THE UNIVERSITY OF SYDNEY
Centre for Veterinary Education
Professional Development Leaders

C&T

CONTROL AND THERAPY SERIES

March 2013 ISSUE 270
Australia's Leading Veterinary Forum

Feature Article

A case of canine symmetrical lupoid onychodystrophy

Enter me in the CVE$500 DRAW

It's easy – read this issue and answer the Question hidden inside

Treatment of penile lesions in bulls

Phil Lindsay, this issue's Major Prize Winner, pictured with 'Tom', 'Jack' and 'Pippi'.

‹e-book› Chance to Win $500 in CVE $ (see page 33)

Centre for Veterinary Education
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CONTROL AND THERAPY SERIES

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CONTENTS

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COVER IMAGE
Anne Fawcett's dog 'Phil' at Bronte Beach, NSW.

Welcome to the first edition for 2013. Once again we have a very mixed bag of articles mainly dealing with companion animals, though there are a couple of cattle articles. Please remember that we welcome your contributions on any topic or species and appreciate the time it takes to prepare articles, so don't be bashful, send them in!

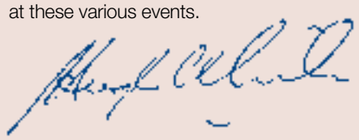
For those of you who receive a hard copy of the C&T, this is just one of the many benefits you get from your membership to the CVE. We know that many practices love their hard copies as they are frequently seen in the vet office, the treatment room or the practice lunch room. Recently we heard of a veterinary colleague looking for a job in England and finding last year's C&T with the Poisons Perspectives featuring prominently in not one, but two English practices.

Our Editor, Lis Churchward, is becoming very concerned that many of you hard copy readers have not taken the time to look at the ebook version of the C&T. We understand that many people are glued to reading hard copies rather than reading articles on the computer or tablet screen, but we urge you to take the time to access the complementary ebook. When you do this you can watch video clips, enlarge images, get instant access to related articles in previous issues and more. The ebook format requires a lot of extra work on Lis' part and comes at an added expense to the CVE, but we believe this is a wonderful resource that adds an even greater benefit to membership of CVE. Please make Lis happy and take the time to wander through the ebook. We know that once you have watched the film clip in this issue you will wonder why you did not do so earlier, and want to look through all of last year's ebook versions.

Lis is so determined to get your attention that she has offered a prize to one of the lucky people who visits and reads the ebook for the first time. See inside for more details.

The CVE has recently introduced a new resource which can be accessed on the CVE website – see http://www.cve.edu.au/saas. This is our new article summaries section which contains two sections – article summaries relating to small animal medicine and feline medicine and surgery journal abstracts provided by the ISFM, the CVE partner in Feline Distance Education. Years ago the PGF used to provide a subscription service to article summaries from a broad range of journals from around the world, but this was discontinued due to rising costs. We are proud to be able to reintroduce this service at no cost to anyone who wants to access the website and scroll through the many articles covering a wide range of subjects.

Take time to study the broad offering of courses being offered by the CVE this year, including great conferences, seminars, workshops, on-line courses and webinars. There is something for everyone and as usual we have tried to reach most geographical regions of Australia. I look forward to meeting up with many of you at these various events.



Hugh White BVSc MVSc MACVSc
DIRECTOR

Graeme Allan awarded DVSc

By Anne Fawcett

Roslyn and Graeme Allan.

Page 1 Page 2 Page 3 Read Graeme's Occasional Address from the Graduation Ceremony

On December 14 2012, as another cohort of students graduated with the Bachelor of Veterinary Science degree, Graeme Allan was awarded the coveted Doctor of Veterinary Science (DVSc) in recognition of his unique and prolific contribution to the field of veterinary diagnostic imaging.

The DVSc is a rare honour, having been bestowed to only some 30 candidates since it was first conferred to Ian Clunies Ross in 1928. It is awarded to outstanding researchers who submit a body of work which is deemed to have made a consistent and distinguished contribution to veterinary science. Candidates must submit a collection of original publications for assessment by no less than three examiners who are considered pre-eminent in their respective research field.

Previous recipients include Ian Beveridge, Hugh (Hughie) Gordon, Cliff Gallagher, Marsh Edwards, Daria Love, Reuben Rose, Paul Canfield, Richard Malik and Peter Windsor. Ian Lean was also conferred with a DVSc in 2012.

According to Professor Rosanne Taylor, Dean of the Faculty of Veterinary Science, 'Graeme is remarkable as being one of the few veterinarians who have earned our highest research degree for their work undertaken, while working as a practitioner, which has greatly contributed to advancing veterinary clinical sciences, nationally and globally.'

But Allan is the first to insist that this was not a solo effort.

'It really was a team effort. Imaging itself is just imaging,' he says. 'I've introduced clinical elements and collaborated with others to combine our knowledge to elucidate strange disorders.'

His thesis, entitled 'Radiological Studies of Disease in Companion and Zoo Animals', is a compilation of more than 40 years of collaborative studies looking into a range of conditions, including pioneering studies on contrast radiography, oesophageal dysfunction, radiotherapy for treatment of cancer in companion animals right through to new forms of rickets in rex kittens and osteocondritis in snow leopards.

The examiners unanimously agreed that Allan's contribution to the field was outstanding. Professor Donald Thrall, best known as the editor for *The Textbook of Veterinary Diagnostic Radiology*, described Allan as 'a renaissance man, with talents in many areas; accomplished veterinary diagnostic radiologist, investigator of numerous problem areas, teacher, and mentor.'

Emeritus Professor Patrick Gavin, of Washington State University, wrote that Allan's thesis 'will facilitate veterinary education, new veterinary radiologists, residents, interns, and veterinary science students.'

He compared the writing in Allan's introduction – in which his early influences and career trajectory are summarised – with that of James Herriot.

Professor Erik Wisner, Chair of UC Davis' Department of Surgical and Radiological Sciences, wrote that 'veterinary diagnostic imaging has only recently emerged as a specialty compared to other clinical disciplines and advances in imaging technology and computing power have caused the specialty to expand and evolve at an astonishing rate. Dr Allan, considered one of the pioneers of the specialty, has successfully navigated and adapted to this ever-changing terrain throughout his career.'

The great irony is that Allan graduated without ever having had a single lecture in radiology. But when one delves into the early life of Allan, imaging doesn't seem a far-fetched career option.

He was born in New Zealand in 1940 and grew up in Waipawa, a quiet rural town.

'Dad was the town doctor, he really liked living in the country,' Allan says. 'So I spent a lot of time within a farming community, playing tennis, fishing the rivers and enjoying that sort of lifestyle. As I thought more about what I would do with my life I thought that living in this sort of community and being a vet would be pretty good.'

That wasn't Allan's only aspiration. He really wanted to be a pilot or a cinematographer.

'At 6ft 3inches I was told I was too tall to be a pilot. I didn't have great eyesight either and when I look back it is patently obvious that it wasn't the right career choice: I would have hated to be responsible for all of those people up in the air.'

When he attended an interview at Film New Zealand, the panel advised him (incorrectly as it would turn out) that there was no future for cinematography in New Zealand. On hearing this, Allan's parents, who had driven him to Wellington for the interview, said it was 'obviously a loony idea'.

A career in veterinary science became top on the list.

'Vets always had a good lifestyle, an interesting job driving around helping people, and that area of New Zealand was really beautiful so it wasn't hard to say I wanted to be a vet.'

At the time there was no veterinary school in the country. Instead, each year the NZ Government provided funding for 25 students to train in Australia – in exchange for working in New Zealand for five years upon graduation.

Travelling overseas was a big deal.

'People didn't just hop on aeroplanes,' Allan says. 'One of my sister's girlfriend's was going to London, it was going to be a five day trip with eight or ten stops and the entire community turned out to her farewell.'

Still, Allan was undaunted.

'Waipawa was a nice town but there had to be a bigger world.'

He enrolled at the University of Sydney.

'We were occasionally shown radiographs but we were not taught how they were taken, how they were processed or a systematic approach to their interpretation,' Allan recalls.

'The mystery of radiography at the veterinary school at that time was known only to Mr Arthur Gee, a faculty employee with a background in medical radiography.'

During his studies, Allan met Roslyn Ward, also a Sydney University student, and his plans to return home fell by the wayside. He opted to pay out his bursary and remain in Australia, a decision he has never regretted. The pair married in 1966.

In 1965, at the end of his studies, Allan took up a locum position in Mildura.

'The vet met me at the train station with his whole family bundled into the car,' Allan says. 'After a brief introduction, he took me to the practice and he and his family disappeared to the coast. That was not an unusual experience in those days.'

It wasn't a particularly edifying one either. The practice had clients all the way from Broken Hill to the south-Eastern border of South Australia. The practitioner had left Allan with his wife's Mini-minor, hardly equipped to endure the tough dirt roads of the outback.

'I was all alone and I just didn't know a damn thing,' he admits. 'I spent a hell of a lot of time driving and getting lost, visiting people who you knew wouldn't pay you...but it didn't occur to me that it was that bad. It wasn't until I learned that some graduates were committing suicide that I realised, and I could understand how some people could be driven to despair in such circumstances. I've always advised new graduates never to join a one man practice; always join a multi-vet practice – you need the support and protection of many people.'

It was a different era, as Allan explained in his address to the graduates of 2012.

'My first boss was a man who had graduated prior to WWII, in the pre antibiotic era. His antimicrobials were antiseptics and sulphur compounds, and when I commenced working with him in 1966 his pharmacy shelves still contained many different sulphur-based medicines. I was lucky, because we had access ▶

Authors' views are not necessarily those of the CVE

Back to TOC page

News

to penicillin, streptomycin and chloramphenicol, the antibiotics of that time.'

Vaccinations for common viral diseases had become available, but many animals remained unprotected.

'I remember as a boy many of our pet cats dying pitifully of feline enteritis. Equally, distemper was no stranger either. When I was working in a NSW country town a drover with a mob of sheep came into town. He bought his dogs to the clinic, but we couldn't help him, and had to watch as one by one they died of distemper. Those dogs were his life. It was heart-breaking.

I'm not sure what happened to the man, or the sheep that he was droving, but I remember his pleas for help.'

Upon graduation, Allan accepted a position at North Shore Veterinary Hospital which possessed a small X-ray machine (it had an output of 90kVp and 30mA – the sort of machine fondly referred to in imaging circles as a 'bullet detector'). Under the mentorship of Rowland Pursell, a veterinarian who had pioneered the development of tick antiserum, Allan was bitten by the discovery bug.

This signalled a change from his university days, when Allan admits he wasn't a stand-out scholar.

'I wasn't a good student in the traditional sense. I didn't like lectures and I didn't learn from the reams of notes that were handed out.'

But reading case reports and journal articles related to the conditions of animals he was treating in practice was endlessly fascinating.

'I found that reading journal articles and looking at clinical material and trying to work out what was going on was a tremendous way for me to learn more about what I did. For the first 10 years after I graduated I worked in clinical medicine and that is what I did – so all of my early publications related to what I was seeing and doing in practice.'

'I didn't attempt to do a PhD because I had no reason to want to do one,' he says. 'I wasn't an honours graduate, and at the time you really had to be dedicated to an academic career to undertake a PhD - that didn't sound like my job description.'

Nonetheless, Allan has made a prolific contribution to veterinary knowledge.

'When you read journals a lot, writing a paper isn't a huge jump – it seemed a natural thing to do, so that was an activity I did in the evenings.'

North Shore was an unusual practice in that two of the four veterinarians working there were female. One, Jennifer Edols, helped the young Allan write up his very first case report. The patient was a cocker spaniel presented for a routine spey. However, the discovery that the bitch possessed an os penis caused much head scratching.

'This led to karotyping the dog, something which was completely new to me and made me realise that there was so much that I didn't know. It also led to cooperative work with members of the medical profession, who had the skills and laboratory facilities that enabled us to stretch our investigations.'

The article, entitled 'A case of male pseudo-hermaphroditism in a cocker spaniel' was published in the *Australian Veterinary Journal* in 1968.

The realisation that practice constantly generated new knowledge was to see many papers follow.

'I found it was through clarifying my own ideas by reviewing the literature, and writing, that I could best further my knowledge as a new graduate.'

Allan immersed himself in his work, probably – he reflects – a bit too much.

'At our recent reunions people from my year have talked about the fact that back then we expected to work seven days a week, were expected to be on call seven nights. We knew we'd be back at work after dinner, we would have to work on weekends and when the phone rang at 2am we would have to get out of bed – it came with the territory,' Allan says.

'I remember we had a baby sitter who was a university student. My wife agreed she could observe our family for a case study which was an important part of her course, and she spent a considerable amount of time at our house. I was shocked to find that in her report she determined that ours was a single parent family because I was rarely there'.

No matter how long the hours, Allan enjoyed his work.

'My wife once told me that she didn't know anyone who looked forward to going to work in the morning the way I did. I still do.'

Allan was fortunate at the time to have two close friends – Rolfe Howlett (then a PhD candidate investigating bone pathology), and Bruce Duff (then training in veterinary pathology). The trio exchanged ideas about cases and acted as a 'brains trust' for one another.

IMAGING PEARLS OF WISDOM FROM GRAEME ALLAN

- When in doubt, change the developer.
- Invest wisely in digital radiography systems – there are a lot of systems out there, some are good, some aren't.
- Everybody can learn to do ultrasound. It's just about familiarity and application. You can't do one a week and get good at it.
- With ultrasound, image resolution is everything.
- For all the things missed by not knowing, there are 10 things missed by not seeing.

No one in Allan's practice at the time really knew what to do with the X-ray machine, least of all him.

'My shortcomings as a radiographer became manifest as frustration with the modality. I used to spend my weekends radiographing animals and hanging the film on pegs on the clothesline in the backyard. I realised that not only did I not know anything about radiology, but hardly anyone else did either.'

He commenced a Masters by research under the supervision of Richard Dixon at Sydney University. Dixon had recently returned from Colorado State University where he had received advanced training in radiology and radiotherapy. He proved a wonderful mentor, helping Allan investigate contrast media in cholecystography of dogs.

Another faculty member, Graham Cotton, arranged for Allan to observe radiology rounds at the Royal North Shore Hospital.

'These weekly sessions introduced me to a medical radiology community. While I found these sessions richly rewarding, the medical radiologists in turn were fascinated by the range of skeletal disorders that we encountered in dogs and cats.'

Another key player at the time was John Holt, a leader of the growing small animal contingent of veterinary practitioners.

'John had a lot of ideas, he had been to America, visited a lot of practices and invited them to address practitioners in Australia,' Allan said. 'He was so impressed with the American Association of Animal Hospitals' system of accrediting practices that he was dedicated to improving the standards of his own practices. Through the ASAVA he started the *Australian Veterinary Practitioner* (AVP).'

Aside from contributing to the AVP, Allan helped coordinate printing.

'I have a theory that there is a time in your life when you are very energetic and these sorts of things are quite easy to achieve during that energetic phase,' he explains. 'You do come to a point where you become more distracted and that is when it is important to have other energetic, enthusiastic people around.'

Soon after completing his masters, Allan joined Bob Kibble and Greg Ross at the Ku-Ring-Gai Veterinary Hospital, where he investigated dwarfism disorders in German Shepherds.

'It's never easy accumulating data in a practice situation where expensive tests and procedures are required, and invariably the cost of pursuing interesting case material was self funded,' Allan says. 'This is a situation that has existed for my entire career.'

Allan, along with Clive Huxtable, Rolfe Howlett, Rob Baxter, Bruce Duff and Brian Farrow, published the seminal paper on pituitary dwarfism in German shepherds in the *Journal of Small Animal Practice* – at the time the most prestigious small animal medicine journal in the Commonwealth.

While at Ku-Ring-Gai, Allan took charge of refurbishing the radiology suite, installing a high output X-ray machine and an automatic film processor. But the frustration of not being competent at radiography proved overwhelming. Allan knew that training in North America was the only solution.

'This was an agonising decision as I was a part owner of the practice. My family, Rolfe Howlett and Richard Dixon were all influential in helping me decide to leave my safe veterinary practice environment, sell my shares in the practice, and move to Ithaca in upstate New York.'

Allan completed a residency at the New York State College of Veterinary Medicine under the supervision of Victor Rendano. His overseas experience opened up new worlds – Allan had a taste of diagnostic ultrasound, radiotherapy, contrast studies and advanced imaging as well as radiography. Allan sat his board exams and passed with flying colours – though not without the help from colleagues.

'I was never that interested in equine radiology and it wasn't one of my strengths. Some former candidates worded me up. They told me one of the examiners regularly showed a radiograph of a horse with an osteosarcoma, even though they are as rare as rocking horses. They said the radiograph would have metal seeds in it, signifying that the horse had had radiation therapy, and that it would be smart to consider the possibility that the osteosarc was radiation induced.'

'So the exams came around and sure enough the examiner pops out this radiograph, and I looked at it quizzically and I said, "You don't suppose it could be a bone tumour, induced by this particular radio isotope?" He said "You're brilliant" – I wouldn't have come through the equine section any other way.'

Upon returning to Sydney he worked hard to create a viable practice relying solely on referral work, also working with Holt and Lindsay Hay for several years while he found his feet.

'John had a fluoroscope and I installed a radiation therapy machine for treating squamous cell carcinomas on cat's faces. They were happy days,' Allan recalls.

It was a dry period. Allan spent much of his spare time writing case histories for the *AVP*, covering diseases which were and remain difficult to diagnose.

'These cases were designed to demonstrate that ordinary practitioners could practice "on the edge", just as effectively as many better qualified colleagues,' he says.

Allan developed one of the first Distance Education programs offered by then Post Graduate Foundation in Veterinary Science, now the Centre for Veterinary Education.

'The idea arose out of symposia that were organised by Doug Bryden,' says Allan. 'Originally we had 3-4 day sessions where practitioners would have intensive workshops and focus on a particular subject. Doug asked me what I thought about developing a radiology distance education course. Having an understanding of what distance education entailed – the resources, the time commitment from tutors and participants, and the need to constantly update and maintain material, I told him I thought he was crazy.' Bryden, not known for backing away from a challenge, said 'do it anyway'.

Giselle Alegria, who had just graduated from secretarial school, gave Graeme boxes of cassettes and a dictaphone. He dictated the course as he drove to and from Canberra, which was part of his practice, and Giselle painstakingly transcribed each recording.

'Giselle deserves enormous credit for creating not only my DE material, but that of several others too' Allan says.

The program – along with internal medicine – was so successful that it ran again. And again. Continuously from 1991 to 2012. Other programs were added, but radiology remains one of the most popular distance education modules. After a twelve month hiatus, the course will run in 2014 in a slightly different format. (See www.cve.edu.au in April 2013 for our DE brochure containing details on this and other CVE Distance Education courses available in 2014. Alternatively, contact ***cve.disted@sydney.edu.au*** or call (02) 9351 7979. ▶

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Back to TOC page

Together with Robert Nicoll – also a tutor on the DE course – he established Veterinary Imaging Associates, a diagnostic imaging consultancy which provides services to veterinarians around Australia. Allan and Nicoll taught diagnostic imaging at the University of Sydney for over a decade while maintaining their private practice.

At the age of 72, Allan shows no signs of slowing down. He continues to work a four-day week, visiting practices in the Sydney region to read films and perform ultrasound while mentoring dozens of practitioners and sharing his wisdom.

'I see a very broad range of practice standards and styles, and it amazes me how some people have adapted to the modern era and some people haven't,' Allan says. 'I've always maintained that practices select their clientele. Practices with low standards often end up with clients who are unlikely to want sophisticated services. But practices which have maintained contact with the times select clients who want that sort of service.'

Retirement is not imminent, but Allan is asked almost daily when he might give up the game.

'You do get to a time in your life when people are obviously thinking "you are an old bugger and why are you still here?"' he says. 'There is a palpable bias against older people in the profession and you do hit that wall. I will continue doing this while I enjoy it, but I couldn't go back to University teaching. The attitudes of today's students to teaching and learning are so different to what I am used to I am not sure I would like to adapt – but I also wouldn't put up with the ageism that is so widespread at universities.'

The secret seems to be the enthusiasm for discovery and thirst for knowledge that bit him with his very first paper.

It also helps to have an incredible support network.

'I think you have to have a sympathetic partner,' Allan says. 'Roslyn has put up with a lot. For me it's been an extremely satisfying and rewarding thing to do. It's not hard for me to want to keep doing it.'

'At a recent reunion one of my year mates told me he hated being a vet all his life – and I thought that was really strange. I can't imagine not enjoying what we do. There are so many clinical syndromes that we come across in practice all the time, and if you keep your eye open you can build very interesting case histories. Imaging is just imaging, but where I've been successful is in collaborating with others to help elucidate strange disorders. I've been so lucky to be surrounded by inquisitive and enthusiastic people.'

NOTE: An earlier version of this article was published in *The Veterinarian* magazine. It should be noted that while every effort was made to mention those who played a key role in Dr Allan's career, it is simply not possible to include everyone. The author apologises for any such omissions.

A list of recipients of the DVSc up to the year 2010 can be found in Paul Canfield's *One Hundred Years: The School at the Foot of the Hill*, available to download from the Veterinary Science Foundation website at ***http://sydney.edu.au/vetscience/centenary/publications.shtml***

CVE Clinical Competency Awards for 2012

Each year we take great pleasure in inviting each Australian and New Zealand Faculty of Veterinary Science to choose a recipient for our Clinical Competency Award.

This prize of $1,000 worth of CVE publications and/or other CVE products (events/courses etc) is offered to the graduating student who has been recognised by their Faculty as being the most competent in clinical skills over the clinical portion of his or her undergraduate years.

We are delighted to award the prize to the following graduates:-

Charles Sturt University	**Stuart GEARD**
James Cook University	**Abigail PRIESTLAND**
Massey University	**Kate GRIFFITHS**
Murdoch University	**Alana KIRK**
University of Melbourne	**Kai NGOR**
University of Queensland	**Sarah LIVINGSTONE**
University of Sydney	**Lucy KOPECNY**

Abigail Priestland is presented with her award by Dr Ruth Sutcliffe of JCU.

Call for more Large Animal C&Ts

It's been a long time since we published a C&T on Goats, Sheep, Pigs, Llamas, Alpacas or Deer.

If you work on an interesting large animal case, please take pics – **even better, film it on your mobile for the C&T Series e-book version of each issue** – write it up and send it in to us. Email: elisabeth.churchward@sydney.edu.au

We need YOU, our members/readers, to ensure that the Series remains broad and interesting.

You don't have to be a CVE Member to contribute articles to the C&T Series.

Bad hair day – 'Muesli' (a Boer goat) in the process of shedding her winter coat. image courtesy of Scott Reid.

Congratulations to all our 2012 DE Participants

On behalf of our Distance Education Tutors and DE Co-ordinators, we would like to congratulate the following participants for their dedication to continuing veterinary education and pursuing Tom Hungerford's *Goanna Track to Success*. Tom Hungerford OBE BVSc FAVSc HDA, our first Director, was a legend in the veterinary profession and famous for this *Goanna Track* philosophy i.e. vets should take one area of veterinary practice and become thoroughly familiar with all aspects of it – conquer it completely. Then, you train others to run this aspect and proceed, like a goanna, to the next challenge and do the same again.

Behavioural Medicine
TUTOR: Kersti Seksel, NSW
Anne-Lise Blum, United Kingdom
Linh Bui, Vietnam
Karen Ellis, United States
Frederique Hurly, South Africa
Eloise Koelmeyer, QLD
Coralea Larsen, NSW
Simon Lee, NSW
Marika Ley, VIC
Fiona McNeil, VIC
Sally Nixon, VIC
Jennifer Pearson, United States
Melanie Rockman, VIC
Annabel Shepherd, QLD
Robin Wiley, Canada
Nicola Wilson, New Zealand

Cardiorespiratory Medicine
TUTOR: Nick Russell, United States
Rose Anderson, ACT
Joyce Chi Taiwan, Province of China
Louise Dawson, NSW
Clinton Eichelberger, Hong Kong
Delwyn Fenby, ACT
Sheila Gadaloff, QLD
Karlo Gicana, Philippines
Doug Gray, QLD
Nina Khin, Hong Kong
Alexandra McLeod, NSW
Alison Moore, VIC
Timothy Nottage, New Zealand
Annie Rose, QLD
Pongrawee Saenpanya, Thailand
Piyanuch Tangjarukul, Thailand

Diagnostic Imaging
TUTOR: Graeme Allan, NSW
TUTOR: Robert Nicoll, NSW
Chris Andersen, ACT
Paula Baskeyfield, Hong Kong
Justin Cardwell, QLD
Jeffrey Clyne, SA
Jaclyn Eng, Singapore
Katrina Garrett, QLD
Stephen Gibson, NSW
Reza Kamgarpour, QLD
Helen Kwan, Hong Kong
Kee Chee Jacqueline Lam, Hong Kong
Jeremy Mannix, VIC
Karen Phillips, SA
Panida Pongvittayanon, Thailand
Sally Rekers, VIC
Michelle Sutherland, NSW
Jonathan Young, ACT

Emergency Medicine
TUTOR: Sandra Forsyth, NZ
TUTOR: Trudi McAlees, VIC
Siti Abdul-Khalid, QLD
Michael Bell, VIC
Betty Chow, United Kingdom
Sophie Curtis, QLD
Michelle Eads, VIC
Kellie Farraway, QLD
Lisa Ferguson, New Zealand
Claire Flaherty, VIC
Brigid Hales, VIC
Viv Hill, United Kingdom
Alan Hillier, TAS
Hollie Hodgkinson, SA
Lin Hoi, Hong Kong
Nicola Hooper, Hong Kong
Liia Kelman, NSW
Karin Kuh, Hong Kong
Julie Anne Mackenzie, NSW
Bridget Macleod, QLD
Marietjie Malherbe, VIC
Leah Manning, NSW
Peter Prendergast, NSW
Nor Rahman, Malaysia
Lau Sang Sang, Malaysia
Sittinard Sawatmongkornkul, Thailand
Greer Sheridan, VIC
Olivia Wade, SA
Kai Yin Wong, NSW
So Yang, NSW

Equine Medicine
TUTOR: Darien Feary, SA
TUTOR: Tony Mogg, NSW
Sarah Cavill, VIC
Surita Du Preez, QLD
Cindy Dudgeon, VIC
Rachel Kent, QLD
Anders Kragh, Denmark
Gian Macolino, NSW
Claudia Marescotti, Italy
Roisin Mc Quillan, New Zealand
Tamara McElroy, NSW
Robin Moore, South Africa
Olivia Nugent, VIC
Miguel Pajate, QLD
Deborah Rogers, QLD
Morgan Weber, WA
Clare Williams-Paterson, VIC
Lianne Wright, VIC

Equine Reproduction
TUTOR: John Chopin, NSW
TUTOR: James Rodger, NSW
Jason Andrews NSW
Tony Batterham NSW
Andrew Bennett NSW
George Corones NSW
Benjamin Czerwonka-Ledez QLD
Andrew Daniels NSW
Melissa Ford SA
Christopher Reardon QLD
Raina Scott NSW

Feline Medicine
TUTOR: Andrea Harvey, UK
TUTOR: Wayne Mizon, ACT
TUTOR: Carolyn O'Brien, VIC
TUTOR: Andy Sparkes, UK
TUTOR: Richard Malik, NSW
Aarti Amin, United Kingdom
Kerry Burke, New Zealand
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Yaiza Mejias, Spain
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Freddy Nilsen, Norway
Gitty Otte, Netherlands
Marisa Palmero, Spain
Chia-Kang Peng, Hong Kong
Jakub Pernak, United Kingdom
Pia Rapp, Finland
Uwe Reinicke, Germany
Muna Roberts, United Kingdom
Maya Robinson, QLD
Candice Schilder, VIC

Back to TOC page

Nadia Sternberg, QLD
Sarah Tawse, WA
Johanna Todd, NSW
Claire Williams, United Kingdom
Catherine Williams, United States
Angeline Wong, Hong Kong
Barbara Wright, VIC

Internal Medicine: Keys to Understanding

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TUTOR: Darren Merrett, VIC
TUTOR: Jennifer Brown, VIC
TUTOR: Kate Hill, NZ
Angie Armstrong, QLD
Stephen Baumberg, New Zealand
Jennifer Coates, NT
Sarah Cooper, VIC
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Duy Ngo, Vietnam
Brigit Pitman, NSW
Christie Robinson ,New Zealand
Emily Sandow, SA
Lucy Shum. Hong Kong
Annabel Sutch. Hong Kong
Agus Wijaya. Indonesia

Internal Medicine: Problem Solving

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Valerie Ng, Singapore
Tara O'Connell, QLD
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Patricia Pak Si Poon, Hong Kong
Jessica Poulsen, VIC
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Kang Li Wong, Malaysia

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Catherine Forman, TAS
Kanokwan Kewsuwan, Thailand
Ramona Maier, Germany
Alison Neef, NSW
Ataya Pibulvej, Thailand
Supreeya Sreesampan, Thailand
Sonja Vorster, QLD
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Wendy Chee, Singapore
Janet Cridland, NSW
Supitsara Hathailak, Thailand
Victoria Onus, NSW
Noppadon Saohong, Thailand
Shiyamala Sivaji, NSW
Nareuthorn Suthiyotha, Thailand
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Ruminant Nutrition

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TUTOR: Guy Yates, VIC
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Benjamin Trotter, WA
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Jeremy Watson, VIC
Bradley Wundke, SA

Congratulations to our DE Early Bird Winners for 2013

Paying early ensured the following vets not only secured a place in the DE course of their choice for 2013, they also received a hefty discount and were the 3 lucky winners in our Early Bird draw, winning an iPad each. Congratulations Nicholas, Luke and Libby and to all our DE Participants who secured a place in our 2013 programme.

Nicholas Chng – Surgery


Luke Annetts – Equine Reproduction 1


Libby Harriman – Beef Production

EVENTS IN 2013

17 Mar	Wound Management & Reconstruction	Launceston
23 Mar	Basic Echocardiography	Dubbo
24 Mar	Advanced Echocardiography*	Dubbo
14 Apr	Hot Topics in Feline Medicine	Perth
19 Apr	Senior Moments – An Insight into Geriatric Cats & Dogs	Sydney
28 Apr	Looking Down the Microscope	Adelaide
5 May	Diabetes	Melbourne
18/19 May	Hip & Stifle in the Dog	Perth
18 May	Canine Rehabilitation I: Massage Therapy	Sydney
29 May	Canine Rehabilitation II: Intensive Care	Sydney
2 Jun	Hot Topics in Feline Medicine	Canberra
14 Jun	ecoCPD: Practical Radiology for the General Practitioner	Sydney
24-28 Jun	Cardiorespiratory Conference	Melbourne
13-14 Jul	Approaches to Avian & Exotics	Sydney
19-21 Jul	External Fixators	Melbourne
27 Jul	Basic Echocardiography	Townsville
28 Jul	Advanced Echocardiography*	Townsville
23-26 Sep	Surgery Conference	Fremantle
13 Oct	Diabetes	Brisbane
27 Oct	Looking Down the Microscope	Port Macq.
8 Nov	ecoCPD: Behaviour	Sydney

* Prior learning will be required to attend this workshop.

ONLINE COURSES IN 2013

18 Feb - 17 Mar	TimeOnline: Dermatology
25 Feb - 24 Mar	TimeOnline: Marine Wildlife
1 Apr - 28 Apr	TimeOnline: Treating Burns (Students)
8 Apr - 5 May	TimeOnline: Pain Management
13 May - 9 Jun	TimeOnline: Wildlife
20 May - 16 Jun	TimeOnline: Animal Welfare
3 Jun - 30 Jun	TimeOnline: Rabbits & Rodents
15 Jul - 11 Aug	TimeOnline: Wildlife (Students)
5 Aug - 1 Sep	TimeOnline: Respiratory Physiology
26 Aug - 22 Sep	TimeOnline: Small Animal Behaviour
2 Sept - 29 Sep	TimeOnline: Marine Wildlife (Students)
28 Oct - 24 Nov	TimeOnline: Anaesthetic Complications
4 Nov - 1 Dec	TimeOnline: Avian

Listed dates are subject to change. Refer to **www.cve.edu.au**, for any updates.

CVE 2013 SHORT COURSES

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

Not sure about our short courses? Our **conferences** and **workshops** offer highly intensive learning providing you with a large hit of CPD points in a short time. Our 1-2 day **seminars** are a practical way to receive a thorough update or refresher. **TimeOnline courses** are delivered wholly online, giving you the flexibility to study when and where you wish and complete your course at your own pace.

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

Visit our website (www.cve.edu.au) to find out more about our programs or you can register your interest by emailing us at **cve.events@sydney.edu.au**.

NEW CVE MEMBER BENEFIT

Small Animal Article Summaries

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Thank you to all contributors

… and more C&T articles and Perspectives needed
Thanks to every author who contributed articles or comments to the *Control & Therapy Series* (C&T). Without your generosity the Series would cease to exist. If you have treated a Large Animal, Reptile or any Wildlife lately, please write up the case and send it in. We aim to keep the Series broad and interesting.

Winners

Major Prize

Entitling the recipient to one year's free membership of the CVE
- **Philip Lindsay:** A case of canine symmetrical lupoid onychodystrophy

CVE Publication Prize Winners

- **Heather Shortridge & Tracey Gowen:** Treatment of penile lesions in bulls
- **Aine Seavers:** Update on C&T 4984: Frozen backs and clinic freezers
- **Heather Shortridge:** 'Ruby' the Lazarus dog or 'When should I give up on ventilation?'
- **Aine Seavers:** Reply to C&T No. 5270 Flea Treatments: Frontline® resistance
- **Jodi Vermaas:** Perspective No. 95 Anxiety in Small Animals, Case 2.

Winner of Best Film Clip
Mimi Dona for the clip demonstrating restraint of:-
- Small Mammals (Bandicoots, Native Rodents and Small carnivorous marsupials)

All CVE Members who receive the print copy of the C&T Series are entitled to the e-book version

The e-book is the perfect complement to your print version.

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Look for these symbols

To be emailed the link to the e-book you must provide CVE with your current email address

Contact cve.membership@sydney.edu.au or call Jacqui Kennedy and receive your Login and Password details. Then visit www.cve.edu.au/candtebook which allows you access to this current issue in e-book format and the 4 prior issues from 2012.

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

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

'…not the academic correctitudes, not the theoretical niceties, not the super correct platitudes that have passed the panel of review… not what he/she should have done, BUT WHAT HE/SHE DID, right or wrong, the full detail, revealing the actual 'blood and dung and guts' of real practice as it happened, when tired, at night, in the rain in the paddock, poor lighting, no other vet to help.'

WINNER OF BEST FILM CLIP

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

Part 4: Wildlife Flashcard Series

Mammals

C&T No. 5279

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

Mimi Dona

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

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

Small Mammals (Bandicoots, Native Rodents and Small carnivorous marsupials)

Go to www.cve.edu.au/candt2012 to view 12 film clips featuring restraint of Amphibians, Birds, Turtles, Lizard & Snakes – available to all.

Part 4.1

SMALL MAMMALS (BANDICOOTS, NATIVE RODENTS AND SMALL CARNIVOROUS MARSUPIALS)

* This flash card does not include large carnivorous marsupials (Tasmanian Devils, Quolls) due to specific expertise being required in care and handling.

Be aware:-

- They are capable of defending themselves using their teeth and claws.
- Most species are solitary, nocturnal and terrestrial.
- Most species have strong home ranges – essential to obtain accurate details of rescue location.
- Rodents are placental mammals and give birth to developed young (and do not have a pouch).
- Bandicoots and Small Carnivorous Marsupials (e.g. Phascogale, Antechinus) – always check females for pouch young. The male scrotum of a marsupial is distinctly pendulous.
- Bandicoots can slough or deglove their tails; if you cannot identify do not tail as a handling technique.
- Rodents can be mistaken as a non-native/feral species (particularly young); native rats are very important to the ecosystem. Seek advice or refer to identification charts for assistance to accurately identify.
- Bandicoots use their nose to detect food; it is very sensitive and they must be housed in a way to prevent damage.
- Marsupial young are born very under-developed and until the stage of development that we are able to successfully hand rear young joeys, they are considered to be 'unviable'. Consult a trained/experienced person for assistance with identification and confirmation on viability.
- Small mammals don't get tick paralysis.

Handling

- To pick up adult Bandicoots grip around the back of the head/skull and hold the hind legs with your other hand. Use a towel to protect from being bitten or scratched.
- Or make a 'V' with your fingers around the head and shoulders and hold the hind legs with your other hand. Do not restrain by the tail.
- To pick up adult Rodents or Small Adult Carnivorous Marsupial use the 'V' technique above. If you are confident it is not a bandicoot, hold with the other hand around the base of the tail to prevent it from scratching with its hind feet. Use a cotton pouch or small pillow case to restrain its body if you are not confident, as they can be difficult to restrain and exceptionally quick to bite.
- Juveniles can be cupped in the palm of your hand if not biting.

Figure 1. Marsupials may have pouch young so be careful when

restraining

Figure 3. Juveniles can be cupped in your hand if not biting.

Figure 2. The 'V' technique; bandicoots should never be restrained using their tail.

Housing the sick Small Mammal

- Preferred enclosure temperature is between 28˚ and 32˚ Celsius.
- Bandicoots: Small carry cage (top opening) lined with towels – do not use wire cages OR
- Rodents: Plastic aquarium or smooth sided ventilated tub lined with towels.
- Alternatively a clean rubbish bin with holes drilled in the lid works very well.
- Always provide a hide-box, towel or toilet roll.
- Make sure the lid is secure as they are very good at escaping and jumping.
- Place orphans in a cotton pouch. Heat must be given to orphans - just furred/furred 28˚ - 32˚ Celsius and unfurred 32˚ Celsius; ideally they should be housed in a Vetario® or Humidicrib and monitored with an indoor/outdoor thermometer or alternatively place in a plastic aquarium with a sealed/ventilated lid.

Figure 4. Small mammals (e.g. antechinus) should be offered shallow dishes to prevent the risk of drowning.

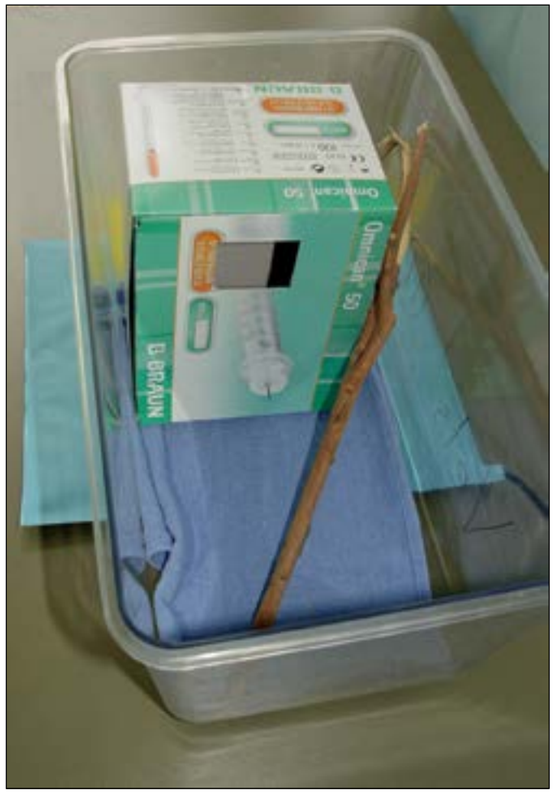

Figure 5. A makeshift hollow created with a syringe box makes an excellent hide to reduce stress.

Emergency diet

- Only offer food once rehydrated.
- Small mammals have a high calorie requirement and need to be fed frequently; they may require fluids if not eating.
- Offer adults a variety of live insects, fruit, vegetables and soaked dog kibble.
- Rodents and bandicoots can be offered birdseed.
- Orphans can be given water and glucodin initially for first 2 feeds, then suitable milk replacer (Divetelact®, Biolac® M 100 or Wombaroo® Kangaroo Milk Replacer >0.7. This can be given either via a 1mL syringe with catheter tip, bottle and appropriate sized teat or in a bowl if lapping.

Assessment under gaseous anaesthetic

Use an anaesthetic mask at 5% induction; it can take 1 – 2 minutes. Or, if difficulties handling, place in an anaesthetic box which will take an additional few minutes. Maintain using a mask on Isoflurane® at 1.5 – 2% with an oxygen flow rate of 1 L/min.

Anaesthetic agents

Alfaxan® CD RTU 3 mg/kg – (I/M). Due to the difficulty with intubation of small mammals and intermittent positive-pressure ventilation in case of apnoea, gaseous anaesthesia is recommended.

Intubation

Cuffed endotracheal tube or catheter tip; intubation is very difficult and most species will be too small. Insert the endotracheal tube with the aid of an anaesthetic spray and tie in with shoelaces.

Recovery

Use a bair hugger® or heat mat and room temperature to maintain the patient's core body temperature throughout the procedure due to small size and rapid heat loss. Vetario® or Humidicribs are ideal for post-operative recovery.

Fluid Therapy

It is important to remember to warm the fluids being administered. Using 0.9% sodium chloride, dose the patient at 5% of its bodyweight. Fluid therapy can be administered subcutaneously or by using standard I/V infusion rates. Syringe pumps are ideal to use in small mammals if available.

Preferred routes for drug administration (similar to domestic pet mammals)

- Subcutaneous – administered in loose skin dorsal neck/ shoulders and cranial thigh area.
- Oral – given via a syringe.
- Intramuscular – dorsal lumbar muscles, cranial and caudal thigh and gluteals.
- Intravenous – jugular, saphenous or cephalic veins, difficult and only suitable for larger animals.

Euthanasia methods

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

- If administering by intracardiac or intraperitoneal, the small mammal must be anaesthetised first. Pinkies don't always reach appropriate levels of anaesthesia in a gas chamber; Alfaxan® I/M is recommended.

Figure 6. There are many different dichotomous keys and books to assist with the correct identification of native or introduced species; the tail in native rodents is shorter than its body length.

Has anyone seen anything like this before?

C&T No. 5280

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Figure 1. Bengal kittens

I have just recently seen a curious case – see Figure 1 above.

A Bengal queen had 4 kittens over 4 hours, then 12 hours later passed these 2 kittens which are healthy and feeding well. The back lesions are raised subcutaneous tissue which appear unattached to the underlying connective tissue or spine. Without further investigation (Owner just brought in for some topical cream to apply), I suspect it probably isn't a spina bifida-type problem.

I told the owner it may have been an intrauterine bacterial infection with secondary dermal erosion, but cannot be sure at this stage.

I am very interested to hear if CVE Members/Readers have seen or heard of any cases like this?

Send in your replies to:

Elisabeth Churchward @ elisabeth.churchward@sydney.edu.au or Fax: (02) 9351 7968 for publication in the upcoming June 2013 Issue 271. The author of the best reply will receive a CVE proceedings of his/her choice. Go to: **www.vetbookshop.com**

Grazing Cows

C&T No. 5281

Rick Atwell (Retired Professor)
BVSc PhD FACVSc
M. 0409 065 255

Having not been in large animal practice for decades this may be old hat but I was fascinated by what grass can do to fetlocks as animals feed in well grassed paddocks.

Horses on 'green panic' (*P. maximum*) can get bleeding cranial fetlock wounds as their legs thrust forward in the fresh green growth (small barbs exist on the edge of the grass). With 'Gatton Panic' and cattle, the lesions are less severe (different species (skin) and grass) – more of a bald fetlock syndrome occurring both in dark and light coloured coats. They seem to cause little irritation except for residual fetlock baldness (and its other possible cause(s) to those unaware). It seems to be more erosive on the fore legs* than the hind. (As the leg thrusts forward is it the fetlock that has the most vegetation contact as the animal moves through a thickly grassed paddock?)

Observations were of a well grazed paddock, containing 137 similarly aged and weighted animals (all castrated) of multiple genetic background with well grown Gatton Panic in March 2012 in the Augathella area, on a verified (EU & USA) organic property.

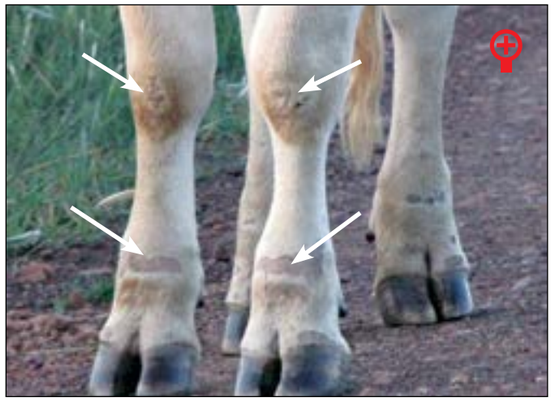

Figure 1.

Figure 2.

WINNERS

Treatment of penile lesions in bulls

C&T No. 5282

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Earlier in the year we made a treatment plan for some bulls for Tracey Gowen, a UQ Vet Student (class of 2013). Tracey took some great photos, which I thought might be good for the *C&T Series*. The bulls had penile injuries, which were treated with slinging and the application of prednoderm.

Figure 1. Distal penile injury of a bull.

This photo shows one of the penile lesions. Tracey cleaned the lesion with iodine, then applied prednoderm, and made slings for the bulls to protect the penises. The bulls were also treated with tolfedine. The bulls also received one shot of long acting penicillin.

Figure 2. This picture shows the sling, made with a hessian sack and tied over the bull's back. As you can see, it is no impediment to the bull moving around.

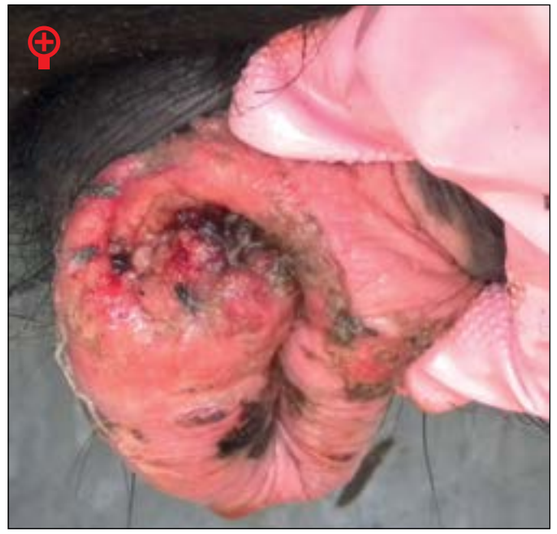

Figure 3. The above picture shows the lesion looking much better on Day 4. At this stage the swelling had resolved enough that the penis could return to the sheath (see below) so the slinging was no longer required.

Figure 4. This treatment resulted in a very rapid improvement in the penile injuries of these bulls. Tracey did a great job with carrying out the treatment and creating an effective sling to protect the penis during healing, and I am grateful to her for allowing me to share these photos with C&T readers.

Back to TOC page

WINNER

Update on C&T 4984: Frozen backs and clinic freezers (March 2009)

C&T No. 5283

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Aine has updated her original C&T which aims to reduce the eventual size and awkwardness of the body in the frozen body bag.

- Place all pets, but especially the larger dogs, in the bag as if in a kneeling position or preferably lying on its back. That way the limbs naturally fold down onto the trunk/torso.
- Fold or wrap the bag over tightly and cellotape or string tie tightly so you end up with a long smooth cylindrical shape. (Don't use computer ties anywhere – they crack off when frozen and become lacerating points of OH&S concern for staff).
  - o When the body then tries to expand in rigor mortis or when freezing, the tucked and wrapped legs don't stick out and get jammed into the sides of the freezer – as latter makes removal harder.
  - o For giant dogs – tuck the neck between the front legs as well to reduce overall length.

The steps outlined above dramatically increase the storage capacity of the freezer as it reduces each body's surface area. Figure 4 shows lots of space left in even the smallest freezer, despite holding 4 big bodies.

Other advantages of this method include:-

- Easier handling as the long rectangular shape allows more people to get hold of the one body properly.
- A less disturbing visual if the owner changes their mind and decides to come back and get the body for a home burial.
- More bodies can fit in the Maroon Body Bins (big garbage bin sized bags provided by the companies which collect and remove the bodies from clinics).
- Easier handling of the corpse at the crematorium as the bodies fit much easier into crematorium ovens which often can be too narrow for very wide amorphous blobs of dead bodies.

*Additionally, a **large fitted bed sheet dropped on the bottom of the freezer assists** when you have to lift a big body from the depth of the freezer, you now have a sling or fulcrum that allows several people to get a hold of and pull the body up with, and leaves the humans in an upright lifting position not hanging in over the freezer sides.*

Figure1. Dog lying on its back or side. Fold line close to the body.

Figure 2. Fold bag over tightly.

Figure 3. Cellotaped into tight cylindral shape.

Figure 4. Labrador/Rottweiler sized bodies in a small freezer. The sheet assists in levering large bodies up and out.

Figure 5. For very large dogs, place dog in centre of body bag and create a handle at each end to give the staff something to grab onto at either end when later needing to lift the frozen body out of the freezers etc... You can slip a sheet under the bag as well to help lever the larger body out of the chest freezer.

MAJOR WINNER

A case of canine symmetrical lupoid onychodystrophy

C&T No. 5284

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Phil pictured with 'Tom', 'Jack' and 'Pippi'.

Phil Lindsay started out as a vet at Melbourne University after graduating from Massey University in New Zealand in 1988. He completed a MVS (Small Animal Medicine) at Melbourne before completing a stint in large animal practice in Gippsland and then moved to work with the dedicated and fun team at Castle Hill Veterinary Hospital in NSW. In 1993 he gained Membership of the ACVSc in Canine Medicine and spent a short period in Small Animal Medicine at Murdoch University. In 1995 he returned to his native New Zealand with his partner and fellow veterinarian, Cathy Opie, and together they established their own practice, The Pet Doctors, on the North Shore of Auckland. Phil, Cathy and their two sons, Douglas and Alexander, live in Devonport with their cats Tom and Jack, and the family Dobermann, Pippi.

In November 2008 a 6-year-old, speyed female black Labrador retriever presented with left forelimb lameness. Pain was localised to the distal part of the 4th digit. The claw of that digit was distinctly abnormal in that it was disfigured (onychodystrophy) with many small splits originating from the distal surface of the wall of the claw and causing 'lamination' where thin pieces of nail broke away laterally (onychorrhexis). The surface of the claw was soft with a powdery residue covering the surface. The claw was separating from the nail bed (onycholysis) and a small amount of pus could be seen at the base of the claw. The bitch had low grade pyrexia, 39.1℃. The claw was manually removed under sedation and the nail bed cleaned and bandaged. Seven day courses of amoxicillin/clavulonate (Vetamox, Ethical Agents NZ) and meloxicam (Metacam, Boehringer Ingelheim) were dispensed.

The affected claw appeared to be healing as expected but I was surprised when the bitch re-presented with similar signs but this time attributable to the claw of the 2nd digit on the same leg.

I had a short 'What the…?" moment and, since the 4th digit had responded so well, I repeated the same therapy and gained good results by the following week for the 2nd digit. Things appeared to be progressing well until just prior to Christmas 2008 when digits 3 & 5 followed the same fate as 4 & 2.

I treated the 2 newly affected claws as described above but I submitted the amputated claws to our diagnostic laboratory for fungal examination. To my diagnostic delight the lab reported that they could see fungal hyphae on both claws and later grew *Aspergillus fumigatus* and a light growth of Malassezia yeast. I remarked to the lab that I thought *Aspergillus* was unusual but they reported that they sporadically identify *Aspergillus onychomycosis*. I was perplexed regarding the source of *Aspergillus* in our suburban Auckland environment until the dog's owner explained that their neighbour was a keen gardener and heavy rain in October 2008 had swept a large quantity of straw garden mulch into the back yard where the dog was kept. The rain persisted for some weeks and, being an 'outside dog,' her feet were constantly wet.

Antifungal treatment was initiated with Griseofulvin* (Griseovet 120mg tablets Virbac 25mg/kg bid) and twice weekly Miconazole nitrate washes (Malaseb 20g/L Dermcare) in late December 2008 and continued until April 2009. Haematology was performed monthly to check for blood dyscrasias associated with griseofulvin therapy. During this time the claws on the affected digits began to show evidence of regrowth although, as is expected with regenerating claws, their shape was slightly twisted and gnarled.

I was surprised that only the claws on the left fore paw had been affected but in late January the bitch began to show progressive signs of onychodystrophy, onychorrhexis and onycholysis which eventually involved all 4 feet, and all the claws were shed. The feet were painful and, presumably from the trauma of the onycholytic claws 'digging into' the nail beds, became infected, necessitating short courses of amoxicillin/clavulonate to control the secondary bacterial infection. This progression of signs appeared to agree with the references I had read regarding treatment of onychomycosis and the expectation that these patients can take 6 months or so before their disease is controlled. Therefore I patiently waited while my treatment 'caught up' with the infection and watched the new claws slowly re-grow. I was pleased that, even though the new claws were misshapen, they no longer had the soft powdery residue that I had noted the previous December.

By April my patience began to wane as claw regrowth was extremely slow and I did not have new, shiny hard claw growing through to replace the misshapen early growth. I began to wonder if an underlying disease was present which had ▶

**Editor's Note: Griseofulvin has no effect on Aspergillus spp. Sorry! It acts on the stratum corneum to make it impervious to dermatophytes e.g.* Microsporium canis.

allowed such an unusual pathogen to colonise the claws. I elected to screen for underlying disease, specifically hypothyroidism and Cushing's syndrome, but the haematology and biochemistry panels were normal and the dog's serum T4 was 34nmol/L (reference 20 – 40nmol/L). I contacted the laboratory to check if there were any other samples I could send to further define an underlying disease and they suggested that I could amputate and submit a representative digit for histopathology.

Rather than amputate, my client and I elected to proceed to a second antifungal treatment and Itraconazole (Sporonox 3.5mg/kg bid) was commenced in April 2009 for 10 weeks. To our delight the claws showed another level of improvement and within 3 weeks we started to see harder, smoother walled claws growing from the nail bed. By August the claws were much improved, and no longer being shed. Considering the chronicity of the disease I didn't expect the claws to be totally normal. The claws were generally weak with onychorrhexis (splitting) especially toward the distal part of the claw.

The dog's claws remained normal for approximately 1 month and at a scheduled revisit we noticed that 1 of the claws on the left hind foot began showing signs of onychomalacia (softening), onychorrhexis (splitting and breaking) and onychodystrophy (malformation). Since it was August, we thought we were dealing with a recurrence of the fungal infection. However, rather than repeat antifungal treatment, I elected to submit the claw for mycology because I thought it would be pretty easy for the lab to see the characteristic form of *Aspergillus* on potassium hydroxide preparation if it were there. The lab was unable to demonstrate any fungal elements and my patient began to show signs of licking her feet and became reluctant to exercise which I attributed to onychalgia (claw pain). The nail beds were inflamed and purulent exudate (paronychia) developed in that area. I treated her with meloxicam and amoxicillin/clavulonate and her signs improved but an increasing number of her claws became brittle and cracked, and she resented me examining her feet. The fungal culture returned and no pathogenic species were detected.

By this time I was happy that I had cleared any fungal infection from the claws but I was perplexed that the claws were not now growing normally and the dog's feet were still noticeably painful. I contacted Dr Richard Malik and he suggested that I may be dealing with a case of symmetrical lupoid onychodystrophy (SLOD) and kindly introduced and referred me to Dr Aine Seavers. Coincidentally, Aine had published an article on SLOD in *The Veterinarian* (August 2009) at the time.

Aine worked through the case and explained to me that nobody anywhere on the planet has developed a hard and fast treatment protocol for this disease. So, using her article from *The Veterinarian* as a template, we started with a basic treatment regime that we could expand later, depending on the response. The protocol started in September 2009 included:-

1. Anaesthetising the dog to have the fur clipped on the toes to expose the ungula to facilitate skin contact with Cortavance (Virbac, NZ) spray. Any remaining damaged claws were removed and all claws were filed smooth.
2. Megaderm (Virbac, NZ): The dosage was incrementally increased from 8mL x1 sachets with food daily for 7 days to 2 8mL sachets the following week etc until we were giving 24mL per day to our 28kg patient, a process that took about 1 month to achieve. Aine suggested that we take this approach in order to reduce the risk of triggering a bout of pancreatitis with such a high level of oil in the dog's diet. At that time only sachets were available in NZ but we are indebted to Mark Kelman (Virbac Australia) who kindly supplied several 1L bottles directly from Sydney for our case. Ian Denby (Virbac NZ) also helped us to secure a supply of Megaderm sachets for our 'heavy user' of this product.
3. Cortavance (Virbac, NZ) spray applied once daily to all feet.
4. z/d® diet (Hill's Pet Nutrition, NZ) Trial in case of food allergy.

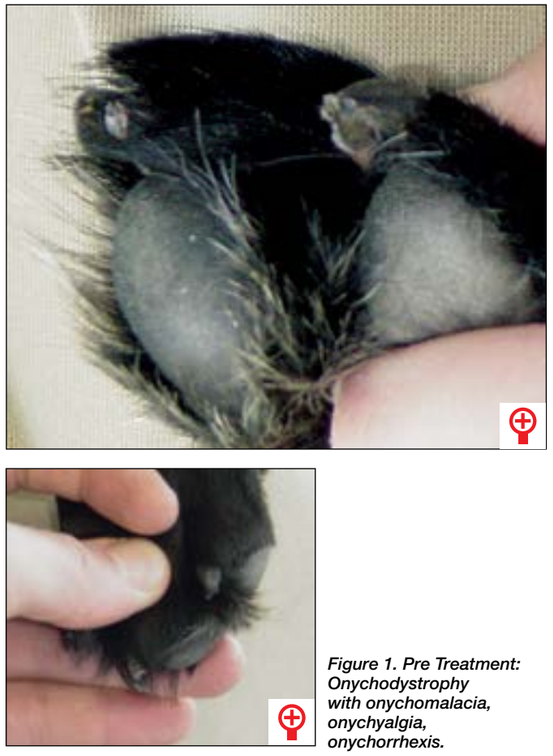


Figure 1. Pre Treatment: Onychodystrophy with onychomalacia, onychyalgia, onychorrhexis.

At the time of initiating the treatment, all claws on all 4 feet were affected to various degrees with signs typical of SLOD: Onychodystrophy with onychomalacia, onychyalgia, onychorrhexis and onychomadesis (shedding of claws). We then monitored with monthly revisits.

As expected with such a chronic disease it took approximately 2 months before any tangible improvement could be appreciated. The initial response was not in fact physical but behavioural. The owner reported that the bitch was no longer licking her feet, was more playful and running about more freely so we attributed this improvement to a reduction in pain from the onychodystrophic digits.

During the 3rd and 4th months of treatment the claws showed signs of physical improvement. To my surprise, the claws did not improve symmetrically or simultaneously, with some claws on the same foot showing massive improvement and yet the immediately adjacent claws still showing advanced signs of disease. Our impression was that generally the claws were getting stronger and developing a more normal, smooth surface for the first half of their length and beyond that they were weak, fracturing and disorganised and essentially self destructing. Every 2-3 weeks the bitch would develop severe splitting (onychorrhexis) of the stratum corneum (wall of the claw) or avulsion of 1 or 2 claws which required them to be removed and secondary infection of germinal tissue of the 'quick' or nail bed to be treated.

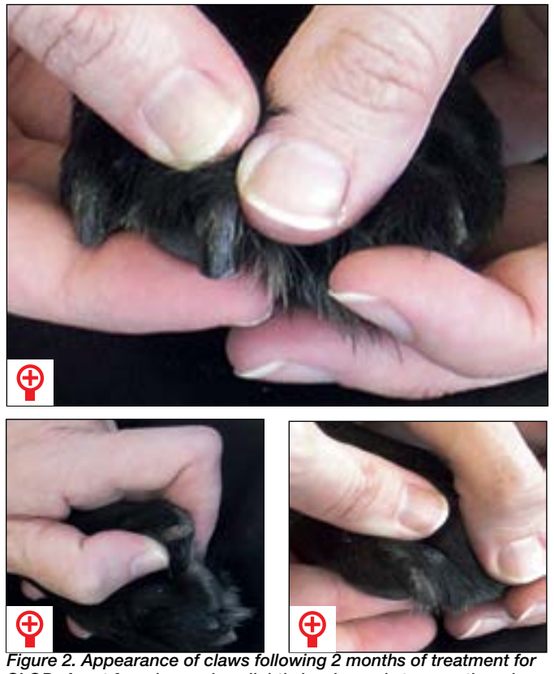
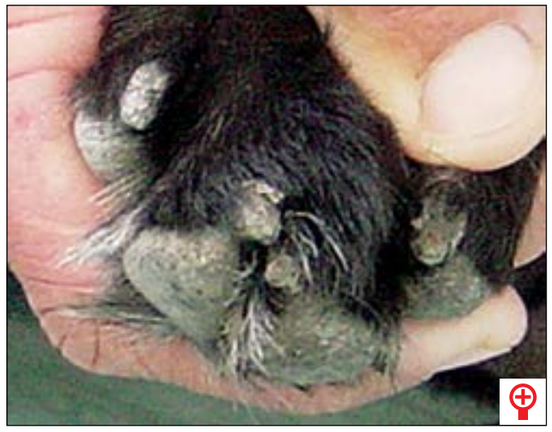


Figure 2. Appearance of claws following 2 months of treatment for SLOD: Apart from becoming slightly harder and stronger there is still obvious signs of SLOD but the most noticeable change was that the bitch's feet were much less painful.



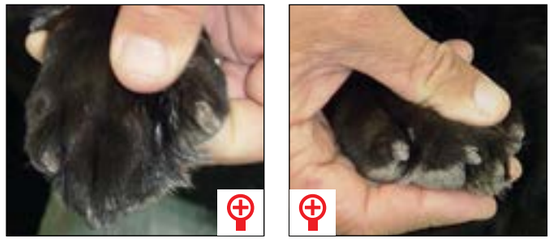


Figure 3. Following 3 months of treatment for SLOD: The feet are more comfortable and the claws were getting stronger and developing a more normal, smooth surface for the first half of their length but beyond that they were very weak, fracturing and disorganised.

However, since tangible improvement was evident we persisted with the same treatment for a further 4 weeks but by the 5th month of therapy the bitch showed no further improvement. I discussed the bitch's signs with Aine Seavers and some colleagues at the (outstandingly informative) Centre for Veterinary Education Dermatology Course in Sydney (February 2010) and received a wide variety of suggestions as to how to progress with this case. I confess openly that I was in the corticosteroid camp before talking to Aine, but her arguments were compelling for the avoidance of steroids in these cases. We instead elected to utilise the immune modulating effects of doxycycline (Vibravet tablets, Pfizer NZ) and placed our patient on 5mg/kg bid for 3 weeks, in addition to the existing Cortavance/Megaderm/ z/d® Hill's protocol.

Figure 4. Following 5 months of treatment for SLOD: We persisted with the treatment but by the 5th month of therapy the bitch showed no further improvement.

At the 6 month revisit the owner had noticed the bitch had achieved another level of improvement in her pain, reporting that she was now more confident and was playing freely with other dogs at the park, whereas previously she had avoided vigorous play. The claws were now more uniformly improved and were no longer fracturing longitudinally. None needed any trimming or removal. The walls of the claws were harder and stronger. The distal halves of the claws had a more normal, pointed appearance although they were shorter than expected suggesting they were wearing more quickly. The surfaces of the claw walls were slightly pitted and dull. The response was very encouraging and so we elected to maintain the doxycycline/Megaderm/Cortavance/z/d® Hill's regime and revisit a month later. ▶

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Back to TOC page

Figure 5. Following 8 months of treatment for SLOD: The structure of the claws was now improved for the first 2/3's of the length of the wall and the distal 1/3's of the walls of the claws were still slightly pitted and dull but much stronger and more normal in appearance. Her distal limbs were more slender and she no longer resented examination of her feet.

My outstandingly diligent clients showed at last that they were in fact human and missed the next revisit but came in a month after that. I was surprised at the now 8 month revisit that not only had the feet improved but the bitch's distal limbs and feet appeared more slender. In retrospect, at previous visits I believe her feet, especially around the interphalangeal joints, had been "puffy." She had no apparent digital pain and her owner said the dog was keen to exercise and no longer licked her feet, as evidenced by the return of black pigmented fur replacing the rusty brown fur around the base of the claws and interphalangeal joints. The structure of the claws was now improved for the first 2/3's of the length of the wall. The distal 1/3's of the walls of the claws were still slightly pitted and dull but nevertheless much stronger and more normal in appearance.

That last revisit was almost 12 months ago at the time of writing this article (insert date) and the owner has maintained the dog on the Hill's z/d®/Cortavance / Megaderm protocol. In the last 4 months the owner reduced the frequency of Cortavance to twice weekly and the Megaderm dosage to 8mL once daily. Early signs of recurrence of SLOD have manifested and it appears the bitch will need more intensive maintenance therapy to control her signs in the future.

I would like to most gratefully acknowledge **Drs Seavers & Malik** for their willing, able and friendly help with this case. This was one of the most protracted medical cases that I have treated and throughout both Dr Seavers & Malik maintained email contact with me and ensured the best possible outcome for this patient. Many thanks also to **Mark Kelman** and **Ian Denby** from Virbac Australia & New Zealand for their ready and enthusiastic assistance in the large quantities of Megaderm that we required to treat this patient. **Dr Nick Cave** from Massey University generously helped me gain a better understanding of current scientific knowledge regarding the effects of unsaturated fatty acid supplementation on the immune system.

Sudden onset of swelling in the right carpus in a Staffordshire Terrier

C&T No. 5285

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Note: Irene now works at the RSPCA, Yagoona.

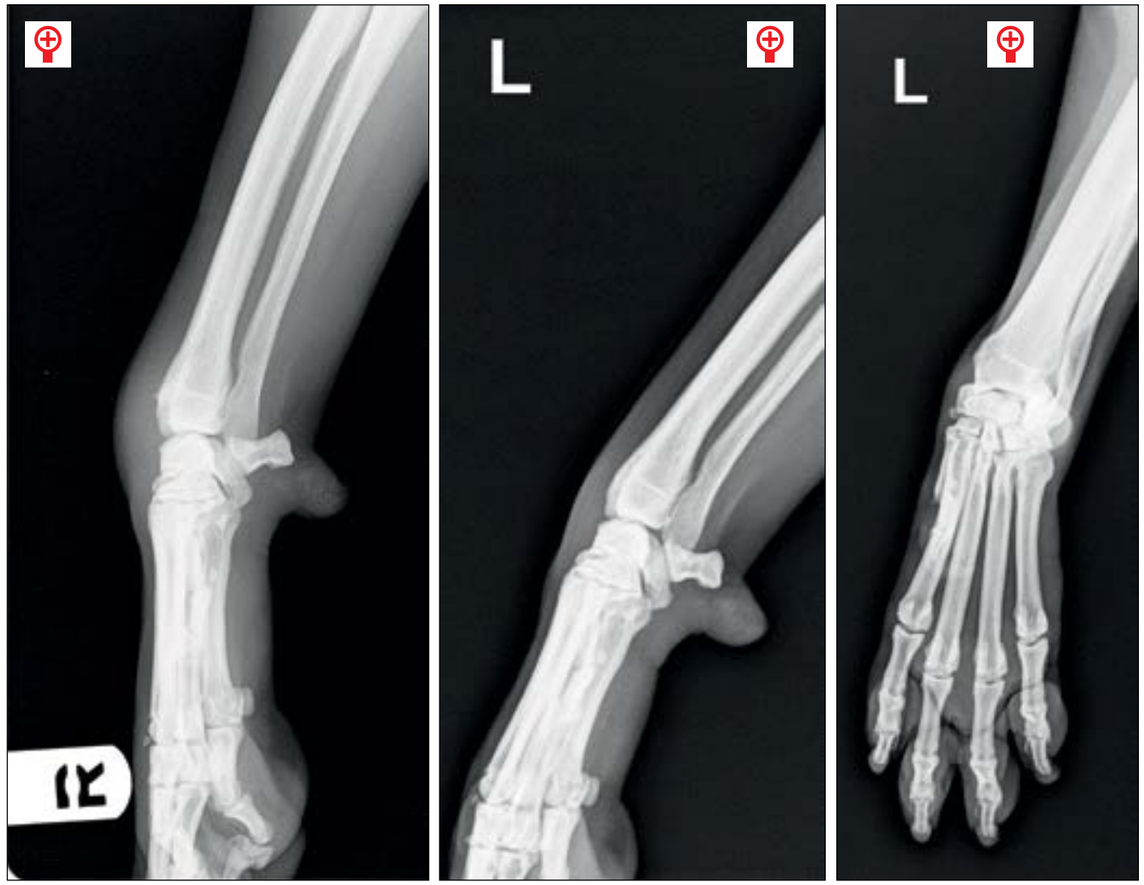
'Carma' is a 4-year-old, male entire, white Staffordshire Terrier cross who presented at Stewart St Veterinary Hospital with a sudden onset of swelling in the **right carpus** with no observable history of lameness. Radiographs revealed a slight mineralisation of the extensor tendon in the area of the distal radius. A differential diagnosis of tendonitis or soft tissue inflammation was made by Yoonmi. Carma was treated with meloxicam (0.1mg/kg SID) for 4 days and amoxycillin-clavulanate (14mg/kg BID) for 10 days and asked to come back for a recheck in 10 days.

Two months later, Carma returned with worsening lameness (grade 1/4) in his **left front** leg and very swollen carpus on physical examination with minimal pain on flexion and extension. The owners felt that Carma may have improved on the 10 days of antibiotics so he was given another course and booked in for repeat radiographs and a further workup in 7 days.

On admission, he was sedated with butorphanol (0.1mg/kg), atropine (0.01mg/kg) and acepromazine (0.02mg/kg) which gave good sedation. Radiographs (lateral, DP and flexed lateral of each carpus and DV, L and R lateral chest) and joint taps were performed. The joint taps were unrewarding. A haematology and serum biochemistry were performed which revealed elevated cholesterol and triglyceride but were otherwise unremarkable. Chest radiographs showed a lack of intrathoracic pathology.

Carpal radiographs are shown in the following images.

Veterinary Imaging Associates were sent the radiographs and kindly provided the following opinion: *'Chronic recurrent extensor tendinopathy with probable tenosynovitis and dystrophic mineralisation' – characteristic of a chronic recurrent injury* ▶

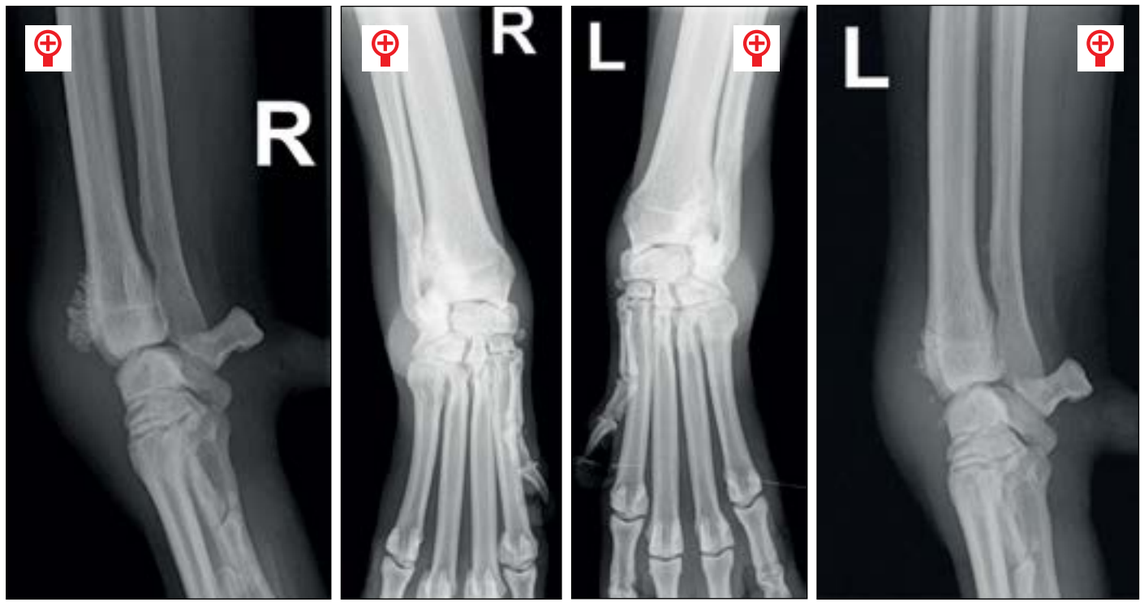


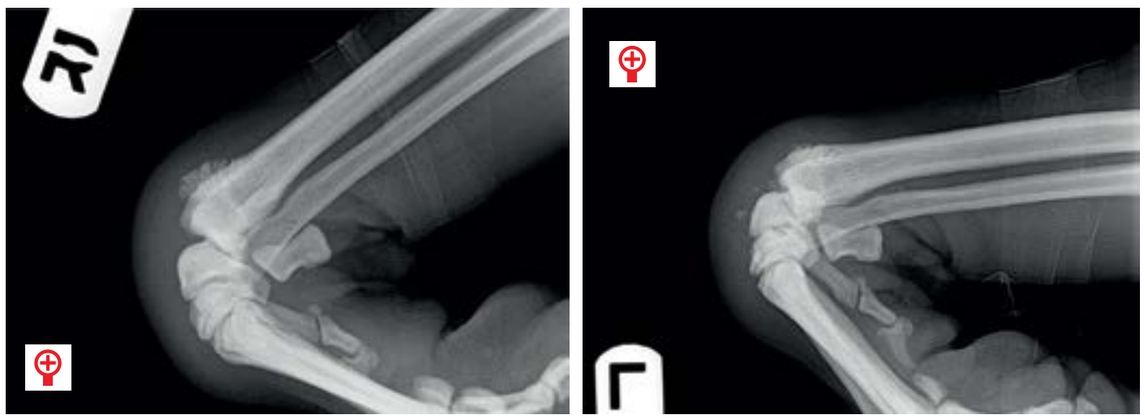
Figures 1 to 3. Initial radiographs of the patient – lateral right and left carpi and dorsopalmar left carpus and manus. Note the significant soft tissue swelling on the dorsal aspect of the right carpus compared to the left carpus. There is a small mineral focus on the dorsal aspect of the distal right radius, just distal to the level of the distal physeal scar.

rather than aggressive change – a consequence of repeated injury and difficult to manage.'

Carma will be managed on a combination of non steroidals or steroids in the future to manage his pain and obviously will be kept as quiet as possible. On further questioning, it was revealed that he was not a dog that jumps off the back of the ute or one who performs excessive strenuous activity.

Postscript: No histology was taken and unfortunately Carma was lost to follow up so the authors are unable to provide further details on the dog's progress and whether or not he remained lame. The diagnosis is therefore still open but the radiographic lesion is interesting enough that CVE believed the article merited publication.





Figures 4-9. Two month follow-up radiographs the carpi – lateral, dorsopalmar and flexed lateral views. The swelling on the dorsal aspect of the carpi is now present bilaterally and accompanied by more significant mineralisation on the dorsal aspect of the right distal radius, with similar but a lesser degree of mineral on the dorsodistal aspect of the left radius. Small mineral foci are also present at the level of the proximal row of carpal bones in the left carpus. Note that the mineral opacities, while irregular are well-defined and there are no areas of destructive or erosive change involving the adjacent bones.

Special thanks to Robert Nicoll from Veterinary Imaging Associates for his assistance with this article.

WINNER

A novel approach to managing axillary/collar wounds

C&T No. 5286

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I am sure many clinicians have been haunted by these darned non-healing collar wounds in a feline patient's axillary region. I have managed a handful and have certainly been asked by colleagues about many of these failed wound repairs. The usual story unfolds something along the lines of, 'after removing the sutures – the wound simply fell apart' – sound familiar? There are many reports illustrating repair techniques that improve success rates in repairing these notorious wounds; from reconstruction of the elbow skin flap, thoracic advancement skin flaps and even using omentum to provide a healing scaffold in these wounds. But even with the utmost surgical skill and attention to detail, some of these wounds still fail.

We were recently presented with a case that had already had several failed surgical attempts at repair. Not wanting to add to this record, I contacted a colleague Dr Rhett Marshall at The Cat Clinic in Brisbane for any pearls of wisdom he had in managing them. I am reliably informed that he has successfully repaired more than 50 of these cases at their practice. His preferred approach is to completely excise granulation tissue and wound edges and close wound with a rotating cranial epigastric advancement flap. However, the intriguing thing was the post-operative management, where he applies a large compression bandage/body wrap into axillary-thoracic region and cage rests for 7 days. Since augmenting his surgical management with these wraps, his first round success is almost perfect, irrespective of the surgical technique employed.

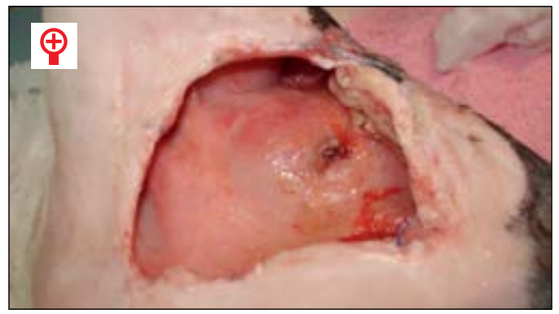
An absorbent cotton wool type dressing is placed into the axilla, wrapping around the body several times, both in front of and behind the affected limb. The aim is to build up a thick soft mass of dressing in the axilla that sits right up into the axilla. The region is then carefully compressed with Elastoplast around the entire dressing and thorax, criss-crossing between and behind forearms. Additional cotton wool is easily pushed up into the bandage under the axilla region each day to ensure wound stays dry and compressed. It is also ideal that the patient be confined in a cage again to limit wound movement and maximise wound healing. The Body bandage is kept in place for 7 days and the sutures removed at day 14-21. NB: most cats DO NOT like being wrapped up like this, so special attention should be given to nursing care, to ensure effective feeding and toileting.

There are likely several factors in these wounds not healing well that the bandage may help address. The axilla is a region of high movement and skin tension – a light compression bandage certainly would aid restricting movement of the forearm and the axillary skin. The compression component will help reduce dead space, a real problem with some of extensive reconstructive techniques often employed. There have been several studies that have also pointed to cats having a slower rate of wound healing in cats compared to dogs. One publication found that sutured wounds at day 7 were only half as strong in cats as in dogs. It has therefore been suggested by some surgeons that wounds such as these should have a delayed suture removal, at least 14 days, but up to 21 days to allow maximal wound strength to develop.

Whether the wound is simply debrided and closed or extensive excision of granulation tissues and advancement flap reconstruction is required, the addition of an axillary-thoracic compressive wrap, exercise restriction and delayed suture removal may be a very simple and effective addition to improving success rates with these problematic wounds. Needless to say, we too have a 100% surgical record in repairing these chronic wounds.

Figures 1-6 (cases 1&2) are from the QLD clinic courtesy of Rhett Marshall – Both are long standing wounds that had had several failed repairs, one had developed extreme granulation tissue and the other a massive non-healing open wound. Rhett performed extensive debridements and then used rotating cranial epigastric advancement skin flaps to close each deficit. Both cases had extensive compression bandages placed and healed without fail.

Figures 7-9 (case 3) are from the VIC clinic courtesy of Richard Gowan – This case had 2 previous failed surgical repairs (including an elbow skin flap repair) with other practices. This wound was very minimally debrided and primarily closed with a body wrap applied for 7 days, sutures removed at 20 days – all fine. ▶



Figures 1-2.

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Back to TOC page

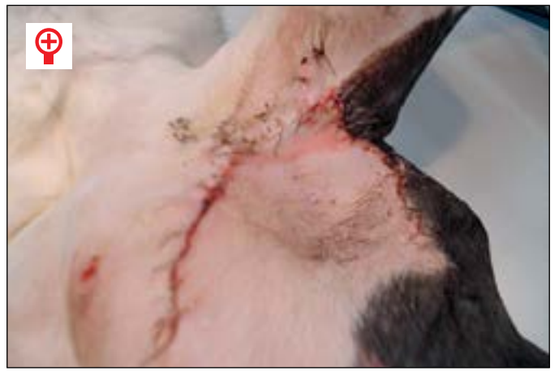

Figure 3. Figures 1&2 (overleaf) & 3 above show Case 1 before, post-op and healed.

Figure 4. Case 2 before surgery.

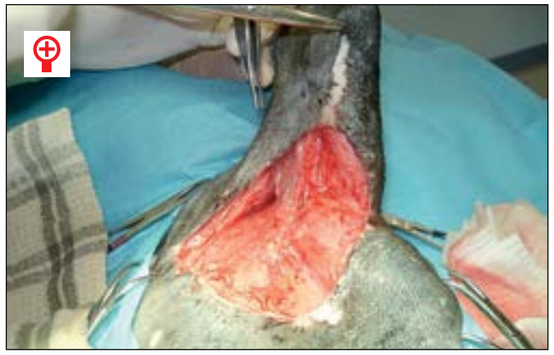

Figure 5. Case 2 during surgery.

Figure 6. Case 2 shows the body wrap post-op.

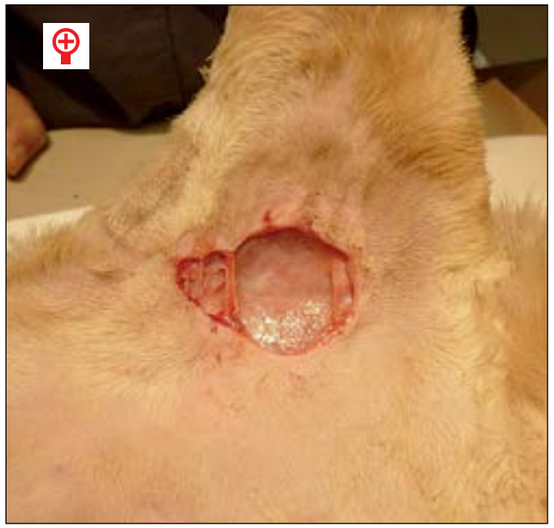

Figure 7. Case 4 shows intro.

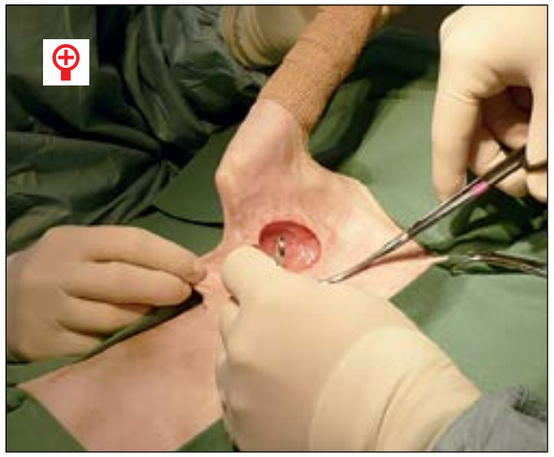

Figure 8. Case 4 pre-op.

Figure 9. Case 4 shows wrap.

Re-published here courtesy of Vetnostics, North Ryde

ADRENALS: What you won't find in a textbook

C&T No. 5287

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Part 4B: UCCR and cALP

As Part 4 of this series looks at diagnostic tests, we can't escape some statistics. So, some very simplistic explanations relative to hyperA are as follows:-

- Sensitivity: the likelihood that the test will detect hyperA
- Specificity: the chance that a positive test is truly hyperA

Then, there are predictive values which take into account the prevalence or likelihood of a disease in addition to sensitivity and specificity.

- Positive predictive value (PPV): the chance of a positive result being indicative of hyperA in dogs with signs of hyperA (e.g. Can we confidently diagnose hyperA when we get a 'positive' result?)
- Negative predictive value (NPV): the likelihood that a negative results eliminates the possibility of hyperA in dogs with signs of hyperA (e.g. Can we rule it out with a negative result?)

Urine corticoid: creatinine ratio (UCCR)

It is commonly stated that this test is highly sensitive but poorly specific, that is, the test picks up most dogs with hyperA but is also positive in lots of other disease states. It is widely advocated as a screening test to RULE OUT hyperA but should we be doing that?

The literature confirms that the test has good sensitivity and poor specificity[1-4] (as low as 21%)[1]. In addition, one study had a positive predictive value of only 3%[2] (i.e. only 3% chance that a dog with increased UCCR actually had hyperA!) so we certainly can't use UCCR for diagnosis.

What about the negative predictive value: can we use the test as a 'rule-out'?

NPVs range from 0.96[1] (Smiley and Peterson) to 0.99[2] (Soffner and Reusch) so it would seem that a normal UCCR should rule out hyperA. However, these papers were all written in the early to mid 1990s and not in Australia. In Jody Braddock's Master's Thesis (2002), **the sensitivity of the UCCR was only 66%**, as 13 of 38 animals with rigorously confirmed hyperA had a UCCR ≤15.[6] It is unclear whether this was because the cases were being picked up earlier (in the 2000s compared to the 1990s) or whether the dog population in Braddock's thesis is different from that in the other studies.

This statistic would confirm my impression at Vetnostics that we do seem to see 'false negatives' at a much higher rate than the 2-4% suggested in the early studies. Thus this test **cannot be used as a rule-out test.**

Given that UCCR cannot be used to diagnose hyperA (poorly specific) and cannot be used as a rule-out either, there is little point in performing this diagnostic test. UCCr does have a role in monitoring treatment with trilostane (to be addressed in a later newsletter).

Corticosteroid-induced alkaline phosphatase (c-ALP)

Increased serum ALP, the most common routine laboratory abnormality in hyperA is due mainly to the induction of a specific ALP isoenzyme by glucocorticoids. The corticosteroid-induced isoenzyme of ALP can be measured by electrophoretic separation, heat inactivation or more usually in commercial laboratories, by levamisole-inhibition. The levamisole inhibition explains why c-ALP is sometimes referred to as l-ALP but this terminology can be confusing as sometimes l-ALP is used to describe the liver isoenzyme; it is also referred to as CAP (corticosteroid-induced ALP) or SIAP (steroid-induced alkaline phosphatase).

The sensitivity of c-ALP has been reported to be 0.81-0.95.[7-9] Specificity is poor (0.18-0.44)[7-9] and PPV in one study was as low as 21.4%[7] thus **this test cannot be recommended as a diagnostic test.** Interestingly, only 50% of glucocorticoid-treated dogs had increased c-ALP in one study and that same study found that **absence of c-ALP increase does not rule out** spontaneous or iatrogenic hyperA (Solter et al 1993).[7]

Percentage c-ALP is often discussed. Wilson and Feldman[9] found that c-ALP comprised 25% or greater of ALP in hyperA dogs and that hyperA could not be distinguished from exogenous glucocorticoid administration, liver disease or diabetes mellitus by percentage in this study.

Similar to UCCr, c-ALP results cannot be used to rule in or rule out hyperA.

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PRACTICE TIP

Optimising the use of glargine insulin in cats

C&T No. 5288

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The use of glargine insulin in diabetic cats has become an important part of the treatment regimen in recent years. We have found the insulin pens to be particularly useful especially with clients who are elderly or who have difficulty using standard insulin syringes or drawing up the smaller volumes of insulin that some patients require. Insulin pens allow doses as small as 1 unit to be administered – smaller doses than this require the very small insulin syringes (0.3mL) to be used (can be drawn up from the 3mL pen cartridge or from the 10mL bottle). Insulin syringe magnifiers are also available to assist in drawing up small doses with a syringe.

To optimise the dosing of small volumes, the way the glargine is stored is important. We have found that using the glargine insulin straight from the fridge seems to influence either the effectiveness or dosage of insulin administered to the patient making it more difficult to stabilise the patient. Storing the insulin pen at room temperature (i.e. less than 25˚C) seems to result in more accurate dosing with a more stable glucose curve being achieved. The post-injection 'drip' on the end of the pen needle after removal from the skin is also reduced in size when administered at room temperature. The product insert actually recommends that the insulin be removed from the fridge 1-2 hours before first use (cold insulin is apparently more painful to inject) and then kept at room temperature after this for up to a month.

After injection it is important to maintain the needle in the skin with pressure on the plunger for at least 10 seconds before removal to ensure that the full dose is administered. We find that the 12mm pen needles are the easiest for the clients to use – the shorter needles increase the risk of 'intra-fur' injection by the client.

Comment courtesy of:

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Ruth provides really useful and practical information here. Aside from the obvious practical advantages, it is also worth noting that insulin dosing pens are much more accurate and precise than insulin syringes for small doses. For example, when administering a dose of 1 U of a 100 U/mL insulin preparation such as Lantas® (glargine) insulin, the mean dose delivered using the AutoPen is 0.93 U (range: 0.63 - 1.20 U) and for the SolarStar pen is 1.02 U (range: 0.60 - 1.40 U). In comparison, a study evaluating the accuracy and precision of insulin dosing by paediatric nurses using 0.3 mL or 0.5 mL insulin syringes found that the mean +SD dose delivered when intending to administer 1 U was 1.64 +0.38 (range: 0.65 - 2.80 U)1. Importantly, it can be seen that most syringe-measured doses exceed the intended dose.

If I want a patient to receive a dose intermediate to, say, 1 U and 2 U, I will recommend 2 U in the morning and 1 U in the evening. Similarly, if I want a cat to receive less than 1 U every 12 hours, I will recommend extending the dosing interval, so that the dose is decreased to, for example, 1 U every 24 hours. This approach works very well in my hands.

Reference
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Quiet Clippers

C&T No. 5289

Email correspondence from the International Society of Feline Medicine's (ISFM) list-serve forum.

The ISFM is CVE's partner in the highly regarded ISFM/CVE Feline Distance Education course. For further information go to: www.cve.edu.au to access our Distance Education brochure. Alternatively, contact **cve.disted@sydney.edu.au** or call (02) 9351 7979. Tutors are:-

- **Andy Sparkes** (UK) BVetMed PhD DipECVIM-CA RCVS specialist in feline medicine
- **Sarah Caney** (UK) BVSc PhD DSAM(Feline) RCVS specialist in feline medicine
- **Carolyn O'Brien** (Australia) BVSc(Hons) MVetClinStud FACVSc(Feline Medicine) MASM
- **Richard Malik** (Australia) DVSc DipVetAn MVetClinStud PhD FACVSc(Feline Medicine) FASM
- **Andrea Harvey** (Australia) BVSc DSAM(Feline) DipECVIM-CA MRCVS RCVS
- **Wayne Mizon** (Australia) BVSc MANZCVSc (Small Animal Medicine, Feline Medicine)
- **Samantha Taylor** (Australia) BVetMed(Hons) CertSAM DipECVIM-CA MRCVS
- **Sheila Wills** (UK) BVSc (London) RCVS ECVIM
- **Elise Robertson** (UK) BS BVetMed MACVSc(Feline) DipABVP(Feline) MRCVS

Andy Sparkes

Q. What is your recommendation for a brand of quiet clippers for cats (clipping for venepuncture etc) either wired or cordless?

DE Participant replied:

The Aesculap GT420 Isis is my all time favorite clipper for cats. It is small, wireless and quiet and if you treat it well (clean and oil) it will go for miles. Not sure if you can buy it in the UK, but I'm sure you can order it online.

Karin Holler replied:

My favourite is also the Aesculap GT420 Isis - nice, small and with a not too large head.

Francesco Gianni replied:

During the London vet show, I remember that Dr Kerry Simpson advised Wella clippers. I am assuming she meant the Contura clippers (?) which are used by hairdressers to trim the back of the neck, are very silent and also 'nipple' friendly. I saw Kruuse was offering them on their website (http://www.kruuse.com/en/ecom/Tilskud_pleje_lopper/Pelspleje/Elek_trim_klip_mask/Wella_Cont_klip_vet/prod_273207.aspx)

Pia Bisgaard Andersen, Denmark replied:

I go with the advice of Dr Kerry Simpson then.

I have used the Wella Contura mini-clippers for many years in small animal practice and feline practice, and they are just so quiet and easy to handle. Most cats, even non-sedated will tolerate them. I love them and the cats love them too. They are cordless and rechargeable. It is possible to buy extra/separate razor heads, when they lose their sharpness.

I mostly use them for venepuncture and smaller areas of pre-op clipping in cats and yes they are very 'nipple friendly' when ventral midline clipping. (Work well on rabbits'/rodents' thin skin too.)

The clipper rarely make cuts in skin, so they are the best choice if you need to clip smaller areas of filtered hair lumps on the soft skin area ventral abdomen or 'trousers' in longhaired non-sedated cats.

If you do a lot of feline work, I prefer to have a clipper/charger unit in each consult room and pre-op theatre, although it's expensive to start with. But otherwise buy one and share it for feline consults. It works well for venepuncture dogs too, but the blades certainly last longer if you treat them well and use for soft clean fur only.

Francesco – the link you mentioned is a Nordic website. I don't know if you could order directly from there or if you need to contact Kruuse in the UK?

But the Wella Contura miniclipper is worth looking for ☺.

Comment courtesy of

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VIN Feline Medicine Consultant (www.vin.com)
\\\\=^..^=////

We first heard about Moser clippers from Richard Gowan. We tried them and were really impressed; they are MUCH quieter than our Oster clippers. We had a set at our old premises but bought 3 more of them when we moved our practice 3½ years ago. The only problem is that they can cause some clipper rash if the blades are not clean or the cat is a bit wriggly (but it is not a common occurrence). There is little maintenance and they have lasted well.

Comment courtesy of

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About 5-6 years ago, I found Moser's clippers to be the only quiet ones and talked them up a lot. Now almost every company has a decent pair of quieter clippers – yet to see a pair of silent ones? Moser's are quiet but expensive. We have 4 different cordless brands in the clinic and all are quiet compared to the old style ones: Moser, Andos, Codos and another cheaper no name brand from China.

What's YOUR Diagnosis?

Swelling on the flank of a cat

C&T No. 5290

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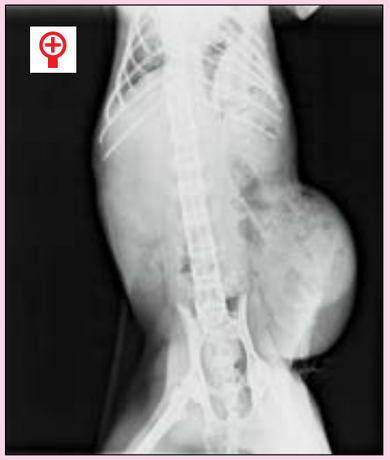

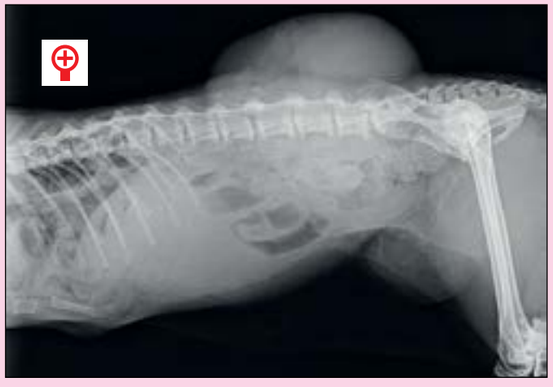

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Send your answers/comments for publication in Issue 271 June 2013 to the Editor, Lis Churchward at elisabeth.churchward@sydney.edu.au or Fax: (02) 9351 7968

Authors' views are not necessarily those of the CVE

Back to TOC page

WINNER

Ruby the Lazarus dog, or ‘When should I give up on ventilation?’

C&T No. 5291

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Figure 1. ‘Ruby’.

Ruby’s owners rang one spring September Sunday with the first snakebite of the season, which came as a surprise as the previous day I had attended a calving and been snowed on! The owner reported the snake looked black and had been viciously attacking Ruby’s leg, so I asked them to come straight in and they said they would bring the snake in too for identification.

I went to the drug fridge and noted, to my consternation, that we didn’t have any of the combined Tiger-Brown anti-venom we usually carry.

Ruby presented bright, very sore on her front right leg but not obviously envenomated. Her pupils were constricting. Clotting was delayed, but her blood did clot. I placed her on a Hartmann’s Solution drip and pre-medicated her with Sol-u-delta-cortef IV and histamil subcutaneously.

I was trying to identify the snake and was in discussion with a more senior vet. Our nurse, Rebecca, was nursing Ruby and when I went to check on her, Ruby suddenly crashed. She immediately turned blue and started doing what looked like agonal gasps.

I intubated Ruby, and we started to manually breathe for her. Ruby’s pupils were fixed and dilated and her eyes staring forwards. The situation looked pretty grim, but her owners opted to go ahead with the CSL Tiger snake antivenom we had (which also is effective anti-venom for black snakes).

For an hour and a half or so, Rebecca and I took it in turns to breathe for Ruby with no response at all. I kept touching her eye for a palpebral reflex but got nothing. Pupillary light reflexes were absent. I rang my colleague Jo again for some tips on prognosis and she suggested I hang in there as some patients recover and some will not. Ruby’s heart was strong so we just kept on breathing for her.

I fetched all the monitoring equipment from surgery and set little Ruby up under our Bair Hugger, and we settled in for a possibly very long afternoon. Finally, the apalert made a beep without our intervention, as Ruby almost imperceptibly began to breathe. She also began to shiver a bit, and I took some hope from these signs.

Ruby’s breathing became increasingly convincing and she began to maintain oxygen saturation above 90%. She stayed intubated and on oxygen for another 2 hours before she spat her tube out. I was able to keep the pulse ox on her tongue so could see that she was still maintaining oxygen saturation once the tube was out. Ruby continued to recover well but was very sore on her leg.

We started to do hot and cold compresses on the leg as often in the day as we could manage. The skin sloughed off a few days post-bite. Six weeks down the track the wound was almost healed and Ruby bright and bouncy.

I wonder if anyone has tips as to when to give up on ventilation? Being in a rural mixed practice, it is sometimes not going to be physically possible to allocate enough staff to manually ventilate a patient to recovery. **Are there any clues as to which patients are likely to recover and which patients are not?**

In this case, our persistence paid off, and the recovery of this delightful little dog made for a very rewarding case.

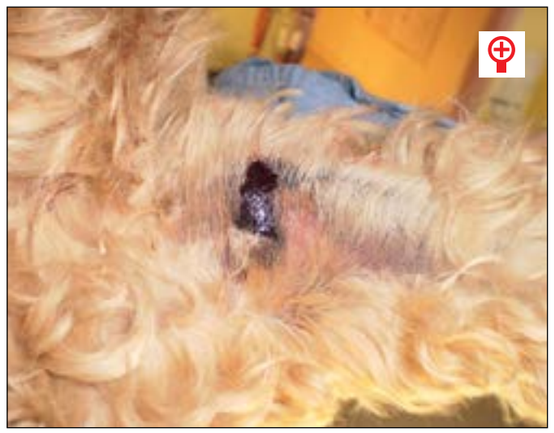

Figure 2. Ruby’s leg Day 1 post bite.

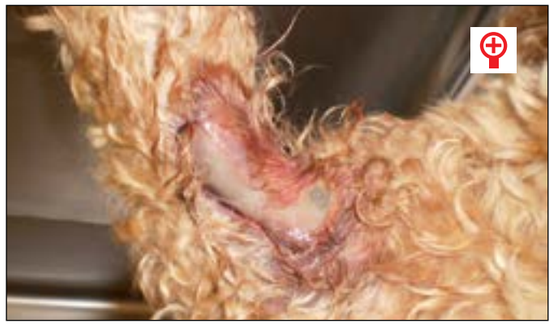

Figure 3. Day 4 post bite.

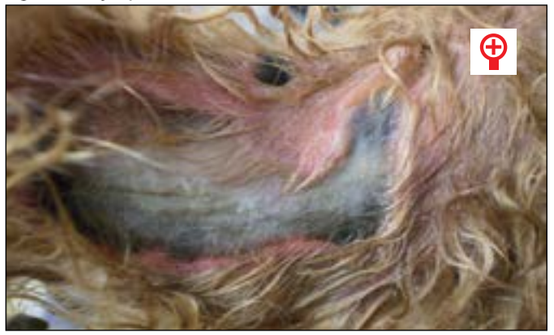

Figure 4. Day 6 post bite.

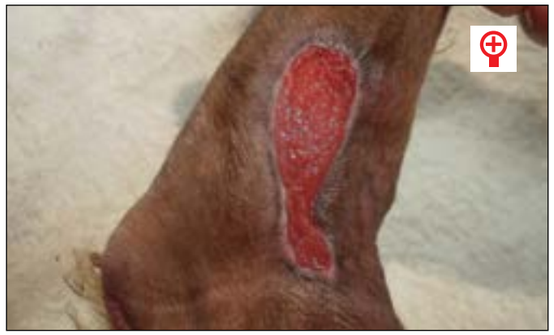

Figure 5. Six weeks post bite.

We love washing soda crystals!

C&T No. 5292

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In the course of treatment for a variety of other medical problems, ‘Max’, a lovable but naughty Schnauzer, ate a large quantity of softban bandaging material. This resulted in the alarming radiograph below (Figure 1), and plans of an impending ex-lap to retrieve the offending material.

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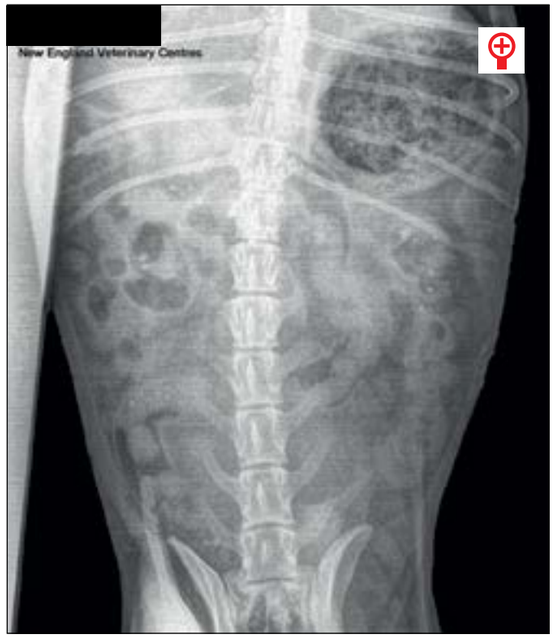

Figure 1.

We often find that we can induce vomiting with washing soda crystals (WSC), but in this case we did not think this would be enough to shift the softban.

Figure 2. The retrieved softban.

But in this case, we are happy to report that shortly after giving the WSC, 60cm of softban was produced by Max!

We find WSC a useful alternative to apomorphine. This is also the first time we have had trouble with a dog eating softban, although it can’t be the first time it has happened!

PostScript Since writing this article I undertook an online toxicology course and was warned that WSC can be quite irritant to the gut. If they are used, only a small amount should be used and followed by lots of water. However, at this stage we have used them many times without any observed issues. ▶

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

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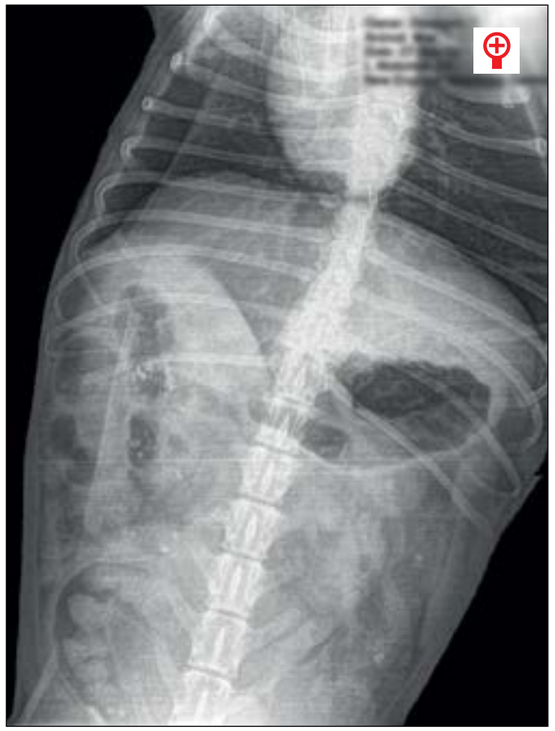

Figure 3. Radiograph of Max's empty stomach post WSC.

Tips to use acetylcysteine vials so the drug doesn't expire on the shelf awaiting a paracetamol poison case

C&T No. 5293

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Watch the antibiotics you use as some of the tetracycline family and erythromycin are not compatible with acetylcysteine.

Liquid Vials

- USA vets use 5% acetylcysteine in artificial tears +/- mixed with antibiotic eye drops TID in rabbits to dissolve the thick pus of dacrocystitis and allow better drainage. Sometimes used under sedation for retrograde flushing.

Ocular ulcers in dogs

- Something I learnt from UK for canine eye ulcers (but not supported by Aussie veterinary ophthalmologists here) is to dilute 1 vial of Omegapharm acetylcysteine (800mg/4mL vial) into 16mLs sterile water and flush the ulcer every 20mins for first 8 hours. Keep bottle in fridge; allow ½ mL in syringe to come to room temperature each time before applying to the affected eye. It has no preservative so discard after the 8 hrs. Probably would now use dog's own serum as beautifully described in C&T Perspective 93 Tick paralysis round table discussion (Dec 2012 Issue 269) to treat these ulcers but acetylcysteine still handy to start using in the interim until/if blood taken/available for topical ocular ulcer treatment.
- Others use it in a nebuliser as used for humans for lung congestion supportive treatment.

Powdered sachet form

- German vets use it in feline rhinitis/severe ocular-nasal congestion: Oral dose 10 mg/kg/daily. There they have sachets with 200 mg Acetylcysteine. Owners dissolve it in 10- 20 mLs of water and then give according to weight either once daily or divided into 2 doses. Most cats take it OK but they do not love it… You could put the acetylcysteine liquid in a gelatine capsule and dose ASAP (before it becomes soggy).

EPI in a cat

C&T No.5294

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We have been tracing an 11-month-old kitten for poor growth/failure to thrive since about 12 weeks of age. She has protein losing enteropathy and her tli is <1 ug/litre.

Q. What would be the best enzyme supplement for her and how would I assess her response to therapy (i.e. just good body condition and good health?) I have been recommended to use Vitamin B12 injections and poss omeprazole as well and wonder what your opinion is?

Reply courtesy of:

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at Rowville Veterinary Clinic - Phone: +613 9763 1799
at Lort Smith Animal Hospital - Phone: +613 9328 3021
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That sounds like a very interesting case. The question of which enzyme supplements are best for the management of EPI is an interesting one. There are no scientific studies that answer this question. Clinicians all seem to have their individual preferences and these are probably mostly based on what they are most familiar with. I think that if an animal with EPI is not doing well on a particular enzyme supplement, then the 2 most common solutions offered are: to increase the dose of the current supplement or to change the brand of supplement. If the latter is chosen and the animal improves, then the new brand might become the preferred supplement for that clinician.

A lot of clinicians like to use Creon® as their first choice of pancreatic enzyme supplement. This is a product designed for use in people. It comes as a capsule that contains granules. The capsule can either be given as it is, or it can be opened up and the granules mixed with the meal. Creon® capsules contain a reliable concentration of lipase at 3 different strengths: Creon® 40000, 25000, and 10000. There is a lot of individual variation in the optimal dose requirement and so it is essentially titrated to effect. I work on the principle that it cannot do any harm and the only side effect reported in people on high doses is constipation (which I have never induced in a dog or cat). I generally start a big dog on 1-2 x Creon® 40000 capsules BID with food, but it is not unusual for an individual large dog to require 4 capsules BID. I even have one cat on 1 x Creon® 40000 BID.

The clinical response to pancreatic enzyme supplementation is typically dramatic. For example, a thin 7-8 kg dog might gain 0.5 kg in the first week and have a marked reduction of gastrointestinal signs. They will typically still require a high calorie intake and defecate 3 or more times a day, but they gain weight and are much more comfortable. Therefore it is not usually necessary to monitor anything other than body weight/condition and the owner's observations. Over time, it might be necessary to increase the dose of enzyme supplementation.

In cats, appetite is linked to hunting behaviour and so polyphagia frequently presents as increased hunting. Cats with EPI with outdoor access will typically catch and eat at least one rat or mouse per day as well as constantly demanding food from their owners. They can also display increased ambush/pouncing behaviour towards people, which might be interpreted as aggression.

Vitamin B12 injections are strongly recommended for this case. I usually give 0.5 mL SC every week for 4 weeks to cats with pancreatic disease and then often continue long term with 0.5 mL SC every 3-4 weeks.

Omeprazole might be helpful, but I would try the pancreatic enzyme supplement by itself at first and see what clinical response you get. If you get a partial response, then I would typically increase the dose of the enzymes before adding omeprazole (but that is just the way I do it and might not be the only way...).

Postscript: Cathy reports that she also spoke to a vet at the ARH who recommended using fresh pancreatic tissue: 1 cm cube with each meal, which was said to be cheap and work well.

Linda Fleeman: Several of my clients have tried to source fresh pancreatic tissue in Australia without success. I would be interested to hear if anyone knows of a reliable source.

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Treating cats with pancreatitis (chronic)

C&T No. 5295

Anonymous

Recently a CVE member posed the following question which is a difficult area and we hope publishing this question stimulates discussion/feedback from members/readers for publication in our upcoming Issue 271 June 2013.

I would love to know your approach in treating cats with pancreatitis (chronic). I have about 5-6 cases which seem to get intermittent flair ups. I generally go with antibiotics (variable amoxyclav, metronidazole or cefovecin (if cannot tablet), maropitant and pain relief. Long term I try to maintain them on a gastrointestinal sensitivity diet, with repeat injections of B12.

Diagnosis is based on U/S and spec FPLi.

Should I be more aggressive in more diagnosis i.e. bile cultures, gastrointestinal biopsies etc?

I feel that I am not getting great outcomes with the cases and welcome feedback from C&T supporters.

Thank you.

Send your answers/comments to
elisabeth.churchward@sydney.edu.au or Fax: (02) 9351 7968.

The author of the best and most comprehensive answer will be entitled to a CVE proceedings of their choice.
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Neuromuscular Diseases CVEP No. 402

The one day seminar Neuromuscular Diseases was held on 21 October 2012 with guest speaker Dr G. Diane Shelton DVM PhD Diplomate ACVIM (Internal Medicine). The aim of the seminar was to provide a practical approach to recognising and diagnosing canine and feline neuromuscular diseases that may be seen in general practice as well as available treatment and management options. These are the 35-page proceedings.

Page 1 *Page 2* **Table of Contents**

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You don't have to be a CVE Member to pose a question to your colleagues, and you can remain anonymous if you prefer. Send your question for publication in Issue 271 June 2013 to elisabeth.churchward@sydney.edu.au or post/fax it in.

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Back to TOC page

Haemophilia in Cocker Spaniel pups

C&T No. 5296

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Note: Marilyn saw this case at Springfield District Veterinary Clinic.

23/02/10: 'Frodo' is a 6½-week-old, **male**, entire Cocker Spaniel Puppy, 1 of a litter of 7 puppies presented for their first vaccination. No abnormalities were detected on the physical exam and all pups were given a C3 vaccination on the right side of chest and microchipped between the shoulder blades. All pups were to be sold to help pay for the owner's upcoming wedding.

25/02/10: 2 days later, Frodo was presented for a large mass between his shoulder blades, likely where the microchip was placed. Apparently the pup had wriggled a lot during the procedure. The mass measured approximately 6cm x 4cm and a fine needle aspirate and Diff-Quik stain revealed red blood cells, no bacteria or neutrophils. He weighed 1.88kg, was quiet, normal temperature, pink mucous membranes, capillary refill time of 1 second, tachycardia (268 bpm) and tachypnoea (156bpm). Further examination revealed a lump on the head over the occiput.

The most likely diagnosis was a haematoma or possibly a reaction to the microchip. He was treated with subcutaneous fluids, Betamox drops BID and Temgesic injection, with a plan to re-visit in 2 days if he remained stable.

Figure 1. Frodo with the haematoma between his shoulder blades (arrow) and over the occiput (arrow head).

26/02/10: Frodo returned the next day as he was very flat, had white mucous membranes, HR 204 bpm, and a grade II/VI left sided systolic heart murmur, which was likely due to his anaemia which had not been present the day before. He had discomfort on abdominal palpation, temperature of 36.3℃, no known vomiting or diarrhoea and no exposure to rat bait. There was no evidence of bruising or petechiae, and no fleas were found.

Differential Diagnoses: Severe blood loss into subcutaneous haematoma, auto-immune reaction after vaccination, liver disease leading to clotting problems, possibly a porto-systemic shunt (I always include PSS in young pups that aren't quite right). A poor prognosis was given; however, the owner had become quite attached to Frodo, and she wanted to treat him.

He was admitted for in-house CBC and MBA, and would likely require a blood transfusion and possible referral. When attempting to place an IV cannular, his cephalics were very flat, and a 25g IV catheter was eventually placed into the right saphenous. Jugular blood was taken which looked very thin and watery.

Abnormalities:

HCT: 5.3% (37.0-55.0)! (Panic and wonder how this pup is alive)

HGB: 1.6 (12.0-18.0)

Platelets: 147 (175-500) not low enough to be causing such dramatic anaemia, possibly non-functional platelets, some clumped platelets were identified on a blood smear.

Albumin: 17(21-36)

Total Protein: 43(48-72) likely from blood loss

Phosphorous: 1.53 (1.65-3.35)

Serum was clear

All other results were within normal limits.

DDx: Possibly a clotting factor problem?

Frodo was given 100% oxygen by mask, his initial SpO_2 was 84%. A blood transfusion of fresh whole blood (62mL) was given over the next 4 hours without cross-matching. He was monitored for a transfusion reaction and, after the first hour, he became a lot brighter; after 2 hours he was able to stand, could eat and drink, and his SpO_2 remained 96% without oxygen therapy.

Frodo's PCV immediately post-transfusion was 28% and TPP 50. He was bright, his mucous membranes were pinker, CRT 1 second, and HR 185bpm, with a gallop rhythm instead of a heart murmur. Once the blood transfusion finished, he was started on IV fluids at maintenance rates using 0.45% saline and 2.5% glucose.

Now to work out what was going on. A Coagulation Profile was the next step to look at clotting factors. After speaking to a pathologist, we realised that if we did a Coagulation profile then, it would reflect the results of the donor dog's blood. Factor VII (prothrombin [PT]) half life is 3 hours and other clotting factors half lives can be days long, and you need to go through several half lives first before the test will be accurate, so it was decided to perform the coagulation profile in 1 week's time.

Frodo was maintained on IV fluids overnight, with a plan to repeat a PCV the next day, and every 2 days until the coagulation profile could be run in a week's time.

27/02/10: Day 2 Post-transfusion: Frodo was bright and happy overnight, mm's pink and CRT <2 sec. Temp 38.2℃, HR 190bpm, mm's pink, CRT 1 sec, PCV 34%/ TPP 50. So far so good; he was discharged and was to be kept quiet.

If all was well the owners would keep him for the next few weeks and would let the new owners know about his history, inform them he had had a blood transfusion, and should be blood-typed if he needed a blood transfusion in the future.

02/03/10: Frodo had been bright and happy at home, eating and toileting normally. Examination revealed his head had become an irregular shape, due to subcutaneous swelling on the dorsal right side of his head. His PCV was 25%/TPP 42 – a decrease from 2 days ago. Frank blood was aspirated from the subcutaneous head swelling. It was still too soon to perform the coagulation profile, and a poor prognosis was given due to the worsening of clinical signs, so the owner elected to euthanase Frodo with a presumptive diagnosis of haemophilia.

09/03/10: 'Hutch' was a **male** Cocker Spaniel pup from the same litter as Frodo, and was presented for lethargy since the night before, crying in pain, and retching but not vomiting. He had some diarrhoea and was eating and urinating normally. There were 2 pups from the same litter left, and they were fine. The bitch had been eating a bone near the pups and the owner was concerned this may have caused the pup to become unwell. On examination, his abdomen and thoracic and lumbar spine was uncomfortable.

Methadone was given subcutaneously and lateral and VD radiographs were taken of the thorax and abdomen. No abnormalities were found. An in-house Activated Clotting Time was prolonged at 5.5 minutes and a coagulation profile was performed, in case he had a similar condition to Frodo. Temgesic was given subcutaneously for pain relief overnight until further results were received.

Brainstorming with a pathologist again revealed that there is a rare coagulopathy affecting male Cocker Spaniel pups – Haemophilia A or B.

10/03/10: Haematomas had formed over both jugular veins after venipuncture the day before.

Laboratory Results: Normal PT, **prolonged APTT** – suggestive of DIC, hereditary coagulation factor deficiency in the intrinsic system (factors VIII (haemophilia A more common) factor IX (haemophilia B less common), factor XI (generally not clinical), or factor XII (generally in cattle, extremely rare in dogs) or occasionally in Von Willebrand's Disease (VWD) (although in most cases APT is normal in VWD).

The next diagnostic step would have been Factor VIII and IX testing, but these were not performed. If more common things occur commonly, then the most likely diagnosis would be Haemophilia A. The prognosis again was poor, and there would likely be ongoing problems, treatment and costs, so the owner elected to euthanase Hutch also.

A coagulation profile was performed on the remaining litter mates (1 male, 1 female) as requested by the owner, although it was probably not necessary for the female pup. Both had normal results and were re-homed, with a recommendation to neuter them both. Factor VIII and IX levels were also discussed, but not performed.

As the bitch was the carrier for haemophilia (as the inheritance is X-linked, with affected patients male and their dam the heterozygous carrier) she was speyed, and had more superficial bleeding than expected, but her activated clotting time was within normal limits.

Haemophilia

Haemophilia is the most severe inherited bleeding disorder, and Haemophilia A is more common than B. Haemophilia A is due to a deficiency in Factor VIII and Haemophilia B is due to deficiency in Factor IX, which are both critical to the coagulation pathway. It is X-chromosome linked, recessive. As males have 1 X-chromosome, and females have 2, the males will express the defect, whereas females can remain carriers if only 1 of their X chromosomes are affected. In asymptomatic animals with a hereditary factor deficiency, PT and APTT may be abnormal or normal.

Female carriers of Haemophilia can be difficult to detect, as factor VIII and IX levels may be normal, however there is a DNA test available which detects the presence (or absence) of a specific mutation in the Factor IX gene for Haemophilia B.

Treatment involves transfusion of fresh frozen plasma or fresh whole blood at times of haemorrhage. Gene therapies may also be available, but also have their limitations.

I would be interested to hear of any haemophilia cases that have been diagnosed and successfully managed.
Many thanks go to Terry King at VSS in Brisbane and all of the pathologists at IDEXX Laboratories for all of their help and advice.

Editor's Note: *Members/Readers are invited to sent Replies and Comments to this article to both the author and CVE at elisabeth.churchward@sydney.edu.au for publication in our next issue. Thank you.*

Colonic torsion in a 4-year-old German Shepherd

C&T No. 5297

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'Locke' Parkin, a 4-year-old entire male German Shepherd was presented after hours at the Animal Emergency Centre Canberra. He had a history of vomiting and lethargy since the morning of the previous day. That afternoon he had been seen at his regular veterinary clinic and treated with an injection of maropitant citrate (Cerenia®) as well as a course of metronizadole for suspect gastroenteritis. While the vomiting had subsided he was depressed, not eating and not defaecating except for some clear fluid on the day of presentation. He urinated normally but the owner had been syringing Lectade® into his mouth as he was not drinking.

On physical examination he was found to be quiet, alert and responsive, with a heart rate of 120 beats per minute, a capillary refill time of less than one second and tacky pink mucous membranes. There was decreased skin elasticity. No constipation was palpable. He had purulent material discharging from his anal sacs, which had been noted the previous day. There was a doughy and painful abdominal mass in the caudo-dorsal abdomen.

After sedation with methadone and acepromazine conscious radiographs were obtained that showed a well defined loop of bowel with contents of soft tissue opacity, as well as a large amount of gas in the proximal bowel. This looked overwhelmingly like an obstruction and an exploratory laparotomy was decided upon after consultation with the owners.

The dog was started on surgical rates (10mL/kg/hr) of intravenous (IV) Hartmann's Solution®, induced with 4mL alfaxan-CD® IV, intubated and placed on isoflurane maintenance. He was given 300mg cephazolin slow IV before the operation.

After routine clipping and preparation a midline incision was performed from xiphisternum to pubis. It revealed a blackened and enlarged loop of colon (Figure 1) as well as an enlarged spleen with discoloured plaques on its surface (Figure 2). ▶

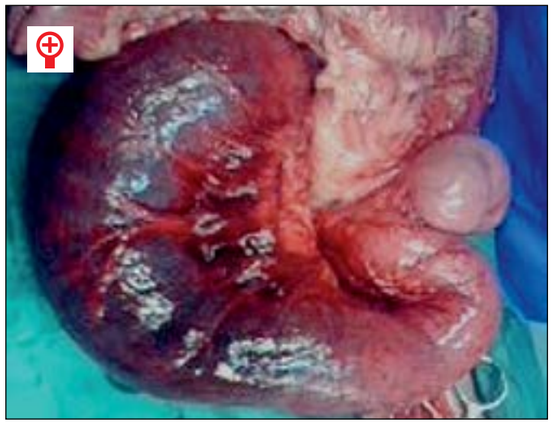

Figure 1. Blackened and enlarged loop of colon.

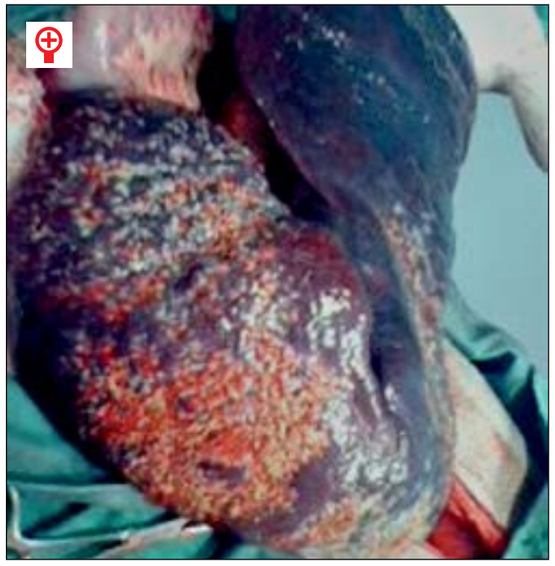

Figure 2. Enlarged spleen with discoloured plaques on its surface.

On further exploration to explain the appearance of the colon it was found to have developed a very tight torsion, very much like one we would expect to see in a horse. The bowel was untwisted and examined. It showed necrosis from about 4 centimetres aboral to the caecal junction, all the way to the rectum, which appeared normal (Figure 2).

There was no arterial pulse in its mesentery. We estimated that 90% of the colon was dead. The owners were contacted and informed. They opted for euthanasia due to the grave prognosis for survival and, should Locke survive, for bowel function and quality of life.

Discussion:

After a search through the literature we only found references to 4 colonic torsions in dogs: 3 of them in Great Danes, 1 in an unstated breed. One of these survived as there were 12 inches (30 cm) of viable bowel, approximating two-thirds of its total length in that dog. This was described as the minimum amount of bowel needed for adequate nutrient absorption. This patient was treated at the Chesapeake Veterinary Referral in Maryland, USA, by Dr Krista Evans. She is quoted as saying she had only seen 2 or 3 papers describing the condition in 20 years and that 90% of them die during surgery. There is no known cause for this catastrophic event other than they all occurred in large breed dogs who share increased risks of gastric dilation and torsion (GDV).

An article by C. Carberry and J. Flanders in *Veterinary Surgery* Vol 2, Issue 3, pages 223-228, May 1993 mentions caecal-colic volvulus in 2 intact male Great Danes with 1 survival. The clinical signs and physical examinations and diagnostic findings were unspecific for large bowel volvulus. They consisted of peracute onset of vomiting, mild abdominal enlargement and pain, lack of faeces, and tenesmus. The radiographs showed severe dilation of bowel loops. All these were consistent with our own findings.

There was no mention of splenic changes in the articles we found and we have no way of telling whether they were pre-existing, or related to the torsion. The only link between these cases other than the size of the dogs is that they all happened in intact males.

We would like to know whether anyone else has encountered cases like these and whether they can offer some comments.

Editor's Note: *Members/Readers are invited to sent Replies and Comments to this article to both the author and CVE at elisabeth.churchward@sydney.edu.au for publication in our next issue. Thank you.*

Invited Commentary courtesy of

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This is an interesting case from both the breed predisposition angle and the surgical decision making points of view.
For some reason German Shepherds are over represented in reported cases of gastric dilation and volvulus, mesenteric intestinal volvulus and specific large intestinal torsion or strangulation.[1-3] This may in some way be related to an established genetic predisposition that the German Shepherd breed has to inflammatory bowel disease.[4-8] A condition has been reported in German Shepherds that have developed intestinal entrapment with strangulation associated with rupture of the duodenocolic ligament.[1] German Shepherds have also been reported to develop intestinal volvulus as a late complication after gastric dilation and volvulus.[9] The only 2 cases of this we have seen over the past 12 years in a specialist/emergency practice were both German Shepherds and in each case the intestinal volvulus was peracute, severe and fatal occurring a week and a month after gastropexy. It is perhaps a 'long bow to draw' but worth mentioning that the suppurative discharge from the anal sacs reported here would make one think of perianal fistulae/anal furunculosis which is another German Shepherd disease that has also been associated with inflammatory bowel disease in this breed. One could speculate that anatomic and motility changes associated with inflammatory bowel disease would be possible contributing factors leading to the colonic torsion seen in this dog.

On the decision making front it is important to distinguish the impact that an extensive resection of the **small intestines** has on a dog from the effect of resecting most or all of the **large intestines** (colon). *Short Bowel Syndrome* is a term usually applied to the potential problems of insufficient absorption of nutrients seen when most of the **small intestines** are removed. It is unlikely to occur with a 70% or less resection of the small intestines. Even with 70-90% removal the malabsorption issues may be quite manageable and any malabsorption issues often improve over time. In my experience the theoretical concerns about short bowel syndrome in a given case are rarely proven to be justified. We rarely are required to contemplate removal of so much of the small intestines to make it an issue. At the Animal Referral Hospital we have seen a bulldog that had its mid-descending duodenum anastomosed to the ileum that did very well with appropriate medical management.

In contrast, the predominant function of the large bowel in the dog and cat is to dehydrate the remaining ingesta after the digestion and absorption by the small intestines. Removal of the entire large intestine tends to result in severe intractable diarrhoea in the dog. Cats with chronic megacolon seem to cope with removal of the colon (by either total or subtotal colectomy) better than dogs. If the necrosed segment of large intestines observed in the German Shepherd of this report had been resected the absorption of nutrients would have been largely unaffected. It appeared that the proximal colon was viable and that the resection of the segment followed by an end-to-end colonic anastomosis could have been performed. It is possible to say that if the cranial rectal artery was intact in this case, as the picture suggested, the resection and anastomosis of the colon would have been viable.[2] Preservation of the ileocolic sphincter and a small segment of the proximal and distal colon in this dog would have probably given it less risk of the severe diarrhoea that follows removal all of the entire colon and the ileocolic junction. There is at least 1 report of a dog having ileorectal anastomosis following colonic torsion that survived.[10]

The subcapsular pale grey plaques seen on the surface of the spleen are a common enough age related change that is not likely to be of significance in this case.

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Reply from Madeleine

Thank you Dr Simpson for the detailed answer and particularly for the breed predisposition angle. I had not known of the incidence of peracute intestinal volvulus in the weeks following a GDV and gastropexy in German Shepherds. Something more to discuss and educate owners about!

Just another day at the clinic...

C&T No. 5298

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Figure 1. 'Rembrandt'.

My colleagues and I saw the following case while I was working at Sylvania Veterinary hospital.

Clinical Case

'Rembrandt', a lovely 15-yr-old MN Siamese cat was presented to us for a check up before 10 days boarding at our veterinary hospital.

During the physical exam, he seemed to be in good health apart from some moderate dental disease. As an elderly cat, we performed an in-house pre-anaesthetic blood test before performing the dental procedure and it revealed 'Rembrandt' had a mild azotaemia, USG of 1.030 and the UPC ratio of 0.1. The dental procedure finished smoothly with the assistance of intravenous fluid therapy and he boarded with us with no notable events in the next 10 days.

Approximately 3 weeks passed before Rembrandt was re-presented to us and this time, the family was not happy. According to the owners, 'Rembrandt' was inappetant, losing weight and for the last week had been sleeping a lot more than usual. The owners had 'heard' that there had been 'a cat in hospital with diarrhoea' and decided Rembrandt must have 'caught whatever it had', and wanted a full refund of the laboratory work, dental, medications and boarding, and of course, wanted their cat cured!

Owners mentioned that there was no polyuria/polydipsia, no vomiting and no diarrhoea. To make things more vague, there were no abnormalities detected in the physical exam. The only thing we could think of was the mild azotaemia from the previous laboratory work, so we advised them that we should really recheck those analytes. ▶

Both urea and creatinine levels were border-line abnormal (actually improved from the last visit) but the ALT was mildly elevated. The USG was still 1.030 but there was a moderate amount of blood in the urine. Diagnostic possibilities - urinary tract infection? Uroliths? Neoplasia? Early stage of chronic kidney disease? +/- Mild hepatopathy?

We recommended an abdominal ultrasound and, surprisingly, the owners were very keen for us to go ahead. The abdominal ultrasound showed:-

– Bladder: normal

– Right kidney: normal

– Left kidney: abnormal. It had a roundish mass, approximately 2 cm in diameter that was invading the cortex and medulla. The mass was sampled using a FNA technique and sent away for cytology analysis.

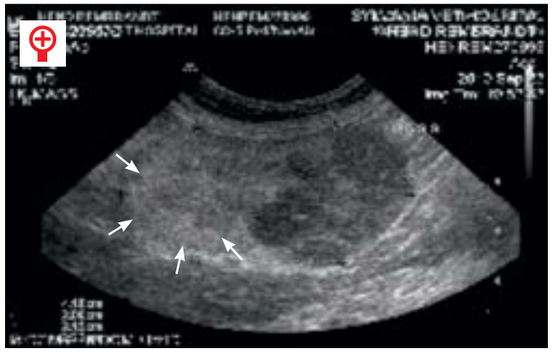

Figure 2. Abdominal ultrasound scan showing mass at the pole of the left kidney.

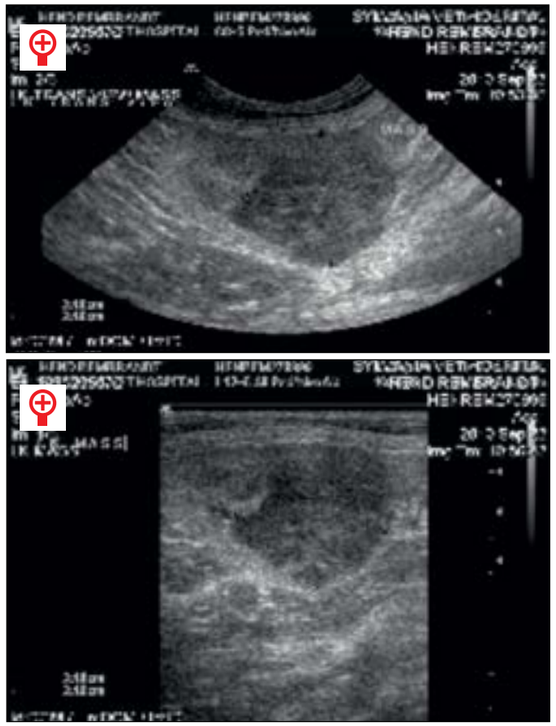

Figure 3.

The smears were moderately cellular with good cell morphology. A moderate background of blood was present (and moderate amount of ultrasound gel in one slide). The nucleated cell population was dominated by medium lymphocytes with round nuclei, single nucleoli and small volume of basophilic cytoplasm. Also present were smaller numbers of small lymphocytes, neutrophils and macrophages. No other significant cellular population or microorganisms were identified.

Interpretation: *Consistent with lymphoma.*

Comments: *The predominance of medium lymphocytes in this sample is consistent with lymphoma. If further classification is required, biopsy for histology +/- Immunohistochemistry is recommended.*

So what happened to Rembrandt? We thought the realistic options were:-

1. Chemotherapy: COP protocol
2. Palliate with prednisolone +/- Chlorambucil
3. Do nothing?

The owners chose option 3. Despite the images and the cytology results, Rembrandt's family said we were 'wrong'. They could not believe that after only 1 week of clinical, yet vague illness, Rembrandt could possibly have an aggressive cancer.

We strongly recommended that they at least try option 2, but they declined. They planned to revisit us in a few months for a re-ultrasound to see if 'that lump' was growing/changing and if so, the would consider chemotherapy then. We sadly warned the owner that that time frame might be too late,and that Rembrandt could be dead, or the cancer metastasise making chemotherapy ineffective. The owners could not be dissuaded from option 3 and took Rembrandt home.

Three days later, the owners came back (without Rembrandt) to pick up some prednisolone tablets. We started him on a dosage of 3mg/kg once daily for the first week, reduced to 2mg/kg once daily for the second week. Sadly, 10 days after commencing prednisolone therapy they returned with Rembrandt to euthanase him.

This case left us with some unanswered questions and and of course we wondered – could it have been different?

Q1. Improving azotaemia: why did Rembrandt's azotaemia improve when he had renal lymphoma? Are they not usually progressive?

Q2. Palliative care: is there anything better than prednisolone +/- Chlorambucil?

Q3. Was it 'wrong' for us to allow the owners to take Rembrandt home with no treatment plans?

Q4. Does this job ever get easier?

Editor's Note

We wecome Comments/Replies from Members/Readers. Please send them to elisabeth.churchward@sydney.edu.au or by mail or fax: (02) 9351 7979.

Perspective 94

Open Access Publishing

Richard Malik, CVE & Nandita Quaderi, BioMed Central

Open Access Journals

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Traditionally, scientists, doctors and vets publish by submitting 'papers' to established journals for peer review. This means an Editor or Associate Editor looks over the paper and then selects 1 or 2 anonymous Reviewers with particular expertise in the field who then read the paper in more depth and decide on the validity and merit of the work. After some 'tooing and froing', and often with subsequent modification, the paper may be accepted for publication. To ensure impartiality, sometimes the Reviewer is 'blinded' to the names of the authors but they often can work out who the author/s is/are if they are writing on a subject that is their area of expertise. The basis for this process is that peer review is supposed to be a 'check and balance' on the logic and conclusions of the study or case series. This is the time-honoured system but there are a number of issues with this process, namely:-

1. The Reviewers aren't paid (and therefore they can be hard to source) and often struggle to meet deadlines
2. Editors & Associate Editors are paid – but not very much – and are usually busy people who undertake these tasks in addition to their main employment.
3. Sometimes it is near impossible to get your paper published if it challenges accepted orthodoxy e.g. the 2005 Nobel Prize in Physiology or Medicine awarded to Barry Marshall and Robin Warren who, with tenacity and prepared minds, challenged prevailing dogmas.
4. **The publishers get a great deal of money** for selling the articles online or through subscription fees for the journal i.e. individual and institutional subscriptions e.g. libraries, medical/veterinary organisations.
5. Unless you have a university or other affiliation you have to pay for the entire article if you are not a subscriber to the journal – only the abstract is free online through Google or PubMed.
6. The authors may need to contribute to 'page charges' for prestigious journals, especially for publications using colour plates.

Due to these issues, the concept of Open Access Journals developed and there is an increasing trend towards publishing in this format. In this model:-

1. The Reviewers do not get paid, but generally guarantee that they will produce the review quickly – often in 2 to 3 week's time.
2. Peer review occurs but the stringency of the process depends on the reputation and standing (impact factor and other metrics) of the journal. Many Open Access journals are of the highest standard and are highly prestigious.
3. Once the paper is accepted for publication, the authors pay the publisher in the order of $1200- 2000 for typesetting/design/other ancillary IT costs as well as editorial costs. The incentive for authors is that there is a greater chance of publication and less length restriction.
4. The final paper is freely available to **anyone** as a downloadable PDF in full colour. Any vet/scientist/author can access it. Even clients can access the articles!
5. Some 'traditional' journals provide the same facility if the authors pay money to 'unlock' their own article. For example, the *Vet Record* now does this.
6. There are moves in the UK to ensure that all government funded research is published in the Open Access format. This is because proponents of this move believe that if taxpayers funded the work they should not then have to pay third parties (e.g. Elsevier) for the privilege of reading about that research.

Caveat emptor – prospective authors should investigate the reputation of the Open Access Journal before proceeding with publication because not all journals maintain the same high ethical standards. Some journals may just want your $2,000 and not conduct the proper checks and balances.

Interested in checking out Open Access Journals yourself?

Here are 4 papers which I had published in Open Access Journals. You can simply click on the links provided below to access them (in our complementary e-book version of this issue - another great reason to ensure you contact CVE TODAY with your email address!) but it's more interesting to try finding them yourself using Google and an appropriate key word search.

- The first article is about genetic recombination of *Cryptococcus gatti* in WA. (Although I am exceedingly proud of the work I'm not sure most C&T Series readers will find it as riveting as my mycology colleagues would!) It can be found at: **www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0016936.** But try looking for it yourself using Google.*
- The second article is a really good retrospective study by a great team of veterinarians representing a summation of Julian Lunn's Masters Degree: **www.parasitesandvectors.com/content/5/1/70** A few years ago, I would have asked Jules to write a perspective for C&T based on this paper but because the journal is Open Access, you can download the PDF directly yourself and we can save the space in the C&T Series for something else!
- The third article concerns Haemotropic mycoplasmas in dogs living in Aboriginal communities in Central Australia. **http://www.biomedcentral.com/1746-6148/8/55****
- The fourth concerns the molecular genetic basis for hypokalemic polymyopathy of Burmese cats. Reference Barbara Gandolfi[1], Timothy J. Gruffydd-Jones[2], Richard Malik[3], Alejandro Cortes[1], Boyd R. Jones[4], Chris R. Helps[5], Eva M. Prinzenberg[6], George Erhardt[6], Leslie A. Lyons[1]. 1 Dec 2012. First WNK4-Hypokalemia Animal Model Identified by Genome-Wide Association in Burmese Cats. *PLOS ONE*. **www.plosone.org.** Volume 7. Issue 12. e53173.

*To test the user friendliness of the search I asked a non-vet to try and find this article. She found the article first go by typing in the following sequence of key words in Google – Cryptococcus gatti in WA Australia open journal. It's that easy and such a valuable resource.

***Editor's Note:** The CVE paid for publication of the middle two papers mentioned in this article.*

Seen any neural angiostrongyliasis cases since 2005?

Finally, on an entirely different subject, if you have seen any cases of neural angiostrongyliasis I would love to briefly hear about your experiences. Just email me at **richard.malik@sydney.edu.au.** Thank you.

A brief introduction to the open access publishing model

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Nandita Quaderi is Publisher (Biological Sciences) for BioMed Central. She was previously a Wellcome-Trust funded Principal Investigator at the MRC Centre for Development Neurobiology, King's College London.

Traditionally, access to scholarly, peer-reviewed research has been restricted to individuals with either an institutional or personal subscription to the relevant academic journals. The price of journal subscriptions continues to rise in an economic climate in which library budgets are being drastically cut. Thus, for those not affiliated to a subscribing institution, it is becoming increasingly difficult to keep abreast of research advances and current expert opinion in any given field.

However, over the last decade the open access (OA) publishing model has revolutionised the availability of scholarly literature by allowing anyone with internet access to freely read, download, translate or otherwise re-use OA articles (on condition that these articles are cited appropriately). Open access articles, as opposed to articles that are simply free to read, are defined by 2 fundamental criteria:-

1. Price barriers (e.g. subscription fees) are removed
2. Permission barriers (e.g. copyright restrictions) are removed

The subscription-free access to scientific literature provided by the OA model is particularly useful for researchers based in the developing world and also for professionals, such as vets, who may not have access to an academic library but nevertheless benefit from knowledge of the most recent advances in their field.

BioMed Central (www.biomedcentral.com) was a pioneer of the OA model, launching their first OA journals in 2000, and the company continues to be at the forefront of the OA movement. We currently publish almost 250 peer-reviewed, PubMed-indexed journals in the biomedical sciences including the top-ranked (by Impact Factor) veterinary science journal *Veterinary Research* (www.veterinaryresearch.org/), *BMC Veterinary Research* (www.biomedcentral.com/bmcvetres), *Virology Journal* (www.virologyj.com), *Parasites & Vectors* (www.parasitesandvectors.com), *Acta Veterinaria Scandinavica* (www.actavetscand.com), and *Irish Veterinary Journal* (www.irishvetjournal.org).

All BioMed Central journals are online only. The online format removes the need for the space and size restrictions imposed by traditional print journals. For example, there are no limits on article size or charges for the inclusion of colour figures. Additionally there are no limits on the number of articles that can be published in a given period, unlike for print journals that often have constraints on the size of individual issues that are related to the cost of producing and distributing paper copies. Removing the need to limit article numbers means that the decision whether or not to publish an article can be made solely on the scientific merit of that article rather than logistical factors.

A summary of the benefits of OA, for both readers and authors, can be found in Table 1. As there are no subscription or licensing fees associated with OA publications, many OA publishers charge an article-processing charge (APC) to cover the costs of publishing. These costs include peer review and copy editing, article production, marketing and promotion, hosting journal websites and maintaining a permanent archive of all published content. The APC for a typical BioMed Central journal ranges from £1075 - £1230. The APCs for BioMed Central journals and those for other major publishers can be found here: **www.biomedcentral.com/about/apccomparison/.** The OA model has widespread support from funding bodies, many of which allow APCs to be covered by research grants and for OA publishing costs for to be included in grant proposals. Several major funding agencies provide central funds to cover APCs and have also introduced mandates that require research funded by them to be published in an OA format (for example: http://www.rcuk.ac.uk/research/Pages/outputs.aspx). The Australian Research Council has recently announced their pro-OA policy – details can be found here: www.arc.gov.au/applicants/open_access.htm

BioMed Central operates a Membership scheme whereby authors affiliated to Member Institutes have their APCs, either in part or in full, paid by the Institute. The list of over 20 Australian Member Institutes can be found here: www.biomedcentral.com/inst/

Authors from more than 90 low-income countries receive automatic waivers on submission and do not have to pay APCs (http://www.biomedcentral.com/authors/oawaiverfund/). Furthermore, any researcher who does not have access to funds to cover the APC for a BioMed Central journal can request that the APC is waived.

In many ways OA journals are the same as subscription journals, they have Editors-in-Chief, Editorial Boards and adhere to robust peer-review standards – the only difference between reputable subscription and OA journals are in relation to the accessibility of published articles and the business model used by publishers to generate income. Considerable respect has been earned over the last decade by OA publishers such as BioMed Central and Public Library of Science (PLOS) and the growing prestige of OA titles such as *Genome Biology* (www.genomebiology.com) and *PLOS ONE* (www.plosone.org). As with traditional journals, different OA journals have different editorial thresholds for publication. So for example, *BMC Veterinary Research* will accept articles that contain 'sound science', as judged by peer-review (blogs.biomedcentral.com/bmcseriesblog/2012/06/01/to-reject-or-not-to-reject-that-is-the-question/), whereas *Veterinary Research* operates a more selective policy that also takes the general interest level of the article into account.

The Directory of Open Access Journals (www.doaj.org/) provides a comprehensive list of OA titles. The successful integration of the OA model as part of the biomedical publishing landscape, together with the relative ease with which it is possible to start a new OA journal, has resulted in a flurry of new OA launches over the last few years. The Open Access Scholarly Publishers Association (OASPA) has been created to maintain the integrity of the OA movement; in order to become a member of OASPA (www.oaspa.org/membership/members/), publishers must agree to abide by a professional code of conduct (www.oaspa.org/membership/code-of-conduct/).

Further reading

Further details of the OA model can be found here:
www.earlham.edu/~peters/fos/overview.htm

Some misleading myths regarding OA are tackled here:
www.biomedcentral.com/about/advocacy12

Benefits of OA for readers	Benefits of OA for authors
Removal of subscription barriers allows immediate electronic access to peer-reviewed articles	High visibility and maximum exposure of articles – increased chances of articles being noticed, read or cited
Easy to search for articles of interest – can use Google as well as traditional indexing services such as PubMed	No limits on article length or additional charges for inclusion of colour figures, video clips or large data sets
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Table 1.

Perspective 95

Anxiety in Small Animals

Jodi Vermaas, Robert Irvin, Kevin Reineck & Kelly Halls.

FROM THE DE FILES

Kersti is the CVE's Distance Education Tutor for Behavioural Medicine

Kersti Seksel BVSc(Hons) MRCVS MA(Hons) FACVSc (Animal Behaviour) Dipl ACVB CMAVA DipECVBM-CA

Link to BMed on website: **http://www.cve.edu.au/debehaviouralmedicine**

Go to the DE page of our website to view course information for 2014: http://www.cve.edu.au/distanceeducation

The behaviour that an animal exhibits at any given time is influenced by 3 main factors: Genetic predisposition, learning from previous experiences and the environment. The genetic or inherited component predisposes an animal to behave in a certain way and influences how behaviour may be expressed. Learning occurs every time an animal interacts with people, other animals and the environment in which it lives. Therefore all previous experiences, good or bad, are influential. The socialisation period (which occurs between 3 and 12 weeks of age in dogs and between 2-7 weeks in cats), is a particularly important and impressionable time. Finally, the environment or current situation in which the animal is in at any particular time will also influence the behaviour that is exhibited at that time.

Anxiety disorders are thought to occur in about 20% of dogs and cats. Anxiety is caused in part by a problem with how the brain processes information so the pet becomes worried about things and events which are not truly dangerous or a threat. Anxiety is a medical problem – just like diabetes is a medical problem caused by a problem with how the pancreas processes glucose. In the brain, information is conveyed between different parts by neurotransmitters. There are many different types of neurotransmitters that have varying effects on the pet's emotional state. These messengers bind to neuroreceptors which then pass the message along. Low levels of neurotransmitters such as serotonin or noradrenaline or a problem with the neuroreceptors may result in increased feelings of anxiety. These feelings may then lead to other responses in other parts of the brain and body. In the brain this may lead to difficulty remembering new information. The result is anxious animals have a hard time learning new things. They may also present with recurrent dermatological or gastrointestinal problems.

When a pet is fearful or anxious it may exhibit any or all of the 4 F's: the Fight, Flight, Freeze or Fiddle (displacement) behaviours. Which response is exhibited at any time depends on the pet's genetic predisposition, its previous experiences as well as the current environment.

A common misconception is that dogs (and cats) just need training and to be shown who is boss to stop unwanted behaviour. This can be detrimental for anxious pets and can make their behaviour worse in the long term. Dogs do not have fixed dominance hierarchies or dictatorial pack leaders who control everything. Many dogs, especially dogs with anxiety disorders, find dominance style handling very confusing and frightening. Many studies have shown that using punishment, even using a stern voice, can increase the chances of an aggressive response. Dogs, especially anxious dogs, need careful, kind and consistent handling to help them feel safe and secure in a stable environment. See **www.dogwelfarecampaign.org** for more information on training.

The treatment program for all anxious pets has 3 key areas – the 3 M's: environmental Management, behaviour Modification and Medication.

The following cases are indicative of the variety of ways that anxiety may be expressed and the effect it has on the human animal bond. They also illustrate how important it is that owners have realistic expectations of the pet and the crucial role they, as owners, play in being able to achieve a successful outcome.

But just as importantly the role of veterinarian is critical, not only in recognising anxiety as a medical problem, but also providing ongoing support for the owner. Of even more significance is that the veterinarian needs to appreciate that neither the owner nor the owner's behaviour is causing this common medical condition even though the condition may appear to be more prominent in the presence of the owner. Veterinarians need to stress to owners that punishment has no role in the treatment of the condition, any more than punishment has any role in the treatment of diabetes or any other disease process.

CASE 1:

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A dog presented to the clinic with sudden onset of aggression directed towards all people which first manifested itself after the owner had been absent from the house for a couple of hours doing her shopping.

The dog was a 6-year-old 8.2kg female spayed Shi-Tzu crossbreed named 'Missy'.

The dog had been the pet of the current owner's grandson. When he had acquired the dog as a pup it had lived inside and been taken everywhere with the grandson and his wife and treated like their baby. When they had a human baby the dog was put outside in a kennel, not allowed indoors and not given any time for play or interaction. The dog had been coping with the change and the grandson was not concerned about the dog's wellbeing. However, the current owner had recently lost her old Shi-Tzu and was concerned that Missy was not being given the attention she deserved. When the current owner adopted Missy she had been an 'outside dog' for eight months. Missy had pre-existing thunderstorm anxiety, which had been treated with diazepam as needed, with poor success, by the grandson. Missy had also once exhibited aggression towards the original owners when she hurt her stifle. This resolved with treatment of the pain.

Missy had been living indoors with the current owner for 3 months prior to the presenting event. The owner was mostly at home ▶

alone with Missy, but had 2 visitors, her daughter and a neighbour, who each came for an hour or so each day. Missy begged food regularly from the current owner and had gained a large amount of weight, sat with the owner on her chair for most of the day and was walked once daily for 20 minutes by the neighbour who visits. She slept in the owner's bed. Missy was very excited to see visitors, especially the neighbour who walked her, greeting them by approaching with tail wagging, jumping and vocalization in a high pitch.

Missy was generally anxious according to the owner and frightened by loud noises, thunderstorms and at times even rustling branches on the roof or window. She shadowed the owner in the house and trembled and whined when a noise startled her or the wind blew strongly. On one occasion she had bitten the owner when they were in bed and the owner presumed she had been frightened by a loud noise but was asleep when it happened so couldn't be sure. The owner reported she would often startle over nothing and start trembling and whining.

Missy displayed inter-dog aggression on lead while walked or when in the front yard with barking and growling. She was not known to have interacted with other dogs off leash at any time in her life.

Missy had been taken to the daughter's vet a month earlier for anxiety and was prescribed clomipramine 10mg twice daily. The owner, however, had only been giving Missy the 10 mg once daily and had been doing this for 4 weeks. There had been no change in her anxiety level.

On the day of presentation the owner had been shopping for 1 hour. When she returned home Missy cowered and growled at her and ran and hid under the kitchen table when the owner tried to approach her. The owner asked her neighbour to come over but Missy would not come out from under the table and growled with bared teeth and snapped when approaches were made. The daughter was then called in and she managed to get the growling Missy out from under the table with a blanket and she was immediately brought to the clinic.

On examination 2 months earlier for vaccination, Missy had not shown any anxiety and was compliant to examination and treatment at the clinic. On the day in question, Missy walked into the clinic on her lead with the daughter and seemed okay with wagging tail, ears forward and no hesitation to enter the consulting room. When Missy was approached she backed into the corner with her tail between her legs, ears flat, bared her teeth and growled. A towel was used to pick her up and a muzzle was fitted. A clinical examination could locate no focus of pain; however, Missy was growling and struggling throughout the examination.

The owner was too scared to take Missy home and very distressed at this sudden onset of aggressive behaviour. She felt the dog was betraying her kindness and should understand that she had rescued Missy so wouldn't hurt her.

My initial assessment was that Missy could have a painful complaint that could not be detected as she had exhibited aggression previously when in pain or that she had become anxious to the point of panic precipitated by an event or noise that had occurred while the owner was out.

I was unsure of the prognosis, as I was unsure of the diagnosis.

I elected to administer 1mg/kg of diazepam by intravenous injection and hospitalize the dog for observation and reassessment. Missy was kennelled in the hospital with food and water. The diazepam was administered at 5pm and as the clinic closed at 7pm Missy was left overnight. She seemed relaxed and happy in her cage. We did not attempt to provoke a reaction from her before leaving or re-examine her.

The next day Missy was happy and greeted the nurses with wagging tail and ears forward and was easily picked up, handled and examined. Oral diazepam at 1mg/kg was administered without issue and we informed the owner she could try taking Missy home and see how she responded. The owner was not going to continue to give the clomipramine as, although the dose given was below therapeutic levels, the owner felt it might have contributed to the current behaviour.

Missy did not exhibit any further aggression or fearful responses at human approach when collected or at home over the following week without any further medication.

At this time a revisit was booked to discuss behavioural modification to introduce stability to Missy's routine with a 'learn to earn program' and settle exercises, and desensitization and counter conditioning for the inter-dog aggression. The owner and the neighbour attended this visit. Missy was also prescribed diazepam for use during thunderstorms or periods of excessive anxiety.

Follow up 1 month later by telephone found Missy to be much less anxious in general but not a lot of progress had been made with the inter-dog aggression.

The owner was very pleased and happy that she and Missy had a mutually beneficial relationship.

WINNER

CASE 2:

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A cat was presented to the clinic for aggressive outbursts directed at the cohabiting cat (with which there had previously been a good relationship) and urine spraying.

The cat was a 3-year-old, castrated male, ginger Domestic Short Hair called 'Tom'.

The cohabiting cat was also a castrated male who was a littermate and had entered the household they currently resided in with Tom when they were both 7-weeks-old. They slept together and up until the week prior to presentation at the clinic had been normally social with each other. The cats were both housed indoors only. The owner had not previously thought of one cat being more dominant than the other as they had seemed perfectly content with each other's company.

The aggression between the cats was mostly at night with the owners waking up to a catfight yowling and screaming. The other cat urinated and defaecated at times, hair was pulled out of both cats and after they separated Tom would continue to hiss and stalk the other cat that crouched and hid or ran away.

Since the fights started the other cat was constantly hiding, slinking from feeding areas to hiding spots and not coming out to interact with the owners. Tom was sleeping less and didn't sit with the owners on the couch but prowled around the house, jumping up onto high spots and sharpening his claws and face rubbing on objects around the house more than usual.

Both cats used the 2 litter trays that were provided and the trays were cleaned daily with no house soiling or inappropriate elimination prior to the owner seeing Tom spray on a window to the side of the house 2 weeks earlier. She had since seen him spray in this location 3 further times. Urinalysis was unremarkable. The owner was distressed that Tom was being so 'mean' to his brother. The owner was also concerned about the physical injury that may occur and that the cats were no longer relaxing to be around. The owner did not appreciate the urine spraying but it was not as concerning to her as Tom's 'nasty' behaviour. She said that she would have to have him euthanased if this behaviour couldn't be stopped.

This behaviour was also a problem to the other cat which was constantly anxious to avoid Tom's aggression.

My initial assessment was that both behaviours were likely to be caused by anxiety related to the presence of another cat or animal and leading to redirected aggression and urine marking.

As the cats were housed entirely indoors I thought the prognosis for control of exposure to the supposed stimulus from the side window and resolution was good but if exposure could not be controlled then the prognosis would become poor.

The owners were instructed to put a cardboard or other covering over the side window where Tom was seen spraying after cleaning it with an enzymatic laundry powder.

They were also instructed to use a Feliway® diffuser in the main room the cats normally spent time to help with the anxiety being experienced by both cats and Feliway® spray around the window now covered over to discourage further urine marking.

Tom was prescribed 0.5mg/kg diazepam to be given twice daily for 3 days.

The owners were also instructed to obtain a cat trap from the local shire to see if they could confirm and possibly remove the source of the problem.

After 1 week there had been no further aggressive or spraying incidents seen and the other cat was coming out and interacting with the owners normally but keeping his distance from Tom.

Tom was sleeping more and seemed more relaxed.

In the following week the owners got the cat trap and on the first night trapped a large entire male cat that on presentation to our clinic was not micro-chipped or collared and very aggressive. It was assessed to be a feral cat and, after the appropriate council notification, euthanased.

The owners removed the window covering but continued to use the Feliway® spray and diffuser for the rest of the month by which time at follow up both cats were reported to be back to normal. At this time no further Feliway® was used and there was no relapse of anxious behaviour from either cat.

CASE 3:

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Statement of problem

A cat was evaluated because of a sudden onset of aggression towards the owner, involving spitting and swatting, as well as urination in unacceptable places.

Signalment

The cat was a 20-month-old desexed female 3.7kg domestic shorthair.

History

The cat was acquired at approximately 1 year old. Previous behavioural history was unknown. She had so far been a friendly cat at home, and was normally affectionate and interactive with the owner. The cat also seemed relatively at ease in the vet clinic, and around other strangers. The cat lived in a household with 3 other cats and 2 dogs, that all normally lived harmoniously. The owner lived with her adult daughter in a semi-rural setting. The cat lived approximately 50% indoors and 50% outdoors.

The owner had noticed a sudden change in the cat's behaviour when the cat began to urinate on the floor of the pantry. The owner had discovered at least 2 occurrences. The cat had previously been well litter trained, as were the other cats in the household. There were no new introductions to the household, and the owner was not aware of any stray cats around, although due to the owner's location this could not be entirely ruled out.

Around the same time as the onset of urination in unacceptable places the cat had suddenly begun showing aggressive behaviour towards the owner. When the owner tried to pat her or pick her up, the cat would hiss, swat and growl. The cat also seemed unwilling to move as much as normal, and was not jumping up on furniture as was her normal habit. There had been no prior reports of any other behavioural problems.

The cat's appetite had remained stable, and there was no history of polyuria/polydipsia or other medical issues.

The owner was concerned by the cat's sudden behavioural change, as well as the potential for scratches or bites when handling was attempted. The cat's choice of location of urination was also frustrating for the owner. The owner and cat had previously shared a healthy bond, but threats of aggression and inappropriate toileting were placing an understandable strain on the friendship.

Physical Examination Findings

The cat was in lean body condition, and most of the rest of the examination was unremarkable. The cat coped well with the examination, until the spine was palpated. There was repeatable spinal pain over the thoracolumbar junction, which resulted in the cat trying to move away and hissing. By this stage, the cat had become stressed, and no further neurological exam was possible. No laboratory workup was done at this point.

Initial assessment

The sudden onset of aggressive behaviour was attributed to the cat's spinal problem, where the cat seemed to anticipate handling or movement which would result in pain. The urination out of the litter tray was less clear – possibilities included reluctance to move to the litter tray (at the other end of the house), an inability to get into the tray due to the spinal issue, or anxiety induced by pain. The pantry may have appeared to be a good option to the cat – a dark, relatively private place with a flat surface. The voiding of a full bladder would also be inherently rewarding for the cat, which may have lead to repeats of the behaviour. The smell of urine may have also contributed to the repetitive nature of the urination in this location.

While the exact cause of the spinal pain was not known, it was suspected that some form of trauma may have taken place. The cat was a known tree climber, and a fall was suspected. Intervertebral disc inflammation was likely, with muscular tears, thoracolumbar vertebral or cord trauma, osteomyelitis and neoplasia being considered as differentials.

Management Plan and Prognosis

The management of this case was squarely aimed at resolving the cause of the cat's anxiety, which was thought to be her spinal pain. As the cat's hydration was normal and no other known medical conditions existed, a short course of meloxicam oral liquid was commenced. The owner was advised to shut off access to the pantry, and an enzymatic cleaner was recommended (repeated use). Litter trays were to be made more easily accessible so the cat would not have to walk as far. If needed, the owner could cut the front out of the cat's normally favoured tray. The owner was also advised to leave her alone if showing any signs of aggression. The prognosis at this stage was fair to good. Within days, the cat was moving much more comfortably, and was no longer swatting or spitting when the owner approached her. The cat returned to using the litter tray almost immediately. Even when the pantry door was left open, there were no attempts to urinate there again. The meloxicam was discontinued after 7 days, and by this stage the cat was behaving normally. ▶

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

Had the cat not responded, a more thorough workup would have been required, including a neurological exam, spinal radiographs (and CT/myelogram if required), as well as urinalysis and biochemical/haematological screening.

Had the aggression persisted beyond the resolution of spinal issues, a strategy involving the above plus environmental management, behavioural modification and long term medication (e.g. fluoxetine and Feliway®) would have been instituted. If the inappropriate urination had persisted beyond resolution of the spinal issues, a strategy involving the above plus addressing litter tray problems, location problems, substrate problems, anxiety related problems and cleaning issues would be appropriate.

CASE 4:

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'Harvey' was a 5-year-old desexed male Dachshund who had recently been rehomed to a family from a breeding property. Harvey had had limited human social contact in the previous 5 years and had been housed with 10 other dogs. He presented to the clinic for a behaviour consult because he was showing signs of timidity and aggression towards the male members of the family but particularly the older son who was 17-years-old. When the son entered the room, Harvey would bark and growl at him until he retreated. If the son approached him, Harvey would growl and then run away still barking. He was strongly bonded to the female of the house (the mother) and followed her everywhere in the house or the yard. When she was not at home, he would hide outside in his kennel. When the mother was home and the men were not, Harvey would confidently leave her side and roam around the yard on his own. When they returned, he again would become a 'Velcro-dog' and stick by the mother at all times.

The barking towards the son was seen to be a huge problem as it was incessant and as the dog would not leave the mother's side, the son was beginning to avoid being in the same room as the dog and therefore the mother. The barking and growling was unable to be stopped while the son was in the room. The human-animal bond in this case was extremely strong as the owner of the dog was suffering from a terminal condition herself and had only a limited amount of time left (6-12 months). Her emotional bond to the pet was, by her own admission, far too strong and based on the emotional situation of her medical problems. She had taken on the task of resolving this situation to leave her family with a happy dog when she passed away. She could not stand the thought of causing the dog to be re-homed again if she did not rectify the situation in the time she had left. We discussed whether working on this problem was a sensible or realistic option for her but she would accept no other alternative.

It was assessed that the dog was showing signs of anxiety towards the son although the reason for this was unknown. Harvey perceived the son as a threat and was displaying behaviour to avoid interactions with him. The son was the only member of the family who did not go to the shelter to pick out the new dog when Harvey was adopted but he had never shown any aggression, 'dominance' or even much attention towards Harvey. It was also seen that Harvey had formed an unnaturally close bond with the owner although he did not show signs of separation anxiety when she was not at home. It was discussed that this problem alone could have a reasonably good prognosis although the effect of the emotional situation within the household may affect that and that the time-frame that we had to work within also put extra pressure on the outcome. A guarded prognosis was given.

Harvey was placed on Fluoxetine medication and a program of training, desensitization and counter conditioning was prescribed. He was also put on the 'nothing in life is free' routine. Calm behaviour in the visual or auditory presence of the son was to be consistently rewarded at all times and once basic training had been achieved, this was to be attempted at times when the son could be heard. Recommendations to disallow Harvey to sit with the owner on the couch were made and he was provided with a bed at her feet. Initially the son was to completely ignore Harvey and all members of the family were to ignore the barking and growling behaviour. When he got to the stage of being able to sit on cue in the son's presence, he could then attempt to approach Harvey but was to offer treats at all times. Advice was given for the son to avoid eye contact and to adopt body language and posture that was not challenging to Harvey at all.

Over time, Harvey learned to accept the presence of the son and the owner was extremely diligent in her behaviour modification program. No attempt has yet been made to wean Harvey off the Fluoxetine. At last contact with the owner, she was extremely happy with the progress made and was confident that it would continue to improve. Harvey still barked when the son entered the room but quickly stopped and was able to be asked to lie still on his bed in the son's presence. They were again able to sit together as a family to watch TV or to eat dinner in the dog's presence. The owner was confident that the dog would be able to continue to live with the family once she passed away. The human-animal bond was stronger than ever. It was decided to continue the medication to help ease the transition once this occurred.

Postscript
I treated this little dog right at the beginning of my behaviour course and now I understand a lot more about behaviour modification strategies. In hindsight, it would have been useful if a more formal de-sensitisation and counter-conditioning program had been targeted towards the older son, whom Harvey had taken a dislike to. It had been suggested by me to the family but unfortunately it was not implemented strongly enough and I had concentrated more on providing Harvey with the 'Nothing in life is free routine'.

As the owner progressed in her treatment of terminal breast cancer, regular medication of Harvey fell by the wayside, as did his behaviour modification exercises. The mother passed away in March 2012 and unfortunately Harvey is as bad as ever with the older son. He is much more relaxed with the father and the younger sons but constantly barks at the eldest. The family seem to just cope with the problem and live with it; further help was declined, as was further medication.

CASE 5:

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Problem

The cat had been recently adopted by the owners from a breeder that had used him for breeding purposes. The cat came into a household with 1 other cat (female spayed) that had been the only cat in the household for its whole life (5 years). Initially the cat was very unsocial and hid under the bed for most of the day. This lasted for about a week and eventually the cat started to gain confidence and venture out and around the house. Both cats were kept indoors at all times. The cat was not used to being an indoor cat in the past and had been kept in a cat run out in the backyard. The amount of socialization with humans was very limited and with cats was limited to matings and to visual interactions only.

The cat started to interact with the initial household cat after about a week of hiding and meowing under furniture, but the interaction was not beneficial for both cats. He became increasingly forceful in his play and would ignore warnings or negative responses from the female cat. This in turn stressed all involved and the owners as well. The female cat became very unsocial and would hide from the new cat. The new cat started to pace around the home in search of the female. He would also become stressed if he was restricted access to her or to the owners of the house. This made isolating the new cat difficult because it would cause continual vocalization in the home, making sleep and relaxing difficult for the owners. The problem in this case was to multiple individuals. The initial cat (female) changed her behaviour from a social loving cat into an unsocial unhappy cat. The owners were losing patience with monitoring interactions and growing tired of the amount of vocalization the new cat was making while prowling for the female cat. The new cat was showing signs of anxiety both initially (hiding) and after gaining some confidence (continual pacing, vocalizing) when he was unable to gain access to either the female cat or the owners.

Initial assessment and management plan

My initial assessment of this case was that the new male cat was having difficulties becoming accustomed to the new lifestyle. It's hard to say that the cat had no social interactions during its growth but from the description it is a definite possibility. If this had been the case a sudden change in lifestyle into a well populated area can cause cats to be fearful, stressed, or show social anxiety. This was the case with this particular cat, but over a short period of time the cat started to became overly social to the point of having signs of separation anxiety when removed from the owners and the other cat.

With this case I suggested to start medicating with Clomicalm® and using Feliway®. I also gave the owners a handout on suggestions of how to introduce a new cat to the household (a bit late but suggested starting from scratch – better late than never). I informed the owners that the process would take time and at this stage the owners became very hesitant. They had been dealing with the situation for about a month and were approaching their limits of tolerating the cat's behaviour (mostly the vocalization and the stress the other cat was undergoing). The consult was cut short because of the owners unwillingness to proceed further once they were informed about the possible timeline (months) and also the chance of difficulties in reducing unwanted behaviours (vocalization and obsessing of other cat). The owners were willing to search for a new home for the cat and I recommended one with no other cats in household and an owner(s) that was able to spend more time with cat. If a new home was unable to be located the owners would consider euthanasia.

Prognosis:

Prognosis for this case was guarded. The owners had a limited time frame that they desired the male cat to correct its behaviour. They had allowed the situation to escalate before finally bringing their issue to someone (myself), so they were at wits end. Once informed about the process and how long it may take to control vocalization/prowling – if it was able to correct at all – the owners appeared to give up and found the only resolution was to find the cat a new home.

Was it resolved?

The male cat was re-homed within about a month of the consult. The new owners were an elderly couple with no other cats. They were both retired and able to devote more attention to the cat than its previous owners. In a way it was resolved – although the cat's behaviour may not have changed, the new household was well informed about that behaviour and willing to take the cat in.

WINNER

Reply to C&T No. 5270 Flea Treatments: Frontline® resistance (Dec 2012, Issue 269)

C&T No. 5299

C&T No. 5270

C&T No. 5271

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Aussie Fleas are like Aussie Men – an Incomparable World apart from their British Counterparts.

I tell every European vet who comes to work here to take everything they ever did with fleas in the UK and bin it because it ain't going to work over here. Aussie fleas are the Rambo version of fleas with environmental habitat variation that just doesn't exist overseas.

Just like Aussie men the Aussie flea is a tough creature – not easily vanquished and very stubborn and persistent in how they go about their daily life in this amazing country.

1. **Environment:** We don't reach -10°C in the soil here in the most highly populated areas of Oz for one night let alone the 10 needed, so fleas survive. Frontline® was originally launched here with a 12 week efficacy from overseas trials – it lasted 3 weeks if we were lucky. For the first year post launch, complaints by vets were ignored by the manufacturer until finally sheer numbers must have made them accept that monthly not seasonal application was the baseline for this product in Australia.

   I advise Advantage® based[1] products by the pallet-load – great for persistence in our sandy warm cocoon soils and great for renters as its environmental activity means households may not have to be nasty sprayed when renters leave as the Advantage® off the dog in environment treats the property safely.

2. **Repellency:** Advantage® and Frontline® make no claims to be flea repellent – the flea has to get ON the pet, come in contact with the chemical and die off.

   Fed, Fat, Slow lumbering fleas are a product failure.

   Skinny, Fast moving fleas are an irritated dying flea from product efficacy not product failure.

   KNOW HOW YOUR PRODUCT WORKS and stop saying it's a failure when it is not.

3. **Water Immersion:** Here dogs swim much more often than in the UK as we have greater and longer access to swimmable water. Dogs just don't go to the beach once a week for A **swim** or have A **shampoo**. Here they go to the beach for **The day** and swim **Many times** then go home and get hosed down or shampooed. Often, back they go to the beach the next day etc. And for those who live near lakes and rivers, swimming is a daily occurrence all year around. So I don't buy any manufacturer's data on water resistance for a topical product.

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Back to TOC page

4. **Shampoos:** Clients often wash their dogs weekly here all year around so it amazes me how many vets recommend a Flea/Tick shampoo to be used before Frontline® when so many such shampoos have piperonyl in them. Piperonyl inactivates Fipronil so you might as well squirt it up the wall as on the dog and that is not Frontline®'s fault. User error!

5. **Product Binding:**
(a) Sebum:
Frontline® came out with a 2 day gap between application and washing as it needs to bind to sebum and be deposited into the sebaceous glands for pulsatile release. Then suddenly, when product use guidelines for Advantage® instructed users to apply the same day, the instructions for Frontline® then changed also. But the reality was that permission to use the same day as the shampoo was the decline of the product and caused an upsurge in complaints.

   Use Frontline® if you wish but leave a 2 day gap and use Capstar® for those 48hrs.

   Virbac now has a new generic priced fipronil in a different vehicle base so some Frontline® allergic dogs may tolerate the Virbac rather than the Merial version.

   (b) Hair:
Selamectin/Revolution® can be impeded by hair.

   Advantage uses hair to disperse.

   Permoxin binds to Clean hair.

   So if you have just shaved and then washed a dog, then maybe Revolution® might be a better one-off topical application or oral Capstar® or Spinosad products until you either get sebum back in production for Fipronil or hair regrowth for Advantage® use.

6. **Elanco – support the companies that support vets**

   If your preferred product doesn't work for dogs which regularly swim or are regularly well washed (forget the company's assurances that it is water fast) then advise vets to recommend Elanco's Comfortis® for their patients. Vets must point out to owners that Comfortis® **must not be fed to dogs on an empty stomach or given to epileptic dogs.**

   Note: Should the dog vomit Comfortis® a second time then Elanco will reimburse costs if the product was purchased from a vet. Elanco does not support claims from products sold from any other outlet.

   So support Elanco where it is the best suit for that pet but know when **not** to use Comfortis®/Panoramis® fully before you dispense it and report any ADRs to the APVMA – even vomiting. My qualifications that allow me to make the above statements are:-

   Having been in Vet practice in the UK and dated several English men in various global locations, I have been in Vet practice now in Oz for over 20 years and stayed married to the same Aussie male for 23 years so I reckon my pontifications have great contextual validity.

Also, the December 2012 C&T No. 5276 Useful mnemonics from the college days of 1960s refers to Phil Rogers' experiences, not mine. My college days were the 1980s in case those of you who know my husband Mark might think I cradle snatched him!

Reference

1. Accumulation & Persistence of flea larvicidal activity in immediate environment of cats treated with imidacloprid – *paper available from Bayer*

Reply to Merial Australia Pty Ltd's comment in the same issue

I am bemused – if I am being kind – by Merial's lack of information that allows them to write to C&T claiming that **resistance** to Fipronil *in the field* has not been documented. Initially a clever play on words as it allows them to exclude the MK1 strains of fipronil resistance fleas used in laboratories for research.

BUT, there are also documented cases *from the field* (cf reference below for starters) so **resistance exists[1] and is documented in both the laboratory and the field.**

I, too, have seen many fat fleas on properly treated Frontline® dogs so the product does **fail**. For the company to claim otherwise is disingenuous and insulting to vets.

Reference

2. Efficacy of Nitenpyram Against a Flea Strain with. Resistance to Fipronil... Suppl *Compend Contin Educ Pract Vet* Vol. 23, No. 3(A), 2001. Page 5

Reply to Perspective No. 93 Round Table Discussion Part 1 Replies to Tick Paralysis in the cat

1 2 3 4 5

Go to the e-book and rollover to read Tick Paralysis C&T 5251

C&T No. 5300

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Anti-Tick Serum – IV or IP for cats?

I recently attended a meeting of an emergency vet service on the Northern Beaches of Sydney (tick country), at which around 15 practices were represented. I asked for a show of hands regarding the preferred route of administration of ATS to cats and got virtually no support for IV infusion. Which prompts my question – why has the IP route become the dominant (near exclusive) recommendation? I offer the following comments based purely on experience and observation.

1. It is easier and cheaper – sure, but not a good reason on which to base any treatment.
2. Maybe the IP route filters out some of the 'nasties' in the serum? – ATS does appear highly particulant, possibly contributing to adverse reactions if given IV. Any benefit to cats would also apply to dogs, and yet few vets advocate IP serum to dogs.
3. It is safer – why? In my hands, the IP route is an extremely variable technique for drug delivery. The easiest drug to gauge IP effectiveness is euthanasia solution. I've had some animals die within seconds of IP lethabarb, and others that have required a 2nd or even 3rd dose to achieve the desired result. No doubt my technique is to blame, but I bet I'm not alone. I would assume that the rate of uptake of IP ATS is equally erratic, with some cats receiving a therapeutic dose within minutes and others not receiving any at all. Safer? Surely the safest use of a drug is to administer it at a known effective dose and a known rate, and for serum of any nature this means the slower the better.

We use IV ATS in all cat patients including those with known prior hypersensitivity reactions. Since using the following protocol, we have treated 482 cats with no fatal adverse reactions. The vast majority of cats have no observable adverse reaction, maybe 5-10% require the serum administration to be stopped for 30-60 minutes, and only 3 cats have required adrenaline injection. We get a consistently good response to treatment, and usually suggest a good prognosis for cats in stages 1 or 2. On average, comparing cats of similar presenting stage, there is a clear difference in the rate of recovery for cats treated with our technique versus the cats treated with IP serum that are transferred back to our hospital from the emergency vet.

Here's our protocol:-

1. Premedication with methadone and acepromazine (ACP). The dose rate will vary with the degree of toxicity and the anxiety level of the cat. Methadone 10mg/mL at 0.02 - 0.03 mL/kg (0.2 – 0.3 mg/kg) and ACP 2mg/mL at 0.02 - 0.05mL/kg. I still use these unfashionably high dose rates if necessary in order to perform Step 2 with a minimum of struggle/stress. We do not administer antihistamines or corticosteroids.
2. Insert an IV catheter and drip line attached to a 100mL bag of saline. We use an injection T-port to connect the catheter to the giving set. Take out 50mL saline in a large syringe and put aside for later, leaving around 30mL left in the bag (primed drip line uses 10-20mL). Add ATS to bag – 5mL for most cats with 1-2 ticks regardless of stage of paralysis, up to 10mL if multiple ticks. Place cat is a quiet cage, run drip at 30mL/hr, and leave alone for 5-10 minutes.
3. By the time the diluted serum reaches the bloodstream (10-15 minutes depending on length of giving set) the cat should be as relaxed as possible in the cage. Now's the time to watch closely for adverse reactions, and they will usually occur in the first few mLs if at all. Diarrhoea, vomiting, weak pulse, pale gums, restless or struggling cat. If any symptoms are observed, STOP ADMINISTRATION OF ATS – try doing that with IP serum. If necessary, administer adrenaline via the T-port – we use 1:1000 at .03mL/kg but would be happy to learn of a better dose. Leave the cat alone to recover.
4. 30-60 minutes after adverse reaction (assuming cat is still alive), restart the ATS drip at a slower rate 10-20mL/hr. I've noticed that most cats don't react a 2nd time – any ideas why?
5. When drip is finished, add the 50mL syringe of saline into the empty bag and slow rate to 5-10mL/hr. This will flush the ATS remaining in the drip line as well as provide a little rehydration. For the older cat with questionable renal function, we will maintain a slow IV drip 5-10mL/hr for the first 24hrs, increasing the rate if cardiopulmonary function allows.
6. I have no particular expertise in the subsequent management of the more life-threatening complications of tick poisoning, although this is more of a dog problem. Cats have a habit of staying alive.

By using this protocol, I have control over the ATS – I know the cat has received the appropriate dose at the slowest rate, I can suspend treatment as necessary, I can immediately counter adverse reactions through the IV access, and I can leave the cat in peace. The main danger is failure to watch the cat for adverse reactions – all too easy to set up the IV drip and plonk the cat in a cage and forget about it!

By the way, we use the same protocol for dogs – a significant percentage of dogs will have adverse ATS reactions, maybe not as celebrated as the cat's but an equally good reason to slow the rate of administration.

In my opinion, the blanket recommendation to treat cats with IP serum needs to be seriously reconsidered.

Reply to Perspective No. 93 Round Table Discussion Part 1 & 2 Replies to Tick Paralysis in the cat (Sept & Dec 2012, Issues 268 & 269)

Download Perspective 93 Round Table Discussion – Part 2: Replies to Tick Paralysis in the cat C&T No. 5193 by Frank Gaschk, Dec 2011, Issue 264

Or go to our Dec 2012 ebook to read a High Res PDF of this article.

Download Part 1 (C&T 5251 from issue 268) of this discussion from Issue 268, Sept 2012. Go to page 46 to download)

1 2 C&T No. 5193

C&T No. 5301

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Where is CVE's Surgery Conference in 2013?

Hint : See calendar on page 9!

Don't forget to provide your full name.

Q from Kim

I presume you get huge numbers of animals with tick poisoning around you? How long do you think it takes from attachment to poison production from the tick? The quoted is 3 to 6 days but the research was done in 1945 – see Rick Atwell's comment. And any 'tick case tricks' up your sleeve? I have had the worst tick case I've seen – though not terrible by your standards I'm sure – and I'd like to be better prepared for the next one. I usually only see 1 a year, but with the successful fox and feral cat eradication in this area, even the bush turkeys are depositing paralysis ticks (anecdotally – seems unlikely a bird could be part of a mammal tick life cycle but certainly the possums are). I have had 5 already this year.

Reply from Owen

Yes, we are in the middle of our tick season now. We see fairly large numbers each year, but not as many as they get up at Mossman and on the Tablelands. **74 cases so far this year (2012) and have used 1,295mLs of anti-tick serum (A/S).**

I don't think I have any special secrets. I try to keep the treatment fairly simple, partly because I reckon it doesn't matter how fancy you get with your treatment, the success rate does not change much; and also it is really important with cats to minimise the stress from too much handling etc. But I am sure you will be fully aware of that. ▶

My routine for cats is to anaesthetise them first thing with about

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Back to TOC page

1mL Alfaxan with a bit of ACP in the same syringe I/V, before I do anything other than be convinced it is a tick case. Then I can do the rest while they are asleep for about 20-30 mins without stressing them out.

Then:-

- Express the bladder
- Put 5 to 8mLs A/S directly IP with a 18 or 19G needle
- Attach a fluid bag to that needle and give about 100mL Hartmann's Solution IP (so I am less worried about them dehydrating during the next 48 hours)
- Search them quickly for any more ticks
- Soak them with Fido's Flea and Tick wash
- Wrap them in a towel and put them into the quietest cage we have with a curtain on the door
- Either warm them or cool them depending on body temperature
- Then basically observe and wait with fingers crossed.
- We may suction throats if necessary. We administer oxygen sometimes, probably should do more proactively. We withhold food and water until we think they can cope with it. Any respiratory signs and we will start injecting Clavulox and Baytril.

Our success rate is pretty good, but it is certainly not 100%. I tell people it is in the high 90 percentile and am careful to warn clients that I cannot guarantee success, and also that I cannot predict the outcome. Usually if they come in to us early enough and we give a good dose of A/S they will be OK. BUT sometimes ones that seem only mildly affected at the time of treatment will deteriorate despite our efforts and be determined to die; yet the opposite happens occasionally and patients that look near death on presentation make a miraculous recovery!

I used to get really upset by the unpredictability, but after so many years of seeing that the success rate per year is good, and having the warning of the clients routine pretty polished, it is not such a worry.

The other trick I use to reduce the stress (on us that is) is that **we have a standard estimate for total treatment cost and we get the client to pay the consult fee plus half of that estimate as a deposit before treatment.** Then if all goes well, they pay the balance when the pet goes home. Otherwise, if their pet dies, we do not ask for any more money. This has worked wonderfully – no need to worry about chasing payment for a dead pet; can concentrate on offering sympathy etc. The small monetary loss for the small numbers of deaths each year is negligible; besides the deposit covers the costs anyway.

Comment courtesy of:

Rick Atwell (Retired Professor)
BVSc PhD FACVSc
M. 0409 065 255

Qs from Kim Kendall & As from Rick Atwell:

Do you think this might be that the Frontline® was not 'real' (there are reports of active-ingredient-free topicals coming from Thailand and China)? Could this situation be a real possibility?

I always give Proban® when I see a cat with a tick (on the basis it should kill any unfound ones who are attached at least). However, I have just had a cat with 1 tick (removed) and Proban® given who was fine for 36 hours – then into grade 2 – and another tick found (5mm across the back) still alive.

Does moxidectin/milbemycin work on *Ixodes holocyclus*?

Will the Seresto® flea collar kill *Ixodes holocyclus*? It is flumethrin so it should…

Re Proban®, it has as many failures as do the others but as it's perceived to be cheaper and as older people generally complain less, perhaps that explains fewer client complaints? I have seen the original data on Proban® done a long time ago but, even allowing for our increased knowledge of how to do trials, you would be scathing if you saw the results. So to me the support for Proban® is low. You need a high dose and there is little cat data; it's all data from dogs and extrapolated. All fail. If TAS has been given, and there are other ticks, it will be OK for a few days as half life of TAS is days, not weeks. The best data I have ever got in random controlled blinded (as much as is feasible in TT trials) was with 2 products i.e. collar and another different mechanism topical 100%. Of course this was never published because of ownership of data re ticks and efficacy. If you watch ticks load, as I have done over many hours in a ward full of dogs, they are very fussy. They do not attach readily and are virginal in their selection of host and site even if they take a long time to attach there is very little hair contact and so very little chance of chemical absorption. They walk so high in the hair coat like water spiders on a pond – no water contact except for their boots! So harder to kill ticks generally because of their modus operandi and then single site usually hair contact if there but also attach to clear hair areas e.g. clear of inner pinnae. So one can see why products fail as there is so little chance of intoxication in many ticks.

Is there an actual documented time of minimum tick attachment to poison production ?

It's now well established, due to gene up regulation time, that *Ixodes holocyclus* as a species not *Ixodes holocyclus* specifically (as experiments not done) but it takes 3 days to get attachment right. Then genes for salivary toxins upregulate and function, then gut enzymes then gut transport for e.g. iron then reproductive genes develop. Three day fits clinically where tick attachment is known, fits experience where 1 to 10 ticks per dog (up to 5 in cats) will not induce signs until a day later i.e. 4mm across on the post 72 hour observations. All being equal, clinical gene studies and experiments (controlled attach time, humidity caged etc) all say 3 days so can use as a guide for travelling dogs etc. Contact times more likely to be longer with old ticks or too cold/hot conditions that do not encourage biological activity and so slower feeding and so slower intoxication as secrete - suck - secrete cycle.

I have no data on cats but IP TAS would be historically done e.g. 40 years ago routinely. Also cats' unique diaphragm access from peritoneal cavity supports the use of IP. IP is also so easy in a cat compared to dogs (who bent the catheters much more easily as thicker wall etc). IP prevents problems from rapid dog sera insult from IV administration (still low at about 1%) and yet the TAS will be IV pretty quickly (no times at my fingertips but have data in Australian lymph text massive tome on lymph which also supports my hypothesis on why dogs and cats differ so much with fluid accumulation with RSCHF).

You don't have to be a CVE Member to submit articles or replies and comments.
We welcome contributions from Vets, Vet Nurses and affiliated professionals working in animal welfare.
Send them to: cve.publications@sydney.edu.au or by post or fax.

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