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Welcome to your February edition of Feline Focus. In this issue we cover a range of areas starting with a fascinating article on tick paralysis from Rachel Korman who works in Australia. Nicola Ackerman tells us about life stage nutrition which complements her webinar on the subject this month. (Remember, if you miss a webinar you can download it later.) Traumatic injuries are all too common in cats, and Rachel Perry and Ann Stanford describe a case with serious jaw injuries. Ann also authors an article on nursing a cat with an oesophagostomy tube. For our Keeping Cats Safe topic, we look at paracetamol (acetaminophen) toxicity, something well-meaning owners may cause by inadvertently administering a tablet. Make sure your clients are aware of the dangers and remind them to contact you for advice rather than giving any human medication. Finally, we want to say congratulations to Sarah Dawson, who is the winner of our Cat Friendly Clinic competition. Sarah told us about a cat which benefited greatly from Cat Friendly Clinic principles and no longer needs to be sedated for nail clipping (see page 64). Sarah will receive either £250 off the cost of our ISFM distance education courses (see http://icatcare.org/learn/nurses/distance-education-course) or a print of her choice from our 2016 calendar. If you have any cat friendly tips or tricks that you think help cats in your clinic do take pictures and email them to distance-education@icatcare.org and we can publish them in Feline Focus.

Best wishes,

Sam Taylor, Veterinary Editor

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PROVEN RESULTS IN 9 OUT OF 10 CATS*

*Source: Mills 2001. Evaluation of a novel method of delivering a synthetic analogue of feline facial pheromone (Feliway®) to control urine spraying by cats. FLW046-004 UK.
Tick paralysis in cats

Tick paralysis is caused by injection of a neurotoxin by *Ixodes holocyclus* ticks. Paralysis ticks are found along the eastern coastline of Australia (mainly southern and central Queensland, NSW). The disease is also reported in America, caused by a different tick species. Cats with tick paralysis deteriorate extremely rapidly and early treatment is imperative for successful recovery. Toxin can be neutralised by the administration of tick antiserum. Respiratory failure is a common cause of death in cats with tick paralysis so interventions such as intubation and mechanical ventilation may be required. Cats with tick paralysis require intensive nursing to address and prevent secondary complications including gastric reflux, oesophagitis, oral ulceration and lower urinary tract complications.

Tick paralysis occurs in cats injected with a neurotoxin following a bite from an *Ixodes holocyclus* tick, otherwise known as the paralysis tick, dog tick or bush tick (Figure 1). Paralysis ticks are found along the eastern coastline of Australia (mainly southern and central Queensland, NSW).

The paralysis tick requires three hosts to complete its life cycle. Common natural hosts include native fauna such as koalas, long-nosed bandicoots, echidnas, possums and kangaroos. These hosts are resistant to toxin effects due to continued exposure. Tick eggs hatch as larvae, then feed on host blood, moult to nymphs, feed again, then moult into adults. Female adults feed again prior to producing large numbers of eggs within leaf litter. These eggs hatch following periods of suitable warmth and high humidity.

**Key point**

Other species are resistant to the effects of toxins produced by *Ixodes holocyclus* but cats (and dogs) can be severely affected with ascending paralysis and death if untreated.

Rachel Korman graduated as a veterinary surgeon from the University of Queensland, Australia, in 2000. She has worked in small animal practice in Australia and the UK. Rachel was an International Cat Care Resident at the University of Bristol, UK, and has specific interests in liver disease, geriatric medicine and feline haematological disease. Rachel currently works at Veterinary Specialist Services in Brisbane, Australia.
Cows, sheep and horses can be infested with paralysis ticks; however, generally only dogs and cats develop severe paralysis following envenomation. All life stages (eg, larva, nymph, adult) are toxic; however, adult females cause most envenomations. The tick injects saliva containing various toxins (holocyclotoxins) during feeding. Toxins act on the neuromuscular junction, inhibiting acetylcholine release, resulting in lower motor neuron (LMN) paralysis, typically affecting the hindlimbs first (ascending paralysis). Laryngeal dysfunction and megaesophagus later develop, predisposing to regurgitation and aspiration of pooled saliva or gastric contents. Toxin effects on potassium channels may also reduce myocardial contractility. Death occurs mostly from respiratory failure due to respiratory muscle fatigue or secondary pneumonia and sepsis.

**Clinical signs**

Cats with tick paralysis deteriorate extremely rapidly and early treatment is imperative for successful recovery. Patients exhibiting clinical signs (ascending paralysis, dyspnoea/tachypnoea, changes in phonation, retching/regurgitation) require emergency treatment. Unfortunately, presenting signs can be vague (eg, vomiting, mild ataxia, tachypnoea) and may go unnoticed, particularly in outdoor cats.

At presentation, patients are graded according to paralysis severity (numerical score) and degree of respiratory compromise (letter score) (see Box 1).

Cats with severe respiratory distress are placed immediately into an oxygen cage with two oxygen lines (5-10 l/min each) to maintain a sufficient fraction of inspired oxygen (Figure 2). These cats have severe anxiety and further distress may result in cardiorespiratory arrest. Sedation (see later) is typically beneficial and patient handling (eg, for intravenous catheter placement) should be kept to an absolute minimum.

**Figure 2:** A cat with respiratory distress due to tick paralysis in an oxygen cage

**Box 1: Grading severity of paralysis and respiratory compromise**

<table>
<thead>
<tr>
<th>Gait score</th>
<th>Respiratory compromise score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Mild ataxia or paresis</td>
<td>A No clinical respiratory compromise</td>
</tr>
<tr>
<td>2 Able to stand unaided but cannot walk</td>
<td>B Mild compromise, respiratory rate &gt;30 breaths per minute</td>
</tr>
<tr>
<td>3 Unable to stand but can right themselves</td>
<td>C Moderate compromise, restrictive breathing with increased effort, gagging and retching</td>
</tr>
<tr>
<td>4 Unable to right themselves</td>
<td>D Severe compromise, dyspnoea, cyanosis, imminent death without intervention</td>
</tr>
</tbody>
</table>
minimum until the patient appears more stable.

**Box 2: Treatment goals**

Treatment goals for tick paralysis are to:
- inactivate the toxin via administration of tick antiserum (TAS);
- support respiratory function;
- address or prevent secondary complications (eg, bladder dysfunction, corneal ulceration, inadequate nutrition).

**Tick antiserum (TAS)**

Toxin is neutralised by administration of TAS (Figure 3). TAS is produced from the sera of hyperimmune dogs and contains antibodies that inactivate unbound holocyclotoxin. Anaphylaxis is more common in cats than dogs and can cause devastating effects; particularly in patients administered TAS previously. For this reason, cats are premedicated with prednisolone sodium succinate and adrenaline, prior to administration.

TAS is diluted with saline and administered via slow intravenous infusion (eg, over 30–60 minutes) to give rapid and reliable distribution throughout the body. Intraperitoneal administration is described; however, antitoxin delivery is unpredictable and administration cannot be stopped in the event of a reaction — thus this approach is not recommended.

Cats must be monitored closely (eg, behaviour, heart rate, respiratory rate, mucous membrane colour ± systolic blood pressure, electrocardiography and pulse oximetry) throughout TAS administration. Adverse reactions include the Bezold-Jarisch reflex typified by bradycardia, pallor and hypotension, secondary to severe reductions in cardiac filling. If this occurs, rapid administration of isotonic crystalloid fluids (eg, Hartmann’s, 5–10 ml/kg over 10 minutes), oxygen therapy and atropine (0.02–0.05 mg/kg IM or IV) is given. The infusion is stopped for a period and potentially restarted at a slower rate, depending on the severity of the paralysis.

Other adverse effects include urticaria or facial swelling, which increases suspicion for anaphylaxis. Patients receive chlorpheniramine (0.5 mg/kg IV), the TAS infusion rate is reduced and they are monitored closely for signs of developing systemic anaphylaxis including vocalisation, tachycardia, dyspnoea, vomiting and hypotension. If these develop, TAS administration is immediately suspended, oxygen provided and adrenaline

**Figure 3:** Tick antiserum is given as a slow infusion and cats should be monitored for anaphylaxis

**Key point**

Tick antiserum can result in anaphylaxis, which is more common in cats than dogs. Hence, premedication with dexamethasone or prednisolone sodium succinate and adrenaline is advised.
State of the art

(0.1 mg/kg IV) and prednisolone sodium succinate (1–2 mg/kg IV) given. Rapid isotonic crystalloid fluid boluses are given intravenously, titrated to effect (ie, heart rate reduction, improvement in blood pressure). If hypotension persists, a constant rate infusion (CRI) of adrenaline may be required.

When a cat has a tick, there is likely to be another lurking somewhere! An important aspect of tick paralysis treatment is ensuring no further ticks are on the patient. Once the patient is stable and sedated, the cat is clipped and frequent tick searches are performed, paying attention to the face, under the chin, ears and around the tail (Figure 4). Tick baths are also performed.

Supporting respiratory function
Respiratory failure occurs in cats with tick paralysis secondarily to:
- increased oxygen consumption due to increased respiratory effort;
- thoracic respiratory muscle fatigue or paralysis, reducing ventilation;
- laryngeal dysfunction causing upper airway obstruction and increased respiratory effort; and
- pulmonary compromise secondary to aspiration or cardiac dysfunction.

Multiple therapeutic interventions are often required including sedation, oxygen therapy, endotracheal (ET) intubation and intermittent positive pressure ventilation (IPPV) using mechanical ventilation (MV).

Anxiety or stress can increase oxygen consumption by 50–100%, predisposing to respiratory muscle fatigue. All efforts should be made to reduce anxiety in cats presenting with tick paralysis. Handling, canine contact and noise around the cat should be kept to a minimum. Reducing anxiety with sedatives aids ventilation and can be lifesaving. Drug choices include butorphanol (0.2–0.6 mg/kg IV or SC, CRI 0.1 mg/kg/h) or acepromazine (0.01–0.03 mg/kg IV or SC). If profound sedation is required in patients with severe anxiety, a midazolam CRI (0.1–0.3 mg/kg/h) and a butorphanol CRI are used.

If cats are tachypnoeic or dyspnoeic despite sedation, oxygen therapy is indicated. Although oxygen therapy can be provided by flow-by

Key point
Avoiding stress is vital in cats with any cause of respiratory dysfunction, including those with tick paralysis, so contact with dogs and handling should be minimised and sedation may be required.
administration, Elizabethan collars with plastic wraps and intranasal oxygen catheters, these methods may distress patients and oxygen cages are preferred for cats.

If hypoxaemia or dyspnoea does not resolve, anaesthesia, ET intubation and tracheal oxygen therapy is administered to reduce oxygen consumption, protect the airway and provide efficient oxygen supplementation (50 ml/kg/min titrated to effect) (Figure 5).

Anaesthesia or heavy sedation is maintained via butorphanol, midazolam and alfaxalone CRIs.

Respiratory function is monitored in patients using a combination of respiratory rate and effort, thoracic auscultation, pulse oximetry (SPO₂), arterial or venous blood gas analysis and capnography. In patients with deteriorating respiratory function, IPPV is considered, provided either by hand or preferably using MV (Figure 6). Mechanical ventilation does the work of breathing for a patient, controlling the amount of oxygen inhaled, breathing rate and pressure within the airways.

An arterial oxygen partial pressure (PaO₂) of 60 mmHg is the minimum acceptable in cats on oxygen supplementation before intubation and/or ventilation is required (this equates to an SPO₂ of 90%). Hypercapnia (PaCO₂ >45 mmHg) is better tolerated than hypoxia (PaO₂ <80 mmHg). Ventilation is commenced when the arterial partial pressure of carbon dioxide (PaCO₂) is greater than 60 mmHg or, if only venous blood is available, when the partial venous pressure of carbon dioxide (PvCO₂) is over 65 mmHg. These are similar to the end-tidal CO₂ level on the capnograph.

Figure 5: Cats remaining hypoxic or with ventilation abnormalities after oxygen supplementation should be intubated. Note this cat has his eyes covered as his eyelids are taped shut to help prevent corneal ulceration.

Figure 6: Mechanical ventilation may be required, necessitating constant intensive care.

Tip
Respiratory failure is a common cause of death in cats with tick paralysis.
Interventions such as intubation and mechanical ventilation may be required if the PaO₂ falls and PaCO₂ rises despite oxygen supplementation.
Treatment goals for IPPV are to maintain adequate arterial blood gas values (\(P_{\text{aO}_2}\) 80–100 mmHg; \(P_{\text{aCO}_2}\) 35–50 mmHg) using the least aggressive ventilator machine settings possible to reduce the risk of lung injury. Patients are maintained under intravenous anaesthesia using butorphanol, midazolam and alfaxalone CRI s. A multimodal approach is best to reduce the adverse effects of any single agent.

**Box 3: Complications during mechanical ventilation**

Complications during mechanical ventilation may include:

- ventilator induced lung injury associated with inappropriate inflation pressures or tidal volumes resulting in over-distension of alveoli;
- ventilator associated pneumonia: patients are predisposed to pneumonia due to loss of normal airway protective mechanisms, patient immunosuppression, pulmonary atelectasis secondary to recumbency and gastric reflux;
- cardiovascular compromise secondary to increased intrathoracic pressure impairing venous return and cardiac output;
- pneumothorax.

Thoracic radiography is performed in cats with deterioration in respiratory status (not explained by respiratory muscle fatigue or paralysis), or with abnormal thoracic auscultation (eg, crackles) to identify signs of aspiration pneumonia (Figure 7) or other pulmonary pathology (eg, pneumothorax). If aspiration pneumonia is identified, broad-spectrum antibiotic combinations (eg, first generation cephalosporin/penicillin plus metronidazole) are provided, preferably based on culture and sensitivity results obtained by bronchoalveolar lavage. Additionally, intravenous fluid therapy and airway nebulisation help improve airway health. Sterile ET tubes are used, suctioned using sterile techniques every few hours and are replaced every 6–8 h.

Duration of MV is variable, but typically between 12–72 h. Patients without pulmonary pathology require shorter MV times. For successful weaning to spontaneous ventilation (ie, the patient is breathing for themselves without MV), the patient needs sufficient respiratory drive, adequate neuromuscular function and must maintain adequate gas exchange. Anaesthesia drugs are tapered to restore respiratory drive and the amount of assistance provided by the ventilator is reduced. Eventually, patients are disconnected from MV, but may remain intubated for longer. Attempts are made to wean from MV every 6–12 h. The cat is

**Figure 7:** A lateral thoracic radiograph of a cat with tick paralysis and aspiration pneumonia
monitored for signs of tachycardia, respiratory distress, hypoxaemia, hypoventilation and hypertension. If ongoing respiratory compromise is evident, MV is reinstated.

Mechanical ventilation is most successful in cats with poor respiratory function, but minimal lung pathology (eg, aspiration pneumonia). Patients with lung pathology are more difficult to stabilise and maintain on MV and survival rates are lower (50%). Unfortunately, the financial contribution required for MV for long periods often has a negative impact on outcome.

**Addressing or preventing secondary complications**

Cats with tick paralysis require intensive nursing to address and prevent secondary complications including gastric reflux, oesophagitis, oral ulceration, lower urinary tract complications (eg, bladder atony), corneal ulceration, muscle atrophy and pressure sores.

Tick paralysis causes a flaccid oesophagus (megaoesophagus) and loss of lower oesophageal sphincter tone. Antiemetics (eg, maropitant, metoclopramide CRI) and gastroprotective (eg, esomeprazole) and prokinetic agents (eg, raniditine) may reduce gastric acid reflux into the oesophagus and the subsequent vomiting and regurgitation that predisposes to aspiration pneumonia.

Unfortunately, these medications don't prevent oesophageal pooling of oropharyngeal secretions. Oesophageal and oral suctioning aids secretion removal but can cause discomfort and requires heavy sedation. Patient positioning in sternal recumbency helps prevent aspiration. Placement of a naso-oesophageal feeding tube is useful for regular decompression and the provision of nutrition (eg, trickle feeding) at a later stage. Nasogastric tube placement is avoided to prevent further gastric reflux.

Due to the megaoesophagus and altered gastrointestinal motility, patients receive nil per os for the first 24 h. Intravenous fluid therapy is required, with rates and fluid type tailored to maintain hydration and address ongoing losses. Careful monitoring of respiratory rate, effort, heart rate, blood pressure and urine output is imperative to avoid volume overload.

Regular bladder monitoring is indicated, with expression or placement of an indwelling urinary catheter to prevent detrusor muscle atony, overflow incontinence or urinary tract infections (UTI). Indwelling urinary catheters are treated in a sterile fashion and any exposed portion is cleaned regularly to prevent iatrogenic UTI. Urine output measurement helps monitor fluid balance. Additionally, barrier creams are applied to prevent urine and faecal scalding.

**Key point**

Secondary complications of tick paralysis include oesophagitis and megaoesophagus as well as corneal ulceration and bladder atony.

Patients with tick paralysis often cannot blink and narcotic use further potentiates corneal ulceration. This can be devastating, resulting in marked ocular pain and potential enucleation. Regular artificial tear administration is required and should be preservative-free as
preservatives have been shown to induce corneal ulceration in humans. In cats receiving MV, contact lenses may be placed and the eyelids taped closed. Corneal ulceration must be identified quickly and treated with topical antibiotics and potentially a temporary tarsorrhaphy.

Most tick paralysis patients are unable to position themselves, and intensive nursing care improves patient comfort and recovery. Soft, dry bedding and regular turning helps prevent pressure sores. Gentle physiotherapy (eg, passive range of motion, massage) reduces muscle atrophy. Moistening of the oral mucous membranes is performed regularly and pulse oximetry probes are covered with moistened swabs and the site changed frequently to prevent oral ulceration. Padding is also provided around ties to anchor endotracheal tubes (Figure 8).

Recovery from tick paralysis
Recovery from paralysis can take anywhere from 24 h to 5–7 days. Patients with complications such as aspiration pneumonia or oesophagitis have prolonged recovery periods. Once regurgitation has stopped and a gag reflex has returned, patients are offered water, then food. Cats may be discharged when they are ambulatory and not ataxic, when they can eat and drink without gagging and are able to urinate voluntarily.

Conclusions
Tick paralysis can have devastating effects..Ticks are indiscriminate, affecting young and old, and sick and healthy cats alike. Treatment must be instigated early and aggressively with intensive nursing care to improve survival. If cats are treated early and avoid secondary complications then complete recovery is possible.

Further reading
Life stage nutrition in felines

Cats’ nutritional needs change as they age, and as a result life stage diets/feeding can be helpful. During gestation and lactation a queen has increasing nutritional demands and should have ad libitum access to a growth diet. Kittens learn to eat solids from 5 weeks of age by tasting the queen’s food. During the adult life stage cats may gain weight and should have body and muscle condition scores monitored. Diets designed for neutered cats contain fewer calories to prevent post-neutering weight gain. Senior cats may not tolerate dietary deficiencies as well as younger cats and, unless they have chronic kidney disease, have a higher requirement for easy digestible protein.

Life stage diets in felines are as important as in other species because nutritional requirements differ throughout life. This article will discuss these nutritional differences and how use of life stage diets can benefit all cats.

Gestation and lactation
Prior to breeding the queen needs to be in good condition and should have:
• a good nutritional status;
• an up-to-date vaccination status;
• received prophylactic treatment for endo- and ectoparasites.

A good body condition score (BCS) and muscle condition score (MCS) is required (Figure 1).1 Queens that are overweight could possibly present with dystocia during parturition. Obesity can also affect fertility, as demonstrated in humans.

The feeding of queens during pregnancy differs to that of bitches. Queens increase food intake soon after conception, and this increases with the duration of gestation. During the first third of pregnancy the queen will lay down fat reserves, which will be used towards the end of gestation and during lactation.2

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VTS(Nutrition) A1 V1 C-SQP HonMBVNA

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Key point
A queen has increased nutritional demands during pregnancy and lactation and should have ad libitum access to a growth diet.
Lactation places huge nutritional demands on the queen. The nutritional demands can increase from 3–6 times that of maintenance. This is dependent on the size of the litter. A growth diet needs to be fed free choice (ad libitum). Large volumes of water will be required and must be available at all times. Peak lactation demands will occur when the kittens are 3–6 weeks of age, and further nutritional support of the queen will be required. Colostrum is vitally important to all mammals; it contains many antibodies from the queen, aiding the early immunological status of the kittens. Queens will sustain a loss of body weight during lactation. Ad libitum feeding during pregnancy will allow for the increase in body tissue that will be metabolised during lactation and to meet foetal energy demands. The weaning body weight of the queen should not be less than the body weight at conception.

When nursing, kittens should be vigorous and active. For the first 4 weeks feeding exclusively from the mother is possible if she is healthy and well nourished. Expected weight gain of kittens should be 10–15 g/day (or at least 10% gain per day). Kittens which are not receiving sufficient milk will cry, become restless or extremely inactive and fail to achieve the expected weight gain. It should be remembered that littermates do differ, and gain weight at different rates.

**Weaning**

Solids can be introduced to the kittens from 5 weeks of age. A mixture of the mother’s diet combined with warm water is ideal. At this age the kittens will show an interest and taste the food, but will not be consuming enough to meet nutritional requirements. Observing the mother eating the diet is important, and will encourage the kittens to start lapping. When eating their first solid food, kittens innately do not choose the most palatable.
Back to basics

food. They will choose what their mother eats, even if her food is unusual. From 6–8 weeks of age complete weaning can be achieved. In the majority of cases the queen will wish to spend less time nursing, and may start to get annoyed or frustrated with her kittens when they try to suck.

On weaning, kittens need to be fed a good quality complete growth/kitten diet. The majority of breeders that use complete growth diets will rehome with the recommendation that the animal remains on the same diet during this transitional period. There is no reason to change the diet unless the animal isn’t gaining weight adequately, if the diet isn’t nutritionally balanced or if the animal is grossly overweight.

Overfeeding in all kittens should be avoided, as it can lead to obesity in later life. Due to the limited capacity

Figure 2: Kittens should be weighed regularly to assess their growth (photograph courtesy of Richard Murgatroyd Photography)

<table>
<thead>
<tr>
<th>Key to Body Condition Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>IDEAL WEIGHT</td>
</tr>
</tbody>
</table>

Figure 3: Feline growth charts (courtesy of Hill’s Pet Nutrition)
of the stomach, which restricts the quantity of food able to be consumed in each meal, growth diets are highly energy dense. Slight underfeeding, which does not induce a reduction in full growth potential, can prevent the development of obesity and later health problems, but must be monitored closely.

Assessment of weight gain needs to be performed on a daily basis for the first few weeks of life. Once weaned and rehomed (normally at 8-10 weeks or often later for pedigree kittens) the kitten should be weighed at least fortnightly, until 3 months old, and then monthly until 6 months old (Figure 2). This conveniently fits in with the required worming regime. Use of growth charts can be invaluable, and will enable trends in weight gain to be monitored (Figure 3). Too slow a weight gain can reflect insufficient calories being consumed, or that an inadequate quality of protein is being fed.

Docosahexaenoic acid (DHA) has been shown to improve cognitive ability and visual acuity, and has been introduced into premium commercial kitten diets.

**Adult maintenance**

The adult phase is defined from when maturity has been reached until physiological changes occur due to the ageing process. The age at which the adult phase starts depends mainly on breed variations. Smaller breeds can reach full maturity from 6 months, larger and giant breeds from 1 year to 18 months (eg, Maine Coons and Norwegian Forest Cats). Each individual animal should have its diet altered to meet its own needs. The quantity of diet fed will depend on the quality of the diet, the amount of exercise the animal receives, neutering status and metabolism. This period is a time when weight gain can occur so cats should have their weight, BCS and MCS monitored regularly to avoid the development of obesity. Changes in metabolism post-neutering should be discussed with owners, and the use of ‘light’ diets or diets specifically aimed at neutered cats may be advocated. These diets are designed to prevent weight gain, not aid in weight loss.

**Neutered adult diets**

Many diets have now been introduced to the market specifically designed for neutered cats. As previously discussed, the animal’s metabolism decreases post-neutering. In entire cats, energy expenditure in both female and male animals is $57 \pm 2$ kcal/kg. Once neutered, this value decreases to $50 \pm 3$ kcal/kg in males, and $51 \pm 2$ kcal/kg in females. There are, however, marked differences in other factors as demonstrated in Table 1. Changes in insulin resistance can suggest predisposition of neutered cats to diabetes mellitus.

**Senior diets**

The senior or geriatric phase of life starts at varying ages due to breed size, though cats are normally deemed as senior at 8-10 years of age. Other factors such as nutritional status, environment, genetic makeup and clinical health will affect these
Changes that occur with age include less obvious ones such as alteration in the physiology of the digestive tract, immune system, kidneys and other organs. Generally, the capacity to absorb and utilise nutrients is not decreased in older animals, but the body becomes less able to tolerate excesses and borderline deficiencies, and the ability to respond to dietary changes may also be decreased. ‘Geriatric’ screening should be considered in all animals once reaching a senior age. A critical part of this screening should include evaluation of nutrition and body condition.

Nutritional changes in senior cat diets are aimed at supporting the physiological changes that occur within this life stage. Previously it was presumed that the energy requirements of senior animals were reduced, due to a decrease in activity levels and expenditure. In cats, however, the maintenance energy requirements do not decrease with age.\(^4\) This could be because cats tend to sleep a lot throughout their lives, balancing bursts of energy with rest, a pattern maintained in many older cats’ lifestyles. Certainly the proportion of obese cats tends to increase until

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**Table 1: Differences between male and female cats, and the consequences of neutering (from Dethioux et al)\(^5\)**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Male</th>
<th>Female</th>
<th>Consequence of neutering</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body fat (as % of total body weight)</td>
<td>23.8 ± 1</td>
<td>30.1 ± 1.7</td>
<td>Increase: male: 32.9 ± 1.7, female: 35.5 ± 1.8</td>
</tr>
<tr>
<td>Energy expenditure (kcal/kg)</td>
<td>57 ± 2</td>
<td>57 ± 2</td>
<td>Decrease: male: 50 ± 3, female: 51 ± 2</td>
</tr>
<tr>
<td>Caloric requirements</td>
<td></td>
<td></td>
<td>Requirements reduced in both sexes</td>
</tr>
<tr>
<td>Serum leptin (hormone made by adipose cells and involved in satiety)</td>
<td>Regulation of leptin secretion by testosterone</td>
<td>No demonstrated oestrogenic control in cats</td>
<td>Male: increase in the male&lt;br&gt;Female: less noticeable change</td>
</tr>
<tr>
<td>Insulin resistance</td>
<td>May be present</td>
<td></td>
<td>Male: continuance of insulin resistance&lt;br&gt;Female: appearance of insulin resistance</td>
</tr>
</tbody>
</table>

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**Key point**

Older cats may not tolerate deficient diets as well as they did when they were young. This age group requires more frequent assessment of body condition score and body weight to assess for disease and nutritional adequacy of their diet.
the age of 7. After this it decreases, especially from the age of 10 years. Chronic kidney disease and a reduction in renal function should be considered in all senior animals, with screening urinalysis recommended for cats over the age of 10 years. A reduction in protein quantities within the diet could be beneficial if renal damage has occurred. However, the quality (biological value) of the protein should be increased, as older cats are prone to skeletal muscle loss due to the inability to downgrade protein metabolism.

Reduced protein digestibility is also experienced in geriatric cats. In healthy adult cats, protein digestibility is typically 85-90%. In geriatric cats this digestibility can be reduced to less than 77%. Diets which have a severely restricted protein level or proteins of a low quality/biological value can predispose cats over 12 years to negative nitrogen balance, and loss of lean body mass. Therefore, protein restriction prior to a diagnosis of chronic kidney disease may not be beneficial. The amount of protein required to support protein turnover actually increases in older cats. Feline senior diets designed for cats with normal kidney function have restricted protein and should thus be avoided. The restriction of phosphate in the diet is indicated in the management of chronic kidney disease; a decrease in kidney function can also lead to an increased loss of the water-soluble vitamins, due to the kidneys, decreased ability to concentrate the urine. This can also lead to a reduction in hydration levels of the animal. Senior animals have a reduced sensitivity to thirst, and thus there is a greater risk of dehydration in this age group. Older cats should be encouraged to drink by altering the provision of water. (See How to increase water intake in cats. Feline Focus 2015; 1[8]: 287-290.)

Most senior diets are formulated to have softer kibbles in order to accommodate any dental problems such as a reduction in the number of teeth or a drop in musculature of the jaw. Moving to a moist diet can benefit the animal if they are having difficulties in mastication. A moist diet will also aid hydration levels.

As the animal ages, smell is the first sense to decline. As the animal’s sense of smell deteriorates the animal may eat less. The aroma of

Figure 4: Senior clinics run by nurses and technicians can allow regular weighing of senior and geriatric cats (picture courtesy of Richard Murgatroyd Photography)
the diet is particularly important in diets aimed at senior animals in order to encourage consumption.

The use of antioxidants for senior animals has been advocated as free radical production can increase with age and illness (cardiovascular disease, arthritis). Geriatric animals, especially cats, have a reduced ability to digest fats. This may lead to fat-soluble vitamin deficiencies, again highlighting the importance of feeding a high quality, balanced diet with adequate digestible fat.

Conclusions
Nutritional requirements change as a cat ages and nurses and technicians should advise owners on the appropriate diets to feed at each life stage (Figure 4). Cats should also be monitored for complications of inadequate or excessive nutrition; for example, with regular assessment of BCS and MCS at each life stage.

References
Online how-to videos
what all cats would want their owners to watch

International Cat Care has produced 16 short videos to demonstrate correct cat care and help owners with various cat care issues.

General
- Apply a spot-on product
- Apply ear drops and clean your cat’s ears
- Apply eye drops or ointment
- Brush your cat’s teeth
- Clip your cat’s claws
- Fit a collar for your cat
- Give subcutaneous fluids to your cat

Diabetes
- Home blood glucose testing for your cat
- Collect your cat’s urine
- Give your cat a subcutaneous injection
- Test your cat’s urine for substances like glucose and ketones

Giving a cat a tablet
- Two people giving a tablet
- Using a pill popper
- Hiding a tablet in a treat
- Crushing a tablet and mixing with water
- Crushing a tablet and mixing with wet food

Available at www.youtube.com/icatcare

International Cat Care is the parent charity of the International Society of Feline Medicine (ISFM). Find out more about International Cat Care at www.icatcare.org.
Oesophagostomy feeding tubes: a nurse’s perspective on placement and care

Oesophagostomy feeding tubes are used to provide assisted nutrition that is required for more than 1 week. These tubes allow the feeding of thicker diets. Placement is simple and complications unusual. Short anaesthesia is required and monitoring may be hindered by the placement of the tube. The stoma site should be aseptically managed and the dressing changed at least daily with the site monitored for the development of cellulitis. Cats should be fed a third of their resting energy requirement (RER) on day 1, and the full RER by day 3 to avoid re-feeding syndrome. Food should be given slowly, at room temperature and the cat monitored for nausea or discomfort.

Oesophagostomy feeding tubes are a popular choice when considering assisted feeding in feline patients. They are straightforward to insert and only simple equipment is required for their placement, making them inexpensive. Complications are rare and, in addition, they are well tolerated and a wide variety of foods can be administered. They are indicated when a cat is unlikely to consume adequate calories for more than a week (see Box 1). For shorter periods a naso-oesophageal (NO) feeding tube may be suitable, but the diameter of NO tubes is such that thicker diets will not easily pass down the tube, limiting the types of food fed. Another advantage of an oesophagostomy tube is that owners can be taught to use them and cats can therefore be discharged for ongoing care at home.

Key point

Oesophagostomy tubes are simple to place and complications are rare, making them good choices for inappetent cats or cats with orofacial traumatic injuries.

Ann Stanford
RVN BSc(Hons) Grad DipVN

After qualifying as a veterinary nurse in 2006 from the Royal Veterinary College, UK, Ann Stanford stayed at the university referral hospital for a period before deciding to locum. She then joined a first opinion and referral practice on the south coast. This is where she completed her graduate diploma in advanced veterinary nursing in 2014. She is now involved in advancing others’ knowledge by performing in-house CPD and guest speaking at the local college.

How to...

**Box 1: Indications for placement of an oesophagostomy feeding tube**

When cats are consuming less than the resting energy requirement for more than 3 days assisted feeding of some kind should be considered. For example:

- cats with maxillofacial injuries (see the case study on pages 65-72 of this issue);
- cats with pancreatitis;
- cats with hepatic disease such as hepatic lipidosis;
- cats with gastrointestinal disease (particularly useful post-surgery to ensure nutrition and medication and therefore promote recovery);
- cats with severe cat flu (Figure 1);
- to facilitate administration of longer term medications (eg, a cat with mycobacterial infection).

**Placement**

Brief anaesthesia is required for placement of an oesophagostomy tube. The nurse should take into account that the majority of the surgeon’s time will be spent at the head end of the patient, restricting the nurse’s ability to do vital and reflex checks on their patient. Monitoring equipment should be used such as pulse oximetry, blood pressure monitoring and, if available, capnography. An oesophageal stethoscope cannot be placed due to the access required to the area for the procedure.

Once anaesthetised with an appropriately sized and well placed endotracheal tube, the patient should be positioned in right lateral recumbency. An area level from the base of the pinna to the shoulder and from the dorsal and ventral aspects of the neck should be clipped, and prepared aseptically using a chlorhexidine-based skin scrub, for the surgical placement of the oesophagostomy tube (see Box 2 for equipment required) (Figure 2).

The length of oesophagostomy tube to be placed should be measured from the planned incision point to the seventh rib and marked on the tube with a pen or small piece of tape. The veterinary surgeon will use forceps via the mouth to enter the oesophagus and turn them laterally to tent the skin in the desired location. An incision is then made through the skin on to the forceps, thus using blunt dissection through the subcutaneous tissues to make an incision into the oesophagus itself. The forceps are then used to grasp the distal end of the tube and thread...
it back through the incision and pharynx to exit via the mouth (Figure 3). At this point the surgeon will detach the forceps and bend the tube back into the mouth and feed it into the oesophagus to the previously marked point. A thoracic radiograph should be taken to confirm correct placement of the tube in the distal oesophagus. To keep the tube secure, a roman sandal/Chinese fingertrap suture is placed (Figure 4). The tube site should be covered with a suitable dressing.

**Dressing**

Dressings are often placed by the attending nurse or technician. Gloves should be worn at all times when applying a dressing to prevent infection. Dress the site using the following steps:

- The site should be cleaned and dried before a dressing is applied.
- Sterile swabs are placed around the stoma site to absorb any discharge and to keep the site dry.
- A gentle layer of soft bandage material is then applied over the site and around the neck to keep the swabs in place.
- The last layer is conforming

**Box 2: Equipment**

Equipment required for placement of an oesophagostomy feeding tube:
- surgical glove;
- sterile drape;
- curved or right-angled forceps;
- oesophagostomy tube of choice;
- scalpel blade and handle;
- suture material;
- marker pen;
- needle holders;
- rat tooth forceps;
- Mayo scissors.

*Figures 2–4 are courtesy of Dr Sarah Caney, www.vetprofessionals.com*
How to...

bandage, which is used to prevent the site becoming soiled. Take care to lay the bandage out non-stretched before application as it could recoil and restrict the patient's airway.

An Elizabethan collar is not needed and would not prevent patient intervention due to the location of the tube. Oesophagostomy tubes are usually well tolerated. If a cat seems uncomfortable or bothered by the tube the attending veterinary surgeon should examine the cat as cellulitis or other problems may have developed.

Management
The stoma site should be checked, cleaned and dressed daily, as a known complication of oesophagostomy tubes is localised cellulitis. The dressing should also be changed if a foul smell is noted, the bandage slips or the patient is uncomfortable with the tension around the neck.

Key point
Cellulitis at the tube insertion point is possible, so manage the tube aseptically. Clean the stoma site and change the dressing at least daily.

Feeding
In cases of anorexia, feeding is introduced slowly as the stomach would have decreased in size and over expansion or re-feeding syndrome can be seen if full requirements are fed immediately. The general rule is if a patient has had a reduced food intake for greater than 3 days, the patient's full resting energy requirement (RER) (see Box 3 for calculations the

Box 3: Calculation of resting energy requirements (RER) in cats

Use current bodyweight in kg.

\[ \text{RER (kcal/day)} = \text{bodyweight}^{0.75} \times 70 \]

Or for cats over 2 kg:

\[ \text{RER} = (30 \times \text{bodyweight}) + 70 \]

author uses in practice) is reintroduced gradually over 3 days. This is undertaken by administering one-third on the first day and increasing by a third each day until the full amount is reached. It should be noted that RER calculations are a rough guide and patients should be weighed on a daily basis to check that the animal's needs are being met. We currently always body condition score (BCS) all patients within the hospital so that feeding requirements can be adjusted accordingly.

Feeds should be split over a 24-h period, but time should always be left for the patient to sleep. The author finds that six feeds given between 6 am and midnight are best tolerated.

Often, depending on the reasoning behind placing a feeding tube, the patients can still be offered food to eat orally as soon as they have recovered from anaesthesia. Patients can be encouraged to eat by physical interaction such as stroking, quiet talking and placing food on the patient's paws and, where tolerated, gently on the gums. Food should always be fresh and warmed, especially if the olfactory system is compromised, such as by obstructed nostrils.
Potential complications
A blockage within the tube is one of the most common complications seen. Best practice to help prevent this is to flush the tube regularly and ensure that the food being administered is well mixed or, if blended, that it is completely smooth before use.

A proven technique for unblocking tubes is using 10–15 ml of a carbonated drink. The bubbles help break down the blockage and, if allowed to sit in the tube for 10 mins, will usually move it with some flushing.

The food should always be administered at room temperature and cats should be fed slowly and monitored for discomfort, retching, hypersalivation or gagging. These signs may indicate overfeeding (Figure 5). If these signs are noted feeding should be stopped and the amount decreased and given at a reduced rate. Most animals will not tolerate over 10–20 ml/kg per feed.5

Pain and nausea should always be controlled prior to tube placement and uncontrolled vomiting would be a contraindication for this type of feeding.

Prior to feeding, the tube should always be flushed with water. To prevent blockages and to check the placement of the tube, it should also be flushed with water post-feeding. If any coughing is noticed on the administration of water, no food should be administered and placement should be checked with radiography.

Conclusions
The veterinary nurse’s role is vital in cases where assisted feeding is required. A good understanding of the management of the tube and the patient’s feeding regime will aid in the success of the assisted feeding and enhance the patient’s recovery.

References
EXTREMELY DANGEROUS TO CATS

Every year cats die because they have been poisoned by antifreeze – don’t let your cat be one of them. Here’s how to keep your cat safe.

What is antifreeze?
Ethylene glycol, otherwise known as antifreeze, is a chemical used to prevent freezing. It is used in car radiators, screen washes and de-icers, and in water features to prevent them freezing up.

Why is it so harmful to cats?
Unlike most chemicals, cats seem to be attracted to the taste of antifreeze. However drinking just a tiny amount will cause serious kidney damage, often so severe that the cat will die.

How can I protect my cat?
› Avoid using antifreeze-containing products
› NEVER use in outdoor water features
› If you do have antifreeze products, store safely away from animals (and children) – a single lick from a bottle can be fatal for a cat
› Clean up any spillages immediately and keep cats away from affected areas until dry
› IF YOU SUSPECT YOUR CAT HAS INGESTED ANTIFREEZE, SEEK IMMEDIATE VETERINARY ATTENTION

Download free practice posters at http://icatcare.org/advice/keeping-cats-safe

icatcare.org/keeping-cats-safe

INTERNATIONAL CAT CARE - KEEPING CATS SAFE CAMPAIGN
Paracetamol (acetaminophen) poisoning in cats

Cats are particularly sensitive to paracetamol (acetaminophen) toxicity because of their limited ability to metabolise the drug to non-toxic metabolites. Alternative metabolite pathways lead to the production of toxic metabolites which cause methaemoglobinemia, cyanosis, anaemia and jaundice. Even a single paracetamol tablet can cause severe toxicity in a cat. Treatment is therefore aimed at ensuring adequate oxygenation, and preventing further metabolism of paracetamol to toxic metabolites with the use of antidotes, particularly acetylcysteine, to prevent damage to the liver and red blood cells.

Paracetamol (also known as acetaminophen or APAP in some countries) is a very widely and readily available non-narcotic analgesic. It is not used therapeutically in cats.

Paracetamol is sold under many brand names and is available in many formulations including tablets, capsules, liquid suspensions and powder preparations (usually for mixing with water). Tablets are typically 500 mg (or 250 mg for children over 6 years of age) and suspensions 120 mg/5 ml or 250 mg/5 ml. Paracetamol is also commonly available in compound products with other drugs including codeine, caffeine, aspirin, ibuprofen and decongestants.

Exposure
Cats may be exposed after eating a paracetamol-containing product but poisoning can also occur when an owner, believing the cat to be unwell, misguidedly gives it part of or a whole tablet or doses the cat with a few millilitres of a paediatric paracetamol suspension.

Nicola Bates has worked in human and veterinary toxicology for 25 years and has been with the Veterinary Poisons Information Service (VPIS) since it started. As well as providing emergency advice via the telephone she has written extensively on veterinary toxicology. In addition to service provision, she is involved in training of VPIS staff and veterinary professionals. She is currently the congress abstract editor for the European Association of Poisons Centres and Clinical Toxicologists (EAPCCT).

The Veterinary Poisons Information Service (VPIS) is a 24-h telephone emergency service providing information on the management of actual and suspected poisoning in animals. It provides direct support to veterinary professionals worldwide.

See http://vpisglobal.com/ for more information and how to sign your practice up to receive this vital service.
**Mechanisms of toxicity**

**Cats are different**

Cats are very sensitive to paracetamol. In particular they may develop methaemoglobininaemia (high blood levels of methaemoglobin; an oxidised form of haemoglobin which cannot bind oxygen), haemolytic anaemia, Heinz body formation and hepatic necrosis.

In all species, paracetamol is metabolised in the liver by glucuronidation, sulphation and oxidation (Figure 1). The glucuronide and sulphate conjugates are non-toxic and are excreted in bile and urine. In most species, the oxidation pathway is minor, and glucuronidation is the major pathway of paracetamol metabolism. Cats, however, have a restricted ability to conjugate with glucuronic acid as they have low levels of glucuronyl transferase, the enzyme that catalyses the final step of the glucuronidation pathway.

The toxic metabolite

The oxidation pathway produces a highly reactive compound called N-acetyl-p-benzoquinone imine (NAPQI). Normally this compound is conjugated with glutathione, then further metabolised to non-toxic metabolites. At low dosing this is an effective and efficient detoxification pathway despite the fact that cats have low glutathione concentrations. At higher paracetamol doses, the toxic metabolite can accumulate and cause liver damage.

**Key point**

Cats have a limited ability to metabolise paracetamol to non-toxic metabolites due to their reduced capacity for glucuronidation in the liver.
glucuronidation and sulphation routes are saturated and the oxidation pathway increases in activity. This results in increased production of NAPQI, causing glutathione depletion in the liver. NAPQI then binds with cellular molecules and proteins causing cell death in the liver.

**Oxidising metabolites**
Alternative metabolic pathways also allow accumulation of oxidising metabolites that may induce methaemoglobin formation, Heinz body formation and denaturation of erythrocyte membranes. In the presence of glutathione, methaemoglobin will be reduced to haemoglobin. However, as glutathione becomes depleted insufficient quantities will be available for this reduction reaction. Methaemoglobin concentrations in blood rise (it is typically measured as a percentage of haemoglobin) and tissue hypoxia occurs. Heinz bodies are denatured chains of haemoglobin with oxidised sulphhydryl groups. They precipitate and migrate towards cell membranes where they render cells fragile. Haemolysis and restricted passage of erythrocytes through the microcirculation and spleen may result in anaemia.

**Tip**
Cats with suspected or confirmed paracetamol toxicity should be treated promptly. The antidote acetylcysteine can be obtained from local hospitals.

It has been suggested that the metabolite para-aminophenol, and not NAPQI, is responsible for the methaemoglobinaemia seen in cats and dogs with paracetamol poisoning. This is a minor metabolite of paracetamol that is removed as N-acetyl conjugates. Cats have only one of the enzymes responsible for this reaction and dogs have none. This means that both species are less efficient at removing this toxic metabolite which is known to undergo reactions with oxyhaemoglobin.

Humans, who frequently overdose with paracetamol and possess both enzymes, do not develop methaemoglobinaemia due to paracetamol toxicity.

**How the antidotes work**
**Acetylcysteine (previously N-acetylcysteine, NAC)**
The most widely used antidote in paracetamol poisoning is acetylcysteine because it can reduce the toxicity of the drug by three main mechanisms:
- Acetylcysteine is a precursor of glutathione and is metabolised to form a substrate for glutathione synthesis in red blood cells and the liver.
- Acetylcysteine acts directly on the reactive metabolite NAPQI to form an acetylcysteine conjugate which can be excreted (although this reaction is slow).
- Acetylcysteine is oxidised in the liver to form sulphate, thereby increasing the capacity of the sulphation pathway.

Administration of acetylcysteine has been shown to reduce the half-life of paracetamol by half in cats.2

**S-adenosyl-methionine (SAMe)**
S-adenosyl-methionine (SAMe) is also a precursor of glutathione, reducing methaemoglobin to haemoglobin. In mice, SAMe significantly reduced paracetamol 2(2) feline focus 59
keeping cats safe

toxicity and was more potent than acetylcysteine in reducing liver toxicity. Administration 1 h after paracetamol ingestion provides protection by reducing oxidation and Heinz body formation, but treatment after 4 h in cats may be of limited benefit.

**ascorbic acid**
Ascorbic acid can be given to reduce methaemoglobin to haemoglobin, although the reaction occurs slowly. There is some evidence to suggest it may also scavenge NAPQI before it binds to proteins, possibly reducing it back to paracetamol. Ascorbic acid is normally given in combination with the other antidotes.

**methionine**
Methionine is another glutathione precursor and has some use in paracetamol poisoning — it was widely used in cases of human poisoning. It may be given where acetylcysteine is unavailable or used in conjunction with acetylcysteine therapy, but is not widely available and only as an oral preparation.

**methylene blue**
(methylthioninium chloride)
In cases of severe methaemoglobinemia, methylene blue has been used in cats. This drug increases reduction of methaemoglobin back to haemoglobin. Although methylene blue can itself cause methaemoglobinemia and haemolytic anaemia, particularly in cats, it is safe to use at correct doses.

**cimetidine**
Cimetidine is a potent inhibitor of cytochrome P450 metabolism in the liver and theoretically administration could result in inhibition of the oxidation pathway, but its efficacy has not been evaluated for paracetamol poisoning cases in cats.

**toxic dose**
The toxic dose of paracetamol in cats is 50–100 mg/kg. In an experimental study cats given 90 mg/kg showed a rapid increase in methaemoglobin formation within 4 h of ingestion. One cat died at 24 h without treatment. A second cat that had been treated with SAME 1 h after dosing was found in distress at 36 h, failed to respond to supportive care and was euthanased. In another study a dose of 60 mg/kg in cats produced a methaemoglobinemia of 22% in 4 h whereas a dose of 120 mg/kg produced a concentration of 45%.

**key point**
It is important that owners are informed that any amount of paracetamol may be toxic. One tablet of paracetamol is enough to cause severe toxicity and death in a cat.
In essence, one tablet of paracetamol is likely to cause severe toxicity in a cat (Figure 2).

**Time course**
Paracetamol is rapidly absorbed from the gastrointestinal tract under normal conditions and clinical manifestations of paracetamol ingestion may occur within 4 h in cats, but definitely within 6–24 h. Recovery in treated cats usually occurs within 2 days, depending on the severity of signs, but biochemical abnormalities may take several weeks to resolve.

**Tip**
Clinical signs of paracetamol toxicity include cyanosis with tachypnoea and dyspnoea, facial and paw oedema, depression, vomiting and haematuria. If a cat presents with consistent clinical signs the owner should be questioned about exposure to paracetamol.

**Clinical signs**
**Early effects (typically 1–4 h)**
Progressive cyanosis is the most striking sign in cats with paracetamol toxicity and is associated with tachycardia, tachypnoea and dyspnoea. Mucous membranes appear brown in colour, and weakness and lethargy may be observed.

**At 4–24 h**
Facial and paw oedema may be observed in some cases. Depression, vomiting, anorexia and vocalisation may occur and dark brown blood may be noted indicating the presence of methaemoglobinaemia. Haematuria, anaemia, and evidence of haemolysis may be present. Less common effects include hyper- or hypothermia, ataxia and lethargy.

**Later effects (day 2–7)**
Although raised liver enzymes and bilirubin have been reported in cats, hepatic necrosis is not the principal cause of fatality in cats as they usually die as a result of severe methaemoglobinaemia. Haemoglobinuria, intravascular haemolysis, jaundice and other evidence of liver damage may be seen in animals that survive the initial stages of paracetamol poisoning. Coma, convulsions and pulmonary oedema are occasionally reported. Oliguria and renal damage can occur after high doses, although the exact dose is unknown in cats.

**Prognosis**
The prognosis of cats with paracetamol toxicity is good with prompt treatment but depends on the severity of methaemoglobinaemia. It also appears that time between ingestion and treatment may be as important, or even more important, than the dose ingested. Coma, convulsions and pulmonary oedema are poor prognostic signs.

**Diagnosis**
Diagnosis of paracetamol is based on clinical signs and history. It is important to ask the owners if they have given their cat any medication. Laboratory changes may also aid diagnosis, with changes in haematology, methaemoglobin concentration, liver enzymes and urinalysis noted (Box 1).

**Treatment**
**Decontamination**
The aim of treatment for a cat with paracetamol toxicity is to ensure adequate oxygenation and prevent
further metabolism of paracetamol to toxic metabolites with the use of antidotes and to prevent damage to the liver and red blood cells. Any cat with signs consistent with paracetamol toxicity should be treated irrespective of the time since ingestion or the dose ingested. If ingestion was recent an emetic and activated charcoal can be considered, depending on the clinical condition of the cat.

Antidotal therapy
Antidotal therapy should be started in any cat with signs of toxicity or that has ingested a potentially toxic dose (Table 1). Acetylcysteine is the antidote of choice and if not available in the practice can usually be obtained from your local hospital emergency department (since it is used for humans with paracetamol poisoning). It can be given by intravenous infusion or orally; however, it has a sulphurous smell and taste which can cause significant hypersalivation so it needs to be diluted to improve palatability or given by naso-oesophageal tube if clinically indicated. SAMe and ascorbic acid can also be given in combination with acetylcysteine.

Monitoring and supportive care
Other treatment is essentially supportive with monitoring for signs of hypoxia, methaemoglobinemia, liver damage, anaemia, haemolysis and renal impairment. Oxygen will be required in cats with cyanosis and, in cats with severe respiratory signs, oxyglobin (haemoglobin

<table>
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<th>Drug</th>
<th>Dosage</th>
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| Acetylcysteine | • **IV or oral:** 140 or 280 mg/kg diluted in 12–25 ml/kg of fluid IV over 6 h, followed immediately by 70 mg/kg IV (over 15–20 minutes) every 6 h for 36 h or more dependent on the clinical condition of the animal  
  • Continue treatment until cat is clinically well  
  • Can be diluted in dextrose or saline |
| SAMe         | • **oral:** 90 mg twice daily for 3 days, then 90 mg daily for 14 days |
| Ascorbic acid | • **SC:** 30–40 mg/kg every 6 h for at least 36 h                         |
glutamer) can be given. Whole blood transfusions may be required in cats with evidence of severe haemolysis, significant decrease in packed cell volume (PCV) or severe anaemia.

Conclusions
Increased metabolism of paracetamol via the oxidative pathway with unopposed production of toxic metabolites causes toxicity in cats following paracetamol administration. The characteristic picture is one of methaemoglobinemia, cyanosis, anaemia and jaundice. Death usually occurs from progressive methaemoglobinemia or, more rarely, from severe hepatic necrosis.

The aim of treatment for the cat with paracetamol toxicity is to ensure adequate oxygenation and prevent further metabolism of paracetamol to toxic metabolites with the use of antidotes, particularly acetylcysteine, and to prevent damage to the liver and red blood cells.

References
3 Terneus MV, Brown JM, Carpenter AB, et al. Comparison of S-adenosyl-L-methionine (SAMe) and N-acetylcysteine (NAC) protective effects on hepatic damage when administered after acetaminophen overdose. Toxicology 2008; 244; 25-34.

Want to become a Cat Friendly Clinic?

Then, register for Cat Friendly Clinic materials and access the accreditation criteria and process.

The Cat Friendly Clinic (CFC) programme is run by the ISFM in collaboration with several different partners across the world. It is designed to help create more cat friendly veterinary clinics, reducing stress for the cat as well as both the owner and veterinary staff treating the cat.

Find out more at:
www.catfriendlyclinic.org
Starting with a blank slate in a brand new veterinary practice I was asked to implement my ideas and the cat waiting area and consultation room were born at Walton Vale Vets4Pets, UK. The area has a cat tree for pet carriers, Feliway (CEVA) diffuser in use at all times, towels for owners to cover their pet carriers and up-to-date information for owners to read offering advice on how they can reduce stress for their cats on vet trips. The exclusive cat only consultation room was created opposite the cat waiting area, thus reducing the distance the animals would need to travel in the practice, with equipment and materials kept just for use on our feline patients. With warm surfaces, toys for distraction, a cat play area with raised surfaces for the more adventurous cat, and a gentle cat friendly approach, my colleagues and I began to notice a big difference even in the most nervous of cat patients.

We noticed that cats seemed more relaxed, owners reported a change in their cats’ behaviour compared with trips to other practices, and owners told us the information we provided was new to them and useful.

There was one cat in particular who caught my attention. I was pre-warned that a very nervous little puss would be visiting me to have her nails trimmed and the owner reported previous attempts had resulted in the cat exhibiting frustration and aggression. Eventually she had been sedated to have her nails trimmed. When this lovely girl arrived, I ensured she was not kept waiting too long and was quickly called in to her appointment to reduce the time spent in the waiting area. The owner’s worry was obvious, but with a gentle, confident approach and by using our high sided basket (see picture) with a cosy cushion for the cat to bury into, I managed to trim the nails without any fuss. Since then I have seen this cat on several occasions for nail trimming and each time there have been no issues and a calm relaxed happy owner, which is a great feeling.

This case showed me how a feline friendly approach can make a huge difference to individual cats as well as the practice as a whole.

If you have any cat friendly tips that you think help cats in your clinic, take pictures and email them to distance-education@icatcare.org so that we can share them in Feline Focus.
Case study

Nursing care of a feline patient with mandibular injuries

Feline patients may sustain maxillofacial and dentoalveolar injuries when falling from heights (so-called ‘high rise syndrome’) as in this case of a 13-month-old domestic shorthair cat who sustained a mandibular fracture and symphyseal separation. Anaesthesia, analgesia, fluid therapy and a plan for provision of nutrition were made pre-operatively following discussion between the attending veterinary surgeon and veterinary nurse. The cat was treated with interdental wiring and an intraoral bisacrylate composite splint, and an oesophagostomy feeding tube was placed.

Feline patients are often involved in traumatic accidents, whether road traffic accidents or falling from a height, and can sustain multiple maxillofacial and dentoalveolar injuries.1,2 These patients often require prolonged hospitalisation and nursing care while immediate stabilisation is performed, followed by definitive fracture repair. This case illustrates the role of the veterinary nurse in providing nutritional support and nursing care to a patient with a jaw fracture.

History
A 13-month-old female neutered domestic shorthair cat was referred to the authors’ practice for definitive fixation of a mandibular fracture sustained after falling from a second storey window 2 days previously. The mandibular symphysis had separated and there was an open fracture of the left mandibular body plus a fracture of the left mandibular third premolar (tooth 307). The patient was unable to close her mouth properly, and was not eating or drinking voluntarily (Figure 1). Intravenous fluid support had been given using a balanced isotonic solution, along with antibiotic cover.
Case study

(cefovecin at 8 mg/kg subcutaneously) and analgesia (meloxicam at an initial dose of 0.2 mg/kg intravenously and repeated after 24 h at 0.05 mg/kg per os and buprenorphine at 20 μg/kg intravenously q8h and last given 12 h previously). Nutritional support had been given by repeated nasogastric intubation, but was poorly tolerated.

The patient was admitted by the veterinary surgeon for general anaesthesia for definitive fracture repair and placement of an oesophagostomy feeding tube. The nurse in charge of the case performed a check of vital signs upon admission: bodyweight 3.6 kg (body condition score 4/9), heart rate 180 beats per minute with matched pulses, respiratory rate 50 breaths per minute with equal inspiratory and expiratory phases. Systolic blood pressure was 120 mmHg.

Nursing treatment

Anaesthetic plan

The veterinary surgeon and nurse in charge of the case discussed a suitable anaesthetic plan for the patient. This included the provision of an opioid for premedication (methadone at 0.2 mg/kg given intramuscularly using a fine gauge insulin needle and syringe into epaxial muscles). The cat was kept warm after pre-medication by the use of an electric heat pad placed underneath the cage bedding, and covering the patient with a blanket until induction. The use of a circulating warm-air blanket was planned for the procedure. A non-rebreathing circuit (Mini Lack) was chosen to deliver the volatile anaesthetic gas (isoflurane) mixed in oxygen.

Pre-anaesthetic checks of equipment were performed by the nurse. Namely:
- attachment of oxygen supply;
- circuit attached and leak checked;
- adjustable pressure limiting valve (pop off valve) open;
- isoflurane levels checked;
- scavenging attached;
- endotracheal tube leak tested.

Nursing priorities

- anaesthetic plan for fracture repair;
- nutritional support;
- fluid therapy;
- pain assessment and management;
- infection control;
- grooming.

Tip

The veterinary surgeon and nurse should work together to formulate an appropriate plan for their patients including provision of analgesia, anaesthesia, nutritional support and fluid therapy.

Figure 1: A 13-month-old female neutered domestic shorthair cat presented with a left mandibular fracture and symphyseal separation. The subsequent malocclusion can be seen with the left mandibular canine tooth (304) visible lateral to the upper lip.
A full induction tray was assembled for the patient including all equipment and drugs required for the induction of the patient and any further drugs that may be required during the procedure to prevent the nurse leaving the patient at any time.

Monitoring equipment was assembled and checked for the patient’s use, including the selection of the appropriate blood pressure cuff size for the patient. The patient was to be preoxygenated to prevent hypoxia at induction. A co-induction of midazolam (0.2 mg/kg IV) plus propofol to effect was to be given to induce anaesthesia. Monitoring of anaesthesia was to be performed by the use of a multi-parameter monitor and visual inspection.

The following parameters were measured continuously throughout the procedure and recorded every 5 minutes:
- oxygen saturation;
- heart rate;
- respiratory rate;
- body temperature;
- end-tidal CO2;
- non-invasive blood pressure.

Visual inspection can be limited in these cases because the veterinary surgeon’s attention is on the cat’s head. Therefore, jaw tone, eye position, mucous membrane colour and capillary refill time were also assessed and recorded every 15 minutes.

The eyes were to be lubricated every 30 minutes by use of a carbomer lubricant, to prevent the patient’s corneas drying due to lack of blink reflex. The patient was intubated after laryngeal desensitisation using a 4.0 mm internal diameter siliconised PVC endotracheal tube, which was inflated until the escape
Case study

Key point

During dental procedures, visualisation of the head can be limited and therefore additional anaesthetic monitoring not requiring access to that area (eg, capnography, blood pressure monitoring and pulse oximetry) can be useful.

of gases around the tube was just prevented. Care was taken when handling the patient’s head to prevent any further discomfort.

Evaluation and dental radiography under anaesthesia was performed by the veterinary surgeon. This confirmed the presence of a symphyseal separation and fracture of the mandibular body (Figures 2, 3 and 4).

In preparation for fracture fixation, and to provide postoperative analgesia, a left mandibular nerve block was performed using an

Figure 5: Intraoperative view: exposure of the mesial root of tooth 307 is clearly seen

Figure 6: Postoperative view: interdental wiring was used, plus an intraoral bisacrylate composite splint

Figure 7: Postoperative view of mandibular fracture repair using bisacrylate composite splinting and interdental wiring. Occlusion is checked by temporary extubation of the patient

Tip

Local anaesthesia provides very effective analgesia for oral surgery and dental procedures.
intraoral technique and bupivacaine local anaesthetic (0.25 mg/kg). The veterinary surgeon took swabs for bacterial culture and sensitivity, and the wound was then debrided and decontaminated prior to fracture fixation. Soft tissues were sutured using a fine gauge, absorbable suture material (1 metric poliglecaprone 25). An intraoral bisacylate composite splint was placed by the veterinary surgeon, using an interdental wiring technique to add fixation support (Figures 5 and 6). The patient’s occlusion was checked by temporary extubation (Figure 7). This was to ensure that the splint material did not obstruct mouth closure. Prior to extubation the patient’s depth of anaesthesia was checked to prevent the patient waking in this period.

The patient was re-intubated, and then positioned in right lateral recumbency for preparation for the oesophagostomy feeding tube placement. An area level with the base of the pinna to the shoulder and from the dorsal and ventral aspects of the neck was clipped and prepared aseptically using a chlorhexidine-based skin scrub for the surgical placement of the oesophagostomy tube. A thoracic radiograph was taken to confirm correct placement of the tube in the distal oesophagus.

Nutritional support
To allow provision of the daily nutritional requirements for the patient, at a site distant from the oral cavity, oesophagostomy tube placement was chosen. Oesophagostomy feeding tubes are well tolerated by cats, are inexpensive, and easy to place under general anaesthetic. Due to the lumen of the tube a variety of foods can be delivered and they are unlikely to block. Nasogastric tubes, however, are not always well tolerated (especially in cases of maxillofacial trauma), and can be prone to becoming blocked due to the small diameter of tube (oesophagostomy tubes are 8–16 French, compared with 3–5 for nasogastric tubes).

The oesophagostomy tube was placed in a routine manner. The patient care plan was constructed and a feeding plan was prepared, based on the patient’s resting energy requirements (RER). As the patient had reduced food intake for <3 days, it was planned to re-introduce the patient to full RER requirements over 3 days. On day 1, one-third of the RER would be given, two-thirds on day 2, and the full RER on day 3.

Pain assessment and management
The patient was placed in sternal recumbency with a slightly elevated head position for recovery and was closely monitored. A plan was then made to perform a pain score assessment q4h. Meloxicam was continued q24h (0.05 mg/kg given via the feeding tube). In addition, it was anticipated that the regional anaesthesia given intraoperatively would provide post-anaesthetic analgesia.

Key point
Oesophagostomy tubes are well tolerated by cats and the larger tube lumen, compared with a naso-oesophageal feeding tube, means they are less likely to become blocked. Owners can also be taught to use them at home.
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A pain score assessment was additionally written on the patient's care plan for the period of 8 h post delivery of these drugs to ensure the multimodal analgesic plan was adequate. Infection control

Results from culture and sensitivity testing showed a profuse growth of Pasteurella multocida which was sensitive to cefovecin. No further antibiotics were therefore given. Infection at the entry site of the oesophagostomy tube is, however, a concern. To prevent this, meticulous nursing care of the entry site is required. The cervical site was clipped and surgically prepared with a chlorhexidine-based skin scrub before tube placement. A dressing of sterile swabs was then placed around the site and covered with a soft, loose bandage with a waterproof vet wrap layer to prevent soiling. The dressing was changed and the stoma site checked twice daily. Gloves were worn at all times when handling the patient and cleaning and re-dressing the site.

Grooming

Grooming all feline patients that are hospitalised is an essential aspect of nursing care. This is critically important in cats with oral and/or dental injuries or diseases. This patient made a quick recovery and was able to be discharged the day after surgery. With in-patients who

Cat friendly practice priorities

- Having a separate cat ward provided this patient with a quiet environment in which to recover from surgery. Every effort is taken to ensure the cat ward remains a quiet environment, such that all staff communicate in quiet tones, and calming music plays on a radio at low level.
- Comfortable bedding was provided, along with access to fresh drinking water and a clean litter tray.
- The patient was clinically examined by the ward veterinary surgeon twice daily, and all parameters recorded on a hospitalisation sheet. In addition, nurses performed pain scoring four times per day.
- Grooming of the coat was carried out daily. Despite the patient having a feeding tube in place, it was still offered food as soon as recovery from anaesthesia was complete.
- The patient was encouraged to eat by physical interaction such as stroking, quiet talking and placing food on the patient's paws and gently on her gums if tolerated. Food offered to feline patients should always be fresh and warmed, especially if the sense of smell is reduced by nasal discharge or facial injuries.
are hospitalised for longer periods, grooming and cleaning should be incorporated into the patient’s care plan at least twice a day, more frequently for patients that are hypersalivating or have nasal discharge.

**Outcome**
The patient was discharged 24 h after surgery as she had started to eat voluntarily. The patient attended the referring veterinary practice 2 days later and was reported to be progressing well at home. The cat was eating adequately voluntarily and tube feeding was not required; therefore, the feeding tube was removed shortly afterwards. The cat returned after 6 weeks to have the intraoral splint removed under general anaesthesia, and further radiographs were obtained. Dental radiographs showed good fracture healing, so the splint was removed. The premolar involved in the fracture line was extracted at this time. Further dental radiographs were advised to monitor the canine tooth for ongoing vitality.

**Discussion**
Feline patients are often presented with maxillofacial and dentoalveolar trauma. One common cause of such trauma is falling from a height, so-called ‘high-rise syndrome’. The veterinary nurse will be required to play an essential role in the initial triage and stabilisation of the patient. In addition, if extended hospitalisation is required, the nurse can play a vital and integral role in the patient’s swift recovery and recuperation.

Commonly encountered injuries with ‘high-rise syndrome’ include:
- hard palate fracture;
- mandibular fracture;
- symphyseal separation;
- tongue injuries;
- other facial soft tissue injuries; and
- dentoalveolar trauma.

Definitive fracture repair is usually undertaken once the patient is effectively stabilised.

Once the patient’s oral cavity is returned to normal occlusion and the fracture stabilised, they will often start eating voluntarily. However, nutritional support should be provided until the patient is eating voluntarily and should aim to minimise loss of lean body mass. In addition, support should aim to reduce catecholamine release (inducing catabolism) by controlling hypotension, pain and infection.

Nutritional support may be given by enteral or parenteral methods. Enteral means are easy and safe, and more physiological for the patient. Enteral support is ideal if the patient has a functional gastrointestinal system but is physically unable to take on board the sufficient number of calories (‘if the gut works, use it’). The chosen route should take into account the patient’s injuries. Naso-oesophageal feeding tubes can be used for short-term support if the nasal cavity and pharynx are not injured and if no maxillary fractures are present, but rely upon a liquid diet being fed.
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Oesophagostomy tubes are easy to place, well tolerated and positioned distantly from any maxillofacial fracture. In addition, a wider range of foods may be administered due to the wider tube bore size. For the calculation of energy requirements see Box 3 on page 54 of this issue (Oesophagostomy feeding tubes: a nurse’s perspective on placement and care. Feline Focus 2016; 2(2); 51–56).

It is erroneous to assume that because a patient has a feeding tube in place it should not be offered food to eat orally. Cats with stabilised maxillofacial injuries should be offered food to eat, and encouraged to do so by diligent nursing. The tube may be removed when no longer required. The dressing is removed, the retaining suture cut and the tube gently pulled out. The stoma site should rapidly heal.

References