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Authors’ views are not necessarily those of the CVE.

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- 23 Oct Medical Management of Uveitis & Glaucoma
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DE FEEDBACK
This year I’m doing my first DE course, Sonorology. Not only am I learning how to use the ultrasound properly, but also I am reviewing and consolidating internal medicine. I believe it is adding me to 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 done, 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.

MULTIPARTITE NAVICULAR BONES OF A RACEHORSE

C&T NO. 5406
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Introduction
Bipartite and tripartite sesamoid bones have been described in the horse previously. This case study records a racehorse with concurrent quadrupartite, 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 provocatory 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 navicular bone with three of four pieces similar in size and a fourth entity with a transverse long axis. Each DSB possesses two surfaces, one articular and the other a tendon surface. Each DSB articulates with the second and third phalanges and the right hind limb. 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 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.

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 unencumbered 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.’

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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THANK YOU TO ALL CONTRIBUTORS
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.

Dissection of the right hind foot exposed 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.

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 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.

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 (polyarthritits). 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 polyarthritis (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 carrier. 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 struck, 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 inudder 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 Biosafety 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.

**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 bipartite 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 suci may pressure an intact DSB to divide it into pieces.

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 Biosafety 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.
Johne’s disease. Spread for CAE and mycoplasmosis and is also important for the feeding of pooled goats’ milk is the main method of infection. In one approach of never feeding bulk goats’ milk to kids. This case study also supports the Australian Goat Industry Association but is aimed at meeting traceability and food safety e.g. 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 but is aimed at meat traceability and food safety e.g. from agricultural and veterinary chemical residues (http://www.mla.com.au/Meat-safety-and-traceability/Livestock-Production-Assurance/vendor-declarations).

References

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KILLING SERRATED TUSSOCK WITH GRANULAR FLUROPANATE HERBICIDE

CAT NO. 5408
Peter Davies

A healthy dairy goat.

Jennie pictured here with her pet goat, “Norman”.

Sadly enough, this case study presented by Dr Baverstock 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 unwatering 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 veterinarian 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., Septicoccus agalactiae, Staphylococcus aureus, Bovine Viral Daemona Virus (BVDV), vibrios, and trichomacizes, 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 in line 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 Veterinarians (formerly RLPR and LHAP). LLS District Veterinarians are a valuable resource for locating relevant, up-to-date, independent advice and information on biosecurity and animal health issues to land holders and veterinarians (http://www.livestock.health.nsw.gov.au/livestock-livestock/advice). I often tell my clients when they think I am being a nagger about biosecurity and quarantine that they can pay me now or pay me later.

3. Once a Tussock plants dies, you need to replace the bare ground that this creates in order to prevent a natural 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 other infections of mycropastoral 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 mechanised spot spray methods, and even spot spraying with a backpack is not practical or safe.

I have calculated that these spot spray 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 infestations in 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 flupropionate 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 flupropionate, 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 eradicate 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.

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, 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 Flupropionate 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 few still several challenging problems with this type of herbicide application.

1. Seed still blows in from neighbouring properties, especially where 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 prevent 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 other infections of mycropastoral 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 mechanised spot spray methods, and even spot spraying with a backpack is not practical or safe.
Sure enough, come January 2014, whilst chasing some time frames. I would die, but it takes a little longer than normal spot spraying arranged the application, and they assured me that the plants one! I spoke with the team at the local rural supplies store who agitated, and wondered if this high cost investment was a wise decision, to enable the poison to be taken up by the plant) and not traditional sprays. As soon as the herbicide had been applied, 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 pointed out to me 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.

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 spraying time and energy into creating better, more diverse 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 treated 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.

Tableted 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 infestations, 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 1. The helicopter rig used to distribute the granular flupropanates.

Figure 2. View from the chopper.

Figure 3. Microclena 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 also be 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.
ANISOCORIA & NYSTAGMUS IN A CAT

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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

‘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 “he’s 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.

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
Fly/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 thoracolumbar 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 4.37 kg. He was eating very little for his owner.

I started Andy on Clindamycin 25 mg BID and Thiama 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: 102.2

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 tuns (for human consumption), I gave the owner a can of ‘Dine Desire Virgin Tun’s’ 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.

**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 + Thiama 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/lower 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 thought.

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.

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**CLOPIDOGREL (PLAVIX®)**

**C&T NO. 5410**

**Dr James M. Euclis BVSc**

Cattlovers Veterinary Clinic 18 Overport Road, Frankston VIC 3199 www.vetbook.org E. seacontainers@hotmail.com

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 thromboembolism and it is used as a prophylactic, particularly at-risk patients with Grade II/IV or greater murmurs.

When given to cats at the recommended dose of 10 - 20 mg/ kg/1, 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, benzazepir or antipine.

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.

References


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**DERMATOLOGY**

**A DE ASSIGNMENT CASE REPORT**

Nundah Village Veterinary Clinic 1514 Sandgate Rd Nundah QLD 4012 T. (07) 3266 2000 E. kmcc@vetmail.info

C&T NO. 5411

Karen McCormick
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 (F3L), 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 ischiatric tuberosity. The radiographs were examined by a surgical specialist, who recommended 4 weeks of cage confinement rather than surgical intervention. Pain relief involved 0.05 mg of Buprenorphine subcutaneously, and placement of a 25 mcg 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 ischiatric 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 Urocitriasis
- Autimmune disease e.g. Ocular/epithelial Urocitriasis

**Treatment**

Truffle began a course of Amoxycillin/Clavulanic Acid (75mg orally every 12 hours). He was also given an anti-histamine 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 continued. His pyrexia resolved and his appetite and demeanour remained normal.

Hydrocortisone 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 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 that stage was 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 informed 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 maloccluded 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
- Perifollicular fibrosis
- Mild perifollicular infiltrate
- Fibroplasia along the dermo-epidermal junction, with or without perifollicular infiltrate
- A moderate dense infiltrate of inflammatory cells in the subcutis
- Pseudofolliculitis

Gross findings are consistent with this case, including: a glistering appearance to the skin, lack of pain on palpation of the surrounding area, and ephelion of hair before deeper ulcerated wounds become apparent (1, 6). 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 (SIB2/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.

Bibliography


Secondary References


"Otis", an 8-year-old MSH 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 non-reflexive. 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 biventricular 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 euthanised.

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

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

C&T NO. 5412

Louise Beveridge

BVMS MRCVS
CVE/ISFM Feline Medicine DE participant 2014

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CVE Control & Therapy Series – Issue 276 September 2014
CALL FOR CASES – WE NEED YOUR HELP!

FLEXOR TENDON CONTRACTURE

CAT NO. 5413

Leonie Thom

Wilton 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 contractures of the distal forelimbs (A). A close-up is provided in (B).

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.

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 owner, 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.svmrx.co.uk which reformulate a number of tablets into cat doses i.e. very small tablets: Metronidazole, amiodarone, gabapentin, tramadol, clonidine. 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

Nicolee 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.

Tips for young veterinarians...

From the DE Files

The CVE is partnered with the International Society for Feline Medicine (ISFM) for our feline medicine distance education program

Handy Hints from the ISFM Forum

Feline-Medicine@MailTalk.ac.Uk

FROM THE DE FILES

We need your help! A syndrome involving flexor tendon contracture of the forelimbs is being investigated by Leonie Thom at Wilston Vet. Please share your experiences with this condition if you have seen it in your practice.

Vale

Rosemary Janet Bryden
2 May 1941 – 30 June 2014

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

Read more...
Transdermal versus Topical

- TD – therapeutic drug concentration in systemic circulation
- Topical – local therapeutic drug concentration in surface organs (skin, eyes, ears, 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 T.D than orally
- Possible longer duration of action without peak side effects – “drug depot in skin”
- 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 bioconversion 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 – pinnas, axils, inguinal
- Some cats may resent the administration
- Increases prescription cost
- Some drugs may irritate or cause burning

Properties of good TD drugs

- Relatively high lipid solubility – need to transverse waxy stratum corneum
- Low melting point – readily converted from a solid to a liquid at body temperatures
- Less polar compounds – very polar compounds
- Small compounds – molecular weights <500 daltons
- Achieve high local drug concentrations

Permeation enhancers

MOAs of permeation enhancers

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

1. PLO
- Pluronic F127 lecithin organogel
- Permeation enhancer
- High molecular weight surfactant that forms drug micelles
- Leachtin – from eggs or soybeans
- Increase the fluidity of the stratum corneum
- Leads to exfoliation of the stratum corneum and low grade inflammation with chronic use which enhances drug penetration
- 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
- Pain
- 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 lipopholic 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
- Glycerol ethers

Many are irritating so combinations of enhancers at lower doses have been used to decrease irritation that occurs with higher doses

Other Ways to Deliver Transdermal Medications:

Formulation of oral drugs into ‘duplex pro-drugs’

- 2 molecules of the same drug linked by an ester bond
- More lipohilic
- Better absorbed across stratum corneum
- Than cleaved locally by esterases in the skin
- E.g. nalbuphine (used to treat alcohol and opiate dependency in humans)

Transdermal Patches

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

- Reservoir type
- 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
- EMLA cream – 10s pretreatment with ULF speeds onset of action in humans from 60 mins to 5 mins
- EMLA cream is safe in cats
- Requires 1hr delay after application – need a study on sonophoresis for this

2. Microneedles
- EMLA cream is safe in cats
- Some cats may resent the administration
- Do not refrigerate
- Do not ship in cold temperatures

3. Iontophoresis, electroporation
- Use electric field to enhance skin penetration
- ‘Poke with patch’ – tiny arrays of microneedles in drug-imregnated patch
- ‘Coat and poke’ – needles surface coated with drug

Microemulsions
- ‘Drug depot in skin’
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Many are irritating so combinations of enhancers at lower doses have been used to decrease irritation that occurs with higher doses

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

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/cm²
- Patch size limited to 50cm²

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 unpalatable 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 – 0.05ml is the usual preferred volume; in cats on multiple drugs, practical volumes per drug are 0.025 – 0.05ml. A total volume of 0.3ml is too much.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Bioavailability</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerin</td>
<td></td>
<td>227mg/mol ointment Used TD for decades in veterinary patients to reduce preload in cardiac failure (44-45) - causes local vasoconstriction, increases local blood flow Used in humans to treat neuropathic pain after thoracotomy (46)</td>
</tr>
<tr>
<td>Rufaxidine</td>
<td>&lt;10% compared to oral when given as a single dose (47-49)</td>
<td>But gave 10x oral dose gut plasma concentrations equivalent to oral (46-47) Repeated topical ointment caused dermatitis</td>
</tr>
<tr>
<td>Diliazem</td>
<td>&lt;10% compared to oral when given as a single dose (47-49)</td>
<td></td>
</tr>
<tr>
<td>Desmethylthoxine</td>
<td>&lt;10% compared to oral when given as a single dose (47-49)</td>
<td></td>
</tr>
<tr>
<td>Buprenovine</td>
<td>&lt;10% compared to oral when given as a single dose (47-49)</td>
<td></td>
</tr>
<tr>
<td>Amntrifylline</td>
<td>&lt;10% compared to oral when given as a single dose (47-49)</td>
<td></td>
</tr>
<tr>
<td>Methimazole</td>
<td>Multiple doses in PLO 4(34-38) 336g/mol patch Effective in post-operative pain management in dogs and cats</td>
<td></td>
</tr>
<tr>
<td>Gabapentin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amlodipine</td>
<td>0.625mg q24hrs in lipoderm 2(42)</td>
<td></td>
</tr>
<tr>
<td>Amitryptiline</td>
<td>&lt;10% compared to oral when given as a single dose (47-49)</td>
<td></td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>&lt;10% compared to oral when given as a single dose (47-49)</td>
<td></td>
</tr>
<tr>
<td>In PLO - 'poor absorption – not reliable' (50)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pregabalin</td>
<td>No cat studies yet?</td>
<td></td>
</tr>
<tr>
<td>TD cream</td>
<td>114mg/mol More recent studies have been published</td>
<td></td>
</tr>
</tbody>
</table>

**Insecticides and Acaricides**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fipronil</td>
<td>Spot-on Distribute to hair, stratum corneum, sebaceous glands but not to systemic circulation (44-45)</td>
</tr>
<tr>
<td>Imidaclopridine</td>
<td>Spot-on Distribute to hair, stratum corneum, sebaceous glands but not to systemic circulation (44-45)</td>
</tr>
<tr>
<td>Selamectin</td>
<td>Higher bioavailability in cats (74%) than dogs (74%) - thinner skin, grooming ingestion (44-45)</td>
</tr>
<tr>
<td>In PLO - negligible absorption after single dose (47-49)</td>
<td></td>
</tr>
<tr>
<td>Oxadiazonide</td>
<td>Human studies, none in cats? TD cream</td>
</tr>
<tr>
<td>Prederaline</td>
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**C&I NO. 5416**

Sheryl van Nuenen¹ & Paul Canfield²

**ON BEHALF OF TICK-INDUCED ALLERGIES RESEARCH AND AWARENESS (TIARA)**

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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**Introduction**
Veterinarians are in an ideal position to practice ‘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 *bodes holoculus* is rare in people**
Veterinary practitioners would be aware that *bodes holoculus*, commonly known as the Australian paralysis tick due to the effect of *its* holocylotoxin 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 holocylotoxin; although rarely, 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 *Finders 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 saliva 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 saliva proteins are associated with galactosylated myelophages, and alphagal has been found in tick gut in *bodes ricinus* (Horstet 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
The knowledge gathered from known tick anaphylaxis patients has led us to develop some guidelines regarding tick removal in humans known to be allergic to ticks:

**GUIDELINES**

- **DO NOT SCRATCH ANYTHING YOU CANT 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**

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.tara.org.au. Tick-induced Allergies Research and Awareness (TARRA) 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.medient.usyd.edu.au
- www.allergyfacts.org.au

Reference


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!

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 tickidal permenin is currently advised

**SNAILBAIT TOXICOSIS**

**C&T NO. 5417**

Tristan Robinson

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 is it not if there’s access to the packet!) I thought it may benefit publication.

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 out 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 60g0 packet contains 60g/kg of iron EDTA, which calculates as 36g of iron EDTA 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 50kg Alaskan Malamute was bright, alert and responsive and clinically well on physical examination. The main abnormal findings were tancy mucous membranes, moderately diated pupils with poor pupillary light reflexes, and mildly panting on abdominal palpation. There was evidence of red staining around the anus and 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 iron.

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.

**Urine**: 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 anaphylaxis pain relief 0.3mg i/d (0.01mg/kg) pethidine (Tempgesic®) 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 atactic 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 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

**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 10xusual 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.

Gastrogyl, 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 aide 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

**WINNER!**

**T. 02 6926 0900**

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**SNAILBAIT TOXICOSIS**
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 sulorbate 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:

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The use of iron EDTA (also known by the International Nonproprietary Name [INN] as sodium ferrousdate) as a small 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 by researcher Colin Young. The product was specifically developed 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. Positive 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 diarrhea 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 4.77mg. A 30kg dog consuming a whole pack therefore has a maximum exposure of 156mg iron/kg bodyweight. It should be recognised that Multiguard contains more than 50% wheat flour which may also contribute to toxicity. In addition, the product contains dextranum benzate. While it is highly likely that use of iron EDTA reduces dog intoxication by other mollicutes, there are still a number of important research questions, previously emphasised by Haldane and Davis (2003). There must be a very clinically relevant and important DVN, 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 dextranum benzate 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.

References


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.

CASE MANAGEMENT

A female juvenile cat found abandoned on a rural property was presented with a presumed littermate to a veterinary clinic for respiratory signs. 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 thoracic 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 visera), no treatment was instituted prior to the immediate pre-anaesthetic period.

Eight days after initial x-rays the kitten was scheduled for surgery. Methadone 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 (0.3mg/ kg combined with ketamine 6mg/kg given intramuscularly). Induction was completed with delivery of isoflurane (2%) in

Figure 1: Dorsal-ventral thoracic radiograph diaphragmatic hernia in a 1.2kg kitten. Note poorly defined diaphragmatic border and gas filled intestinal loops in chest.
oxygen via mask. The patient was then intubated with a size 1.2kg kitten. Note indistinct line of diaphragm, obscured cardiac silhouette and pneumothorax (Fig.3).

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.

Figure 3: Dorsal-ventral thoracic radiograph showing post-operative pneumothorax. Note marked collapse of pulmonary parenchyma. Diaphragmatic border is no longer visible.

Figure 4: Dorsal-ventral thoracic radiograph showing reduction of post-operative pneumothorax.

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 was used to appose the wound edges in a simple interrupted suture pattern. The final three sutures were all preplaced before ligature and care was taken to ensure adequate diameter remained for the passage of the caudal vena cava through the caval foramen. Close communication between the 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 pleural lungs as the herniorrhaphy was completed. A 23 gauge intravenous catheter 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 tongue was detected in the oral mucous membrane 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, 3-way tap® and 10mL syringe. Approximately 500mL 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. A repeat thoracic radiograph one hour after discovery provided evidence 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 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 is in her new home.

DISCUSSION

Presentation and Pathophysiology

Acquired or traumatic diaphragmatic hernia (TDH) results from rupture of the thin muscular diaphragm and associated injuries.2,26 These may be the result of either blunt abdominal trauma or penetrating thoracic trauma.2,26 Contributing factors for TDH 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 (fractures, pulmonary contusions), as well as the systemic effects of pain and hypovolemic shock. TDH associated respiratory compromise ultimately results in hypovolaemia, 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 hypovolemic shock are not unusual. Gibos et al (2000) reported a substantial 71.4% incidence of concurrent injuries. Given the severity of the injury and the large size of the hernia, it is reasonable to assume the lungs were severely compromised.

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.4,17,22,23 Thus radiography is always indicated to confirm the diagnosis of TDH, and in addition is helpful to ascertain the presence of concurrent injuries.2,26

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.2,26

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.2,26 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 hypoxaemia unresponsive to fluid resuscitation, or unrelenting abdominal pain.2,26

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...
Anesthesia and Patient Preparation

Attention to anaesthetic technique and patient preparation is essential in the patient with respiratory compromise. Inappropriate selection and administration of anaesthetic agents may precipitate decompensation even in the asymptomatic patient.10 Premedics 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 muscle relaxants, used alone or in combination.11 Case series as early as 196910 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 positive pressure ventilation (PPV) with IPPV, and IV access obtained. The incidences of respiratory failure, ileus, or atelectasis post-operatively in TDH patients are frequent, and intubation allows IPPV to avoid potential complications of tracheostomy tube placement.12 It is well recognized that the incidence of atelectasis post-operatively with IPPV, by avoiding forceful inflation of the lungs with IPPV, but performing transdiaphragmatic/thoracostomatic atelectasis at completion of hemicorporectomy. As indicated by the striking pneumothorax pictured, iontophoresis of surgically induced pneumothorax post-operatively via thoracostomy or indwelling thoracostomy tube are recommended to prevent RPE.12,13,14 Restrictions to IPPV do not allow for preservation of the use 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 (RPE), potential re-expansion pulmonary oedema (RPE), and iatrogenic re-expansion pulmonary oedema (RPE) by placing a minimal tidal volume.12,14 Thus, further, the routine practice of taking a post-operative x-ray following surgical repair of diaphragmatic hernia would be defensible, allowing identification of pleural space abnormalities and pulmonary oedema in a timely fashion.

Monitoring of the TDH patient post-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 correctly.

The definitive method of quantifying the efficacy of ventilation and gas exchange is arterial blood gas analysis.15 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.16 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 anesthesia, with particular value in the respiratory compromised individual. It is a non invasive method of determining arterial oxygen saturation (SpO2) in arterial blood. Although SpO2 is not linearly related to PaO2, pulse oximetry can still provide clinically relevant information regarding tissue oxygen delivery of oxygen. The advantages of pulse oximetry in comparison to arterial blood gas analysis are that there can be provided a continuous, non-invasive estimation of oxygenation. However, pulse oximetry data must always be interpreted critically, as readings are subject to artefact and non-pulmonary factors.17 A further obvious disadvantage is that no information is provided regarding arterial carbon dioxide levels.18 Capnometry is the spectrosopic technique for measuring carbon dioxide in respiratory gases. Early tidal capnography allows the determination of adequacy of ventilation, because elimination of CO2 by the patient is the best determinant of alveolar ventilation.19 Although end-tidal capnography has unique features that may be helpful in monitoring the critically ill or to individually to tailor IPPV, studies have shown that end tidal readings can diverge significantly from arterial readings particularly in the diseased patient.19,20 Hence arterial analysis remains the definitive method for evaluating carbon dioxide levels.20 Capnometry, particularly with bedside analysers, also has limitations in patients such as ours that have a very small tidal volume.21

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. Particularly in our patient, close monitoring for hypercapnia would have signalled ventilatory issues prior to hypoxia developing as a result of the pneumothorax. Pneumothorax-induced hypercapnia and subsequent respiratory acidosis was almost certainly the cause of delayed anesthetic recovery in our patient, as it can result in loss of consciousness/stupor in the absence of anaesthetic agents.15-18,20 Furthermore, the routine practice of taking a post-operative x-ray following surgical repair of diaphragmatic hernia would be defensible, 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. Meladone Injection; Ikm.
b. Midazolam Injection; Propofol

References:

COMMENT ON PERSPECTIVE 107: LIVE ANIMAL EXPORT IS UNETHICAL BY PETER KERKEZOV
(FIRST PUBLISHED IN THE C&T SERIES JUN 2014, ISSUE 275 EBOOK)

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

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 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 Overview (2014). Both are available online as they have been published open access. 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 (conceded) 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 and VALE is the lack of independent veterinarians on live export ships; this was also noted as a concern in the Kenya Enquiry. Veterinarians are currently employed by the exporters. Veterinarians such as Dr Peter Kerkezov, 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 AUSL (Australian Standards for the Export of Livestock)1) on live export voyages, was removed from her position in the Animal Welfare Unit of 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.

References
heat_stress_paper.pdf
S1060023114001014
com.au/policy/151-live-animal-export
5. See Australian Standards for the Export of Livestock (Version 2.3) 2011:
standards-v2.3.pdf
export-trade/submissions-export-livestock

COMMENT

Dear Editor

I would like to thank Peter Kerkezov 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 Kerkezov’s article has a wealth of experience backed by powerful images which make it invaluable.

M Richard BVSc

INTRODUCTION

A recent fatal stonefish envenomation of a dog in Redcliffe (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 venom 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 verrucosa) and the estuarine stonefish (Synanceia horrida, synonym S. rachynus). Both are ambush predators and are typically superbly camouflaged, often with their own epiphyton of algae. True stonefish have 13 non-branching venom spines (Figure 2). This characteristic distinguishes it from species that are bataness mimics (many of which are also venomous, but less so than true stonefish).

Stonefish are distributed from Woolloongoa 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 Caribbean. Stonefish are typically found in relatively shallow waters with the reef species preferring coral reefs or story/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. 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).[2]

As with most stonefish, their venoms are complex mixtures and contain about 15% protein. A number of 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 depolarising neuromuscular blockade[1]. Limb paralysis and death from respiratory muscle paralysis are noted features of stonefish venoms in experimental rodents.

• The cardiovascular effects are mediated by muscarinic receptor activation. The associated hypotension is supported by sympathomimetic (alpha) receptor activation.

• The neuromuscular effects are mediated by depolarising type neuromuscular blockers. The venom also contains potent sodium channel blockers.

• Stonefish venoms contain a potent excitatory and inhibitory amino acid neurotransmitters.

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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 [6-8].

Stonustoxin from the eel skinstonefish also produces depolarizing paralysis of the diaphragm, contributing to respiratory paralysis [10].

- The stonefish venom lethal factor (stonustoxin) has a LD50 and is about 17 times lower than crude whole venom [11]. Stonustoxin is a potent hemolysin 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 pressure/vasoconstrictor activity of noradrenaline (notably in the aorta) [13]. The central arterial dilator activity of stonustoxin, combined with volume loss due to its oedema forming action, is considered to be the major cause 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 hemolysic 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 anecdotal, 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 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 mortal 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°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 offers benefit in about 75% of human cases and it is 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 adrenaline and noradrenaline. Additionally, given the effects of stonefish venom on vascular integrity, the risk of pulmonary edema following aggressive treatment with intravenous 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.

References


1. http://www.couriermail.com.au/newsquest/news/moreton/mum-warns-mum-warns-eminent-treatable-with-antivenom-and-analgesia. 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
RAT BAIT

QUESTION POSED ON THE CVE/ISFM DE FELINE MEDICINE LISTSERVE

C&T NO. 5421

Nikki Frost

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:

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC</td>
<td>2.74 x 10^12/L</td>
<td>5.5-12.2</td>
</tr>
<tr>
<td>HCT</td>
<td>11.60%</td>
<td>30.3-52.3</td>
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<tr>
<td>HGB</td>
<td>4.39 g/dL</td>
<td>6.8-16.2</td>
</tr>
<tr>
<td>MCV</td>
<td>42.3 fL</td>
<td>35.9-53.1</td>
</tr>
<tr>
<td>MCHC</td>
<td>15.7g/dL</td>
<td>11.8-17.3</td>
</tr>
<tr>
<td>MCHC</td>
<td>37.1 g/dL</td>
<td>28.1-35.8</td>
</tr>
<tr>
<td>RBC</td>
<td>11.6 x 10^6/L</td>
<td>2.87-17.02</td>
</tr>
<tr>
<td>Toluen</td>
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<td></td>
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<tr>
<td>Tolamin</td>
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<tr>
<td>Torabine</td>
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</tr>
<tr>
<td>Tolcain</td>
<td>0.1</td>
<td></td>
</tr>
<tr>
<td>New</td>
<td>0.22 x 10^6/L</td>
<td>1.24-10.2</td>
</tr>
<tr>
<td>lym</td>
<td>1.96 x 10^6/L</td>
<td>0.92-6.88</td>
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<td>mono</td>
<td>0.37 x 10^6/L</td>
<td>0.05-1.67</td>
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<tr>
<td>eos</td>
<td>0.01 x 10^6/L</td>
<td>0.17-1.57</td>
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<tr>
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</tr>
<tr>
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<td>350k/L</td>
<td>151-900</td>
</tr>
<tr>
<td>Protime</td>
<td>100 sec</td>
<td></td>
</tr>
</tbody>
</table>

So did Vt 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 Vt K. PT was normal with a PCV of 38%, 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

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/catprofessionals/free_downloads.html

POSSIBLE ALFAXAN REACTION

C&T NO. 5422

Geoff Haynes

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 IV, 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 and then finally realised this was probably anaphylaxis or an anaphylactoid reaction to alfaxalone. I gave 20µg adrenaline (0.02mL of 1/1000 adrenaline), 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 adrenaline (0.02mL of 1/1000 adrenaline), 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 adrenaline when the kitten suddenly slurred and ‘came around’. She was very disinorientated but within 30 minutes was ‘quite good’. She was successfully deessed 2 days later with a premix 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. 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 APVMA 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 adrenaline, 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 inapplicable 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 (alfalone 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 ciclohexidrin vehicle.

Handy tip from de feline medicine listserv

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

Possible Alfaxan reaction

Note: When searching on pubmed, ALFAxALone (US & UK) retrieves entries for both spellings, but ALFAxALONE (Australia) will only retrieve the subset that use this spelling.
Thank you for giving us the opportunity to comment on this C&T. Jurox is the manufacturer of Alfaxan® Anaesthetic. Dr Hayes reported this case to Jurox, and we collated the information involved. Our discussion suggests there is no evidence 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 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. Alfaxalone has also been shown to modify cardiovascular function and is a weak myocardial depressant. The formulation also contains hydroxy-propyl betacyclodextrin, salive 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 anaphylactic 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 Mejigan 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 is similar adverse reaction reports. However, if these reactions are all physiologic, and indeed myself I 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 would 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 had a ‘case mortality’ or ‘case morbidity review’, some of which went 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.

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INVITED COMMENTARY COURTESY OF:

Stephen Page
Advanced Veterinary Therapies
PO Box 905 Newcomb NSW 2402
E. stephen@avth.com.au

The report from Geoff Hayes describes an adverse experience in a feline 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 administered, to remember available information on other 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 these 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 approved by CVMA) available at http://www.fda.gov/AnimalVeterinary/ProductsApprovedAnimalDrugProducts/FOMADSummary.cfm. This 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/10845. For medetomidine, over a 27 year period there are 5 reports of pulmonary oedema in cats 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, identified as “respiratory problems” and one report of an alfaxalone (IT) 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 list 16 reports as “edema, lung congestion” for cats. These reports are for allergies, 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; and rechallenge; and

rechallenges. 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

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

Carla Appelgren, Small Animal Surgery
Susan Carr, Small Animal Medicine
Karen Chalmers, Medicine of Cats
Jennifer Chau, Veterinary Radiology (Small Animal)
Jeffrey Clyne, Veterinary Emergency & Critical Care
Abbie Couper, Veterinary Emergency & Critical Care
Mihai Coasa, Veterinary Emergency & Critical Care
Julie Culver, Veterinary Behaviour
Natsuko Drury, Veterinary Emergency & Critical Care
Rebecca Frances, Surgery of Cats
Nicola Gaut, Medicine of Cats UK
Joanna Goldman, Veterinary Behaviour
Kyle Grant, Small Animal Surgery
Clifford Ho-Ie, Veterinary Emergency & Critical Care
Karen Holker, Medicine of Cats
Nicole Hoskin, Small Animal Dentistry & Oral Surgery
Marethe-Hoeyer, Medicine of Cats
Mathison, Veterinary Behaviour
Joanna McLachlan, Veterinary Behaviour
Dawn Mills, Avian Health (Fowl)
Ingird Nash, Veterinary Emergency & Critical Care
Jennifer Nesbitt-Hawes, Veterinary Behaviour
Joanna Pitlon, Veterinary Radiology (Small Animal)
Helen Purdham, Veterinary Behaviour
Ema Rankin, Small Animal Medicine
Muira Roberts, Medicine of Cats
Sarah Robinson, Veterinary Pathology
Margaret Roser, Veterinary Pathology (includes Anatomical & Clinical Pathology)
Natalie Roulatt, Veterinary Radiology (Small Animal)
Reza Sangam, Surgery of Horses
Nadia Sternberg, Medicine of Cats
Rachael Straton, Veterinary Behaviour
Michelle Sutherland, Avian Health (Caged & Avairy Birds)
Sarah Tawse, Veterinary Emergency & Critical Care
Sandra Torchill, Medicine of Cats
Leah Wright, Small Animal Medicine
Genevieve Zhang, Small Animal Surgery

Authors’ views are not necessarily those of the CVE

Back to Table of Contents
Kerrie has won a DVD of her choice from www.vetbookshop.com

A BIT OF FUN!

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

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

RADIOGRAPHS, CT & SAMPLING THE LUNG

Zoe Lenard
BSVc(Hons) FANZCVS(Radiology)

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 (enrofloxacin and amoxicillin-clavulanic 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.
A diagnostic dilemma: the clinical signs and imaging findings are consistent with two types of lung sampling (bronchoalveolar lavage, and ultrasound-guided fine needle aspirate). The patient returned 1 week later for a tru-cut guided biopsy of the left lung.

After induction of anaesthesia using propofol and maintenance with isoflurane and oxygen, a 14-gauge tru-cut biopsy was obtained from the left lung, using ultrasound guidance. The biopsy tract was 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 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 to refine a differential diagnosis list and accurately confirm disease, in the fewest of tests. The lung pattern on the initial radiographs (Fig 1A 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 tissue 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 entirely 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.

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References

Figure 1A: transverse CT image in the dorsal thorax, showing a right-sided pneumothorax (left lower quadrant, arrows). To the left of the left caudal lung lobe (asterisk) is a poorly defined round opacity. The left caudal lung lobe is marked. Other normal anatomical features include the caudal pulmonary veins (asterisks) and part of the liver and gall bladder (*).
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 be less than optimally (and in some cases even euthanased) due to restricted owner finances, so effective pet insurance could be a real lifeline for many 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.

Vet professionals and insurers need to understand and communicate with each other 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, bombarded 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 practising small animal vet from the northern beaches of Sydney has described the stalling and delays of access to face to face than an insurance call centre 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 pieces 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 rate 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 owners.’


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 ligature.
- 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 ctitis externa requiring total ear canal ablation surgery.

Yet sometimes insurers broadly exclude all 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 an episode of mild superficial pyoderma years before. What absurd nonsense! If a human has an episode of acne 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 declaring 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 size, such as a melanomata gland adenoma of the eyelid, 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 overkill 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’.
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, could it be a scam or are some insurers are an honesty with their external scanning contractors, and the claim should be finalised within 5-7 days. (It wasn’t).

I have personally heard several examples when ‘forms that were never received’ were where the 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).

On February 14th, at the request of the owner we made an clinical examination, especially if pathology free, or document loss associated with the passing of time).

The level of public trust in us is well established – surveys repeatedly find that veterinarians are amongst the most respected professionals. Pet insurance hasn’t been known to ‘go down in public esteem’ and always submit honest and accurate records. In NSW at least, under the new Veterinary Practice Regulation 2013, the Veterinary Practitioners Code of Conduct states that: a record of a consultation, procedure or treatment is altered, the alteration must be clearly identified in the record. 

Veterinary Practitioners Code of Professional Conduct states that: a record of a consultation, procedure or treatment is altered, the alteration must be clearly identified in the record. 

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Veterinary Practitioners Code of Professional Conduct states that: a record of a consultation, procedure or treatment is altered, the alteration must be clearly identified in the record.
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 panelists 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 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, 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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