of elemental calcium at a rate of 60–90 mg/kg/day (eg, 100 mg/ml [10%] calcium gluconate solution contains 9 mg/ml elemental calcium; 100 mg/ml [10%] calcium chloride solution contains 27.3 mg/ml elemental calcium). Remember that calcium salts should not be supplemented into fluids containing lactate, acetate, bicarbonate or phosphate due to the potential for precipitation of calcium (Table 1). Calcium gluconate is preferred, as calcium chloride is more irritant to the vein. The rate of administration should be adjusted to maintain normocalcaemia.
- Spontaneous recovery of calcium homeostasis may take days to weeks in iatrogenic hypoparathyroidism post-thyroidectomy.[18] Therefore, oral vitamin D_3 (calcitriol) supplementation should be given

Treatment is indicated when ionised calcium is <0.8 mmol/l; or when total calcium is <1.5 mmol/l, with supporting clinical signs.

Table 1 Supplement compatibilities for commonly used intravenous fluids

Supplement	Compatible crystalloid fluids	Incompatible fluids
Potassium chloride	0.45% NaCl, 0.9% NaCl, lactated Ringer's solution (LRS), Ringer's	
Potassium phosphate	0.45% NaCl, 0.9% NaCl	LRS, Ringer's
Calcium gluconate, chloride	0.45% NaCl, 0.9% NaCl, Ringer's	LRS
Glucose (dextrose)	0.45% NaCl, 0.9% NaCl, LRS, Ringer's	
Magnesium sulphate	0.45% NaCl, 0.9% NaCl, 5% dextrose	LRS, Ringer's

alongside calcium. Start calcitriol at 0.03–0.06 µg/kg for 3–4 days, then titrate to effect. Calcitriol is available in the UK as Rocaltrol (Roche) capsules 0.25 and 0.5 µg; for small doses the liquid may need to be aspirated from the capsules for dosing. An alternative preparation is alfacalcidiol, which requires hepatic conversion to vitamin D_3 (One-Alpha; Leo Laboratories liquid 2 µg/ml). The onset of action of vitamin D_3 is 1–4 days, during which time calcium supplementation can be transitioned to an oral form (eg, calcium carbonate 0.5–1 g/day in divided doses), and then gradually tapered off, aiming to maintain calcium levels at the lower end of the reference interval using vitamin D_3 alone. Other forms of synthetic vitamin D (ergocalciferol and dihydrotachysterol) have a more prolonged onset of action and duration, which increases the risk of excessive supplementation and toxicity.

Hyponatraemia

Before hyponatraemia can be confirmed, pseudohyponatraemia must first be excluded. This can be caused by hyperlipidaemia, hyperproteinaemia or raised serum viscosity, which may lead to a spurious sodium result by plasma sample dilution. Hyperglycaemia and mannitol also effectively dilute or reduce sodium levels by causing fluid expansion of the patient's circulating volume.

Clinical signs depend on the rate of development of hyponatraemia and are primarily referable to the central nervous and neuromuscular systems. Cerebral oedema develops in acute hyponatraemia due to rapid influx of water into neurons. Signs include lethargy, weakness, incoordination, seizures and coma. There may be few clinical signs if hyponatraemia has developed more slowly (eg, >48 h).

Treatment

- Careful monitoring is required when correcting hyponatraemia (or hypernatraemia). Aim to change the plasma sodium concentration at a rate of ≤0.5 mmol/l/h; more rapid correction may cause cerebral dehydration, haemorrhage and demyelination.
- For initial volume expansion a fluid that has a similar sodium concentration to the patient (± 6 mmol/l) should be chosen.
- For replacement and maintenance fluid requirements continue with isotonic crystalloids (0.9% NaCl or lactated Ringer's), monitoring sodium levels every 1–2 h initially, and adjusting the fluid type to ensure sodium is increasing at ≤0.5 mmol/l/h.

No treatment is indicated in pseudohyponatraemia. Hyponatraemia in the presence of hyperglycaemia does not require specific treatment either, with therapy targeted to management of hyperglycaemia.

Nutrition and the acutely ill cat

Assessment of a cat's nutritional status should be performed once emergency complications have been stabilised. Evaluate recent food intake and the degree of recent weight loss, and look for clinical signs of malnutrition (eg, muscle atrophy, dull coat condition, poorly healing wounds, and peripheral oedema due to severe hypoalbuminaemia).

Cats are obligate carnivores with a constant high protein requirement.[19] They are unable to down-regulate protein requirements, even in the face of illness and anorexia, resulting in catabolism of endogenous proteins (muscles) and thereafter hypoproteinaemia. Hepatic lipidosis is a further potential complication of anorexia in both obese and normal/underweight cats.[20,21]

Attending to nutritional requirements should be considered as important as fluid therapy in critically ill cats.

Nutritional support may include the use of antiemetic therapy, assisted/tube feeding and appetite stimulants. The type of tube chosen will be influenced by the disease, anticipated duration of assisted feeding and the cat's suitability for general anaesthesia:

- **Naso-oesophageal tubes** are the simplest to place but are only appropriate for short-term feeding (5–7 days) and are not suitable if the cat has nasopharyngeal or oesophageal disease, is obtunded or is vomiting.
- **Oesophagostomy tubes** are often a suitable option for short- to medium-term feeding and can be placed under a very short anaesthetic.
- **Gastrostomy tubes and jejunostomy tubes** can be considered if the oesophagus cannot be used.
- **Parenteral nutrition** may be an option for patients unable to tolerate tube feeding or unsuitable for general anaesthesia.

The cat should be fed to meet its resting energy requirement (RER), building up to the full ration over 48–72 h:[22]

$$RER\ (kcal/day) = 70 \times Bodyweight\ (kg)^{0.75}$$

If the patient is not gaining or maintaining weight, the caloric intake can be increased by 25%. Overfeeding can result in gastrointestinal disturbances and metabolic complications ('refeeding syndrome'), and so should be avoided.[23]

Attending to nutritional requirements should be considered as important as fluid therapy in critically ill cats.

Hypernatraemia

Clinical signs of hypernatraemia are similar to those associated with hyponatraemia, with the rate of plasma sodium change likewise being significant. Hypernatraemia results in a shift of water from the cerebral neurons to the intravascular space, which may lead to vessel rupture, cerebral and subarachnoid haemorrhage and irreversible neurological damage.

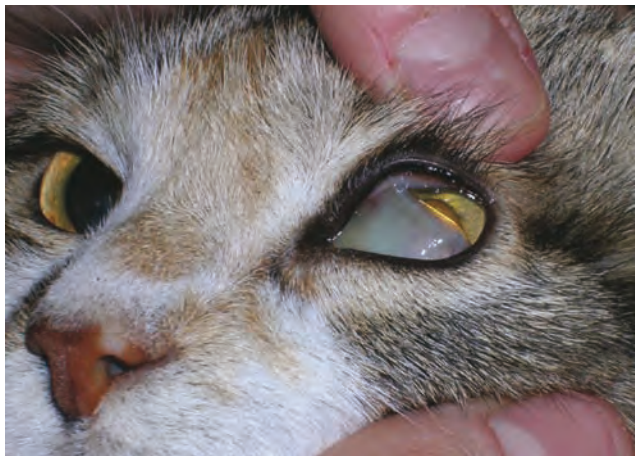


Figure 4 Mildly icteric third eyelid in a cat diagnosed with neutrophilic cholangitis

Treatment

- Use 0.9% NaCl for initial volume expansion, to prevent an excessively rapid drop in sodium levels.
- Provide maintenance fluids using 0.45% NaCl or 5% dextrose to correct the patient's free water deficit over 2–3 days. Reassess sodium levels every 1–2 h initially, adjusting the fluid type to ensure sodium levels drop at ≤0.5 mmol/l/h.

Hyperbilirubinaemia

Hyperbilirubinaemia will be grossly evident on physical examination once bilirubin levels are >40 µmol/l. Often the easiest sites to detect this initially are the conjunctiva, palatine mucous membrane and inside of the pinna (Figure 4). Hyperbilirubinaemia does not require specific treatment per se, aside from searching for and managing the underlying cause. However, it is thought that high levels of bilirubin may be toxic to renal tubular cells, and fluid therapy will increase renal excretion.

Hyperbilirubinaemia is common in critically ill cats and could be a secondary complication – for example, due to sepsis-induced cholestasis or hepatic lipidosis.

What else?

The list of database abnormalities covered in this article is not complete; rather, the focus has been on important abnormalities in the critical patient. Additional laboratory abnormalities that might need consideration include other haematological and biochemical aberrations; for example, thrombocytopenia, azotaemia, abnormal liver enzymes, coagulation abnormalities and analysis of body cavity effusions. However, discussion of these is beyond the scope of this article. Blood–gas analysis can also be very useful in assessing and monitoring the emergency patient but is not widely available in practice and likewise is beyond the current scope of discussion.

Anaemia

The rate of development and degree of anaemia will dictate whether a patient requires immediate oxygen-carrying support in the form of a blood transfusion or haemoglobin-based oxygen carrier (if available). Cats with chronic anaemia often appear to be coping with very low packed cell counts (often between 7 and 10%). In acute haemorrhage or haemolysis, however, there is less time for adjustment to the reduced oxygen-carrying capacity and a more rapid requirement for transfusion. While the transfusion is collected, supplemental oxygen and crystalloid fluids can be given to support perfusion.

Hypoproteinaemia

Hypoproteinaemia, and in particular hypoalbuminaemia, is an important finding on an emergency database and can guide further investigation and treatment. Hypoalbuminaemia can be a result of increased protein loss through the gastrointestinal or renal tracts, through exudation from the skin or external blood loss; or a result of decreased production due to hepatic insufficiency, malnutrition, maldigestion or malabsorption or through sequestration into body cavity effusions. Hypoalbuminaemia may also occur in inflammatory disease due to a negative acute phase protein response; hypoalbuminaemia is more commonly seen in association with sepsis than non-infectious SIRS.[4]

History and physical examination may give some indication as to likely cause(s) of the hypoalbuminaemia and this must be coupled with assessment of haematology and biochemistry (including bile acids to evaluate hepatic function) and urinalysis (including urine protein:creatinine ratio to quantify proteinuria). Globulins can also be decreased in patients with intestinal disease. Changes in plasma proteins due to malnutrition are usually mild. If malnutrition is suspected as a cause of hypoproteinaemia then careful attention to support of the patient's nutrition should be given (see box on page 196).

KEY POINTS

- **Effective management of the critically ill cat requires clinicians to institute good monitoring and serial reassessment of the patient, to treat specific problems as discussed in this two-part article, and to be aware that sepsis and SIRS may be the reason the patient is not responding appropriately to the treatment given.**
- **Critical patients are, by their nature, unpredictable and the best way to be successful is to be watchful, considerate and questioning, to ensure excellent teamwork and communication, and to constantly strive for improved patient monitoring.**

Case notes

Venus, a 2-year-old male castrated Russian Snow cat, presented with a 48 h history of dysuria, dull demeanour and progressive abdominal distension.

Venus in acute collapse. An IV catheter has been placed, oxygen is being provided by mask and he is laid on a soft bed with a second bed covering him to maintain normothermia

Presentation Venus was quiet but responsive; he lay in lateral recumbency and was reluctant to ambulate. Initial survey examination revealed pale mucous membranes, a capillary refill time (CRT) of 2 s, and a heart and pulse rate of 240 bpm. Metatarsal pulses were difficult to palpate. The mucous membranes were tacky. He had a fluid thrill and pain on abdominal palpation. Rectal temperature was 37.9°C. The respiratory pattern was normal and thoracic auscultation was unremarkable.

Initial problem list

- Dysuria
- Shock – there are several signs of abnormal perfusion: mucous membrane pallor, extended CRT, altered quality of peripheral pulses and tachycardia. At this point, hypovolaemic, obstructive and distributive shock were possible causes; cardiogenic shock was considered less likely based on thoracic auscultation, and hypovolaemic shock was considered most likely
- Abdominal pain
- Abdominal fluid thrill – ascites
- Dehydration – based on the tacky mucous membranes.

Given all the signs, an acute abdominal disorder was most likely

Steps taken to start stabilising Venus An IV catheter was placed and fluid resuscitation begun using crystalloid fluid. A 10 ml/kg bolus of lactated Ringer's was administered over 10 mins. Blood was taken for an emergency database and 0.1 mg/kg of methadone was given by slow IV injection. Systolic blood pressure revealed hypotension (85 mmHg, RI 120–150 mmHg).

Repeat auscultation of the thorax was performed following fluid administration to check for signs of unmasking of occult cardiac disease (eg, heart murmur, arrhythmia, gallop sound) and was normal.

Interpretation of emergency database This shows a mild hyponatraemia, mild hyperkalaemia, marked azotaemia and moderate hyperglycaemia. The azotaemia could be pre-renal, renal or post-renal. However, in view of the history of dysuria, abdominal pain and distension, post-renal or lower urinary tract disease was considered highly likely. The degree of hyperkalaemia and hyponatraemia did not warrant any specific change in stabilisation, beyond further fluid therapy. Lactated Ringer's was continued due to the presence of a compensated acidosis (mixed disturbance); two further 5 ml/kg boluses were administered.

Venus's emergency database results

Parameter	Result	Reference interval (RI)
Sodium	144 mmol/l	147–162
Potassium	4.5 mmol/l	2.9–4.2
Ionised calcium	1.26 mmol/l	1.2–1.32
Glucose	10.1 mmol/l	3.5–5.5
Urea	43.8 mmol/l	6.5–10.5
PCV	27%	25–35
Total solids	81 g/l	Total protein: 77–91
pH	7.32	7.25–7.4
PCO_2	37.6	28–34
Base excess	–6	–5–+2
Bicarbonate	19.6	16–20

Response After 30 mins, Venus was significantly more responsive and comfortable, with a heart rate of 200 bpm and palpable metatarsal pulses. (The prior tachycardia may have been a combination of shock and pain since it is not unusual for shocked cats to have a normal heart rate or bradycardia.) Systolic blood pressure was 100 mmHg. Oxygen saturation (without supplementation) was 98–99%; thus, further supplemental oxygen was not considered necessary. Having reached appropriate endpoints for fluid resuscitation, intravenous fluids were continued at 6 ml/kg/h.

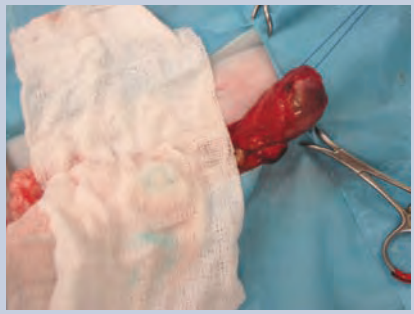
Further assessment Detailed clinical examination revealed reduced skin elasticity, consistent with dehydration of approximately 6–8%, in addition to hypovolaemia. An empty bladder was palpable. Abdominocentesis revealed a pale yellow translucent fluid.

Abdominal fluid analysis revealed:

- Creatinine 2058 µmol/l (serum 505)
- Total protein 9.1 g/l (serum 81)
- Potassium 7.8 mmol/l (serum 4.5)
- Glucose 12.1 mmol/l (serum 10.1)
- Smear examination – moderate numbers of erythrocytes and non-degenerate neutrophils; no evidence of intracellular bacteria. Low numbers of platelets and macrophages

Interpretation of abdominal fluid analysis High levels of creatinine and potassium compared with serum levels confirmed a diagnosis of uroabdomen.[24]

Further investigation Abdominal ultrasound revealed a thickened, irregular bladder wall, with apposition of the mesentery to the cranial pole and a moderate volume of ascitic fluid. Following further stabilisation, an exploratory coeliotomy revealed rupture of the cranial pole of the bladder. Although there was no known history, trauma was considered the most likely cause of the bladder rupture.



Intraoperative appearance of the bladder, showing rupture of the cranial bladder pole, marked erythema and contusion of the bladder wall. *Courtesy of Dr A Gines, University of Bristol*

WHAT THIS CASE DEMONSTRATES

A logical approach to the acutely collapsed cat is important. Venus responded rapidly to stabilisation with fluids, oxygen and analgesia. Despite the severity of the urinary tract trauma, the laboratory changes identified in the emergency database were surprisingly mild. Extensive investigations were not required in order to understand the cause of Venus's collapse and successfully stabilise him.

Acknowledgements

The discussion on hyperkalaemia in this review has been adapted from the 'BSAVA manual of feline practice: a foundation manual' (in press).

Funding

The authors received no specific grant from any funding agency in the public, commercial or not-for-profit sectors for the preparation of this article.

Conflict of interest

The authors do not have any potential conflicts of interest to declare.

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