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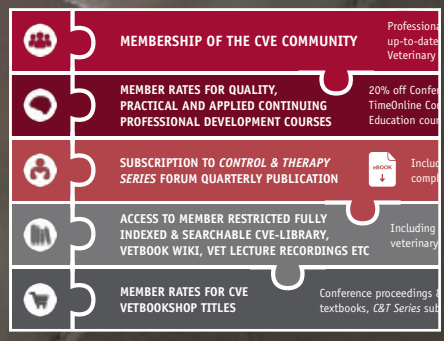

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COVER IMAGE: 10-Month Old Ellie Surridge unstaged – courtesy of her mother Dr Charlotte Surridge

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FROM THE DIRECTOR

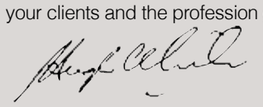
As I travel across the country to various CVE events including seminars and workshops, I am always heartened to hear how many of you appreciate this publication. While the C&T is not technically peer- reviewed, many of the articles contributed are of a very high quality and hopefully add another dimension to a busy practitioner's life. Our editor, Lis Churchward, does a great job squeezing as much information into each publication as she can and yet there is always a waiting list of articles still to be published.

In this edition, there is once again a great variety of articles with something of interest for everyone. You may wonder how an article on the control of serrated tussock has made it into C&T, but given the widespread distribution of this weed, we feel sure that the information contained may be useful to the clients of many of our large animal practitioners. There are a number of articles sourced from our DE files and apart from being interesting and challenging case studies, they show the time and effort that our distance education participants put into their work.

There is a comment from one of our regular contributors, Marshall Thornton, who has been blown away by doing his first distance education course this year. While Marshall is at an age where many of us are considering retirement, he continues to want to improve his skills and knowledge to the benefit of his patients and clients. He has enjoyed the sonology DE course so much that he has signed up for another DE course next year, despite still being very busy in small animal practice. Over the years we have had many participants studying our long DE courses in order to gain a greater understanding of a specific discipline area and uniformly they have reported that they have found the extra work demanding, but at the same time extremely rewarding in both personal and financial terms.

Zoe Lenard, one of our newer distance education tutors, has written a perspective on thoracic radiography for this C&T. Despite being a busy imaging specialist and mother of young children, Zoe has found the time to rewrite the three DE modules on abdominal imaging, present at CVE seminars and post case studies on the discussion forum of the diagnostic imaging course.

Later this year we will be sending out a survey, which we hope you will take the time to complete. It will give you an opportunity to give us feedback and commentary on our products and services. I am sure that you have noticed how much our marketing has been revamped this year. In May we launched our new membership structure and this has been hugely successful, with many practice owners taking up the new option of practice membership and many part-time veterinarians joining for the first time, or rejoining. We started 2014 with a lot of new faces at the CVE and this has created enormous enthusiasm and energy in the office, at a time when our organisation is about to celebrate its 50th anniversary (next year). So, when you receive an invitation to complete the survey, please take a few minutes to click on the link and send us your feedback, so that next year we can do things even better for you, your team, your clients and the profession generally.



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27 Sep	Advanced Echocardiography	Melbourne	**FULL**
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11 Sep	Rabbit Medicine & Dentistry		
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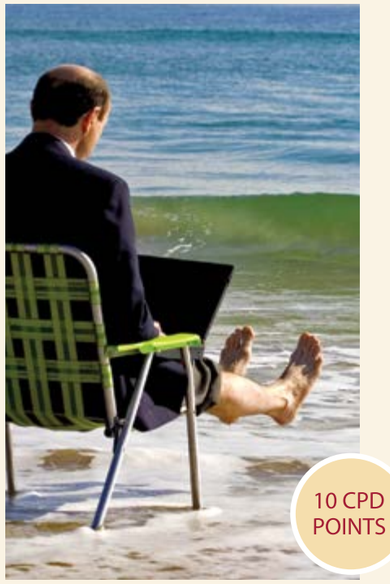
9 Oct	Referring a Behaviour Case: To Whom & When		
23 Oct	Medical Management of Uvetis & Glaucoma		
6 Nov	Surgery – Acute Abdomen		



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15 Sep	Small Animal Behaviour	with Dr Jacqui Ley
22 Sep	Dermatology	with Dr Linda Vogelnest
13 Oct	Wildlife	with Dr David Blyde
3 Nov	Pet & Aquarium Fish	with Dr Alex Rosenwax

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THANK YOU TO ALL CONTRIBUTORS

… and more C&T articles and Perspectives are needed

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

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DE FEEDBACK

This year I'm doing my first DE course, Sonology. Not only am I learning how to use the ultrasound properly, but also I am reviewing and consolidating internal medicine. I believe it is aiding me to be a better practitioner.

I have booked the DE Thoracic radiology course for next year.

I commend you on providing these DE courses. The format this year enables part courses, which will reach more vets who might find the 'full monte' full year course a bit daunting. Well I did, but am thoroughly enjoying the heavy work load and the highly talented tutors.

The C&Ts continue to be as excellent as ever.

Cheers

Marshall Thornton (Long-term CVE/PGF Member)

Unsolicited email sent to CVE June 2014 and published here with permission.

Established in 1965, the C&T is the brainchild of Dr Tom Hungerford, first Director of the PGF (renamed the CVE) from 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.'

MULTIPARTITE NAVICULAR BONES OF A RACEHORSE

C&T NO. 5406

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CASE REPORT
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Ballina NSW 2478

Introduction

Bipartite and tripartite sesamoid bones have been described in the horse previously. This case study records a racehorse with *concurrent quadripartite, tripartite and bipartite navicular bones (distal sesamoid bones).*

Clinical Evaluation

A 3 year old thoroughbred gelding presented with bilateral, forelimb, weight bearing lameness particularly when turning to the left or right on a hard surface. The horse was being prepared for flat racing, however persistent recurring 3/5 lameness interrupted this program. Shortening of the anterior phase of the stride was evident in both forelimbs when walking and a positive pain response was elicited to provocative pressure applied across the heels of both fore feet using conventional hoof testers.

The gelding was administered 2.5mL romifidine (Sedivet) intravenously and digital radiographs taken of both forefeet utilising the standard views including palmaroproximal-palmarodistal oblique (Pa45Pr-PaDiO) or navicular skyline views. The navicular bone or distal sesamoid bone (DSB) of each forefoot was observed to be in sections suggesting the possibility of multiple ossifications centres or acquired fractures.

Normally the DSB of each foot is positioned palmar to the junction of the second and third phalanges as a single bone entity with a transverse long axis. Each DSB possesses two surfaces, one articular and the other a tendon surface. Each has two borders, proximal and distal, and two blunt-pointed extremities. Each DSB articulates with both the second and third phalanges and normally ossifies from a single growth centre.

The owner requested the horse be euthanased thus affording an opportunity to dissect the feet.

Autopsy Findings

Dissection of all four distal limbs revealed significant degenerative, macroscopic changes in the navicular bones, and the third and second phalanges of both fore feet and the right hind foot. The left hind navicular bone appeared normal. Clinical lameness in the right hind had not been observed.

The insertion of the common extensor tendon on the extensor process of the third phalanx in each of the four feet appeared normal however the deep digital flexor tendon (DDFT) of each affected leg appeared abnormally compressed when passing over each of the navicular bones of the fore limbs and the right hind limb.

The left forelimb dissection revealed a *quadripartite* navicular bone with three of four pieces similar in size and a fourth smaller portion. The bone pieces showed signs of remodelling at the articulation with the second and third phalanges and the edges appeared rounded and worn leading to the conclusion that wear had been developing throughout a significant period of the horse's life. The articular surface of the distal end of the second phalanx showed abnormal erosion marks in the medial and lateral condyles.

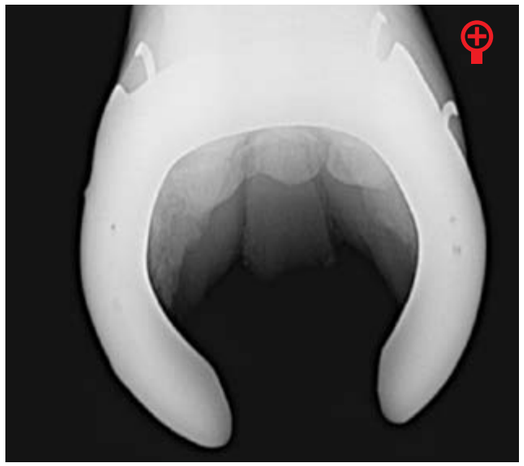

Figure 1A

Figure 1B

Figures 1A & 1B. Left fore quadripartite navicular bone

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Dissection of the right fore distal limb revealed a *tripartite* DSB with the medial piece being larger in size and the lateral portion malformed and associated with a smaller fragment. The separation occurred lateral to the central eminence. The larger portion showed signs of remodelling and the edges between these two larger portions appeared rounded and worn, leading to the same conclusion as with the left forelimb that this abnormality had been longstanding.

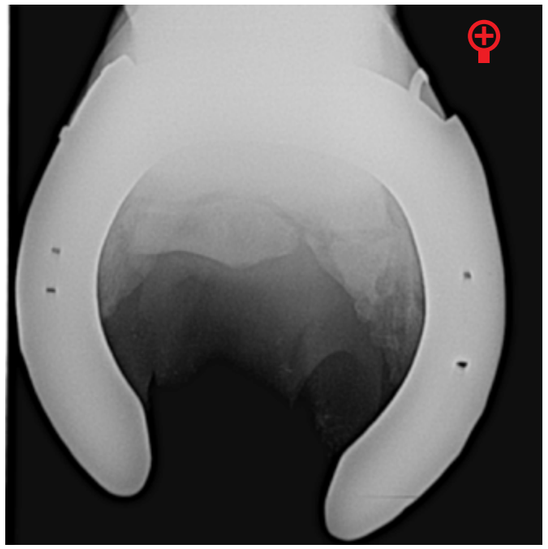

Figure 2A

Figure 2B

Figures 2A & 2B. Right fore tripartite navicular bone

Dissection of the right hind foot exposed a *bipartite* navicular bone with the medial piece being 1/3 in size and the lateral portion 2/3 in size of the navicular bone had it been one piece. The separation occurred medial to the central eminence. The larger and smaller pieces showed signs of remodelling and the edges between these two portions appeared rounded and worn, again leading to the same conclusion as with the forelimbs that the DSB had been in this state for a significant period of the horse's life. The facies articularis sesamoidea of the third phalanx showed an asymmetrical wear pattern and the articular surfaces of the second phalanx showed abnormal erosion marks in the medial and lateral condyles

Figure 3. Right hind bipartite navicular bone

The left hind limb dissection revealed a normal navicular bone and the third phalanx showed what was considered a normal wear pattern at the facies articularis sesamoidea. The joint surface of the second phalanx showed a normal articulating pattern in the medial and lateral condyles.

Figure 4. Left hind normal navicular bone

Conclusion

Limited literature exists describing this pathological condition. Bipartite and tripartite navicular bones have been described but a DSB in greater than three portions appears uncommon, particularly when found in association with tripartite and bipartite navicular bones of the same horse. The pathogenesis of this abnormality is unknown; however various theories have been postulated. One theory is that medial and lateral sulci may pressure an intact DSB to divide it into pieces.

A CASE STUDY OF MYCOPLASMOSIS IN GOATS

C&T NO. 5407

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This case study involves a large commercial dairy milking 300 does that bought another 27 new milking does from another herd. It was 3 to 4 weeks later that the problems started. Fifty-eight kids died or were destroyed in the next 2 months due to arthritis in multiple joints (polyarthritis). There was also a sudden increase in the number of mastitis cases (from 9 cases per 6 months to 29 per 6 months). The mastitis was characterised by agalactia (complete loss of milk) on one side, often with severe illness in the doe.[1]

At the next kidding season the polyarthritis problem with the kids re-occurred with 80 of 200 kids being infected. This was despite this commercial herd having excellent kid housing, good quality feed and dipping every kid's naval cords with 3.5% inorganic iodine. All the kids were fed their dam's colostrum then bulk raw goat's milk. The death rate in the kids by this time had reached 52.7%.

At this time, veterinarians were finally called in from the University of California after the owner had tried unsuccessfully to cure the kids, treating the affected kids with a range of antibiotics i.e. penicillin, tylosin and oxytetracyclines. The veterinarians examined all 200 kids and found that 80 of them had multiple swollen joints, severe lameness, weight loss, poor coats and scours (diarrhoea). Only a few had a fever but half had increased lung sounds on expiration and elevated respiratory rates.

There were 26 kids that were so severely affected that they could no longer stand and these were destroyed and then subjected to necropsy examination. The most striking common finding from these necropsies was a fibro-purulent poly-arthritis (pus with fibrous stands in multiple joints). Various types of lung damage were also a common finding. *Mycoplasma mycoides*, subspecies ***mycoides*** was isolated from both the affected joints and also the bulk milk tank samples.

The next step was to take sterile milk samples from the entire milking herd of over 400 does and try to culture mycoplasma organisms from their milk to identify the carriers. This was done monthly for 6 months. This established that some of the does were only intermittent carriers and many showed no outward signs of any infection. However, when a heat wave stuck, 40 of these carrier does died of peracute mastitis. The added stress of the hot conditions tipped them over the edge and they died suddenly.

The University veterinarians instituted a supervised control program which had these components:

- Culling all kids with swollen joints
- Feeding only treated colostrum and pasteurised milk or milk replacer to kids
- Culturing the milk form each does for mycoplasma and segregating the herd into infected and clean milking herds
- Milking infected does last
- Washing all udders in udder wash and drying with disposable paper towels
- Dipping all teats in an iodine based teat dip after each milking
- Good hygiene in the milking area
- Culturing sterile samples of each new doe's colostrum for mycoplasmas and these does were hand milk separately until the results were known
- Infected milkers would be culled when economically possible

Within 2 weeks of the introduction of this control programme, all new cases of polyarthritis ceased and no more were seen that year. However, in the next year not all kids were placed on the control programme and 20 of these kids were subsequently culled.

The most disturbing feature of this case study was that when the herd of origin of the 27 introduced does that started the outbreak was investigated, it was found that the herd owners already knew they had a mycoplasmosis problem and were taking sterile samples of their own milking does and testing them for mycoplasma. These herd owners were irresponsibly selling their positive/carrier milking does as milkers and not for meat.

This case study stresses the need for goat producers to thoroughly investigate the health status of the herds of all animals they want to purchase and introduce. Is the Australian system of National Vendor Declarations (NVD) or Goat Health Statements enough protection for your goat herd's health to prevent a similar situation? The answer is no. Every goat herd needs a Biosecurity plan and a quarantine policy for new introductions developed with the help of their veterinarian. Veterinarians who develop these plans need to know the goat

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A healthy dairy goat.

herd's management well and about common goat diseases e.g. that mycoplasmas can be cultured from the ears of goats and can be spread by ear mites moving from goat to goat.[2]

The NVD system was developed by the Meat & Livestock Authority but is aimed at meat traceability and food safety e.g. freedom from agricultural and veterinary chemical residues (http://www.mla.com.au/Meat-safety-and-traceability/Livestock-Production-Assurance/Vendor-declarations).

The National Goat Health Statement is attached to the NVD or waybill but only asks about the herd status in regard to CAE, Johne's disease, footrot and lice. Many other important diseases such as mycoplasmosis and anthelmintic resistant gastro-intestinal worms, are not covered. Also there are no clear-cut rules in the National Goat Health Statement – see http://www.farmbiosecurity.com.au/wp-content/uploads/2014/06/National-Goat-Health-Statement.pdf. Different goat keepers could be comfortable with different levels of risk, preparing to accept a higher level of risk in order to increase their numbers quickly or introduce better genetics. In the latest issue of the 'Australian Goat World' there was an advertisement for a dairy goat herd that was certified by blood test as being free of Caprine Arthritis Encephalitis (CAE), Johne's disease and mycoplasmosis. Let's hope this is the start of a trend.

This case study also supports the Australian Goat Industry Council's National Kid Rearing Plan and associated checklist in their approach of never feeding bulk goats' milk to kids.[3] The feeding of pooled goats' milk is the main method of spread for CAE and mycoplasmosis and is also important for Johne's disease.

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INVITED COMMENT COURTESY OF:

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Jennie pictured here with her pet goat, 'Norman'

Sadly enough, this case study presented by Dr Baxendell is essentially identical to a scenario at a Queensland goat dairy that I was consulted on a few years ago. Like the case study, this dairy was in expansion mode and purchased several nannies from another breeder that came with a *National Goat Health Statement*. The owner believed that this declaration was sufficient to ensure that the purchased animals were healthy.

Regrettably, this was not the case and the dairy lost most of the goat kids the first year of the mycoplasma outbreak and the milking nannies were reduced to less than half the original population due to culling of carrier and clinically infected animals over the next two years. It is an all too common story.

So how did this happen? As pointed out in the case study, the goats were sold with the disease whether it is unwitting or malicious. ***Caveat emptor*** applies now as it did in Roman times…let the buyer beware. While the *National Goat Health Statement* is very important for controlling diseases like Johne's disease, CAE and virulent foot rot, it is not a substitute for a veterinary health examination. Animals purchased at dispersion sales or as replacement animals are commonly implicated in disease outbreaks. And it is not just mycoplasma that can be 'bought' into your herd. Commonly introduced pathogens/diseases include: *Salmonella spp., Streptococcus agalactiae, Staphylococcus aureus*, Bovine Viral Diarrhoea Virus (BVDV), vibriosis, and trichomoniasis, anaplasmosis, caseous lymphadenitis and virulent foot rot. Unfortunately, it is uncommon for a livestock producer to seek veterinary advice regarding purchases. We are often made aware of the 'new arrivals' when the disease outbreak is at peak when we are called out to put out the 'fire'. The excuses producers use for poor biosecurity and quarantine are many, varied and often money is an under riding issue.

How do we prevent it from happening again? Open up a dialog regarding biosecurity and quarantine well before the need arises. Encourage your clients to develop a biosecurity plan and a quarantine policy for all new introductions with your assistance. The plans need to be simple, realistic and tie in with herd management practices and levels of risk the producer is willing to take. A resource, often overlooked by farmers, producers and veterinarians, is the Local Land Services (LLS) District Veterinarian (formerly RLPB and LHPA). LLS District Veterinarians are a valuable resource for locally relevant, up-to-date, independent advice and information on biosecurity and animal health issues to land holder and veterinarians (http://www.lls.nsw.gov.au/livestock/livestock-advice). I often tell my clients when they think I am being a nag about biosecurity and quarantine that they can pay me now or pay me later.

KILLING SERRATED TUSSOCK WITH GRANULAR FLUPROPANATE HERBICIDE

C&T NO. 5408

Peter Davies

Tally Gang Station
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My name is Peter Davies and I own a property called 'Tallygang Station', which is around 200 hectares in size, located about 4 km from Wombeyan Caves in the southern highlands New South Wales. This mountain property is at an average altitude of 840m, and consists of hilly, rocky and, in some areas, very steep granite country which is approximately 70% cleared with around 700mm average annual rainfall.

In the past, the property ran sheep and was well known in the area for producing high quality fine wool. These days, mostly due to wild dog problems, I run a small Hereford breeding herd in combination with one of my friends, a neighbour with a similar land holding.

In this area we are surrounded by National Park and smaller semi-bush blocks. All of this land, including mine, has medium to heavy infestations of Serrated Tussock – to name but one of my weed problems! In some cases there are nearby unmanaged properties that are now virtually 100% Serrated Tussock.

Over the past 14 years of owning my property I have continually conducted spot spraying of Serrated Tussock. I have invested heavily in spot spray equipment over the years, including a 100 litre tractor mounted hose spray unit, a 70 litre Quad bike hose spray unit, several backpacks and, at times, contractors using 4x4 mounted hose spray units. By my calculations, over the years I have applied over 30,000 litres (mixed) liquid Flupropanate on the more accessible parts of my land. Although these applications have been relatively effective in slowing the spread of the Tussock on my property I feel that there are still several challenging problems with this type of herbicide application.

1. Seed still blows in from neighbouring properties, especially where no regular weed controls are in place.
2. Serrated Tussock seed remains viable in the ground for a number of years.
3. Once a Tussock plants dies, you need to replace the bare ground that this creates in order to ensure a preferred pasture species takes its place – seeding can be expensive and logistically difficult on my property.
4. No matter how careful I am with spot spraying, there still seems to be significant over-spray issues – therefore killing both the Serrated Tussock and the other grass species that I wish to promote.
5. The very nature of this beautiful mountain country means that large infested parts of the land are inaccessible to larger more efficient mechanical spot spray methods, and even spot spraying with a backpack is not practical or safe.

I have calculated that these spot spraying applications cost around $50 per hectare when I use a contractor on my property. This cost only applies to the spraying of the cleared paddocks. Such an approach does not treat lightly wooded sections of the paddocks, nor steep hills and gullies. This means that I have large sections of land that have never been treated, mostly due to accessibility issues. So not only do I have seed spreading from neighbouring properties, but also from those less accessible areas of my own land!

One day, in a casual conversation in the local pub with the owner of the rural supplies business in town, he mentioned his trials of a new **granular flupropanate** that he was applying to properties in the area by helicopter. As I recall, he said that this was a quick and efficient way to apply flupropanate, that his test applications showed highly selective weed kill, and that the granules would remain viable in the ground for up to 2 years.

I could see that if this system was effective, it could resolve a number of the challenges that I was experiencing with the existing spot spraying method I had been using on my property. In particular, I could have the granules applied to areas on my farm that have NEVER been treated, especially as some of these areas are geographically situated on the property so that the prevailing winds take the tussock seed over the rest of my farm as well as my neighbours'. In addition, if he was right about the selective nature of the granules, I could avoid the on-going problem of 'over-spray' i.e. killing the pasture immediately around the tussock that I was targeting. The bonus, and ultimate selling point, for me was that he told me that the granules would remain viable for around 2 years, therefore not only eradicating the plants I could see on the ground now, but also the next generation of seedlings that I have noticed would ultimately come through shortly after typical spot spray applications.

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Figure 2. View from the chopper.

Although this type of application was expensive I decided to go ahead and have one small test paddock treaded with the granules. This covered an area of 27 hectares which included the paddock with the old shearing shed, and sheep yards – so heavily grazed in the past. It is also a section of my property that had some very steep, heavily serrated, tussock-infested areas on it that had never been treated.

So in October 2013 the helicopter came (Figure 1) and, at a cost of around $133 per hectare, I had the 27 hectare area I selected treated with granules in less than an hour (Figure 2). Spot spraying the same area would have taken about 4 days, and still I would not have been able to treat the steeper hills and gullies in this paddock. Two of my neighbours on adjacent properties took the opportunity of also treating their serrated tussock infestations, which reduced the cost of bringing the helicopter and granules to our district from Taralga.

After heavy rain in November 2013 (which is what is needed to enable the poison to be taken up by the plant) and not seeing any dead Tussock on the ground, I started to get a little agitated, and wondered if this high cost investment was a wise one! I spoke with the team at the local rural supplies store who arranged the application, and they assured me that the plants would die, but it takes a little longer than normal spot spraying time frames.

Sure enough, come January 2014, whilst chasing some cattle I happened to walk the treated paddock including the steep hills which had never previously been sprayed. To my relief I saw dead tussock everywhere I looked. Even better, I could see no evidence of any other plant species having been killed. On the down side, I still had a strip of live serrated tussock plants through this paddock. This is due to the fact that I have power lines running over this paddock, and the helicopter was not able to apply granules over this area – something that was not pointed out when I first gave the go ahead for the treatment of this paddock. This untreated strip will need to be followed up with spot spraying at some point.

Figure 1. The helicopter rig used to distribute the granular flupropanate.

Recently I had the local Agronomist Dale Chalker look over the treated area, and he confirmed that there appeared to be no or very little damage to more desirable grass (including native species) and legume species and most or all of the tussock dead (Figure 3). I now await to see if any new Serrated Tussock seedlings come through to test the longer term viability of the granules.

In summary, I feel that if I, my neighbours and National Parks invest in this granular application strategy, we have the best chance ever to get on top of this particular weed problem, and then start to divert our spot spaying time and energy into creating better, more dense ground cover, either in the form of trees or pasture. For me, this is the first time in the 14 years of me owning this property that I have seen an entire paddock – including, gullies, steep hill sides, open country and under trees – where a flupropanate treatment has been effective on 99% of the areas where herbicide has been applied and not also killed a significant number of other species.

Next steps – watch for seedlings in the treaded paddock, and treat the rest of the property!

Editor's comment: *Serrated tussock is a very significant weed throughout much of NSW, and this new technology was developed in Australia. The apparent success of this method, if it holds its early promise, will greatly improve the productivity of much of NSW, with flow on effects to farmers and the local community.*

Granular Flupropanate herbicide is a product that has been proven to be effective in the control of Serrated Tussock. The active ingredient flupropanate is a residual herbicide that is taken up by the plant's roots.

When applied in liquid form by boom spray it can do damage to more than just the targeted plants, leaving bare patches and killing off otherwise beneficial species, but its granular form is proving an effective weapon against Serrated Tussock, even in large scale application.

Tablelands Agronomist Bryn Rees said liquid flupropanate application had an uneven kill rate across a wide area when applied by air. Granular flupropanate proved to be much more effective even in timbered areas, with little effect on native grasses or improved species like legumes, cocksfoot and phalaris.

'The granules fall through the canopy and land on the ground near the target.'

In trials, heavily Serrated Tussock infected paddocks on tablelands properties near Taralga, where the constant battle to keep the Serrated Tussock in check had had limited success, were treated with Granular Flupropanate. The Granular Flupropanate had wiped out nearly all the Serrated Tussock but left all the native pasture. Clover was thriving just 2 months after the treatment of the infested pasture.

For smaller areas of spot treatment of Serrated Tussock infestation, precision ground spreaders and hand spreaders have proven to be quite effective. Shaker packs are available for use around individual plants.

Contact John Brotchie on 0488 480 702 to book an inspection and mapping of your affected area in readiness for the coming spring aerial campaign.

Figure 3. Microlaena amongst dead serrated tussock

LAUNCH OF NEW TRANS-TASMAN CAT SURVEY

Do you have a good understanding of pet ownership habits and the lifestyle of cats in your area?

A major new survey has been launched this month by Boehringer Ingelheim to cat owners across Australia and New Zealand.

Supported by the Australasian Society of Feline Medicine & the Centre for Veterinary Education, the survey aims to help Veterinary professionals better understand and track the priorities and practices of feline care across Australia and New Zealand.

Dr Richard Gowan, Chairman of the Australasian Society of Feline Medicine (ASFM) said 'We know from smaller surveys that data collected directly from cat owners can deliver an insight into healthcare awareness and practices of cat owners'. The survey results may help guide future initiatives for the ASFM, helping improve the health and welfare of cats in Australia & New Zealand.

In addition to the lifestyle questions around habits of cats, the survey will also ask owners about why they visit their vet as well as their knowledge and awareness of preventable cat diseases.

Mr John Heath, Head of Boehringer Ingelheim Animal Health Australia and New Zealand said, 'With a range of leading vaccines and pharmaceuticals targeted at feline health, Boehringer Ingelheim is proud to conduct this survey. We are excited about what insights the survey may discover and we look forward to sharing these with veterinary professionals across Australia and New Zealand.'

Results from the 'Insights into Feline Health and Lifestyle 2014' survey are expected to be published early in 2015.

HOW TO PARTICIPATE:

Encourage your clients to take the short 5 minute survey by visiting www.catsurvey.com.au.

Boehringer Ingelheim Territory Sales Managers will be also delivering materials for you to share with your clients. If you would like to know more or request materials, contact Laura Johnston on laura.johnston@boehringer-ingelheim.com.

Authors' views are not necessarily those of the CVE

CVE *Control & Therapy Series* – **Issue 276** September 2014

BEST 'VET PET'

The word 'Pet', apart from its obvious meaning as a domestic or tamed animal kept for companionship or pleasure, has come to be associated with strong and positive human interactions. We use the term to indicate when people are pampered and treated with special favour or affection such as 'she's the class pet', or to indicate strong feelings and emotion as in 'that is his pet love/project'.

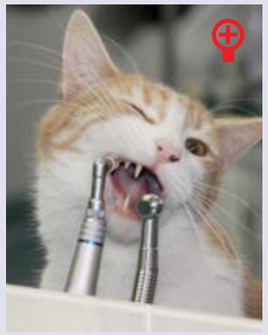
IS YOUR PET THE NEXT C&T COVER BOY/GIRL? FIND OUT AND WIN CVE$500!

Contributing vets enjoy seeing their pets in an issue of the C&T Series, so we thought we'd run a fun competition and encourage our Members/Readers to send in their favourite pet pics. Post your pics onto the CVE's Facebook page (www.facebook.com/cvesydney) and the owner of the pet with the most 'Likes' will win CVE$500 towards a CVE course/product of their choice (see www.cve.edu.au) AND their pet will feature on our December 2014 Issue 277 cover. The 2 runners up will feature on the December eBook cover.

Think of this as '*Australia's Next Top Pet'* competition. If you'd love to see your favourite pet on the next C&T cover AND win CVE$500 towards a CPD course/ product of your choice, go to our Facebook page and upload your pic.

Entries (clockwise from top) from C&T Series supporters and contributors, Doctors:-

- Gary Norsworthy (Alamo Feline Health Center, USA),
- Anne Fawcett (Sydney Animal Hospitals Inner West, Stanmore)
- Kerrie Rodgers (Patterson River Vet Centre)



PRIZES generously supplied by Hill's:-
WINNER - Hill's™ Science Diet™ or Hill's Prescription Diet™Dog or cat food to the value of $200
RUNNER UP - Hill's™ Science Diet™ or Hill's Prescription Diet™Dog or cat food to the value of $150

FROM THE DE FILES

Winner of Best Film Clips FROM THE DE FILES

ANISOCORIA & NYSTAGMUS IN A CAT

C&T NO. 5409

Moira van Dorsselaer
2014 CVE/ISFM Feline Medicine DE participant

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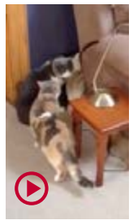
Moira shared this case with her tutors and fellow DE participants seeking advice, and wishes to state that a definitive diagnosis was not obtained. Therefore, we seek feedback and comment from you, our Members/Readers.
Please email to: Elisabeth.churchward@sydney.edu.au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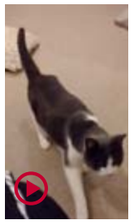
Video 2 21/7/14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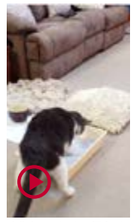
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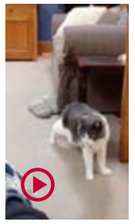
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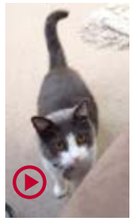
Video 6 25/7/14


Video 7 28/7/14.

'Andy' is a 3-year-old Domestic Short Hair. BCS: 4/9 4.98 kg. He is mostly an inside cat but does go outside occasionally. His owner brought him in to the clinic because she saw Andy falling over intermittently. She described it 'As if he is drunk & falling over'.

Andy was a very nervous cat so he wouldn't move out of a crouch position in the consultation, so I didn't get to see him falling over. I asked his owner if she would take some movies for me on her smart phone and email them to me so I could see what he was doing at home. His left pupil was more dilated than the right; in the photo it doesn't look as obvious as it was. He also had a slow, subtle horizontal nystagmus to the left. Pupillary light reflex was normal and consensual and menace was also normal.

There were no sign of uveitis or corneal damage and the eye otherwise appeared totally normal. I do not have a Tonopen so I couldn't measure the pressure in his eyes. Ear examination was completely normal and tympanic membrane normal and non-inflamed.

Andy had lost 2kg in 3 months as I had advised the owner at his previous visit that he was overweight so she had been working on it. I observed that 2kg was quite a large weight loss though, for Andy's frame. Abdominal palpation was unremarkable. Heart rate and rest rate: Purring heavily > unable to stop. Mucous membranes were pink and moist and CRT 1 second. Andy did not appear to be in any pain at the initial consultation.

USG: 1.040

Sediment & Dipstick: unremarkable.

PROBLEM LIST

- Nystagmus
- Weight loss
- Anisocoria

Nystagmus Diagnosis

- Inflammatory: Vestibular (Although no head tilt), brain
- Infectious: Ear
- Neoplastic: Brain tumour (unlikely in cat this age)
- Toxic: No exposure according to Owner

Anisocoria Diagnosis

- Trauma: Corneal injury (not obvious), Head trauma (possible, no history)
- Neoplastic: Possible (? age of cat)
- Inflammatory: Uveitis, Retinal disease (I did not look at retina at initial consultation : Later date was totally normal)
- Brain or Nerve lesion
- Glaucoma:

I took bloods for general profile but unfortunately did not do

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FIV/FeLV which I probably should have. I was waiting to see what they revealed before doing anything else, if in fact I did do anything else.

Andy's blood results came back completely normal. I arranged to do a neurological examination and examine his retina's. We took a conscious lateral thoracic/abdominal radiograph to rule out any cavity effusions. Blood was taken for Cryptococcosis and Toxoplasmosis serology. At this consultation Andy had lost further weight and reduced to 4.87 kg. He was eating very little for his owner.

I started Andy on Clindamycin 25 mg BID and Thiamine 100mg SID on advice from the Tutors of the DE course.

The next step was to anaesthetise Andy and take radiographs of his tympanic bulla and check his larynx/pharynx for polyps. The final diagnostic plan was to either perform a CSF tap or a CT scan depending on clinical picture and owner finances.

HISTORY

CHECK UP

25/7/2014 Vet: MV BCS: 4 Wt.: 5.17 Temp:

On checkup, Andy still had diarrhoea most likely related to the antibiotics. Andy has improved significantly and his eyes no longer had anisocoria, with the pupils now the same size. Andy still would not walk around the consult room but his owner showed me a new movie of Andy walking almost normally. As he was only eating canned tuna (for human consumption), I gave the owner a can of 'Dine Desire Virgin Tuna' and encouraged her to try Andy on this instead, as it is more balanced for cats. Andy was to continue on all current medication for the time being.

HR: Regular & normal amplitude: 160

Pink/moist mm CRT 1 sec

Eyes: NAD

Ears: NAD > no discharge/inflammation

We arranged a visit the following week to monitor Andy's weight. The owner emailed later to say he was happily eating the sample Dine and had jumped out of the carrier without falling over – a huge improvement.

Question to DE listserve (after the owner sent through the video of Andy at the Litter Tray): Anything else I should have done or planned to do?

QUICK UPDATE

Amazingly, Andy appears to have made a full recovery, which I will admit totally surprised me. I was expecting the worst. I continued him on his current medication, Clindamycin 25mg BID + Thiamine 100mg SID, I was reluctant to change anything at that point. I am awaiting the Toxicology results which I suspect will be negative, especially now that he has improved.

My thoughts are that he either:-

1. Had a Middle/Inner ear infection that responded to Clindamycin
2. Was in fact Thiamine deficient because he was not eating due to his owner taking away his food he liked and he went on a bit of a 'hunger strike'. Is that plausible?
3. Had vestibular disease and just needed time and none of my treatment made any difference. Andy's age doesn't really fit with this though.

I was pleased either way as I thought we were going to lose him. I will continue to weigh him weekly and gradually wean him off his medication.

CLOPIDOGREL (PLAVIX®)

C&T NO. 5410
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I have been using clopidogrel (Plavix®) for a number of cardiac disorders of cats. Clopidogrel is an oral thienopyridine-class antiplatelet agent that has been used in cats for a number of years. I usually reserve it to cases where cats have recovered from aortic thromboembolisms and it is used as a preventative, particularly at-risk patients with Grade III/VI or greater murmurs.

When given to cats at the recommended dose of 10 - 20 mg/kg1, the drug is well tolerated. I have been using this drug for 3 years now, on over 20 cats, with no side-effects. A number of geriatric patients with cardiomyopathy have concurrent hyperthyroidism and/or chronic renal disease, and there doesn't appear to be an inter-reaction between this drug and carbimazole, benazepril or amlodipine.

I have also found this drug beneficial in younger cats with stable cardiomyopathy as a preventative against thromboembolism, although this is 'anecdotal' due to insufficient numbers of patients or long-enough scale of usage, or the presence of a control cohort of cats.

Although clopidogrel has anti-platelet effects, it does not appear to be beneficial for treatment of acute thromboembolic cases2, in my experience.

In humans, side-effects are common when clopidogrel is used in conjunction with NSAIDs. I have not tried the concurrent use of aspirin with cats that are on this drug, although low-doses, as recommended by several prominent veterinary cardiologists and in cats that require long-term management with anti-thrombotic agents, clopidogrel may well be a more superior product.

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FROM THE DE FILES

DERMATOLOGY
(A DE ASSIGNMENT CASE REPORT)

C&T NO. 5411

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DE Feline Medicine participant 2013

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Signalment and History

'Truffle' was a male/neutered 18-month-old Domestic Short Hair cat. He was originally adopted from Nundah Village Veterinary Clinic as a stray kitten. He was up to date with vaccinations (F3), and had no ongoing health issues as reported by his owner. He received regular monthly flea treatments. His only prior notable physical problem involved malocclusion of his canine teeth due to a previously healed mandibular fracture as a kitten.

Truffle first presented at the after-hours Emergency Centre for unwillingness to walk upstairs and painful hindquarters. No history of trauma had been observed by his owner, but it was suspected that Truffle had been hit by a car. Although painful on palpation, he did not exhibit neurological abnormalities. He appeared to have no external injuries, but radiographs revealed several nondisplaced pubic fractures and a fractured right ischiatic tuberosity. The radiographs were examined by a surgical specialist, who recommended 4 weeks of cage confinement rather than surgical intervention. Pain relief involved 0.05mg of Buprenorphine subcutaneously, and placement of a 25mcg transdermal Fentanyl patch. Truffle was confined to a large crate, and his owner later reported he seemed comfortable. He was bright and affectionate, fairly mobile within his cage, and eating well.

Two weeks later he re-presented at the Emergency Centre after hours when his owner noticed hair had begun to fall out along his dorsal spine. She reported no relevant history to explain the hair loss. The area was clipped, and several palm-sized ulcerative wounds were discovered which extended down over his flanks. Purulent exudate was present at the wound edges. The edges of the wounds were not thickened. Truffle was pyrexic (temperature 39.2°C). He also vomited during the consultation and produced a tapeworm. His mucocutaneous junctions and nailbeds appeared normal. His lymph nodes were not palpably enlarged. There was no evidence of external parasites. No other abnormalities were found on physical examination.

Unfortunately due to cost constraints it was not possible to take a biopsy of the lesions for histopathology.

Problem List

- Non displaced fractures of the pubis and ischiatic tuberosity due to unseen trauma 2 weeks prior, possibly a car accident
- Ulcerative open wounds present over dorsal spine and flanks with purulent exudate
- Pyrexia
- Tapeworm

Differential Diagnoses

- Ischaemic Dermatopathy secondary to trauma
- Thermal or chemical burns
- Vasculitis secondary to a drug reaction
- Feline Idiopathic Ulcerative Dermatosis
- Autoimmune disease e.g. Epidermolysis Bullosa

Treatment

Truffle began a course of Amoxycillin/Clavulanic Acid (75mg orally every 12 hours). He was also given an anthelmintic effective against tapeworms. Transdermal Tramadol 0.05mg was applied to his medial pinna every 12 hours for pain relief. Truffle was re-examined at the day clinic every 3 days, while antibiotics and pain relief were continued. His pyrexia resolved and his appetite and demeanour remained normal.

Hydrocolloid Duoderm™ dressings were applied to the wounds and held in place with soft bandages and tube netting. The dressings were replaced every three days. When a healthy layer of granulation tissue was observed and the wounds had begun to contract, Melolin™ dressings were used. Truffle continued to eat well and his temperature remained normal. He remained confined to his crate for the full four week period.

The aim of treatment was to provide pain relief and infection control while the wounds healed as much as possible before considering surgical closure if necessary. After 6 weeks, the wounds had formed a complete epithelial layer so at that stage we decided against going ahead with any surgical intervention.

After Truffle's recovery he was returned to the clinic for a vaccination 4 months later. His owner reported that he was 'back to normal', and no new lesions had been observed. The skin over his thoracolumbar spine appeared thin and glistening with areas of patchy alopecia. Truffles showed no discomfort on palpation of the area. He was otherwise physically normal on examination.

Discussion

There were several aspects to Truffle's history which influenced our choice to treat his injuries as a case of 'Traumatic Ischaemic Dermatopathy'. Truffle originally came to the clinic as a stray kitten and he had been examined several times since then to ensure his malocclused canine teeth were not impinging on his gingiva and causing pain. No dermatological abnormalities had been observed during these examinations. His owner reported no history of trauma after the initial incident 2 weeks previously, and at the time the lesions were first noticed, Truffle was safely confined to his crate.

On examination his mucocutaneous junctions and nailbeds appeared normal. His lymph nodes were not enlarged. His owner applied regular flea control and he showed no evidence of external parasites. There was hair loss but no hair breakage around the area, and excessive grooming or self-trauma had not been observed by his owner. We discussed the possibility of an autoimmune disease but in light of his recent history of trauma and the lack of other physical findings this seemed unlikely. Truffles developed no new lesions after treatment with antibiotics and bandaging was initiated.

Unfortunately we were unable to collect a biopsy sample for histopathology which would have helped enormously with arriving at a definitive diagnosis. Other cases of ischaemic dermatopathy secondary to traumatic pelvic fractures in cats have shown consistent histopathological findings ([1], [2]):

- Follicular and adnexal atrophy, follicular telogenization
- Mildly acanthotic epidermis
- The stratum corneum is focally lifted from the epidermis, or absent in areas
- Fibroplasia along the dermo-epidermal junction, with a mild perifollicular infiltrate
- A moderate to dense infiltrate of inflammatory cells in the subcutis
- Perivascular fibrosis

Gross findings are consistent with this case, including: a glistening appearance to the skin, lack of pain on palpation of the surrounding area, and epilation of hair before deeper ulcerated wounds become apparent ([1], [3]). In similar cases the timeframe between the initial trauma and the development of skin lesions has reportedly ranged from approximately 2 to 6 weeks ([1]).

The exact pathomechanism of traumatic ischaemic dermatopathy remains unknown. It has been postulated that shearing forces during the initial trauma cause damage to the epidermal blood supply resulting in ischaemia and resulting tissue necrosis([1]).

After the experience of managing Truffle, I am more aware of the possibility of post-traumatic skin necrosis which may become apparent several weeks after the initial incident. In future this should be discussed with owners after a cat's post-trauma recovery, and observed for when rechecking the patient after the initial recovery period.

Figure 1. Specialist surgeon recommended 4 weeks of strict confinement

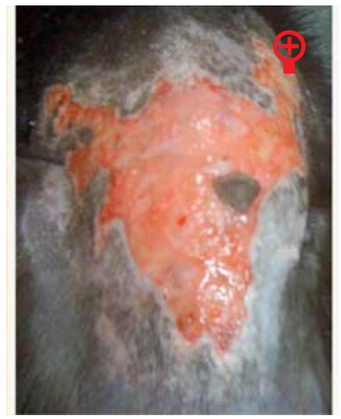

Figure 2. Easily epilated hair along spine progressing to a large ulcerated wound extending along flanks with pyrexia (39.2°C)

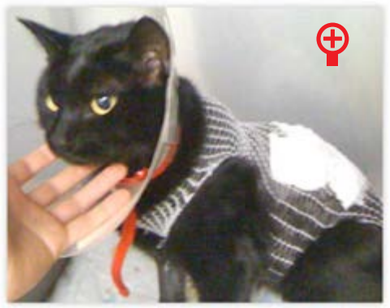

Figure 3. Duoderm Hydrocolloid dressings applied, held in place with soft bandages and tube netting to protect the area and prevent self-trauma. Melolin dressings were used once a healthy layer of granulation tissue was apparent and the wounds began to contract. 'Truffle' remained confined to his crate and no new lesions became apparent.

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FROM THE DE FILES

AN UNUSUAL CASE OF THROMBOEMBOLIC DISEASE IN AN 8-YEAR-OLD CAT

C&T NO. 5412
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'Otis', an 8-year-old MN DSH cat presented to us due to sudden onset right foreleg lameness. He had not been seen by a veterinarian for over 2 years and his last clinical examination was within normal limits. The owner reported Otis had been well in the preceding days and that the lameness had only occurred in the last few hours despite Otis being indoors and no known trauma having occurred.

On physical examination, Otis seemed agitated and distressed. He could ambulate but was dragging his right foreleg; it was noted that his shoulder extensors were functioning and the upper limb was moving forward. However, his distal limb (distal to elbow) was limp and flexed at the carpus, which was dragging along the floor.

Otis's mucous membrane colour was pink with a normal capillary refill, respiratory rate was 40bpm and there were brief moments of open mouth breathing. He was normothermic. The heart rate was 220bpm and there was an irregular rhythm and pulse deficits. A basic neurological exam was unremarkable.

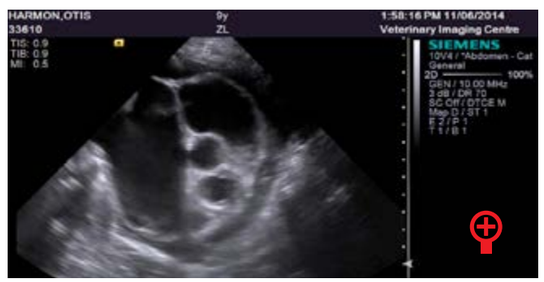
Closer examination of the affected limb revealed a moderate degree of pain and Otis became fractious with gentle manipulation of the leg. His paw was cold to touch and there was a mild cyanotic colour change of his pads and nail beds on the affected forelimb. With the accompanying cardiac abnormalities and these clinical findings there was concern that Otis had cardiac disease with a subsequent thromboembolism. Otis was given 1mg ACP and 0.06mg buprenorphine IM and referred for an immediate cardiac ultrasound.

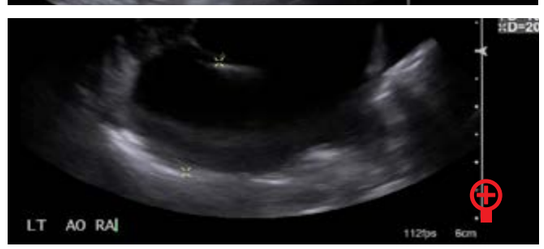
His cardiac ultrasound showed massively dilated left and right atria (24 and 25mm; normal <12mm) and the ultrasonographer also commented there were areas of ventricular myocardial infarction. There was significant 'smoke' (spontaneous echo contrast) in the left atrium and there were also moderate pleural and pericardial effusions present.

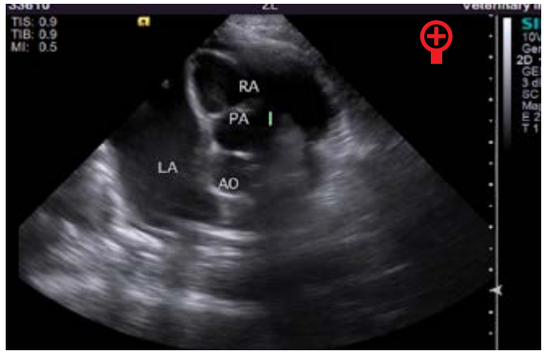
A diagnosis of end-stage congestive heart failure with a brachial thromboembolism was made.

I found this an interesting as my initial differentials for Otis were a traumatic injury or neurological disease (injury, fracture, brachial plexus avulsion.) This was the first case of a thromboembolism I had seen in practice that did not involve the typical location of the aortic bifurcation (i.e. a 'saddle thrombus'.) The first main branch artery to leave the ascending aorta is the right subclavian artery and it is hypothesised in this case that a thrombus from the left atrium entered this artery leading to its occlusion. Sadly, the prognosis for cats with the more common presentation of thromboembolic disease (FATE) is poor and given the severe cardiac changes Otis showed his prognosis was grave and he was euthanased.

Figure 1. A different cat who presented with similar symptoms as Otis: abnormal foreleg position.







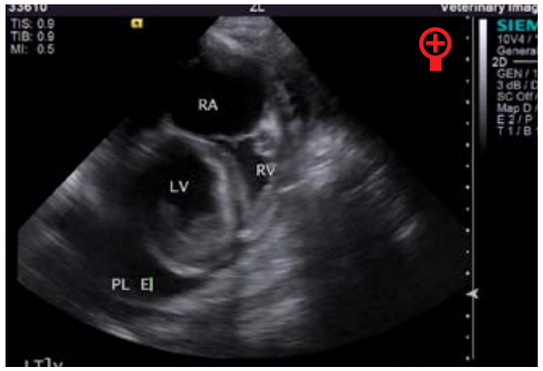


Figure 2-5. Ultrasound images of Otis's heart: significant findings include left and right atrial enlargement, increased La:Ao, pleural effusion and 'smoke' present in the left atrium.

VALE
ROSEMARY JANET BRYDEN
2 MAY 1941 – 30 JUNE 2014

LtoR: Breck Muir, Doug Bryden and Rosemary Bryden at CVE's office

On behalf of the veterinary profession, we offer our sincere condolences to Doug Bryden and family.

Read more…

CALL FOR CASES – WE NEED YOUR HELP!

FLEXOR TENDON CONTRACTURE

C&T NO. 5413
Leonie Thom

Wilston Vet
50 Newmarket Rd Windsor QLD 4030
T. (07) 3357 3882
E. info@wilstonvet.com.au www.wilstonvet.com.au
Winner 2010 Quest Business Achievers Awards

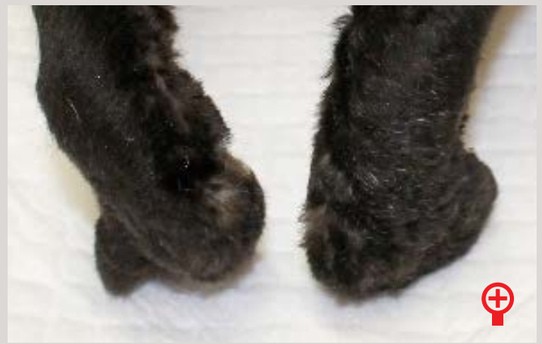
We are currently investigating a syndrome in cats which results in flexor tendon contracture of the forelimbs – see Figures 1 & 2 – and Rex cats may be over-represented.
Cats generally present with non-painful gait abnormalities associated with the gradual contracture of the phalanges, then the carpus. The condition may be unilateral or bilateral.
It is believed to affect only the forelimbs but cases involving the hindlimbs would also be of interest.

HAVE YOU SEEN ANY CASES LIKE THIS?

If so, we'd very much appreciate your help. Would you please contact me via e-mail: leonie_thom1@bigpond.com

Thank you!

Figure 1. Note the spectacular flexor contracture of the distal forelimbs (A). A close-up is provided in (B).



FROM THE DE FILES

HANDY HINTS FROM THE ISFM FORUM FELINE-MEDICINE@MAILTALK.AC.UK

THE CVE IS PARTNERED WITH THE INTERNATIONAL SOCIETY FOR FELINE MEDICINE (ISFM) FOR OUR FELINE MEDICINE DISTANCE EDUCATION PROGRAM

C&T NO. 5414
David Culley

HOW ON EARTH DO YOU GET RELUCTANT CATS TO TAKE TABLETS?

I am disappointed that some cats that I treat do not realise the benefits that I am bestowing on them by giving them medications.

I have taken a thorough history, performed a comprehensive diagnostic work up, arrived at a diagnosis, discussed the condition with the cat's patient owners, given very informative handouts – usually downloaded from the FAB site – and prescribed treatment. You would think, therefore, that the cat would take the medication without any fuss, but…!

Please can you let me know your hot tips for administering a tablet, e.g. benazepril, to a cat that has other ideas!

REPLIES

Reply No. 1

Helen Dennis
I have found Webbox pretty successful – the majority of cats love it and it is easy to mould around a tablet.

Reply No. 2

Samantha Taylor
E. taylorvet4@GOOGLEMAIL.COM

Is everyone aware of Summit Pharmaceuticals http://www.svprx.co.uk which reformulate a number of tablets into cat doses i.e. very small tablets: Metronidazole, amlodipine, gabapentin, tramadol, cisapride. Very helpful and not too expensive.

Reply No. 3

Sally Stockson
I have had an email from Summit Pharmaceuticals today launching methimazole transdermal gel… does anyone have experience of this?

Reply No. 4

Nicolette Joosting
Vancouver Feline Hospital
E. feline1@TELUS.NET

Yes, we use it a lot – for over 10 years now. We use a lot of transdermal meds now, and are looking for funding to get the efficacy studies done.

Methimazole studies have been done, it does work.

In terms of transdermals, most are compounded in PLO gel – we find this causes residue so owners need to be aware to clean the residue off the inside ear pinnae.

We are finding we are preferring lipoderm gel formulations – it is not temperature sensitive so it doesn't separate in cold weather (or if clients put it in the fridge!); there is less residue on the skin; it is considered a more stable base; there are several forms available – plain (buprenorphine); high molecular weight (for fentanyl, insulin, antibiotics, gabapentin); and anhydrous (for non-hydrous drugs like SAMe, ASA); and there is lipoderm max for transporting several molecules together.

Transdermals are in heavy use for human patients here, and we are lucky to have several local compounding pharmacies properly trained with proper quality control to script out to. I am always hesitant to re-sell a compounded drug (sorry Summit, but it gives me the creeps). Developing a relationship with the local human hospital or hospice care's compounding pharmacist is well worth it!

Tips for young veterinarians...

Cartoon courtesy of long-standing CVE supporter Frank Gaschk – veterinarian, cartoonist and animator.

TRANSDERMAL DRUG THERAPY

C&T NO. 5415

Nicolette (Nicky) Joosting

Vancouver Feline Hospital
Vancouver, BC, Canada
E. feline1@telus.net

Transdermal versus Topical

- TD – therapeutic drug concentration in systemic circulation
- Topical – local therapeutic drug concentration in surface organs (skin, eye, ear canal)

Benefits of Transdermal

- Better acceptance – easier to do – better compliance
- Potentially less GI irritation and side effects
- Avoidance of first pass intestinal and hepatic metabolism – e.g. isosorbide dinitrate is better TD than orally [4(6)]
- Possible longer duration of action without peak side effects – 'drug depot in skin'[4]
- Ability to custom formulate the drug concentration to patient's size

Limitations of TD

- Inappropriate for drugs that act locally on GI
- Some drugs are poorly absorbed transdermally and never reach therapeutic drug concentrations
- NOT appropriate for antibiotics/antimicrobials because of risk of poor absorption, sub-therapeutic plasma concentrations and potential selection for resistant bacterial strains
- May be ineffective for pro-drugs dependent on hepatic biotransformation for efficacy
- No immediate effect in emergency setting, except nitroglycerin
- Constrained by the physical limitations of permeation enhancers and practical limitations in how much skin coverage patient will accept
  - Need to apply to areas with relatively thin skin – pinnae, axilla, inguinal
  - Some cats may resent the administration
  - Increases prescription cost
  - Requires compounding – stability data often unavailable, need knowledgeable pharmacist

Properties of good TD drugs

- Relatively high lipid solubility – need to transverse waxy stratum corneum [2,4(8,9)]
- Low melting point – readily converted from a solid to a liquid at body temperatures [2,4(8,9)]
- Less polar compounds [2,4(8,9)] – very polar compounds (e.g. aminoglycosides, peptides) are poorly absorbed without additional interventions (e.g. electrical field, micronicelles, ultrasonic disruption of the stratum corneum)
- Tend to be effective at very low dosages (e.g. fentanyl, lidocaine, nicotine, nitroglycerine, scopolamine, oxybutinin, contraceptive hormones – doses range from 0.1-32mg/day in humans). Drugs that require higher dosages e.g. >50mg /day, are unlikely to be adequately absorbed, especially through the relatively small surface area of a cat's pinnae.
- Small compounds [2,4(7,8)] – molecular weights <500g/mol (500 daltons)
- Achieve high local drug concentrations [4]

Permeation enhancers

MOAs of permeation enhancers [4(16,18)]

- Increase fluidity of stratum corneum
- Solubilization of lipids between corneocytes
- Generation of pores on surface of corneocytes
- Exfoliation of stratum corneum

1. PLO

PLO= pluronic F127 lecithin organogel

- Pluronic = isopropyl palmitate and a poloxamer
  - surfactant that forms drug micelles [4(10)]
- Lecithin – from eggs or soybeans
  - increase the fluidity of the stratum corneum [4 (11,12)]
  - leads to exfoliation of the stratum corneum and low grade inflammation with chronic use which enhances drug penetration [4(11,12)]
- separates at cold temperatures (discard when this happens)
  - do not refrigerate
  - do not ship in cold temperatures
- greasy feel and residue build-up

2. Lipoderm

- Commercial product with proprietary ingredients
- Contains lecithin
- Not temperature sensitive – can go in fridge
- Less residue on skin
- Little more expensive 10%
- Stable base in several formulations
  - Plain
  - High molecular weight – insulin, antibiotics, gabapentin
  - Anhydrous – for non-hydrous drugs – SAMe, ASA
  - Lipoderm max – for transporting several molecules together

3. VanPen

- Commercial product with proprietary ingredients
- Used for more lipophilic drugs

4. DMSO

- Dimethyl sulfoxide
- Excellent permeation enhancer
- But very irritating with strong odor and thus not recommended for use in cats

5. Other vehicles used

- Oleic acid
- Propylene glycol
- Ethanol
- Glycol ethers

Many are irritating so combinations of enhancers at lower doses have been used to decrease irritation that occurs with higher doses [4(19)]

OTHER WAYS TO DELIVER TRANSDERMAL MEDICATIONS:

Formulation of polar drugs into 'duplex pro-drugs'

- 2 molecules of the same drug linked by an ester bond
- More lipophilic
- Better absorbed across stratum corneum
- Then cleaved locally by esterases in the skin
- E.g. naltrexone (used to treat alcohol and opiate dependency in humans) [4(20)]

Transdermal Patches

'Matrix' type

- High concentration of drug in a matrix or solvent with one or more permeation enhancers
- Rely on skin permeability to regulate drug delivery

'Reservoir' type

- As for matrix but with additional semi-permeable membrane to control rate for drug delivery

Physical disruption of stratum corneum – getting bigger molecules and more polar molecules across

1. **Sonophoresis -** Low frequency ultrasound [4(21,22)]
   - EMLA cream – 10s pretreatment with LFUS speeds onset of action in humans from 60 mins to 5 mins [4(23)]
   - EMLA cream is safe in cats [4(24)] but requires 1hr delay after application [4(25)] – need a study on sonophoresis for this!
2. **Microneedles** [4(26,27,28)] – humans report this as painless, studied in context of TD delivery of insulin, desmopressin
   - 'Poke with patch' – tiny arrays of microneedles in drug-impregnated patch
   - 'Coat and poke' - needles surface coated with drug
3. **Iontophoresis, electroporation**
   - use electric field to enhance skin penetration [4(1,29)]
   - insulin,calcitonin,vasopressin, octreotide, opioids, NSAIDs, antiemetics
   - commercially available iontophoric devices (e.g. Iontopatch 80) well tolerated by human patients
   - not evaluated for veterinary patients

TRANSDERMALS IN CATS

Data to compare the efficacy of PLO, Lipoderm, VanPen in the delivery of veterinary drugs

None [2]

Successful commercial formulations that have combined PLO/lipoderm/VanPen with systemic drugs

None [3]

Drugs suited to transdermal, commercially available or currently in empirical use:

In humans

Fentanyl, buprenorphine, scopolamine, nitroglycerin, contraceptives, HRT, nicotine, oxybutynin, clonidine, testosterone, lidocaine, insulin, desmopressin, calcitonin, vasopressin, octreotide, opioids, NSAIDs, anti-emetics, PTH, ondansetron and probably others….

NSAIDs – penetrate 3-4mm at site of application, with additional delivery to skin, muscle and joint tissues through local blood supply [4(4)] – effective for local treatment of acute musculoskeletal injuries in most placebo-controlled studies in humans, with minimal side-effects [4 (25)]

In cats

Constraints in human TD therapy (we do not know what they are in cats)

- total daily dosage requirement <50mg/day
- therapeutic plasma concentration in ng/mL range
- absorption limited to 1mg/cm^2
- patch size limited to 50cm^2

Considerations for the use of TD drugs without absorption or efficacy data

- Select a drug with large margin of safety - No real way to extrapolate dosages when bioavailability or efficacy data is unknown – could be higher, same, lower
- Select only a drug with a quantitative therapeutic endpoint (e.g. serum T4 for methimazole,). In cases where only qualitative therapeutic endpoint exists (e.g. pain management) use documented data – signs of pain/pain scoring, appetite, BW, frequency of vomiting, repeated ultrasounds with applicable measurements and beware of placebo effect and all the other pitfalls….
- Adjust dosage to effect
- Empirical dosing of antimicrobials should be avoided – if titrating from a low dose to avoid toxicity, dose titration can lead to antimicrobial resistance (I advise avoid antimicrobials in TD formulation)
- Use if other proven routes of admin are not possible – the uninjectable unpillable cat, intestinal malabsorption, intestinal side effects, palliative care, older cats with cervical and TM joint stiffness, owner abilities, need for compliance
- Use only if an immediate clinical response is not needed (ie can wait ten minutes)
- Inform the client that the appropriate dosage is not established for this route
- Make sure the pharmacy can provide information on the constituents of the formulation – should a local or systemic hypersensitivity reaction develop
- Make sure the pharmacy can provide stability information or a shelf-life for the formulation, and that this is included on the label instructions.
- Ask the pharmacy to label each TD syringe with drug name and expiry date! Cats can be on multiple TD drugs and the syringes all look the same.
- Ask the pharmacy and always remind the owners to wear fingercots when applying TD drugs. Most important for methimazole, prednisolone and buprenorphine.
- Use the highest concentration to give the most practical volume – 0.05mLs is the usual preferred volume; in cats on multiple drugs, practical volumes per drug are 0.025 – 0.05mLs. A total volume of 0.3mLs is too much.

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Drug	Bioavailability /Efficacy studies	Molecular weight	Formulation	Notes
Nitroglycerin		227g/mol	ointment	Used TD for decades in veterinary patients to reduce preload in cardiac failure [4(31,32)] – causes local venodilation, increases local blood flow Used in humans to treat neuropathic pain after thoracotomy [4(33)]
Fluoxetine	<10% compared to oral when given as a single dose [2(18,25,57,102), 3(15) 4(50)]			But if gave 10x oral dose got plasma concentrations equivalent to oral [3(15)4(50)] Repeated topical admin caused dermatitis
Diltiazem	<10% compared to oral when given as a single dose [2(18,25,57,102)]			
Dexamethasone	<10% compared to oral when given as a single dose [2(18,25,57,102)] In PLO negligible absorption after single dose [3(18)4(52)]			
Buspirone	<10% compared to oral when given as a single dose [2(18,25,57,102)] In PLO –poor absorption – not reliable [3(19)]			
Amitryptiline	<10% compared to oral when given as a single dose [2(18,25,57,102)] In PLO – poor absorption after single dose – not reliable [3(19)4(53)]	227g/mol		
Methimazole	Multiple doses in PLO P-K study poor absorption [3(14,17) 4(48)] Clinical studies – effective in lowering serum T4 in hyperthyroid cats, with fewer GI side effects than oral, risk of idiosyncratic toxicity (facial pruritits, hepatotoxicicty, blood dyscrasias) appear to be same [2(80), 3(13), 4(49,47)]	114g/mol	TD cream	More recent studies have been published
Carbimazole	Similiar to methimazole [2(10)]			
Glipizide	~20% compared to oral when given as a single dose [2(5)] 4-30% [3(16)]			Despite low absorption, has been associated with delayed decrease in BG [2(6)]
Atenolol	Modest efficacy in propylene-glycol-glycerin-Tween at 6.25mg q24hrs [2(53)]			
Amlodipine	0.625mg q24hrs in lipoderm [2(42)]			
Gabapentin	No cat studies yet?			
Pregabalin				– cannot get pure compound – so pharmacist feels there is a lot of impurities if they use the product from the pharmaceutical supplier and that will adversely affect absorption
Fentanyl	[4(34-38)]	336g/mol	patch	Effective in post-operative pain management in dogs and cats [4(34-38)] Less sedation and hypothermia than injectable narcotics [4(35,36)] Analagesic plasma concentrations 3-5 days in cats [4(39,40)] Variable absorption among individuals [4 (39,42)] and local/body temps [4(43)] Time to effect 12-24hrs [4 (39,42)]
Fentanyl	In PLO – single dose – 'undetectable' serum conc [4(51)]		TD cream	In clinical practice, we see a definite effect; dose controllable and titrat-able as the TD form; clinical response usually within 10minutes
Buprenorphine	No cat studies yet		TD cream	In clinical practice, effective; TD dose controllable and titrate-able to patient needs; much better compliance than with TM dosing! Clinical response 5-20minutes.
Insecticides and Acaricides				
Fipronil			Spot-on	Distribute to hairs, stratum corneum, sebaceous glands but not to systemic circulation [4(43-45)]
Imidaclopramide			Spot-on	Distribute to hairs, stratum corneum, sebaceous glands but not to systemic circulation [4(43-45)]
Selarnectin	Higher bioavailibilty in cats (74%) than dogs (4%) – thinner skin, grooming ingestion [4(46)]		Spot-on, in dipropylene glycol ether & isopropanol	Systemic absorption
Ondanstron	Human studies, none in cats?		TD cream	In clinical practice effective, dose varies from 1-5mg/cat; used when no other option exists
Prednislone	Steroids are well absorbed TD, human studies, none in cats?		TD cream	Finding 5mgTD bio= 5mgPO in clinical cases, clinical response in IBD, pancreatitis, lymphoma, mast cell, palliative, chemo, dermatitis and asthma; owner-preferred!

References:
1. J.E. Maddison, *Special considerations in Feline Therapeutics, chapter 1* in *Feline Medicine & Therapeutics*, 3rd Edition; E.A. Chandler, C.J. Gaskell, R.M. Gaskell; (2004); Blackwell
2. Lauren A. Trepanier, *Guidelines and Precautions for Drug Therapy in Cats, chapter 4* in *The Cat: Clinical Medicine and Management*; Susan Little; (2012); Elsevier
3. Mark Papich, *Drug Therapy in Cats: Precautions and Guidelines*, *Chapter 31* in *Consultations in Feline Internal Medicine*, Vol 5; J.R August; (2006); Saunders-Elsevier
4. Lauren A.Trepanier, *Transdermal Therapeutics*, *Chapter 18* in *Consultations in Feline Internal Medicine*, Vol 5; J.R August; (2006); Saunders-Elsevier

HOW MUCH DO YOU KNOW ABOUT HUMAN TICK-RELATED DISEASES?

FOR EXAMPLE, HAVE YOU HEARD OF RED MEAT ALLERGY? IF NOT, THEN THIS INFORMATION WILL PROVE USEFUL FOR YOUR CLIENTS AND YOU

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C&T NO. 5416

Sheryl van Nunen[1] & Paul Canfield[2]

ON BEHALF OF TICK-INDUCED ALLERGIES RESEARCH AND AWARENESS (TIARA) – WWW.TIARA.ORG.AU

Introduction

Veterinarians are in an ideal position to practise 'one medicine' when an agent of disease can affect both animals and their owners. Tick-related disease is an exemplar of that situation, where veterinary practitioners, when treating an affected animal, are ideally placed to inform pet owners of potential human hazards and to act as a conduit to health professionals who can provide further advice and treatment. Consequently, we provide this information on human tick-related diseases to veterinary practitioners in the hope that they will assist us in better educating pet-owners about potential human risks related to tick bites.

Paralysis related to *Ixodes holocyclus* is rare in people

Veterinary practitioners would be aware that ***Ixodes holocyclus,*** commonly known as the Australian paralysis tick due to the effects of its holocyclotoxin in animals, is endemic 20-30km inland along virtually the entire eastern seaboard of Australia. However, many of you may not be aware that paralysis in humans due to this tick is usually limited to local effects of holocyclotoxin; although rarely, especially in children, the paralysis will be more severe. ***What is more important in humans is the concern over possible infectious disease transmission and increasingly prevalent local and systemic allergic reactions induced by tick bites.***

What are the tick related disease issues for humans?

Around the world, a variety of tick species are very well recognised causes of infectious disease transmission in both animals and humans. The commonest infectious disease they transmit to humans in Australia is tick typhus (associated with Spotted Fever group rickettsiae, including Queensland Tick Typhus [R. australis] and Flinders Island Spotted Fever [R. honei]). Lyme disease (Lyme borreliosis) is an infectious disease caused by at least three species of bacteria belonging to the genus Borrelia. Lyme disease occurs in the USA, the UK, Europe and Eurasia, but is not known to occur in Australia. However, symptoms not unlike Lyme disease occur in Australian patients and therefore the Department of Health has set up a Clinical Advisory Committee on Lyme Disease to examine these issues (http://www.health.gov.au/lyme-disease).

Whilst these infectious diseases are of concern, the most common serious medical complaints caused by ticks are allergic reactions. These reactions manifest as:

- large local reactions at the site of the tick bite (Figure 1)
- tick-induced anaphylaxis (an immediate IgE-mediated reaction to tick salivary proteins) and
- mammalian meat-induced anaphylaxis (an IgE-mediated reaction directed against alphagal, typically several hours after mammalian meat ingestion).

The last condition is not fully understood, but it is known that the bite of a tick in some people can lead to the production of an allergic class of antibody called IgE which binds to a carbohydrate present in muscle from non-primate mammals (e.g. sheep and cattle) and New World monkeys. This carbohydrate is called galactose-alpha-1,3-galactose and is abbreviated to alphagal. Humans, great apes and Old World monkeys do not have alphagal in their muscle and are, therefore, capable of producing anti-alphagal antibodies. Non-mammalian meat derived from birds, reptiles and fish does not contain alphagal and, therefore, is not a trigger for anaphylaxis. It is assumed that tick saliva contains a substance that is the same or similar to alpha-gal, which is injected into the person to induce the production of anti-alpha-gal IgE antibodies. Whilst that actual substance in tick saliva has yet to be identified and confirmed; it is known that some tick salivary proteins are associated with galactosylated moieties, and alphagal has been found in tick gut in ***Ixodes ricinus*** (Hamsten ***et al***, 2013). Sometimes a hive-like rash occurs in sensitized individuals, but in some people a dangerous generalised anaphylactic reaction occurs after ingesting mammalian meat.

Whilst tick-induced anaphylaxis and red meat allergies are primarily mediated through the release of histamine and other compounds, rarely do humans have underlying mastocytosis. When they do, they may suffer from both tick and mammalian meat anaphylaxis. A minority of

The knowledge gathered from known tick anaphylaxis patients has led to us developing some guidelines regarding adult tick removal in humans known to be allergic to ticks:

GUIDELINES

- DO NOT SCRATCH ANYTHING YOU CAN'T SEE
- DO NOT DISTURB A TICK
- KILL THE TICK IN SITU (preferably with an ether-containing spray)
- IF THE TICK DOES NOT FALL OFF AFTER DEATH, THEN ALLOW A HEALTH PROFESSIONAL TO REMOVE THE DEAD TICK

individuals (2%), who do not have underlying mastocytosis will have both tick and mammalian meat anaphylaxis. Children, in particular, and some adults, have only gut symptoms or delayed angioedema or both after mammalian meat ingestion.

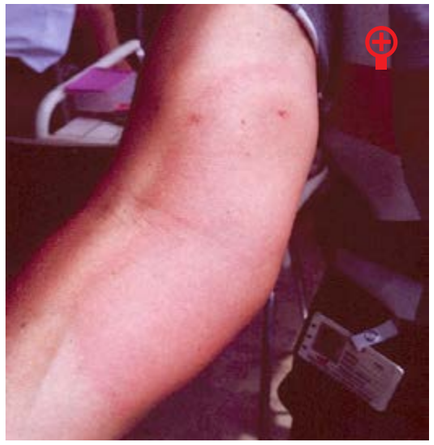

Figure 1. A very large local reaction to a tick bite on the inner aspect of the upper arm.

Recommendations for tick removal in humans

We know that every veterinary practitioner has their favourite method of removal of ticks from animals. Recommendations for tick removal in humans also vary, but we believe that the study of known tick anaphylaxis sufferers offers insights into *optimal* tick removal techniques. People who have anaphylaxis to ticks typically have the onset of their symptoms as soon as they remove or disturb the tick. Disturbing the tick and removing the tick whilst it is alive likely results in allergic reactions in those so prone due to either local dissemination of tick salivary allergens or the unwitting compression of the tick's salivary glands, resulting in tick allergens being injected into the vascular system of the host. Some patients who have killed the tick in situ, but then tried to remove the tick with tweezers have developed anaphylaxis following tick removal. ***However, known tick anaphylaxis patients who kill the tick in situ and either allow the tick to drop off after its death or remove the tick after killing it with fine forceps (tweezers) without compressing the tick salivary glands, very rarely, if ever, have an anaphylactic reaction!***

Figure 2. An example of an ether-containing spray that can be used to kill ticks in situ.

Ether-containing sprays (e.g. Wart Off Freeze® - Figure 2) will freeze a tick, killing it instantly. Larval and nymph stage ticks are not easily removed and are usually multiple (when the area to be covered makes ether spray use impractical) and tickicidal permethrin is currently advised

WHERE TO FIND FURTHER INFORMATION

If you or one of your pet-owners wishes to know more about tick-related disease issues in people, then further information is available at www.tiara.org.au. Tick-induced Allergies Research and Awareness (TiARA) was established at Royal North Shore Hospital in 2013. Its aims are:

- To promote awareness of tick-induced allergies by the public, health professionals, those in at-risk occupations, educators and government.
- To provide resources and support for sufferers of tick-induced allergies who live remote from expert medical and dietetic advisors.
- To promote research into the prevention & cure of tick-induced allergies.
- To disseminate established tick management strategies and help develop novel, proven tick management measures.

Additional sources of information are:

- www.allergy.org.au
- www.medent.usyd.edu.au
- www.allergyfacts.org.au

Reference

Hamsten et al. Identification of galactose-alpha-1,3-galactose in the gastrointestinal tract of the tick *Ixodes ricinus*; possible relationship with red meat allergy. *Allergy 2013* Apr; 68(4) 549-5552. doi:10.1111/all.12128.Epub 2013 Feb 18.

WINNER!

SNAILBAIT TOXICOSIS

C&T NO. 5417

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I treated this case last October and as none of the other 4 vets within the clinic had seen a case of Multiguard® Snail/Slug Killer intoxication in a dog, and as the product is marketed as a 'pet safe' alternative (which it is not if there's access to the packet!) I thought it may benefit publication.

I I must make mention of the useful article from a newsletter extract found on Google and written by the Southern Animal Referral Centre in Victoria.

Presenting complaint: Ingestion of unknown quantity of 'Multiguard®'

The owner heard 'Indi', a neutered 3-year-old Alaskan Malamute, as a little distressed in middle of the night. Indi went outside to toilet and remained outside for the night. The following morning the owner noticed many piles of red liquid and was unsure if they were vomit or diarrhoea (later presumed to be diarrhoea.) At this stage the owner found an open box of Multiguard® Snail Bait with most of the packet likely consumed. The active ingredient of the bait is Iron EDTA complex.

The 600g packet contains 60g/kg of IronEDTA, which calculates as 36g of IronEDTA which is the maximum ingested dose the dog may have received.

The reported lethal dose for iron is 100-200mg/kg.

Dog Weight: 30kg.

36,000mg (dose ingested)/30kg =1,200mg/kg i.e. 6-12 times the lethal dose.

The 30kg Alaskan Malamute was bright, alert and responsive and clinically well on physical examination. The main abnormal findings were tacky mucous membranes, moderately dilated pupils with poor pupillary light reflexes, and mildly painful on abdominal palpation. There was evidence of red staining around the mouth and anus relating to the red dye in the bait.

The patient was admitted to hospital and a treatment protocol started. Blood was collected for full analysis and a serum tube sent for iron levels.

An in-house VetScan coagulation profile returned normal aPTT and PT times. The PCV was 0.43 L/L and the TPP was 58g/L.

Bloods: mild left shift indicating some inflammatory demand. Biochem was unremarkable.

Urinalysis USG 1.017 (after 20mins of intravenous fluid therapy), colour normal and dipstick unremarkable.

Started on Hartmann's solution IV via with cephalic catheter at 5mL/kg /hr.

Premedication with IV dose of butorphanol and acepromazine.

Induction with 40mg alfaxalone IV and maintained on isoflurane and oxygen.

High volume water enema to evacuate red diarrhoea; also gastric lavage with warm water. Able to flush moderate amount of red dye and stomach contents.

Post anaesthesia pain relief 0.3mg (0.01mg/kg)buprenorphine (Temgesic®) and 1mL/10kg maropitant (Cerenia®)

Ranitidine 2mg/kg SLOW IV followed by oral 1g carafate tablet 30mins later.

The dog was monitored throughout the afternoon and became lethargic/sedate, with tongue partially hanging out and pupils more dilated than previously. HR remained stable; however, SPO2 was ~90% and respiration rate increased to approx 50. The dog was also mildly ataxic and had urinary incontinence.

After ringing around 5 local pharmacies to source desferoxyamine it became clear that it was very expensive and no-one stocked it on the shelf. A desperate call was made to the local base hospital which had a small supply available.

The desferoxyamine is a chelating agent and was started as a CRI at the recommended 15mg/kg/hr. The total dose given to the dog was 10xvials of 500mg.

The urine was monitored for an apparent colour change to 'rust' colour; however, we found this very difficult due to constant urine dribbling and being a female.

Gastrogel, which contains magnesium hydroxide, was also administered orally. MgOH, also known as 'milk of magnesium' helps slow absorption of iron from GIT. Activated charcoal does not work.

The dog remained stable late into the afternoon; however, due to low SPO2, nasal oxygenation was considered but not undertaken.

Furthermore, a blood lactate with Accutrend machine was done, results below:-
3pm; 3.1 mmol/L (normal<2.5mmol/L)
9pm: 2.4mmol/L

The dog remained in hospital for a further 2 days, and slowly improved. The urinary incontinence resolved, tongue tone returned, appetite was good, and faecal colour and consistency returned to normal. However, the PLR remained reduced.

The patient was discharged and went home on oral ranitidine and sulcrafate tablets.

The serum iron result was 64umol/L (15-42).

Post-script

Unfortunately the client did not bring the dog back for follow-up bloods, but a phone call in the few days following discharge indicated that the dog did return to normal.

INVITED COMMENTARY COURTESY OF

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All veterinarians and pet owners are encouraged to report any adverse events resulting from the approved use of a registered product. We've recently updated our website with new information on the program which can be found at **http://apvma.gov.au/node/69.** The take home message is that the APVMA is dependent on people reporting anything unexpected or adverse resulting from the use of a registered product – it's all about making sure that the directions for use on the label are appropriate to ensure the continued safe and effective use of a product.

INVITED COMMENTARY COURTESY OF:

Stephen Page

Advanced Veterinary Therapies
PO Box 905 Newtown NSW 2042
E. swp@advet.com.au

The use of iron EDTA (also known by the International Nonproprietary Name [INN] as sodium feredetate) as a snail and slug killer was the invention of the University of Melbourne researcher Colin Young. The product was specifically developed by Multicrop as an alternative to metaldehyde and methiocarb with a more favourable environmental profile and greater margin of safety to mammals and birds. Intoxication of companion animals consuming iron EDTA (available as Multiguard Snail & Slug Killer) has not been the subject of many reports since it was first registered in Australia in 1997. Prior to the current case from Tristan Robinson there is a single publication describing the presentation and management of 5 dogs at the University of Melbourne (Haldane and Davis 2009). More recently, Hall (2013) included iron EDTA is a review of iron toxicology. The APVMA annual reports of adverse experiences contain no reports of intoxication. However, the manufacturer has received a small number of reports. Interestingly, when first registered the manufacturer worked with the Australian Veterinary Association and a veterinary toxicologist to put together a treatment protocol which recommended no treatment unless signs of systemic iron toxicity are observed. A more recent treatment algorithm is included in the peer reviewed toxicology brief on iron toxicity by Albretsen (2006) (highly recommended and available at http://veterinarymedicine.dvm360.com/toxicology-brief-toxicity-iron-essential-element). The algorithm suggests that chelation therapy does not need to commence until serum iron concentration is greater than 500µg/dl = 90µM. However, it is essential to realise that all forms of iron are not equivalent and the toxicology brief does not specifically refer to iron EDTA. While iron EDTA is a preferred form of iron to fortify foods and is accepted as safe for this use, there is no information on the highest no observable adverse effect level in the dog. It may not be appropriate to extrapolate toxic doses from the more common forms of iron, particularly ferrous sulfate, ferrous fumarate and ferrous gluconate). These forms of iron were principally involved in over 30% of the deaths from accidental ingestion of drug products by children in the US between 1983 and 1991 (Manoguerra et al 2005) and remained an important cause of death until regulatory changes led to reduced exposure. But are treatment protocols for these forms of iron suitable for iron EDTA? An expert consensus panel assessing iron ingestion in humans (Manoguerra et al 2005) concluded that the literature did not provide solid evidence on a referral threshold dose of iron, but did conclude that a dose of elemental iron of 40mg/kg bodyweight was a reasonable threshold. Onset of symptoms was unlikely to extend beyond 6 hours. Mild symptoms such as vomiting and diarrhoea, should not necessarily prompt referral for treatment. So it may be reasonable to manage suspected intoxication conservatively on presentation if clinical signs have not developed.

The amount of elemental iron ingested is the starting point for calculations of exposure. Iron EDTA (as the trihydrate) has a molecular weight of 421 and the elemental iron content is 13%. A 600g pack of Multiguard which contains 6% iron EDTA therefore contains 0.78% iron = or 4,771mg. A 30kg dog consuming a whole pack therefore has a maximum exposure of 159mg iron/kg bodyweight. It should be recognised that Multiguard contains more than 80% wheat flour which may also contribute to toxicity. In addition, the product contains denatonium benzoate.

While it is highly likely that use of iron EDTA reduces dog intoxication by other molluscides, there are still a number of important research questions, previously emphasised by Haldane and Davis (2009). There must be a very clinically relevant and important **DVM, honours or masters project** in elucidating answers to the following questions:

1. What is the minimum toxic dose of iron EDTA in the dog?
2. Based on available evidence, what is a reasonable treatment algorithm for managing intoxicated dogs?
3. Does denatonium benzoate influence ingestion by dogs (no published data!!)

The manufacturer is very interested in answers to the first 2 questions and I expect would cooperate completely with any project.

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Hall, J. O. (2013). Chapter 51 - Iron. *Small Animal Toxicology* (Third Edition). M. E. Peterson and P. A. Talcott. Saint Louis, W.B. Saunders: 595-600.

Manoguerra, A. S., et al. (2005). Iron Ingestion: an Evidence-Based Consensus Guideline for Out-of-Hospital Management. *Clinical Toxicology* 43(6): 553-570.

MAJOR WINNER!

The Major Prize Winner is entitled to a year's free membership to the CVE.

PERI-OPERATIVE MANAGEMENT OF TRAUMATIC DIAPHRAGMATIC HERNIA IN A JUVENILE CAT

C&T NO. 5418

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ABSTRACT

An abandoned juvenile cat with mild respiratory compromise was subsequently diagnosed with a traumatic diaphragmatic hernia via plain radiography. Coeliotomy revealed a primarily radial diaphragmatic tear with a circumferential component. The patient was manually ventilated during herniorrhaphy. Minimal adjunctive monitoring devices were available to assess adequacy of ventilation and patient oxygenation, and financial restraints precluded placement of a thoracic drainage system. A post-operative pneumothorax was diagnosed during anaesthetic recovery. Therapeutic thoracocentesis resulted in resolution of respiratory embarrassment.

This case report aims to document diaphragmatic hernia in a paediatric patient managed with multiple practical and financial restraints, and compare this to historical findings and current recommendations from the literature.

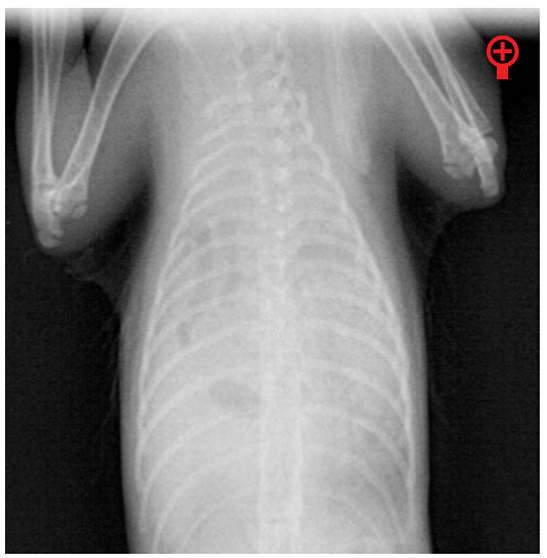

Figure 1: Dorso-ventral thoracic radiograph diaphragmatic hernia in a 1.2kg kitten. Note poorly defined diaphragmatic border and gas filled intestinal loops in chest.

CASE MANAGEMENT

A female juvenile cat found abandoned on a rural property was presented with a presumed littermate to a veterinary clinic for re-homing. On arrival at the clinic the kitten was estimated to be 12 weeks old and weighed 1.2kg. Initial health examination revealed no overt abnormalities.

Several days after presentation it was noted that the kitten tired more rapidly than her littermate and on more than one occasion was observed to be tachypnoeic. Thoracic auscultation at rest was unremarkable, as was a major body systems assessment. Conscious plain radiographs were obtained to investigate further (Fig. 1 and Fig. 2). A mild increase in respiratory rate was noted during restraint for radiographs but dyspnoea did not ensue. Normal respiratory function resumed without the need for supportive care as soon as the patient was released.

Radiographs revealed an indistinct diaphragmatic border ventrally, markedly increased thoracic opacity with loss of normal thoracic details (cardiac silhouette not visible), and evidence of cranial herniation of abdominal viscera.

A presumptive diagnosis of traumatic diaphragmatic hernia was made. Differential diagnoses included hiatal hernia, and congenital diaphragmatic or pericardio-diaphragmatic hernia.

A generous offer from a surgeon colleague resulted in surgical exploration and correction being pursued on a charitable basis. The kitten was in a stable condition and apart from exercise restriction to prevent development of a respiratory crisis, and close monitoring for secondary gastrointestinal complications (e.g. entrapment or torsion of herniated viscera), no treatment was instituted prior to the immediate pre-anaesthetic period.

Eight days after initial x-rays the kitten was scheduled for surgery. Methadone[a] 0.2mg/kg was given subcutaneously as a premedicant and pre-emptive analgesia. Pre-oxygenation with 100% oxygen via face mask was attempted prior to induction. Due to difficulties establishing intravenous access prior to induction, the patient was sedated with midazolam[b] 0.3mg/kg combined with ketamine[c] 6mg/kg given intramuscularly. Induction was completed with delivery of isoflurane[d] 3% in

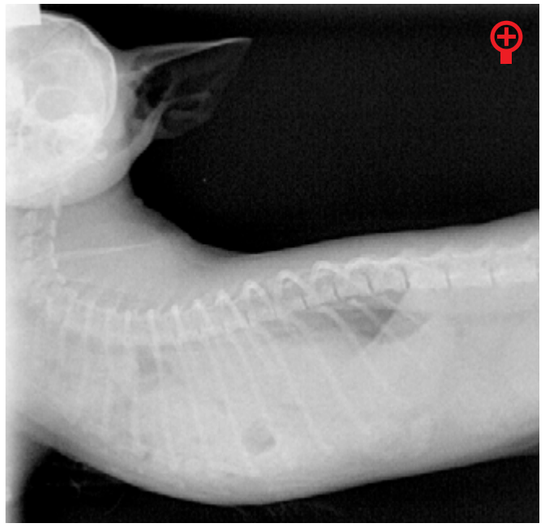

Figure 2: Right lateral thoracic radiograph showing diaphragmatic hernia in a 1.2kg kitten. Note indistinct line of diaphragm, obscured cardiac silhouette and gas filled structures within chest.

oxygen via mask. The patient was then intubated with a size 3 cuffed silicone endotracheal tube after desensitization of the larynx with local anaesthetic[e]. A surgical plane of anaesthesia was maintained with isoflurane[d] in oxygen gas, delivered with a non-rebreathing circuit. Intravenous access was established and isotonic crystalloid fluids[f] were administered intra-operatively at 10mL/kg/hr. Systolic blood pressure was monitored using Doppler flow detection and sphygmanometer[g]. Rectal temperature was monitored intermittently and thermal support provided via an external controlled heating device.

Intermittent positive pressure ventilation was commenced immediately following intubation to minimize the risk of hypoventilation and hypoxaemia. A rate of approximately 6 -8 breaths per minute was used, varied as necessary to maintain patient oxygenation and appropriate anaesthetic depth. Standard manual parameters (mucous membrane colour, capillary refill time, heart rate, pulse quality , ocular reflexes etc) in conjunction with Doppler blood pressure measurement were used to monitor adequacy of ventilation and anaesthetic depth, as appropriate adjunctive monitoring devices were not available.

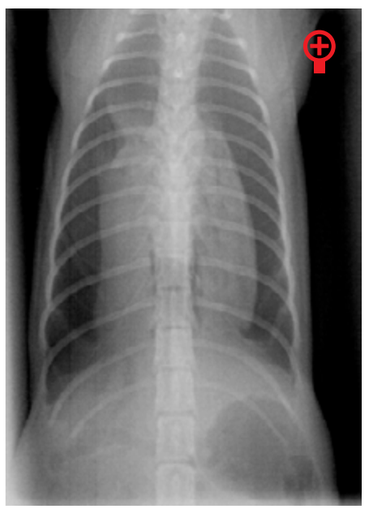

Figure 3: Dorso-ventral thoracic radiograph showing post-operative pneumothorax. Note marked collapse of pulmonary parenchyma. Diaphragmatic border is now visible.

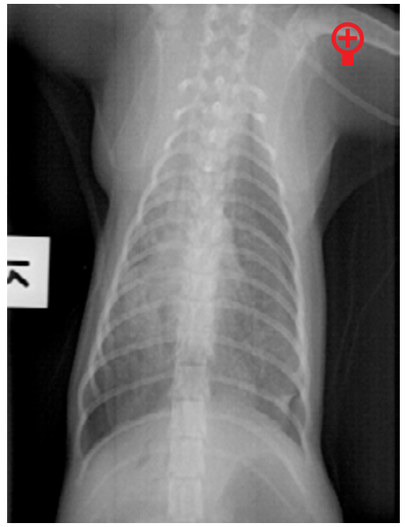

Figure 4: Dorso-ventral thoracic radiograph showing reduction of post-operative neumothorax.

The patient was positioned in sternal recumbency for as long as possible prior to sterile preparation of the surgical site and commencement of surgery. A ventral midline coeliotomy was performed from the xiphoid to the pubis. On entry to the abdomen the hernia was characterized: the left lobes of the liver, portions of the stomach, left limb of the pancreas, intestines and greater omentum were herniated through the diaphragmatic tear. Gentle traction was used to reduce the hernia contents before thorough examination of the diaphragmatic tear was performed. The tear was primarily radial, extending from the ventral abdominal wall to the caval foramen. A small circumferential component to the tear was additionally identified. No debridement of the edges of the hernia was performed. 4.0 polydioxanone[h] was used to appose the wound edges in a simple interrupted suture pattern. The final three sutures were all preplaced before ligation and care was taken to ensure adequate diameter remained for the passage of the caudal vena cava through the caval foramen. Close communication between surgeon and anaesthetist was employed to attempt gentle partial re-inflation of the lungs intra-operatively, but no attempt was made to fully inflate the atelectic lungs as the herniorrhaphy was completed. A 23 gauge intravenous catheter[i] was positioned through the suture line of the diaphragm prior to closure of the hernia and used to partially alleviate the surgical pneumothorax once the final sutures were tightened. The catheter was then removed prior to abdominal closure in a routine manner.

Discontinuation of gaseous anaesthetic was initiated on completion of the surgery and the patient was returned to sternal recumbency during the recovery period. 100% oxygen was supplied via reduced rate IPPV as the patient was monitored for return of spontaneous ventilation. Complications were suspected in the recovery period as the patient was slow to recover spontaneous ventilation, and dyspnoea was evident when respiration did commence. A cyanotic tinge was detected in the oral mucous membranes and breath sounds appeared reduced on thoracic auscultation. Radiography revealed a large volume pneumothorax (Fig.3).

Unilateral thoracocentesis was performed using a 23G butterfly needle with extension[j], 3-way tap[k], and 10mL syringe[l]. Approximately 50mL of air was evacuated from the pleural space. Repeat radiography confirmed adequate drainage (Fig.4). After relieving the pneumothorax the patient responded rapidly, resuming normal spontaneous respiration and recovering oxygenation. Breath sounds were improved on repeat thoracic auscultation. Anaesthetic recovery proceeded normally and the patient was extubated in the standard manner. Thermal support and intravenous fluids were continued in the immediate post-operative period, as was oxygen supplementation via flow-by. Analgesia was provided in the form of buprenorphine[m] 0.02mg/kg subcutaneously as required. The patient henceforth made a rapid and uneventful recovery from surgery and at 6 month follow-up was clinically well (Fig.5).

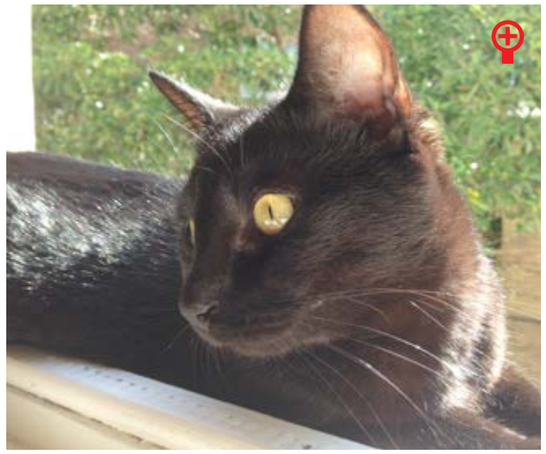

Figure 5: The Patient recovered and content in her new home

DISCUSSION

Presentation and Pathophysiology

Acquired or traumatic diaphragmatic hernia (TDH) results from rupture of the thin musculotendinous structure separating the thoracic and abdominal cavities, and is considered to be almost exclusively the result of blunt trauma to the abdomen.[26] Motor vehicle accidents have long been reported to be a leading cause in small animals in retrospective studies.[1,5,20,25] However, as here, there may be no discernible history of trauma of any kind; Garson et al (1980) only recorded a positive history of trauma in 28 of 56 cases in cats and dogs.

As reported in this case, the most common presenting sign with TDH is respiratory compromise.[4,5,17,23,25] The severity of respiratory compromise may vary considerably between individuals, and the pathophysiology has been attributed to multiple factors. Contributing factors may include loss of diaphragmatic function, pleural space filling defects (caused by abdominal organ herniation, fluid or air), pulmonary atelectasis due to compression by herniated viscera, secondary trauma-associated thoracic injury (rib fractures, pulmonary contusions), as well as the systemic effects of pain and hypovolaemic shock. TDH associated respiratory compromise ultimately can result in hypoventilation, hypoxaemia, tissue hypoxia, and cellular dysfunction or death.[1,26]

Given the severity of trauma required to cause diaphragmatic rupture, concurrent injuries such as rib, pelvic or long bone fractures, additional soft tissue injuries, and hypovolaemic shock are not unusual; Gibson et al (2005) reported a substantial percentage of concurrent injuries. Unsurprisingly, concurrent injuries may be associated with increased mortality rates; in a study by Schmiedt et al (2003) 83.3% of cats that did not survive had concurrent injury. No concurrent injuries were identified in our patient and she was haemodynamically stable at presentation and throughout the pre-surgical period. When analysed in terms of existing literature, this is likely to have had significant bearing on pre-operative stability and potentially post-operative survival.

Diagnosis

Respiratory dysfunction is the most common presenting sign of TDH. Clinical examination findings such as muffled/absent lung sounds, empty abdomen, and intra-thoracic borborygmi have also been described in diagnosis of TDH but their presence is variable or may not be pathognomonic for TDH.[4,17,23,25] Thus radiography is always indicated to confirm the diagnosis of TDH, and additionally is helpful to ascertain the presence of concurrent injuries.[2,23,26]

Diagnostic radiography must be performed with the utmost care to avoid aggravating respiratory compromise. The risk of respiratory failure and death in an already compromised patient should never be underestimated. In 1971, Wilson et al reported 2 out of 39 peri-operative TDH deaths were caused by positioning for radiographs. Hence stabilization for shock and respiratory distress in at-risk patients should occur prior to diagnostic radiography. Additional recommendations include pre-oxygenation, taking the fewest views required for a diagnosis (with the projection causing the least positional distress to the patient taken first), and use of minimal restraint.[2,26] Radiological findings in our patient were in agreement with typical findings in TDH which include loss of diaphragmatic line, obscuring of cardiac border, increased thoracic density, and intestinal gas shadows within the thorax.[2,4]

Occasionally a definitive diagnosis may not be reached using plain radiography alone. In such cases alternative diagnostics include contrast radiography, ultrasonography, or rarely exploratory laparotomy.

Pre-Surgical Management

Surgical correction of diaphragmatic hernia is ultimately required to restore diaphragmatic integrity and normal respiratory function. There has been conflicting reports in the literature regarding the recommended timing of surgery.[1,5] In the absence of a consensus on this issue, a decision on timing of surgery in the individual patient should be based on haemodynamic stability, the degree of respiratory compromise and presence and severity of concurrent injuries. Accepted indications for emergency surgical intervention include gastric herniation with tympany, ongoing haemorrhage and hypovolaemia unresponsive to fluid resuscitation, or unremitting abdominal pain.[26]

In order to minimize the already significant anaesthetic risk that TDH patients present, it is important that the patient is stabilized as much as possible prior to surgery. Indicated procedures or therapies may include intravenous fluid resuscitation, thoracocentesis, gastric decompression, analgesia, oxygen

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therapy, blood pressure monitoring, and blood and urine analyses.[3,20,21,26] Appropriate analgesia is particularly important in TDH patients to avoid the detrimental effects of pain (surgical or from concurrent injuries) on respiratory function.

It was fortunate that our patient was very stable on presentation and pre-operatively, thus required minimal interventions.

Anaesthesia and Patient Preparation

Attention to anaesthetic technique and patient preparation is essential in the patient with respiratory compromise. Inappropriate execution of general anaesthesia may precipitate decompensation even in the asymptomatic patient.[3]

Premedicants in TDH patients should be selected with the purpose of avoiding significant respiratory or cardiovascular depression. Recommended agents include low-dose phenothiazines, opioids, benzodiazepines, and low-moderate dose dissociatives, used alone or in combination.[3,11] Case series as early as 1965[23] report the benefits of pre-oxygenation via face mask in TDH patients, providing it does not result in further distress. Induction agents should be selected with the same criteria as premedicants. Intravenous induction is generally preferred to limit patient distress (and thus further respiratory compromise) and permit rapid control of the airway. More attention to achieving suitable sedation pre-operatively in our patient may have resulted in IV access being established more easily, allowing an intravenous induction and more rapid capture of the airway.

Immediately following induction the TDH patient should be intubated to allow protection of the airway and facilitate intermittent positive pressure ventilation (IPPV). IPPV, whether manual or mechanical, is an essential part of maximal support techniques in the patient with respiratory insufficiency.[3] The cumulative effects of ineffective diaphragmatic function, occupation of the thorax by abdominal viscera, surgically induced pneumothorax, and depressant effects of anaesthetic agents, make ventilatory support an absolute indication even in the asymptomatic TDH patient.[15] Sullivan and Reid (1990) attributed 2 deaths in their case series of 60 cat and dogs with TDH to a delay in commencement of IPPV following induction of anaesthesia.

Manual ventilation is labour intensive, tidal volumes may be erratic, and there is a tendency to hyperventilate.[13] However, as in this case, mechanical ventilation is often not available in first opinion practice. Potentially harmful effects of either form of IPPV include barotrauma and reduced venous return and cardiac output.[7,15] Recommended tidal volumes for IPPV are 10-20mL/kg at a rate of 8-20 breaths per minute, with peak inspiratory pressures limited to 20cm H_2O.[3,7,13,24] Earlier reviews[3] suggested that higher peak inspiratory pressures may be required in conditions where there is reduced pulmonary compliance, however it is now considered that over-zealous IPPV may have a role in the development of re-expansion pulmonary oedema, a potentially fatal complication associated with surgical correction on TDH.[11,27] Thus careful monitoring of airway pressures during IPPV is recommended to avoid barotrauma, and anaesthetists performing IPPV should not attempt to reinflate chronically collapsed lungs.[27] Close attention was paid in our patient to appropriate force and tidal volume particularly given her small size. However the inability to measure peak inspiratory pressures and administered tidal volumes was a disadvantage.

Patient positioning after induction and during surgical preparation is of utmost importance in TDH cases to prevent potentially fatal aggravation of respiratory compromise. Garson et al (1980) described death in a feline TDH patient after inadvertent positioning in lateral recumbency with the affected side uppermost.

Patient Monitoring and Potential Complications

Recognised complications of surgical TDH repair include re-expansion pulmonary oedema (RPE) and iatrogenic pneumothorax,[14,26] in addition to ischaemia-reperfusion injury reherniation, gastric torsion, shock, haemorrhage and cardiac arrhythmias.[17]

RPE is characterized by the onset of moderate to severe pulmonary oedema following rapid re-expansion of the pleural space or atelectic lungs.[18] It has been described as a cause of post-operative death in small animal patients undergoing TDH repair, and in a cat after correction of pectus excavatum.[4,17-20] RPE appears to be more problematic in feline patients. Various causes have been suggested but most authors agree the pathogenesis is multifactorial. Garson et al (1980) found that forceful lung reexpansion to control surgically induced pneumothorax likely contributed significantly to the development of fatal RPE. Thus it is now recommended that gradual re-expansion of chronically collapsed lungs is performed. Airway manometry during IPPV to avoid barotrauma, and gradual alleviation of surgically induced pneumothorax post-operatively via thoracocentesis or indwelling thoracostomy tube are recommended to prevent RPE.[4,19-20,26-27] Restrictions particular to our case did not allow for placement or maintenance of a thoracic drainage system, and facilities were not available to ensure appropriate airway pressures. We attempted to balance the risks of the major anticipated post-operative complications of re-expansion pulmonary oedema and pneumothorax by avoiding forceful inflation of the lungs with IPPV, but performing transdiaphragmatic thoracocentesis at completion of herniorrhaphy. As indicated by the striking pneumothorax diagnosed post-operatively, indwelling thoracic drainage may have been useful to allow gradual reduction of the pleural space.

Monitoring of the TDH patient peri-operatively is crucial to assess adequacy of ventilation, and in early detection of life threatening complications. While meticulous manual monitoring is essential, the judicious use of ancillary tools can provide extremely valuable information when interpreted critically.

The definitive method of quantitating the efficiency of ventilation and gas exchange is arterial blood-gas analysis.[3] The partial pressure of carbon dioxide in arterial blood (PaCO2) defines alveolar minute ventilation, and the partial pressure of oxygen in arterial blood (PaO2) defines the ability of the lungs to oxygenate the blood.[6] A disadvantage is the need for an arterial sample. In our paediatric patient serial blood gas analysis would have necessitated a femoral arterial catheter placed by surgical cutdown – invasive and impractical even had the facilities been available.

Pulse oximetry is widely advocated to monitor arterial oxygen saturation in any patient under anaesthesia, with particular value in the respiratory compromised individual. It is a non invasive method of measuring the oxygen saturation of haemoglobin (SpO2) in arterial blood. Although SpO2 is not linearly related to PaO2, pulse oximetry can still provide clinically relevant information regarding tissue delivery of oxygen. The advantages of pulse oximetry in comparison to arterial blood gas analysis are that it provides a continuous, non-invasive estimation of oxygenation.[8] However, pulse oximetry data must always be interpreted critically, as readings are subject to artefact and non-pulmonary factors.[24] A further obvious disadvantage is that no information is provided regarding arterial carbon dioxide levels.

Capnometry is the spectroscopic technique for measuring carbon dioxide in respiratory gases. End tidal capnography allows non-invasive dynamic assessment of adequacy of ventilation, because elimination of CO2 by the patient is the best determinant of alveolar ventilation.[12] Although end-tidal capnography has unique features that may be helpful in monitoring the critical patient or to individually tailor IPPV, studies have shown that end tidal readings can diverge significantly from arterial readings particularly in the diseased patient.[9,22] Hence arterial blood gas analysis remains the definitive method for evaluating carbon dioxide levels.[16] Capnography, particularly with sidestream analysers, also has limitations in patients such as ours that have a very small tidal volume.[16]

In the described case, minimal adjunctive monitoring devices were available to assess adequacy of ventilation and patient oxygenation. In light of current literature recommendations, and complications that occurred in our patient, even the availability of a pulse oximetry unit would have been extremely beneficial. Pulse oximetry would have allowed detection of hypoxaemia and instigated a search for the cause before the patient became clinically cyanotic. Despite the impracticalities for our particular patient, serial blood gas analyses would have provided valuable information – identification of hypercapnia would have signalled ventilatory issues prior to dyspnoea developing as a result of the pneumothorax. Pneumothorax-induced hypercapnia and subsequent respiratory acidosis was almost certainly the cause of delayed anaesthetic recovery in our patient, as it can result in loss of consciousness/stupor in the absence of anaesthetic agents.[6,10] Furthermore, the routine practice of taking a post-operative x-ray following surgical repair of diaphragmatic hernia would be sensible, allowing identification of pleural space abnormalities and pulmonary oedema in a timely fashion.

Acknowledgements

Thanks to Richard Malik and Chris Tan for their contribution and assistance with this article.

Footnotes:

a. Methadone Injection; Ilium.
b. Midazolam Injection; Pfizer
c. Ketamine Injection; Parnell
d. Isoflurane Inhalation Anaesthetic; Pharmachem
e. Xylocaine 4% Topical; AstraZeneca
f. Hartmanns' Solution; Baxter
g. Ultrasonic Doppler Flow Detector Model 811-B; Parks Medical Electronics, Inc. Aloha, Oregon, U.S.A.
h. Surgicryl; SMI
i. SurFlo IV Catheter 23 x ¾", Terumo
j. BD Vacutainer Safety Lok Collection Set; Becton, Dickinson and Company
k. BD Connecta; Becton, Dickinson and Company
l. BD 10mL Syringe; Becton, Dickinson and Company
m. Temgesic Injection, Reckitt Benckiser

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COMMENT ON PERSPECTIVE 107: LIVE ANIMAL EXPORT IS UNETHICAL BY PETER KERKENEZOV

(FIRST PUBLISHED IN THE C&T SERIES JUN 2014, ISSUE 275 EBOOK)

C&T NO. 5419
Dr Sue Foster

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Vets Against Live Export (VALE)

The issue of animal welfare in live export has become very sharply focused in the last few years since revelations of cruelty to Australian animals exported to Indonesia.

Vets Against Live Export (VALE) is an incorporated body of veterinarians whose aims include providing a source of objective scientific information on the welfare of animals exported live from Australia. While the treatment of Australian animals in importing countries is a major cause for concern, VALE believes there are problems of an equivalent magnitude associated with other aspects of live export, particularly the long-haul sea voyages.

VALE members include veterinarians who have been on live export voyages, veterinarians with extensive experience in abattoirs, animal welfare specialists, veterinary behaviourists and large animal veterinarians. Members and advisors also include senior academics from Australian and US veterinary schools.

VALE concentrates on veterinary and welfare aspects of all stages of the live export process but particularly on welfare associated with the voyages. VALE members have now published two peer reviewed articles in ***The Veterinary Journal***: ***Heat Stress: a major contributor to poor animal welfare associated with long haul live export voyages*** by Caulfield et al (2014) and ***The welfare of Australian livestock transported by sea*** by Foster and Overall (2014). Both are available online as they have been published open access.[1,2] In addition, VALE regularly contributes to veterinary and public media and provides relevant expert veterinary opinion on health, welfare, disease and slaughter when requested.

VALE analyses all high mortality voyage information in addition to any information made available by the Department of Agriculture, Fisheries and Food (DAFF) under Freedom of Information. The latter is notoriously difficult to retrieve, is invariably heavily redacted (censored) and is often expensive to access. Routine requests for information and clarification are made to DAFF as can be seen from VALE's website (www.vale.org.au). Such requests usually result in delayed replies, incomplete replies or no replies. The lack of transparency by the body regulating this trade, highlighted by this correspondence, is concerning to veterinarians attempting to assess animal welfare in the live export trade.

Of equal concern to both the Australian Veterinary Association[3] and VALE is the lack of independent veterinarians on live export ships; this was also noted as a concern in the Keniry Enquiry.[4] Veterinarians are currently employed by the exporters. Veterinarians such as Dr Peter Kerkenezov, Dr Lloyd Reeve-Johnson, Dr Tony Hill and others who have made critical comments to DAFF, have not been re-employed. Experienced live export veterinarian Dr Lynn Simpson, who made a public submission regarding breaches of ASEL (Australian Standards for the Export of Livestock)[5,6] on live export voyages, was removed from her position in the Animal Welfare Unit at DAFF and is still on stress leave. Thus, critical veterinary assessment that could result in improved animal welfare is actively discouraged. It should also be noted that earlier this year, the DAFF Animal Welfare Unit ceased operation.

If this trade is to continue there must be easy access to comprehensive data, independent veterinary oversight both pre-embarkation and on-board and active compliance to standards with appropriate penalties imposed. Currently, all three are lacking. It is likely that all three will be vigorously resisted by the industry and government. Until independent data confirming compliance to current standards and acceptable welfare at all stages of the live export trade is available, VALE will continue to actively and strongly oppose this trade.

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1. See Caulfield et al: http://www.vale.org.au/uploads/1/0/4/3/10438895/heat_stress_paper.pdf
2. See Foster and Overall: http://www.sciencedirect.com/science/article/pii/S1090023314001014
3. See AVA Position Statement 15.1 Live Animal Export: http://www.ava.com.au/policy/151-live-animal-export
4. See Livestock Export Review: http://www.daff.gov.au/__data/assets/pdf_file/0008/146708/keniry_review_jan_04.pdf
5. See Australian Standards for the Export of Livestock (Version 2.3) 2011: http://www.daff.gov.au/__data/assets/pdf_file/0010/1904365/australian-standards-v2.3.pdf
6. See submissions: http://www.daff.gov.au/animal-plant-health/welfare/export-trade/submissions-export-livestock

COMMENT

Dear Editor

I would like to thank Peter Kerkenezov for the C&T Perspective No. 107 on live animal export. I was myself already opposed to the practice on welfare grounds, but his fine exposé of the industry has added to my knowledge and determination. I deeply believe that veterinarians are custodians of animal welfare and was dismayed that the leadership of the AVA chose to support business interests (that of the farmers exporting overseas, as well as of the veterinarians they employ) in the face of the stresses inherent to the voyages and the lack of control over treatment of the surviving animals by the destination countries. The article has made me aware of Vets Against Live Export Inc (VALE) which I have now joined. I have in the past written to my local MPs to increase awareness of this issue in parliament and was pleased to find out that that my local ALP member for Throsby, Stephen Jones, had made a speech against this practice in front of the NSW House of Representatives. Indeed, other MPs oppose the trade and can be appealed to for further action. I believe however that the veterinary profession should be a leader in this debate, and that Dr Kerkenezov's article has a wealth of experience backed by powerful images which make it invaluable.

M Richard BVSc

TOXICOLOGY BRIEF: STONEFISH ENVENOMATION IN DOGS

C&T NO. 5420

Rhian B Cope BVSc BSc(Hon 1) PhD cGLPCP DABT ERT FACTRA
Rosalind Dalefield BVSc PhD DABT DABVT

Introduction

A recent fatal stonefish envenomation of a dog in Redcliff (near Brisbane) presents a timely reminder to veterinarians that these potently venomous animals are a serious risk to dogs that enter coastal waters within their range. The recent case occurred with a dog that was paddling in the shallows of a residential canal development. This illustrates that these fish are fully capable of living in the normal human and pet urban environment. Indeed, indigenous Australians who live in coastal areas with endemic stonefish populations were very much aware of the need to coexist (i.e. completely avoid) these animals and have a traditional dance that specifically warns about the danger of encounters with stonefish [1]. The traditional dance conveniently includes a wooden model of the fish complete with the characteristic 13 dorsal venomous spines in order to aid identification (and avoidance) [1]!

On average there are about 25 human stonefish envenomations per year in Australia, with most occurring in Queensland and the Northern Territory. Stonefish antivenom is the second most used antivenom in humans in Australia.

Veterinary-Relevant Zoological Features

There are currently 2 known species of stonefish (Figure 1) in Australian waters: the reef stonefish (***Synanceia verrucos***) and the estuarine stonefish (***Synanceia horrida***, synonym ***S. trachynis***). Both are ambush predators and are typically superbly camouflaged, often with their own epiphyton of algae. True stonefish have 13 non-branching venomous spines (Figure 2). This characteristic distinguishes it from species that are batesian mimics (many of which are also venomous, but less so than stonefish).

Stonefish are distributed from Woolgoolga on the northern New South Wales coast (just north of Coffs Harbor), over the entire Queensland coast, across the Northern Australian coast and down the Western Australian coast to as far south as Geraldton. As with scorpion fish, their distribution is likely to expand with ongoing oceanic warming. The same species are found in numerous coastal areas in the Indo-Pacific region as well as along the Florida coast and the Carribean.

Stonefish are typically found in relatively shallow waters with the reef species preferring coral reefs or stony/rocky bottoms. The estuarine species can also be found in sandy areas or in mud and seems to be more tolerant of fresh water.

In Queensland, humans and their pets in and around boat ramps and in residential canal developments commonly encounter stonefish. Local residents of these areas are usually well aware of the need to wear protective, thick soled, footwear when wading in stonefish habitat.

Stonefish have also developed a following amongst salt-water aquarium hobbyists. One of the authors has been presented with an aquarium kept specimen for veterinary treatment: a significant professional hazard since these animals should be handled with a very high level of caution, if at all. If in doubt, do not touch or handle!

Venom Characteristics

Stonefish are the most venomous fish species in Australian waters and their venoms are amongst the most toxic known marine venoms. The venom is heat labile (indeed in some countries such as China, Japan and the Philippines, stonefish are eaten as a delicacy and are perfectly safe provided they are adequately cooked – heating inactivates the venom). The acute toxicity in mice of stonefish venom is comparable to snake venoms (mouse LD_{50} 0.36 mg/kg or about 11 mg for a 30 kg dog, assuming similar sensitivity).

As with most venoms, stonefish venoms are complex mixtures and contain about 13% protein. A number of the important subcomponents and venom activities have been investigated:

- Stonefish venoms contain a very potent hyaluronidase (many-fold more potent than those found in snake venoms; aka 'spreading factor'). The hyaluronidase may be responsible for the extensive necrosis that is occasionally (i.e. rarely) encountered with stings in humans [1-4].
- Stonefish venoms are potent cardiovascular and neuromuscular toxins that produce bradycardia (even in the presence of substantial hypotension) and depolarizing neuromuscular blockade [1]. Limb paralysis and death from respiratory muscular paralysis are noted features of stonefish venoms in experimental rodents.
  The cardiovascular effects are mediated by muscarinic receptors and adrenoceptors. Depolarizing neuromuscular blockade associated with the estuarine stonefish (*S. horrida*) venom is due to a massive and sustained release

of acetylcholine from the pre-synaptic membrane and is mediated by activation of the SNARE system combined with formation of non-selective membrane pores by trachynilysin, a venom protein [5-9].
Stonustoxin from the estuarine stonefish also produces depolarizing paralysis of the diaphragm, contributing to respiratory paralysis [10].

- The stonefish venom lethal factor (stonustoxin) has a LD_{50} and is about 17 times lower than crude whole venom [11]. Stonustoxin is a potent hemolytic and oedema-forming toxin [12]. The oedema forming action is not dependent on the release of histamine and can be severe and long-lasting e.g. a 30% increase in leg weight lasting more than 24 hours in experimental envenomation of rodents. Stonustoxin is also a potent inducer of hypotension and counteracts the pressor/vasoconstrictor activity of noradrenalin (notably in the aorta) [13]. The central arterial dilator activity of stonustoxin, combined with volume loss due to its oedema forming action, are considered to be the major causes of the severe (and often difficult to treat) hypotension that can occur with stonefish stings.
  A second stonustoxin-like protein, neoverrucotoxin, has also been isolated [14].
- Verrucotoxin is a glycoprotein venom factor that is hemolytic and induces hypotension. It is a calcium channel blocker and affects myocardial K^+(ATP) channels via the muscarinic M3 receptor pathway [15, 16].

Epidemiology

Perhaps predictably, currently available data indicates that most human stings (and anecdotally, most stings in dogs) occur in the summer an occur in visitors to tropical areas that have stonefish habitats (i.e. have no awareness of the risk of stonefish stings) [17]. There are possibly two factors involved with this phenomenon: humans are more likely to enter stonefish habitats during the summer and stonefish may have a period of seasonal dormancy during the colder months.

Unfortunately there are very few veterinary case reports available. However, stonefish envenomation of dogs is known to occur based on media reports[1] and the lead author is aware of fairly regular anecdotal reports from particular locations in Queensland (coastal urban areas around Moreton Bay, Queensland).

Clinical Toxidrome

Sadly, the authors are not aware of any dogs that have survived being stung. All cases of canine envenomation that the authors are aware of have been uniformly fatal, despite attempts at treatment in some of the cases. Dogs with significant stonefish stings appear to develop immediate severe pain, rapidly collapse and develop hypotension that is difficult to treat. Bradycardia has been noted in some cases.

This contrasts with the situation in humans where stonefish stings are very painful and unpleasant, but they are usually eminently treatable with antivenom and analgesia. Stonefish stings in humans are only very rarely lethal with one documented fatality occurring in Australia in 1915 [18]. In contrast with the toxidrome in dogs, severe systemic toxicity in humans (i.e. collapse, hypotension, bradycardia, pulmonary edema and respiratory paralysis) are very rare.

Treatment of Dogs with Stonefish Stings

The authors are not aware of any successful treatment of significant stonefish envenomation in dogs . At least in theory, immersion of the affected limb in hot (45^{o}C) water as soon as possible following the sting should help to reduce the pain and inactivate the venom (the venom is heat labile). In humans, hot water immersion seems to offer benefit in about 75% of human cases and it s the mainstay of treatment in a number of countries where antivenom is not available [17, 19].

It is assumed, but currently unproven, that rapid administration of antivenom would be helpful in canine cases. Stonefish fish antivenom treatment is often impractical in veterinary situations because it is expensive and rarely stocked in veterinary practices. Limited stocks of the antivenom may be available in selected human hospitals.

Current clinical experience indicates that even aggressive treatment of shock and hypotension following stonefish stings in dogs is generally not successful. It should be noted that stonustoxin counteracts the arterial pressor actions of catecholamines such as adrenalin and noradrenalin. Additionally, given the effects of stonefish venom on vascular integrity, the risk of pulmonary edema following aggressive treatment with intravascular crystalloids may be increased. Breakdown of vascular integrity may also pose additional risks with the use of colloidal volume expanders.

Prevention

Stonefish stings in dogs are generally completely preventable. Lack of owner awareness is the most common challenge, particularly for owners who have recently moved into coastal areas with known stonefish populations.

Dogs should not be allowed to paddle or wade in known stonefish habitats, particularly in the warmer summer months. Dog boots, which are usually relatively thin, are unlikely to offer adequate protection against stonefish spines.

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Figure 1: The reef stonefish,* Synanceia verrucos. *The upper image is a wild coral reef specimen that is camouflaged to resemble coral. The bottom image is an aquarium-kept specimen

Figure 2: Stonefish dorsal spines. Each of the 13 dorsal spines has a venom gland located at the base of the spine. If a spine is stepped on (as in the upper illustration), the pressure forces a large volume of venom up a groove in the spine and into the dermal and subcutaneous tissues. The spines are capable of penetrating through thin-soled shoes (e.g. surfing/diving booties).

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WINNER

RAT BAIT

QUESTION POSED ON THE CVE/ISFM DE FELINE MEDICINE LISTSERVE

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Just wondering what everyone else would have done in this situation.

A 3-year-old Ragdoll presented to the clinic with haematuria. A urine sample was taken, SQ fluids given and pain relief whilst awaiting the results.

Two days later, presented to me as cat gone downhill. Cats MM are WHITE. Temperature is low and the cat is really flat. Took more history from the owner, and it turned out that the cat was playing with containers that had rat bait paste in them.

So did bloods:

Test	Result	Range
RBC	2.74 x 10^12/L	6.54-12.2
HCT	11.60%	30.3-52.3
HGB	4.3g/L	9.8-16.2
MCV	42.3fL	35.9-53.1
MHC	15.7pg	11.8-17.3
MCHC	37.1 g/dL	28.1-35.8
RDW	20.20%	15-27
Retic	35.6K/µL	3.0-50
WBC	11.6x10^9/L	2.87-17.02
%neu	79.4	
%lym	17.2	
%mono	3.2	
%eos	0.1	
%baso	0.1	
neu	9.22x10^9/L	1.4/8-10.29
lym	1.99x10^9/L	0.92-6.88
mono	0.37x10^9/L	0.05-0.67
eos	0.01x10^9/L	0.17-1.57
baso	0.1x10^9/L	0.01-0.26
plt	35K/µL	151-600
PT	>100 secs	15-12.2

So gave Vit K, IV fluids, warming but needed clotting factors NOW.

Here is the question: Fresh dog blood (had a German Shepherd willing to make a donation) or dog fresh frozen plasma?

In the end, I decided canine plasma was safer and gave that, given that PCV of 11% was just compatible with life. The cat survived the night and just came back for PT retest after 10 days Vit K. PT was normal with a PCV of 39%, so the cat did well but did I just get lucky? Should I have given the whole blood?

Really, I know the ideal would be to have typed 'donor cats ready and waiting' but we don't really have any willing staff cats. But at least we are now looking at getting typing kits in instead of having to send blood off to the lab.

Please email your replies for publication in the December 2014 Issue 277 to: Elisabeth.churchward@sydney.edu.au

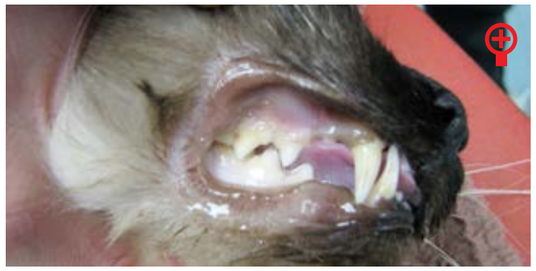

Figure 1. Gums at first presentation.

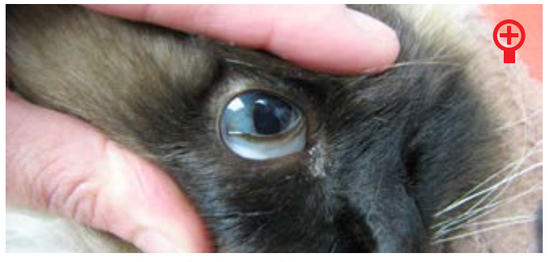

Figure 2. Conjunctivae at first presentation – very pale!

Figure 3. Gums after 10 days of vitamin K.

HANDY TIP FROM DE FELINE MEDICINE LISTSERVE

Great resources to measure blood pressure in cats can be found at Sarah Caney's website: http://www.vetprofessionals.com/catprofessional/free_downloads.html

WINNER

POSSIBLE ALFAXAN REACTION

C&T NO. 5422
Geoff Hayres

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I had a clinically normal female kitten (1.3kg) presented for desexing. I administered premed S/C of 0.1mL (100µg) medetomidine, about an hour later. Zero point 3 mL *Alfaxan®-CD* (10mg/mL alfaxalone) was given slowly I/V, the kitten intubated with a 2mm non-cuffed endotracheal tube and connected to isoflurane/oxygen. Our pulse oximeter would not provide a reading. As I incised the skin my very alert nurse noticed a lot of fluid in the kitten's mouth. I also noted the cut surface of the incised skin was poor; the gums were dark red and the pupils totally dilated with very loud fluid rales in the lungs on thoracic auscultation. I gave 5mg frusemide I/M and 0.15mg dexamethasone I/M, then 'reversed' with 0.05mL (25µg) atipamezole, 5mg/mL, also I/M. Nothing much changed, so I gave another 5mg of frusemide and then finally realised this was probably anaphylaxis or an anaphylactoid reaction to alfaxalone. I gave 20µg adrenalin (0.02mL of 1/1000 adrenalin), also I/M. Still little changed; the rales were unchanged, so I extubated the kitten and suspended it by the back legs to 'drain' the lungs of fluid; this certainly reduced the rales. Oxygen was continued by mask, another 5mg frusemide was given I/M. I was about to repeat the adrenalin when the kitten suddenly stirred and 'came around'. She was very disoriented but within 30 minutes was 'quite good'. She was successfully desexed 2 days later with a premed of ACP, I/V propofol, isoflurane and oxygen maintenance.

Since this experience I have heard of 2 similar cases: one had methadone premed, the other medetomidine, then I/V alfaxalone induction and isoflurane/oxygen maintenance. In the crisis, neither had adrenaline; one lived, one died. On the AVPMA website, I looked at 3 years of reports (2009/2010/2011) and noted about 10 reports each from those years, with comments like 'rales', 'respiratory problems' and 'pulmonary oedema'.

I have used large amounts of *Alfaxan®* over the years and I like the product. This reaction certainly took me by surprise, and the point of this article is to create awareness as to the possibility of anaphylaxis/anaphylactoid reaction to alfaxalone, which does not seem to be a new phenomenon, and to remember adrenalin, for which the dose in cats is 10-20µg/kg. I think the frusemide, dexamethasone and 'draining' also played significant roles.

Note: Members/readers are reminded to report all such cases to both the manufacturer and APVMA.

EDITOR'S NOTE
from Richard Malik, CVE Valentine Charlton Specialist

Geoff vividly describes a near death following induction of general anaesthesia in an apparently normal kitten. We need to consider 2 things:-

1. What is the pathophysiology of the 'episode'?, and
2. Was it related to the Alfaxan™ or the medetomidine, or both, or was it related to an undetected comorbidity e.g. congenital heart disease?

My view is this kitten had acute pulmonary oedema. I have no idea if it was cardiac, or non-cardiac. Knowing the protein concentration of the expectorated fluid would have been informative. Anaphylaxis in cats has been studied in cats after incompatible blood transfusion (type A blood to type B cat) and after administration of crushed heartworm, and the key features are bradycardia, hypotension, reduced cardiac output and bronchoconstriction. So I am by no means convinced the patient had a reaction characterised by massive widespread mast cell degranulation. Instead, I suspect the cat had acute left-sided heart failure, or perhaps a pulmonary thromboembolic event. An echocardiogram soon after the event may have been informative.

Anaphylactoid injections were common after administration of Saffan (alfaxalone in cremophor vehicle). These reactions were highly variable in extent, but a feature was swelling of the distal limbs (angioedema) due to histamine release. The culprit was the vehicle, not the active drug, and to the best of my knowledge such events have not been seen with the cyclodextron vehicle.

Read full explanation here...

Note: When searching on pubmed, ALFAXALONE (US & UK) retrieves entries for both spellings, but ALPHAXALONE (Australia) will only retrieve the subset that use this spelling.

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INVITED COMMENTARY COURTESY OF:

Meaghan Bell BSc (Hons) BSc BVMS
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Thank you for giving us the opportunity to comment on this C&T.

Jurox is the manufacturer of Alfaxan® Anaesthetic Injection. Dr Hayres reported this case to Jurox, and we collated the information about the case and submitted a report to the APVMA as part of our international pharmacovigilance obligations.

When considering this individual case

- Whilst it may seem semantics, Alfaxan currently available in Australia is Alfaxan Anaesthetic Injection – this is a different product to Alfaxan CD, which was a product registered in the early 2000s and has not been available since about 2002. That product was different in a number of ways to the current formulation. Our discussions with Dr Hayres indicate that this case was associated with Alfaxan Anaesthetic Injection.
- The patient received three (3) medications before the clinical signs of apparent pulmonary oedema. Whilst the time between administration of the Alfaxan and the clinical signs is suggestive of a causal effect, this is at best a suggestion.
- Dr Malik points out that the signs may fit more with acute left-sided heart failure. The premedication regime of medetomidine and the maintenance agent isoflurane have been well recognised to modify cardiovascular function. Alfaxalone has also been shown to modify cardiovascular tone, albeit in supraclinical doses in unpremedicated patients. The potential for the signs seen in this patient to be a result of a combination of the drugs administered and the animal's own underlying physiology always needs to be considered.
- The fact the patient was anaesthetised successfully in subsequent days without Alfaxan is not necessarily supporting evidence of a causal link to Alfaxan, especially considering that ACP was used as a premedication, a drug with significantly different cardiovascular effects to medetomidine.
- Jurox and the APVMA have both classified this case as having a 'possible' causal link to Alfaxan, using the APVMAs causality algorithm.

From a more broad perspective

- Anaesthesia is not a benign process. It is essentially applied pharmacology and physiology. The process of becoming anaesthetised itself changes cardiovascular and physiologic functions. When a young animal (neonate or juvenile) is added to that mix, compensatory mechanisms that can be expected to create a buffer for an adult animal may not act in the expected manner and a patient may respond differently.
- An independent survey in 2011 showed that Alfaxan is the most common first choice anaesthetic induction choice in Australian veterinary practices. Many hundreds of thousands of anaesthetics are performed annually with Alfaxan around the world.
- As a result of the above two points, Jurox investigates many reports of possible 'adverse effects' associated with Alfaxan and our other peri-operative medications. These possible 'adverse effects' take many forms, including those described in this report and even, occasionally, the angio-oedema that Dr Malik comments on.
- Such events can be very difficult to assign a causal link to any one factor or drug. Polypharmacy is practiced as part of balanced anaesthesia and the protocols of anaesthetic care and monitoring vary greatly between reported cases. Many of the cases that are reported to Jurox could be considered as 'multifactorial anaesthetic associated adverse events' rather than being causally linked to any one drug or aspect of the patient and procedure.
- Any drug is capable of inducing an 'anaphylactic' or 'anaphylactoid' reaction. It must be remembered that it is the 'drug' not necessarily the active. Alfaxalone is a neurosteroid molecule with a molecular structure very similar to progesterone. It is difficult to comprehend that a steroid molecule can cause anaphylaxis as these are created endogenously. The formulation also contains hydroxy-propyl betacyclodextrin, saline and various electrolyte buffers. There is little evidence of any of these components acting as allergens; however, it is possible that this can occur. Having said that, it is difficult to see a steroid molecule as a likely allergen; the formulation of methylprednisolone sodium succinate used in human hospitals has been recognised to pose a risk of anaphylaxis or similar reaction. Again, this may be formulation rather than active associated.

Pharmacovigilance is an important part of pharmaceutical safety. Readers are advised to contact pharmaceutical companies and/or the APVMA if they have any concerns about potential adverse drug events.

REPLY TO JUROX'S COMMENT FROM GEOFF

Thanks for the info from Jurox, all fair enough, and Meaghan was good to deal with from the start. I have always liked Alfaxan and continue to use it in both dogs and cats. Interestingly, I personally have heard of 2 similar cases and there are similar adverse reaction reports. However, if these reactions are all physiologic, and indeed I myself have always regarded cats as an 'inflammatory reaction waiting to happen', then the article will at least alert others as to what can happen and help with what to do should it occur. I knew this article may stir up some controversy and even mentioned I would understand if CVE didn't want to publish it. In my professional life I have always has a 'case mortality' or 'case morbidity review', some of which went on in my head all night for long periods of time, I have always tried to understand not only what went wrong but try to stop it happening again. I would be much quicker to react myself should this ever happen to me again, I hope it may prevent one of my colleagues from losing a patient that may have otherwise have been saved.

INVITED COMMENTARY COURTESY OF:

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The report from Geoff Hayres describes an adverse experience in a female kitten following the administration of medetomidine (SC), alfaxalone (IV) and isoflurane/oxygen (IT). In investigating the relationship between the adverse event and the medications administered it is useful to confirm the dose rate(s) administered, to review available information on adverse experiences, and to consider the criteria for assessing causality.

The dose rates described are all consistent with label recommendations. But has the adverse syndrome been described before? Complicating this case is the fact that a number of medications were administered and it is not obvious if the adverse experience is associated with one or a combination of medications.

A very valuable starting point when searching for previous reports is the US Center for Veterinary Medicine Freedom of Information (FOI) summary for each product (but only available if the product is also approved by CVM) available at http://www.fda.gov/AnimalVeterinary/Products/ApprovedAnimalDrugProducts/FOIADrugSummaries/.

The FOI summary for Alfaxan presents the results of studies on 572 cats (480 from efficacy studies and 92 from safety studies). The studies include the (safe) use of Alfaxan in juvenile cats of similar bodyweight to the kitten in the current case. Furthermore, the compatibility of medetomidine as a preanaesthetic agent prior to Alfaxan was studied in 6 adult cats. While the duration of anaesthesia was extended with this combination in some cats, adverse effects are not described. At the recommended dose, adverse experiences were uncommon and not life threatening. Only at the high dose of 25mg/kg were adverse signs of excess upper airway sounds and clear fluid in the endotracheal tube observed.

Other useful information can be gleaned by reviewing online adverse experience reports. For example, the APVMA Adverse Experience Reporting Program annual reports from 1995 to 2012 are available at http://apvma.gov.au/node/10946. For medetomidine, over a 27 year period there are 5 reports of pulmonary oedema in cats (3 between 1995 and 2003, and one report in each of 2004 and 2007). For alphaxalone, the annual reports for 2003 to 2012 include a total of 101 possible or probable adverse experiences, including 5 reports of pulmonary oedema, 5 of rales, 12 identified as 'respiratory problems' and one report of an anaphylactoid reaction (2004). It is important to note that the APVMA reports refer to the active ingredient only and not to the specific formulation. Therefore, it is unclear how many of these reports refer to the same product used in the current case.

The CVM Adverse Drug Experience (ADE) cumulative reports from 1 January 1987 to 30 April 2013 are available at http://www.fda.gov/AnimalVeterinary/SafetyHealth/ProductSafetyInformation/ucm055369.htm. The entry for medetomidine in the cat lists 16 reports as 'edema, lung(s)/trachea'. There are no reports for alfaxalone, presumably because the product has only been recently approved.

Finally, causality assessment is essential. While there are a number of approaches, the APVMA applies six criteria – previous experience with the product; alternative aetiological candidates; evidence of overdose, timing of events; dechallenge; and rechallenge. Applying these criteria to the current case, with respect to either medetomidine or alfaxalone yields a possible association with use of either agent. Greater certainty or strength of the relationship can only be determined if all observed suspected adverse experiences are reported to the manufacturer and the regulator (APVMA in Australia) and Geoff is to be applauded for raising this case for discussion.

ANZCVS MEMBERSHIP EXAMS

FROM THE DE FILES

Congratulations to CVE Distance Education Alumni who successfully passed their examinations in 2014

Carla Appelgrein, Small Animal Surgery
Susan Carr, Small Animal Medicine
Karim Chammas, Medicine of Cats
Jennifer Chau, Veterinary Radiology (Small Animal)
Jeffrey Clyne, Veterinary Emergency & Critical Care
Abbie Couper, Veterinary Emergency & Critical Care
Mihai Csatai, Veterinary Emergency & Critical Care
Julie Culver, Veterinary Behaviour
Natsuko Druery, Veterinary Emergency & Critical Care
Rebecca Francis, Small Animal Medicine
Nicola Gaut, Medicine of Cats UK
Joanna Goldman, Veterinary Behaviour
Kylie Grant, Small Animal Surgery
Clifford Ho-le, Veterinary Emergency & Critical Care
Karin Holler, Medicine of Cats
Nicole Hoskin, Small Animal Dentistry & Oral Surgery
Marie-Theres Hoyer, Medicine of Cats
Olutunbi Idowu, Small Animal Medicine
Isabella Lam, Veterinary Emergency & Critical Care
Katie Le Messurier, Small Animal Medicine
Leah Manning, Small Animal Medicine
Lynn Mathison, Veterinary Behaviour
Joanna McLachlan, Veterinary Behaviour
Dawn Mills, Avian Health (Poultry)
Ingrid Nash, Veterinary Emergency & Critical Care
Jennifer Nesbitt-Hawes, Veterinary Behaviour
Joanna Pilton, Veterinary Radiology (Small Animal)
Helen Purdam, Veterinary Behaviour
Ema Rankin, Small Animal Medicine
Muna Roberts, Medicine of Cats
Sarah Robson, Veterinary Pharmacology
Margaret Roser, Veterinary Pathology (includes Anatomical & Clinical Pathology)
Natalie Rouillard, Veterinary Radiology (Small Animal)
Reza Singam, Surgery of Horses
Nadia Sternberg, Medicine of Cats
Rachael Stratton, Veterinary Behaviour
Michelle Sutherland, Avian Health (Caged & Aviary Birds)
Sarah Tawse, Veterinary Emergency & Critical Care
Sandra Tothill, Medicine of Cats
Leah Wright, Small Animal Medicine
Genevieve Zhang, Small Animal Surgery

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WINNER OF BEST PICS IN THE ISSUE!

A BIT OF FUN!

C&T NO. 5423

Kerrie Rodgers
– 2013 DE Feline Medicine Participant

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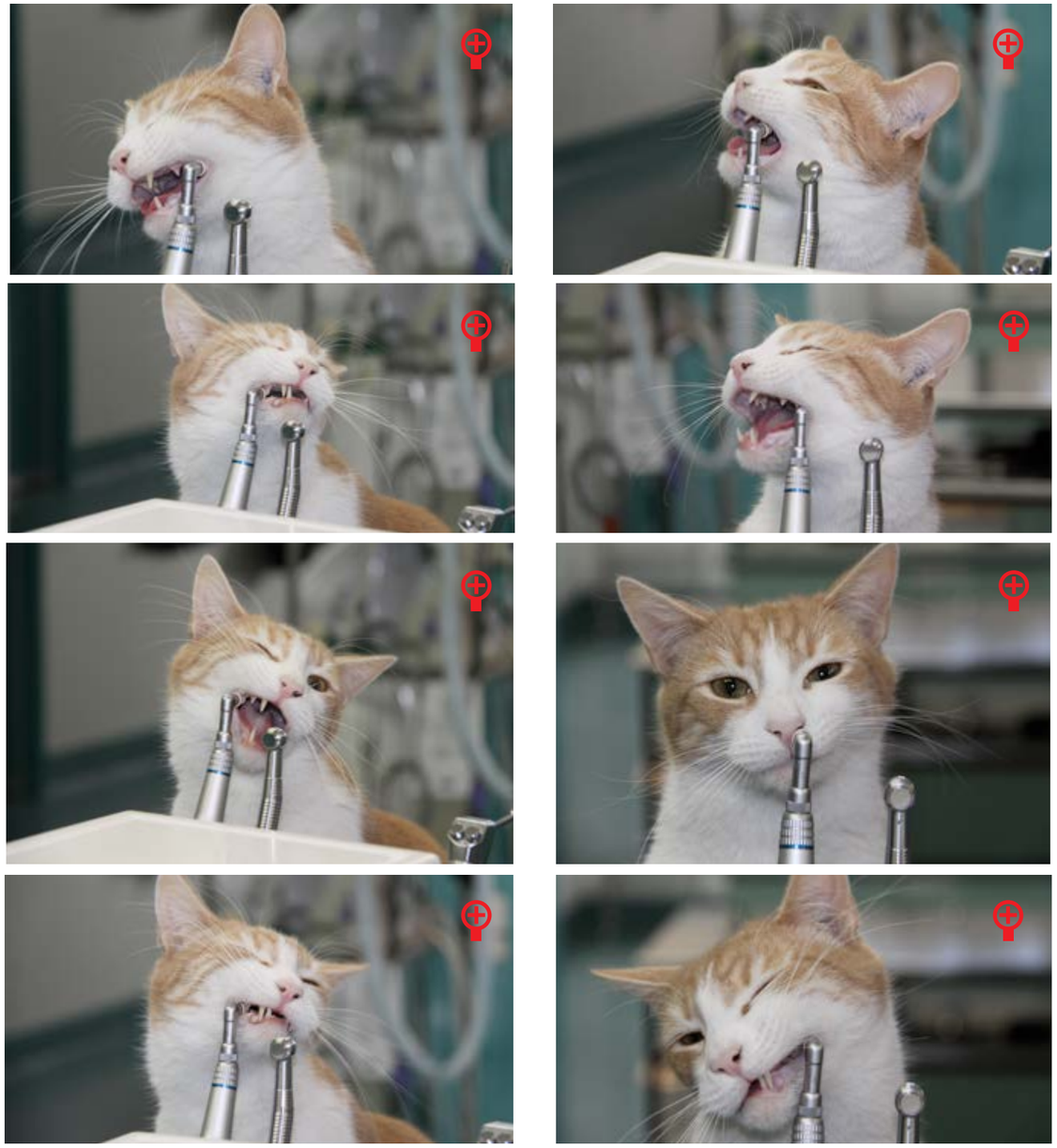
Kerrie shared this montage with her fellow participants as a bit of light hearted relief…

This is 'Frankie' – our beautiful clinic cat – having a go at 'DIY dental'! We are thinking of employing him as he really seems to have the hang of dental scaling.

Kerrie has won a DVD of her choice from www.vetbookshop.com



PERSPECTIVE 108

RADIOGRAPHICALLY COMPLEX LUNG DISEASE – WHAT ARE WE TRYING TO PROVE?

RADIOGRAPHS, CT & SAMPLING THE LUNG

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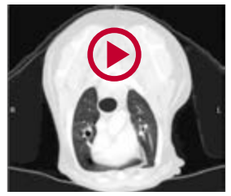
Zoe attended the University of Sydney and obtained a Bachelor of Veterinary Science in 1999. She worked in small animal practice in inner Sydney for 4 years, before moving to Perth, Western Australia.

At Murdoch University, Zoe undertook a residency in Diagnostic Imaging (2003-2006) and became a Fellow of the Australian and New Zealand College of Veterinary Scientists in Radiology in 2007. Zoe is a Director of the Veterinary Imaging Centre and Perth Veterinary Specialists, and an adjunct Senior Lecturer at Murdoch University.

Zoe is also the Tutor for the CVE's Distance Education Abdominal Imaging program

Go to www.cve.edu.au/candtebook to view the following film clip. If you have forgotten your Username and Password, contact cve.membership@sydney.edu.au or call (02) 9351 7979.



Transverse post contrast CT of the thorax, windowed to demonstrate lung and soft tissue, running cranially to caudally ('Right' is displayed on the left). Note severe and extensive lung disease (with mineralisation) on the left, the well inflated 'normal' segments of lung on the right and the ease of detection of the intermittent nodules (right and left).

An 8-year-old castrated neutered Standard Schnauzer dog presented to our practice with a 4 month history of a dry, retching cough. Pyrexia (39.2°C) was present. Radiographs (Figure 1) showed a poorly defined soft tissue mass lesion in the periphery of the left caudal lobe, with a couple of other separate pulmonary nodules throughout other parts of the left cranial lobe. The pattern was certainly mixed and unusual; several radiologists may have interpreted these images differently. One can speculate about whether the tracheobronchial lymph nodes (located dorsal to the carina) were involved, but the answer is not definitive on this study. A neoplastic aetiology seemed more likely; however, other unusual differentials (including inflammatory lung disease with focal consolidation, granuloma formation, eosinophilic bronchopneumonopathy, or several of these processes concurrently) could not be ruled in or out.

Bronchoscopy was performed in order to sample the lung. Some purulent material was noted in the left caudal lobar bronchi but no signs of a foreign body were visualised. Samples showed multinucleated* cells and increased neutrophils, suggestive of an infectious process. Antibiotics (enrofloxicin and amoxcillian-clavulonic acid) were started. Mycoplasma culture was negative. One week later the dog returned with little change in the cough, but normothermic (37.8°C).

A CT scan of the thorax was performed to better characterise the lung lesions. We routinely perform thoracic CT scans under general anaesthesia, with the dog positioned in sternal recumbency; we induce hyperventilation immediately prior to the scan followed by breath-hold during the scan. Intravenous contrast was then administered (600mgI/kg) and a further CT scan obtained. The CT confirmed extensive changes throughout the left caudal lung lobe with coalescing nodules (1-30mm diameter), several of which contained extensive mineralisation. Interstitial infiltrates were present (i.e. the lung tissue between the nodules was considered to be more dense or 'white' than normal. Figures 2-3). The tracheobronchial lymph nodes (located dorsal to the carina) were severely enlarged (up to 35mm diameter), with patchy mineralisation and evidence of necrosis (Figure 4). Separate smaller pulmonary nodules were identified in the right caudal and left cranial lobes. These changes were considered to be highly suggestive of pulmonary neoplasia (e.g. bronchial carcinoma), and ultrasound-guided fine needle aspirates were obtained from several left sided nodules and masses.

The lung aspirates were interpreted to contain severe necrosis with moderate mixed inflammation and mineralisation. The pathologist commented that changes present in some of the epithelial cells in the smears were suggestive of neoplasia; however, too few intact epithelial cells were present to be conclusive, despite the lack of presence of any infectious agents.

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A diagnostic dilemma: the clinical signs and imaging findings are consistent with neoplasia, but this could not be proved with two types of lung sampling (bronchoalveolar lavage, and ultrasound-guided fine needle aspirate). The patient returned one week later for a tru-cut guided biopsy of the left lung. After induction of anaesthesia using propofol and maintenance with isoflurane and oxygen, 4 x14guage tru-cut biopsies were obtained from the left lung, using ultrasound guidance. The biopsy tracts were visualised with ultrasound and only abnormal, non-aerated lung was biopsied. Immediately following the biopsies, a small volume pneumothorax was detected with ultrasound; however, the dog maintained excellent oxygenation. The dog was placed into left lateral recumbency (i.e. abnormal side down, with the aim of trying

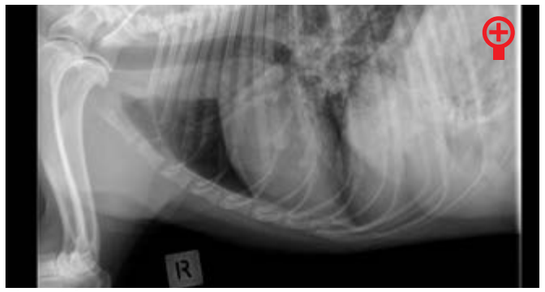

Figure 1.A

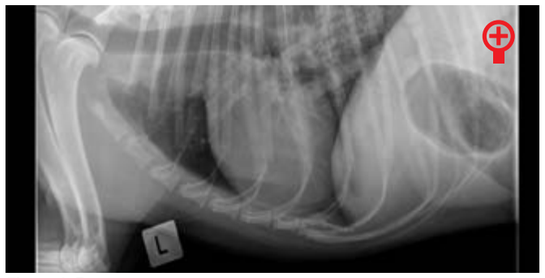

Figure 1. B

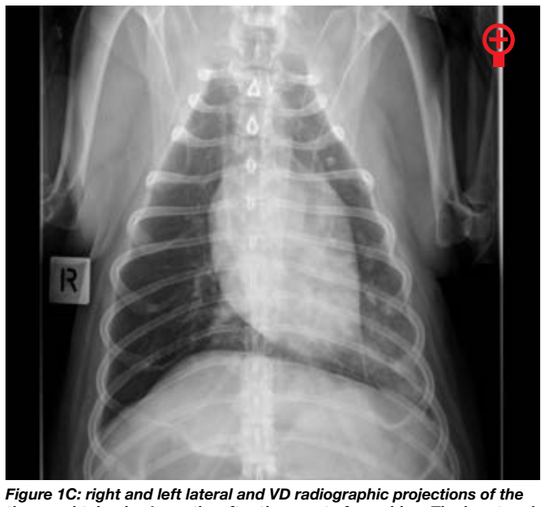

Figure 1C: right and left lateral and VD radiographic projections of the thorax, obtained ~ 4 months after the onset of coughing. The heart and pulmonary vessels are normal. There is a poorly defined increase in soft tissue opacity confined predominantly to the left caudal lobe (so best visualised on the right lateral and VD projections). On the right lateral projection, two discrete nodules are present superimposed over the heart and not easily detected on other projections. The region of the tracheobronchial lymph nodes (asterisks) is poorly defined.

to 'seal' any leaky biopsy tracts). He was allowed to recover from anaesthesia and closely monitored, but showed no clinical signs of pneumothorax. On extubation, a small volume of blood was noted in the lumen of the endotracheal tube.

Histopathology of the lung biopsies was interpreted as squamous cell carcinoma. Given the disease affected multiple lung lobes and had spread to the regional lymph nodes based on the CT findings, the owners elected for palliative therapy alone.

This case is interesting to me, as a radiologist, for several reasons. The dilemma each clinician faces is how to refine a differential diagnosis list and accurately confirm disease, in the fewest of tests. The lung pattern on the initial radiographs (Fig 1 A to C) was poorly defined and mixed, and whilst it is natural to leap to a conclusion of neoplasia in a middle aged dog with a chronic history of coughing, there is no guarantee that neoplastic disease was actually present. The patient progressed to bronchoalveolar lavage, which is an excellent test for sampling the airways. The finding of inflammation is common in respiratory disease (whether neoplastic or not) BUT a purely inflammatory lesion did not match the clinical signs and the dog failed to respond to appropriate treatment.

Thoracic CT is always indicated in cases of complex respiratory disease in small animals – it is a much more sensitive test for the detection of small pulmonary nodules (Alexander et al, 2012), and for differentiating airway and airspace disease. The lack of superimposition of lung and body wall tissues allows a good index of suspicion for the detection of other pulmonary diseases, like pulmonary fibrosis (Johnson et al, 2005) and can be useful for detecting embolic disease (Goggs et al, 2014). The benefits of CT are demonstrated in this case – extensive mineralisation of the both the abnormal lung and tracheobronchial lymph nodes was not visualised on radiographs taken only 1 week earlier. The fact that the tracheobronchial nodes were severely enlarged on CT when equivocal on radiograph further demonstrates the insensitivity of radiographs. Yet, with all of this information pointing towards neoplasia, the guided lung fine needle aspirates were not conclusive.

This emphasizes the limitations of fine needle aspirates for diagnosis of non-exfoliative disease. I can think of many cases in my career where lung aspirates have been inconclusive in proving respiratory neoplasia. The concurrent severe inflammation present in many lung cancers confuses the picture somewhat. Regardless, needle aspirates are a relatively safe, quick and non-invasive test, with very low complication rates; hence, it is easy to argue lung fine needle aspiration has a place. I would prefer lung biopsy any day.

Percutaneous lung biopsy is not performed commonly in small animals in my experience, but lung biopsy is common in people, often via bronchoscopy (Ishida et al, 2012). The risk of pneumothorax, whilst present, should be low if the biopsy sample site is poorly aerated; in our case, contingencies for pneumothorax were in place, but fortunately not needed. Lung biopsy is more common in horses (Venner et al, 2006). In this case, being able to obtain a pathological diagnosis that matched the clinical and imaging findings justified the test to the owners, who wanted to be sure about what they were dealing with. It also reiterates to me that we should interpret all of our pathology tests with the rigour that we apply to any test – if the pathology does not match the clinical and imaging findings, try sampling again. It seems right in this case that histopathology (assessing tissue architecture) was the superior test for making the diagnosis. With

the benefit of the CT assessment, the decision to proceed to ultrasound-guided lung biopsy was straightforward and the risk of complications considered low because we had an accurate indication of the extent of disease.

In conclusion, thoracic CT is indicated in all cases of complex respiratory disease, assuming the patient can tolerate the procedure. General anaesthesia during CT is useful but will increase the risk in severely compromised animals. The choice to perform a minimally invasive lung biopsy (via ultrasound guidance) was justified in this case, given the extent of the CT disease. This case confirms how difficult it can be to accurately obtain a representative sample from the lungs with other means.

Acknowledgements: Jevan Christie – internist; Devon Thompson – radiologist; John Jardine, Jon Meyer, Jason Stayt – pathologists

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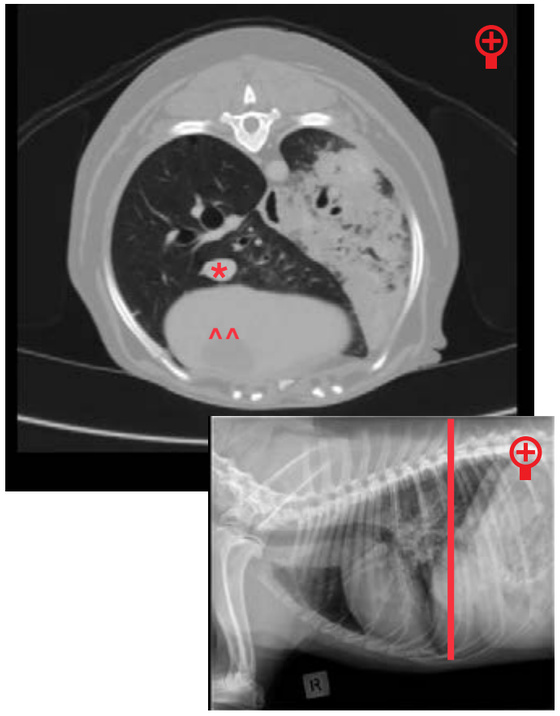


Figure 2: transverse CT image, post IV contrast, in a lung window through the level of the left caudal lung lobe (see the corresponding line on the radiographic image). This study was obtained ~ 1 week after the radiographic image. There is a dense soft tissue mass obliterating the left caudal lung lobe, preserving lung volume. The loss of aeration in the left lung lobe is marked. Other normal anatomical features include the caudal vena cava (asterisk) and part of the liver and gall bladder (^^).

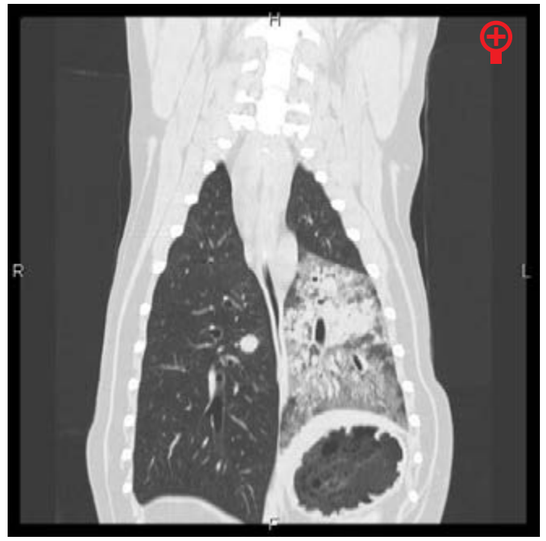
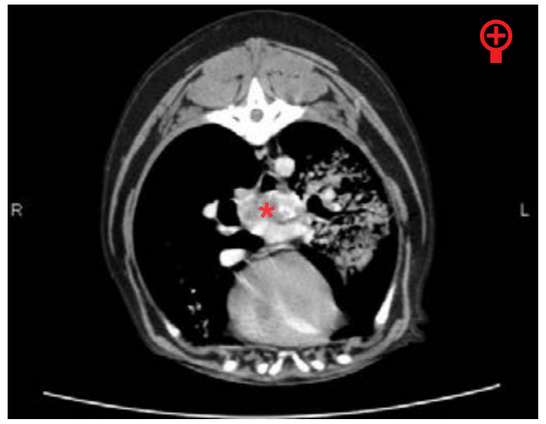


Figure 3 – dorsal CT image through the dorsal third of the thorax, the head is oriented towards the top of the image and the right lung is displayed on the left. The inflated right lung is visible as predominantly normal; however, a discrete small nodule is present within the middle of the lung. The extent of the abnormality in the left lung, with a mix of dense and less dense tissue, is evident.



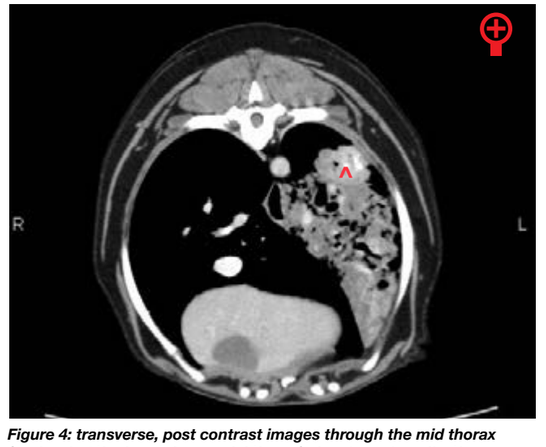


Figure 4: transverse, post contrast images through the mid thorax (a – level of the heart, b – caudal thorax) showing mineralisation and necrosis in the tracheobronchial lymph nodes (asterix) and mineralisation in the lung (arrowhead). The mineralisation was not detected radiographically.

PERSPECTIVE 109

PET INSURANCE – PROBLEMS AND POTENTIAL

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These are solely his personal opinions. He does not accept payment or commissions from any pet insurer.

Figure 1. 'Dexter' with his owner – alive and well – thanks to pet insurance!

INTRODUCTION

Pet insurance offers a better future for our profession, our clients and our patients. Even in 'well off' areas, many pets are treated less than optimally (and in some cases even euthanased) due to restricted owner finances, so effective pet insurance could improve animal welfare. In the United Kingdom and Sweden, pet insurance levels are much higher than Australia. Some Australian veterinary specialists claim that the majority of pets they see are now insured. I don't think there is much debate that owners with insured pets seek veterinary attention more readily than those who are uninsured.

Our profession has exceptionally high levels of altruism. Vets often work in challenging, dangerous or even life threatening conditions – with aggressive animals, on farm outcalls, or with horses potentially infected with Hendra virus (with the development of the vaccine, hopefully the risk of the latter is declining). Vets donate their services treating injured wildlife, performing community volunteer work, or volunteering for overseas welfare organisations. Many vets volunteer for professional organisations such as ANZCVS, AVA, ASAVA and others. This is admirable, but not all industries or workers share this degree of altruism.

-Some vets get agitated that 'Insurers are only in it for the money!'

-NEWS FLASH: Pet insurers are in the market to make a profit.

-There is nothing wrong with that.

-Insurers have to make a profit or the product will fail.

As individuals, insurance workers may well be kind to their grandparents, contribute to charity, or volunteer in the community – but let's accept the obvious truth – insurers ARE primarily in it for the money. Insurers can and do fail. Older readers may remember the catastrophic collapse of FAI / HIH Insurance in 2001 and its costly aftermath. There have to be boundaries to pay outs and not every claim will be accepted.

However, the veterinary profession should not support pet insurance unless it offers good cover, good service and good value for money for pet owners, and is respectful of the value of our veterinary time.

Veterinarians and insurers need to understand and communicate with one another better. Insurers emphasise their low margins. Insurers need to understand that vet science is also a low margin industry and that we work extremely hard to achieve a modest living. Our time is valuable, and cannot be wasted through repeated requests for documents that have already been submitted or completion of overly complex claim forms.

Vets are in a unique position to understand pet insurance, compared to individual owners. We shouldn't have to 'go into battle' for honest clients who have been treated poorly by insurers, bamboozled by scientific or legal jargon. Insurers and some vets frequently ask, **'Why should vets get involved – why not leave claims to the pet owner?'**

Pet insurance becomes our problem if we recommend it, yet forms are 'not received' or owners are denied legitimate claims. The vet profession is much easier to access face to face than an insurance call centre. It is often we who bear the brunt of dissatisfaction and anger when claims are unfairly denied. The vet profession can be the greatest advocate or strongest critic of pet insurance.

Vets have legitimate concerns about the way pet insurance is operating

Sometimes it really does seem as though insurers are looking for any reason to reject claims. One practicing small animal vet from the northern beaches of Sydney has described the stalling and delays of some insurers as 'an orchestrated and deliberate strategy to deny or prolong legitimate claims so that clients just give up'. It is particularly upsetting to owners who have approved treatment in good faith when claims are declined, leaving a significant burden on the family budget.

This is a real problem and has attracted adverse media commentary

Opinion piece from the Sydney Morning Herald April 15, 2013:

'The vet says they have had similar problems with many pet health insurance companies and it takes a huge amount of their time helping clients to claim legitimate refunds. If a ''people'' health insurance company behaved like this they would be closed down.

They effectively rake in hundreds in premiums and pay out next to nothing. I have visions of their office having one staff person who is the call centre, assessor and reviewer for a few hundred thousand clients.

There needs to be a proper exposure of their practices. Not everyone has the time, skills and determination to follow through to the ombudsman. Basically these companies are stealing money from unsuspecting pet lovers.'

http://www.smh.com.au/comment/no-dog-in-the-manger-complaint-20130414-2htnt.html#ixzz2V2ySLbl7

http://www.smh.com.au/comment/no-dog-in-the-manger-complaint-20130414-2htnt.html?rand=1366001094802

I do not endorse some of these wilder assertions, but note them as an example of adverse media commentary on pet insurance. Most pet insurance works very well, and insurers usually do not refuse legitimate claims. However, I have certainly seen legitimate claims denied.

THE PROBLEMS – AND SOME POSSIBLE SOLUTIONS

1. Legitimate claims rejected due to blanket exclusion of so-called 'pre-existing' conditions as opposed to true congenital, pre-existing or chronic disease

Problem: This is an area of on-going concern to pet owners and vets. AVA is currently working to address the issue of 'pre-existing conditions', which are not tightly defined and are not analogous to true congenital or chronic disease.

No reasonable person would object to an insurer refusing to cover true congenital, pre-existing or chronic disease that was identified prior to insurance being taken out. Examples include:

- A puppy or kitten with congenital patent ductus arteriosus requiring cardiac stent placement or ligation.
- A puppy with severe hip dysplasia that will ultimately require total hip replacement surgery.
- An adult dog with a cruciate ligament rupture that had occurred prior to insurance, requiring cruciate orthopaedic surgery on the affected or the opposite limb.
- An adult dog with end stage otitis externa requiring total ear canal ablation surgery.

Yet sometimes insurers broadly exclude whole body systems, based on a single trivial episode, years before, completely unrelated to later illness. This is deceptively categorised as a 'pre-existing condition'. Just because an animal has had a condition of a particular body system, it does not make that animal 'pre-disposed' to all conditions of that body system.

A real life example – Surgical removal of a skin lump from a dog

A claim was refused, arguing that a skin lump was a pre-existing skin condition, as the dog had one episode of mild superficial pyoderma years before. What absurd nonsense! If a human has an episode of tinea as a teenager, then years later has a melanoma excision from their face, the melanoma is not a pre-existing condition.

Strategy: The veterinary profession should not promote pet insurance unless exclusions are identified when insurance commences (rather than when declining claims, possibly after years of cover). A cooling off period should apply within which owners can withdraw from pet insurance, commencing when they are informed which exclusions, if any, are to be applied.

2. Cease the policy of insurers declining claims when there is 'no definitive diagnosis'

A fatuous reason for rejection of claims is that 'no definitive diagnosis' occurred. Many conditions are treated presumptively or symptomatically, such as **vomiting**. Often no definitive diagnosis is made, yet with simple symptomatic treatment, complete recovery occurs.

These rejections are unconscionable.

A real life example: Surgical removal of a skin lump - case 2

A claim for excision of a skin lump was refused, as there was no conclusive diagnosis. This is just plain silly, and potentially more costly to insurers, as it may promote extra submissions to pathology for lumps excised with wide margins or considered low risk (yet that still need excision due to their site, such as a meibonium gland adenoma of the eyelid, which though benign may injure the cornea if further growth occurs). In the case mentioned, I still had the mass in formalin and rang the insurer to advise them that as they wanted a definitive diagnosis I would send it off for histopathology and they would pay.

Granulomatous meningoencephalitis (GME) is often diagnosed presumptively and by exclusion. In some cases GME can often only be diagnosed definitively on post mortem. By that logic insurers may be suggesting that they would prefer to pay for a post mortem than for effective therapy. To deny claims based on a lack of definitive diagnosis is ridiculous and could lead to overservicing in pathology submissions and needlessly escalating costs to owners and indeed insurers themselves.

Solution: Insurers must stop denying claims when there is 'no definitive diagnosis'.

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3. Document security and document loss

Some insurers claim that forms posted 'never arrived'. Whilst forms may be genuinely misplaced or never sent by owners, I can attest from personal experience that this has happened with several claims I have posted registered mail. Is this deliberate stalling, or are some insurers unable to organise an office?

Preparing this article, I was beginning to wonder if my concerns were out of proportion or overstated, when a typical case occurred. The owner has agreed to allow me to share her story.

A real life example: 'Wilma' B, a desexed female Rhodesian Ridgeback

In December 2013, the dog presented suffering haematuria and pollakiuria. Basic in-house urine testing confirmed a urinary tract infection, which was successfully treated with antibiotics. All insurance documents were posted via registered mail on January 8th, 2014 and delivered to the insurer on January 9th. It may be searched here: http://auspost.com.au/track/ (Tracking number 5090394744016).

On February 14th, at the request of the owner we made enquiries. We were told that no documents had been received and it was requested that we resubmit them. We declined, and gave them the tracking number. We advised we would not be resubmitting, but would direct the owner to the Financial Industry Ombudsman. Within 2 hours the insurer phoned back, having located the documents. They claimed that their temporary loss was due to an error by their external scanning contractors, and that the claim should be finalised within 5-7 days. (It wasn't).

I have personally had several examples when 'forms that were never received' were suddenly found when a registered mail tracking number was provided. Is it any wonder that vets and owners get exasperated?

Solution: Avoid loss of completed forms by owners or insurers by completing and sending them yourself via registered mail. It also improves the chances of the client getting paid quickly. **Ensure ALL paperwork is completed and signed**. The best option is registered mail, which is more expensive than regular post. **Record the mail tracking number** on the patient record for future reference.

Probably best are e-submissions via email. Scan forms and put the pet insurance or claim number in the email subject line. Hopefully all insurers will accept e-submissions in the future. This could reduce a concern of many vets – insurers requesting a complete history be given to pet owners, who then spend hours on line 'researching' every aspect of the patient history online, getting confused and agitated about scientific terms, possible diagnoses, treatments and miracle cures. (Petplan will accept e-claims, Petsure / Hollard are working on it).

The **cruciate, patella and hip examination form**, which has to be completed prior to insurance by some insurers, and without which a claim cannot be made for orthopaedic surgeries involving those regions, is particularly important. **Also make sure you make a complete medical record for all new patients describing stifle, patella and hip examination.** It is important when it comes to Orthopaedic / Cruciate / Hip examination that the status of the relevant joints is recorded in your medical records at initial examination, especially if pathology free, so that insurers do not say that a condition was pre-existing.

Long-term solution: AVA and insurers are working towards e-submissions across all insurance products, which should reduce document loss and improve processing times.

4. Clearer communication between the veterinary profession and pet insurers

At a personal level from insurance staff to veterinarians: Some insurance call centre staff are almost impossible to communicate with, with poor English, let alone an understanding of veterinary scientific terminology. This needs to change.

At a company to profession level: A major 'blow out' in claim times occurred during the transition to a new computer software program in late 2012 by a major insurer, which caused great dissatisfaction amongst clients. The profession was only made aware of this after the problem had been resolved, when the AVA engaged with insurers in late 2013. With better communication much of the irritation could have been defused. Instead, widespread frustration developed and the value of pet insurance itself was questioned. On a positive note this episode did lead to better lines of communication between vets and insurers.

5. Clearer communication between pet insurers and their clients

Clearer communication would help insurers too. Despite noteworthy attempts to promote pet insurance by regular practitioners and through the media by vets such as Dr Kersti Seksel on ABC 702 Sydney radio, adverse feedback on pet insurance in the same show damaged confidence. In early 2014 a caller to Kersti's show said (paraphrased) 'We phoned the insurance company prior to the procedure, they assured us the surgery would be covered, but when we went to claim, it was denied.' This kind of media commentary **destroys public confidence in pet insurance.**

An insurance staff member revealed that this certainly happens occasionally. They also revealed that **all insurance telephone calls are recorded and logged** and in the event of a dispute, the calls can be reviewed. If an insurer has given a verbal 'go ahead', this must be honoured, and clients are legally able to request a review of such conversations.

6. Promote better veterinary technical expertise amongst insurers

Individuals with limited veterinary knowledge are refusing claims on dubious grounds. Whilst no one would expect insurance assessors to 'know it all' they should have better access to veterinary resources and knowledge.

There is a marked absence of veterinary technical expertise in some insurance companies. A respected veterinary specialist told me that an insurance claim for one of his patients was rejected because the insurance assessor could not comprehend the distinction between a differential diagnosis and a diagnosis! The assessor thought that one of the conditions listed as a differential was the diagnosis. The specialist argued the point at which point the insurer said, 'Well that is what we think the animal has and we're rejecting the claim'. Reassuringly, some insurers do employ significant numbers of veterinarians.

Solution:

a. Promote better veterinary expertise within the pet insurance industry by increased employment of Australian registered veterinarians and experienced veterinary nurses.
b. The veterinary profession should engage with insurers to assist, when needed, in provision of technical expertise or referral for expert or specialist advice.
c. Veterinary assessment of an insurance decision should be completed, if requested, by a registered Australian veterinarian, familiar with Australian diseases and clinical practice. That vet should have the conviction to stand by their decision, in other words put their name and registration number to an assessment, and be prepared to discuss, review or defend that decision.

7. Agreed processing times

Prolonged delays for insurance payouts stress family budgets, and cause aggravation.

Solution: Insurance claims should be determined within 30 days of receipt of all relevant documentation. If requested, a vet review by a registered Australian vet or, ideally, a vet with further qualifications e.g. MANZCVS or FANZCVS should be completed within 21 days of receipt of any additional required documentation.

8 Remove misleading information from claim forms

The implication that claims must be submitted within 60 or 90 days or they will not be processed is wrong. There are legitimate reasons why starting a claim may be delayed, such as a serious family illness or crisis. Under Australian law claims must be processed, even if submitted years later. (It is desirable, however, for owners and vets to make claims promptly, to avoid confusion or document loss associated with the passing of time).

WHY BOTHER WITH INSURANCE?

When pet insurance works well – and it usually does – it is a blessing. Pet insurance is growing fast and there is significant future potential. Veterinarians who have practiced in the UK know how widespread and effective pet insurance is there.

Case study – a successful insurance claim – happy client, happy pet, happy vet

'Dexter', a 9-year-old Groodle dog has been treated over the past 2 years for intractable epilepsy by a general and specialist veterinary practice. His owners have claimed over $10,000 for vet treatment, which has been paid promptly. Dexter, who is a lovely dog, would not be alive without pet insurance, and is stable and doing well.

Building pet insurance in your practice

Insurers have some highly educational staff training seminars on pet insurance. By training and familiarising your staff with pet insurance, many potential problems can be reduced or eliminated.

Vets should not offer advice in advance of treatment as to whether they believe an illness is covered by insurance or not.

A non-confrontational way of raising pet insurance with pet owners is for vets to ask 'Do you need any insurance paperwork signed?' Some insurers also provide convenient stickers to paste onto invoices saying 'this would have only cost $X with pet insurance'. These support receptionists who have to deal with clients who are polite to vets but moan about costs to 'defenceless' reception staff.

WHEN THINGS GO WRONG – THE CONSUMER'S DEFENCE – THE FINANCIAL INDUSTRY OMBUDSMAN SERVICE

Clients who have unresolved problems with legitimate claims can lodge a dispute with the Financial Ombudsman Service (FOS) formerly Banking and Insurance Industry Ombudsman - See www.fos.org.au. The FOS is the appropriate forum to consider disputes between consumers and their pet insurance provider and is a not-for-profit organisation that offers a free and independent external dispute resolution service. The Financial Ombudsman Service (FOS) works with consumers and member financial services providers to help resolve disputes that fall within their Terms of Reference, if they have been unable to resolve the complaint through internal dispute resolution processes. As an impartial organisation, FOS does not provide any general, financial or legal advice. FOS may be contacted on 1300 78 08 but using the internet is far easier: http://www.fos.org.au

FOS releases comparative tables that present dispute statistics. Pet insurance disputes are classified under Personal and Domestic property but are now noted as a separate category at the request of AVA and insurers.

Comparison tables can be accessed via the following link: http://www.fos.org.au/publications/comparative-tables/

If a claim is refused and should have been paid according to the terms of the Product Disclosure Statement, and providing you've submitted all the information required, owners should not waste time battling through insurance call centres.

Don't argue the point if insurers are talking nonsense. Just let the FOS do the hard work.

FRAUD AND ETHICS – A GENUINE CONCERN

We can be proud of the high ethical standards of our profession. The level of public trust in us is well established – surveys repeatedly find that veterinarians are amongst the most respected professions. It is imperative that vets maintain this 'high moral ground' and always submit honest and accurate records. In NSW at least, under the new Veterinary Practice Regulation 2013, the Veterinary Practitioners Code of Professional Conduct states*: If a record of a consultation, procedure or treatment is altered, the alteration must be clearly identified in the record.*

Insurers have mixed views on fraud. One of the two major insurers the AVA has met with felt that the problem was minimal; the other thought it was highly significant. Manipulating a record to suggest that a condition or disease was not present at an earlier time may constitute fraudulent behavior. Insurers have sophisticated computer programs that can help identify fraud. Don't be tempted to consider this kind of practice as a 'favour to struggling pet owners'. Fraud is not just unethical, it is criminal.

Maintain absolute impartiality in recommendation of pet insurance by vets

To avoid any perception of bias do not accept commissions or any direct or indirect monetary benefit from any insurer.

FUTURE DIRECTIONS

Pet insurance should be easier to understand, and less vague and open to speedy interpretation.

The vet profession should continue to engage with insurers to make the system work better and **tackle problem areas** to the benefit of all (for example in vet and client education, and by reducing document loss in transfer to insurers). Vets should not be afraid to question obvious poor service or support a client who has been blatantly wronged.

Veterinary bodies should seek feedback from our profession before they consider a blanket recommendation to advocate or promote pet insurance. Otherwise, we risk damaging our professional credibility by associating ourselves with an industry (banking and finance) that consistently finds itself in poor favour with our clients. Our energy may be best spent educating vets and clients about pet insurance.

Disputes and a Veterinary review panel / advisory board
We need to be very careful regarding any suggestion to establish a **'veterinary review committee'** for insurance disputes, not that one has been one suggested to date. Whilst it is likely vets and specialists would be happy on occasions to offer an expert opinion, it would be convenient for insurers to refer disputes to a volunteer vet committee, made up of unpaid vets doing insurance work for free. On the other hand, a neutral independent review panel of paid vets funded by insurers might benefit all stakeholders. Suitable panellists could include experienced practitioners including those who are injured, retired, semi-retired, or on maternity leave (yet able to work part-time in a non-clinical situation or from home). The potential for the panel to become an insurance dispute dumping ground[1] make it important that there is a financial penalty reducing spurious disputes, such as a fee paid to the Australian Companion Animal Health Fund for research.

Alternatives to an external vet review committee could include:

a) An independent veterinary review board affiliated with the Financial Ombudsman Service to assist in evaluation of some disputed claims. By building confidence in the integrity of pet insurance the vet profession would be able to recommend it with greater Confidence.
b) Representatives of the veterinary profession liaising with the FOS when required and, if necessary, suggesting an independent expert veterinarian in the field relevant to the dispute. The insurer or FOS should remunerate vets for their time.

The profession needs an avenue for vet feedback to insurers about experiences with pet insurance
Initially, at least, we should seek feedback from veterinarians. The AVA's February 2014 member survey was an excellent starting point. Perhaps further market research is needed to allow us to better understand experiences with pet insurance from both our profession and our clients. An emailable survey link and pet owners' competition sent to our client bases would likely provide an excellent response and valuable data.

Establishment of an HICAPS style payment system for pet insurance. This would allow Medicare type instant payments at point of service, relieving the financial burden on pet owners. In conclusion, pet insurance offers some problems and great potential – if insurers engage with vets we can solve many problems and make insurance work better for all – and, most importantly, for patients like 'Dexter'.

FEEDBACK REQUESTED PLEASE
Any other ideas folks? Please feel free to join the conversation via *Control and Therapy* or at AVA Forums under Small Animals, Experiences with Pet Insurance.

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