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

‘Whip it good’
Nasal Mites

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News

Calendar - DE 2014 registration OPEN!

From the Director

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 WIA Division of AVA has been very active in running a mentoring scheme for new graduates and the WIA is keen to roll out this initiative 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/anxiety/courses 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 signaling 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 possibile-suicide

This quarter we have another ‘bump’ issue with 64 pages, as our editor Liz Churchill 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 CVE 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 AustraliaNZ. 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
Director CVE

Directors

Calculated by the CVE & the Discipline of Psychiatry, University of Sydney

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

Cost: CVE members $201 (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 veterinary’s mental health 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 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).

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From surgery to rehabilitation, the CVE has a wide range of conferences, workshops and online courses.

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All CVE courses are presented by leading experts in their field, so you can confidently choose the CVE to provide you with the quality professional development you seek to become a better practitioner and ensure the continuing success of your practice.

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

<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
<th>Location</th>
</tr>
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<tbody>
<tr>
<td>23-26 Sept</td>
<td>Orthopaedic Conference</td>
<td>Fremantle</td>
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<tr>
<td>5 or 6 Oct</td>
<td>Hip &amp; Stiffe Workshop</td>
<td>Brisbane</td>
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<td>13 Oct</td>
<td>Diabetes</td>
<td>Brisbane</td>
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<tr>
<td>19 Oct</td>
<td>Basic Echocardiography</td>
<td>Sydney</td>
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<tr>
<td>20 Oct</td>
<td>Advanced Echocardiography*</td>
<td>Sydney</td>
</tr>
<tr>
<td>27 Oct</td>
<td>Looking Down the Microscope</td>
<td>Port Macq.</td>
</tr>
<tr>
<td>8 Nov</td>
<td>ecoCPD: Behaviour</td>
<td>Sydney</td>
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* Prior learning will be required to attend this workshop.

ONLINE COURSES IN 2013

<table>
<thead>
<tr>
<th>Date</th>
<th>Course</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 Sept - 29 Sept</td>
<td>TimeOnline: Marine Wildlife (Students only)</td>
<td>2013 AND save up to $200 on course fees</td>
</tr>
<tr>
<td>28 Oct - 24 Nov</td>
<td>TimeOnline: Anaesthetic Complications</td>
<td>CVE and ask for 10% discount on another TimeOnline course</td>
</tr>
<tr>
<td>4 Nov - 1 Dec</td>
<td>TimeOnline: Avian</td>
<td>TimeOnline course will be offered in 2014</td>
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Hugh White
Director CVE

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Back to TOC page
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/isfmfcc (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 tachypnoeic, 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 be tachycardic and pyrexic, and let down patients are stressed? We simply can’t begin to practice good feline medicine until we can address these factors. The ISFM ‘Cat Friendly Clinic’ accreditation scheme is designed to help and encourage veterinary clinics to be proactive in making visits less stressful for both 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 been 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.

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.

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
Welcome to the latest issue of Control & Therapy Series – 272 SEPTEMBER 2013.

Thank you to all contributors.

... 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

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View

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

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Contact cve.membership@sydney.edu.au or call Jacqui Kennedy on (02) 9351 7979.

Then visit www.cve.edu.au/candtebook which allows you access to this current issue in e-book format and the quarterly link to the ebook by email.

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.

Part 4.3

ECHIDNAS

Be aware of:-

- Endothermic; 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 Echidnas 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.
- Males 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 themselves 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.

Contact

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

Don’t miss reading the ebook!
Housing the sick or injured 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. Stomach tubing may be required if not self-feeding, specialist advice with this technique is recommended.

Euthanasia methods
- Injection of Sodium Pentobarbitone® can be administered either caudal to the external nares. 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.

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.

Echidnas and the platypus are the only egg-laying mammals, known as monotremes.
Stabilisation of a fractured mandible in an Eastern Grey Kangaroo

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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 fracture 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 fracture of bone should be more stable if it was 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 symphalangism 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 (Figures 1 & 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.

Surgical correction of bilateral flexural forelimb deformity in a foal

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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 sucking reflex and was dehydrated with an increased skin tent. The foal remained in sternal recumbency and, upon orthopedic 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 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 (GammaCheck® E, Plavacc Pty Ltd) was performed and indicated failure of passive transfer, and a plasma transfusion was administered (Equiplas®, Plavacc Pty Ltd).

Also during this visit the limbs were reassessed for deformity. The foal was classified as having a Grade 2/5 bilateral congenital forelimb flexural deformity as he was born with the inability to completely extend the carpi 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.

The foal’s physical condition appeared to improve with time and growth. By the age of 8 weeks the foal began to show signs of increased movement. The foal was classified as having a Grade 0/5 bilateral congenital forelimb flexural deformity (contracted tendons), as well as angular deformity of the carpal regions.

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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. 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.

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.

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’. Consultation with a farrier was sought to modify the angle of the toe over several remittances to bring the sole into contact with the ground and reduce the strain on the flexor tendons.

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.

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 allow him 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 metacarpo-phalangeal joint15, as opposed to the carpal joint. Tenotomy of the deep digital flexor tendon directly would also only release flexion mainly of the distal limb16. Further research revealed that tenotomy of the flexor carpi ulnaris and ulnaris lateralis tendons has been used for the surgical

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.

Figure 5. Splint 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.

Figure 2. Bilateral radiographic examination of the carpus of the foal. (A) Lateral/proximal view of the left carpus, (B) lateral/proximal view of the right carpus, and (C) bilateral dorsopalmar view of the carpus. All radiographs show 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.

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 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. 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.

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.

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’. Consultation with a farrier was sought to modify the angle of the toe over several remittances to bring the sole into contact with the ground and reduce the strain on the flexor tendons.

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.

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 metacarpo-phalangeal joint15, as opposed to the carpal joint. Tenotomy of the deep digital flexor tendon directly would also only release flexion mainly of the distal limb16. Further research revealed that tenotomy of the flexor carpi ulnaris and ulnaris lateralis tendons has been used for the surgical
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. 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. Therefore, these could have been used instead of, or in conjunction with, correction trimming. Excessive exercise was avoided during treatment as this can also contribute to fatigue of the flexor tendons and potentially aggravate the problem.

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

References

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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 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 CootaAcoo near the Dandenongs in Victoria. Our first year, spent learning establishing protocols, was run by a spate of late term abortions, still births and failure to thrive: lovely countryside but iodine deficient. Fortunately once diagnosed the condition was 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 and I contacted my ‘go to’ man in Victoria, Dr Inow Professor Ivan Cape 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 Wimbeyan Caves. Figure 2. Below) Comparison of thyroid gland mass from normal and goitrous newborn kids.
Follow on from C&T No. 4636: Curettage and diathermy of feline nasal squamous cell carcinoma

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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.
Curettage and diathermy of feline nasal squamous cell carcinoma

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A routine treatment for feline squamous cell carcinoma of the nasal planum is cryotherapy. A more aggressive procedure is noseectomy permitting wide margins. 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 -80°C in all regions of the tumour, including the lateral and deep margins, is considered adequate to completely devitalise neoplastic tissue. The outer edge of the iceball at 0°C is inadequate for tissue destruction 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. A 90 second double freeze thaw cycle of cryotherapy with a 4 mm 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 ‘grazes’ 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 1 mm 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 area is allowed to heal by secondary intention.

Petroleum jelly

Diathermy destroys approximately a 1mm rim of tissue. The and again diathermied. If necessary a third cycle is repeated. The procedure is repeated. The diathermied base is curetted

References
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 anaesthetised, or the loss of blood is so great you need to stop where it is possible/safe to have a sedated animal sedated/sedated/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 prepuret sheath, 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 atarax and – hurricane injections to cover the 6 weeks until withdrawal 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 impendence 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 CAT Series.

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Eye watering tips for suturing techniques in Tom’s Paddock

C&T No. 5328
Economic validation for stock-incommonly used antibiotics and antimetics on the drug shelf

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Cerena® is wonderful antibiotic and is less expensive than it was, but it’s still expensive. Vomiting dogs are not overly concerned in the weeks of it – (always keep an eye kBid chemotherapy or chiral 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.

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**Small Animals**

Clinical abnormality is up to 10mm swelling at injection site, lasting up to a day for isolated injections. At higher doses/multiple injections at some sites, 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 in ≤6 weeks or during pregnancy/lactation.
- Use with caution in animals with liver disease.
- Ketamine squirted into the mouth of a feline cat also works but you have to have it fairly confined in the first place to do that.
- Immobilization fallen out of favour and whilst Domitor® was and remains a very popular drug used overseas, it fats auto-antibodies 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 of use.

**Cocktail B**

- A dry bottle of Zoletil (tiletamine & zolazepam) diluted with 2.5 mL Domitor and 2.5 mL Torbugesic (10 mg/mL). 1 mL per cat or 5 kg dog, 0.2 mL per 0.5 kg dog, 0.3 mg for a 20 kg dog, 0.4 of this stuff in the rum 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 concoctions.

Note from Dr Bernstein: The cocktail was recommended to me by the Virbac distributor in Israel under the name of ZDT (Zol Domitor Torbugesic) aka TD or TTdx (when using doxymor versus doxymor – careful! – the concentrations). 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 hipped-up animal. The article states that a higher dosage can be used for anesthesia although I have never used it without tocainide. See also Dr Ko’s handy dosage chart for TTdx.

Injectable Anaesthesia Update by Dr Jeff Ko

Jeff Ko’s Dosage Chart for TTdx

1 2 3 4

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 at and 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 handling of an animal that has gone into anaesthesia as well 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 are both so palpably stressed that you as a vet can’t concentrate properly given the fear/hystera/grief/distress phenomena bouncing around the room. This is especially useful if the dog is blind – those pets far more likely to panic and start shaking 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 nowness 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’ to cats as I believe the cat requires more sedation (a S/C injection or a slow LM push) and I give Cerenia® to cats in advance of euthanasia in the belief that the cat owners want to sit in on, or where the cat’s history suggests manual restraint is not something the cat tolerates. Use a needle for the injection and the dog 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-back...
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.
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. 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.

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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. Pemphigus foliaceus.

Figure 1 to 3.

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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Canine behaviour – have we got it right?

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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 / Redirect / Distraction / Ignore unwanted Behaviour (RRDIB) techniques adopted by Canine Veterinary Behaviourists (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 would hold up. 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 dog owners.

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 qualifications.

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 raises 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 RRDB.

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 noisy 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 RRDB alone.

Another case I saw was a Pug cross that had noise phobias and a deeply rewarding experience for the dog lover. This is not possible using RRDB alone.

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

This is 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 dominance but, most importantly, rank without rancour.

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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). The University of Sydney Faculty of Veterinary Science

The University of Sydney Faculty of Veterinary Science

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

**Invited Comment courtesy of:**

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The University of Sydney
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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 spurn the scientific community to examine skills deployed by the most effective humane practitioners. This process will demystify the so-called ‘dominating’ 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.

Reference


**WINNER**

The hidden perils of paradise

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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.

Two months ago one of the dogs, a three-year-old Jack Russell Terrier was stung on the ear by a jumper ant.

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 ear, 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 leg.

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 advisable to consider the animal in the same way as a human. To be safe for our pets and when considering species specific treatment for cases of anaphylaxis.

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 ear, 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 leg. The dog 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 stictic 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.

**Invited Comment courtesy of:**

Kersti Seksel.

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

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**Images sourced from the internet.**

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These dogs 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.
**OBITUARY – Dr John Holt**

By Anne Fawcett

**PLEASE NOTE: a version of this article was published in The Veterinary 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 cabinet maker and brief career in industry, John purchased St George Animal Hospital (SGAH) from Richard Boon in 1959 and developed it into a showpiece practice. Veterinary Association conference and the word dog or cat would not be heard in John's practice.

Freeman and Neville Japp, Holt co-founded a group which later became the Australian Small Animal Veterinary Association.

According to Mary, John's care of animals continued until his death, as he was an active supporter of the campaign against live animal export.

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

John received multiple awards throughout his career, including an award from the Australian Veterinary Journal (AVJ). He was the first small animal veterinarian to receive this award and the first mainstream veterinary publication to honour small animal practice in particular.

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

John 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 articles often amplified insights the dog was to my knowledge the first recorded case of its kind diagnosed and successfully corrected.”

It's worth considering 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 AVJ.

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

Allan recalls a Sydney practitioner’s branch meeting, attended mostly by veterinarians.”

Friend Henry Hirschhorn feels that John's contribution was “greatly underestimated” and that John was “a pioneer who opened my heart to the power of stereoscopy.”

The Association’s first office in Hurstville was possible at a time when small animal practice was much less sophisticated than today.

There 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 care of animals.

PLEASE NOTE: a version of this article was published in The Veterinary Magazine.

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“Recently I faced a very unfortunate situation with one of my clients and one of its 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. He followed up with blood tests and found that he was in renal failure.

I asked his owner if he had any illnesses inside his apartment and he reported ‘no’. He was aware, as I had previously told him (and many other clients) especially clients with indoor-only cats and I see a lot of them that likes 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 brought 2 plants at the Tenterfield 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. I certainly brought it in. I identified it. It was, indeed, Lucky Bamboo. He also said that he had noticed Cuba eating it and then vomiting.

Anyhow, to cut to the chase, I ‘googled’ Lucky Bamboo and unfortunately not out that this plant is actually a ‘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 it is, as well as every other vet I have spoken to, including vets at The Cat Clinic at the RSPCA’s Emergency vets at VBSQ in Brisbane, said that they had no idea Lucky Bamboo was a toxic plant. It is a common plant; forests make very artistic designs with it (you can train it to form spiral formations) and it has a very good Fosh Shui for your home.

But ‘Lucky Bamboo’ is a silly 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 all 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 Lili NOR is it 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 FRM, DAVS**

Registered Specialist in Veterinary Pathology

Honorary Research Associate, Queensland Herbarium & Biosecurity Queensland

Life Member, Australian Veterinary Association

“Yapunyah!”

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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 Dracaceae) or for some botanists, Family Asparagaceae. 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, p157). There are about 60 Dracaena species named worldwide and he lists them as grown in Australia. To call the plant in question Dracena sandersiana for any other species for that matter, we would need to examine by a botanist. Is the plant still available for examination? If so, Dr Paul Foster (q村落) at the Queensland Herbarium, IPAR, can access the VETTOX Discussion Group (based in the UK) and if not, he might 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 Toxics Database has a few entries on Dracaena. In one, Stilla Ossendorf (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 Dracena sandersiana (lucky plant). Other entries are records of a couple of discussion strings within the VETTOX Discussion Group (based in the UK). Stilla summarised, they indicate that Dracena 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. For my part, 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 a
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 9780643050697

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

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**Cultivation and uses**

**Lucky bamboo spiral houseplant**

*Dracaena sanderiana* and related species are popular houseplants. *D. draco* or *D. draco* 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 in water in a fish aquarium. The water should be completely changed every two weeks. The water should be boiled prior to planting, soft tap water with little fluoride, or even water from a filtered, established aquarium. It does best in bright, indirect lighting and temperatures from 10°C to 25°C (60°F to 77°F).

Yellow or brown leaf edges may be caused by too much direct light, crowded roots, or fluctuating or chlorinated water. The latter of which can be prevented by leaving tap water exposed to the air for a day before plant sale. Salt 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* is 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 the natural elements of water, fire, earth, wood and metal into balance within the environment. Lucky bamboo is believed to be an ideal form 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 and especially the Spring stalks 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’.

References

2. ‘Cocky Tips: Lucky Bamboo’

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**Dracaena sanderiana** (Source From Wikipedia, the free encyclopedia)

**Scientific classification**

- Kingdom: Plantae
- Class: Monocots
- Order: Asparagales
- Family: Asparagaceae
- Subfamily: Nolinoideae
- Genus: Dracaena
- Species: *D. sanderiana*

**Binomial name:** Dracaena sanderiana

**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 (0.6 in) broad. It is marketed in the developed world as a Chinese decorative plant ‘Lucky Bamboo’. (although Bamboo and not native to Asia), propagated from short cuttings, usually in water.

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**What is a trigger point?**

Myofascial trigger points have been defined in human medicine as a ‘hypersensitive spot in skeletal muscle that is associated with a hyperirritable 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 inflammation, 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 Gladstone Veterinary Hospital (GVH) with hindlimb lameness, which was owned concern 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 his 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 only once a day. The lameness did improve initially on re-examination 2 days later, and on physical examination it was found that there was moderate swelling associated with the gracilis 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 dragging her left hind toe 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 wasted left hind biceps femoris, quadriceps, tibialis, fascia latae and middle gluteal muscles.

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

From physical examination findings the following problem list was generated:

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**Trigger points**

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**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 due to 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.

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**Invited Comment courtesy of Dr Selina Ossedryver BVSc (Hons) MVSc**

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

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

Przypanied zatrus: dracaena u kotow.


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**Dracaena sanderiana** (Lucky bamboo spiral houseplant) **References**

2. ‘Cocky Tips: Lucky Bamboo’

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**www.vetbookshop** Australia’s poisonous plants, fungi and cyanobacteria

by Ross McKenzie published by CSIRO and available at CVE’s www.vetbookshop.com


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**Scientific classification**

- Kingdom: Plantae
- Class: Monocots
- Order: Asparagales
- Family: Asparagaceae
- Subfamily: Nolinoideae
- Genus: Dracaena
- Species: *D. sanderiana*

**Binomial name:** Dracaena sanderiana

**Dracaena sanderiana** is a species of the genus *Dracaena*. It 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 (0.6 in) broad. It is marketed in the developed world as a Chinese decorative plant ‘Lucky Bamboo’. (although Bamboo and not native to Asia), propagated from short cuttings, usually in water.

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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 dilatation 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 gracilis 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 paraparesis. 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 fascia latae in which the trigger point was present was conducted using a Pointer Excell II TENS device (Lhase™). Following TENS therapy, stretch of the tensor fascia latae 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 contraction.

**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.

**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 1998).
Feline eosinophilic proliferative glossitis case

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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 scallops around head and neck. I diagnosed this as ECG 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 4.8kg, mouth unable to examine due to ferocious nature, ventral throat soft swelling, skin surface over swelling aseptic. Heart sounds and lungs sounds were fine with no abnormalities detected, palpation no 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 stipped, 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 granulomatus 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

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‘Poochie’, a 1½-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 citalimycin 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 skin rads to check for bone lysis and the necessity for either a hemmoraphilactomy 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 gangrenous tissues. The pathologist commented on the neoprosy 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 nucleus 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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www.cve.edu.au/distanceeducation
Tale of a broken tail

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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 fray ed, 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 sacrococcygeal 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 lumbar spinal spine were intact. There was complete avulsion of the caudal vertebrae 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 sacrocaudal 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 q14hr 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 (abiet 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 traumatised by licking/biting and the skin at the base of the tail 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 neuropathia which resolves rapidly (Davis & Walmsley, 2012) which it did in this case. Concurred 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.

• Peineal sensation and anal tone are key prognostic indicators. Where urinary bladder function is affected, the absence of peineal 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 counselled accordingly.

References

In Practice 34:27-33.

Australian Veterinary Journal 84:53-66.

Small Animals

Figure 1. The tail on presentation.

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

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

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

Figure 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.

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 18mos 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 her 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 Claudox®, 1.5mL Baytril® given SC. We all tried to ignore the horrid sound of the sucking air from the wound. Once on O2, with an IV line and pain relief on board, I elected to anaeastheticise. It was approximately 10 minutes from time to 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/O2 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 32.9°C.

Ktly® was placed around the wound and the RHS chest wall and sternum were prepasted 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 O2 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 2nd 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 incision in about the 5th intercostal space through the skin and SC. We were waiting on anaesthetic for the peritoneal lavage. 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 mono. 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 defect that was present...

We postponed our routine surgery and kept her on the heated table running, 4-8 hours 0.1mL ACP was given IV to help sedate her if she seemed uncomfortable.

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 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 to get a better feel for any changes. A methadone infusion of 0.1mg/kghr for 24 hours was started. Every 4-6 hours 0.1mL ACP was given IV to help sedate her if she seemed uncomfortable.

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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 into 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 omeprazole or oral amoxiclav/Baytril®/metrogyl once her appetite was on track. 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 omeprazole or oral amoxiclav/Baytril®/metrogyl once her appetite was on track. She wasn’t so keen on the softer canned recovery diets. Her pain relief was well managed by the ongoing methadone infusion.

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

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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.

Dog 1 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 entirely scientific so I set 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 endoscopy: 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 endoscopy 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 invoked 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 really stop 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 resolve this. I rang Dog 5’s owner who had staying with Dog 6! and she was 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!

Photos: 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 colitis. The ivermectin dosage for heartworm prophylaxis is much lower (6 μg/kg PO) and unlikely to be effective against nasal mites.

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. Foliacular hyperplasia in the nasopharynx should arouse suspicion even when mites are not visualized. Flushing the nasal cavity with saline may result in mites migrating caudally into nasopharynx where they can be seen more easily.

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.

An 11-y-o working Border Collie from Alice Springs had epistaxis on 3 occasions, snorting, vigorous reverse sneezing at times, ulcerative lesions on the nasal planum and nares (see photographs below) and serious 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: inserting the scope Jody actually saw the mites skittering 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 setting 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.

Dog 1:
- a 2 y.o. Kelpie x from Kalgoorlie – client’s dog
- 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 mites migrating caudally into nasopharynx. Flushing the nasal cavity with isoflurane resulted in mites visualized. Diagnosis was then confirmed and a treatment trial with milbemycin before scoping. The resident thought pattern recognition based on n=1 was not entirely scientific so a treatment strategy was set up a big bill, found the mites and then treated successfully with milbemycin.

Dog 2:
- a vet nurse’s dog
- presented with really severe reverse sneezing.

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 – another friend’s dog

Dog 6:
- a 12 y.o. MN Kelpie x – friend-of-a-friend’s dog

Video courtesy of Sue Foster

The story

Jody suggested that nasal mites were probably quite frequently overlooked and under-diagnosed, 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 endoscopy: 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 endoscopy 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 invoked 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 really stop this terrible gagging.’ Dogs 3 and 5 had stayed with Dog 6! and she was 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.

...and one more from Jody Braddock

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

Rhinoscopy from Dog 1 showing nasal mites in the nasopharynx

...and one more from Jody Braddock

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

Rhinoscopy from Dog 1 showing nasal mites in the nasopharynx
Case Report: A canine urogenital carcinoma on the neck of a domestic shorthair cat

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Keywords: Apocrine ductular carcinoma, feline, neoplasia, pathology

Introduction
Apocrine adenocarcinoma comprises a group of rare primary cutaneous cancers, and has been reported rarely in dogs and cats, usually in aged animals

The cat was FIV and FeLV negative serologically and had an ulcerated lesion of approximately 2 cm diameter with self-surgery. Initial examination of the lesion revealed a pruritic circular ulcerated over the surface of an underlying rather poorly differentiated mass. The excised mass was placed in formalin and processed for a second opinion. The cat was given 0.5cc Cefovecin11 subcutaneously prior to discharge. The tissue deficit was performed. The cat was given 0.03 mg atropine subcutaneously, followed 20 minutes later with 10mg sufentanil intravenously. A 3.5 mm endotracheal tube was placed and anesthesia was maintained with 2% isoflurane / 1L oxygen per minute using a non-re-breathing circuit. Lactated Ringer’s 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 leucocytes 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 hair shaft that was ulcerated over the surface of an underlying rather poorly differentiated mass composed of trabeculae and islands composed of variably sized acini, trabeculae and solid nests of pleomorphic epithelial cells surrounded by a profuse reactive fibromyxoid stroma. Small nests of these epithelial cells were invading the surrounding stroma on the periphery of the mass. The mass was divided into two parts. Some of the larger nodules in the neck and the head of the cat contained necrotic cytoplasmic and necrotic cells plus melanin debris. Other islands were better differentiated and consisted of tubules formed of epithelial cells with a more rounded 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 its metastatic spread to lymph nodes.

The tumour appeared to have been fully excised surgically.

Discussion
Since few apocrine ductular carcinomas have been reported in cats, their biological behaviour is not well established. However, they may be common in 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 lymph nodes as well as distal lymph nodes and lungs1. A diagnosis of feline apocrine ductular carcinoma was made in an aged male neutered domestic short-hair cat presented with a vague history of an ‘abscess’ associated with an inverted, ulcerated and circumcised lesion (1 cm diameter) on the middorsal region of the neck.

The cat had been treated by a previous veterinarian with a two-week course of broad spectrum oral antibiotics (amoxicillin-clavulanate). Temporary resolution of clinical signs, primary pruritus and the size 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 purulent circumscribed ulcerated lesion of approximately 2 cm diameter with self-infected exfoliations at the margins of the wound. The cat was FIV and FeLV negative serologically and had received annual core vaccinations (F1P3 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 referable to the skin lesion were obvious. The cat was later admitted for surgical biopsy.

The cat was anaesthetised with 1.0 mg acetylpyrazalone and 0.03 mg atropine subcutaneously, followed 20 minutes later with 10mg sufentanil intravenously. A 3.5 mm endotracheal tube was placed and anaesthesia was maintained with 2% isoflurane / 1L oxygen per minute using a non-re-breathing circuit. Lactated Ringer’s 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.

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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 circumcised lesion (1 cm diameter) on the middorsal region of the neck.

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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.

References
9. Ferr-o-sac 3, Fort Dodge, Australia
10. Parim Injection 5mg/ml: Pameld Laboratories, Australia
11. Convex, Piller, Australia

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What’s YOUR Diagnosis?
C&T No. 5343
Suzanne Pears
E. suzannepears@hotmail.com

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

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 cytological diagnosis?

CAT No. 5345

George Reppas
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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.

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

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

Figure 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?

CAT No. 5346

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

e-book readers rollover or download C&T 5280 here

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 it 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 Hornsby 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.vetbookshop.com

Replies and Comments

Email your answer to elisabeth.churchward@sydney.edu.au and be in to win a CVE proceedings of your choice. Go to www.vetbookshop.com to peruse our titles.
Why I want to be a veterinary specialist & Why I support the CVE

Amy Lam BVSc(Hons) GradCertVetStud MACVS (Small Animal Medicine) Registrar in Small Animal Medicine

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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 vets at the time. He was passionate about helping pets, and owners alike. He was and still is an incredible inspiration.

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Amy Lam is an Australian trained registrar in Internal Medicine currently working at Willows in the UK. She has also done in private practice in Melbourne and Canberra (with her Dad), undertaking 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.

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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 involvement by the owner of the dog and the clinician managing the case and appropriate individualisation of the dog’s therapeutic and monitoring regime. The veterinary clinician must invest time to educate the owner about canine diabetes and its management, as well as to provide support and guidance which is necessary, especially when initially introduced 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.

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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 advantages as hospital-derived ones. It is preferable to use the same scale each week. Scales designed for weighing adult humans are not suitable for small and medium sized dogs. Scales designed for babies are a much better option.

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.

Appendix

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 >30°C or light for prolonged periods. An expert method of routine handling of insulin is therefore necessary. The possibility of insulin resistance is to be new to a vial of insulin, suspension must be thoroughly mixed prior to administration or doses should be based on the available and inadvertently changing to a different type of syringe can lead to dosing errors. Dosing errors are less frequent with insulin pens. However, insulin pens and syringes, however, insulin pens dosing 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. In this instance, treatment involves performing haematology, serum biochemistry, urinalysis, and microbial culture and susceptibility testing of the urine. Almost any concurrent condition might cause insulin resistance that affects diabetic control. Once the concurrent disease state has resolved, insulin sensitivity can be expected to improve, and previously inactivated risk of hyperglycaemia unless the insulin dose is decreased.

Hypoadrenocorticism 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 improved weight loss with weight in the order of 8% of body weight per year.

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 cats. There is a typical period of poor glycemic control that is followed by deteriorating glycemic control despite increasing insulin doses. The period of poor glycemic 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 clinical history and investigation.

In insulin-treated diabetic people, hypoglycaemic events are 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 improved weight loss with weight in the order of 8% of body weight per year.

The findings of a serial blood glucose curve should always be related to the clinical history and weights, and changes in body weight before a final decision is made to increase or decrease an insulin dose. If a diabetic dog is lethargic, has a stable body weight, has no ketonuria, is drinking <0.6 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, serial blood glucose dosage adjustments of no more than 1 U are advised, regardless of the dose of insulin being receiving.

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

<table>
<thead>
<tr>
<th>Blood glucose range</th>
<th>Insulin recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;10 mmol/L (180 mg/dL)</td>
<td>Decrease the 12-hourly insulin dose by 50%</td>
</tr>
<tr>
<td>5-8 mmol/L (90-144 mg/dL)</td>
<td>Decrease the 12-hourly insulin dose by 20% (rounded down to nearest U of insulin)</td>
</tr>
<tr>
<td>3-5 mmol/L (54-90 mg/dL)</td>
<td>Decrease the 12-hourly insulin dose by 10% (rounded down to nearest U of insulin)</td>
</tr>
<tr>
<td>&lt;3 mmol/L (54 mg/dL)</td>
<td>Increase the 12-hourly insulin dose by 50%</td>
</tr>
<tr>
<td>&lt;2 mmol/L (36 mg/dL)</td>
<td>Increase the 12-hourly insulin dose by 100%</td>
</tr>
<tr>
<td>&lt;1 mmol/L (18 mg/dL)</td>
<td>Increase the 12-hourly insulin dose by 200%</td>
</tr>
</tbody>
</table>

The standard protocol for generating a serial blood glucose curve is to perform a ‘routine’ serial blood glucose curve immediately after each insulin dose. The time position after each dose.

It is also important to check that the dosing dial has returned to the ‘zero’ position after each dose.

The protocol can be modified on the next visit 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.

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BROMINISIM/BROMISM (BROMIDE TOXICITY) – CAUTIONS WITH DOGS ON BROMIDE AID THERAPY

‘Harvey’ – a 10-year-old male neutered Australian Terrier

Long-term antiepileptic drug therapy (phenobarbitone and KB,E) for grand mal seizure disorder and well-controlled, non-ambulatory, non-ambulatory, obtunded patient with reduced serum levels of both mid-range therapeutic levels. 

Had been on Hill’s u/d™ Diet (99ng Sodium per 100kcal ME) exclusively since a severe pancreatitis bout a few years previously. Diet was changed two months ago to Hill’s u/d™ Diet (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 KB,E (by 33%) dosages were steadily increased over those 2 weeks, presuming the seizures were being controlled. 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 15 years of marital neglect.

Terry 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, AVA Qld. He has been an external examiner, guest tutor and tutor at UQ’s School of Veterinary Science as well membership examiner for ANZCVSc.

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 regular veterinary practice in 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) because it was 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 general clinician with a special interest in the emergency and critical care patient.

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Terry, born and bred in 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) because it was 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 general clinician with a special interest in the emergency and critical care patient.

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Terry King with Aussie icon - the Victory

Hypoglycaemia – Some Tips on Treatment

Glycose 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 insulinaemia, extrapancreatic neoplasia (leiomysarcoma, plasma cell myeloma, hepatoma, lymphocytic leukaemia, lymphosarcoma, malignant melanoma, mast cell adenoma, haemangiosarcoma), liver disease, hypoadrenocorticism, systemic infections (sepsis), toxicities (kilyl, ethanol, salicylates, beta-blockers), and insulin overdosage.

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 profound development of the nervous system’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 you know that it has been clinically proven that cut grass (freshly mown lawn) releases a chemical called Seracensin 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).
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 used as a sugar substitute in sugar-free candy, gums, and baked goods, desserts, beverages, cereals and toothpaste. It is also used as a sugar substitute in sugar-free candy, gums, and baked goods, desserts, beverages, cereals and toothpaste. It is also used as a sugar substitute in sugar-free candy, gums, and baked goods, desserts, beverages, cereals and toothpaste.

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.

INTRAVENTRICAL LIQUID EMULSION (ILE) THERAPY FOR INTOXICATIONS: MAGIC MILK? OR WILL THE BUBBLE BUST?

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 (20% intralipid) (aqueous 10%, lipids 90%) is an osmotically active agent. The resultant inotropy increase may defeat the pharmacological actions of the calcium channel antagonist. The resultant inotropy increase may defeat the pharmacological actions of the calcium channel antagonist. The resultant inotropy increase may defeat the pharmacological actions of the calcium channel antagonist.

With some alarming human reports of its efficacy in reversing IV local anaesthetic toxicity, and some less impressive human and veterinary reports of its effectiveness and a case report of lack of neutralizing molecules. The presence of >80% of drugs is irreversible, log P may be an accurate predictor of a compound’s behaviour in the body. Chemists claim that the Distribution Coefficient (log D) is a more correct descriptor for lipophilicity with irreversible compounds (log D at pH 7.4) and irreversible compounds (log D at pH 7.4).

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

- Is the toxicant highly lipophilic as well as the toxol?
- Are the sodium ions (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 cases) has a log P value around 0.5, hence one would expect it not to be significantly negated by ILE therapy.

ILE therapy may be of value, especially in the local anaesthetic induced cardiac arrest situations:

1. Restoration of myocardic function by increasing intracellular calcium. The resultant inotropy increase may defeat the pharmacological actions of the calcium channel antagonist. The resultant inotropy increase may defeat the pharmacological actions of the calcium channel antagonist.

- Supplementation of cardiac energy supplies, as fatty acids are the primary substrate for myocardial ATP production. The local anaesthetics inhibit the enzyme carnitine–acyclic translocase thus depleting available energy, via impeding fatty acid transport into cardiac mitochondria. This fatty acid translocase may be overcome by ILE supplying large amounts of fatty acid substrate.

What does this all 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 candidates for therapy as their log D values range from 0.5 to <0.7, indicating high lipohelicity. Remember, that the ILE therapy may interfere with other therapeutic drugs used, e.g. the vasodilators (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 cases) has a log P value around 0.5, hence one would expect it not to be significantly negated by ILE therapy.

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1. Restoration of myocardic function by increasing intracellular calcium. The resultant inotropy increase may defeat the pharmacological actions of the calcium channel antagonist. The resultant inotropy increase may defeat the pharmacological actions of the calcium channel antagonist.
Inflammatory bowel disease in cats (IBD) is an important and relatively common 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 gut epithelial cells, large intestine, or stomach; in some cases, all parts of the gastrointestinal tract are affected. It results in recrudescent 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 cannot 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 owing to multifocal and a slowdown of movement of hair through the intestinal tract.

Causes/Transmission

By definition, the cause of IBD is 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 the way it’s going’. 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 found in abundant 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 is 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 a case with an endoscope and with an endoscope and with an endoscope, 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 ‘eosinophilic 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 kidneys, liver, and pancreas should also be ruled out. When a cause has been 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 hypertrophic 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. Instead of a low protein diet we use a modified diet. For example, a food trial is used to use a modified diet. In this diet the protein is furnished by a diet that contains a protein source to which the cat has not 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 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 be terminated. 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 steriod 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 sufficient 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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