SPECIAL ANNIVERSARY EDITION IV
Control & Therapy Series, December 2015 | Issue 281

Centre For Veterinary Education

CELEBRATING OUR FUTURE

CELEBRATING 50 YEARS OF SERVICE
TO THE VETERINARY PROFESSION IN 2015

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

C&T
CONTROL AND THERAPY SERIES

December 2015 – Issue 281

Australia's Leading Veterinarian Forum

PUBLISHER
Centre for Veterinary Education (CVE)
Tel: + 61 2 9351 7979
Fax: + 61 2 9351 7968
cve.publications@sydney.edu.au

www.cve.edu.au

Veterinary Science Conference Centre
Level 2, B22 Regimental Drive
The University of Sydney, NSW 2006
Print Post Approval No. 224792/0012

DIRECTOR
Hugh White BVSc MVSc MACVSc

EDITOR
Elisabeth Churchward:
elisabeth.churchward@sydney.edu.au

VETERINARY EDITORS
Richard Malik
Hugh White

ADVERTISING
Ines Borovic: ines.borovic@sydney.edu.au

CVE XMAS SHUTDOWN

The CVE closes down on Thursday 17 December 2015, reopening on Monday 4 January 2016.

CONTENTS

Back to table of contents

FROM THE EDITORS

Elisabeth Churchward & Richard Malik

2015 has been a terrific year for the CVE.

In our 50th anniversary year, we have recorded the highest-ever number of enrolments for our 2016 Distance Education (DE) program, but places are still available for most courses. In an increasingly competitive continuing veterinary education climate, we believe this increase is due to several key factors. Primarily, it is the calibre of our tutors – their expertise, experience and dedication. Between them, they have a wealth of corporate memory. They have used it to design, refine and fine-tune their programs. Added to this is the commitment and enthusiasm of everyone at the CVE involved in providing content, formatting, liaison and administrative support. Finally, it's due to the participants themselves. Word-of-mouth is the best advertising for us!

The changing demographics in the veterinary profession have been dramatic over the last 20 years. More women are graduating than men, by a ratio of over three to one, and more vets are working part-time due to balancing work and family, or pursuing additional interests. To cater for the change in demand, our traditional Time-Out face-to-face program has morphed into the digital Time-Online. The quality and variety of the tutors and content, plus flexibility and affordability, has seen Time-Online be enthusiastically embraced by vets, with lots of new topics and content for 2016.

Our 2015 Events & Workshops attracted excellent participation rates and we thank everyone who enrolled, listened and participated. Thanks also to colleagues at the Australian veterinary schools and Massey, NZ, for promoting our expanded 'Recent Graduate Survival Seminar' to their alumni and graduating class of 2015. Our 2016 March issue will announce the winners of the CVE Clinical Competency Awards at Australasian vet schools.

The unique *C&T Series* veterinary forum, so loved by the profession, is now in its 46th year and we have published over 5,500 C&Ts and 120 Perspectives. The Series continues thanks solely to the generosity of our contributors who write the articles and supply the images and videos for no recompense other than the satisfaction of sharing veterinary knowledge with their peers.

None of this success would be possible without YOUR support, whatever Member category you are: Practice, Professional, Part-Time, Recent Graduate, Student, Academic or eMember, and the support of our generous Sponsors from trade and industry. Together, your financial support for the CVE enables us to continue the work started 50 years ago by our first Director, Dr Tom Hungerford, and colleagues.

Thank you to everyone who is involved with, and supports, our work. An advantage of being a not-for-profit organisation is that profits generated are redirected back to the CVE to enable us to continue to provide relevant, quality and unbiased continuing veterinary education. Our milestone 50th year is drawing to a close but, as our cover boldly proclaims, we look forward to an exciting and successful future advancing the veterinary profession and helping you become a 'better vet'.

Season's Greetings and a Prosperous New Year to you and yours.

CALENDAR 2016

2016 Major Conferences

SYDNEY
Medical Imaging Conference + Brain Masterclass
Monday 22 – Friday 26 February, 2016

MELBOURNE
Valentine Charlton Feline Conference + Masterclass
Monday 20 – Friday 24 June, 2016

BRISBANE
Emergency Conference + Masterclass
Monday 24 – Friday 28 October, 2016

Seminars

SYDNEY
Sports Medicine: Theory & Practice
Friday 11 – Sunday 13 March, 2016

HOBART
Clinical Pathology Seminar
Sunday 13 March, 2016

ADELAIDE
Critical Care Seminar
Sunday 1 May, 2016

CANBERRA
Clinical Pathology Seminar
Sunday 29 May, 2016

PERTH
Feline Medicine Seminar
Saturday 23 – Sunday 24 July, 2016

SYDNEY
Ophthalmology: Theory & Practice
Friday 26 – Sunday 28 August, 2016

PORT MACQUARIE
Critical Care Seminar
Sunday 9 October, 2016

TOWNSVILLE
Feline Medicine Seminar
Saturday 12 – Sunday 13 November, 2016

January

Su	Mo	Tu	We	Th	Fr	Sa
31					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30

February

Su	Mo	Tu	We	Th	Fr	Sa
	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29					

March

Su	Mo	Tu	We	Th	Fr	Sa
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

April

Su	Mo	Tu	We	Th	Fr	Sa
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30

May

Su	Mo	Tu	We	Th	Fr	Sa
1	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28
29	30	31				

June

Su	Mo	Tu	We	Th	Fr	Sa
			1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30		

July

Su	Mo	Tu	We	Th	Fr	Sa
31					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30

August

Su	Mo	Tu	We	Th	Fr	Sa
	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30	31			

September

Su	Mo	Tu	We	Th	Fr	Sa
				1	2	3
4	5	6	7	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29	30	

Hands-on Workshops

SYDNEY
Anesthesia Workshop
Friday 12 February, 2016

SYDNEY
Diagnostic Ultrasound Workshop
Friday 26 February, 2016

SYDNEY
Hip & Stifle Workshop
Saturday 27 February, 2016

SYDNEY
Bone Plating Workshop
Sunday 28 February, 2016

SYDNEY
Approaches to Bones and Joints Workshop Friday 13 May, 2016

SYDNEY
Basic Echocardiography Workshop
Friday 7 October, 2016

SYDNEY
Advanced Echocardiography Workshop
Saturday 8 October, 2016

TimeOnline – Online CPD
PodcastPLUS

Check out our website for upcoming TimeOnline and PodcastPLUS topics...
www.cve.edu.au/timeonline
www.cve.edu.au/podcastplus

Calendar Key

- 2016 DE commences
- Major Conferences
- Seminars
- Hands-on Workshops
- TimeOnline start dates
- PodcastPLUS
- School holidays (NSW)
- CVE holiday closedown

CELEBRATING 50 YEARS OF SERVICE TO THE VETERINARY PROFESSION IN 2015

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

The C&T is the brainchild of Dr Tom Hungerford, one of the founders of the PGF* (established in 1965) and the first Director (1968-1987), who wanted a forum for uncensored and unedited material.

> ***'...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.'***

The first *C&T*, contributed by Dr R M Kibble from Kurring-gai Animal Hospital, Turramurra North, NSW was on 'Infertility – Uterine Conditions' and was published on 29 April 1969. CVE Members are reminded that this and other C&Ts, Perspectives, Proceedings and veterinary publications are available to CVE members through the CVeLibrary. Contact cve.enquiries@sydney.edu.au or call us at +61 2 9351 7979 if you've forgotten your Username and Password for access.

Thank you to all contributors

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

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

WINNERS

MAJOR PRIZE WINNER

Lessons Learned from a Chihuahua's Posterior: Adventures & Misadventures in Urogenital Surgery & Wound Healing, *Heather Shortridge*

CVE PUBLICATION PRIZE WINNERS

Entitling the recipient to a CVE proceedings of their choice: www.vetbookshop.com

Mycobacterial Infection in a Dog, *David Lee*

Vitamin K Responsive Dermatopathy, *Penny Reeves*

Oncology in the Cat, *Xiangting (Kitty) Huang*

*The Post Graduate Foundation in Veterinary Science of The University of Sydney (PGF) was renamed the Centre for Veterinary Education (CVE) in 2008.

Thank you to our C&T industry supporters

Your financial support sponsors the production of the *C&T Series* in high quality colour print format as well as the complementary digital eBook version (facilitating the inclusion of film clips, downloads, rollovers and enlarging of images) and postage costs.

Boehringer Ingelheim

Sponsor since 2011 www.boehringer-ingelheim.com.au

Virbac

Sponsor since 2011 www.virbac.com.au

asfm australasian society of feline medicine

www.asfm.com.au

B|BRAUN SHARING EXPERTISE

www.bbraun.com.au

BOVA Your prescription, your way.

www.bovavet.com.au

ceva

www.ceva.com.au

iM3® The Veterinary Dental Company

www.im3vet.com

MSD Animal Health

www.msd-animal-health.com.au

RADINCON X-RAY

www.radincon.com.au

PROVET A Henry Schein Company

www.provet.com.au

CVE NEWS

THE AUSTRALIAN RHINO PROJECT GALA DINNER

16 September, 2016

Anne Fawcett

CVE Member

E. fawcettanne@gmail.com
W. www.smallanimaltalk.com

Left to right: Dr Chris Brown, Ray Dearlove, Margaret Dearlove, Peter McLoughlin & Dr Pete Morkel. Photo courtesy of Phil Hines, phil@philhinesphotography.com.au

If The Australian Rhino Project's inaugural Gala Dinner is anything to go by, the ambitious project will be a roaring success.

The Australian Rhino Project (TARP) was formed in 2013 with the goal of establishing a breeding herd of rhinoceros in Australia as insurance against extinction of rhinoceros species in South Africa. The biggest threat to rhinos is poaching. It is estimated that one rhinoceros is poached every 8 hours, an unsustainable level that will see rhino species extinct within our lifetime.

TARP aims to transport up to 80 rhinoceros to Australia, breed them and eventually return them to South Africa. The not-for-profit organisation has partnered with Investec Australia, Taronga Conservation Society, the Faculty of Veterinary Science and Business School at the University of Sydney, Zoos South Australia and the Classic Safari Company.

Master of Ceremonies Dr Chris Brown encouraged guests to throw their support behind the project, and introduced the many special guests of the night. TARP Founder Ray Dearlove discussed the challenges, including securing in principal support from the Australian Government and the logistics of moving mega-beasts across the world.

Dr Jane Goodall OBE could not attend, but shared a pre-recorded video with guests, thanking them for supporting the initiative. *'It is such an important initiative, the Australian Rhino initiative,'* she said. *'I've seen the decline of rhinos in Africa and its absolutely shocking, they are critically endangered now, and it's just because people in Asia, particularly in Vietnam, feel there is medicinal value in their horns. Do you know some people even take the rhino horn powder for hangovers? That's just appalling.'*

'There is an awful lot of poverty in Africa, and we need to alleviate that poverty. We need people to withstand bribes and if they're really poor they won't. We need to educate, we need to help the people and governments understand that these animals are worth more alive than dead. The tourist industry is really providing an awful lot of income for many of these African countries.'

Speakers included Tim Jarvis AM, an environmental scientist, author and adventurer whose extreme exploits have been documented on the Discovery Channel. He talked about the challenges of 'big' projects – and the need to assemble the right team. His description of re-enacting Shackleton's 1916 Journey – including the risks of falling through ice and living off blubber for weeks at a time – was a reminder that the seemingly impossible is possible with the right team.

Veterinarian Dr Peter Morkel, who through the course of his career has worked in 16 African countries and is on the IUCN African Rhino Specialist Group Executive, discussed his experience with rhinos – including seeing several subspecies become extinct in his lifetime.

Dr Morkel, who has a long-standing interest in the physiology and pharmacology of immobilisation of large animals, described the unique challenges of rhinoceros anaesthesia. These include practical challenges such as anaesthetic monitoring to potential complications like compartment syndrome. He described the devastating nature of injuries (often fatal), suffered by rhinoceros attacked by poachers.

It is estimated that it will cost approximately $100,000 to transport a single rhinoceros from South Africa to Australia. The aim of the dinner was to raise at least $150,000. Ray Dearlove confirmed that 650 people attended and *'we exceeded our expectations in terms of fundraising – people were extremely generous...'* By the end of the night, between a silent auction and a nail-biting auction run by James Kennan, well over $300,000 had been pledged. The most vigorous competition was for a one-in-a-lifetime place on the B777F cargo plane which will transport the animals from Johannesburg to Sydney.

The date of that journey is yet to be finalised. ■

CVE NEWS

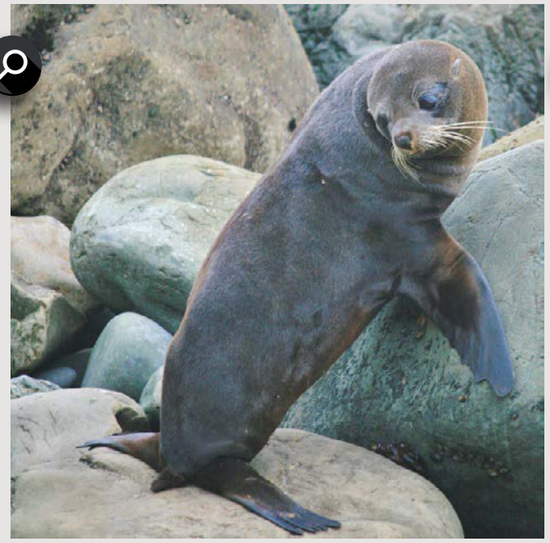
BEST WILDLIFE PIC FACEBOOK COMPETITION

Emily Ainsworth was the winners with the most ‘likes’ and won tickets for 2 for the Save the Rhino Project gala dinner.

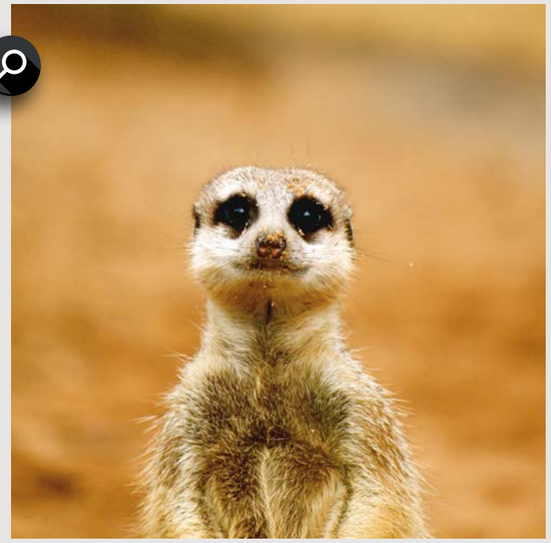
2nd and 3rd respectively appear below. All 3 winning photos will be mounted and displayed in the CVE offices.

Thanks everyone for your support.

CVE Team



2nd Place: Catherine Purvis with ‘Having the sealiest time’.



3rd Place: Georgia Englund with ‘Meerkat Manor’.

1st Place: Emily Ainsworth with ‘Elephant Sedation – Bush Style’.

FROM THE ISFM FORUM

WHAT IS YOUR DIAGNOSIS?

Pete Coleshaw

Jaffa’s Health Centre for Cats
52 St Francis Road Salisbury UK

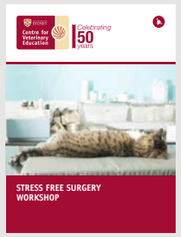
P. 01722 414298
E. jaffa@jaffavets.com
W. www.jaffavets.com

C&T NO. 5502

This cat had a pruritic skin condition with most irritation centred on the head.

Question:

- **What is your diagnosis?**



Please email your answers to: elisabeth.churchward@sydney.edu.au

The vet with the most complete and correct answer wins a CVE proceedings of their choice.

Go to: www.vetbookshop.com to peruse our list of titles.

Figure 1. What’s your diagnosis?

CVE DIAGNOSTIC IMAGING

WHAT IS YOUR DIAGNOSIS?

Robert Nicoll

BSc (Vet) BVSc DACVR

C&T NO. 5503

Prior to specialising in diagnostic imaging, Dr Robert Nicoll worked in mixed veterinary practice in Bathurst, NSW for several years. After undertaking his residency training at the University of Wisconsin, Madison, USA, he returned to Australia. With Graeme Allan he formed Veterinary Imaging Associates and more recently, their teleradiology practice Online-Vets.com, providing an international diagnostic service. Since 1998, Robert has been an associate tutor with Graeme in the Diagnostic Imaging Distance Education course and has worked with Graeme on developing a special digital radiography stream for those who have made or are looking to make the leap into filmless radiography.

Signalment: 10 weeks old female Kelpie crossbred.

History & Clinical Findings: Sudden onset of lameness 3 days ago and not weight bearing on hindlimb since. Currently only toe-touching but not bearing any weight on the limb. Mild pain response when limb palpated and extended.

Views: Left hind leg – lateral and craniocaudal

Questions:

- What radiographic changes/abnormalities are evident in the two views provided?
- What is your diagnosis?
- What would you do next?

VIEW SOLUTION

Or download the full article

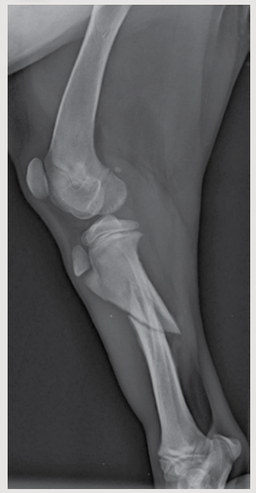


Figure 1. Craniocaudal left hind limb.

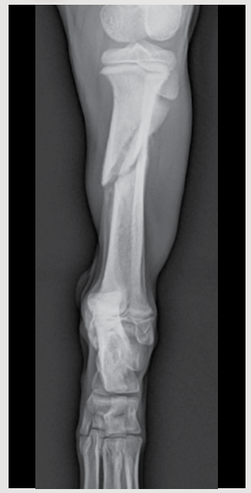


Figure 2. Craniocaudal left hind limb.

LARGE ANIMAL

COMMENT FROM AUTHOR OF PERSPECTIVE NO. 29

UTERINE PROLAPSE IN CATTLE *C&T SERIES*, OCT 1999

Geoff Manefield

BVSc, MSc

E. geoff.manefield@bigpond.com

C&T NO. 5504

I have noted the reference to the 'both hind legs backward' technique for reinversion of the prolapsed bovine uterus. It is of very significant help in cases in downer cows.

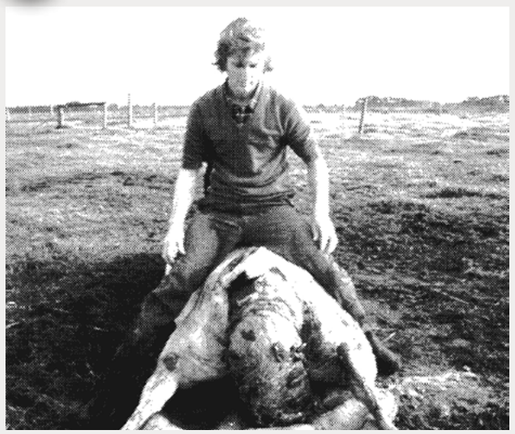
When I first described the technique Dear Old Uncle Tom Hungerford received reports from so many happy veterinarians that he concluded, and wrongly wrote, that I put standing cases down to use the leg back position.

Since the technique in downers is to produce some intra-abdominal space, and reduce the naturally positioned animals' antagonistic expulsion power, I have never found it necessary to put the standing cow down.

In fact, in the standing case, since even any activity in excess of gentle walking can cause rupture of middle uterine, and/or other involved, artery with fatal consequences, I believe that putting the cow down is contra-indicated. Standing cases should be walked very patiently to a crush and bail, or some other structure that will allow appropriate gentle restraint, and the prolapse dealt with in the standing animal. ■

Editor's Note: *Thanks to Geoff for this clarification which he has provided in response to the reference to his Perspective 29 mentioned on page 13, Sept 2015 Issue 280.*

New CVE Members/Readers are invited to read Perspective 29 in the complementary eBook.



The Manefield Inverted Uterus Technique.

LARGE ANIMAL

ANSWER TO C&T NO. 5482 WHAT IS YOUR DIAGNOSIS?

Mandy Lugsdin

Ovens & Kiewa Veterinary Hospital
79-81 Myrtle Street, Myrtleford VIC 3737

T. (03) 5752 1235
E. care@ovensandkiewavets.com.au
W. www.ovensandkiewavets.com.au

C&T NO. 5505

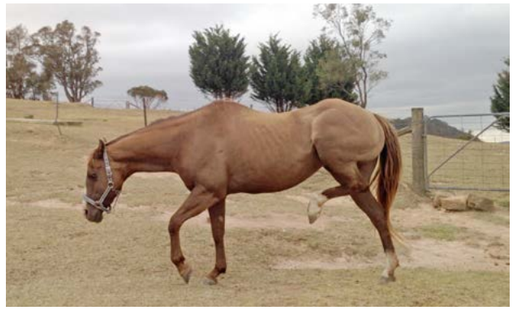


Figure 1. Starlet.

What is your Diagnosis: Australian Stringhalt

Aetiology: The condition of stringhalt has been attributed to many factors over the years. The prevalent theory is that stringhalt is a neurological condition, induced by a toxin which affects the nervous system.[2] The exact aetiology of the disease is still obscure, however the involvement of False Dandelion (Hypochoeris radicata) or an associated mycotoxin appear most likely aetiological influences.[2] The combination of the likely presence of this weed, poor quality pastures, the timing of a dry summer and drought conditions lead to a highly suggestive presumptive diagnosis. When the features of the clinical exam are considered, it is likely drought conditions especially force horses to become indiscriminate grazers. Other plant species may be involved (sheep's sorrel (Rumex acetosella) and couch grass (Elymus repens)).

Treatment: Proposed treatment of stringhalt includes rest, removal of animals from affected pasture, lateral digital extensor myotenectomy, and medical treatment using phenytoin (may not be on the market for equine use anymore), mephenesin (central muscle relaxants) or baclofen.[2] Most authors recognize that the majority of horses will recover spontaneously without treatment, however, there is a known protracted recovery period, taking place over a few weeks up to over twelve months.[1] A spontaneous recovery is dependent on the ability of the axons to regenerate. In severely affected cases, larger myelinated fibres become affected and thus a longer regenerative process is likely, which potentially may be an incomplete process.[3] If grazing a particular area of pasture has produced the disease in the past, it is recommended to avoid using this paddock throughout the summer and autumn months, especially in drought like conditions.[2] An alternative would be to consider pasture renovation. Mild and early cases have been reported to especially benefit from removal from pasture and conservative treatment.[3] In a study undertaken between 1991 and 2003 of 13 horses, complete resolution of clinical signs was seen in 11 of 13 horses studied who were treated by myotenectomy of the lateral digital extensor muscle and tendon.[3] Sometimes this condition does not resolve, however this is impossible to predict at this time. In extreme cases that become recumbent, the necessitation of euthanasia may be the only feasible option. ■

References:

1. Barry, W.C. (1956). The incidence of Australian Stringhalt in horses in New Zealand. *N.Z. Vet. J.* 4: 26-27.
2. Cahill, J.I., Goulden, B.E. and Pearce, H.G. (1985) A review and some observations on stringhalt. *N.Z. Vet. J.* 33: 101-104.
3. Torre, F. (2005). Case Report: Clinical diagnosis and results of surgical treatment of 13 cases of acquired bilateral stringhalt (1991-2003). *Equine Vet. J.* 37: (2) 181-183.
4. Church, S. (2007) The University of Melbourne – EQUINE 2 – Lecture notes. Page 30.

Editor's Note: *Thank you to all the vets who emailed an answer to this C&T. Mandy's answer was judged to be the most complete and therefore she is the winner of a proceedings of her choice. See vetshop.com.au for a list of CVE titles available.*

If you have a great photo or image suitable for the 'What's YOUR diagnosis?' column, please email it to: elisabeth.churchward@sydney.edu.au

---

Comment from Richard Malik, CVE

We own a 300 acre property on which we run a menagerie of small ruminants and about 20 horses, give or take a donkey or two. 'Starlet' – the old mare in the photo – was given to me by a small animal client who bred quarter horses because a uterine infection had made Starlet infertile. Starlet is broken to halter, but not to saddle – and she just takes it easy as the oldest horse on the property. About 2 years ago, we had a very dry spring and there wasn't much standing feed. A yellow weed implicated in causing stringhalt was abundant, although the horses did not obviously seek it out. The onset of stringhalt was apparently abrupt; either that, or I am a very poor observer. Starlet was moderately affected, as per the video. My Arabian gelding 'Aladdin' was very mildly affected, really only noticeable during backing. All the other horses were unremarkable.

Starlet was treated in a standard manner. We moved her to a home paddock, and started feeding her lucerne hay. Aladdin came in for a few days, but then he seemed normal, so he went out with the others. I did a GOOGLE search to re-familiarise myself with the literature, and elected to treat Starlet with phenytoin, rather than the alternative of baclofen (which, ironically, I had done some work on many years ago with Nick Kannegieter). BOVA Compounding was able to post me Dilantin capsules by express post within 48 hours, and I used a dose first recommended by the Werribee group. The phenytoin was first given in bread, and was subsequently sometimes mixed in with horse pellets, 'Breed and Grow' and lucerne chaff; it has an unpleasant taste, but this can be masked by dilution. Within 24 hours the extreme hyperflexion characteristic of stringhalt was less evident, and she moved better and seemed more comfortable. We continued the drug for several weeks, kept her in for 2 months, and she made a complete recovery. Aladdin's signs never returned, and we got good rain in summer, which gave the horses good standing feed. I spent many hours with the mower getting stuck into the worst patches of the yellow weed. Good for cardiovascular health!

We currently have another horse with stringhalt – 'Bentley' – a retired thoroughbred. His signs seem worse in winter and improve in summer. When his signs are at their worst, we add phenytoin to his rations.

I have also seen stringhalt in association with nasal neoplasia in a retired blood donor we took on from the Rural Veterinary Centre, Camden, presumably as a paraneoplastic neuropathy. Maybe that's Bentley's problem, and we just haven't found a tumour. Or maybe he has an old age, dying back neuropathy.

The diagnosis of stringhalt is a classic 'illness script', but it's important to see if environmental conditions are consistent, and that there isn't any other potential cause of peripheral nerve dysfunction. The main aspect of treatment is removal from the suspect pasture areas, but there is no doubt that drug therapy ameliorates the signs, and that's good symptomatic relief which is a positive welfare outcome. Starlet is still alive and enjoying her old age. I needed to buy a new mower. ■

If you missed the video, view it in the complementary eBook available at: www.cve.edu.au/candtebook

INTERESTING LINK

Dr Lydia Tong has shown vets how to tell the difference between bone fractures caused by accidents and those caused by abuse.

Pet abuse and domestic violence are closely linked. Dr Tong's fracture identification methods are giving vets the added confidence to identify cases of violence against pets and could serve as a warning of domestic violence.

http://sydney.edu.au/news/84.html?newsstoryid=13915

WILDLIFE

REHABILITATION OF AN EASTERN GREY KANGAROO

Colo Heights Wildlife Clinic and Refuge
P. (02) 4565 0323
E. macmcg@spiderweb.com.au

Jim Phelan
BVSc, Dip Wild Med

Christine McGregor
Vet Nurse & Wildlife Rehabilitator

C&T NO. 5506

Before commencing this case history, I would like to thank Jim Phelan as the attending veterinarian. Without his selfless dedication in donating his time and service to our injured and orphaned wildlife, cases like Carl's might never have been considered for treatment. Also, heartfelt thanks to Peter Burgess BVSc, Linda Zapletal BVMS, BSc (Hons), MRCVS and finally Rosemary and Steve Garlic, all of whom who have made their time available, often after-hours, to share their advice and experience.

The Colo Heights Wildlife Clinic was started by Alan Warner BVSc MAppSc (Wildlife Health) MANZCVS (Avian Health) along with myself, Chris McGregor Vet Nurse and Independently Licensed Wildlife Carer, and my wonderful long-suffering partner Michael Adams. With Alan's sad passing in 2013, Jim has taken over as attending veterinarian.

We are totally self-funded, receiving no donations, grants or subsidies; hence we make no profit. We offer a 24 hr call-out service in our area for all injured and orphaned native wildlife with the exception of marine mammals and reptiles. This is fortunate as Colo Heights is a damned long way from the sea!

So many wildlife cases are deemed too difficult, too costly, or too time-consuming that the 'easy' answer is frequently euthanasia. Here at the Colo Heights Wildlife Clinic and Refuge, all cases are assessed and treated on an individual basis. If the animal has a chance of recovery sufficient to permit eventual release, we attempt treatment and rehabilitation unless the prognosis is hopeless.

CARL'S STORY

We received a call-out at 8.00 pm on 14/03/2015. On arrival, we found 'Carl', and Eastern Grey Kangaroo (EGK) with an open wound to his right hind leg with the tibia exposed. Nevertheless, he was still able to hop faster than we could run. With the help of some locals we were able to capture him with a minimum of chasing.

He was a wild 10 kg Joey at foot but no sign of his mother or the rest of his mob. According to locals, he had been at the area for at least 12 hrs. Once captured, he was immediately given Valium® 20 mg and Tramal® 25 mg IM and transported for assessment. The wounds were considered to be most consistent with a dog attack with some remaining damaged skin tissue showing evidence of bite wounds.

The muscle and other tissue surrounding the exposed tibia was grey and showed no sign of active haemorrhage but was reasonably clean. The skin wound was 50 mm long with tibia and tendons exposed. There was some gas under the skin in the area of the thigh. Most likely this was traumatically-induced subcutaneous emphysema. The wound was flushed with saline then a weak solution of Iovone® surgical scrub and again flushed with a saline. Sulfadimidine powder was sprinkled onto the wound and covered with Jelonet® then sterile swabs moistened with normal saline were applied.

Cotton gauze bandage, cotton wool, Krix® cotton bandage and finally Vet Wrap® with a small amount of Elastoplast® were used to secure the top and bottom of the bandage to the skin. 0.25mL of Benzyl® penicillin was given I/V along with 1.0mL Betamox® LA (Amoxycillin 150 mg/mL) IM. An unrecorded volume of Hartmann's both IV and SC was given and the patient was warmed on a large heating pad in the hospice. He was supplement fed 3-hourly with 50mL Biolac® M200 milk formula at standard concentration via bottle and teat and was supplied with lucerne hay, fresh grass and water ad lib. Four drops of Tramal® oral drops (100 mg/mL) tid were mixed in the milk as needed.

15/03/2015

6.00 pm: Toes were observed beginning to swell distal to the dressing. He was sedated with 2.0 mLs of Valium® 5 mg/mL injectable by IM and we proceeded to general anaesthesia then he was masked down with Isoflurane® initially at 4.0 % and maintained at 1.0- 1.5%.

The bandage was removed and replaced essentially identical to the original. 0.7 mL 'Equivac TAT' tetanus antitoxin was given SC on the left flank plus 0.3 mL '5 in 1' vaccine SC on the right flank.

The prognosis appeared very poor at this point due to the extent of the wound and exposure of bone. It was difficult to estimate if healing could ever occur in terms of both degree and time required. Even if all went well, it could take many months. Because Carl appeared to have a calm temperament and was already settled in, it was decided to continue with treatment. As with all injured larger joeys and adult macropods, an inside enclosure is required. If they can be kept without any visual or other stimuli originating from the outside environment they tend to remain remarkably calm and accept nursing in most cases. Modecate® (fluphenazine) mg/mL) can be administered IM as a long-acting sedative/ neuroleptic agent in these circumstances but in Carl's case this was not needed

17/03/2015

Bandage changes were carried out under anaesthesia every second day. A small amount of necrotic tissue was debrided around exposed bone. The appearance of the bone suggested it was viable. At this stage, debridement was kept to a minimum as the soft tissue present provided at least some protection for the underlying bone, despite the fact that some of that soft tissue would eventually be lost through necrosis.

The protocol for treatment was established in an ad hoc fashion and remained largely unchanged till complete healing had occurred. In summary, that protocol was:

- Pre-medicate with IM Valium® (0.5-1 mL) and SC Atropine® (1.0 mL standard dose). The dose of Valium® was varied based on the animal's clinical condition at the time.
- GA induced and maintained with oxygen/isoflurane (4.0 reduced to 1.5%) by mask.
- All bandage changes were performed under conditions aimed at maximising asepsis. Gloves, drapes and all in-contact dressing materials were sterile.
- Flush wound with saline and then weak solution Iovone® and swabbing any loose necrotic tissue followed by a second flush with saline.
- Solosite® Gel – clear colourless used to hydrate and protect exposed bone.
- Sulfadimidine powder on surrounding soft tissue.
- Flamazine® ointment (1% silver sulfadiazine) on surrounding tissue.
- Jelonet® – swabs used as first layer in direct contact with exposed tissues.
- Sterile swabs soaked in saline over the Jelonet® swabs.
- Bandage materials remained the same as per initial dressing.
- Betamox® LA was administered every second day at 1.0 mL per 10 kg BW.
- Tramal® oral drops bid or tid as needed – dosage as per previous.

19/03/2015

At this dressing change, the surface of the bone appeared to be drying out. A bit of creativity was employed here by making do with what's available. A human neonatal nasogastric tube was cut to the right length to stretch from thigh to tibia. The distal end was sealed with heat and the tube was perforated with small holes (via a heated needle) in that section which covered the area of exposed bone. The top of the nasogasteric tube had a flip closure which prevented contamination. The tube was sutured to the sterile saline soaked swabs which were part of the usual dressing.

This tube was flushed with saline 5mL BID without removing any part of the dressing, thus helping to keep the exposed bone moist. This seemed to work well. The modified tube was flushed with Iovone® solution at each bandage change and re-sutured in place.

23/03/2015

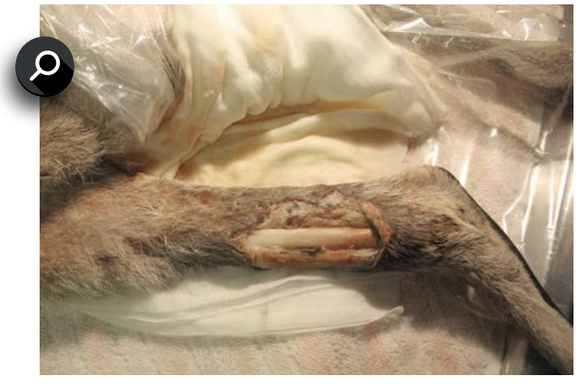


Figure 1. Wound post clean-up Day 10.

31/03/2015

Acute onset diarrhoea of uncertain cause. Treated with single dose of 5.0 mLs Baycox® PO in case of possible coccidiosis based on previous similar experiences. The question of intestinal coccidiosis in juvenile macropods is not entirely understood but experience suggests that any acute diarrhoea in this type and age of animal should include coccidiosis in the differential diagnosis and prompt therapy is always likely to be of more clinical benefit than any delay that awaits diagnostic confirmation.

01/04/2015

This date appeared to correspond to the time when the size of the skin defect attained its maximum, measured at an

oval shaped area approximately 90 mm x 40 mm. Bandage changes were done every second day until this date after which time the interval was reduced to every third day.

Necrotic tissue continued to either slough unaided or was debrided until both lateral and medial aspects of the distal tibia were exposed. Connective tissue then began to appear; one unidentified tendon was lost but the subsequent long-term ability to use the leg was not adversely affected.

7/04/2015

We first observed the beginnings of the development of a peculiar creamy mucoid tissue that first formed at the junction of the skin margin and its attachment to the exposed bone. Over the next few weeks, this unidentified material proceeded to migrate over more of the exposed bone till the covering was eventually complete. It also seemed that there was a gradual inward migration of what looked very much like fine blood vessels. *(**Author's Note:** We have no idea what this material or tissue is and our own research has not been fruitful. I speculate it may be something which has developed in conjunction with our use of the Flamazine® gel and/or the Solosite® gel but have no proof. Perhaps someone can enlighten us?)*

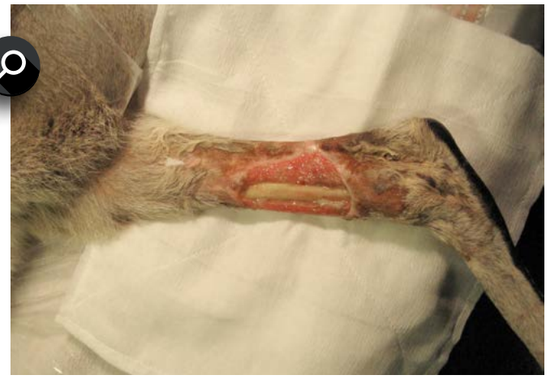


Figure 2. Mucoid tissue starting to develop Day 25.

19/04/2015

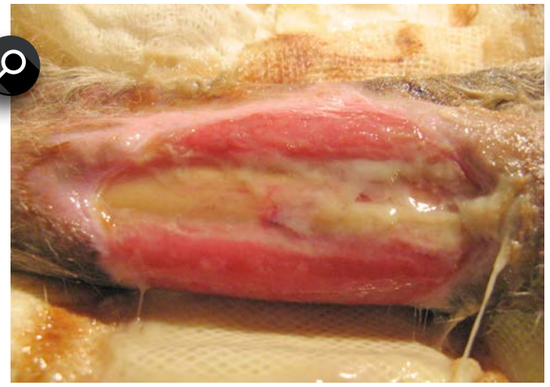


Figure 3. First appearance of blood vessel formation Day 37.

1/05/2015

Reduce bandage changes to every 4 days.

5/05/2015

Leg looks good. The bone is now completely covered with creamy mucoid tissue, Blood vessels are now starting to grow through this tissue (as previously mentioned).

9/05/2015

Something similar to granulation tissue is starting to grow over mucoid tissue. Betamox® LA now only given at bandage change rather than every second day.

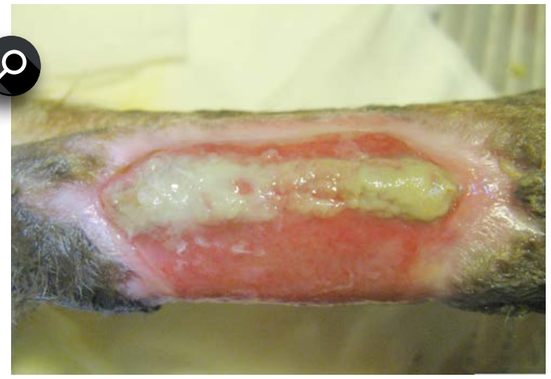


Figure 4. Granulation tissue growing through remaining mucoid tissue Day 57.

25/05/2015

Granulation tissue completely covering bone. Edges of skin starting to close in over the wound approximately 7.0 mm all around. Leave bandage change to 5 days.

5/06/2015

Looks very good. Leave bandage changes for 7 days.

27/06/2015

Decided from today to only use Solosite® and Flamazine® under Jelonet®. No injectable antibiotics from here.

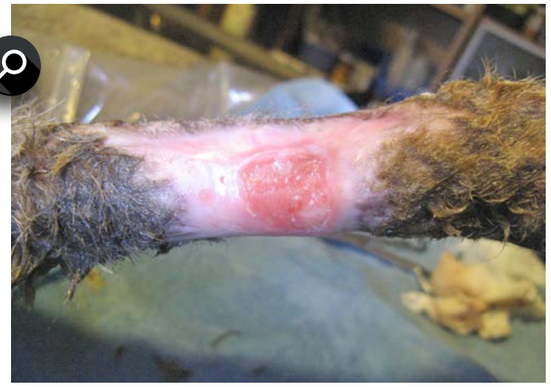


Figure 5. Wound continues to heal, no injectable antibiotics. From here Day 106.

4/07/2015

Size of wound is now reduced to 10 mm diameter. Anaesthesia no longer necessary for bandage changes. Now carried out after light dose of Valium®.

11/07/2015

Wound now only 5 mm diameter. Opt to change to Wound Gel® (available at any pharmacy) which contains Manuka honey along with Solosite® under Jelonet®.

19/07/2015

Wound is now fully healed. Continued to bandage with a light dressing for a week only to protect new hairless skin from inadvertent or self-inflicted damage.

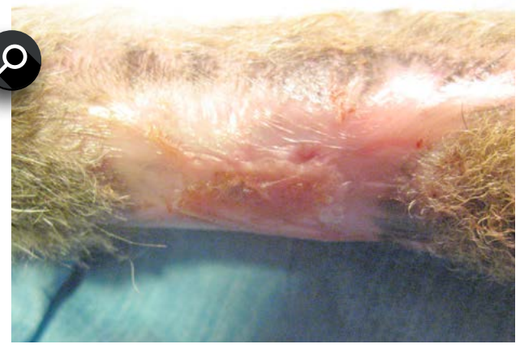


Figure 6. Wound has total coverage Day 128.

FOOTNOTE:
Chris McGregor – Vet Nurse & Wildlife Rehabilitator

Carl is now undergoing rehab to build up muscles by exercising in a large compound during the day and returns to the hospice at night. In a short time he will be bonded into a mob of approximately 5 joeys, all of which will continue to be in care until they are approximately 20 kg. (This is the normal time EGKs become totally independent of their mother). At this time, they will be soft released into the wild on our property at Colo Heights.

Hopefully, they will return from time to time as nearly all our similarly rehabilitated macropods do. These EGKs behave just as their normal wild relatives do and will not allow anyone to approach them. They will head for the bush if they see a human. Most will not even come near my partner. However, they do retain a bond with me (their 'mother') and will come and greet me. I have even had one return after living unseen by me in the wild for 3 years. She came to me, we touched noses and she licked my face. However, she would immediately leave if anyone else approached.

Incredible animals… and yet, as proud Australians, with apparently little respect for one of our major national icons, we hunt them, we shoot them, we eat them and export their flesh for consumption? Perhaps they deserve better from us. ■

FREE VETERINARY RESOURCE

Please read this open-access article and to share it with colleagues. Go to: http://dx.doi.org/10.1016/j.cimid.2015.07.004

Westman ME, Malik R, Hall, E, Sheehy P & Norris JM, Oct 2015, Determining the feline immunodeficiency virus (FIV) status of FIV-vaccinated cats using point-of-care antibody kits, *Comparative Immunology, Microbiology & Infectious Diseases*, **42** (2015) 43–52.

***Note:** The CVE funded production of the images in colour to enhance the educational value of this article.*

Comment courtesy of Mark Westman & Jacqui Norris at the Veterinary Faculty of The University of Sydney

The introduction of the FIV vaccine in 2002 complicated feline immunodeficiency virus (FIV) diagnosis because FIV vaccination reportedly resulted in the production of antibodies to FIV indistinguishable from those produced in response to natural FIV infection. In other words, veterinarians were informed that FIV-vaccinated cats would test positive using point-of-care FIV antibody test kits, irrespective of their actual FIV infection status. For this reason, diagnosis of FIV infection in FIV-vaccinated cats and cats of unknown FIV vaccination status shifted towards more expensive molecular methods such as nucleic acid amplification (PCR).

In our recent study of Australian FIV-vaccinated client-owned cats, we found that the FIV infection status of FIV-vaccinated cats was accurately assigned using two inexpensive, fast, simple to use, antibody detection kits made by different manufacturers (Witness FeLV/FIV and Anigen Rapid FIV/FeLV). Anigen Rapid reported no false positive results (i.e. correctly identified 114/114 FIV-vaccinated/FIV-uninfected cats), while Witness reported a small number of false positive results (6 false positive results; i.e. correctly identified 108/114 FIV-vaccinated/FIV-uninfected cats). SNAP FIV/FeLV Combo could not distinguish FIV-vaccinated from FIV-infected cats, reporting 114 false positive results. Where FIV vaccination is practiced, we suggest either Anigen Rapid or Witness should be used for initial screening of FIV infection, particularly in shelters where large numbers of cats need to be assessed quickly and affordably and where vaccination history is often unknown. Since no diagnostic test procedure is 100% accurate, we still recommend confirming any positive result using the other antibody test kit or by PCR assay. ■

DISTANCE EDUCATION / FELINE MEDICINE

FROM THE ISFM DISCUSSION FORUM **TREATMENT OF POST-OBSTRUCTION DETRUSOR AGONY?**

The Cat Doctor
21 Westleigh Avenue, Coulsdon
Surrey, United Kingdom

Amy Bergs

E. info@thecatdoctor.co.uk

C&T NO. 5507

The CVE is grateful to all the vets who allow us to publish email correspondence from the ISFM and DE listserves. Readers should bear in mind that the CVE is a unique forum and that these C&Ts originated as 'informal' exchanges intended for the recipients in the emails. Care should be taken not to 'skim read' and misinterpret these exchanges.

I have a 4-year-old male neutered DSH who is currently unable to urinate on his own and was wondering if you might be able to help?

He has a history of FLUTD, managed initially with urinary wet food, until the owner decided to discontinue it. Did well for 2 years, then had to move house. Two days after the move, the owner came home to find the cat practically non-responsive. Instead of calling the vet, she gave him Metacam® and went to bed. By some miracle, he was still alive the next morning and gradually started eating and drinking again and acting near normal by the next evening. However, he started to go in and out of the litter tray, trying to urinate, and licking his rear end constantly. A full 3 days later, the owner finally rings the vet as he is leaking urine all over the house. The only other complaint was that he was reluctant to run or jump, otherwise perfectly normal in himself.

When I saw him, his bladder was the size of a tennis ball, though not entirely firm as a typical blocked cat. He was not dehydrated, and otherwise well in himself (aside from what appear to be rodent ulcer-type lesions on his lip margins...), good body condition, bright and responsive and chatty. Good anal tone, colon palpated normal, gait and tail all normal. He is apparently a very stoic cat, as this couldn't have felt good.

He was unblocked with great ease, no resistance to catheterisation under sedation, bladder was flushed and catheter was pulled as the unblocking had been so easy (which I regret in hindsight), subcutaneous fluids given. Blood sample taken, but still pending as over the weekend so will likely just repeat. Urine showed no crystals in-house (though at his last incident a few years ago, he had lots of struvite present) and USG 1.036, otherwise unremarkable, going out for full analysis and culture tomorrow. Kept in hospital for observation and began buprenorphine and prazosin.

Twenty-four hours later, he was showing no efforts to use the litter tray (tried multiple substrates), and did not seem at all uncomfortable. Bladder gradually getting bigger. Now, about 30 hours after unblocking, he is starting to lick and leak urine again (the licking stimulates a visible trickle of urine). Attempts to manually express have been unsuccessful despite a pretty well-behaved cat. Eating and drinking well, temperature, pulse and respiration unremarkable, active and chatty and purring and rolling for rubs.

Diagnosis

I suspect his initial non-responsive episode was actually a blockage, and that he is now suffering from post-obstruction detrusor atony.

Plan

1. Continue prazosin and buprenorphine.
2. Re-sedate to insert urinary catheter and this time suture in place for 2-3 days to allow bladder muscle to heal.
3. Add in bethanechol to stimulate bladder contraction.
4. Submit new bloods, add in IV fluids if any sign of azotaemia though not currently dehydrated.
5. Await urine culture results, add in antibiotics only if indicated.
6. Speak seriously with owner about: management of FLUTD and the seriousness of this condition, returning the cat to a wet urinary diet.

Questions

1. I know X-rays to check for stones would be ideal, but is it necessary? Would require transporting him to a different facility and money is becoming an issue. Urine was always clear yellow, no blood.
2. Am I missing anything, or would you recommend I change my current plan at all?
3. How long is this likely to last, and is there any way to tell if it is improving or do we just pull the catheter and hope for the best?

I've never seen one of these before so any comments would be appreciated, thanks.

DE / FELINE MEDICINE

REPLY

Richard Malik

CVE Valentine Charlton Consultant

Centre for Veterinary Education
The University of Sydney

E. richard.malik@sydney.edu.au

C&T NO. 5508

Many people have said sensible things, but perhaps to be controversial, may I chip in?:

1. My own view is when the bladder is stretched – the damage is done, and you need to wait for the gap junctions to heal between the smooth muscle cells in the bladder wall.

2. I have never been convinced that any of the drugs work (at the level of the bladder wall smooth muscle), and I have read a recent comment from Lulich and Osborne to that effect, which makes me more bold in saying what I have felt for years.

3. My own experience is that although the bladder smooth muscle is in terrible shape, the urethral sphincter still can work well, too well! – which makes expressing them hard; for this reason, using a benzodiazepine e.g. midazolam can make expressing the bladder much easier. If you are worried about Valium and idiosyncratic liver necrosis, then use midazolam 0.3 mg/kg subcutaneously – works in about 5 minutes – and they eat very well when they stop being wobbly. Another way is to mask them down with isoflurane or sevoflurane – express the bladder, and then wake them up.

4. Long term catheterisation or urinary diversion (cystostomy tube) are ideal – although there is always risk of ascending infection. In people, they can leave indwelling catheters in for years – although of course in people it's easier to change them and keep them clean and so forth.

5. People have experimented with percutaneously inserted bladder catheters – but there currently seem to be too many complications – see recent article from the Davis group in *JFMS.*

6. Finally, although FAR FROM IDEAL – for certain owners where money is an issue and long term hospitalisation is not acceptable, leaving an indwelling urinary catheter *in situ* for 1-2 weeks (with an E-collar in place) and the cat in a restricted area with clay litter or foam pockets for litter – is a way that lets the bladder heal; ascending bacterial infection is common – but you can start antibiotics based on culture and susceptibility **after** you remove the catheter. Ideally, use a gentle silicone catheter that is long enough to get into the bladder. Catheter length is important, as in big cats – you need a LONGER catheter. The Slippery Sam and MILA brands are both excellent.

7. I agree about wet food diets, although if there are crystals in the mucus plug I favour canned Hill's s/d® or the like for the first few weeks. ■

Post-script from Amy:

It took a further 7 days in hospital for his bladder function to return enough for him to go home under close supervision but then he did very well and had no further complications. I was most impressed by the fact that he managed to clear his initial blockage - he's lucky to be alive! Some 18 months later, the cat is now doing brilliantly, his owner has followed all of our advice and he even survived a move to Germany without issue.

The CVE & ISFM jointly run the Feline Medicine DE program. See www.cve.edu.au/de/feline-medicine

DISTANCE EDUCATION / FELINE MEDICINE

FROM THE ISFM DISCUSSION FORUM
CAT BODY CONDITION ASSESSMENT & WEIGHT LOSS NIGHTMARE

C&T NO. 5509

The CVE is grateful to all the vets who allow us to publish email correspondence from the ISFM and DE listserves. Readers should bear in mind that the CVE is a unique forum and that these C&Ts originated as 'informal' exchanges intended for the recipients in the emails. Care should be taken not to 'skim read' and misinterpret these exchanges.

Figure 1. Bagpuss

I'd appreciate some input on my own cat. 'Bagpuss' is approximately 5 years old (estimated – he was a stray) and is neutered. When I got him in May 2014 he weighed 5.2kg, but was covered in wounds and had just come into rescue. By November 2014 he weighed 5.8kg and I decided he was fat. I was guilty of feeding him rubbish (yeah, we shared croissants) so I shamefacedly put him on a diet.

He's on 100% wet food. He has 3 small cans daily. I have the calorie content of the food from the manufacturer and 3 cans is 180 kcal per day. I calculated that 80% of his resting energy requirement was 225 kcal, so he is getting less than that. And HONESTLY, I have been strict. DEAD strict. I don't want a diabetic cat. He has no extras any more, all his training treats are taken out of his cans (I pull the bits of meat out – gross) and nobody else is feeding him – hubby is well on board.

Bagpuss does steal food occasionally (from my plate!) but by occasionally I literally mean a morsel every couple of weeks or so. He goes outdoors once in a while, maybe once a week or so, his choice. He does get at least 30 minutes exercise a day because my husband adores the cat and is always playing with him. They run up and down the stairs like loons.

I weighed Bagpuss today, having been on his diet regime for nearly three months. His weight? 5.8kg! ARGH!

I would welcome non-biased and honest opinions about his weight. I appreciate photos are more limited than a 'hands-on' examination, but I'm a bit worried about where to go from here. He is healthy in every other respect, apart from a minor eye issue (another story).

Pete Coleshaw: Shosh, forget the calorie count – what's the carb-count? The answer, as always, lies in your freezer at work!

Shoshannah McCarthy: I can try cutting him down to 2 tins daily. He'll die of hunger, lol! Pete – it's not raw, but it's not bad. >95% meat and no grains or veg.

Pete Coleshaw: Shosh, I'll let you off! I do find raw tends to satisfy hunger more – usually! I think the thing that ruins the theory is castration and the metabolic changes it induces. As you know, I am dead against routine castration of male dogs but of course it is different for smelly, fighting tom cats but neutering does mess up their metabolism. I would still go raw if I were you, on the basis that un-adulterated animal-based protein is more likely to induce satiety and a bit of bone can only help fill him up but unfortunately it doesn't work for all, at least short-term. Allow some time – as Zaila says, you've stopped any increases in weight, now work slowly on the decrease.

Shoshannah McCarthy: Pete, Bagpuss does love raw, but the reason I moved him onto the tins was because, rightly or wrongly, I thought it might be easier to modulate his intake more precisely. I'm not ruling it out again for the future, but I will stick with the tins for now and see if more time melts more weight. I will try cutting him down to 2 tins. I suspect the ear plugs and blindfold I must don in order to avoid his pitiful hunger cries may make keeping a cat slightly pointless, seeing as I will be unable to see or hear him!

Richard Malik (Feline Specialist): Replace one meal with a chicken drumstick. In fact, a drumstick once a day come close to a perfect diet. He will end up gnawing on the bone, and it gives him something to focus on if he is hungry. A lamb shank will do much the same – but that will definitely be enough for a day.

Shoshannah McCarthy: I will definitely try that. I used to give him chicken wings so hopefully he'll manage a drumstick. :)

Sam Taylor (Feline Specialist): Not trying to be controversial but don't those of you in the UK have concerns re raw chicken given the high proportion contaminated with *Campylobacter* (nearly 70% I think)? I am not keen on raw feeding for various reasons but would strongly advise against doing so for any house with older people or children.

What do you advise hygiene wise? I have visions of cats dragging *Campylobacter* covered meat all over people's houses.

Not trying to provoke a raw versus commercial cat food diet debate, more interested in that aspect as the contamination of chickens is such an issue in the UK.

Nikki Duckworth: Sam, it is in New Zealand also where if 1 chicken on the line that is contaminated then the next 10,000 are apparently.

Andrea Harvey (Feline Specialist): As a relatively recent convert to feeding part of the diet as raw meat, I can share my experiences:-

1. Healthy cats guts can cope very well with *Campylobacter* and *Salmonella* – what about wild cats and cats that hunt? Rare to have a problem. And they eat the intestines of rodents and birds, plus reptiles.

2. Important to feed it very fresh. Bacteria including *Campylobacter* proliferates quickly with time and so shouldn't feed anything not super fresh and high grade (either from a butcher where it has come in that day, or supermarket premium grade with longest shelf life). Don't get the 'reduced for a quick sale' packets!

3. Most cats won't touch it once there is significant bacterial load on the surface (i.e. too old) so this is also protective.

4. I don't feed raw meat to cats that have any intestinal disease as likely to be more susceptible to infectious agents, so I am careful and cautious with those.

5. In terms of how to feed and not get a disgusting house – because, yes, they would drag it all over the house if allowed half the chance! I recommend feeding them whilst they're locked in the bathroom or shower recess which is easily cleanable and prevents raw meat getting taken through the house. I imagine it would be trickier if there are kids in the household, for sure. I wouldn't worry about old/immunosuppressed though as just like preparing meat for themselves – as long as you wash hands after handling.

I used to be dead set against feeding raw too, but based on what I had been taught and common opinion, rather than experience; sort of like an anchoring phenomenon, like which religion you are brought up to accept. So I decided to keep an open mind and not be against something I never had experience with previously; rather, to try it first before making up my mind.

I have now been feeding my cats part raw meat for 5 years – never had a problem at all and I've noticed lots of health benefits. In fact, our cats more frequently get vomiting or diarrhoea when given commercial food (if they eat too much and/or too quickly, or after a sudden change of brands); hand-on-heart they have never had any GI upset from raw meat. I actively avoid pork – too fatty.

It is also commonplace in Australia to feed cats raw meat and at least 50% of my clients, if not more, feed some raw meat and I have honestly never come across a problem. It is, however, important to be careful; I only advise feeding fresh, high quality **human grade** meat and not the raw meat sold in pet shops, and nothing economy, just high grade! And I stick to chicken, beef and lamb.

Richard (Malik) has been feeding his cats like this for many years longer and, again, has never had a problem. He is also old(er) and immunosuppressed and he has never had a problem himself from this feeding practice – but has had severe food poisoning from eating out in restaurants – a much more likely source of problems for people.

We don't however have kids, and I do think it would be very tricky feeding cats raw meat with young children in the household, unless you can lock them out of the room used for feeding and clean it afterwards. I expect mums with young children have better things to do and I think it is wise to avoid the practice if you have young children.

I hope these experiences help. I would never in a million years thought I would have recommended feeding cats raw meat 10 years ago!

Richard Malik: If you worry about *Campy* – you cannot feed raw chicken. The way suppliers prepare the carcass there is contamination, and the number is closer to 100% than to 70%, the last time I checked. But cats have been eating the guts of birds since the dawn of time, and their microbiome usually prevents them being anything more than transient commensals.

Feed lamb instead. If you are worried about toxoplasmosis, freeze it first. Avoid kangaroo meat, especially pet food grade – risk of Q fever is an issue in breeding queens.

But in Australia I have at least 5 human infectious disease clinicians as friends who feed their cats and dogs raw chickens, or parts thereof. **They just wash their hands before eating meals.**

Sam – I have sad new for you. When your cat licks your face, frequently it's just cleaned its bum, which is why you get *Salmonella* and *E. coli* and the like as transient bacteria in cats' oral cavities.

I choose to let my cat lick my face. What about you? What about your kids?

Melanie Dobromylskyj (Specialist Veterinary Pathologist):

Figure 2. Melanie and 'Ping'.

Another UK raw chicken feeder – my cats eat raw chicken wings, and we have 2 young children in the house. Just be sensible about washing hands etc. We feed the cats in the evening, overnight – once the kids are in bed. We clear up the few remaining bones in the morning… Never had any problems and have been raw feeding for about 11 years now. The cats love it!

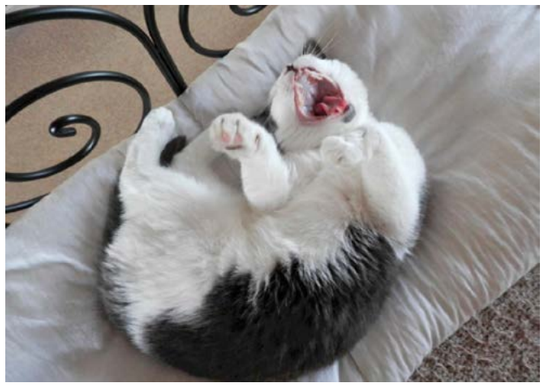


Figure 3. 'Ping'

Carla Nienhuis (Registered Veterinary Nurse and ISFM CertFN): I have the same (or sort of) problem with my cats. One is overweight (4.8 kg) and the other is 4 kg (that's OK!). We are going to renovate our scullery and we will create 2 eating houses with this kind of cat flap: www.sureflap.com/en-us/pet-doors/microchip-pet-door. At this moment we feed the cats at 6am, 11am, 4pm and 10pm. But my cats don't agree... they what to eat at 4 am as well! While we are sleeping... so I'll use the eating houses and SureFlaps to create an extra feeding moment. We will program the flap so the eating houses will be open around 4am. If we let it open all day, the overweight cat would start eating as soon as she could... that means immediately after her last meal :) The houses will be big enough for some feeding puzzles inside.

A friend of mine made cat houses to control her cat's eating habits. I'll ask her if I may show some of her pictures on this forum... And of course I'll show you my eating houses as soon as the renovation is ready.

Sam Taylor: I do want one of those SureFlap things but they cost £100 and I was thinking of my bank account, not my poor skinny cat. It is hard as 'Fatty' nicks her food (also sometimes vomits as she eats too quick) then she finds 'Eric' and puts her bum in his face until he runs off, then she eats his food! If you shut him in he won't eat (likes to see all exits!) All ideas welcome as she is a walking pre-diabetic...

Zaila Dunbar: Aha, well Sam you need the SureFlap microchip activated cat feeders!

Sam Taylor: Thanks everyone for comments on raw feeding! Actually, we have a fat cat (too ashamed to post photo) and thin cat, very difficult to manage!

DE Participant: You've really cheered me up!

I felt guilty for feeding my mad kitty raw food, from the point of view of Toxoplasma, *Campylobacter*, etc. I've never worried about her catching disease from raw food, but I've been ticked off by other vets from the disease transmission point of view.

Mad kitty was hand-reared by me from 3 days old; my Rottweiler bitch did bum duties. She has all the behavioural problems of a hand-reared cat, vomits occasionally , gets idiopathic cystitis, bites, scratches, is rough in her affection – I suppose that's why I love her! I'm obviously a terrible mother.

But she does really well on raw food, she'll occasionally gobbles down her wet food, and then lavishly throw up. She never throws up raw food. I've never investigated her sporadic vomiting because: she's not losing weight; is healthy; I'm not brave/suicidal; and she's always done it sporadically, especially when anxious. I think investigation would be so traumatic, it's not worth it, and I presume it's behavioural.

Has anyone any views on emotional emesis in cats? I'm still not planning on investigation!

Pete Coleshaw: There is a slow-feed cat bowl available over here – the gobble-stop!

(Image sourced from the internet)

Figure 4. Gobble Stopper.

Richard Malik wrote: If you slow down the rate she eats wet food it will probably be fine. Feed in 2 sittings and smear about 40 grams all over a flat plate – this means they don't eat it too quickly. Some people think the high histamine content of wet food makes them vomit. TRY THIS TRICK – see if it works.

Shoshannah McCarthy: Yes, that is what I meant, from a social stress point of view, giving them the option of eating alone. It is amazing how many cystitis patients' owners say they are not stressed but then when you start asking, it turns

out 3 cats share 1 bowl and 1 cat will block access to that or the cat flap.

Jane Ehrlich: Absolutely agree with Zaila. I've seen too many cases of inter-cat aggression where the owner has insisted on one large bowl for both cats. The problem was softened immeasurably when there were separate eating – and litterbox – stations.

Zaila Dunbar: I'm not sure what Nikki was referring to with 2 cats at the same bowl but I would advise against it from a behavioural/stress perspective. It's OK if they choose to eat from the same bowl, but they need to be given an option so not competing over resources. We see massive issues with stress related disease (FIC) and cats in households falling out with each other where we are, and many mixed sex neutered sibling pairs fall out significantly once they hit social maturity.

Andrea Harvey: Being a fellow veggie, I was also very repulsed about ripping up raw chicken initially (retch!), but once I got used to it, not a problem, and I'm careful re hygiene! I do think it would be a nightmare with kids though! You would find Leo sat in the bathroom gnawing on a drumstick! I think I would avoid that possibility!

I do sometimes get anxious advising clients though, as you know what clients are like listening to you; I had a half hour conversation with one client about what to feed and how, and what to avoid, and the next time I saw them, they said 'the cat is loving the pork tripe from the petshop'. Didn't you hear me say AVOID PORK, AVOID RAW PET SHOP MEAT?! I nearly died.

I think there is a big difference between just advocating raw diets in general, and doing it the way we now do - we are VERY careful. In fact, often Richard won't allow ME to buy the meat for HIS cats because he doesn't trust me to get the right brand/quality/date. I was in trouble last week for buying the wrong type of steaks! So feeding a raw diet yourself, and recommending that to every client is also a different ballgame!

Sam Taylor: Thanks all – very interesting. I am not too worried about the cats – more the human health risks given the way chickens are killed and prepared (but then I have been veggie for nearly 30 years!).

Yes, 'Richard' my cat licks my children and, worse, I have a dog and my youngest child licks him as he thinks it is funny. He also shares food with him! One lick for dog, one for baby! Good for the immune system I hope! I take Andrea's point about fresh chicken and using a butcher. I think that must be important.

Pete do you advise caution if people have kids? Andrea suggests feeding in the shower. What about others? Just thinking practically.

Pete Coleshaw: Totally agree with Richard and Andrea on all points. I have sold maybe 20 tonnes of raw minced chicken – never had an issue – and I am certain that much, if not all, is contaminated with Campys of various species. The pH in the stomach of raw-fed carnivores drops much lower than those fed starchy diets and an animal fed an evolutionary diet should have optimal gut immunity, with a microbiome closer to natural. I suspect that a complement of good bugs blocks the bad bugs anyway – is raw the perfect prebiotic?

I would agree, though, that it is scarey the first time you advocate it to a client, when everything you have been told is contrary. A bit like the bones sticking into their throat; how on earth have they evolved over the millenia to eat prey if this is an issue? If Campy really is a concern, then whole chicken pieces can be blanched in boiling water for a few seconds; it will kill the surface contaminants without denaturing the rest. I guess you could even do the same with minced meat-with-bone. ■

Editor's note: *Even specialists can have widely differing opinions about this and all manner of things. No one who knows who is right. But we all should have a view and be open to other people's experiences.*

WINNER FOR BEST VIDEO IN THE ISSUE!

Thanks to Carla for not only taking these pictures for this C&T article, but also sending us a video as well for the eBook. Carla wins a DVD of her choice from www.vetbookshop.com

Post-script from Carla Nienhuis: I have just re-read my comment and I'm no longer completely behind it.

Earlier, I wrote that the cat flap will not be open all the time. It is not possible for the cat, however, to see if the cat flap is open or closed, with the result that the cat will not understand the way it works and will try to open it or refuse to use it. This may result in frustration. We have therefore chosen to let the cat flap stay open 24 hours (so we do not use the time function) and at night we put an automatic feeder in the eating houses. This feeder will open at 3am (the cat will hear a click and can see the valve is open, so it's clear she can eat).

Unfortunately, the eating houses are not big enough for feeding puzzles, so at least once a day we feed the cats with puzzles outside the eating houses. A friend of mine made houses which are big enough for feeding puzzles, but unfortunately I do not have those pictures to share.

Please see pictures of my eating houses.

Eating from a dish is very boring for cats... Let them work for their food!

Carla kindly went to the additional trouble of videoing the cat feeding houses in action. View in the eBook.

FELINE MEDICINE / DISTANCE EDUCATION

HOW WOULD YOU TREAT THIS CASE?

Collaroy Plateau Veterinary Clinic
24A Aubreen Street, Collaroy Plateau NSW

Caroline Wood
E. cpvets@outlook.com

C&T NO. 5510

'Riley' is a 7-year-old male neutered DSH who presented 2 weeks ago with lethargy and moderately reduced appetite. On initial physical examination, his mucous membranes were pale, he had a previously undiagnosed heart murmur, he was very underweight, and he had a palpable abdominal mass with a mild to moderate amount of abdominal fluid. His initial bloods/urine showed a regenerative anaemia (PCV 16%), leucopaenia (WBC 2.6, neutrophils 1.0, lymphocytes 1.3, monocytes 0.3, eosinophils 0.1, no basophils) and decreased platelets (estimate 128) with no evidence of *Mycoplasma haemofelis*. He was also hypoproteinaemic (TPP 57, globulins 23, albumin 34) with well concentrated urine (1.048) and no liver enzyme increase or increased spec fPL.

He had a typed whole blood transfusion, then an abdominal ultrasound by a specialist the following day which showed abdominal fluid, several enlarged mesenteric lymph nodes (measuring 1.18cm and 1.33cm across), and a massively enlarged heteroechoic spleen, with no other abnormalities. Fine needle aspirate biopsies of the spleen and a sample of the abdominal fluid was sent to IDEXX and a tentative diagnosis of lymphoma was made; he was started on 10mg prednisolone PO SID and amoxyclav PO BID. His post-transfusion PCV was 19% which had increased to 20% by the following day. His previously detected heart murmur disappeared after the transfusion.

The owners then took him home for the weekend as he seemed much brighter and we were waiting for the results. The results came back as:

Cytology

1. Abdominal fluid

Samples consist of overall low number of nucleated cells on an amphophilic proteinaceous background with moderate numbers of red blood cells. Nucleated cells are 28% large mononuclear cells, 52% small mononuclear cells, and 20% non-degenerate neutrophils. Occasional metarubricytes and rubricytes are also seen. Some large mononuclear cells have a moderate amount of basophilic cytoplasm and a single round nucleus with smooth chromatin and a single prominent nucleolus. Anisocytosis and anisokaryosis are mild; mitotic figures are not seen. Other large mononuclear cells have abundant pale basophilic cytoplasm and a single round nucleus with stippled chromatin and a single nucleolus. Anisocytosis and anisokaryosis are mild to moderate; mitotic figures are not seen.

2. Abdominal mass

Samples consist of low to moderate numbers of lysed cells with occasional stromal aggregates/material, low, but noticeably increased number of large round to less often slightly elongated mononuclear cells, low numbers of small lymphocytes, and moderate to large numbers of red blood

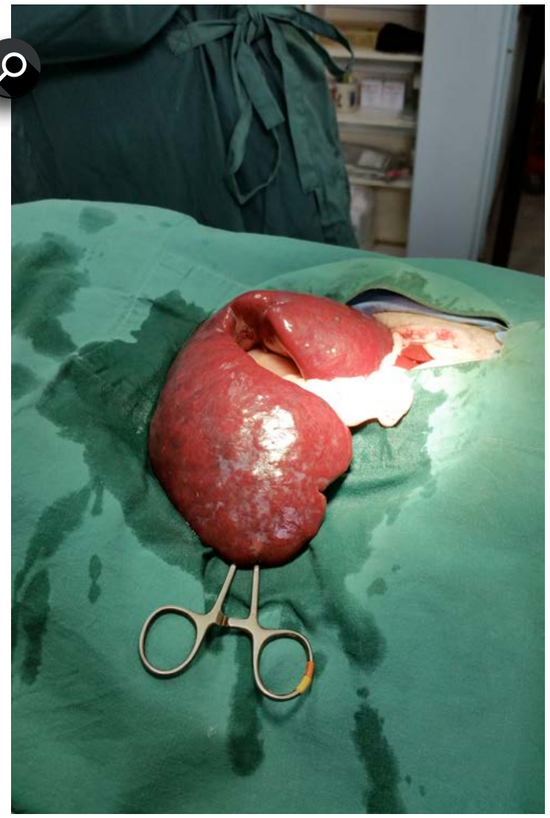


Figure 1. Massively enlarged spleen at exploratory laparotomy.

cells on an amphophilic background. Large mononuclear cells have a moderate amount of basophilic cytoplasm and a single round nucleus with smooth chromatin and a single prominent nucleolus. Anisocytosis and anisokaryosis are mild; mitotic figures are not seen.

Interpretation

1. Abdominal fluid: High protein low cellular modified transudate with possible increased percentage of small lymphocytes.

2. Abdominal mass: Concerning for neoplasia – see comments.

Comments: Modified transudates can be seen with underlying neoplasia, cardiac disease, or long standing transudates. Some of the large mononuclear cells may be reactive mesothelial cells and there may be a degree of increased percentage of small lymphocytes which can be seen with a degree of loss of lymph rich fluid/impairment of lymphactic drainage. Low numbers of neoplastic cells cannot be completely excluded within the fluid given the concern for underlying neoplasia in the abdominal mass. The abdominal mass is of uncertain origin, but there may be splenic involvement given occasional stromal aggregates. The large rounded cells are concerning for large lymphocytes/lymphoma, although given the large degree of cell lysis and the possibility of splenic aspiration, a definitive diagnosis is not possible. Other rounded appearing cell types are also possible (such as epithelial or mesenchymal), but are thought less likely. Histopathology (if clinically indicated) is recommended to aid in further characterization.

The owners brought him back for a recheck on the Monday and said he seemed well over the weekend but then on Monday morning he seemed a bit flat again. His PCV had decreased to 16% again, the heart murmur returned, and he had moderate abdominal fluid. His CBC was checked to make sure his WBC count had increased, and it had, though it was still low: WBC 4.6, neutrophils 3.4, lymphocytes 0.7, monocytes 0.4, eosinophils 0.1, no basophils. Platelets were estimated to be about 214.

He had another typed whole blood transfusion, then a splenectomy was performed. I have attached a picture of his spleen at the time of surgery; unfortunately, no mesenteric lymph nodes were taken nor were chest radiographs taken. His spleen weighed 350g, dropping his weight to 5kg, which was way too thin for a cat of his size who should at least be 6-6.5kg!

He recovered well after surgery and his PCV has continued to increase post-surgery (with no audible heart murmur). The histopathology results (sent to Vetnostics) came back initially as this:

MACROSCOPIC EXAMINATION

Portion of spleen: A portion of spleen 75x35x30mm. RST in 3B. (vs/ss)

MICROSCOPIC EXAMINATION

Diffusely infiltrating the spleen and effacing much of the normal architecture is a sheeted population of neoplastic large round cells. The cells have a small to moderate amount of lightly eosinophilic cytoplasm with indistinct margins containing a round to indented nucleus with a prominent central nucleus. There is mild to occasionally moderate anisocytosis and anisokaryosis, scattered individual necrotic cells along with many mitotic figures often >7 per HPF with atypia. Amongst the neoplastic cells are small lymphocytes, few plasma cells, macrophages, erythrocytes and small groups of haematopoietic cells.

Neoplastic cells do not contain metachromatic granules.

SUMMARY

ROUND CELL MALIGNANT NEOPLASM.

COMMENT

There are very few distinguishing features of these cells that allow for definitive classification. They are not mast cells. They could be of lymphoid origin and this possibility can be further investigated with immunohistochemistry for an additional fee ($83). Please call me if you wish to discuss.

We then requested immunohistochemistry and this came back:

IMMUNOHISTOCHEMISTRY REPORT

The neoplastic cells are negative for CD3 (T cell) and CD79a (B cell) lymphoid markers and are not considered to be lymphocytes. Further staining* shows tiny very light blue granules within the cytoplasm of these cells.

REVISED COMMENT

I now think this is a very poorly granulated/histiocytic variant of mast cell tumour that is occasionally recognised in cats. It clearly lacks typical metachromatic mast cell granules. The diagnosis of this condition is often difficult and made by elimination of other possibilities. Further confirmation is not possible because we don't have other specific mast cell immunohistochemical markers.

FINAL DIAGNOSIS

SPLENIC MASTOCYTOSIS (POORLY GRANULATED/ HISTIOCYTIC TYPE)

Now I don't know what to do. I spoke to one internal medicine specialist who suggested going back in to take a sample of mesenteric lymph node, but that seems pretty extreme if we already have a diagnosis, don't we? Is this a case that is suitable for referral for chemotherapy? ■

***Note:** The pathologist confirmed that the stain he performed to visualise the granules was tol blue and he did so before immunohistology but did not see any granules. After he got the immunohistology back and found it was negative he repeated the tol blue stain as he doubted the first results and then saw the granules. He is confident it was splenic mastocytosis.*

Postscript November 2015

I actually managed to speak to Riley's owner today who came in for more prednisolone; they decided not to pursue chemotherapy/a specialist appointment, so they are just continuing Riley on prednisolone only. They reported he has gained a lot of weight and has his normal exuberance and zest for life back, which is excellent news!

HOW WOULD YOU TREAT THIS CASE...?

Please email your answer to: elisabeth.churchward@sydney.edu.au

Winner of the best and most complete answer is entitled to a CVE proceedings of their choice.

Please see www.vetbookshop.com to peruse out list of titles.

C&T RESOURCES

Please visit www.cve.edu.au/candt2015 to download these articles or view them in the eBook:

Diagnosis of toxoplasmosis and typing of Toxoplasma gondii Quan Liu, Ze-Dong Wang, Si-Yang Huang and Xing-Quan Zhu.

Baysol® Snail and Slug Bait *Treatment advice for accidental ingestion by dogs and cats.*

FROM THE DE FILES

2015 ANZCVS RESULTS

Congratulations to CVE DE Alumni who successfully passed their examinations in 2015

Jillian Kelly	Animal Nutrition
Yvette Ellen	Medicine & Management of Laboratory Animals
Surita Du Preez	Medicine of Horses
Celine Lee	Medicine of Horses
Stephanie Williams	Medicine of Horses
Lianne Wright	Medicine of Horses
Penelope Brown	Small Animal Medicine
Daniel Edwards	Small Animal Medicine
Esmee Koh	Small Animal Medicine
Alina Lavelle-Fry	Small Animal Medicine
Pui Pui Liem	Small Animal Medicine
Sophie Tyler	Small Animal Medicine
Julie Ward	Small Animal Medicine
Sarah Warren	Small Animal Medicine
Amanda Miller	Small Animal Surgery
Brendan Sinnott	Small Animal Surgery
Nicole Lobry de Bruyn	Veterinary Behaviour
Michelle Gray	Veterinary Emergency and Critical Care
Mariko Ike	Veterinary Emergency and Critical Care
Jennifer Philbey	Veterinary Emergency and Critical Care
Shelley Wiltshire	Veterinary Emergency and Critical Care
Mary Carr	Veterinary Epidemiology
Daniel Lawrence	Veterinary Radiology (Small Animal)
Vickie Saye	Veterinary Radiology (Small Animal)

SMALL ANIMAL

MAJOR WINNER

LESSONS LEARNED FROM A CHIHUAHUA'S **POSTERIOR** ADVENTURES AND MISADVENTURES IN UROGENITAL SURGERY AND WOUND HEALING

Locum

Heather Shortridge

E. heathershortridge@gmail.com

C&T NO. 5511

'Chico', a 9-year-old 2.83kg male Chihuahua presented me one of the most challenging and extensive cases of my career thus far. I am sure Chico has put some grey hairs on my head, and he has given me a lot to think about, some of which will hopefully be helpful to others.

Figure 1. Chico in hospital

Chico presented on a Wednesday in January 2014. He had been very unwell, just laying around, and not eating. Chico is an epileptic dog, and he had been having more seizures than usual. (He was on phenobarbitone 7.5mg SID and had been on this dose for a long time). Chico had only passed a small amount of faeces and had been dribbling urine. On physical examination a large bladder was palpable. Chico was very lethargic and had a prominent heart murmur.

Radiographs were taken of Chico's abdomen which showed a number of stones in his urethra.

Chico was started on IV Hartmann's solution and given 0.15mL methadone. I attempted unsuccessfully to pass a 4 French urinary catheter. I then gave Chico 0.2mL alfaxalone IV and could partly pass a 6 French catheter. My colleague and I used a retropulsion technique to flush the stones back into the bladder and pass the urinary catheter.

This involves an assistant placing digital pressure ventrally via a finger in the rectum, flushing the catheter with saline to build up pressure within the catheter, and then the assistant releasing digital pressure so the stones are blasted back up the urethra into the bladder. This was successful and allowed us to drain the bladder.

Urinalysis was performed on the collected urine, and calcium oxalate crystals were observed on microscopy.

Chico presented late in the day so it was decided to stabilise him on fluids overnight at 10mL per hour while his owner decided which way to proceed.

I discussed performing a cystotomy on Chico to extract the stones, but his owner was very reluctant for Chico to have abdominal surgery. My colleagues suggested we could instead perform a pre-scrotal urethrostomy to extract the stones, and then leave this open to heal by secondary intention.

Chico's owner was very concerned about any surgery but agreed we could proceed with the urethrostomy. Chico was anaesthetised, and following the procedure as described in Fossum, Small Animal Surgery (2006) a urethrostomy was performed. We retrieved a lot of sharp yellow stones from the incision site. I used PDS sutures to close down around the urethral incision site to reduce the risk of any leakage under the skin.

Chico was started on amoxicillin-clavulanic acid and meloxicam, and continued on intravenous fluids. At this point I had 3 days off work. The first day I was off work (Friday) Chico was quite bright and so was discharged. The next day he re-presented for medicating as his owners were struggling to do this at home. His bladder was small and soft and the incision site was not painful. My colleague noted that he did look bruised around the testicles.

Chico re-presented on Monday morning very subdued. He was also sore around his left hindleg and testicles. I re-started him on intravenous fluids, and took blood, particularly to check for azotemia.

Chico's results were as follows:

- TP 74 (54-82)
- BUN 55.2 (2.5-8.9)
- Creat 195 (27-124)
- Gluc 8.9 (3.3-6.1)
- ALT 79 (10-118)
- ALP 2362 (20-150)

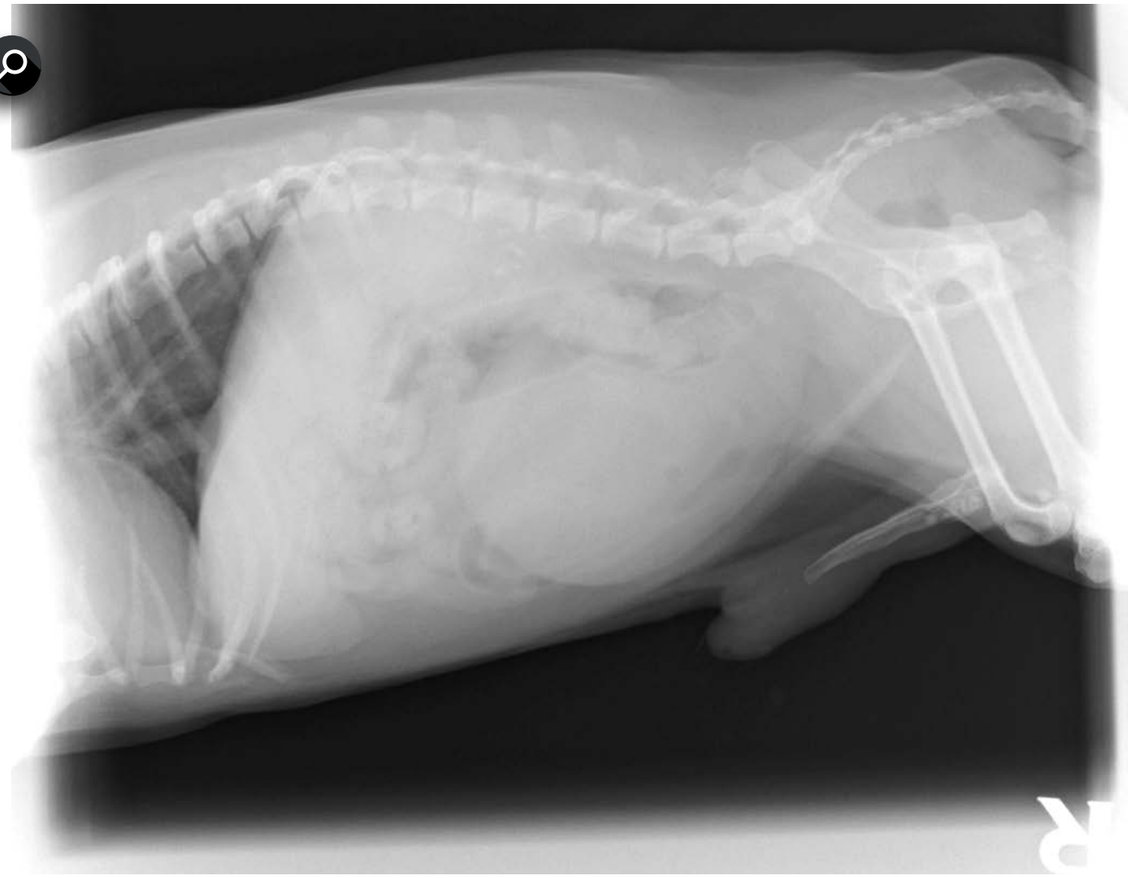


Figure 2. Lateral radiograph showing numerous stones blocking the distal urethra

Metacam was stopped due to the presence of azotemia. Chico was maintained in hospital on fluids. Flamazine and vaseline was applied around the urethrostomy site. By Tuesday, the distal end of Chico's scrotum was looking jelly-like. I discussed with Chico's owners that we needed to control the azotemia, but that it looked like Chico was going to need to be castrated and have a scrotal ablation.

On Wednesday, Chico's urea and creatinine were much improved (BUN 35.3 (2.5-8.9), Creat 113 (27-124). Unfortunately, Chico himself was looking much worse. Where the previous day, Chico had had a small amount of irritation below his rectum, by Wednesday almost the entire skin between scrotum and rectum and extending quite far laterally, looked really sick.

Chico's owners were very concerned about whether he could tolerate more surgery, but I discussed with them that it was really a matter of life and death for Chico as he seemed to be suffering from rapidly worsening subcutaneous urine scalding. Chico's owners agreed we could go ahead with surgery. Chico was preoxygenated and given 0.01mL of acepromazine 2mg/mL IV, followed by 0.3mL of alfaxalone IV, and intubated with a 3.5 cuffed ET tube.

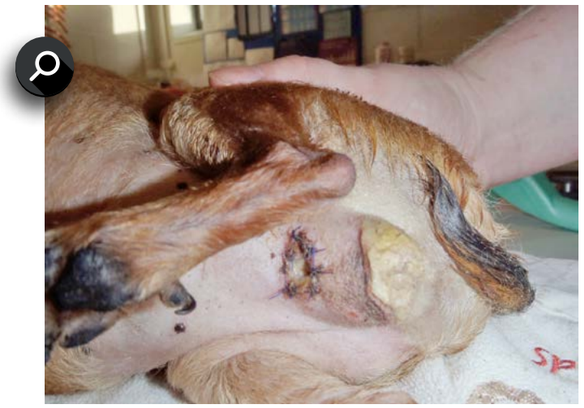


Figure 3. Chico's urethrostomy and rump pre-operation.

I castrated Chico and performed a scrotal ablation. Unfortunately, it was obvious that a large area of skin over Chico's rump was devitalised. I was very frustrated as Chico had looked so flat and sick the previous day and I had not wished to rush into surgery with him again, but the devitalisation of the skin looked so much worse on Wednesday than it had on Tuesday.

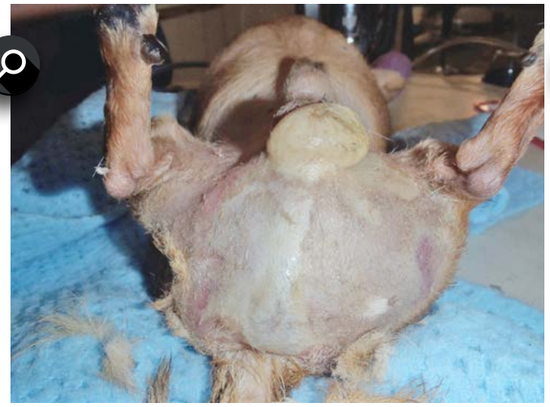


Figure 4. 'Jelly' scrotum and sick skin preoperatively.

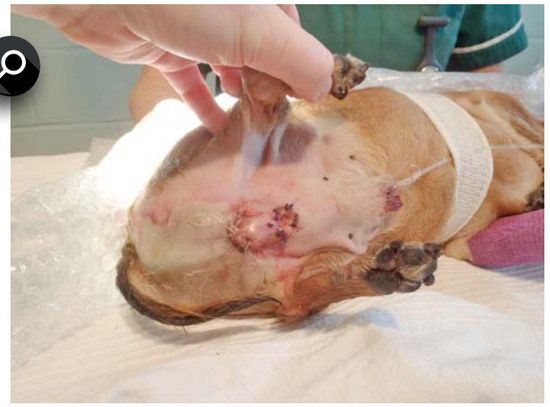


Figure 5. Extensive devitalised tissue visible post scrotal ablation.

Before waking Chico, up I placed a 6 French urinary catheter and stitched it into place. Figure 5 shows Chico post-operatively; it is possible to see how devitalised the skin is dorsal to the urethrostomy site, and also some nicks I made in the devitalised skin for drainage.

I had posted about Chico on the Veterinary Information Network (www.vin.com) and it was suggested that Chico could have a urethral tear, and if so, we should leave a urinary catheter in situ for 5-7 days. Ideally, we should have done a contrast study to evaluate the urethra, but we had already spent much of the owner's budget so it was decided to leave the catheter in place and see what happened after allowing enough time for healing. I started Chico off with a closed urine collection system and on Thursday he was brighter and urine was flowing freely.

On Friday, I informed the owner it looked like Chico was going to lose a lot of the skin from his rump and that we would manage this as it happened.

On Friday night, Chico pulled his catheter out. By Saturday morning, it already looked like there was fluid accumulating under Chico's skin, so we anaesthetised him and replaced

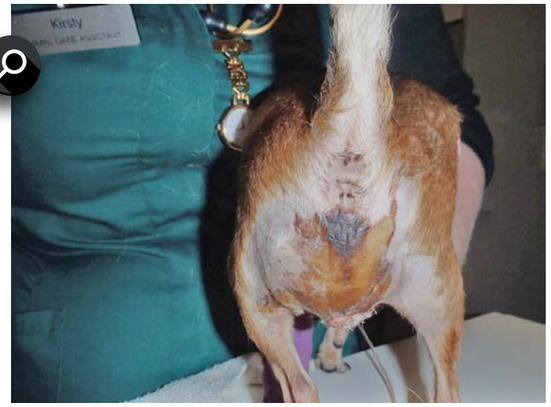


Figure 6. Chico's rump Friday.

the catheter. As well as stitching in the catheter with a Chinese finger trap suture, I also stitched it to his belly bandage in several places. It was critically important the catheter remain in situ.

Chico kept twisting his catheter up and, in the end, acknowledging that this is terrible from a microbial point of view, I eventually left his catheter free in his cage. At this point I figured it was a matter of life or death that the catheter stay in, while a UTI could be dealt with later.

Chico's condition improved over the next few days, and it was an anxious wait for his owners and me. By 5 days post scrotal ablation the skin was obviously breaking down.

We started Chico on potassium Citrate 50mg per kg bid at this time, to try to keep his urine pH between 7.1 and 7.7, on the assumption his urinary stones would be calcium oxalate.

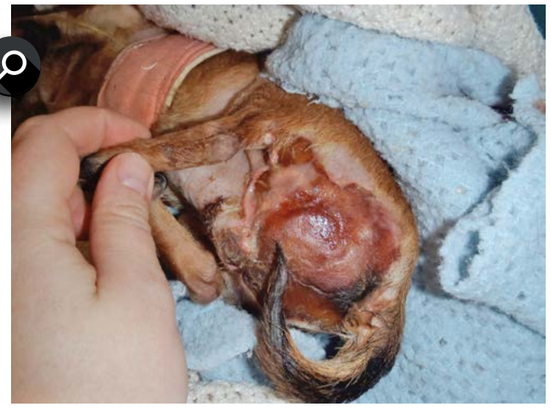


Figure 7. Six days post scrotal ablation / recatheterisation.

Anxious not to pull the urinary catheter too quickly, further reading had suggested it can take up to 14 days for urethral healing to occur. We decided to leave the catheter in place for longer. Chico had been having injections of amoxycillin clavulanic acid but I decided to give him Convenia® (cefovecin) as he was resenting frequent injections. I had

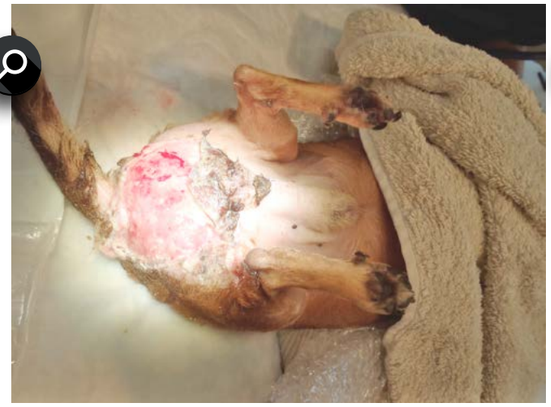


Figure 8. Granulation tissue revealed when dead skin removed.

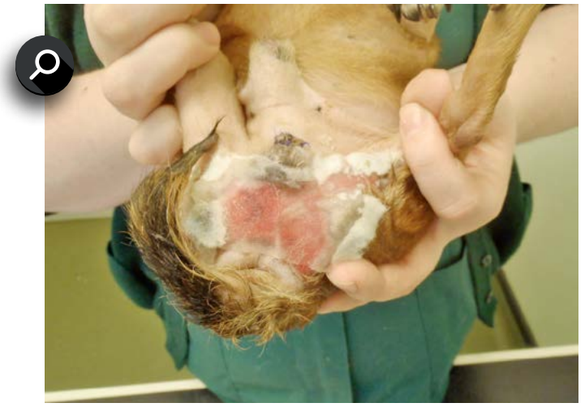


Figure 10. A novel way of bandaging? Medihoney and kaltostat 'bandage'.

also been giving buprenorphine 3 times daily for pain control while Chico was in hospital.

On day 12 post scrotal ablation, I induced anaesthesia with alfaxalone and flushed Chico's bladder with sterile saline, before removing the urinary catheter. I held my breath, waiting to see if Chico would continue to urinate.

To my delight, Chico continued to urinate freely with no evidence of urine leakage under the skin.

Two days after removing the catheter, I persuaded Chico's owner to let me debride the dead skin from his rump under general anaesthesia. The tissue under the wound looked good. I applied medihoney to the granulation bed, and kaltostat, and then applied a bandage (dubbed 'Chico's superpants' in the clinic)

Chico was discharged from hospital once following wound debridement. Unfortunately, Chico's super pants were super impractical. Chico returned 3 days later and had been urinating on the bandage, and struggling to get around. I removed the bandage, flushed the wound with sterile saline, and then applied medihoney to the granulation tissue. I

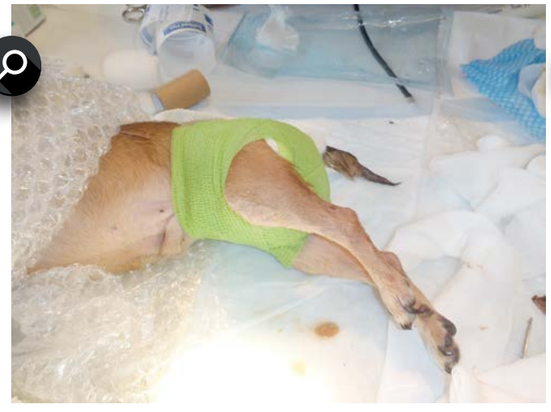


Figure 9. Chico's superpants.

moistened some kaltostat and stuck it to the wound over the medihoney, and stuck down the edges to Chico's fur using surgical glue.

Over the next few weeks I saw Chico every 3 days or so, removed any bandage material that remained, flushed the wound with sterile saline and reapplied my kaltostat dressing.

Results came back from the Minnesota Urolith Centre that the stones had been 30% Calcium Oxalate Monohydrate and 70% Calcium oxalate dihydrate. Chico was to continue having potassium citrate and was to avoid cheese, milk, tea, coffee, chocolate, vegetables, fruit, nuts, and salty foods (an adjustment for him, as he had been a cheese and pork pies dog!)

To my delight and intense relief, Chico's rump eventually healed. By the time I left the clinic only a small area of wound remained.

My colleagues gave me an update that 6 months later Chico was doing well, had gained weight again, and was back to his normal happy self.

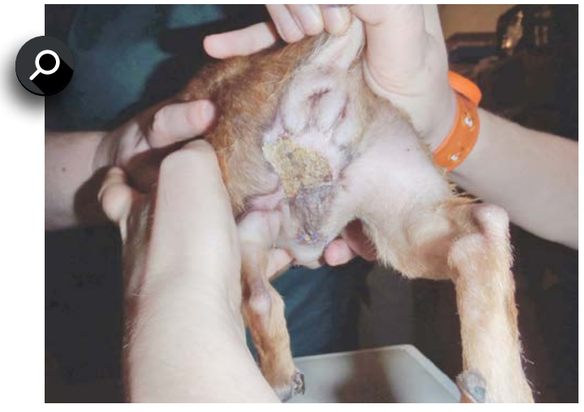


Figure 11. Almost healed!

Figure 12. Relieved vet with healed Chico, 7 weeks post operatively.

Discussion:

Key point 1: Pushing initially for a castration and scrotal urethrostomy may have been a better choice than a pre-scrotal approach, given the size of the dog? While on VIN, commentators suggested a urethral tear was likely the cause of the urine leakage. I do wonder if the tiny size of the dog meant that a pre-scrotal incision was inclined to result in urine pooling under the testicles? Alternatively, given that we could retropulse all the stones into the bladder, perhaps we should have pushed harder to perform a cystotomy to remove them? Chico's owner was very against abdominal surgery, but maybe we should have tried harder to persuade him?

Key point 2: In future, if I ever see what I think is urine scald, I will try to act immediately, even if the patient is not an ideal candidate for anaesthesia. I was shocked by how quickly Chico's condition worsened and would act swiftly if faced with anything similar in the future.

Key point 3: I was very disheartened when faced with the large area of Chico's rump that had lost its skin. I was amazed how well he healed and how much the wound contracted down, given patience, time, and wound care. Alternatively, given that we could retropulse all the stones into the bladder, perhaps we should have pushed harder to perform a cystotomy to remove them? Chico's owner was very against abdominal surgery, but maybe we should have tried harder to persuade him.

This was a highly stressful case, although a good learning experience, and I was very relieved when things turned out well for Chico in the end. ■

INTERESTING LINK

JFMS Open Reports publishes case reports, short case series and short communications that are relevant to feline practice and **are freely accessible.**

Note: *All articles are available as PDFs – great to access on your tablet.*

http://jfm.sagepub.com/content/17/4/279.full

SMALL ANIMAL

WINNER

MYCOBACTERIAL INFECTION IN A DOG

David Lee

BVSc (Hons), MVSt, MANZCVSC (Avian Health, Small Animal Medicine)

Allison Crescent Veterinary Hospital
Shop 13, 72-80 Allison Crescent Menai NSW 2234

T. (02) 9543 5843
E. david@allisoncrescentvet.com.au

C&T NO. 5512

QUESTION

We had an 8-year old Hungarian Visla FN which presented for a large lump that was expanding rapidly on the dorsal neck area. The dog had suffered from pancreatitis and was treated in hospital for a few days and recovered, 5 weeks prior to presenting with this lump. During the pancreatitis treatment, the dog had received injections of amoxicillin/clavulanate in the dorsal neck area, where this lump had now appeared.

The lump was very firm, and initial FNAs suggested spindle cells and suggested a soft tissue sarcoma, but on incisional histopathology it was confirmed to be *pyogranulomatous panniculitis*. On further staining, they detected mycobacteria (see histopathology report below).

Histopathology

Both tissues show similar changes. The deep dermis and subcutis contains multiple foci of pyogranulomatous inflammation, some of which are separated by fibrovascular septa, and which often contain a central area of necrotic or vacuolated cells with accumulation of neutrophils. Peripherally some foci are surrounded by lymphocytes and plasma cells. Small numbers of multinucleated giant cells are seen.

Diagnosis

Pyogranulomatous panniculitis

Comments

Possible causes include bacterial, mycobacterial or infection, possibly associated with a puncture wound or foreign body Alternatively, this lesion may represent a form of so-called sterile granuloma syndrome. This latter syndrome is characterised by granulomatous to pyogranulomatous inflammation, coupled with an often dramatic response to glucocorticoids, suggesting the likelihood of an immune dysfunction, perhaps associated with persistent antigenic stimulation.

The dog has now had radical surgery to resect this lesion and the tissue has been submitted for culture. He is doing OK at the moment, but there are certainly a few questions and the owners are concerned as to the possibility that the dog contracted the mycobacteria while she was in hospital.

Discussion and thoughts

1. Several unanswered questions in regards to this case remain. Could the dog have contracted mycobacteria while she was in hospital for pancreatitis treatment? I understand that mycobacteria gets into the body via abraisions in the skin, and needles do break the skin, but I am struggling to find any literature on cases where they have contracted mycobacteria through needle stick lesions or through open surgical wounds in hospital settings, like an MRSA.
2. Would the mycobacteria be expected to be around in a hospital setting? On the ends of multi-use injection bottles containing commonly used, everyday antibiotics?
3. Are there any cases of mycobacteria like this where they have contracted the bacteria after an injection or vaccination?
4. What antibiotics would be the best ones to use while the culture results are pending? I know that culture can take a long time, and also that it can actually be very difficult to culture mycobacteria. But at the same time, while we wait, we would need some empirical antibiotic cover. Also, if it takes so long to culture, how 'long' is 'long enough' for empirical antibiotic cover? Would 5 weeks *in vivo* be enough time for the bug to grow? Is there any evidence out there, that supports the theory that the mycobacteria could have caused such an aggressive infection only 5 weeks after instillation from the pancreatitis treatment? Given the mycobacterial culture is expected to take over 12 weeks, would this be enough evidence to disprove the theory that the dog contracted the mycobacteria from the prior treatment. I guess so, if *in vivo* and *in vitro* times for mycobacterial growth is expected to be different.

Addendum Comments (12.06.13)

Special stains demonstrate the presence of occasional beaded acid fast bacilli consistent with a Mycobacterium spp. Culture is recommended. ■

REPLY FROM RICHARD MALIK

We will never know for sure what happened but we need a diagnosis and if you can't get one we can arrange for PCR on your formalin fixed tissue or even the Diff-Quik stained smears. I don't know what drugs the dog is on now but a combination of doxycycline 5 mg/kg twice a day and enrofloxacin (standard dose) would be a great start. In the future, we would probably recommend doxycycline plus pradofloxacin.

Possibilities

- Dog fight injury – you don't know about?
- Dirt on the skin – followed by injection of fatty injection
- Injection multi-use bottle – contaminated by mycobacteria
- A culture will help

It's usually an OIL injection amoxicillin/clavulanate acid (which is in peanut oil) that is the most likely candidate.

Ideas: Get the bottle of amoxicillin/clavulanate acid and give it to Jacqui Norris at the University of Sydney for culture.

Tip: If it was in the bottle, you would have had a NUMBER of cases – not just one.

REPLY FROM DAVID LEE

I don't know if we can culture the amoxicillin/clavulanate acid bottle now, given that it was over 3-4 months ago and I would have to assume that it has been used up. The dog is on enrofloxacin and doxycycline so looks like we are doing the right thing.

So far, we have not seen any more cases of suspect mycobacterial infections like this patient, and the dog is doing well.

So, just to reiterate, this lump started off the size of a golf ball. See pictures below:

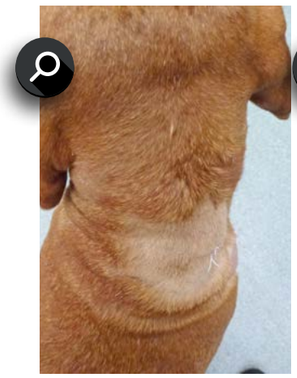


Figure 1. Coco before surgery, incisional biopsy taken at the suture site.

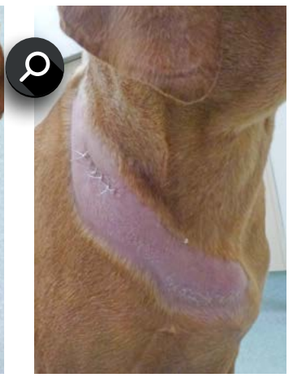


Figure 2. Coco before surgery. NOTE: This is not clipped fur, but hair loss over the granulomatous lesion.

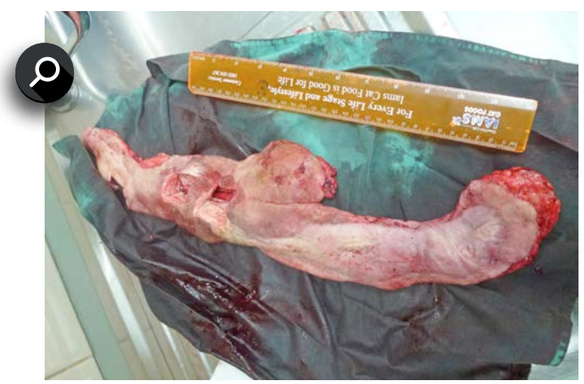


Figure 3. Coco lesion resected during surgery. Measures about 45cm length, and 8-10cm thick!

Want to win **$5,000**

to spend on CVE courses and events?

SYNOVAN™ INJECTION FOR DOGS

THE UNIVERSITY OF SYDNEY
Centre for Veterinary Education
Est. 1965

Send us a case report that explains how Synovan has contributed to the health and well-being of one of your patients and you could win a $5000 CVE voucher, valid till December 2017.

The winner will also have their case report published in the March 2016 edition of CVE's Control & Therapy Series.

For a copy of the entry guidelines, case report template and full terms and conditions, please contact Ceva at marketing.australia@ceva.com.

Entries close 31st January 2016.

Abridged Terms & Conditions:

Entry only open to veterinarians registered to work in Australia. Promotion commences on Monday 28th September 2015 and ends at 11.59pm on Sunday 31st January 2016 ("Promotional Period"). Entries must be submitted by emailing to marketing.australia@ceva.com in electronic format before the end of the Promotional Period. To be valid, case studies must incorporate administration of Synovan as part of the patient's treatment. The winning entry will be judged by staff at Ceva Animal Health Pty Ltd on 15th February 2016, based on the criteria set out in the entry form template; the winner will be notified by phone or email within two (2) business days. The winner will receive a Centre for Veterinary Education voucher valued at AU$5000 which will be applied as a credit to the winner's individual CVE account. Promoter: Ceva Animal Health Pty Ltd (ABN 54 002 692 426) of 11 Moores Road, Glenorie NSW 2157. Telephone: (02) 9652 7000. Contact marketing.australia@ceva.com for full Terms and Conditions.

THE UNIVERSITY OF SYDNEY
Centre for Veterinary Education

LAST CHANCE TO ENROL & STUDY IN 2016

Enrolments close 31 January, 2016.

www.cve.edu.au/distanceeducation

Stand out from the pack!

DISTANCE EDUCATION

2016 DE COURSES

- **Behavioural Medicine**
  Kersti Seksel
- **Cardiorespiratory Medicine**
  Nick Russell, Niek Beijerink
- **Clinical Neurology**
  Laurent Garosi, Sam Long, Simon Platt
- **Clinical Pathology**
  Sandra Forsyth
- **Dermatology**
  Ralf Mueller, Sonya Bettenay
- **Diagnostic Imaging:**
  - **Thoracic** Robert Nicoll
  - **Abdominal** Zoe Lenard
  - **Musculoskeletal** Sarah Davies
- **Emergency Medicine / LIMITED PLACES**
  Trudi McAlees, Sandra Forsyth
- **Feline Medicine**
  Sarah Caney, Richard Malik, Andy Sparkes, Mike Lappin, Carolyn O'Brien, Wayne Mizon, Elise Robertson, Jessica Quimby, Susan Little, Samantha Taylor
- **Internal Medicine: A Problem Solving Approach / SOLD OUT** Jill Maddison, Sue Bennett
- **Internal Medicine: Keys to Understanding**
  Darren Merrett, Jen Brown, Kate Hill
- **Medical Oncology**
  Peter Bennett
- **Ophthalmology**
  Robin Stanley
- **Ruminant Nutrition / LIMITED PLACES**
  Paul Cusack
- **Sonology / SOLD OUT**
  Karon Hoffmann, Cathy Beck
- **Surgery / LIMITED PLACES**
  Wing Tip Wong, Guy Yates

Need help choosing the right course? Contact us by email at: cve.disted@sydney.edu.au or by phone on: +61 9351 7979.

WHY CHOOSE DISTANCE EDUCATION [DE]?

Because you are:

- ✓ Dedicated to becoming a better vet
- ✓ Keen to reinvigorate your love of veterinary science
- ✓ Looking to enhance your practice's capacity
- ✓ Interested in expanding your knowledge in a specific area
- ✓ Considering changing your veterinary focus
- ✓ Returning to the workforce
- ✓ Preparing for ANZCVS membership exams

...or simply love learning

SMALL ANIMAL

CANINE NEURAL ANGIOSTRONGYLIASIS

EOSINOPHILIC MENINGOENCEPHALITIS DUE TO MIGRATION OF LARVAE OF THE RAT LUNGWORM *ANGIOSTRONGYLUS CANTONENSIS*

Richard Malik
CVE Valentine Charlton Consultant
Centre for Veterinary Education
The University of Sydney
E. richard.malik@sydney.edu.au

Derek Spielman
Senior Lecturer in Veterinary Pathology
Faculty of Veterinary Science
The University of Sydney
E. derek.spielman@sydney.edu.au

C&T NO. 5513

History, Signalment and Clinical Signs

Canine neural angiostrongyliasis (CNA) is a disease of dogs caused by migration of the larvae of the rat lungworm *Angiostrongylus cantonensis*. To date, this is a disease of dogs, wildlife (especially the tawny frogmouth) and animals housed in zoos, although other species such as horses can be affected. Importantly, it can cause disease in people, and young children are at greatest risk. The disease is behaving as an emerging infectious disease and has been spreading south (into NSW) from south eastern Queensland since the early 1990s.

Although dogs of any age can be affected, the disease is most common in puppies because infection generally occurs after ingestion of snails or slugs. Perhaps 8% of the population of the most common garden snail in Sydney contain infective larvae. As indiscriminate eaters, puppies eat anything, including slugs and snails, especially if they appear on or near the food bowl. Snails are more likely to be infectious in environments where rats are plentiful, often on acreage, or near aviaries or food stored outside. Adult dogs can also be infected, although often their infections are less severe than in pups. Cats are generally not infected, because (at least with experimental infections), snails are vomited after ingestion. For geoclimatic reasons, infections are most

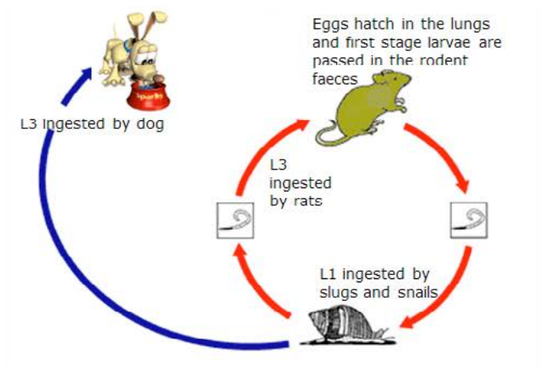


Figure 1. Life cycle of Angiostrongylus cantonensis.

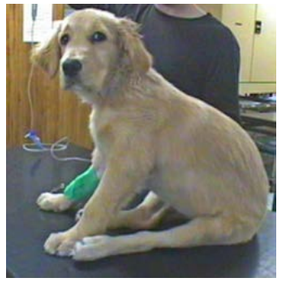


Figure 2. An affected Golden retriever pup with neural angiostrongyliasis. Note peculiar hind limb stance. This pup recovered completely with no residual neural deficits.

common in early autumn along the eastern coast of Australia, although they occur in any month; infections are rare in winter. The disease is seen in coastal localities from Jervis Bay to tropical northern Queensland.

Dogs develop infections after eating molluscs containing infective larvae. Larvae leave the gut and travel to the caudal spinal cord and meninges. Migrations of the larvae cranially within the spinal cord and meninges cause an ascending eosinophilic meningoencephalitis. Clinical signs result from a combination of the physical damage caused by migrating larvae and the eosinophilic inflammatory response to the excretory products of the metazoan parasite. The clinical picture depends on when the animal is presented, as the signs initially reflect a more caudal neuroanatomical localization, whereas later the signs are of an ascending paralysis.

The most salient feature of CNA in dogs is spinal pain, or hyperaesthesia. The pain can vary from moderate to excruciating, which can be so diffuse as to defy localization. Early on pain is generally most prominent at the tail base. Later the whole spine is affected, including the cervical spine. Detailed neurological testing usually results in a combination of upper motor neuron signs and lower motor neuron signs, which can be somewhat confusing; however the hallmark of the disease is spinal hyperaesthesia. Dogs which ingest few larvae have less florid disease, and their signs can be less syndromic, including cranial nerve abnormalities.

Interestingly, clinical signs can develop suddenly when pups or young adult dogs are given monthly flea treatments

containing macrocyclic lactones (ivermectin-like drugs) that can precipitate disease in dogs incubating CNA, as the drug kills migrating larvae *in situ*, causing sudden development of eosinophilic meningitis. Such cases often respond especially well to appropriate therapy (see later).

Diagnosis

History, signalment and clinical findings are often quite suggestive of the diagnosis, and sometimes the signs are so distinctive that a presumptive diagnosis can be made on the examination table, especially if there is an environmental history of rats and exposure to snails. Haematology often shows peripheral eosinophilia. Definitive diagnosis requires collection of CSF, either from a lumbar tap or the cisterna magna. Eosinophilic pleocytosis is strongly suggestive of CNA and a definitive diagnosis can be made by submitting CSF for an in-house ELISA assay developed by Dr Rogan Lee (a veterinary scientist) at Westmead Hospital. Dr Damien Stark at St Vincents Hospital has developed a real time qPCR for *A. cantonensis*, although so far CSF from affected dogs has failed to produce a positive result, presumably because insufficient larval nucleic acid is released into the CSF.

Neosporosis can occur in young dogs and occasionally produces somewhat similar signs; however hyperaesthesia is rarely as prominent (indeed, it is usually not evident) and, early on, the signs in the hind limbs are unambiguously lower motor neuron in type, rather than mixed or upper motor neuron in type.

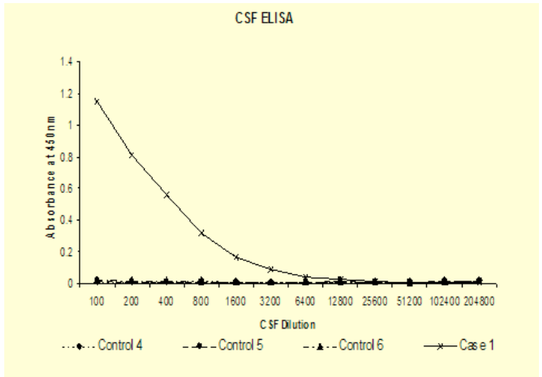


Figure 3. ELISA results for CSF form a case of neural angiostrongyliasis.

Treatment and prevention

The cornerstone of therapy for this condition is corticosteroids to dampen the eosinophilic response. Usually prednisolone is given at a dose of 1 mg/kg twice daily. In dogs with extreme pain, prednisolone can be given intravenously at the same dose rate, or replaced initially by a single injection of dexamethasone at a dose rate of 0.2 mg/kg (IV or SCI). Usually corticosteroids provide very good control of pain and hyperaesthesia, although in severe cases they can certainly be supplemented by opioids such as methadone, morphine or buprenorphine and possibly gabapentin. Although controversial, Richard Malik believes that once there is substantial clinical improvement (i.e. after 3-4 days), recovery is hastened by actually killing the larvae *in situ*, which can be achieved slowly over several days using fenbendazole or with a single dose of moxidectin (by applying Advocate™ topically). After 7-10 days, the dose of prednisolone is reduced by 50% every 5-10 days, depending on the clinical response. Tapering can be faster in young animals, where long courses of corticoids are avoided because of their adverse effects on the growing skeleton.

Although specific research has not been undertaken, or at least published, it is highly likely that monthly application of a moxidectin formulation will prevent this disease by killing larvae before they reach the spinal cord and meninges. An alternative approach would be to administer ProHeart SR-12™ from 12 weeks of age, with a further injection at 6 months when the dog is close to its adult weight, as likely the concentration of moxidectin produced from this depot preparation would be sufficient to prevent larvae reaching the central nervous system. Such an approach would currently be 'off-label'. ■

References

Lunn JA, Lee R, Smaller J, et al. Twenty two cases of canine neural angiostronglyosis in eastern Australia (2002-2005) and a review of the literature. *Parasites & Vectors*. 2012;5:70. doi:10.1186/1756-3305-5-70. (Open Access)

Malik R, Spielman D, Šlapeta JB, Gone in the back legs, *Microbiology Australia* (http://microbiology.publish. csiro.au/paper/MA13004.htm)

SMALL ANIMAL

WINNER

VITAMIN K RESPONSIVE DERMATOPATHY

Penny Reeves
BVSc, CertVetAc
E. rutherglenvet@gmail.com

Rutherglen Vet Clinic
149 Main St, Rutherglen VIC

C&T NO. 5514

'Mika' is an 8-year-old female neutered kelpie cross, suffering from a congenital keratinisation defect. This defect is characterised by excessive scale, poor elasticity and flexibility of the skin. Mika was sore to touch, had difficulty walking due to foot pain, her skin was tight and wouldn't 'tent', she was largely inactive, suffered from pyoderma and otitis, severe dandruff and large pieces of skin would slough regularly.

Mika was managed with Phytosphingosine shampoo weekly (relating molecule for skin turnover), Acetretin (Roaccutane®) 20mg sid (to slow down epidermal turnover) and was placed on a low fat diet (due to the retinoid side effects of Acetretin affecting lipid metabolism). This treatment improved her skin elasticity, there was less skin sloughing, less severe and fewer pyoderma episodes while still being itchy. She had an improved activity level. Her otitis was not resolving and due to the side effects of Acetretin, she also had dry eye.

Mika ingested Rat Sac and was given Vitamin K for 3 weeks. At this time the owner ceased her Roaccutane. Mika's skin started to show improvement soon afterwards and continued to improve over the 3 weeks. Improvements were seen in elasticity, less scale, glossy coat, no itch and she only had to be bathed every 3 weeks, compared to weekly.

Figure 1.

Soon after the Vitamin K was stopped the benefits were lost and her condition deteriorated to what it was prior to starting the Vitamin K.

Vitamin K was once again administered at a dose of 12.5mg sid with the Roaccutane®. Her response was less dramatic and slower. As the Vitamin K needs to be administered with a fatty meal we decided to stop the Roaccutane® (which must be given to a patient on a low fat diet) and her condition continued to improve.

Two months later her behaviour has changed; she is now described as affectionate, energetic and playful, and she likes to be petted. She runs and jumps to catch a ball, her skin is elastic, she can walk on concrete and she no longer has ocular discharge from the dry eye side effects of the retinoids. She is only bathed monthly.

Over 6 months later she still has occasional otitis and a small amount of dandruff (she had one relapse when the Vitamin K was not administered with a fatty meal).

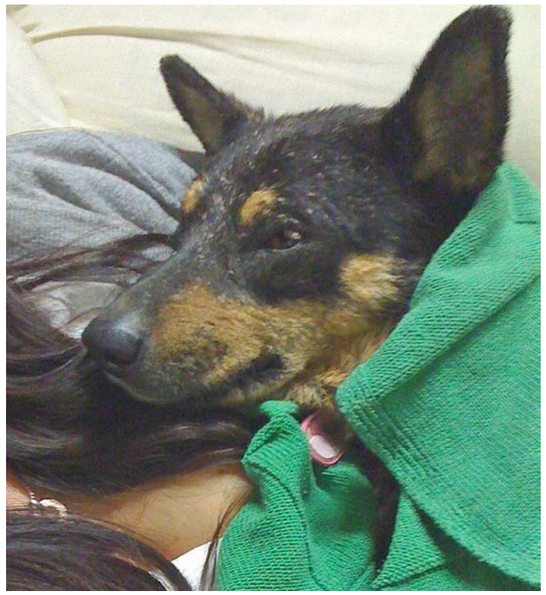


Figure 2. Mika before Vitamin K.

Currently we are trialling a higher dose of Vitamin K.

Vitamin K1 Phylloquinone is a fat soluble vitamin which is not stored in the body; it is found in leafy green vegetables such as lettuce, broccoli and spinach. Vitamin K2 (menaquinones) can be synthesized from K1 by microflora in the gut and are also found in the diet in meat and fermented food products like cheese.

Vitamin K is said to be involved in the carboxylation (activation) of the Matrix GLA protein (Matrix ɣ-carboxyglutamic acid protein) which inhibits soft connective tissue calcification including in the skin and the cardiovascular system. By preventing calcification of the elastin within the skin, this may be why Mika's skin is becoming more elastic.

Disruptions in gastrointestinal absorption can give rise to a deficiency in the vitamin level though Mika never displayed any signs of gastrointestinal disease. I would encourage any further discussion on Vitamin K's role in skin disease. ■

Figure 3. Mika on Vitamin K.

Post-script from Penny November 2015

Unfortunately, we euthanased Mika last month as she had a peripheral lymphadenopathy, with her inguinal and prescapular lymph nodes most affected. Biopsies confirmed a malignant round cell tumour...

Lab comments: The histologic morphology of the neoplastic cells is most suggestive of a malignant plasma cell tumour; however, an anaplastic lymphoma cannot be ruled out.

These tumours had restricted her venous blood flow and were causing oedema of her legs.

The owner was happy that at least for the past 12 months she had had a playful, energetic dog.

SMALL ANIMAL

INVITED COMMENT COURTESY OF

Greg Burton
BVSc (Hons), MACVSc (Small Animal Surgery), FACVSc (Dermatology)
Melbourne Veterinary Referral Centre &
The University of Melbourne
E. derm@melbvet.com.au

C&T NO. 5515

My initial contact with Mika was a request to review a repeat biopsy from 'Mika'. The history stated 'Mika was seen by Mandy Burrows in Perth as a puppy and her original biopsy was reviewed by Ken Mason and a histological diagnosis of Idiopathic Seborrhoea given'. I reviewed the second biopsy of Mika in 2010 (aged 3 years) and the histological features at that stage were difficult to interpret due to massive bacterial and fungal overgrowth leading to epidermal hyperplasia (epidermal hyperplasia is not typical in primary keratinisation defects).

I subsequently examined Mika in Wodonga (regional clinic) in 2010 and Mika's clinical features supported a diagnosis of a primary keratinisation defect with massive surface infection. Resolving the infection did not resolve the generalised hyperkeratosis.

I prescribed the phytosphingosine treatments and the isotretinoin therapy as both have actions to promote terminal differentiation of keratinocytes and normalise keratinisation.

I saw Mika while on retinoids and phytosphingosine in 2012 and then again in 2015 while on vitamin K and my examination is below:-

Thursday, 20 September 2012

AISRecLoc:0001150300 Physical examination (8:50 pm Greg Burton (GW)/GGB)

Weight: 15.700 kg, 34.54 lb, 0.63 m2

Mika's physical examination revealed Mica is travelling really well. There is excessive scale still but this is really minimal compared to previous visits and the skin underneath is almost normal. The ventral abdomen is still less flexible but there is no fissuring and the no scale and no infection!

The ears are very colonised with mixed bacteria but no pus and minimal itch. There is moderate keratinous debris. Joints palpate normally.

Thursday, 26 February 2015

AISRecLoc:0001402123 Physical examination
(5:38 pm Greg Burton (GW)/GGB)

Weight: 15.700 kg, 34.54 lb, 0.63 m2

Mika's physical examination revealed dramatic improvement. The skin was still scaly but mild and patchy rather than generalised. There was almost normal elasticity in the ventral abdominal and axillary skin and Mika is much more comfortable and cytologically free of infection on her skin. The ears were very itchy and ulcerated with purulent exudate but even the ears were better in that there was no compaction with scale.

My comment on Thursday, 26 February 2015

Mika is definitely improved by the vitamin K therapy. As discussed, there is evidence that vitamin K plays a role in calcium metabolism in the various tissues and calcium gradient affects differentiation in the skin and expression of some of the skin adhesion molecules so it may be that this is the link in Mika's case.

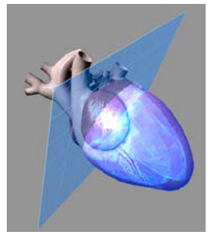
NOTE: There were no dermal changes on biopsy. Loss of elasticity was due, in my clinical opinion, to the total lack of hydration of the epidermis due to profound skin barrier dysfunction and increased trans-epidermal water loss. This is an epidermal disease.

I did recommend (to the owner) that a repeat biopsy was indicated while on vitamin K as this would facilitate publication as a case report. ■

Note: *Look out for Mandy Burrows' comment on this case in the upcoming March 2016 Issue 282.*

INTERESTING LINKS

From the ISFM Forum: Thanks to Aga Zoltowska for sharing these 2 links:



An interesting interactive app about heart... Worth checking out....

http://www.kpn-interactive.com

Useful toxicity app by ASPCA

https://itunes.apple.com/gb/app/apcc-by-aspca/id954896166?mt=8

DISTANCE EDUCATION

CONGRATULATIONS TO THE DISTANCE EDUCATION CLASS OF 2015

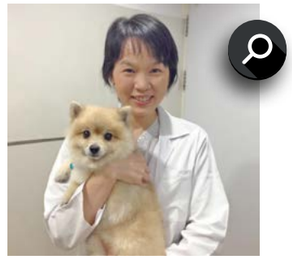
Distance Education is demanding and requires dedication and commitment, especially when juggling study requirements with work and family.

Congratulations to you all for successfully completely a vigorous but rewarding year of continuing professional development.

CVE Tutors & Staff.

2016 DE EARLY BIRD – WINNERS

Paying early ensured the following vets not only secured a place in the DE course of their choice for 2016, they also received a hefty discount and were the 3 lucky winners in our Early Bird draw, winning an iPad 3 mini each.



Congratulations to:-

- Dr Ellena Hillbrich, Australia: Sonology
- Mrs Nan Choisunirachon, Thailand: Sonology (pictured above)
- Dr Joyce Tang, Hong Kong: Abdominal Imaging

and to all our DE Participants who enrolled and secured their place in one of our 2016 courses.

SMALL ANIMAL

INVITED COMMENTARY ON C&T NO. 5499 VOMITING AND WEIGHT LOSS IN A YOUNG CAT

(Sept 2015 Issue 280)

Read C&T NO. 5499 in the eBook

C&T NO. 5516

Jan Šlapeta

MVDr PhD GradCertEd (Higher Ed)

Associate Professor in Veterinary Parasitology
Laboratory of Veterinary Parasitology
Faculty of Veterinary Science, The University of Sydney

Deputy Editor: ***International Journal for Parasitology***

E. jan.slapeta@sydney.edu.au
T. +61 2 9351 2025

Cryptosporidium is only rarely detected in stool samples of cats. The most comprehensive Australia wide study on this subject by Palmer *et al*. (2008) detected *Cryptosporidium* in 3.5% of 491 refuge cats and 1% of 572 cats in vet practices. Such scarce records make it very difficult to access material to be used in validation of antibody based tests and PCR tests. Most in-clinic rapid antigen tests are only validated for the zoonotic *C. parvum* bovine genotype and *C. hominis*. The more reliable test is to use antibodies that target the oocyst wall; these antibodies detect conserved surface molecules and we know that they can label all cryptosporidia we have so far handled including *C. molnari* from fish (Barugahare *et al*. 2011). It is the gold standard test and also our preferred test for 'strange' cryptosporidia at the Parasitology Lab, FVS, USyd. Application of PCR is potentially a good approach, but note that not all PCRs will be pan-*Cryptosporidium*. Therefore, checking with the lab if their test detects *C. felis* or *C. muris* is always worthwhile.

One probably ponders what is the real zoonotic potential of the cryptosporidia involved in this case? During routine typing of 14,469 human cases of cryptosporidiosis in the UK, *C. felis* ranked the fourth most commonly encountered species - 426 (n = 38), following the top three: *C. parvum* bovine genotype, *C. hominis*, and *C. meleagridis*. In this comprehensive UK study, significant risk factors were contacts of subjects with immunocompromised cats (Elwin *et al*., 2012). *C. muris* was on several occasions detected in HIV-positive human subjects in their stool samples. Therefore, *C. felis* and *C. muris* are considered of moderate and minor public health significance, respectively (Šlapeta, 2013). ■

References

Barugahare R, Dennis MM, Becker JA, Šlapeta J. Detection of Cryptosporidium molnari oocysts from fish by fluorescent-antibody staining assays for Cryptosporidium spp. affecting humans. *Appl Environ Microbiol*. 2011 Mar;77(5):1878-80. doi: 10.1128/AEM.02691-10. Epub 2011 Jan 14.

Elwin K, Hadfield SJ, Robinson G, Chalmers RM. The epidemiology of sporadic human infections with unusual cryptosporidia detected during routine typing in England and Wales, 2000-2008. *Epidemiol Infect.* 2012 Apr;140(4):673-83. doi: 10.1017/S0950268811000860. Epub 2011 Jun 7.

Palmer CS, Thompson RC, Traub RJ, Rees R, Robertson ID. National study of the gastrointestinal parasites of dogs and cats in Australia. *Vet Parasitol*. 2008 Feb 14;151(2-4):181-90. Epub 2007 Nov 1.

Šlapeta J. Cryptosporidiosis and Cryptosporidium species in animals and humans: a thirty colour rainbow? *Int J Parasitol*. 2013 Nov;43(12-13):957-70. doi: 10.1016/j.ijpara.2013.07.005. Epub 2013 Aug 20.

Andrea Harvey

BVSc DSAM(Feline) DipECVIM-CA MRCVS MANZCVS (Assoc)
Registered Veterinary Specialist, NSW

PhD Scholar, Centre for Compassionate Conservation
School of Life Sciences, University of Technology Sydney

E. andreaharvey.cat@gmail.com

Wow, what an interesting case and so well written by this astute veterinarian. Thank you for writing this up to share with C&T readers! I have certainly never come across this before myself. I think that it is a really important reminder for veterinarians to remember to consider infectious causes of these clinical signs, albeit if they are further down the list. Although we need to also remember that 'common things occur commonly' and to be sensible and practical in our approach, with inflammatory bowel disease probably being top of the differential diagnosis list, it can be all too easy to reach for the corticosteroids without complete investigation or thoughts regarding infectious aetiologies, for which immunosuppressive doses of corticosteroids could have disastrous consequences!

Whilst I haven't seen *Cryptosporidium* causing these signs and pathology in cats, I have seen an increasing number of cats that appear to have clinical signs associated with spiral bacteria. It is always hard to prove cause and effect, but the cases I have seen either had convincing pathology with large numbers of spiral bacteria present, and/or already had extensive treatment trials with diet, corticosteroids and additional symptomatic treatments, with no improvement, and then had resolution of their clinical signs with combination treatment with omeprazole, amoxycillin and metronidazole. So, these cases to me seem convincing that either spiral bacteria were the cause, OR there is another unknown antibiotic responsive condition in these cats. This *Cryptosporidium* case reminds me of these cases and the importance, as the author points out, of having more

unusual causes of these clinical signs on your radar by thinking through all differential diagnoses at the start, and reviewing your differential diagnosis list if the case isn't progressing as you hoped.

In this case, although corticosteroids may have been required to reduce secondary inflammation, it would have been interesting to see if there was clinical response to the azithromycin alone. I guess what we don't know in this case is cause and effect; was all the pathology caused by the *Cryptosporidium*, or did the cat have underlying inflammatory bowel disease and the 'damaged' intestinal epithelium as a result allowed *Cryptosporidium* to proliferate?

It is often difficult to prove cause and effect in veterinary medicine, and this case had a great outcome which is the most important thing. I suppose what I might have done differently was treat with say 1 week of azithromycin first, and if not sufficient improvement, then add in the corticosteroids. I think with the gastric pathology, I would have also been tempted to add some gastroprotectants like omeprazole and sucralfate. However, there is no right or wrong answer, and the cat had a great outcome, so well done to the veterinarian treating this case! ■

SMALL ANIMAL

PET OWNERSHIP & PHYSICAL HEALTH

Thanks to Linda Mayer for sharing this interesting abstract from: Robert L. Matchock, 2015, Pet Ownership and Physical Health, *Curr Opin Psychiatry*, 2015;28(5):386-392.

Among owners attached to their dogs, the longer dogs gazed at their owners, the higher the level of oxytocin in the owners, at least as measured by urinary oxytocin, which may not be the most reliable way to sample oxytocin. Furthermore, if dogs are given intranasal oxytocin, dogs affiliate more with, and have more social orientation toward, their human owners. Beetz *et al.* cogently argue that most beneficial psychological and physiological effects of HAIs are mediated by the pituitary peptide hormone, oxytocin (e.g., attenuating both components of the hypothalamic-pituitary-adrenal axis – glucocorticoids and cardiovascular responsiveness). Oxytocin is traditionally viewed as being implicated in parturition and lactation, but its importance in social bonding is starting to be recognized. More research is needed on the biochemical correlates of HAIs, especially cortisol and oxytocin. ■

SMALL ANIMAL

THE DEBATE ABOUT FERAL CATS

Andrea Harvey

BVSc, DSAM (Feline), Diplomate ECVIM-CA, ANZCVS (Associate), MRCVS
Registered Veterinary Specialist, NSW

PhD Scholar, Centre for Compassionate Conservation
University of Technology Sydney

E. andreaharvey.cat@gmail.com

C&T NO. 5517

For clarification, the term 'feral cats' is being used here to describe completely wild cats that are born outside of human society and live in wilderness areas away from towns and cities, and have no contact with people. It is important to differentiate this population from unowned or stray cats since the issues of these two populations are very different. Unowned or stray cats consist of domestic cats that have been lost or abandoned, and unwanted kittens from these cats. They tend to live in larger colonies than feral cats, live in urban or semi-urban areas and are largely dependent on human society for food and shelter.

Just days before International Cat Day, the Australian government announced its plans to kill two million feral cats by 2020. What is the stance of veterinarians on this?

Many veterinarians would initially accept this as a necessity and simply seek to ensure that this is undertaken as humanely as possible; we are all led to believe that lethal culling is the only way to control destruction of native wildlife by feral cats. But is this true? According to well-respected ecologists, Dr Arian Wallach and Dr Daniel Ramp, 'Killing cats achieves only one outcome with consistency: it produces dead cats.'

Wallach & Ramp's recent article in *The Conversation* (29 July 2015, http://theconversation.com/lets-give-feral-cats-their-citizenship-45165) entitled 'Let's give feral cats their citizenship' not surprisingly attracted much controversy, but offered a refreshing alternative view to the feral cat debate, encouraging readers to question both the ethics of mass killing of feral cats, and the ecological effectiveness in achieving its aims.

Figure 1. Feral cats are typically portrayed as hissing and aggressive looking, which enhances the negative perception of them, but this body language is simply illustrating that the cat is feeling very anxious in its current situation – hissing and growling is its way of asking to be left alone! (Source: http://www.abc.net.au/news/2015-07-20/feral-cat-growls-in-the-bush/6634036)

They suggest that it is naïve to think that killing cats will simply have the desired outcome of allowing native wildlife to flourish. What if the predominant cause of loss of native wildlife is related to habitat destruction by humans? What if feral cat populations continue to proliferate because humans are also killing Australia's apex predator, the Dingo, causing disarray of the trophic cascade? What if killing cats has knock-on ecological impacts, which just create another problem such as explosion of rodent and rabbit populations? There is also evidence that killing feral cats may not have positive ecological impacts because cats from other areas simply fill their niches.

Wallach and Ramp go on to explain; 'ecosystems are notoriously, and wonderfully, complex things. They are comprised of dense networks of interactions that bind the fate of species to one another. Cats have become deeply entangled in this web of life.'

So it turns out that killing feral cats may not have the perceived benefits that we have been led to believe by many conservation movements. Decisions regarding large scale lethal culling directly impacts animal welfare, so as veterinarians we need to be able to critique the evidence surrounding the necessity and impacts of culling. It isn't acceptable to take the view that this is beyond our remit.

What about the ethics of killing any healthy sentient being? Many veterinarians would struggle to make the decision to humanely euthanase a perfectly healthy cat because its owner no longer wanted it. If we struggle with the ethics of this, why would we think it ethically acceptable to kill millions of unowned cats, with some vague unproven hope that this would result in native wildlife flourishing?

As veterinarians, every week we go to great extents in our efforts, time and resources to extend the life, perhaps only for a few months, of often terminally ill patients, and often at great financial expense to the owners; how can we justify this, and then readily accept killing millions of healthy animals just because they don't have an owner?

Figure 2. Feral cats killing native wildlife is the justification being used for a widespread cull of feral cats, but does predation justify the large scale killing of predators? (Source: http://www.theaustralian.com.au/national-affairs/greg-hunt-calls-for-eradication-of-feral-cats-that-kill)

The vision of International Cat Care (of which the International Society of Feline Medicine is the veterinary division), is that all cats, owned and unowned, are treated with care, compassion and understanding. Many veterinarians exhibit a huge amount of care, compassion and understanding for their feline patients, but is this regularly extended to unowned and feral cats as well?

Interestingly, similar questions were recently raised surrounding the worldwide outrage at the killing of Cecil the lion, by the trophy hunting American dentist. The general public throughout the world seemed to be united in the view that killing Cecil was ethically unacceptable. So if we agree that it was wrong to kill Cecil the lion, why would we accept it is OK to kill any other lion? Many lions are killed with no reaction at all from the public, so what is the difference about Cecil? He had a name, people knew who he was, he was part of a research project, and he had an individual identity. It is common in our society for more value to be placed on individual animals that have a name, or other personal identity, compared to an unidentified member of the same species, but of course there is no difference in their degree of sentience.

Similarly, if we accept that it isn't ethical for our neighbor to kill our cat if it toilets in their flowerbed, why would we think it ethical to kill any healthy cat, the only difference being that it doesn't have an owner or an individual identity recognized by us? The difference that often drives societies' decisions is that the latter would be against the law, whereas the former is encouraged by the law. As the Honorable Michael Kirby (former Chief Justice of the High Court of Australia) recently stated at an animal law conference 'the law can be oppressive, ignorant and often fails, it needs continual reconsideration'. Thus the law should not be relied upon as a guide to ethical decision making.

When it comes to naming animals, the prejudice goes even further in the division between owned and unowned animals. Typically, animals that society has chosen to consider undesirable, are grouped together with a negatively biased name such as 'pest', 'feral', 'invasive', introduced' etc. These terms do little more than further exacerbate the prejudice against them, and is clearly not an ethical way of distinguishing between species, or between members of the same species. Similarly, 'feral' cats are usually illustrated as a growling hissing cat, suggesting they are nasty and aggressive when, as feline vets, we know that this body language simply represents a highly anxious cat that feels threatened. All of

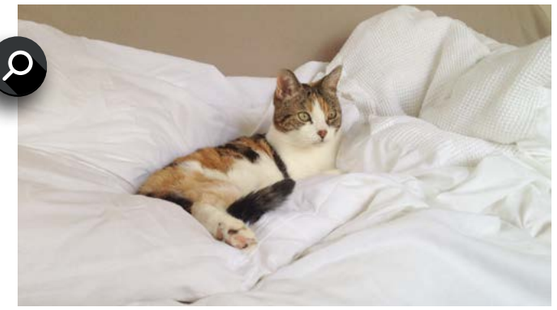


Figure 3. 'Binks' has a plethora of comfortable beds to choose from and favourite treats on demand. Is it ethical to place more value on individual cats that we name and which share our lives whilst also condoning the killing of feral cats that have the same degree of sentience?

this results in the connotation that feral cats are ‘bad’ and helps society to justify killing them.

To this end, Wallach and Ramp ingeniously suggested renaming the countries feral cats, ‘Australian wild cats’. This would certainly help a transition to embracing them as part of Australia’s present day ecosystem.

Conditioned ethical blindness is a common occurrence in society where some kind of ‘reward’, which may simply be obtaining or holding down a particular job, conditions people to ignore the ethical issues around them. This is very much the case in the conservation field, where ‘pest control’ has become a huge ‘industry’. Within these industries, academic fields, and also wider society, there is also much pressure to conform to accepted opinions; which most often is that ‘introduced species damage the ecosystem and should be killed’.

As ecologists, not animal welfare scientists or veterinarians, Wallach and Ramp are to be applauded for bravely pushing through ethical blindness, and past the pressure to conform to conservationist dogma, leading the way in bringing ethical decision making to the table through a compassionate conservation approach.

Most veterinarians probably wouldn’t accept the notion that the best outcome is not to intervene with the cat populations at all, and would worry about the welfare costs of this. However, most veterinarians are probably more familiar with stray/unowned populations than true feral cats (see definitions in dialogue box). Certainly in stray/unowned populations we know that morbidity and mortality is high, and that individuals in these populations have very poor welfare. They typically start reproducing at one year of age or less and produce multiple litters a year. Feline infectious diseases are particularly common in this scenario, contributing to significant morbidity in kittens. The physiological strain on the queen is also considerable.

International Cat Care acknowledge that controlling unowned cat populations is a major international welfare challenge. Reducing population growth rates can significantly improve health and welfare at an individual and population level. Trap-neuter-return programmes can be successful in some parts of the world, but in others are inadequate or unachievable. The Alliance for Contraception in Cats and Dogs is a non-profit organisation that serves to advance methods of non-surgical reproductive control, and significant research is ongoing in this area, as highlighted in a recent Special Issue of *Journal of Feline Medicine & Surgery*, dedicated to ‘Non-surgical Feline Fertility Control’ (*JFMS* Sept 2015, Vol 17, Issue 9).

It is important, however, to differentiate the issue of stray/unowned cats from that of feral cats, which is the topic of discussion here, as they are different populations which will require different management strategies. As Wallach and Ramp point out, the wildcat populations in Australia roam remote, vast landscapes, desert and dense bush in often inaccessible areas, and intervention of reproduction as can be performed in unowned/stray populations is not practical in true wild populations. These cats are also more commonly solitary, with much lower reproduction rates to unowned/stray cats that tend to live in colonies e.g. around garbage tips or university campuses.

Instead, Wallach and Ramp propose that coexistence of native wildlife with wild cats is possible, stating that ‘the major forces that influence the ability of prey to coexist with cats include vegetation cover and larger predators’, and so this is where efforts would be best focused.

Whether or not readers agree with Wallach and Ramp’s views on the feral cat debate, the authors’ excellent discussion points force us to question each step of the decision making in wildlife management, and our own ethical values, and encourage us to step out of states of conditioned ethical blindness, and away from the pressure to conform with dogma.

Wallach and Ramp conclude, ‘the aim of conservation is not to generate an ever increasing body count, but to guide human behaviour to enable the rest of the Earth’s species to flourish. Embracing cats is a paradigm shift. It means embracing the entirety of Australia’s modern ecosystems - native and feral - and letting go of the past.’

After all, which introduced species has done and continues to do most damage to the Australian environment, wildlife and ecosystems? Introduced humans of course. With all the advances in other areas of science and medicine, there has to be better solutions than repeated large scale lethal culling for infinity of species we have singled out to place blame on. There has to be a better way forward into the future. ■

SMALL ANIMAL

NUTRITION

Dr Billinghurst Lecture Recording

C&T NO. 5518

View the video of Dr Billinghurst’s lecture in the eBook

Dr Billinghurst graduated BScAgr in 1966 from Sydney University and in 1976 obtained his BVSc with Honours. Working in small animal practice, he developed a strong interest in evolutionary nutrition and studied extensively in this area. Dr Billinghurst has published 3 books on companion animal nutrition and lectured around the world on this topic for the past 20 years.

Now retired from clinical practice, Dr Billinghurst focuses on evolutionary nutrition; he consults, conducts clinical and literature research, write and lectures on this topic.

Interesting article by Dr Billinghurst here:
http://titlestand.com/ebook/ebook?id=10084147&ts=1#/26

Thank you to Duncan Houston, 3rd year Sydney University Vet Student, for supplying this link. ■

SMALL ANIMAL / FROM THE DE FILES

ONCOLOGY IN THE CAT

Xiangting (Kitty) Huang

E. kitty.hxt@gmail.com

Kitty was a 2013 CVE & ISFM Feline Medicine DE participant and this C&T was an assignment in one of the Oncology Modules.

Singapore

WINNER

C&T NO. 5519

QUESTION 1

Lymphoma (or lymphosarcoma) is the most common malignant tumour of cats. Treatment of this cancer with sequential multi-agent chemotherapy is inexpensive in terms of the actual drugs, but few vets offer this service to clients, and often clients cannot afford the expense of treatment at a referral centre.

Consider your own practice and chose a suitable chemotherapy protocol that is both user-friendly and affordable for your clients. Work out the cost of treating a 4kg cat for 6 months. Include all costs such as drug administration fees, costs of drugs, laboratory testing and adjunctive drugs e.g. anti-emetics etc and disposal of waste.

	Treatment	*Dosage for 4kg cat (=0.252 m²)*	*Cost*
Week 1	Cyclophosphamide Vincristine Prednisolone	x1 50mg tablet PO 0.13mls IV (new bottle) x2 5mg tablets PO daily for 7 days IV catheter set up	$5.00 $55.00 $(0.8 x14)= $11.20 $20.00
Week 2	Vincristine Prednisolone	0.13mls IV – using the same vial from week 1 x1 5mg tablet PO alternate days IV catheter set up	No charge $(0.8 x4) = $3.20 $20.00
Week 3	Vincristine Prednisolone	0.13mls IV – using the same vial from week 1 x1 5mg tablet PO alternate days Iv catheter set up	No charge $(0.8 x4) = $3.20 $20.00
Week 4	Cyclophosphamide Vincristine Prednisolone	x1 50mg tablet PO 0.13mls IV – using the same vial from week 1 x1 5mg tablet PO alternate days Iv catheter set up	$5.00 No Charge $(0.8 x4)= $3.20 $20.00
Week 5	Vincristine Prednisolone	0.13mls IV (new bottle) x1 5mg tablet PO alternate days Iv catheter set up	$ 55.00 $(0.8 x4) = $3.20 $20.00
Week 6	Vincristine Prednisolone	0.13mls IV – using the same vial from week 5 x1 5mg tablet PO alternate days Iv catheter set up	No charge $(0.8 x4) = $3.20 $20.00
Week 7	Cyclophosphamide Vincristine Prednisolone	x1 50mg tablet PO 0.13mls IV – using the same vial from week 5 x1 5mg tablet PO alternate days Iv catheter set up	$5.00 No Charge $(0.8 x4)= $3.20 $20.00
Week 8	Vincristine Prednisolone	0.13mls IV – using the same vial from week 5 x1 5mg tablet PO alternate days Iv catheter set up	No charge $(0.8 x4) = $3.20 $20.00
	Weekly CBC Weekly Review		$(40 x8) = $320.00 $(20 x8) = $160.00

Table 1. Induction with COP Protocol (6-8 weeks).

Chemotherapy Protocol for GI Lymphoma in Cats

Induction with COP Protocol (6-8 weeks): (See: Table 1.)

C= Cyclophosphamide (Cytoxan)
200-300mg/m^2 PO q3wks
Comes in 50 mg tablets

O= Oncovin (Vincristine)
0.5mg/m^2 IV weekly
Comes in 1 mg in 1 ml vials

P= Prednisolone
50mg/m^2 PO q24h for 1 week then 20mg/m^2 PO q48
Comes in 5 mg tablets

- Weekly Complete Blood Count (CBC) is recommended to monitor if any cytopenias occur. If significant, delay chemotherapy for a week and repeat CBC.
- Toxicity during induction is minimal.
- Treatment with cyproheptadine (periactin 4mg) @ 1-2mg/cat PO BID as required if cat becomes anoretic. Estimated cost a week is $(0.25 x7) = $1.75 if necessary.

The cost of chemotherapy during induction period including weekly CBC checks is approximately $798.60.

Maintenance with LMP Protocol after 6-8 weeks induction (See: Table 2.)

L= Chlorambuil (Leukeran)
20mg/m^2 PO q2wks
Comes in 2mg tablets

M= Methotrexate
2.5mg/m^2 PO 2-3 times/ week
Comes in 2.5mg tablets

P= Prednisolone
20mg/m^2 PO q48h
Comes in 5 mg tabs

- Review every 6-8 weeks for a CBC. (A CBC cost $40.00 and a review consult cost $20.00)
- * = Dosage rounded down a little to prevent splitting of tablets.
- ^ = Requires splitting of tablets.
(Owners are given option to have drug compounded. Advice gloving when handling all chemotherapy drugs. Split tablet is placed into an empty capsule before administration).
- Toxicity during maintenance therapy is minimal and intensive monitoring by veterinarian is not necessary.
- Methotrexate can cause GIT signs such as anorexia, vomiting and diarrhoea.

The approximate cost of maintenance chemotherapy for a month is $48.80.

Therefore for 4 month period it would cost **$195.20.**

In a 4 month period, approx 2 CBC and 2 reviews will be done; this will cost **$120.00**

Total cost for 4 month maintenance therapy is $315.20.

Total cost for chemotherapy for 6 month period with the above protocol is estimated to be $1,113.80.

The following drugs may also be required during the chemotherapy period:

- Cerenia injection to prevent or treat vomiting ($30.00 per injection)
- Cyproheptadine or mirtazapine for appetite stimulation ($1.75 per week)
- Prokolin for diarrhoea ($28.00 per tube lasting a week of treatment).

	Treatment	*Dosage for 4kg cat (=0.252 m^2)*	*Cost*
Week 9	Chlorambucil Methotrexate Prednisolone	x2 of 2mg tablet PO * x1/4 of 2.5mg tablet three times a week ^ x1 5mg tablet PO alternate days	$(6 x 2)= $12.00 $(3 x 1)= $3.00 $(0.8 x 4)= $3.20
Week 10	Methotrexate Prednisolone	x1/4 of 2.5mg tablet three times a week x1 5mg tablet PO alternate days	$(3 x 1)= $3.00 $(0.8 x 4)= $3.20
Week 11	Chlorambucil Methotrexate Prednisolone	x2 of 2mg tablet PO * x1/4 of 2.5mg tablet three times a week x1 5mg tablet PO alternate days	$(6 x 2)= $12.00 $(3 x 1)= $3.00 $(0.8 x 4)= $3.20
Week 12	Methotrexate Prednisolone	x1/4 of 2.5mg tablet three times a week x1 5mg tablet PO alternate days	$(3 x 1)= $3.00 $(0.8 x 4)= $3.20

Table 2. Maintenance with LMP Protocol after 6-8 weeks induction.

QUESTION 2

Choose an interesting oncology case that you have dealt within the past 1-2 years. Also make a multimedia PowerPoint presentation of the case using the CVE template. Try to choose a case with good pictures – patient, lesion, radiographs, ultrasound, cytology/histology etc. In your report, summarise the signalment, history, presenting signs, investigations, lab tests etc, differential diagnosis, diagnosis, treatment and outcome. The case does not have to be a 'perfect' case, but try to analyse what could have been done differently or better and what you might do differently next time.

Case Study Report

Signalment, history, presenting signs and physical examination

'Socks' is a black and white male neutered domestic shorthair cat, approximately 10-years-old that lives at a cattery.

In the past 6 months, Socks had on-and-off diarrhoea that was not responsive to different medications and gastro-intestinal diets. He was last seen by a colleague approximately 7 months ago at the clinic for diarrhoea with blood. During that visit, no obvious abnormalities were detected on physical examination and Socks was treated symptomatically with Stormogyl®, deworming, tramadol and subcutaneous fluid therapy. He was also started on Hill's i/d® diet. A liver and kidney profile blood test done at that time was also unremarkable.

According to the caregiver at the cattery, the diarrhoea did respond to treatment initially and no blood was noted in the stools anymore. However, the problem reoccurred every now and then but because Socks was clinically well, no action was taken.

On 22nd July 2012, Socks was presented for chronic diarrhoea and weight loss. Caregiver mentioned that Sock's appetite was still good and he was still active. Unsure about any vomiting episode but cat was otherwise well.

Socks was bright, alert and responsive on presentation. Other physical examination findings are as follows:

- Weight 3.6 kg and body condition score 2/5
- Temperature 39.2°C
- Mucous membranes pink and CRT within normal limits
- Eyes and Ears OK
- Heart and Lung auscultation OK
- Two firm masses palpable along intestinal tract. One approximately 3cm in diameter and the other approximately half the size.

Socks did not appear to be in pain or uncomfortable during abdominal palpation.

Diagnostics, results, case management and outcome

Blood was collected for CBC and Comprehensive profile. Results are as follows: (See Figure. 1)

Blood test results were fairly non specific. White blood cell count was borderline high and platelet count slightly below reference range. Elevated amylase may be associated with pancreatic disease/intestinal mucosal disease/intra-abdominal disorders. Elevated globulin count was likely to be associated with an inflammatory or neoplastic process.

The caregiver was informed about the findings and discussion was made with regards to performing an ultrasound scan versus going straight to an exploratory laparotomy and biopsy.

To save cost, the caregiver opted for an exploratory laparotomy and biopsy. Intravenous fluid therapy was initiated and Socks was fasted for surgery the following day (day 2). During the exploratory laparotomy, 2 masses were found along the jejunum. The larger mass was located proximally and was approximately 3cm in diameter while the smaller mass was approximately 1.5cm in diameter. Mesenteric lymph nodes were also enlarged.

A segment of jejunum was resected and anastamosis was performed to have the larger mass removed. A mesenteric lymph node was also removed for histopathology. All other organs appeared grossly normal. Socks had cefazolin antibiotics intravenously and had a fentanyl patch placed for continual pain relief. A single dose of buprenorphine was given post operatively.

Socks had an uneventful recovery post operatively and was alert and responsive on day 3. He was offered some tinned Hill's i/d® and ate well. Socks was discharged on day 4 and was sent back on a course of amoxicillin clavulanate acid antibiotics.

Histopathology findings are as follows:

- Intestinal mass (jejunum): Lymphoproliferation atypical, large cell
- Mesenteric Lymph node: Lymphoproliferation atypical, large cell

Comments: From the sections examined, there is a marked dissecting proliferation of moderately large lymphocytes with 2-5 mitosis per high power field effacing the mural and submucosal layers with encroachment upon the mucosal layer interspersed by multifocal to moderate mixed inflammation, oedema, necrosis and moderate multifocal mural immature fibrovascular proliferation; **a malignancy of the lymphoid lineage is indicated.** Additional clinical diagnostics and surveillance are warranted.

With the histopathology findings, lymphoma was concluded as a diagnosis and chemotherapy treatment options were discussed with the caregiver. The option of referral was

given to the caregiver if she wished for the slightly more complex chemotherapy protocol. The caregiver opted for the cat to remain under my care and so we started on the COP induction protocol (cyclophosphamide, vincristine and prednisolone) and LMP maintenance protocol (chlorambucil methotrexate and prednisolone) reflected in answer of Q1.

Socks responded extremely well to the chemotherapy and did not exhibit any side-effects or complications. Weekly CBC was not performed due to cost concerns and chemotherapy was performed as long as Socks was clinically well. The caregiver was aware of potential risks and complications associated with chemotherapy. The smaller

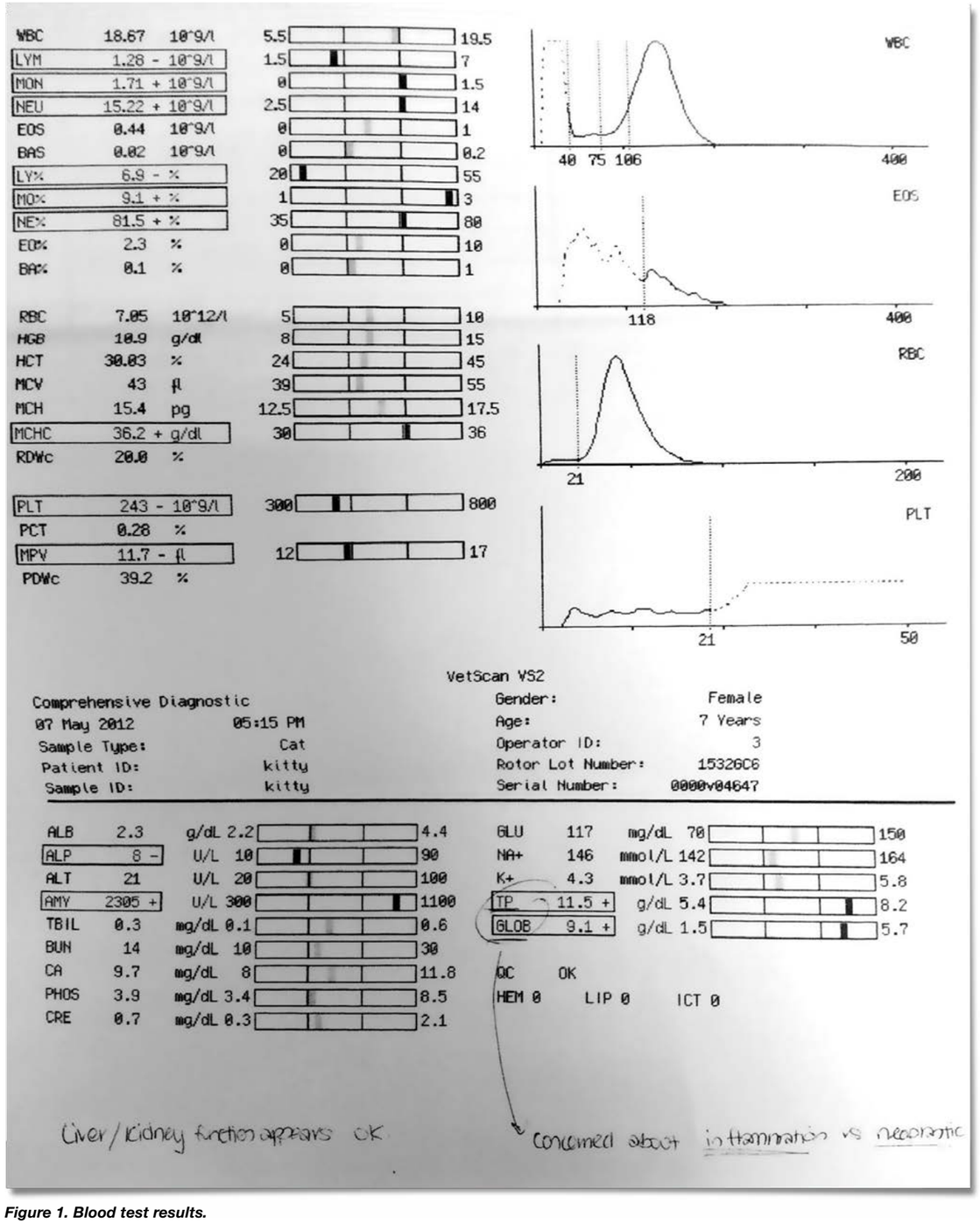


Figure 1. Blood test results.

mass along the jejunum was not palpable after the 2nd chemotherapy session and the diarrhoea gradually improved over the first few sessions of chemotherapy. Diet was also switched from Hills i/d® to Royal Canin indoor formula once the diarrhoea resolved. Sock's weight also gradually increased from the initial 2.8kg to 3.5kg.

COP induction protocol was maintained for 8 weeks and then over to LMP maintenance protocol for approximately 4 months. As Socks was doing really well, caregiver stopped the chemotherapy completely at the end of the 4th month into the maintenance protocol.

Discussion

I haven't seen another case of alimentary lymphoma since Socks but, reflecting on how it all went previously, I am happy with the outcome and will definitely encourage owners to consider chemotherapy for alimentary lymphoma in the future. The owner's commitment and patient's co-operation throughout the chemotherapy is important to ensure a good outcome.

I will consider adding L-asparaginase to the traditional COP protocol as it appears to increase survival times.

Having read more about alimentary lymphoma in this module, differentiating the type of alimentary lymphoma is important and can help guide treatment options and prognosis. I find the histopathology report from Agri-food and Veterinary Authority of Singapore can be quite vague occasionally and would definitely consider sending biopsy samples to other laboratories such as IDEXX where immunophenotyping and PCR are available, if required. I guess the limitation will always be the cost involved.

Comment from DE Tutor, Richard Malik:

This oncology report on the cat with alimentary lymphoma was excellent, as was the costing for treating lymphoma in a cat. Therefore, we requested Kitty's approval to share it in the C&T Series *and we thank Kitty for giving us permission to do so.*

I do not have anything to criticize, although it should have been possible to get the diagnosis using a fine needle aspirate – either 'blind' or using ultrasound guidance. (Some people prefer surgery –because some tumours can perforate after starting chemotherapy).

Postscript Oct 2015

Socks continued to do well for approximately 1.5 years after remission in December 2012. Socks had soft stools most of the time but as he was doing well otherwise, owner was reluctant to recheck if the lymphoma returned.

He was diagnosed with kidney failure in October 2014. Supportive care was provided until December 2014 when Socks passed away.

When he was diagnosed with kidney failure, no further test were done to determine if kidney failure was associated with lymphoma. ■

SMALL ANIMAL

FELINE HYPERCALCAEMIA

Bruce Mackay
BVSc, FANZCVS
E. BMackay@vss.net.au
T. (07) 5530 6370

Veterinary Specialist Services, Brisbane
104 Eastlake Street, Carrara QLD

C&T NO. 5520

When investigating a patient with hypercalcaemia, it is prudent to repeat the analysis of serum calcium, ideally with an ionised calcium (better reflection of the biologically active form of calcium), as the investigation of hypercalcaemia ***may*** involve a CBC, repeated biochemistry, chest radiographs, abdominal ultrasound, bone marrow aspiration, cytology or biopsies, parathyroid hormone, vitamin D etc and be expensive and demanding on client and veterinarian. Lipaemia and haemolysis can both cause spurious hypercalcaemia.

Calcium is tightly regulated by 3 hormones. Calcitriol is the active form of vitamin D, and increases the formation of a calcium binding protein in the intestinal epithelium – functioning to increase calcium absorption. PTH is secreted by the parathyroid gland in response to changes in serum calcium. PTH increases calcium mobilisation from the skeleton; however, it also causes phosphaturia – net effect is hypercalcaemia and hypophosphataemia. Calcitonin has a mild blood calcium lowering effect by decreasing osteoclast activity.

There are many causes of hypercalcaemia. In dogs, the most common causes are lymphosarcoma, anal sac carcinoma, hypoadrenocorticism, primary hyperparathyroidism (parathyroid neoplasia), other neoplasia (myeloma, other carcinomas), chronic renal failure, granulomatous disease and vitamin D toxicity.

In cats, the most common cause of hypercalcaemia is probably 'idiopathic', followed by renal failure and malignancy (lymphoma being the most common). Some of the previously diagnosed 'idiopathic' patients may actually be associated with vitamin D toxicity.

A bit of my poetic licence means that 'idiopathic' maybe actually means 'idiot / pathetic' – i.e. in some cases, we did not look hard enough!?

Following is a discussion on the approach to several cats over the last few years. ***Always interesting to note how your approach changes over years!***

'Noodle' – 3-year-old Tonkinese presented with reduced appetite, weight loss and occasional vomiting. Diet: chicken, meat, canned tuna and Iams biscuits. Noodle was hypercalcaemic (tCa 3.95 mmol/L and iCa 1.96 mmol/L), moderate azotaemia (BUN 29.9 mmol/L and Cr 287 mmol/L) and hyposthenuric urine. NSF on physical exam. Normal CBC, normal chest radiographs, renomegaly with medullary rim sign, ultrasound guided biopsies revealed hypercalcaemic nephropathy. Noodle was treated initially with i/v saline and ranitidine and discharged on alendronate 5 mg twice weekly flushed with 5 mL water and prednisolone 5 mg bid. Close monitoring was stressed with the owner and Noodle was discharged to be monitored by the referring veterinarian (1 hour's drive away). Noodle went off my radar until I was asked to present a session on hypercalcaemia at a conference about 1 year later. I rang the owner to enquire about Noodle. 'He is doing fine but I was worried about the possible side effects of the medication and therefore stopped them'. The owner was about to hop on a plane to Hong Kong and I requested we get Noodle in for some blood tests when she got back. All normal! Normal kitty and calcium and BUN and Cr! – Idiopathic? Vitamin D toxicity? Vitamin D assays had not been analysed…

About 5 years ago, I was presented with a family of 4 Persian cats over a few months.

'Jasmin', a 2-year-old Persian speyed female presented with a low grade fever, reduced appetite and weight loss. Biochemistry revealed hypercalcaemia (tCa 3.4 mmol/L, iCa 1.67 mmol/L), mild azotaemia (BUN 13 mmol/L, Cr 276 mmol/L. Jasmin's diet was dry and canned Royal Canin and Ultima canned. Abdominal ultrasound revealed free fluid in the right retroperitoneum, a very 'reactive' retroperitoneum and dilated ureter and an IVP revealed a leaking right ureter. A laparotomy was performed, a nephrectomy (*Enterococcus* cultured from kidney and *Strep* from urine). Jasmin was treated postop with i/v fluids, cephalothin and supportive care before being discharged. The ureteroliths were calcium oxalate.

Several months later, a litter mate – '– presented with a history of being unable to jump onto the lounge and weight loss (5.06 kg to 4.1 kg) over the last 6 months. Tiger Lilly's diet was Science diet biscuits, Royal Canin (Persian, Dental or Indoor cat) and Dick Van Pattern's Natural Balance. Tiger Lilly was hypercalcaemic (tCa 3.94), normophosphataemic, not azotaemic, thrombocytopaenic and had a lymphocytosis (9.2 and repeated at 8 – 'activated lymphs'). Physical examination, chest radiographs and abdominal ultrasound were all normal; a bone marrow performed in light of the lymphocytosis was also normal. Tiger Lilly had a markedly elevated 25 hydroxy vitamin D (> 375 nmol/L with a reference range of 88-168) and a PTH of < 12.0 pg/mL with a reference range of 22-122) i.e. appropriate PTH in a hypercalcaemic patient. Tiger Lilly was treated with alendronate 5 mg twice weekly and prednisolone 5 mg bid with a diet change to meat and commercial canned food. His calcium took approximately 4 weeks to reduce to normal and his vitamin D took about 8 weeks.

Due to the presence of 2 hypercalcaemic kitties in 1 household, we examined the other 2 litter mates as well. 'Anna' was clinically normal and had a normal calcium. 'Toby' was clinically normal; however, he was hypercalcaemic (iCa 1.46 mmol/L) and had an elevated vitamin D level (> 375 nmol/L).

The cats all 'grazed' on cat grass regularly. Cat grass (*Dactylis glomerulata*) allegedly has very high vitamin D content. The cat grass was removed from the cats' enclosure and the 3 cats all did well long term. Follow up vitamin D levels at 2 months were normal.

Recently, we have had 2 Sphynx cats presented with hypercalcaemia. 'Sphynxstar' presented with a poor appetite and occasional vomiting. She was normal on physical examination. Diet consisted of Royal Canin digestive sachets, Nutrigel, Fancy Feast Roast Chicken, Ultimate tuna / chicken and Canidae pure sea dry biscuits, 'greenies' and she had her teeth cleaned daily with a 'veterinary toothpaste'.

She was hypercalcaemic (tCa 3.6 mmol/L) and not azotaemic. Sphynxstar had a lymphocytosis (7.7) with lymphs 'activated'. She had a CBC / biochemistry, chest radiographs and abdominal ultrasound. Sphynxstar was treated with i/v saline and her serum calcium returned to normal over 10 days and she was discharged on a strict diet change to Hills c/d. She remained well with a normal calcium.

'Miss Kizzy', an 18 month Sphynx presented a few weeks later with a poor appetite. Again a physical examination was normal. She was on the same diet as Sphynxstar. Miss Kizzy was hypercalcaemic (tCa > 4 mmol/L, iCa 1.93, normal BUN and Cr and PO4 in the normal range (1.7 mmol/L with reference range of 1-2). Normal chest radiographs and abdominal ultrasound. Miss Kizzy had a PTH of 10.3 (reference range of 1-84) and a markedly elevated vitamin D (671 nmol/L with a reference range of 65-170). Again, Miss Kizzy's calcium returned to normal over 10 days and clinically she did very well with the only change being dietary. The problem was that she was on 'sooo' many different components to her diet. ■

I very quickly realised how fantastic a network we have here in Australia. I emailed a query to Richard Malik and within days had feedback from Richard, David Fraser (Emeritus Professor of Animal Science at Sydney Uni), Sue Foster and Linda Fleeman with suggestions and help with where to get both serum and dietary vitamin D analysis run. Nick Cave's lab at Massey Uni can run dietary vitamin D levels as can the National Measurement Institute in Melbourne. 'You need to run both Vit D2 and D3 as seafood can be high in cholecalciferol (vitamin D3) and supplementation in pet food is often ergocalciferol (vitamin D2 – plant derived and cheaper)' – Sue Foster. Running vitamin D assays (both D2 and D3) can be very expensive when a patient is on several different diets / supplements ~ $500/sample!

Thanks also to Sue Foster / QML for support running the vitamin D assays in the '4 cat household'. The more recent vitamin assays were run at Michigan State and therefore had a several week turnaround.

Investigating hypercalcaemic patients can be complex and expensive! Our feline patients are probably more difficult because of the 'idiopathic' group but remember that many may only be 'idiopathic' because we don't look hard enough! Carefully question the owners on diets, supplements and cat grass.

And then a colleague suggested that maybe the Sphynx kitties get vitamin toxicity because they have no coat and get too much sunlight! – Well – maybe not.

Editor's Note: *Bruce's last comment is meant to be ironic.*

SMALL ANIMAL

COMMENT COURTESY OF

David Fraser
Emeritus Professor of Animal Science
The University of Sydney

The National Measurement Institute (NMI) in Melbourne has developed a very sensitive and accurate assay for measurement of vitamin D (cholecalciferol) and its analogues in food and other biological material. NMI offers a commercial service to assay samples. As expected, these assays are moderately expensive but the results are very reliable. ■

FELINE OROFACIAL PAIN SYNDROME (FOPS)
CALL FOR DNA SAMPLES!

Are you treating a Burmese cat with FOPS? Are you able to help a group of researchers find the genetic cause of the disease, thus helping to prevent and treat this painful and debilitating condition? The diagnosis of this syndrome is relatively straightforward – it is a painful condition centred in the oral cavity, often triggered by mild trauma (e.g. teething) or inflammation (e.g. resorptive lesions), causing **disproportionate pain and often self-mutilation** (see below). Diagnosis is by exclusion of causes of oral pain and can be made by any small animal clinician. If doubts exist as to the diagnosis, please contact Clare or Richard for advice on diagnosis and clinical management, or even referral.

A recent genetic association study on Burmese cats affected by FOPS suggested several promising candidate genes and to continue this study we would like to perform whole genome sequencing.

If you are able to provide either an EDTA blood sample or cheek swab* then we would be very grateful.

Please submit sample to:

Richard Malik **(Australasia)**

Richard Malik, Centre for Veterinary Education,
Level 2, Veterinary Science Conference Centre B22,
The University of Sydney, NSW 2006
richard.malik@sydney.edu.au

Clare Rusbridge **(Europe)**

Clare Rusbridge, Fitzpatrick Referrals, Halfway Lane,
Godalming, Surrey, GU7 2QQ, United Kingdom
D +44(0)1483 423761 F +44(0)1483 527590
ClareR@fitzpatrickreferrals.co.uk

Blood instructions (UK)
http://www.veterinary-neurologist.co.uk/FOPS/

Leslie Lyons **(North America)**

Leslie Lyons, College of Veterinary Medicine,
E109 Vet Med Building, 1600E. Rollins Street,
University of Missouri Columbia, MO, 65211, USA

Submission form
http://felinegenetics.missouri.edu/wp-content/uploads/2013/07/Submission-Form.pdf

Blood instructions (USA)
http://felinegenetics.missouri.edu/dna-sampling-and-shipping/blood-samples-1

***in the UK, due to our Home Office guidelines, blood may only be submitted that is excess to diagnostic testing, for example from a haematology evaluation. If this is not available then a DNA swab can be provided – please contact c.rusbridge@surrey.ac.uk**

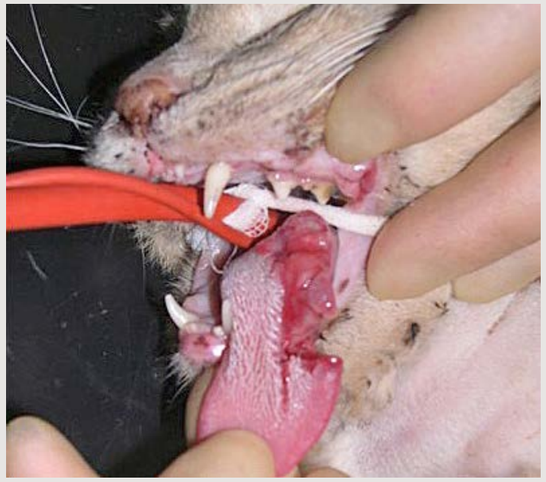
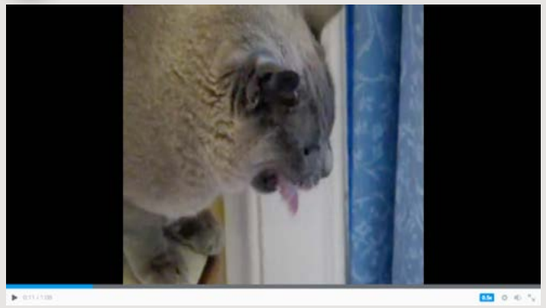


Figure 1. Tongue Mutilation in a Burmese cat with FOPS. Photograph courtesy of Jamie Finney MVB MRCVS, Abbeycroft Veterinary Centre

ABOUT THE DISEASE

Download all the necessary forms from the eBook or visit: www.cve.edu.au/candt2015



Feline orofacial pain syndrome (FOPS) is characterized by behavioural signs of severe oral discomfort. This condition is seen in a variety of feline populations, although Australian and European lines of Burmese cats predominate, suggesting a genetic basis for this **neuropathic pain disorder**. Dental pain, for example permanent teeth eruption and periodontal disease, can trigger the condition. Environmental factors can exacerbate the condition and individuals with poor social coping strategies in multi-cat households appear to be more vulnerable. Affected cats are presented most commonly with pawing and mutilation of the mouth especially the tongue. In many patients discomfort is elicited by movements of the mouth such as eating, drinking or grooming. The apparent pain is typically unilateral and can be episodic with variable pain-free intervals. (see https://vetvideo.surrey.ac.uk/media/video-figure-6).The syndrome is often recurrent, and with time may become unremitting, with up to 10% of the cases being euthanized as a consequence of the condition.

GENERAL

AG GAG LEGISLATION INTRODUCED IN NSW

Desmond Sibraa

Solicitor, Author of Food Legislation NSW

E. dsibraa@optusnet.com.au

C&T NO. 5521

To use the words of Sir Humphrey Appleby, of 'Yes Minister' fame, the NSW government has made a very courageous decision to introduce such draconian legislation to protect those animal handlers from the public scrutiny of their cruel activities that have been disclosed by animal activists over many years. Such groups include: Animals Australia, Voiceless, Animal Liberation and other whistle-blowers who provide information to these groups. Animal advocates have become increasingly effective in gathering and releasing undercover footage captured in agricultural facilities. Much of this undercover footage exposes extreme examples of animal cruelty, neglect and violations of animal protection laws. The ABC recently broadcast greyhound live baiting that is now prohibited by the legislation. There has been much public support for the publication of these reports and the Government may face adverse publicity at the next election.

The 2015 Biosecurity Bill passed in its entirety, after amendments put forward by the ALP, the Greens, and the Animal Justice Party were rejected.

Niall Blair, the Minister for Primary Industries, said that the bill would 'provide strict new penalties for anyone who intentionally or recklessly breaches their biosecurity obligation'.

'Our farmers are suffering as a result of unlawful farm trespass – financially, emotionally and physically,' Mr Blair said.

'Aside from the intolerable biosecurity risk farm trespass creates, it is also an unjust invasion of the privacy of farmers,' he said.

The legislation prohibits animal activists from trespassing to expose animal cruelty and imposes penalties of $1,000,000 and / or 3 years imprisonment for an individual and for organisations penalties of $2,000,000. Officials have the power without a warrant to use force to enter premises based on a 'suspicion' of wrongdoing.

In 2013, the NSW Minister for Primary Industries, Katrina Hodgkinson observed that 'it seems every week now where you've got animal activists breaking into intensive farms… these people are vandals. These people are akin to terrorists.'

The real purpose of the legislation is to provide anyone engaged in cruelty to escape detection and public awareness of their cruel treatment of animals. The exposure of these monsters is extremely bad for their businesses. If there was no cruelty bring carried out, there would be no need for any ag-gag legislation. An alternative would be to follow Animal Liberation's proposal to install cameras in all places where animals are handled and increase penalties for breaches. There has also been a failure of government authorities to enforce their powers to prevent cruelty by proper inspections and cancellation of licences in appropriate cases. Activists can find these cases and record the cruelty whereas government authorities seem to be unable to use their powers to detect cruelty to animals. The NSW Food Authority officers have power to enter farms where food production animals are raised and produce food. They would not be trespassing. Evidence of animal cruelty could then be referred to the RSPCA.

The cruelty exposed by Animal Liberation at the Hawksbury Valley Wilberforce abattoir caused the Food Authority to make abattoir managers employ animal welfare officers.

Shot undercover over 6 days at the abattoir, the footage shows pigs being dragged onto the sticking table and being belted with what looks like an iron bar. The pigs should be rendered unconscious by a stunner before their throats are cut, but the footage shows that it has not been done properly in some incidences. On one occasion a pig's head was pummelled 7 times. A minute later the same worker beat another pig over the head 13 times. Animal Liberation's Emma Hurst says the footage shows 'grotesque cruelty'.

There has been an increase in the discovery of cruel treatment of animals in all premises licensed by the NSW Food Authority and little action is evident by the Authority that has the power to licence these premises. Despite the appointment of Animal Welfare Officers by the abattoir management, the cruelty seems to be increasing according to reports from Animal Liberation activists. While the Authority inspectors have numerous powers of investigation, to enter and inspect premises and search records, the activists have only needed to install video cameras to demonstrate the extent of the cruelty. This raises the question of how many offences remain to be discovered and why is the discovery only discovered by activists from Animal Liberation?

The thugs committing these offences are personally liable and the employers are also vicariously liable, see section 123 of the Food Act. A defence is available if the employer can establish that the employer had no knowledge of the contravention and could not have prevented the offence by the exercise of due diligence. Where an employer is situated on the premises it would seem impossible to claim due diligence.

ABC's *Lateline* broadcast images of turkey cruelty on 21 March 2013. See the horrific video at: www.abc.net.au/news/2013-03-20/video-shows-poultry-cruelty/4585382

The footage was secretly filmed over 2 weeks in an area of the Tahmoor abattoir where workers take the birds from cages and place them into shackles to be stunned and slaughtered.

'I think this is definitely some of the worst that we've [ever] seen,' Animal Liberation's Emma Hurst said.

Workers can been seen kicking the birds, sometimes up to 9 times.

Other birds are punched, bashed against walls or the cages and stomped on until they are still. Sometimes co-workers kick or hit the same bird as it moves along the production line. It appears to be common behaviour. Sometimes the workers appear to celebrate while those who see what is happening fail to intervene.

One worker even appears to try and separate a bird's head from its body by stretching and slotting it in the edge of a cage while it is attached to the moving production line.

In the High Court case Lenah Game Meats Pty Ltd (Lenah) operated a possum meat processing plant in Tasmania. Unknown individuals unlawfully entered Lenah's premises and installed hidden cameras without Lenah's knowledge or consent.

The hidden cameras were used to capture footage depicting the possum slaughter process. The footage was being supplied to Animal Liberation Ltd, who later passed the footage on to the ABC.

On becoming aware of the ABC'S plans to broadcast the footage on the current affairs program, the *7:30 Report*, Lenah sought an interlocutory injunction to restrain the broadcast. While the ABC was not involved in the trespass or the installation of the surveillance equipment, it was aware that the footage had been obtained unlawfully, at least after ABC's application to the court.

In his judgement, Justice Michael Kirby, defended the use of surveillance by animal activists on public interest grounds:

'Parliamentary democracies, such as Australia, operate effectively when they are stimulated by debate promoted by community groups. To be successful, such debate often requires media attention. Improvements in the condition of circus animals, in the transport of live sheep for export and in the condition of battery hens followed such community debate.'

The majority of the High Court found in favour of ABC by rejecting Lenah's argument and held that the interlocutory relief was unavailable to Lenah.

Even moderate lobby groups like Voiceless believe in the value of illegally obtained video. 'It's an important tool in raising consumer awareness about where their food comes from,' says Voiceless' legal counsel, Emmanuel Giuffre.

In a taste of what's to come, undercover vision and social media have had a dramatic impact on farming.

In 2011 secret footage of an Indonesian abattoir broadcast on ABC TV's *Four Corners* led to the suspension of live cattle exports to Indonesia.

Over the past 5 years, a concerted social media campaign against the use of sow stalls in piggeries led to Coles announcing it would only sell 'sow stall free' pork.

Officials have also raided a number of piggeries after the exposure of serious abuse of animals.

This month it was the Australian wool industry on the defensive after secretly filmed footage from shearing sheds emerged, allegedly depicting sheep being punched and hit with electric shears.

However, there are warnings that legislative action could backfire on farmers. The world's best known authority on humane handling of livestock, Dr Temple Grandin, says hardline 'ag-gag' laws in United States have done more harm than good.

'I think it's the dumbest thing that farmers ever did. What you should be doing when you're getting bashed is opening doors not closing them,' she says.

'Basic things like how you raise cattle and pigs and how you slaughter them, that's not private information.'

Dr Grandin says if intensive livestock industries have nothing to hide, they should consider live webcam streaming to the public, citing US examples.

Giuffre says Australian animal rights activists have become increasingly effective in gathering and releasing undercover footage exposing animal cruelty and highlighting the truth behind factory farming. 'We've seen footage from over a dozen Australian piggeries of pigs being beaten, of endless rows of sows imprisoned in metal and concrete stalls, unable to take a step forward or back; unable even to turn around.'

'We've seen footage of thousands of ducks crammed into sheds, wading in their own filth, their legs unable to support their weight because they've been deprived of much-needed water for their entire lives.'

There has been footage, Giuffre says, of countless live export atrocities, 'footage of Australian cattle and sheep being brutally abused, being slaughtered on the streets in backyard butcheries and unauthorised slaughterhouses overseas'.

Wally's Piggery, near Yass, New South Wales, was forced to close after footage obtained by Animal Liberation NSW in 2012 caused a public outcry. It showed pigs being bludgeoned and kept in filthy, cramped conditions.

The organisation **Animals Australia** tells some horrific stories on its website, including the one about Nature's Child, a prize-winning thoroughbred mare, who was discarded by the Australian racing industry. Her life ended in horrendous cruelty at a Victorian knackery. 'She was shot in front of her equine companion, then dragged by a tractor – while still alive – to a killing floor, where a worker cut off her tail and slit her throat. Other horses were beaten with pipes, transported while sick and injured, or left dead in the holding yard.'

The evidence was collected by the **Coalition for the Protection of Racehorses.**

Investigations by Animals Australia in the Middle East and South East Asia exposed cruelty in the live export trade and resulted in the first ever suspension of live animal exports – to Egypt in 2006, then Indonesia in 2011 – and sweeping reforms to the operation of the entire industry.

The organisation's investigation into the factory farming of pigs in Australia was a catalyst to the pig industry agreeing to restrict the use of stalls for pregnant sows by 2017.

Giuffre says there is a growing movement of ethical consumers who care where their food comes from and want to see farm animals treated with respect.

'This is not just an animal protection issue; it's an environmental protection issue and a consumer protection issue; it's civil liberties generally and I think that any attempts by governments to try to suppress free speech or suppress dissent – and that's exactly what it is – would be opposed by a large contingent of our politicians, but of course they need to be made aware of the issue.'

American journalist Will Potter, who wrote the book **Green is the New Red** and was recently on a speaking tour of Australia, says ag-gag is coming to Australia because Australian animal advocates have been incredibly effective.

Australians have an opportunity that activists lacked in the US, Potter says; they are better informed and can identify and stop ag-gag proposals before they become law.

There is a long history of open rescues and undercover investigations in Australia, Potter says, and activists such as Patty Mark and Animal Liberation Victoria are known internationally for their pioneering work. Also, national media exposés such as the *Four Corners* programme about live exports, 'A Bloody Business', had provoked public outrage.

Finally, a quote from Sir Paul McCartney — ***'If slaughterhouses had glass walls, everyone would be a vegetarian.'*** ■

GENERAL

PRACTICE TIP: SEPARATE DIAGNOSTIC OCULAR MEDICATIONS FROM OCULAR TREATMENT MEDICATIONS

Aine Seavers

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

C&T NO. 5522

A talk by Kate Hindley from the Small Animal Specialist Hospital at the October 2014 Shoalhaven AVA meeting (latter always so well run by the Berry boys with great food and atmosphere) was a good source of practical tips for safer and better diagnostic clinical examinations. Kate's emphasis on the need to separate our diagnostic ocular medications from our ocular treatment meds was a timely good clinical practice reminder.

Consideration should be given to stock individual single-use disposable vials for diagnostic tests where possible. There are about 14 drops approx. in the usual Minims vial but studies have shown serious risk of cross infection so each vial should be strictly single-patient-use only and disposed of promptly.

The multi-dose bottles are often better kept for those medications that can be dispensed for home treatment of an ocular condition.

For local anaesthesia during examination, rather than using multi-dose Alcaine® bottles Proxymetacaine hydrochloride (24hr time frame once opened…), consider Minim's Proxymetacaine hydrochloride 0.5% w/v, Eye Drops, solution single use vials.

Tropicamide 0.5% or 1% single-use vials are preferable for in-clinic use due to rapid onset (20-30mins) and shorter duration of action (4-12hrs) and hence of benefit in a diagnostic setting. Cause less marked salivation in cats than with atropine. Not as effective as atropine for relieving ciliary body muscle spasm associated with uveitis. 1 drop per eye, repeat after 20-30mins if needed.

Atropine Minims 1% single use vials for diagnostic in-house use. The dog can go home on a multi-dose bottle format but alert the client that such a multi-dose container has a 30 day maximum use once opened.

Single-use Minims fluorescein vials in 1% or 2% are especially good if you find clients get queasy when you use the dry paper fluoret strips on the pets' eyes in the clinic. *Caution:* Some Fluorescein Minims do contain lignocaine.

Serum/EDTA can be used where there is a concern of bacterial inflammation/melting cornea. Serum is better as a multifactorial natural polypharmacy but one can use the EDTA found in the bigger blood tubes. Add 5mLs of normal saline and use 4-5 time per day for melting (not indolent) ulcers. ■

MEDICAL IMAGING CONFERENCE + MASTERCLASS

THE INTERPLAY OF DIAGNOSTIC IMAGING & INTERNAL MEDICINE

Join us for four days of diagnostic delights, where imaging specialists will combine with internists to provide the best ways to maximise the information provided by different imaging modalities in the light of signalment, history, physical examination and laboratory findings.

The fifth day will be dedicated to a masterclass on the brain, where neuroimaging and clinical neurology will combine to expand your understanding of the pathophysiology of CNS disease. Although the focus will be on imaging brain disease and dysfunction, there will also be clinical vignettes addressing diagnosis and therapy.

Early Bird rates apply, enrol before 11 January, 2016.
ENROL NOW >> www.cve.edu.au/conference/imaging

SPEAKERS

- **Sarah Davies** (Convenor) BVSc, MS, DACVR, Assoc MACVSc
- **Cathy Beck** BVSc (Hons), DipVetClinStud, MVS, FACVSc (Radiology)
- **Karina Graham** BVSc (Hons), FANZCVS (Small Animal Medicine)
- **Christine Griebsch** DrMedVet, DECVIM (Small Animal)
- **Belinda Hopper** BSc, BVMS (Hons), MVS, FANZCVS (Radiology)
- **Fleur James** BSc, BVMS (Hons), FACVSc
- **Sam Long** BVSc, PhD, Diplomate ECVN
- **Mariano Makara** DrMedVet, Diplomate ECVDI
- **Darren Merret** BVSc, MVS, Cert SAC, FACVSc
- **Marjorie Milne** BVSc, FACVS
- **Robert Nicoll** BSc (Vet), BVSc, DACVR

CONFERENCE

Monday 22 –Thursday 25 February, 2016
9.00 – 5.00

MASTERCLASS

Friday 26 February, 2016
9.00 – 5.00

VENUE

Veterinary Science Conference Centre
B22 Regimental Drive
The University of Sydney

PERSPECTIVE NO. 120

SWEET SUCCESS: TIPS FOR MANAGING DIABETES IN DOGS & CATS

Karen Brenner
BVSc, Dip.ACVIM
Centre for Animal Referral and Emergency
5 Hood Street, Collingwood VIC 3066
T. (03) 9417 6417
E. admin@carevetspecialists.com.au

Linda Fleeman
BVSc, PhD, MANZCVS
Animal Diabetes Australia
at Boronia Veterinary Clinic, Rowville
Veterinary Clinic & Lort Smith Animal Hospital
E. L.Fleeman@AnimalDiabetesAustralia.com.au
W. www.AnimalDiabetesAustralia.com.au

Do dogs and cats with DKA need insulin?

We are aware of anecdotal reports suggesting that insulin should be withheld from patients with diabetic ketoacidosis (DKA) until the animal is rehydrated; and in some practices, this seems to be a reasonably common approach to treatment. It must be recognized, however, that almost all of the metabolic derangements associated with DKA are attributable to insulin deficiency (either relative or absolute) and fluid therapy alone is therefore inappropriate. In fact, before insulin was commercially available, DKA was an almost uniformly fatal condition.

While it is true that fluid therapy will cause blood glucose concentrations to decrease, this will not switch off ketogenesis. Fluid therapy will promote urinary loss of glucose and ketones and is an important means to address fluid deficiencies, improve renal blood flow, and correct electrolyte derangements; however, fluid therapy alone will not suppress ketogenesis, the catalyst for DKA. It is of course acceptable to commence fluid therapy before insulin therapy, especially when the clinician is too busy at the time of admission to set up the treatment protocol or is waiting for laboratory results. Nevertheless, deferring insulin therapy should not be the standard course of treatment – insulin therapy is indicated as soon as practical. The only exception is when there is severe hyperosmolality, but this presentation is very uncommon, requires careful intensive treatment, and is associated with a poor prognosis.

Published protocols describing the management of DKA in dogs and cats have been available for decades. These protocols include the administration of low doses of insulin either as a constant rate infusion (CRI) or repeated intramuscular injections (Figure 1). The goal of insulin therapy is to promote the transportation of glucose into cells and consequently, to switch off ketogenesis while addressing fluid and electrolyte derangements. The animal's blood glucose concentration needs to be monitored closely and the insulin titrated so that the blood glucose gradually decreases into the range of 10-15 mmol/L (Figure 2). The animal's blood glucose is then maintained in this range by the administration of both insulin plus intravenous (IV) glucose until the dog or cat begins to eat. Resolution of metabolic derangements and return of appetite can be achieved in most patients over a period of 24-48 hours. Prompt insulin administration is necessary for a timely recovery. Withholding insulin in veterinary patients with DKA is likely to increase the length of hospitalization and subsequently, the cost to the owner.

For those not familiar with insulin CRI protocols, there is sometimes concern about the adsorption of insulin to the plastic IV line. Although it is true that some insulin will adsorb to the lining of the infusion bag and giving set, this soon reaches a steady state and all remaining insulin is delivered to the animal. It is not necessary to prime the line. It is also not necessary to run the insulin CRI through a separate IV catheter. In fact, it is prudent to run concurrent insulin and glucose infusions through the same catheter to ensure that both infusions cease at the same time if the catheter fails.

If you are interested in incorporating an insulin CRI into your practice's DKA management protocol, you may find the following resources helpful:

- Feldman, Nelson, Reusch and Scott-Moncrieff. Canine and Feline Endocrinology, 4th Edition. *Saunders* 2014
- https://www.cve.edu.au/files/concurrent-pancreatitis-and-diabetes.pdf
- https://www.cve.edu.au/files/flow-chart.pdf
- https://www.cve.edu.au/files/feline-diabetic-ketoacidosis.pdf

How reliable is your diagnostic laboratory's IGF-1 assay for diagnosis of cats with suspected acromegaly?

Endocrine assays are much less reliable than most of the routine biochemical assays with which we are familiar. This issue is especially important in Australia where there are no dedicated veterinary endocrine diagnostic laboratories.

It has recently been suggested that acromegaly might be the underlying cause of diabetes in many more cats than previously recognised and some authors have even recommended routine screening of all diabetic cats. Diagnosis of acromegaly is made difficult without a reliable feline IGF-1 assay in Australia and so screening is not feasible at this time. Furthermore, there are currently no practical and reliable treatment options for this condition in Australia.

Acromegaly describes the syndrome that arises as a consequence of excessive growth hormone (GH) and thus insulin-like growth factor-1 (IGF-1) production. Acromegaly was once thought to be an uncommon disorder but it is now being increasingly recognised in cats; in fact, some prevalence studies from the UK suggest that acromegaly may be present in as many as one third of feline diabetic patients (Niessen S, Petrie G, Caudiano F, *et al.* Feline acromegaly: an undiagnosed endocrinopathy? *J Vet Int Med* 2007; **21**: 899-905), with male cats over-represented.

Elevated GH concentration causes catabolic derangements via insulin antagonism leading to hyperglycaemia. The slower onset, anabolic effects (which create the traditional physical features of acromegaly) are due to elevated IGFs. Therefore, the development of the acromegalic phenotype is often insidious and may not be clinically apparent at the time that biochemical testing is suggestive of acromegaly. In fact, one study in which diabetic cats were assessed for the presence of acromegaly demonstrated that in only 24% of cases did the clinician consider acromegaly based on the cat's phenotypic appearance alone. The clinical signs associated with acromegaly can include polyuria/polydipsia, polyphagia, weight gain (despite poor glycaemic control), organomegaly, hypertrophic cardiomyopathy, broad facial features, prognathia inferior, and inspiratory stridor. Cats with acromegaly may also have clinical signs reflecting the space occupying effects of a pituitary macroadenoma.

There are no pathognomonic clinicopathologic abnormalities

Figure 1. Low doses of rapid acting, crystalline insulin are recommended for treatment of dogs and cats with DKA.

Figure 2. The blood glucose needs to be monitored while the animal is receiving insulin and fluid therapy.

on a routine screening blood test. The IGF-1 assay is currently advocated; however, not all IGF-1 assays have been created equal. It is very important to ensure that the assay employed by your reference laboratory is one that has been validated for use in cats. In the past, the Royal Prince Alfred hospital in Sydney provided a reliable assay for feline IGF-1. Unfortunately however, this laboratory changed to the Immulite IGF-1 assay more than 3 years ago. In a personal e-mail communication to Linda Fleeman on 21-12-12 on the reliability of the Immulite IGF-1 assay, Dr Stijn Neissen from the Royal Vet College at the University of London gave the following response: 'We have flirted with Immulite ourselves but found it terribly unreliable during thorough testing using well phenotyped acro and non-acro cats. I would be surprised if anyone else got it to work.' It is important to note that many diagnostic clinical laboratories in Australia continue to send samples for IGF-1 assessment to the Royal Prince Alfred hospital in Sydney more than 3 years since the assay was changed. Therefore, it is recommended that prior to sample submission, clinicians should confirm where their diagnostic clinical laboratory will send the sample for IGF-1 assay. It is important to ensure that an assay validated for feline IGF-1 is always used and preferable that the laboratory performing the assay participates in at least 1 of the 2 external quality assurance schemes for veterinary endocrine assays. This currently requires that Australian samples are sent overseas for IGF-1 assay. To optimise the chance of detecting an increased IGF-1 concentration, diabetic cats must first be treated with insulin for at least 4-6 weeks. This is because portal insulin induces IGF-1 production and acromegalic cats can produce normal IGF-1 results when they are insulin deficient.

Research into medical inhibition of pituitary GH hypersecretion is still ongoing but recent work has shown some efficacy using pasireotide; unfortunately though, this medication is cost prohibitive in almost all cases. Hypophysectomy is currently carried out quite successfully at a limited number of facilities world-wide; however, success appears to be directly proportional to the experience and expertise of the surgeon with the procedure and of the intensive care team with the perioperative care including hormone supplementation. Finally, radiation therapy can also be considered. This latter modality can reduce tumour size and tumour hormone production but the overall effectiveness, onset of altered glycaemic control, and duration of reduced GH production can be difficult to predict. ■

Editor's Note: *Readers are directed to read this outstanding article written by Linda Fleeman and colleagues Ann Thompson and Patty Lathan and published in April 2015: Update on insulin treatment for dogs and cats: insulin dosing pens and more.*

Available through the eBook version of this issue, or through OPEN ACCESS.
See: http://dx.doi.org/10.2147/VMRR.S39984

WATCH OUT FOR LINDA'S 2016 TIMEONLINE

Masterclass in diabetes management in dogs and cats

Dates: 18 July – 15 August, 2016

This course will provide practical guidelines that will enable you to achieve successful outcomes for your diabetic patients. Learn about protocols that are easy to follow, new improved treatment and monitoring tools such as insulin dosing pens and continuous subcutaneous glucose monitors, and tips on how to take the stress out of the management of diabetic dogs and cats with concurrent illnesses.

Visit www.cve.edu.au/timeonline for the full TimeOnline Calendar for 2016

Most of us veterinarians graduated loaded with knowledge but short on experience and self-confidence based on that. Would it not be nice to have access to experience on your day of graduation? The private non-profit VetCoach project collects veterinary professional career learnings from veterinarians around the world and shares them with veterinary students and young colleagues with the objective to inspire and motivate these young professionals. Over 15,000 books have been produced for distribution world-wide and the 8th VetCoach edition for Australasia is in print and will be available early 2016 in Australia and New Zealand. In this new book the career reflections of 107 authors will feature of which 34 are from Australia and 10 from New Zealand. The rest presents a nice mix from countries like USA (28), UK (7), South Africa (3), several European countries as well as from some individual colleagues in India, Iran, Kenya and Sudan. There are 63 male and 44 female authors.

VetCoach AU includes supportive welcome messages from the presidents of the WVA, WSAVA, FECAVA, AVA and NZVA and is executed in partnership with ProVet Australasia (a Henry Schein Company) and Merial Australia. Books can be ordered from March 2016 onwards from ProVet and Merial and via the VetCoach website www.vetcoach.info. The most recent information about the project and the status of this new VetCoach AU edition can be found in Facebook under > vetcoachproject <. Dr. R.C. Nap, DVM, PhD, Dipl ECVS & Dipl ECVCN, Uppertunity Consultants. ■

PERSPECTIVE NO. 121

MANAGEMENT OF FELINE HYPERTHYROIDISM

Andrea Harvey

BVSc, DSAM (Feline), Diplomate ECVIM-CA, ANZCVS (Associate), MRCVS

Registered Veterinary Specialist, NSW

PhD Scholar,
Centre for Compassionate Conservation
School of Life Sciences
The University of Technology Sydney

E. andreaharvey.cat@gmail.com

Hyperthyroidism is well recognised to be the most common feline endocrinopathy encountered in clinical practice. Whilst most practitioners have vast experience in treating these cases, recently new treatments and new insights into treatment have become available. Furthermore, although the majority of cases may be straightforward to diagnose and treat, there are also many cases that prove to be much more challenging. It is important that the clinician recognises the potential difficulties in management, and the complications that may arise, to enable owners to be well informed and involved in the decision making as to the most appropriate treatment option for their cat.

Management options: curative treatments vs daily control of thyroxine synthesis

There are broadly 4 management options available: surgical thyroidectomy, anti-thyroid medications, radioiodine treatment and dietary management. In order to emphasise an important consideration with management, these options will be discussed in 2 different groups: curative treatments for hyperthyroidism and daily control of thyroxine synthesis. The reason for doing so is that this important difference is often overlooked when clinicians are choosing how best to manage hyperthyroid cats. Certainly daily control options can be very useful in some circumstances, such as cats with significant co-morbidities that might not be expected to live for very long, or as a way of stabilising the thyrotoxic state in the short-term prior to undergoing a curative treatment. However, these daily control options are not considered appropriate to use in the long term, when curative treatment options exist. It has long been the feeling of most eminent endocrinologists, that curative treatments are far superior since controlling thyroxine synthesis does nothing to halt the underlying disease process which will continue to progress. This means that controlling thyroxine synthesis can become more challenging over time, the volume of hyperfunctional thyroid tissue increases over time, and this may also predispose to neoplastic transformation. A recent study confirmed these fears, that hyperthyroid cats on long-term medical treatment show a progressive increase in the prevalence of large thyroid tumours, intrathoracic thyroid masses, and suspected thyroid carcinoma.[1] In Peterson's studies, less than 2% of recently diagnosed hyperthyroid cats had thyroid carcinoma, whereas up to 20% of cats treated medically for longer than 4 years had evidence of thyroid carcinoma on thyroid imaging. Furthermore, it can be difficult to titrate medical management effectively, and measuring total T4 is not likely to be an accurate indicator of the degree of control, particularly when total T4 may frequently be falsely suppressed by concurrent non-thyroidal illnesses. This, together with often a lack of close monitoring, means that many cats on long-term daily control may still have ongoing effects of thyrotoxicosis, which may be particularly relevant in ongoing cardiac effects.

There are other potential concerns of the individual daily control options, which are highlighted further in the individual sections below.

Cost of different treatment options, and clients' financial limitations are commonly quoted as a reason not to pursue a curative treatment in a given individual. However, although there is some variation in costs of different medical treatment options, diets, surgery and radioiodine treatment at different centres, broadly speaking, when the costs are broken down,

Figure 1. A cat with classic signs of thyrotoxicosis - weight loss despite a good appetite, and tachycardia. Thyrotoxicosis may not be adequately controlled with treatments that act as a daily control of thyroxine synthesis.

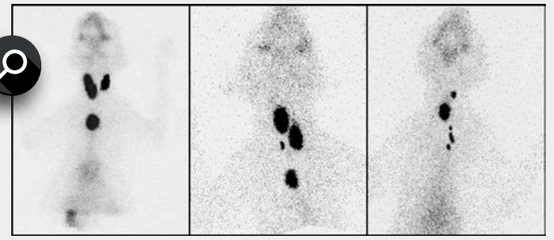


Figure 2. Scintigraphic images of hyperthyroid cats. More than 2 areas of hyper functional thyroid tissue, and areas quite far down in the chest are not uncommon. Radioiodine treatment is a curative treatment regardless of location or volume of hyper functional thyroid tissue.

the average cost of either surgery or radioiodine treatment is approximately equivalent to that of 1 year of treatment using daily control options. Thus, this is not really a valid reason not to pursue curative treatment in most circumstances unless it is clearly predicted that the cat will not live beyond a year or so post-diagnosis. Despite this and the concerns with daily control of thyroxine synthesis as a long-term treatment, anti-thyroid medication was the preferred method of management in a survey of UK practitioners, with cost being the biggest reason for this choice.[2]

Availability of the curative treatment options is another reason commonly used to justify use of long-term daily control management options. This will clearly still be an issue in some circumstances; however, thyroidectomy is not a technically difficult surgery, and new radioiodine treatment centres are setting up all the time. Veterinarians should therefore make every effort to keep re-evaluating availability of these preferred treatment options in their areas. Even flying a cat interstate for a week in hospital to receive radioiodine is preferable in many situations compared to the cat being managed for several years with a daily control treatment.

Having said this, daily control options certainly do have a place in managing feline hyperthyroidism. Firstly, particularly when thyroidectomy is planned, stabilisation of the thyrotoxic state prior to surgery will significantly reduce anaesthetic risks. Secondly, hyperthyroidism is a disease of older cats, and therefore there are many patients that may have co-morbidities that may influence predicted survival, and therefore treatment choice, and/or could be a contraindication for one of the curative treatments. A recent study evaluating routine health screening in apparently healthy older cats highlighted the high prevalence of occult disease in this population of cats.[3] A further study evaluating the presence of comorbid disease in cats referred for radioiodine treatment showed a large proportion with comorbid disease that led to a change in the advised treatment for their hyperthyroidism in almost 20% of the cases.[4] Contraindications for curative treatment may be related to anesthetic risk for surgery, or the hospitalisation time following radioiodine treatment. The latter, however, needs to be interpreted within the individual circumstances, as there is significant variation between countries in the required hospitalisation time. For example in the UK, radiation safety requirements used to dictate that 5 weeks hospitalisation was required post-iodine treatment. This meant that in some cats with co-morbidities or requiring other medications, this was not a suitable treatment option. These hospitalisation times have since reduced in the UK, but in other countries such as the US and Australia, only 5-7 days hospitalisation is usually required, and therefore presence of comorbities or need for other medications are less of an issue and do not necessarily preclude radioiodine as the best treatment option.

The presence of concurrent azotaemia in hyperthyroid cats, prior to treatment, is a situation where daily control options may be preferable since survival times in these cats are known to be poor.[5] This is a situation where dietary management with an iodine deficient diet may be useful (see below). The importance of differentiating between cats that are azotaemic prior to treatment of their hyperthyroidism, and cats that become azotaemic after treatment for their hyperthyroidism, needs to be emphasised, as the latter group usually have good long-term survival, and in this group of cats development of azotaemia is not related to outcome except in cats that become hypothyroid following treatment. New data has shown that restoration of euthyroidism in medically treated hyperthyroid cats with iatrogenic hypothyroidism causes a reduction in plasma creatinine concentrations, and thus might improve renal function.[6]

This summary highlights that a lot of individual factors should be considered in choosing the appropriate treatment option for each hyperthyroid cat, and that in the majority of cats a curative treatment is usually preferable, but specific situations exist where daily control options are advantageous.

Treatment options for the daily control of thyroxine synthesis

1. Anti-thyroid medications

These medications work by blocking the synthesis of thyroid hormone within the thyroid gland. There are now different formulations available of methimazole, and carbimazole (which is metabolised to methimazole following absorption). Historically, only the human formulation of carbimazole (Neomercazole) was available, and in Australia this has still been the case until recently. Titration of dose can be difficult, and the medication should be given every 8 hours to give good control. There is now a slow release formulation of carbimazole available as a veterinary product, Vidalta™ (MSD Animal Health). This allows once daily dosing to be effective, which offers a huge advantage to those cases requiring medical management. It is important that tablets are not crushed as this destroys the slow release formulation. Good control can be achieved in the majority of cases, although when Vidalta™ was introduced to the UK, the author had experience with several cases that had been

previously well controlled on oral methimazole and were not able to be controlled on Vidalta™. The reason for this is unclear.

A veterinary formulation of methimazole (Felimazole™; Dechra Veterinary Products) has also been available for some years in the UK and recently introduced to Australia. This is a twice daily medication, although in some cats once stabilised, it is possible for them to be maintained on once daily medication. There was some concern in the early days of its use in the UK that a higher prevalence of side effects was being encountered compared to using carbimazole. There is no rationale for this since carbimazole is metabolised to methimazole, and no studies have supported this.

Transdermal methimazole formulations are also widely used with reported good efficacy. They are likely to be less efficacious than oral formulations, but have been shown to be associated with less gastro-intestinal side effects. Therefore, these formulations can be a useful alternative where tablet administration is not possible or where gastrointestinal side effects with oral anti-thyroid medications occur.

The most common side effects with anti-thyroid medications are anorexia, vomiting, and diarrhoea. Often these are mild and self-limiting, but they can be severe and persistent, necessitating ceasing treatment in some cases. Facial pruritus and excoriation is a well recognised side effect, which can be severe and necessitates stopping treatment in all cases. Other potential side effects may not be clinically apparent in the immediate term and therefore regular monitoring of haematology and biochemistry are critical in these patients. Various haematological disorders including leucopenias, thrombocytopenias, haemolytic anaemia and coagulopathies, and hepatotoxicity have all been encountered. Less commonly, lymphadenopathy and myasthenia gravis have also been reported.

2. Dietary management

Recently, an iodine-restricted diet (Hill's y/d®) has become manufactured and is now available in Australia. The theory behind this diet is that by depleting iodine, the ability to synthesise thyroid hormones is reduced. In a study evaluating efficacy of this diet, there was a significant reduction in clinical signs and total T4 within 4 weeks of feeding this as the sole diet.[7] The diet was evaluated in both indoor and outdoor cats with no difference in efficacy found. Another finding in hyperthyroid cats that were fed this diet was a reduction in serum creatinine concentration. This has also been identified in clinical cases when azotaemic hyperthyroid cats have been managed with the diet, suggesting that it may be especially useful as a management modality for hyperthyroid cats with pre-existing concurrent azotaemia. The reason for this reduction is creatinine is not clear, but it is also a protein and phosphate restricted diet.

Figure 3. The new state of the art radioiodine treatment facility at Small Animal Specialist Hospital (SASH), Sydney.

Figure 4. The new design of radioiodine cages are much more aesthetically pleasing for owners, and more comfortable for cats with multiple resting shelves and a large window that they can look out of.

The diet is not palatable to every cat, however, and efficacy can be variable. Monitoring needs to ensure that adequate quantities are being ingested as otherwise weight loss and muscle wasting can easily occur as the diet is relatively low in protein. It may therefore not be ideal for initial treatment for cats with advanced thyrotoxicosis and very poor body condition. The diet is relatively high carbohydrate and low protein and so is also not considered ideal for cats with concurrent diabetes or in diabetic remission.

Concerns have been raised about the safety of feeding an iodine restricted diet in the longer term. There is some controversy as to whether the diet is actually iodine deficient, rather than simply iodine restricted, and whilst it may control thyroid hormone synthesis, it may contain insufficient iodine for other essential extra-thyroidal functions. One of these concerns is in relation to the role of iodine in the immune system and its anti-inflammatory and anti-oxidative actions. In people, iodine deficiency is widely associated with increased prevalence of infectious diseases, and iodine supplementation was historically used as a therapy for many infectious diseases. Extrapolating from this information, there are some concerns that cats fed an iodine deficient diet for prolonged periods may have compromised immune function. The issues surrounding long term safety of the diet are particularly controversial since demonstration of safety would be a requirement if this were a veterinary licensed drug, but because this isn't classified as a drug, different regulations apply.

In summary, caution should be exercised in recommending this as a long term management strategy for otherwise healthy hyperthyroid cats. However, as a short term therapy to stabilise cats prior to curative treatment, or as a treatment for hyperthyroid cats that have concurrent azotaemia before treatment, or another comorbid disease meaning that only short term survival is likely, then this could be a very useful alternative treatment option, particularly for cats that may suffer side effects with anti-thyroid medications, or where the owner is unable to administer medications.

Curative treatment for hyperthyroidism

1. Thyroidectomy

A full discussion of thyroidectomy is beyond the scope of these notes. However, surgical thyroidectomy remains a good choice of treatment in cats where this is no ectopic hyperfunctional thyroid tissue. Owners, however, should be warned about the possibility of ectopic tissue, and failure to cure the hyperthyroidism, as well as possibility of recurrence in the future. Williams et al reported 1 in 3 cats having thyroidectomies did not have permanent resolution of hyperthyroidism. One study on scintigraphic findings of hyperthyroid cats reported up to 20% had ectopic thyroid tissue[8], however such a high percentage of cats with ectopic thyroid tissue was not supported in a much larger study, where only 4% had ectopic tissue.[9]

There are more significant potential risks associated with thyroidectomy compared to radioiodine treatment. Naan *et al* 2006[10] reported a 2% mortality rate, 6% that had post-operative complications and 5% that had recurrence of hyperthyroidism. Cats should be appropriately stabilised with medical therapy, prior to surgery, to reduce anaesthetic risk. Another concern associated with thyroidectomy is the development of post-operative hypoparathyroidism and subsequent hypocalcaemia. In most instances, this will resolve with time, and treatment with calcium supplementation and vitamin D is only required in the short term until parathyroid function is regained. However, occasionally cats can remain hypoparathyroid and require long term treatment with vitamin D +/- calcium supplementation.

2. Radioiodine treatment

Radioiodine treatment is considered the gold standard treatment for feline hyperthyroidism worldwide. It is a curative treatment that does not require anaesthesia and has no significant side effects. Efficacy is not dependent on the location of hyperfunctional thyroid tissue, so presence of ectopic hyperfunctional thyroid tissue does not affect outcome. Reported survival times are significantly longer for cats treated with radioiodine than with other treatment modalities.[11, 12]

Historical 'issues' associated with cost and availability have already received comment above. In Australia, radioiodine is now widely available throughout NSW, VIC, ACT, QLD, SA and TAS. With required post-treatment hospitalisation times in Australia usually only being 5-7 days, there is rarely contraindication for this as a treatment.

Sometimes veterinarians report that owners are reluctant to pursue radioiodine treatment. However, this is perhaps in part due to a lack of information. Interestingly, in a survey of UK owners of hyperthyroid cats, cost of treatment, travel distance and waiting time for treatment had a low impact on owners' treatment choice.[13] Owners' main concerns with treatment choice were hospitalisation length and resultant concerns about the cat being unhappy and/or the owner missing the cat. We are fortunate in Australia that the hospitalisation time is such that these potential concerns are less of an issue, and therefore it would be anticipated, based on these results, that most owners would be happy to pursue radioiodine treatment if it was advised and they had received adequate information.

Iodine-131 is the isotope used for treatment. This has a half-life of 8 days and emits both beta-particles and gamma radiation. The iodine is concentrated within the thyroid gland and its emitted radiation destroys surrounding functioning thyroid cells. It is β-particles that causes most of this damage, and because they travel <2mm in tissue, radiation damage does not affect surrounding structures. Thyroid cells that are not destroyed immediately develop abnormalities reducing their survival time. The thyroid damage therefore is both immediate and ongoing, resulting in euthyroidism usually within 4-30 days, although a small proportion of cats can take significantly longer to become euthyroid. The treatment is usually a single treatment,

and can be given via subcutaneous or intravenous injection, or administration of a capsule orally, the latter being more common in Australia and much less expensive.

Many centres offering radioiodine treatment historically used fixed dosing protocols, most commonly around 4mCi (148MBq). However, fixed dose protocols mean that a proportion of cats may not receive an adequate dose, whilst some cats may be overdosed resulting in hypothyroidism. Development of hypothyroidism did not used to be a concern, and clinical evidence of hypothyroidism is rare. However, recent work has shown that iatrogenic hypothyroidism appears to contribute to the development of azotemia after treatment of hyperthyroidism, and reduced survival time in azotemic cats.[5] The aim of radioiodine treatment, therefore, is to cure the hyperthyroidism without causing hypothyroidism, and a variable individual dosing regime is more likely to achieve this. Individual doses are calculated based on severity of hyperthyroidism, taking into considering clinical parameters, T4 concentration, the size of the thyroid, age of the cat and presence of any concurrent diseases.

Significant changes in kidney function occur within 4 weeks post-treatment and none thereafter,[14] which would make sense when a sudden large reduction in thyroxine occurs during this time. As a result of this recent knowledge regarding hypothyroidism, and changes in renal function post-treatment, a sensible idea that has been utilized in some radioiodine treatment centres now for several years (R Malik, & S Pegrum, *personal communications*) is to supplement cats with thyroxine immediately post-treatment, weaning off treatment over 2 months. Total T4 and renal parameters are then reassessed once thyroxine is stopped 2 months post-treatment, in order to assess efficacy of the radioiodine treatment, and presence of azotaemia and/or persistent hypothyroidism. If hypothyroidism is present at this stage, particularly in cases that have developed azotaemia, then thyroxine supplementation is continued. Thyroxine crushed within their food is easily accepted by cats, so medication is rarely problematic. In these cases, curing the hyperthyroidism, and supplementing with physiological doses of thyroxine is considered much more preferable than trying to manage hyperthyroidism with long term daily control of thyroxine synthesis. ■

Figure 5. Even anxious cats tend to settle in these new radioiodine cages very well, and cats that are usually too anxious to eat when in hospital tend to eat very well in the comfort and quietness of these cages.

References:

1. Peterson ME, Broome MR. Hyperthyroid cats on long-term medical treatment show a progressive increase in the prevalence of large thyroid tumors, intrathoracic thyroid masses, and suspected thyroid carcinoma. *J Vet Intern Med* 2012;v26:1523.
2. Higgs P, Murray JK, Hibbert A. Medical management and monitoring of the hyperthyroid cat: a survey of UK general practitioners. *J Feline Med Surg* January 2014; online
3. Paepe D, Verjans G, Duchateau L, *et al*. Routine health screening: findings in apparently healthy middle-aged and old cats. *J Feline Med Surg*. 2013; 15: 8-19
4. Boland LA, Hibbert A, Harvey AM. Incidence of comorbid disease in cats referred for radioiodine treatment. *J Vet Intern Med* 2009; 6: 1347
5. Williams T, Elliott J, Syme H. Association of iatrogenic hypothyroidism with azotemia and reduced survival time in cats treated for hyperthyroidism. *J Vet Intern Med* 2010; 24:1086-1092
6. Williams TL, Elliot J, Syme HM. Effect on Renal Function of Restoration of Euthyroidism in Hyperthyroid Cats with Iatrogenic Hypothyroidism. *J Vet Intern Med* April 2014, online
7. van der Kooij M, Becvarova I, Meyer HP, Teske E, Kooistra HS. Effect of an iodine-restricted food on client-owned cats with hyperthyroidism. *J Feline Med Surg* 2014; 16 (6): 491-498
8. Harvey AM, Hibbert A, Barrett EL et al. Scintigraphic findings in 120 hyperthyroid cats. *J Feline Med Surg* 2009; 11 (2): 96-106
9. Peterson ME and Broome MR. Thyroid scintigraphy findings in 2096 cats with hyperthyroidism. *Vet Radiol Ultrasound*. May 2004, online
10. Naan EC, Kirpensteijn J, Kooistra HS, Peeters ME. Results of Thyroidectomy in 101 Cats with Hyperthyroidism. *Vet Surg* 2006; 35 (3): 287-293
11. Milner RJ, Channell CD, Levy JK and Schaer M. Survival times for cats with hyperthyroidism treated with iodine 1131, methimazole, or both: 167 cases (1996-2003). *J Am Vet Med Assoc* 2006; 228: 559-563
12. Peterson ME and Becker DV. Radioiodine treatment of 524 cats with hyperthyroidism. *J Am Vet Med Assoc* 1995; 207: 1422-1428
13. Boland LA, Murray JK, Bovens CPV, Hibbert A. A survey of owners' perceptions and experiences of radioiodine treatment of feline hyperthyroidism in the UK. *J Feline Med Surg* January 2014, online
14. van Hoek I, Lefebvre HP, Peremans K, Meyer E, Vroubels S et al. Short and long term follow up of glomerular and tubular renal markers of kidney function in hyperthyroid cats after treatment with radioiodine. *Domestic Animal Endocrinology*. 2009; 36: 45-56

Back to table of contents

preventic®

The First Line of Defence

Against Deadly Paralysis Ticks

PARALYSIS TICKS take only a few days to affect your dog – and can be deadly soon after.

Preventic is ***FAST*** – It starts working within ***24 HOURS*** against Paralysis Ticks.

Virbac

Shaping the future of animal health