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Analgesia challenges in a canine burn victim

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## Contents

### Large

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Author(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Comparing lameness between group-housed sows on rubber-covered floors and group-housed sows on concrete floors</td>
<td>Kirstin Pinto</td>
</tr>
<tr>
<td>5</td>
<td>Sudden death caused by ‘Phalaris’ in some ewes</td>
<td>Jeremy Rogers</td>
</tr>
</tbody>
</table>

### Wildlife

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Author(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>42</td>
<td>Winks and Blinks</td>
<td>John Morris</td>
</tr>
</tbody>
</table>

### Small Animal

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Author(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>If you could only do one intervention/test—what would that be?</td>
<td>Aine Seavers</td>
</tr>
<tr>
<td>7</td>
<td>Hepatic amyloidosis in a young Siamese cat presenting with severe anaemia, clinical and therapeutic thoughts</td>
<td>Emmy Pointer, Michael Brown &amp; Nicholas Robinson</td>
</tr>
<tr>
<td>10</td>
<td>Delayed neurological signs secondary to carbon monoxide poisoning after smoke inhalation</td>
<td>Emma Billing</td>
</tr>
<tr>
<td>12</td>
<td>Diagnosis of carbon monoxide poisoning</td>
<td>Jennifer Brown</td>
</tr>
<tr>
<td>14</td>
<td>Ballistic intravesicular foreign body</td>
<td>Orla Fitzpatrick</td>
</tr>
<tr>
<td>15</td>
<td>Ocular ouch: Clerapliq</td>
<td>Aine Seavers</td>
</tr>
<tr>
<td>16</td>
<td>Yunnan Baiyao</td>
<td>Aine Seavers</td>
</tr>
<tr>
<td>18</td>
<td>Cow Pox in a cat</td>
<td>Maria Macarena Sanchez Martel</td>
</tr>
<tr>
<td>21</td>
<td>Why do dogs and cats react differently to heartworm?</td>
<td>Rick Atwell</td>
</tr>
<tr>
<td>23</td>
<td>Severe sunburn in three dogs presenting to veterinary clinics on the Atherton Tablelands of Far North Queensland</td>
<td>Thomas De Ridder, Justine Campbell, Tamara Olley, Tahleah Haddow, Graham Brown &amp; Paul Reddell</td>
</tr>
<tr>
<td>34</td>
<td>Oestrogen overdose as an cause of mammary gland hyperplasia in young Donskoy tomcat</td>
<td>Martina Naceradska &amp; Katerina Horackova</td>
</tr>
<tr>
<td>38</td>
<td>Acute renal injury in a cat called Asterix</td>
<td>Pete Coleshaw</td>
</tr>
<tr>
<td>39</td>
<td>Multi-modal analgesia challenges in a canine burn victim</td>
<td>Chris Sun</td>
</tr>
<tr>
<td>44</td>
<td>Feline ureteral transitional call carcinoma and concurrent alimentary small cell lymphoma—complications of treatment and management</td>
<td>Lan-Hsin Kuo</td>
</tr>
</tbody>
</table>

### Perspectives

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Author(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>51</td>
<td>No. 143 Ethical dilemmas in companion animal practice</td>
<td>Anne Fawcett</td>
</tr>
</tbody>
</table>

Disclaimer: Knowledge and best practice in the field are constantly changing. As new research and experience broaden our knowledge, changes in practice, treatment and drug therapy may become necessary or appropriate. Readers are advised to check the most current information provided (1) on procedures featured or (2) by the manufacturer of each product to be administered, to verify the recommended dose or formula, the method and duration of administration, and contraindications. It is the responsibility of the practitioner, relying on their own experience and knowledge of the patient, to make diagnoses, to determine dosages and the best treatment for each individual patient, and to take all appropriate safety precautions. To the fullest extent of the law, neither the Publisher nor the Editors/Authors assumes any liability for any injury and/or damage to persons or property arising out of or related to any use of the material contained in this publication.
Welcome to the mid-year edition of C&T, which is once again an expanded edition due to the amount of material we have received. As usual, this edition contains articles by seasoned C&T contributors, as well as several written by newcomers to this forum.

Recently, we conducted a survey to ascertain how well the CVE is serving the needs of its members as well as non-members in the veterinary profession. The results are very interesting. Thank you to all those people who took the time to complete the survey, your contribution will help us to make sure that what we do in the future remains relevant to all vets.

One of the things we wanted feedback on was what the attitude is to hard copy publications, as we continue to print and distribute C&T, Conference Proceedings from seminars and workshops, and the CPD Handbook. While all these publications could be available only as digital versions, the majority feedback still favours hard copy. Most universities are actively reducing the production of hard copy materials and many journals (including the Australian Veterinary Journal and Australian Veterinary Practitioner from July) are now only available online. So, the question is: why do we keep getting requests to continue to provide hard copies?

Over the last few years I have been carefully watching the participants at our various conferences and workshops and the overwhelming majority can be seen writing notes in the margins of the proceedings and in the blank pages at the end of each presentation. Whenever I visit a vet practice, the office or vet library invariably contains old copies of PGF or CVE proceedings, with well-thumbed pages and copious notes, so maybe old habits die hard? Educational psychologists often find that people engage more with their learning and have better retention when they commit pen to paper, so there is some science to back up this practice! Indeed, some early adopting schools that introduced tablets to replace a case full of textbooks have reverted to ‘old technology’, based on evidence-based data from their students’ assessments and feedback.

If you did not take the survey or would like to offer us feedback on these or other matters, we would welcome your input to add to the responses of over 1,000 vets.

This edition of C&T has a range of diverse articles and comments. As usual, we would value your feedback on any of the articles in C&T. It is easy for us at the CVE to believe that it occupies a useful place somewhere between the fully peer reviewed journals and veterinary blogs, but if you do not agree, please let us know. If C&T no longer holds any interest or relevance for you or your colleagues, we need to know; if you would prefer only an eBook version, let us know; if you have any other comments or observations, please tell us!

Please send your feedback to cve.enquiries@sydney.edu.au

The CVE Critical Care and Surgery conference to be held in Melbourne later in June is now booked out, due to overwhelming demand. Registrations are open for the Neurology conference in Cairns in September – this will also be a fantastic four-day program led by Steven de Decker from the RVC. Steven gave a very thought-provoking Podcast-Plus a couple of months ago, with a very active discussion forum after the event, so make sure that you do not miss this opportunity to elevate your understanding of neurology to a new and exciting level.

We also hope those of you in the bush get some rain, while the keen skiers get a good season on the slopes.

Hugh White
Director
## Calendar Key

- Orange: Conference
- Yellow: Seminar
- Green: Workshop
- Blue: TimeOnline
- Pink: PodcastPLUS
- Black: Theory & Practice

### Workshops

- **Anaesthesia**
  - Sat 2 November 2019
- **Advanced Anaesthesia**
  - Sat 3 November 2019
- **Hip & Stifle**
  - Sat 9 November 2019
- **Bone Plating**
  - Sun 10 November 2019
- **TOWNSVILLE**
  - Back to Basics: Diagnostic Ultrasound
    - Sat 10 – Sun 11 August 2019
- **QUEENSLAND**
  - **Soft Tissue Surgery I**
    - Sat 2 November 2019
  - **Soft Tissue Surgery II**
    - Sun 3 November 2019

### TimeOnline

- **Shock & Fluid Therapy**
  - 17 June - 14 July 2019
- **Goat Medicine & Husbandry**
  - 8 July - 4 August 2019
- **Canine Abdominal Ultrasound**
  - 15 July - 11 August 2019
- **Feline Behaviour**
  - 5 August - 2 September 2019
- **Heart Murmurs & Coughing in Dogs & Cats**
  - 5 August - 1 Sept 2019
- **Masterclass in Diabetes Management**
  - 12 August - 8 September 2019
- **Rabbits & Rodents**
  - 12 August - 8 September 2019
- **Clinical Pathology**
  - 19 August - 15 September 2019
- **Avian Surgery**
  - 26 August - 22 September 2019
- **Emerging Wildlife Diseases**
  - 2 - 29 September 2019
- **Feline Cardiology**
  - 9 September - 6 October 2019

### PodcastPLUS

- **Update on Drugs in Behaviour Medicine**
  - Thu 27 June 2019
- **Salmonella - Cast it Out!**
  - Thu 25 July 2019
- **Adrenal Disease in Cats**
  - Thu 29 August 2019

### Skin & Ear Cytology & Culture

- 16 September - 13 October 2019

### Pain Management

- 23 September - 20 October 2019

### Trauma Patient Management

- 30 September - 27 October 2019

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CVE Control & Therapy Series – Issue 295 June 2019
Comparing lameness between group-housed sows on rubber-covered floors and group-housed sows on concrete floors

Kirstin Pinto
4th Year DVM student. University of Sydney.

Abstract

Sow lameness poses adverse economic, welfare and health issues to the pig industry. Rubber mats may be a practical and cost-effective way to reduce lameness in group-housed gestating sows but the effectiveness of this strategy is unclear. A critical appraisal was conducted to determine whether sows group-housed on rubber-covered floors have reduced lameness compared to sows group-housed on concrete floors. A focussed literature search of CAB Abstracts, Medline and Web of Science resulted in 37 papers, of which five met the eligibility criteria for appraisal. These articles reported on trials which measured lameness parameters between sows group-housed in pens with concrete flooring and flooring covered with rubber mats. The results were conflicting; two articles reported significantly lower lameness in sows on rubber mats and three detected no significant difference between groups. All trials had limitations including small sample sizes, non-random allocation to treatments, non-blinded assessors, and short exposure time to mats.

It is suggested that another trial be carried out, rectifying these limitations, to confirm the usefulness of rubber mats in reducing sow lameness. The willingness of producers to implement rubber mats in their herds has also been assessed based upon the feasibility of rubber mats, and the level of agricultural subsidies and concern for animal welfare across countries.

eBook download: The full article can be read in our eBook.
Sudden death caused by ‘Phalaris’ in some ewes

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Introduction

This producer has a small farm in the Mt Pleasant area that he visits most days, as he does not live on the property. One Saturday morning in late November he visited to find 15 Dohne ewes dead and one moribund. Local vets were unavailable to assist in a timely manner.

The initial suspicion was a poisoning event and the moribund sheep and 2 others were examined. There was no obvious pathology but Phalaris toxicity was diagnosed on histopathology. The pasture available was of good quality- a mix of rye grass, clover, cocksfoot and Phalaris, with no obvious poisonous weeds, and other sheep and cattle grazing nearby appeared unaffected. The remaining sheep in the paddock were moved to an adjacent paddock and no further deaths occurred.

History

The owner is a careful and particular manager and is well educated in sheep production. This mob of 230 five or six year-old Dohne ewes in score 3+ condition were shorn and drenched about 2 weeks previously. On Saturday 24th November 15 ewes were found dead in a paddock, 1 more was moribund, while the balance of the flock still in the paddock were grazing happily. The ewes appeared to have died overnight or early in the morning, and 4 or 5 had been attacked by foxes. The owner has not experienced mortalities like this in the past. On close examination of the pasture, it appeared that clumps of Phalaris had been selectively grazed in preference to other species. The weather had been cool with some light rain in the prior week and so it is likely that young Phalaris shoots had been available.

Test results

A moribund ewe was euthanased and autopsied, along with two others that appeared to have died within 4-6 hours. All had full rumens and were in good body condition, with a slight reddening of the abomasal mucosa seen as the only notable post mortem change.

Blood tests showed very high urea, AST and CK levels in particular, and ocular fluids were negative for nitrate/nitrite levels. Histopathology on brain tissue established the diagnosis:

Brain and cranial cervical cord (Sheep 1-3): Neuronal pigmentation consistent with phalaris toxicity. Small intestine (Sheep 1): Enteritis, eosinophilic, multifocal, minimal to moderate; with protozoan parasites.
Interpretation

Phalaris toxicity is well documented, but rarely seen in this area of the Adelaide Hills. It typically occurs in cooler, mild weather in autumn to early summer when fast growing shoots are available and both sudden death and a ‘staggers’ syndrome can be seen. The toxic principle is dimethyltryptamine and phenylethylamine (alkaloids) that act on cardiac muscle and brain tissue. 1 Sudden death is from heart failure, but the incoordination syndrome (‘staggers’) occurs after prolonged exposure, when sheep are disturbed and driven. These symptoms may appear weeks after exposure and are very variable.

Discussion and Recommendations

Government veterinary services provide an important role in assisting diagnosis and disease surveillance activities particularly where situations are complex, remote or private veterinary services are stretched or diminishing. This producer contacted me having been to a workshop some years ago that PIRSA presented, reaffirming the fact that producers will contact the people they know first for advice.

Although Phalaris poisoning (acute death or staggers) cannot be treated in affected animals, removal from the source does aid recovery, and in high risk situations administering cobalt prior to exposure does seem to have a protective effect. This can be done orally at weekly intervals (28mg/ week) or in fertilizer applications 1 but in this situation, simply waiting until Phalaris pastures are more mature before grazing with sheep should be sufficient.

A Flock & Herd paper that might be of interest is included in the references.

References:


2. Phalaris Poisoning Syndromes Associate Professor Peter Windsor, Farm Animal Health, University of Sydney Posted Flock & Herd September 2016 flockandherd.net.au/sheep/reader/phalaris-poisoning-syndromes

If you could only do one intervention/test—what would that be?

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Rescue dog displaying problem behaviour. Go to the eBook (cve.edu.au/control-and-therapy) to see video. Luna is an adult female-neutered rescue dog already on Clomipramine and Clonidine.

Suddenly 3 months into her new home she begins to madly hump the male owner 24/7 to the point that neither dog nor owners have had any sleep for 3 days.

What is the one simple thing you can do to bring immediate resolution and cessation of issue?

Send answers to joanne.krockenberger@sydney.edu.au and best answer receives a DVD from our library. Visit vetbookshop.com.
Hoke, a 2-year-old male castrated Siamese cat, presented to our referral hospital on 22/2/16 for further evaluation of severe anaemia. Hoke had no known previous medical problems, was a well-cared for indoor only cat in good body condition, and was FIV/FeLV negative. He had been purchased from a breeder as a kitten in New Jersey, USA. Two days prior to presentation, he had vomited at home, and was subsequently very lethargic.

His family veterinarian performed a complete blood count and chemistry panel prior to referral. Hoke’s hematocrit was 8.5%, with a mild increase in reticulocytes (56.2x10⁹/L), normal red cell indices, mild neutrophilia (16.85x10⁹/L), monocytosis (1.19x10⁹/L), and thrombocytopenia (85x10⁹/L). His total protein was normal at 71g/L (with normal albumin and globulin fractions; albumin 35g/L, globulin 36g/L). He had mild azotemia (BUN 20.71mmol/L, creatinine 185.6µmol/L); no urine sample was obtained at that time. His total bilirubin was elevated at 22.23 µmol/L and his ALT did not register on the in-house machine. ALP and GGT were normal.

Given the presence of regenerative anaemia, normal total solids, and increased bilirubin, the referring veterinarian made a very logical list of differentials including *Mycoplasma haemofelis* infection and immune-mediated hemolytic anaemia (IMHA).

However, after referral to our internal medicine practice, an abdominal ultrasound was performed. The liver was notably enlarged, with rounded borders and irregular and mottled parenchyma. Hyperechoic and hypoechoic nodules were present. The kidneys were also abnormal—they were small, hyperechoic, and with poor corticomedullary definition. Most concerning was the presence of a moderate volume of echogenic free fluid. A sample of the fluid was obtained, and the PCV was 22%, consistent with a haemoabdomen. His ALT, with dilution, was 3589U/L. A slide agglutination test was negative, and a blood smear was unremarkable.

Hepatic amyloidosis in a young Siamese cat presenting with severe anaemia: clinical and therapeutic thoughts

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Figure 1. Hoke.

Figure 2. Liver ultrasound.
After some quick research was performed about this unusual case, systemic amyloidosis was strongly suspected.

In the absence of a traumatic event, haemoabdomen in the cat is a rare occurrence. Interestingly, neoplastic and non-neoplastic aetiologies were fairly evenly represented in a recent study of feline spontaneous haemoabdomen. The most common non-neoplastic causes identified in the aforementioned study included coagulopathies (secondary to sepsis, rodenticide and pancreatitis) and hepatic necrosis. Hepatic disease, in general, was the most common cause of haemoabdomen in cats according to the study. Hepatic amyloidosis with associated liver fracture was the specific cause in 4/35 (11.4%) of the non-neoplastic cases and 4/65 (6%) of the total cases.

Systemic amyloidosis occurs when amyloid protein accumulates excessively in various organs. The accumulation causes interference with normal organ function, as well as tissue and vascular fragility. Amyloidosis in the cat has been described most often in the Siamese, Abyssinian, and other Oriental breeds, but has also been identified in the domestic short hair (DSH)/mixed breed cat. Causes include chronic inflammation, infection, neoplasia, and of course, familial inheritance. No specific cure exists for amyloidosis, and the prognosis is poor to grave, especially when clinical signs are present.

Hoke received a blood transfusion the day he was admitted, and was discharged the following day on anti-inflammatory dose of prednisolone, Vitamin K, and Denamarin (SAMe-S-Adenosylmethionine and silybin extract from milk thistle). Grape seed extract was soon added based on a 1999 case report describing a prolonged survival time in similar cat given proanthocyanidin antioxidants. Additional therapies, described in more detail below, were gradually added to address his renal disease and associated symptoms.

Hoke was rechecked at our hospital 8 weeks later. The appearance of his liver on ultrasound was improved, and no free fluid was noted. His hematocrit was 36%. Six months after that, he remained clinically normal at home, with a normal haematocrit.

Nineteen months after he was initially admitted to the ICU, Hoke experienced a similar episode of significant anaemia (PCV 16%, total protein 69g/L) and lethargy. His azotemia had progressed (BUN 28.84mmol/L, creatinine 274µmol/L). His family allowed another blood transfusion, and Hoke spent one night in the hospital again.

In September 2018, 31 months after his initial presumptive diagnosis, Hoke began vomiting and was experiencing abdominal pain. He was inappetant and weak. Blood work showed severe azotemia (BUN and creatinine levels too high to read), mild non regenerative anaemia (hematocrit 28%), and a moderate increase in ALT (625U/L). Hoke’s family took him home for one evening to say goodbye prior to euthanasia, but he passed away at home.

Necropsy showed bilaterally severely shrunked kidneys with irregular contour. The liver was swollen with heterogeneous colouring, and the tissue was noted to be extremely friable.

Histopathology confirmed severe, diffuse amyloidosis in the liver, with associated marked hepatocellular degeneration and atrophy. The kidneys showed evidence of acute and chronic infarcts, with regions of glomerular amyloidosis (to a lesser extent than that found in the liver).

Hepatic amyloidosis isn’t a common disease, but it is important to consider this differential in a young anaemic cat, certainly of Siamese and related breeds, but in mixed breed cats as well. It is also important to resist the temptation to completely eliminate the possibility of blood loss anaemia in a cat with normal total solids, as Hoke had normal total solids during both of his documented hepatic fracture/haemoabdomen episodes, and normal total solids have been noted in other case reports of this disease as well. In reading other case reports, it seems quite common for clinicians to consider a working diagnosis of IMHA or _M.felis_ first as a cause of regenerative anaemia and normal total protein +/- elevated bilirubin in these cases (as both the referring vet and I did prior to abdominal ultrasound). An abdominal ultrasound was a key diagnostic tool in this case.

Given the grave prognosis for this disease, I quite was happy with 31 months of good quality life post-diagnosis, as was the family. Of note as well is that Hoke died of kidney disease, not anaemia or hepatic failure.

Hoke’s clotting times weren’t checked during his initial hospitalisation, but he was treated presumptively with daily oral Vitamin K, based on previously published literature about this condition. Denamarin was used for general hepatic anti-oxidant effects. Hoke’s family was concerned about steroid side effects, and while I initially started prednisolone at a 0.5mg/kg dose, we dropped to 0.2mg/kg for most of his life per their wishes (increased again to 0.5mg/kg at the time of the second hepatic fracture, then tapered again). Hoke received oral Cerenia almost daily, oral mirtazapine as needed, and sublingual Buprenex as needed. Hoke ate a therapeutic prescription renal diet. Yunnan Baiyao, a proprietary traditional Chinese medicine used in human and veterinary medicine for hemostatic effects, was added after his second hospitalisation. Lastly, Hoke received oral grape seed extract (20mg a day) continuously after his initial diagnosis. A case report from the Control & Therapy series 20 years ago describes a 3.5yr old Siamese cat with histologically confirmed severe hepatic amyloidosis, who was still alive 3 years post diagnosis, and the only treatment provided to the cat during that time was antioxidant tablets containing Mediterranean pink bark and red wine grape seed extracts. The author of the case report postulated that feline hepatic amyloidosis is associated with a hyper-stimulated immune response that results in excessive free radical formation, and that free radical scavenging by these very potent antioxidants was the cause of the clinical remission.
The truth is that I don’t know what helped Hoke live for as long as he did, and perhaps the same clinical outcome would have occurred with a different therapeutic protocol, or with no specific therapy at all. However, given the dearth of information about the specifics of this disease and the lack of rigorously tested treatments, my therapeutic approach to my next case of feline hepatic amyloidosis would be the same, as Hoke’s survival time is certainly on the far right of the bell curve for this disease.

References


Figure 3. Liver. Severe diffuse amyloidosis. Amyloid appears here as a lightly eosinophilic homogenous material. H&E.

Figure 4. Kidney. Amyloid is present in perivascular and peritubular areas of the medulla. Congo Red.

Figure 5. Liver. Sinusoids are markedly expanded by amyloid. Hepatic cords are attenuated and atrophic. H&E.

Figure 6. Liver. Sinusoids are markedly expanded by amyloid. Congo Red.
Delayed neurological signs secondary to carbon monoxide poisoning after smoke inhalation

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George is a 5-year-old male neutered Jack Russell terrier cross who presented late one winter night to the clinic after the house he was residing in caught fire. George had been staying with his owner’s daughter and her 2 dogs. All occupants escaped or were rescued and there were no fatalities. The neighbours (who had retrieved George, and his 2 friend dogs, from the house) presented him at approximately 11pm. One of his friends was deemed ok on rescue from the house, whilst the other was taken to another vet by another neighbour and discharged an hour later.

On physical exam George was hypothermic (T 34.8°C), presumably because the neighbours thought he needed fresh air after he’d inhaled smoke so had him in the ute tray, driving through town and whilst they waited. While not entirely wrong, the ambient temperature in July at 11pm can be -5°C to -10°C without wind chill.

George was taken inside and active warming was initiated whilst I examined him. He was unconscious and unresponsive to pain. RR was 30/min with deep, effortful breaths and with harsh large airway sounds and crackles and wheezes over both sides of the chest. HR was 120/min, mucous membranes were a muddy, partially cyanotic colour, with CRT>2s.

George was started on an oxygen mask while an IV line was secured and warmed fluids (Hartmann’s) administered at shock rates for half an hour, then reduced to twice maintenance. Within this time George’s mucous membranes became quite red with CRT <1s. Nasal oxygen was placed and run at 1.5L/min.

George was also given Meloxicam 0.2 mL subcutaneously (SQ) with an estimated body BW of 5kg, as well as enrofloxacin 5mg/kg SQ to prevent pneumonia. Unfortunately, I could not get the pulse oximeter to read consistently on his tongue or prepuce etc. It appeared to be having one of those temperamental moments.

George was grossly very dirty, sooty with a strong smoke odour but no other injuries were found. Monitoring and support was continued for the next 2 hours, during which time George did not regain consciousness but did begin to respond to toe pinching and other noxious stimuli. He coughed if moved, but his RR improved to 20/min- still with deep effort, but less than on presentation. His HR decreased to 100/bpm. Nasal O₂ was reduced to 1L/min, then 0.5L/min and RR remained steady, as did HR and MM colour (still quite dark pink). Temp was nearing normal at this stage.

By the next hour George was voluntarily moving slightly, and if I called his name his eyes would roll up, but roll back down again. He was normothermic but HR and RR remained the same, however, the respiratory effort had reduced along with most of the crackles. Fluid rate was then reduced to just below maintenance for the remainder of the night so as to not encourage pulmonary oedema in his already damaged lungs.

The next morning George was conscious and having bouts of prolonged coughing, followed by production of white froth (pulmonary oedema). Mucous membranes were pink, and very moist with a 1s refill. George was normothermic, HR and RR were within normal limits, and chest sounds had decreased. George’s SpO₂ remained normal without intranasal O₂ which was good as he had pulled it out overnight anyway: He would eat soft foods if hand fed, with an exaggerated swallow that indicated what a sore throat he had (poor guy), but he was otherwise interactive and affectionate, if exhausted. George would walk outside for toilet breaks, but got tired very quickly and slowed down, had an increased RR and would begin coughing with production. George was started on oral enrofloxacin and meloxicam and discharged with full support that afternoon as he appeared stable, and was ambulatory and eating. The owners were to monitor the RR, the coughing and report any colour or blood in the production, a reduction in activity or if George became generally quiet.

Four days later George came in for a revisit as the owners had found him unable to walk that morning. He had been going really well at home, interacting well, going outside and sleeping. The coughing had continued but had been reducing in frequency and production. George was eating, drinking, urinating and defecating normally but had not
defecated in the last 24hrs so the owners had given him olive oil on his food.

Physical exam revealed conscious proprioceptive deficits in both hind limbs with the left slower than the right. Patellar reflexes seemed reduced. George showed significant paresis of his hind limbs when attempting to walk, though the forelimbs seemed unaffected. Anal tone was good, spinal palpation exhibited no pain or response. Temperature was normothermic. Chest sounds were clear, although George still coughed occasionally. George also appeared to be partially centrally blind - he was having trouble navigating around objects in the room, and identifying treats in front of his face. Both pupils were normal, with consensual pupillary light reflexes. Fundic exam was difficult as George wouldn’t keep still but there appeared to be no issues with the anterior chamber or lens that would interfere with vision. The owners also felt he had suddenly become quite deaf as he would not respond to his name often, and seemed to have no interest in exploring the yard whereas before he would sniff the boundaries (lack of sense of smell?). He also only turned to the right in consult and exhibited some pacing which seemed almost compulsive in nature. The clinical signs were consistent with a widespread neurological problem, or a multifocal one and I was a bit stumped. The owner was not open to referral and imaging.

I sent George home on continued meloxicam and supportive care such as slings to assist walking, touch to...
help navigate, leash walks outside, diet changes to more odiferous foods such as fishy cat foods etc. Whilst I tried to work out what was going on. I found a single paper on a case of delayed carbon monoxide toxicity in a dog with smoke inhalation ([https://www.researchgate.net/publication/229956606_Full_recovery_following_delayed_neurologic_signs_after_smoke_inhalation_in_a_dog](https://www.researchgate.net/publication/229956606_Full_recovery_following_delayed_neurologic_signs_after_smoke_inhalation_in_a_dog)) as well as other mentions of delayed neurological deterioration in cases of carbon monoxide toxicity ([https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1476-4431.2005.00140.x](https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1476-4431.2005.00140.x)). All animals in these papers eventually made a recovery if they survived the initial event.

So… it would appear this was what was happening to George, and that really it was only the tincture of time. I got the owner to keep a diary of what signs George was showing and to keep in regular contact so that we could support him and change the plans if need be.

Table 1 contains the owner’s report:

George continued to improve but maintained some defensive aggression towards the other dogs he was present with at the time of the fire. He also exhibited increased aggression and increased anxiety whenever he visited the house that burned, that was not evident when he was at home. The owner was advised to keep visits minimal and short and George was given Zylkene® to assess if this could improve his anxiety pre-visit as well as reduce his aggression towards the other dogs. The owners were also advised to allow George his own space and not force him to be with his ‘friends’ again. The behavioural changes and management would have needed more consultation and much more time, which the owners did not want to pursue.

George never appeared to regain full vision as he still found it difficult to track balls thrown by his owner, but he recovered almost fully in every other way (mobility, hearing, and balance, no more vomiting).

George was lost to follow up after this but it serves as an interesting possible complication of getting an animal through the initial crisis of severe smoke inhalation, only to be served with neurological problems for weeks afterwards. Despite this, however, most animals seem to make a pretty good recovery with proper support.

Smoke inhalation is a very uncommon presentation in the veterinary world, yet a reasonably common presentation in the human medical world. Treatment modalities appear very similar, with the same delayed neurological degeneration described in up to 20% of human patients. Oxygen therapy, ABC support as needed, N-Acetylcysteine (Mucomyst) nebulisation (if you have NAC), and cover against pneumonia appear to be the cornerstones of human treatment.

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Diagnosis of carbon monoxide poisoning

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Carbon monoxide (CO) is a tasteless, colourless, odourless gas. 50,000 cases of CO poisoning in people are reported in the US each year.1 CO exposure is most commonly from car exhaust systems, faulty heaters, fires and industrial accidents. There are relatively few reports of pure CO poisoning in the veterinary literature.

CO poisoning causes cell damage due to tissue hypoxia. Hemoglobin has a 250-fold greater affinity for CO than oxygen. CO completes with oxygen for binding to hemoglobin and by displacement of oxygen, reduces the oxygen carrying capacity of blood. Additionally, the binding of CO causes a conformational change of hemoglobin, leading to a shift of the oxyhemoglobin dissociation curve to the left (increasing oxygen affinity for hemoglobin and decreasing oxygen release to tissue).

Symptoms of CO poisoning in humans are extremely non-specific, including headache, dizziness, fatigue, nausea/vomiting, altered mentation, chest pain, shortness of breath, and loss of consciousness.1

Blood levels of carboxyhemoglobin do not directly correlate with the degree of clinical severity nor the prognosis. As well as causing hypoxia, CO causes multiple other toxic effects, including oxidative stress following the tissue hypoxia and cellular damage due to inflammatory processes. Organs with high oxygen demand are most susceptible to hypoxic injury, and brain and cardiac effects dominate acute signs of CO poisoning and also feature in delayed complications in both human2 and veterinary cases.3-6

The classic ‘cherry red’ mucous membranes we were taught occur with CO poisoning tend to occur only when carboxyhemoglobin blood levels reach lethal levels in humans.1 The bodies of cats that died from CO poisoning had bright red skin, muscles and bright red discolouration of the abdominal serosa7, however alive dogs and cats that presented with CO poisoning had normal pink mucous membranes.6

In humans, the diagnosis of CO poisoning is made by a clinical triad.1

1. History of recent exposure to CO
2. Symptoms consistent with CO poisoning
3. Elevation of arterial or venous carboxyhemoglobin level (>3-4% in non-smokers or >10% in smokers)

Oxygen saturation measurements by use of conventional pulse oximetry (SpO₂) are typically normal in cases of CO poisoning. Conventional pulse oximetry uses two wavelengths of light (660 and 940 nm) emitted across a vascular bed to calculate the relative proportions of oxy- and deoxyhemoglobin, based on their different absorption spectra. Normal deoxyhemoglobin has relatively higher absorbance at 660 nm, whereas normal oxyhemoglobin has relatively higher absorbance at 940 nm. Carboxyhemoglobin has similar absorbance to oxyhemoglobin at 660 nm. Therefore, when carboxyhemoglobin levels are elevated, pulse oximetry gives falsely elevated arterial oxygen saturation. In a prospective study of 16 adult humans that had CO exposure, oxygen saturation as measured by conventional pulse oximetry failed to decrease to less than 96% despite carboxyhemoglobin levels as high as 44%. In one published case series of CO poisoning, all animals (2 cats, 4 dogs) had SpO₂ levels of 97-100%.

Demonstration of elevated carboxyhaemoglobin can be made directly with CO-oximetry (requiring a blood sample), or, indirectly with multiwave pulse oximetry (also known as pulse CO-oximetry) using a probe placed on the skin.

CO-oximeters (these are different to pulse oximeters!) perform spectrophotometry on injected arterial or venous blood and directly measure the concentrations of oxy-, deoxy-, carboxy-, and methemoglobin. Many newer blood gas analysis machines have inbuilt CO-oximeters. CO-oximeters are the method of choice for the diagnosis of CO poisoning.

Similar to conventional pulse oximetry, but using multiple wavelengths of light, multiwave pulse oximeters are available which can estimate four different types of hemoglobin (oxy-, deoxy-, carboxy-, and methemoglobin) in a non-invasive method. Multiwave pulse oximeters may miss patients with CO poisoning, so if CO poisoning is suspected and multiwave pulse oximetry is normal, CO-oximetry is still recommended. Multiwave pulse oximetry is useful for the diagnosis and monitoring of animals with CO poisoning or smoke inhalation (carboxyhemoglobin) or animals that have methemoglobinemia.

References


Cruz is a male neutered domestic longhair, indoor-outdoor cat rehomed through a cat rescue charity as an adult cat. He is presumed to be about 5-years-old. His owners were told that he had a history of urinary issues (mainly blood in the urine) and was on Hills prescription c/d™ feline dry as recommended by the shelter vet. He lived in a house with one other older cat and 2 dogs. He was a nervous cat and the other cat in the household was known to attack him.

We saw him for the first time in late 2015 for a recurrence of his haematuria and on examination a wound on his back was found that was presumed to be related to a cat fight. He was overweight and nervous in consult so it was difficult to palpate his bladder. The owner had reported that the haematuria was sporadic and she noticed it had returned with urinating around the house, specifically the kitchen.

He was treated for a cat fight abscess with a course of antibiotics and non-steroidal anti-inflammatories. A presumptive diagnosis of idiopathic cystitis or sterile haematuria was made given his history and the results of an in-house urinalysis (dipstick and sediment exam) so we discussed environmental enrichments, pheromone sprays and other treatment options to try to prevent recurrence.

Cruz continued to have repeated episodes of haematuria and multiple urine samples (from non-absorbent litter) examined in-house and at an external lab for sediment and culture repeatedly showed the presence of blood and only on one occasion were a small amount of struvite crystals seen. Biochemistry was also unremarkable.

When Cruz continued to have haematuria long after the passing of the owner’s other resident cat, the owner finally agreed to further diagnostic tests in April 2018. We were definitely not expecting to see the air gun pellet floating inside the bladder on the lateral radiograph! We are unsure if the pellet was present from his shelter days or a more recent acquisition given his long history of haematuria. With this new discovery, a coeliotomy was performed the following day and the pellet was removed from the bladder. He has not had an episode of house soiling or haematuria since the surgery. Although free floating pellets in bladders are pretty uncommon, we have found this case a useful aid to get owners to agree to a more thorough work up of our urinary cases.
I thought I would share this relatively new (for Australia) eye product: Clerapliq. As vets we should ‘Anticipate, Assess and Alleviate’ pain wherever and whenever we can in our patients.

I have an ongoing concern that for ocular conditions, effective home analgesia is often inadequate.

CLERAPLIQ® has been shown to be effective in healing ulcers and is well known to veterinary and human ophthalmologists in Europe.

It has also been documented that RGTA (Regenerating Agents- polysaccharides specifically designed to replace degraded heparin sulphate in injured tissues) molecules alleviate pain.

To date, no adverse effect has been reported.

I have not used it on enough ulcers to declare it is faster or better than any other methods in use; but what I can say is that the change in the afflicted dogs’ behaviour does indicate that it certainly alleviates pain.

A healthy canine cornea may be less sensitive than a human cornea but a diseased cornea hurts, regardless of the species.

The addition of Clerapliq to our pet ulcer regime provides a level of analgesia I have not seen with any other home analgesic regime. The dogs sleep all night and wake the next morning more alert, social and interactive; to the point that the owners comment on how they didn’t realise how bothered the pet actually was by the eye condition.

I would love to hear others’ experiences and obviously any caveats about when to absolutely avoid the product.

It’s available through Clifford Halem CH2 as a normal stock item or you can buy direct from Silverglide themselves:

SilverGlide.com.au

The site also has an expanded explanatory video on the product for those who want to know more.
Yunnan Baiyao

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Background

Yunnan Baiyao (pronounced YUN nan BYE yow) was used in the Vietnam War where the powder/tea was packed into bullet wounds, allowing Northern Vietnamese soldiers to survive long enough to get back to medical help. Today it is sold as a tea with the leaves used directly on wounds. It is also available in powder, capsule and spray forms.

It is attracting interest in both human and veterinary medicine for treating bleeding lesions in the bladder. In emergency medicine and first-opinion practice, where haemorrhaging cases tend to be first presented, there is a move to use this Chinese (secret) herbal combination to stop bleeding. Emergency vets instil it up the nose of a dog having spectacular epistaxis and also sprinkle it on wounds and bleeding tumours.

I have used it for:

› Inoperable masses, especially in geriatric patients where the animal is coping with the mass but the owner is not coping with the blood flicked everywhere and may be considering euthanasia sooner rather than later. Yunnan Baiyao sprinkled on the mass stops the bleeding and dries up the area to buy the owner and the pet some more time. The photo (Figure 1) is of a 19-year-old amazingly happy cat not bothered by the mass on its head; however, the in-laws minding the cat were bothered by the blood flecks. Yunnan powder kept the cat alive until owners came back and then for several months more.

› Yunnan Baiyao also worked well on an ancient but mostly healthy dog with inoperable rectal ring tumours that bled everywhere—client happy.

I also have clients keep capsules at home if their pet has lesions that are likely to bleed and who have difficulty getting veterinary attention quickly i.e. out in remote country areas or unable to transport their dog because of the size of the pet or owners who do not drive. Yunnan Baiyao has been wonderful to stop the bleeding until vet assistance can be organised. Even small lesions can bleed spectacularly, so it is good for owners to keep on hand to sprinkle on them.

Other colleagues report great success with:

› Cutaneous hemangiosarcomas that bleed everywhere

› After nasal biopsies which tend to bleed

› Stopping nail bleeding after too high nail trim. It will still take time to stop the bleeding but faster than not using it with the bonus being that it’s relatively safe if ingested by the animal as well.

› Stuffing the contents of the capsule into the nose for tumour epistaxis

› Bleeding lesion at tympanum of an elderly dog. Three weeks’ later, there was no hemorrhage and the area was easily visualized—a lobulated, pink mass. Two months later, on two occasions, when the owner had run out of Yunnan Baiyao the lesion began bleeding again three days later when the dog shook its head. At no other time when using it (with continued cleaning, etc.) had there been any bleeding.

Other colleagues report temporary success with oral squamous cell carcinoma, thus buying the pet some time before eventual euthanasia.

Doses for Yunnan Baiyao

Doses vary so I contacted some overseas vets and one of the biggest exporters of Yunnan Baiyao to see what is recommended. This seems to be the most popular regime but use it at your own discretion.

For any active internal bleeding:

Dog/cat: 1 capsule (0.25 g) per 15-20 lb (6.8kg-9.07kg) given orally 4 times daily for 1-2 days.

Horses: 8 g given orally 3 times daily for 1-2 days.
After bleeding is stopped:
Dog/cat: 1 capsule (0.25 g) per 20-30 lb (9.07kg-13.6kg) given orally 2 to 3 times daily for 3-7 days.
Horses: 4 g given orally 2 times daily for 3-4 days.

Maintenance dose:
Dog/cat: 1 capsule (0.25 g) per 30-50 lb (13.6kg-22.7kg) given orally 2 to 3 times daily.

External Bleeding or any open wound:
Topical application of Yunnan Baiyao.

Editor’s note:
1. Although the exact formulation is obscure, the herb (*Panax notoginseng*) is believed to be the principal anti-haemorrhage constituent, although it has also been speculated that the haemostatic action may be due to microscopic plant fibres that stimulate platelets to aggregate.
2. Tranexamic acid (IV, SC, orally) has a similar range of indications and is a bit more conventional. Widely used in people for menstrual bleeding, nose bleeds, polytrauma and any major orthopaedic surgery (e.g. hip replacements). However, it is more expensive, short dated and cannot be used topically on wounds.

Note: Yunnan Baiyao can be purchased from most Chinese food stores or supermarkets which are often found close to universities with overseas students. Costs about $12 a box.

Disclaimer:
I am not an herbal vet in any way—I am a mainstream western vet—so if anyone knows more about why we shouldn’t be using Yunnan Baiyao leaves and powder, I’d be happy for you to share it in the next C&T issue. Please email your reply to: Joanne.Krockenberger@sydney.edu.au. But, in the interim, when faced with unfixable/hard to access haemorrhage Yunnan Baiyao has been superb.
Cow Pox in a cat

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Buzz Walls is a 2-year-old, male neutered, domestic shorthair, outdoor cat who lives in Barnsley, South Yorkshire. He has been in the Walls family since he was a 2-month-old kitten. He has been coming to my practice since his first vaccination and is up to date with vaccinations and with his flea and worming treatment. Our patient is an excellent hunter and never forgets to bring lovely presents to his parents, such as dead mice and other small animals.

Buzz was brought to my branch in September 2017, Mrs Walls reported that he hadn’t been himself for the last couple of days—he had been a bit lethargic, with decreased appetite and not wanting to go outside.

On clinical examination Buzz seemed quite shy, not as lively as in previous consultations although his demeanour was good. The clinical examination was unremarkable. My recommendation at that point was to monitor him closely, and keep him indoors. A single injection of NSAIDs (meloxicam) was given and he was to be re checked in 24 hours.

The owner couldn’t make the appointment the day after but reported that Buzz seemed better, although he still wasn’t 100% right.

Five days later Buzz came back to the clinic with the same signs but this time Mrs Walls thought that he had been fighting as he presented with several wounds on his face, head, neck, back and fore limbs. The skin lesions were small nodules strongly scabbed with crusts, predominately on his face and forelimbs, but there were some others on his neck, and back. These lesions were small, between 2 and 4 mm, the bigger ones were ulcerated and seemed to be pruritic.

After a discussion with Mrs Walls we both agreed that the best thing for Buzz would be for him to be admitted for intravenous fluid therapy support and deeper investigations.

Problem list
Our patient presented at this point with some skin problems but systemic signs were also developing.

1. Nodules heavily crusted and scabbed
2. Mild pruritus
3. Ulceration of scratched scabs
4. Cellulitis
5. Lethargy and anorexia

My differential diagnoses started with the following, listed by most to least likely:

› Eosinophilic granuloma complex
› Immune-mediated dermatoses like pemphigus or lupus erythematosus
› Miliary dermatitis
› Neoplasia
› Cat fight (cat bite abscess)
Initial blood and dermatological tests

With these signs I decided to perform a full biochemistry with electrolytes and a complete blood count. I also added a bidirectional flow ELISA snap test for FeLV/FIV.

Haematology results were unremarkable, biochemistry parameters were all within normal limits but with a slightly low urea (4.8 mmol/L). The FeLV/FIV snap test was negative.

Once Buzz was stable the dermatological tests were performed under GA:

TRICHOGRAM: performed in-house, fur was selected on healthy part of skin but also near the lesions. There was a normal telogen: anagen ratio and the majority of hair shafts remained intact, although some were broken. This was possibly caused by self-trauma due to pruritus.

SKIN SCRAPING: in-house testing was done and I also sent off a sample to an external laboratory. The selected areas were the lesions on head and neck. No parasites, e.g. demodex, as an underlying condition were seen and no other findings were reported by the external lab.

DERMATOPHYTE TEST: I didn’t perform a Woods lamp test as I didn’t suspect there would be any superficial mycosis. However fur was sent to an external laboratory, placed in a sterile container for them to do the culture on a dermatophyte test medium.

SKIN BIOPSIERS: three skin biopsies were taken from three lesions: one from head, second from dorsal neck, third from right fore-limb. I used an 8 mm biopsy punch for the first two and a 6 mm biopsy punch for the one on the forelimb. Areas were gently clipped but not cleaned to avoid losing any important information.

SWABS FOR CULTURE AND SUSCEPTIBILITY: 2 swabs were taken from lesions on head and back and placed on charcoal medium to be sent off to the lab.

Unfortunately I didn’t perform cytology as I don’t trust my microscopy skills. In future cases I will start doing this to improve my skill set in this area.

Test results and Buzz complications

Buzz recovered very well from the anaesthesia and was discharged with a cefovecin injection (Convenia® [Zoetis] 8 mg/kg SC) and a single injection of Dexamethasone (Dexafort® [intervet] 0.5 mg/kg). Both of these injections were to cover the secondary infection and help (or at least I thought that I was helping) with the pruritus.

Two days after being discharged, Buzz had to be seen out of hours by one of my senior colleagues. Buzz’s skin had deteriorated very quickly, he had many new lesions and they were now oozing and he was extremely lethargic. The beauty of experience is that as soon as my colleague examined Buzz she realised that my list of differential diagnoses was missing an important disease. She strongly believed that we were looking at a cowpox virus case, as she had seen a similar case 15 years ago. Buzz was immediately admitted for intravenous fluid support and opioid pain relief was administered as he was in severe discomfort. On the same consultation my colleague asked Mrs Walls if any member of the family was presenting any skin lesions. Her younger son, a teenager, had just been diagnosed with impetigo a few days previously. My colleague suggested Mrs Walls go back to the GP and report that Buzz was possibly affected with cowpox virus.

The morning after I arrived at work and found out what had happened to Buzz. Talking to my senior vet I felt terrible, as I had administered corticosteroids to him, causing immunosuppression and systemic infection.

The confirmation of my senior vet’s suspicions by the lab took only a few days. The histological evaluation showed the presence of necrotic and ulcerative dermatitis with mixed inflammation and intracytoplasmatic viral inclusions diagnostic for feline cowpox virus infection as aetiology.
In addition, swabs results came back. Buzz had a combination of methicillin-resistant *Staphylococcus pseudintermedius* (MRSP) and methicillin-resistant *Staphylococcus aureus* (MRSA). Following the sensitivities results, the antibiotic we selected to treat buzz was marbofloxacin (Marbocyl [Vetoquinol] 2 mg/kg), having and intermediate sensitivity for MRSP and good sensitivity for MRSA.

**Hospitalisation and treatment**

There is no specific treatment for Cowpox infection in cats but omega interferon has been shown to be helpful¹. Therapy is mainly supportive, such as broad spectrum antibiotic for secondary bacterial infections but definitely corticosteroids should be avoided… In this case as immunosuppression had already happened and systemic signs were seen, Buzz was hospitalised for supportive treatment. In recent articles they have suggested the use of Feline interferon omega.

Buzz was admitted and kept in isolation and barrier nursing. His treatment consisted of intravenous fluid support and pain relief therapy. Initially he had a Convenia injection but once we had the culture results we started with marbofloxacin. Ulcers were cleaned with chlorhexidine and a buster collar was placed to avoid self-trauma.

**Outcome**

Buzz made a complete but steady recovery. He ended up being hospitalised for nearly three weeks. In this time his wounds dried up and the scabs subsequently fell off. His appetite returned and he was getting stronger and happenier. Antibiotics were stopped after 2 weeks of treatment.

After two negative swabs Buzz was discharged with no further treatment, but keeping the buster collar and cleaning the remaining wounds with hibiscrub (always with gloves).

Fortunately Buzz made a good recovery, the only reminder of the disease are a few alopecic areas where the bigger ulcers were.

This case has been very challenging for me and has made me realise that even uncommon diseases can present themselves on your doorstep. This has highlighted to me the importance of getting a good anamnesis and complete differential diagnostic list.

Having seen and dealt with this disease will help me in the future, as I’ll be more likely to recognise the signs faster. Out of 14 vets that work at my practice only one recognised the disease. I made Buzz’s condition worse with the steroid injection. However this could have been worse as cowpox has been reported to be involved in severe respiratory problems or even death.

**References**


**Editor’s note**: Buzz lives in the UK. Cow Pox is not present in Australia.
Why do dogs and cats react differently to heartworm?

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Cats and dogs may vary as to how they ‘see’ dead heartworm material.

Segments (1cm lengths) of one D.immitis adult female filaria were placed individually in vials of cat (n = 3) and dog (n=3) blood, immediately after venous collections were made into EDTA. Fragments were left in place for 30 minutes and the vials stored in a water bath at body temperature; all procedures being undertaken in the same time / same venue re any environmental influences. The only variable was the blood sources.

The fragments were then prepared for scanning electron microscope (SEM) and the resultant photographs (n = 52) evaluated independently i.e. samples were not identified for species or animal number and were assessed by an (independent) pathologist (Dr R. Sutton).

SEM showed that dog platelets hardly ‘saw’ the fragments whereas cat platelets overwhelmed the fragments creating large clumps, assumed to be mainly platelet-derived.

So cats may ‘see’ dead filariae more strongly and activate a clotting cascade that is far superior to that of the dog, helping to perhaps explain why dogs and cats react to dead filariae so differently – i.e. cats can die (with a single dead or ruptured worm) whereas dogs can have many thrombi and survive (they have the fastest clot lysis times of all species, including humans). This could be another explanation as to why dogs survive such massive pulmonary thrombosus (and break-down debris from filariae) compared to other species, especially the cat.

(Atwell - Trial B16 (1995) Pfizer HWD Research Conference, Marthers Vinyard, USA)
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I have used a number of DR systems in the past, both in veterinary and human practice (Schick, Sirona, Kodak and Genoray), but I would have to say that the results and image quality that I am getting with the iM3 CR7 Vet is the best so far.

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Dr. Anthony Caiafa
BVSc BDSc MACVSc (SA Surgery and Veterinary Dentistry)
Severe sunburn in three dogs presenting to veterinary clinics on the Atherton Tablelands of Far North Queensland

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Abstract

Three dogs were presented with near identical dorsal skin necrosis to two veterinary clinics on the Atherton Tablelands in far North Queensland in 2017. Initially the origin of these lesions was unclear, but further investigations strongly suggested an aetiology associated with dorsal thermal necrosis (DTN), a rarely reported condition caused by excessive and prolonged exposure to UV radiation. One case in particular had all the key features for differential diagnosis of DTN: an incident of prolonged exposure to UV radiation with high ambient temperature and associated symptoms of heat exhaustion, a large necrotising wound but with delayed eschar formation, and the absence of hair singeing or chemical or abnormal odours that would characterise a more typical thermal burn from other causes. To our knowledge these are the first documented cases of DTN in Australia. We also describe our approach to treatment of the resulting wounds and the time course of progression of the wounds until full closure.

Introduction

Thermal burns are relatively uncommon in dogs and are usually caused by flame burns or scalding (e.g. from hot water in hoses, heating pads, latex gloves filled with hot water for heating during surgery, heating lamps, hot packs, car mufflers, stoves, radiators, and electrical cords).¹⁻³ The depth of the burn is usually classified as superficial (outermost epidermis), partial thickness (epidermis and a portion of the dermis), or full thickness (involving the full thickness epidermis, dermis and sometimes deeper structures).

In contrast to these ‘typical’ thermal burns, there are reports from the USA of burns seen across the back of dogs likely caused by exposure to solar UV radiation. This condition has been described as ‘dorsal thermal necrosis’ (DTN). DTN was first reported in 1999 in a case study of a Dalmatian puppy that presented with full thickness cutaneous burns along the back after prolonged exposure to the sun in a high ambient temperature.⁴ A second record of a full thickness thermal burn attributed to DTN was published in a brown coated Dachshund in 2016⁵ and, most recently, a retrospective analysis detailed a further 16 cases from the south western USA between 2009-2016.⁵⁻⁷ The original publication recording DTN hypothesised that areas of dark or black hair acted as ‘heat sinks’ when a dog was exposed to extreme or prolonged solar radiation, resulting in a thermal burn and skin necrosis.⁴⁻⁶ It has since been shown that animals with tan or brown coats are also more susceptible to DTN.⁴⁻⁷

The aetiology and occurrence of DTN is characterised by a common history of prolonged exposure to the sun (usually >1 - 4 hours) in high ambient temperatures (ranging from 23.9°C to 42.6°C) either through being left outside in areas with little shade (tethered or free), going on long walks or hikes, or simply spending the whole day at the beach. Signs of heat exhaustion were frequently described, some requiring veterinary intervention, others being managed by the owners through a combination of cooling with water, fanning and resting indoors. The time between exposure to solar radiation and high ambient temperatures and the presenting to a veterinary practice for dorsal necrotising skin lesions ranged from 2 days to 2 weeks.⁵⁻⁷
Skin lesions associated with DTN typically appear as large firm coalescing nodules that form along the dog’s back in the initial progression of the condition, and over time open and drain. In cases of later stage presentation, plaques, eschars and full thickness necrotising tissue and fat are reported. Histology shows coagulative necrosis, with full thickness necrosis of the epidermis, adnexa and dermis. In serious cases it can continue down into the panniculus and deeper subcutaneous tissues, with infiltrating inflammatory cells along with erosion, ulceration and serocellular exudation and crusting.

From the literature, the suggested management of DTN depends on the stage at which it is suspected and the severity of the burn. Following initial treatment of any heat exhaustion that may have occurred due to the solar exposure and high ambient temperatures, the burn itself is managed similarly to other thermal burns (debridement of eschars, systemic and topical antibiotics, NSAIDs and pain relief) and can include treatment to stabilise metabolic and systemic effects in severe cases.

In this report we describe three cases of canine dorsal skin necrosis, consistent with DTN, that presented at veterinary clinics on the Atherton Tablelands in 2017. We believe these are the first published records of DTN in Australia and that other cases may go undiagnosed because the condition is not widely recognised. We also outline our treatment approach to facilitate healing of the resulting wounds.

Overview of cases

Presentation and clinical features

Three cases of dorsal skin necrosis in dogs presented to veterinary clinics on the Atherton Tablelands, in Far North Queensland in 2017. Case 2 occurred in April 2017 and Cases 1 and 3 occurred very close together in October 2017.

Of the three cases, Case 1 showed all the characteristic features, clinical presentation and history of DTN and it is from this basis that we compare the other two cases. Figure 1 shows the first photographic record of each case. As illustrated, the presence of extensive dorsal necrosis and eschar formation were the striking features.

Table 1 compares the three cases here with the reported clinical history and features seen in DTN cases from the US literature. Further details can be found in the individual case descriptions, but in summary, what all three cases have in common are:

- Excessive solar exposure (heat exhaustion and the high ambient temperature on the day of injury).
- Dorsal lesions consistent with thermal injury, in particular necrosis, exudation and delayed eschar formation.
- They were not hosed down by the owner using a garden hose, ruling out a scalding thermal or ‘wet’ burn.

<table>
<thead>
<tr>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 21*</td>
<td>Day 27*</td>
<td>Day 5*</td>
</tr>
</tbody>
</table>

*Days calculated from appearance of first lesions
Table 1. Why we think DTN is the most likely cause of dorsal necrosis in the three cases seen, bold italics indicates history or clinical signs indicative of DTN. 3,5,7

<table>
<thead>
<tr>
<th>History</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolonged UV exposure and high ambient temperatures (\text{range 23.9 to 42.6 °C})5</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Heat exhaustion</td>
<td>✓</td>
<td>✘</td>
<td>✘</td>
</tr>
<tr>
<td>Garden hose used to cool dog3</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
</tr>
<tr>
<td>Hair singeing, chemical or abnormal odours present</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
</tr>
<tr>
<td>Mouth lesions (seen in licking of chemicals, electrical burns)</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
</tr>
<tr>
<td>Any other known access to causes of thermal, electrical or chemical burns</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
</tr>
<tr>
<td>On photosensitising drugs</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
</tr>
<tr>
<td>Other known chemicals or products applied to coat/skin in previous week</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
</tr>
</tbody>
</table>

**Clinical presentation**

| Dorsal skin lesions (which can extend from head to sacral area)         | ✓      | ✓      | ✓      |
| Skin lesions found extending down the sides, limbs or chest away from dorsum | ✘      | ✘      | ✘      |
| Initial raised nodules that coalesced to form partial or full lesions    | ✓      | ✓      | ✓      |
| Delayed dorsal necrosis and formation of eschars #                      | ✓      | ✓      | ✓      |

**Histological**

- ‘Wicking or tracking’ necrotic areas seen histological around hair follicles
  - N/A
  - N/A
  - ✘

*Maximum temperature recorded at Mareeba airport on the day of injury*

# as opposed to contact thermal burns where immediate coagulation and eschar formation is seen, dorsal necrosis thermal and scald burns develop eschars in later stages of the disease

Δ a distinct histological feature for scalding thermal burns

^ No histology samples taken

Histology samples were only available for Case 3. One of the main differential diagnosis for DTN would be from a scalding burn injury. In Australia, this is most likely from a hot garden hose with left over water being heated by the sun. Cases have been reported in the US with a similar histological and clinical presentation to that of DTN with the exception of a particular histological finding. Scalding or ‘wet’ burns tend to have a ‘tracking or wicking’ affect down the hair shaft which gives the appearance of necrotic hair follicles surround by normal, viable collagen. This has not been reported in the histological findings of DTN cases. Interestingly, this histological feature was not reported in Case 3 (Figure 2 & Table 2).

Figure 2. Day 5 (showing the depth of the lesions and the three biopsy areas).
Table 2. Histological similarities of Case 3 and the histological findings reported in the literature for DTN. 3–5, 7

<table>
<thead>
<tr>
<th>Reported histological changes for DTN</th>
<th>Case 3 histology sections</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diffuse necrosis across epidermis and dermis, sometimes extending into subcutis depending on depth of injury</td>
<td>✓</td>
</tr>
<tr>
<td>Infiltration of the epidermis through to subcutis of inflammatory cells (including neutrophils)</td>
<td>✓</td>
</tr>
<tr>
<td>Crusting (serocellular in many cases) and ulcerated surface</td>
<td>✓</td>
</tr>
<tr>
<td>Neutrophil exudation</td>
<td>✓</td>
</tr>
<tr>
<td>Epidermal erosion/ulcerations</td>
<td>✓</td>
</tr>
<tr>
<td>Fibrosis/dermal fibroplasia</td>
<td>✓</td>
</tr>
<tr>
<td>Hair follicles showing hyperplasia/dysplastic changes</td>
<td>✓</td>
</tr>
</tbody>
</table>

The histological diagnosis for Case 3 was a subacute ulcerative dermatitis, possible thermal burn to the epidermis with a secondary bacterial infection in some areas.

Treatment strategy

The treatment of the three cases followed basic principles for veterinary management of burns, including debridement of the necrotic tissue and eschars, systemic and topical antibiotic medication, anti-inflammatory and pain management. In addition, a small molecule epoxy-tigliane that is under early stage development as a potential wound healing agent was used on some of the wound area in all three cases. Table 3 outlines the various treatments across the three cases.

Table 3 Treatments received by the three cases presented.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Case 1 (30.6kg)</th>
<th>Case 2 (32kg)</th>
<th>Case 3 (30kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cooling by owner (no cooling with garden hose occurred)</td>
<td>✘</td>
<td>✓</td>
<td>✘</td>
</tr>
<tr>
<td>Initial stabilisation and hospitalisation</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
</tr>
<tr>
<td>Systemic antibiotics</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Topical lotions/washes</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>NSAIDS/Pain Relief</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Surgical intervention - debridement of eschar</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Dose of experimental epoxy tigliane used (mg)</td>
<td>4.4</td>
<td>1.6</td>
<td>0.1</td>
</tr>
<tr>
<td>Wound dressing and changes</td>
<td>✘</td>
<td>✘</td>
<td>✘</td>
</tr>
</tbody>
</table>
Final outcome and time to full wound resolution

Figure 3 shows the final healed outcome for each case. Interestingly, Case 3 was the fastest to resolve, it was also the only case to have the eschars removed surgically within a week (5 days) of the first appearance of dorsal lesions. Subjectively the lesions seen in Case 3 were also more superficial when compared to Case 1 and 2. Case 3 was also the only one to develop loss of hair pigmentation, possibly due to the more superficial initial injury and its resulting effects on the melanocytes of the hair follicles. Interestingly Case 1 had very little visible alopecia or scarring when compared to the other cases. The final healed wounds of DTN cases in the literature commonly reported residual scarring and alopecia.5

Figure 3. Final healed outcome and appearance.

<table>
<thead>
<tr>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image1.png" alt="Image" /></td>
<td><img src="image2.png" alt="Image" /></td>
<td><img src="image3.png" alt="Image" /></td>
</tr>
</tbody>
</table>

Day 91*  Day 146*  Day 87 *  (long term follow up)

*Days calculated from appearance of first lesions

The time frame for healing can be affected by many factors and it is always hard to derive definitive conclusions especially in relation to therapy, from individual cases. From the data that was able to be collected in these cases, removal of the eschar appears to be one of the most important considerations to achieve faster wound resolution. Healing times for DTN reported in the literature is also limited. Table 4 presents a comparison of the three cases compared to the range reported on six other individual cases (Table 4).5
Table 4. Time taken for wound closure and healing for three presenting cases.

<table>
<thead>
<tr>
<th>Case</th>
<th>Total days to heal*</th>
<th>Days to heal after eschar removal</th>
<th>Reported in the literature</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Total days to heal (n= 6) *</td>
</tr>
<tr>
<td>Case 1</td>
<td>146</td>
<td>112</td>
<td>45 – 99 days (Median 75 days)$^5$</td>
</tr>
<tr>
<td>Case 2</td>
<td>91</td>
<td>66</td>
<td></td>
</tr>
<tr>
<td>Case 3</td>
<td>31</td>
<td>26</td>
<td></td>
</tr>
</tbody>
</table>

*From first appearance of lesions

Individual case descriptions

Case 1. ‘Bandit’: Entire male, Australian cattle dog, blue and black in colour, 3 years-of-age.

Table 6. Presenting history and treatment for Bandit.

<table>
<thead>
<tr>
<th>Date</th>
<th>History</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>12/10/2017</td>
<td>• Left chained to the clothes line, entangling himself so that he was unable to find shade under the trees in the owner’s backyard.</td>
<td>• Active cooling by owner. No garden hose was used</td>
</tr>
<tr>
<td></td>
<td>• Full sun exposure until late afternoon when owners returned.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Max temperature at the Mareeba Airport 30.6$^\circ$C</td>
<td></td>
</tr>
<tr>
<td>13/10/2017</td>
<td><strong>Day 0</strong></td>
<td><strong>Day 3</strong></td>
</tr>
<tr>
<td></td>
<td>• Presented to TVS on the 13th of October 2017 for a possible burn or abrasion to his back.</td>
<td>• Sent home with a topical antibiotic lotion as acute moist dermatitis (hot spot) was suspected initially suspected.</td>
</tr>
<tr>
<td></td>
<td>• Serous like exudate along his back.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Hair was clipped and cleaned with dilute chlorhexidine.</td>
<td></td>
</tr>
<tr>
<td>16/10/2017</td>
<td><strong>Day 3</strong></td>
<td><strong>Day 25</strong></td>
</tr>
<tr>
<td></td>
<td>• Pain and sensitivity along the dorsum and the owner was unable to apply the topical lotion.</td>
<td>• Debridement and eschar removal.</td>
</tr>
<tr>
<td></td>
<td>• Revisit to TVS</td>
<td>• The wound area was then lavaged with 0.9% saline and cleaned with gauze swabs</td>
</tr>
<tr>
<td>07/11/2017</td>
<td><strong>Day 25</strong></td>
<td>• An experimental epoxy tigliane was applied to the entire wound area, and then left for secondary intention healing.</td>
</tr>
<tr>
<td></td>
<td>• Well adhered thick eschars, with necrotic tissue and purulent discharge along entire dorsum.</td>
<td>• Systemic antibiotics were ceased</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• No further topical applications of any other medication were applied.</td>
</tr>
<tr>
<td>12/01/2018</td>
<td><strong>Day 91</strong></td>
<td><strong>Day 91</strong></td>
</tr>
<tr>
<td></td>
<td>• Full wound resolution</td>
<td>• No further treatment</td>
</tr>
</tbody>
</table>

*From appearance of the first skin lesions
Figure 4. Case Progression for Bandit.

| Day 18* | Day 25* | Day 32* | Day 91* |

*From appearance of the first skin lesions

On Day 25 the extensive eschars were surgically removed. Data collected over the time lesions developed showed a rapid decrease in total wound surface area immediately after eschar removal as is seen in Figure 7.

Figure 5. Changes in total wound surface area and rate of closure in Case 1 following eschar removal at wound day 25.
Case 2. ‘Ellie’: spayed female mastiff tan in colour, 6 years of age.

Table 7. Presenting history and treatment for Ellie.

<table>
<thead>
<tr>
<th>Date</th>
<th>History</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| 06/04/17   | • Ellie picked up from the kennel by the owner’s son and left her tied to the ute in the car park of a local gym for a few hours.  
            | • Maximum temperature recorded at Mareeba airport was 28.4°C.  
            | • Over the next 5 days a small red raw area developed where the dog was scratching and this continued to develop into a large wound that was scabbed over and where further smaller spots were appearing | • Nil                              |
| 11/04/17 Day 5* | • Routine vaccinations and to check a rash along her back  
                       | • Necrotic areas along dorsum sloughing in places.  
                       | • No reported singed hair or abnormal odour coming from her coat.  
                       | • No previous history of medications or products applied to Ellie prior to the lesions appearing. | • Cephalexin and topical antibiotic lotion |
| 03/05/17 Day 27* | • Eschar developed and fully attached, with only small edges able to be removed while the dog was conscious. | • An experimental epoxy tigliane was applied to the entire wound area and left for secondary intention healing. |
| 10/05/17 Day 34* | • Eschar fully removed | • An experimental epoxy tigliane was applied to the entire wound area and left for secondary intention healing.  
                       | | • No further antibiotics or other topical medications used. |
| 30/8/2017 Day 146* | • Full wound resolution | • No further treatment |

*From appearance of first lesions
Figure 6. Case Progression - Ellie

*From appearance of first lesions

Figure 7. Case Progression for Bonnie.

*from appearance of the first skin lesions

# note the white discolouration of the hair on the edges of the scar tissue, not seen in previous cases 1 & 2.
Case 3. ‘Bonnie’: spayed female Boxer-cross tan in colour, 11-years of age.

Table 8. Presenting history and treatment for Bonnie

<table>
<thead>
<tr>
<th>Date</th>
<th>History</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>22/10/17</td>
<td>• Escaped 5 days previously from the back-yard by going under a barb wire fence, and disappeared for the day.</td>
<td>• Home treatment of lesions with weak betadine and honey.</td>
</tr>
<tr>
<td></td>
<td>• Maximum temperature recorded that day was 27.2°C at Mareeba Airport.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Lesions appeared on back, owners thought were from the barbed wire.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• No known drugs, medications or chemicals had been used on the dog in the last few weeks but they were not able to say if she had got in contact with something when she disappeared for the day.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Home treatment of lesions with weak betadine and honey.</td>
<td></td>
</tr>
<tr>
<td>27/10/17</td>
<td>• Presented to Central Veterinary Clinic in Mareeba. Lesions on back were not healing and becoming larger.</td>
<td>• Owners opted for general anaesthetic and surgical debridement of the necrotic tissue and eschars.</td>
</tr>
<tr>
<td>Day 5*</td>
<td>• Large areas of eschar along dorsum with purulent discharge and matted hair.</td>
<td>• Entire back clipped and cleaned with dilute malaseb.</td>
</tr>
<tr>
<td></td>
<td>• On attempt to remove the eschar, the dog was extremely sensitive and painful.</td>
<td>• All eschars debrided to fresh tissue.</td>
</tr>
<tr>
<td></td>
<td>• Numerous other open wounds were visible along the dorsum with no lesions found anywhere else and no lesions seen orally.</td>
<td>• Three sections of skin removed over the thoracic, thoraco-lumbar and lumbar regions of the dorsum for histology</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Cephalexin and meloxicam dispensed.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Home on dilute betadine and honey topical application</td>
</tr>
<tr>
<td>8/11/17</td>
<td>• Wounds healing extremely well</td>
<td>• Experimental epoxy tigliane applied to the deeper lesions near tail base.</td>
</tr>
<tr>
<td>Day 17*</td>
<td>• Lesions towards tail base that were the deepest still quite large but healing well</td>
<td>• No other systemic or topical medication used.</td>
</tr>
<tr>
<td>22/11/17</td>
<td>• Full wound resolution</td>
<td>• No further treatment needed</td>
</tr>
<tr>
<td>Day 31*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*From appearance of first lesions
Conclusions

To our knowledge, these three cases are the first documented in Australia with patient history, clinical presentation and, in one case wound histology, consistent with DTN. We felt it worthwhile to report these cases to (a) increase awareness of the potential occurrence of DTN amongst veterinarians and owners and (b) improve future approaches to treatment and management in everyday veterinary practice.

These cases along with other recorded occurrences in the United States highlight a real risk to dogs (and potentially other species) in areas of high ambient temperatures and sun exposure. Owners should be made aware of the possibility of this potent and severe thermal injury if dogs, and other animals, are left in high ambient temperatures in full sun. Similarly, veterinarians can use this history to aid in diagnosis of suspected DTN where other dorsal necrosis aetiologies have been ruled out.

While normal approaches to the care of burn wounds (systemic and physiological management of heat exhaustion, surgical debridement, pain relief and management of secondary infections) also underpin the treatment and management of DTN, there are some key take home messages specific to DTN from the cases reported here including:

› Removal and debridement of the developed eschar should occur as soon as possible and would appear to be a key treatment intervention to consider in reducing healing time in DTN cases. Once debridement and removal of the eschar occurred, the total wound area reduced dramatically. This was well demonstrated in Case 1 (see Figure 5).

› Following eschar removal in Cases 1 and 2, no systemic or topical anti-microbials or other medications were used. Judicious use of anti-microbials is becoming an important consideration in veterinary practice to aid in minimising the spread of drug resistant micro-organisms and the over use of anti-microbials when not needed.

› There was no evidence of infection or adverse healing by leaving the wounds open in all three cases. An important difference from the literature where cases generally had regular wound dressing and bandage changes.

› Biopsies of future cases would be useful to further define and determine aetiology, especially how DTN lesions differ histologically from other types of burns.

It is hoped that from these reported cases there will be an increased interest and reporting of future occurrences of DTN in Australia to build on the understanding, prevalence and management in dogs and other species.

References

Oestrogen overdose as an cause of mammary gland hyperplasia in young Donskoy tomcat

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²Veterinary practice U Stříbrné kočičky, Prague, Czech Republic
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Abstract

Case summary: This report describes the first reported case of oestrogen overdose in an entire male Donskoy cat. The cat was presented with mammary gland hyperplasia and was urine marking outside the litter-tray. Studies describing fibroepithelial mammary hyperplasia mention only progesterone as a causal agent, but in this instance high concentrations of oestradiol were observed together with clinical signs consistent with mammary gland hyperplasia. All the symptoms present in this tomcat were alleviated after withdrawal of exogenous oestrogen.

Relevance and novel information: To the best of our knowledge, this is the first report of oestrogen overdose in an entire tomcat. In addition, this report describes the mechanism of oestrogen overdose in a cat as a result of secondary drug consumption as a result of its owner undergoing transdermal hormone replacement therapy.

Introduction

Feline mammary fibroepithelial hyperplasia (FEH) is a benign, fibro-glandular proliferation of one or more mammary glands. It may occur in both the female and male cat. It is thought to be a progesterone-dependent phenomenon, i.e. after the administration of progestational agents, or after prolonged luteal phase of the oestrus cycle. Hyperplasia is characterized by a sudden onset within a short period of 2 to 5 weeks¹ and may occur up to 2 months after hormone administration (endogenous or exogenous).² Clinical signs can be observed in intact queens of any age, in pregnant females and in female and male cats receiving progestin treatment¹ e.g. megestrol acetate, medroxyprogesterone acetate (Depo-Promone) or proligestone (Covinan).³ A few reports mention the occurrence of FEH in males under treatment with the progestin delmadinone acetate (Darden or Tardak)² and anti-androgen progestin cyproterone acetate (Androcur).³ In both males the drugs were used to prevent urine spraying.²,³ Both oestrogen and progesterone receptors are present in FEH tissue. Progesterone receptors were detected in epithelial cells and stromal cells. Oestrogen receptors were detected in suprabasal or luminal epithelial cells.⁴ Binding of progesterone to progesterone receptors results in local secretion of somatotropin (STH) and insulin-like growth factor 1 (IGF-1) which results in growth of the mammary complex.⁵ Clinical signs in most cases are an increase in the volume of the mammary glands. This may be either isolated or multiple, in otherwise clinically healthy animals. The size of the enlarged glands is variable, ranging from 1.5 to 18 cm.¹ Progesterone concentrations in serum may be elevated.¹ The most accurate diagnostic tool is biopsy. Cytological differentiation between benign and malignant mammary lesions is difficult. The reliability of cytological differentiation is low. Cytological examination should be interpreted together with the symptoms and the sudden onset of the clinical signs.⁶ The progesterone receptor antagonist aglepristone (Alizine®,Virbac, France) can be used to medically manage FEH.⁷-⁹ Aglepristone competitively binds to the progesterone receptor without activating the hormone response-cascade in target tissues. This drug binds with a 9-fold affinity to progesterone receptor, and according to the manufacturer, its half -life in the organism is of 6 days duration, if administered once at a single dose of 20mg/kg or twice at 10mg/kg.² It is not licenced for use in cats and its use is ‘off label’. Cabergoline (Galastop®, Cave Santé Animale, France) is a dopaminergic agonist that produces a selective and long-lasting inhibitory effect on prolactin secretion, which in turn may be helpful to supress lactation.¹ Its use was described in association with castration in a tomcat.⁶ The first author has used cabergoline as a successful treatment for mammary hyperplasia in one queen. Prognosis for uncomplicated feline mammary fibroepithelial hyperplasia is good. If there is mastitis or ulceration, the prognosis may be more guarded, especially if the condition has been left untreated for a long period. Some patients become anaemic and require blood transfusion therapy. Spontaneous regression of the enlarged mammary glands after removal of the
progesterone may occur, and this can be facilitated by ovariectomy in cats which are sexually entire and in which normal cycling is the cause of the problem developing. Exogenous oestrogens can be administered transdermally in human patients due to the lipid soluble nature of steroid hormones, and both oestradiol and ethinyloestradiol are used for hormone replacement therapy in post-menopausal women. Both hormones moieties are lipophilic and together with suitable auxiliaries contained in transdermal sprays or ointments can easily penetrate the skin to achieve clinically effective concentrations in the systemic bloodstream.10-12

After dermal penetration, steady state concentrations of exogenous oestrogen occurs after 7 days of regular application. If an animal has been regularly in contact with a preparation applied to owner’s skin, the absorption through contact or licking is likely and the steady state of oestrogen levels can occur in the in-contact animal as well. This warning is mentioned in the summary of product characteristics (the official document published by state authority), unfortunately it is not in the product information label added to these products.10

Oestradiol is the most active oestrogen, responsible for normal growth and development of the female reproductive organs and development and maintenance of secondary female sex characteristics. The skeletal system is influenced by oestrogens: epiphyseal closure is accelerated; calcium deposition and bone formation is increased. Oestrogens have a slight anabolic effect and increase sodium and water retention. There is strong influence on gonadotropin release by oestrogens and androgen secretion is inhibited in presence of higher oestrogen levels.13

Case description
A 14-month-old intact male Donskoy cat (a hairless breed of Russian origin; 3.2 kg) was presented with enlargement of all mammary glands. This condition developed, apparently acutely, 2 days prior to presentation.

He was bought as a 3-month-old kitten from a breeder. At the age of 11 months he started urine marking at home and scratching the walls and furniture around the window. Urine was examined elsewhere, with no abnormalities being detected, but the tomcat underwent a 3 week course of amoxicillin-clavulanate, which did not change the urine marking behaviour. The situation deteriorated further when the owner brought home a new girlfriend. The cat started to urinate in front of the owner and she started to beat the tomcat and prevent it from moving around the flat.

The owner attended the practice of the first author when the cat was 13 months-old because he was urinating outside the litter tray, and for clinical signs of upper respiratory tract infection of one month duration. At the first visit, urine was examined and no abnormalities were found on Dipstick examination and sediment cytology and urine culture was negative. Ultrasound examination of the abdomen was unremarkable; FeLV/FIV Snap test was negative. Clinical examination was unremarkable except for abundant ear wax with low numbers of Malassezia yeasts and mild signs of gingivitis, rhinitis and conjunctivitis. Smears from the conjunctiva, nose and throat were PCR positive for Mycoplasma arginini and Bordetella bronchiseptica. The cat was successfully treated with pradofloxacin for 3 weeks. Feliway diffuser was used and the cat’s environment enriched and the tomcat permitted to scratch around the window provided, but according to the owner, none of these measures helped prevent the tomcat from urinating outside the toilet. Castration or Suprelorin implant weren’t possible because the owner wanted to use this tomcat in a breeding programme as soon as possible.

Enlargement of the mammary glands occurred 25 days after the first visit (Fig. 1), and the testes of the tomcat were smaller than the other tomcats of the same age. Interestingly, the owner mentioned that they looked smaller compared to the photos from autumn (Fig. 2). Further history taking revealed that the cat’s owner was undergoing sex reassignment surgery, necessitating the use of many female hormones and testosterone antagonists. The owner was not aware of the tomcat eating any of his/her medication, but had lost one Androcur tablet one month previously. The owner, who looked like a woman, mentioned that she had also been using an oestrogen spray since November 2016 (Lenzetto 1.53 mg/ dose transdermal spray) and used it several times a day, without washing her hands subsequently, and that she petted the cat frequently.

Fine needle aspirate biopsy from the hyperplastic mammary gland did not show any cells with malignant features; a few foamy macrophages, adipocytes and rare neutrophils were observed together with eosinophilic background. Blood test results are presented in Table 1. Oestradiol levels in this intact tomcat were two-times higher than the upper reference interval of the laboratory for females (oestradiol radioimmunoassay (RIA) with

![Figure 1. Mammary glands of intact male Donskoy tomcat. (A) Mammary hyperplasia on the first day of examination. (B) Mammary hyperplasia largely resolved one week after oestrogen exposure withdrawal.](image-url)
extraction 119 pg/mL), whilst the progesterone level was low (progesterone <0.2 ng/mL). A strong suspicion of oestrogen overdose in the tomcat was made according to this data. The tomcat was referred to the hospital for abdominal ultrasound, in an attempt to rule in or out the presence if ectopic ovarian tissue, adrenal masses and other pathology which might be responsible for female hormone production. The examination was unremarkable except for bilaterally enlarged adrenal glands (Fig. 3) and ultrasonographic findings in the mammary glands were consistent with benign mammary gland hyperplasia.

The treatment was withdrawal of the exogenous source of oestrogens. The owner was advised to wash hands and apply the spray to an area inaccessible to the cat. After one week clinical examination of the tomcat revealed a reduction in the size of mammary glands of approximately 80%! The owner also reported that the tomcat stopped urinating outside the toilet almost immediately after the oestrogen withdrawal.

Discussion

Since the owner was receiving strong female hormones and anti-testosterone medication it was highly likely, that the tomcat could be overdosed with female hormones. The owner mentioned that she missed one Androcur tablet 1-2 months ago. Tomcats can develop FEH after medication with cyproterone acetate approximately 2 months after administration.³

Because FEH is typically caused by progesterone excess, its serum concentration was measured. In our tomcat the progesterone level was low. This might be as a result of progesterone not being the causal agent or because the assays are specific for progesterone hormone and do not detect exogenous progestin molecules.¹

To the best of the authors’ knowledge, there is no publication describing FEH as a consequence of high oestrogen concentrations, such as in this tomcat. But according to the literature, oestrogen receptors are present in mammary gland cells⁴, thus hyperplasia might be caused by higher levels of oestrogen too. Similar clinical signs were observed in dogs with hyperoestrogenism syndrome referable to Sertoli cell tumour of the testis, often with oestrogen myelotoxicity, severe bone marrow damage with non-regenerative anaemia and pancytopaenia.¹⁴ In this tomcat, no anaemia was present despite long time exposure to the exogenous oestrogen and the well-known sensitivity of cats to oestrogen overdose. The drop in red blood cell numbers was small within one month; maybe mild bone marrow toxicity was actually present.

Congenital adrenal hyperplasia secondary to 11β-hydroxylase deficiency was described in a domestic cat, in which penis and empty scrotum was present together with ovaries and a uterus.¹⁵ Presence of ovaries and uterus was not confirmed in this tomcat on ultrasound, so that congenital adrenal hyperplasia is less likely to be a cause in this tomcat.

Adrenal hyperplasia is mentioned in connection with Cushing’s disease in cats.¹⁶ But no other findings consistent with hyperadrenocorticism including clinical signs except adrenal hyperplasia were observed in this tomcat.

It is thought that the FEH being a progesterone-associated disorder, the therapeutic approach should focus on the removal of the progesterone influences in order to correct the changes in the mammary glands. Thus, discontinuation of any ongoing hormone therapy is mandatory.¹ According to this information the basic approach in this case was withdrawal of possible oestrogen application which was the suspected aetiology in this patient. If this approach...
was not successful, aglepristone would be used according to already published recommendations. 7-9

The fact that the inappropriate urination stopped with withdrawal of the oestrogen preparation might be because the higher oestrogen level was the cause of the marking behaviour as is the case in queens who sometimes mark using their urine during oestrus. 17

Conclusions

This might be first documented case of exogenous oestrogen causing FEH. Oestrogen overdose in cats might not be rare at all, because more and more owners are undergoing oestrogen treatment with the use of skin sprays, unaware that this medication could be easily transferred to the animal’s skin. Therefore, in odd cases good history taking is extremely important. In our tomcat, careful use of the medication by the owner was sufficient to resolve the mammary hyperplasia and inappropriate urination.

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Table 1. Blood work of the presented tomcat.
Acute renal injury in a cat called Asterix

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Is it just me or do acute renal cases just creep up on you?

A cat that has been sick a couple of times, quiet, not eating for 36 hours – all very vague and not over-exciting. Except that ‘Asterix’ had a round very painful structure mid abdomen. And mum had no money.

My nurse waved the blood results in one hand with a bottle of green dream in the other: creatinine >1000, urea >65, phosphate 3.95 – clip the leg up, curtains!

But whoah, not so fast. Let’s check it out; ultrasound showed one huge right kidney and a smaller left kidney. But no pelvic dilatation on the large one! I would have expected both to be enlarged with lily poisoning, and unilateral ureteric/pelvic dilatation with obstruction. However, the left kidney was probably defunct (22mm long axis, and with indistinct architecture) so maybe unable to expand with toxicity? Perhaps an obstruction had cleared but a painful swollen kidney had resulted? – can this happen?

In any case we had an acute renal injury which could be either treated or not. No money, guarded prognosis, heaven beckons. But I love potential euthanasia cases – sounds awful, but where death is in the sights, the options open up and the challenge is made. So how might we manage the case? The ‘book’ is very clear – intense fluid management for a few days monitoring input and output, maintaining hydration, attending to nutritional needs. But it’s Friday – so I can’t even do a charity no-fee job with the weekend looming. And most of you will not have the privilege of that option any time of the week. (I am my own boss!)

However an o tube is cheap, the client is willing. Asterix was anaesthetised with IV propofol after 0.25 mL buprenorphine so we ran the drip through the day and disconnected him in the evening to send home. The post-op tube-check X-ray showed some mineralised material in the right kidney but further investigations were precluded by a lack of funds.

Armed with the only 2 cans of liquid convalescent diet on the shelf (recent supply issues) we gave instructions on how to liquidise and double-sieve a pate-style moist food and attempted to demonstrate tube-feeding.

Asterix thought differently…..

Bloodied, we squirted 100mg of gabapentin liquid down the nozzle and called them back 2 hours later to complete our demo. The plan was to do the same at home, sedating as required but reducing the dose down as soon as he settled, aware that renal clearance of gabapentin might be delayed in this cat. Oral buprenorphine completed the protocol.

The clients were devoted parents, and after 48 hours giving 20 -30mL per waking hour the gabapentin was superfluous and he was eating some food voluntarily. The next examination was for tube removal 7 days later when bloods confirmed resolution of azotaemia. Attitude precluded cystocentesis for specific gravity and a katkor (non-absorbent cat litter) sample was requested, result SG: 1.016

So what lessons are there to be learned here?

Figure 2. Lateral radiograph showing placing of O tube and disparity in kidney size.
Personally I’m frustrated that there is so often deemed to be the ‘correct way’ for treating a specific condition – and nothing else is ‘permissible’. And too often I think the fear of litigation discourages folk from trying anything that is not standard practice. With a compliant cat, and open wallet I will run with the top standard-of-care formulated by others with more in-depth knowledge and understanding. But if there are no funds available for this, why not be inventive? – In this case there were no potential welfare issues that would not also have existed with a standard fluid regime, and no guarantee of success – but ditto for intravenous therapy and monitoring. We know that the degree of azotaemia is not predictive of outcome, and our ‘fudge’ may have less chance of success than a hospitalised regime. If one’s best efforts fail, at least one has tried, and the client will appreciate your efforts.

So in conclusion, no claims that this approach is in any way unique or clever – oesophagostomy tubes are a recognised means of supporting longer-term CRD cases and I wont be the first to use one in the early stages of management. But it wasn’t on my radar before this case, and I just wanted to share the concept with anyone coming across the same scenario.

Introduction

Pain relief in pets suffering from burns can be a major challenge, in particular progressive thermal burns involving the epidermis, subcutaneous and underlying muscle layers. It has been reported that third degree burns involving the formation of an eschar of the epidermis and dermis results in the absence of pain, while partial or second degree burns are the most painful.1,2 Analgesia is the most important welfare consideration in these patients both in the initial and subsequent presentations until surgical treatment to remove the eschar. Inadequate analgesia may result in maladaptive pain responses such as patient related aggression causing unnecessary suffering.

Case

Charlie is a 3-year-old Cavalier King Charles cross-bred dog presented to Sydney Animal Hospitals Norwest after the owner reported he had been stuck inside a house fire for about an hour. She had returned home to a burnt house and he was inside. Charlie was unwilling to move and was quiet. Before the fire he was eating, defecating, urinating and drinking normally.

On examination, Charlie was bright, alert and responsive with normal chest auscultation (HR-140bpm) and clear breath sounds. He was covered in ash but had a normal respiratory rate (28bpm) without dyspnoea. No obvious burns were seen on his body. He did not cough and was amenable to a thorough clinical exam.

He was not painful on examination and SPO₂ at room air was at 95%. He was placed in an oxygen cage due to concerns about inhalational pneumonitis, and he was maintained on intravenous Hartmann’s solution at twice maintenance throughout the day. Later that night it was noted that the left ventrolateral thorax had a pink burn approximately 4 x 3cm in diameter and did not appear to
be swollen and painful. Oral meloxicam was prescribed at 0.2 mg/kg PO SID and Flamazine® was applied to the affected area. The owners elected to take him home but were advised to take him to an after-hours referral hospital if he worsened.

Charlie re-presented the following day having spent the night at the after-hours referral centre. On presentation, he had a quiet demeanour and poor oxygen saturation 81%. He had mild inspiratory dyspnoea and normal lung sounds were present with no obvious crackles or wheezes. His bloodwork showed a mild lymphocytosis and he was placed back in an oxygen cage and conscious chest radiographs revealed a diffuse interstitial pattern. At this stage he was tentatively diagnosed with delayed smoke irritation with early signs of upper respiratory tract infection or pneumonia. He was not tolerant of oral antibiotics and was given Cefovecin 8 mg/kg SC once off. Charlie was still relatively pain free and tolerated repeated physical examination. He was still eating well when fed by the owner and his burn wound was not larger. His pain relief at this point was still Meloxicam oral and once again he was sent home.

Four days later (six days after initial presentation), Charlie re-presented due to a 12 hour period of inappetence, was shaking and reportedly bit the owner when she attempted to apply the Flamazine® on the wound. He had an elevated temperature of (39.8°C) and was not amenable to physical examination. The wound was now 4 x 5 cm in diameter and appeared mottled and darkened (Figure 1). This area also appeared swollen and extremely painful to touch. When touched anywhere he would quickly snap around and bite. His SPO₂ remained stable at 94-97% without oxygenation and no dyspnoea or abnormal lung sounds were detected.

Charlie was started on methadone 0.3mg/kg IV q4h and a 25µg fentanyl patch was applied to his left hind leg. Gabapentin was discussed with the owners but given the difficulty of oral administration of medication, it was not prescribed. He improved clinically and began eating again and the owners elected to take him home. He developed some mild diarrhoea and meloxicam was stopped temporarily.

It was recommended that surgery be performed to remove the large area of necrotic eschar now developing; however, the following day showed a further increase in the size of the eschar. He was now inappetent, screaming when touched, constantly looking at his wound and becoming aggressive. Charlie was re-admitted into hospital and started a fentanyl lignocaine ketamine infusion (FLK) (F: 1-2mcg/kg/hr; L 1mg/kg/h, K 0.12mg/kg/h) in addition to his fentanyl patch. Throughout the next few days, the eschar darkened in colour and extended almost 6cm in diameter on the left side of Charlie (Figure 2). He was occasionally interested in boiled chicken in hospital which was an improvement from the preceding 24 hours.

Surgery for the removal of the eschar was performed 12 days after initial presentation. The eschar was now approximately 7 cm in diameter and extended from the left axilla from around the point of the elbow occupying the entire left thorax (18% of body). 22mg/kg IV Cephazolin was given 30 minutes pre-op and induction with Alfaxan 3mg/kg IV given to effect, and maintained on 1.5%-2% Isoflurane. Charlie’s heart rate was stable at 100 bpm but as soon as the wound was clipped and palpated his plane of anaesthesia became lighter as evidenced through movement, HR increasing to 200bpm and RR to 60. A ring block was performed around the proposed incision site of the eschar given it was not possible to penetrate any needle through the tissue to perform intercostal nerve blocks cranial or caudal to the eschar. A large flap of eschar including necrotic dermis and subcutis was removed en bloc. The open wound was bandaged with a wet to dry dressing using sterile saline.

Charlie recovered uneventfully and was continued on a FLK infusion. His fentanyl patch was replaced. After recovery, he ate his dinner in the afternoon and continued on meloxicam. Throughout the next few days his demeanour and appetite improved although he was still aggressive to touch. We began a course of gabapentin 5mg/kg PO BID to manage his pain along with meloxicam, fentanyl patch and FLK. At the time of this article, Charlie was still resenting touching of the contracting wound during examination.

Figure 1. Wound 6 days after initial presentation.

Figure 2. Wound 10 days after initial presentation.
Points

1. Despite formation of an eschar, inadequate analgesia when burns progress may still result in transduction and transmission of nociception. In Charlie’s case, an eschar (third degree burn) involving at least 18% of his body eventuated, but he still showed marked signs of pain.

2. Hospitalisation of burn victims should be the standard of care regardless of initial presentation. Earlier hospitalisation and aggressive multi-modal analgesia of Charlie may have reduced wind up and should incorporate all of NSAIDs, Opioids, Gabapentin and local analgesia where appropriate. Eschars may take up to a week to be visible and it is important that appropriate analgesia be provided until then.

3. In cases where hospitalisation is not possible or the owner is unwilling (as was Charlie on acute presentation) a pain scale sheet should be provided to the owners to ensure to allow the client to bring in the patient for pain intervention as soon as possible. Many versions of pain scaling exist, including validated scales for assessing acute pain in companion animals in hospital such as the Glasgow Composite Pain Scale, Melbourne Pain Scale and the Colorado Acute Pain Scale. For outpatients, the client should use a pain scale which is suitable and easy for owners to do at home such as the University of Pennsylvania Canine Brief Pain Inventory (Canine BPI) which owners can download online (Fig 3; www.CanineBPI.com).

4. Charlie continues to demonstrate allodynia (pain perceived from a non-noxious stimulus). This is most likely due to maladaptive pain even after removal of the eschar. Although ketamine was used to reduce dorsal horn wind up in this case, there is the potential for maladaptive pain to be lifelong. An ongoing pain plan for Charlie should be aimed at reducing the modulation of refractory pain involving gabapentin, tricyclic anti-depressants, opioids and NSAIDs and benzodiazepine analogues.

Follow up

April 2019: A further surgery was required to achieve primary closure of the wound as contraction appeared to have ceased, but the dog is now doing very well and is pain-free.

References:


Winks and Blinks

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Introduction

John Morris is a neurologist who has turned his interest in watching human patients into a passion for watching birds. He was a pioneer of the subspecialty of movement disorders in neurology in Australia, is an excellent teacher and one of his publications ‘The neurological short case’ is still used by physician trainees. He also made video recordings of movement disorders and made them available to neurologists and trainees throughout Australia (for free). He was always interested in blinking. For instance, people with extrapyramidal disorders may blink in order to initiate voluntary eye movements.

11 years ago, I was at the busiest time in my life as a neurologist at Westmead hospital. On the way to work one morning, I went to see a colleague at his office about the results of a recent PSA test. He examined me and told me his findings. What he said caused me to drive home. I never worked again. After surgery, I started to go on walks in bush in the valley below my house. For the first time I found myself wanting to know the names of the birds I heard, and sometimes saw. So I bought a super zoom camera and began to photograph them, rushing home to identify them from a field guide. The camera turned distant spots of blurred colour into the detail of feather filaments, beaks and, most attractive of all, eyes. I could imagine what van Looewenhoek must have felt when he first put a drop of pond water under his microscope and saw the details of teeming life.

Many of my pictures of birds were spoiled by blinks - the beauty of their eyes obscured by an opaque film. Often the bird moved just as I was taking the picture. Sometimes, the bird did something interesting, spur winged plovers for example have a habit of giving a little nervous jump. Stills missed this. I decided to go back to videoing what I saw. For much of my career I had videoed my patients with movement disorders. It seemed only natural to return to the medium with which I was most comfortable.

Now an even more exciting world opened up to me. Many birds blinked almost every time they turned their heads. Why did they do this? Using the video frames as a crude measure of time, I began to compare the duration of blinks with the duration of the head turns. There was a strong correlation. But why? When we move our eyes quickly, our brain renders us momentarily blind. Retinal images which would be blurred by eye movement never reach our consciousness. Were birds, which move their heads as we move our eyes, achieving the same end by blinking?

While chewing on these thoughts, I began to notice other unusual things. Often, during a blink, the bird’s eyes would wobble alarmingly for a second or so. I only saw this with high speed videoing. Blinks also varied in their speed and duration. Nictitating membrane blinks were usually rapid and brief. In parrots, owls and pigeons the upper eyelids joined in. Lower lid blinks were slow and prolonged and occurred when the bird was drowsy or during preening. Some birds blinked every time they pecked.

Once I began to get a feel for how birds blinked, I began to wonder how this compared with other creatures. Most fish don’t blink. Why not? Crocodiles are the closest living relatives to birds, so I began to visit zoos. While there I was astonished to find that meerkats blink with their nictitating membranes every time they turn their heads - just like birds.

And so a passing interest which started while rehabilitating myself from surgery, became a pursuit which gave me enormous pleasure. I was surprised to find that there has been little interest in blinking for more than a century. Moreover, there was little scope for publishing a descriptive classification of blinking. This type of study was all the rage in the century before the last one.

So I have put together my findings on a website which I hope you will enjoy visiting. And if you have thoughts or observations you would like to pass on to me, I shall be very pleased.
Figure 1. Elevation of the lower eyelid in drowsiness in a female Mallard (Anas platyrhynchos).

Figure 2. White-lipped tree frog. At 160ms after the onset of the blink, the eyeballs are retracting into the head. This is most obvious in the left eye which is seen in profile. There is narrowing of the palpebral fissure, which is due to the retraction of the globes. An opaque membrane has appeared above the lower eyelid, travelling vertically in an upward direction. This is often called a nictitating membrane, but it is an extension of the lower eyelid which is folded underneath the eyelid when the eye is fully open.

Figure 3. Nictitating membrane blink on head turn in an ostrich (Struthio camelus). The blink lasts 280ms.

Figure 4. Upper lid and nictitating membrane blink in a greater masked owl (Tyto novaehollandiae).

Figure 5. Nictitating membrane blink on head turn in a meerkat with no associated closure of the eyelids.
Feline ureteral transitional cell carcinoma and concurrent alimentary small cell lymphoma—complications of treatment and management

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Abstract
A ureteral transitional cell carcinoma was diagnosed in an 11-year-old male neutered domestic shorthair cat with concurrent alimentary small cell lymphoma presented with hydronephrosis. The uncommon location of the transitional cell carcinoma resulted in a delayed recognition of a neoplastic process as the cause of the ureteral obstruction though the location was identified by ultrasound and computed topography antegrade pyelogram. The concurrent existence of two kinds of neoplasia, transitional cell carcinoma and alimentary small cell lymphoma, may have contributed to the surgical complication and complicated the long-term management of this case. After surgical correction of the ureteral obstruction, the decision was made to treat his alimentary small cell lymphoma with close observation for recurrence of urinary tract signs.

Introduction
The common locations of transitional cell carcinoma (TCC) are in the urinary bladder and the urethra. It is most common in older dogs but rarely found in cats (Wilson 2007). Ureter is an uncommon site for TCC in cats. To the author's knowledge, feline ureteral TCC has been previously reported in one brief case report (Anderson 1986) and in one case report of an abdominal mass circumflexing both ureters (Cohen 2012). In the human literature, urothelial cell carcinoma of the upper urinary tract (UUT-UCC, including TCC of the ureter and kidney) is also uncommon and accounts for only 5-10% of urothelial carcinoma (Olgac 2004; Margulis 2009). The distal ureter appears to be more frequently affected, presumably due to greater stasis (Browne 2005; Vikram 2009). From the literature, we know that UUT-UCC behaves differently from bladder cancers: 60% of UUT-UCC are invasive at diagnosis compared with only 15% of bladder tumours in humans (Babjuk 2008; Jemal 2009). In cats, TCC of the urinary bladder appears to be a rare and aggressive disease that frequently develops at sites away from the trigone (Wilson 2007).

There is limited data regarding treatment and prognosis of feline TCC. The response of feline urinary tract TCC to chemotherapy is not well documented (Knapp 2001). A retrospective study of 20 cases of feline TCC of the urinary bladder showed a median survival time of 261 days with or without treatment and the median progression-free interval was 89 days (Wilson 2007). There were insufficient numbers of cats to associate a significant survival advantage to any one treatment or to evaluate the effect of surgery on survival. One cat in this study had concurrent alimentary small cell lymphoma and was treated with chlorambucil and prednisone before and after diagnosis of TCC. The cat was treated surgically for its TCC with incomplete surgical margin. Tumour recurrence was detected 79 days after surgery, and it had an overall survival time of 261 days. Treatment of another cat with urethral TCC via surgery and radiation therapy has been reported (Takagi 2005). A retrospective study of 17 cats with TCC of the urinary bladder, which were treated with meloxicam, had a median survival time of 311 days (Bommer 2012). The study suggested that non-steroidal anti-inflammatory drugs (NSAIDs) such as piroxicam and meloxicam might have a role to play in the palliative management of TCC in the urinary bladder of the cat.

Case report
An 11-year-old male neutered domestic shorthair cat was referred for investigation of his hydronephrosis and suspected ureteral obstruction. He presented to the referring veterinarian with inappetence and vomiting two days before being referred to the specialist hospital. Serum biochemistry showed mild elevation in urea and creatinine (table 1). He had a history of diffuse enteropathy with suspected reactive mesenteric lymph nodes observed sonographically four months prior to the presentation of his current illness. Treatment trial with vitamin B injection,
1. Royal Canine Low Fat Diet
2. Carbimazole, Amdipharm

metronidazole and diet alternation to a Low Fat Diet seemed to relieve the gastrointestinal (GI) signs including vomiting and decreased appetite. No further investigation was pursued at that time. He also had hyperthyroidism, which has been well controlled by carbimazole.

On presentation, he had persistent azotaemia (Table 1) and a firm left kidney on palpation. Abdominal ultrasound showed left renomegaly (47 mm long; normal range 30-42 mm) and bilateral pyelectasia (left renal pelvis measuring 6.9 mm on the sagittal plane and 4.4 mm on the transverse plane; right 4.6 mm on the sagittal plane and 4.8 mm on the transverse plane) (Fig. 1). Left ureter was mildly dilated (3.7 mm wide just distal to the hilus) and could be followed to the caudal abdomen where it tapered abruptly and could no longer be seen on ultrasound. The gastric wall (4 mm; normal <3.7 mm) and duodenal wall (3.2 mm; normal <2.5 mm) were mildly thickened with normal wall layering. Lymph nodes were within normal limits.

Computed topography (CT) excretory urography was unable to confirm left ureteral obstruction. The study showed failure of delivering contrast into the left renal pelvis and distally despite good opacification of the left renal parenchyma (Fig. 2a). A CT antegrade pyelography was then performed and showed good opacification of the left renal pelvis and left ureter. The ureter was dilated and tortuous, and tapered at the level of the trigone of the urinary bladder and became invisible (Fig. 2b), which was consistent with a distal left ureter obstruction.

No intramural or intraluminal abnormalities were identified by either ultrasound or CT but the location of obstruction was consistent in all studies. A distal left ureteral obstruction was strongly suggested by the antegrade pyelogram. The renal function of the left kidney could not be accurately assessed prior to correction of the obstruction.

A blockage was identified approximately 2 cm cranial to the entry of the left ureter into the urinary bladder wall during exploratory laparotomy. A partial ureterectomy of the segment distal to and including the obstruction site was performed, which was followed by ureteroneocystostomy. A psoas cystopexy was performed to accommodate the shortened left ureter.

During the surgery, the left ureter was observed to be dilated with several small rounded black soft materials in the luminal side of ureteral wall and with further focal dilatation around the materials. The right kidney and ureter were grossly normal. A ventral midline cystotomy was performed. The mucosa of the urinary bladder appeared oedematous but otherwise normal. The blockage was identified by antegrade flushing of the left ureter through an incision at the junction of the ureter and the renal pelvis using a small tomcat urinary catheter and observing the mild expansion of ureter proximal to the obstruction. The left ureter was amputated from the site of blockage and the amputated segment was sent for histopathology.
Full thickness biopsies of stomach, duodenum, jejunum and ileum were obtained for histopathology examination. The defects were closed by simple continuous suture of 3-0 Monosyn®. An oesophagostomy tube was placed at the end of surgery.

The histopathology analysis of the GI biopsies suggested a diagnosis of small cell, low grade, epitheliotropic lymphoma (Fig. 3). The histopathology of the ureter segment showed multifocal to diffuse dysplasia and atypia of the urothelium with atypical epithelial cells present within the submucosa, consistent with TCC (Fig. 4a & 4b). The proximal third of the ureteral segment contains an intense neutrophilic infiltrate (Fig. 4c), areas of chromatin streaking, fibrin exudation and multifocal small haemorrhages (Fig. 4d). The inflammation gradually decreased distally and remained most intense in the superficial submucosa but the epithelial changes with the presence of atypical cells remained evident.

The patient recovered uneventfully from general anaesthesia (Day 0). Post-operative treatment included IV

3. 3-0 Moneys, Braun
4. Oesophagostomy tube, Unomedical
fluid\textsuperscript{5} containing 20 mmol/L KCl\textsuperscript{6} at twice maintenance rate, fentanyl\textsuperscript{7} CRI (2-6 µg/kg/h) (replaced by buprenorphine\textsuperscript{8} 12 µg/kg sc tid since Day2), ranitidine\textsuperscript{9} (2 mg/kg slow iv bid), amoxicillin-clavulanate\textsuperscript{10} (12.5 mg/kg po bid) and carbimazole (1 mg/kg po bid). An oesophagostomy tube was used for nutritional management (one 156 g tin canned food\textsuperscript{11} mixed with 50 mL water, given 50 mL q4h).

The patient was unable to urinate without a urinary catheter in place post-operatively. His urine output was adequate and his azotaemia reduced when a catheter was in place (Table 1) but stranguria/dysuria recurred after removal of urinary catheter at Day 5. The catheter was difficult to advance beyond the bladder neck. Ultrasonography revealed free abdominal fluid. Positive contrast cystogram and retrograde urethrogram showed abnormal urinary bladder neck and persistent filling defect at the junction of the urethra and the urinary bladder (Fig. 5). The post-operative anaemia (PCV/TP 15/65) became worse (PCV/TP 12/62) and the patient developed hypothermia (33.9°C). A total of 50 mL type A blood was transfused, and antibiotic therapy with cephazolin was commenced.

A second exploratory laparotomy was performed to evaluate the urinary bladder neck and to relieve the obstruction. The proximal urethra appeared to be kinked when the urinary bladder was full. The urinary bladder was repositioned and the left ureter was reinserted followed by a cystopexy and a nephrocystopexy to release any potential tension placed on the urinary bladder. A cystostomy tube was placed.

Dehiscence of ileac and duodenal biopsy sites was repaired using 3-0 Monoplus and strengthened with serosal patches. The site of the gastric biopsy was also dehiscent and closed with 3-0 Monoplus in a double layer inverting pattern. Two Jackson pratt drains were placed on the right and left sides of the abdomen and the abdomen was lavaged with warm saline prior to closure.

Analysis of the abdominal effusion collected during surgery confirmed septic peritonitis, and \textit{Escherichia coli}, \textit{Enterococcus faecalis}, another \textit{Enterococcus} sp.

\begin{table}[h]
\centering
\begin{tabular}{|c|c|}
\hline
Drug & Manufacturer/Brand Name \\
\hline
Hartmann’s & Fresenius Kabi \\
KCl 20mEq & Astra Zenica \\
Fentanyl & Hospira \\
Buprenorphine & Reckitt Benckiser \\
Ranitidine & Claxo Smith Kline \\
Clavulox & Pfizer \\
Hill’s a/d & \\
\hline
\end{tabular}
\caption{Drugs and their manufacturers/brands.}
\end{table}
and a Klebsiella sp. were cultured. Antibiotic therapy with ticarcillin-clavulanate12 (40 mg/kg slow IV qid) and enrofloxacin13 (5 mg/kg sc qid) was commenced along with dolasetron14 (0.62 mg/kg sc qid), fentanyl CRI (6 µg/kg/h) and ketamine15 (0.3 mg/kg/h) for anti-emesis and analgesia. His hyperthyroidism was kept under control by carbimazole (1 mg/kg po bid). IV fluid and oesophagostomy tube feeding (as previous plan) were administered post-operatively.

Abdominal ultrasound performed at D26 revealed a mild increase in serum creatinine with normal serum urea (Table 1). His anaemia had largely resolved but persistence of left pyelectasia. The left renal pelvis measured 5.2 mm in the transverse plane and 6.2 mm in the sagittal plane. The size of both kidneys and the diameter of the left ureter (Fig. 6) were similar to the previous examination. His urea and creatinine values remained within reference ranges 24 hours after discontinuing IV fluids (Table 1). His haematocrit slowly increased though he remained anaemic (PCV/TP 18/66). The patient was discharged at D28 with PCV/TP 26/69. The owners were pleased with his progress at home, although he remained unwilling to eat voluntarily. The feeding tube could then be removed once his appetite had returned to normal. His appetite and drinking voluntarily.

We encountered several problems in the management of this case. Long-term management of his alimentary small cell lymphoma with prednisolone and chlorambucil was recommended to commence two to three weeks after surgery. The median survival times for alimentary small cell lymphoma were 18 months with good quality of life under proper management. However, the ureteral TCC carried a poor prognosis as it might lead to recurrence of urinary tract signs in the near future but use of NSAIDs for palliative treatment was problematic with concurrent prednisolone administration.

The patient had a recheck examination eight days after discharge. The owners were pleased with his progress at home, although he remained unwilling to eat voluntarily. He had gained 200 g body weight and his ventral midline surgical wound was healing well. Renal analytes at the time showed a mild increase in serum creatinine with normal serum urea (Table 1). His anaemia had largely resolved (PCV/TP 26/69).

Table 1. Repeated serum biochemistry analyses of renal values over time.

<table>
<thead>
<tr>
<th>D-2</th>
<th>D00</th>
<th>Reference</th>
<th>D01</th>
<th>D03</th>
<th>D04</th>
<th>D05</th>
<th>D07</th>
<th>D08</th>
<th>D09</th>
<th>D10</th>
<th>D11</th>
<th>D14</th>
<th>D15</th>
<th>D16</th>
<th>D24</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea (mmo/L)</td>
<td>12.3</td>
<td></td>
<td>17.3</td>
<td>10.2</td>
<td></td>
<td>23.7</td>
<td>7.7</td>
<td>16</td>
<td>10.5</td>
<td>16.2</td>
<td>5.9</td>
<td>7.8</td>
<td>8</td>
<td>9.3</td>
<td></td>
<td>5.4-10.7</td>
</tr>
<tr>
<td>Crea (µmol/L)</td>
<td>311</td>
<td>272</td>
<td>71-212</td>
<td>330</td>
<td>210</td>
<td>310</td>
<td>130</td>
<td>210</td>
<td>130</td>
<td>180</td>
<td>140</td>
<td>180</td>
<td>160</td>
<td>180</td>
<td></td>
<td>70-160</td>
</tr>
<tr>
<td>Urea (mg/dL)</td>
<td></td>
<td>16.3</td>
<td></td>
<td>7.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>3.6-10.7</td>
<td></td>
</tr>
<tr>
<td>Crea (mg/dL)</td>
<td>324</td>
<td></td>
<td>193</td>
<td></td>
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<td></td>
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<td>27-186</td>
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</tr>
</tbody>
</table>

The owners were instructed to reduce the amount of food given through the oesophagostomy tube to encourage the patient to eat by himself. The feeding tube could then be removed once his appetite had returned to normal. His antibiotic courses would continue for another two weeks, after which time the chemotherapy for his alimentary small cell lymphoma would be commenced and managed by the referring veterinarian.

Discussion

Ureteral TCCs are difficult to diagnose. Occasionally ultrasound may be able to visualize the obstruction. Excretory urography may not provide suitable images. In the absence of sonographical visualization of the

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12. Timentin, Glaxo Smith Kline
13. Enrofloxacin, Apex
14. Dolasetron, Sanofi-Aventis
15. Ketamine, Parnell
16. Marbofloxacin, Pfizer
obstruction, an antegrade positive contrast pyelogram should be performed. In human medicine, several diagnostic imaging modalities have developed to provide more information for diagnosis of upper urinary tract (UUT) malignancy, including multidetector CT urography (MDCTU) (Wang 2010), MRI urography (MRU) (Takahashi 2010) and diffusion-weighted MRI (DWI) (Yoshida 2011).

Several surgical techniques have been developed to manage ureteral obstruction (Hardie 2004). In cats with obstruction in the distal two thirds of the ureter as in this case, it is preferred to be managed by partial ureterectomy and ureteroneocystostomy. Ureteroneocystostomy techniques can be divided into intravesical techniques such as the mucosal apposition technique (Gregory 1996), which is what we used in this case, and extravesical techniques, which does not require a cystotomy (Bernsteen 2000). The latter is preferred in cats, particularly when there is minimal preexisting ureteral dilation, because it is associated with a reduced degree of post-operative swelling and ureteral obstruction (Mehl 2005). Renal descensus and psosas cystopexy can be performed to reduce tension (Stone 1992; Kyles 1998).

The main reason for using an intravesical approach in this case was to gain better access to the distal ureter and therefore obtain a more precise evaluation of the obstruction. Both antegrade and retrograde flushing were performed to determine the degree of patency of the ureter and to identify the location and length of the blockage. A cystotomy also enabled a thorough examination of the bladder mucosa.

With the subcutaneous ureteral bypass (SUB) device becoming widely available nowadays, placement of a SUB device may allow wider surgical margins by removal of a larger portion of the ureter and a partial cystectomy in the current case. However, the SUB device was not used widely at the time of the surgery.

The dehiscence of the GI biopsy sites might be attributed to multiple factors. The prolonged active acute uraemia might have compromised the GI wall. Although a previous study has concluded that cats with alimentary lymphoma did not appear to be at high risk of post-operative dehiscence after full-thickness GI surgery (Smith 2011), the renal values of the cats included in the study were not mentioned and no cat in the study was anaemic. The surgeries conducted in the above study were all associated with their GI problems including biopsy, mass removal and GI obstruction. There was no other concurrent disease other than one lymphoma case mentioned in the article. A combination of uraemia, anaemia and alimentary small cell lymphoma may predispose the animal to wound dehiscence.

Ureteronephrectomy has been suggested for ureteral tumours that have not metastasized or invaded locally (Dobson 2010). However, the decision was made to leave the left kidney in place. First, the function of the patient’s right kidney had not been fully evaluated and the mild elevation of renal values at presentation might suggest a functionally compromised right kidney. Furthermore, we did not know if the TCC has metastasized. Last but not least, there is no study supporting that ureteronephrectomy provides longer survival times in cats with ureteral TCC.

There is limited information regarding chemotherapy for TCC in cats. Single-agent treatment with piroxicam in dogs with urinary bladder TCC has been reported to yield a median survival time of 181 days (Knapp 1994). The value of NSAIDs as an anti-cancer therapy has not been fully evaluated in cats. NSAIDs should be used with caution in cats due to the fact that feline patients are more prone to their adverse effects. However, piroxicam had been well tolerated by the cats in a previous study (Wilson 2007). Combination of mitoxantrone and piroxicam has also been suggested to be potentially beneficial for cats with urinary bladder TCC according to the data of canine TCC. In addition, a recent study showed cats with TCC, which were treated with meloxicam therapy (with or without surgical removal), had a medium survival time of 8 months (311 days) (Boomer 2012). The data suggested that meloxicam might be beneficial in the palliative management of urinary bladder TCC in cats. However, the presence of alimentary small cell lymphoma and the treatment regime including prednisolone in this case excluded the concurrent administration of NSAIDs. Radiation therapy could potentially be an alternative therapy if the adverse effects could be tolerated by the patient. In addition, a ureteral stent (Berent 2010) or a SUB device (Horowitz 2013) can be placed with minimal invasion to alleviate further ureteral obstruction should the regrowth of the TCC occur in this case to maintain a good quality of life for his remaining time.

The study by Boomer et al revealed that the medium survival time for the meloxicam-treated cats was longer in COX-2-negative cats (375 days) compared with COX2-positive cats (123 days) (Boomer 2012). However, further studies with a larger number of cases may be required to confirm this finding.
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Ethical dilemmas in companion animal practice

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In July 2018 I presented a podcast on ethical headaches in companion animal practice to CVE members. The aim was to provide an overview of frameworks used to approach ethically challenging situations that may arise in practice settings, and to help people work through some of these situations to their satisfaction, with the aim of (hopefully) improving clinical outcomes and reducing moral stress. A bit ambitious for one hour!

Ethically challenging situations are common in veterinary practice, with the majority of veterinarians experiencing at least one ethically challenging situation at least once per week, and some veterinarians experiencing these multiple times per day (Batchelor and McKeegan, 2012, Kipperman et al., 2018). For 9% of US-based veterinarians, ethically challenging situations are the leading cause of work related stress, while 42% reported that they were ‘one of many equal causes of work related stress’ (Kipperman et al., 2018). Less experienced veterinarians, primary accession veterinarians, associates and female practitioners were more likely to report experiencing ethically challenging situations, and more likely to be stressed by them (Kipperman et al., 2018). How frequently you experience ethically challenging situations depends what you consider ethically challenging. For example, for many veterinarians, a request for ‘convenience euthanasia’ of a companion animal is very stressful, because they need to determine whether they serve the interests of the animal or the interests of the owner. But for some veterinarians there is no ethical challenge – the animal can be killed humanely, therefore animal welfare is not compromised, and the client’s interests are served (Rathwell-Deault et al., 2017b). What doesn’t elicit a second thought for one practitioner may be experienced as an ethical crisis for another.

Themes discussed following the podcast

The Podcast discussion forum, which was open to all registrants, was extended due to participant interest. The general themes may be of interest to CVE members and so I’ve provided an outline here.

De-identified posts from the discussion forum (excluding those made by the author) were uploaded into NVivo Pro 11 qualitative and mixed methods software. A word-frequency query was used to generate a word cloud (Figure 1). This provides an overview of words that came up most commonly.

An informal thematic analysis was performed (for a discussion of thematic analysis, see (Terry et al., 2017)). Briefly, this involved rereading all of the posts and coding them inductively for themes. The same post could have several different codes assigned, so that any extract of a post could remain uncoded, be coded once, or be coded multiple times. The list of codes was examined to identify clusters of codes and complex codes which were grouped together identified as themes. Themes were reviewed for both internal coherence and distinctiveness from other themes. This involved reading all coded extracts from each theme. Where data extracts did not fit a theme, these were either reallocated to a more appropriate theme, allocated to a new theme, or discarded from the analysis. Themes that overlapped were merged. I will discuss key themes in order of their frequency of appearance, below.

1. Conflict between veterinarians and employers/practice policy

Some participants mentioned experiencing an ethically challenging situation when they felt that the wishes of employers, practice culture or policies went against/conflicted with their professional or personal values, or perceived interests of the client/animal. Whose interests should they ultimately prioritise? For example, a number of veterinarians raised concerns about employers or practice policies, which discouraged or were perceived to discourage referral. This is at odds with veterinary professional codes (for example (Veterinary Practitioner’s Board of NSW, 2013)), but some veterinarians would prefer...
to leave their job or remain in a workplace that causes them moral stress, than raise concerns about the policy with an employer.

One of the limitations of much of the veterinary literature, and possibly tertiary veterinary ethics education, is that it overlooks this issue, focusing instead on the conception of the veterinarian as balancing the interests of the client and those of the animal. This simple ‘triad’ fails to recognise that veterinarians have obligations to employers, professional bodies and the community, and may create an unrealistic or distorted conception of the veterinarian as a ‘moral hero’ (Ropohl, 2002). To what extent is the individual veterinarian responsible for changing workplace culture? What if s/he is unable to?

Workplace conflict (for ethical and other reasons) is a concern because it may impact personal wellbeing of veterinarians, but also retention in a profession where career attrition is an acknowledged problem (Heath, 2001). Veterinarians and nurses may change jobs in an attempt to find congruence between personal and organisational values (Page-Jones & Abbey, 2015). Indeed, a number of participants mentioned leaving an employer due to such conflict.

An investigation into factors associated with work and life satisfaction of veterinary practitioners in Germany found that female employed veterinarians reported the most stress, and their work satisfaction was highly influenced by their satisfaction with their supervisor and colleagues (Kersebohm et al., 2017). In this study, respondents prioritised ‘a good working atmosphere’ over a ‘reasonable salary’ and ‘holidays and leisure time’, suggesting that there is room for practices to develop a collaborative and supportive culture. The authors of the study call for better training of veterinary managers and supervisors. In addition to that, communicating about and understanding values may be helpful for all parties in establishing a positive workplace culture.

Interestingly, interviews with veterinarians and nurses revealed that some ‘equate being a commercial organisation with being unethical’, indicating a significant challenge in finding a path that is commercially successful and ethically acceptable to the profession (Page-Jones and Abbey, 2015).

A related issue is the autonomy of employed veterinarians. Many veterinarians undertake CPD but may be constrained regarding changes they can put into place if they are not a key decision-maker in the practice. For example, a veterinarian may undertake CPD with a focus on minimising fear, anxiety and stress in veterinary patients. But they may not be in a position, or may not perceive they are in a position, to request interventions such as non-slip examination table surfaces, longer consultations for fearful animals, or waiting rooms that cater to species specific needs (Edwards et al., 2019). This is a potential source of moral stress which may lead to frustration and disengagement.

### 2. Lack of time for ethical analysis/decision making

Some participants felt that they made decisions too quickly, then subsequently ruminated or experienced ‘decision regret’ (sometimes for many years). In addition, some felt that because of client or management constraints, that a decision needed to be made on the spot, even though the veterinarian felt that they did not have all of the necessary information to make an appropriate decision. This may be exacerbated by financial constraints which limit diagnostic testing. Alternatively, time constraints meant that it was sometimes easier to order further investigations and treatments rather than assess patient quality of life and reconsider diagnostic/treatment goals.

As discussed in the Podcast, a number of ethical decision making tools, from a simple cost: benefit analysis to an ethical matrix, require time to identify stakeholders and concerns, as well as research to consider the costs/risks and benefits of particular courses of action. There may be a need to consult a third party such as a specialist or insurer, to review legislation or relevant Codes of Conduct, or to discuss a decision with colleagues.

Well-developed practice policies based on consultation may be helpful in saving time where similar ethically challenging situations recur (for example, the treatment of wildlife or stray animals). This takes time, but if team members feel that their opinion has been heard and valued, and it saves time in the long run, it is surely time well-spent.

There are situations where time can be bought. Not every ethically challenging situation needs to be addressed immediately. Some participants had developed strategies including the use of revisits, follow-up calls, or even admitting the patient, to facilitate decision making in particularly challenging cases. However, if lack of time is a consistent problem, it points to a management issue. If veterinarians, as professionals who subscribe to a code of ethics, don’t have time to address ethically challenging situations, the public may lose trust in the profession (Susskind & Susskind, 2015). Are there ethical grounds for extending the length of consultations?

Hopefully, veterinary undergraduate training and continuing professional development will increasingly focus on helping prospective and practicing veterinarians think through common ethically challenging situations ahead of time. (As an aside, in addition to complicating ethical challenges, it is important to consider the impact of being time-poor on veterinary team members. Given concerns about wellbeing of members of our profession, this author has argued elsewhere for lunch breaks (Fawcett and Brailey, 2017)).

### 3. Euthanasia refusal

There are situations where some veterinarians do not agree that an animal should be humanely killed, often referred to as ‘convenience euthanasia’ or humane killing of an
animal purely for the interests of the owner. What counts as ‘convenience euthanasia’ differs between veterinarians (Rathwell-Deault et al., 2017a).

In a study of Australian veterinarians, performing euthanasia ‘for reasons you do not agree with’, was one of the least frequent ethically challenging situations experienced by veterinarians, but one of the most stressful (second only to situations where the veterinarian suspects animal abuse) (Crane et al., 2015).

One option is to refuse to perform the service (Yeates and Main, 2011, Magalhães and Ana, 2016), but this raises concerns about the consequences to the animal and owner, as well as issues around responsibility for the animal. Kipperman and colleagues found that 80 per cent of survey respondents reported refusing to euthanise an animal at some point (52 per cent reporting declining such a request every few years, 32 per cent reported declining such a request a few times a year) (Kipperman et al., 2018). The most common reasons for reluctance to decline such requests were fear that the client would seek other options that may worsen the animal’s welfare (65.3 per cent), difficulty refusing such a request when a client had already reached a decision (50.8 per cent) and concerns about jeopardising their relationship with the client. On this basis, for every euthanasia actually refused by a veterinarian, there may be many others that veterinarians perform despite not feeling comfortable with this decision. A sub-theme that arose here were situations where veterinarians were asked by clients to humanely kill their pet when they, the client, died. Opinions were divided about the appropriate course of action but the discussions often revealed important contextual information was obtained by exploring the issue in depth with the client before refusing outright. This issue was widely discussed in the media recently: www.cbsnews.com/news/owner-had-health-dog-euthanized-so-they-could-be-buried-next-to-each-other/

4. Euthanasia for behaviour problems

Euthanasia for behaviour problems was a major theme, in part because many veterinarians felt they did not have expertise in behaviour, and/or they did not know enough about the animal (for example, the problem behaviour was not demonstrated in the consultation room). Concerns included challenges in verifying behaviour problems and questions around liability if euthanasia is not performed. There was also uncertainty around assessing how behaviour problems impact companion animal quality of life. Such challenges could be exacerbated by lack of time, which may limit the ability to take a comprehensive behavioural history or consider environmental management or training.

This is consistent with the findings of a study of mortality resulting from undesirable behaviours in dogs aged under three years attending primary-care veterinary practices in England (Boyd et al., 2018). A retrospective review of records, via the VetCompass database, found that veterinarians had recommended referral for an undesirable behaviour in just 10.3% of cases where dogs had subsequently died (either by euthanasia or motor vehicle trauma), and dispensed nutraceutical, pheromone or pharmacological treatment for an undesirable behaviour in 3% of cases where dogs had subsequently died (either by euthanasia or motor vehicle trauma). The study also revealed that a record of owners trying to address the undesirable behaviour(s) was present in only 12.9% of cases, and only 34.4% of that small percentage had consulted their veterinarian previously about their dog’s behaviour. The authors speculate that in some cases, veterinarians or owners may have deemed some behaviours too severe to treat, but it is likely that there is a knowledge gap among both owners and veterinarians about early identification and management of undesirable behaviours.

Such consultations may require lengthier discussions, and may benefit from more information (for example, owners presenting video footage in instances of recurrent behaviour). Additionally, as participants pointed out, veterinarians could refer such cases where possible, or consult Specialists or Members of the ANZCVS Behaviour Chapter about strategies for management.

5. Discussion about ethically challenging situations reduces feelings of isolation

Discussion around, rather than the resolution of, ethically challenging situations was considered very important by participants. Of course, this was mentioned by veterinarians actively participating in a discussion forum, so one might assume self-selection bias, i.e. participants in the discussion forum are those who appreciate the opportunity to discuss cases.

In a recent discussion about the establishment of a clinical ethics consultation service at North Carolina State University, the authors noted that there is good evidence from the human medical field that ‘effective ethics consultation can relieve or diminish many...stressors and promote better patient (and family) care’ and that the consultation service had ‘been very positive in relieving stress for staff and house officers who previously felt alone in their moral distress when dealing with challenging situations’ (Adin et al., 2019).

A number of participants expressed interest in ‘ethics rounds’ (mentioned in the Podcast), employed in the nursing and medical fields (Silén et al., 2014, Watts et al., 2013). Ethics rounds has been documented in some veterinary teaching settings (Graber, 1983), but not among practitioners.

6. Financial barriers to veterinary care

This theme included concerns about clients who were unable to pay for care, strategies to improve accessibility of veterinary care (including pet insurance, payment plans, wellness plans and so on), if and when to treat...
animals for free, and compromise in standard of care to save an animal. Batchelor and McKeegan identified client financial constraints as the most commonly encountered ethically challenging situation (Batchelor and McKeegan, 2012). Situations where the owner would not pay for treatment were one of the most common ethical stressors experienced by Australian veterinarians (Crane et al., 2015). Client economic limitations affected the ability to provide desired care for patients on a daily basis for 57 per cent of US veterinarians (Kipperman et al., 2017).

This suggests that veterinarians should expect to encounter clients with economic limitations, and pro-actively address barriers to veterinary care. Yet often this is not the case. For example, Danish companion animal practices encounter financially limited clients often (33.8 per cent 3-4 times per month; 24.6 per cent 5-10 times per month) but only 9 per cent reported having a written practice policy on handling clients with financial limitations (Kondrup et al., 2016).

The Access to Veterinary Care Coalition released a report in December 2018 (Access to Veterinary Care Coalition, 2018) identifying barriers to veterinary care in the US. The report states that millions of pets do not receive adequate veterinary care because the costs are beyond the family’s ability to pay. This may be the most significant animal welfare crisis affecting owned pets in the United States. Furthermore, it claims that ‘…there is wide recognition within the community of veterinary health service providers and public health officials that a care gap exists in veterinary medicine, which negatively impacts the health and well-being of underserviced pets and their families.’ (Access to Veterinary Care Coalition, 2018).

One of the issues raised in the report is the conflicts between the interests of animals, whose welfare is at stake, the interests of owners in ensuring the health of their companion animals and enjoying the benefits of the human-animal bond, and the interests of veterinarians in earning a reasonable income (according to a recent report, the median annual income of a full-time veterinarian in Australia was $84,240, with a student debt of up to $150,000 (Hanley and Butt, 2019)). This conflict is likely to increase given escalating student debt that veterinarians carry. The report reveals that many private veterinary practitioners feel they are in competition with low-cost clinics, and suggests that student debt forgiveness for pro-bono veterinary work may help address the ‘care gap’.

7. Owner unwilling to treat a particular condition appropriately

Another emergent theme was veterinary concerns about owners electing not to treat a condition. For example, participants described situations where owners failed to recognise, or did not appreciate the severity of, conditions such as problem behaviours, chronic pain and dental disease. Alternatively, there were situations where owners used inappropriate treatment (for example, behavioural modification that the veterinarian deemed unacceptable) or sought treatment such as anaesthesia-free dentistry.

Such situations can give rise to moral stress because the veterinarian knows the ‘right’ thing to do for an animal, but may feel constrained by limitations imposed by the owner. It also raises questions about whether veterinarians should act as strong patient advocates (Coghlan, 2018).

Some of these challenges may be resolved through communication and education. Position statements, media releases and guidelines from professional organisations can be helpful to share with owners (for example, see the AVA’s media release on Anaesthesia free dentistry https://www.ava.com.au/node/85991 or the American Veterinary Society of Animal Behaviour’s position statement about use of punishment for modification of animal behaviour https://avsab.org/wp-content/uploads/2018/03/Punishment_Position_Statement-downoad_-_10-6-14.pdf).

Other themes discussed were:

- Prudent use of antimicrobials (including the dilemma of when to accede to client demands);
- Situations where veterinarians felt they had to choose between servicing a community or population of animals in need, or taking a job offering better working conditions/remuneration;
- The refusal of owners to euthanase an animal with poor quality of life and a poor prognosis in the face of a strong veterinary opinion that this is in the best interest of the animal;
- Determining what counts as a ‘treatable condition’ when financial/practical resources are limited (for example, complex medical or behaviour cases in shelter settings; wildlife; stray animals);
- The challenge of performing a quality of life assessment in a clinical setting (especially with episodic conditions e.g. seizures).

Conclusion and resources

The only conclusion to draw is really that there is no conclusion! This was not a formal study, and there are undoubtedly numerous ethically challenging situations that were not discussed in the forum. Not everyone who downloaded the podcast participated in the discussion forum, and participants were limited to veterinarians and vet students (not veterinary nurses or owners).

I did not collect demographic information, although it would be interesting to explore potential associations with the nature, frequency and perceived stressfulness of ethically challenging situations and gender, role in the practice (employer, employee), education (including CPD), years in practice and so on. This is something I hope to explore in a formal study.
There may be scope to incorporate training about specific ethically challenging situations in veterinary undergraduate training, as well as in continuing professional development.

Participants did mention that they found characterising their own ethical views an interesting exercise. You can do this by registering for free at http://www.aedilemma.net/ and then completing the quiz and reviewing the feedback.

Another useful resource is a series of short videos based on the work of Mary Gentile (Gentile, 2017), about articulating one’s own ethical position in workplace settings. They can be viewed for free here: https://ethicsunwrapped.utexas.edu/series/giving-voice-to-values

Finally, the St James Ethics Centre runs ETHI-CALL, a free ethics counselling hotline. It would be fabulous to fund such an initiative for veterinarians! http://www.ethics.org.au/ethi-call/ethi-call

For me the discussion forum underscored the need for a safe (as in confidential, non-judgemental) space in which veterinary professionals can talk about ethically challenging situations.

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She is currently undertaking a PhD about ethically challenging situations confronted by veterinarians, veterinary nurses and animal health technicians under the supervision of Professor Paul McGreevy (Sydney School of Veterinary Science) and Dr Siobhan Mullan (Bristol University).

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Reference
8698.0 – Waste Management Services, Australia, 2009–10

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