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C&T
CONTROL AND THERAPY SERIES

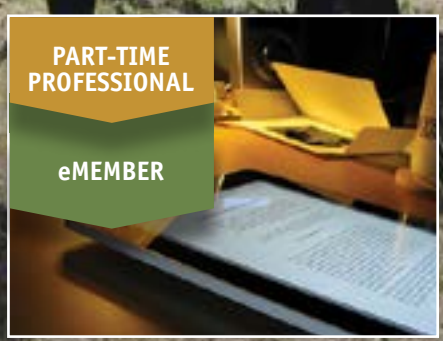
JUNE 2014 ISSUE 275
Australia's Leading Veterinary Forum

eBOOK

FROM FELINE MEDICINE TO SAVING AUSTRALIA'S BRUMBIES

by Andrea Harvey, pg 39

FEATURE ARTICLE:
NECROPSY SUMMARY
by Adam Gordon & Lydia Tong
Fig. Haemorrhage within 4th ventricle of brain



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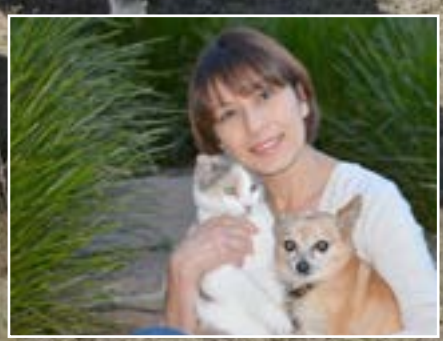

NEW AWARD:
'CELEBRATING OUR VETS'
Awarded to Joeanne Katsikaros
See Sue Foster's article on pg 25

C&T
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FROM THE DIRECTOR

This edition of C&T contains our usual range of high quality contributions from a diverse range of sources across a variety of topics.

Many people comment about our use of the goanna in the C&T and also in some of our promotional material. As the CVE approaches its 50th year, many of our younger readers may wonder why we continue to use this image so frequently. The first Director of the CVE, Dr Tom Hungerford, was an incredibly energetic veterinarian who built up a very large and successful practice in western Sydney and was a prolific writer. He wrote numerous textbooks and constantly advocated that knowledge can lead you anywhere – in fact he frequently wrote about how any vet could 'follow the goanna track to success' by reading all that was available on a subject and by immersing oneself in a subject. We at the CVE believe that everything we provide is following along that theme, to help to lead you along the goanna track to success.

Readers will notice an article featuring the goanna, promoting a new range of membership categories available when you join or renew your membership with the CVE. Each of these membership options provides a range of benefits, which we have designed in response to feedback from both our existing members, as well as other veterinary professionals not currently members. There is something for everyone, as our membership program provides relevant support at every stage of your professional life. We have recognised that with the changing face of the veterinary profession there are many disparate demands on your time and money, not to mention the constantly changing nature of information delivery, so the CVE team has come up with different membership options to suit your special needs.

We hope that our membership drive will be well received, as stronger membership means that we can continue to provide new and better benefits, while you gain access to an increasing range of products, at prices that suit your current situation.

The CVE Distance Education (DE) program for 2015 has been released with early bird registrations now available. Thousands of veterinarians have benefitted from our DE courses which have been running for over 20 years. Historically, over 30% of those who do DE courses go on to study for and pass their ANZCVS Membership exams. Others study a particular course simply because they want to be a better vet. Interestingly, many participants complete DE courses because they actually enjoy studying and challenging themselves – when near enough is no longer good enough.

Distance Education requires a large commitment in time and effort, but testimonials from satisfied vets who have completed the courses speak for themselves. Many derive a greater sense of satisfaction from their work and others have reported that they have actually increased their earning capacity, through being able to provide their patients with a higher level of care.

Our new membership program now offers member rates for DE courses. Easy payment options remain available. Look out for the CVE 2014/2015 Continuing Professional Development Program booklet, which will appear in your mailbox or at your practice very soon. There is something in there for everyone!

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19 Oct	Small Animal Dentistry	**Port Macquarie**

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20 Jul	Bone Plating - FULL	**Sydney**
9 Aug	Feline Surgery	**Sydney**
10 Aug	Canine Surgery	**Sydney**
16 Aug	Feline Surgery	**Townsville**
17 Aug	Canine Surgery	**Townsville**
26 Sep	Basic Echocardiography	**Melbourne**
27 Sep	Advanced Echocardiography*	**Melbourne**
11-12 Oct	Diagnostic Imaging Workshops	**Sydney**
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28 Jul	Anaesthetic Complications
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8 Sep	Feline Emergencies *NEW*
15 Sep	Small Animal Behaviour
22 Sep	Dermatology
13 Oct	Wildlife
3 Nov	Pet and Aquarium Fish

WEBINARS

26 Jun	IMHA and how to prevent pulmonary thromboembolism
24 Jul	Tick paralysis treatment update
31 Jul	Backyard Chooks
7 Aug	Blue Tongues, Turtles and Frogs
21 Aug	Congestive Heart Failure in dogs with mitral valve disease
11 Sep	Rabbit medicine and dentistry
25 Sep	Elapid snake envenomation
9 Oct	Referring a behaviour case: to whom and when
23 Oct	Medical Management of Uveitis and Glaucoma
6 Nov	Surgery - Acute Abdomen

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View the full 2014-2015 CPD calendar on our website: cve.edu.au

Back to Table of Contents

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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- **Adam Gordon & Lydia Tong**: NECROPSY SUMMARY

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- **Mimi Dona** – MAMMALS: POSSUMS & GLIDERS

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- **Nikki Frost:** JUST NOT CRICKET! CASE OF AN UNUSUAL FOREIGN BODY
- **Sue Foster:** TWO DIET-ASSOCIATED DISEASES IN THE ONE DOG

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Established in 1969, the C&T is the brainchild of Dr Tom Hungerford, first Director of the PGF (renamed the CVE) from 1968-1987, who wanted a forum for uncensored and unedited material.
Tom wanted to get the clinicians writing.

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

Authors' views are not necessarily those of the CVE

Back to Table of Contents

WINNER OF BEST FILM CLIP

PART 4: WILDLIFE FLASHCARD SERIES
POSSUMS AND GLIDERS

C&T NO. 5387

Mimi Dona
Lecturer on Animal Studies and Sustainability
at the Metropolitan South Institute of TAFE

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

RESTRAINT OF:-

squirrel gliders

sugar gliders

possums

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▶	Bandicoots, Native Rodents & Small Carnivorous Marsupials
▶	Bats
▶	Echidnas
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▶ ▶	Macropods

Film clips courtesy of Lincoln Williams
fotomedia.com.au

Possums and Gliders

Be aware:

- They can inflict a nasty bite and scratch.
- Marsupials – always check for pouch young (gliders usually have twins).
- Marsupial young are born very under-developed and until the stage of development that we are able to successfully hand rear young joeys, they are considered to be 'unviable'. Consult a trained/experienced person for assistance with identification and confirmation on viability.
- All species have strong home ranges – essential to obtain accurate details of rescue location and release at night, as they are nocturnal.
- Gliders – live in social groups (except Greater Glider). They have a gliding membrane from their forearm to hind leg.
- Possums – All species are arboreal and nocturnal. They have prehensile tails.
- Native marsupials don't get tick paralysis.

Handling

- Both possums and gliders can be hostile and may attempt to bite and scratch.
- Possums are best restrained by simultaneously grasping the back of the neck with one hand and the tail with your other hand, with the aid of a towel and/or gloves. When holding the back of the head you will find there is no scruff and you will need to use a grasping technique for an adequate grip. With smaller possums, you will be able to get a better grip by using your whole hand, making a 'V' with your fingers.
- Gliders can be grasped at the back of the head with your thumb on one side and your forefinger on the other side; a small hand towel is highly recommended to avoid being bitten and to contain the body.

Housing the sick or injured possum and glider

- Preferred enclosure temperature for Adults is 28˚C. Orphaned joeys just furred to furred 28˚ - 30˚C and 32˚C for unfurred.
- Adult possums can be housed in a carry cage (preferably one that opens from the top); line with soft towels or small blankets and always provide a box to hide in and cover.

Figure 1a. Unlike a cat, possums lack the loose skin around their neck to scruff so a grasping technique is required for an adequate grip. Towels and/or gloves are recommended to avoid being bitten or scratched.

Figure 1b. Possums are best restrained by simultaneously grasping the back of the neck with one hand and the tail with your other hand.

- Adult gliders can be housed in a plastic aquarium or smooth sided ventilated tub with towels to line the bottom and a secure lid as they are very good at escaping. Always provide a box to hide in and a cover.
- Place orphans in a cotton pouch. Heat must be given to orphans; ideally they should be housed in a Vetario® or Humidicrib and monitored with an indoor/outdoor thermometer. Alternatively, place in a plastic aquarium with a sealed/ventilated lid.

Emergency diet

- Only offer food once rehydrated.
- Native vegetation (flowers for nectar and pollen and foliage for gleaning tiny arthropods) are an important part of this species diet but should only be offered if you are familiar with the species.
- Gliders have a high metabolic rate and it is important to give them oral or subcutaneous fluids if not self-feeding. They can be offered a variety of foods: native leaves, flowers and fruits, liquid nectar mix, small quantities of Wombaroo Lorikeet and Honeyeater mix, fruit, vegetables, mealworms, soaked dog kibble. Greater Gliders must be given leaves and flowers from eucalypts ONLY.
- Possums can be offered leaves, flowers and fruits from a variety of native plant species (e.g. eucalyptus, acacia, lilly pilly,

Figure 2a. Using a small hand towel is highly recommended when handling a sugar or squirrel glider to avoid being bitten, and to contain the body.

Figure 2b. For an adequate assessment, gliders are best examined under anaesthesia.

Back to Table of Contents

Figure 3. If unsure, consult a trained/ experienced person for assistance with identification and confirmation on viability of orphaned joeys.

Figure 4. Native vegetation is an important part of this species' diet but should only be offered if you are familiar with the species.

Figure 6. Orphans can be given water and glucodin initially for the first 2 feeds, then a suitable milk replacer.

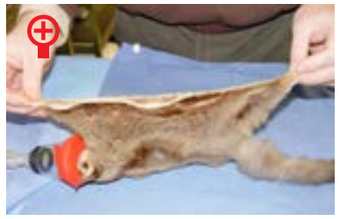

Figure 7. Gliders have a gliding membrane from their forearm to hind leg.

Figure 8. Always check for pouch young (gliders usually have twins).

bottle brush) – preferably with good quality tip, mealworms, fruit, vegetables and soaked dog kibble. Ringtail possums are hindgut fermenters and fruit should not be offered or be limited.

- Orphans can be given water and glucodin initially for first 2 feeds, then suitable milk replacer (Divetelact®, Biolac® M 100 or Wombaroo® Possum Milk Replacer >0.8. This can be given either via a 1mL syringe with catheter tip, bottle and appropriate sized teat or in a bowl if lapping.

Assessment under anaesthetic

Gaseous

- Use an anaesthetic mask at 5% induction; it can take 2 - 3 minutes. Or, if there are difficulties handling, place in an anaesthetic box, which will take an additional few minutes. Maintain using a mask on Isoflurane® at 1.5 – 2% with an oxygen flow rate of 1 L/min.

Anaesthetic agents

Due to the difficulty with intubation of small gliders and possums and IPPV in case of apnoea, gaseous anaesthesia is recommended. Alfaxan CD RTU 3 – 5 mg/kg – (I/M)

Figure 5. Adult possums can be housed in a carry cage (preferably one that opens from the top); always provide a box to hide in and keep covered to reduce stress.

Intubation

Is very difficult with small marsupials. With some of the larger species, use a cuffed endotracheal tube or catheter tip and insert the endotracheal tube with the aid of an anaesthetic spray and tie in with shoelaces.

Recovery

Use a Bair hugger® or heat mat at room temperature to maintain the patient's core body temperature throughout the procedure, using a cloacal thermometer to monitor. With joeys, Vetario® or Humidicribs are ideal during post-operative recovery

Fluid Therapy

It is important to remember to warm the fluids being administered. Using 0.9% sodium chloride, dose the patient at 5% of its bodyweight. Fluid therapy can be administered subcutaneously or standard I/V infusion rates. Syringe pumps are ideal to use in small mammals. IP fluids (under GA) are an option if unable to get a vein and advanced dehydration.

Preferred routes of drug administration

- Subcutaneous – loose skin at lateral neck/shoulders, side of abdomen or over thigh area (be careful of membrane in Gliders).
- Oral – given via a syringe. For orphans use a 1mL syringe with a 24g or 22g cannula attached.
- Intramuscular - dorsal lumbar muscles, cranial and caudal thigh, upper arm (only larger species).
- Intravenous – cephalic is the preferred route of drug administration, alternatively lateral caudal tail vein, saphenous, jugular (difficult).

Euthanasia methods

Injection of sodium pentobarbitone can be administered either by intravenous, intracardiac or intraperitoneal routes.

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

ENDOMETRITIS AND RENAL THROMBOEMBOLIC INFARCTION IN A HEIFER

C&T NO. 5388

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A necropsy was performed on a 2-year-old Poll Hereford X heifer that had been down for 3 days. Prior to euthanasia, she was in lateral recumbency and straining intermittently, with a purulent vaginal discharge. No foetus was present; however, the uterus contained malodourous purulent material.

The uterine wall was thickened and the uterus was twice its normal size. Both kidneys were grossly discoloured, mottled red and pale brown, with the left kidney more affected than the right. The liver was pale brown red with slightly rounded margins. The abdominal adipose tissue was bright yellow.

The kidneys demonstrated 2 distinct patterns: pale yellow raised multifocal, pinpoint foci with hyperaemic margins, and depressed well demarcated multifocal regions varying in colour from pale pink to yellow with dark red margins and measuring 2 to 5 cm in diameter. On the cut surface, the latter regions were in a wedge pattern within each lobule. Within the adipose tissue of the left retroperitoneal space, a relatively large cavitated fluid-filled space was present that was surrounded by brown discoloured adipose tissue.

Samples (fixed and fresh tissue and gel swabs) were sent to the Department of Primary Industries Brisbane for histopathology and bacteriology. The results were: suppurative metritis, thromboembolic infarction of the kidneys and steatitis. Salmonella chester and Trueperella pyogenes (formerly Arcanobacterium pyogenes) were isolated from the uterine swabs. An almost pure growth of Salmonella chester was isolated from the kidneys.

The endometritis was the most likely cause of the renal lesions. This was supported by the isolation of mixed organisms in culture and a pure isolation from the kidneys, together with the infarctions caused by bacterial thromboemboli.

Bacteraemia and the resultant endotoxaemia would have been the cause of death in this heifer if the euthanasia had not been performed.

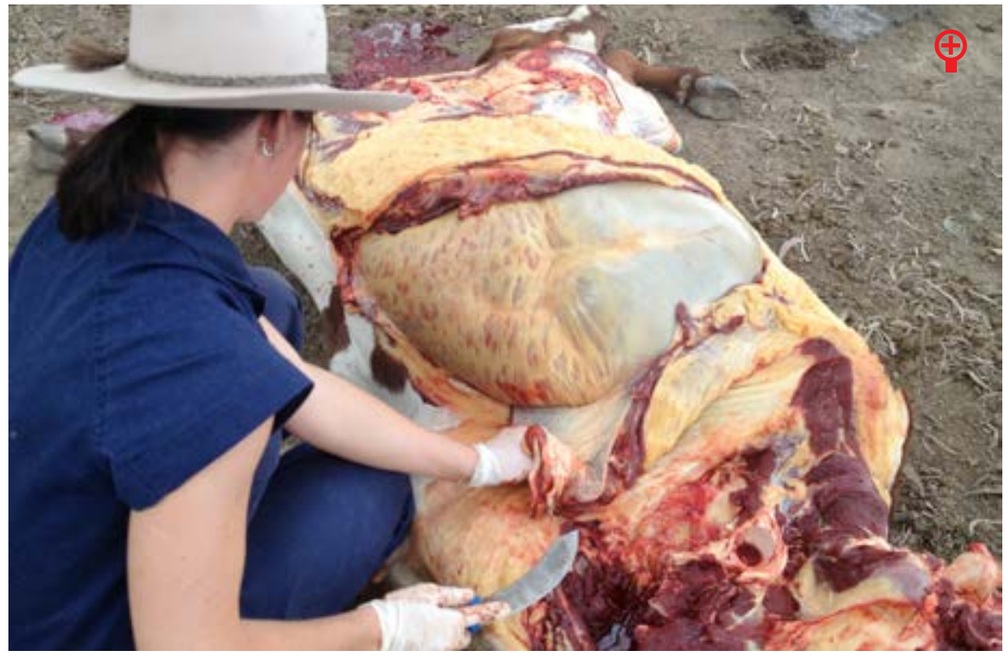

Figure 1: Getting to the display stage. Note the yellow adipose tissue.

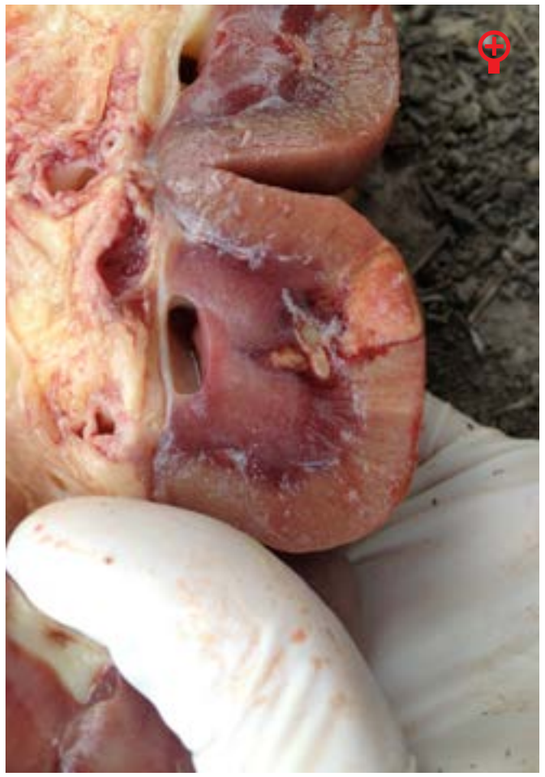

Figure 2: Left Kidney. Note the two distinct patterns, raised pinpoint foci and the larger depressed regions of infarction.

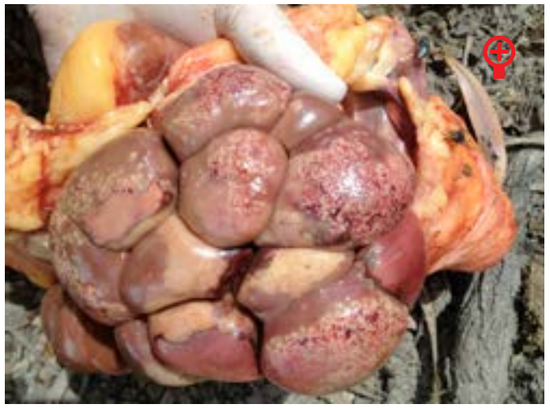

Figure 3: Left Kidney, cut surface. Note the well-demarcated pale cream foci with hyperaemic margins, leading to a wedge of necrosis within the renal cortex.

INVITED COMMENTARY COURTESY OF:

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This is an interesting case report of fatal septicaemic salmonellosis associated with likely abortion and metritis.

It is presumed that the endometritis and bacteraemia were precipitated by *Salmonella* abortion in a beef cow, although further information is required to confirm that the cow had been pregnant and at what stage of pregnancy the event occurred.

The potential for a dual primary role of *Salmonella* and *Trueperella* is unclear in this case. It is to be presumed that the isolation of *Trueperella pyogenes* (formerly *Arcanobacterium pyogenes*) from the uterus is due to secondary invasion post abortion, although the possibility of it playing a role in the presumed abortion cannot be excluded.

The presence of pure growth *Salmonella chester* in the kidney specimens associated with gross findings consistent with infarction confirms the role of bacteraemia and thromboembolic disease in this case. The gross images of the kidneys are classical for the appearance of suppurative embolic nephritis with occasional areas of tissue necrosis consistent with consequent infarction. It would be interesting to know whether there was also an endocarditis present or vasculitis evident elsewhere in the body.

The source of *Salmonella* in this case is not speculated upon in the case report and it is not clear to me a likely source given my preconceptions about the likely pastoral operation from the region. Given the current weather conditions in that region, the potential for contaminated supplemental feeding to be involved in the exposure to *Salmonella* seems likely. It would be interesting to note whether this is a sporadic case or whether there was more than one animal affected.

In the images it appears that there may be yellow discolouration of the fat. The pathogenesis of the presumed jaundice is not resolved from the information provided. It is possible that it relates to a prehepatic jaundice associated with intravascular haemolysis associated with the exotoxin production of *T. pyogenes* or endotoxaemia associated with Salmonella septicaemia; or hepatic jaundice associated with generalised hepatocellular injury due to endotoxaemia/septicaemia or with multifocal embolic hepatic disease. The latter seems less likely given the gross description of mild hepatic swelling rather than multifocal hepatic disease. The possibility of concurrent unrelated hepatic disease cannot be excluded, from the information provided. Histopathological examination of the liver may have helped discriminate the pathogenesis of presumed jaundice in this case.

Reply from Michaela:

Thanks Mark for your comment. To answer your questions:-

At the time of necropsy, cotyledons were present within the uterus and the uterine discharge that is consistent with a 5 month old pregnancy.

No endocarditis or vasculitis was evident grossly.

This was a sporadic case, and the source of the Salmonella is unknown as a paddock inspection was not completed at the time. The cattle were not on supplementary feed, the conditions were dry and stock were feeding on native grasses and old sorghum stubble.

POLLNESS AND INTERSEX GOATS

C&T NO. 5389

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The sheep industry is looking to use DNA technology to breed poll sheep varieties. Unfortunately this will be a pipe dream for goat breeds. Just as well for cashmere goats which are shorn standing up with their mouth and nose in a leather holder on a solid stand and a chain passed behind the horns to keep them locked in. Angoras are also handled by holding onto horns. However horns are dangerous to goat owners who can suffer real damage, even accidentally from a goat quickly raising its head. Dairy and pet goats should therefore be disbudded a few days after birth, as soon as the horn buds can be felt. Horned goats have whorls in the hair on the top of their heads. Most goat breeders do this rather unpleasant task themselves.

Why not breed poll goats then? The reason is that in goats the dominant gene for pollness is associated with a recessive effect for intersex, with incomplete penetrance.[i]

There is no need for a DNA to test for poll genes in goats. French research is available on how to tell if goats were either heterozygous or homozygous for the poll gene by looking at the top of their heads (which would be a lot cheaper than any DNA test). The work was referenced in Chapter 4 of 'Goat Production' edited by C Gall and published by Academic Press in 1981. The author of Chapter 4, Genetics: Breeding Plans, was the original researcher, G Ricordeau and he also published photos of plaster casts of the top of head of PP (both genes for pollness) and Pp (1 gene only for pollness) goats.[ii] The former (PP) i.e. those carrying both poll genes have just one large rounded bump on each side. The Pp (i.e. one poll & one horned gene) have bumps shaped like a large bean (with a middle indentation each side and they form a V shape pointing forward. It is best to look at the top of the head at 5-6 months of age, which is fortunately before most goats are breed. Kids born with the genes for horns have hair whorls, which are seen before the horn buds can be felt.

As all PP females are infertile as masculinised in the uterus and half the PP males are infertile, telling the difference between PP and Pp is very important. Females which are intersex vary a lot in their external appearance. Sometime just the clitoris is enlarged (often the size of a pea and easily seen between the vulvar lips) but in other cases the external genitalia are a mixture between male and female. Sometimes the female looks normal but has no cervix (which is confirmed via a vaginal examination). Sometimes the female can even look like a buck with 1 or 2 testes in a scrotum.[iii]

Most authorities agree that horned goats do not have this problem of intersex, although they can still be problems with freemartins (where kids in the uterus share a blood supply). Freemartins are rare but more likely if very large number of kids born in a litter. Hence Anglo-Nubians with their larger kid numbers are more likely to be affected.

Also there is Israeli research showing that beside intersex (pseudo-hermaphrodites) poll to poll matings result in fewer female and more male kids - even if all the intersex kids are counted as females. For these reasons it is not possible to breed a poll goat breed, unlike the situation with sheep and cattle.

So the bottom line for vets is:
Check the top of the head of any goat presented for infertility for poll bumps and put intersex at the top of your differential diagnosis list, if you see just one large bump per side.

And the bottom line for goat breeders is:
Never mate a poll goat to a poll goat

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*CVE Members may access this and other proceedings in the CVe-library.

Goat image courtesy of Scott Reid

Back to Table of Contents

THE ROLE OF GLUTEN ALLERGY IN THE ETIOPATHOGENESIS OF BREED-SPECIFIC FELINE DIARRHOEA

C&T NO. 5390

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A pair of 12-week-old Ragdoll kittens were purchased by a breeder in Victoria. Within a week of purchase, the owner reported a profuse watery yellow-brown and pungent diarrhoea in both kittens which was non-responsive to secnidazole therapy.

These kittens came from a breeder with known problematic issues with Tritrichomonas spp infection.

On clinical examination, both kittens were of normal weight for age (2.9 and 3.3 kg, respectively) and clinically healthy, with no evidence of other diseases apart from chronic diarrhoea. No other cats or dogs were present in the household.

Routine fecal analysis was performed at the initial consultation by IDEXX, which revealed the following:-

- Fecal color brown
- Fecal consistency – soft to watery
- Fecal starch – normal
- Fecal muscle – normal
- Fecal neutral fat – normal
- Free fatty acid – normal
- Fecal trypsin – positive
- Fecal occult blood – positive
- Fecal floatation – nil eggs and cysts

Despite therapy with antirobe (25mg orally once daily for 4 weeks) and secnidazole (100mg orally every 3 days for 2 weeks), followed by depot prednisolone (depomedrol 10mg SQ), the diarrhoea continued in both cats for the next month unabated, interspersed with intermittent bouts of firmer 'cow-pat' stools.

Various elimination trials with HS Sensitive Stomach diet for 4 weeks followed by a fresh-food diet (primarily meat and organs) for a further 4 weeks failed to alleviate the diarrhoea, which at times was voluminous and explosive, often with concurrent flatulence.

The owner eventually submitted both cats for euthanasia at 6 months of age due to house soiling and lack of self-cleaning of the cats, particularly in the hindquarters.

In desperation, both cats were hospitalised and fed exclusively on Felidae, a potato-based dry food[1], with a recommendation for euthanasia if no response was noted within a week. Both cats were withdrawn from all medication and fed only Felidae and fresh water ad lib.

Within 24 hours, normal stool motions were evident and this continued until discharge 7 days later.

Cats have a known hypersensitivity to specific proteins within their diet, as evidenced by remission of inflammatory bowel disease when fed hydrolysed protein diets (e.g. HS Z/D[2])[3]. However, a dramatic response to a potato-based non-hydrolysed diet as opposed to wheat/rice based suggests a possible underlying gluten allergy in the etiology of symptoms rather than a protein-induced hypersensitivity.

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1. www.canidae.com.au
2. Hill's feline Z/D diet
3. Mandigers, PJ et al (2010) Efficacy of a commercial hydrolysate diet in eight cats suffering from inflammatory bowel disease or adverse reaction to food. *Tijdschr Diergeneeskd* 135(18):668-72

TERBINAFINE IN THE TREATMENT OF FELINE CRYPTOCOCCUS

C&T NO. 5391

Cryptococcus is a relative uncommonly diagnosed disease in my clinic. Most cases appear to involve territorial fighting between cats and endemic possum populations which I assume are natural reservoirs for the fungus.

A recent patient presented with bilateral submandibular swellings which were confirmed on biopsy to contain budding cryptococcus. Having experienced poor responses to azole therapy in the past, I tested a large dose regimen of terbinafine, which has a high therapeutic index in this species. This drug has not been reportedly used in feline patients for treatment of this condition, although it has been used in dogs[1,2].

At 25 mg/kg given orally once daily, the patient showed no side-effects from the medication, and within a 3 week period, marked tumour reduction was evident and weight gain was noted.

This medication should be considered as a safe alternative to drugs such as itraconazole in severe cases of feline cryptococcus.

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WE NEED YOUR HELP

PLEASE CONTRIBUTE TO THIS NEW FELINE MYCOBACTERIAL DISEASE WEBSITE WWW.MYCOVET.COM

C&T NO. 5392

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Tutor in CVE/ISFM Feline Medicine DE program
PhD Candidate, University of Melbourne - Faculty of Vet Science

Following on from a PhD in the epidemiology of environmental mycobacterial infections, Dr Carolyn O'Brien, an Australian specialist in feline medicine, has launched a new website dedicated to collecting further data on the incidence of feline mycobacterial diseases from a world-wide perspective.

Dr O'Brien hopes that veterinarians and owners will contribute details of affected animals via an online survey over the next few years, to build up a large database of cases. The data collected will not be passed on to a third party or be used for any financial gain, and are purely for epidemiological purposes.

The site also contains useful information for cat owners regarding the diagnosis and management of feline TB, leprosy and environmental saprophytic mycobacterial infections (for example, rapidly growing mycobacteria and members of the *M. avium* complex).

Also listed are useful contacts for veterinarians, such as details of Mycobacterium Reference Laboratories, and experts in the diagnosis and treatment of feline mycobacterial disease.

Dr O'Brien intends to build on the resource, and any suggestions/requests for additional information or contributions such as images (with owner permission) are most welcome. The site can be found at www.mycovet.com and Dr O'Brien can be contacted via links on the site.

Figure 1. Localised* M. avium *infection on the head of a cat

Figure 2. Conjunctival granuloma caused by M. sp 'Tarwin'

Figure 3. Periocular feline leprosy lesions

Figure 4. Solitary skin lesion typical of feline leprosy

Figure 5. Mulitple cutaneous lesions secondary to disseminated mycobacteriosis

Back to Table of Contents

SUGGESTIONS FOR OUR CAT WAITING AREA?

C&T NO. 5393

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Conversations from the ISFM Forum

The CVE is partnered with the International Society for Feline Medicine (ISFM) for our FELINE MEDICINE DISTANCE EDUCATION PROGRAM

Q. I am part of a large mixed practice so getting anything done specifically for cats is a struggle. We have a cat ward and now have managed to secure a small area of the waiting room for cat waiting when we refurbish the waiting room. It will be small (maybe 3-4 chairs only) so need any advice on what you consider are priorities. How important are shelves off the floor for the cat baskets etc... Any ideas welcome. At the moment cat-only times are not an option. Also, any ideas on how to improve the consult room to make it cat friendly as consult rooms will be the next phase! I have Feliway® diffuser on and often use the windowsill for examinations but most of the new rooms will be internal with no windows.

REPLIES FROM THE LISTSERVE

REPLY NO.1
Nikki Gaut
Charter Veterinary Group
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I've been lucky enough to work in 2 cat-only clinics and have now recently ended up in a big small animal group in Devon – very rural with LOTS of dogs. However, I've been able to take lots of things from the cat-only clinics to help me tweak things in my present clinic (which sounds similar to your set-up) to really improve things for our feline patients.

I find that in small waiting areas you can divide the room effectively with office dividers. These prevent visual contact and provide an area to place posters – on one side doggy stuff and on the other feline. They come in all colours so you can even keep with your practice 'theme'! In one clinic I literally had 2 seats to work with so I bought some low-slung small tables to place either side of each seat with a pile of Feliway®-sprayed fleecy blankets underneath for clients to place over their cat boxes. The clients then placed the box on the table and sat the other side. I plugged in Feliway® and then put out some cat magazines to calm the owner as much as the cat! At reception I've divided the desk into a cat area where the owner can place the carrier so it's off the floor, and placed a dog lead hook the other side so dog owners know to go that side. As Pete points out, I've found educating your reception staff will be your biggest asset.

As for the consult room I've worked with windows and no windows and I don't have a preference. The darker room can be more calming and seem cosier so don't be put off! As long as there's somewhere for the cat to settle raised up from the floor (with a blanket or vetbed as Sarah suggested) then I find they're happy. Always keep a stash of catnip toys (washable) for interaction with nervous kitties and some healthy treats (like Thrive®) to tempt. Cat scales are an absolute must and having everything to hand is important so you don't have to keep leaving the room – quiet clippers, vaccines etc.

REPLY NO. 2
Zaila Dunbar
Queens Park Vets, London
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I basically said a combination of what Sarah and Pete said but with the addition that I work in a clinic where it is not possible to have a separate waiting area or a dedicated consult room or cat-only consult times but we are hugely successful at being cat friendly purely by all the staff having great cattitude and being clever with booking appointment slots, whipping cats to a quiet space out back if there's any hint of a noisy or bouncy dog in the waiting room and never allowing baskets to touch the floor. Noise and activity are controlled almost to the level of seeming a bit neurotic to colleagues/staff who don't usually work with us. We're obsessive about stopping cats catching glimpses of other cats (or dogs) around the clinic. It's amazing what you can achieve simply by changing what all staff do rather than actually making the physical changes without everyone's heart and understanding being in it.

We love our dog patients/clients but our cats and cat owners are in no doubt about how well we recognise and cater to their special needs.

Reply from Lisa

Thanks all for your encouragement. I just have the waiting room and consult room to go before being able to comply with the feline friendly practice standards, so quite an achievement in a rural/farming area! But news is spreading about the 'cat vet' and one day (in my dreams) maybe cat-only clinic...

Go to our complementary e-book version to download these articles:

-C&T No. 5356 Cat friendly clinics, Pete Coleshaw, Issue 273, Dec 2013

-Creating a cat friendly clinic and working towards ISFM Accreditation, Andrea Harvey, pg 2, Issue 273, Dec 2013

WINNER!

PLEURAL HAEMORRHAGE IN A 2-YEAR-OLD KELPIE CROSS

C&T NO. 5394

Madeleine Richard BVSc Hons I

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Note: Madeleine worked on this case whilst she was employed at the Animal Emergency Centre, Canberra

'Darla', a 25 months old female desexed kelpie cross was presented to us after hours for lethargy and increased breathing effort. On examination she had white mucous membranes, her lung sounds were harsh but not crackly and there was a distinctly increased inspiratory effort. The respiratory rate was 40bpm. She was also hypothermic (36.0˚C) and her haematocrit was 34% (0.34 L/L) and total protein 48g/L with a clear plasma. This suggested a recent internal haemorrhage. There was no sign of haemolysis.

Darla had spent the day in the secure yard of a Canberra suburban house, with other dogs that showed no sign of illness. There was no history of trauma or of access to rodenticides. The possibility of a snake bite was deemed to be slim by the owners although ever present in Canberra.

Radiographs were taken that showed a pronounced pleural effusion, as well as an opacity over the base of the heart.

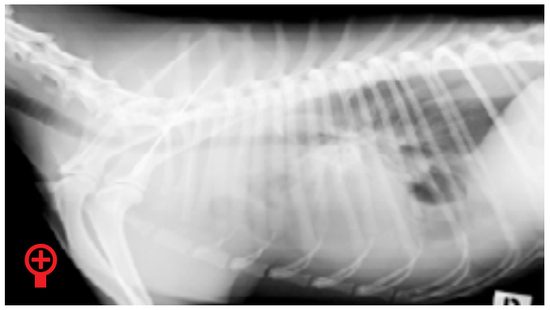

Figure 1

Differential diagnoses were of pyothorax, intrapleural haemorrhage, chylothorax and right-sided heart failure. The plan, as outlined to the owners, was to do an ultrasound-guided thoracocentesis and examine the fluid collected. The amount of fluid in the chest precluded further radiologic interpretation.

As finances were limited, our hope was that the diagnosis would be of pyothorax, a condition common in our area during grass seed season. We find that with aggressive draining of the chest followed by appropriate antibiotic therapy a full resolution

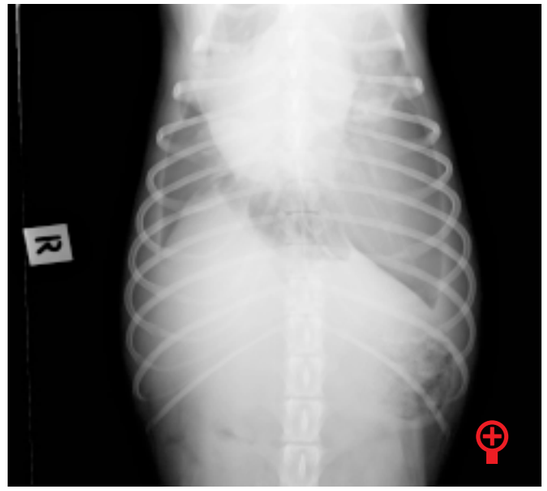

Figure 2

is usually achieved. A concurrent loss of red blood cells into the effusion may have accounted for her mild anaemia.

The owner opted to go ahead. The ultrasound revealed free fluid, as well as a flapping circular mass adjoining (but not part of) the heart. It was assumed to be a fibrinous deposit.

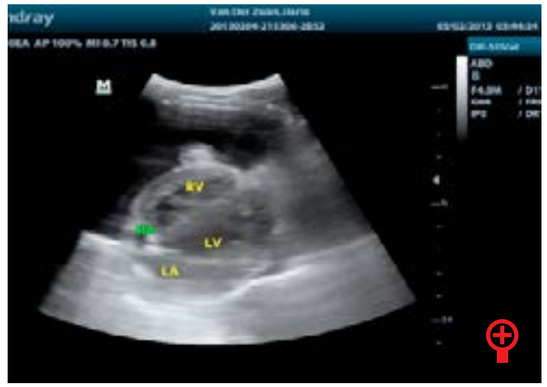

Figure 3

Back to Table of Contents

The right cranial thorax was clipped and prepped routinely, lignocaine infiltrated between the 4th and 5th ribs at the level of the costo-chondral junction. A 20g catheter advanced into the chest, the stylet withdrawn and an extension set with a 3-way tap connected sterilely. After two 60 ml syringes were filled with the effusion it became clear that it was frank blood rather that an exudate. The blood did not clot, confirming that the heart hadn't inadvertently been penetrated.

A quick cytological examination with DiffQuick® stain confirmed the lack of bacteria whether free or within white blood cells. The cell distribution was consistent with straight blood. The fluid's haematocrit was higher than her blood's at 38%. There were adequate platelets and evidence of a regenerative anaemia on blood cytology. There were no spherocytes or other signs of an immune-mediated destruction of red blood cells.

At this point we decided to auto-transfuse the effusion intravenously to give her an edge while ongoing diagnostics were initiated. A total of 120ml of effusion was given back to her over 30 minutes. During this process her temperature stabilised back to 37.5ºC, climbing to 39.0ºC when she was placed back in her cage with heat pads and on fluids at 1.5 maintenance rate.

Another case describes the same condition in two 6 month-old littermate Shetland Sheepdogs.[2] A cause was not established and only one of the two survived. The second was necropsied to confirm diagnosis. In these cases the haemorrhage was restricted to the thymus and mediastinum and radiographs had provided an ante-mortem presumptive diagnosis.

The presence of free blood in the chest with no history of trauma or rodenticide toxicity made us consider neoplasia as well as snake envenomation as differential diagnoses.

Due to financial constraints a snake venom detection test was not considered and we opted for a creatine kinase level. It came back at 473 U/L (10-200). Her urine was clear yellow. A snake bite seemed unlikely. An activated clotting time test of four minutes was inconclusive after a large amount of bleeding. PT and APTT tests were not performed, again for financial reasons.

Her status remained stable but critical for the next two hours. After further discussion with the owners they opted for euthanasia on financial grounds. Permission for a necropsy for diagnostic reasons was obtained.

The findings were of marked intrapleural haemorrhage that originated from a ruptured outpocketing of the cranial mediastinum, at the level of the heart. The mediastinal space was completely filled with a haemorrhagic mass (Figure 4). A sample was obtained and fixed in case of further queries and confirmed that it was a soft but solid mass rather than another fluid pocket. Histopathology was not done however.

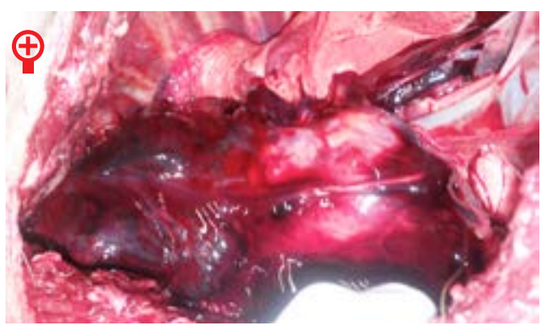

Figure 4

Discussion

The scientific literature describing similar presentations refer to cases of thymic haemorrhages in very young dogs from spontaneous or traumatic causes or associated with rodenticide toxicity.

One case describes a spontaneous haemorrhage of the involuting thymus in a 10 months old Dalmatian. Surgery was performed to remove a 10 by 15 cm haemorrhagic mediastinal mass that was confirmed on histopathology as a haematoma originating from the thymus.[1] The dog recovered fully after surgery.

Another case describes the same condition in two 6 month-old littermate Shetland Sheepdogs.[2] A cause was not established and only one of the two survived. The second was necropsied to confirm diagnosis. In these cases he haemorrhage was restricted to the thymus and mediastinum and radiographs had provided an ante-mortem presumptive diagnosis.

A 1987 study of twenty dogs with thymic haematomas found them caused in four cases by dissecting aneurysms of the aorta, and in seven by an overstretch of the neck causing trauma or even microtrauma. The other dogs had no identifiable pathogenesis.[3]

A 2002 retrospective study was done of 10 cases of thymic haematomas in young dogs (9-24 weeks old). It confirmed rodenticide toxicity in five cases. The thymic medulla was the primary site of haemorrhage and in severe cases normal lobular architecture was lost. The results stated: 'Lesions in confirmed cases of anticoagulant rodenticide toxicosis also are compatible with published descriptions of idiopathic and spontaneous thymic hemorrhage, but are inconsistent with normal thymic involution.' [4]

All these cases seemed to have been in a younger age group than Darla, as the dissecting aneurysms are not given a signalment. The lack of definitive histopathology is frustrating in this case as we must continue to surmise the aetiopathology of her condition. A thymic haemorrhage would be consistent with a solid mass. On the other hand trauma (such as a stretch to the neck) causing a mediastinal bleed that initially clotted would also be possible. The subsequent haemorrhage would have resulted from clotting factor consumption.

Editor's Note: It seems most likely that this dog did have bleeding into the mediastinum associated with involution of the thymus, although anticoagulant rodenticide cannot be excluded without determination of PTT and APTT. If these owners had a greater financial and emotional commitment, transfusion of fresh whole blood, vitamin K1 subcutaneously and possibly administration of tranexamic acids may have resulted in a favourable outcome.

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SULPHITE PRESERVATIVES IN PET MEATS

C&T NO. 5395

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Sulphite preservatives (e.g. sodium metabisulphite) in pet meats and pet food rolls causing thiamine deficiency and resultant brainstem haemorrhage has been characterised for at least 20 years.

When we first see a cat at Paddington Cat Hospital (and at every annual visit), we ask the simple question: 'What does your cat eat?' and we are amazed to find a significant number of cats still being fed pet meats. We explain the problem and recommend that these owners feed alternatives to their cats.

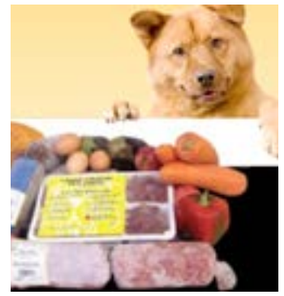
The pet food industry in Australia is self-regulated via the Pet Food Industry Association of Australia (PFIAA) which sets its own standards using a voluntary code of practice. A major deficiency of this system is the lack of any legal requirement for full and accurate labelling of the ingredients in pet food, i.e. listing ingredients (including preservatives) is optional.

One very clever client of ours was shocked that pet meats that specifically claim on the label 'no preservatives' may indeed contain such a dangerous ingredient.

She obtained a sulphite test from http://fedup.com.au/information/support/sulphite-tests (2 test strips came with each card) and obtained a reading of approximately 60-200 ppm. See the picture: in real life, the colour on the strip was somewhat between the two panels, whereas in the photo it seems to be closer to the second one.

These results were from: http://www.caninecountry.com.au/ feline roo meat. Their website prominently displays the words: 'Preservative Free'.

Low levels like this may not necessarily result in clinical signs, particularly if owners are feeding other foods also which may explain why we don't see more cases with neurological signs as a result of these preservatives. However, the levels should be zero (as labeled); extrapolating from the human experience, many individuals may experience adverse effects – skin rashes, gastrointestinal issues and so forth (e.g. many people who get headaches from drinking red wine get them because of the high sulphite content in the wine) and we can ensure NO clinical cases if we ensure our clients don't feed these foods to their cats or dogs.



Canine Country Pet Food (Images sourced from www.caninecountry.com.au/)

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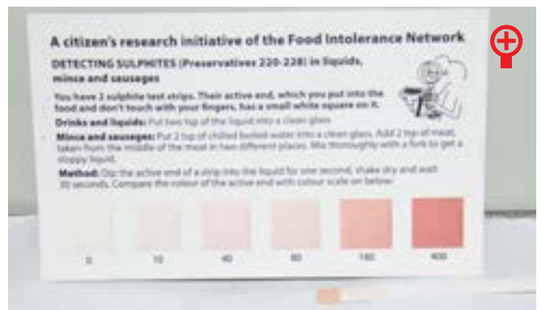

A LITTLE MIRACLE TO START THE YEAR

C&T NO. 5396

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Just thought I'd share an interesting case we've had over the last couple of weeks. 'Maddie' is a 13-month-old, speyed DSH (3.3 kg) that presented 3 weeks ago with a history of lethargy over the preceding weeks which had progressed to inappetance. No history of vomiting/diarrhoea/coughing. On physical exam she had white mucous membranes, tachycardia and murmur. No signs of jaundice. Bloods taken showed a PCV of 10, with no signs of regeneration. The white cell lines and platelets were within normal limits (WNL), biochem all WNL. Possible haemotropic Mycoplasma spp seen on blood film. No access to toxins, no history of medications.

We started her on doxycycline 5mg/kg BID and ran a PCR for M. haemofelis. Whilst awaiting PCR results, she presented 3 days later collapsed and dyspnoeic. PCV was 6% and still no sign of regeneration. We started her on supplemental oxygen and next day organised a blood transfusion (thank goodness she was type A, as was the only donor cat we had access to). PCV came up to 12% with transfusion.

PCR for haemotropic mycoplasmas came back negative the next day – dammit. Abdominal radiology and ultrasonography was unremarkable. We discussed FIV/FeLV testing and bone marrow biopsy with owners (up to date with vaccinations for both FeLV and FIV) but the owners declined any further investigation as the cat was looking much better since the transfusion.

In a last ditch effort I decided to trial high dose prednisolone (10mg BID) and cyclosporine (25mg BID) (on the basis of one report of successful treatment of pure red cell aplasia in a cat in Germany with this protocol – Mishke R. Cyclosporin a therapy in in a cat with pure red cell aplasia. Berl Munch Tierarztl Wochenschr 1998; Nov-Dec;111(11-12):432-7).
The owners were reluctant to use this treatment as 'the internet' says cyclosporine is bad. They waited until the cat started looking unwell again before actually starting.

We rechecked the haemogram and though the PCV was still only 10, the fantastic news was we finally had reticulocytes at 7.3%!

We hope that the owners continue with meds and that she'd recover fully, but part of me just couldn't believe that we were finally getting somewhere with her, and feeling very much like I was really able to use the skills from the course to better manage this case (blood transfusion, literature searches). Thanks guys.

Follow Up

A follow up visit and repeat haemogram ten days after the first appearance of reticulocytes had shown further improvement with an increase in the haematocrit to 22% and a reticulocyte count of 1.4%. At this point the cyclosporine dose was reduced to 25mg SID and the prednisolone dose kept at 5mg BID.

A repeat haemogram a further 10 days later finally had a normal haematocrit of 25% with a reticulocyte count of <0.1%. At this stage the owners felt that she was all better and despite repeated conversations on the seriousness and long term nature of her disease, didn't come back for their recheck appointments until approximately 3 months later, by which time the poor kitten was back in a miserable state of lethargy and collapse, again with a haematocrit of 10%.

They had continued the prednisolone but had discontinued the cyclosporine. They were advised to start the cyclosporine again, but instead preferred to pursue more natural therapies as promoted by the internet. A follow up phone call to the owners a couple of weeks later confirmed our fears that she had passed away, and they weren't forthcoming on any further details, so presumably the kitten died shortly after our last appointment with her.

A most frustrating end to what was initially a hugely rewarding case, and so sad to see such a beautiful cat die after such a long struggle to get her responding to treatment.

REPLY COURTESY OF:

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RCVS Recognised Specialist in Feline Medicine
European Veterinary Specialist in Internal Medicine
CVE Feline Distance Education Tutor
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Well done Sasha! We see quite a lot of cats with pure red cell aplasia that respond well to treatment so well done for thinking of this and treating for it. Some cats, even if they respond well, can take a couple of months to start regenerating well – so this is great to be seeing such a quick response.

I often don't start with both steroids and cyclosporin first off but I agree that you have made the right decision in this case because the cat's anaemia was severe and you can't afford to wait a long time for treatment to work. Many cats that take a while to respond will need, in the interim period, a 2nd and sometimes even a 3rd transfusion, but some owners will give up before you get to this stage and sounds like you only had one more chance with these owners so I think you have made the right decision with treatment here so again, well done for doing that.

A couple of words of caution – there isn't yet much evidence – but it appears that the side effects of cyclosporin in terms of immunosuppression and infectious diseases is much more likely if they are on treatment with steroids concurrently. It is a balancing act and primarily you need to treat the cat's anaemia. But once she is responding well, I would advise weaning off the steroids more quickly than you would do if she were on steroid monotherapy, obviously while monitoring.

Secondly, most importantly at the moment – this is a very high dose of cyclosporin – Atopica for cats has just been licensed and launched in Australia – but it is a liquid which allows more accurate dosing. But importantly, in the safety and efficacy trials that Novartis did in the licensing process they demonstrated that 7mg/kg/day was efficacious. This was for cats with atopy, not other immunomediated diseases, but I would use the same dose. Above this dose, the risk of side effects in terms of infectious diseases such as toxoplasmosis will increase.

I have certainly used cyclosporin at higher doses in the past, albeit not often, but never above 5mg/kg BID so this is really quite a high dose (a doses used to prevent kidney transplant rejection). My advice would be to reduce your dose to 25mg ONCE daily of cyclosporin.

With regard to the corticosteroids, I would normally for an immunosuppressive dose just use prednisolone at 1mg/kg BID. In this case I think it would be reasonable to use both to start with but I would want to reduce the prednisolone as soon as possible if things were going well. Normally I would reduce by about 25% every month but if you are using concurrent cyclosporin I would reduce by 25% every week if she continues to be improving during this time. Continue if she stops regenerating and PCV stops increasing.

This would be my approach but other people may have a slightly different approach; there isn't necessarily a right or wrong here, but this is what I would try and do.

If money and cyclosporin are an issue, you could do it the other way around and keep on high dose prednisolone but wean off cyclosporin earlier. The main thing is close monitoring and changing treatments depending on response rather than sticking to a rigid protocol.

Please keep us posted! Will keep fingers crossed for her! I would recheck PCV in 7-10 days. Well done again for getting so far with her.

FROM THE DE FILES
WHAT'S YOUR DIAGNOSIS?

C&T NO. 5397

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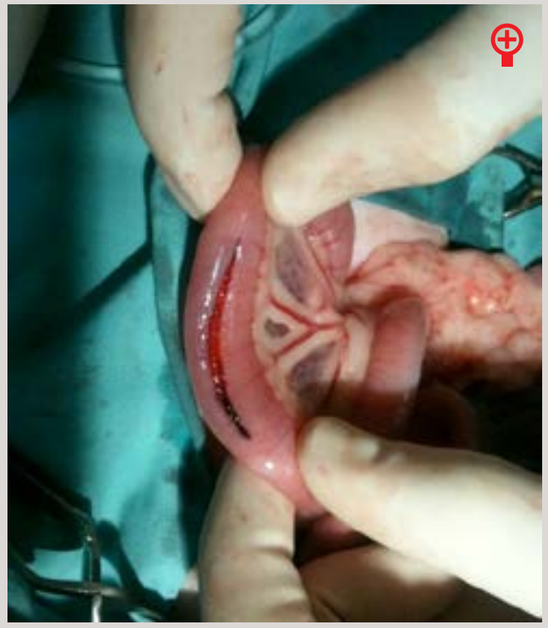

Figure 1. 'Bella's' bowel.

Email your answer to elisabeth.churchward@sydney.edu.au

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Back to Table of Contents

WINNER!

JUST NOT CRICKET!
CASE OF AN UNUSUAL FOREIGN BODY

C&T NO. 5398

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Case history

'Bear', a 6-year-old spayed female domestic shorthair cat presented with a weepy left eye and third eyelid prolapse. A presumptive diagnosis of conjunctivitis was made and the cat was sent home on antibiotic eye ointment. One month later the cat presented to me for the eye not healing. The owner had noticed that she appeared to be having trouble closing her eye and that the eye seemed bulgier. There was also still ocular discharge.

Upon examination, there was left sided exopthalmos with third eyelid prolapse (see Figure 1). The cornea and iris appeared normal but there was episcleritis occurring. Upon retropulsing the eye, discharge started appearing in the mouth that wasn't there when the mouth was examined prior to examining the eye. A presumptive diagnosis of retrobulbar abscess was made and the cat was admitted for examination under anesthesia.

Bear was anaesthetized with meditomidine, ketamine and butorphanol intramuscularly. After retropulsing the eye again, a black object appeared from a hole in the soft palate medial to the last molar (see Figure 2). The object was carefully removed and found to be the leg of a black field cricket *Teleogryllus commodus* (see Figure 3). The hole was then opened up using forceps and the retrobulbar space gently flushed. Bear was then sent home on meloxicam (Metacam) and amoxicillin plus clavulonic acid (Clavulox).

Discussion

Clinical signs are listed in Table 1.

Table 1: Clinical signs of retrobulbar disease.[1]
Unilateral exopthalmos
Third eyelid protrusion
Conjunctival hyperaemia
Ocular discharge - serous or mucopurulent
Episcleral vessel congestion
Pain on palpation of periorbital region
Pain on mouth opening
Swelling of oral mucosa posterior to last molar.

Retrobulbar disease can include retrobulbar abscesses and/or cellulitis, which may be due to trauma, foreign bodies, abscessed caudal upper molars, dacyrocystorhinitis, aberrant migration of *Dirofilaria immitis* and *Ancylostoma* sp. larva, fungal pyogranulomas, sialodenitis, zygomatic salivary gland abscesses, granulomatous meningoencephalitis, and extension of paranasal sinus or nasal cavity infections, orbital neoplasia, and orbital pseudotumors.[2]

Ultrasound placed onto the cornea can help differentiate different causes with malignancies appearing as highly echogenic masses, mostly in the caudal part of the globe and, with inflammatory disease, the most consistent abnormality is blunting of the posterior aspect of the globe.

Treatment of inflammatory lesions consists of incision and establishing drainage and systemic antibiotics with expected resolution within 3-10 days.[3]

Bear was showing most of the clinical signs in table 1, even at first visit when it was assumed she was suffering from conjunctivitis.

If retropulsion had been performed at this visit, it may have given a clue to something occurring behind the globe. Retropulsion is an easy quick test that should be performed in all cases of suspected retrobulbar disease and, in this case, gave us a diagnosis as the pressure caused the foreign body to be milked out of the retrobulbar space. We assume that the leg penetrated the soft palate while Bear was trying to eat a live cricket that was struggling in her mouth and then broke off, leaving itself embedded in the retrobulbar space.

See enlarged images in eBook

Figure 1: Exopthalmous of the left eye as viewed from above.

Figure 2. Visible foreign body exiting retrobulbar space from hole in soft palate

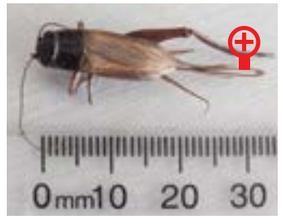

Figure 3. Teleogryllus commodus – *Field cricket.*

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FROM THE DE FILES
WINNER!

URETHRAL OBSTRUCTION

C&T NO. 5399

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Introduction

Urethral obstruction is a common presenting problem in male cats in general practice,[11] often occurring as a result of urethral plugs, urolithiasis, and strictures. In many cases the cause of the obstruction is unknown (idiopathic urethral obstruction) and probably involves some degree of urethral spasm. Less common causes include neoplasia and phimosis.[4] The following case study documents a traumatic urethral rupture and subsequent obstruction and its treatment.

Case Report

A 3-year-old desexed male domestic shorthair cat (5kg) was presented afterhours after being found in pain and with blood staining around the hind end. His owners had heard a cat fight the previous night but had seen no evidence of injuries at the time. Upon returning home from work in the evening they had found that the cat did not want to move. He was very grumpy when touched and bloody fluid was evident on the back legs. On examination, the cat was alert with pink mucous membranes, capillary refill time <2 seconds, and rectal temperature 39.2°C. He was able to walk on all 4 legs though was favouring the left hind leg and there was shredding of the nails on the right front foot. He was difficult to examine fully around the hind end and resented his tail being lifted. He was also sore on palpation of the pelvis. There was watery blood stained fluid which had the smell of urine, dripping from the preputial area and on the paper lining of the carry cage. A golf ball sized bladder was palpable in the caudal abdomen and was non-painful and soft. After examination the cat squatted on the floor and passed normal faeces.

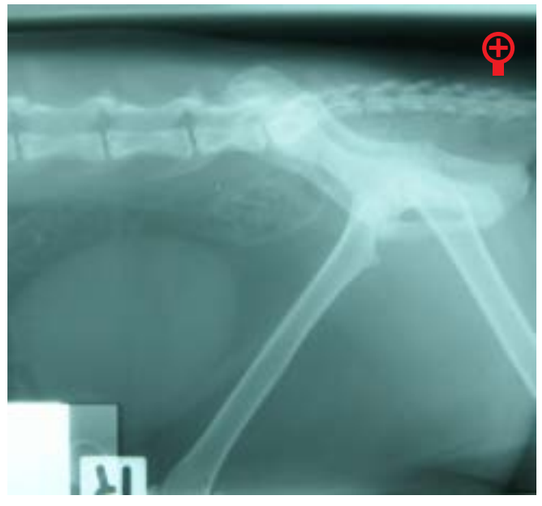

Fig 1. Plain lateral radiograph of caudal abdomen/pelvis

After the initial examination – the main problems identified were pelvic/perineal pain and haematuria. Main differentials included trauma (hit by car, animal attack), and other causes of haematuria (UTI, FIC, coagulopathy etc). The cat was admitted and given pain relief (1mg butorphanol) and antibiotics (43.75mg amoxycillin/clavulanic acid injection). Next morning the cat was re-examined and found to have a fluidy swelling on the inside of the left thigh and was still resentful of examination. No urine had been passed in the litter tray overnight. He was sedated for further examination and found to have deep puncture wounds from a suspected cat fight to the perineum between the anus and the prepuce and smaller punctures extending down the caudal left hind leg. There was extensive swelling and bruising of the inner left hind leg. The bladder was still palpable within the abdomen. Plain radiographs were taken which showed a large bladder silhouette consistent with findings on abdominal palpation and no pelvic or hindlimb fractures (Fig 1&2). Attempts to catheterise the bladder were unsuccessful with the catheter being able to be passed only approximately 2.5-3cm before encountering resistance. A retrograde urethrogram was performed with Urograffin (Sodium Amidotrizoate / Meglumine Amidotrizoate) to assess the integrity of the urethra. There was leakage of contrast material into the surrounding tissues consistent with a tear in the urethra (Fig 3&4).

Problems identified at this stage were:

1. Urethral rupture secondary to trauma (cat fight wound)
2. Cellulitis from leakage of urine into subcutaneous tissues and injury from initial cat fight

Back to Table of Contents

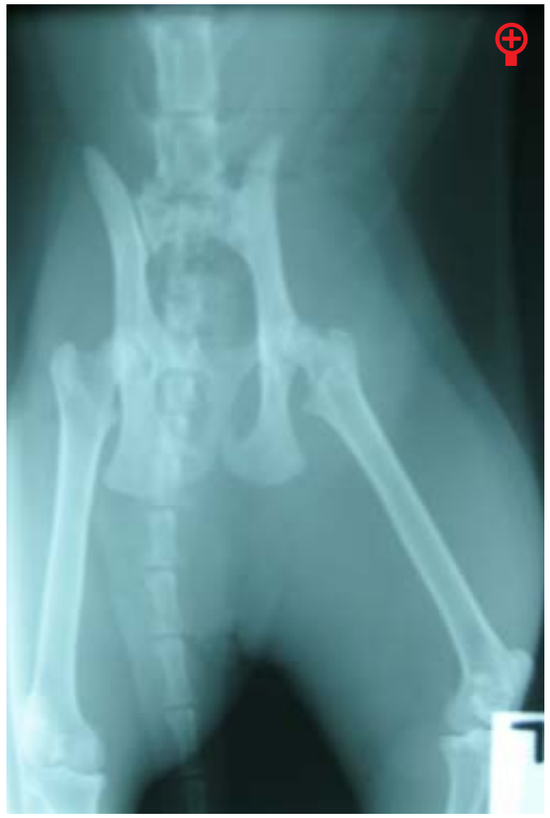

Fig 2. Plain ventrodorsal radiograph of caudal abdomen/pelvis

Repair of urethral rupture is by either primary repair if possible or if enough intact urethral tissue is present, then by second intention healing after placement of a urinary catheter (as a stent). The option of referral for specialist treatment and surgical repair was declined by the cat's owner.

The cat was placed on intravenous fluids and given a general anaesthetic. The perineum and prepuce was incised and explored – there was extensive swelling and bruising to the area making identification of the intrapelvic urethra past the tear difficult. Rather than potentially causing more damage to what remained of the urethra, a decision was made to perform a laparotomy and cystotomy and pass a guiding catheter normograde through the urethra to identify the pelvic component of the damaged urethra. A soft catheter was then placed into the bladder following the guide. Urethral tissue was present longitudinally with a 5mm long deficit present on the dorsal aspect. Not enough urethral tissue remained to achieve a primary repair so secondary healing was attempted by leaving in place the catheter to act as a stent for the regrowth of urethral epithelium. The area was flushed and closed with a double layer closure. The cystotomy and laparotomy were closed routinely. The cellulitis on the left hindleg was opened and flushed and drain tubes placed prior to closure. A closed urinary collection system was set up and analgesia (1.25mg meloxicam q24h x 3d) and antibiotic cover (50mg amoxycillin/clavulanic acid q12h) was continued over the subsequent week while the fight wounds were healing. The urine sediment was evaluated regularly for evidence of inflammation/infection, though urine culture was not performed.

Then, due to the size of the urethral tear, the urinary catheter was left in place for 3 weeks before removal (the cat was hospitalised during this time) and hospitalisation was continued for 2 days after this to ensure the cat was able to urinate adequately. The cat was discharged with instructions to closely monitor urination over the next few weeks.

The cat represented 3 days later – he had been going well at home for the first 2 days, passing small amounts of urine frequently and very happy, but in the past 24 hours he had progressed from being grumpy and urinating in inappropriate places, to vomiting (once) and not eating and no urine passed despite straining to urinate over the previous 4 hours. On examination he had a full, firm, painful bladder consistent with urinary obstruction. He was given pain relief (1.25mg meloxicam and 1mg butorphanol) and 1.5mg diazepam to help relax the smooth muscle in case of urethral spasm but there was no improvement over the following 60-90 minutes. He was anaesthetised and catheterisation was attempted but an

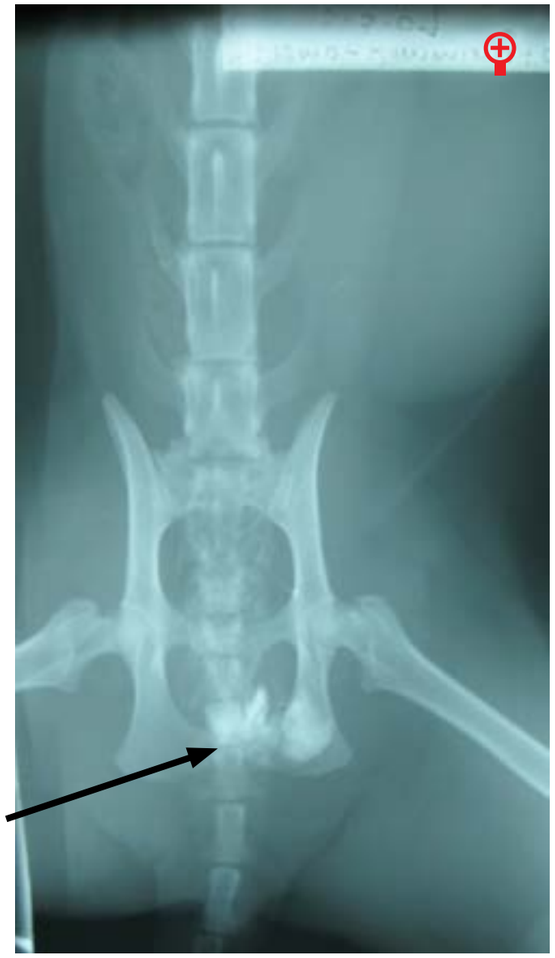

Fig 3. Positive contrast urethrogram (ventrodorsal view) showing leakage of contrast material into surrounding tissues (arrow)

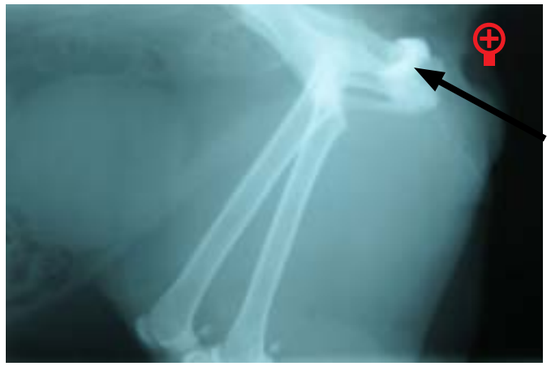

Fig 4 . Positive contrast urethrogram (lateral view) showing leakage of contrast material into surrounding tissues (arrow)

obstruction was encountered at the level of the original injury. It was suspected that either a stricture had formed at the site of the original injury or that the wound had not healed adequately and there was wound breakdown. Urine was removed from the bladder via cystocentesis to relieve some of the pressure in the bladder while treatment options were discussed with the cat's owner. Options at this stage included referral for specialist treatment, urinary diversion surgery or euthanasia. Referral for specialist treatment was once again declined. Perineal urethrostomy was not an option due to position of the initial injury and extensive scar tissue in the area. Pre-pubic urethrostomy was chosen as the most appropriate diversion surgery in this case. Potential complications of the surgery include wound breakdown, urinary incontinence, urine scalding, abdominal muscle pressure causing obstruction and increased risk of urinary tract infections.

The cat's owner consented to the procedure and the cat was anaesthetised. A caudal ventral midline laparotomy was performed and the urine in the bladder was drained via cystocentesis. The urethra was carefully released from any soft tissue attachments and as much length of the abdominal urethra was retained as possible before transection from the pelvic urethra (the remaining pelvic urethra was tied off with 4/0 Vicryl). The proximal section of urethra was directed through the caudal abdominal muscles to the skin whilst maintaining as direct route as possible to avoid potential kinking of the urethra. The end of the urethra was splayed and sutured to the skin with 4/0 Prolene. A catheter was able to be passed easily into the bladder at this stage. The surgery site was flushed, the abdominal muscles and subcutaneous tissues closed with 2/0 Vicryl and the skin closed with Prolene. The cat was maintained on intravenous fluids (0.9% saline) until he was eating normally, pain relief (1mg butorphanol) and antibiotics (75mg cephalexin) post-operatively and an Elizabethan collar was used to prevent self-trauma.

The day following the surgery, minimal urine was being produced despite a full bladder and attempts to catheterise the stoma were unsuccessful. The cat was re-anaesthetised and a stitch cranial to the stoma in the abdominal muscles was removed and the subcutaneous sutures cranial to the urethra were removed. A catheter was able to be passed easily again after this. Following the corrective procedure the cat was able to pass urine quite easily and was continent. Anti-inflammatories (1.25mg meloxicam q24h) and antibiotics (75mg cephalexin q12h) were continued whilst the cat was in hospital for the subsequent 3 days. He was sent home on antibiotics for another 5 days (oral meloxicam for feline use was not commercially available at the time of the surgery).

The wound was rechecked a week after the surgery – the cat had urinated on the bed that morning and had some dribbling of urine after coming out of the litter tray. There was some irritation around the surgery site and it was suspected that the cat was rubbing the area with the edge of the Elizabethan collar. A larger collar was applied and Vaseline applied to the skin around the surgery site to help reduce urine scalding. At removal of stitches a week later, the cat was continent and all irritation had settled down.

It has been several years since the surgery at the time of writing and despite the increased risk of infection, the cat has had only one incident of an ascending bacterial urinary tract infection in that time. He is now kept as an indoor cat to avoid any further altercations with outside cats.

Discussion

Urethral rupture is most commonly a result of trauma (vehicular accidents, bite wounds, penetrating injuries), urethral calculi or iatrogenic (poor catheter placement),[3,6] and should be suspected whenever there is pelvic trauma, excessive swelling and bruising around the perineal region or inability to pass a urinary catheter. The best method of identifying a urethral rupture is by positive contrast retrograde urethrogram.[8] Plain radiographs should be taken prior to using contrast media to evaluate the area for fractures and other injuries, especially following traumatic episodes.

Leakage of urine from the damaged urethra results in an inflammatory reaction that can progress to tissue necrosis.8 Management of urethral rupture requires initial patient stabilisation, correction of any metabolic disturbances and provision of urinary diversion.[6] Urinary diversion is a priority to reduce urine leakage either by placement of indwelling urinary catheter or via surgical placement of a cystostomy tube.[8] It is recommended that baseline haematology and biochemistry be performed so that any abnormalities can be corrected prior to anaesthesia. As the cat was quite bright at initial presentation, was still eating and drinking and in-house pathology was not available at the time (24 hour turn around time for outside lab), routine blood tests were not performed in this case but certainly would have been helpful – in particular with the second surgery where the cat had become more unwell.

Partial ruptures of the urethra may heal by second intention once urinary diversion has been achieved and larger defects can also heal if an intact strip of urethral mucosa remains[2,9]. Primary repair is challenging due to the small size of the feline urethra and is associated with the potential complications of dehiscence and stricture[6]. A transurethral catheter should be maintained for 3 weeks but strictures can be a frequent complication[8] – possibly due to the presence of the catheter itself.

The risk of a urinary tract infection increases the longer the urethral catheter remains in place,[10] even when using a closed collection system as in this case. Giving antibiotics will not necessarily prevent infection and may actually predispose to the acquisition of resistant bacteria.[1] Antibiotics were used to treat the initial infection from the cat fight wounds and urine leakage cellulitis in this case but were stopped once these wounds had

Back to Table of Contents

healed. Ideally, the urine should be cultured regularly or at the very least following catheter removal.[7] Using aseptic technique while maintaining a closed collection system and minimising the length of time the catheter is in place also helps reduce the risk of infection. Urine sediment was evaluated regularly in this case but urine culture was not utilised – this may have improved the management of this case.

The development of a stricture or wound breakdown following attempts at urethral repair is quite common. Strictures may occur days to weeks after the procedure and present in the same manner as other causes of urethral obstruction – i.e. dysuria, pain, unproductive attempts at urination, depression, the presence of a hard full bladder on abdominal palpation and inability to pass a urinary catheter. The presence and position of the stricture can be confirmed by performing a positive contrast retrograde urethrogram (same as for urethral rupture).

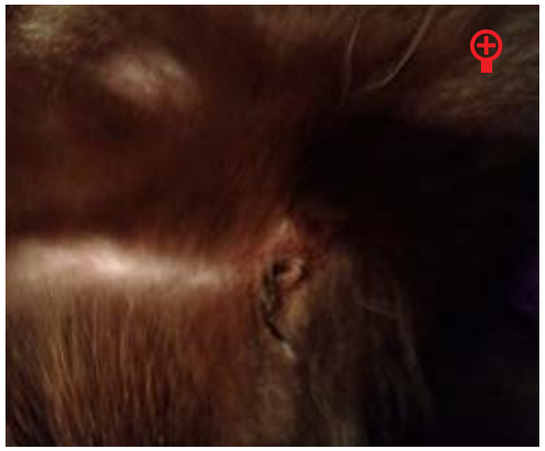

Figure 5. Appearance of the prepubic urethrostomy stoma once fully healed

Surgical options for urethral strictures include cystostomy, cystostomy combined with urethrostomy, perineal urethrostomy, prepubic urethrostomy and transpelvic urethrostomy.[11] Penile trauma due to catheterisation is the main indication for urethrostomy in the cat[3] – repeated catheterisation predisposes the male cat to urethral inflammation, trauma and stricture. Perineal urethrostomy is indicated for strictures within the penile urethra.[3] Intrapelvic strictures, or those associated with trauma and scar tissue formation require a prepubic urethrostomy (as in this case). Transpelvic urethrostomy is associated with a higher complication rate and so is not often utilised. Potential complications of urethrostomy include wound dehiscence, urine scalding, urinary incontinence, stricture formation and increased risk of urinary tract infection due to the shortened urethral length. Bacterial urinary tract infection is the most frequent post-operative complication seen.[3] Due to the high complication rate, urethrostomy is considered a salvage procedure and should only be performed when other treatment options have failed and the potential complications need to be fully explained to the patient's owners.

Recently, self expanding metallic urethral stents have been used for urethral strictures in cats with some success.[5] This may become a more readily available treatment option in the future.

SUMMARY

A. Protocol for urethral trauma

- Stabilise patient – correct any underlying cardiovascular and metabolic abnormalities. Assess for other injuries (especially if trauma case) and treat as appropriate
- Baseline haematology, biochemistry, urinalysis
- Plain radiographs, contrast radiographs of the urinary system to check for position of tears, urine leakage
- Provide urinary diversion to prevent secondary complications of tissue necrosis, uroabdomen etc.
- Monitor diversion method for signs of complications e.g. regularly perform urine culture, and use aseptic technique.

B. Protocol for urethral strictures

- Stabilise patient – correct any underlying cardiovascular and metabolic abnormalities especially if obstruction has occurred.
- Baseline haematology, biochemistry, urinalysis
- Relieve obstruction/bladder pressure – catheterisation, cystocentesis if catheter unable to be passed
- Plain radiographs, contrast radiographs of the urinary system to check for position of stricture
- Plan any surgeries based upon position of stricture, surgeon's abilities – consider referral for surgery, ensure client fully aware of potential complications and expectations of surgery.

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WINNER!

TWO DIET-ASSOCIATED DISEASES IN THE ONE DOG

C&T NO. 5400

Sue Foster

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Signalment: 'Bambi', ShihTzu/ Malt x 5.7 y.o. FN

History:

Periods of stupor noticed by the owner over a week, 3-4 days increasing lethargy and inappetence, yellow pasty diarrhoea for 24 hours and possible polydipsia. The dog had had episodes of shaking and 'collapse' and would appear to 'fall asleep while standing'.

PE abnormalities: Vague and disorientated mentation with one mild short duration seizure during examination; vertical nystagmus, anisocoria, slight head tilt to the left, weak, ataxic, trembling/ twitching slightly, decreased hindlimb reflexes, slightly decreased forelimb reflexes.

Diet:

- Woolworths Select Dog Food Chilled Beef & Vegetable Roll
- VIP Dog Food Chilled Roll Gourmet Chicken
- JerHigh Chicken Sampler Treats/jerky (Thailand)
- Pet mince from the local butcher (not known whether preservatives added)
- Supplemented with chicken breast fillet cooked and mixed with vegetables

Haematology: normal

Biochemistry: normal apart from slightly decreased potassium (3.2 mmol/L)

UA: 4+ glucose (blood glucose 8.7 mmol/L), USG 1.008

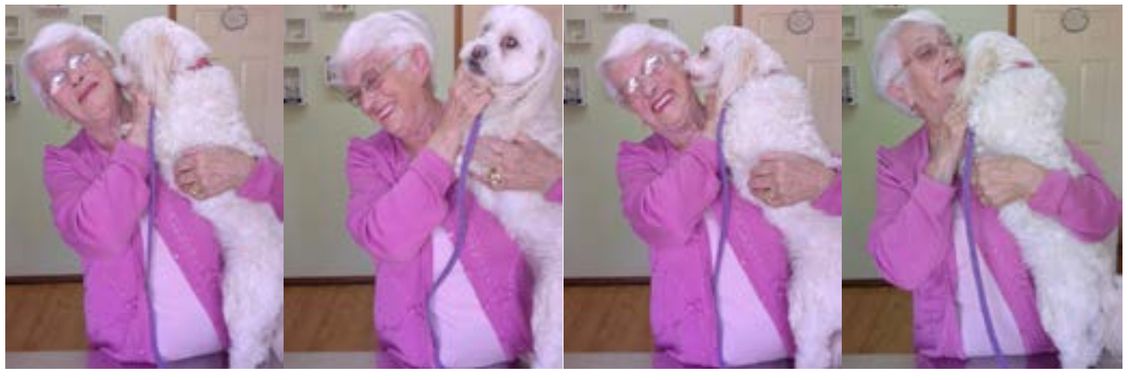
Urine cytology normal and no growth on bacterial culture. Urinary fractional excretion of electrolytes was normal.

Prescription: IV fluids with added KCl

Outcome after 36 hours: Bambi started eating small amounts. She improved slightly in demeanour and would try to wag her tail, but was not quite alert and occasionally appeared to have periods of stupor and trembling.

My suggested diagnosis: Likely acquired proximal renal tubulopathy to account for gastrointestinal signs, polydipsia and inappropriate glucosuria. Neurologic signs didn't really fit but the dog seemed to be responding to IV fluids so I ignored them (bad mistake…if using pattern recognition, then the pattern has to be right!).

Progress: Three days after admission, she started to deteriorate neurologically with circling to the left, falling to



Happy reunion...

Back to Table of Contents

the left and, occasionally, falling to the right also. Over the next 24 hours this progressed to anorexia, depressed mentation, lateral recumbency, inability to stand and knuckling on all 4 limbs when held in standing. The owners had limited funds for further investigation so the veterinarian involved provided a great deal of the treatment for free and Vetnostics diagnostic laboratory provided free urine and serum electrolytes for the fractional excretion of electrolytes. Finally, I thought about the neuro signs and the diet: processed dog roll and pet mince both previously having been reported to result in thiamine deficiency in Australia due to added sulphites as preservatives. No thiamine injection was available but a large dose of B complex was given to provide 40 mg thiamine whilst the veterinarian sourced oral 100 mg thiamine tablets from the health shop/pharmacist.

Outcome: Glucosuria and neurological signs resolved, normal dog and delighted owners (lovely old couple). There was a rapid response to thiamine (some mild improvement noted within 4-5h) and steady improvement to near normal over 4 days (on 100 mg thiamine PO SID), when the dog was discharged.

FOLLOW UP SULPHITE ANALYSIS

VIP (the manufacturers of both dog rolls) responded rapidly to our enquiry and informed us that there were no sulphites in these products. Masterpet also responded promptly to our questions. Dr Joe Katsikaros however, paid to have both dog rolls, the JerHigh treats and the pet mince analysed for sulphite content. The culprit for the added sulphite was the mince from the butcher!

FOLLOW UP ACQUIRED PROXIMAL TUBULOPATHY

There is little doubt that one of the components of this dog's diet was responsible for the acquired renal tubulopathy (acquired Fanconi syndrome) as the pattern for this was similar to that in the cases reported by Thompson et al (2013). The tubulopathy and glucosuria reversed with withdrawal of these food sources and has not recurred. The case was logged with AVA PetFAST, a site for reporting potential adverse reactions to pet foods or treats.

MORALS OF THE STORY

1. That this dog had not 1 diet-related problem but 2 serves as a reminder to check and analyse the diet carefully always.
2. Do a careful problem oriented approach and don't dismiss the signs that don't fit as is often done, in my experience, with inappropriate glucosuria but in this case vice versa, with the inappropriate glucosuria detracting from the even more critical issue of thiamine deficiency!

FINAL NOTE

The owners of this dog, elderly and both recently out of hospital, are determined to pay back as they can, but there is no doubt that their dedicated and compassionate veterinarian, Dr Joe Katsikaros, saved this dog's life by the care she provided at her own cost. That she also went on to identify the cause of the thiamine deficiency has provided us with a

reminder that not all food sourced from butchers is safe and that 'pet meat' cannot be regarded as being safe in dogs (or cats) regardless of its source.

This case was logged with PetFAST, as all suspected adverse food and treat reactions should be. The pet food companies involved were quick to respond to the case and help with investigations. As acquired tubulopathies are continuing to occur in Australia, we do request that any suspected cases are logged on PetFAST. I am always happy to take enquiries on the issue (as are Drs Linda Fleeman and Mary Thompson).

Reference

Thompson MF, Fleeman LM, Kessell AE, Steenhard LA, Foster SF. Acquired proximal renal tubulopathy in dogs exposed to a common dried chicken treat: retrospective study of 108 cases (2007-2009). *Aust Vet J* 2013;91:368-371

Comment from Linda Fleeman

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Representative of the AVA on pet food issues

This case highlights several issues relating to safety of the food that we feed our pets in Australia.

The first is that veterinarians are encouraged to the use the PetFAST system (http://www.ava.com.au/petfast) for reporting all suspected adverse events relating to pet food, pet treats, and pet meat. This improves communication between veterinarians and manufacturers and facilitates more rapid identification of outbreaks of food-related adverse events in Australian dogs or cats. The AVA's PetFAST website also provides a detailed checklist for veterinarians dealing with a suspected adverse event, with information on relevant history taking and sample storage.

The second issue here is that 'pet meat' is not subject to either the regulation relating to meat for human consumption or to the regulation relating to pet food. 'Pet meat' is raw or unprocessed meat and comprises a substantial proportion of the pet food market in Australia. It comprises meat from various sources including kangaroo, beef, lamb, chicken etc, and is sold throughout this country in supermarkets, pet stores, pet barns, and in both retail and wholesale butcher shops. Some products are packaged with informative labels that include ingredient lists and statements about preservative content. Other pet meats are sold from bulk refrigerated tubs with no information available to the consumer on source of the meat(s) or the inclusion of additives. A common error among consumers is to assume that the absence of labelling information regarding preservatives means that the product is 'preservative-free'. In contrast, it is legal and often quite routine to add sulphite preservatives to pet meat to extend its shelf life without informing the consumer. One reason why sulphite preservatives are not permitted for meat intended for human consumption is that they impart a red colour to the meat and thus give a false impression of freshness.

It is important to recognise that 'pet meat' is specifically excluded from the Australian Standard for the Manufacturing and Marketing of Pet Food. The definition of 'Pet Food' in the Australian Standard is, 'Food for dogs or cats including all types of dry, semi-moist, retorted, pasteurized and other food manufactured for consumption by domesticated dogs or cats but excluding pet meat'. There are historical reasons for the distinction between 'pet food' and 'pet meat' in Australia which have resulted in different regulation for each. Regulation relating to 'pet meat' was first developed in the 1970s to prevent substitution and contamination of meat for human consumption. The regulation varies among the States and Territories. Furthermore, there is currently no industry body representing pet meat producers.

The current regulation of the 'pet meat' industry does not adequately protect dogs and cats consuming pet meat products from adverse health events such as thiamine deficiency caused by consumption of sulphite preservatives. Therefore there is requirement for improved education of, and communication with, pet meat producers about the dangers of using sulphite preservatives in pet meat. These issues could be addressed by improved regulation of the pet meat industry and one solution is that pet meat producers be encouraged to become members of the Pet Food Industry Association of Australia (PFIAA).

Shirley and Douglas were delighted to be reunited with 'Bambi'

CELEBRATING OUR VETS

The CVE is a membership organisation and we are keen to celebrate vets in the CVE community, particularly long-standing supporters such as Joeanne, who have contributed financially to the CVE/PGF for many years to support our work.

As Sue notes in this article, there is no doubt that JoeAnne saved this dog's life by the care she provided at her own cost. She also paid for the analysis which identified the butcher's mince as the culprit and her efforts have allowed us to publish this article to share with members/readers.

The CVE is awarding Joeanne a CVE course or seminar or her choice for her selfless and long-standing dedication to her patients and their owners.

Back to Table of Contents

REPLY TO INVITED COMMENT ON C&T NO. 5332 CANINE BEHAVIOUR – HAVE WE GOT IT RIGHT?

Grahame Baker

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C&T NO. 5401

Rollover or download these articles in the eBook

CANINE BEHAVIOUR – HAVE WE GOT IT RIGHT, C&T 5332 by David Bligh, Issue 272, Sept 2013

INVITED COMMENT ON THE ABOVE ARTICLE, Kersti Seksel, pg 43, Issue 273, Dec 2013

REPLY TO THE INVITED COMMENT, C&T 5386 by David Bligh, Issue 274, Mar 2014

This was my reply to the news that a colleague was moderated from the Behaviour special interest group for forwarding an earlier post of mine, so I decided to send it to the C&T forum.

Bad move, we'll both be excommunicated! You have to be PC these days or get moderated.

The rat bag dogs (and kids) are the ones with the most potential, initiative and drive, if you pull them into line at the start. Timid dogs need to be taught confidence not praised for lack of it. Throwing any on the scrap heap is unforgivable. Dogs are receptive to instruction; the limitation is usually the human's ability to communicate. Guidance has to be done within the boundaries of friendship and affection. All my dogs have been quiet, obedient and gentle but started as all sorts – ex pig dogs, euthanasia cases for problem behaviour, unsocialised timid dogs. If I asked them to behave, they did. If I praised them for growling at a stranger they would have probably have knocked them down and asked what I wanted next…

I checked out an alarm at the hospital once, walked the dark back lane with the truck headlights behind me and a dog either side. One dog disappeared which was totally out of character but I kept going with the other one. When I walked back behind the truck lights dear old 'Emily' was asking two police to wait while I sorted out the problem. She had great faith in my problem sorting-out ability but she didn't like people walking up behind me in the dark. Praise her? Her party trick in play was to nip the skin over the femoral artery. I asked her to come behind.

Anyway, it's a punishable offence to encourage a dog to bite. (Re owners praising dogs as they bite the Vet - do you praise kids for playing in the traffic?)

Figure 1. Smokey (forefront) pictured with his daughter Becky – 1975

I treated a 40 kg pig hunting dog years ago, bull terrier cattle dog + other, the owner used to put him on the table where he sat growling at me every time, looking at me sideways with his little piggy eyes. I trusted the owner but not 'Smokey'. I asked if Smokey would bite and the owner always said no, then in a well-choreographed performance he would backhand Smokey's ear but Smokey, without looking, always twisted his head slightly just enough to avoid contact. The growl reduced to a low grumble of complaint but I could do anything to Smokey without fear.

When Smokey was dying, no longer up to pig hunting or disciplining the pack, they left Smokey with me for some peace before he died, as Smokey had always regarded the hospital as a restful place. Smokey lived another 13 years with the more gentle lifestyle; I got to know him well, loved him dearly. A good watch dog but obedient, and if I praised him for growling at a stranger he would have knocked them down without question after he gave them a chance to back off. (He was always fair).

It wasn't my training but his old owners; men were fair game if Smokey thought they were out of line, women weren't a threat and children were to be protected no matter what. Give him a baby possum and ask him to look after it and he would sit through the consults with it on his head and warn off other dogs.

Dogs and kids need positive guidance not mindless praise for whatever random thought might be rattling around their brain. Telling people to praise fear behaviour is incorrect advice approaching negligence.

We need to watch out for the politically correct brigade.

REPLY TO INVITED COMMENT ON DAVID BLIGH'S C&T NO. 5386

Aine Seavers

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C&T NO. 5402

Seven Roads to Rome, six ways to castrate a calf, five ways for stifle surgery but only ONE way to train a dog – Why?

This new drive to reward biting dogs with food treats is not a potential nightmare – it is an actual current day horror. Many experienced dog trainers – lay and veterinary alike – are aghast at these pups coming from vet and pet shop puppy schools where nurses/trainers rewarded biting dogs with treats. I didn't believe it the first time I heard it but other vets are now hearing the same.

This new training 'trick' is a violation of common sense and unfair to the dog, to its family and to their surrounding community. I have no idea how it started out as a concept but given how flawed humans are in putting any message into practice, perhaps the 'concept' is actually being misunderstood and hence misapplied. If so, then ALL people enrolled in these courses need an immediate email from the originator of the concept to re-educate the trainers in the technique.

Training should be like all aspects of veterinary medicine – customised for the particular patient, its setting and the animal's purpose in life, all done with common sense and avoidance of cruelty. And that includes avoiding cruelly training a dog to get a positive reward when it bites!

We have a plethora of proven training techniques, be it gentle whispering/calming strokes/short verbal instant reprimands and equally immediate praise/gentle but firm restraints/food rewards but all have to be applied sensibly to avoid confusing the animal. We are losing the plot with some of the crazy new ideas coming into behaviour training, unfettered by commonsense, relevant experience or by an acknowledgement that dogs are not shrunken little humans with hairy coats.

MANAGEMENT OF DIABETIC DOGS & CATS REQUIRING ELECTIVE GENERAL ANAESTHESIA

C&T NO. 5403

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DOGS REQUIRING ELECTIVE GENERAL ANAESTHESIA

1. Half the usual dose of insulin should be administered in the morning when food is withheld.
2. Whenever possible, schedule general anaesthesia of diabetic dogs early in the day so that they can be recovered and discharged to the home environment before the next insulin injection and meal is due.
3. Intravenous administration of an insulin constant rate infusion is recommended while the dog is hospitalised with the goal of maintaining blood glucose concentration at 6-15 mmol/L. Blood glucose concentration should be monitored every 1-2 hours and intravenous fluids continued until the dog is discharged from hospital.
   (a) If the blood glucose concentration is 6-15 mmol/L, intravenous infusion of insulin at 25 mU/kg/hr (0.5 mL/kg/hr of 50 mU/mL solution (25 U insulin in 500 mL saline) with an infusion of 2.5% dextrose in 0.45% saline with 20-40 mmol/L potassium at 6 mL/kg/hr is recommended.
   (b) If the blood glucose concentration is >15 mmol/L, intravenous infusion of insulin at 25 mU/kg/hr (0.5 mL/kg/hr of the solution described above) with an infusion of lactated Ringer's solution with 20-40 mmol/L potassium is recommended.
   (c) If the blood glucose concentration is <6 mmol/L, an infusion of 2.5% dextrose in 0.45% saline with 20-40 mmol/L potassium at 6 mL/kg/hr is recommended and insulin infusion should be withheld until the blood glucose concentration is >6 mmol/L.

CATS REQUIRING ELECTIVE GENERAL ANAESTHESIA

1. The entire usual dose of insulin should be administered in the morning when food is withheld.
2. Whenever possible, schedule general anaesthesia of diabetic cats early in the day so that they can be recovered and discharged to the home environment before the next insulin injection and meal is due.
3. For cats with excellent glycaemic control as evidenced by persistent negative glucosuria, intravenous administration of an insulin constant rate infusion is recommended while the cat is hospitalised with the goal of maintaining blood glucose concentration at 5-10 mmol/L. Blood glucose concentration should be monitored every 1-2 hours and intravenous fluids continued until the cat is discharged from hospital.
   (a) When the blood glucose concentration is 5-10 mmol/L, intravenous infusion of insulin at 25 mU/kg/hr (0.5 mL/kg/hr of 50 mU/mL solution (25 U insulin in 500 mL saline) with an infusion of 2.5% dextrose in 0.45% saline with 20-40 mmol/L potassium at 6 mL/kg/hr is recommended.
   (b) When the blood glucose concentration is >10 mmol/L, intravenous infusion of insulin at 25 mU/kg/hr (0.5 mL/kg/hr of the solution described above) with a maintenance infusion of lactated Ringer's solution with 20-40 mmol/L potassium is recommended.
   (c) If the blood glucose concentration is <5 mmol/L, an infusion of 2.5% dextrose in 0.45% saline with 20-40 mmol/L potassium at 6 mL/kg/hr is recommended and insulin infusion should be withheld until the blood glucose concentration is >6 mmol/L.
4. For cats with less than excellent glycaemic control as evidenced by positive glucosuria, intravenous administration of an insulin constant rate infusion is recommended while the cat is hospitalised with the goal of maintaining blood glucose concentration at 6-15 mmol/L. Blood glucose concentration should be monitored every 1-2 hours and intravenous fluids continued until the cat is discharged from hospital.
   (a) If the blood glucose concentration is 6-15 mmol/L, intravenous infusion of insulin at 25 mU/kg/hr (0.5 mL/kg/hr of 50 mU/mL solution (25 U insulin in 500 mL saline) with an infusion of 2.5% dextrose in 0.45% saline with 20-40 mmol/L potassium at 6 mL/kg/hr is recommended.
   (b) If the blood glucose concentration is >15 mmol/L, intravenous infusion of insulin at 25 mU/kg/hr (0.5 mL/kg/hr of the solution described above) with an infusion of lactated Ringer's solution with 20-40 mmol/L potassium is recommended.
   (c) If the blood glucose concentration is <6 mmol/L, an infusion of 2.5% dextrose in 0.45% saline with 20-40 mmol/L potassium at 6 mL/kg/hr is recommended and insulin infusion should be withheld until the blood glucose concentration is >6 mmol/L.

Back to Table of Contents

MANAGEMENT OF DIABETIC DOGS & CATS REQUIRING GENERAL ANAESTHESIA

For cats with excellent glycaemic control as evidenced by **persistent negative glucosuria**, IV administration of an insulin CRI is recommended while the cat is hospitalized with the goal of maintaining blood glucose concentration at **5-10mmol/L**. Blood glucose concentration should be monitored every 1-2 hours & IV fluids continued until the cat is discharged from the hospital.

The **entire usual dose of insulin** should be administered in the morning when food is withheld.

Preparation

BAG A – Insulin Bag
Add 25U ActRapid (0.25mL) to 500mL of saline (50mU/mL)
Mix well & label
Cover & protect from light

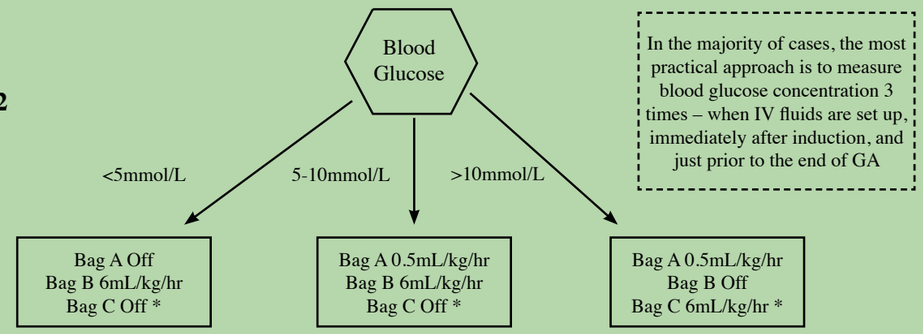
Bag B – Glucose Bag
0.45% NaCl 2.5% gluc + 20-40mmol/L KCl
Mix well & label

Bag C – Hartmann's Bag
Hartmann's Solution + 20-40mmol/L KCl
Mix well & label

Use

CATS

Protocol 1 of 2



In the majority of cases, the most practical approach is to measure blood glucose concentration 3 times – when IV fluids are set up, immediately after induction, and just prior to the end of GA

Whenever possible, schedule GA of diabetic cats early in the day so that they can be recovered and discharged to the home environment before the next insulin injection is due.

*If higher total fluid rate is required at any stage, then increase Bag C to the required rate. Eg. Add 4mL/kg/hr to get surgical rates of 10mL/kg/hr

MANAGEMENT OF DIABETIC DOGS & CATS REQUIRING GENERAL ANAESTHESIA

For cats with less than excellent glycaemic control as evidenced by **positive glucosuria**, IV administration of an insulin CRI is recommended while the cat is hospitalized with the goal of maintaining blood glucose concentration at **6-15mmol/L**. Blood glucose concentration should be monitored every 1-2 hours & IV fluids continued until the cat is discharged from the hospital.

The **entire usual dose of insulin** should be administered in the morning when food is withheld.

Preparation

BAG A – Insulin Bag
Add 25U ActRapid (0.25mL) to 500mL of saline (50mU/mL)
Mix well & label
Cover & protect from light

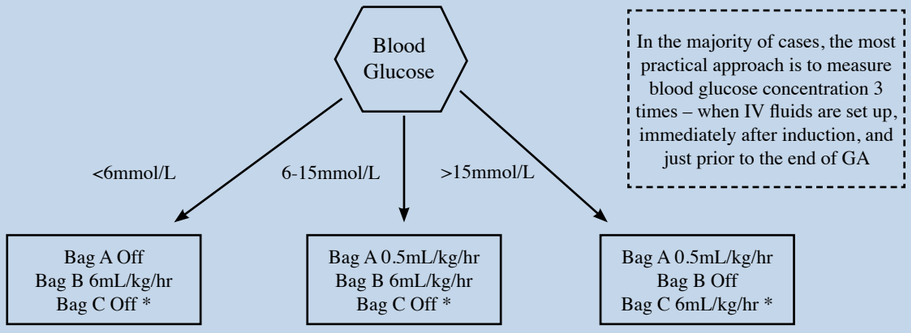
Bag B – Glucose Bag
0.45% NaCl 2.5% gluc + 20-40mmol/L KCl
Mix well & label

Bag C – Hartmann's Bag
Hartmann's Solution + 20-40mmol/L KCl
Mix well & label

Use

CATS

Protocol 2 of 2



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MANAGEMENT OF DIABETIC DOGS & CATS REQUIRING GENERAL ANAESTHESIA

IV administration of an insulin CRI is recommended while the dog is hospitalized with the goal of maintaining blood glucose concentration at **6-15mmol/L**. Blood glucose concentration should be monitored every 1-2 hours and IV fluids continued until the dog is discharged from hospital.

Half the usual dose of insulin should be administered in the morning when food is withheld.

Preparation

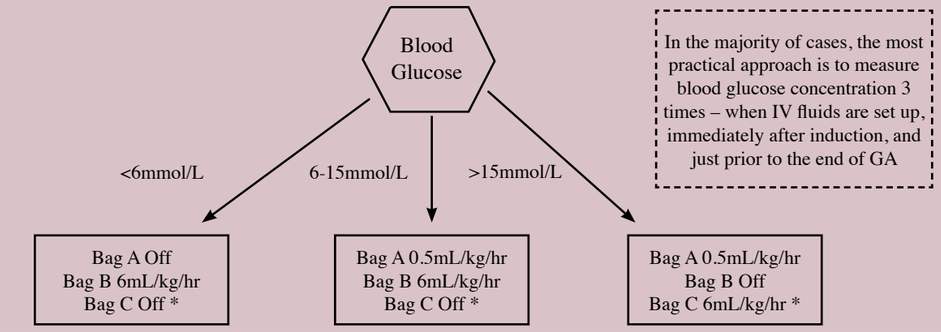
BAG A – Insulin Bag
Add 25U ActRapid (0.25mL) to 500mL of saline (50mU/mL)
Mix well & label
Cover & protect from light

Bag B – Glucose Bag
0.45% NaCl 2.5% gluc + 20-40mmol/L KCl
Mix well & label

Bag C – Hartmann's Bag
Hartmann's Solution + 20-40mmol/L KCl
Mix well & label

Use

DOGS



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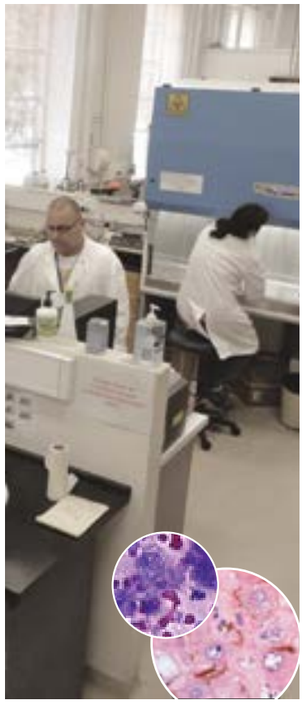
*If higher total fluid rate is required at any stage, then increase Bag C to the required rate. Eg. Add 4mL/kg/hr to get surgical rates of 10mL/kg/hr

eBOOK For cats with excellent glycaemic control as evidenced by **persistent negative glucosuria**

eBOOK For cats with less than excellent glycaemic control as evidenced by **positive glucosuria**

eBOOK For management of dogs



See NECROPSY SUMMARY – Major Winner, co-contributed by Lydia Tong, VPDS Vet Pathology Resident p. 33

MAJOR WINNERS!

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

NECROPSY SUMMARY

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C&T NO. 5404

Adam Gordon[1] & Lydia Tong[2]

CLINICAL PRESENTATION

A 3-year-old, neutered male Burmese cat named 'Runty' presented with acute onset of hind limb paralysis and respiratory distress. He is actually my cat, but this happened while the cat was boarding in my practice, as I was just in the process of finishing an overseas trip. This meant the clinical signs were picked up exceedingly quickly by my diligent team.

On presentation, there was flaccid paralysis of both hind limbs and the tail. The cat was non-ambulatory and exhibited tachypnoea, occasional open mouth breathing and panting. Deep pain sensation was absent in the hind limbs and tail. The nail beds and pads of the hind limbs appeared cyanotic and were cold on palpation. Femoral pulses were absent bilaterally.

Thoracic auscultation revealed a heart rate of 220 beats per minute and tachypnoea (56 breaths per minute). There was no murmur and an intermittent gallop rhythm was present. Crackles and increased bronchovesicular sounds were auscultated over the lung fields. The rectal temperature was 35.0°C.

Clinical findings were consistent with aortic thromboembolism as a sequella of cardiac disease. Thoracic radiographs (Figure 1 A and B) showed increased opacity of the perihilar and caudal lung fields extending to the periphery, prominent interstitial markings and enlarged pulmonary veins consistent with cardiogenic pulmonary oedema. Echocardiography revealed an unclassified cardiomyopathy characterised by severe global systolic dysfunction of the left ventricle, with minimal remodeling of the left atrium. Spontaneous echogenic contrast was seen in the left atrium.

Initial treatment was to place the cat in an oxygen cage, provide opioid analgesia using buprenorphine (Temgesic, Reckitt and Colman) 0.03mg/kg IV, dalteparin (Fragmin; Pfizer) 100 IU/kg SC, frusemide (Frusemide; Ilium) 4mg/kg, pimobendan (Vetmedin; Boehringer Ingelheim) 1.25mg per os and tissue plasminogen activator (Actilyse; Boehringer Ingelheim) 20mg intravenously administered as a constant rate infusion at 4mg/hour over 5 hours.

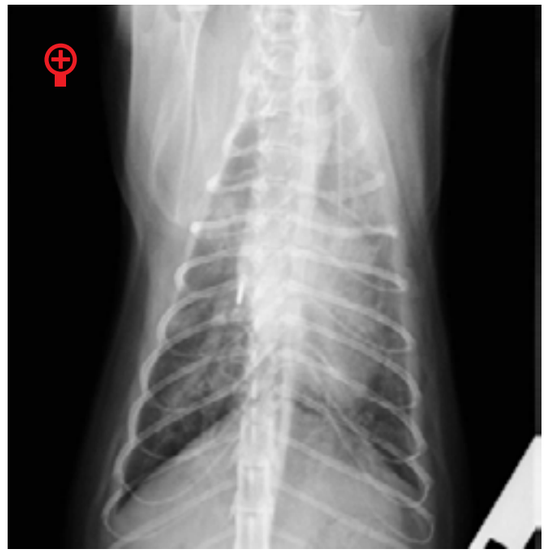

Figure 1 A. Dorsoventral thoracic radiograph taken shortly after stabilisation in an oxygen cage.

Serum biochemistry revealed hyperkalaemia, hypochloridaemia, pre-renal azotaemia, hypocalcaemia, hyperglobulinaemia, hyperglycaemia, elevated alanine aminotransferase and lipase. Urine specific gravity was 1.040.There was trivial to very mild mitral regurgitation and spontaneous echogenic contrast in the left atrium.

Ongoing treatment consisted of buprenorphine analgesia (Temgesic, Reckitt and Colman), dalteparin (Fragmin; Pfizer), pimobendan (Vetmedin; Boehringer Ingelheim), frusemide (Frusemide; Ilium) and clopidogrel (Plavix; Sanofi Aventis) 18.75mg daily. On day 2 after presentation respiration appeared more normal and the rate dropped to 28 breaths per minute. Thoracic radiographs confirmed resolving pulmonary oedema.

BIOCHEMISTRY RESULTS		
Analyte	Value	Reference Range & Unit
Albumin	36	22 - 40 g/L
Alkaline Phosphatase	22	14 - 111 U/L
Alanine aminotransferase	490	12 - 130 U/L
Urea	18.6	5.7 - 12.9 mmol/L
Calcium	1.91	1.95 - 2.83 mmol/L
Creatinine	177	71 - 212 µmol/L
Glucose	10.05	4.11 - 8.83 mmol/L
Lipase	2406	100 - 1400 U/L
Total Protein	108	57 - 89 g/L
Globulins	72	28 - 51 g/L
Sodium	156	150 - 165 mmol/L
Potassium	6.4	3.5 - 5.8 mmol/L
Chloride	106	112 - 129 mmol/L

M-MODE ECHOCARDIOGRAPHY MEASUREMENTS		
Parameter	Measurement	95% Confidence Interval
IVSd	3.0 mm	3.0-6.0
IVSs	3.0 mm	4.0-9.0
LV chamber -d	16.0 mm	10.8-21.4
LV chamber -s	14.0 mm	4.0-11.2
LVWd	3.0 mm	2.5-6.0
LVWs	3.0 mm	4.3-9.8
FS	12.5%	40-67
Ao	8.0 mm	6.0-12.1
LA	11.0 mm	7.0-17.0
LA:Ao	1.38	0.88-1.79
EPSS	1.0 mm	0.0-2.0
Ao flow velocity	0.41 m/s	
AO PG	0.7 mmHg	
PA flow velocity	0.42 m/s	
PA PG	0.7 mmHg	

Unfortunately, Runty developed paresis of the left forelimb. The paw was cool, the pads were pallid and there was no deep pain sensation. No pulse was detected using Doppler probe.

On day 3 after presentation the respiration still appeared normal, but there was no change in the neurovascular status of the hind limbs and the left forelimb. Furthermore the cat now appeared profoundly obtunded. Given the deterioration in central nervous system function and grave prognosis the decision was made to euthanase the patient.

GROSS POST-MORTEM EXAMINATION

Gross examination was performed on a normally developed, 3-year-old male neutered Burmese Cat. Significant findings were identified in the heart, lungs, left forelimb, aorta and brain.

The right heart was moderately enlarged, with a dilated and thin right ventricular free wall. There were focally extensive regions of pallor affecting 40% of the left ventricular epicardium. The heart weight was 27g, the right ventricular free wall measured 1mm, the left ventricular free wall measured 5mm, and the interventricular septum measured 6mm (Figure 2).

The lungs were pale to dark red with a diffuse patchy pattern, were smooth-surfaced and mildly heavy and wet.

The left manus exhibited severe reddening of the subcutaneous tissues and the digits were red to black, extending up to the mid antebrachium. There were additional foci of subcutaneous reddening at the caudal elbow, and dorsomedial to the scapula.

Within the distal aorta there was a well-defined, smooth edged clot (55x8x7mm), which varied from pale and slightly firm at the distal end, to red-black and mucoid at the proximal aspect.

Sectioning of the brain following fixation revealed a large region of dark red to black material (clotted blood) within the fourth ventricle.

The following tissues were grossly normal: Thyroid glands (tan, ovoid, L 20x5x2mm, R 20x5x2mm), liver (prominent lobular pattern), kidneys (35x25x10mm), adrenals, stomach (empty excepting a small amount of bilious mucoid material), spleen (contracted 20x30x5mm), pancreas (pancreatic/duodenal vein is dilated 140x20x4).

Histological Examination

Examination of the heart revealed an extensive, irregular, multifocal to coalescing, interstitial fibrosis and replacement of myocardial cells of the left ventricular free wall and interventricular septum (Figure 3). Fibrosis was preferentially affecting the inner third (endocardial region) of the myocardium, (Figures 4-5) though it did project transmurally toward the free wall of the left ventricle multifocally, and the epicardium of the left ventricular free wall. Fibrosis was poorly defined, irregular and disordered. The fibrosis tended to surround large vessels. The papillary muscles were also markedly affected. Adjacent to regions of fibrosis there was myocardial cell loss and myocardiocyte degeneration. Myocardial disarray was not a feature. Sporadically, in regions of fibrosis individual myocardial cells contained dense purple granules (mineralisation).

The interventricular septum (left side) was less affected and appeared to be at an earlier stage of degeneration. Throughout, there was a mild increase in inflammatory cells, with scattered lymphocytes and neutrophils seen around medium sized blood vessels. The right ventricular myocardium was normal, though it is thinned; multifocally the right ventricular free wall was thinned to 1mm.

Fibrosis was confirmed with Masson's trichrome staining (Figure 5). Weigert's elastic stain showed that elastin deposition was normal.

The distal aortic thrombus consisted of large numbers of clustered, slightly degenerative erythrocytes, large regions of fibrin which

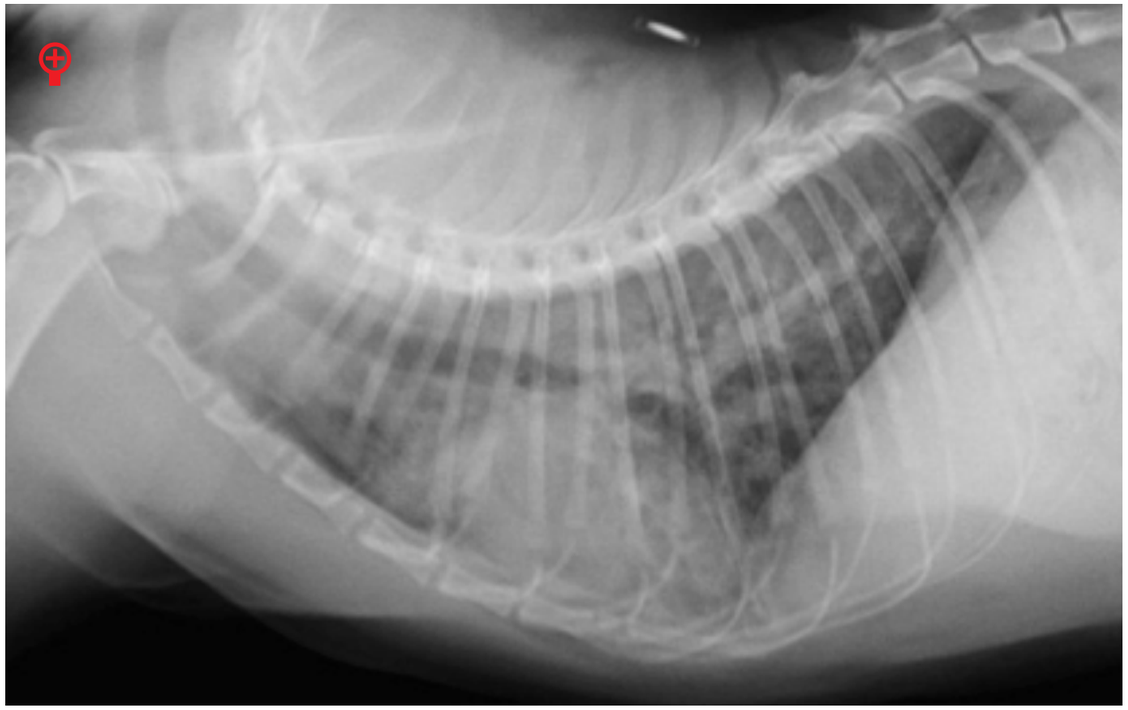

Figure 1 B. Lateral thoracic radiograph

formed poorly defined layers in parts, and small numbers of mixed leukocytes admixed with the erythrocytes. Intermittently there were thin endothelial cells lining the thrombus surface.

Within the fourth ventricle of the brain is a large cluster of degenerative erythrocytes, fibrin, and occasional leukocytes (blood clot) (Figure 6). The clot adhered to the adjacent brain parenchyma, and erythrocytes extend a short distance into this and beneath remnants of choroid. Intermittently there were thin endothelial cells lining the thrombus surface. Brain parenchyma is otherwise normal.

Tissue from the left manus contained a marked band of haemorrhage spanning deep dermis, through hypodermis and extending to the musculature. Occasionally there was haemorrhage within the dermis, extending to the epidermis. There were two large hypodermal vessels containing fibrinous, well-defined thrombi which were lined intermittently by endothelial cells.

The liver was affected by marked, eccentric, centrilobular hepatocellular fallout and pallor (degeneration and necrosis).

Normal tissues: Thyroid, parathyroid, spleen, urinary bladder, pancreas. Not examined: Lung

Diagnoses:

1. Left ventricular fibrosis and myocardial atrophy, marked, transmural, with predilection for the subendocardium, with marked right ventricular dilation, and moderate cardiomegaly, (ratio of heart weight to body weight: 6.3)
2. Thromboembolic disease, subacute (3-7 days), left forepaw, distal aorta
3. Intracranial haemorrhage (4th Ventricle), ante-mortem, recent <7 days
4. Hepatic centrilobular degeneration, marked (hypoperfusion injury)

Discussion

Runty was diagnosed with a severe fibrosing cardiomyopathy of the left ventricle preferentially affecting the subendocardial myocardium with multifocal transmural extension. Multifocal thromboembolic disease is considered likely to be a sequel of poor cardiac output (decreased blood flow) and increase turbulence of flow within the left chambers due to the cardiomyopathy. Haemorrhage, such as in the fourth ventricle may be associated with heparin therapy, and/or depletion of clotting factors.

Fibrosing cardiomyopathies preferentially affecting the subendocardial tissue of Burmese cats were described, albeit reports are limited in number.[1] These reports are typically of young kittens and endocardium (alone) is replaced and thickened by both fibrous tissue and elastin.[1,2] These cats tend to have concurrent severe left sided chamber dilation and enlargement. The condition is referred to as endocardial fibroelastosis (EFE).

Gross and histological changes observed in this case do not align well with these reports. Runty's interstitial fibrosis is tending to affect the subendocardial myocardium, but also extends transmurally to affect a significant amount of subepicardial myocardium of the left ventricular free wall.

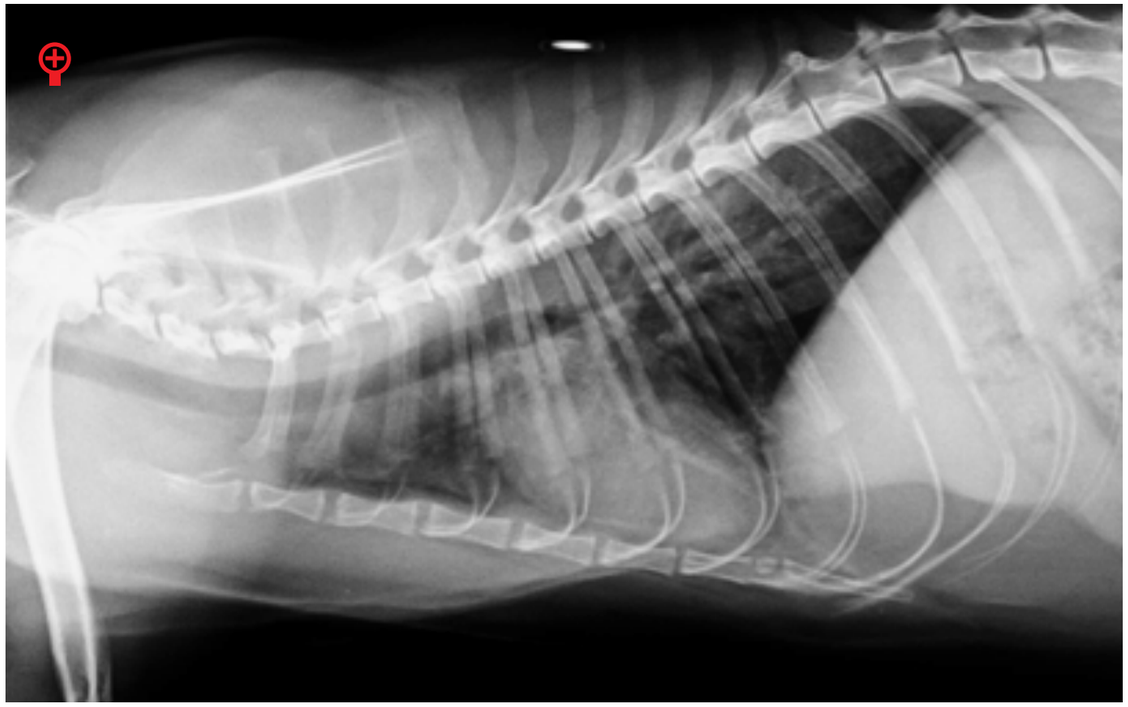

Figure 1 C. Lateral thoracic radiograph after frusemide and other measures (see text)

Weigert's elastic stain showed no excessive elastin deposition within affected heart, *cf* descriptions of EFE. He is also considerably older than previous reports of affected cats, who generally show clinical signs between 3 weeks and 4 months (and dogs <6 months).[1,2,4]

There is some resemblance in this case to the histopathological alterations reported with arrhythmogenic right sided cardiomyopathy (ARVC), other than the predilection for inner myocardium in Runty's case. ARVC tends to affect outer myocardium, although can also become transmural. Left sided ARVC is now an accepted phenotypic variant of human ARVC, and ARVC with marked involvement of the LV has been described in the cat (although in this case there was also significant right-sided involvement not apparent with Runty).[3]

Both of these conditions (EFE and ARVC) are thought to have genetic components, as well as multiple other proposed aetiologies – such as viral myocarditis, myocardial necrosis, diet and environmental factors, as well as genetic predisposition.

Therefore, while Runty has a fibrosing cardiomyopathy, his pathology not sufficiently typical to allow categorisation with either of these conditions described in cats. There are anecdotal reports by clinicians and pathologists in Sydney that other cats, particularly Burmese, may have presented similar pathologies. Collation of these cases may provide additional information on the spectrum of fibrosing cardiomyopathy in Burmese cats.

References:

1 B.C. Zook, L.H. Paasch, 1982, Endocardial fibroelastosis in Burmese cats, *American Journal of Pathology*, 106 (1982), pp. 435-438

2 N. Rozengurt, 1994, Endocardial fibroelastosis in common domestic cats in the UK, *Journal of Comparative Pathology,* 110, pp. 295-301

3 Ciaramella P et al, Arrhythmogenic right ventricular cardiomyopathy associated with severe left ventricular involvement in a cat, *Journal of Veterinary Cardiology* (2009) 11, 41-45

4 M. H. M. A. Larsson, et al, (1997), Endocardial fibroelastosis in a dog, *Journal of Small Animal Practice* 38, 168-170

5 T.L. Cushing, 2013, Endocardial Fibroelastosis in a Quarterhorse Mare, *Journal of Comparative Pathology*, Available online 4 May 2013.

Fig 2. Heart: Transverse section showing right and left ventricles, with moderate dilation of the right ventricle

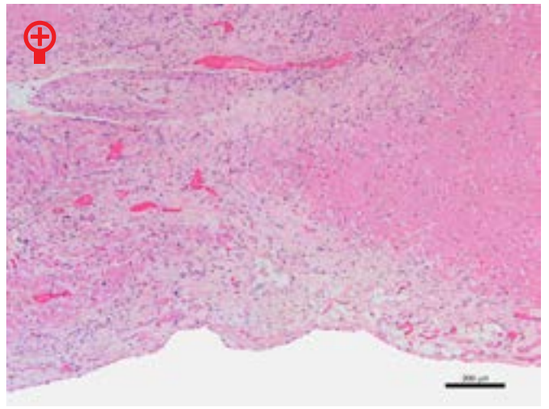

Fig 3. Heart: Subendocardial and perivascular replacement and interstitial fibrosis (fibrosis pale pink, cardiomyocytes deep pink), haematoxylin and eosin. [Magnification 100x]

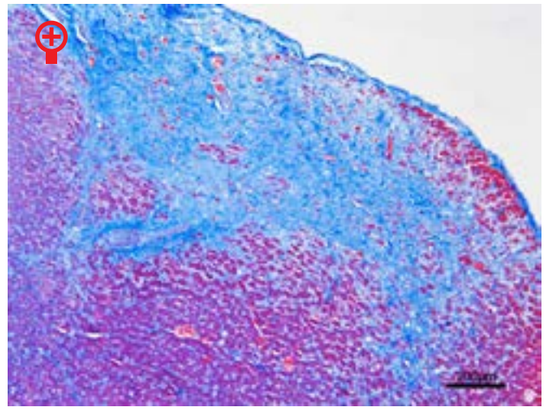

Figure 5. Additional section of Fig 3 stained with Massons Trichrome highlighting the subendocardial replacement and interstitial fibrosis (Fibrous tissue is blue, myocardiocytes are red. [Magnification 100x]

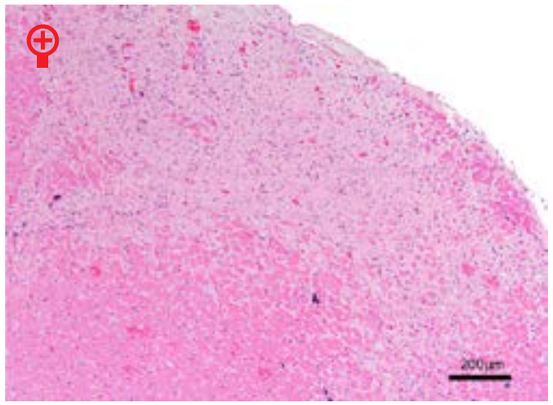

Figure 4. Heart: Subendocardial replacement and interstitial fibrosis (fibrosis pale pink, cardiomyocytes deep pink), haematoxylin and eosin. [Magnification 100x]

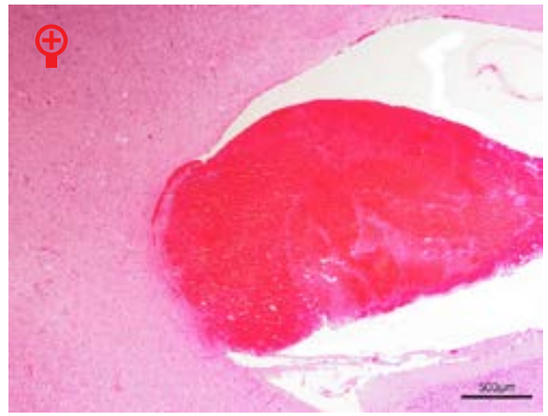

Fig 6. Brain: Sections showing a large recent antemortem haemorrhage within the fourth ventricle. [Magnification 40x]

Back to Table of Contents

INTERESTING WEBSITE

www.scientificamerican.com/article/where-house-cats-roam/

Where House Cats Roam; August 2011; Scientific American Magazine; by Madhumita Venkataramanan; 1 Page(s)

Anyone who has ever owned an outdoor cat knows that it tends to disappear for hours, sometimes days, at a time. Where do cats go when they are lurking out of sight? The question is of interest not just to pet owners but also to conservation scientists who study the impact of free-roaming cats on wildlife populations. Scientists at the University of Illinois and the Illinois Natural History Survey recently attached radio transmitters to the adjustable collars of 18 pet and 24 feral cats in southeastern Champaign-Urbana and tracked the animals by truck and on foot for more than one year.

The research, published in the *Journal of Wildlife Management*, shows that pet cats maintain a rather lazy existence: they spent 80 percent of their time resting. They devoted another 17 percent to low-activity pursuits such as grooming and only 3 percent to high-activity pursuits such as hunting. Unowned cats rested just 62 percent of the time and spent 14 percent, mostly at night, being highly active. Feral cats roamed far more widely than researchers had expected: up to 1,351 acres. In contrast, pet cats stayed within an average of about five acres of home.

The small cats' behavior is similar to that of their larger cousins. "Maintaining a ranging area is a very intrinsic behavior to cats," says Alan Rabinowitz, CEO of the conservation organization Panthera. Like small cats, wild cats like to stay close to humans for easier access to food. Jaguars in Latin America, for example, slink quietly through massive stretches of human land. It's part of a cat's nature to live on the interface of wild and human-inhabited land.

HANDY TIPS!

C&T NO. 5406

Aine Seavers
Oak Flats Vet Clinic
58A Central Avenue
Oak Flats NSW 2529
E. vets@oakflatsvet.com.au

Rabbits LOVE Lectade!

Even a semi- moribund dehydrated rabbit will drag itself to the bowl and doggedly drink it until it self- rehydrates. One can almost see the rabbit resurrect itself before one's own eyes

Photo courtesy of Anne Fawcett – pictured above with rabbit friend.

If you have seen an interesting rabbit case lately, please write it up for the C&T and email it to: elisabeth.churchward@sydney.edu.au

CAN SOCRATES HELP US IN CLINICAL REASONING?

C&T NO. 5405

Courtesy of Paul Canfield DVSc PhD GradCertEdStud, FANZCVS FRCPath MRCVS, Professor Emeritus, Faculty of Veterinary Science of The University of Sydney

Here is something that I read somewhere and have paraphrased because it tugged at a memory:

After serving in a war, Socrates devoted himself to the pursuit of truth. His reputation as a philosopher soon spread around Athens and beyond. When he was told that the Oracle of Delphi had revealed to one of his friends that Socrates was the wisest man in Athens, Socrates responded not by boasting or celebrating, but by trying to prove the Oracle wrong. In typical Socrates style, he decided he would try and find out if anyone knew what was truly worthwhile in life, because he deduced that anyone who knew that would surely be wiser than him. He set about questioning those with wise reputations, but no one could give him a satisfactory answer. Instead all pretended to know something they clearly did not. Finally, he realised the Oracle might be right after all: he was possibly the wisest man in Athens because he alone was prepared to admit his own ignorance rather than pretend to know something he did not!

Moral for clinical reasoning? You are not alone in your ignorance of disease, but be accepting rather than pretend to know something you don't know! Additionally, be critical and perhaps even cynical of what you read or hear from those with expert reputations!

PERSPECTIVE NO. 105

FROM FELINE MEDICINE TO SAVING AUSTRALIA'S BRUMBIES

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RCVS Recognised Specialist in Feline Medicine
European Veterinary Specialist in Internal Medicine

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One of the wonderful things about the veterinary profession is its diversity, and flexibility. Veterinarians can work in clinical practice, research or industry. In practice you can choose to work with a variety of species, or choose to specialise in one or two species from an infinite selection, or in a particular body system area. However specialised you may become, it doesn't mean that an interest cannot be maintained in other areas or species and it is always possible to change career direction within the profession relatively easily. The other aspect of being a veterinarian that is unique is that whatever corner of the profession we end up in, we are all brought together by our core love of animals, and desire to make a difference for them.

CVE feline DE tutor, feline specialist at SASH, and ISFM Australasian representative, Andrea Harvey tells her story of how a British feline specialist brought up on the Island of Guernsey came to live on a farm in the Southern Highlands of NSW and be a passionate advocate of the Australian Brumby.

Andrea's 3 adopted Brumbies: from left to right, Hero, James Brown & Sonic when she first met them as yearlings, at Save The Brumbies Inc sanctuary near Armidale

Back to Table of Contents

It was a cold windy night in Guernsey as I pulled up my blankets and sleeping bag to snuggle up on the stable floor for the night next to my recumbent pony, Merrylegs, who was battling with a lacerated digital flexor tendon sheath. There were concerns that it was infected, and I was told that it was touch and go whether she would survive. I was 14-years-old and determined to nurse her through and prove the equine vet wrong. After a cold uncomfortable night, as the sun rose the next morning, Merrylegs' condition began to turn the corner. That was the moment that I committed wholeheartedly to becoming a veterinarian.

The yearling Brumbies were naturally anxious on arrival at Andrea's after a 3 day journey, but quickly realized that Andrea meant food!

They were soon settled enough to be able to go out in 150 acres with the other 10 horses at the farm

So my prime reason for becoming a vet was my passion for horses. I fell in love with horses at about the age of 10 when I was finally allowed my first riding lesson. Soon after that I was working all hours at the stables, cycling there early in the morning, mucking out and filling water buckets, in exchange for rides. Of course I was desperate for my own pony, and every weekend at lunch break from the stables I would cycle to my Gran's for lunch and sneakily write 'pony' on her shopping list, but it never came! Eventually, I suspect thinking they could put a stop to my badgering, my parents said 'OK we'll buy you a pony if you pay for its keep'. Away they went thinking 'ha that will keep her off our back for awhile, what chance has a 14-year-old got of paying for the keep of a pony'. Two weeks later I had a job working in the local Fish & Chip shop, a month later I presented my parents with the money for the first month's upkeep for a pony. They had to admit defeat and do something they had never in a million years planned on doing, buying me a pony. That is when Merrylegs came into my life, a lively grey 13.2hh mare who stayed in my life through GCSEs, A-levels, Vet School application, the whole of Vet School, my first jobs in practice, the whole of my residency training, certificate, diploma exams and my first job as a Specialist. She only passed away, sadly about 3 years ago, at the grand old age of 35 years. I often think about Merrylegs; through having her in my life I learnt so many fundamental things: just how much you can achieve with nothing more than pure determination, how the elating feeling an animal recovering from illness makes going that extra mile all the worthwhile, and from a client perspective of just how important animals are to people's lives. The patient sitting on the consult table, standing in the paddock or stable, has often been so important to its owner through all sorts of key life events that we will never know about, just like Merrylegs had been with me.

So, how did I end up becoming a cat vet? I always loved cats too of course, and had my first cat, 'Tiger', when I was 8-years-old, but I wanted to be an equine and large animal vet right up to about 4th year of Vet School. During preparation for getting in to Vet School and during the early years of Vet School I had also developed a love for working with farm animals. There was a dairy farm down the road from where I lived in Guernsey and I would keenly cycle down at 4am to help with milking, and they would call me at any hour when a calving was happening. I also have fond memories of 1st year Vet School vacation time spent lambing in the Cumbrian hills in the far north of England. But it was in the clinical years of Vet School that the harsh realities of being a farm animal vet hit me, and much of being an equine vet was about lameness, and I had come to realize that orthopaedics was not my strong point! Small animal medicine, on the other hand, seemed fascinating and being lucky to have mentors like Tim Gruffydd-Jones, Andy Sparkes and Martha Cannon, at a time when feline medicine was still in its infancy, my interest in feline medicine quickly flourished. I never intended on becoming a Specialist; I still really wanted to go into mixed practice when I first graduated. I really 'fell' into being a feline specialist simply because I really wanted to do the absolute best for all of my patients and realized at an early stage in practice that that was impossible when dealing with lots of different areas and different species. I quickly gained a lot of cat clients in my first job and realized that cats were often not managed optimally in practice and so decided to try and undertake a residency in feline medicine to learn more about this enigmatic species. Little did I know that that would end up leading me down the pathway of becoming a feline Specialist and really just working with cats (and some dogs) for several years.

When I moved to Australia to live on my partner's farm in the Southern Highlands, I was quickly re-united with caring for many other species: sheep, alpacas, goats, chickens, geese, turkeys as well as horses. This re-kindled my love of working with these species and, although I couldn't give up my day job as a cat vet, I enjoy dabbling in 'pretending' to be a multi-species vet! With over 60 animals on our farm, there is always something that needs attending to; just recently we have been treating one of our horses for Australian Stringhalt, another with a severe limb wound, some of our chickens for 'scaly leg', and we recently

bottle reared a weak lamb, and castrated a couple of our lambs and a miniature colt that we bred.

It is working with the horses in my spare time though that still gives me the greatest pleasure. We already had 10 horses, and when I moved from the UK I couldn't move without my current horse, Connie, so along she came too, together with my cat, Thomas. Despite not being short of equine company now, I have always had the dream to have a horse from a youngster. Here in Australia, living on 250 acres, I decided that now was the time. As I searched the web for young horses, I stumbled across the website of 'Save the Brumbies' (www.savethebrumbies.org), and this is where my incredible journey with Brumbies began. I learnt of the plight of Australia's wild horses and how the charity had been set up following a horrific aerial slaughter of over 600 horses in Guy Fawkes River National Park of NSW in 2000, just the same year that the Brumby featured prominently in the opening ceremony of the 2000 Sydney Olympic games. Brumbies are widely known across the world as an Australian Icon, epitomising the spirit and freedom of Australians. Many people across the world know of Brumbies, immortalised in the film 'The Man from Snowy River', the 'Sliver Brumby' series of books and the 19th century 'bush poetry' of Banjo Paterson. Coming from oversees I naively assumed that these beautiful wild horses were highly valued in Australia and I was therefore shocked to find out that in this day and age, in their home country, this Australian Icon is actually considered a feral pest, with the wishes of the government being to try and eradicate them from many National Parks.

Not knowing much about the history of horses in Australia, I was fascinated to read in the beautiful book 'Brumby: A celebration of Australia's wild horses' that these horses originated back in 1788 with the arrival of the First Fleet. Horses were imported by early European settlers, with the long journey by sea meaning that only the toughest horses even survived the trip. There were still only about 200 horses thought to have reached Australia by 1800. The rising popularity of racing around 1810 brought an influx of Thoroughbred imports, mostly from England. By 1820, around 3,500 horses were present in Australia. By the 19th century horses had significantly grown in number and were utilised widely from ploughing fields, building roads and railways, carrying mounted police and bushrangers, and for day to day transport.

It is believed that the wild horse population in Australia originated from a combination of some horses escaping from settled regions of Australia, and horses being released to fend for themselves when pastoralists abandoned their settlements, between 1804 and the 1840's. At this time, these initial stocks would have comprised Thoroughbreds, Clydesdales, Timor ponies and Arabians. Only the toughest of these horses were to survive the harsh environments, and so natural selection has seen the evolution of tough, highly intelligent, genetically diverse, magnificent horses, in what is known today as the Australian Brumby. The term Brumby is thought to have come about as a result of horses left behind by Sergeant James Brumby when he left Australia in 1804.

Horses from the Northern Tablelands of NSW were then captured and bred for the remount trade and were known as Walers (comprising a combination of Thoroughbred, Arab, Cape horse, Timor pony and Clydesdale or Percheron). These horses became indispensible to Australians in the Light Horse mounted forces in the Second Boer War and World War 1, with 16,314 horses dispatched overseas from Australia during the Boer War, and 121,324 horses being sent overseas in World War 1. Over 70,000 of these horses lost their lives in World War 1. Due to quarantine restrictions the light horses could not return to Australia and were either sold to the British Army as remounts for Egypt and India or destroyed. Only one Waler, called 'Sandy', the mount of Major-General WT Bridges, an officer who died at Gallipoli, is known to have been returned to Australia.

It didn't take the Brumbies long to settle in at Andrea's farm and make friends with the other horses, sheep and alpacas (Photo courtesy of Anne Fawcett)

Back to Table of Contents

As an insight into how important these Walers were to Australians during World War 1, I can thoroughly recommend the book 'Bill the Bastard'. This particular story focuses on a Waler called 'Bill', one of thousands of horses sent with the ANZAC mounted force to the Middle East in the Great War at Gallipoli. Bill was initially known for always bucking when asked to gallop, but became famous for gallantly saving 4 Australians outflanked by the Turks, by carrying 5 men at once, ¾ of a mile galloping through soft sand without a single buck. The book is an enjoyable and fascinating read for anyone.

As demand for remounts declined in the 1940s, the commercial Waler trade ended and remaining unwanted Walers were again set free into the bush, left to fend for themselves. It is currently estimated that 400,000 to 1 million horses roam freely in Australia, making it the largest population of wild horses in the world.

From snow-capped peaks and wild bushland to the arid Centre and Northern and West Australian Outback, Brumbies have evolved to be as diverse and magnificent as the continent they roam (K Massey and M Lee Sun). The Brumbies found today in different states of Australia all have slightly different origins. The Brumbies roaming free today in Guy Fawkes River National Park are said to be direct descendants of the Walers used in World War 1, described by an English cavalry officer as 'undoubtedly the finest cavalry mounts in the world' (Lieutenant Colonel RMP Preston, The Desert Mounted Corps).

Having found out the fascinating origins of the Australian Brumby and the battle that they are facing today, I was determined to help in some way. I contacted Save the Brumbies (STB) and was soon off on a trip to visit their sanctuaries near Bellingen and Armidale. There I was met by an incredible woman, Jan Carter, the founder and president of the charity. Despite being in her 70's she single handedly runs the Bellingen sanctuary at her home where she cares for Brumbies that have been physically or mentally injured and are unsuitable for adoption. Here, they remain in Jan's loving care for the rest of their natural life. I was blown away by how quiet, calm and affectionate the Brumbies here were, and many of these Brumbies had never seen a person for the first few years of their life, and been severely traumatised on capture. But with Jan's gentle handling and patience, they had grown to love being around people. The main sanctuary near Armidale is a 1,400 acre property all run by Megan Hyde, a volunteer. Here a group of older mares and stallions are kept to live in a natural habitat to that which they were living in before being removed from Guy Fawkes National Park. Younger horses removed from the park are assessed, vetted and have basic handling before being offered to approved people for adoption. I was delighted that they considered me suitable for adopting a Brumby and off I went to meet the youngsters. Given that I would be travelling a yearling a long distance to the Southern Highlands, I thought it unfair to travel one alone, so had already decided that I would adopt 2 colts and had learnt of 2 that were good friends, 'James Brown' (JB) a buckskin colt and 'Sonic' a bay colt, so was all set for adopting

The beautiful Hero (left) and Sonic (right)

them. However, when I met them, it turned out they had a 3rd friend, 'Hero' a palomino. What a dilemma, I couldn't possibly take 2 and leave one of them without his mates, so I decided to adopt all 3, and how glad am I that I did!

Hero was 18-months and JB and Sonic were about 15-months-old when they arrived at our farm. Megan and Jan had done a fabulous job with handling them and they travelled really well, but of course they didn't know us and being youngsters away from everything they knew they were anxious on arrival. We left them be in a paddock near the house and for the first few weeks I would just feed them and hang around in their paddock so they just got used to me being around, without trying to approach them. It wasn't long before they realized that I meant food, and began approaching me with curiosity. Once they were settled we introduced them to our other horses and let them out in 150 acres with the other horses. It didn't take long before they were coming up to see me every time I went out to

From woolly anxious yearlings (above) to handsome, calm & confident 2 year olds (below). (Below and to the right photos courtesy of Anne Fawcett)

Hero & Andrea enjoy a special moment

Although they have free range of over 150 acres, Andrea often finds the Brumbies waiting at the back gate for her! (Photo courtesy of Anne Fawcett)

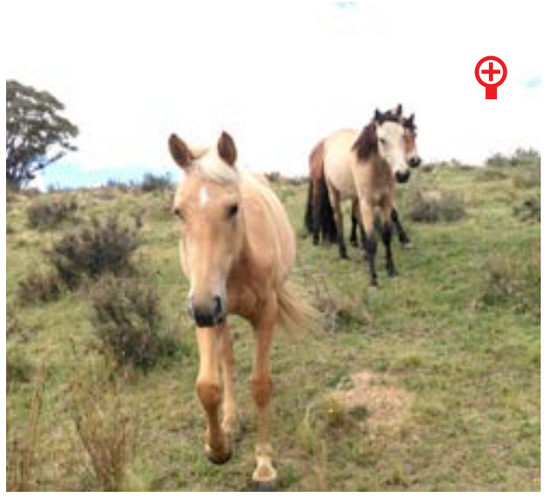
James Brown & Sonic check out the saddle before having it on their backs (Photo courtesy of Anne Fawcett)

Back to Table of Contents

the paddock, often leaving the other horses to follow me back to the house, or just turning up at the back gate waiting for me to come out. After a couple of months I could easily slip halters on them out in the paddock and they quickly took to leading, being the best behaved horses to lead that I have had. Another month later we were trimming their feet, again behaving as if they had been having their feet trimmed for years, and being much quieter than many of our other horses. They are extremely intelligent; we have a round yard that I thought I would feed them in so they became comfortable going in there; after only 2 feeds in there, they would see me walking towards the round yard from a long distance away and gallop over, straight into the round yard and wait for me to get there and feed them, leaving our other horses completely oblivious to what was going on. It is hard to put the quality of these ponies into words; they are undoubtedly special. They are full of personality and character, and in some ways behave more like dogs; they come over to me when they see me, follow me everywhere, stand for ages having cuddles, and just love to be around me. They had an incredible bond with me, after only 6 months. Now, having been with us for a year, and rising 3 year olds, they are on their way to being started under saddle and are all taking everything in their stride. Brumbies are known for their loyalty to their carers and the bonds that they develop, and I can now see why. There seem to be so many misconceptions about Brumbies, even amongst intelligent people, and horse lovers. People just need to meet a Brumby for their perception to change. Fellow veterinarian and writer of www.smallanimaltalk.com, Dr Anne Fawcett visited our farm recently and was also overwhelmed by their gentle and affectionate nature. She said 'I expected Brumbies to be wild, flighty, uncontrollable, rearing, kicking creatures. But they were gentle, sweet, affectionate and absolutely in love with Andrea'.

Brumbies are genetically diverse – natural selection has meant they are tough, they have good bones, great feet, they are amazingly sure footed and athletic, they move beautifully and effortlessly, they are versatile, quiet, calm, gentle, affectionate and highly intelligent. Are these not all the features that we seek in domestic horses? It seems to me that a vast selection of any horse owner's dream horses are sprawled across our doorsteps in Australia, yet they are regarded as feral pests recklessly subjected to terror to be shot from the air or captured and shipped off for slaughter, and meanwhile domestic horses are bred to try and achieve these qualities with many of those horses also tossed aside in the process too.

It is tragically ironic to think that these majestic horses' ancestors saved the lives of many Australians bravely carrying them in war, and now they have to fight for their own future in lands that have been their home for centuries. There are many ways for anyone to support Australia's Brumbies and more information can be found by visiting the STB website; www.savethebrumbies.org. STB is a particularly admirable charity, not only for the great work they do in finding loving homes for hundreds of Brumbies that would otherwise have been slaughtered. But, also for the way that they work tirelessly, with only a tiny handful of volunteers and no paid staff, to not only look after the Brumbies at their sanctuaries, but also to raise public awareness of the importance of protecting the Australian Brumby, establishing the Brumby as a proven quality riding horse, lobbying to dispel the negative myths and misconceptions that seem to have become associated with these magnificent animals, and working closely with National



It didn't take long before the Brumbies were coming running up to Andrea when they saw her, and following her everywhere

Parks & Wildlife Service (NPWS) to bring about radical changes in wild horse management, all without any funding from NPWS or the government, so relying solely on funds raised by themselves.

STB president, Jan Carter, seems to have boundless energy, passion and determination to find an ethically acceptable solution to wild horse population control. Off her own back, she has been researching the use of immunocontraceptive reproductive control utilising porcine zona pellucida (PZP) vaccine which has been used successfully to manage wild horse populations in areas of the US including Maryland, North Carolina, California, Colorado, Wyoming and Nevada. With the support of their local equine vets, STB are planning on undertaking a trial using PZP in a group of mares at the New England Brumby Sanctuary near Armidale. There are difficulties in importing PZP into Australia, but no hurdles phase Jan or dampen her determination. Will PZP be the answer? I have no idea, and this is all way out of the comfort zone of my own narrow area of knowledge. But for Jan, a non-veterinarian, a musician by profession, to be searching for answers to controlling Australia's wild horse population and pro-actively trying to initiate and embark on clinical research in the area, deserves respect and admiration, as well as support from the veterinary profession in trying to find a viable solution.

Wanting to further my knowledge in this area to get a better understanding of PZP and current data, I have now found myself with a list of references to read in Journals such as Reproduction, Wildlife Research, Journal of Wildlife Management and Journal of Reproductive Fertility, journals that I had never heard of before let alone ever thought I would be reading. As a consequence of my wider reading, I am now planning with STB a fertility control trial using GnRH vaccine which is more readily available in Australia than PZP. It can be refreshing to maintain interests in other species outside of our comfort zone, and embrace this unique aspect of our profession. I discovered that it is very hard finding out information about wild horse management and fertility control, because of course most veterinary work and funding involving equine reproduction is directed at breeding horses, not stopping horses breeding!

In this crazy world in which we live, the amount of money in

James Brown comes to greet Andrea in the paddock. (Photo courtesy of Anne Fawcett)

some equine industries such the racing industry is phenomenal, whilst the Australian Icon and ancestors of brave Australian warhorses are branded as feral pests and indiscriminately destroyed. Can the veterinary profession do more to turn this around? Boasting the highest population of wild horses in the world, of such historic significance with such highly sought after equine qualities – surely Aussie veterinarians can rise to this challenge! Can I help to make any difference to the management of Australia's Brumbies? I have no idea, but I'm going to try and make a difference. Life gets more complex as we get older and lots of things get in the way of our aspirations, but if I can summon up the same determination that I had as the 14-year-old girl in Guernsey, then maybe just maybe I can help to do something to preserve the future of the Australian Brumby.

As I come indoors to finish writing this article, I realize that 27 years further down my chosen career path, not much has actually changed in my spare time since a 10-year-old girl; I've just spent the morning picking up horse poo and filling water buckets, only now we have 16 horses of our own! Sometimes it is good to reflect on just why we chose to join this profession in the first place, and to remember not to let everyday challenges, frustrations or politics get in the way, but to stay true to our values and aspirations that we had as a child, when many of us made that life defining decision to become a veterinarian.

Further reading

www.savethebrumbies.org

Brumby: a celebration of Australia's wild horses, 2013. Kathryn Massey and Mae Lee Sun. Exisle Publishing

www.australianbrumbyalliance.org.au

www.lighthorse.org.au

www.australiansatwar.gov.au

www.awm.gov.au/atwar

www.sentient.org.au

Hero excitedly trots over to Andrea when he spots her walking across the paddock

Back to Table of Contents

Sonic, James Brown & Hero have now been joined by another 3 adopted Brumbies; Archie, Dinky & Banjo (which is thought to be Hero's father)

New Sentient Brumby Working Group

Since writing this article, Andrea has adopted another 3 Brumbies and also set up a Brumby Working Group, within Sentient, The Veterinary Institute for Animal Ethics.

Andrea's vision is to form a group of veterinarians and other animal scientists to become a central resource of scientific information about Brumbies, and hopefully with time be considered the 'go to' organisation for welfare advice regarding their management.

Key roles for the group would be facilitating and compiling research into specific areas that would advance the welfare of Brumbies, forming strategic alliances with other organisations to develop and propose policies for their humane management, and advocating on behalf of their welfare to government and environmental groups.

Some of the key issues for Brumbies historically have been claims of their negative impact on the environment, welfare issues arising from mass aerial shooting, limited alternative options for population control (such as fertility control and passive trapping and adoption), the fragmented approach to advocating for their needs, and the lack of independent scientific evidence upon which to base decisions about the most humane management of these unique wild horses.

Andrea is seeking interested people to get involved – if you are interested in becoming involved with Sentient's Brumby Working Group, please contact Andrea on andreaharvey.cat@gmail.com

PERSPECTIVE NO. 106

GETTING FRIENDLY WITH ANATOMY

Corinna Klupiec
(pictured above with 'Nimble')

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Corinna started her veterinary career with a combination of small animal practice and post-graduate study, before moving into veterinary technical services.

Things took a detour in 2000 when she ventured into educational video production in order to pursue a long held interest in audiovisual communication.

After a formative stint producing science and technology videos for high school students, Corinna had the opportunity to combine her interest in video and veterinary science by producing a DVD, 'Ophthalmic Examination in Cats and Dogs', in collaboration with Dr Mark Billson and the Centre for Veterinary Education at the University of Sydney.

Seeking to delve further into an educational role, Corinna took up the post of lecturer in veterinary anatomy at the Faculty of Veterinary Science, University of Sydney. There, she (re) discovered a zeal for anatomy and a passion for helping students to embrace this fundamental veterinary discipline.

Corinna is currently living on the south coast of NSW enjoying a 'sabbatical' that provides the rare opportunity to reflect and write, indulge some sadly neglected hobbies and enjoy more time with her family.

Could there be a more fascinating field of study than anatomy? Surely not! Or perhaps I just feel that way because I have had the privilege of teaching it. We all know there is nothing like teaching something for developing an understanding of, and perhaps even a passion for, a subject that might previously have seemed overwhelming and impenetrable (not to mention tedious and boring). Sadly, due to the density of their courses and the pressures on their time, many students never encounter the total immersion experience that unlocks the magic and wonder of anatomy.

This is a shame because, for the practising veterinarian, a fervour for anatomy pays significant dividends. Or, as specialist veterinary ophthalmologist and ardent anatomy advocate Dr Mark Billson[a] likes to remind people, 'anatomy is your friend'! It is hard to imagine truer words being spoken. The most fundamental of veterinary procedures, the physical examination, is based on a sound understanding of what is 'normal', in order to recognise abnormal. This should be in every new graduate's anatomical inventory. Beyond this, more specific interventions such as diagnostic imaging and surgery draw heavily on knowing where things are, what they are doing there and what they normally look like. Much of this comes with experience, and there is ongoing discussion about how much a veterinary graduate should know on 'day one'. My former manager and mentor, the mighty Dr Glenn Shea[b], believes that new graduates should have enough anatomical knowledge to assess and apply at least basic first aid to any trauma case that crosses their path. He takes this view because trauma does not discriminate. It could involve a broken limb, a deep cut to the abdomen, a blunt impact to the chest or head – the list goes on and on. What structures lurk in the affected area, what purpose do they serve and what does this mean for managing the case? Of course, veterinarians handling a trauma patient also need to enlist their knowledge of surgical techniques, wound healing, physiology and microbiology, to name just a few other parts of a complex whole, but it remains very helpful to have an understanding of the topography of the affected region.

So, can you prepare for this without the 'total immersion experience' I mentioned earlier? Can you be competent in anatomy without being passionate about it? Well let's put it this way, you can certainly pass anatomy exams without passion.

Back to Table of Contents

Gritty determination and hard work will usually get the job done. But does that make you competent? What does that even mean? These are curlier questions. I would contend that the way you learn anatomy, and the relationship you develop with it, can significantly influence your enjoyment of your overall veterinary education, as well as your confidence and competence as a clinician. And the best way forward, in my opinion, is to make anatomy 'your friend' for life.

It's tempting to say that the way to do this is to keep envisaging how the anatomy you are learning will apply to your life as a veterinarian. I don't disagree with this, and I think integration of clinical examples into anatomy teaching is hugely important. But it can be hard to draw all your motivation from this, especially in the early stages of your studies. So what am I suggesting? Well, I don't have a magic formula, but I think it's important to reclaim and retain our sense of curiosity and fascination, which is often one of the first things to go out the window when we are feeling pressured and/or intimidated by the subject matter. Unfortunately, I can't give an example of how I did this as a student, when my flirtations with fascination were subverted by a fear of failure, but I can describe how it worked for me as a teacher.

This time last year I would have been preparing to teach the anatomy of the eye and ear. These are two of my favourite topics, the eye for its beauty and anatomical elegance, the ear because it is so damn difficult. I remember only too well the first time I had to teach the anatomy of the ear. After reading the (as it turns out excellent) lecture notes left by my predecessor, I was completely flummoxed. Sure, the outer ear was pretty straightforward. The middle ear you could also get your head around (no pun intended) with a little bit of effort. But the inner ear. OMG. Completely baffling. My first thought was 'how on earth are my students going to feel when confronted with this?' Anything but empowered, most likely. As the one who had to teach it, I wasn't feeling particularly empowered either. But then I sat down to navigate the inner aural maze, one challenging

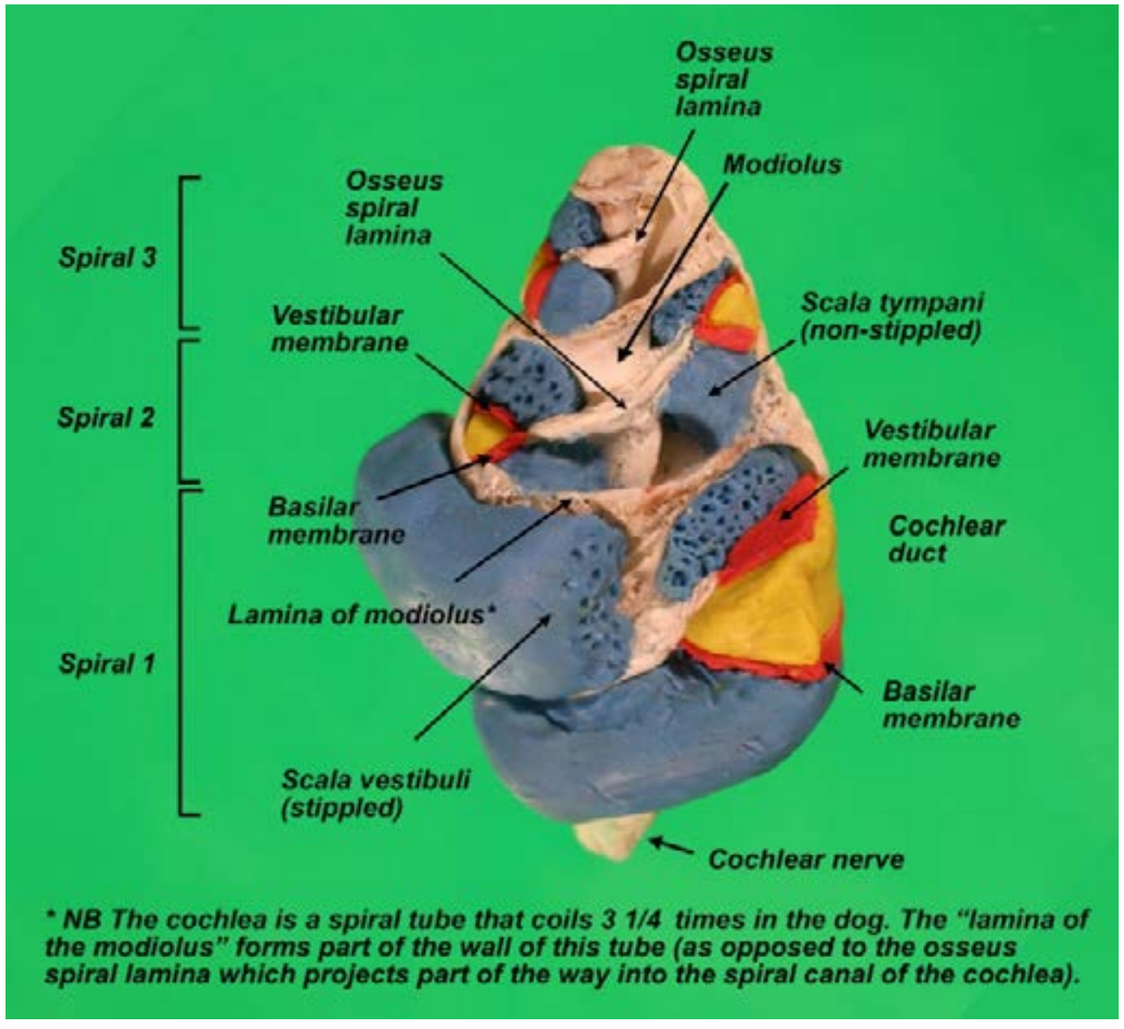

Figure 1. Plasticine and shell model of the cochlea of the dog. C Klupiec, 2010

concept at a time, with time to consult various textbooks and - crucially - time to think about what I was reading and to piece it all together. Ultimately I also made a plasticine model (see picture) - a painstaking and frustrating but ultimately therapeutic and rewarding activity. The whole process was embarrassingly satisfying. I had new respect for the inner ear (!) and I felt elated that I could now regard the lecture notes as a helpful guide (friend??) rather than a document full of mystery and threat.

Of course, most of this in-depth study was conducted on my own time, not 'company time', as I would never have been able to fit it into my normal working hours. There were opportunity costs - not a lot of housework got done that week, for example. The same thing applies to students, but the opportunity costs are a bit more serious than wearing fruity clothes because you didn't have time to do the washing. Time you spend studying one thing is time you don't spend studying something else, or earning money to get you through university, or exercising to maintain your physical and mental health, or – indeed – taking a much needed break from the rigours of study. A veterinary student might also argue that some topics, like the anatomy of the inner ear or the histology of the epididymis, are of relatively limited practical application, yet may still turn up in exams, depending on what the teachers decide to include in the great exam question lottery.

So even if you are in a head space where fascination and curiosity prevail, where do you find the time to convert your anatomy study from an exercise in memorisation into a transformative personal learning experience?

In this regard, let's compare veterinary anatomy students with backpackers. Both are motivated to see and learn about new things. Both have limited resources and are strategic in how they spend them. Both share information to help each other have the best possible experience. Both find that some things pique their interest, others seem hardly worth the effort. Both can take a few knocks as long as the journey is punctuated with highlights and moments of satisfaction. Both want to come out the other end intact, preferably with a good story to tell.

One might argue that a major difference between backpackers and students is that one group does it for fun, the other does it for the more earnest purpose of pursuing a career. OK, maybe, but backpacking is not all fun (think dodgy hotels, cancelled flights…), and it would be a great shame if studying was all drudgery. Indeed, innovation researchers Clayton Christensen and colleagues[1] purport that the two main 'jobs' that students (school students, in this case) want to get done during their education are to have fun with their friends and feel a sense of progress and achievement[1]. Sound a bit like backpacking???

So maybe there is some mileage in tackling anatomy from the perspective of a traveller, being smart about how you expend your resources so you can explore and marvel at the world around you, while also coming to grips with the more mundane bits that glue the 'wow' moments together. Not that I am the first to relate learning anatomy to travel. James E. Smallwood of North Carolina State University writes wonderfully about anatomy teachers taking the role of tour guides, regaling their audience with sometimes less, sometimes more information than they really wanted, but giving them a sense of familiarity with their surroundings and making it easier for them to orient themselves the next time they pass that way[2]. Also implicit in Smallwood's approach is the very important acknowledgment that not everything can be remembered on first viewing. That's what travel photos and textbooks are for. The important thing, though, is that you are inspired to return for another look.

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a Dr Mark Billson, Small Animal Specialist Hospital, North Ryde, NSW, Australia.

b Dr Glenn Shea, Senior Lecturer in Veterinary Anatomy, Faculty of Veterinary Science, University of Sydney, NSW, Australia.

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Back to Table of Contents

PERSPECTIVE NO. 107

LIVE ANIMAL EXPORT IS UNETHICAL

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Read the comments provides by 'Vets Against Live Export' (VALE) here

Peter is a graduate of the University of Queensland Veterinary School and the Australian Maritime College (University of Tasmania). He is a master mariner (unlimited ship size and area of operation) in the merchant navy as well as a veterinary surgeon. Peter has had command of numerous merchant ships up to 50,000 gross tonne, worked as a commercial diver to 60m deep, and practices medicine and surgery of horses. Peter's wide-ranging education and experience permits him to view the Middle East - African live export trade from a different perspective.

Live animal export is a story of corporate profiteering, politics, deceptive information and profound cruelty. It is also a story of division within society where the opponents of the trade are unyielding in their fight to end the prolonged horror meted out to the animals selected for export.

Generally it appears, most exponents for the export of living animals from Australia's shores have a pecuniary interest and their interest in economics far overshadows any compassion for the entities being exploited. The truth is that there is a very dark side to this trade and there is no way that live animal

Figure 1. Sadistic slaughter following starvation and neglect

Figure 2. Discharging and loading live cargo

export can be ethical. There is no ambiguity of the inherent cruelty of *long hauls at sea and deplorable acts of inhumanity on foreign soil.*

After viewing horrific footage of Australian domestic animals being tormented and sadisticaly butchered alive in Qatar, Pakistan, Egypt, Jordan, Israel, Kuiwait, Mauritius and Indonesia it is now evident to most Australians that exporting live animals to these countries is unethical and only supports further depravity. 'You may choose to look the other way, but you can never say again that you did not know' (Lyn White, 16/06/2011).

Live animal export is reported to be 0.3% of total exports from Australia (2012)[1]. The importance of exporting live animals could hence be considered not that important to our economy especially when evidence[2] exists that the alternative option of processing meat at the closest point of production would be more productive in terms of monetary return and employment. It is a myth to suggest banning cattle export would destroy the beef industry. Australians do not owe the rest of the over populated world sustenance in the form of live animals to be sadistically slaughtered after being tormented to the extreme. Australia is a reliable and trustworthy trading partner; however, the recipients of our live animals repeatedly fail to respect our animal welfare standards. Once the animals depart the wharf there is no guarantee of safe passage and that the persons receiving live animals will be benevolent and all animals stunned before slaughter. Australian boxed meat has a shelf life of 4 months and is the most efficient means of sending meat overseas. In those countries where refrigeration is an issue, those without usually cannot afford to eat red meat anyway. Without refrigeration, slain animals need to be consumed as soon as possible, or salted down for preservation. Most cargo vessels already lift frozen products either in their own refrigerated rooms

Figure 3. Old converted bulk carrier

or on or below deck in refrigerated containers.

Meat importers in the United Kingdom have said they will take as much frozen boxed meat that Australia can provide and even that would not be enough.

Animal welfare has been researched for many years and slaughter without stunning is highly controversial. Welfare issues are far greater for cattle than sheep as they take longer to lose consciousness after having both carotid arteries severed. In cattle the anatomy of the brain is somewhat different in that they have vertebral arteries that are usually not severed by the slaughter technique (Kosher or Halal). These vertebral arteries continue to supply the brain with oxygenated blood. That is, when the carotid arteries are severed during slaughter the brain still has a blood supply. Professor Temple Grandin from Colorado State University concluded cattle take a longer period of time to become insensible. 'The time to loss of insensibility when good cutting technique is used will range from 17 sec to 85 sec (Blackmore 1984, Blackmore et al 1983, Gregory and Wotton 1984, Grandin 2010, Daly et al 1988, Gregory et al 2010). Some cattle may have prolonged periods of sensibility lasting up to 385 seconds (Blackmore, 1984)...'

One would expect dairy cows and breeding stock to have some level of 'protection' commensurate with their value; sadly, however, this is not the case. Numerous occurrences have been recorded of sheep and cattle dying of neglect in the Middle East and elsewhere. On 18th September 2012 the Australian Broadcasting Corporation reported one typical case where high value Victorian dairy cattle were sent to Qatar. An Australian veterinary technician (AVT) was invited to inspect the farm and found a lack of infrastructure to support the thousands of sheep, goats and cattle coming from Australia. The AVT agreed to stay on as a consultant to remedy the problems on the farm and subsequently returned to Australia for a 10 days break. Upon her revisit to the Qatar farm she discovered the animals hadn't been fed whilst she was away and were dying in 50ºC heat. 10,000 sheep had been exported to the Qatar farm in February 2012 and 7,000 sheep had perished from lack of feed, water, and heat stroke (failed thermoregulation). The farmer of the dairy cows (vendor) became informed as to her animals' fate and pledged she 'will never export cattle ever again'.

The live export ships are frequently converted versions of a previous trade. Blowers and extraction fans have always been part of a ship structure and of course there should be adequate

Figure 4. Extreme fear, unpreventable disease (multi-system failure and subsequent death) and suffering are synonymous with 'long hauls' at sea

feed and water however a veterinary surgeon is not always on board. Stocking density has always been an issue and the pens' structure, including flooring, and size is not always satisfactory.

There is a marked difference between vessels undertaking *short hauls* to ports close to Australia, such as in Indonesia, as opposed to *long hauls* to the Middle East, Russia and Africa. On the long hauls where the majority of ships commissioned to transport Australian animals are old former oil tankers, bulk carriers, container ships and vehicle transporters. Most are 30 years old or more. Many have had their names changed to conceal a poor history. No livestock ship is registered in Australia but flagged out to other countries such as Singapore and Panama. All operate with foreign crew and none carry Australians apart from an Australian Government accredited veterinarian (AAV) (when carried), a head stockman, and very rarely an Australian master. Some ships do, however, have Australian names such as the Wellard owned vessels 'Ocean Swagman' and 'Ocean Drover' (previously MV 'Becrux') that are registered in Singapore. The intent is to earn some public approbation. Machinery breakdowns do occur and will continue to occur despite backup systems. Ventilation and adequate air changes, and many other aspects of the whole seagoing operation are always at risk of becoming compromised. In many instances the ventilation is grossly inadequate and heat stress is an ongoing issue in equatorial zones.

Animal based indicators and clinical evidence of disease obtained from veterinary examination, such as excessive panting, coughing, cleanliness, lameness, demeanour, injuries, skin lesions, nasal discharge, and diarrhoea provide reliable signs that the welfare of the animals on board a ship is severely impaired. Indicators and clinical signs of this nature, and death, are common place on long hauls. Weight gain on short hauls is not a reliable indicator of animal welfare.

Notwithstanding the improvements in ship design, improved technology, scientific and practical knowledge, unpreventable *morbidity* and *mortality* will always occur. The mortality rates will vary from what are considered within normal limits to excessive. Proponents of the trade argue that low death rates are an indicator of trifling cruelty. For example, because only 0.44% of the sheep die out of 65,000 sheep then that represents very little cruelty having occurred. This, of course, is an incorrect assumption or fallacy.

The Veterinary services provided to export animals have too frequently been sub-optimal and not at a standard acceptable elsewhere in the wider community. Photographic evidence is available to demonstrate repeat violations of the Australian Standards for the Export of Livestock (Table A3.1.2 ASEL) in that veterinary inspections have failed to detect and reject animals according to this code (ASEL). Examples of this include: failure to detect at pre-selection and final inspection cases of excessive weights of cattle, fly blown dehorned cattle, excessive wool, and salmonella infected carriers, cases of keratoconjunctivitis, scabby mouth, advanced pregnancies, hernias, foot rot, and inanition (failure to eat). Relying only on visual inspections to detect 'diseased' animals seems ineffective. We know sheep carrying salmonella, whilst not showing evidence of disease, walk on the ships undetected. In addition, there have also been cases where sheep with severe diarrhoea have been loaded. Laboratory testing (bacterial culture, ELISA, PCR, etc.) is not routinely carried out because many mobs of sheep may show up positive to *Salmonella* spp. Extreme fear, heat stress, unpreventable disease, injury, brutality and agonising death, all take place at some stage throughout 'long hauls' at sea.

Australian society expects their veterinarians to look out for the welfare of all animals and it is enshrined in some states legislation to do so. NSW veterinarians now have to declare they will promote the welfare of animals under the Veterinary Practice Act 2003. Unfortunately veterinarians on-board the live export ships are not independent. They are employed by the exporter. In 2011 there were 121 listed AQIS* accredited veterinarians and only 7 did three or more voyages. Whilst veterinarians are being selectively chosen and paid by the exporters there will be a serious conflict of interest. Independent veterinarians employed by DAFF Biosecurity (AQIS) have been recommended repeatedly but resisted by industry. Many on-board vets have had their employment terminated because they have submitted disapproving voyage reports. Verification is available to demonstrate AAV's (employed by the exporters) and AQIS veterinarians have repeatedly breached Table A3.1.2 ASEL in that they have failed on many occasions to comply with rejection criteria.

Conducting review after review is not going to improve animal welfare when ongoing calamities of the trade are unpreventable and successful prosecutions for breaches of state government Prevention of Cruelty to Animals (POCTA) legislation and the Australian Standards for the Export of Livestock (ASEL) have not occurred. As time goes by, it becomes more evident that any investigation undertaken by the Department of Agriculture, Fisheries and Forestry (DAFF) is tainted by the fact that DAFF both promotes and investigates the trade therefore by law their findings would be invalid. It follows then that these continuing reviews amount to being a sham. The industry has been beleaguered by ongoing disasters year after year since the late 1970's notwithstanding successive Governments, pertinent primary and subordinate legislation, veterinary research, enhanced shipping technology, numerous reviews, numerous

Figure 5. Evidence of extremely poor animal welfare

Figure 6. One of 650 diseased cattle mid Indian Ocean bound for Saudi Arabia

*Note: The Australian Quarantine and Inspection Service (AQIS) is now the Department of Agriculture, Fisheries and Forestry (DAFF) Biosecurity.

Authors' views are not necessarily those of the CVE

Back to Table of Contents

CVE *Control & Therapy Series* – **Issue 275** June 2014

Figure 7. Sheep so sick they will unlikely receive an adequate dose of antibiotic

reports and the development of the Australian Standards for the Export of Livestock (ASEL). An Order under the Australian Meat and Livestock Industry Act 1997 refers to ASEL however there exists a divergence of opinions as to whether these standards are actually enforceable.

As it stands at the moment, DAFF requires a veterinary surgeon to accompany animals on voyages > 10 days, and on 10% of other randomly chosen voyages (these would be the 'shorthauls'). This recommendation by the Keniry Review meansa veterinarian is not on all ships exporting live animals. DAFF may also place a veterinarian on any voyage it deems fit and I assume this decision would be in accordance with the exporter's risk assessment. Decisions to mitigate risk should be based on scientific, ethical, and practical merit and not influenced by economics particularly where the welfare of the animals is at stake.

When you consider the high risks to animals on the ships it is difficult to accept that the treatments offered to mitigate some of these risks such as thermal injury are entirely genuine. For example, to expect one veterinarian to thoroughly look after say, 120,000 sheep or even 65,000 sheep plus 650 cattle 24hrs a day X 7days a week for 3 weeks is an impossible task. An AAV's role is to make reports, perform autopsies, diagnose disease, and attempt treatments.

Since Australia is a signatory of the World Organization of Animal Health (OIE) then full compliance with the Terrestrial Animal Health Code should be a priority. Poor veterinary services, incorrect recording, falsifying biostatistics, poor animal husbandry practices including working animals in extreme heat, the use of electric goads and inattentiveness to density when loading and transporting, all amount to poor governance and disregard for animal welfare. Having exposed inconsistency and inaccuracy, reliance on any biostatistics offered by export companies, DAFF, MLA and LiveCorp must be viewed with caution.

Stress is a major factor in live export trade. It is accepted in the scientific forum that extremely stressed animals are more likely to submit to disease processes such as Salmonellosis/ Enteritis Complex.

In the wake of the 'Four Corners' expose in 2011 it became evident Australian Livestock Export Corporation (LiveCorp), Meat and Livestock Australia (MLA), and the Australian Quarantine Inspection Service (AQIS) disgraced themselves by not reviewing performance more thoroughly and initiating corrective action when required, long ago. In a letter to members published on the MLA website, Chairman of the day Don Heatley wrote: 'A decade's worth of industry and government representatives, plus independent animal welfare experts had not seen such cruelty - or something would have been done.' It is difficult to believe MLA, LiveCorp Board, and members of AQIS did not know what fate awaited these animals. The marketing manager of Wellard Rural Export admits he knew. It appears so many people have failed dismally in their responsibilities to guarantee our export trade.

Figure 8. An Australian sheep two weeks out of Fremantle bound for the Middle East

The marketing manager for Wellard at this time claimed to have personally sold more than *1.5 million* head of cattle into Indonesia since 1991. He divulged the largest privately owned abattoir in Jakarta killed 4,000 to 6,000 cattle a month and was 'working on getting a stun system in place'. If the estimated figures quoted are correct then approximately 72,000 cattle were killed yearly without stunning at this abattoir. Another independent source reported 450,000 Australian cattle were killed without stunning in Indonesia. The marketing manager said 'I have watched literally thousands of cattle slaughtered in the boxes in Indonesia. Yes, there are problems... but 98% of the cattle I watched killed was quick and without fuss'. If this account is to be believed, 2% of 72,000 head of cattle equals 1,440 cattle that, one must assume, were brutalized in a similar manner as seen on Four Corners at just that single abattoir. The manager admitted the eight or nine other crews that slaughtered cattle at the same abattoir nightly had even lower standards. Simon Illingworth wrote 'A handful of rogue slaughtermen captured on film should not have stopped a billion dollar industry that supplies food'. Attempting to justify and reduce the enormity of their actions by equating to dollars is contemptible.

Large amounts of money have been granted to recipients of Australian livestock so as to educate them in matters of animal husbandry and animal welfare. Egyptian abattoirs were granted large amounts of money by a humanitarian institute in order to stop the insane brutality when slaughtering livestock however on subsequent inspection nothing had changed (Pers comm: Philip Wollen AOM, 2013).

The value of Australia's live cattle exports in 2012-13 was *$590 million*. Live cattle export hence only amounts to approximately 1.26% of the total value of agriculture. It may not be far from the truth to accept live export amounts to only 5% of the whole livestock industry, and of this about 3% would be sheep and goats on long hauls (> 10 days) to the Middle Eastern and African countries. The Australian Bureau of Statistics (ABS) is the officially recognised Australian government agency that collects and publishes statistical information about Australia and its people. In 2011-12 the value of Australia's total agriculture was *$46.7 billion*. In recent times, the agricultural commodities with the highest value of production by Australian farmers have been cattle and calf slaughterings, followed by wheat, milk, vegetables, fruit and nuts, sheep and lamb slaughterings, and wool. In 2012-

Figure 9. Veterinary surgeon performing daily autopsies

Back to Table of Contents

13 Australia exported 67% of its total beef and veal production to more than 100 countries. The value of total beef and veal exports in 2012-13 was *$5.06 billion*. The beef industry contributes 13% to total Australian farm exports

Farmers are the key players in live animal export. It is farmers who need convincing there are equally profitable and more humane alternatives domestically. Financial reasons should not constitute justification for the inherent cruelty of long hauls at sea and deplorable acts of inhumanity in foreign countries; however, faced with indoctrination about financial hardship that trade cessation will cause, farmers are left confused with tough choices when agents, exporters, Meat and Livestock Australia, shipping companies, importers and overseas processors (who, at times, are the same entity. Example: Wellard) all push their own agendas, and encourage them to keep producing animals for trade. These corporations paint a grim picture of the farmers' future without it, despite independent economic analysis that suggests otherwise. There are still farmers who will not be discouraged from sending more of their animals to these locations. Farmers do have a choice at sale yards by stipulating the sale of their animals is for 'meat only' and not for live export.

Figure 10. Peter and his wife Susie (in earlier years) with a heifer bound for Lord Howe Island

Compliance with the Commonwealth Government new system of tracing animals from farm gate to slaughter (Export Supply Chain Assurance Scheme (ESCAS)) has also failed miserably and will continue to fail as Australia cannot possibly regulate conditions worldwide. One of the exporters stated it was too ambitious and another, when faced with sheep being hurled and booted out of the ship in Israel recently, stated it was very difficult to control the situation at the end destination.

Like farmers, veterinarians also play a pivotal role in the live trade and as the farmers are becoming divided so is the veterinary profession. A difference of opinion has evolved between those for and those against. As a consequence of the utter failure of successive Commonwealth Governments to protect the welfare of animals throughout the live export chain, a growing number of veterinarians have registered their opposition to live export by forming an association called Vets Against Live Export Inc. (VALE). VALE acts as a repository of information about the live export industry and associated animal health issues. The association specifically aims to focus on veterinary and scientific concerns and expose corrupt practices in live export. VALE also endeavours to raise public awareness and provide objective information to the public and veterinary profession on issues relevant to the live export of farm animals hoping ultimately to influence and change government policies relating to live export.

This manuscript is not intended to be about politics but about the millions of innocent animals sent by Australian farmers on long haul sea transport vessels to recipients that practice ongoing sadistic acts of cruelty. The policy of exporting animals live should cease immediately and this should be non-negotiable.

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ANIMAL WELFARE – VIEW THE ROBERT DIXON ANIMAL WELFARE VIDEOS

The greatness of a nation and its moral progress can be judged by the way its animals are treated
– Mohandas Gandhi*

Since 2011, The University of Sydney has hosted the annual Robert Dixon Animal Welfare symposium. Go to our ebook to view videos of past Robert Dixon Memorial Symposia or visit www.cve.edu.au/animalwelfare.

2014	Does Australia breed companion animals ethically?
2013	The economics of welfare in intensive farming
2012	How should animals be used in the teaching of veterinary medicine and animal science?
2011	Should Australia export live animals for slaughter?

Dr Robert Dixon (1951-2011)
Champion for Animal Welfare

Robert Dixon graduated from the University of Sydney in 1973 with a BSc(Vet) degree before completing his BVSc degree in 1974. Robert completed his PhD in 1980 in New Zealand and returned to the University of Sydney in 1983 to take up an academic appointment within the Faculty of Veterinary Science. For many years Robert held the Faculty position of Sub-Dean Animal Welfare as well as serving on the University Animal Ethics Committee. Robert impressed his friends and colleagues with his determination, passion, positive attitude and sense of humour as he battled with ill-health throughout his life.

*Robert's emails finished with this quote.

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