C&T

Issue 292 | September 2018

Centre for Veterinary Education

Control & Therapy Series

MAJOR WINNER

Uterotomy of a North Island Brown Kiwi (Apteryx australis)

p6

C&T

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Control & Therapy Series

Australia's Leading Veterinarian Forum

Centre for Veterinary Education

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Contents

From the director

What a variety of articles await you in this edition of C&T! I mentioned previously that Joanne Krockenberger has joined the CVE as Associate Editor and you will see that she is already starting to make her mark. For those of you who know Richard Malik, you will see his hand in some of the contributions as we continue to draw upon his wide network of colleagues and friends for material.

I am ever hopeful that with the wide range of topics presented in this issue, it will stimulate others to submit interesting cases and articles, or to comment on previous articles. After all, that was the forum originally promoted by Tom Hungerford half a century ago, before the evolution of the internet, blogs and electronic social media.

The CVE is frequently congratulated on continuing to provide single theme conferences rather than multi-stream events, where everyone can get a high-quality update on particular subject areas. I am also pleasantly surprised at how many people still value the hard copy proceedings and continue to write notes in the expansive margins while the speakers are giving them valuable information, tips and tricks, etc. Next year will be no different, with the February Sydney Cardiology conference being led by Etienne Cote (regarded by some as the Ettinger of this generation) and supported by Niek Beijerink and his selection of local experts.

The June Melbourne conference will be a combination of Critical Care & Surgery, presented predominantly by Sophie Adamantos and Mickey Tivey from the UK, while the 'holiday destination conference' in September will be held in Cairns.. Neurology has been chosen as the topic with a program designed to be interesting, educational and highly relevant to general practitioners, being led by Steven de Decker from the UK and supported by Sam Long and Pat Kenny. The last Neurology conference held by the CVE was in Fiji in 2011 and it was so successful that the participants stayed for every lecture while their golf clubs gathered dust!

The CVE Distance Education courses for 2019 are filling well once again, which is pleasing given the intense competition and broad offering of continuing professional development programs available around the world. As our marketing says, the CVE aim is to continue to provide high quality continuing education free from bias, and 2019 will be no different from any other year.

Enjoy the following articles as there is something for everyone, and don't forget to use the e-book to explore the additional materials and images available through those links.

For those of you who live and work in drought affected areas, our thoughts are with you. I know from years of experience the impact severe drought has on the whole community and how this can have a negative impact on veterinary practice and all of those involved with practices. It is times like this that we all need inner strength and resilience to cope with extremely challenging conditions. Take care of yourselves and those around you.

Hugh White
Director

Calendar

Intensive Seminar + Workshop

SYDNEY
Practical Ultrasound – Intensive Short Course
Mon 29 October – Fri 2 November 2018

Conference

SYDNEY
State of the Heart Cardiology Conference

Mon 18 – Fri 22 Feb 2019

MELBOURNE
Critical Care & Small Animal Surgery

Mon 17 – Thu 21 June 2019

Seminars

SYDNEY
Oncology: Theory & Practice
Fri 26 – Sat 27 October 2018

SYDNEY
Applied Anatomical Allsorts Seminar
Wed 31 October 2018

Hands-on Workshops

SYDNEY
Back to Basics: Diagnostic Ultrasound Workshop

Fri 21 September 2018

USA
Feline Medicine Interactive Workshop
25 - 26 September 2018

SYDNEY
Advanced Echocardiography Workshop

Sat 6 October 2018

SYDNEY
Stress Free Surgery Workshop

Sat 20 October 2018

2018 - 2019

September

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Hands-on Workshops

MELBOURNE
Back to Basics: Diagnostic Ultrasound Workshop
Sat 27 Oct 2018

SYDNEY
Bone Plating Surgery Workshop
Sun 11 Nov 2018

Experience of a lifetime

SOUTH AFRICA
Fri 7 - Fri 21 Dec 2018

TimeOnline

Ophthalmic Surgery
Mon 17 Sep – Sun 14 Oct 2018

Avian Medicine
Tue 2 – Mon 29 Oct 2018

Animal Forensics: Animal Cruelty & Interpersonal Violence
Mon 22 Oct – Sun18 Nov 2018

Herd Emergency Medicine
Tue 5 Nov 2018

Prevention & Treatment of Neonatal Calf Disease - Mon 25 Feb 2019

2019 program coming soon...

PodcastPLUS

Wildlife Rehabilitation
Thu 27 Sep 2018

Azotemia in the Cardiac Patient
Thu 25 Oct 2018

2019 program to start on 28 Feb

Distance Education

Important course dates:

31 Oct 2018 Early Bird ends

See flyer for this issue or visit cve.edu.au/education for the full list of DE topics for 2019.

1 Feb 2019 - DE program start!

Calendar Key

- Intensive Short Course
- Conference
- Seminar
- Hands-on Workshop
- Distance Education
- TimeOnline
- PodcastPLUS
- CVE closed

THANK YOU TO ALL CONTRIBUTORS

> ***I enjoy reading the C&T more than any other veterinary publication.***
>
> Terry King, Veterinary Specialist Services, QLD

Follow Tom Hungerford's 'goanna track to success'...

Since its establishment in 1969, the *C&T Series* has been a unique forum:

> ***… not what he/she should have done, BUT WHAT HE/SHE DID, right or wrong, the full detail, revealing the actual "blood and dung and guts" of real practice as it happened…***
>
> Tom Hungerford

Thank you to all contributors

…and more C&T articles and Perspectives are needed.

Thanks to every author who contributed articles or comments to the *Control & Therapy Series* (C&T) and to those who supplied images and visuals. Without your generosity the Series would cease to exist.

Winners

MAJOR PRIZE WINNER

Uterotomy of a North Island Brown Kiwi (Apteryx australis)
Orla Fitzpatrick **p6**

BEST VISUALS

Uterotomy of a North Island Brown Kiwi (Apteryx australis)
Orla Fitzpatrick **p6**

CVE PUBLICATION PRIZE WINNERS

Some Thoughts on Mobile Veterinary Services
Wendy Mashado **p4**

Ketofol as a General Anaesthetic Induction and Maintenance Agent
Adam Gordon **p16**

Concurrent Diabetes and Hyperthyroidism
Kate Mowbray **p27**

Leg rope for anaesthetised horses to improve safety
Nick Scott **p42**

PRIZE ENTITLEMENTS

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Best Visuals: DVD of your choice
Winner: A CVE proceedings
Visit **cve.edu.au/vetbookshop** to peruse our list of titles.

GENERAL + WINNER!

Some Thoughts on Mobile Veterinary Services

Wendy Mashado

e.wmashado@aapt.net.au

Wendy's Mobile Vet Service

C&T No. 5686

The evolution of my mobile service was directed more by circumstance than intent but I have now been offering a mobile, (home visit), service for 5 years. I don't advertise at all, anywhere, but we are increasingly busy. I live in a rural area, word of mouth and other people's social media spreads the word.

I have a medium-length wheel-base, high roof, Mercedes van which I have fitted out as a consult room. It is high enough for all but the tallest people to stand in. Opposite the sliding door, along the long side, is a small bench for a computer, a large lockable, multi-draw, tool box for medications, (Xtreme tool box), a sink and a camper fridge. There is a narrow bench along the short side with shelving underneath and a lift up table for consults. There is a high shelf all around and some scales behind the front seats which we drop down as required. On the roof is a solar panel that supplies the power for the fridge, computer and tyro machine. Large dogs are looked at on the floor, outside or in the owner's house. Small dogs and cats can be examined in the van or in the owner's house.

I have a vet nurse with me all day. It takes an hour in the morning for us to organise the day's appointments and to package up medications, clean and restock the van. Once home in the evenings the routine book work of a practice occurs as well as attending to any lab work we may have generated through the day. The nurses drive to appointments whilst I write up the notes of the previous case, act as receptionist, nurse, etc.

This form of veterinary service is great

- For the elderly and infirm
- For people with young children
- For people with multiple pets
- For animals that hate the vet clinic
- For euthanasias
- For seeing animals in their home environment and picking up subtle things which can lead to a diagnosis. This cannot be done in a 10-15min consult. I do like to have the time to watch and to listen. (I was sitting in a client's family room taking a history for their coughing cat when I noticed the door mat was a little ragged, and yes, the cat chewed at it when he was playing. Remove the mat and problem was solved.)

It is more personal and you develop better relationships with owners and their pets.

On the other side of the equation:

- There has to be a back-up clinic.
- You then lose control of the care of your patient. This can be frustrating for both medical and surgical patients if you are used to dealing with both.
- There is a limit to the breadth of care you can offer. This carries with it a degree of guilt as far as emergency services go and managing medical patients that require hospitalisation. Again this can be frustrating.

We have a daily pick-up for pathology, blood results are back the next day. We look at blood smears, can do PCV's, TP, FNA's, sticky tapes, UA's, all the simple lab work. This is all done at the end of the day.

I believe that having many years of practice experience is essential for the success of this type of service. I feel that the ideal situation would be for a mobile service, with dedicated staff, to be just one of the services offered by a Veterinary Hospital. The clients like it, the patients like it. It's a great way for older vets to slow down but still be involved in the industry in a meaningful and valuable way.

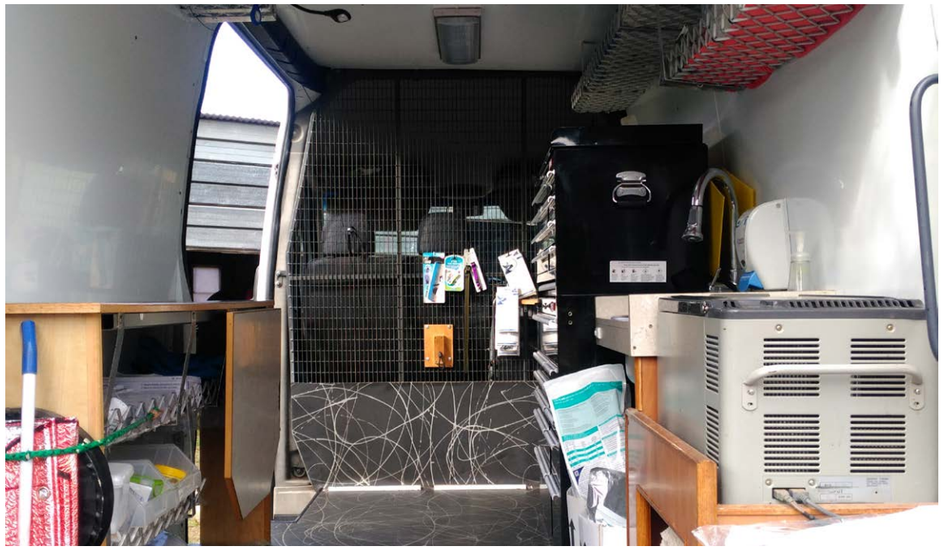

Figure 1. Interior of the van

Figure 2. Whale watching during a break

In-spite of the professional frustrations this form of practice can be very rewarding. There is less pressure, less stress and more time to appreciate the simple joy that can be found in our work. And we take the time to watch the whales as they cruise down the coast!

Figure 3. Interior of the van

AVIAN + MAJOR WINNER!

Uterotomy of a North Island Brown Kiwi *(Apteryx australis)*

Orla Fitzpatrick

VetEnt
Queenstown New Zealand

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C&T No. 5687

Tawahi, an 8 year old 2.5kg breeding female on display at Kiwi Birdlife Park Queenstown was brought in to the clinic at VetEnt Queenstown after keepers noticed the sudden onset of what they presumed were seizures in her enclosure. The keepers alerted the park manager and Tawahi was brought to the clinic.

On arrival at the clinic at 4pm, Tawahi was collapsed and recumbent. Her abdomen was firm and swollen. Tawahi had a recent history of laying soft shelled eggs that were cracking easily during incubation. It had been a while since her last egg was laid and she was in an enclosure with her mate. She had been off her food and was drinking more than normal. Her temperature was normal at 37.5° celsius.

A blood sample was taken (from the medial metatarsal) and sent to Gribbles Lab Christchurch and Tawahi had

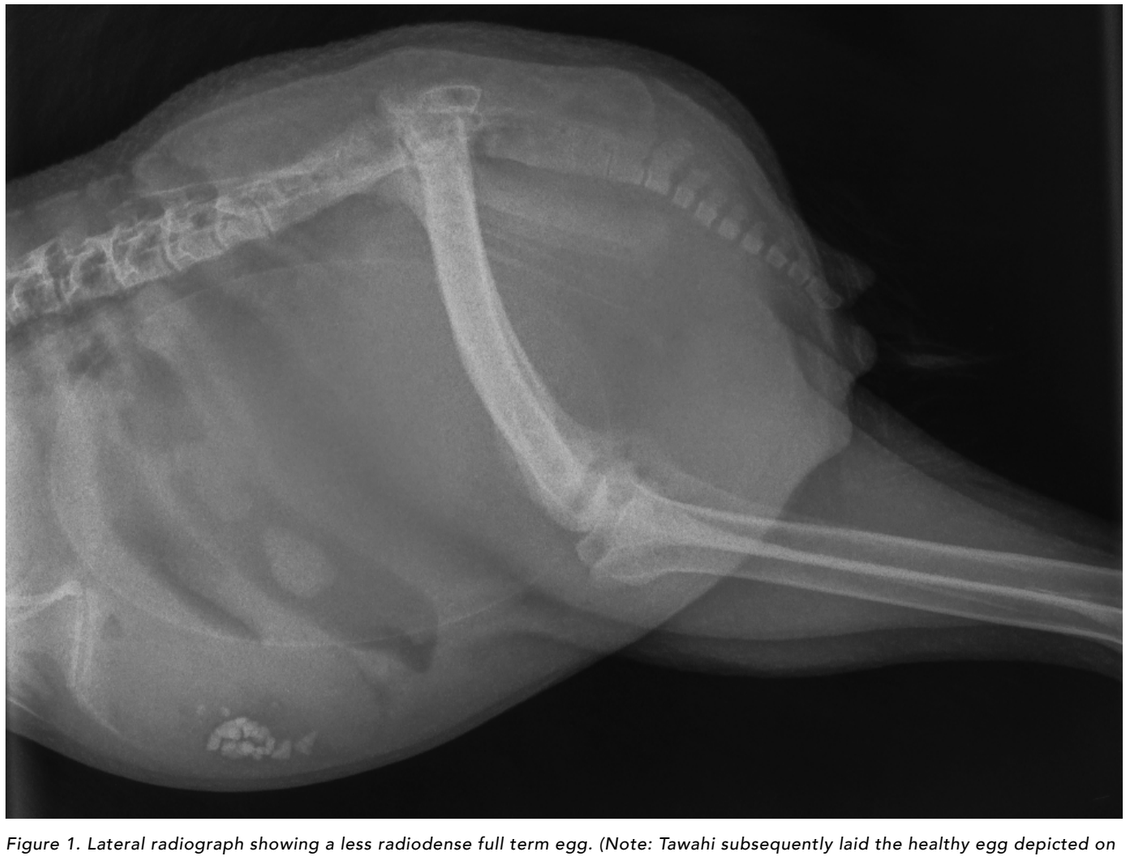

Figure 1. Lateral radiograph showing a less radiodense full term egg. (Note: Tawahi subsequently laid the healthy egg depicted on the cover).

Figure 2. Tawahi receiving oxygen in her cage.

an intravenous catheter placed in the medial metatarsal vein and was started on fluid therapy (Hartmann's = lactated Ringers solution 10% total BW over 24hrs). As her breathing was rapid and shallow we placed her on oxygen (see *Figure 2*)

Abdominal radiographs showed a large full term egg taking up most of her abdomen *(Figure 1)*. The egg appeared less radiodense than a normal full term egg, suggesting that it was poorly calcified.

We were concerned that Tawahi was 'egg bound' and potentially hypocalcaemic so calcium gluconate was added to her fluids (10mg/kg IV).

Cloacal palpation showed the presence of the egg but the utero-vaginal sphincter was not dilated. On consultation with Brett Gartrell of Wild Base Hospital Palmerston North it was recommended to apply Prostin E (a prostaglandin E2 human vaginal gel) to help open the sphincter. With many phone calls to local midwives we finally sourced some gel at the nearest maternity hospital 3 hours drive

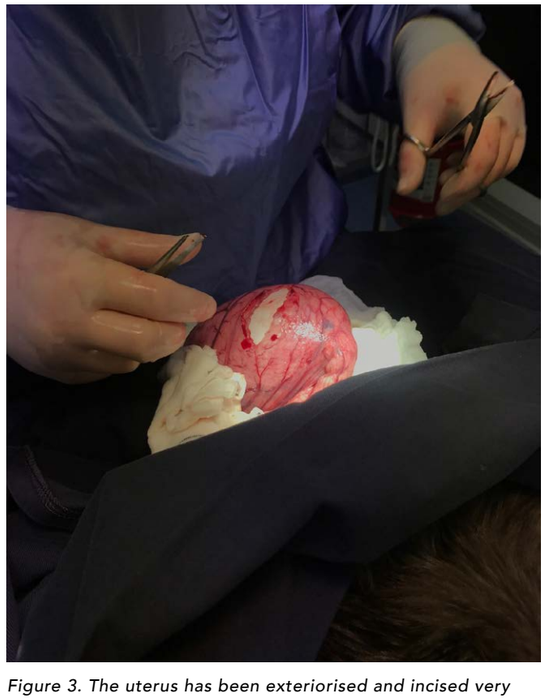

Figure 3. The uterus has been exteriorised and incised very carefully to avoid rupturing of the fragile egg.

Figure 4. Tawahi in the immediate post-op period needed to be intubated for quite a while.

Figure 5. The egg, weighing 400 grams, was unfortunately soft shelled.

from the clinic and our veterinary nurse found family friends to collect and deliver the gel to us.

We applied the gel at 8pm (very small amount in the cloaca) but Tawahi was rapidly deteriorating, becoming more distressed and appeared to have aspiration pneumonia from her contractions so, at 10pm we knew Tawahi was not going to survive the night if left and we made the call to go in surgically.

We induced Tawahi with Isoflurane and Oxygen (we have engineered a special Kiwi induction mask using a 60mL syringe to accommodate for their long beak). Once induced, we then intubated with an endotracheal tube. The intravenous fluids were warmed with a fluid line warmer and she was on a patient warming system throughout the procedure to help maintain her body temperature.

Due to the size of the egg a large area of feathers were plucked on her ventrum as a large skin incision would need to be made. Routine skin sterilisation using chlorhexidine scrub was carried out. We limited the alcohol to prevent hypothermia.

A midline coeliotomy was performed and the large egg within the uterus was quickly visible. The thin walled uterus and oviduct were exteriorised, the abdomen packed off using moistened laparotomy sponges and an incision was made in the uterus to remove the egg. As the egg was soft shelled, care was needed when attempting to remove it from the oviduct to reduce the risk of rupture and contamination of the abdomen with its contents. Once the egg was removed, the oviduct contracted very quickly. A biopsy of the uterus was taken before closure and sent for histological assessment. The egg weighed 400 grams which was a healthy size Kiwi egg but unfortunately as it was soft shelled it was not viable, so it was not incubated.

The oviduct was closed with one layer of 4-0 monofilament continuous inverting suture. Flushing the abdomen was not required due to the sterile technique and also not recommended due to the fact that there is continuity with air sacs.

The peritoneum and muscle layers of the abdomen were closed with 3-0 monofilament simple interrupted suture, subcutaneous layer using 3-0 braided simple continuous suture and the skin was closed using 2-0 non-dissolving cruciate sutures.

On a few occasions during closure the nurse raised concern as she noticed the heartbeat weaken. Adrenaline (1:1000 -0.01mg/kg NB extrapolated from companion animal dose) and atropine (0.01mg/kg IV) had to be given and the bird appeared to have a good response.

Isoflurane was turned off as soon as the last skin suture was placed but the kiwi needed to be left intubated for at least 90 minutes following the surgery. She was kept warm with extra heat discs, bubble wrap and 'hot hands'. When she was ready for extubation we propped her into sternal recumbency, and used a mask to deliver oxygen.

At 2:30 AM I took Tawahi home to monitor throughout the night. We were not very hopeful that she would survive but I was delighted to see her sitting up in her cage and much brighter a few hours later. With very little sleep we met

Figure 6. Tawahi propped up after the surgery. She took a few hours to recover.

back at the clinic at 7am to medicate and feed Tawahi. The Park Manager and head keeper brought her special diet and helped us out considerably as I had a very busy morning of consults.

Due to the aspiration pneumonia we started her on enrofloxacin (10mg/kg PO BID) and Itraconazole (5mg/

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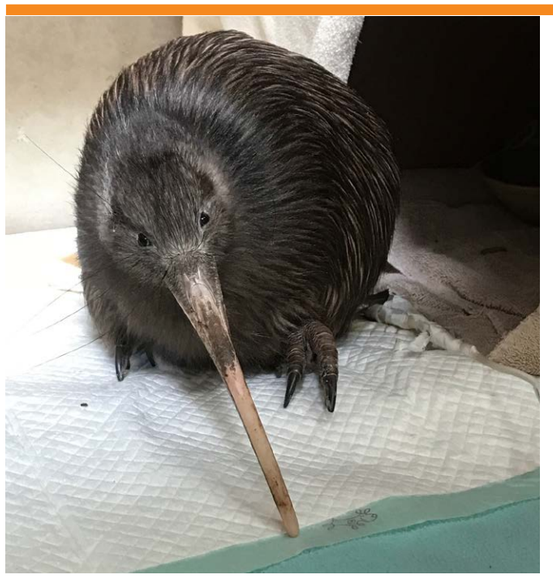

Figure 7. Less than 12 hours after the surgery, Tawahi is alert, moving around her cage and eating well.

kg PO BID) and she was given Metacam® (0.1mg/kg PO SID) and butorphanol (2mg/kg IM BID) for pain relief. Fluid therapy was continued and Tawahi was hospitalised over the next few days to monitor and medicate.

Her morning and evening meals were weighed to monitor her dietary intake. We hand fed small grape sized balls

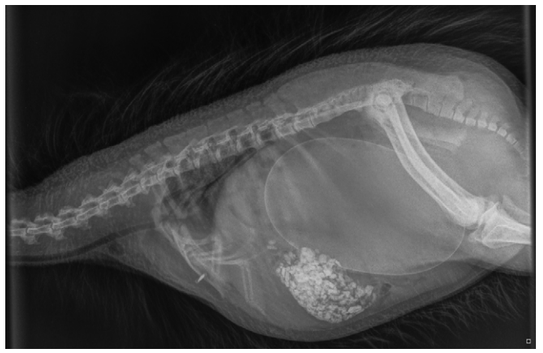

Figure 8. Radiograph showing Tawahi's healthy egg

Figure 9. The second egg

of her diet containing her medication twice daily to help supplement her nutritional intake but Tawahi recovered quickly and was eating her normal daily intake within a few days of surgery.

Tawahi made an incredible recovery suffering only from some bruising around the incision site and went back to the Park after a few days, she was kept separate from her mate for a few weeks to prevent breeding. The skin sutures were removed 12 days post operatively. Tawahi and her mate were reunited and were soon showing very healthy breeding behaviours. We had been a little concerned about the future breeding risks with Tawahi but thankfully Tawahi laid a perfectly healthy egg with no need for intervention at the end of March that is currently being incubated (Kiwi eggs take 80 days to hatch), her latest chick, Mahuta, a one year old male, is going to be released in a few weeks and the park hope to release Tawahi within the next few years.

View more images on this case in the complementary eBook.

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ORLA AND ALAN

Originally from rural County Down in the North of Ireland; Orla graduated from UCD School of Veterinary Medicine, Dublin in 2005 and after a brief stint as a mixed animal practitioner, she decided to focus on companion animals while working as a locum in Australia, the UK and Ireland. Orla first visited New Zealand in 2008 while on holiday and fell in love with the incredible scenery, outdoor lifestyle and friendly locals. She returned in early 2010 with a job offer and has worked in the resort town of Queenstown ever since with no plans of leaving.

As a keen conservationist, Orla loves being able to utilise her degree to help vulnerable NZ native wildlife through her work with the local wildlife park, various conservation groups and by educating clients on responsible pet ownership. Despite the extremely enjoyable work with natives, Orla is actually a cat nerd and is studying towards her feline memberships. She is servant to a toothless rescue cat called Alan.

ACKNOWLEDGEMENTS

Orla would like to take this chance to acknowledge the flawless anaesthetic monitoring of Tawahi by Vet Nurse Georgia Affleck who was truly impressive in her handling of such a difficult patient and thanks the staff at Kiwi Birdlife Park for their support.

LARGE

Primary Photosensitisation in Sheep in South Australia

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C&T No, 5688

Introduction

An extensive outbreak of photosensitisation in sheep was observed across South Australia's Eyre Peninsula and Northern Adelaide Plains from late September to October 2017. Large numbers of producers reported lambs and older sheep with swollen ears & faces, some with visibly sunburnt facial skin. These sheep had been grazing a variety of pasture types, but predominantly pastures with a high content of legumes such as medic and vetch species. Some flocks grazing cereals were also affected.

Affected producers contacted PIRSA, livestock agents, farm advisors and nutritionists for information and advice. Most producers moved affected sheep from the pasture and the syndrome rapidly resolved, although a small number of severely affected sheep died. Some testing was conducted on affected sheep, and this indicated some raised liver enzyme levels, and hepatopathy (liver damage) was noted in two severely affected animals.

On affected properties producers observed heavy infestations of aphids on the pastures and the theory that aphid consumption was contributing to the condition gained some traction. Other producers observed black smut like fungal growth near and on some plant species and a suspicion of mycotoxin induced hepatopathy was also discussed. However, some affected producers did not report either a heavy aphid infestation or fungal issues.

Primary photosensitisation in livestock occurs sporadically in association with some *Brassica* species and sometimes on Lucerne pasture. Occasionally the condition occurs in small numbers of sheep on other legumes, particularly in monocultures. On this occasion, an estimated 25 000 sheep on mostly legume pastures were affected over a short period. This condition has never been reported on this scale in South Australia. Disease investigations ruled out exotic diseases such as bluetongue and confirmed most cases as primary photosensitivity.

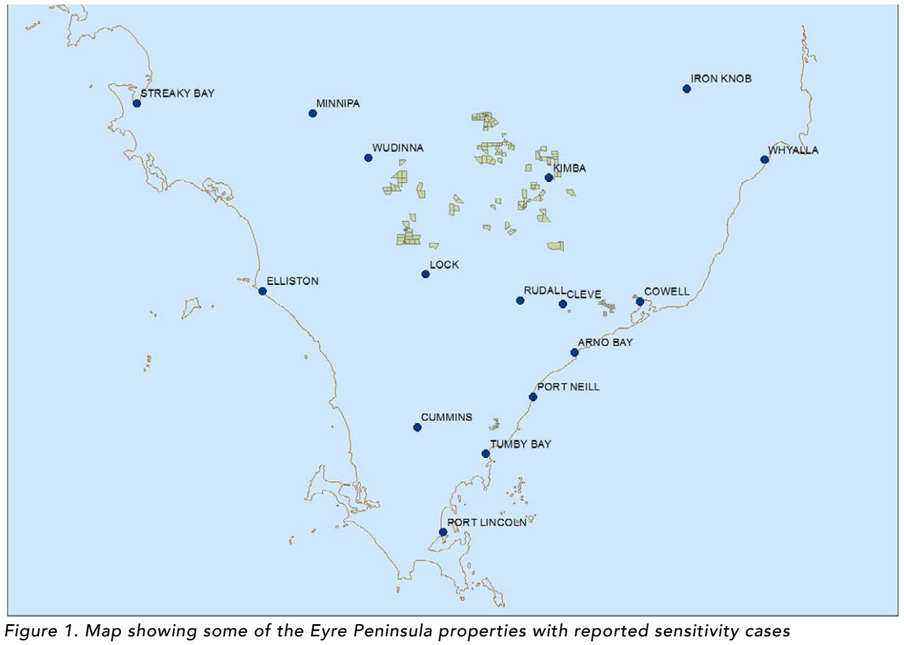

Figure 1. Map showing some of the Eyre Peninsula properties with reported sensitivity cases

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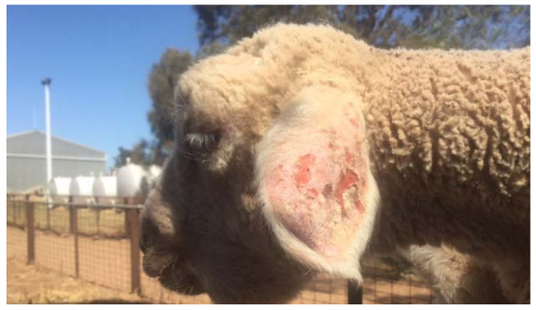
Figure 2. Possibly mould beneath medic plants



Figures 3,4,5,6,:Photosensitivity affected sheep on Eyre Peninsula properties showing crusty lesions on face, lips and pinnae.

Figure 7 Aphis craccivora

History

Cases of photosensitisation in sheep began to be reported to PIRSA in late September 2017. Reports and alarm escalated through October as more sheep and properties were detected.

PIRSA Animal Health collected data and blood and plant samples from eight properties on the Eyre Peninsula. Another 17 affected properties were reported via Landmark agents and anecdotally there were many more properties affected than this.

The syndrome was reported in ewes, lambs and wethers grazing rye grass, vetch, vetch and medic, and medic pastures. Six of nine producers interviewed reported aphids present, but with varying levels of infestation. One producer reported a black smut or mould growing underneath or near medic plants.

Lesions observed included swollen ears, swollen lips and face, scabby lips and ears, severe conjunctivitis and blindness in some sheep and deaths in a small number of sheep but most affected sheep recovered quickly when removed from the affected pasture. Some affected sheep responded to injected corticosteroids and confinement in shearing sheds, away from sunlight. Severe cases (mostly lambs) had crusty, thickened skin on the face, lips and ears and failed to grow well after being affected.

Some producers reported that they managed the problem by controlling aphids in pastures using insecticides such as LeMat 290 (Bayer) before reintroducing sheep. Aphids were identified as cow pea aphids, *Aphis craccivora*.

Test Results

Four post mortem examinations were conducted by PIRSA, and a private Veterinary clinic conducted one investigation. Eleven blood samples were collected from nine properties.

Diagnosis

Primary Photosensitivity:

Most blood samples submitted showed only mildly elevated levels of the enzymes which indicate hepatic (liver) damage. This suggests that liver damage was not severe, and therefore primary photosensitivity was suspected in most cases.

Secondary Photosensitivity

Preserved liver samples in two cases of severely affected sheep did show evidence of hepatopathy with changes consistent with secondary photosensitisation. This suggests that in at least some of the severely affected sheep, liver damage was likely to have exacerbated the photosensitivity symptoms.

Discussion

Sporadic primary photosensitisation in sheep is well documented and known to be associated with particular plants and crops at specific stages of development. The condition normally affects small numbers of animals within a group, some more severely than others, and is often associated with very good growing conditions (Salmon *et al,* 2015). Growing conditions across most of the Eyre Peninsula had generally been poor prior to spring, with well below average biomass production across the region but above average rainfall in August with a warm spring produced good fodder growth on some of the affected properties, particularly in the Kimba area. However, not all affected properties experienced this above average fodder growth.

Sporadic, individual cases of primary photosensitization associated with fast growing, high protein pastures are reported in this area from time to time but the scale of this outbreak and the concurrent severe aphid infestations have not been reported previously in SA.

In this event an estimated 20,000-30,000 sheep were affected across a wide area, with face and ear lesions in young and adult sheep the predominant characteristics. In contrast, the *Salmon et al* report noted feet lesions as well. Understandably there was some confusion about the co-involvement of parapox virus infection (scabby mouth), and in one case this virus was confirmed. Some exotic diseases involving face lesions (vesicular diseases and bluetongue) were excluded on six properties using serology. Only sheep were reported to be involved in this event, and mainly merino or merino cross-bred sheep.

Primary and secondary photosensitisation have been described from a wide variety of green leafy plants, *Brassicas*, millets, medic species, Lucerne and grasses (Radostits *et al* 2000). **Primary photosensitisation** is described as '…ingestion of plants containing light sensitive substances' (Robson, 2007). These substances are ingested in amounts that exceed the animal's ability to detoxify through liver activity, and metabolites accumulate in the skin and are transformed into phylloerythrin by sunlight, and this damages the skin. Livestock are generally affected 4-5 days after going on to pastures and new cases cease when removed from the pasture affected. All grazing species may be affected but there can be individual and species susceptibilities.

In some of these cases, some sheep appeared to respond to injected corticosteroids and/or being confined out of sunlight in shearing sheds to reduce symptoms. Some lambs appeared to be severely affected (possibly some secondary liver damage) and lost considerable condition and value when sold one month later (pers comm).

Secondary photosensitisation occurs following liver damage, often due to fungal or plant toxins such as those occurring in lupinosis or in Heliotrope (potato weed) poisoning. These toxins damage the liver and allow metabolites to circulate that are activated by sunlight, such as they do in primary photosensitivity. Typically, severe cases of secondary photosensitivity do not respond well to treatment, and involve liver damage detectable on blood tests (significant elevation of GGT and AST) and are often fatal. Livestock disease investigations on the Eyre Peninsula sometimes confirm cases of secondary photosensitisation, usually associated with Lupinosis or Heliotrope poisoning (pyrrolizidine alkaloid toxicity). In cases of predominantly primary photosensitisation, individual sheep or even individual mobs may present with more severe symptoms of secondary photosensitivity, possibly due to underlying chronic liver pathology from their earlier grazing history.

While there are very few papers in the literature describing any association between aphid consumption and photosensitivity in sheep, Ferrer *et al* (2007) investigated whether photosensitivity in sheep grazing Lucerne was due to *Aphis craccivora* and/or seven-spot ladybirds (*Coccinella septempunctata*) larvae. These authors concluded that the aphids were not implicated in the photosensitisation cases, while the ladybird larvae were. McClymont and Wynne (1955) proposed the possibility of aphids causing photosensitisation in sheep in NSW, but no research was conducted to establish this.

Other theories of fungal or mycotoxin involvement associated with aphid excretions were circulating during this event, but were not investigated. Not all photosensitivity events were reported and while some cases reported an associated aphid infestation, others did not.

Photosensitivity may also be due to biochemical and other defence mechanisms that plants have evolved to protect themselves from insect and mammalian herbivores (War *et al*, 2012; Launchbaugh *et al*, 2001). These ***anti-grazing*** attributes in plants reduce their palatability, reduce their digestibility, or induce toxic effects when consumed and some of these attributes are induced by particular seasonal conditions or by grazing pressure (including herbivorous insects), or by an interaction between these factors and growth stage. Launchbaugh *et al* (2001) describe how grazing animals have developed mechanisms to contend with the anti-grazing attributes of plants. They discuss how grazing animals manage potentially harmful plant compounds by:

1. Grazing selectively. Diet selection skills involve cautious sampling, consuming a varied diet and consuming plants in a cyclic, intermittent or carefully regulated pattern.

2. Possessing internal systems to detoxify or tolerate ingested plant toxins.

Primary photosensitisation appears to be more commonly associated with monoculture situations and the ecological interactions which may have contributed to this animal health event are not well understood. Whilst the ability of sheep and other grazing animals to protect themselves from the harmful aspects of plants has been studied at length (Launchbaugh *et al*, 2001), these protective grazing strategies tend to be less available to animals grazing pastures with less species diversity and the mix of plant species available to grazing livestock varies with

SUMMARY OF PROPERTY SAMPLING

Collection Date	Animals affected	Samples Submitted	BTV Exclusion	Pasture type	Aphids noted	Frozen pasture available	Liver pathology noted
					✘		
29/09/17	Lambs only (on ewes)	Blood	✓	Rye Pasture	✘ (Suspected scabby mouth)	✘	✘
3/10/17	Ewes and lambs	Blood	✓	Vetch and medic	✓	✓	Mild
3/10/17 11/10/17	Ewes, lambs, wethers	Blood Blood, liver	✓	Vetch	✓	✓	Yes
3/10/17	Lambs	Blood	✓	Medic	✘	✘	Mild
3/10/17	Ewes and lambs	Blood	✓	Medic	✓	✘	Mild
12/10/17	Ewes and lambs	Blood liver	✘	Medic	✓	✓	Mild
12/10/17	Ewes and lambs	Blood liver	✘	Medic	✓	✓	Mild
12/10/17	Ewes and lambs	Blood liver	✘	Medic	✓	✓	Moderate/ chronic
28/9/17	Ewes & lambs	Blood liver	✓	?	?	✘	Yes

PIRSA sample submissions for Eyre Peninsula photosensitivity cases

seasonal conditions, agronomy, ecology, soil nutrition or interactions between any of these.

In cases where animals have chronic liver damage (possibly by longer term exposure to toxins) their ability to detoxify recently ingested material will be compromised, and toxicity symptoms will be more pronounced and slower to resolve. Cereal Hay without green matter is suggested as the safest feed for any photosensitivity affected animals (Robson, 2007). To safeguard against photosensitivity risk situations, one strategy may be to allow access to cereal hay prior to grazing risky fodder, and to continue feeding palatable hay throughout the risk situation.

Since a major role of Primary Industries Departments in Australia is to detect and respond quickly to new large scale disease events it is pleasing that producers in SA quickly contacted trusted sources of advice, including PIRSA. Producers tend to trust sources known personally to them and in this case the local PIRSA Animal Health Officer was very quickly involved. Producers were able to access funding to assist with investigations and testing and PIRSA Animal Health liaised with producers and collected samples for subsidised veterinary pathology testing and diagnosis. SA *Sheep Connect* (an industry / government partnership) also organised a helpful webinar at short notice with a presentation from Dr Colin Trengrove, and this benefitted regional producers across South Australia.

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PIRSA would also like to acknowledge the advice and assistance of Dr Rob Suter, Victoria DPI, and Dr Kym Perry, Research Scientist / entomologist SARDI.

GENERAL

Koala Health Hub

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Figure 1.

Disease is recognised as a potential threat to koalas, although there is no comprehensive understanding of the impacts of disease across populations. Without consistent collection, analysis and reporting of disease information it is difficult to reach consensus about whether disease is a cause or symptom of population decline and what implications disease has for resilience and recovery of koala populations. NSW Office of Environment and Heritage (OEH) recognises that samples and data collected from sick and injured koalas by veterinarians, koala rehabilitation facilities and field studies are an extremely valuable resource that, for many valid reasons, is not currently being used to its full potential. OEH also recognises that private practitioners often support koala care with limited training or financial support and that there is a need to better support these activities.

As a first step to addressing these issues, OEH is consulting widely with people involved with koalas to identify who is collecting samples and data from koalas in NSW, as well as to identify issues and a series of potential models or protocols for coordinated storage and sharing of such data and samples. The Saving Our Species Iconic Koala Project identifies the need to formalise a protocol for collection, analysis and reporting of koala disease samples and information as a key action to help secure koalas in the wild.

OEH has engaged the Koala Health Hub (KHH), University of Sydney to conduct and report on the first stage of this project due to their expertise in this area. This activity comprises one component of a broader program, the aims of which are:

- To develop a catalogue of existing sample stores and databases with information related to koala disease in NSW.
- To develop standard protocols for the collection, storage, analysis and reporting of information on koala disease in NSW.
- To identify options for koala disease data collection and sharing to facilitate systematic research and effective conservation approaches.

The Koala Health Hub is an initiative of the University of Sydney to benefit koala welfare and conservation by connecting researchers across the country with people on the coalface of koala care and management. Through communication and collaboration, and provision of essential services and expertise, we aim to create an inclusive, diverse and innovative source of support for koala health management in Australia. The Koala Health Hub was founded on public donations collected by the Koala Park Sanctuary, West Pennant Hills; and receives additional support from the Koala Preservation Society, Australia. Its aims are to:

- Create a national network of expertise, support, and co-ordination to those responsible for koalas in care or in the wild;
- Provide free- of- charge diagnostic and clinical support to practitioners caring for koalas;
- Provide opportunities for training in the management of diseased koalas;
- Generate collaborative research benefits to aid further improvements in koala health, conservation and welfare.

If you care for koalas and would like to engage with the Koala Health Hub or the OEH project, please contact **damien.higgins@sydney.edu.au** and visit the KHH website at **www.koalahealthhub.org.au**

Figure 2: Necropsy training workshop at 'Friends of the Koala'

SMALL + WINNER!

Ketofol as a General Anaesthetic Induction and Maintenance Agent

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Ketofol is a ketamine-propofol admixture. The first randomized double-blind study comparing ketofol with propofol was a study of human paediatric patients undergoing cardiac catheterization, published in 2005. It is now used widely in Emergency Department procedural sedation and analgesia, especially in paediatrics.

This popularity is due to its ability to provide rapid and effective sedation and analgesia with short, smooth recoveries and good maintenance of haemodynamic parameters.

In my hands, ketofol has been a very reliable, well tolerated and 'safe' (if there is such a thing as a safe anaesthetic) induction and maintenance agent.

Obviously it is preferable to have access to a syringe pump to administer ketofol as a constant rate infusion, though a dedicated and competent veterinarian or veterinary nurse can manually administer it.

Ketamine and propofol have been co-administered with the intent of counteracting the undesirable individual effects of these drugs. Prospective, randomised, controlled, 'blinded' studies have shown that there are benefits to using the mixture, both in dogs and cats, as opposed to using propofol alone, namely superior haemodynamic parameters.

Ketamine, a phencyclidine derivative, stimulates sympathetic efferent activity, resulting in an increase in heart rate and arterial blood pressure. It is unique amongst anaesthetics in that it maintains or increases cardiac output as a consequence of this sympathomimetic activity.

Ketamine given alone intravenously can cause muscle rigidity, convulsions and violent anaesthetic recoveries. These effects can be countered by co-administration of a benzodiazepine or propofol.

Propofol, an alkyl phenol with sedative-hypnotic properties causes cardiopulmonary depression. Propofol administration is generally associated with decreased pulse rate and arterial blood pressure as a consequence of dose dependant lowering of sympathetic tone. It is also a direct negative inotrope. The addition of ketamine to propofol helps offset the myocardial depressant effects of propofol.

Ketamine at clinical doses has minimal effect on respiratory drive. Propofol produces respiratory depression (and apnoea if given too rapidly) during induction of anaesthesia.

Benefits seen with Ketofol compared to Propofol alone include:

- Ketofol allowed tracheal intubation with a smaller propofol dose than when propofol was administered alone.
- Ketofol use was associated with higher pulse rate and mean arterial pressure.
- Quality of tracheal intubation and induction of anaesthesia was assessed as being of superior quality with ketofol.
- The lower dose of propofol required for induction of anaesthesia results in less respiratory depression.
- Ketamine has analgesic effects, so that the ketofol mixture provides analgesia whilst propofol alone has no analgesic properties. In a clinical trial where ketofol was used as total intravenous anaesthesia (TIVA) in cats undergoing ovariectomy, all cats had low post-operative pain scores and no rescue analgesia was necessary, even when no other analgesic drug was administered.

Contents

Ketamine and propofol in a 1:1mg/mL combination mixed in the same syringe has been shown to be physically compatible for at least 3 hours.

Table 1 describes the constitution of the admixture and dose rates.

In summary, this is how Ketofol is made up:

1. Draw 10mL of propofol up into a 20mL syringe.
2. In a 10mL syringe, draw up 1mL of 100mg/mL ketamine and 9mL of normal saline and mix.
3. Add the ketamine/normal saline mix (which is now ketamine 10mg/mL) into the 20mL syringe that already has 10mL propofol in it.
4. Mix well and it is ready to use.
5. Repeat in 2nd 20mL syringe if you wish to use all of the 20mL propofol vial.

Assuming pre-medication with acepromazine and an opioid, dosing is as follows:

1. Induction: 0.4mL/kg of mix
2. Maintenance: Constant Rate Infusion (CRI) 0.03mL/kg/min.
3. Obviously rate can be titrated to manage required depth of anaesthesia.

Table 1. Ketofol constitution and administration

Preparation of 20mL syringe			
Drug			
Propofol 10mg/mL	None	10mL	10mg/mL
Ketamine 100mg/mL	1mL in 9mL NS	10mL	10mg/mL
ie. 5mg/mL of each drug in syringe mix			
Induction			
4mg/kg=0.4mL/kg of mix	[equates to 2mg/kg of each drug]		
Maintenance CRI			
20mg/kg/hr=2mL/kg/hr	[equates to 10mg/kg/hr of each drug]		
0.33mg/kg/min	[equates to 0.17mg/kg/min of each drug]		
CRI of mix 0.03mL/kg/min			

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SMALL

A Cost Comparison of Various Anaesthetic Induction and Maintenance Agents

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With the increased availability of generic formulations of propofol and midazolam, I thought it would be an interesting exercise to do a cost comparison of the various general anaesthetic induction agents, as well as costs of maintenance anaesthetic agents.

Table 1 compares the costs of the various agents.

The following assumptions were made:

- Patients were 10kg dogs
- Patients had been pre-medicated with acepromazine and an opioid such as methadone or morphine.

Notes:
Midazolam offers several advantages over diazepam, namely:

- It is water soluble and absorption is excellent after intramuscular injection, making it useful for use in combination with ketamine in fractious patients.
- It is safer for intravenous use in cats, as propylene glycol as a carrier agent in diazepam has been documented to cause problems such as Heinz body anaemia and the solvent is very irritant to the endothelial lining of veins and causes transient hypotension

Table 1 the cost of various volatile agents used in maintenance of general anaesthesia, with costs shown below in AUD$.

Maintenance Agent Cost Calculations							
Agent	mLVapor/mL Liquid	Agent cost	Bottle size (mL)	O2 flow (mL/min)	% Agent	Cost per minute	Cost per hour
Sevoflurane Branded	183	$336.93	250	1500	3.5	$0.39	$23.20
Sevoflurane generic	183	$252.00	250	1500	3.5	$0.29	$17.35
Isoflurane	195	$57.10	250	1500	2	$0.04	$2.11
Desflurane	207	$469.01	240	1500	8	$1.13	$67.97

Reproduced with permission from Veterinary Anesthesia & Analgesia Support Group; vasg.org

I have included 'Ketofol' as an induction and maintenance agent.

Table 2. Cost of injectable anaesthetic agents

Drug	Volume Purchased (mL)	Cost Per bottle/ box vials	Cost per mL	10kg Dog Dose in mL	Cost Volume 1	Cost Volume 2	Total Cost
Propofol[a] 200mg/20mL	100	$7.00	$0.07	4.0	$0.28		$0.28
Aquafol[b] injection 100mL (Propofol 28 day)	100	$123.80	$1.24	4.0	$4.95		$4.95
Ketamine[c] 100mg/mL	50	$18.60	$0.37	0.5	$0.19		
Diazepam[d] 5mg/mL (PamLin)	20	$27.10	$1.36	0.5		$0.68	$0.86
Midazolam[e] 5mg/mL (B.Braun)	10	$8.40	$0.84	0.5		$0.42	$0.61
Alfaxalone[f] 10mg/mL	20	$86.30	$4.32	2.0	$8.63		$8.63
Thiopentone[g] 50mg/mL	100	$47.20	$0.47	3.0	$1.42		$1.42
**Ketofol 10mg/mL (Induction)	N/A	N/A	$0.06	4.0	$0.24		$0.24
**Ketofol 10mg/mL (Maintenance - 10kg Dog)	Time:	60 minutes	30 minutes	15 minutes	10 minutes		
**Maintenance CRI 20mg/kg/hr	Volume (mL):	20.0	10.0	5.0	3.0		$3.00
** = Maintenance CRI 2mL/kg/hr	Cost/Unit Time:	$1.20	$0.60	$0.30	$0.18		$0.18

a. Propofol Lipuro 1% MCT/LCT AMP 200MG/20mL B.Braun
b. Aquafol Injection 100mL Ceva
c. Ilium Ketamil 50mL Troy Laboratories
d. PamLin Injection 20mL Ceva Animal Health
e. Midazolam 5mg/mL 1mL Pkt 10 B.Braun
f. Alfaxan-CD RTU 20mL 10mg/mL Jurox
g. Thiobarb powder+water for inj 5G powder + 100ML SWFINJ Jurox
*** The total cost in the midazolam and diazepam rows refer to the cost of ketamine as well as the benzodiazepine.**
****See Ketofol preparation sheet**

SMALL

Acquired urinary sphincter mechanism incompetence (USMI) in a queen

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Millie is a Burmese neutered female, acquired at 2 years of age. She presented with urinary incontinence and severe gingivitis, the latter confirmed to be due to severe periodontitis, a not unfamiliar picture in the pedigree cats I see.

Gross examination of the genitalia and urinalysis was unremarkable, incontinence occurring only when asleep. No dysuria was noted. At the time (2014) I found very little in the literature relating to hormonally-responsive urinary incontinence in neutered queens, but based on experience in dogs I commenced phenylpropanolamine (Propalin® 40mg mL) 0.8 mL TID. This gave short-term relief confirming the diagnosis, but it presented compliance issues. Next port of call was an oestrogenic, and further to Richard Malik's suggestion, a gelatine capsule containing both phenylpropanolamine (liquid) and Incurin® (0.25mg) was given daily. Initially this was effective, a 1 week course of this giving 6 month's resolution before recurrence. However, it was a messy impractical solution – I guess we should have split the phenylpropanolamine capsule and done it all dry!

Many years previously in the days of stilboestrol it was common for dogs to go many months after a short course of this medication, suggesting that the mechanism was based on more than hormonal shortage, or that stilboestrol had an exceedingly long persistence in the body. In computer terms, to me it seems as though it sets a receptor back to its default position though I will leave the mechanism to be debated by others with more academic abilities. Anyway, the long relief achieved suggested that the Incurin® was the effective part of the combination so I decided to just treat with this in short courses of 7 days, avoiding further medication until incontinence recurred.

The time to recurrence varied between 1 and 3 months, and we then moved onto regular dosing reducing frequency until we controlled it on ¼ tablet (0.25mg) weekly.

Based on a discussion on the ISFM forum, we tried a GnRH implant (Suprelorin®) in its smaller 4.7mg size. The cat is currently symptom-free 7 months after implantation.

Discussion

USMI does occur in queens though it would appear to be uncommon. In dogs, I always used a positive response to phenylpropanolamine as a quick confirmation of diagnosis, precluding the need for more-complex urinary tract investigations other than urinalysis checking for secondary UTI. I suggest this may be equally valid in cats.

Further to using the deslorelin implant, I found the following paper where a similar long-term resolution of incontinence was achieved:

Effectiveness of deslorelin acetate subcutaneous implantation in a domestic queen with after-spaying urinary incontinence. *J Feline Med Surg*. April 2014; 16(4):366-8.

Reference

S.P. Gregory, Mar-Apr 1994, Developments in the understanding of the pathophysiology of urethral sphincter mechanism incompetence in the bitch, *Br Vet J*, 150, pp. 135-150

eBook download:

C&T No. 5685 (Issue 291 June 2018)

Read My Life as a Vet

GENERAL

Veterinarian Alleges Animal Welfare & Surgical Principles Being Violated During Shark Catch & Release Programs in Australia

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Surgical implantation of transmitters into the body cavity of sharks is being performed by non-veterinarians, a situation of great concern for those interested in animal welfare.

CVE Member and C&T contributor Peter Kerkenezov believes the veterinary profession should weigh in on this subject matter: 'Currently marine biologists are performing atrocious procedures on all sorts of marine animals'.

Due to space constraints, please go to the complementary eBook version of this issue to download the submission:

eBook download:

Something Fishy About Shark Tagging Program, The Master Mariner, Dec 2015

Submission Number 08 (by invitation) to Environment and Communications References Committee re Shark Mitigation Strategies (Federal Senate).

Total submissions to date = 54

Figure 1. Non-sterile invasive abdominal surgery to insert a foreign body (V16 69 kHz acoustic tag) without anaesthetic into a shark is performed underwater by non-veterinary surgeons.

Figure 2. The recent death of an Orca in Canadian waters all but confirms what many people have been saying: That the risks of invasive tagging is significant, and in many cases leads to undue stress, infection, maiming and death of the recipient animal.

SMALL

Get a Grip

When Slip-Sliding-Along Ain't Cool

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Modern house floor surface and coverings and our pets are often a bad mix.

I see anything from minor or major disruption to households and to the pets where these modern shiny floors cause pets major problems resulting in a compromise for welfare in terms of limiting 'the where, the when and the degree' by which pets can chose to interact with their owners. Some animals will simply choose to withdraw into safer zones resulting in less spontaneous social interaction and greater stress and anxiety for the pet. Some pets will struggle on, but many owners of elderly dogs often seem overwhelmed by how difficult it is for their dogs to walk around the home without slipping and sliding, to the point the owner is considering euthanasia. Younger dogs often seem to become airborne on the wooden/tile floors then slide and/or slam into a door or furniture resulting in injury.

Figure 1. Anti-slip underlay

Solution 1 is easy and cheap

A trip to Bunnings to purchase as many metres of anti-slip underlay as needed by the owner in the colour of their choice. cf Figure 1 (Top, fourth and fifth rung products).

Owners then can create a cheap washable replaceable walkway against the wall or create transition walkways and paths to other areas to allow the pet to traverse across the slippy floors. The same grip principle applies to non-slip slippers and boots for pets; but my clients find the slipper stays on but the dog just rotates around in the sock and then the sock falls off. The home-made anti-slip mat runways, custom cut to the width and length needed for each house and for each pet, work much better.

Solution 1B

For larger or heavier dogs, you may need to use a carpet runner on top of the anti-slip underlay for better traction. Bunnings also sells a variety of about 60cm wide rolls of carpet runner in various colours and designs you can have cut to the required length (about $19 a metre long) to then sit on the underlay so carpet mat/runner does not move. **bunnings.com.au/our-range/paint-decorating/flooring/carpet/runner**.

Solution 2

In April, I came across a new non-slip pet product – Paw Friction-from Silverglide Australia.

My initial reaction was negative and that it could cause reactions on the pads or if licked.

But if it didn't cause any issue then I wondered about it as an option both indoors and outdoors for dogs. The movie

Contents

Figures 2A and B. Silverglide Paw Friction

Figure 2C. Image courtesy of Dr Kim Coyner, (Dermatology Clinic for Animals, USA)

eBook video: The movie shows Max before and after application of PawFriction

provided by the company shows a huge benefit in the dogs the product is applied to, so do watch the link:

I asked around and got the following feedback from a vet colleague:

> ***"It would not replace boots but did seem to help my elderly dogs walk on my vinyl floor with less slipping. They didn't lick at it or seem bothered and it was easy to apply though a bit messy. Downside is it only lasts about a week and has to be reapplied.***

Given the distress and exhaustion otherwise healthy old dogs experience trying to get weak legs under them, the product is still worth considering.

My own additional thoughts were that if Paw Friction is non-irritant and safe to use then, besides being helpful to slipping pets, the additional aid might be in allergic pets.

- If the product adheres safely to the pads and coats the pads… AND
- If it is the lipid-poor foot pad that allows more hydrophilic antigens like pollens to be absorbed in through the pads, would this paw product work as an allergen barrier for those allergic dogs who don't tolerate boots?

Solution 3

Whilst there are many dog boots/shoes around now, many are not so good indoors.

Thank you to Emily Rothstein Bassell for the link to this pack of 12 disposable rubber slip-on disposable boots that claim to give traction on slippery surfaces **pawzdogboots.com/pillars-of-health/**.

Don't leave the product on the dog unsupervised as it could represent an intestinal blockage risk if eaten and obviously don't leave it on for too long so that the dog runs the risk of developing circumferential pressure sores.

Solution 4

Toe Grips.

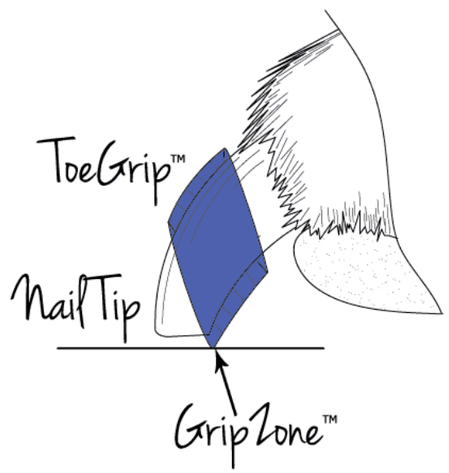

Image sourced at **toegrips.com/**

These are *NOT* suitable for dogs with sore nails or who hate their feet being touched but for other dogs they

work well. Some good videos on their site. You apply a lubricating gel and push the grip on firmly. You might need to stretch the grip open with a tweezers to help slip on over the nail. Really have to pick your patient for these ones but they will work for particular dogs depending on the cause of the dog's weakness. Don't allow your dog to chew and eat them either!

Solution 5

Leg braces have become mainstream in vet medicine now for injuries and rehab.

Several online sites offer various options on braces for one or both legs to enable stability in rising.

Additionally, we used to advise owners to protect their own backs and the pet's back by running a large sheet or towel under the dog's hind quarters and belly to help hoist the dog up safely into a standing position, whereupon most dogs can then move around on their own. Now owners can also purchase a variety of different commercially made body slings on ebay from $13-$40 which is very affordable but always check any seller out before you commit to buy: bit.ly/bodyslings

Slip-sliding spotlight on cats

If we are rethinking how we provide correct litter tray design, appropriate indoor ambient temperatures and cat carriers for cats in general then we need to address cat-specific issues for feeding as well.

Cats don't show their gait problems on slippy floors as clearly as dogs do, but cats do have problems just the same with wooden/tiled floors and these issues are significant when it comes to the cat's access and use of feeding bowls and litter trays.

Old cats will often lose grip with either their back or front legs respectively as they push into or jump back out of the litter trays. We solved that for our cat by having some Granny Grip materials on the 'runway' approach to our cat's litter trays.

eBook download:
Issue 284 September 2016 C&T No. 5568
Urination Issues for Cats - When the Bladder is the Victim, not the Primary culprit;When traditional litter trays need a new shape!

The biggest issue is however *feeding time*.

The elderly cats get exhausted holding their stance to avoid slipping whilst they eat. Often, they are so exhausted that they stop finishing their meal and start picking at their food in small bursts of activity.

Just putting the bowl on the grip material doesn't solve the issue; you also have to provide a large enough mat of the grip material *to accommodate both the bowl and the cat* so the cat can stand on the mat and eat from the bowl also on the same anti-slip mat. Suddenly your capricious appetite cat scoffs his whole bowl of food down in one session.

Very elderly cats, or cats with elbow arthritis, may need even more help than just the bowl on a large anti-slip mat. Those extra needs elderly cats can assume a kneeling position on their carpi that is distressing to see. For those cats, the food bowls need to be elevated up – but perhaps not in the straight-way-up we do for dogs.

An elevated feeding tiered/layered design to allow the cat to stand upright and lean into the elevated bowl was constantly preferred by one of my own elderly Siamese. I only found this out by accident when I found him kneeling to eat and rushed to make him a stand by grabbing 3 old books of odd widths tapering to a flat top that was narrower than the base. This base was then covered with a large sheet of anti-slip. He constantly chose this tiered stand even if other options were available so there must be something about it that allows a more comfortable stance and feeding action. It was such a joy to see him enjoy eating again by just the most basic modification to his environment. Several clients have commented how much better their elderly cat is once the food tray was elevated for their pet.

SMALL

Dog parasites are going for the fireworks!

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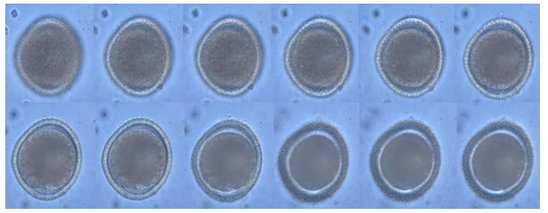
As a parasitologist and an educator, I am tasked to challenge and possibly disrupt dogma and general assumptions. Topics that I like to tackle revolve around dogmas our students feel close and personal about, because they revolve around their beloved dog or a cat. By the way, it goes beyond student cohorts, to practicing veterinarians. Reflecting on our own actions, if guided by evidence or assumption is always good. A reflection on the use of antiparasiticides is one that fits well within the sustainable pet ownership and comparisons with antibiotic use and stewardship to combat AMR (antimicrobial resistance). The text that follows is challenging not just for student but also for owners and veterinary practitioners. Parasitology has one major advantage over many other infectious disease disciplines - the testing is as simple as 1-2-3 (at least for the common roundworm). We all learned how to do a faecal float right? So we can test in no time if the animal needs to be treated for a parasite or not. I question why don't we do it? Why giving an antiparasiticide to an animal that has no evidence of parasitosis is considered normal? Here is a reflection that brings much needed evidence from data published by colleagues in the Netherlands.

Do you own a dog?

There's a good chance that you do, because 39% of households own a dog in Australia and there are estimated to be 4.2 million pet dogs.[1]

If you own a dog, no doubt you are constantly told that dogs have worms and 'you need to do something about it'. 'To do' means: to use worming tablets or Spot-on products (anti-parasiticides or anthelmintics) to remove the worms from their gastrointestinal tract and do this on



regular basis, perhaps every 3-4 months. As an obedient dog owner, you most likely do that. The anti-parasiticides are special types of antibiotics paralysing or killing those parasites. These chemicals are generally very safe. On the other hand, as far as chemicals, common sense dictates: if there is no worm, there is no need for chemical treatment. And they can be expensive!

Does your dog have dog roundworm?

If the answer is YES: go ahead and de-worm them, but if there is zilch, give it a miss. All veterinarians should know how to test for it. It's easy!

What do we know about this dog roundworm egg (known to veterinarians as Toxocara canis) pictured above?

It is here, but the latest census (2008) found evidence of the dog roundworm only in 1.2% of 1,400 poo specimens from dogs in Australia.[2]

They looked at dogs that visit veterinary clinics and found a mere 0.4% of them were infected. In dogs that did not visit veterinary clinics, it was only marginally higher at 2.4%. Those are really low numbers. So the chance that your dog has this worm is rather slim.

Hang on, isn't the reason for such a low number the frequent use of antiparasiticides?

Until now, I did not have an answer to that.

Now, a group of veterinary parasitologists from Utrecht University in the Netherlands has undertaken a bold study to investigate if it is actually worth using routine antiparasiticides for dog roundworms in owned dogs.[3]

The study neatly shows that dog roundworm is rare and routine de-worming is not needed for most adult non-breeding dogs. Best is to just occasionally test them and treat only those that need it.

The Dutch team recruited around 1,000 owned dogs and tested them for dog roundworm every month over 3 years. Normally they would be giving these dogs the anti-parasitic pill 4 times a year, this time they did not. *The conclusion is: over a 3 year period, two thirds of dogs never had faeces testing positive for dog roundworm.*

Dog roundworm is an intestinal worm. In the intestine of the dog, roundworms mate and each female roundworm will be shedding thousands of eggs over its lifespan. The eggs will appear in the dog faeces - for veterinarians to detect them. There are a few twists to this life cycle. The dog roundworm has lived with dogs for many years, possibly millions of years, with canine ancestors and the host and parasite co-evolved. By co-evolved I mean, adapted to the dog's biology to maximize its reproductive success. Essentially, making sure that it gets from mum to its off-spring safely. To do so, dog roundworms don't just end up in the intestines of their victims, they undergo migration through the dog's organs. In adult dogs (older than 6 month), the migrating parasites are arrested in dogs' organs until there is some trigger to allow them to progress further. The natural trigger is pregnancy of the victim. In the pregnant dog, the parasite will start to migrate again, but this time also to the unborn pups (via placenta and milk). From the parasite's point of view, this is perfect. Pups are infected even before they were born.

Back to the non-pregnant, adult dogs - are there any triggers? This time, again, the Dutch study provides some interesting insights.

Winter seemed to be the trigger, because there were more positive samples for dog roundworm in winter compared to summer months. It is further suggested that some type of stressor is behind this winter phenomenon.

In Europe the winters are cold and coincide with the festive and hectic Christmas season meaning shorter walks, less time for the pet and all this followed by the New Year celebration with ubiquitous fireworks displays.

You might be pondering what fireworks have to do with parasites?

To be honest, I was surprised as well but I do actually find some truth to it.

It is no secret that dogs do not enjoy fireworks. But the parasites probably enjoy the fireworks! The Dutch authors indicate that fireworks, in fact, play a role in increased presence of the dog roundworm. Fireworks are stressful to dogs, but whether it implies reactivation of the arrested roundworms is up to some scrutiny. It is definitely a worthwhile hypothesis.

All this said, dog roundworm is a serious parasite that should not be taken lightly. It can find its way to us humans as the migrating parasite through our bodies as well. In pregnant dogs and puppies, it is one of the top pathogens and every veterinarian will advise how to control it. In Australia, the ever-present cat fleas and danger of canine heartworm requires their prevention with drugs that often kill dog roundworms as well. So, most likely, you and your dog are safe.

eBook download:
Please go to the eBook for references.

SMALL

Practice tip: Packing for feline caudal clearances

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Doing something for a long time does not make it right nor proper. However, I have been doing routine caudal clearances in cats since last century. My consideration is that if you remove only the diseased teeth, one at a time, I can guarantee the cat won't tell anyone when their teeth hurt again, and the owner may well delay or deny further treatment. So one out-all-out has become my default procedure. 'Gummy' cats also seem to live longer and certainly have reduced oral pain. Your method of removal of all the teeth caudal to the canines will be different to mine. I do remove diseased canine teeth but not at the same time if I can help it - the cat seems to take longer to figure out how to eat again. The packing procedure I have developed removes the need for stitches in the gum (tricky especially since most cats have rather friable gums by this stage) and there is nothing for them to try to scratch out of their mouths.

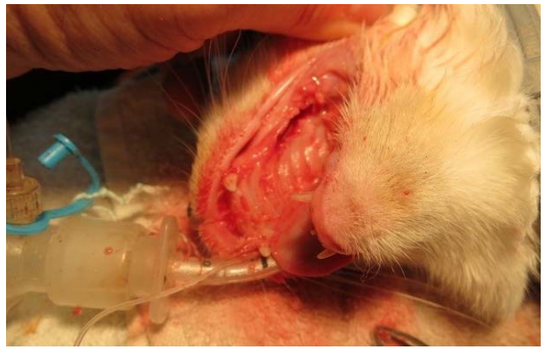

Figure 1. Large deficit visible immediately post extractions.

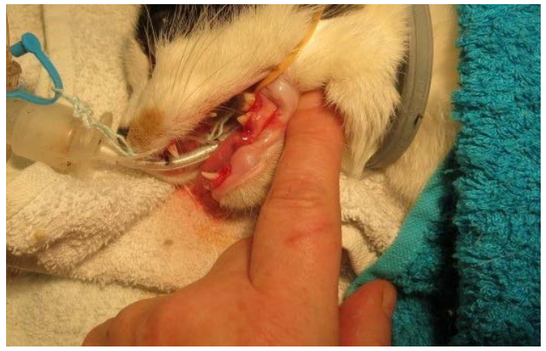

Figure 2. Rubber band holding in endotracheal tube. Tampon used as packing.

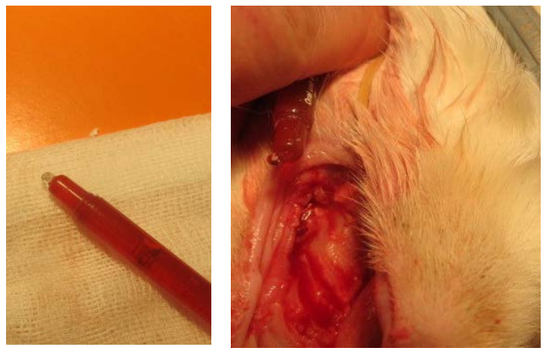

Figure 3. Poloxamer gel going into cavity. Poloxamer gel from BOVA at room temperature (gel). It is the same product used in paediatric Coloxyl® drops. It is used to pack the sockets first, followed by the gelfoam.

I do these as a front line procedure commonly – cats don't like repeat visits to your clinic (or mine) and every anaesthetic carries a risk. This is a 4-year-old DSH female from a 'rescue group' presented with periodontal osteitis, gingivitis and halitosis. All except the canines are removed.

The most discomfort seems to come from the ET tube (the surgery takes up to 90 minutes).

About 1 in 20 cats get food stuck in the cavities, and that will present either as smelly breath, or not eating properly after a couple of days. Those I can usually just flush out in the consult room

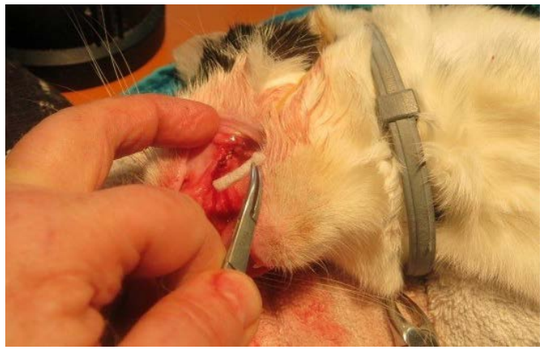

Figure 4. Gelfoam sliver packing placed after poloxamer gel.

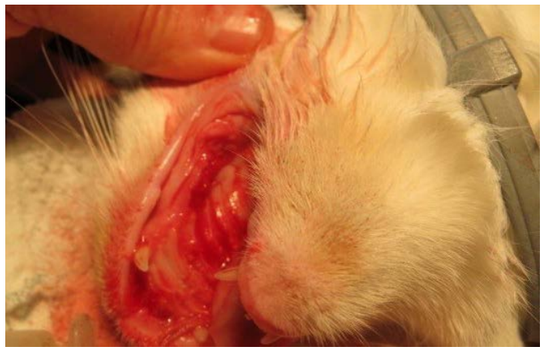

Figure 5. Appearance immediately post procedure.

VERY occasionally, a piece of bone breaks off, or a root is not completely removed (called 'dry sockets' by human dentists) and those need a sedation to fix.

I do use a long acting amoxi injection, and vary the post-op pain relief between triamcinolone injections (Triamolone TM) 0.1 - 0.2 cc sq. or meloxicam orally for a few days. Cats over 8 years old always get an IV drip intra-op. Younger cats get some IP fluids.

Cats can still catch rats and even disembowel them, so other than not being able to chew on bones or grass, there is no downside. The medical upside is pain free oral health.

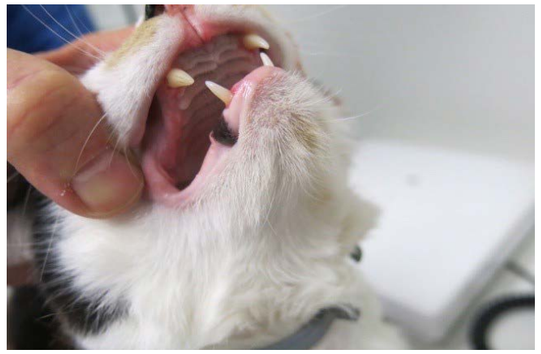

Figure 6. Appearance 3 weeks post procedure.

SMALL + WINNER!

Concurrent Diabetes and Hyperthyroidism

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C&T No. 5697

These 2 endocrine diseases are encountered regularly in cats, however, not as often in a cat at the same time! My own cat Midas is one of the lucky few, and through his own diagnosis and treatment I discovered some of the challenges this tricky situation can present.

Diabetes mellitus occurs at an estimated rate of 0.74% cats in Australia, and hyperthyroidism occurs at a rate of approximately 10% in cats over 10 years of age. This makes the chance of the 2 conditions occurring together unlikely, though certainly possible, especially as the cat ages. In humans there is a link between hyperthyroidism and type 1 diabetes mellitus (both being primarily autoimmune diseases) but the pathogenesis for diabetes and hyperthyroidism is different in cats and having the 2 conditions concurrently does not appear connected.

Midas is our 8-yr-old MN fluffy ginger, diagnosed 18 months ago with diabetes mellitus. We settled into a

routine of 3 international units glargine BID, with a diet of Royal Canin diabetic dry food. I was 'monitoring' him at home. This was probably not as ideal as it could have been but he appeared happy and stable in weight.

After approximately 12mths, I tried him on Purina diabetic dry as I had heard this was a lower carbohydrate option (23%) than the Royal Canin (32%). Around this time he started to develop softer/smellier stools and occasionally vomited. I noticed I was needing to feed him more to maintain him at a stable weight. The blood glucoses I was obtaining at home had started to gradually creep up. Eventually we went back to the Royal Canin Dry but these issues persisted until I finally took him in for wellness bloods and a urinalysis.

His bloods were good except for the glaring Total T4 result of 150nmol/L! (Ref range 11- 46nmol/L). Sure enough, on palpation there was a unilateral thyroid enlargement. Thankfully his renal function looked good.

Diagnosis

There is a lot of overlap in clinical symptoms of diabetes mellitus and hyperthyroidism (i.e. weight loss with good appetite, polyuria/polydipsia, GI signs) and so it could be quite easy to assume one or the other condition was worsening unless the clinician was open to the possibility of 2 conditions occurring together and running appropriate tests (including careful thyroid palpation).

A sufficiently elevated TT4 in combination with compatible clinical symptoms, i.e. palpable thyroid nodule plus the presence of a loud fast heart on auscultation, is usually sufficient for a diagnosis of hyperthyroidism in a diabetic cat, though a free T4 by equilibrium dialysis, or a TSH determination, could also be performed or the TT4 repeated after a month if results were more in doubt.

In the opposite situation (a cat already hyperthyroid), a sufficiently elevated blood glucose and glucosuria would indicate diabetes mellitus. If results were borderline, unfortunately fructosamine levels can be artificially lowered by hyperthyroidism and are not advised.

Hyperthyroidism is a known cause of insulin resistance, and it is possible if the hyperthyroidism can be controlled, then the cat may go into remission.

Treatment

There are 4 treatment options for hyperthyroidism

1. *Hills Y/d® diet:* not suitable for diabetics (too high in carbohydrates)
2. *Carbimazole/methimazole:* commonly used medical treatment – possible to use with diabetics, as long as no adverse effects i.e. vomiting/diarrhoea/loss of appetite. Will mean ongoing treatment and regular blood tests to keep things in balance.
3. *Thyroidectomy:* for reasons unrelated to diabetes has fallen out of favour. If unilateral thyroidectomy is performed there is a high chance of recurrence from the contralateral gland in the future (although this can take several years to occur).
4. *Radioiodine:* in general considered gold standard treatment for hyperthyroidism, with greater than 95% chance of cure with one treatment.

When investigating radioiodine treatment, *multiple clinics refused to take a diabetic patient.* This was because of:

1. *The perceived increased risk to staff* due to their needing to come into close contact with him twice daily for insulin injections. According to ARPANSA (Australian Government Primary Authority on radiation protection and nuclear safety) guidelines, staff attending to radioactive cats must wear gown, disposable gloves and overshoes. Access is limited to 15 minutes per day per person, i.e. a different staff member twice daily. Ideally, the cat would remain at least at arm's length.
2. *The risk of hypoglycaemia.* If Midas stopped eating or became hypoglycaemic for any reason while in hospital, staff would be unable to offer medical treatment for him while he was highly radioactive.

I did have to evaluate the risk/benefits for Midas but felt in his case I still wanted to proceed and eventually I located a clinic that was willing to take him.

Normally cats would be fasted prior to sedation and administration of the radioactive capsule, however Midas was given a small amount of food and no insulin the morning of administration and there were no problems.

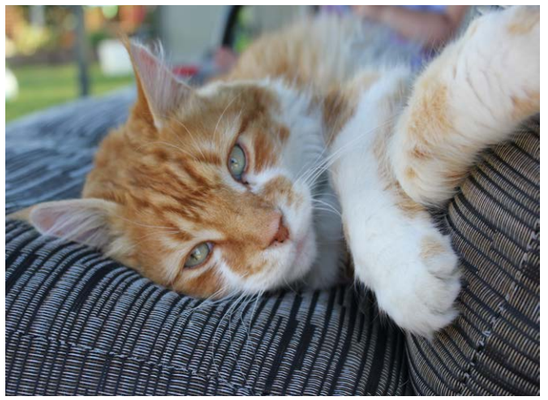
To err on the side of caution (or in this case hyperglycaemia rather than hypoglycaemia), he was given a reduced dose of insulin while in hospital isolation (2 units BID) x 7 days, had no problems with his appetite and had an uneventful stay.

Post Treatment

Once thyroid levels come into the normal range, it was advised that insulin requirements may decrease. However, spot monitoring at home over the next few weeks didn't show a lot of changes.

At 4 weeks post radioiodine treatment, we performed follow-up bloods, which showed a TT4 of 17nmol/L, (ref 11-46) and the rest of the panel was normal. An in-clinic blood glucose curve performed over the day had glucose falling to 3.5mmol/L so I did end up reducing his insulin to 2units BID.

Three months later his stools are back to normal and he doesn't vomit any more. His weight has been creeping back up so he is now on stricter rations! One of the most remarkable changes though has been in his coat – he is now back to a beautiful fluffy boy with no mats.



Discussion

Ah, the benefits of hindsight… My cat Midas probably was developing hyperT4 for at least 6 months by the time it was diagnosed. This underscores the importance of being alert to concurrent disease and of repeating the physical examination and running full blood profiles in addition to glucose home monitoring.

He was probably a bit unusual in that he had no liver enzyme elevations (apparently up to 10% of hyperthyroid cats don't and that his insulin requirements had not changed dramatically prior to diagnosis. I probably should have retested his TT4 or run a free T4 for additional level of confidence in the result, but his TT4 was so high and I could feel a thyroid nodule that I accepted the diagnosis.

I also considered medical treatment for a month prior to the radioiodine to check renal function and any immediate effect on insulin requirements but Midas's urea/creatinine were low normal to begin with, and I felt the 33% reduction in insulin while in hospital would be sufficient to cover any immediate insulin requirement changes.

eBook download:
Please go to the eBook for references.

SMALL

What is your diagnosis?

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C&T No. 5698

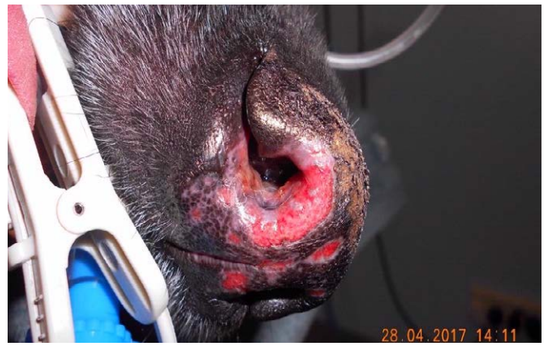
A 3-year-old male German shepherd presented for nasal crusting and depigmentation. No other skin or mucocutaneous lesions were present. There were no signs of systemic disease. He was eating, drinking, urinating and defecating normally.

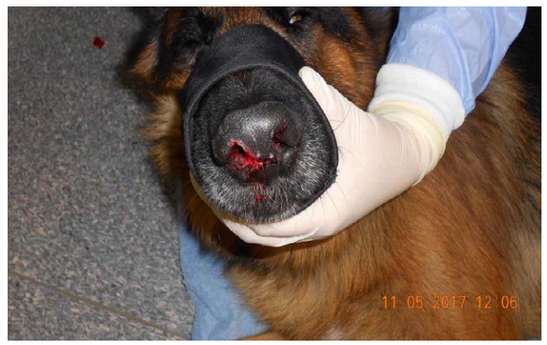
Questions

- What are the diagnostic possibilities?
- How would you investigate this patient further?

Email your answer to

cve.publications@sydney.edu.au Winner of best and most complete answer is entitled to CVE proceeding of their choice. Visit **cve.edu.au/vetbookshop** to peruse our list of titles.





SMALL + WINNER!

What happens in my back yard at night?

Anonymous

Note: The author had reservations about possible negative feedback but we believed it was important to share these observations. Hence, the solution was to publish this anonymously.

C&T No. 5699

Cat doors aren't as secure as you might think!

Earlier this year I attended the CVE's exceptional Small Animal Behaviour Conference and heard Sarah Heath discussing feline behaviour. I learned so much from her talks, but was particularly intrigued by her study of cat doors. It showed – using video evidence – that so-called secure cat doors were being breached by neighbourhood cats, often to the complete ignorance of the owners who simply puzzled about their own cat's behaviour.

Later, I was involved in an episode of redirected aggression. It was around 2am when I woke to the awful sound of guttural cat wailing and raced to the source of the noise. My cat, diligently confined indoors at night, was having a face-off through the window at a tooth-baring neighbour's cat – not so confined. As I raced to the window to shoo the unfriendly visitor away, my cat panicked – turned around and bit me. Hard!

There was regret on both sides. I screamed and limped, he realised he'd mistaken his target, and the neighbour's cat skipped away over the fence. In the morning I realised there had been an episode of *middening* (an Old Norse word referring to unburied stool, acting as a visual and olfactory cue to other cats, a throwing down of the furry gauntlet).

Figure 1. The author's cat with Bushnell camera at back.

I thought it would be interesting to find out if the visitor was visiting often.

I put out the call to borrow a motion activated camera and it was answered. A friend lent me a Bushnell Trophy

Cam HD with no-glow black LEDs. It is very easy to operate – insert 8 AA batteries (they last for months), insert a memory card, set the camera and leave it.

bushnell.com/hunting/trail-cameras/trophy-cam

I got into the habit of downloading the images from the memory card each morning, and discovered much.

Another neighbour's cat was visiting the premises on a much more regular basis than our unfriendly intruder, and weeing in the compost.

The original intruder I had actually set out to photograph, let's call him Fluffy, did visit – less frequently, and would urinate in a different spot, and also leave his calling card (a huge stool) smack bang in the middle of the path that everyone uses.

These two neighbouring cats were never in the yard at the same time each night. It was like they had a roster. And then another cat that I'd never seen before appeared!

During the daylight, my two cats would tread the exact same path as the visitors, sniffing.

There was also footage of a black rat running along the same path.

What to do with this data?

I didn't want to post it on social media as I worry that someone with an anti-feline agenda may use it to make nuisance claims about cats. And I didn't want to confront the neighbours. I had tried diplomacy after the initial bite incident, talking to Fluffy's owners and suggesting that they observe a cat curfew. They may be partially observing this as he visits infrequently. A colleague told me that when her cat suffered a cat bite abscess due to a fight with a neighbouring cat in her own house, she gently let the neighbours know and they subsequently euthanased their cat. I don't want blood on my hands.

But I now have a concrete reason (if I didn't already) to keep my cats in at night and restrict access to the windows. What I love about this camera is that it provides snapshots of animal behaviour without the confounder of an observer. And from an epidemiological perspective, it has given me more information about potential risks for disease spread that I had not considered.

Contents

Figure 2. 'Fluffy' approaches the camera at night.

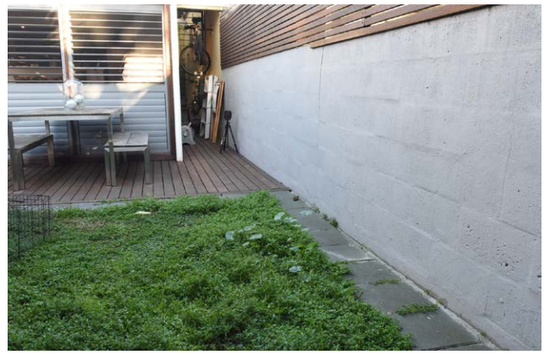


Figure 5. Our yard viewed from the back, showing the position of the camera (adjacent to the wall, right).

Figure 3. 'Fluffy' urinates in the yard beside the cat path.

Figure 6. A random cat visits our yard.

Figure 4. A stool deposited by Fluffy on the bath our cats use.

Figure 7. A bold rat traverses the same path frequented by visiting cats.

GENERAL

Insights into the ecology of ticks can help us to avoid and protect ourselves from ticks

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Figure 1. Researchers at the University of Sydney are studying the roles that wildlife, such as this long nosed bandicoot, play in supporting ticks in and around our cities.

Henry Lydecker is an early career researcher who studies the ecology of parasites and infectious disease in the Anthropocene, as well as the ways in which society shapes and is shaped by infectious diseases. For the last four years, Henry has studied all aspects of the ecology of ticks over the course of his PhD studies at the University of Sydney. In April this year, Henry submitted his PhD thesis titled 'Ticks, hosts, and urbanisation: the ecology of the Australian paralysis tick Ixodes holocyclus in Sydney, NSW'. Besides working with ticks, Henry also studies perceptions and portrayals of infectious disease in published media and social media as part of a project at the Marie Bashir Institute for Infectious Disease and Biosecurity at the University of Sydney.

Introduction

For almost every day of a tick's life, it is following a quest: to find a host animal and successfully feed enough to either moult or reproduce. We use the term 'questing' to refer to when a tick is actively seeking out a host to feed on. The rewards of completing this quest are great, however so too are the risks. Unfortunately, tick bites can lead to many negative impacts for humans and other animals: ticks can transmit pathogens that cause infectious disease, trigger allergic reactions, and inject a toxic venom that can cause paralysis. Ecologists that study ticks endeavour to better understand the tick's quest, and by doing so can help us to avoid tick bites and prevent the negative impacts of these little creatures.

While the vast majority of veterinary practitioners are no strangers to ticks, for those lucky enough to not encounter them, here is an introduction. Ticks are small arachnids that are obligate external parasites of terrestrial vertebrates – meaning that they feed solely on the blood of their avian, mammalian or reptilian hosts. There are around 90 species of Australian tick, however only a few species are known to bite humans, companion animals, and/or livestock with some frequency. The most well-known of these is the aptly named Australian Paralysis Tick *Ixodes holocyclus*, whose bites can cause lethal paralysis in companion animals, and can be found along most of Australia's east coast. In this article, I will shed light on some of the unique aspects of the evolution, ecology, and physiology of ticks, including the Australian Paralysis Tick. I will then discuss how we can avoid ticks and protect ourselves from their bites.

Figure 2. An adult female Australian Paralysis Tick.

Contents

Hard vs Soft ticks?

First, a matter of terminology. There are two main families of ticks, the 'hard ticks' of *Ixodidae* and the 'soft ticks' of *Argasidae*. These two families have very different feeding strategies, and correspondingly divergent morphology. Soft ticks feed briefly, and for many times during their lives. To accomplish this, soft ticks live in or very close to the nests of their hosts. Soft ticks are frequently parasites of animals that live in large colonies, such as sea birds, and are rarely encountered by humans or companion animals. Hard ticks, on the other hand, are the classical ticks that most of us imagine. Hard ticks usually feed two to three times during their entire lives, which can be over a year long. After feeding, a hard tick will moult on to the next stage, and as an adult female it will lay eggs. After each moult, hard ticks increase dramatically in size. Hard ticks feed on almost any vertebrate animal imaginable, even penguins, and these ticks are the ones that are responsible for most bites in humans, companion animals or livestock. While soft ticks are interesting, I will focus on discussing hard ticks, as they are the only ticks that most of us will ever encounter.

A Parasite with a Predatory Past

In the Michael Crichton novel and blockbuster film 'Jurassic Park', fictional scientists extract dinosaur DNA from the blood of mosquitoes in amber: however if such technology was possible, they could have also used ticks. Amber dating back to the Cretaceous has been found containing ticks that are strikingly similar to the ticks of today. Ticks are thought to have evolved at least 100 million years ago, from ancestors that were most likely predators, similar to modern day spiders and pseudo scorpions. Like other predatory arachnids, these ancient precursors of ticks would have likely used a toxic venom to both disable their prey and make it easier to drink up the insides. There is still debate about if the different families evolved their parasitic lifestyle separately, or if the parasitism evolved in a common ancestor, however the predatory origins of these organisms help us to understand the venomous and toxic saliva that modern ticks inject into their hosts.

Australia has a reputation internationally for deadly venomous snakes and spiders, however many people would be surprised to know that the venomous animal responsible for the most human and companion animal suffering in Australia is no snake or spider: it is the tick. All ticks produce some sort of venom, but the Australian Paralysis Tick produces and injects venom in such quantity and potency that even much larger animals can face lethal consequences.

Tick paralysis is the all too familiar condition that can result from a tick bite: ataxia and paralysis leading to possible mortality in dogs and cats. Horses and cattle can also be impacted, but the clinical impacts are rarely lethal, except in calves and foals. The severity of tick paralysis depends on the number and size of ticks biting the host animal, and the size and immune ability of host animal. Wildlife usually become immunized to the toxins after being exposed to low doses from the bites of larval ticks, but it is rare for companion animals to develop similar immunity,except in colonies of dogs exposed gradually to develop antiserum. For obvious reasons, it is not advisable for owners to attempt to immunize their pets in such a manner. Treatment for tick paralysis is very expensive, and while a vaccine is being developed, the best way to prevent tick paralysis right now is to use anti-tick treatments, avoid tick-prone areas, and more frequently check animals for ticks.

What sort of place does a tick like anyway?

To understand the sorts of places where ticks are more likely to be, we need to think from the perspective of a tick. For a tick, life is mainly structured around two things: finding hosts and avoiding dehydration. The first is pretty self-explanatory: ticks need to find hosts to feed on. While some ticks are specialists that focus on just one or a few species of hosts, the Australian Paralysis Tick and other human biting ticks are more of generalists that will feed on whatever they can find. The Australian Paralysis Tick is mostly found in humid coastal forests and rainforests, and there are no clear associations between its habitat and any specific species of host. We know surprisingly little about the host ecology of the Australian Paralysis Tick: we've observed it feeding on many different species of native animals (mostly mammals, but also many birds and reptiles), as well as most introduced mammals. Out of the many species that this tick has been found feeding on, a few stand out as likely important host species because of their abundance, overlapping range, and ability to survive close to where humans live. Several species of bandicoot (imagine a cross between rat, rabbit, and kangaroo) live along the East coast of Australia in the same forests and rainforests as the Australian Paralysis Tick, and they have historically been associated with the Australian Paralysis Tick. Other likely native host species include the Bush Rat *Rattus fuscipes*, the Brush Tailed Possum *Trichosurus vulpecula*, and macropods such as the Swamp Wallaby *Wallabia bicolor*. There are also several species of introduced wildlife that are likely hosts for the Australian Paralysis tick, including both species of introduced rats (*Rattus norvegicus* and *Rattus rattus*), the European Rabbit *Oryctolagus cuniculus*, the Red Fox *Vulpes vulpes*, and of course companion animals like dogs and cats. All of these species of possible hosts live in overlapping ranges, including that of the Australian Paralysis Tick. Determining the roles that these different hosts play in supporting the

Figure 3. A Brush tailed possum (left), house cat (centre), and a long nosed bandicoot (right) can be seen visiting this same tree in the yard of a house in the Northern Beaches of Sydney. Many different species animals that could be hosts for ticks live in and around our cities.

Australian Paralysis Tick remains a major gap in our understanding of ticks.

Thankfully, we know substantially more about the sort of environmental conditions that are favourable for the Australian Paralysis Tick. The drinking habits of ticks are fairly unique among animals: ticks do not usually drink liquid water (fun fact, spiders drink water) and instead depend on absorbing water vapour from the atmosphere or through consuming the blood of their hosts. It should then come as no surprise that the greatest cause of mortality in ticks is dehydration: in order to seek out food, ticks must risk dehydration by climbing out onto exposed vegetation in the hopes that a host will pass by. For much of their lives, ticks hide out in refuges – either dense vegetation or leaf litter – waiting for safe weather to begin questing for hosts.

Australian Paralysis Ticks seem to prefer warm and very humid conditions. Ticks are actually highly resistant to cold temperatures, and are more often limited by too hot temperatures. The Australian Paralysis Tick can survive temperatures between 7°C and 32°C, with temperatures around 27°C considered to be optimal for tick questing. That is of course assuming that there is also adequate local humidity is sufficient. We have not identified a specific lower limit for suitable humidity, however although as humidity increases, questing becomes less risky, and tick activity increases. It is thought that ticks are less active in the rain, but that is likely due to the tendency of most scientists to avoiding doing fieldwork in the rain!. So the environmental conditions that are ideal for tick activity are similar those that are often found during what we know as the tick 'season' between August and February in coastal Eastern Australia: warm and humid weather. On the flip side, conditions that are worse for tick questing are either very cold or very warm, with low humidity, and a good breeze to blow away any humidity.

When ticks aren't on their hosts, they are either questing in vegetation or hiding from dehydration. There are no specific species of plants that are required by the Australian Paralysis Tick, but low and dense vegetation such as tall grasses, ferns, and various thick bushes and shrubs are likely to facilitate tick questing. Areas that are humid and protected from wind, such as valley bottoms, are more likely to have ticks. While you can come across Australian Paralysis Ticks almost anywhere within 30-100km of the East Coast of Australia, you can reduce your chances of running into these ticks by staying on trails when in the bush. Pet owners should not let their dogs off leashes in forests and rain forests, as curious dogs running through dense vegetation are extremely effective at collecting ticks.

Are cities safe from ticks?

We like to think of ticks as only being in rural areas or the bush, however many people encounter them in their backyards or urban parks and green spaces. Cities like Sydney have a lot of green spaces, including fragments of bush and forest, and while the positives benefits of these to human physical and mental health and conservation are clear, they also support urban populations of parasites like ticks. Even houses that aren't adjacent to green spaces can still have ticks: some wildlife species such as Black Rats, Brush Tailed Possums, and birds can move back and forth between backyards and bush areas, possibly transporting ticks around. Because of the possibly lethal consequences of tick bites, pet owners should be made aware of the risks of ticks even if they live in urban areas that aren't typically considered suitable for ticks.

A possibly less tick-y future.

Global climate change will likely negatively impact many species around the world, including the Australian Paralysis Tick. Eastern Australia will become both warmer and more arid in the next century, and this will reduce the amount of suitable habitat for the Australian Paralysis Tick, and maybe even reduce the duration of 'tick season'. However, before anyone gets too excited about the prospect of a tick-less future: it is unlikely that this species of tick will dramatically reduce in range or abundance. We also don't know what ecosystem services are being provided by this tick, and it is not clear if other species of tick might rise in abundance to replace the Australian Paralysis Tick.

Conclusion

Ticks are rightly feared and despised by many because of the impacts they can have on humans and companion animals. While many wish to eradicate ticks from the environment, these animals are resilient creatures that are unwittingly play a role as villains. It is doubtful that we can learn to live alongside ticks, but we can learn to better avoid and protect ourselves from them by studying the biology and ecology of ticks.

SMALL

Pretty Poisons in Yard series: Plumbago/Skyflower/ Leadwort

Aine Seavers

Oak Flats Vet Clinic

58A Central Avenue, Oak Flats NSW 2529

e. reception@oakflatsvet.com.au

C&T No. 5701

This plant causes a severe excruciating blistering skin condition in dogs who lie on or run through these plant beds. You must decontaminate to get the blistering sap off the pet before you lather it with lotions and supportive therapy. Wear gloves.

Don't confuse with Skyflower from Duranta spp. Duranta is also toxic but not by contact.

OPAL Score is 3 which puts it as a low aeroallergen risk. The kicker is the high blistering contact risk. For more info on OP allergen score, I have written a short summary on bit.ly/backyard-2

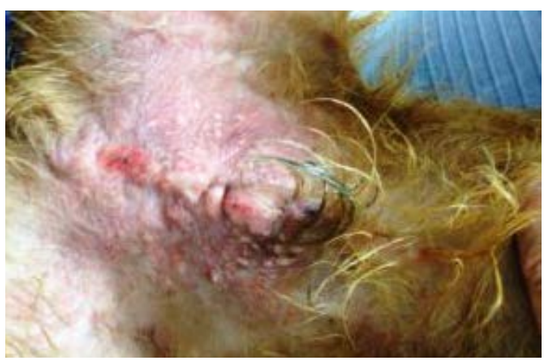

Figure 1 Lesions on skin of ventral abdomen.

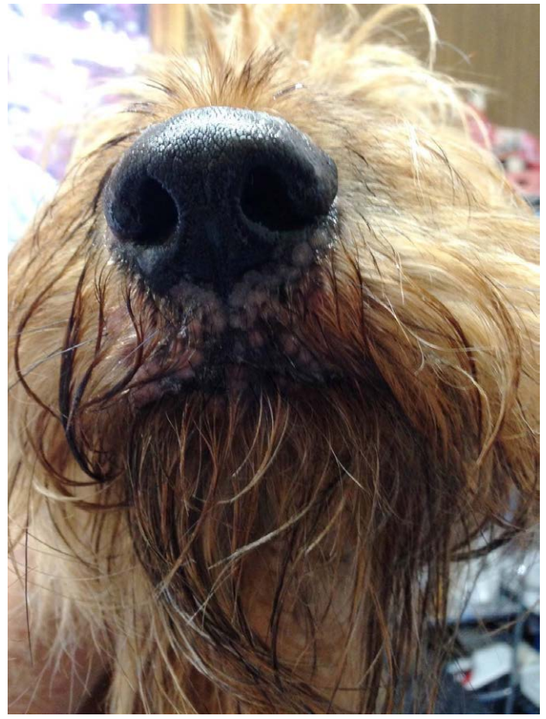

Figure 2. Lesions in muzzle area

Figure 3 Plumbago. OPAL Score 3

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GENERAL

Comparison of the efficacy of sodium pentosan polysulfate alone with the combination of sodium pentosan polysulfate and N'acetyl glucosamine for the treatment of osteoarthritis in dogs

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Abstract

Objective: To compare the efficacy of sodium pentosan polysulfate (PPS) with the efficacy of PPS and N'acetyl glucosamine (PPS-NAG) in the treatment of osteoarthritis in dogs.

Design: A randomised, double-blind, positive controlled clinical trial in dogs.

Methods: Forty eight dogs with radiographically confirmed osteoarthritis were randomly assigned to treatment with 4 weekly injections of 3mg/kg PPS as PPS alone or PPS-NAG. The dogs were assessed by veterinary investigators at weeks 1, 2, 3, 7 and 11 for lameness, range of motion, pain on palpation and overall response to treatment. The dog owners completed a Canine Brief Pain Inventory (CBPI) immediately before (baseline) and then weekly for 11 weeks after treatment. Final analysis was conducted on 36 dogs, 22 treated with PPS-NAG and 14 treated with PPS alone.

Results: Improvement over 11 weeks was seen in both treatment groups with significant reductions in lameness score, overall improvement and CBPI scores over time. Dogs in the PPS-NAG treatment group had statistically significant improvement in CBPI pain severity in weeks 5-9 compared to those dogs treated with PPS alone. There was also a more rapid response to PPS-NAG, with overall improvement score as assessed by the vets being significantly lower in weeks 1 and 2, compared with dogs treated with PPS alone.

Conclusions: Treatment with PPS was shown to result in improvements in symptoms of OA in dogs and the addition of NAG to PPS resulted in a significant improvement in efficacy of treatment.

Acknowledgments

Funding for the study was provided by Ceva Animal Health Pty Ltd, Australia.

Introduction

Osteoarthritis (OA) is a common disease in dogs.[1] Current treatments include nonsteroidal anti-inflammatory drugs which have potentially serious adverse effects.[2] PPS is a safe and widely used disease-modifying osteoarthritis drug (DMOAD), which has been demonstrated to have chondroprotective activity in dogs with OA.3-5 By combining PPS and NAG in Synovan®, both these DMOADs are delivered systemically. The combination of drugs with different mechanisms of action may improve efficacy by targeting different pathways that contribute to OA.[6] NAG has been shown to have anti-inflammatory activity when administered intramuscularly[7] and to inhibit inflammatory mediators involved in OA.[8] The combination of PPS and NAG has been documented in in vitro trials to have enhanced anti-inflammatory efficacy in both equine and canine chondrocytes.[9,10] The efficacy of PPS-NAG, compared with PPS alone, was compared in this double-blinded randomised trial in dogs with confirmed radiographic evidence of OA.

Materials and methods

Forty eight dogs, aged 6-12 years, presented to the Wagga Wagga Veterinary Hospital for lameness were enrolled in the study. The trial was approved by the Charles Sturt University Animal Care and Ethics Committee, approval 14/092 with signed owner consent. Inclusion criteria were no clinical systemic disease noted during the initial examination, lameness, confirmation of osteoarthritis on radiographs, normal haematology and biochemistry, no treatment with PPS or oral joint supplements in the past month and owner consent. Dogs were excluded for severe OA, infectious arthritis, neurological or significant systemic disease.

Treatment: Dogs were randomly assigned to treatment with PPS-NAG (Synovan® Injection, Ceva Animal Health, NSW, Aust) (25 dogs) or PPS (Cartrophen Vet Injection, Biopharm Australia, NSW, Aust) (23 dogs) which was administered by SC injection once weekly for 4 weeks at the label dose rate of 3mg/kg of PPS. Veterinarians and owners were blinded to the treatment. All dogs were treated initially with a 4 day course of oral carprofen or meloxicam but no other medication which could interfere with the study outcomes was administered.

Monitoring: Dogs were examined initially week 0 (baseline) and at the end of weeks 1, 2, 3, 7 and 11 by the veterinarians associated with the project team who performed a lameness examination at each time point. Scores for lameness (0= no lameness to 4=non-weight bearing), pain on palpation (0=no pain to 3=severe pain), and range of motion for the most severely affected limb (0=normal to 3= severely reduced) were recorded at week 0 (baseline) and weeks 1, 2, 3, 7 and 11. Overall improvement was assessed on a four point scale representing improvement from the baseline (week=0) assessment (0=greatly improved, 1=moderate improvement, 2=mild improvement, 3=no improvement). This score was only recorded for weeks 1, 2, 3, 7 and 11.

Dogs were assessed weekly for 12 weeks by owners who completed a canine brief pain inventory (CBPI) pain severity score at the end of each week. CBPI scores provided measures of pain severity score (PSS) (assessed as worst, least, average and now), and of pain interference score (PIS) (assessed as general activity, enjoyment of life, ability to rise, ability to walk, ability to run and ability to climb up) as described by Brown *et al* [9,10]. At weeks 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 and 11 PSS and PIS change variables were defined as the value for each week minus the week 0 (baseline) value for that dog and variable. This meant that a negative value for change from baseline indicated an improvement in PSS or PIS score. The more negative the change value, the more improvement in score.

Statistical analysis: The main outcomes of interest were PSS change and PIS change and the four separate veterinary scores representing lameness, pain, range of motion and overall improvement. At each measurement occasion for each dog, the average PSS and PIS values were created by calculating the arithmetic mean of the four component scores for PSS (worst, least, average and now) and the six component scores for PIS (activity, enjoy, rise, walk, run, climb). The mean week 0 values for each animal and CBPI category (PSS and PIS) were classified as baseline values for that dog and category.

Linear mixed models were used to analyse the data with formulation, week and formulation#week interaction added as fixed effects. A random effect was added for animal ID to adjust for lack of statistical independence as a result of repeated measures from animals.

Model outputs are presented as marginal means and 95% confidence intervals for each combination of formulation and week, derived from the multivariable models. Follow-up tests were performed after multivariable models to compare specific means, using *t*-tests and standard error terms derived from the multivariable models.

Model checking was done through assessment of residual plots including distribution patterns to check normality of residuals (histogram and P-P plots) and through standardised residuals vs fitted values to look for violation of assumptions such as heteroscedasticity. All analyses were undertaken using Stata version 13 (**stata.com**), using alpha=0.05.

Results

Thirty six dogs completed the study, 22 treated with PPS-NAG and 14 treated with PPS. A large number of dogs (25%) did not complete the study for a variety of reasons including development of medical problems, unrelated injuries or owner decision. It was noted during initial examination that a large number of dogs with clinical signs of OA did not have any evidence of OA on radiographs and were thus excluded.

Data from 22 dogs treated with PPS-NAG (A) and 14 dogs treated with PPS (B) was analysed. Retrospective power analysis using data mean and variance, and taking into account the different numbers in the treatment groups, showed sufficient power to detect a 2/10 difference in score (i.e. clinical significance). Preliminary assumption testing was conducted to check for normality, linearity, univariate and multivariate outliers, homogeneity of variance-covariance matrices, and multicollinearity, with no serious violations noted.

Results are presented as graphs of mean change: formulation vs week with error bars at 95% confidence

interval. CBPI change = value for that week – value for week 0 (Baseline), negative score = improvement with larger improvement = larger negative score.

Treatment of dogs with a clinical diagnosis of OA with both PPS-NAG (A) and PPS (B) resulted in reductions in PSS score compared to baseline *(Figure 1)*. The mean PSS for dogs treated with PPS-NAG was lower than the PSS for dogs treated with PPS, with the majority of scores being below -2. At weeks 5-9 the scores for dogs treated with PPS-NAG were significantly lower than for dogs treated with PPS ($p<0.05$). Following treatment with PPS-NAG the PSS scores were significantly lower than baseline ($p<0.05$), whilst for dogs treated with PPS the scores were not different from baseline at weeks 1 and 5-7.

Following treatment with both PPS and PPS-NAG the calculated PIS changes were significantly lower than baseline ($p<0.05$) with the PIS scores for dogs treated with PPS-NAG being consistently lower than for dogs treated with PPS, however this difference was not significant *(Figure 2)*.

There was no significant difference between treatments for lameness score but both treatments result in a decrease in lameness score over time *(Figure 3)*. For dogs treated with PPS-NAG there was a significant decrease in lameness score at weeks 1-2 and weeks 2-11 ($p<0.05$). For dogs treated with PPS there was a significant decrease between weeks 3-7.

The overall improvement scores for dogs treated with PPS-NAG were significantly lower than for dogs treated with PPS in weeks 1 & 2 ($p<0.05$) *(Figure 4)*. The scores for dogs treated with PPS-NAG in weeks 3, 7 & 11 were significantly lower than in week 1 ($p<0.05$). The scores in weeks 7 & 11 were significantly lower than in week 2 ($p<0.05$). The scores for dogs treated with PPS in weeks 3,7&11 were significantly lower than in week 1, the scores in week 7&11 were significantly lower than in week 2 and in week 7 significantly lower than in week 3 ($p<0.05$).

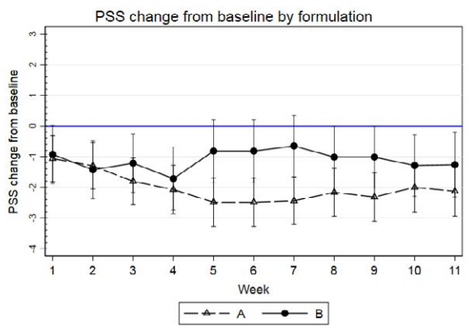

Figure 1. Plot of marginal mean PSS change from baseline by formulation and week, derived from multivariable model.

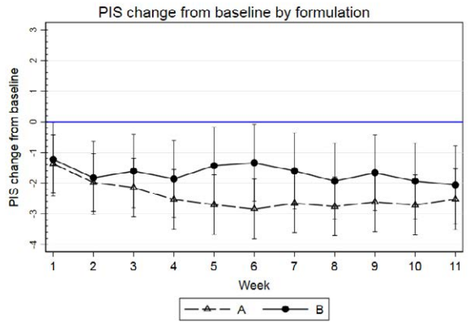

Figure 2. Plot of marginal mean PIS change from baseline by formulation and week, derived from multivariable model

The pain score for dogs treated with PPS was significantly lower than for dogs treated with PPS-NAG at week 3 ($p<0.05$) but there were no other significant differences *(Figure 5)*. The pain scores for dogs treated with PPS-NAG were significantly lower than baseline for weeks 2 and 7 ($p<0.05$). The pain scores for dogs treated with PPS were

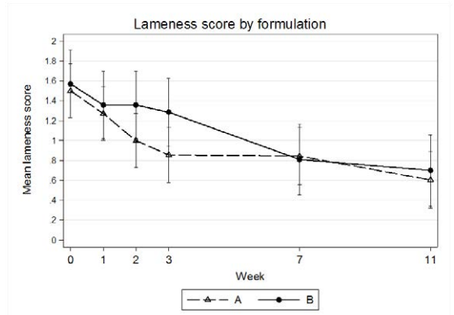

Figure 3. Mean lameness score for each combination of formulation and week

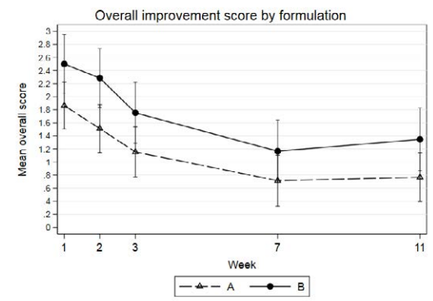

Figure 4. Mean overall improvement score for each combination of formulation and week.

significantly lower than baseline in weeks 3 and 7 ($p<0.05$).

The range of motion score for dogs treated with PPS-NAG was significantly lower than for dogs treated with PPS at week 2 ($p<0.05$) but there were no other significant

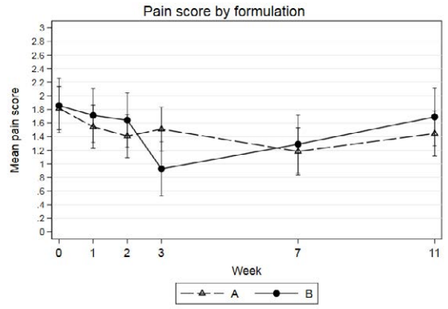

Figure 5. Mean pain score for each combination of formulation and week

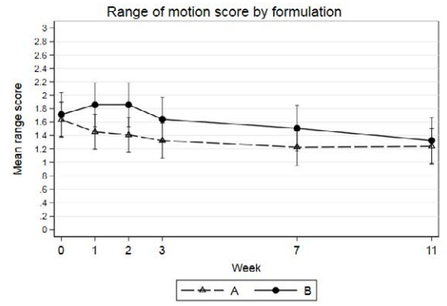

Figure 6. Mean range of motion score for each combination of formulation and week

differences *(Figure 6)*. The range of motion scores for dogs treated with PPS-NAG were significantly lower than baseline for weeks 3, 7 and 11 ($p<0.05$). The range of motion scores for dogs treated with PPS were significantly lower than baseline for week 11 ($p<0.05$) and weeks 7 & 11 were lower than weeks 1 & 2 ($p<0.05$).

Discussion

This study demonstrated that treatment of OA in dogs with a course of 4 injections of PPS at a dose of 3mg/kg PPS, both alone and in combination with NAG, is effective in reducing the clinical signs of OA, as measured by owner assessment by the CBPI, lameness score and overall improvement as assessed by veterinary investigators. Treatment with 3mg/kg of PPS has been reported to result in a significant reduction in clinical signs of OA in dogs with significantly less pain on joint manipulation, improved orthopaedic score and overall response in treated dogs.[5]

In the current study the response following treatment with PPS-NAG was significantly greater than the response following treatment with PPS alone. The PSS and PIS scores following treatment with the combination were consistently lower numerically for the full evaluation period and significantly lower at a number of time points. Similarly for the veterinary assessment of lameness score and overall improvement the scores demonstrated greater numerical improvements consistently for treatment with PPS-NAG compared to PPS and there were significant differences at a number of time points.

The results also supported a more rapid response to PPS-NAG treatment than for PPS treatment in dogs with OA. The lameness scores in dogs treated with PPS-NAG showed a significant decrease in weeks 1-2 and 2-11 compared with dogs treated with PPS, which showed a decrease in lameness later, from weeks 3-7. This has been reported anecdotally by Synovan® users (pers. comm Ceva Animal Health). In addition this is supported by in vitro trials which have demonstrated a greater anti-inflammatory effect for the combination of PPS and NAG compared with PPS alone.[9, 10]

It is recognised in practice that there is a delay in response to PPS and frequently dogs are administered a short course of anti-inflammatories, such as carprofen or meloxicam, as was included in the protocol for this study. The more rapid reduction in pain in the immediate weeks following a course of PPS-NAG may be an important factor in establishing patient loyalty and having those clients return for booster injections. Client's expectations are highest following the completion of the 4 week course.

The results of the subjective measures pain on palpation and range of motion did not follow the same pattern as the other assessments and it is difficult to interpret these findings. These are subjective assessments conducted by the veterinarian at a single time point. It is recognised that pain is subjective, dynamic and multidimensional and that validated pain assessment tools, such as the CBPI are recommended to be used to more accurately assess treatment response in dogs.[13] The CBPI is a valid, reliable, owner-completed questionnaire which was developed as a chronic pain outcome assessment tool in clinical studies.[14,15] Client specific outcome measures such as the CBPI are recognised to be preferred methods for monitoring pain and the effects of therapies for OA in dogs.[16]

The improved response to PPS-NAG over PPS alone supports some form of synergy between the two actives. Pentosan polysulphate is known to stimulate cartilage healing and in particular synthesis of glycosaminoglycans (GAGs) by chondrocytes and hyaluronic acid (HA) by synoviocytes.[17] Glucosamine is the main substrate for the biosynthesis of GAGs and administration of glucosamine results in accelerated GAG synthesis.[8] Co-administration of NAG and PPS is a strategy to ensure that there are high levels of glucosamine in the blood at the same time the PPS is administered, providing a larger pool of bioavailable glucosamine for biosynthesis of GAGs and HA. It has been suggested that oral glucosamine and chondroitin sulfate (CS) may interact in a complementary or synergistic fashion to improve synovial fluid HA content

Contents

in OA.[18] It has been reported that the combination of glucosamine plus CS showed enhanced anti-catabolic and anti-inflammatory effects in OA patients compared with the use of glucosamine or CS alone.[8] PPS, like CS, is a polysulfated GAG and has similar effects on patients with OA and thus it is likely that the combination of PPS and NAG has a similar synergistic action to the combination of CS and glucosamine. This is supported by in vitro trials which demonstrated a greater anti-inflammatory effect for the combination of PPS and NAG compared with PPS alone.[9,10]

This study supports earlier studies[3-5] which reported the benefits of the use of PPS as part of the management of OA in dogs. The study also supports that PPS has ongoing effects after the course of 4 injections, with improvements lasting for the duration of the study which was 12 weeks. Improvements in joint stiffness and pain for 20 weeks after a course of 4 weekly injections of PPS in humans have previously been reported.[19] This study demonstrated an enhanced efficacy for the combination of PPS and NAG, it is recognised that combinations of drugs can assist in the management of OA in humans.[20] Enhanced efficacy and a more rapid response to treatment will not only benefit patients immediately but also may lead to better long term outcomes as this will be noticeable to clients and encourage them to return for future veterinary treatments.

Abbreviations OA, osteoarthritis; PPS, pentosan polysulfate; NAG, N'acetyl glucosamine; PPS-NAG, combination product containing PPS and NAG; DMOAD, disease-modifying osteoarthritis drug; CBPI, canine brief pain inventory score; PSS, Pain Severity Score; PIS, Pain Intensity Score; GAG, glycosaminoglycans; HA, hyaluronic acid; CS, chondroitin sulfate.

Figure 1 Kay Hill Veterinary Teaching Hospital

LARGE

Nick Scott

Compliments & Comments on Perspective No. 138

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C&T No. 5703

eBook download:
Perspective No. 138 Castrating mature brumby (wild horse) stallions with the Henderson technique

I was hugely impressed with the article by Andrea Harvey and Kim Bensch on castrating brumby stallions and the series of photos were an excellent addition to the text.

To me the most striking features were:—

Minimal haemorrhage with the Henderson instrument:— in my practice I always used triple crush emasculators and only rarely had trouble with post op haemorrhage except in stallions or colts that had actually served a mare. With these horses I eventually resorted to routinely applying a fixed ligature to the cord as well as emasculating, as post op haemorrhage or marked swelling occurred in most cases. I have only recently heard about the Henderson method and my initial reaction was that it sounded fearfully cumbersome and traumatic but although the colts I operated on would have routinely been in far better condition than a brumby and thus more likely to bleed, the fact that none of the 25 had severe haemorrhage is most impressive, and I think everyone should consider trying the technique.

Their method of anaesthesia seemed to work very well and I think emphasising minimal disturbance post op was a very good feature.
I have only ever used xylazine as a premed and was far more conventional with my doses of xylazine at 1.1 mgs per kg followed after 5-10 minutes with diazepam (5mgs per mL) at approximately 1mL per 80 kg then ketamine at 2.2 mgs per kg straight after.
I was interested in the authors' comments about romifidine — I did not ever have access to romifidine supplied by Boehringer, so the extra cost was a significant factor but it could well be worth trying. I agreed with their comments about not trying to induce the horse till the head was totally hanging and giving more romifidine as needed. With xylazine also, anaesthetics were always much better when the horse was fully sedated.

I was interested in their use of butorphanol and midazolam or diazepam routinely as further premeds. Unlike many of my colleagues I used to use these drugs but either diazepam or an opiate and I don`t ever remember using them together.
I have never used the lower doses of ketamine they mention except I would reduce the dose to about 1.7 mg per kg if I had also sedated with Giafen for extra relaxation.

Because of poor relaxation with the alpha 2 agonist and ketamine, I often considered trying intra testicular local but just felt it would just slow things down and be of little benefit. Their protocol of injection of lignocaine both into the testicle and then into the spermatic cord preoperatively is excellently described and would be well worth trying as they reported definite benefits.

The pictures show that the yards really seemed to be in a remote outback area. When I was in practice I was always fanatical about trying to do any castration or surgery on as clean a grass area as possible. The results the authors have achieved with very low rates of infection, would suggest that this was not always essential, although stockyards in the more heavy carrying high rainfall country would likely be far more contaminated than these.
Certainly it was a great advantage to be able to give an unhandled horse an IV injection then roll them out of the crush and operate where they fell. (Just after graduation I remember doing the same thing with a 2-3mL IV dose of the dreaded suxamethonium but those unsedated horses used to gallop out for 2-3 steps!)

The authors' comments that these horses were removed from Kosciuszko National Park is very topical when I write this (6/7/18).
We have just had the disastrous political decision by the NSW Government to stop controlling brumbies in the National Park.
I have certainly had an emotional attachment to horses myself but we must put all sentiments aside; these are feral animals in a fragile alpine environment which already has enough challenges with drought, climate change and human usage during winter and summer.
In this environment, overstocking with brumbies will cause just as much damage as feral pigs or rabbits and we must control these animals in whatever way possible, and veterinarians should support all the scientific studies stating that control is essential.

Finally, there is one issue in the article that I must disagree with:— the pictures showing the surgeon kneeling next to

a horse's scrotum while an assistant is just holding the top hind leg up in their hands.

Notwithstanding that the romifidine and then local anaesthetic probably gave better anaesthesia than my routine, they only reported on a series of 25 anaesthetics.

I have only kept records of the last 140 equine anaesthetics I did in practice, but I can assure readers not all inductions, anaesthetics nor recoveries were consistently smooth and uneventful; in mixed practice I found variable anaesthetics occurred in every other species I tried as well.

With no restraint on the hind leg not only is the surgeon in danger of an unpredicted kick, but the person holding the hind leg could easily be jerked off their feet by an involuntary movement landing on top of the surgeon who may be wielding a scalpel at the time.

Eventually I developed a routine for roping the top hind leg out of the way which worked well for me.

LARGE + WINNER!

Nick Scott BVSc

Leg rope for anaesthetised horses to improve safety

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Some other vets used to tie the horse's lead to the back pastern to pull the leg out of the way, but I never favoured this as the horse would look in an unnatural position and I thought at more risk of injury.

I only have 1 picture on file of the way I used to rope legs for castrations or other surgeries and it is only a reasonable image as this horse was haemorrhaging severely from a wound inside the tibia and was already staggering before anaesthetic induction.

So the rope was applied in a great hurry before a long surgery and you can see that the rope is applied much too loosely, myself and the owner were in no danger of being kicked but if the horse`s anaesthetic became too light he would have been at risk of stretching his leg out straining against the rope.

As a student I remember a horse fracturing its femur, as the ropes became loose, when we were being shown how to cast horses without anaesthetic and I have been very conscious of this danger ever since. Ideally the rope should be pulled up towards the shoulder so that the hoof is at least touching the horse`s elbow.

To explain the application:— First a loop is made over the horse`s head then worked down as low on the neck as possible. It helps to lift up the mane to work the rope down further. Then the free end of the rope is fed down BETWEEN the forelegs then looped OVER the pastern and comes back UNDERNEATH the pastern, this will lock the foot inside the rope. Then the free end is placed through the neck loop over the shoulder. Then with the operator standing behind the wither it is quite easy to work the foot up towards the elbow as the rope loop works as a fulcrum to allow leverage. Then I fixed the end of rope to the pastern with 2 half hitches, these could be released quite safely leaning over from the horse`s wither even when it was recovering quite strongly.

Alternatively an assistant could hold the end of the rope after only 1 half hitch and it could be released and removed more quickly. An advantage of this system was that the horse could be pulled up onto his back quite safely and fairly easily using the free end of the rope with perhaps another loop over the upper hock to provide a more even pull.

A couple of times, particularly when working without assistance the horse became too light and rolled right over. This was not as big a disaster as it looked, there were no signs of injury to these horses. The safest way with least effort was to top them up then roll them back to the other side. One time with no assistance I was able to lean over the horse`s neck and release the half hitches and then he was able to slowly work his way out of the rope while I held his head to try and keep him calm.

I initially used a big cotton rope that could tie up both hind legs but it was very bulky to use and the pictures supplied show a rope I used successfully for several years. It was for sale as a show lead in a saddlery and is 3.6 metres long with a diameter of approx. 20 mms. It is synthetic material so very strong and although it has burnt my hands [after lassoing a cow] it has never irritated a horse`s pastern.

The little brass snap lead clips supplied with the lead are never strong enough so I cut it out with bolt cutters and replaced with a steel snap hook or carabiner which are much stronger. I have always been able to find these at local hardware shops as I think they would be far more expensive at a climbing or camping gear shop.

Contents

Figure 1. Rope in a loose coil

Figure 2. Steel snap hook

The white rope did show a lot of dirt, blood etc. but I used to remove the snap hook from the spliced end then put through the washing machine with my overalls. I was able to fit this rope onto some big horses, over 600 kgs and nearly 17 hands but I advise this is the minimum length needed for general practice.

Figure 3. The little brass snap lead clips supplied with the lead are never strong enough so I cut it out with bolt cutters and replace with a steel snap hook or carabiner (Fig 2)which are much stronger.

Figure 4. The rope in action.
Note: Nick uses this rope when working single handedly or with only 1 untrained assistant

Reply to Nick Scott's compliments and comments from co-authors Andrea Harvey & Kim Bensch

Thank you so much for taking the trouble to read and comment on our article, and for making such lovely comments; it's really very much appreciated! As such an experienced equine vet it is really interesting and valuable to hear of your experiences and insights too. We also appreciate the opportunity you have provided us for expanding the discussion in some of these areas.

Your comment about post-operative haemorrhage in stallions/colts that had served mares is interesting; all our stallions we aged between 3-10 years, with most being around 5-6 years so many of those will presumably have served mares. You also mention that you would expect haemorrhage to be more likely in horses in good condition. I should also point out that these brumbies were actually all in good condition; although some are in poor condition when removed from the park, these had been in the owners' care for some time and fed up prior to gelding, so all were in body condition scores of around 5/9. Given their good condition, mature age, and likelihood that many will have served mares, it sounds like you do definitely agree that the lack of haemorrhage is impressive and likely to be a significant finding.

Your comments regarding the anaesthesia are really thought provoking. We don't have a lot of experience using xylazine as a pre-med so our comments are speculative and based on theory rather than on experience, but we can't help thinking that the longer duration of action of romifidine over xylazine (approximately 20 minutes longer duration) really helps to provide a safer plane of anaesthesia for the duration of the procedure (i.e. less chances of prematurely waking up) and a smoother recovery, since they still have the alpha 2 agonist acting after the ketamine has worn off. Furthermore, for the level of sedation, the degree of ataxia tends to be much less with romifidine than xylazine (England *et al* 1992), which is likely to contribute to smoother recoveries and a high success of standing on first attempt. Although we kindly received some donated Sedivet® for our 2nd lot of castrations, we did purchase it for the first lot. Romifidine certainly is significantly more expensive (approx. $25 per average dose) than xylazine (approx. $5 per average dose), but realistically this probably is not prohibitively more for most clients to pay for an anaesthetic, especially if there are genuine benefits as suggested above.

It is also noteworthy that you remark that not many of your colleagues would use butorphanol or midazolam, or both in a pre-med either. Again, we do wonder whether this more multimodal anaesthesia/analgesia was providing more effective anaesthesia and analgesia, and thus we would have a much lower risk of unwanted voluntary movement and premature recovery, making the anaesthetic safer for personnel. Again, we are sure that the use of intratesticular lignocaine would help with this too. We can see that this may slow the procedure down for open castrations or young colts without much testicular fat and fascia, but certainly in these mature stallions there was a fair amount of fat and fascia to strip away from the testes, and so this gave time for the lignocaine to take effect without slowing the procedure. We certainly would not attempt the Henderson technique without this; as we mentioned the horses did react to the twisting of the spermatic cord when we didn't have a deep enough plane of anaesthesia or hadn't given the lignocaine long enough to take effect, and this could lead to a sudden dangerous voluntary movement by the horse.

We are definitely very safety conscious so really take on board your comments about safety and restraint under anaesthesia. There are a few things to add here. Firstly, for open castrations with routine emasculators, Kim routinely positions herself on the dorsal side of the horse and approaches from behind the hind limb, which is a safer position. I however am very short and found that I didn't have good enough reach from this position! Furthermore, when using the Henderson you need to have the emasculators positioned in line with the spermatic cord which makes standing in front of the hind legs on the ventral side of the horse unavoidable at that stage. We must emphasise that we never had a handler holding the hind leg directly with their hands, only with a rope. The particular picture we used probably also wasn't the best picture as it does show the handler quite close to the horse with quite a short length of rope, but in reality, we usually had the handler standing further back with a longer length of rope between them and the horse; we only used long thick strong ropes for this. One of the things that I really like about this, which would be lost with tying the leg back, is that it aids in monitoring the depth of anaesthesia. The handler was instructed on how to monitor muscle tone and to immediately alert us if there was an increase in muscle tone, which allowed the surgeon to step back and the anaesthetist at the head end to immediately top up anaesthesia, and the surgeon would only resume position once the depth of anaesthesia was adequate again. In addition to this, of course having a dedicated veterinary anaesthetist at the head end of the horse meant that they were continually monitoring depth of anaesthesia through respiratory rate, eye position and presence/absence of nystagmus etc., and had IV access being ready to immediately top up with anaesthetic agents if required. Again, the purpose of the anaesthetist sitting on the neck is in no way to provide restraint of any kind, but to be optimally positioned for close anaesthetic monitoring and immediate IV top ups if required. Although unpredictable things can of course always happen, we really feel that this worked very well for us and

was safe. We always give our handler clear instructions before we start regarding muscle tone monitoring, monitoring for tail movement, and communication. We also instructed them to let go of the rope in the event of significant unexpected movement (after the surgeon had jumped out of the way!). We had very good communication within our team; the surgeon would always be checking on the depth of anaesthesia with the anaesthetist prior to key events such as incising the scrotum, placing the emasculators, and starting to twist the cord, and the anaesthetist and handler would always communicate immediately if the depth of anaesthesia was lightening, and the surgeon was always vigilant and ready to jump out of the way if needed. We do believe that these are all also really important factors for personnel safety too. Of course, we had the great luxury of having 2 veterinarians at each procedure, and I would not feel comfortable with these any other way! Obviously, this is very different to having to do an anaesthetic and procedure as the only vet, and in that situation, we can certainly appreciate the benefits of your suggestion.

Finally, we do again wonder whether our anaesthetic protocol and having quite a deep plane of anaesthesia significantly reduces the risk of unpredicted movements. We can certainly imagine that when you add up the differences in our protocols compared to some, that in a situation without butorphanol, midazolam nor lignocaine, and perhaps with the xylazine beginning to wear off, that a horse could certainly suddenly become extremely reactive if it only has ketamine on board and that begins to wear off. Of additional interest, romifidine has also been shown to reduce unwanted movements in response to stimuli during procedures, with a comparative study of detomidine vs romifidine, combined with butorphanol, showing less kicking and defensive movements with romfidine and butorphanol, compared to detomidine and butorphanol (Green *et al* 1996). Other studies have also shown horses to be less arousable to stimuli following romifidine compared to xylazine sedation, with romifidine resulting in superior quality of sedation (Ringer 2012).

We would be really interested in the comments of anaesthetists on this, but we are certain that the multimodal anaesthesia/analgesia approach that we use, together with the longer duration of action of romifidine and its additional effectiveness in reducing unexpected movements, and in combination with the very close monitoring of anesthesia and topping up immediately as required, would significantly reduce the risk of such occurrences. Certainly, with the depth of anaesthesia in our horses and the close anaesthetic monitoring, the risks of sudden unexpected voluntary movements should be significantly minimized. We would expect this protocol to also provide a more optimal welfare outcome for the horses.

Kim has much more experience with equine field anaesthesia beyond these geldings and has only ever had one horse kick out when she was less experienced and in hindsight the horse was very anxious and poorly handled, not adequately sedated at the time of induction, and they did not adequately assess depth of anaesthesia prior to starting incising. Since using the above protocol combined with good handling preparation and careful anaesthetic monitoring, she has not encountered such an event again.

You also mention infection risks and the surface for performing the surgery on. Certainly, we would prefer a nice dry grassy area too. However, you are right, in this situation we were in a remote outback area so there wasn't too much choice there. However, we did ensure that the yards were clean and dry. The dryness isn't always easy to ensure, but we had some luck there. The yards were actually lightly hosed down the morning before in order to dampen down the dust which was more of a potential issue. We did actually have one very unfortunate event where a totally unexpected thunderstorm suddenly came over mid procedure with one horse, and we were gelding in torrential rain, thunder, lightning and strong winds with equipment blowing everywhere! Veterinary medicine is never perfect despite the best laid plans! This horse was fortunately fine, and did not develop any post-operative infection, whilst us veterinarians and our handler were a little traumatized!

Finally, in response to your comment about the new NSW Brumby Bill. We agree that this is a disastrous political decision. I have just completed 3 years researching the ecology and welfare of brumbies in the national parks. I love the brumbies and have created a brumby sanctuary at our 700 acre hobby farm. However, what I found during my research, was that many of the brumbies had terrible welfare due to lack of nutrition, and knock-on consequences of that, in addition to high gastrointestinal parasite burdens, and a high prevalence of ***S. vulgaris***. Therefore, even without considering the environmental aspects, the populations need to be controlled for their own welfare too. I agree that the veterinary profession should have a 'louder voice' in this issue to try and avoid the current situation of policy being made with no expert or scientific input.

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Further Comments on Perspective No. 138 from John Coles (email to co-authors)

Everything in the article correlates perfectly to my own experience. I have become very confident with field anaesthesia of horses and have similar techniques to those used in your study. I now have no interest in performing traditional castration - I would rather a client go elsewhere if Henderson does not appeal to them. Having said that, I have been able to convince every client that it will likely be the best outcome in my hands and proceed.

I have had one disastrous eventration out of about 300 Hendersons that I have done. You didn't address the comparison between the two methods for concern about possible eventration - what are your thoughts there?

Was great to read the article.

Reply to John Cole's comments from co-authors Andrea Harvey & Kim Bensch

Thanks so much for your comments, it is good to know that our experience correlates so well with your own! Of course it was you that first mentioned the Henderson technique to Richard a long time ago, and it was largely off that advice from you, and then further reading and others' experiences, that we decided to give it a go!

May I ask with your field anaesthesia, whether you also use butorphanol and/or midazolam in your pre-meds, and whether you use intratesticular lignoacaine? Also, do you have any experience with romifidine, or do you use xylazine? I ask because I am aware that many simply use xylazine and ketamine, and I don't have alternative experience to the protocol we described, but I feel certain that using the more multi-modal anaesthesia/analgesia approach that we would be getting superior anaesthetics that are also safer for personnel in terms of less likelihood of the horse suddenly moving etc. Do you have any comments on that?

Re eventuation; I don't have enough personal experience, and I guess being comparatively rare, and often breed related (reportedly more common in Standardbreds), that a comparative study would really need to be performed on quite a large number of horses (perhaps just Standardbreds? Was your eventration in a Standardbred?) to know if there was a true difference in the incidence of eventration between the 2 techniques.

However, you may find the following useful which was in the paper of a recent survey of Australian vets regarding castrations they had performed. *'Of 5,100 horses castrated with emasculators, 64 cases (1.3%) experienced eventration of tissue and 5 (0.1%) were reported to have eviscerated.'* Reports of eventration were significantly higher in horses castrated using an open technique compared to other closed or semi-closed techniques. Of 230 horses castrated using the Henderson, 4 (1.7%) were reported to have eventuated from the incision and 1 horse (0.4%) eviscerated. There was no significant association between instrument and the reported rate of evisceration (Owens *et al* 2017).

Obviously, it is still hard to directly compare with this data as far fewer horses in this study were castrated with the Henderson, and there is also no mention of age and breed of the ones that did have eventration. Given there were significantly fewer horses that eventrated with normal emasculators having a closed castration as opposed to open, you would think that the risk using the Henderson might be lower overall, simply because of it being a closed technique.

SMALL

Possible Lyssa Virus Exposure in a Siberian Husky

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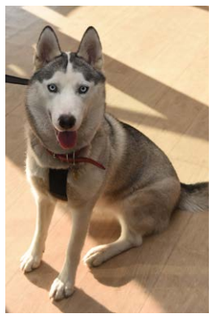

Figure 1. Husky

A distraught man came into the practice and mentioned his dog had killed a bat the night before last (i.e. 48 hours ago) and he was terribly worried about rabies, could we help? He still had the bat's body in a bag at home and he could bring the dog (and the deceased bat) in if we had time for an appointment. The dog was a 1.5 year old female neutered Siberian Husky.

I said I'd make some phone calls and get back to him.

I called the Emergency Animal Disease (EAD) Hotline. They advised me to vaccinate the dog for rabies, in case the bat had Australian Bat Lyssavirus (ABL), so I rang the owner to book in an appointment that afternoon.

When he returned for the consultation the dog was bouncing around the consulting room (Figure 1 shows the dog in a rare still moment).

Figure 2. Dead bat

The owner said he had been walking the dog when a bat flew from the ground, right into the path of the thrilled dog who proceeded to chew the bat (looking at this dog I could imagine an enthusiastic response). Awful shrieking ensued and the owner tried to grab the bat from the dog's mouth. At this point I asked the owner about the scratch I noticed on his hand. This was acquired during the process. At some point the dog gave up, the bat fell out of her mouth (deceased) and the owner and dog walked home. But the owner was unsettled by the event. He returned to the site, realised the animal was a bat and not – as he had suspected, a bird – got online and read about ABL.

The dog was fine: eating and drinking well, very bright but a challenge to examine. There were no obvious neurological deficits but the excitement of the dog and her predisposition to 'nipping' precluded a comprehensive neurological examination. She happily downed two bowls of water during the consult.

Questions for vets to reflect on, courtesy of Dr Siobhan Mor

- Do you know how to counsel clients following an encounter with a bat? Vets are uniquely placed to educate the public about the risks associated with handing bats and are often the first point of contact following an encounter.
- Does your practice have an SOP for dealing with potential ABL exposures?
- Are all staff members who handle bats in your practice vaccinated? NSW Health recommends vaccination of all people who are at increased risk of being bitten or scratched by bats in Australia.
- If you are vaccinated, when was the last time you had your titre checked?
- Do you know the phone number of your local public health unit?
- Do you know where to find government factsheets on zoonoses?

Editor's note: link to Animal Health Australia, Australian Veterinary Emergency Plan disease Strategy, Australian Bat Lyssavirus: http://bit.ly/ozlyssa

Information for the public: http://bit.ly/lyssavirus.

The owner became the focus of concern. I called the EAD again and they patched me through to public health. I passed the phone to the owner as he made arrangements for immediate medical attention, while the staff at the Elizabeth MacArthur Agricultural Institute were on standby to receive the bat.

The owner, a European man who had grown up knowing the risks of rabies, was very concerned despite assurances that the risk was low. It wasn't zero – there had been recent positive bats in the area.

He handed me the bat which he had collected, wrapped and placed in a bag, kept at home (unrefrigerated) for 48 hours (Figure 2 and 3). Fortunately the head was intact. (The NSW DPI advises that people take precautions when handling dead bats, including using sturdy gloves and a shovel to pick up the body, or contacting the local council to do so. Because he didn't know, this gentleman did neither).

I vaccinated the dog for rabies as recommended by the veterinarian I spoke to on the EAD hotline, and the owner took her home before proceeding to a local hospital for post-exposure prophylaxis. Using sturdy gloves, I carefully wrapped the bat, sealing it in an esky so that it could be couriered to the DPI (they were incredibly proactive in arranging a courier for us).

The next day we had the results: the bat was negative for ABL. I called the owner with the preliminary results and he was incredibly relieved.

He later told me that day was R U OK? day. As he waited for the results, colleagues at work asked each other if they were okay.

When they asked him he said 'NO'.

He said he was so scared about contracting rabies, but no one asked him the follow up question: 'why are you not okay?' or 'what's up?' He said he'd never felt so isolated. It wasn't until he recalled this story that I realised how absolutely terrified he had been of contracting a deadly disease.

When the results came through he came in so relieved and said that it was wonderful that vets, doctors and the labs had worked together. And now he is OK. He was very keen to share the story so that others could learn from the experience and has asked me to do so.

References

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CVE AWARDS

cve.edu.au/award-programs

The Paul Gotis-Graham Award for Excellence in Small Animal Surgery

Recipients are entitled to one full registration at a CVE surgery conference or workshop of their choice.

The Paul Gotis-Graham Memorial Award was instituted to honour a veterinary surgeon who gave so much of himself to the College, the CVE and others. It is bestowed on a worthy recipient who has passed the requirements for Membership to the ANZCVS in Small Animal Surgery.

Paul (1967-2014) was an inspirational veterinary surgeon who enjoyed teaching and mentoring his colleagues.

Congratulations to Dr Jason Heng, the 2018 recipient.

Previous awardees:-

- 2017 – Teng Xiang Khoo
- 2016 – Sally-Anne Richter
- 2015 Anna Nutt
- 2014 – Gavin McDonagh

2017 CLINICAL COMPETENCY AWARDS

(Different universities award these at different times, hence the time lag)

Congratulations to:-

- Charles Sturt University: *Jessica Verwey*
- James Cook University: *Jillian Eliza Bell*
- Massey University, NZ: *Corey Regnerus*
- The University of Adelaide: *Conor Brand*
- The University of Melbourne: *Elita Frazer*
- The University of Queensland: *Chi Yan Kan (Lydia)*
- The University of Sydney: *Lyndon James Clearly*

REPLIES & COMMENTS

FURTHER COMMENTS ON PREVIOUS C&Ts

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C&T No. 5706

C&T No. 5293 Tips to use acetylcysteine vials so the drug doesn't expire on the shelf awaiting a paracetamol poison case (A Seavers, March 2013, C&T Issue 270, pg 32)

NAC is now being used in canine otitis cases to decrease biofilm formation and improve antibiotic therapy. 2% is recommended as the optimum dilution as in some dogs a higher concentration could induce irritation and/or pain.

I have read in veterinary forums of the following doses being recommended:-

Using the 200mg/mL 20% vial, dilute 1:9 for 2%

Use sterile water or cool boiled water to dilute

Apply the product daily at home and wait 30mins.

Then apply ear drops

Stability: Ciprodex® in combination with 1.25% NAC): Good stability over a 15day period.

Efficacy: 15-day study period.

Ototoxicity: Low risk if NAC = 2% (but for sure less toxic than a severe deep infection). Medics use NAC systemically to prevent ototoxicity of gentamicin or trans-tympanic injections to avoid ototoxicity of cisplatin.

Note: The concentration of NAC changed in 2010 – it is now 800mg in a 4mL vial so be aware of that in Australia

C&T No. 5329 Economic validation for stocking uncommonly used antidotes and antiemetics on the drug shelf (A Seavers, Sept 2013, C&T Issue 272, pg 20)

Cerenia in Giardia cases

Our clinic Giardia Protocol is daily Drontal® x 3 days in dogs.

The problem was a subset of dogs either vomit within ½ hour of oral Drontal® (needing re-pilling) or after 2 hours (not needing re-pilling). Either event makes for poor ongoing client compliance.

We now pre-med with Cerenia® several hours before the Drontal® tablet each time with great success.

A question of my own

Has anyone considered Cerenia® in rhododendron poisoning in the scenario of a pet sheep or valuable breeding ram or ewe being poisoned? Obviously not practical in a flock situation, but the small hobby farmers (often vets/nurses) are keen to treat these cases so hence my thought about Cerenia. The distressing spasms and heaving are caused by the grayanotoxin acting directly on the vagus. The Vagus has NK1 receptors and Sub P. in its dorsal motor nucleus. Cerenia® would target those receptors directly so would that be an indication to use Cerenia® almost as an anti-dote in these cases? We also know that Cerenia is a powerful analgesic, especially in gut pain, so again another indication for these poison cases?

Reading material

Spotlight on the perioperative use of maropitant citrate. B. Kraus. *Veterinary Medicine*: Research and Reports 08/2017.

eBook download:
C&T No. 5293 Tips to use acetylcysteine vials

eBook download:
C&T No. 5329 Economic validation for stocking uncommonly used antidotes and antiemetics

USING YOUR MEMBER BENEFITS...?

Looking for more information on ticks, for example...? Search cve.edu.au/cvelibrary using your username and password. See proceedings chapters: Tick Paralysis 1: Pathophysiology, Controversies and Initial Management & Tick Paralysis 2: Critical Care Of Patients With Tick Paralysis (By Rob Webster, Proc No. 411 Emergency Conference)

PERSPECTIVE
NO. 139

Pathology for Practitioners: Feline Hepatobiliary and Pancreatic Disease

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This paper provides an outline of talks delivered at the Wanaka Veterinary Conference, Edgewater Resort, Wanaka in August 2016 (**vetsontour.com.au**). These talks covered a subset of feline hepatobiliary and pancreatic disorders in which biopsy is often helpful in reaching a definitive diagnosis. The advantages of various biopsy methods were discussed, as well as special staining methods, and ancillary diagnostic procedures such as immunohistochemistry and lymphocyte clonality testing.

LIVER: Hepatic Response to injury

The liver is a remarkably resilient and adaptable organ, with a large functional reserve and impressive regenerative capacity. Up to 70% of the parenchyma can be surgically removed without causing clinical insufficiency and, in a matter of weeks, the liver is restored to its original mass via regenerative processes[1]. This confers on the liver an extraordinary ability to recover from injury; however, it also results in late diagnosis of insidious diseases. This has implications for histologic examination, particularly in long standing cases of progressive inflammatory liver disease, in which fibrosis and regenerative changes may predominate, occasionally obscuring the underlying process.

Normal hepatic architecture is critical to liver function, and recovery following hepatic injury is dependent on survival of an intact reticulin framework, normal blood flow and biliary drainage. Severe or chronic damage may result in fibrosis that significantly alters hepatic function. Both biliary and hepatocellular injury readily result in rapid proliferation of myofibroblasts, with deposition of collagen in portal tracts and extension of fibrous septa into the surrounding parenchyma, eventually bridging neighbouring portal tracts. This pattern of fibrosis can dramatically alter perfusion of blood through the hepatic lobule, leading to portal hypertension and liver dysfunction.

Fibrosis within hepatic sinusoids is comparatively subtle and may be impossible to detect without the use of special histochemical stains for collagen. Delicate collagen fibrils that extend into the space of Disse interrupt interactions between hepatocytes and sinusoidal plasma, and alter the structure of the sinusoid endothelium in a process known as capillarisation. Deposition of collagen is potentially reversible when the source of the injury is addressed; however, ongoing injury may eventually result in chronic hepatic fibrosis with impaired hepatic function.

The distribution and pattern of lesions within the hepatic parenchyma is directly related to disease pathogenesis and recognition of these patterns is critical to arriving at a diagnosis. Focal lesions may be suggestive of a localised inflammatory process (e.g. abscess) or slowly progressive tumour. Multifocal lesions may correlate with haematogenous localisation of pathogens, or be associated with a metastatic process. Generalised or diffuse changes are often more difficult to detect with imaging and biopsy may be particularly helpful in these cases. Generalised liver disease may result from toxic insults or inflammatory disorders such as lymphocytic cholangiohepatitis. Infiltrative neoplastic processes such as lymphoma or cholangiocarcinoma may also have a poorly demarcated, extensive distribution. The pattern of lesions within the liver should inform the diagnostic approach with respect to biopsy technique and the number of samples submitted. Because the pattern and distribution of lesions within the liver is related to the underlying pathogenesis of disease, it is critical to provide some description of these to your pathologist!

KEY POINTS

- The number and distribution of hepatic lesions is closely related to disease pathogenesis
- This information should be included on the biopsy submission form

- Early fibrosis is often not detectable without special stains for collagen

Biopsy Technique

A few guiding principles apply when choosing the most appropriate biopsy technique, regardless of organ or tissue. Cytologic specimens are appropriate when information on tissue architecture is not critical to the diagnosis. There is a risk that this strategy will not yield a representative sample of the lesion. It is therefore most appropriate when a lesion is essentially diffuse and diagnosis may be reached via evaluation of individual cell morphology or detection of pathogens. Small samples (e.g. core needle biopsies) provide some information regarding tissue organisation but only survey a tiny portion of the organ and are most suitable when a lesion is relatively widespread. Larger incisional or excisional biopsies are ideal in scenarios in which a broad scale view of the tissue landscape is required for diagnosis and prognosis, for example tumour invasion and surgical margins or tissue response to injury, which is often important in hepatic disease.

When it comes to biopsy of the liver, cytology is often inadequate as disease processes are frequently patchy and complicated, involving multiple cell types and reparative responses such as fibrosis. Feline hepatitic lipidosis is a notable exception, as cytologic changes are widespread and characteristic. The various challenges associated with obtaining either a core needle or larger biopsy of the liver should be evaluated against the suspected pathologic process and the likelihood of sampling the lesion. Selection of biopsy technique will be influenced by the distribution and size of the lesion, the presumptive diagnosis and clinical parameters (coagulation status, anaesthetic and surgical risks) [2]. Pursuit of additional tests (culture, immunohistochemistry, molecular clonality testing) will also be influenced by the availability of sufficient tissue.

Ultrasound guided core needle biopsy is minimally invasive and may be sufficient for unambiguous lesions; however, the small size of core needle biopsies is often a major limitation. In human medicine, examination of 15 portal triads is required in order to provide an accurate evaluation of diffuse or multifocal disorders, and specimens >15mm in length are advised. This exceeds the median needle biopsy specimen length in cats. The small number of portal triads and hepatic lobules sampled in a typical core biopsy likely explains the poor concordance between core needle and wedge biopsies[2].

Laparotomy provides an opportunity to closely inspect the gross appearance of the liver and collect a variety of larger, more representative samples, including wedge and punch biopsies. Ideally, multiple sites should be sampled if possible.

KEY POINTS

- Cytology is appropriate when disease is diffuse, individual cell morphology or pathogen detection can yield a diagnosis, and evaluation of tissue architecture is not important
- Core needle biopsies are limited in size and may not adequately represent the lesion
- When lesions are multifocal, provide multiple samples if possible

Inflammatory Hepatopathies

The distribution and character of inflammatory infiltrates within the liver vary with aetiology, severity and chronicity of disease, route of entry and pathogenesis. Most infectious causes of hepatitis result in either patchy or randomly distributed inflammatory foci (consistent with a haematogenous route of entry; Fig 1.) or an infiltrate that is concentrated around portal tracts (inflammation originating in the biliary tree). Core needle biopsies that adequately represent portal triads should convey the information needed to make a diagnosis of the latter. If imaging findings are suggestive of randomly distributed inflammatory foci (e.g. abscesses), make sure to include this information on your submission form.

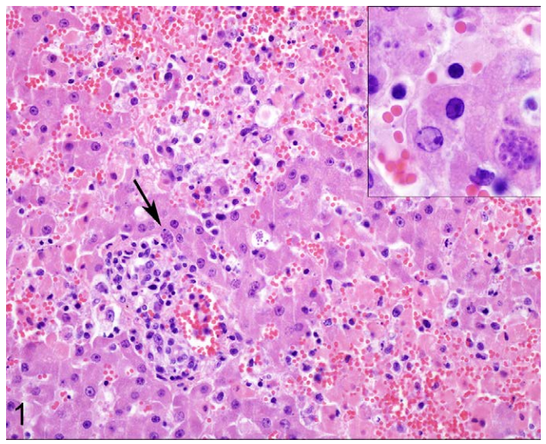

Figure 1. Toxoplasmosis, necrotising and haemorrhagic hepatitis with intracellular and extracellular tachyzoites, liver, cat. Multifocal areas of severe haemorrhage and necrosis are randomly distributed throughout the hepatic parenchyma, consistent with haematogenous distribution of Toxoplasma gondii. Protozoa are present within the cytoplasm of hepatocytes and macrophages (arrow). High magnification inset: Hepatocytes contain intracytoplasmic 2-3 µm crescent shaped basophilic tachyzoites. Haematoxylin and eosin (HE).

Acute inflammatory insults within the liver tend to be characterised by a neutrophilic infiltrate, accompanied by activation of Kupffer cells and variably extensive hepatocellular necrosis. Acute liver injury, regardless of aetiology, will not result in progression to significant fibrosis unless inflammation and parenchymal damage is particularly protracted. Chronic hepatitis refers to a necroinflammatory process lasting 6 months or more. In veterinary medicine, most chronic liver disease is idiopathic, reflecting our limited understanding of disease pathogenesis.

Neutrophilic cholangitis/cholangiohepatitis

Neutrophilic cholangitis is relatively common in the cat and tends to occur in conjunction with other disorders such as pancreatitis and enteritis. Shared entry of the biliary and pancreatic ducts to the duodenum provides an avenue for ascending enterobacterial infection of both systems[3]. Recognising neutrophilic cholangiohepatitis in biopsies is relatively uncomplicated provided sufficient tissue is provided for adequate evaluation of portal tracts. Occasionally, systemic inflammatory processes (bacteremia, sepsis) may alter the population of leukocytes trafficking through hepatic sinusoids, resulting in a slightly confusing histologic picture that resembles hepatitis.

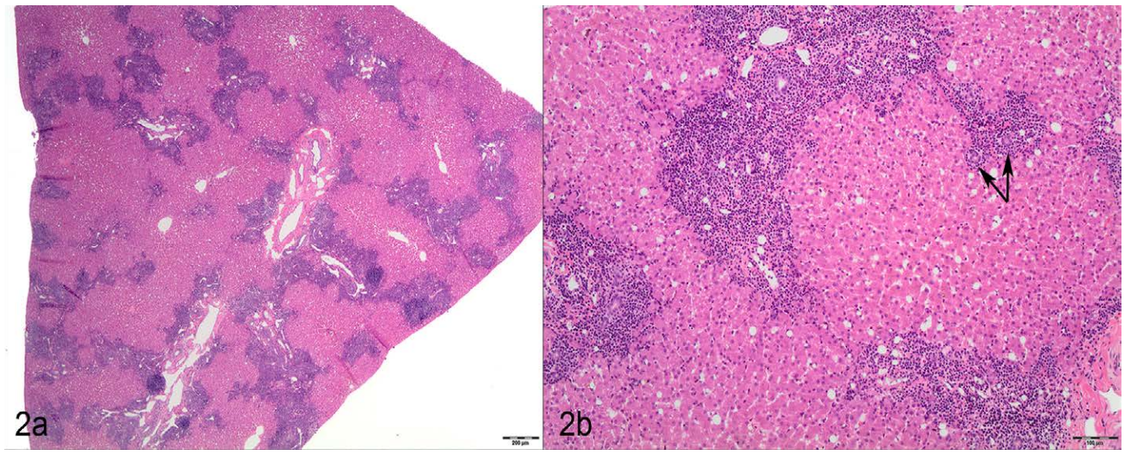

Figure 2. Lymphocytic cholangiohepatitis, liver, cat. 2a. At low magnification, portal tracts are obscured by a prominent cellular infiltrate that bridges adjacent portal areas. 2b. Neighbouring portal tracts are bridged by infiltrating round cells, accompanied by mild biliary hyperplasia (arrows). HE.

Lymphocytic cholangitis/cholangiohepatitis

This is a distinct, slowly progressive inflammatory disease with a poorly understood aetiology. A proposed immune-mediated pathogenesis is supported by the predominance of T lymphocytes within portal inflammatory infiltrates (fig. 2). Again, an association exists between lymphocytic cholangitis, pancreatitis and inflammatory bowel disease (triaditis). Sections of liver are characterised by portal infiltrates of small lymphocytes with variable peribiliary fibrosis and bile duct proliferation (fig. 3a). Distinguishing these reactive lymphocytes from neoplastic lymphocytes (lymphoma) can be particularly challenging when the inflammatory infiltrate extends into the hepatic parenchyma. Immunohistochemistry (IHC) for T and B lymphocytes (fig. 3b and 3c) may assist in differentiating the two processes by identifying a mixed population of cells[4]. PCR for antigen receptor rearrangements (PARR) may also assist diagnosis when histologic findings cannot distinguish the two processes with confidence. These PCR-based assays evaluate the diversity of T and B lymphocyte antigen receptor rearrangements in order to differentiate reactive (polyclonal) lymphocyte populations from neoplastic (monoclonal) infiltrates (fig. 3)[5].

KEY POINTS

- Lymphocytic cholangitis and lymphoma can be difficult to distinguish
- A staining panel that includes routine HE, Masson's trichrome (for peribiliary fibrosis), and IHC for T & B cells, and cytokeratin (epithelial marker) helps differentiate the two
- PARR may also be indicated

Neoplasia

Lymphoma is the most commonly encountered haematopoietic neoplasm in feline livers. Diagnosis is often complicated by histologic features that overlap with those of lymphocytic cholangitis (see above).

Primary hepatic epithelial neoplasms (hepatocellular carcinoma, cholangiocarcinoma) occur infrequently in cats.

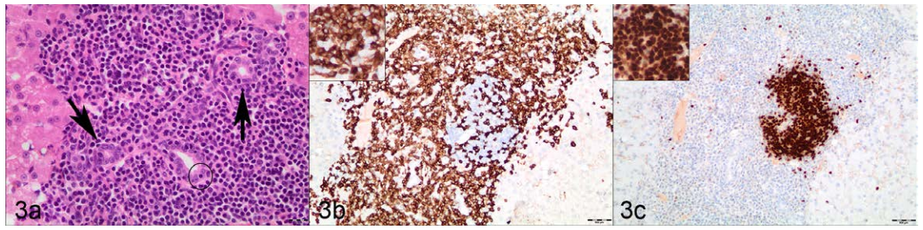

Figure 3. Lymphocytic cholangiohepatitis, liver, cat. 3a. Portal tracts are expanded and effaced by cellular infiltrates composed predominantly of small lymphocytes, which breach the margins of the portal tract and extend into the surrounding hepatic parenchyma. Lymphocytes infiltrate the biliary epithelium (circle) and small bile ducts are increased in number (biliary hyperplasia; arrows). HE. 3b. Immunohistochemistry (IHC) for CD3, a T cell marker, indicates the majority of infiltrating cells are T lymphocytes. High magnification inset: Strong membranous labelling for CD3 is detected in the majority of infiltrating round cells. 3c. IHC for a B cell marker, Pax5, demonstrates the presence of a lymphoid follicle within the portal inflammatory infiltrate. High magnification inset: Cytoplasmic expression of Pax5 confirms the presence of scattered B lymphocyte-rich lymphoid follicles, consistent with a chronic inflammatory process, rather than lymphoma. In this case, no evidence of T cell receptor clonality (i.e. neoplasia) was detected using PARR, confirming the inflammatory nature of the lesion.

Although hepatocellular carcinoma (HCA) is reportedly the second most common primary hepatic neoplasm in cats, prevalence is estimated at only 1-3% of all feline neoplasms[6]. HCA are typically slowly progressive and late to metastasize, presenting as solitary, locally expansile masses. Due to the benign histologic appearance of neoplastic cells, core biopsy is not recommended for definitive diagnosis. Even with wedge biopsies, histologic features of HCA may be difficult to distinguish from nodular hyperplasia and or regeneration. A detailed description of the lesion (imaging, appearance at surgery) is therefore critical to assist interpretation of histologic findings. When sampling large masses, biopsy of the margin of the mass is recommended to avoid sampling a necrotic centre.

KEY POINTS

- HCA is uncommon in cats and usually presents as a single, slow growing, expansile mass
- Diagnosis may be challenging with small samples (both core and wedge biopsies) as tumours often closely resemble normal tissue

Amyloidosis

In cats, hepatic amyloidosis tends to occur in the context of systemic amyloidosis associated with familial AA amyloidosis; however, amyloid deposition may occur secondary to chronic inflammatory or neoplastic disease (with overproduction of amyloid A). Familial AA amyloidosis is described in Abyssinian cats and is suspected in Siamese and Oriental breeds. Renal amyloidosis tends to predominate in this condition; however, amyloid deposition is often detected in the liver, surrounding portal tracts and within hepatic sinusoids, resulting in atrophy of surrounding hepatocytes (fig. 4). Affected livers are enlarged and predisposed to rupture and bleeding. Histologic detection of amyloid is often relatively straight forward at the time of diagnosis; however, additional stains (Congo red) may be required when amyloid deposition is not extensive.

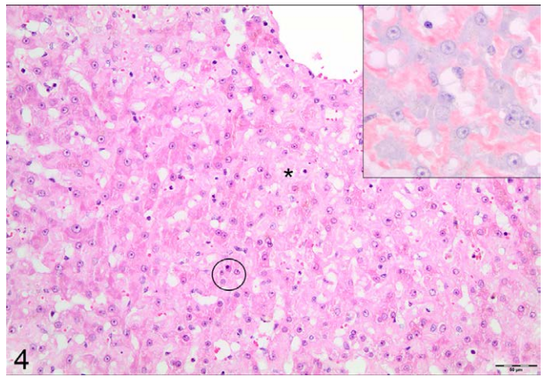

Figure 4. Amyloidosis, liver, cat. Diffuse accumulation of homogenous, pale eosinophilic extracellular matrix within hepatic sinusoids (asterisk) is accompanied by marked attenuation and atrophy of hepatic cords and frequent hepatocellular degeneration and loss (circle). Inset: A Congo red stain confirms the presence of amyloid, which is stained salmon pink (congophilic), with apple green birefringence under polarised light (not shown).

Hepatic Lipidosis

The terms hepatic lipidosis or steatosis are used to broadly describe the accumulation of triglycerides within the cytoplasm of hepatocytes. Lipidosis may be physiologic (e.g. late pregnancy or lactation) or pathological, as with feline hepatic lipidosis. Lipid accumulation also occurs readily in injured hepatocytes when the rapid transit of fatty acids and triglycerides may be obstructed at various points in the lipid metabolism pathway. Primary lipidosis should not therefore be confused with secondary lipidosis associated with an underlying hepatocellular insult (e.g. toxicity, hypoxia). Providing your pathologist with a relevant clinical history should avoid any confusion regarding the origin and significance of any hepatocellular lipid accumulation.

Hepatic lipidosis is readily recognisable in biopsy sections (in fact, lipidosis is probably the only disorder that can be reliably diagnosed by FNA). Triglyceride accumulation results in the formation of distinct clear vacuoles within the cytoplasm of hepatocytes. Accumulated triglycerides are not directly harmful to hepatocytes, but may reflect the duration and severity of the underlying injury. Steatosis is typically reversible, but renders hepatocytes more vulnerable to a range of toxic and nutritional insults.

The pathogenesis of feline hepatic lipidosis is poorly understood and likely multifactorial, with both increased uptake of non-esterified fatty acids and altered lipoprotein formation. It is possible dietary inanition reduces the availability of precursors of lipoprotein synthesis. Lipidosis also occurs secondary to other medical disorders, such as diabetes mellitus, hyperthyroidism, acute pancreatitis, small intestinal disease, neoplasia and concurrent inflammatory liver disease.

Hepatotoxicity

The liver is particular vulnerable to toxic insult due to its central role in biotransformation. Additionally, the portal vein drains the gastrointestinal tract, exposing the liver to high concentrations of ingested xenobiotics, including drugs, environmental toxins and plant and fungal-derived metabolites consumed in food. Factors affecting hepatotoxicity relate to the toxin in question, the mechanism of hepatic biotransformation and the susceptibility of the individual with respect to species, breed, age, nutritional status and intercurrent disease. Hepatic biotransformation follows a complicated series of pathways that varies with compounds and between species[1]. Briefly, Phase I reactions are mediated by cytochrome P450 monooxygenase enzymes. These are concentrated in the centrilobular zone, accounting for the stereotypical distribution of hepatocyte necrosis resulting from compounds metabolised by this system. Phase 2 reactions inactivate phase I metabolites through conjugation with glutathione and phase 3 reactions rely on transporter proteins to move conjugated molecules across the hepatocyte membrane into the canaliculus for excretion in bile. Differences in phase 1 and 2 activity between various species and breeds account for their distinctive susceptibilities to different toxins. Cats have limited phase 2 metabolism due to reduced activity of specific enzymes within this pathway, resulting in particular susceptibility to acetaminophen toxicity. Expression of transporter proteins required for phase 3 reactions also shows striking inter-species variability.

The gross and histologic characteristics of acute toxic liver injury range from centrilobular microvesicular lipidosis to massive coagulative necrosis and haemorrhage. Chronic hepatotoxicity is more variable in histologic appearance and may include areas of lipidosis and necrosis, fibrosis, nodular regeneration and bizarre cytologic changes. Chronic hepatotoxins favour the selective growth of hepatocytes that resist their mitotic inhibitory effects and may therefore be carcinogenic (e.g. aflatoxin).

Cats are typically quite discerning and ingest contaminated food and environmental toxins with less frequency than dogs. In cases of sub-lethal intoxication, biopsy may assist diagnosis. In cases of acute fatal hepatoxicity clinical findings are fairly consistent and animals often die before biopsy is possible. Cats may present with dullness, anorexia, abdominal pain, icterus and neurologic symptoms attributable to hepatic encephalopathy. Decompensation is often rapid and evidence of synthetic dysfunction (e.g. petechiae, ecchymoses, and hypoglycaemia) may be present. Gross and histologic changes are consistent regardless of the causative toxin; however, toxicology (from tissue obtained at necropsy) may be indicated when other animals and people are at risk of exposure.

KEY POINTS

- Acute hepatotoxicity is often characterised by centrilobular lipidosis and necrosis
- Necropsy may assist definitive diagnosis in cases of acute lethal hepatotoxicity

Extrahepatic Changes

Rarely, animals may present with a primary complaint of dermatitis, secondary to severe liver disease[7]. Hepatocutaneous syndrome has been reported in cats and like dogs, is associated with severe vacuolar hepatopathy. Skin lesions are characterised by erythema, crusting, exudation, ulceration and alopecia with a distribution around the eyes, mouth, anogenital region and on pressure points such as paw pads.

Getting the most out of your Biopsy/Pathologist

Interpretation of hepatic histopathology is made within the context of the clinical picture. At an absolute minimum, describe the number and distribution of lesions within the liver on your submission form – this may be based on imaging findings, or your observations at surgery. Ideally, also provide relevant clinical information including signalment, duration/chronicity, pertinent haematology and biochemistry findings, evidence of intercurrent disease and your primary differential diagnoses. The latter is particularly important since

changes within the liver often constitute a stereotypical or non-specific response to injury. Providing a differential list helps your pathologist understand how various histologic changes might correlate with your clinical findings.

Some pathologists routinely run additional histochemical stains to assist diagnosis, clarify histologic changes or provide practitioners with additional information that may assist prognostication and treatment. For example, a trichrome stain for collagen may indicate the onset of fibrosis, warranting anti-fibrotic therapy. Some of the changes we can detect in liver sections include:

CHANGE	HISTOCHEMICAL STAIN
Collagen (fibrosis)	Masson's trichrome
Iron	Perl's prussian blue
Copper	Rhodanine red
Reticulin fibres	Reticulin (silver stain)
Bile	Hall's bilirubin
Amyloid	Congo red (fig. 4)

Immunohistochemistry for lymphocytic and epithelial markers is usually available at an additional cost and may assist in distinguishing aggressive inflammatory lesions from neoplastic infiltrates (e.g. lymphocytic cholangitis vs lymphoma), and identifying/phenotyping neoplastic infiltrates. Your pathologist may contact you for permission to run these additional tests; occasionally practitioners will give permission to spend the additional money (up to a point) on their submission form in order to streamline the process.

In some instances, a definitive diagnosis may not be determined by biopsy, particularly when severely injured livers are characterised by marked fibrosis and remodelling. When this is the case, additional discussion with your pathologist may be instructive, particularly if response to therapy or changing clinical signs refine the differential list.

Pancreas

This section is brief, as biopsy is not typically the primary approach to diagnosis of either exocrine or endocrine pancreatic disease.

Acute feline pancreatitis is often managed medically without biopsy. At laparotomy, gross changes are often easily recognisable and histologic verification may not be necessary. Chronic pancreatitis is more challenging to recognise grossly and biopsy may be required to confirm the presence of inflammation and fibrosis.

Often inflammatory pancreatic disease is investigated in the context of concurrent hepatic and enteric inflammation. Biopsy is required for definitive diagnosis of triaditis, so that characteristic inflammatory infiltrates can be demonstrated in each tissue[8]. Histologically, chronic pancreatitis is characterised by interstitial lymphoplasmacytic infiltrates and dissecting bands of fibrosis, often given the parenchyma a slightly nodular appearance. This may be confused with exocrine nodular hyperplasia, an incidental age-related change that is not associated with any preceding pancreatic injury.

Exocrine pancreatic adenocarcinoma is uncommon in cats and is often diagnosed late in disease progression, when metastasis is widespread. The gross appearance of pancreatic adenocarcinomas can be variable. Occasionally tumours present as solitary, circumscribed masses, although more often, tumours are infiltrative with poorly circumscribed margins, and an accompanying connective tissue reaction that may mimic severe pancreatic fibrosis. Rarely, cats with pancreatic adenocarcinoma may present with a paraneoplastic dermatopathy characterised by alopecia of the face, legs and ventrum[9].

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