THE UNIVERSITY OF SYDNEY
Centre for Veterinary Education
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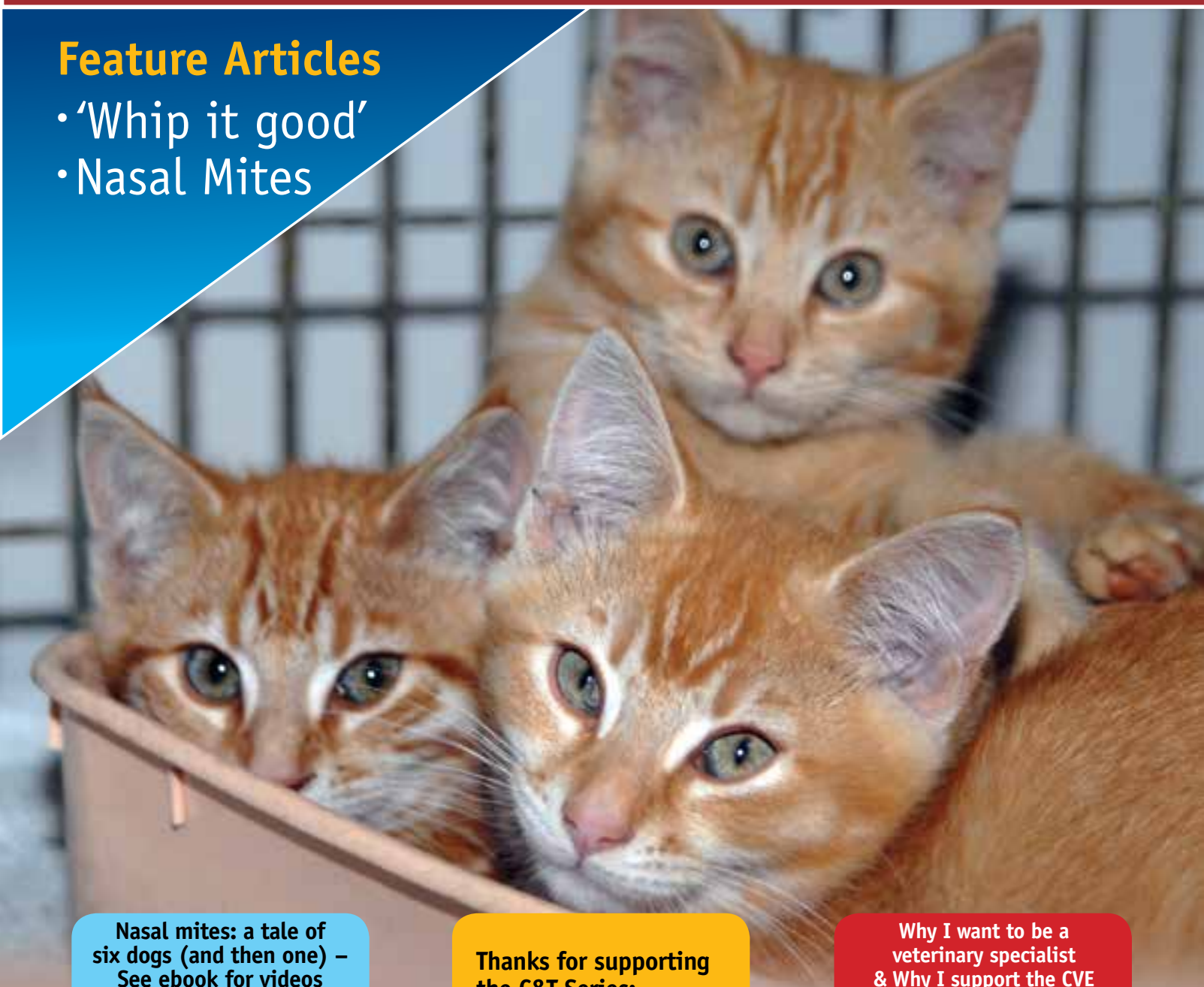
C&T

CONTROL AND THERAPY SERIES

September 2013 ISSUE 272
Australia's Leading Veterinary Forum

Feature Articles

- 'Whip it good'
- Nasal Mites



Nasal mites: a tale of six dogs (and then one) – See ebook for videos

Thanks for supporting the C&T Series:-

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Why I want to be a veterinary specialist & Why I support the CVE

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C&T
CONTROL AND THERAPY SERIES

The Centre for Veterinary Education (CVE)
T. (02) 9351 7979
F. (02) 9351 7968
E. cve.publications@sydney.edu.au
www.cve.edu.au
Level 2, Vet Science Conference Centre,
B22, Regimental Crescent, The University of Sydney, NSW 2006.
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SEPTEMBER 2013 ISSUE 272 Australia's Leading Veterinary Forum

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PUBLISHER
The Centre for Veterinary Education (CVE)
Director
Hugh White BVSc MVSc MACVSc
EDITOR
Elisabeth Churchward
T. 9351 7979
E. cve.publications@sydney.edu.au
Veterinary Editors
Hugh White
Richard Malik
ADVERTISING ENQUIRIES
Elisabeth Churchward
T. 9351 7979
E. cve.publications@sydney.edu.au
DESIGNERS
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COVER IMAGE
Courtesy of Anne Fawcett

A couple of months ago we learned of the sudden and unexpected death of one of our Distance Education participants. As always, this came as a great shock to her tutor, the DE team and all of us at CVE. It reinforced the fact that suicide is an ever present reality, particularly for younger idealistic vets in Australia. Sadly, suicide rates in veterinarians are nearly double the national average.

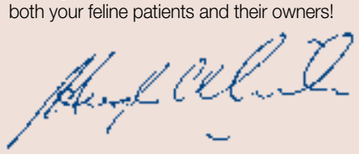
At the last AVA annual conference in Cairns, there was a lot of discussion about this issue and the various factors which may contribute to the problem. The WA Division of AVA has been very active in running a mentoring scheme for new graduates and the AVA is keen to roll this out as a national initiative – see http://www.ava.com.au/system/files/private/Mentor%20program%20guide%20final.pdf

The CVE has also partnered with the Department of Psychiatry at Westmead Clinical School, Sydney Medical School, University of Sydney to advertise and promote CPD programs for the benefit and welfare of veterinarians. CVE members can get discounted registration and earn CPD points for these courses. So far, two courses have been offered this year – **Anxiety Disorders** see http://www.cve.edu.au/news/anxietycourses and **Take Control of your Worry**, which starts on 14th October – see http://www.cve.edu.au/takecontrolofyourworry

All of us within the profession should be aware of the insidious signs signalling anxiety and depression and be aware of the help available to those in need. The broader issue of addressing the causes of depression so that suicide may be averted, is a subject much larger than can be addressed in this column, but is a discussion the whole profession needs to have around work conditions, support, low wages and self-esteem issues. If you wish to read more about what we should be looking for in others around us, the following article is well worth a read – it contains many useful references for further reading: http://www.veterinaryteambrief.com/article/impaired-veterinarian-recognizing-depression-possible-suicide

This quarter we have another 'bumper' issue with 64 pages, as our editor Lis Churchward has been overwhelmed with material for publication, resulting in an unacceptable backlog. Once again we wish to thank all our contributors and the companies whose advertising support has helped us to absorb the cost of a larger publication. Recently we have had many positive comments about C&T and the new interactive PDF format, which literally brings many articles to life. Don't miss the contributions by our regulars – Aine Seavers, Anne Fawcett, Marshall Thornton and Peter Howe, nor the three Perspectives by Linda Fleeman, Terry King and Gary Norsworthy.

There is also a special article promoting the **Cat Friendly Clinic Accreditation Scheme**, which is an initiative of ISFM – the International Society of Feline Medicine. ISFM is a partner with CVE in our Feline Medicine Distance Education course, where we have roughly equal numbers of the 60 participants from Europe/UK, Asia and Australia/NZ. Read Andrea Harvey's article on page 4 about how your clinic can benefit from participating in this scheme, which is being promoted around the world and which will be a winner with both your feline patients and their owners!



Hugh White BVSc MVSc MACVSc
DIRECTOR

Long-term CVE supporter Anne Fawcett and 'Phil' meet the Deputy PM

SCRAP THE CAP!

www.scrapthecap.com.au

Deputy Prime Minister Anthony Albanese held a meet and greet recently in the inner west. It was a great opportunity to meet him in person and discuss the proposed $2000 cap on tax deductible self-education expenses. Whilst I have written to Mr Albanese and made a submission in response to the treasury discussion paper, there are doubtless many other important issues that politicians have to consider and it's easy for concerns about one matter to be lost in the noise. So I took my dog Phil and was able to chat with Mr Albanese (a proud pet owner himself) for about 10 minutes. He was certainly sympathetic to the argument that the cap should be scrapped, but I firmly believe that whether this will occurs depends on the response from the veterinary profession as well as others. If you haven't yet had a chance to contact your local member it's a good time to do so.

DISTANCE EDUCATION 2014

Early Bird Option 2 ends 31 October 2013

Register and pay the $1,000 deposit to secure your place and then pay in full by 31 October 2013 AND save up to $200 on course fees

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Visit our website (www.cve.edu.au) to find out more about our programs or you can register your interest by emailing us at **cve.events@sydney.edu.au.** Listed dates are subject to change. Refer to www.cve.edu.au, for any updates.

EVENTS IN 2013

23-26 Sept	Orthopaedic Conference	Fremantle
5 or 6 Oct	Hip & Stifle Workshop	Brisbane
13 Oct	Diabetes	Brisbane
19 Oct	Basic Echocardiography Workshop	Sydney
20 Oct	Advanced Echocardiography* Workshop	Sydney
27 Oct	Looking Down the Microscope	Port Macq.
8 Nov	ecoCPD: Behaviour	Sydney

* Prior learning will be required to attend this workshop.

TimeOnline

ONLINE COURSES IN 2013

2 Sept - 29 Sept	TimeOnline: Marine Wildlife **(Students only)**
28 Oct - 24 Nov	TimeOnline: Anaesthetic Complications
4 Nov - 1 Dec	TimeOnline: Avian

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For more information on any of our TimeOnline courses please visit www.cve.edu.au/timeonline or email cve.timeonline@sydney.edu.au

Take control of your worry

Co-presented by the CVE & the Discipline of Psychiatry, University of Sydney

Cost: CVE members $261 (Non CVE members $290, inc. GST)

6 CVE CPD points (awarded on confirmation course has been completed)

According to the latest research, veterinarians have higher levels of depression, anxiety, stress and burnout than the general population. It has been suggested that opportunities to enhance veterinarians' cognitive and coping skills be provided throughout their veterinary career.

The CVE has partnered with the Discipline of Psychiatry, Sydney Medical School at the University of Sydney to bring you a brand new psychoeducation course, Take Control of Your Worry, a three-week structured program designed for those who wish to apply proven effective strategies to gain control of their excessive worry and anxiety.

Tutor Dr Lisa Lampe is author of the book 'Take Control of Your Worry' (and participants receive a free copy).

www.vetbookshop.com

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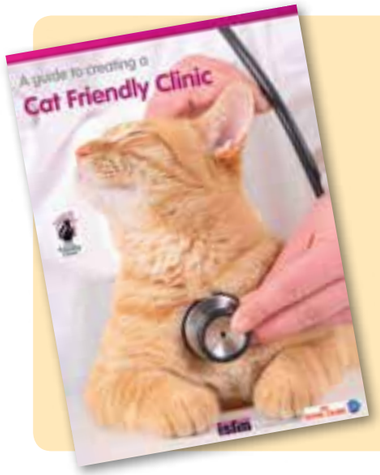
CVE News

ISFM Cat Friendly Clinic Accreditation Scheme is Launched in Australasia



The International Society of Feline Medicine (ISFM) has just recently launched their 'Cat Friendly Clinic' accreditation scheme in Australasia, in collaboration with Royal Canin and CEVA. Andrea Harvey – a CVE Feline Distance Education Tutor and feline Specialist at Small Animal Specialist Hospital (SASH) in Sydney – is the Australasian Representative for ISFM and is overseeing the accreditation scheme in Australasia. Here, Andrea tells us more about the scheme, and how to get signed up for further information.

In the December issue of C&T, a more in-depth article will feature Andrea's top tips on how to create a more 'cat friendly' environment within veterinary practices.

FREE INFO PACK

Receive a FREE 'cat friendly clinic' information pack which includes a full veterinary guide, all the details of scheme and how to apply for accreditation, by visiting: http://tinyurl.com/isfmcfc (or go to the International Cat Care website for further information: www.icatcare.org)

The number of pet cats, and the value of cats as part of the family, is increasing worldwide, but many cat owners avoid veterinary visits because they know it is stressful for both their cat and them. Making veterinary clinics 'cat friendly', and letting clients know that we understand their concerns are vital in addressing this. Unless we get these basics right we won't get many feline patients coming through our clinic door, and continuing to come back!

A stressed cat may be more difficult to handle, they may exhibit fear aggression and be challenging to examine or take blood samples from. When you do examine them they may be tachypnoiec, tachycardic and pyrexic, simply as a result of stress. You may perform further diagnostic testing, and a stressed cat may be hypertensive, hyperglycaemic, even alkaluric. If you hospitalise them, they may not eat, urinate or defaecate. And how do you assess the demeanor of a stressed cat outside of it's normal environment? When we are trying to diagnose disease, monitor illness, monitor and treat pain, how can we possibly do a good job as veterinarians if our feline patients are stressed? We simply can't begin to practice good feline medicine until we can address these factors. To do this, we need to understand the way that cats think, what makes them stressed and why.

I am absolutely passionate that having a 'cat friendly clinic' forms an essential foundation for all feline practice. The majority of factors that cause anxiety in cats can be at least partially overcome, and seeing the transformation in your feline patients when you do so is extremely rewarding. Often as vets we are good at focusing on complex problems and missing the small simple things that make a big difference. Creating a 'Cat Friendly Clinic' is often about simple things that can be easily achievable.

The ISFM 'Cat Friendly Clinic' accreditation scheme is designed to help and encourage veterinary clinics to be proactive in making visits less stressful to cats and improving the standards of veterinary care for cats, in addition to recognising clinics that do take a different approach to cats and demonstrating these differences to clients.

The ISFM Cat Friendly Clinic was started in the UK initially as a competition in 2006, and after huge success and enormously positive feedback from clients, veterinary clinic staff and business owners, the more formal accreditation scheme was launched, first in the UK and then gradually being rolled out to other European countries as well as having being adapted with the AAFP and taken up in North America. Having been heavily involved in developing the Cat Friendly Clinic scheme since its infancy, I was keen to roll out the scheme in Australasia when I moved here in 2012, and so I am really excited that it has now been officially launched in Australasia with the generous support of Royal Canin and CEVA. There has already been great interest in the scheme with over 100 vet clinics throughout Australasia having registered their interest so far.

Being committed to 'practicing what I preach' and ensuring that criteria are achievable, I immediately set out to transform the clinic that I had started working at in Sydney into a 'Cat Friendly Clinic' to be able to provide a good example for other clinics to follow. Following a visit from Dr Andy Sparkes (ISFM Veterinary Director) earlier this year, I was delighted that SASH has been awarded the first ISFM gold standard cat friendly accredited clinic in Australasia. Dr Andy Sparkes commented that he was particularly *'impressed how everyone at SASH had embraced the ethos of the programme so fully to create a truly Cat Friendly environment'*. Every clinic presents its own challenges, but the key starting point for any clinic is exactly this; getting all the veterinary clinic staff on board so that everyone buys into the 'cat friendly clinic' ethos. I was lucky to work with a fantastic team at SASH where everyone pulled together to further improve all the feline facilities, and protocols. Investing time in educating staff, involving them in decisions and having leadership from a 'feline advocate' in the clinic really pays off in being able to implement any necessary changes successfully.

For more information, top tips, common hurdles and ways to overcome them, look out for Andrea's 'cat friendly clinic' article in the December issue of the C&T Series.

Andrea Harvey
BVSc DSAM(Feline) DipECVIM-CA MRCVS
RCVS Recognised Specialist in Feline Medicine
European Veterinary Specialist in Internal Medicine
CVE Feline Distance Education Tutor

FOR MORE INFORMATION, CONTACT ANDREA

As well as overseeing the accreditation of clinics in Australasia, Andrea is always happy to take enquiries and help provide any guidance required on how to become more 'cat friendly'. Having first-hand experience with implementing changes required for accreditation she is only too familiar with the challenges and hurdles that clinics can be faced with.

If you have specific queries about the accreditation scheme, please do not hesitate to contact Andrea at: aharvey@sashvets.com

Authors' views are not necessarily those of the CVE

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… and more C&T articles and Perspectives needed
Thanks to every author who contributed articles or comments to the *Control & Therapy Series* (C&T). Without your generosity the Series would cease to exist.

In order to reduce the publishing 'queue' we have produced a 'bumper' 64-pager this issue and in recognition of the calibre of articles in this issue, have awarded 2 Major Prizes.

Winners

Major Prizes

Entitling the recipients to one year's free membership of the CVE

- **Naomi Lessels:** 'Whip it good'
- **Sue Foster & Jody Braddock:** Nasal Mites

CVE Publication Prize Winners

- **Harry Corbett:** Iodine Goats.
- **Deborah Marriott:** The hidden pearls of paradise.
- **Al Warner:** Stabilisation of a fractured mandible in an Eastern Grey Kangaroo

Winner of Best Film Clip

- **Mimi Dona:** Echidna restraint

Winner of Best Pictures

- **May Chin Oh & Anne Fawcett:** Linear foreign body in a dog

Don't miss reading the ebook!

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- Sue Foster's Nasal Mites video
- Aine Seavers' 'reverse sneezing' videos and much more…

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Then visit **www.cve.edu.au/candtebook** which allows you access to this current issue in e-book format and the 4 prior issues.

Contact
For all enquiries regarding the *Control & Therapy Series*, please contact The Editor, Elisabeth Churchward at **cve.publications@sydney.edu.au** or call (02) 9351 7979.

The C&T and Perspective Series is the brainchild of Dr Tom Hungerford, first Director of the PGF (1968-1987), who wanted a forum for uncensored and unedited material. Tom wanted to get the clinicians writing.

'…not the academic correctitudes, not the theoretical niceties, not the super correct platitudes that have passed the panel of review… 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, when tired, at night, in the rain in the paddock, poor lighting, no other vet to help.'

WINNER OF BEST FILM CLIP

Compiled at the Currumbin Sanctuary Wildlife Hospital by Mimi Dona © 2010

Part 4: Wildlife Flashcard Series

Mammals

C&T No. 5322

*This series is the result of collaboration between Mimi Dona & Dr Michael Pyne of Currumbin Wildlife Sanctuary Veterinary Hospital. Non CVE members can access these flashcards and videos at **www.cve.edu.au.***

Mimi Dona

Senior Veterinary Nurse – Currumbin Wildlife Sanctuary Veterinary Hospital (CWS) & Lecturer on Animal Studies and Sustainability at the Metropolitan South Institute of TAFE.

(e-book) Film clip courtesy of Lincoln Williams, Fotomedia **(www.fotomedia.com.au)**.

Echidnas

Part 4.3

ECHIDNAS

Be aware of:-

- Ectothermic; body temperature is influenced by their surroundings.
- Nocturnal during warmer months, often seen out through the day during colder months.
- Echidnas can die from heat stress; they are unable to cool down using familiar mammalian tactics.
- Echidnas have a very sensitive beak; clear frothy nasal discharge is normal.
- Fractures will often only be detected via radiographs; the beak is commonly fractured when subjected to road trauma.
- Sexing Echnidas is difficult; experienced Wildlife Veterinarians use ultrasonography (internal testicle) or palpate the penis in males once anaesthetised.
- Females contract abdominal muscles to form a pouch to carry their young; in the second stage of parental duties the mother leaves and periodically feeds the Puggle in a burrow. This needs to be considered in their treatment plan and release location, as the Puggle will still be milk dependant.
- Females do not have nipples but instead excrete milk via their pores in their 'pouch'.
- The easiest method to take blood is from the blood filled sinus vein that is just caudal to the external nares on the dorsal aspect of the beak. Anaesthetise the echidna first and use a 25 gauge winged infusion needle to gently obtain 1 - 2 mLs.
- Ticks are commonly seen on Echidnas.

Handling

- To pick up an Echidna wear gloves or use a large towel (folded-over), place hands on either side of its body between the forearms and hind legs. It will naturally curl into your grip, lift up and support against your body. Bare hands can be used once comfortable with the technique.
- Sometimes when strong and active they will push themself into the corner of the container; lift up as a ball and correct your grip once they open out.
- Young Echidnas (un-spined or just-spined) can be gently picked up with one hand by supporting the body and cupping your hand. ▶

Figure 1. Gloves will not impede manual dexterity when handling an echidna.

Figure 2. Echidnas naturally curl around your hands making a secure hold.

Figure 3. A juvenile echidna is called a puggle.

Housing the sick or injured Echidna

- Preferred enclosure temperature is between 18˚ - 25˚ Celsius.
- If cold, heat can be given by placing under half of the enclosure. Never provide heat if the patient is unable to move. This must be monitored carefully and care taken not to over-heat.
- If hot, reduce the temperature by placing an ice brick under a towel or a corner of the container.
- Adults can be housed in a smooth sided ventilated tub with towels to line the bottom and create a hide (clean rubbish bins with holes drilled in the lid work very well).
- Make sure the sides are high and the lid is well sealed, as they are very good at escaping.
- Unspined orphans can be placed in a cotton pouch in an aquarium or Esky® with lid removed or wedged open. Place in a cool spot and monitor the temperature closely with an indoor/outdoor thermometer keeping below 25˚ Celsius.

Figure 4. Towels or ripped up newspaper can be used to help the echidna make a 'hide'.

Figure 5. Garbage bins are excellent for transporting, or temporarily housing an echidna.

Emergency diet

- **Adult** echidnas have a very specialised diet and if unsure can go weeks without food if in good body condition and hydrated or being given fluids. Adults can be fed soaked and mashed up dog kibble or Hills a/d® on a shallow dish. Always offer a bowl of water. Stomach tubing may be required if not self-feeding, specialist advice with this technique is recommended.
- Echidnas can be offered termite mounds as a natural source of food.
- **Puggles** require specialist care and need fostering immediately. Experienced carers will feed a hydrated Puggle milk formula (Divetelact®, <0.3 Wombaroo Echidna Milk®, >0.3 Wombaroo Echidna Milk®) by dribbling milk into their cupped palm or bowl. This mimics how it naturally feeds off mum (lapping). Stomach tubing may be required if not self-feeding, specialist advice with this technique is recommended.

Figure 6. Juvenile echidnas are fed by dribbling milk into a cupped palm or bowl; this mimics how it naturally feeds off mum.

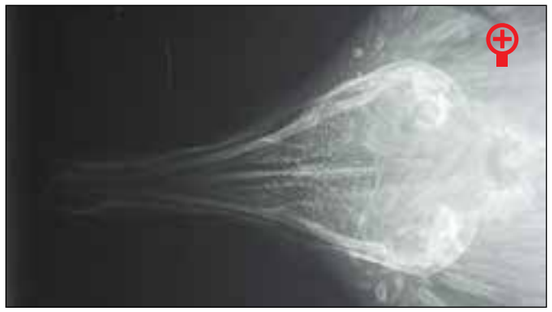

Figure 7. The beak is commonly fractured when subjected to road trauma; this can only be detected via radiographs.

Assessment under anaesthetic

It is not uncommon for Echidnas respiratory rate to drop very low under general anaesthesia; 2 breaths per minute or lower. The heart can be very difficult to auscultate.

Gaseous

Due to their unusual shape beak it is best to make a mask out of a syringe (end removed) or syringe case, wrapping co-flex at the end to create a seal. Two people are required to deliver the anaesthesia, one to hold and the other to hold up the beak and try and place the mask. This can require some patience as the Echidna burrows its nose into itself, if not successful you can use this to create a temporary gas chamber until it relaxes enough for you to get placement.

Use an anaesthetic mask at 5% induction, can take up to 5 minutes. Or if there are difficulties handling the echidna, place in an anaesthetic box. Maintain using a mask on Isoflurane® at 1.5 – 2% with an oxygen flow rate of 1 L/min.

Figure 8. For a better induction, make a mask out of a syringe case to fit their unusual shaped beak.

Figure 9. Echidnas and the platypus are the only egg-laying mammals, known as monotremes.

Injectable

Alfaxan® CD RTU 3 mg/kg – (I/M or I/V)

- Injectable agents in Echidnas can lead to long recovery times. Due to this and the difficulty with intubation, gaseous anaesthesia is recommended.
- If the beak is damaged and gaseous anaesthesia is not effective due to the limited oxygen intake then an analgesic injection is recommended (Methadone® - 0.3mg/kg). If Injectable anaesthesia is the only option careful monitoring and a low dose is required due to the inability to intubate.

Recovery

Check cloacal temperature and use the room temperature to maintain the patient's core body temperature throughout the procedure; only give heat if required and monitor closely.

Fluid Therapy

It is important to remember to warm the fluids being administered. Using 0.9% sodium chloride, dose the patient at 5% of its bodyweight. Fluid therapy can be administered subcutaneously or by standard I/V infusion rates. If using the sinus vein the patient must be anaesthetised.

Preferred routes for drug administration

- **Subcutaneous** – administered in loose skin on underbelly.
- **Oral** – given via a syringe or stomach tube.
- **Intramuscular** – administered to forearm muscle, hind limb muscle and gluteals. You need to use a long 1 ½ inch needle to get between the spines.
- **Intravenous** – beak sinus (preferred), cephalic or femoral vein.

Figure 10. Echidnas have a blood filled sinus vein on their beak, just caudal to the external nares.

Euthanasia methods

Injection of Sodium Pentobarbitone® can be administered either by intravenous, intracardiac or intraperitoneal routes.

- An Echidna requires a general anaesthesia for euthanasia unless moribund.

WINNER

Stabilisation of a fractured mandible in an Eastern Grey Kangaroo

C&T No. 5323

Al Warner BVSc, MAppSc (Wildlife)
Holroyd Veterinary Clinic
381 Merrylands Rd
Merrylands NSW 2160
T. (02) 9637 6075
E. alan@holrodyvetclinic.com.au

A female Eastern Grey joey of approximately 11-months-of-age was presented by a wildlife carer who had observed the animal to take fright and collide with a solid fence.

There was swelling in the middle region of the body of the right mandible. The animal had been sedated to travel to the clinic with diazepam and on presentation general anaesthesia was induced by masking her down with 5% isoflurane. Radiology revealed a fracture of the right mandible just rostral to the first premolar. Some movement could be detected at the fracture site by digital manipulation but there was only a slight misalignment of the mandibular segments.

In this species the 2 bodies of the mandible are not fused in the midline at a symphysis. Instead there is a fibrous joint that allows movement between the 2 rami that is said to allow a scissor-like action with the 2 large chisel shaped lower incisors while grazing.

Various methods of stabilizing the fracture were considered and I elected to anchor the rostral end of the fractured segment to the opposite mandible at the rostral extremities of the mandibular bodies. I did not attempt to stabilise the caudal segment in which only slight movement could be induced on manipulation. We reasoned that the fragment of bone should be more stable if it were fixed at 1 end than if both ends of the fragment were free to move. I proposed to stabilise the rostral segment in a similar manner to stabilising a symphaseal fracture in a cat. However due to the shape of the incisors it is not possible to simply wire around the base of the teeth as is possible in a cat. The method I choose was to drill an approximately 1 mm hole in a rostro-caudal direction through the center of each incisor with a dental burr. Through these holes size 0 Supramid™ (Braun) non absorbable suture material was passed and knotted (Figures1 & 2).

Figure 1. Incisors drilled & Supramid™ inserted.

Figure 2. Supramid™ knotted & incisors stabilised.

After the incisors had been secured there was very little movement at the fracture site. This joey was an orphan that was being hand reared on formula and had recently been introduced to solid food. Oxbow Critical Care[1] coarse ground formulation was added to the milk formula so that the joey did not have to chew and masticate solid food but still had fibre and essential nutrients in her diet. We estimated the fracture would heal over a matter of 3 or 4 weeks and that we could remove the Supramid™ suture and repair the defects in the incisors at the end of that period. As it happened the repair of the holes was done after a period of 4 weeks due to floods preventing travel to the clinic. (Any other year it would likely be bushfires). There was no movement palpable when the animal was examined under anaesthesia prior to the repair of the holes.

We decided to repair the drilled holes in the incisors with light cured glass reinforced ionomer which is presented in kit form and manufactured by Fuji Corp[2].

Preparation of the teeth to be filled entailed undermining the dentine between the anterior and posterior enamel layers to a depth of approximately 1 mm and the application of the cleaner to the dentine and surrounding enamel surfaces. Undermining the dentine creates a cavity that the composite can key into to prevent the infill from dislodging. The composite is prepared by mixing predetermined amounts of the powder and liquid components. There is a 2½ minute working time before the mixture starts to set. The setting of the composite is facilitated by the application of blue light from the visible light spectrum for 20 seconds. A small hand held cold blue light source is used for this procedure. The result of the repairs can be seen in Figure 3.

Figure 3. Defects in incisors repaired.

Figure 4. Fuji BOND dental composite (sourced from the internet).

The successful outcome of any surgical procedure on wild animals depends on the age, size, temperament and condition of the particular animal and the skill and commitment of the carers who nurse them. I am fortunate to work with dedicated, knowledgeable and experienced carers. Few practicing veterinary surgeons have the time and facilities to adequately nurse and rehabilitate macropods.

I am indebted to Dr Bill Sundin, a friend and Dental Practitioner, who lent me the kit to repair the holes in the incisors.

References

1. Oxbow Critical Care. Oxbow Animal Health. 29012 Mill Road, Murdock, NE 68407 USA info@oxbowanimalhealth.com
2. Fuji Corp, Tokyo, Japan.

Surgical correction of bilateral flexural forelimb deformity in a foal

C&T No. 5324

Dr Melissa Ford BSc. BVMS and Mr James Meyer BSc.PV
Strathalbyn Veterinary Clinic
32 Love Avenue
Strathalbyn SA 5255
T. (08) 8536 2722
E. james.n.meyer@student.adelaide.edu.au

Introduction

Congenital abnormalities can occur in a range of systems and are often clinically observed early in the life of a neonate. The musculoskeletal system can be affected by a wide array of conditions that usually result in co-ordination difficulty exhibited by the foal in the immediate postpartum period. 'Spider', an 8-week-old, bay Thoroughbred colt, underwent extensive medical and surgical therapy to correct bilateral congenital forelimb flexural deformity.

Initial Visit

Born of the 6th October 2011, the foal presented having not suckled up to 6 hours postpartum. The foal was weak on all 4 limbs and was not able to remain standing without assistance. The front limbs were observed to cross over readily and the foal appeared to have very little control over his movements.

All vital signs were within normal limits. The foal had a weak suckling reflex and was dehydrated with an increased skin tent. The foal remained in sternal recumbency and, upon orthopaedic examination neither forelimb was able to be extended fully at the carpus.

Differential diagnoses for the limb abnormalities include: flexural deformity (contracted tendons), as well as angular deformity of the carpal regions. Flexural deformity is the most probable diagnosis as it commonly presents bilaterally[1] and this case is not associated with valgus, or varus, rotation of either forelimb.

The aim of the first 24 hours was to provide nutrition, fluids and maternal antibodies. 500mL of the mare's colostrum was administered via a nasogastric tube. Confinement was also recommended to allow the foal time to strengthen his forelimbs before trying any other management. The following day an IgG test (Gamma Check® E, Plasvacc Pty Ltd) was performed and indicated failure of passive transfer, and a plasma transfusion was administered (Equiplas®, Plasvacc Pty Ltd).

Also during this visit the limbs were reassessed for deformity. The foal was classified as having a Grade 2/3 bilateral congenital forelimb flexural deformity as he was born with the inability to completely extend the carpii to its normal position. The decision was made to administer oxytetracycline 20mg/kg IV (Terramycin 100, Pfizer Australia Pty Ltd), along with splinting of the legs, to aid in the stretching of the contracted tendons of the forelimbs.

Over the next 6 weeks splint therapy, with a repeat dose of oxytetracycline 3 days after the initial administration, was continued with periods of disuse in between to prevent pressure sores from developing and to better assess the success of the treatment. The combination of oxytetracycline and splints achieved a 30-40% improvement in relaxation of the forelimbs that allowed the foal to stand up on his own. However, he displayed a lack of muscle development in the shoulders and forelegs due to minimal use of the limbs and, as can be seen in Figure 1, marked contraction of both forelimbs was still evident.

Figure 1. (A) Spider after 6 weeks of therapy with oxytetracycline and splints to correct flexural forelimb deformity. (B) Highlighting the large angle still displayed in the forelimb despite 6 weeks of treatment. Pictures taken on the 25th November 2011.

Little improvement was subsequently made so at 7 weeks of age it was deemed unlikely that continued treatment with the current regime would produce any further improvement in the foal's condition. The next step in treatment would involve surgery to release the contracted tendons and/or ligaments to allow the foal to straighten his legs.

Surgery

On the 2nd of December 2011, the mare and foal were brought into the clinic to perform the bilateral forelimb tenotomy on the foal. Documented procedures indicated that it was important to palpate the flexor tendons under sedation, prior to surgery, to ensure that the correct tendon(s) of interest were actually involved in the deformity[1,6]. Radiographs of the carpal joints (Figure 2) were then performed to ensure no other developmental abnormalities were present that may decrease the prognosis of surgical intervention.

Some cases may involve incomplete ossification or wedging of the carpal bones and this can greatly decrease the prognosis of surgical intervention, because although the flexor tendons are to be released, the carpus may still be unable to extend to a normal position or have decreased range of motion. All of the radiographs showed normal neonatal carpal anatomy indicating that no reduction in range of motion should occur from other structures in the carpus during release of the flexural tendons and therefore the prognosis for surgery was good.

Figure 2. Bilateral radiographic examination of the carpus of the foal. (A) Lateromedial view of the left carpus, (B) lateromedial view of the right carpus, and (C) bilateral dorsopalmar view of the carpus. All radiographs show normal neonatal carpal anatomy indicating that no added resistance should occur from the carpus during release of the flexural tendons.

Surgery

Following sedation and radiographs, surgery was commenced, with the foal placed in left lateral recumbency. Following surgical skin preparation a 5cm lateral proximal-distal skin incision was made, centred at the level of the distal radial. The tendon of insertion of the ulnaris lateralis muscle, deep to the fascia, was identified and exposed by blunt dissection. The tendon was transected approximately 2cm above the insertion at the accessory carpal bone (Figure 3A). Care was taken to avoid the lateral palmar vein and nerve during transection.

The flexor carpi ulnaris tendon was then confirmed deep to the incision and was palpated for tension, and the muscle was taut on palpation. Therefore it was transected at the same level as the ulnaris lateralis tendon. Final palpation of the remaining tendons was performed to ensure no undue tension existed and then the incision was closed in a standard fashion. Once closed, the range of motion of the limb was tested and showed almost complete extension.

The foal was then rotated into right lateral recumbency to allow access to the left forelimb. The surgery was repeated in the same manner on the contralateral limb. On range of motion testing, improvement was noted but there was still a small degree of limb contracture.

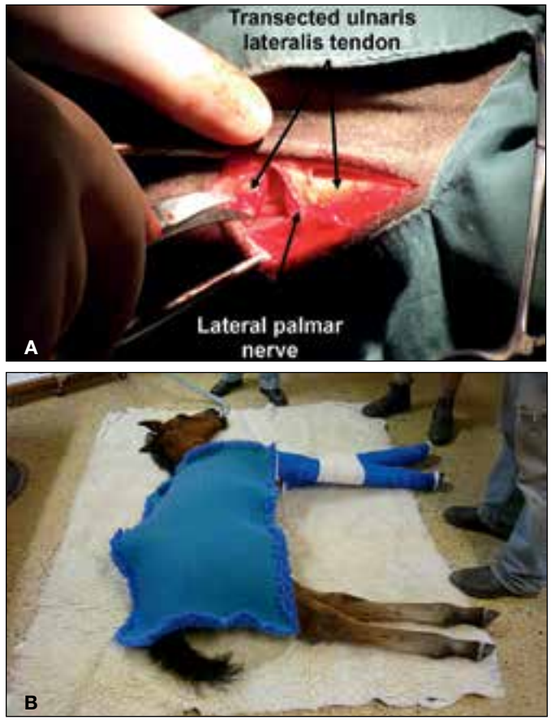

Figure 3. (A) Showing ulnaris lateralis tenotomy. Care must be taken to avoid the lateral palmar nerve when blunt dissecting to expose the tendon before transection. (B) Showing recovery of the foal on oxygen with splints applied to both forelimbs. Pictures taken on the 2nd December 2011.

The splints were reapplied (Figure 3B) to support the limbs while healing took place and provide additional extension of the remaining intact tendons. It was recommended that the splints remain in position for 5 days post surgery and to confine the mare and foal in a small yard or box for 2 weeks until suture removal[7].

Outcome

Overall, the surgery proved to be successful. The angle of the limbs had greatly improved (Figure 4A). The splints were removed and it was evident that they had caused excoriation of the dorsal surface of the pasterns from the pressure of the bandage and/or splints on the skin (Figure 4B). These lesions healed without complication when treated as an open wound.

The foal spent the majority of the time recumbent after surgery, which resulted in a number of pressure sores. These rapidly resolved without treatment once he regained mobility. Upon standing without the splints, the carpus appeared to buckle slightly probably due to a lack of stability from removal of sections of the stay apparatus. With muscle development in the other structures of the limbs, this should not be an ongoing problem.

Once the foal was walking again, it was evident that the digits had become hyperextended (Figure 4B). This may be due to increased strain on the superficial and deep digital flexor tendons as a consequence of the tenotomy leading them to stretch and become 'dropped at the fetlocks'[7]. Consultation with a farrier was sought to modify the angle of the toe over several trimmings to bring the sole into contact with the ground and reduce the strain on the flexor tendons.

Figure 4. (A) Showing greatly improved angles of both forelimbs in the foal 12 days post-surgery. (B) Shows post surgical complications involving extension of the toe and excoriation of the dorsal aspect of the pastern due to pressure from the splints. Pictures taken on the 14th December 2011.

The sutures were removed at 12 days post surgery and the mare and foal moved into a larger yard to promote use of the forelimbs in hope that this would cause adequate muscle gain to stabilise the carpus. Approximately 1 month post-surgery all of the pressure sores and excoriation wounds had healed completely. The foal's mobility had increased, the limbs remained straight, but buckling of the carpus was still present.

Final Follow-up

Over time, from continued use, it is expected that shoulder muscle development will occur that will result in strengthening of the affected joints. A large increase in muscle development was observed at 6-months-of-age (Figure 5). The foal's coordination during locomotion had improved at this time and continues to become stronger as time passes with less flicking of the toes. While the prognosis for the foal does not include a racing career, it is expected that he will develop sufficient function in the forelimbs to be used as a pleasure/trail riding horse.

Figure 5. Spider at 6-months-of-age. Marked improvement in the flexure of the forelimbs can be seen when compared to Figure 1. Digital hyperextension had decreased from Figure 5, suggesting that corrective farriery had been beneficial. Muscle development of the shoulder had also improved. Picture taken on the 11th April 2012.

Discussion

It was clear that the combined therapy of oxytetracycline and splints improved the foal's flexural deformity enough to allow him to stand and suckle on his own and this will often be enough to fully resolve mild cases. However, surgical intervention was required in this case to correct the deformity enough to improve his quality of life in the long term. Initial opinion was to perform a desmotomy of the superior check ligament to release the digital flexor tendons and allow extension of the limbs. However research suggested that this technique is best suited to releasing flexion of the metacarpophalangeal joint[1,12], as opposed to the carpal joint. Tenotomy of the deep digital flexor tendon directly would also only release flexion mainly of the distal limb[1]. Further research revealed that tenotomy of the flexor carpi ulnaris and ulnaris lateralis tendons has been used for the surgical ▶

management of carpal flexural deformities[1, 6, 10]. The difference in regional joint extensions between the 2 surgical procedures relates to the anatomical origins and insertions of the relevant tendons involved. The flexor muscles of the foreleg are similar in many respects, such as their origin from the caudomedial aspect of the humerus, except for 1 difference; their insertions. The flexor carpi muscles, radialis and ulnaris, attach near the carpus to the medial splint bone and accessory carpal bone respectively while the digital flexor tendons, deep and superficial, attach to the phalanges[13] (Figure 6).

While the digital flexors have action on the carpus through pressures placed upon the check ligaments during contraction, the main effects of transection of these muscles or the check ligaments is to release contractual force upon the distal limb where they insert. This is therefore the indication for tenotomy of the flexor carpi ulnaris for release of the carpal joint from flexural deformity. The ulnaris lateralis muscle has 2 branches of insertion, 1 to the accessory carpal bone and another to the lateral splint bone, and is also involved in direct flexion of the carpus[13].

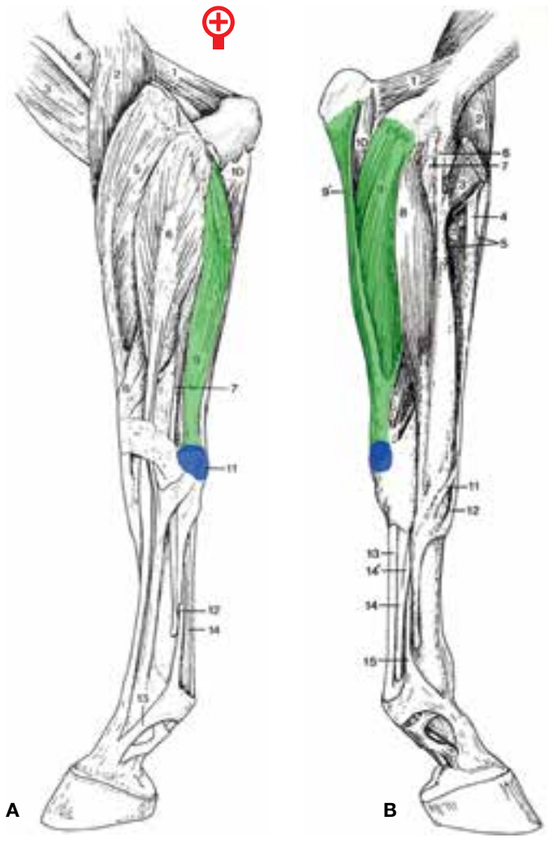

Figure 6. (A) Distal muscles of the left forelimb; lateral view showing ulnaris lateralis muscle (green) insertion onto the accessory carpal bone (blue) and the deep digital flexor tendons (14) inserting on the phalanges. (B) Distal muscles of the left forelimb; medial view showing flexor carpi ulnaris muscle (green) insertion onto the accessory carpal bone (blue) compared to the superficial (13) and deep (14) digital flexor tendons. Adapted from Dyce, KM, Sack WO, Wensing CJG 2002, Textbook of Veterinary Anatomy, third edition, Elsevier, USA.

Hyperextension of the digits in the forelimb was an unexpected post-surgical complication that required ongoing correction. While modification of the angle of the toe resulted in the desired effect, another method has been proposed that may have been a viable alternative. Application of toe extensions, or similar devices, helps to maintain contact between the sole and the ground[6]. By maintaining contact with the sole, and in particular the toe of the hoof on the ground, pressure is removed from the digital flexor tendons and spread over the entire hoof[6]. Therefore, these could have been used instead of, or in conjunction with, correctional trimming. Excessive exercise was avoided during treatment as this can also contribute to fatigue of the flexor tendons and potentially aggravate the problem[6].

Conclusion

Surgical correction in an 8-week-old foal, following non-invasive splint and oxytetracycline therapy, was successful in correcting bilateral flexural deformity of the carpus. Transection of the flexor carpi ulnaris and ulnaris lateralis muscles allowed adequate extension of the joints to facilitate straightening of the forelimbs. Minor post-surgical complications were successfully resolved and the foal is expected to develop sufficient function to perform as a pleasure riding horse.

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Call for more Large Animal C&Ts

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WINNER

Iodine Goats

C&T No. 5325

Harry Corbett
330 Canterbury road
Bayswater NSW 3153
T. (03) 9720 6800 F. (03) 9738 1064
E. harryc@myaccess.com.au

In the late 1970s I was given a challenge: Would I be interested in setting up an embryo transfer unit for goats? At that time there was big interest in angoras and certain of the exotic sheep types such as fat tails. The offer was made by my ex-brother in law Dr Bertram Wainer.

Although seasonal, this work occupied me for the best part of 8 to 10 years but eventually it succumbed to the inevitable fate that awaits agricultural 'schemes'. Along the way I met a succession of incredible characters, too numerous to mention here.

Memories flooded back the other day when Richard Malik happened to send me an image of his farm (see Figure 1). Clearly a lovely little spot, the undulating nature of the land reminded me of the farm where we established the unit at Cockatoo near the Dandenongs in Victoria. Our first year, spent largely establishing protocols, was ruined by a spate of late term abortions, still births and failure to thrive: lovely countryside but iodine deficient. Fortunately once diagnosed the condition is easily prevented with injections but the condition brought more grief several years later when I did some work in New Zealand.

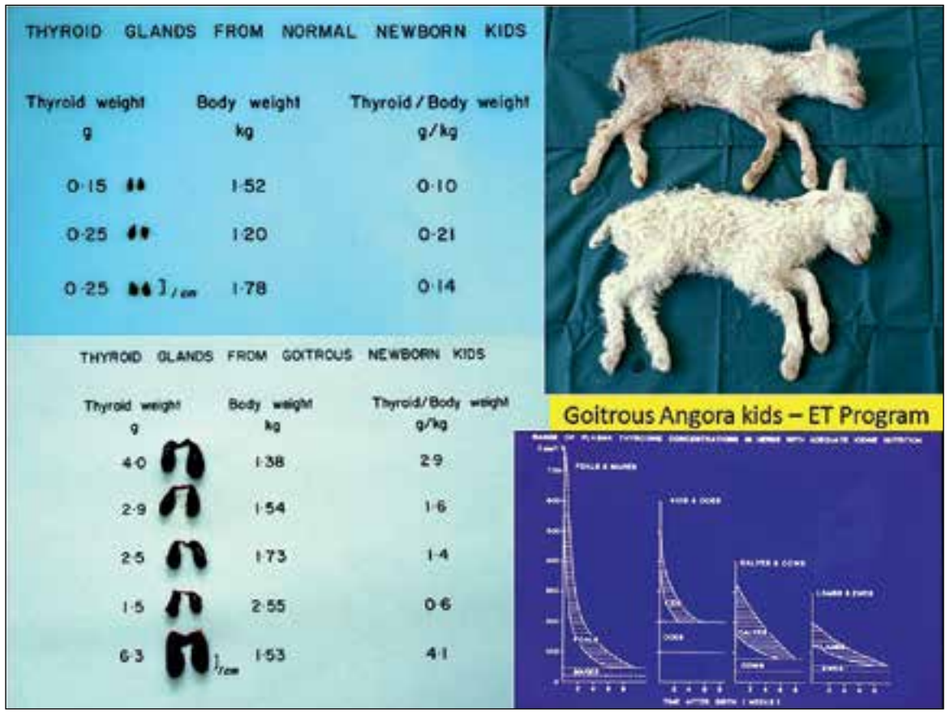
I arrived late in the day and by the time we got to the farm it was quite dark. On the way the clients explained that other farms in the area that they had just moved to had experienced late term abortions etc. I asked about iodine deficiency but they told me the tests that had been done did not indicate it as the cause. When I awoke in the morning and before breakfast I walked the farm: lovely rolling country. I contacted my 'go to' man in Victoria, Dr (now Professor) Ivan Caple and he suggested more definitive testing be done, testing that indicated an iodine deficiency (Figure 2).

As a veterinarian with too many years at the coalface I cannot stress enough the importance of observation, gut feeling and questioning. Edward Hargreaves was struck by the similarity of the country around Bathurst to that he had seen in the goldfields of California and we all know what that led to.*

*(***Editor's Note:** For non-Australians, Hargreaves discovered gold at Bathurst which led to the Gold Rush…)*

Figure 1. Richard's farm 3km from Wonbeyan Caves.

Figure 2. (below) Comparison of thyroid gland mass from normal and goitrous newborn kids.



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Follow on from C&T No. 4636: Curettage and diathermy of feline nasal squamous cell carcinoma

C&T No. 5326

Robyn Jarrett BSc BVSc MVM MANZCVS
Veterinarian
Pukekohe Veterinary Centre
11 Edinburgh St, Pukekohe 2120, NZ
T. (09) 238 6897 F. (09) 238 9905
puke@vetcentre.co.nz

Paul Jarrett FRACP FRCP
Dermatologist
Department of Dermatology, Middlemore Hospital, Private Bag 93311, Otahuhu, Auckland 1640, NZ
Honorary Senior Lecturer, Department of Medicine, Auckland University

In October 2005 the CVE (then the PGF) published a prize winning C&T article by CVE member Robyn Jarrett and husband Paul Jarrett, a Dermatologist – re-published overleaf – about a novel technique to treat nasal planum SCCs in cats. It generated some controversy at the time. It is very pleasing to us at the CVE that the work was extended, subjected to peer review and then satisfied the qualities of evidence-based medicine. With colleagues Norman & Gibson, they recently had an article on this topic published in the *Journal of Small Animal Practice* (2013) 54, 92–98 – see a brief description below.

Curettage and diathermy: a treatment for feline nasal planum actinic dysplasia and superficial squamous cell carcinoma

Jarrett RH, Norman EJ, Gibson IR, Jarrett P.

Source: Pukekohe Veterinary Centre, 11 Edinburgh St, Pukekohe, 2120, New Zealand.

Abstract

Aim: To evaluate curettage and diathermy as therapy for actinic dysplasia and superficial squamous cell carcinoma of the nasal planum of cats.

Methods: 34 cats assessed to have actinic dysplasia and superficial squamous cell carcinoma involving less than half of the nasal planum were treated with 3-cycles of curettage and diathermy. Response to treatment, adverse effects, owner impressions, time to recurrence and proportion disease free at 1 year were evaluated.

Results: Lesions ranged from actinic keratoses to invasive squamous cell carcinoma. Complete response to therapy was obtained in all cats. The median follow-up time was 18.2 months. Two cats had recurrence of lesions at 161 and 192 days after treatment. The probability of remaining disease-free after 12 months was 0.94. Median time to recurrence was not reached. The procedure was well tolerated by the cats with a good cosmetic outcome and no substantial post-operative complications.

Significance: This study shows that curettage and diathermy is an effective treatment for feline actinic dysplasia and for superficial squamous cell carcinoma involving less than 50% of the nasal planum. Curettage and diathermy is easily mastered and requires minimal equipment.

Note: The unit in the article is a Geiger TCU unit which currently costs US$625 – see the Delasco website for further information.

The step-by-step procedure as presented at College Science Week in 2011

Figure 1. Pre-operative

Figure 2. Curettage

Figure 3. Post-curettage

Figure 4. Diathermy

The authors wish to acknowledge Richard Malik's enthusiasm and encouragement for this project – Thank you Richard.

Republishing our Winner C&T No. 4636, Issue 240 from our October 2005 mailing

WINNER

Curettage and diathermy of feline nasal squamous cell carcinoma

Robyn Jarrett BVSc Veterinarian
Cameron Rd Veterinary Clinic,
1071 Cameron Road, Gate Pa, Tauranga,
New Zealand
T. (07) 5787858 F (07) 5787850

Paul Jarrett FRACP, FRCP Dermatologist
The Skin Centre,
171 Cameron Road, Tauranga New Zealand
E. pjarrett@xtra.co.nz

A routine treatment for feline squamous cell carcinoma of the nasal planum is cryotherapy. A more aggressive procedure is nosectomy permitting wide margins[1]. Curettage and diathermy is proposed as an alternative treatment modality. This technique can be a useful treatment for some human skin cancers.

Cryotherapy is a non selective destructive procedure. Achieving a temperature of -50 to -60°C in all regions of the tumour, including the lateral and deep margins, is considered adequate to completely devitalise neoplastic tissue[2]. The outer edge of the iceball at 0°C is inadequate for tissue destruction[3] and thermocouple control is not routine practice in veterinary or human medicine. This degree of freezing may not be achieved in large tumours and therefore small and low risk tumours only should be selected for this modality. The extent of tumour on the nose of a cat can be difficult to assess clinically. Although a visible iceball is seen, the degree of freezing may be inadequate in the deeper and lateral tissue. Healing is prolonged and recurrence can occur.[4] A 30 second double freeze thaw cycle of cryotherapy with a 4mm margin for superficial basal cell carcinoma in humans can take up to 6 weeks to fully heal.

Cell adhesion of neoplastic tissue is diminished, allowing it to slough easily. Curettage uses this feature to delineate the neoplastic from the normal skin. With practice, this tissue plane can be easily discerned during the procedure. Initially, the neoplastic tissue is easily removed and then the curette 'grates' against healthy dermis. (Figure 2) The curetted bed is often larger than would have been expected clinically. Normal appearing tissue can slough easily indicating subclinical tumour extension. Vigorous diathermy of the entire curetted bed aims to destroy any remaining neoplastic tissue and also permits haemostasis.(Figure 3) The curetted fragments can be sent for histology. (Figure 5)

The procedure is repeated. The diathermied base is curetted and again diathermied. If necessary a third cycle is repeated. Diathermy destroys approximately a 1mm rim of tissue. The area is allowed to heal by secondary intention. Petroleum jelly (Vaseline®) is applied daily until the area has healed. The healing is expected to be quicker than adequate cryotherapy as no devitalised tissue remains to be sloughed.

This easily mastered technique gives confidence that all tumour margins have been destroyed. The equipment is inexpensive to purchase—a small curette, and a diathermy unit. (Figures 6,7,8,9) Any diathermy source (thermocautery or electrocautery) is suitable provided adequate charring of tissue is achieved. The unit pictured can be purchased with a variety of tips (loop and ball) from Delasco www.delasco.com for $US495. A setting of between 4-6 is suitable for this purpose.

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Figure 1 Squamous cell carcinoma

Figure 2 Curettage in action

Figure 3 Diathermy

Figure 4 Post Diathermy

Figure 5 Curetted samples in formalin

Figure 6 Curette

Figure 7 3mm diameter curette head

Figure 8 Thermocautery unit (Geiger TCU)

Figure 9 Thermocautery tip (Loop)

Laparoscopes – what is old is new again

C&T No. 5327

Peter Howe
Leslie Street Veterinary Clinic
Leslie Street, Umina NSW 2257
T. (02) 4342 0500
E. pandh@tac.com.au

I used to pick up the laparoscope as a matter of routine. I used it for laparoscope assisted ovariohysterectomies, ovariectomies, liver biopsies and in-lieu of exploratory laparotomies. I have also used it for vasectomies in cats (for teaser toms in cat breeding establishments), for infertility exams where uterine pathology is suspected, cystotomies for tumour biopsy or urolith removal etc. Its use is only limited by one's imagination.

These days I use the laparoscope much less frequently, although I recently started to overhaul my equipment with some renewed enthusiasm. The younger vets do not seem overly enthusiastic and older colleagues do not like change. I believe that laparoscopy gives a greater chance of a definitive diagnosis at times compared to other modalities because actually seeing the lesion/pathology in-situ and in colour and in relation to all of the abdominal organs allows for an accurate diagnosis, and more importantly prognosis, for much less money. In humans, for things like endometriosis, laparoscopy is still the gold standard test.

Thoracoscopy also has a great deal to offer.

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Eye watering tips for suturing techniques in Tom's Paddock*

C&T No. 5328

Aine Seavers
Oak Flats Vet Clinic
58A Central Avenue
Oak Flats NSW 2529
E. reception@oakflatsvet.com.au

Sometimes you just don't get to have help/time or be in a place where it is possible/safe to have a sedated animal sedated/anaesthetised, or the loss of blood is so great you need to stop it now – not in 10mins and a staple suture gun will just not do the trick. What to do?

I raised this question with some overseas colleagues to find out how I could improve on my – 'only allowed place one suture scenario' – and here are some tips.

First, if haemorrhage is not an issue, say a mad Staffie that you have to reduce and replace its extruded erect penis:-

- Normally I reduce first by either applying chilled KY Jelly® to the penis – (always keep some in the ice box – great for reducing eye lid chemosis) or chilled sugar solution and/or obstetrical lubricant. Some vets swear by just applying the lubricant – loosening the hairs trapped prepuce and then have the dog walk backwards. Do whatever works.
- Once reduced; I apply xylocaine numbing gel or EMLA cream to the prepuce shaft, pinch skin and place one single suture anterior to pinch – FAST!
- A work around for that is; you can actually thread the suture through the eye of a 22G injection needle first before passing it through the skin. That way you don't need to have very sharp eyes and a steady hand. Just leave a loop out at the end and pull the needle back leaving the suture pre-placed and ready to tie (Raymond de Villa).
- Then fast the dog for desexing the next day – You can use Tardak anti-hormone injections to cover the 6 weeks until castration is chemically effective.

For other sites – a single cut on a dog on a call-out – a large injection needle pre-threaded or not, as you see fit, gives you a wound repair in seconds.

I don't normally catheterise prolapsed utero-vaginal masses but I get the odd one that has been kinked for so long one wonders about the urethra.

Or that the penis has been enlarged for so long that the prepuce has stretched to the point that the penis is reduced back into the sheath but there is a pocket of prepuce between the penis and outside – a pocket into which the dog urinates and scalds itself.

So another tip I learnt was suturing the whole lot in – catheter and all – with the pre-threaded injection needle. Karen Thomas who told me about the technique first learnt it by watching a large animal vet anchor a jugular catheter in a horse. Richard Glassberg catheterises the dog then puts the needle through one side of prepucal skin, the catheter and then prepucal skin the other side. He then puts the suture through the needle and withdraws the needle so he can tie the suture. Obviously leaving catheters in carries its own issues and one might not do this in a non-hospitalised animal but it does ensure urine flow if there is concern that there has been impediment to outflow for a dangerously long time.

*This is a reference to Tom Hungerford's quote on our 'Thank you to contributors' page 6 which sums up the philosophy of the *C&T Series*.

Economic validation for stocking uncommonly used antidotes and antiemetics on the drug shelf

C&T No. 5329

Aine Seavers
Oak Flats Vet Clinic
58A Central Avenue
Oak Flats NSW 2529
E. reception@oakflatsvet.com.au

Cerenia® is a wonderful antiemetic drug and is less expensive than it was, but it's still expensive. Vomiting dogs are not overly common on a daily/weekly basis so it goes out of date quickly which means less regular demand and price stays too high. A bottle had a 28 day expiry here; overseas it's been used for 3mths but I notice the new post-cat registration claim that **the vial now says 2013 discard after 90 days so great news there.**

For many of us, the sole use nowadays for Rompun® (Xylazine 20mg/mL) would be for some early presentation Baysol® poisonings. But with even those poisons becoming a rarity in certain regions, the drug often goes out of date without a single drop being used.

Therefore we need to look at legitimate other ways to validate having these valuable antidotes and antiemetics sitting on our shelves.

Cerenia®

Caveat – some of this cannot be endorsed by Zoetis Inc – the following is purely 'early days' anecdotal feedback from discussions amongst 1,000 vets in clinical practice around the world on list serves in which I participate.

- Some published work[1] suggests that given IV it is as effective a visceral gut analgesic in dogs as morphine! Other studies have demonstrated reduced sevoflurane MAC in dogs and cats subjected to painful stimuli.[2,3] ***Subcutaneous injection is the ONLY registered route here but I wondered about*** it post exploratory laparotomy/abdominal surgery as a post op analgesia. I am finding the product superb in patients with pancreatitis to control vomiting and visceral intra-abdominal pain. Colleagues have used it for abdominal cancer patients as a great palliative care option for months despite the 5 day maximum rule.

I have been using it in pyometra – we so often fail to address the suspected pain, nausea and reflux this group of patients must have, given a large compressive intra-abdominal mass from a reproductive tract that is in itself very effective at producing high levels of pain chemicals as well as compressive and distension distress. I have no concerns using it, especially if pyometra is open and discharging, but probably should be a bit more cautious of closed pyometra until we know more about the level of contractions it could induce in this tract.*

- 'Early days' feedback from colleagues who asked me about megaoesphagus cases suggests it has efficacy in controlling the regurgitation symptoms.
- It is widely given before oral I^{131} to prevent vomiting of the radio-iodine capsule.
- It can reduce nausea and vomiting associated with administration of chemotherapy drugs to cancer patients. Prevention of vomiting/nausea (1mg/kg at least 1 hour in advance – lasts for 24 hours same as dogs so if giving for example for chemo then give it the night before).
- Cerenia® may be useful in paraneoplastic pruritus with a small number of individuals with T cell lymphosarcoma gaining relief from pruritus.
- Some vets have used Cerenia® to reduce itch in atopic patients. And/Or as an adjunct therapy to other drugs such as cyclosporine in humans[4-6] but success in dogs has been variable[4-6]. There has been some reported anecdotal success in cats with hypersensitivity disorder but early days yet.
- Use in Antifreeze poisoning cases; Where clients' finances don't allow 4MP I/V ethanol treatment regimes[7]. Ariane Goerlich DVM uses Cerenia® S/C at 1mg/kg then 2mg/kg PO after 24hrs start with oral ethanol/vodka (former at 20% ethanol) 5mg/kg/PO q 6hrs x 5 doses then q 8hrs x 4 doses. Oral fluids at 1-2 x maintenance divided every 2-3hrs PO[7]. *For more on the use of Cerenia® in anti-freeze presentations – go to the CVe-library to read the discussion between an emergency specialist and a toxicology specialist following my query to them.*
- Used for post op ileus and inappetence – will cause peristalsis so care in young or case presentations at risk of intussusception*.
- Used in refractory coughing, again because substance P is involved in many types of inflammation.
- Feedback from the troops in the field was that when Cerenia® is used in ataxic vestibular disease syndromes to palliate a concern over co-existing nausea whilst tincture of time does the healing then the symptoms of ataxia resolve much faster than normal. I am certainly finding it very helpful in severe cerebrovascular accidents or idiopathic geriatric vestibular disease where the dogs are so nauseous and inappetent from the occular strobing and nystagmus that either they can't perceive how to co-ordinate prehension of food or are too motion-sick to consider eating. Within an hour there is remarkable improvement (not resolution) of some of the severe ataxia/head tilt/torticollis.
- I worry about its QRS prolongation so am not confident about using it in tick cases until someone starts publishing lots of ECG-tracked cases for 1-5 days so we can have data on that possibility. If it's safe, it might be good for tick paralysis cases.

Questions

<u>Drug Instructions: Do not use beyond 5 days.</u>

1. If that 5 day rule comes from human data – how do our pets and their dopamine receptor levels compare to the levels in humans?

   Vets are using it for much longer than 5 days and not seeing these issues so we need some extra information from Pfizer on that aspect.

2. Cerenia® isn't supposed to be used in puppies under 16 weeks due to risk of medullary hypoplasia, particularly up to week 11. Has anyone had experience of such an effect?

Explanations and answers given to overseas vets by their Pfizer contacts to the question: Why not administer Cerenia® beyond > 5 days?

A. *'As for the 5 day indication, there is some bioaccumulation of maropitant when dosed beyond 5 days. Further, in the initial safety and toxicity studies high doses (10 x registered dose rate) resulted in inappetence so there is the risk that continued dosing at the registered dose rate would result in accumulation to such a degree that it suppresses appetite.*

B. *Substance P is an important neurotransmitter in the CNS. In the dopaminergic system, substance P is primary neurotransmitter. So, if we entirely deplete substance P via blockade with maropitant Cerenia®) from the CNS, the dopaminergic system does not function properly. Depletion of substance P will lead to tremors and signs typically seen with dopamine depletion like Parkinson's disease and especially Huntington's disease. So, it is best given 5 days or 2 days off, or every other day etc, but not continually.*

C. *(1) The label is limited to 5 days for acute vomiting because that was the length of time that our clinical trial used. The 5 days was a result of our surveying a group of GI specialists that recommended that the longest period of time an antiemetic should be used without knowing the underlying cause is 5 days.*

   (2) The pharmacokinetics of the drug are non-linear and after 5 days in our initial PK studies, the drug stayed in the therapeutic range for another 2 days. We have done more PK studies to date – see ACVIM Forum abstracts 2011 available on VIN or in ACVIM Journal – that have shown that after about 14 days the drug levels off to a steady state. This is not on-label nor is the drug approved for use in this chronic manner.

   (3) You also asked me to comment on the substance P concern. I contacted our clinical pharmacologist and we have no evidence of any tremors with our labeled dosing. I do not believe that it is possible to block all substance P in the brain with Cerenia®. NK1 receptors are in high numbers in the vomiting center, so Cerenia primarily works there.

Since going to press, additional information has come to hand. Therefore Aine's ebook article will be published in December 2013 Issue 273 instead. We apologise for any inconvenience.

Invited Comment courtesy of:

Stephen Page BSc(Vet)(Hons), BVSc(Hons), DipVetClinStud, MVetClinStud, MAppSc(EnvTox), MANZCVSc

A target animal safety study in dogs summarised in the US CVM freedom of information summary led to the conclusion that 'maropitant injectable solution (10 mg/mL) was well tolerated when administered subcutaneously to healthy 16-week-old dogs for 15 days at up to 5 mg/kg'.

Re substance P (a neuropeptide member of the tachykinin family that acts on neurokinin 1 and 3 receptors) – while dopamine is the principle neurotransmitter within the various dopaminergic projections within the CNS, there are a large number of other neurotransmitters that serve to modulate dopamine neurotransmission, acting via muscarinic, cannabinoid, NMDA and other receptors – including NK1 and NK3 receptors.

Use in Cats:

<u>Same dose as dogs (1mg/kg)</u>. Younger cats seem to eliminate it quicker so you might notice it wearing off sooner. Most common ▶

Authors' views are not necessarily those of the CVE
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clinical abnormality is up to 10mm swelling at injection site, lasting up to a day for isolated injections. At higher doses/multiple injections at same site – the lesions were bigger, lasted longer, and presented more consistently.

- Safety of Cerenia® in cats when used concurrently with other centrally acting agents (e.g. sedatives and anaesthetics) has not been studied
- Safety has not been established <16wks or during pregnancy/lactation
- Should not be used concurrently with Ca channel antagonists (heart meds = Verapamil and Diltiazem)
- Used with caution in animals with liver disease

A Fellow of the American Academy of Veterinary Pharmacology and Therapeutics (Pharmacology/Toxicology) advises:-

> *As 'the main neurotransmitter involved with mast cells is substance P, Cerenia® could be considered in CNS inflammation, CNS vomiting center, gastric and intestinal pain, allergic disease (mast cell degranulation), tissue oedema, local tissue injury, burns, nerve pain (damaged nerves – neuropathic pain), muscular inflammatory pain, post-surgical pain, joint pain, bladder wall pain, sinusitis, lung and bronchial inflammation, pancreatitis, etc.'*

Therefore, could we one day be using Cerenia® for bladder pain in FIC cats?-cf When the bladder is the victim not the culprit article (see Perspective 90 Small Animal Behaviour, June 2012 Issue 267, pg 29 in the CVe-library).

ebook readers – Rollover or Download Perspective 90 here.

1 2 3 4 5 6 7 8

So maybe Cerenia® will become cheaper again and less likely to go out of date!!?

ROMPUN

Rompun Cocktails

For many of us, the sole use nowadays for Rompun® (Xylazine 20mg/mL) would be for some early presentation Baysol® poisonings. But with even those poisons becoming a rarity in certain regions, the drug often goes out of date without a single drop being used.

Where we worked in the UK, the area was over-represented with aggressive Rotties and psychotic male box-headed Golden Retrievers (who would try to take your face off without a hint of a growl or warning – most muzzled in the car before coming in the clinic!). As many of those dogs were presented by owners who couldn't handle their own pet – it having bitten some if not all family members by the time it was presented, then handling such dogs normally tested the First Commandment of General Practice; 'Thou Shalt at all times strive to remain out of Hospital'.

- If you knew in advance when they were booked in or where the owner was totally useless at holding them, then the owner was given a mixed pill pot containing phenobarbitone 30 or 100mg tabs, as many as you think it needed then that dose tripled, plus 1mg/kg acepromazine tablets to premedicate the dog at home.
- When they turned up in the waiting room unannounced and the owner could restrain them for a short time, local practices used a 6mL intra-dog 'Cocktail A'-pre-euthanasia of 2mLs of acepromazine (2 mg/mL) drawn up then needle change, 2mLs of Xylazine 20mg/mL (there is a 100mg/mL horse version one could use) then a vial of 2mLs of diazepam (5 mg/mL) – all in the one syringe intra dog.

Hugh Bain tells me that dogs will drink lethabarb in milk on house call-outs so that is useful.

Ketamine squirted into the mouth of a feral cat also works but you have to have it fairly confined in the first place to do that.

Immobolin® had long fallen out of favour and whilst Domitor® was and remains a very popular drug used overseas, it flattens veins thus increasing difficulty of venepuncture so not something you want pre-euthanasia. However, in more recent years an esteemed Israeli colleague Dr Michael Bernstein uses a Domitor combination and as he has never been wrong in almost 2 decades of advice, then his recipe below is also of use.

- *'Cocktail B'; A dry bottle of Zoletil (teletamine & zolazapam) diluted with 2.5 mL Domitor and 2.5 mL Torbugesic (10 mg/mL).0.1 mL IM per cat or 5 kg dog, 0.2 mL IM for a Cocker and 0.3 for a 30 kg dog. 0.4 of this stuff in the rump will flatten a 40 kg Rottie in 5 minutes. This flattens them out for 30-45 minutes and is reversible – unlike my concoction above. Note that in many countries once you add 2 drugs to each other (even drugs into a fluid bag) you now have an unlicensed product so be careful how and when you use such combinations.*

Note from Dr Bernstein: The cocktail was recommended to me by the Virbac distributor in Israel under the name of ZDT (Zoltil Domitor Torbgesic) aka TTD or TTdex (when using dexdomitor instead of domitor –careful - ½ the concentration). An internet search shows that the combo was published by Dr Jeff Ko. I certainly can vouch for efficacy, not only for pre-euthanasia, but also as a premed when you are on your own with a fractious hyped-up animal. The article states that a higher dosage can be used for anaesthesia although I have never used it without Isofluorane. See also Dr Ko's handy dosage chart for TTdex.

Injectable Anaesthesia Update by Dr Jeff Ko 1 2 3 4

Jeff Ko's Dosage Chart for TTDex 1 2

I still use euthanasia 'Cocktail A' where the owner wants to drop the dog off and leave before the euthanasia takes place. For OHS reasons we do not allow the owner to leave until the dog is first given the cocktail and it is allowed 10-20 mins to take effect. Then the sleeping dog is muzzled (Rompun/Domitor can allow a sedated animal to suddenly sit up, bite and then fall back sedated so we use a muzzle always), the owner leaves and we immediately perform the euthanasia (so no risk of animal vomiting unattended). The safety of my staff and the giving to an animal of as gentle and as stress-less an euthanasia as possible are my priorities.

I also administer 'Cocktail A' to any dog where I am not confident the event will go smoothly i.e. either the dog or the owner or both are so palpably stressed that you as a vet can't concentrate properly given the fear/hysteria/grief/distress pheromones bouncing around the room. This is especially useful if the dog is blind – those pets far more likely to panic and start shrieking the place down than a deaf animal or one in pain. That happened to me in my first years out of Uni and I have never recovered from the trauma of that particular episode.

If there is any distress shown by the dog I stop and advise the owner that I refuse to fight with any animal at euthanasia. We will wait 15mins for the injections to kick in and then we will give the I/V. I often tell this to a nervous owner in advance and you can see them physically relax. Clinic room lights are turned off, the door closed and the dog and owner left alone for a short time. When you return – everyone is much calmer.

I give a smaller version of 'Cocktail A' or else straight Zoletil S/C I/M to cats in advance of a euthanasia that the owner wants to sit in on, or where the cat's history suggests manual restraint is not something the cat tolerates. (Use a new needle for the injection and the sting is massively reduced – the nurses holding the cats will love you for it!)

I charge out the additional cost of the injections on the euthanasia bill if we have had to call them into play; if not needed then we don't charge.

Some USA vets have commented on the use of Rompun pre-black bag where a round smooth non-corrosive foreign body (FB) cannot be afforded to be removed by normal means by the owner. In such cases Rompun in a dog positioned in a head down position is given in order for it to vomit back up the FB. I have not personally used this technique though I am aware of several vets who have done so successfully. I would have to be very sure of the FB type, sure that it is in the stomach and have no other options bar euthanasia to first try this.

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For more on the use of Cerenia® in anti-freeze presentations – go to the CVE website to read the discussion between an emergency specialist and a toxicology specialist following my query to them

Reading

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External markers of internal disease; the 'not-so-humble' nail clip - Part 2

C&T No. 5330

Aine Seavers
Oak Flats Veterinary Clinic
58A Central Avenue
Oak Flats NSW 2529
E. reception@oakflatsvet.com.au

Grateful thanks to my overseas colleagues who generously collaborated to produce this 'pictorial' to follow up C&T No. 5312. Pictures of both canine and feline nail disease presentations illustrate that cases presented as lameness/neuro are in fact primary dermatology/neoplastic/bad management conditions.

No. 1

Ana Rostaher DVM dipl ECVD
Small Animal Clinic - Department of Dermatology
Vetsuisse-Faculty - University Zurich
Winterthurerstr. 260, CH-8057 Zurich

Centre Hospitalier Vétérinaire Fregis
43, Avenue Aristide Briande
F- 94110 Arcueil
E. ana_rostaher@yahoo.com

Figure 1. Bucher Pitschi lip erosions

Figure 2. Bucher Pitschi ulcer mouth

Figure 3. Bucher Pitschi claw deformations

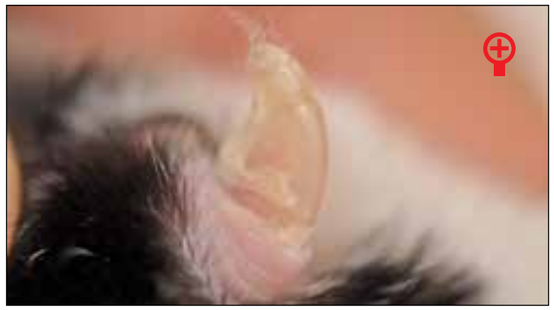

Figure 4. Bucher Pitschi claw deformations

Figure 5. Bucher Pitschi crusts erosions foot pads

Figure 6. Bucher Pitschi ear erosions

Figure 7.

Figure 8. Nail biopsy parcial Bucher Pitschi 2

No. 2

Kimberly Coyner DVM DACVD
Dermatology Clinic for Animals of Las Vegas
USA
www.dermvetvegas.com
E. dermlink@gmail.com

Sadly the only cat nail disease picture I have is of cutaneous lymphoma, but I hope its helpful. This cat was certainly painful and lame for the several months of antibiotics and Depo injections before it came to see me... the pictures are pre and post cleaning of the purulent exudate.

I have also included a bacterial paronychia in a dog secondary to atopy. Another thing that I would mention as commonly misdiagnosed as ortho or neuro disease is the 'corn' syndrome in Greyhounds.

Figure 1. Bact paronychia allergy

Figure 2. Bact paronychia allergy

Figure 3. Greyhound corn

Figure 4. Kharma lymphoma

Figure 5. Kharma lymphoma

No. 3

Neil MacRae Smith BVM&S BSc CertVD MRCVS
Haven Veterinary Practice
Clacton on Sea
United Kingdom
E. sprock2002@yahoo.co.uk

Figure 1.

Figure 2.

Figure 3.

Figure 4.

Figure 5.

Figure 6 & 7. taken post pred treatment.

No. 4

Rendina McFadden, DVM
Bass Lake Pet Hospital
New Hope, MN USA
E. kubyren@yahoo.com

Figures 1-2. ▶

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Figures 3-4.

The first case (Figures 1 to 4) was an 11-year-old male neutered DLH (domestic long hair cat). We were unable to biopsy due to financial constraints but the cat had numerous other lesions that were suggestive of neoplasia and the paronychia was suspected to be a paraneoplastic process.

Figures 5-6.

Figures 7-8.

The second case (Figures 5 to 8) was a 7-month-old spayed female DSH (domestic short hair cat). Histologic diagnosis: *'severe, ulcerative, eosinophilic and to a lesser extent granulomatous, lymphoplasmacytic, neutrophilic dermatitis.'* A hypersensitivity/allergic reaction and/or eosinophilic granuloma complex lesion was suspected.

No. 5

Dr Robert Hilton BVSc(Hons) MACVSc (Canine Medicine) CertVD MRCVS
PO Box 42 Yarrambat VIC 3091
E. ozskinvet@yahoo.com.au

Figures 1.

Figures 1 to 3. Pemphigus foliaceus.

What is YOUR Diagnosis?

C&T No. 5331

Email your answer to:
elisabeth.churchward@sydney.edu.au

Winner is entitled to a CVE proceedings of their choice.

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No. 6

Guillermina Manigot DMV UBA
Dermlink
Buenos Aires, Argentina
E. dermlink@gmail.com
www.dermlink.com.ar
www.teledermatologia.com.ar

Figure 1. Poodle toy, bad feet conformation, showing looks after grooming. See the flat feet, staining of toes (atopic patient).

Figure 2. Same kind of patient as in Figure 1, but owner didn't allow clipping.

Figure 3. ▶

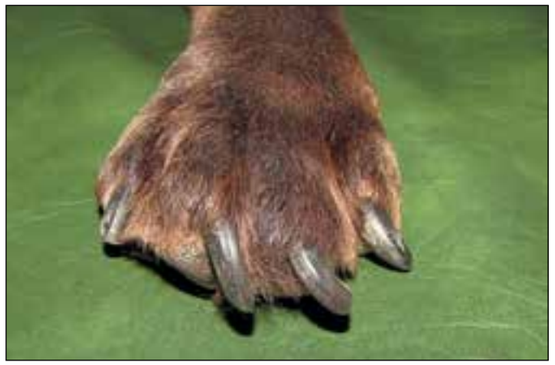

Figure 4.

Figure 5. Pyonychia in a cat. Not pemphigus.

Figure 6. Long nails unkempt in a young Basset Hound dog, causing lameness and paw-pad dermatitis (licking).

Figure 7.

Figure 8.

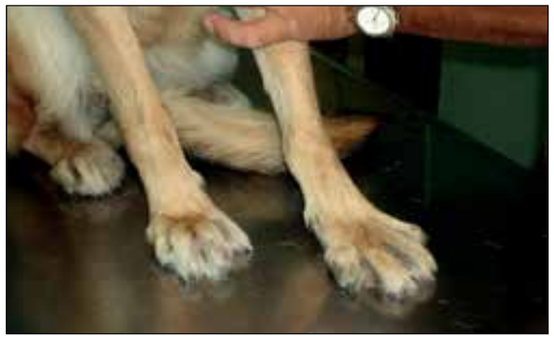

Figure 9.

Figure 10. Mixed breed dog with what I call 'Wheel Nails' or 'Dog-on-Wheels'.

Figure 11.. Traumatic exungulation caused by trapping the nail in a metal mesh.

Canine behaviour – have we got it right?

C&T No. 5332

David Bligh BVSc (Hons)
Rathmines Veterinary Hospital
5 Immarna Rd
Rathmines NSW 2283
T. (02) 4975 3366
E. rathminesveterinary@bigpond.com

Of all the specialties in Vet Science, canine behaviour is the only one that must compete with the outside world. For this reason there's been a need to define itself and have a point of difference. This, and the ugly sight of an angry human trying to 'correct' an out of control dog, has led justifiably to more non-confrontational techniques; hence the positive Reinforcement / Redirection / Distraction / Ignore unwanted Behaviour (RRDIB) techniques adopted by Canine Veterinary Behaviouralists (CVB). I'm on board with these methods and feel they are an invaluable tool when training and treating behavioural problems. If these methods where used solely and perfectly from puppyhood they alone would be sufficient to have well adjusted happy canines.

Unfortunately, we don't live in a perfect world and are faced daily with a myriad of unwanted canine behaviour.

One area that worries me greatly is the current dogma propagated by CVB that dogs do not need to know who's in charge and they are what they are. I checked my texts from the 1990s and this was not the case then. Why a total U-turn from 2000 years of human–canine interaction has been necessary, in such a short period, perplexes me. The thinking originated from CVB in the USA and seems to have been accepted without argument. Perhaps it's a knee jerk reaction to the dog whisperer juggernaut or maybe the debate has been hijacked by anxious non-assertives.

Observing a pack of dogs is used to support the above contention as once frameworks have been established the pack runs smoothly and cooperatively with not so much as a growl; dogs follow those dogs that know how to access food, water etc and learn from them. Harmony can exist for long periods. The other side of the coin which CVB have chosen to ignore is the challenge when new dogs enter the frame or younger dogs mature. Growling then becomes commonplace and maturing adolescents that push the boundaries are gripped round the back of the neck and forced to submit (fortunately we don't need to do this); confrontation and disharmony exist until a new authoritative framework is formed.

Building an initial authoritative framework allows RRDIB to work spectacularly well and behavioural issues are able to be completely resolved rather than minimised.

Of the dogs we see in practice, think of those that have authoritative frameworks. They tend to be calm tail-waggers who have an easy sociability and those that don't are anxious, agitated and prone to aggression. This, I know, is a bit of a generalisation, but the more you think about it the more it holds up.

So how can we build an authoritative framework that fits in with in Veterinary ethics and thinking?

With this current thinking the 'correction' has been completely banned without qualification.

I'd like to suggest a name change as 'correction' is associated with punishment or discipline which I'm not advocating. Let's call it the reminder.

For reminders to be used these 4 boxes must be ticked:-

1. A reminder is never painful, is never angry and is never frustrated. It is in fact gentle, the verbal component being the most important.
2. Before a reminder is given the sit/stay command needs to be perfected. Number (1) applies to this too. This is not hard to do and is easy to show owners. Simply squat next to the dog holding the collar with one hand and when the 'sit' command is given the animal's rump is pushed to the ground (again gently and not aggressively) with the other hand. This often needs to be repeated anywhere from 3 to 20 times. Once the dog is sitting the 'stay' command is given; if the dog rises the 'sit' command is repeated until the dog is sitting calmly next to the handler. This is then reinforced over time with patience and consistency. Success is reached when the dog automatically sits next to the handler without being told; this often takes days rather than weeks.
3. Reminders should never be given to fearful dogs. The one exception is when the dog has a trusting bond with the handler (i.e. not fearful of handler) and is acting aggressively due to its fear.
4. Reminders should be given on the periphery of the inciting cause, when the ears prick and visual contact is first made. If the cause of the dog's unwanted behaviour is right in its face retreat is the best course of action .

I understand that a small percentage of people are incapable of the above and these people should be moved straight to RRDIB.

Reminders merely help to establish an authoritative framework from which harmony grows – often for the life of the dog.

As an example, take the common problem of hypervigilant bossy dogs that bark and want to attack other dogs they see (or fear aggression see (3) above). With reminders, the dog over weeks and sometimes months can gradually get closer and closer to other dogs and, with patience and good timing and some other minor techniques, can relax around other dogs.Once they're sniffing each other the dog can be let off lead to play with his canine buddy – a truly liberating experience for the dog and a deeply rewarding experience for the dog lover. This is not possible using RRDIB alone.

Another case I saw was a Pug cross that had noise phobias that caused 'panic attacks'. I saw it when presented by the owner's father; it was on massive doses of Prozac® as the problem was so intractable and was exhibiting classic signs of serotonin toxicity. These symptoms stopped when Prozac® was stopped. The interesting part was that these panic attacks did not occur at the father's place. The worst noise for the dog was the whipper snipper and the father could whipper snipper around the dog while it lay there peacefully. The father was calm and assertive and experienced with dogs; the daughter was passive and anxious and inexperienced with dogs. A great indicator of the importance of authoritative frameworks.

I struggle to think of a canine behavioural problem that can be cured without this authoritative framework. RRDIB improves and minimises but does not cure. Complete resolution is feasible and surely should be aimed at, using all available techniques.

And this is the elephant in the room – success rates.

Currently I'm embarrassed to admit I would refer dogs with any aggressive disorder to a good dog trainer rather than a CVB. ▶

Invited Comment courtesy of:

Prof Paul McGreevy
Faculty of Veterinary Science
The University of Sydney
E. paul.mcgreevy@sydney.edu.au

In abandoning the concept of the hierarchy, are we in danger of throwing the social-order baby out with the heavily soiled hands-on-domination bathwater?

This a thorny issue, especially as we become more aware of the role of anxiety in canine behaviour anomalies. These days in dog behaviour circles, dominance is referred to as the D word. The notion of dominance brings with it some emotional baggage and conjures images of domination and dominators. And, as we have seen, this can unfortunately be misinterpreted as an endorsement of the use of force and the so-called alpha roll. Clearly, we don't want to oppress dogs but nor do we want dogs to displace us from resources. So, we need to have a relationship that avoids conflict and helps us to go unchallenged. To do this, we must acknowledge that there are howling gaps in our knowledge of social order in dogs. The harmony that groups of dogs achieve without violence offers an elegant model for our interactions with dogs but we have to accept that there are limitations to our ability to become honorary dogs. Nevertheless, we can craft our interactions with dogs to deploy the *other* D words – deference and displacement – to achieve implied ranking but, most importantly, rank without rancour.

With a team of passionate professional canine ethologists and veterinary behaviourists, I recently co-wrote a peer-reviewed article that explores this topic in detail (McGreevy et al., 2012).

I strongly encourage veterinarians who care about dogs to read it in full but, in the meantime, here is the abstract:-

This article reviews the literature on the complex and variable nature of the dog–human dyad and describes the influence of terms such as 'dominance' on attitudes that humans have toward dogs. It highlights a legacy of tension between ethology and psychology and notes that some practitioners have skills with dogs that elude the best learning theorists. Despite the widespread appeal of being able to communicate with dogs as dogs do with one another, attempting to apply the intraspecific dog ethogram to human–dog and dog–human interactions may have limited scope. The balance of learning theory and ethology on our interactions with dogs is sometimes elusive but should spur the scientific community to examine skills deployed by the most effective humane practitioners. This process will demystify the so-called whispering techniques and permit discourse on the reasons some training and handling techniques are more effective, relevant, and humane than others. This article explores the mismatch between the use of nonverbal communication of 2 species and offers a framework for future studies in this domain. Technologies emerging from equitation science may help to disclose confusing interventions through the collar and lead and thus define effective and humane use of negative reinforcement. The case for a validated intraspecific and interspecific canid ethogram is also made.

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Note: *Please see December's issue for another Comment courtesy of Kersti Seksel.*

WINNER

The hidden perils of paradise

C&T No. 5333

Associate Professor Deborah Marriott
Department of Microbiology and Clinical Infectious Diseases
St. Vincent's Hospital, Sydney NSW 2010
T. 61 2 8382 9196
F. 61 2 8382 2989
E. dmarriott@stvincents.com.au

Four very lucky rescue dogs spend their weekdays on the standard quarter acre block in Sydney but almost every weekend travel one hour north to 50 acres of rainforest and tall timbers on the Central Coast. They always enjoy bushwalking, swimming in the dams and chasing each other for possession of the best stick on the property. In general the main hazard of this canine paradise is paralysis ticks which of course are manageable with the appropriate oral and topical agents. At certain times of the year, particularly when the weather is wet and moist, leeches abound and after a walk it is necessary to remove up to 20 leeches from between the paws of the dogs. However they are not troubled by this, although on occasions they have been retrieved from more awkward sites such as the nose, the oral cavity or the perianal region.

Figure 1.

Recently a more significant hazard has emerged. Intermittently, the nests of a large aggressive black ant called the jack jumper ant (also known as the jumper ant, the hopper ant or the jumping jack) appear on the property. This species of bull ant, *Myrmecia pilosula*, is native to Australia, in particular the south east. (Figure 1).The ants have a very characteristic appearance with a black or red and black body and contrasting orange-yellow legs, antennae and mandible. (Figures 2 and 3) The nests consist of mounds of fine gravel from which the ants can be seen entering and exiting. They also have concealed nests under rocks where the only indication may be sentry ants, present at the entrance.

Figures 2 & 3.

To date the humans bushwalking on this property have avoided jumper ant bites, generally by wearing leather hiking boots. However the canine members of the family have been bitten on several occasions. The 13-year-old Corgi/Jack Russell, the 7-year-old Terrier cross and the aging Kelpie all experienced sudden severe pain at the site of the bite. Ants were seen in close proximity on each occasion. The animals sat down in obvious distress, holding the affected paw in the air and refusing to walk any further. The dog often licked at the paw in a frantic attempt to gain relief. The pain lasted approximately 30 minutes during which time the 'patient' was in obvious distress and reluctant to use the affected limb. Recently the 6-month-old Kelpie cross was bitten on the paw. He also developed severe pain at the site of the bite but within 20 minutes developed profound facial swelling with near closure of his eyes and marked perioral oedema. He was very subdued but did not lose consciousness and did not appear to have stridor or respiratory difficulties. The swelling resolved over the next 4 hours.

Human allergic reactions to jumper ants are very common with 2-3% of people in endemic areas having generalised allergic reactions and in about half of these the reactions can be life-threatening with several deaths recorded in recent years. In humans, approximately 70% of those with allergy will have another allergic reaction if they were stung again.

Research of the literature revealed that description of canine anaphylaxis is very uncommon in the peer reviewed literature*. However, given that recurrent anaphylaxis is well described it would be prudent to carry an epi-pen when undertaking bushwalks in the future.

Vets should consider asking owners about jack jumper ants when confronted with dogs with angioedema.

*There is material in texts, but little in the way of evidence based medicine.
** Images sourced from the internet.

OBITUARY – Dr John Holt

By Anne Fawcett

Image courtesy Mary Holt

PLEASE NOTE: a version of this article was published in *The Veterinarian Magazine*

The profession is mourning the loss of Dr John Holt, an Australian veterinarian credited by many as the man who put small animal practice on the map.

John graduated from Sydney University in 1954. After a brief stint as a cattle vet and a brief career in industry, John purchased St George Animal Hospital (SGAH) from Richard Boon in 1959 and developed it into a showpiece companion animal practice. He married Mary, a pharmacist, in 1960.

Colleague Graeme Allan said that John became a passionate advocate for small animal practice "at a time when you could go to the Australian Veterinary Association conference and the word dog or cat would not be mentioned."

Allan recalls a Sydney practitioner's branch meeting, attended mostly by meat inspectors and Government employees, when the conversation turned to treating squamous cell carcinoma in cats.

"This person [John] popped up and asked why weren't people using colchicine because it's an anti-mitotic agent," Allan said. "I'd never heard of it and neither had anyone else. We thought it was pretty sophisticated."

John's practice became known for setting the standard.

The business expanded, incorporating 6 practices in Sydney and employing 11 veterinarians and 56 para-veterinary staff. The practice produced 8 University professors.

According to former colleague Lindsay Hay, one of John's biggest drivers was the need for the profession to recognise small animal practice as "a legitimate endeavor".

"Whilst it is hard to imagine, in the 1970s when I graduated the predominant view was that real vets saw real animals – which didn't include dogs and cats," Hay said. "Our lecturers were very dismissive of anyone who didn't want to treat a horse or cow – the idea that you might set up a small animal practice in the suburbs was looked down on."

At its peak in the 1980s SGAH employed seven veterinarians, had its own fully equipped lab and radiology suite – including a fluoroscopy unit.

"There were times in the 1970s and 1980s when up to 80 animals a day were treated in hospital and 15 surgical procedures were performed a day," Hay said. "It was not unusual to do ten speys before lunch and three orthopedics after lunch."

Hay said that John could be impatient at times, because he was a real doer.

"He knew what he wanted and he would just get in and do it."

Hay said this made the practice a very exciting place to work.

"Surgeries were all performed with mask, gowns and gloves, and all animals were premedicated with an opioid and maintained on halothane anesthesia many years before such procedures were regarded as standard best practice. This was at a time when some vets were still doing speys on the back of toilet doors."

John was inspired by small animal practice overseas, and wanted to share its developments with Australian colleagues. He travelled to North America on multiple occasions, acting as a visiting guest lecturer at numerous institutions including Canada's Guelph University and Washington State Veterinary School. Friendships he struck up there led to numerous eminent veterinarians – including Steve Ettinger, Carl Osborne, Joe Bojrab and others – visiting Australia.

"John used his extensive contacts in North America to bring many speakers to Australia for conferences and workshops and made a huge contribution to veterinary education and practice standards by showing the profession what was possible at a time when small animal practice was much less sophisticated than now."

Together with like-minded practitioners, including Keith Baker, Noel Freeman and Neville Japp, Holt co-founded a group which later became the Australian Small Animal Veterinary Association.

John financed and edited the organisation's journal, the *Australian Veterinary Practitioner* (AVP), and provided administrative support for the organisation for many years. The first committee provided personal guarantees to support the Association's first office in Hurstville.

"It is difficult to see how the ASAVA would exist without John's vision and support," said Hay.

Friend Henry Hirschhorn feels that John's contribution was "greatly unappreciated for the work he did for the profession in general and for small animal practice in particular."

The AVP was established following a series of rejections of articles by the *Australian Veterinary Journal* (AVJ).

"We had submitted a set of articles to the AVJ which were rejected on the grounds that they added nothing new to veterinary knowledge," Hirschhorn said. "In fact, John's article on the diagnosis and surgical correction of misplaced ureters in the dog was to my knowledge the first recorded case of its kind diagnosed and successfully corrected."

It – together with an article by Hirschhorn on the correction of anterior cruciate ligament rupture by use of the anterior tibial tendon transfer – was published in the first edition of the AVP.

John was president of the World Small Animal Veterinary Association (WSAVA) from 1986 to 1988.

John had a rich life outside practice, travelling the world extensively (visiting at least 65 countries), collecting art and representing Australia in the shooting team at the 1960 Rome Olympics.

"John was passionate about small animal practice," Mary said. "He loved the animals and also really enjoyed working with young people – both nurses and veterinarians."

John received multiple awards throughout his career, including an award for meritorious service to the ASAVA, the Waltham Award for International Service to the Profession and a string of honorary memberships across a range of organisations including the American Animal Hospital Association.

According to Mary, John's care of animals continued until his death, as he donated to numerous animal welfare groups. In later years John was a very passionate supporter of the campaign against live animal export.

He also shared his life with 'Rosie', adopted from the Cat Protection at 7- years-old with chronic renal insufficiency.

"Only John would have adopted Rosie," Mary said. "The staff told us that we had to be very patient and it would take many weeks for her to settle in – they were correct. At one stage we thought she would never let us touch her but remarkably she is now the most cuddly cat you can imagine. This was due to John's patience and persistence."

WSAVA President Jolle Kirpensteijn said that he had always listened to John's "wise consults" and that John was "a pioneer who opened my heart to the global veterinary community."

John passed away on June 24, 2013 at the age of 82 years.

'Unlucky' bamboo for cats

C&T No. 5334

Camille Stephenson
Teneriffe Veterinary Surgery
29 Florence St
Teneriffe QLD 4005
T. (07) 3257 1099
E. camilles@iinet.net.au

Recently I faced a very unfortunate situation with one of my clients and one of his cats. This owner had 3 Burmese cats of varying ages and all indoor only. He brought them all in for vaccinations a few weeks ago. His middle cat 'Cuba' had lost a bit of weight and he reported that he was concerned about this. He said after I questioned him that he had noticed Cuba 'hanging over the water bowl' but not obviously drinking it – apart from that, nothing else to report.

I told him I was concerned about that since although the owner reported no obvious PD/PU with Cuba, I said that we should check his urine/kidneys. Cuba was 5-years-old. His urine had a very low USG with protein shedding. I followed up with blood tests and found he was in renal failure.

I asked his owner if he had any lilies inside his apartment and he reported – 'No'. He was aware, as I had previously told him (and my other clients, especially clients with indoor-only cats and I see a lot of them) that lilies are toxic to cats and cause renal damage and hence possible failure.

We admitted Cuba, placed him on IV fluids etc. His owner told me that he had recently bought 2 plants at the Teneriffe Festival from a stall. He said he thought it was 'Lucky Bamboo'. I told him I didn't think that plant was a problem but asked him to bring it in so I could identify it. It was, indeed, Lucky Bamboo. He also said that he had noticed Cuba eating it and then vomiting.

Anyway, to cut to the chase, I 'googled' Lucky Bamboo and unfortunately found out that this plant is actually a lily – not what I either knew or expected.

Unfortunately, Cuba crashed and burned rather dramatically with acute renal failure and was put to sleep.

The reason I'm sharing this story in the C&T Series is that I, as well as every other vet I have spoken to, including vets at The Cat Clinic as well as the Referral/Emergency vets at BVSC in Brisbane, said they had no idea Lucky Bamboo was a toxic plant. It's a common plant; florists make very artistic designs with it (you can train it to form spiral formations) and it has allegedly very good Feng Shui for your home!

But 'Lucky Bamboo' is a lily and **not** lucky for cats. Needless to say, this owner is totally devastated that a 'lucky plant' he brought into his home killed his cat.

Just thought the message should go out to all vets that this common plant is lethal to cats.

Comment from Richard Malik, CVE

Camille provides circumstantial evidence that this plant can cause kidney failure in cats. I see it at the shops all the time. But what makes no sense to me – is that it grows from a stalk, and not a bulb, so it doesn't seem a Lilium species to me. That doesn't mean it isn't nephrotoxic.

Ross replies: This plant is NOT a lily NOR is it a bamboo. Common names should NOT be the basis for scientific investigations. Rather, vets should get a botanical name from a competent authority.

Invited Comment courtesy of

Dr Ross A. McKenzie PSM, DVSc
Registered Specialist in Veterinary Pathobiology
Honorary Research Associate, Queensland Herbarium & Biosecurity Queensland
Life Member, Australian Veterinary Association
'Yapunyah'
26 Cypress Drive, Ashgrove, Brisbane QLD 4060
T. (07) 3366 5038 M. 0458 012 352
E. yapunyah.house@bigpond.com

First, accurate identification of the plant associated with this clinical case has not been established (judging only by the report above), so we do need to proceed with caution because cultivated house plants can be difficult to identify, particularly the ones that do not usually flower. You need a scientific name to access the literature.

Second, common names are notoriously unreliable as a means of identification. Don't trust them without further investigation. Don't trust nursery labels, either.

Having got that off my chest, we can **probably** assume that the plant was a member of the *Dracaena* genus (Monocotyledons in Family Dracaenaceae, or for some botanists, Family Agavaceae). Please don't call this plant a bamboo. Bamboos are grasses in Family Poaceae; quite different! According to Roger Spencer's (2005) Horticultural Flora of South-eastern Australia (Volume 5, p375), there are about 60 *Dracaena* species known worldwide and he lists 7 as grown in Australia. To call the plant in question *Dracaena sanderiana* (or any other species for that matter), it would need to be examined by a botanist. Is the plant still available for examination? If so, Dr Paul Forster of Queensland Herbarium has expertise with this group of plants and **may** be available to give his opinion. In any case, take the plant to the Queensland Herbarium at the Mt. Coot-tha Botanical Gardens, Toowong, and ask them for an identification. They may be able to do that 'on-the-spot', but don't expect to get it back if they need to keep it for close examination.

The Biosecurity Queensland Natural Toxicants Database has a few entries on *Draceana*. In one, Selina Ossedryver (current curator of the database and my replacement in the role) and I were asked by a local veterinarian to identify a plant thought to be linked to renal failure in a cat in 2006 and the Queensland Herbarium called it *Dracaena sanderiana* (lucky plant). Other entries are records of a couple of discussion strings within the VETTOX Discussion Group (based in the US). Summarised, they indicate that *Dracaena* species have been suspected, but not firmly linked to renal failure in cats and dogs. They indicated that cats and dogs will chew the plant but gastrointestinal (GI) signs are the most likely outcome, if any. There was no peer-reviewed report of poisoning by this genus to 2009, when I retired. I have not searched the literature since then. I have included *Dracaena* species in the Digest section of my (2012) field guide/handbook on the poisonous plants, fungi and cyanobacteria of medical and veterinary importance in Australia (CSIRO Publishing), giving them a low risk rating. I listed GI signs, but have not mentioned ▶

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renal failure because the available evidence was not strong enough. Of course, this is not to say that renal failure is not a possible outcome.

I would be interested to hear if the plant is formally identified, and if any more evidence comes to light.

Australia's Poisonous Plants, Fungi and Cyanobacteria: A guide to species of medical and veterinary importance
Ross McKenzie

CSIRO PUBLISHING (http://www.publish.csiro.au/pid/6507.htm)
ISBN: 9780643092679

CVE Members receive a 15% discount on all CSIRO titles, including Ross's 'Poisons Bible'.
Go to www.vetbookshop

Book Review

Download here to read a comprehensive review from Stephen Page, also available on our website at www.cve.edu.au/candt2012 resources.

Editor's Note: We give the last word to Ross:-

I have not written this as an academic text. There are no literature citations, just a short reading list, and I have tried to write in a plain language style so that non-professionals will have a chance of understanding. I want it to be accessible to animal owners and managers so that they can use it to prevent poisonings.

Readers should realise that it has 2 functions: (1) information source and (2) exercise machine (it weighs 3 kg and repeated use will strengthen your arm muscles).☺

Dracaena sanderiana (Sourced From Wikipedia, the free encyclopedia)

Scientific classification

Kingdom:	Plantae
clade:	Angiosperms
clade:	Monocots
Order:	Asparagales
Family:	Asparagaceae
Subfamily:	Nolinoideae
Genus:	*Dracaena*
Species:	***D. sanderiana***
Binomial name:	***Dracaena sanderiana*** Sander ex Mast.[1]

Dracaena sanderiana is a species of the genus **Dracaena.** The species was named after the German-English gardener, Henry Frederick Conrad Sander (1847-1920). It is also known as *Dracaena braunii*, **Ribbon Dracaena, Lucky Bamboo, Belgian Evergreen** or sometimes **Ribbon Plant**. It is one of a group of small, shrubby species with slender stems and flexible strap-shaped leaves that grow as understory plants in rainforests. It is native to Cameroon in tropical west Africa. It is an upright shrub growing to 1.5 metres (5 ft) tall, with leaves 15-25 cm (6-10 in) long and 1.5-4 cm (1-2 in) broad at the base. It is marketed in the developed world as a Chinese decorative plant 'Lucky Bamboo' (although unrelated to Bamboo and not native to Asia), propagated from short cuttings, usually in water.

Cultivation and uses

Lucky bamboo spiral houseplant
Dracaena sanderiana and related species are popular houseplants, with numerous cultivars sold. It can survive in many indoor conditions, but indirect lighting is best as direct sunlight can cause the leaves to turn yellow and burn.

Although it grows better in soil, it often is sold with the roots in water. The water should be completely changed every two weeks. The water should be bottled water, soft tap water with very little fluoride, or even water from a filtered, established aquarium. It does best in bright, indirect lighting and temperatures from 15°C to 25°C (59°F to 77°F).

Yellow or brown leaf edges may be caused by too much direct light, crowded roots, or fluoridated or chlorinated water, the latter of which can be prevented by leaving tap water exposed to the air for a day before plant use. Salty or softened water can also cause this.

Twisted shapes can be produced by rotating the plant with respect to gravity and directed light sources. This is difficult to achieve for most home users, but not impossible with a lot of spare time and a lot of patience.

Often in large chain pet shops it will be sold as an aquatic plant. While it will live for months like this, it will eventually rot unless the sprouts are allowed to grow above the surface.

Other information

- *Dracaena sanderiana* in a more natural form, in this case at Ragunan Zoo, Jakarta,Indonesia.
- *Dracaena sanderiana* is toxic to pets.
- *Dracaena sanderiana* can flower in Autumn, Winter, and early Spring.

Dracaena sanderiana has long been associated with the Eastern practice of Feng Shui – or the bringing of natural elements of water, fire, earth, wood and metal into balance within the environment. Lucky bamboo is believed to be an ideal example of the thriving wood and water element, with the addition of a red ribbon sometimes tied around the stalks which is believed to 'fire' the positive flow of energy or chi in the room. The number of stalks also has meaning: three stalks for happiness; five stalks for wealth; six stalks for health. Four stalks, however, are always avoided since the word 'four' in Chinese sounds too similar to the Chinese word for 'death'.[2]

References
1. "Dracaena sanderiana information from NPGS/GRIN". www.ars-grin.gov. Retrieved 2008-03-19.

2."Quick Tips: Lucky Bamboo".

Invited Comment courtesy of

Dr Selina Ossedryver BVSc (Hons) MVSt
Senior Veterinary Pathologist & Curator,
Natural Toxicants Database
Biosecurity Sciences Laboratory
Biosecurity Queensland, DEEDI
Health & Food Science Precinct, 39 Kessels Road,
Coopers Plains
PO Box 156, Archerfield BC QLD 4108
T. (07) 3276 6070 F. (07) 3216 6620
E. selina.ossedryver@deedi.qld.gov.au
www.deedi.qld.gov.au
Customer Service Centre 13 25 23

Biosecurity Queensland is a service of the Department of Employment, Economic Development & Innovation

In addition to the cases from our Natural Toxicants Database that Ross has discussed above, a literature search revealed the following paper:

Przypadki zatruc dracena u kotow.

Adaszek,. ; Garbal, M. ; Kutrzuba, J. ; Winiarczyk, S. ; Kwiecinski, P. 2009. Case of *Dracaena* intoxication in cats. *Zycie Weterynaryjne* Volume: 84 Issue: 8 Pages: 655-657.

Abstract: The aim of this study was to present a case of two cats (1.5 and 2.5-years-old), that were presented to the veterinary clinic with pupils dilatation, drooling, diarrhea and vomiting. Information from the owner indicated possible *Dracaena* poisoning. Blood for routine hematological and biochemical examinations was taken from the sick animals. In one animal (cat #1), leukocytosis was evident and in both cases biochemical examination revealed a decrease of total serum protein concentration. Supportive fluid therapy (with aminoacids) and cocarboxylase treatment resulted in a gradual recovery. Detailed anamnesis and the positive response to the therapy indicated that both cats were intoxicated with *Dracaena* leaves they consumed.

I will certainly add Camille's report to the database for future reference.

Trigger points

C&T No. 5335

Sam Kovac
60/60 Princes Highway
St Peters NSW 2044
T. (02) 9516 0234
E. sam@southerncrossvet.com.au

Objective

Despite being accepted as a common cause of pain and dysfunction in humans, myofascial trigger points are greatly under-diagnosed by veterinarians, even though they are also common and a significant cause of morbidity in veterinary patients. Perhaps this is a result of a lack of published evidence on the matter (only 3 papers are in existence on myofascial pain syndrome in dogs). Only if clinicians take an interest in recognising myofascial trigger points in the clinical picture of lameness will more animals benefit.

What is a trigger point?

Myofascial trigger points have been defined in human medicine as a *'hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is tender when pressed, and can give rise to characteristic referred pain, motor dysfunction and autonomic phenomena'* (Simons, Travell and Simons, 1999). There is controversy over whether trigger points are a local or central nervous system disease; however, regardless of the pathophysiology, trigger points represent regions of localised muscle contracture, which are associated with capillary bed compression and profound hypoxia. These focal muscle contractures may cause pain (including referred pain) and interfere with function, for example muscle shortening may lead to altered stride length and altered gait.

Diagnosis

Locating a myofascial trigger point in veterinary medicine relies mainly on palpation. Careful palpation across a muscle belly may demonstrate a significant pain response on palpation of a focal area but no pain response in the area immediately surrounding the particularly tender area.

CASE STUDY:

Presenting complaint

'Emma' presented to Gladesville Veterinary Hospital (GVH) with hindlimb lameness, which the owner was concerned could have been bone neoplasia due to the genetic predisposition for greyhounds to develop osteosarcoma. The owner's previous greyhound also died of osteosarcoma, which had prompted her visit to GVH.

Significant history

One year ago, Emma experienced a suspected subluxation to her left coxofemoral joint, which was treated conservatively with rest and 50 milligrams of carprofen orally once a day. The lameness did improve slightly on re-examination 2 days later, and on physical examination it was found that there was moderate swelling associated with the gracillis muscle of the left hind leg. Carprofen was continued at the same dose rate and physiotherapy was recommended if there was ongoing lameness.

Four days prior to presentation Emma sat behind her owner with her tail between her legs and was shivering; this was followed 2 days prior to presentation with an episode of not wanting to walk. According to the owner, Emma was slightly scraping her left hind toes on the ground and occasionally yelped when she rose from a laying position.

Physical examination:

Emma was walked away from, towards and beside the clinician and student in order for her gait to be analysed. It was found that her left hind leg was slightly outwardly rotated and slightly abducted. Deep palpation of Emma's epaxial musculature elicited no pain response. Emma's 5th digit of the left hind leg had a reduced range of motion compared to the contralateral 5th digit. The other left hind limb joints had no abnormalities detected with no crepitus and full range of motion achieved in each joint. Emma had mild wasting of the left hind biceps femoris, quadriceps, tensor fasciae latae and middle gluteal muscles.

On deep palpation for trigger points, Emma exhibited a pain response to the left hind limb tensor fasciae latae muscle. No other trigger points were found in any other leg.

From physical examination findings the following problem list was generated: ▶

1. Reluctance to walk
2. Scraping of left hind toes on the ground
3. Outward abduction of left hind leg
4. Decreased stride length of left hind leg

Referred pain from an acute abdominal disorder such as gastric dilation and volvulus (GDV), gastritis, and intestinal volvulus was possible but Emma was not showing any other systemic signs, making her problem much more likely to be a musculoskeletal disorder.

Trauma to the hip abductor muscles was a possibility. This was likely as, on gait analysis, the lameness was associated with an outward rotation of the hip and Emma did have a gracillis muscle strain diagnosed ultrasonographically 2 years ago.

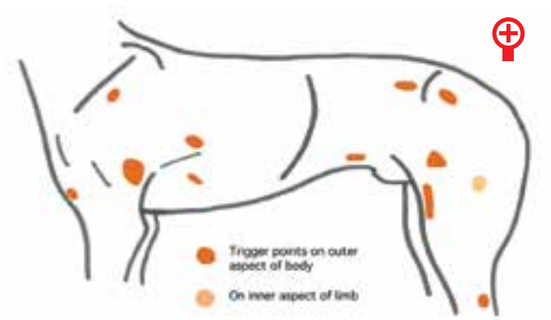
A type I or II Hansen disk herniation into the spinal cord at T3-T13 is possible but unlikely. A disc herniation could lead to the neurological deficits of the hind legs; however, herniations tend to manifest as bilateral paresis. Emma's lameness was unilateral and there was no paresis.

Trigger points in the hip abductor muscles causing contractile shortening of these muscles and thus abduction of the limb was a likely diagnosis at this point and could explain all of the physical examination findings.

Transcutaneous electrical nerve stimulation (TENS) of the motor end plate of the region of tensor fasciae latae in which the trigger point was present was conducted using a Pointer Excell II TENS device (Lhasa™). Following TENS therapy, stretch of the tensor fascia lata by extending the hip was undertaken. It is thought that electrical stimulation using TENS temporarily allows stretching of the affected muscle to reduce the localised contracture.

Result

The next morning when the owner was contacted she reported that Emma was completely healed and there was no evidence of lameness or discomfort. At the check-up appointment, no trigger points were identified and Emma's lameness was not evident. At 3 weeks post appointment when the owner was contacted by phone, Emma had no evidence of lameness and was much brighter in herself.



WINNER OF BEST PICTURES

Linear foreign body (LFB) in a dog

C&T No. 5336

May Chin Oh & Anne Fawcett
Sydney Animal Hospitals Inner West
1A Northumberland Ave, Stanmore NSW 2048
T. 02 9516 1466
E. innerwest@sydneyanimalhospitals.com.au

A 13-month-old male neutered Mastiff presented with a history of vomiting and inappetance following ingestion of a large bone approximately 20 hours earlier. The owner admitted that the dog was also a chewer of inanimate objects in general including toys, plastic and socks.

Physical examination

On physical examination the dog was in good body condition (body condition score 3/5, **Figure 1**) but profoundly depressed – although a tail wag could still be elicited. Abdominal palpation revealed a rounded soft mass, approximately 8cm in diameter, in the mid abdomen which elicited marked pain on palpation.

Further investigation & treatment

The patient was premedicated with methadone (0.2mg/kg SC) and placed on intravenous Hartmann's solution at surgical rates. Keflin (25mg/kg IV) was administered in anticipation of abdominal surgery. Left and right lateral abdominal radiographs revealed plication of the small intestine with gas accumulation more evident in the left lateral view (**Figure 2a & 2b**).

Anaesthesia was induced with alfaxalone CD (2mg/kg IV to effect) and maintained with isofluorane in oxygen following intubation.

A ventral midline coeliotomy was performed. The jejunum was plicated (**Figure 3**) and difficult to expose due to tension created by the linear foreign body. The LFB extended from the pylorus, where it was anchored, to the jejunum. There was no indication of enteric rupture, peritonitis or mesenteric torsion.

An enterotomy was performed at the site close to the distal end, allowing partial removal of a string-like foreign body impacted with faecal and plant matter (**Figure 4**). Two additional enterotomies were performed to remove most of the remainder of the LFB. Finally a gastrotomy was performed at the anchoring point and the stomach was suctioned. A large knotted bunch of string-like material anchored in the pylorus was easily removed.

All three enterotomies were closed with 3/0 Monosyn in a simple continuous pattern (**Figure 5**). The gastrotomy was closed with 3/0 Monosyn in a simple interrupted pattern.

The abdomen was lavaged copiously with several litres of warmed saline. A routine three layer closure was performed.

The patient received methadone and maropitant at standard doses post-operatively for pain relief.

The owner later identified the foreign material retrieved as the remains of a rope chew toy (Figure 6). The patient ate Hills i/d approximately 24 hours after surgery. Given his incredible energy and ravenous appetite it was decided that it was best to board the patient post-operatively to allow the surgical wound to heal and to prevent misadventure. This proved a wise decision.

Comments

During the time that the owner went home and "dog proofed" the entire house to such a degree every non-furniture item in the house was removed – with the exception of the TV remote control. The patient, upon arriving home, was seen to "eat" the remote control. Fortunately the remains of the remote control were later found in the house – badly chewed and written off, but not inside the dog which was all we were concerned about. We always warn owners that despite undergoing major surgery, most foreign body eaters are repeat offenders if given the opportunity!

Anderson *et al* (1992) described a single enterotomy technique for LFB removal in dogs and cats, which where possible reduces surgical time significantly. The technique involves making a single enterotomy to remove the anchor and tying this to a catheter or similar (e.g. sterile needle cap), then milking the catheter aborally and removing per rectum (or via an additional enterotomy). The sheer size and abrasive nature of the foreign body in this case precluded the use of such a technique. The bulkiness of this particular rope-like foreign body resulted in several smaller anchor points contributing to the plication – thus multiple enterotomies were unavoidable.

In one major study comparing the use of continuous and simple interrupted suture patterns for enteric closure and intestinal anastomosis in dogs and cats, the dehiscence rate was similar (2% in continuous closures versus 4% in simple interrupted closures, one animal in each case), with 98 per cent of patients surviving with no evidence of dehiscence overall (Weisman *et al* 1999). ▶

Figure 1. The patient, a male neutered mastiff weighing approximately 30kg.

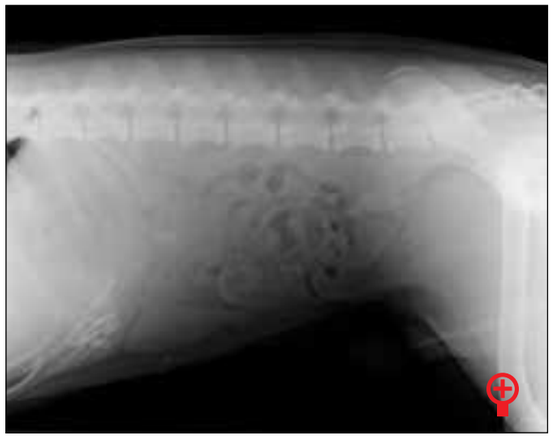


Figure 2a & 2b. Left and right lateral abdominal radiographs reveal plication of the small intestine with gas accumulation more evident in the left lateral view.

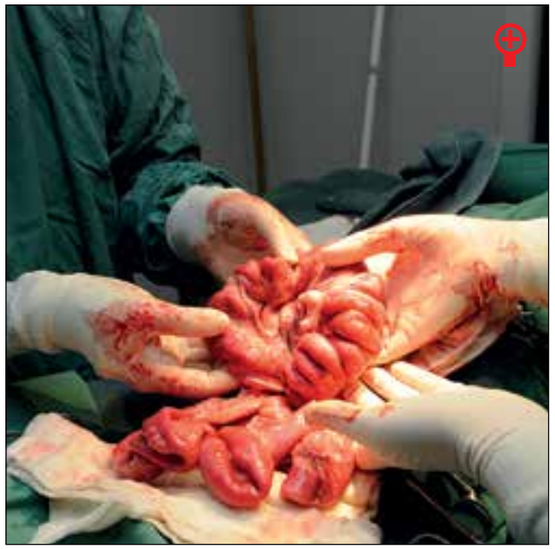


Figure 3. Exploratory laparotomy revealed marked plication of the small intestine.

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Figure 4. Fibrous material and hair were removed via two enterotomies and a gastrotomy. The bulk of the foreign body was anchored in the pylorus.

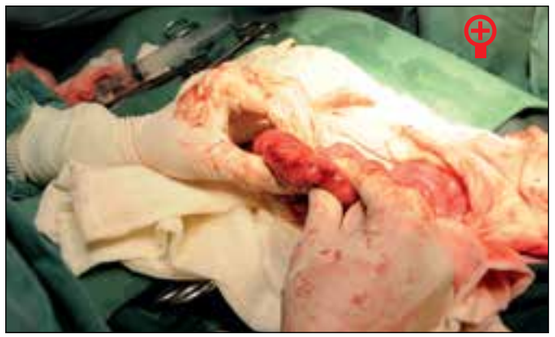

Figure 5. The thickness of the foreign body and adhesion to the intestinal mucosa necessitated large enterotomies, one of which is pictured closed here.

Figure 6. What was saved of the foreign material. Much broke down upon removal but the largest part, found in the pyloris, is just above the 15cm length ruler, followed by lengths of the remainder of the foreign body which was sectioned prior to removal.

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Anderson S, Lippincott CL, Gill PJ (1992) Single enterotomy removal of gastrointestinal linear foreign bodies. *Journal of the American Animal Hospital Association* 28(6):487-490.

Weisman DL, Smeak DD, Birchard SJ, Zweigart SL (1999) Comparison of a continuous suture pattern with a simple interrupted pattern for enteric closure in dogs and cats: 83 cases (1991-1997). *Journal of the American Veterinary Medical Association* 214(10):1507-10.

Feline eosinophilic proliferative glossitis case

C&T No. 5337

Marshall Thornton
West Cessnock Veterinary Hospital
2a Percy Street, Cessnock NSW 2325
T. 02 4990 4400
E. wcvh@tpg.com.au

Figure 1. Note proliferative lesions on the tongue and palatoglossal arches.

Cat 'Smokey', domestic shorthair MN around 10-years-old, first seen June 2009 with old RH leg fracture, which we amputated. Smokey lives with a nearby relative, so the owner does not see him that often.

Smokey has had untreated all over military eczema sporadically, also a lip ulcer that has come and gone for years.

In March 2011 Smokey had the linear thickened upper lip ulcers, abraded tongue tip, linear plaque on hard palate spreading into soft palate. He had punctate scabs around head and neck. I diagnosed this as EGC complex, and he responded well to 2 20mg injections of methylprednisolone acetate 3 weeks apart.

Early January 2012 Smokey presented again with a large diffuse soft submandibular swelling. Since he does not live at home, the owner was not that specific on history, but he thought Smokey had been scratching the hair off under his throat maybe 2 or 3 weeks prior. The swelling had been present 2 days or so and was enlarging.

CE: Weight 4.9kg, mouth unable to examine due to ferocious nature, ventral throat soft swelling, skin surface over swelling alopecic. Heart sounds and lungs sounds were fine with no abdominal abnormalities detected, palpation no abdominal abnormalities detected, skin tent 0, temperature 38.1˚C.

A fine need aspirate was watery grey/yellow fluid – DQ – non lytic neutrophils mainly, with some lymphocytes, some giant multinucleate cells (activated macrophages?), no bacteria seen. Further note – my microscope does not have a camera mount. The giant cells seen were at least 20 times the size of a neutrophil. Some had U-shaped basophilic nuclei, some appeared to be binucleate. The cytoplasm was lightly basophilically stippled, some were vacuolated. The neutrophils were not the typical degenerative types seen in a typical abscess smear. I did not notice any large numbers of eosinophils.

Diagnosis: sterile abscess unknown aetiology

The following day I anaesthetised Smokey. On examining his mouth there were proliferative lesions on and around the tongue. The photos show the lesions. I took a 6mm punch biopsy of the dorsal tongue lesion. The submandibular swelling had gone down over night. I put a small cross incision into where the middle of the swelling had been, to facilitate any further drainage. The skin was detached from the subcutaneous tissue for a wide area under the mandible and over the ventral throat surface. There was no pyogenic membrane. From the tongue base oedema I saw, I thought the submandibular fluid had come from there.

Histopathology report

A prominent eosinophilic granulomatous glossitis is present with numerous well formed eosinophilic granulomas in the subepithelial tissue. Several of these contain foreign bodies. One is a refractile colourless rectangular structure. The other is yellowish with a cuticular wall that could be a hair or possibly a setae of a caterpillar. This would appear to be a foreign body eosinophilic granuloma rather than one induced by allergy.

Diagnosis: Eosinophilic Granulomatous Glossitis

Comment: There is no evidence of neoplasia. This appears to be a foreign body reaction because the granulomas have discrete foreign bodies mostly in the centre. Caterpillar setae from hairy caterpillars such as processionary caterpillars can cause these sort of oral lesions in cats when they play with the caterpillars or put them in their mouths.

With thanks to Dr Angela Begg (Vetnostics).

Amelanotic malignant melanoma

C&T No. 5338

Linda M Schoenberg VMD PhD DABVP (Feline Practice)
Just Cats Hospital
4125 Mayfield Road
South Euclid, Ohio 44121-3041
Check OK to publish email
E. drcats@sbcglobal.net

'Poochie', a 11½-yr old DLH FeSp was seen initially 4/5/10 because of 'sores' in the mouth. The previous veterinarian had seen the cat for an annual wellness exam 3/19/10 and dispensed clindamycin on 4/3/10. We noted gingival hyperplasia on the left upper arcade. We performed a dental prophy, performed a gingivectomy and biopsied the tissue from the left upper arcade. It was also noted that the R upper canine had some proliferation of gingival tissue as well. The biopsy came back as fibromatous and ossifying epulis of periodontal ligament origin, incompletely excised.

On 12/20/10, the cat returned because of recurrence of the epulis and we repeated the gingivectomy. On 8/4/11 the cat returned again but this time, there was weight loss and now, the hard palate on the L side appeared abnormal. We consulted with a radiologist, oncologists and a surgeon who recommended skull rads to check for bone lysis and the necessity for either a hemimaxillectomy or radiation therapy afterwards. The owner declined to do any further diagnostics at that time.

We saw the cat on 1/21/12 because of multiple growths on the face. The masses were on the L side of the face: nostril, muzzle, upper eyelid, side of noses and gingiva. The owner elected euthanasia and I was given permission to biopsy the tissues. After the initial histopathology was performed, immunohistochemical staining S100 was performed to confirm the diagnosis of amelanotic malignant melanoma.

Postscript: The lab that performed the histopathology on both occasions is a wonderful facility. My guess is that when we first biopsied the tissue, we may not have gotten deep enough for an accurate diagnosis to be made. We submitted 3, lobulated 0.5-1.0 cm long irregular pieces of glabrous tissues. The pathologist commented on the necropsy specimen: *It is likely that the previous gingival lesion either was the same tumor at a less aggressive stage or that this tumor developed in or deep to the previous lesion which had lower cellularity, bland nuclear features with inconspicuous nucleoli and a low mitotic rate (the original slide was reviewed). This does not prove that this tumor is a melanoma because the neoplastic mesenchymal cells in sarcomas are capable of phagocytizing melanin pigment in the skin. One of the features of this tumor that would support a melanoma is that the spindle cells infiltrate to the dermal-epidermal junction (or to the junction of the lamina propria and mucosal epithelium) in nonulcerated areas. Sarcomas occasionally also infiltrate to the dermal-epidermal junction).* The lab then did immunohistochemical staining for S100 on the mass by the nose which supported the diagnosis of amelanotic malignant melanoma. ▶

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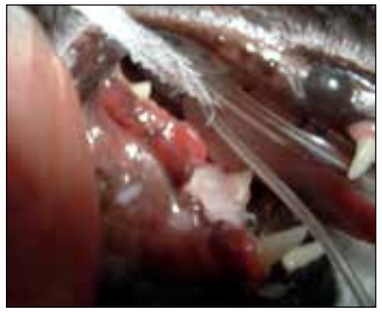
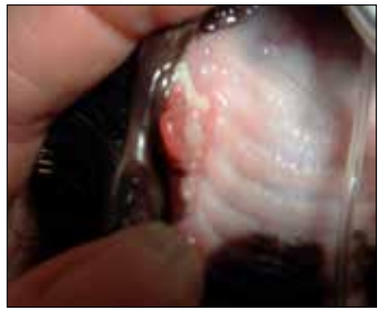
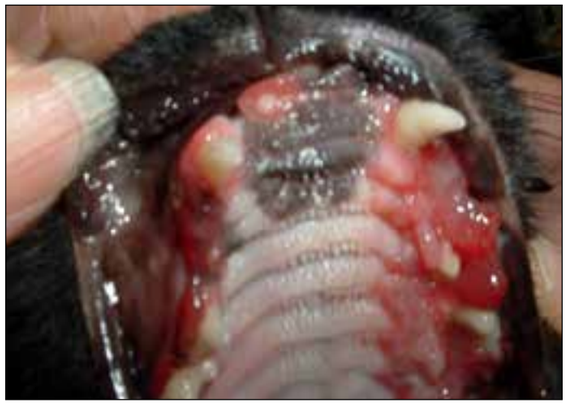




Figures 1 to 3 above taken 6 April 2010.


Figures 4 to 5 Taken 20 December 2010.





Figures 6 to 8 (left). Taken 4 August 2011.
Figures 9 (above). Taken 21 January 2012.

Tale of a broken tail

C&T No. 5339

Kitty Cheung
University of Sydney
E. wche8536@uni.sydney.edu.au

Anne Fawcett
Sydney Animal Hospitals Inner West
1A Northumberland Ave Stanmore NSW 2048
T. (02) 9516 1466
E. innerwest@sydneyanimalhospitals.com.au

A 7-year-old male neutered domestic short hair presented after a suspected altercation with a motor vehicle. The cat had been out all night and returned in the morning, dragging its hind limbs. The cat then urinated in its litter tray, passing frank blood while vocalising loudly.

At presentation the cat was laterally recumbent. Nails on both hind legs were frayed, consistent with trauma. The tail was flaccid, and caudal vertebrae could not be palpated in a segment approximately 4 cm long midway along the tail (Figure 1). No mal-alignment was detected on palpation of the caudal lumbar spine, sacrum or sacrocaudal junction. The presence of anal tone was reassuring, but the absence of deep pain and withdrawal reflexes in both pelvic limbs were a concern. The entire tail felt cold to touch and did not respond to stimulation. With the exception of a superficial graze in the left gluteal region there were no external injuries.

Thoracic and abdominal radiographs were unremarkable. The pelvis and lumbosacral spine were intact. There was complete avulsion of the caudal vertebra between CV10 and CV11 (Figure 2). The avulsed segment of the tail was removed surgically to improve patient comfort and prevent further traction and injury to the caudal nerve roots by removing the dead weight of the tail (Davis & Walmsley, 2012). Incision over the avulsion site revealed crushed tendons of the sacrocaudales muscles and caudal spinal nerves (Figure 3). These were infused with lignocaine and transected. The wound was closed in 2 layers with a 3.0 synthetic monofilament (Biosyn).

Post-operatively, the cat received amoxicillin-clavulanic acid (20.5mg/kg PO q12hr x 7 days) and meloxicam (0.05mg/kg PO q24hr x 7 days). In addition, gabapentin (8mg/kg PO q 12hr x 30days) was dispensed to minimize neuropathic pain.

The cat remained paraparetic following surgery. However, the following morning the cat was able to walk (albeit with an abnormal pelvic limb gait), urinate and defecate. The base of the tail felt warm. In the morning of day 2 the tail stump was completely flaccid. However, by midday the stump was mobile (Figure 4) and slightly warmer to touch. The patient was sent home for some indoor R&R, with an Elizabethan collar.

Five days post-operatively, the cat was re-examined. The tail had been traumatised by licking/biting and the skin at the base of the tail was cold and exudative (the Elizabethan collar had been used intermittently). Osteomyelitis secondary to self trauma of the tail stump was suspected, although no abnormalities were detected on radiographs.

The owners were instructed to use an Elizabethan collar consistently. One week later the skin over the tail stump felt colder to touch and appeared necrotic. A further 4 caudal vertebrae were removed. These were not submitted for histopathology due to cost considerations. The cat made a full and uneventful recovery. Gabapentin was continued for 2 months post-operatively for the treatment of ongoing neuropathic pain.

Discussion

This cat was lucky on 2 accounts. Tail injuries like this are often associated with major traction being applied to the tail, for example by a car or bike tyre, as the cat tries to escape (Davies & Walmsley, 2012). However, traction on the caudal nerves can cause a mild neuropraxia which resolves rapidly (Davis & Walmsley, 2012) which it did in this case.

Concurrent injuries, including pelvic and femoral fractures, are present in up to 84% of cases (Smeak & Olmstead, 1985). Our patient appears to have been one of the small number of cats to escape almost exclusively with a tail injury. In the absence of radiographically apparent abdominal or pelvic injuries, it is likely that the haematuria was due to blunt trauma to the bladder.

The absence of radiographic changes shortly after the first procedure does not rule out osteomyelitis, but necrosis of skin of the tail reflected damage to the blood and nerve supply of the tail due to the initial insult. Neuropathic pain is a likely trigger for self trauma in these cases, necessitating prolonged treatment.

In refractory cases, multimodal analgesia, including local anaesthesia, tricyclic antidepressants and NMDA receptor antagonists may be required to resolve signs including self trauma (O'Hagan, 2006). There are scant reports of phantom limb pain (PLP) in the veterinary literature, but it is possible that this patient suffered from a form of PLP, the risk of which is increased in patients with pre-amputation pain (O'Hagan, 2006). It would be interesting to know if epidural anaesthesia at the time of the first surgery, and increased perioperative anaesthesia such as an intravenous ketamine infusion, may reduce the incidence of post-operative self trauma.

What we learned

- We were a little anxious to palpate the tail extensively, but tail sensation is an excellent prognostic indicator for return of tail function.
- Perineal sensation and anal tone are key prognostic indicators. Where urinary bladder function is affected, the absence of perineal sensation and anal tone for more than 2 weeks was a very poor prognostic indicator.
- Affected cats that are going to regain urinary function tend to do so within 7 days.
- Surgical stabilization or amputation of tail fractures is important in reducing the incidence of chronic tail-base pain.
- Tail pull injuries can take some time to fully declare themselves and owners should be counseled accordingly. ▶

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Davies E & Walmsley G (2012) Management of Tail Pull Injuries in Cats *In Practice* 34:27-33.

O'Hagan BJ (2006) Neuropathic pain in a cat post-amputation. *Australian Veterinary Journal* 84:83-86.

Smeak DD & Olmstead ML (1985) Fracture/luxations of the sacrococcygeal area in the cat: a retrospective study of 51 cases. *Veterinary Surgery* 14:319-324.

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Figure 1. The tail on presentation.

Figure 3. Exposure of sacrocaudales muscles and caudal spinal nerves.

Figure 2. Ventro-dorsal radiograph of the tail illustrating dramatic avulsion of the caudal vertebrae.

Figure 4a. The tail stump is flaccid the morning following surgery.

4b. The tail stump is mobile later in the day.

Figure 5. The tail stump 12 days following surgery.

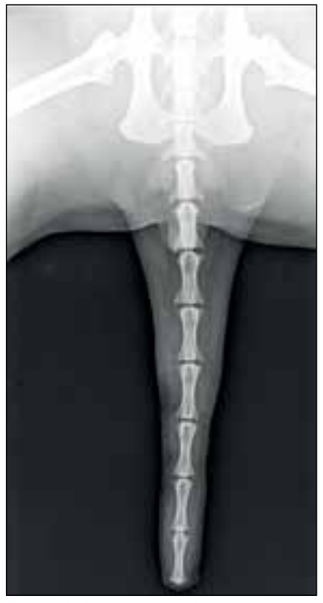

Figure 6. Radiograph of the tail stump prior to second procedure.

MAJOR WINNER

'Whip-it good'

C&T No. 5340

Naomi Lessels
Leslie St Veterinary Clinic
Leslie St
Umina NSW 2257
E. nslessels@gmail.com

Naomi (right) pictured with nurse Sam Tulloch who has sadly moved on up north. Miss you Sam!

I'm a mother of 2 boys (aged 9 and 10 years) and I love bike riding, fishing and skateboarding with them. I'm currently raising money for the ride to conquer cancer – a 200km bike ride in October benefitting the Chris O'Brien Lifehouse at RPA. If anyone would like to donate just search my name under www.conquercancer.org.au

Thanks!
Naomi

'Whip-it', my twin sister's accident prone 13.5kg 18mo FS Whippet suffered a horrifying injury on May 3 2012. I'd told my sister to not throw sticks at the beach but she had felt it would be OK to throw the stick in the water. On this day her 2 dogs came running out of the surf each holding the end of a stick approx 60cm long and 2.5cm wide. As they ran along the beach 'Allie' the kelpie cross dropped her end of the stick and it dug in the sand. Whip-it then thrust on to it. A stranger drove them all to our nearby clinic with half the stick protruding from next to Whip-it's sternum. Thankfully my sister is a paramedic and this proved to be a great help in the immediate treatment and recovery.

By the time I'd arrived a screening radiograph had revealed the stick had penetrated adjacent to the sternum and had stopped at the level of the diaphragm adjacent to the vena cava – see pic. Approx 20cm of stick was in the chest.

Methadone 0.3 mg/kg had been given IM by a colleague.

An IV line was placed and shock fluid rates given-90mL/kg/hr while we assessed. Cefazolin 20mg/kg was dribbled in over minutes and 1 mL Clavulox®, 1.5mL Baytril® given SC. We all tried to ignore the horrible sound of the sucking air from the wound.

Once on O_2 with an IV line and pain relief on board, I elected to anaethetise. It was approximately 10 minutes from time of injury to presentation and 15 minutes from presentation to anaesthetic; like most of the following this is open to debate.

She was induced with 20mg alfaxan, intubated and placed on Isoflurone/O_2 maintenance. The IV fluid rate was reduced to 130mL/hr as her colour improved. We also had to warm her up as she'd come straight out of the surf and her initial rectal temp was only 35.9 C.

KYJelly® was placed around the wound and the RHS chest wall and sternum were prepped as best as possible . Thankfully our theatre table can be positioned in a v shape which really helped with such a deep chested breed. She was on a 45˚ angle allowing access to the wound adjacent to her sternum as well as the right chest wall. Her O_2 stats hovered between 85 and 90 through this. Intermittent positive pressure ventilation (IPPV) wasn't started until after the stick was removed.

I hadn't treated such a trauma before and elected to make an incision in about the 5th intercostal space through the skin and SC approx 15cm long should I need to dive in and stop haemorrhage when the stick was removed.

We braced for stick removal; I placed sterile gauze around the stick and gently pulled. On retrieval her oxygen stats started to improve and thankfully blood didn't start welling in the chest. IPPV was started and continued until the defect in the chest wall was repaired. I guess it was fortuitous that the defect was under the pectoral muscles. Access was initially difficult and I decided to elevate the superficial pectorals then blunt dissect down through the muscle plane of the deep pectorals to get access. I used this overlying tissue to plug the defect by suturing with 2/0 monocryl. A fenestrated chest drain that attaches to a closed collection system was placed. I elected to put this through the defect that was present... Maybe this isn't good practice but we ultimately had a good result. Post op rads showed a non-ideal position of the tube but placement is surely difficult in such a deep chested breed – I felt like I was feeding it in to a deep dark well. In hindsight repositioning her in lateral recumbency so I could have run it in ventrally and caudally would have been better. The trochar which came with the drain greatly facilitated tunneling and exiting the tube. It was fixed with a Chinese fingertrap suture and a routine closure of SC and skin was performed.

Post op rads taken

The chest drain immediately drained approx 100mL haemorrhagic fluid. For a gut wrenching 30 seconds I didn't think it was going to stop. As it turned out we drained approx 300mL in the first 24 hours and another 300mLs over the following 3 days.

A methadone infusion of 0.1mg/kghr for 24 hours was started. Every 4-8 hours 0.1mL ACP was given IV to help sedate her if she seemed uncomfortable.

We postponed our routine surgery and kept her on the heated table in surgery with monitoring equipment on. She remained intubated for a good 2 hours after the anaesthetic was turned off. With her lack of response I was worried she'd suffered cerebral hypoxia and would be blind or worse.

One hour post op I rechecked her PVC/TP and they were 38% and 5 g/L respectively. We had some fresh frozen plasma that we'd frozen in to 20mL aliquots so we thawed 2 under warm water and gave them slowly IV.

Approx 1½ hours post op she had an episode of ventricular tachycardia – well the heart rate shot to 230, an ECG was put on and our monitoring equipment declared ventricular tachycardia. In my haste I didn't print a trace but started giving lignocaine slow IV and told my sister to say goodbye to Whip-it. 20 mg lignocaine IV later she was settling and back on track. It was about an hour after this I extubated the blood and froth ▶

coated ET tube and put a mask on her. She coped well and 2 hours after this removed the mask. During this time we'd organised a blood transfusion.

I managed to speak to Andrew Marchevsky at SASH at this time and debrief a little on the traumatic morning – Thanks Andrew! I felt a bit more reassured about not proceeding to a thoracotomy. Obviously we considered referral for continual monitoring but where we are on the central coast is about 1 ½ hours from any 24 hour Emergency vet. I felt fairly certain she'd require further surgery for pyothorax, removal of necrotic lung lobe, skin necrosis or any other nasty complication possible.

Mid afternoon I gave 250mL whole blood in to a new catheter in medial metatarsal vein. Doppler BP checks over the course of the day continued to be good. Helena, being a paramedic, stayed with Whip-it the whole day and was able to constantly monitor/re-heat hot water bottles, check rectal temperatures etc and all the other myriad nursing things that are helpful in this setting when your regular nurses are trying to answer phone calls and clean or help other vets etc.

I started Whip-it on a course of 20mg/kg cefazolin IV q8 hours for 6 doses as well as the daily SC Clavulox® and Baytril®. We increased the dose of Clavulox® the following day to 20mg/kg SC and the enrofloxacin was the standard 5mg/kg SC. About a week later she developed a SC abscess which I attributed to the Clavulox® injection given in haste at the initial time of presentation.

Whip-it was transferred to the local overnight Animal Emergency Centre for overnight monitoring where thankfully she remained stable. The following day she was transported back to our clinic and we continued to keep the chest drain primed and monitor her temperature. Amazingly she wanted to eat so small amounts of cooked chicken were given. She wasn't so keen on the softer canned recovery diets. Her pain relief was well managed by the ongoing methadone infusion.

After some deliberation she was discharged to my sister's place in a collapsible cage we hired out. I have to admit the nursing here was amazing; continual monitoring with housemate's sautéing chicken thighs on demand. She was dispensed buprenorphine in pre made up syringes 150ug with 0.015mg/kg ACP 12 hourly for 3 days then swapping to tramadol and 20mg onsior with oral amoxyclav/Baytril®/metrogyl once her appetite was on track. I removed her IV fluids 48 hours post op and her chest drain 4 days post op. It helps having a paramedic as the owner – she followed all written instructions perfectly.

When I removed the chest drain I stained a drop of some of the last fluid in the tube with DiffQuik. Thankfully there were very few free bacteria and macrophages galore obviously having been hard at work. The bacteria present were cocci. No rods were seen.

At time of suture removal all was well.

I'm ecstatic Whip-it lived, without lavage of the chest. I was extremely anxious about this decision. I wasn't sure which antibiotic protocol to go with but feel good about the repeat cefazolin IV. I think most people would use Clavulox®/Baytril® combo as their go-to option. I'm sure people in the know can offer more advice on this. On day 4 when I removed the chest drain it was Sue Foster, Medical Consultant from Vetnostics who advised adding in the metrogyl. Thanks Sue, always awesome when I'm in a flap. 200mg bid 10 days was started.

The closed chest drains by ASTRA (Bellovac FG 18 with trocar) are outstanding. I'd only used them before after large mass removals. I try and avoid chest surgery… the only thoracotomies I'm involved with are the dog-attacks-cat or small dog ones…

Figure 1. The ruler and arrow indicate the length of stick that had penetrated.

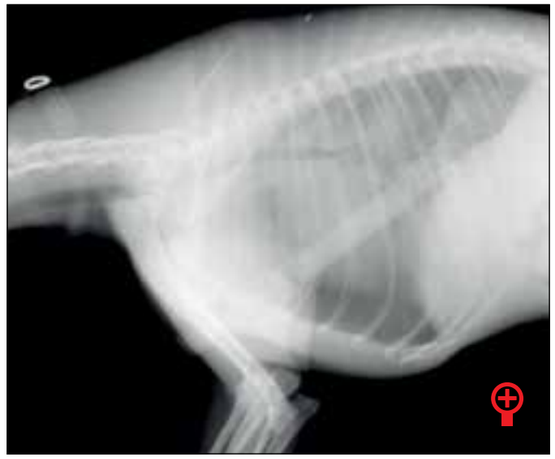

Figure 2. X-ray on initial presentation.

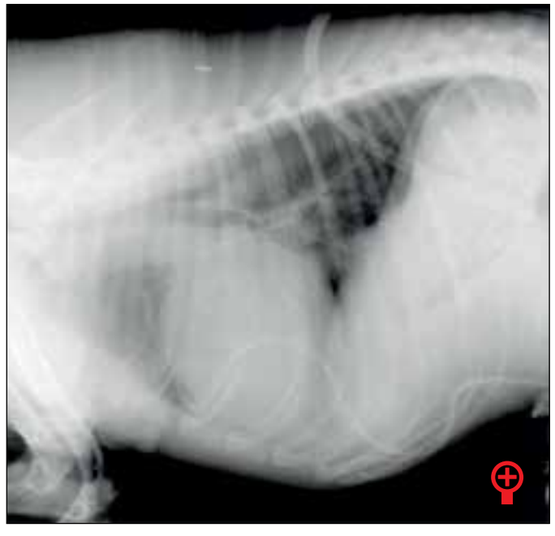

Figure 3. X-ray post op.

Figures 4 and 5. Pampered recovery at home with closed collection system in place.

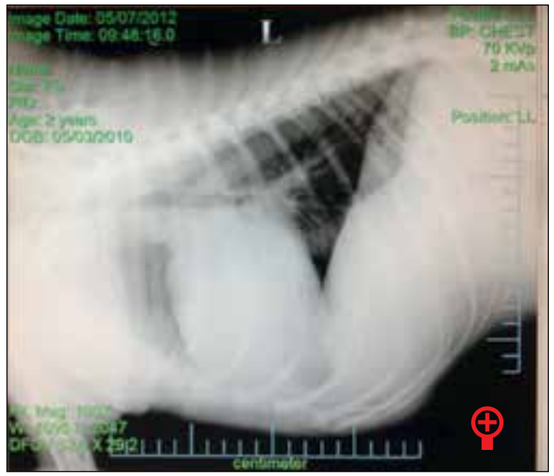

Figure 6. (top right) X-ray at time of drain removal.

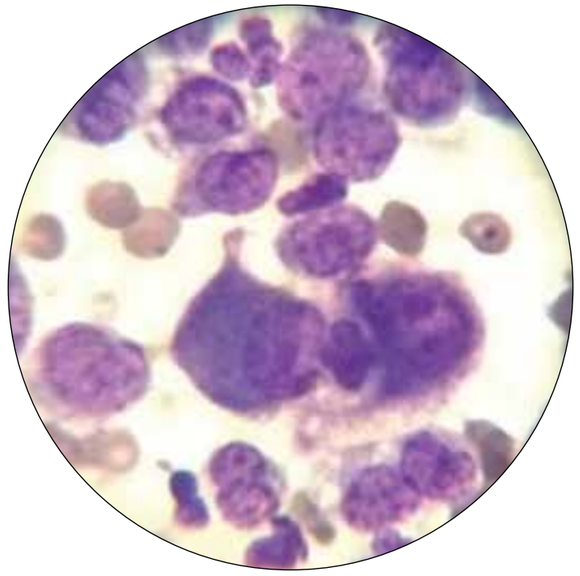

Figure 7. (bottom right) DiffQuik stain of fluid collected when drain removed. Unfortunately no bacteria is shown in this image due to the difficulties of taking pictures with an iPhone attached to the microscope lens; however, in other portions of the slide intracellular bacteria were visible.

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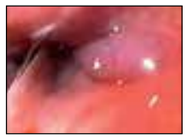
MAJOR WINNER

Nasal mites: a tale of six dogs (and then one)

C&T No. 5341

Sue Foster BVSC MVetClinStud FACVSc (Feline Medicine)
Small Animal Medical Consultant
Vetnostics
60 Waterloo Road
North Ryde NSW 2113
E. sfoster@bigpond.com

& Jody Braddock BVSc (Hons I) MVetClinStud FACVSc
(Canine Medicine Specialist)
North Shore Veterinary Specialist Centre
64 Atchison Street
Crows Nest NSW 2065
E. jodybraddock@bigpond.com

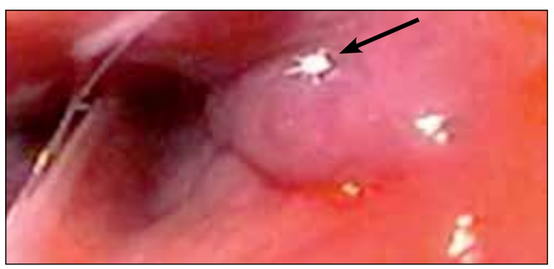


Video courtesy of Sue Foster

The story

Dog 1: a 2 y.o. Kelpie x from Kalgoorlie – client's dog
Dog 2: a vet nurse's dog
Dog 3: a 7 y.o. M red cattle dog – friend's dog
Dog 4: a 5 y.o. FN Border Collie – my dog
Dog 5: an aged MN Bull Terrier x – another friend's dog
Dog 6: a 12 y.o. MN Kelpie x – friend-of-a-friend's dog

Dog 1 presented with really severe and distressing reverse sneezing, worse at night and so bad that the owners were considering euthanasia. Visual inspection of nose and pharynx was normal. Rhinoscopy showed follicular hyperplasia and nasal mites (see below). Treated with milbemycin which elicited an incredibly severe reaction the first night (owners thought the dog was going to die!). Complete resolution of clinical signs after that and no reaction to subsequent milbemycin doses – very grateful owners.



Rhinoscopy from Dog 1 showing nasal mites in the nasopharynx

Dog 2 presented to my resident with reverse sneezing about a week later so I suggested that perhaps we could do a treatment trial with milbemycin before scoping. The resident thought pattern recognition based on n=1 was not terribly scientific so scoped, ran up a big bill, found the mites and then treated successfully with milbemycin.

Dog 3 was heard to snuffle and snort a few times at hockey training so with Dogs 1 and 2 fresh in my mind, I joked with the owner that he was bound to have nasal mites. It became a running joke. She was sure it was because he was a rural dog, allergic to town, as the signs always disappeared up north in the country (where it is significantly warmer). One day, Dog 3 came to stay and within a fortnight my own dog, **Dog 4**, started to reverse sneeze for the first time, just once or twice a day but reverse sneezing nonetheless. Mites no longer seemed a joke, so home came the milbemycin, which was given around 6pm. At 2am, I headed into Murdoch for the prednisolone: Dog 3 was reverse sneezing almost constantly and Dog 4 was having severe episodes every 15 minutes and I had one each side of the bed. Got some sleep after prednisolone kicked in and signs in both dogs resolved after 24h. I rang Dog 5's owner who had also looked after Dog 3 and found that he had been heard to have occasional odd respiratory sounds also. He was dosed with milbemycin and prednisolone (for the first 24h) and resolved without issue.

Dog 6: about 18 months after treating Dogs 3-5, I was sitting at a friend's birthday breakfast when I fielded the inevitable question: 'Do you mind if I ask you a veterinary question?' SIGH. 'No'. 'Our vet is completely stumped as to why our dog just makes these terrible sort of gagging noises. It is really distressing, especially at night and we are going to have to put him down. He has had multiple visits with scoping and radiographs and there is nothing there. He is on prednisolone which helps a bit but the side effects are unpleasant and nothing really stops this terrible gagging.' Dogs 3 and 5 had stayed with Dog 6! I worked out the dose of milbemycin and told them to pick up some Milbemax® from any vet on the way home and to continue the prednisolone. I received the most grateful (but irate) phone call the next week. He was cured. They were furious that their lovely old dog had had to suffer for 18 months without respite when the condition was obviously diagnosable over a champagne breakfast. SIGH. Some vet out there will not be happy with me but I did try and explain that the condition is not widely recognised in Australia and that I would write it up!

The morals of the story:-

1. Nasal mites are out there, they do occur in Australia and they are probably quite frequently overlooked and under-diagnosed.
2. Reverse sneezing is a common presentation and it may be worse at night, possibly related to cooler nocturnal temperatures.
3. Clinical signs can be so severe that owners will consider euthanasia.
4. Nasal mites are easy to treat with milbemycin but if you don't want to be called in the night by a distraught owner, make sure you give prednisolone at 0.5 mg/kg prior to treatment.

It is very reasonable to do a treatment trial of milbemycin in any dog with reverse sneezing as resolution is prompt after drug administration and it is significantly cheaper than a full nasal investigation.

The Facts

The canine nasal mite *Pneumonyssoides caninum* is reported from many countries (probably worldwide) and inhabits the nasal cavity, nasopharynx and frontal sinus. Little is known about its life-cycle. Clinical signs include sneezing, reverse sneezing, and impaired olfaction. Diagnosis is established by direct observation of the mites in or around the external nares or endoscopically in the nasopharynx or nasal cavity. Follicular hyperplasia in the nasopharynx should arouse suspicion even when mites are not visualized. Flushing the nasal cavity with isoflurane may result in mites migrating caudally into nasopharynx where they can be seen more easily.

Photograph: Courtesy of Dr J Braddock

Nasal mites can be effectively treated with milbemycin, ivermectin or topical selamectin. Milbemycin (0.5-1mg/kg, once weekly, PO for 3 consecutive weeks) appears safe and efficacious; 0.5 mg/kg, once monthly, PO is the dose for heartworm prophylaxis. Topical selamectin, 6-24 mg/kg, every 2 weeks for three treatments is also effective but alopecia may develop at the higher doses; 6-12 mg/kg, monthly, topically is the dose used for heartworm prophylaxis. Ivermectin has been effective at various doses (200-400 µg/kg, SC or PO) and dosing frequencies (single dose or multiple doses at various intervals) but all doses have been higher than the licensed dose and these doses can have serious adverse effects in ivermectin-sensitive dogs such as collies. The ivermectin dosage for heartworm prophylaxis is much lower (6 µg/kg PO) and unlikely to be effective against nasal mites.

It is advisable to supply a single anti-inflammatory dose of prednisolone (0.5 mg/kg PO) with the first treatment, regardless of drug choice, to be used either prophylactically or if signs worsen in severity after treatment. The transient increase in severity of signs that occurs in some dogs is presumed to be due to a host reaction to the dead and dying mites. While this reaction is only transient, it can be distressing to both dogs and owners.

And one more from Jody Braddock

For a story that tops all these stories, Dr Jody Braddock provided me with this one:-

An 11-y-o working Border Collie from Alice Springs had epistaxis on 3 occasions, snoring, vigorous reverse sneezing at times, ulcerative lesions on the nasal planum and nares (see photographs below) and serous nasal discharge. The owners knew it would be neoplasia and the dog had been really distressed but they flew her down 'just to be sure before they killed her!' What happened next was unprecedented: on inserting the scope Jody actually SAW the mites skating around on the nasal mucosa (see mite photo above)! 'Never in my wildest dreams did I think that was what I was looking for.' So, for the cost of jetting the dog down to Sydney unaccompanied and referral, she was completely fixed with a couple of Interceptor® tablets. The owners were just overjoyed. Although a working dog, she was still much loved and had given 11 years of loyal service.

Jody commented that she believes nasal mites occur in dogs in Sydney, especially from the Eastern Suburbs, as some dogs with consistent signs do resolve with a milbemycin treatment trial prior to further investigation. ▶

Authors' views are not necessarily those of the CVE
Back to TOC page

Invited Comment courtesy of:
Aine Seavers
Oak Flats Vet Clinic
58A Central Avenue
Oak Flats NSW 2529
E. reception@oakflatsvet.com.au

THANK YOU THANK YOU THANK YOU CVE & SUE FOSTER & JODY BRADDOCK FOR PUBLISHING THIS ARTICLE!

Nasal Mite Reverse Sneezing Complex is **not uncommon nor rare** especially along the Eastern Seaboard – this **condition is uncommonly diagnosed** – a huge difference!

I have been a many-decades-long believer of any reverse sneezing being first and foremost a milbemycin deficiency so this article is sweet music to my ears and will **revolutionise** the treatment and **reduce** the suffering of so many pets which spend years with pharyngeal inflammation and distress.

Additional to reverse sneezing sign, the mites can cause nasal pruritus, serous discharge, epistaxis, hyposmia and more rarely excessive lacrimation, orbital cellulitis and abscessation.

I use Milbemax® as my source of milbemycin. Be a bit cautious using milbemycin as so acutely microfilarcidal: run a heartworm blood test in any dog not on any heartworm preventative. However, in my area most dogs on heartworm preventative so not such an issue.

I am not a lover of Revolution® (selamectin) in a dog with a haired coat as the active ingredient is impeded by hair so it is my second choice depending on heartworm status and pelt type.

I do give a shot of Dexason® IV if concerned about how much phalangeal irritation is present pre miticidal use but prednisolone would be fine as well. However, I have treated many with no premed so was concerned to hear of Sue's experience. I will err on the side of caution now and run them all on a premed regardless.

The mites can survive for 19 days in a cool, humid environment so direct nose-to-nose contact is not always needed to infest pets. Greyhounds can be a way of moving the mites around tracks and post codes – Dapto here is a hot bed of cases for me and there is a big track next to a large puppy and dog training run so perfect conditions.

On inserting the scope, Jody actually SAW the mites skating around on the nasal mucosa. Adult mites are oval, pale yellow 1-1.5 mm in length (2x-3x otodectes) and all legs on the anterior half of the body.

Retrograde nasal flushing as described by Marks and others (1994) appears to be more effective in identifying mites than anteriograde flushing. An antibody (Ab) test has been developed but is not used widely in endemic countries because of the high seroprevalence. A circulating eosinophilia (>2.2 X 10^9 eosinophils/litre) may also be noted in approximately 28% of dogs.

My Sydney Uni story – late 1990s – I was in the Theatre there observing an operation when all hell broke loose at the next table where a greyhound was being scoped whilst on maintenance halothane (which really annoys the mites even more than isoflurane). The surgeon jumped up back up from his chair exclaiming 'It has *Sarcoptes mites* in its nose!' I suggested it was perhaps instead nasal mites *P. caninum* – to not much belief initially…When I suggested their presence had not been written up in Australia the room got all excited, then the surgeon slowly lifted his head and said 'Oh no, I've SQUASHED IT!'.

Depression all around.

Invited Comment courtesy of:
Jan Slapeta MVDr PhD GradCertEd (Higher Ed)
Associate Professor in Veterinary Parasitology
Laboratory of Veterinary Parasitology, Faculty of Vet Science
The University of Sydney
T. +61 2 9351 2025 F. +61 2 9351 7348

Co-Editor-in-Chief *Veterinary Parasitology*

Parasitological enigmas are still out there to be solved. No doubt, the canine nasal mite *Pneumonyssoides caninum* (syn. *Pneumonyssus caninum*) is one of them. The mite was described as new in 1940 from a euthanized dog in the US suffering from urogenital problems. On post mortem and after splitting the head the pathologist noted a large number of mites. The discovery was assisted by a keen veterinary student present at the necropsy that took a couple of these mites and brought them to a parasitologist (Chandler & Ruhe, 1940). The rest is history!

The mite is not unknown to Australia; the first report is by an owner of a healthy greyhound reporting 'small white insects crawling from the animal's nostrils at night, whilst the bitch was sleeping' and later reported in a letter to the *AVJ* editor by my predecessor at the University of Sydney (Gordon & Keep, 1951). The Sydney report revealed only larval stages that at that time prevented definitive identification. The adult mites were soon reported from Queensland (Olds, 1953). The mite is around and about.

The frequency and clinical picture is yet to be understood. Passionate observer and inquisitive veterinarian is the right marriage, as documented in the above case series. Finding them is tough (don't underestimate the mites – they move fast), curing is then quite easy.

For parasitologists the parasite is an enigma from one more reason – no-one has seen them lay eggs or seen the eggs themselves! How do they multiply and more importantly do they multiply outside the dog's nose and sinuses? Over a decade ago, a Swedish group attempted to confirm the transmission from dog-to-dog, yet the outcome is somewhat ambiguous (Gunnarsson *et al.* 1998). They transplanted mites from one dog to a series of experimental dogs. At post mortem they recovered the mites, hence confirming *P. caninum* mites can be transplanted and live in nostrils, but they could not recover more mites that they transplanted. Did the mites multiply in dogs? Well, the jury is out there; however, a report of 250 mites from a naturally infected dog is a good reason to think so. Yet, I believe, we are missing a vital ingredient in this story and *P. caninum* is still outsmarting us. Thank you for this case series, thank you for your inquiry and demonstrating how much clinical practice has to offer.

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View Aine's dog 'reverse sneezing' Part 1 in our ebook

View Aine's dog 'reverse sneezing' Part 2 in our ebook

Case Report: Apocrine ductular carcinoma on the neck of a domestic shorthair cat

C&T No. 5342

Dr Jim Euclid BVSc
Catlovers Vet Clinic
18 Overport Rd, Frankston VIC 3199, Australia
T. +61-3-9769-6999 F. +61-3-9769-6699
www.felipedia.org

Authors: James M. Euclid, BVSc(Hons) BVBiol PhD, Rebbecca A Wilcox BVSc (Hons), BAn Sci., Carl J Muhlnickel BVSc. MACVSc , Judith Nimmo, BSc, BVSc(Hons), DipPath, MSc, Dip ACVP, PhD, MRCVS

Keywords: Apocrine ductular carcinoma, feline, neoplasia, pathology

Introduction

Apocrine adenocarcinoma comprises a group of rare primary cutaneous carcinomas, and has been reported rarely in dogs and cats, usually in aged animals[1]. There are a variety of histological patterns in these tumours depending on whether the tumour has arisen from the secretory or ductular portion of the gland. Ductular carcinomas have a characteristic double layer structure associated with their derivation from or differentiation toward apocrine ducts[2]. Ductal carcinoma have been reported as occurring from glandular tissue in the skin[3], eyelids[4], lips[5], mammary glands[6] and anal sacs of cats[7,8]. Apocrine ductular carcinomas were not named as a specific entity in dogs and cats before 1992 and hence identification and reports of this specific tumour subgroup is quite limited in the veterinary literature.

Case Study

A 13-year-old male neutered domestic short-hair cat presented with a vague history of an 'abscess' associated with an inverted, ulcerated and circumscribed lesion (1 cm diameter) on the mid-dorsal neck region (Figure 1).

The cat had been treated by a previous veterinarian with a two-week course of broad-spectrum oral antibiotics (amoxycillin-clavulanate). Temporary resolution of clinical signs, primarily pruritus and healing of the ulceration, was evident for about one week but relapsed at the conclusion of antimicrobial therapy. A delay of some weeks occurred prior to the cat being presented for a second opinion.

Initial examination of the lesion revealed a pruritic circular ulcerated lesion of approximately 2 cm diameter with self-inflicted excoriations at the margins of the wound.

The cat was FIV and FeLV negative serologically and had received annual core vaccinations (F3[9]) only. The cat was microchipped and had been fed a commercial diet all its life.

Regional lymph nodes were palpably normal. No other clinical signs referrable to the skin lesion were obvious. The cat was later admitted for surgical biopsy.

Anaesthesia was induced with 1.0 mg acetylpromazine and 0.03 mg atropine subcutaneously, followed 20 minutes later with 10mg alfaxan intravenously. A 3.5 mm endotracheal tube was placed and anaesthesia was maintained with 2% isoflourane / 1L oxygen per minute using a non-re-breathing circuit. Lactated Ringers' solution was administered at 10 mL/kg/hr throughout the surgery.

A sterile swab was inserted into the lesion and rotated within the subcutaneous tissue to obtain a sample for microbiological assessment.

The lesion was excised using a wide, elliptical incision and 1 cm margins of the surrounding skin. Routine closure of the skin deficit was performed. The cat was given 0.5cc Cefovecin11 subcutaneously prior to discharge. The tissue biopsy and sterile swab were submitted for laboratory analysis. The excised mass was placed in formalin and processed routinely for histological evaluation.

Laboratory analysis revealed numerous leukocytes and epithelial cells present on the microbial swab. Microbiological report revealed a mixed growth of Gram-negative and Gram-positive bacilli and cocci. Ziehl-Neelsen acid-fast staining failed to reveal the presence of any mycobacteria.

The biopsy specimen was covered by a haired skin that was ulcerated over the surface of an underlying rather poorly circumscribed mass composed of trabeculae and islands composed of variably sized acini, trabeculae and solid nests of pleomorphic epithelial cells surrounded by a profuse reactive fibrosis (desmoplasia). Small nests of these epithelial cells were invading the surrounding stroma on the periphery of the mass. The mitotic index was high (> 4 per hpf). Some of the larger tumour islands had necrotic cystic centres with neoplastic cells forming an outer rim. In some, the islands showed evidence of melanisation of the tumour cells and released clumps of melanin free in the necrotic centres. Other islands were better differentiated and consisted of tubules formed of epithelial cells with a more basaloid appearance arranged in tubules and winding ribbons often with a double layered structure. There was also a prominent lymphoplasmacytic reaction around the advancing edge of the tumour and within the connective tissue between the tumour islands. The tumour appeared to have been fully excised surgically.

Discussion

Since so few apocrine ductular carcinomas have been reported in cats, their biological behaviour is not well established. However, they are most commonly found on the head, legs and abdomen in older cats (11 – 13 years of age)[12]. They are usually locally invasive and tumours also have high metastatic potential, commonly invading regional lymphatics neighbouring these tumours[13].

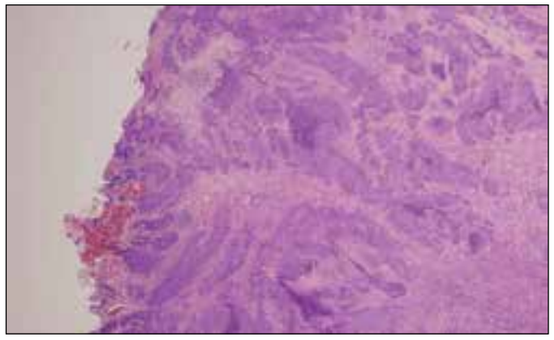
A diagnosis of feline apocrine ductular carcinoma was made in an aged male neutered cat on the basis of the characteristic histopathology particularly the double layer structure of the tubular and ductular structures within the tumour, although the tumour varied from area to area in its degree of differentiation and morphology with solid, tubular and acinar areas. These malignant neoplasms are considered rare in cats and little biological data has been accumulated for the behaviour of these neoplasms in domestic animal species, but they appear to behave more aggressively in cats than in dogs, and are usually locally infiltrative into the adjacent dermis, subcutis and muscle as well as having high metastatic potential.

Apocrine carcinomas in general have a high potential for metastasis to local lymph nodes and visceral organs such as ▶

the lungs. In cats, the solid-cystic apocrine adenocarcinoma subgroup is the most likely to metastasise although the ductular type is also considered to have high metastatic potential. In most cases, regional lymph nodes draining the primary tumour site are likely to be first affected and should be examined by cytology or histopathology to check for metastasis.

Clinically, the differential diagnoses to consider in such cases of focal epidermal or subcuticular ulceration include mast cell tumours, Mycobacterial infections, FeLV-associated fibrosarcoma, and cutaneous lymphosarcoma. Histopathological examination of a fixed biopsy is necessary for definitive diagnosis and should be considered in any lesion that does not respond to initial antibiotic treatment or is suspected to involve neoplasia.

Figure 1. Clinical appearance of lesion prior to surgical removal.



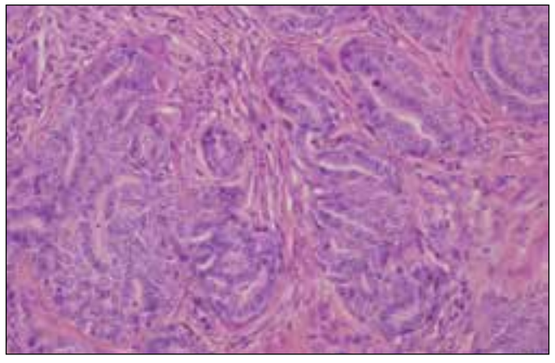


Figure 2. Apocrine ductular carcinoma in the cat (low magnification).

Figure 3. Apocrine ductular carcinoma (high magnification).

Post-script: Jim reports that there has been no recurrence in this case for the last 2 years.

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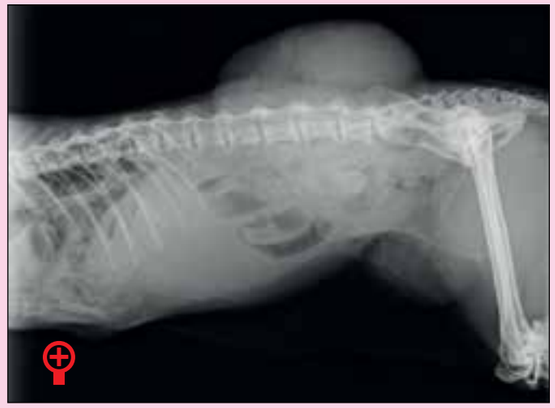
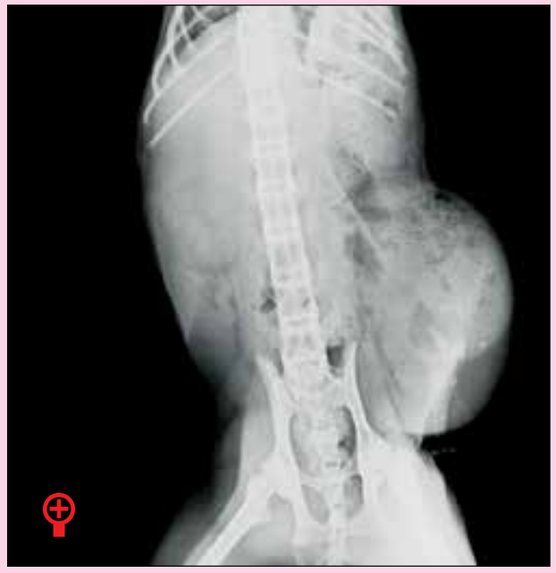
Answer to C&T No. 5290 Swelling on the flank of a cat

C&T No. 5343

Suzanne Pears
E. suzannepears@hotmail.com





Rollover to view enlarged images

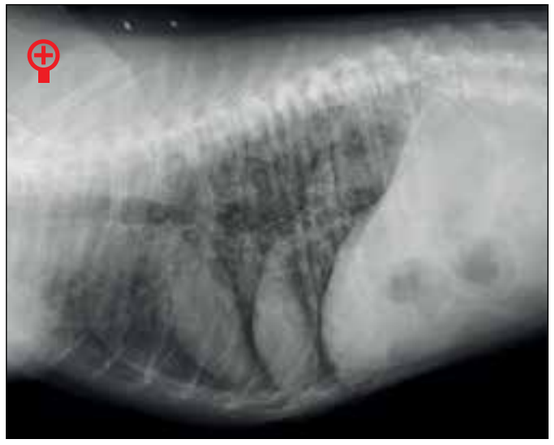
Answer: The cat had a diaphragmatic hernia (made up of stomach and intestines) and a lateral abdominal wall hernia which was made up of kidney, intestines and omentum. Cause unknown but trauma suspected. Diagnosis confirmed with ultrasound and post-mortem.

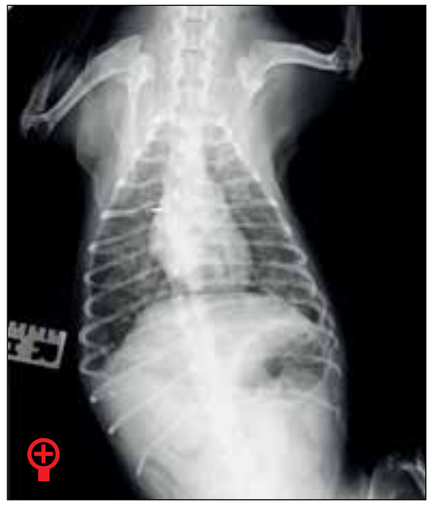
What's YOUR Diagnosis?

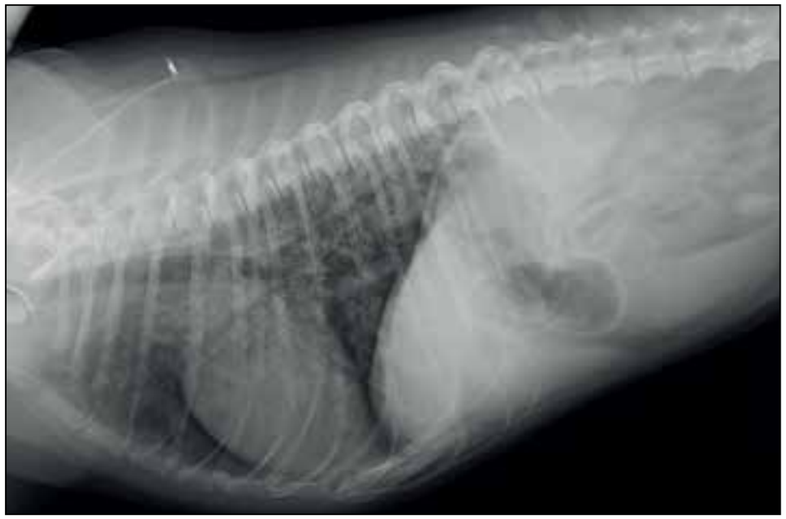
C&T No. 5344

Suzanne Pears
Maroubra Veterinary Hospital
88 Bunnerong Road
Pagewood NSW 2035
T. (02) 9344 8722
E. suzannepears@hotmail.com

A 6-year-old male Fox Terrier Cross presented 3 weeks ago for bilateral clear nasal discharge and noisy breathing. Harsh lung sounds and a small amount of weight loss (T 39.4˚C) were the only notable findings on examination. The discharge resolved following an Amoxyclav and Baytril course (still on these), but the noisy breathing persisted. X-rays showed a diffuse, hazy mixed pattern (with a prominent interstitial component) and lung FNA suggested lymphoid hyperplasia, and no indication of neoplastic disease. Submandibular lymph nodes were enlarged. Haematology was normal and only mild elevation in AST, ALKP and ALT. The rest of biochemistry was normal and heartworm negative. Chest and abdominal ultrasound were normal. ▶







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What's YOUR cytological diagnosis?

C&T No. 5345

George Reppas
Registered Specialist Veterinary Pathologist, Vetnostics
60 Waterloo Road, North Ryde NSW 2113
T. (02) 9005 7012 F. (02)9005 7950
E. george.reppas@vetnostics.com.au

George is a Registered Specialist Veterinary Pathologist working at Vetnostics in North Ryde NSW and is a Fellow in Veterinary Clinical Pathology of the Australian and New Zealand College of Veterinary Scientists as well as a Diplomate of the European College of Veterinary Pathologists. He has written and co-authored articles in many scientific journals and veterinary texts and has served as an examiner of the ANZCVS Fellowship examination in Veterinary Clinical Pathology. Recently he has been collaboratively involved in developing a selective range of advanced diagnostic techniques (Immunocytochemistry & Infectious Diseases PCRs) specifically adapted to veterinary cytology which are available through Vetnostics.

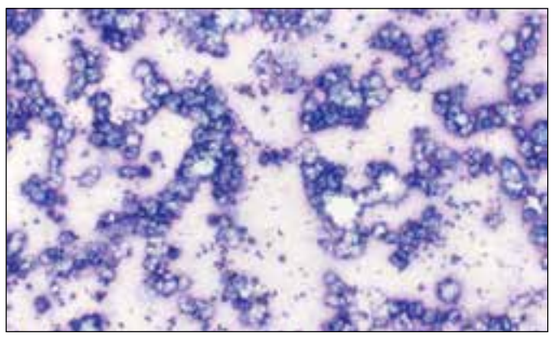


Figure 1. Diff-Quik photomicrograph of FNA smear - low power (x 20)

A 14yo FN DSH Cat presented with multiple firm subcutaneous swellings about the head and swollen/thickened distal limbs and feet. A smear made from the FNA of one of the skin lumps on the cat was stained with Diff-Quik (Figures 1-3 below).

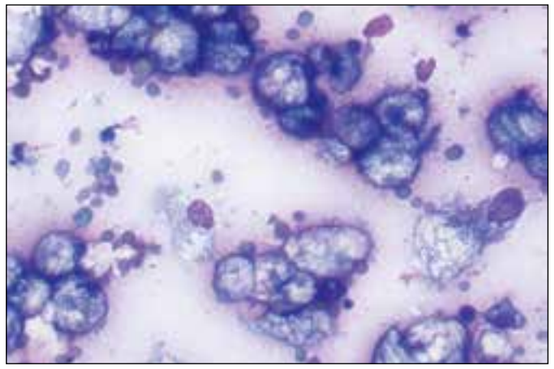
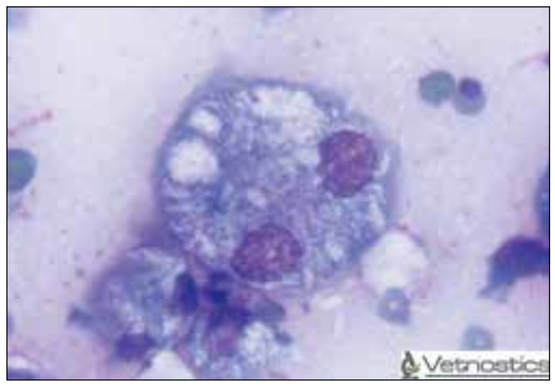


Figure 2. Diff-Quik photomicrograph of FNA smear - low power (x 40)



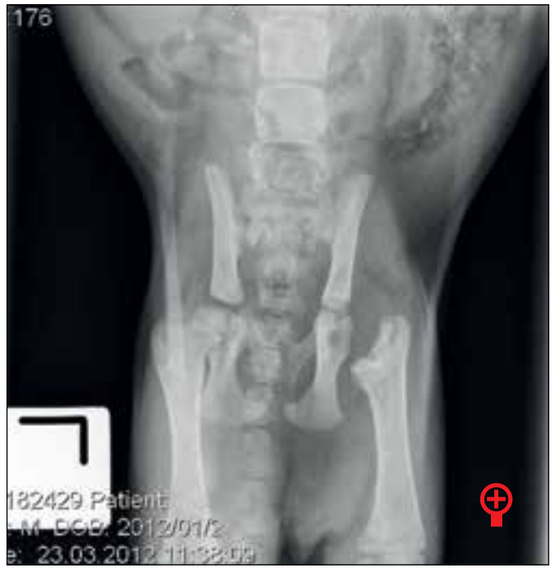
Figures 3. Diff-Quik photomicrograph of FNA smear - high power under oil immersion (x 100).

Email your answer to: **elisabeth.churchward@sydney.edu.au**
Answer in Dec 2013 Issue 273

What's YOUR Diagnosis?

C&T No. 5346

Cathy Birch
North Nowra Veterinary Hospital
205 Illaroo Rd
North Nowra NSW 2540
T. 02 4423 1688 F. 02 4423 1800
E. admin@northnowravet.com.au



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Replies to: We Need Your Help - Has anyone seen anything like this before? (Jim Euclid, C&T 5280, March 2013 Issue 270)

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C&T No. 5347

Reply No. 1
Maribeth Murphy
Old Sale Road Veterinary Services
E. mmurphy1@live.com.au

Any chance they could have one of the mutations causing an Ehlers-Danlos type syndrome?

Reply No. 2
Michael Wallace
Caring Vets
E. contact@caringvets.com.au

Before even looking up the computer or noting that your kittens were Bengals, your case reminded me of a Bengal breeder for whom I delivered a litter on 4 July 2007. Of the 5 kittens born, 1 had an extreme cleft palate (if in fact if was formed at all) and a deficit of almost the entire area of the dorsal cranium i.e. skull and skin. The kitten was euthanased. I have not seen this lesion since in any cat breed. The breeder lived in Horning Sea Park (Western Sydney). The queen is now 7 years and 4 months old.

***Note:** Thanks to Maribeth and Michael for contributing their replies. At this stage we can't confirm a diagnosis but both respondents are entitled to a CVE proceedings of their choice from www.vebtookshop.com*

Why I want to be a veterinary specialist & Why I support the CVE

Amy (nee Aspley-Davis) Lam is an Australian trained registrar in Internal Medicine currently working at Willows in the UK. She has worked in private practice in Melbourne and Canberra (with her Dad), undertaken a rotating internship at the University of Sydney, and a residency at the Small Animal Specialist Hospital (SASH) in Sydney. She is currently working towards her fellowships, and loves medicine of both dogs and cats.

Amy Lam BVSc (Hons I) GradCertVetStud MACVS (Small Animal Medicine)
Registrar in Small Animal Medicine
Willows Veterinary Referrals
Highlands Road, Shirley
West Midlands, UK
E. amy.lam@willows.uk.net

As with many of us, I have wanted to be a vet since I was in kindergarten. I loved going to work with my Dad and seeing him help pets, and owners alike. He was and still is an incredible inspiration.

Dad owned one of Canberra's largest veterinary practices, employing 3 to 6 other vets at the time. He was passionate about quality veterinary care and looking after his staff. Dad wanted to ensure that he continued to attract the best vets to Canberra. He offered CPD courses through the PGF (now CVE) to entice vets to come to his practice.

He attracted some amazing vets to work with him. Many of these vets have gone on to do membership exams of the Australian College or specialty training/PhDs (e.g. Richard Gowan who runs the Cat Clinic in Melbourne, Naomi Hansen who did a residency in emergency and critical care at the University of Pennsylvania, Natalie Courtman who is preparing for American College Boards in Pathology working for IDEXX, Claire Sharp Associate Professor in Emergency and Critical Care at Tufts University, Jacqui Phillips who is the Director of Medical Research at Macquarie University after completing a PhD, Mike Serdy Diplomat of the American College of Veterinary Surgeons working in San Deigo). Some of them have gone on to run or own their own practices and become exceptional veterinary practitioners (e.g. Michael Hayward – Gungahlin Veterinary Hospital, Ben Black – Tuggeranong Veterinary Hospital, Lan Tran – Guildford Veterinary Hospital, Tony Wilson – Gungahlin Veterinary Hospital, Alison Taylor – Kippax Veterinary Hospital).

All of these vets did CVE Distance Education whilst working. They were inspired to continue their professional development, and contributed straight back to the practice.

Throughout high school and college I continued to work in Dad's practice. I worked alongside these amazing vets. I was so delighted to achieve that incredibly high university entrance score to get into Veterinary Science. My first day at vet school was incredible, but I had already realised I wanted more. During vet school, I started to work at a veterinary specialist practice on weekends (ARH) and did extra time at as a student intern at the teaching hospital during my holidays.

When I graduated, I had a strong interest for internal medicine. It was obvious to me, that I wouldn't be satisfied until I became a specialist. I started work in a large small animal practice.

In my first year out, **I entered Jill Maddison's Problem Solving Internal Medicine Short - Course (her year-long course was booked out for 2 years!), and in 2nd year, I enrolled in Internal Medicine Keys to Understanding with Darren Merrett and Boyd Jones.** I was so intrigued by internal medicine that I decided to work on becoming an internal medicine specialist. This started with a rotating internship at the University of Sydney, then a residency at the Small Animal Specialist Hospital. I spent externships at Colorado State University, and the Royal Veterinary College. I am now studying for Fellowships, and hoping that one day, I can contribute back to the association that gave me inspiration and the foundations I use every day to become an internal medicine specialist – the Centre for Veterinary Education.

Perspective 98

How to best monitor diabetes in dogs

Linda Fleeman
Animal Diabetes Australia

Linda Fleeman BVSc PhD MACVSc
Animal Diabetes Australia
at Boronia Veterinary Clinic Phone: +613 9762 3177
at Rowville Veterinary Clinic Phone: +613 9763 1799
at Lort Smith Animal Hospital Phone: +613 9328 3021
E.: L.Fleeman@AnimalDiabetesAustralia.com.au
www.AnimalDiabetesAustralia.com.au/

Monitoring clinical signs and response to therapy

Establishing a practical routine for the dog's owner

Many owners of diabetic dogs welcome the opportunity to monitor their pet's response to therapy, although compliance can be very variable. Compliance is markedly improved if there is close rapport between the owner and the clinician managing the case and appropriate individualisation of the dog's therapeutic and monitoring regimen. The veterinary clinician must invest time to educate the owner about canine diabetes and its management, as well as to provide support and guidance while the owner becomes accustomed to the treatment and monitoring procedures and establishes a practical routine.

The primary aim of therapy in diabetic dogs is to achieve resolution of clinical signs, so it is important to regularly monitor signs such as the volume of water drunk and body weight. If the dog drinks more than 60 mL/kg/day or is lethargic or losing weight, then adjustment of the dog's insulin dose is probably required. Owners of diabetic dogs should be encouraged to keep detailed records of their dog's progress.

1. Appetite, general demeanour and behaviour should be recorded every day.
2. Insulin dose and meal composition should be recorded twice each day.
3. Water intake over 24 hours should be measured at least once each week. If there is more than one pet drinking from the same water bowl, it is useful to measure the volume of water drunk by all the animals. The diabetic dog typically is the reason for most of the variation in water drunk in multi-pet households.
4. Urine glucose and ketones should be measured at least once each week. Diabetic dogs will often need a bit of gentle encouragement to become accustomed to their owner approaching them when they are urinating. However, most will come to readily accept urine collection. Persistent negative glucosuria might indicate an increased risk of hypoglycaemia. If the urine glucose result is consistently 'negative' for 2 weeks, then it is often advisable to decrease the dog's insulin dose. Ketonuria usually indicates illness or very poor diabetic control. **It is necessary to counsel owners that no importance can be attributed to the amount of glucose recorded on the dipstick – that is, there is no important difference between 1+ and 4+ results; it is simply a question of positive or negative glucose. Persistent negative glucosuria identifies periods when the exogenous insulin dose should be decreased.**
5. Body weight should ideally be recorded once each week. It is preferable to use the same scale each time. Scales designed for weighing adult humans are not suitable for small and medium sized dogs. Scales designed for babies are a much better option.

In addition to appraisal of the owner's insulin dosing technique, compliance of both the owner and the patient with the feeding recommendations must be routinely evaluated to ensure appropriate timing and consistency of the meals. An approach that is often successful following each reappraisal is to recommend the 'dose' of food along with the dose of insulin to educate the owner of the importance of carefully weighing or measuring the food for their dog in the same manner that they carefully prepare the dose of insulin.

Measurement of long-term glycaemia: fructosamine

Measurement of fructosamine is an additional way of assessing glycaemic control in diabetic dogs, although **monitoring clinical signs is usually sufficient**. Plasma fructosamine provides approximate measures of average blood glucose concentration over the preceding 2-4 weeks and thus is an indicator of longer-term diabetic control. Measurement of fructosamine is most useful when there is little available information about recent clinical signs of the diabetic dog, or when results of serial blood glucose measurements do not match with the reported clinical signs.

Comparison of serial measurements of fructosamine in an individual diabetic dog allows evaluation of glycaemic response to management changes. A major limitation is that it represents average glycaemia and gives no information about the degree of fluctuation around that average. Therefore it does not indicate the risk of hypoglycaemia on the current insulin regimen. It is recommended that additional monitoring tools be used when appraising insulin dose, such as changes in the dog's water intake, body weight, and urine glucose concentrations.

Monitoring blood glucose concentrations at home

Some owners are interested in performing blood glucose monitoring at home. **Single, sporadic measurements provide little useful clinical information for monitoring glycaemic control**, and serial blood glucose concentration curves that follow the same protocol as those obtained in hospital are required. As with blood glucose curves obtained in hospital, results must be related to the dog's clinical signs; interpretation requires an understanding of the complex interactions involved in glucose homeostasis in diabetic dogs.

A practical approach is to use knowledge of the dog's clinical signs to guide the timing of home-generated blood glucose curves. For example, if there is marked variability of 24 hour water intake, owners can be advised to perform a glucose curve on a day when the dog does not drink much water. This approach would increase the chance of detecting hypoglycaemia. **If clinical signs consistent with hypoglycaemia occur at home, owners accustomed to measuring their dog's blood glucose concentration can quickly confirm whether or not hypoglycaemia is present and so facilitate timely treatment.**

Home-generated serial blood glucose curves are as reliable as hospital-generated curves and have many of the same ▶

limitations. There is considerable day-to-day variability in blood glucose measurements in diabetic dogs. The major advantages of home-monitoring of blood glucose concentration are that measurements can be easily obtained at any time and can be repeated if equivocal results are obtained, the cost is minimal compared with a veterinary visit, and the effect of hospitalisation on appetite and stress hyperglycaemia are avoided.

Samples can be obtained either from the marginal vein of the lateral pinna, by collection of capillary blood for example from the oral buccal mucosa, the medial pinna, or the metatarsal pad, or by direct venepuncture. Once the owner is familiar with the technique, they usually need some practice at sample collection with the dog at home before they develop sufficient skill to generate a serial blood glucose curve.

Owners should be encouraged to purchase a **veterinary blood glucose meter** because human glucose meters give significantly lower results in dogs when compared with both veterinary meters and laboratory reference methods. Veterinary glucose meters currently available in Australia are the **AlphaTRAK meter** (VetQuip Pty Limited) and the **g-Pet meter** (Vepalabs Veterinary Pathology).

Micromanagement with frequent adjustment of insulin dose must be avoided and owners who choose this method of monitoring often need to be advised against over-zealous blood glucose measurement and interpreting the results themselves.

Continuous interstitial glucose concentration monitoring systems

Continuous interstitial glucose concentration monitoring systems such as the Guardian REAL-Time Continuous Glucose Monitoring System (Medtronic) can also be used in the home environment or in hospitalised patients to monitor glycaemia. **Important advantages of continuous monitoring systems over intermittent measurement of blood glucose are that they facilitate detection of brief periods of hypoglycaemia and provide information when the dog is sleeping at night.** One limitation is that they must be calibrated with blood glucose concentration, so there is still a requirement for some blood sampling during monitoring.

Serial blood glucose concentration curves

Serial blood glucose curves are one of the diagnostic tools available to assist with case assessment of diabetic dogs. **The recording of an 'ideal' blood glucose curve is NOT a goal of therapy. Their role is to help answer specific clinical questions when appraising the response to treatment and so should not be scheduled unless clinical problems have first been identified.** A common error is to perform a 'routine' serial blood glucose curve in a diabetic dog that is 'going well'.

Serial blood glucose concentration curves are most useful in cases where the clinical history is poor. However, it is important to recognise that **they are an unreliable clinical tool for evaluation of insulin dose in diabetic dogs because there is a large amount of day-to-day variability in results.** It is advisable to always consider additional indicators of glycaemic control, such as changes in the dog's water intake, body weight, and urine glucose concentrations, when appraising insulin dose.

The large day-to-day variability of the curves and the serious sequelae that might result from insulin overdose justify the need for a conservative approach to dosage recommendation. For example, when assessment of clinical signs indicates that there is good glycaemic control, dosage increases should typically be limited to increments of 0.5 or 1.0 U of insulin.

The principal reason for performing a serial blood glucose curve in a diabetic dog is to evaluate whether the insulin dose can be increased without risk of inducing hypoglycaemia. This is particularly important whenever recommending insulin doses >1 U/kg. Although assessment of clinical signs is valuable for identifying dogs with poor glycaemic control, it is often not effective for identifying dogs at risk of clinical hypoglycaemia.

The standard protocol for generating a serial blood glucose curve is to admit the dog into the hospital before administration of the morning insulin injection and obtain a baseline blood glucose reading. The usual insulin dose and meal is then given. Blood glucose measurements are then obtained every 2 hours until the next insulin injection is due. If the owner administers the morning insulin injection, it provides an opportunity to review their injection technique and correct any problems. **If the dog refuses to eat, subsequent blood glucose values are likely to be lower than if the dog eats normally. In this situation, it is probably best to cancel the serial blood glucose curve on that day and re-schedule it.** The protocol can be modified on the next occasion to allow the owner to take the dog home or to a less stressful environment after the baseline blood glucose reading is obtained so that the usual meal can be fed there. The dog can then be returned to the hospital before the next blood glucose reading is due. Alternatively, the morning pre-insulin blood glucose reading can be omitted from the serial blood glucose curve and the dog can be brought to the hospital after the morning insulin injection and meal have been given at home.

Guidelines for evaluation of serial blood glucose curves in diabetic dogs are primarily based on the nadir, or lowest blood glucose reading obtained, and the two pre-insulin blood glucose values (Table 1). The first pre-insulin blood glucose value is the reading obtained before the morning insulin injection and the second is the last blood glucose taken when the next insulin injection is due, usually 12 hours later. **The nadir gives an indication of the maximal blood glucose response to the current dose of insulin and can occur at any point in the blood glucose curve, including just prior to insulin injections.** The time to nadir influences recommendations for dose but can vary considerably, even in the same dog over consecutive visits. The pre-insulin blood glucose values provide an indication of the likely blood glucose level when the dog is due for an insulin injection at home. This is important because hypoglycaemia is more likely if the effects of the previous insulin injection overlap with the next insulin dose, producing an additive effect.

Severe episodes of hypoglycaemia following insulin overdose can result in compensatory hyperglycaemia, also termed Somogyi phenomenon. This hyperglycaemia sometimes appears to persist for several days, and serial blood glucose assessment performed shortly after an episode of hypoglycaemia may result in a curve similar to that obtained following insulin under-dosing. **If there is ever any doubt about the interpretation of a serial blood glucose curve, it is always safest to err on the side of caution and decrease the dog's insulin dose.**

Problem-solving difficult cases

Insulin resistance

The majority of uncomplicated diabetic dogs are stabilised on an insulin dose of approximately 0.5-1.0 U/kg and few require doses greater than 1.5 U/kg. **Insulin resistance can be defined as poor diabetic control at insulin doses exceeding 1.5 U/kg.**

The 3 major differential diagnoses for insulin resistance are:-

- Error in insulin handling or administration
- Concurrent disease or drug therapy
- Compensatory hyperglycaemia secondary to insulin overdose (Somogyi phenomenon).

1. Error in insulin handling or administration

Insulin can become inactivated if exposed to temperatures >30º C or light for prolonged periods. An expedient method of ruling out the possibility of inactivated insulin when investigating insulin resistance is to change to a new vial of insulin. Insulin suspensions must be thoroughly mixed prior to administration or doses might vary greatly.

Although experienced owners of diabetic dogs rarely report difficulty with administration of insulin to their pet, it is important to review their injection technique for errors whenever insulin resistance is investigated. A wide range of insulin syringes are available and inadvertently changing to a different type of syringe can lead to dosing errors. Dosing errors are less frequent with insulin dosing pens than with needles and syringes. However, insulin dosing pens must be primed prior to administration of each dose to ensure there is no air in the system. It is also important to check that the dosing dial has returned to the 'zero' position after each dose.

2. Concurrent disease or drug therapy

Concurrent disease or drug therapy causing insulin resistance may be suspected based on history and physical examination findings and can be further investigated by performing haematology, serum biochemistry, urinalysis, and microbial culture and susceptibility testing of the urine. Almost any concurrent disease including dental disease might cause insulin resistance that affects diabetic control. Once the concurrent disease state has resolved, insulin sensitivity can be expected to improve and there will be increased risk of hypoglycaemia unless the insulin dose is decreased.

Hyperadrenocorticism and hypothyroidism are two important causes of insulin resistance in diabetic dogs. Both can present a diagnostic challenge in a diabetic dog. Systemic and topical corticosteroids are the drugs most commonly associated with insulin resistance. Obesity causes insulin resistance and improvement in insulin sensitivity occurs with weight loss.

3. Compensatory hyperglycaemia secondary to insulin overdose (Somogyi phenomenon)

Compensatory hyperglycaemia secondary to insulin overdose (Somogyi phenomenon) is one of the most common causes of insulin resistance in diabetic dogs. There is typically a period of good glycaemic control that is followed by deteriorating glycaemic control despite increasing insulin doses. The period of good glycaemic control may be very brief and is sometimes missed, especially if dose adjustment is based only on results of serial blood glucose concentration curves without careful consideration of the dog's clinical signs.

In insulin-treated diabetic people, hypoglycaemic events occur more frequently during the night than during the day and the same is likely true for dogs. Therefore insulin-induced hypoglycaemia is likely to be missed with day-time monitoring. Continuous subcutaneous glucose monitors are a useful diagnostic aid for detecting night time hypoglycaemia. **Compensatory hyperglycaemia often appears to persist for several days following insulin-induced hypoglycaemia.**

For diabetic dogs with poor glycaemic control that are receiving insulin doses greater than 1 U/kg where administration/dosing errors and concurrent disease/drugs have been ruled out, it is recommended that the insulin dose be decreased to 0.5 U/kg and the response to this change monitored. If insulin resistance was due to compensatory hyperglycaemia secondary to insulin overdose, there is typically marked clinical improvement within 1-2 weeks. If there is another cause of insulin resistance, clinical signs typically become much worse within a few days and the previous insulin dose can be resumed and the investigation for another cause continued.

Short- or long-duration of insulin action

Short- or long-duration of insulin action may be suspected based on results of serial blood glucose testing. It is important to recognise that there is great day-to-day variability of serial blood glucose concentration curves and apparent duration of action varies considerably from day to day.

- A diagnosis of short-duration of insulin action must be supported by compatible clinical signs such as polydipsia that repeatedly occurs for only a few hours before an insulin injection is due. Short-duration of insulin action must also be differentiated from compensatory hyperglycaemia secondary to insulin-induced hypoglycaemia.
- Short-duration of insulin action can be associated with insulin resistance and investigation for concurrent disease is indicated. If short-duration of insulin action is confirmed and insulin overdose and concurrent disease have been ruled out, then the same insulin preparation may be administered every 8-hours or a different insulin preparation with an expected longer duration may be trialled.
- Long-duration of insulin action usually responds to a decrease in the 12-hourly insulin dose. It is not usually necessary to decrease the frequency of insulin administration.

Table 1:
Guidelines for evaluation of serial blood glucose concentration curves in diabetic dogs

The findings of a serial blood glucose curve should always be related to the history, physical findings, and changes in body weight before a final decision is made regarding insulin dose. If a diabetic dog is not lethargic, has a stable body weight, has no ketonuria, is drinking <60 mL/kg/day, and glycaemic control is very good, but an increase or decrease in insulin dosage is suggested by the serial blood glucose curve, then insulin dosage adjustments of no more than 1 U are advised, regardless of the dose the dog is receiving.

Table 1	
If the nadir is <3 mmol/L or the dog has shown clinical signs of hypoglycaemia	Decrease the 12-hourly insulin dose by 50%.
If the nadir is 3-5 mmol/L or if either pre-insulin blood glucose value is <10 mmol/L	Decrease the 12-hourly insulin dose by 20% (rounded down to nearest U of insulin).
If the final pre-insulin blood glucose is <5 mmol/L	The evening insulin injection should be withheld and the dog fed as usual. Decrease the 12-hourly insulin dose by 20% (rounded down to nearest U of insulin), but the new dosing regimen should not be commenced until the following morning. If the final pre-insulin blood glucose is <3 mmol/L, decrease the 12-hourly insulin dose by 50%.
If the nadir is 5-8 mmol/L and both pre-insulin blood glucose values are >10 mmol/L	The dog likely has optimal clinical control and does not require an insulin dosage adjustment.
If the nadir is >8 mmol/L and the pre-insulin blood glucose values are >10 mmol/L	Increase the 12-hourly insulin dose by 20% (rounded down to nearest U of insulin).
If very high blood glucose measurements are recorded in an insulin-treated diabetic dog, for example, values in excess of 30 mmol/L	The possibility of insulin resistance should be considered if insulin dose is > 1 U/kg.

Apology & Correction

Our apology to Linda Fleeman for an error in 'Perspective 96: How to best monitor diabetes in cats' (Issue 271, June 2013, pg 40). Units/kg should have read units/cat – see below:-

High insulin requirement (>1.5 units/kg) or an unexpected increase in insulin requirement

Most diabetic cats require doses of 5 units/cat or less of glargine insulin administered every 12 hours. Occasionally an individual cat will need 6 or 7 units before good glycaemic control is achieved. Insulin doses should always be based on estimated ideal body weight rather than actual body weight in underweight or overweight cats. Once persistent negative glucosuria is achieved and insulin requirement decreases, many cats will go into remission and the remainder will typically require 3 units or less of insulin every 12 hours.

Authors' views are not necessarily those of the CVE
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Critical Care Perspective

Terry King
Veterinary Specialist Services

Terry King BVSc MACVSc
Emergency & Critical Care Medicine
Veterinary Specialist Services

Brisbane Hospital – Cnr Lexington & Logan Rds
Underwood QLD 4119
T. 61 7 3841 7011

Gold Coast – 104 Eastlake Street
CARRARA QLD 4211
T. 61 7 5530 6370
E. tking@vss.net.au

Terry, born and bred in Townsville, North Queensland, received his BVSc from University of Queensland in 1975, and Membership of the Australian College of Veterinary Scientists (now ANZCVSc) in 1996 in Emergency and Critical Care.

Terry believes he has been blessed with 3 major career moves, all in small animal practice – 19 fantastic years in general practice on Brisbane's north side, then 7 years as a medical clinician in UQ's Veterinary Teaching Hospital (a time he wouldn't swap for anything) before being extremely fortunate to join Veterinary Specialist Services in 2002, and this will 'see him out'. Terry is not a specialist, but a partner and internal medicine referral clinician with a special interest in the emergency and critical care patient.

Devoted to the veterinary profession, Terry has strived to repay it and his colleagues for the enjoyment and friendships he's received by serving on various committees and sometimes office bearer on the Brisbane Veterinary Practitioner Branch of AVA, AVA Qld, ASAVA, and the ANZCVSc. He has been an external examiner, guest lecturer and tutor at UQ's School of Veterinary Science as well membership examiner for ANZCVSc.

Awards Terry treasures (straight to the Pool Room) are: AVA Meritorious Service Award (1991), ASAVA Practitioner of the Year (2005) and the appointment as Life Member of the UQ Veterinary Student's Association (2002).

Blessed with being able to work and learn with veterinarians all over this country and the world, Terry is adamant that Australian veterinary care is second to none. Terry is a committed family pet practitioner, loves the animal-person bond, and undertakes to prolong it. While all facets of small animal medicine and surgery have progressed enormously over Terry's nearly 40 years of involvement, he believes care of the critical patient as well as palliation (a multi-disciplinary approach and involving multi-model therapy) of the chronically (and terminally) ill lead our advancement in family pet medicine.

Terry loves reading, writing and sharing good beer and wine with family and friends, or anyone he can find to fill a glass with. He considers himself a champion of the bar-b-que; loves walking his dogs, swimming (slowly), anything to do with horses (especially horse racing), and mowing the lawn with his trusty Victa (his favourite pastime). Did you know that it has been clinically proven that cut grass (freshly mown lawn) releases a chemical called Serenascent that makes people happy and relaxed and prevents mental decline in old age? It works directly on the brain, in particular the amygdala (emotions) and the hippocampus (memory).

As well as the above, on his days off work, Terry cooks dinner, takes out the rubbish and does the household chores, trying to make up for the last 35 years of marital neglect.

Terry King with Aussie icon - the Victa

BROMINISM/BROMISM (BROMIDE TOXICITY) – CAUTIONS WITH DOGS ON BROMIDE AED THERAPY

'Harvey' – a 10-year-old male neutered Australian Terrier

Long term AED (antiepileptic drug) therapy (phenobarbitone and KBr) for grand mal seizure disorder and well-controlled, with serum levels of both mid-range therapeutic levels.

Had been on Hills i/d™ Diet (99mg Sodium per 100kcal ME) exclusively since a severe pancreatitis bout a few years previously.

Diet was changed two months ago to Hills u/d™ Diet (53mg Sodium per 100kcal ME) after cystotomy surgery for removal of calcium oxalate uroliths. His owners recently (3 weeks prior) changed his diet to a home-made low oxalate diet (oatmeal, turkey mince, vegetables) with no added salt.

Two weeks after starting his new diet, 'Harvey' started having episodes of hindleg tremors, head bobbing and weakness. His phenobarbitone (by 100%) and KBr (by 33%) dosages were steadily increased over those 2 weeks, presuming the signs were a recrudescence of his seizure activity.

'Harvey' then presented with clinical signs of profound obtundation, comatosed state, dyspnoeic and hypoxaemic. Chest radiographs showed megaoesophagus with severe aspiration pneumonia; blood profile showed marked neutrophilia with a left shift, severe hyperchloraemia (Cl- 173mmol/L) and subsequent phenobarbitone levels were high (197 umol/L; NR: 65-150) as were KBr (40.4 mmol/L; NR: 8.8-25.0).

'Harvey' died of his respiratory complications.

'Monty' – a 7-year-old male neutered Terrier Cross

AED therapy had consisted of phenobarbitone (high range therapeutic level – 140umol/L Ref range: 65-150 umol/L), KBr (high range therapeutic level – 23.2 mmol/L Ref range 8.8 – 25.0mmo/L) and pregabalin 3mg/kg TID which was recently substituted for levetiracetam due to poor seizure control. Seizure control seemed to be much better with this new regime.

Owners sourced Guide Dog leading raw diet (low Sodium) and changed 'Monty' from his Hill's Science Diet dry food™ (227mg/100g Sodium and 678/100g K).

'Monty' gradually became weaker which the owners initially presumed was due to the addition of pregabalin to his AED regime, however he presented 3 weeks after his new diet was begun, non-ambulatory and obtunded.

Blood profile showed marked hyperchloraemia (>120mmol/L. Ref range: 100-120 mmol/L) and subsequently serum KBr levels were reported at 40.8 mmol/L.

Monty was given 0.9% NaCl IV until he was conscious enough for oral alimentation (3 days), his KBr dose was reduced by 25% and he gradually returned back to normal. His previous diet, Hills Science Diet™ was resumed and he remains well and seizure-free some 6 months later.

Discussion

Bromide can be used as a sole or adjunctive anticonvulsant medication. Bromide causes membrane hyperpolarisation by competing with chloride transport across cell membranes of excitable tissues. Hyperpolarisation of the membrane raises the seizure threshold thus reducing the cellular charges which induce epilepsy/seizures.

When orally administered, bromide is absorbed in the SI (also well absorbed when administered rectally), and is concentrated in the ECF. It has similar concentration and distribution in the ECF as chloride. Bromide will cross the BBB and enter into the brain and CSF and across the placenta to be detected in milk. Bromide is renally excreted, and care should be taken when administering to those patients with renal dysfunction.

Brominism can occur due to reduced chloride ion concentration because of the effects of sodium and chloride on bromides renal excretion. Lowering the sodium content of the diet will reduce the amount of sodium renally excreted, increasing the concentration of serum chloride, and hence the concentration of serum bromide, recalling that bromide will have similar concentration and distribution as chloride in the ECF. In noting this, the opposite can occur if sodium chloride is increased in the diet, there will be increased renal excretion of both sodium chloride and bromide, thereby reducing the ictal threshold and increasing the likelihood of a seizure event.

It is recommended to place animals onto a well balanced diet (preferably with known sodium chloride concentration) at the commencement of their AED therapy. Homemade diets are not recommended due to the inherent likelihood of fluctuating sodium concentrations (despite the chef's best efforts). Should the diet need to be altered, it is recommended to monitor bromide and chloride levels and adjust the bromide dose accordingly (lower dietary sodium = reduced bromide dose; increased sodium = increased bromide dose). Also be aware of medications which can affect sodium chloride levels and the excretion of bromide through the kidneys (e.g. diuretics and cardiac medications). Similarly note that KBr and NaBr have different molecular weights, and changing from Na to KBr should be avoided.

HYPOGLYCAEMIA – SOME TIPS ON TREATMENT

Glucose oxidation is the predominant energy substrate for the brain. There are no glycogen stores in the brain; glucose enters the CSF by diffusion, not facilitated by insulin. Glucose enters cells (including neuronal cells) via 2 different types of membrane-associated carrier proteins: SGLT (Sodium-coupled Glucose Transporters) and GLUT (Glucose Transporter Facilitators).

Hypoglycaemia can result secondary to insulinoma, extrapancreatic neoplasia (leiomysarcoma, plasma cell myeloma, hepatoma, lymphocytic leukaemia, lymphosarcoma, malignant melanoma, salivary adenocarcinoma, haemangiosarcoma), liver disease, hypoadrenocorticism, systemic infections (sepsis), toxicities (xylitol, ethanol, salicylates, beta-blockers), and insulin overdose.

Hypoglycaemia in the very young may be secondary to malnutrition, stress, parasitism, immature hepatic systems, or gastrointestinal disease; glycogen storage diseases have also been reported in puppies. Fatty liver syndrome may cause hypoglycaemia in toy breed puppies at 4-16 weeks of age. In the immature patient, glucose transporter levels are low, making them especially susceptible to suffering hypoglycaemia, despite the protective mechanism of the neonate's enhanced ability to metabolise ketone bodies (partly due to lack of body fat and extended time necessary to produce ketones).

Hypoglycaemia at glucose concentrations less than 2.0 mmol/L can precipitate seizures. **However, the severity of signs correlates more with the rate of decrease of blood glucose levels, rather than to the actual glucose concentration.** Hypoglycaemia that slowly develops usually causes weakness, paraparesis, severe depression, or behavioural changes, whereas sudden drops in glucose levels are more likely to cause seizures. Puppies often present markedly depressed or even comatose.

Glucose replacement therapy involves IV administration of 0.25-0.50 grams/kg dextrose to the animal which is obtunded or seizuring. Neonates should receive 1-2 grams/kg (IV, Intraosseous). Administer isotonic replacement intravenous fluids with 2.5 – 5.0% dextrose until the patient is eating and able to maintain euglycaemia without supplementation. Oral glucose administration to a seizuring patient (or with an unprotected airway) is contraindicated. Once conscious, the animal should be fed to provide a longer-acting glucose source.

Blood glucose should be monitored as the likelihood of Somoygi effect causing rebound hyperglycaemia (via hepatic glycogenolysis and secretion of diabetogenic hormones – adrenaline, glucogon, cortisol, growth hormone) is real, especially in the diabetic patient iatrogenically overdosed.

Sometimes, e.g. insulinoma cases, the administration of dextrose solution can promote insulin secretion and exacerbate hypoglycaemia and clinical signs.

Thiamine >2mg/kg (usually given as 25-50mg total dose) IM should be given in advance of glucose therapy, as it is an essential coenzyme in glucose utilisation in the brain.

In situations of refractory hypoglycaemia secondary to iatrogenic insulin overdose, or in insulinoma cases behaving similarly, glucagon (50 ng/kg IV bolus then 20-50 ng/kg/min CRI) can be added to dextrose supplementation. The GlucaGen HypoKit by Novo nordisk contains Glucagon (rys) 1mg (1 IU) as HCl. ▶

With the powder (1 IU glucagon) and solution (1mL) to mix and use IM or IV. Cost is approximately $45 (full cost), kept in the fridge and has an expiry of about a year from time of purchase.

There are occasions where the clinical signs of CNS derangement (including seizures) do not immediately resolve with resolution of hypoglycaemia, especially in the diabetic patient iatrogenically overdosed with insulin. Our practice has seen 2 such canine cases (persistent seizure activity with altered mentation) due to hypoglycorrhachia (abnormally low CSF glucose – 0.7mmol/L & 1.1mmol/L): the first over 24 hours after euglycaemia was achieved (Blood Glucose was measured at 6.9mmol/L → CSF:BG ratio 0.10) and the other some 48 hours after hypoglycaemia was successfully reversed – here, BG was measured at 7.3mmol/L → CSF:BG ratio 0.15; when normoglycaemic, CSF:BG ratio is usually 0.70-0.80. These 2 cases took 4 and 7 days respectively for seizure activity to cease. Both cases showed no MRI abnormalities, scanning performed the same time as CSF Tap. Both cases had resumed insulin therapy (CRI Regular Insulin) to maintain blood glucose in the normal range.

Some years ago I had a feline diabetic patient accidentally overdosed with its insulin that was found in Status Epilepticus the next morning; cluster seizure activity continued for some 2 weeks after normoglycaemia was achieved and the mentally obtunded patient was being maintained on Actrapid Insulin CRI – she was hypoglycorrhachic (CSF glucose 1.0 mmol/L) on day 7 of euglycaemia with CT scan showing no structural brain anomalies.

We have had a few other diabetics presenting hypoglycaemic with similar major CNS disturbances that took some days after normoglycaemia was achieved to regain relatively normal mental status – in these, CSF analysis wasn't performed to demonstrate suspected hypoglycorrhachia.

Neuroglycopaenia (shortage of glucose in the brain) without hypoglycaemia has been described in humans during intensive insulin therapy, in diabetics on CRI Insulin for DKA, and ICU patients with cerebral haemorrhage receiving insulin. It has also been described in a child where it was attributed to temporary dysfunction of GLUT-1. Curiously, this phenomenon seems to be associated with the more rapid drops in blood (and hence brain) glucose concentrations, similar to the severity of clinical signs mentioned earlier. This is supported by the knowledge that in starvation states (after 21 days in people) the decrease in BG is greater than the decline in CSF glucose, resulting in higher CSF:BG ratios (>0.80). I am not aware of any plausible explanation for the failure of the brain's protective mechanism in these instances.

This refutes the common held belief that we can always rule-out hypoglycaemia as being the cause of CNS dysfunction if normal mentation isn't achieved at the same time as (or soon after) euglycaemia.

XYLITOL TOXICITY IN DOGS

Xylitol (a sugar alcohol) is a white crystalline substance that looks and tastes like sugar. It is a popular manufactured sweetener used as a sugar substitute in sugar-free candy, gums, and baked goods, desserts, beverages, cereals and toothpaste. It is also naturally found in low concentrations in fruits, vegetables and mushrooms. This product has a very wide safety margin in people, but in dogs it is a strong stimulator of insulin release and can cause severe hypoglycaemia with ataxia, collapse and seizures. Hypokalaemia may accompany the hypoglycaemia (insulin pushes potassium intracellularly). Clinical signs may occur 30-60 minutes after ingestion but xylitol's prolonged action (due to slow release and absorption) may not show clinical hypoglycaemia for up to 48 hours. Large amounts have been associated with acute hepatic necrosis, coagulopathies and death, with or without preceding hypoglycaemia; illness usually shows about 48 hours after ingestion.

It is tabulated that dogs ingesting >0.1 gram/kg xylitol are at risk for developing hypoglycaemia, and those ingesting >0.5 gram/kg are at risk of acute hepatic necrosis.

There is no antidote for xylitol toxicity. Decontamination procedures may be employed in cases of recent ingestion, but activated charcoal is not indicated as it is ineffective at binding xylitol.

Hypoglycaemia may be prolonged for some days, hence blood glucose needs to be monitored several times a day; IV dextrose is often necessary.

Acute hepatic failure portends a guarded prognosis; intensive care and therapy is essential for a successful outcome.

There are about 190 grams of xylitol in a cup of powder – enough to be lethal to a giant breed dog. Chewing gums sweetened with xylitol contain 1-2 grams per piece – enough to cause hypoglycaemia in a 10kg dog.

The Virbac dental health product, Aquadent, contains xylitol as one of its active ingredients. At the recommended dilution for drinking water, there is less than 1/1000 of the amount suggested to be at risk of hypoglycaemia, and less than 1/5000 of the amount likely to put the dog at risk of acute liver failure. If in the very rare situation that a dog was able to drink a full 500mL bottle of Aquadent before dilution, 2.5 grams xylitol potentially could be ingested, making dogs up to 25kg at risk of hypoglycaemia, and those up to 5kg at risk of acute hepatic failure.

VSS has successfully treated three dogs with hypoglycaemia attributed to xylitol ingestion; a Maltese presenting in acute hepatic failure (jaundice, low-protein ascites, coagulopathic, encephalopathic, hypoglycaemic) was euthanased on financial and prognosis grounds.

This is another example of products safe for humans which are not necessarily safe for pets, joining lilies, macadamia nuts, avocado skin (birds), onions, chocolates, grapes and raisins as items to keep out of the reach of our pets.

Members are reminded that our June 2011 Issue 253 entirely devoted to Small Animal Poisoning cases is available here: www.cve.edu.au/elibrary/edition/5357

INTRAVENOUS LIPID EMULSION (ILE) THERAPY FOR INTOXICATIONS – MAGIC MILK?

OR WILL THE BUBBLE BURST?

ILE therapy has received a lot of recent attention as an antidote for certain poisonings in people and animals. Historically used as a constituent of parenteral nutrition, the 20% lipid emulsion (medium-long chain triglycerides; 260-340 mOsm/L; 2 year shelf-life; pH 8.0) can conveniently be delivered via peripheral IV catheter. It is not cost prohibitive either, a 500mL bag costing less than $100 wholesale.

With some alluring human reports of its efficacy in reversing IV local anaesthetic toxicity, and some less impressive human and veterinary reports (based on anecdotes and case reports), it's little wonder that the 'hype' has been to extrapolate the use of ILE therapy to toxicities of unrelated drugs and toxins. While case reports can be criticised for lacking controls, their credibility is further reduced by the fact that other treatments are also being administered and improvements stated are clinically assessed.

Guidelines for dosing have been extrapolated from the human literature (considered extra-label) and is 1.5mL/kg initial IV bolus (some authors say as high as 4mL/kg) followed by a CRI of 0.25mL/kg/min for 30-60 minutes. The CRI can be repeated in 4-6 hours if the patient is still showing symptoms of toxicity, and if there is no obvious lipaemia. A limit of 8mL/kg/day has been advocated. ILE 20% has a half-life of 30-60 minutes, but can take up to 24 hours to clear from the bloodstream. There have been only rare reports of adverse effects in humans, and none in the veterinary literature in the acute setting – anaphylaxis to the colloid and fat overload syndrome (hyperlipidaemia, fat embolism, haemolysis, jaundice, and coagulopathy).

The most accepted theory for ILE's mechanism of action is the 'Lipid Sink' theory where the ILE forms fat droplets that provide a lipid chamber separate from the aqueous chamber into which lipophilic compounds may dissolve hence there is a less effective concentration of the lipophilic drug available to the tissues. Theoretically, the higher the lipophilicity (log P value; P >1.0 considered to be lipophilic) of the toxin or drug, the more effective ILE will be as an antidote. Log P (the Partition Coefficient of a compound) can be found on Google Searches, the website **www.drugbank.ca/drugs** is a good resource as is **www.cambridgemedchemconsulting.com/reso**, and the paper in *JVECC* 21 (4) 2011, pp 309-320 has a table of pharmacologic drugs whose side-effects may be potentially reversed by treatment with ILE.

There are discrepancies in some of the reports that warrant thought. Reputable experimental animal studies using ILE to reverse local anaesthetic toxicity show benefits including increasing lethal dose, survival time, and improving haemodynamic parameters; however, in the clinical setting, rarely would ILE be as quickly accessible as Bicarbonate which could be rapidly given to effect similar beneficial effects. To add to the intrigue, of nearly 80 human case reports (mostly toxicity to local anaesthetics, cardiovascular drugs, psychotropic agents and anticonvulsants) the response was similar to those drugs that had a log P value <1.5 (i.e. relatively low lipophilicity) as those with log P value >1.5 – a total of 51/76 (67%) of patients showing significant clinical improvement with all of the local anaesthetic toxicity patients showing a positive response to ILE administration. In some of the tricyclic antidepressant cases, recovery was just as rapid without ILE therapy, presumably due to concentrations falling with redistribution in the patient's own lipids. I would have thought that infusing some fat into a patient wouldn't meaningfully add to the lipid in which the drug can sequester as there already is a large extravascular sink for lipid soluble drugs. Further, one experimental animal study showed no change in the mean serum concentration of both amitriptyline (log P 4.92) and nortriptyline (log P 4.51) over 5 hours of increasing ILE infusion concentration, additionally demonstrating increased bioavailability and enhanced toxicity when ILE was given during the absorption phase of these orally administered drugs. To confuse further, an experimental rat model using the topically applied organophosphate parathion, showed ILE therapy ineffective when given in the absorption phase, and when given later, ILE only delayed (not prevented) toxicity effects.

When you look at the pharmacokinetics of plasma protein binding interactions, you'd presume that ILE would increase the expulsion of high (but not low) hepatic clearance drugs given IV or absorbed percutaneously. In orally taken drugs/toxins, this would be expected to be tempered by the increase in bioavailability. Consequently, if absorption is complete, but distribution is continuing, then a beneficial effect of ILE is likely.

Perhaps there is a biological meaning to the log P not being an accurate determinant of lipophilicity for ionisable compounds because it only correctly describes the partition coefficient of neutral (uncharged) molecules. Since >80% of drugs are ionisable, log P may be not an accurate predictor of a compounds behaviour in the changing pH environments of the body. Chemists claim the Distribution Coefficient (log D) is a more correct depictor for ionisable compounds. The website **www.chemaxon.com** has examples of compounds with their log D value at different pH's, listing log D (the Distribution Coefficient) use as a measure of lipophilicity with ionisable compounds (log D at physiological pH of 7.4) and un-ionisable compounds (log P = log D at any pH). Could this be the reason (at least in part) that some of those aforementioned 'high lipophilic' medications were unmoved by ILE administration?

These anomalies portend an argument for 2 lesser heralded theories as to why ILE may be of value, especially in the local anaesthetic induced cardiac arrest situations:

1. Restoration of myocyte function by increasing intracellular calcium. The resultant inotropy increase may defeat the depressive effect of the intoxication. One would envisage this being especially true in calcium channel antagonist toxicity.
2. Supplementation of cardiac energy supplies, as fatty acids are the primary substrate for myocardial ATP production. The local anaesthetics inhibit the enzyme carnitine-acylcarnitine translocase thus depleting available energy via impeding fatty acid transport into cardiac mitochondria. This fatty acid transport blockade may be overwhelmed by ILE supplying large amounts of fatty acid substrate.

What does all this mean for us as clinicians?

When it comes to veterinary clinical reports (so far) of intoxications, these mostly involve the injected Avermectins (Ivermectin, Moxidectin) and the topical Synthetic Pyrethroids (namely off-label Permethrin in cats). We need to be mindful of established successful treatment regimes and general supportive measures. If we believe the 'Lipid Sink' theory, then these are toxins that are ideal to 'tackle' with ILE, e.g. their log P values range from >5.0 to <7.0, indicating high lipophilicity. Remember, though, that the ILE may interfere with other therapeutic drugs used, e.g. the Benzodiazepines (Diazepam, Midazolam) have log P values of 3.0 or more, as does Propofol, and the Phenothiazines (Chlorpromazine, ACP) are 4.0 or more. Methocarbamol (often used successfully in Permethrin toxicity in cats) has a log P value around 0.5, hence one would expect it not to be significantly negated by ILE therapy.

Questions that could be asked to forecast whether ILE therapy is likely to favourably alter drug/toxin kinetics in an overdose situation:

- Is the toxicant highly lipid soluble?
- Are the symptoms severe enough and is the expected mortality rate high?
- How was the toxin administered – oral, injection, topical?
- Does the toxicant have a high hepatic clearance ratio?
- Have traditional treatments failed (e.g. skin or gastric decontamination, activated charcoal, emesis; known antidotes, general supportive measures such as IV fluids, CV and respiratory support, mechanical ventilation, etc) +/- are there financial constraints to prolonged treatment to warrant off-label use of ILE?

With a plethora of clinical case reports and prospective studies in the pipeline, let's hope ILE therapy stands the test of time, but until then, we should proceed with caution, as it is not hard to remember previous fashionable therapies of intoxications supported by simple reasoning that have ended up in the dustbin.

Perspective 100

My Experience with Lomustine for Lymphoma and Inflammatory Bowel Disease

Dr. Gary Norsworthy high fives one of his patients. Alamo Feline Health Center, USA

Note from Richard Malik: *We are very grateful to Gary for allowing us to publish this very practical anecdotal material in the C&T Series. Note that Gary and his associates have administered over 1,500 doses of Lomustine.*

Dr. Gary D. Norsworthy has been in private practice for 40 years, 25 in small animal practice and 15 in feline practice. He is the owner of Alamo Feline Health Center in San Antonio, Texas. In addition to practice, he lectures frequently on feline diseases and is the editor and major author of 6 feline textbooks, the most recent being *The Feline Patient, 4th Edition*, which is being or has been translated into Spanish, Portuguese, Japanese, Italian, and Korean. His practice hosts externs from veterinary schools around the world.* He is a charter Diplomate of the American Board of Veterinary Practitioners in the Feline Practice Category and an Adjunct Professor at the College of Veterinary Medicine, Mississippi State University and Western University.

He was chosen for the 2009 Practitioner of the Year Award in the Medical Specialist category by the Texas VMA.

Lomustine (CCNU) is not considered a first-line chemotherapy agent by veterinary oncologists; it is usually used as a rescue drug when other protocols fail or relapse occurs. However, I have found it both successful and advantageous compared to other protocols. Its use is almost free of side-effects; cats do not show lethargy, vomiting, and anorexia typical of more aggressive protocols. In addition, it is given orally about once per month.

The only significant side-effect is neutropenia. This occurs in about 40% of treated cats and only after several doses. It is also spontaneously reversible and should not call for specific treatment. Use of the drug can continue once the neutrophil count returns into the normal range, which usually takes about 2 weeks. On a positive note, most cats that experience neutropenia are more likely to experience a good response.

Other side effects noted are focal areas of hair loss, sometimes multiple in the affected cats, slow regrowth of hair shaved for surgery, and infections due to immune-suppression. I have seen one case of severe coughing that was responsive to Clavamox®, making it likely to be *Bordetella*. However, I have never seen immune-suppression leading to sepsis and other severe disease.

Successful treatment is defined as creating a clinical sign-free cat. Vomiting and/or diarrhea should stop. The appetite should return to normal, and lost weight should be regained. Thickening of the small bowel, as documented by ultrasound, usually resolves. In my experience, remission occurs about 75% of the time in cats with small cell lymphoma after approximately 6 treatments. Its duration varies greatly but averages about 20 months. Currently, I recommend treatment with lomustine every 4 weeks until remission then every 6-8 weeks long-term. Treatment of lymphoblastic lymphoma is not as successful as treatment of small cell lymphoma, but many of these cats respond surprisingly well to the same lomustine protocol used for small cell lymphoma.

It is not possible to clearly distinguish some cases of small cell lymphoma from IBD using cytology so a full thickness biopsy of the small bowel or stomach with histopathology is needed for accurate diagnosis. There are some ambiguous cases (5-10%) that require immunohistochemical staining and polymerase chain reaction (PCR) testing for clear differentiation. Lymph node cytology is particularly problematic to distinguish IBD from small cell or lymphoblastic lymphoma; therefore, a wedge biopsy is recommended for lymph nodes. However, I do not routinely biopsy mesenteric lymph nodes because many cats with lymphoma of the bowel will not have lymphoma in the lymph nodes; the lymph nodes are read as lymphadenitis. In addition, treatment is the same whether it is in the lymph nodes or not.

Although my primary use of lomustine is for treating small cell lymphoma of the stomach or small bowel, cats with inflammatory bowel disease also respond well. Therefore, if the owner will not permit a laparotomy for full-thickness biopsy of the stomach or small bowel or if the cat is not healthy enough for biopsy, lomustine and prednisolone/prednisone can be used for empirical treatment. This approach is definitely not my preferred way to manage these cats, but some situations make it an option to consider.

The official dose for lomustine is 50-60 mg/m^2. It is supplied as a 10 mg capsule, and can be compounded to other sizes. I give 10 mg to cats weighing 3-5 kg. Prednisolone or prednisone is given simultaneously at about 10 mg per cat q24h PO. If a cat is not pillable, an injection of 20 mg of Depo-Medrol is given q4w.

The capsule is extremely sticky when moist so a clean pilling is necessary followed by a few milliliters of water orally to prevent adhesion to the esophagus. If the capsule gets wet, roll it in corn starch. That will dry it and leave it slick so it can be used again. The current cost is about $10 per capsule when a bottle of 20 is purchased. It can be obtained on prescription at local pharmacies.

Administering lomustine to fractious cats is best done by giving anesthesia or heavy sedation, intubating, and passing a feeding tube into the stomach. (A 12 Fr. red rubber tube works well.) Put the contents of the capsule in 5 mL of tap water in a 6 cc syringe, shake well, attach to the feeding tube, and syringe the lomustine-water into the stomach. Having an endotracheal tube in place keeps the feeding tube from entering the trachea and guarantees it will go into the stomach.

Most cats that respond will be significantly improved by the time of the third treatment. If this does not occur, one should re-evaluate the case to decide if further treatment is advisable.

When recurrence occurs following remission, lomustine has not been very successful for me as a rescue drug. If the client wishes to continue, chlorambucil or the modified Wisconsin protocol should be considered. However, recurrence is usually a very aggressive form of disease so most owners will correctly elect euthanasia.

* Senior veterinary students are encouraged to apply for a 2 to 6 week externship at Alamo Feline Health Center in San Antonio, Texas. Students who plan to spend their career in small animal or feline practice will benefit from daily exposure to real world feline medicine and surgery. An apartment on the second floor of the building is available for use by externs at no charge. Information and applications are available at www.AlamoFeline.com

CLIENT INFORMATION HANDOUT

Inflammatory bowel disease in cats

Gary D Norsworthy DVM DABVP
Alamo Feline Health Center
16201 San Pedro Avenue
San Antonio TX 78232
www.alamofeline.com

Inflammatory bowel disease (IBD) is an important and relatively common medical problem of cats. It is not a specific disease; rather, the term IBD represents several processes which are manifested as inflammation of the bowel. It may involve only the small intestine, large intestine, or stomach; in some cases, all parts of the gastrointestinal tract are affected. It results in recurrent or chronic vomiting, chronic or recurring diarrhea, or both. Weight loss is common in advanced cases.

Contributing Factors

Ingestion of hair can occur with grooming and may lead to development of hairballs, especially in cats that are prolific groomers. Although it might contribute to gastric (stomach) or intestinal irritation, it is more likely that frequent vomiting of hairballs is the result of IBD. As IBD develops the small intestinal walls thicken resulting in a slowing of motility and a slowdown of movement of hair through the intestinal tract.

Causes/Transmission

By definition, the cause of IBD in the cat is unknown.

Clinical Signs

Three general presentations have been identified for IBD:-

1. chronic vomiting
2. chronic diarrhea, and
3. weight loss.

Vomiting or diarrhea often begins as an intermittent event but, over months to years, progresses to as much as several times per day. Sometimes medical care is sought; more often excuses are made for it that include: 1) eating too fast, 2) sensitive stomach, 3) 'just hairballs', and 4) 'that's just the way he is'. Contrary to prior belief, most vomiting is due to disease in the small intestine and not in the stomach. Most cats with vomiting or diarrhea that persists long enough will also have weight loss because the intestinal wall gets so thick that absorption of nutrients does not occur properly.

Diagnosis

The first step in diagnosis is an ultrasound study of the stomach or intestinal walls. If the walls of either organ are thickened, further tests are needed. This disease is considered a 'diagnosis of exclusion' so various tests and treatments are used to confirm a diagnosis of IBD.

Chronic inflammation stimulates immune cells, primarily lymphocytes and plasma cells, to invade the stomach and/or intestinal wall. Occasionally, eosinophils and neutrophils will be found. Thus, the disease is diagnosed when these cells are identified in abnormal levels in the tissue. A pathologist is responsible for this part of the diagnosis; the pathology report usually labels the disease lymphoplasmacytic gastritis (stomach), lymphoplasmacytic enteritis (intestine), or lymphoplasmacytic colitis (colon). Occasionally, the immune cell type involved is the eosinophil. In this case, the disease is called eosinophilic gastritis, enteritis, or colitis.

In order to obtain these cells, a biopsy is required. The majority of the inflammatory response occurs in the small intestine, not in the stomach. Therefore, biopsies of the stomach are usually not sufficient to make the diagnosis. Many cats with lymphoma (cancer) of the small intestine have the same clinical signs and similar ultrasound findings. The only way to distinguish between the 2 diseases is with a biopsy. In the past we have used an endoscope to try to diagnose IBD. However, it is not possible to reach most parts of the small intestine of the cat with an endoscope. In addition, the biopsies taken with an endoscope do not sample all of the layers of the organ; they are not 'full thickness biopsies.' The pathologist needs all of the layers of the affected organ to fully understand the disease. Therefore, surgery is needed to biopsy the small intestine.

Some pathologists may report the diagnosis as 'Inflammatory Bowel Disease'. However, this is a diagnosis that cannot be made strictly from a tissue biopsy. More correctly the pathologist should report 'chronic enteritis' or 'chronic inflammation' then list the cell type(s) involved in the inflammatory process.

While the presence of an inflammatory process is determined with a biopsy, isolating the cause of the inflammation will usually require other tests. Tests or treatments should be performed to rule out stomach and intestinal parasites, cancer, and infections. Diseases such as hyperthyroidism and diabetes are considered. In addition, diseases of the kidney, liver, and pancreas should also be ruled out. When the cause cannot be determined the disease is properly termed Inflammatory Bowel Disease.

Treatment

When possible, the specific disease is diagnosed and treated. Sometimes the above mentioned tests will do that, and sometimes a cause cannot be found. Unfortunately, by definition IBD is a disease for which a cause is not found.

Our treatment protocol is as follows. Note that some of these steps are part of the diagnostic process by eliminating potential causes.

1. A hypoallergenic diet is used to test for food allergy. Since the protein portion of the food is the portion that stimulates an abnormal (exuberant) immune response, test diets are constructed to avoid immune stimulation. One approach is to use a hydrolyzed protein diet. In this diet the protein molecules are broken down into pieces so small that the immune system cannot detect them. Without immune stimulation the allergic reaction cannot occur. Another approach is to feed a diet that contains a protein source to which your cat has never been exposed. Diets composed of rabbit, duck, or venison are the most common.

   A food trial requires time for the body to remove the offending protein that has been in the normal diet. This takes about 6 to 8 weeks. Therefore, a food trial lasts 8 weeks, at which time the signs of vomiting or diarrhea should terminate.

   For a food trial to be successful, your cat must eat the hypoallergenic diet *exclusively.* Eating other cat food, ▶

dog food, table food, or treats is strictly prohibited. Any 'mistakes' requires beginning again.

2. Probiotics are nutritional supplements that supply the bacteria needed for digestion ('good bacteria'). Veterinary probiotics are formulated specifically with the proper bacteria in the correct concentration needed to help cats (and dogs). Proviable® (Nutramax Laboratories) is a capsule containing a chicken-flavored powder. The capsule can be given directly down the throat or opened and sprinkled into canned food. FortiFlora® (Nestle Purina) is a powder that is sprinkled into food. Either product is acceptable and is given for 30-60 days.
3. Vitamin B12 is not synthesized (made) in the cat. It is found in adequate amounts in commercial cat foods. When the food is digested vitamin B12 is absorbed through the walls of the small intestine. Small intestinal disease prevents proper absorption. Therefore, an injectable form of B12 is given subcutaneously (under the skin) for several weeks beginning with twice per week injections then going to once per week injections. One of the side benefits of B12 administration is that this drug often stimulates the appetite of cats that are not eating well. If you are not comfortable with subcutaneous injection technique, we will be glad to demonstrate it or you can go to our Facebook page to the video on giving subcutaneous injections.
4. Some parasites can cause chronic inflammation in the stomach and small or large intestines. Fenbendazole is given orally for 5 days. It is a very wide-spectrum anti-parasitic drug that will eliminate any relevant parasites.
5. Some cats develop chronic intestinal disease due to the overgrowth of certain bacteria. This condition is called dysbiosis. Metronidazole is an antibiotic that can control the 'bad bacteria' very effectively. It is given orally for 30 days.

   The probiotic, vitamin B12 injections, fenbendazole, and metronidazole are started at the same time as the hypoallergenic diet.

If the clinical signs (vomiting, diarrhea, or both) are gone after 2 months of treatment, there is no need for steroids. (If vomiting, weight loss, or diarrhea is severe, we may elect to begin steroids at the onset of therapy to control the clinical signs.)

If the clinical signs are not gone after the first 2 months of treatment, your cat does not have a food allergy. The hypoallergenic diet is discontinued and a different diet is used which is highly digestible, high in protein, and low in carbohydrates. An immune suppressing drug, steroids, cyclosporine (Atopica®), or lomustine, is begun in an attempt to better control the cat's overactive immune system. One or more of the immune suppressing drugs is given long-term.

If the eosinophilic form of inflammation is found by the pathologist, a steroid is given, but 2-4 doses of lomustine may be given with it. This is a form of inflammation that is harder to control so more powerful drugs are needed initially.

Corticosteroids are renowned for causing a variety of side-effects in humans. Fortunately, cats are very resistant to these side-effects as compared to humans. Regardless, to minimize any possible adverse effects, our goal is to use the lowest possible dose that is effective.

Prognosis

In most cases, it is reasonable to expect good control of the disease using a steroid and a highly digestible diet. However, cure should not be expected until research finds the cause of this disease.

Urgency

There is mounting evidence that IBD will transform to lymphoma, a form of cancer, when the disease occurs for months to years. Therefore, it is important to diagnose and treat this disease as early as possible.

HOW DO I KNOW IF MY PHOTOS ARE THE RIGHT RESOLUTION FOR PRINT?

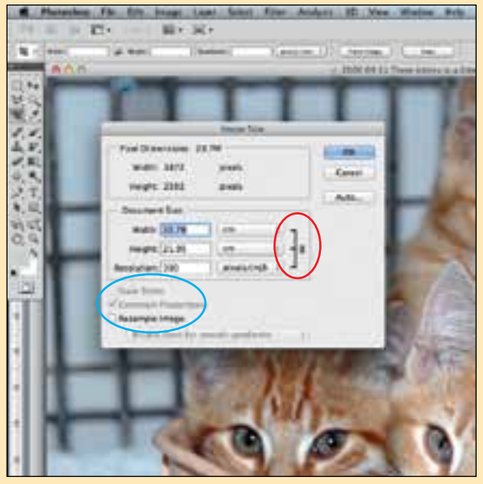
These days most of us have something we can take a decent photo with, like a smart phone or digital camera. Provided the camera has been set to a high quality setting you can probably count on a photo with sufficent resolution for print. But how do you check and what is high resolution? And will it print out okay in C&T?

When it comes to resolution there are two things to keep in mind. First, the physical size (height X width) and second the pixels per inch (ppi). So let's take an image and open it in Photoshop (if you have that!). Select 'image size' under the image menu and a window opens, like the one on the right.

Start by checking that the brackets (circled in red) are all on. Do this by checking the boxes off/on to match this window (circle in blue). This will preserve all the resolution information.

Most images will show either 72, 150 or 180 in the pixels/inch box. Now, go ahead and type 300 in that box. You will notice that the dimensions of the photo become smaller. These final dimensions are the actual size of the image at high resolution. As you can see on the right, the image is roughly A4 size at high resolution.

What about digital resolution? Well that's a different story...



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