C&T

Control & Therapy Series

Issue 309
December 2022

Celebrating the International Day of Veterinary Medicine

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Centre for Veterinary Education

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C&T

Issue 309 | December 2022

***Control & Therapy* Series**

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Small Animal

General

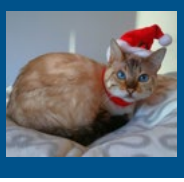
Call for Cases

WildLife

Large Animal

Happy holidays!

Our team is taking a break from Thursday 22 December 2022 and we'll be back on deck on Monday 9 January 2023.



ENGAGE WITH YOUR PROFESSION

Established in 1969, this unique veterinary publication celebrates over 50 years of veterinary altruism. An ever-evolving forum gives a 'voice' to the profession and everyone interested in animal welfare. You don't have to be a CVE Member to contribute an article to the *C&T* Series. Send your submissions to Dr Jo Krockenberger: joanne.krockenberger@sydney.edu.au

"I enjoy reading the C&T more than any other veterinary publication."

Terry King
Veterinary Specialist Services, QLD

Thank you to all contributors

The *C&T* Series thrives due to your generosity. If you're reading this and have been contemplating sending us an article, a reply or comment on a previous *C&T*, or would like to send us a' What's YOUR Diagnosis?' image and question or seek feedback from colleagues, please don't hesitate to contact us.

The *C&T* is not a peer reviewed journal. We are keen on publishing short pithy practical articles (a simple paragraph is fine) that our readers can immediately relate to and utilise. And the English and grammar do not have to be perfect—our editors will assist with that.

Join in—write up that interesting case

C&T authors agree that it is extremely satisfying to read their articles in print (and the digital versions) and know they are contributing to veterinary knowledge and animal welfare.

Winners

Major Winner
Prize: A CVE$400 Voucher

Best Visuals
Prize: Digital video or DVD. Visit cve.edu.au/cveshop to peruse our list of titles.

Winners
Prize: A CVE$100 voucher

FROM THE DIRECTOR

I can't think of a more fitting end to 2022 than with a front-cover celebration of the contribution made by people in veterinary science all over the world. I often ponder (with gratitude) the versatility this profession allows. In clinical practice you can see many species or become an expert in one. You can dabble in multiple aspects of diagnostics and treatment or narrow your focus and specialise. You can be involved in research and leading the way in new understandings and innovations or be a passionate educator of the next – or current – generation of vets and nurses. And the reality is, many of us do more than one of these things – plus a heap of other stuff!

So, here's to everyone in the profession – whatever it is you're doing or how many days a week you spend doing it – for the contribution you make to animal welfare, human wellbeing and global betterment.

As always, there is some ripper content in this edition of the C&T, whatever your area of interest. I found Rick Atwell's recommendations on the use of hot water bottles particularly interesting (41°C doesn't seem that hot – it surprised me that this temperature might cause harm in as little as 2.5 minutes!). Jeremy Rogers' contribution on *Phalaris* toxicity in kangaroos is also fascinating; and for those in NSW I encourage you to take a look at Christine Griebsch' update on leptospirosis. Robin Stanley's ophthalmology myth-busting is essential reading for everyone in the clinic. And here's a challenge to finish things off – try not to say 'aww' when you see Sue England's photographs of cats in onesies!

Happy reading – and all the very best as we usher in 2023.

Simone

Winner

Entitled to a CVE$100 voucher

Small

WINNER OF WHAT'S YOUR DIAGNOSIS?

How would you treat this case?

C&T No. 5947 (Issue 308, Sept 2022)

Robert Mills
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C&T No. 5948

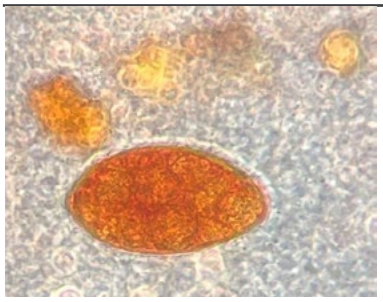


Photo courtesy of Sue Jaensch

This ova looks like Spirometra spp. (*Spirometra erinacei*). These are also known as 'zipper' worms and are a reasonably common tapeworm when you go looking for them. They are far more common in higher rainfall areas.

The egg is embryonated with an operculum at one end of the shell and there are usually plenty of them on the slide when found.

These parasites are most commonly found in cats, particularly cats that are allowed to hunt.

The most important aspect of this diagnosis is to understand that the tapeworm is acquired through predation, as the larval (metacestode) stage is found in the tissue of intermediate hosts (usually fish and frogs but can also be ingested by accidental ingestion of copepods containing the metacestodes when drinking from ponds and waterways). Cats and dogs become infected by ingesting these intermediate hosts.

As the species is a tapeworm, it is treated with praziquantel at a higher than standard dose rate. The dose needs to be increased from the standard dose of 5mg/kg PO to a dose of 4-8 times this (20-40mg/kg PO).

We usually recommend a follow up in-house faecal float in 3 months after a treatment and then every 3-6 months, particularly if it is a farm cat.

Heavy infestation with zipper worm left untreated can cause severe ill thrift and even lead to intestinal obstruction. I have performed one intestinal resection and anastomosis in a cat with such a large burden of Spirometra that it had a small intestine completely obstructed with adult tapeworm. ◆

Congratulations!

Robert wins the voucher which may be used towards membership, to enrol in CVE courses or to purchase CVE products from CVeSHOP.

Robert is a 1995 graduate of Melbourne University Vet School with a background in dairy, mixed and companion animal practice.

He followed the path of many over to the UK to broaden his experiences and thoroughly enjoyed the adventures undertaken through Great Britain, Europe and Africa.

Homesickness brought him back to Australia where he eventually set up a thriving practice in a growing area to the North of Coffs Harbour where he currently works. His practice is predominantly companion animals but they still manage to service a few large animal clients.

As a veterinarian Robert is still very humbled by the vocation that continues to teach you that you will never stop learning from both your successes and your failures (yes, his post mortem knife is still brought out when needed).

His areas of interest mostly involve eyes or anything surgical.

Robert is a keen surfer and loves fishing. His eldest daughter is kept busy with horse riding events after starting at pony club from a very young age.

Looking to the future, he plans to continue to grow the practice and hopefully work smarter rather than harder.

Celebrating the International Day of Veterinary Medicine

December 9, 2022

Meet our Cover Stars!

Each year on December 9, veterinary professionals and organisations around the world are recognised for their amazing efforts in promoting and protecting the welfare of animals and humans alike – we can't thank you enough!

We hope you enjoy this special issue of C&T which showcases vets and vet nurses from Australia and across the globe.

Meet our Vets and Vet Nurses!

Opposite page, from top to bottom, left to right

Vet Nurse Tracy Goodman, Quirindi Vet Clinic (APIAM), NSW

Dr Brad Robertson and Chasca, VSA Auckland

Vet Nurse Simone McCormick and Angel, The Cat Clinic Hobart, TAS

Dr Georgia Burton with Rufus and Flash, Concord Veterinary Hospital, NSW

Dr Jen McCormack and Sir Romeo Salvatore, North Hobart Veterinary Hospital, TAS

Dr Johanna Tait and Odin, Ultimate Veterinary Clinic, VIC

Vet Nurse Hannah Fitzgerald, The Cat Clinic Hobart, TAS

Dr Jorge Baron Morris and Lexi, Ballarat Veterinary Practice, VIC

Dr Leah Puk and Biscuit, Paddington Cat Hospital, NSW

Dr Nadia Burns and Gonzo, Warby Street Veterinary Hospital, VIC

Vet Nurse Montana Green, The Cat Clinic Hobart, TAS

Dr Polly O'Brien and Bertie, Geelong Animal Welfare

Vet Nurse Jordan Nichols and Nicky, The Cat Clinic Hobart, TAS

Dr Samantha Singleton and Hugo, Shoalhaven Veterinary Group, NSW

Dr Sophie Mead, Orchard Hills Veterinary Hospital, NSW

Vet Nurse Tracy Coghlan with Ellie-May and Maggie, Scenic Rim Veterinary Service, QLD

Dr Nonie Coutts and the gang, Dr Nonie Coutts Veterinary Surgery, Bahrain

Dr Rhys Powell and Sampson, Local Land Services – Orange, NSW

Vet Nurse Helena Evans and Louis, The Cat Clinic Hobart, TAS

Meet our Cover Stars

C&T, Issue 309, December 2022

Pictured from top to bottom, left to right

Sarah Daphne Foo and Ivy, The University of Sydney, NSW

Dr Bree Talbot, Byron Bay Wildlife Hospital, NSW

Dr Mitzi Walker and Ali, Greencross Vets – Moorooka, QLD

Dr Sy Woon and Simba

Dr Michael Ferguson, Wauchope Veterinary Clinic, NSW

Dr Georgia Ladmore and Cassie, Orange Vet Hospital, NSW

Dr Anne Quain and Hero, The University of Sydney, NSW

Dr Maayan Tourel and Rutie, Struggletown Vet Hospital, NSW

Katrina Cheney and Ashton, The University of Sydney, NSW

Dr Sam Kovac and Bonnie, Maddie and Clara-Belle, Southern Cross Vet, NSW

Dr Wong Shaw Feei and Kristal, USJ Animal Clinic, Malaysia

Dr Natalie De Souza and Bella, Kirrawee Veterinary Hospital, NSW

Dr Leah Padman and Bentley, Petstock West Gosford, NSW

Dr Jenny Warland and Pepper, Dr Paws Veterinary Clinic, NSW

Dr Adriana Barcia Gorria with Nestor, Uruguay

Dr Jess Lawson and Ned, Your Family Vet, SA

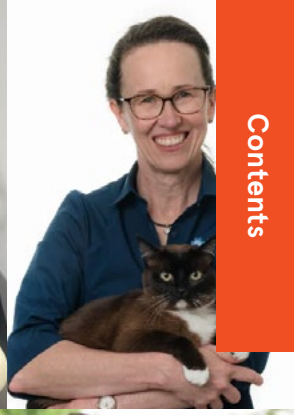
Dr Tunbi Idowu and Kai, Zoetis, NSW

Dr Edward Bassingthwaite, Whole Energy Body Balance, Australia

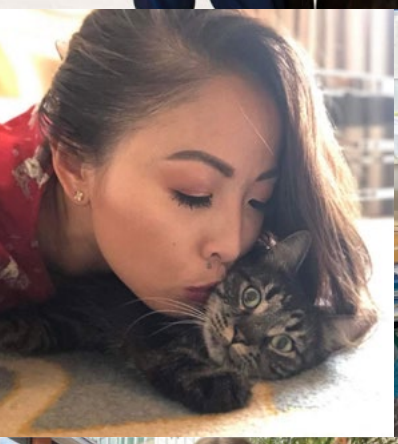
Dr Lesca Sofyan, Orchard Hills Veterinary Clinic, NSW

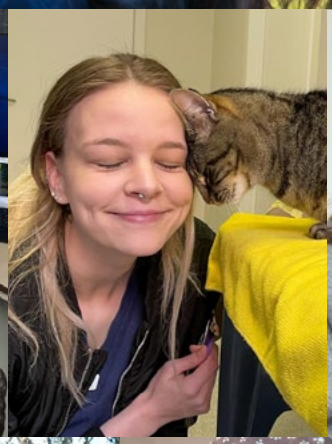















Sophie
Veterinary Nurse



EZYDOG


MAJOR Winner

The prize is a CVE$400 voucher

Small

CANINE *SPIROMETRA* INFECTION

Stephen Laing BVetBiol/BVetSc
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C&T No. 5949

Stephen graduated from Charles Sturt University in 2015 with a Bachelor of Veterinary Biology and Bachelor of Veterinary Science. Over the years he has worked in mixed and small animal veterinary practices throughout regional New South Wales and Victoria.

More recently Stephen operates his own large animal (farm animal and equine) ambulatory practice in the Southern Highlands NSW, whilst also working as a locum in small animal emergency practices.

Stephen enjoys all areas of small and large animal veterinary practice, with particular interests in small animal medicine and cattle reproduction, medicine and surgery.

History

A 7.5-month-old female spayed Labrador presented to the emergency centre for lethargy and reduced appetite after defecating a large tapeworm (Figure 1) 18 hours prior. The dog had been defecating tapeworms for several weeks, but the owner became increasingly concerned as this was the largest, they had seen and the dog was not herself.

Figure 1. The tapeworm that was passed.

At 3-months-of-age, the owner noticed worms (suspect roundworms) in the faeces. The owner started the dog on Nexgard Spectra® (afoxolaner and milbemycin oxime) monthly at the label dose.

At 5-months-of-age the owner noticed tapeworm segments in the faeces. The dog was started on Drontal (febantel, praziquantel and pyrantel) at the label dose every two weeks. Despite this treatment regimen the dog continued to defecate tapeworm segments.

In the two weeks prior to presentation the dog had started intermittently regurgitating / vomiting at night, was slow to eat (normally it was a ravenous eater) and became lethargic.

The dog's diet was commercial dry food, pigs ear treats and occasional fresh bones. No offal was fed. The dog lived in suburbia with no access to carcasses.

The dog regularly swam in the local river.

Examination / Diagnostics

The physical examination was within normal limits.

The owner provided a sample of the tapeworm during the consultation which had the appearance of a zipper down the middle (see photo 2 – note this is preserved and not fresh). This is characteristic of Spirometra tapeworm.

A faecal sample was sent to Vetnostics for a faecal float which identified ova of *Spirometra* tapeworm seen (Figure 3).

Figure 2. The tapeworm at closer resolution, after fixation in ethanol.

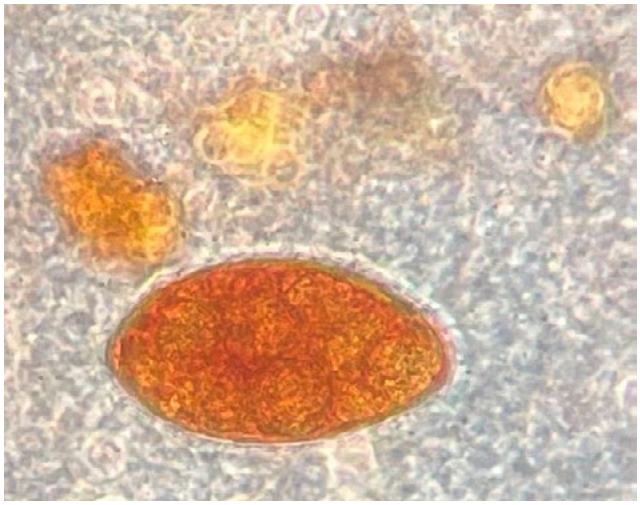


Figure 3. Spirometra oocyst in faecal float. Note the hexacanth embryos. Photo courtesy of Sue Jaensch.

Diagnosis

Based on the appearance of the adult tapeworm and the operculated eggs on the faecal float *Spirometra erinaceieuropaei* ('zipper worm') was diagnosed.

Treatment

Treatment for *Spirometra* tapeworm was started based on suspicion (confirmed diagnosis the next day with the faecal float and morphological examination of the gross specimen) with praziquantel at ~22.5 mg/kg PO q24hr for two consecutive days. **Note this is about four times the standard label dose for tapeworms.**
An injection of maropitant at 1 mg/kg SC was also given to help with nausea and vomiting.

A couple of days after starting treatment the owner advised the dog was back to her normal self.

This case is a reminder that identification of tapeworm species in infected dogs and cats is important given that *Spirometra* spp. require MUCH higher doses of praziquantel.

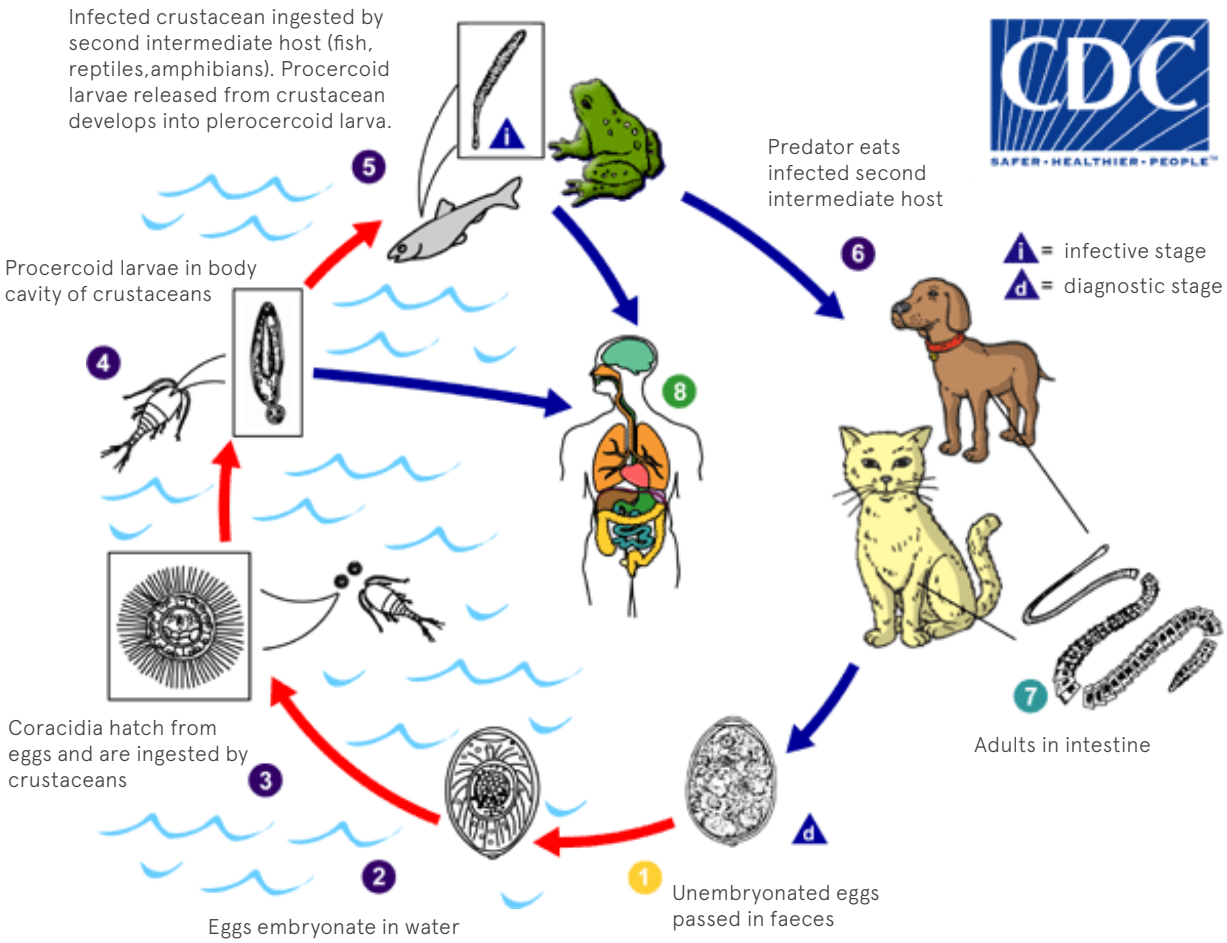


Figure 4. Centers for Disease Control & Prevention
cdc.gov/dpdx/sparganosis/index.html

Discussion

Spirometra spp. have an indirect lifecycle, requiring two intermediate hosts. Dogs, cats or foxes (the definitive hosts) infected with adult *Spirometra erinaceieuropaei* shed eggs in their faeces. In water these eggs embryonate, with coracidia hatching that are then ingested by copepods, the first intermediate host. Within the copepod the coracidia develops into a procercoid.

These infected copepods are then ingested by second intermediate hosts such as snakes, tadpoles/ frogs, lizards, chickens, dogs, cats, pigs, but not fish. Within these second intermediate hosts the procercoid larvae develop into plerocercoid larvae (also called sparganum) and infect the muscle and connective tissues.

Dogs, cats or foxes become infected with adult tapeworms when they ingest an infected second intermediate host, completing the lifecycle. They can also become infected as paratenic hosts if they ingest infected copepods in drinking water, resulting in sparganum in their tissues.

Humans can also become infected with sparganosis by ingesting infected copepods (in drinking water) or under cooked second intermediate hosts.

It is unknown how exactly this dog became infected. The dog swam regularly (multiple times per week) in the local river and so it is possible that given this exposure it was at higher risk to ingestion of an infected secondary intermediate host (i.e. frog).

In addition, many tapeworm species (including *Spirometra* spp.) pose a human health risk, whether that be directly or indirectly.

Acknowledgement

Thank you to Dr Richard Malik for his much-appreciated guidance on this case. Thank you also to Dr Sue Jaensch from Vetnostics for providing the photo of the ova on the faecal float.

References

Australasian Animal Parasites Inside & Out, 2015

parasite.org.au/wp-content/assets/Parasitology2015.pdf

Molecular identification of *Spirometra erinaceieuropaei* infection in a dog with its successful treatment 2018. ◆

Call for Cases

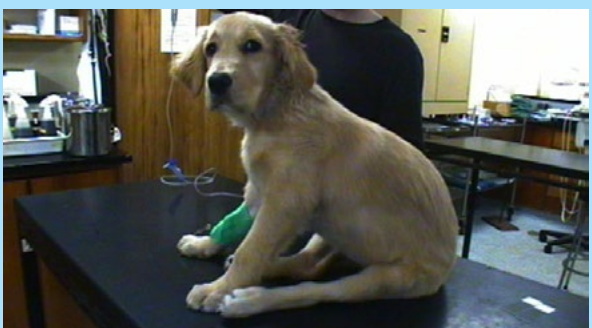
CAN WE PREVENT RAT LUNGWORM DISEASE (NEUROANGIOSTRONGLIASIS)?

Richard Malik, Rogan Lee & Jan Slapeta
CVE, Westmead Hospital &
Sydney School of Veterinary Science
e. Richard.Malik@sydney.edu.au

Rat lungworm disease has become one of the most common causes of meningitis in dogs along the warmer parts of the east coast of Australia.

We think that monthly moxidectin will prevent dogs developing infection based on theoretical concepts and some research in experimental rats. It is proving very difficult to do direct experiments to prove this.

Can you help me?



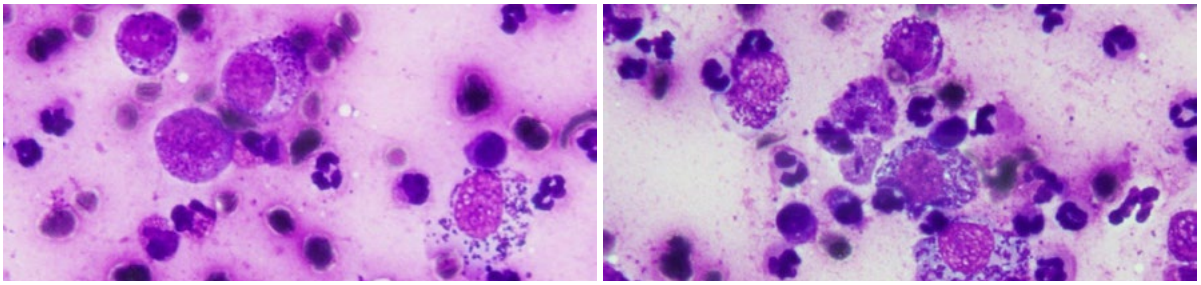
As a result, we are interested in obtaining data on dogs with naturally occurring rat lungworm disease, specifically, what routine heartworm/tick preventative they had been receiving in the lead up to developing the disease. So, we are asking clinicians who have seen cases of this condition to look back into their records and extract data on what and when the dogs were given in terms of prophylactic worming/heartworm prevention e.g. Bravecto, Bravecto Plus, Simparica, Nexgard, Advocate etc. We realise some pups will not be on any monthly preventative and would like to document this also.

Cases	Monthly Preventative Given (which one? Or nil?)	Timing of monthly preventative relative to developing neuro-angiostrongyliasis
Age, Breed, Sex		

Please provide an email address for follow up questions.

We know it takes time and effort to look up records, but we would be very appreciative.

Thank you. ◆



Mast cell tumour cytology. Photos courtesy of Mark Krockenberger Sydney School of Veterinary Science

Small

STELFONTA®: A NEW MAST CELL TUMOUR TREATMENT OPTION FOR DOGS

Dr Joy Yan Ziea BVSc (Hons)

Dr Harvey Saunders BVSc MANZCVS

Dr Becky Leung BVSc (Hons)

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The Australian Animal Cancer Foundation
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C&T No. 5950

Introduction

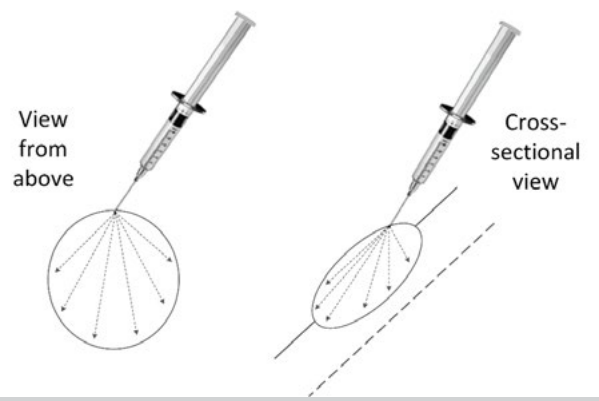
Tigilanol tiglate (STELFONTA®), a novel small molecule derived from the seed of a North Queensland rainforest tree *Fontainea picrosperma*, has been approved by the Australian Pesticides and Veterinary Medicines Authority (APVMA) for the intratumoural treatment of non-metastatic mast cell tumours (MCTs) of any cytological grade. Cutaneous MCTs may be treated anywhere on the body, head, or legs, and subcutaneous MCTs can be treated at or distal to the hock or elbow.[1] Tigilanol tiglate is a potent cellular signalling molecule with a multifactorial mode of action involving induction of a localised acute inflammatory response, immune cell recruitment to the treatment site and disruption of tumour vasculature. At efficacious intratumoural doses, these processes cause haemorrhagic necrosis of the target tumour and its destruction results in the creation of a treatment site tissue deficit that should be left unbandaged and generally heals uneventfully without the need for direct veterinary intervention.[2–7] A randomised controlled blinded clinical trial of tigilanol tiglate for treatment of canine MCTs at 11 sites was carried out in the US. A complete response rate of 87% (68 out of 78 patients) was achieved with one or two injections with a second injection given if a complete response was not achieved at an assessment 28 days after the first.[2] Longer-term response durability was assessed for that study and at 12 months of the evaluable patients that had a Day 28 complete response following a single treatment, 89% (57 out of 64) were still recurrence-free at the treatment site.[8]

Here we report a case study, Lucy, an 8-year-old female neutered Bull Arab cross that was referred to Brisbane Veterinary Specialist Centre for enrolment in an Australian study of tigilanol tiglate in the treatment of canine MCTs at four specialist referral centres during 2021 and 2022.[5]

Treatment and Response

Lucy presented with a single MCT on the medial aspect of her left thoracic limb distal to the elbow (Figure 1). She had no previous history of mast cell tumours, had no palpable regional lymphadenomegaly and the findings on her physical exam were otherwise unremarkable. A fine needle aspirate of the lesion was taken and submitted to Independent Veterinary Pathology (IVP, Australia) for cytological grading using the Camus system.[9] Lucy met the tigilanol tiglate treatment criteria, a treatment day was determined, and the concomitant medications dispensed. An essential aspect of the tigilanol tiglate treatment is the mandatory concomitant medications to reduce the potential risk of side effects associated with mast cell degranulation[2,4,5,10] and to control pain associated with the local inflammatory response induced at the tumour site by the drug. Lucy commenced prednisolone 2 days prior to treatment day and both chlorpheniramine and famotidine on the day of treatment (Table 1).

Table 1. Protocol summary for an intratumoural tigilanol tiglate treatment of a mast cell tumour.

Protocol Component	Description / Comments
1. Concomitant medications	
a. Mandatory	• Corticosteroid (start 2 days prior to treatment day) ○ Prednisolone: 0.5mg/kg b.i.d. for 7 days then s.i.d. for 3 days • H_1 histamine receptor blocker (start on treatment day) ○ Chlorpheniramine: 0.5mg/kg b.i.d. for 8 days OR ○ Diphenhydramine: 2mg/kg b.i.d. for 8 days • H_2 histamine receptor blocker (start on treatment day) ○ Famotidine: 0.5mg/kg b.i.d. for 8 days
b. At clinician discretion	• Pre-emptive analgesia is recommended but agent used, timing and duration is case specific and at the discretion of the clinician
2. Sedation	• May be required at discretion of attending clinician and dependent on ○ patient temperament ○ size, location, and number of lesions
3. Estimation of tumour volume	• Tumour dimensions measured with callipers and volume calculated using a modified ellipse formula where: Tumour volume =0.5×length×width×depth
4. Calculation of tigilanol tiglate dose	• Dose rate: 0.5mg tigilanol tiglate (1 mg/ mL) per cm^3 of estimated tumour volume: Dose (mL)=0.5×Tumour Volume
5. Dose limits	• Minimum dose: 0.1 mL • Maximum dose: 5 mL OR up to a dose rate of 0.25mg / kg
6. Dose administration	
	• *Tigilanol tiglate is:* ○ *injected intratumourally through a single point* ○ *delivered using a 1 or 3 mL Luer lock syringe and a 23-26G needle* ○ *administered with a fanning technique to disperse dose throughout the tumour mass*

The fine needle aspirate result was a cytologically low-grade MCT. The tigilanol tiglate dose was calculated based on tumour volume, calculated using a modified ellipsoid method, and a 50% volume per volume rate (Table 1).

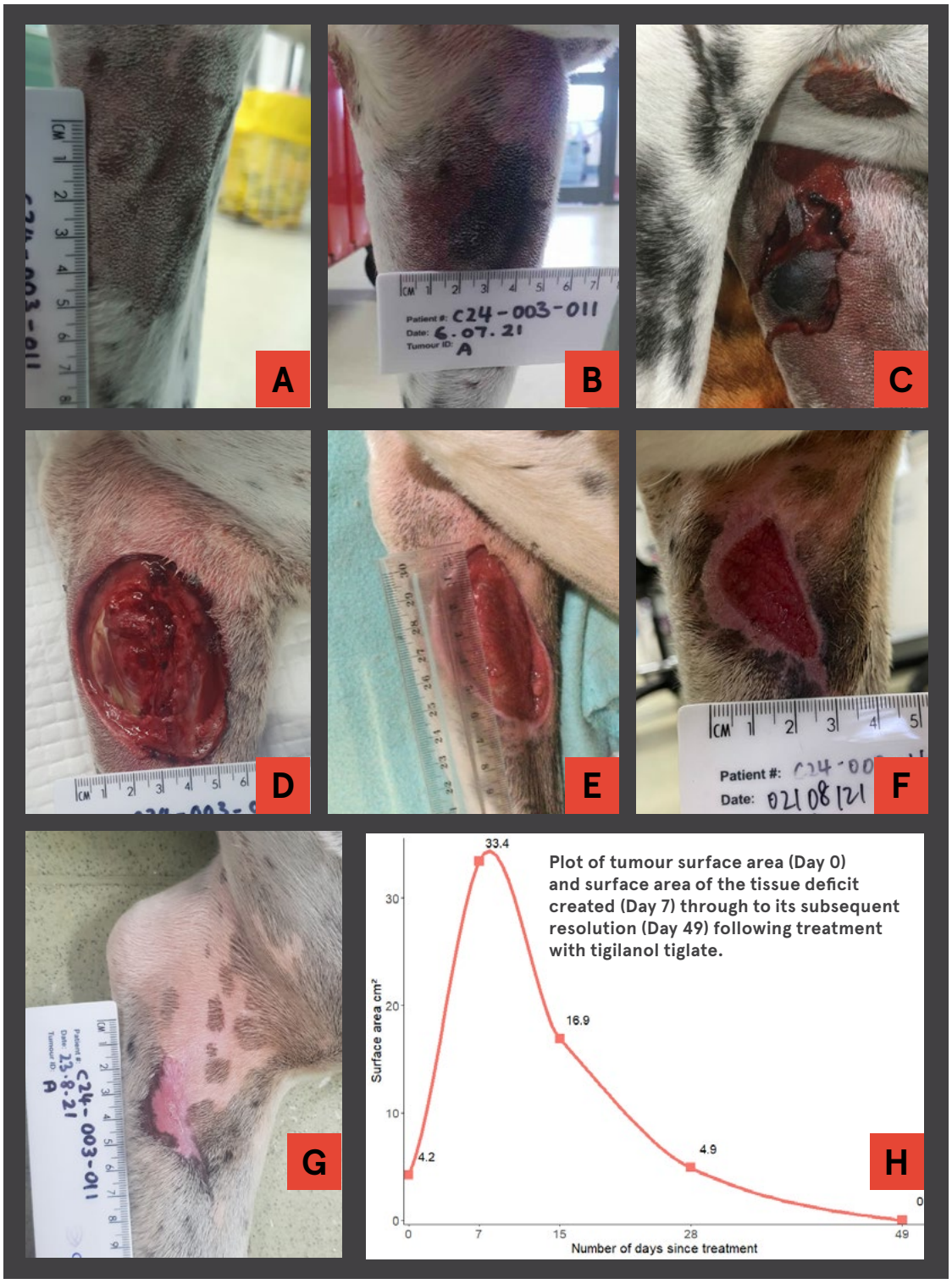


Figure 1. Photo series following the progression of a mast cell tumour response after an intratumoural tigilanol tiglate injection.

A. Treatment day (Day 0), tumour volume = 2.5 cm^3.
B. Day 1, bruising and significant discolouration of treatment site 24 hours after treatment.
C. Day 3, serosanguinous discharge emanating from treatment site from ongoing haemorrhagic necrosis and formation of an eschar.
D. Day 7, slough of the eschar.
E. Day 15, contraction of tissue deficit and infill with granulation tissue.
F. Day 28, rapid closure of tissue deficit.
G. Day 49, wound resolution, and a complete tumour response recorded using RECIST criteria.[11] There was no evidence of recurrence 12 months after treatment.
H. Plot of treatment site healing rate.

Lucy's tumour dimensions were measured with digital callipers and her tigilanol tiglate dose calculation workings are below:

- Tumour volume (modified ellipsoid method)=0.5×length(cm)×width(cm)×depth(cm)
- Lucy' s tumour volume=0.5×2.4×1.9×1.1=2.5 cm^3
- Tigilanol tiglate dose (mL)=0.5×Tumour Volume
- Lucy' s tigilanol tiglate dose=0.5×2.5 =1.3 mL
- Lucy' s tigilanol tiglate dose rate=1.3/39=0.033 mg/kg

The maximum tumour volume for a tigilanol tiglate treatment is 10 cm^3. Multiple MCTs may be treated on a single treatment day and if this is the case, the sum of tumour volumes must be ≤10 cm^3.[5] In addition, the patient dose rate must be ≤0.25 mg/kg. Lucy's tumour volume and dose rate were within these parameters. The tigilanol tiglate dose was drawn up into a Luer lock syringe with a 23G ¾" needle. The dose was administered intratumourally by dispersing the dose throughout the tumour mass using a fanning pattern (Table 1). Personal protective equipment in the form of disposable gloves and protective eyewear were worn while the Stelfonta dose was administered.[2,4,5]

The treatment response was typical of that observed in patients treated with tigilanol tiglate. Significant tumour discolouration and bruising was seen around the treatment site on examination the next day. By Day 3 a serosanguineous discharge emanated from the treatment site as the tumour necrosed and an eschar formed. By Day 7 the eschar had sloughed. From days 7 through to 49 there was rapid contraction and infill with granulation tissue until there was resolution of the tissue deficit (Figure 1). On Day 49, there was no evidence of MCT assessed using response evaluation criteria in solid tumours (RECIST).[11] During the process, the treatment site deficit was left uncovered, the patient was allowed unrestricted access to the treatment site and antibiotic therapy was not required as there were no local or systemic indicators of infection. Lucy has no evidence of recurrence at the treatment site 12 months post treatment.

Stelfonta presents veterinarians with a new option for treating canine non-metastatic MCT. Wound creation following intratumoural treatment can be confronting both to the clinician and the owner without prior understanding of tigilanol tiglate's novel mode of action and the initiated healing response without intervention. Lucy did not have any evidence of metastatic disease and her tumour volume and dose rate were within label indication.

Lucy's lesion healed well within 49 days and no additional wound management was required. Stelfonta is a new and exciting local therapeutic option for mast cell tumours with the right case selection.

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Small

NOT ALL THAT IS A NASAL MASS IS A TUMOR: NASAL CRYPTOCOCCUS IN A DOG

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C&T No. 5951

Case summary:

A 3-year-old female spayed Staffordshire Terrier *(Figure 1)* was presented to the Small Animal Specialist Hospital on the Central Coast with a 4-week history of right sided ocular serous discharge, intermittent mucopurulent to sanguineous right sided nasal discharge and progressive worsening in frequency of sneezing. The owner reported that the pet was systemically well with no reduction in appetite or energy levels. Prior treatment by the primary veterinarian consisted of a 2-week course of Amoxicillin-clavulanate, which moderately improved the clinical signs. Diagnostics at the referring veterinary hospital included nasal radiographs which revealed possible loss of turbinates in the right nasal passage. A blind nasal flush was also performed which retrieved only mucopurulent discharge at the oropharynx.

Figure 1.

Physical examination by the medicine specialist documented: Panting with no increase in respiratory effort, mild serous nasal discharge at the right naris, and reduced airflow through the right naris. There was absent nasal pain, normal facial symmetry and normal retropulsion. Oral examination revealed grade 3 periodontal disease. There was moderate submandibular lymphadenopathy with the right side more pronounced in size than the left.

The primary differentials for the nasal signs in this patient included, in order of priority:

1. Nasal tumour (although the young age for this presentation was considered 'odd')
2. Foreign body with development of a secondary granuloma
3. Mycotic rhinitis
4. Allergic rhinitis

The patient underwent screening blood work, fine needle aspiration of the submandibular lymph nodes and a general anaesthetic for nasal CT.

The CBC and biochemistry prior to anaesthesia revealed a mild inflammatory leukogram and mild hyperglobulinaemia. The lymph node aspirates were submitted to an external clinical pathology lab and subsequently returned as bilaterally reactive lymph nodes, likely secondary to periodontal disease and/or nasal disease.

After the nasal CT was performed the radiologist reported:

- Invasive osteolytic mass occupying the right nasal cavity
- The mass displaces the nasal septum to the left and is partially invading the left nasal cavity
- The lesion is predominantly soft tissue attenuating
- There is moderate contrast enhancement, which is relatively uniform
- The lesion extends caudally into the nasopharynx

The radiographic findings were supportive of nasal neoplasia e.g. adenocarcinoma.

Given the presence of a substantial nasal mass occupying the entire length of the right nasal passage, blind nasal biopsies were performed.

The retrieved right nasal biopsies were submitted for histopathology. The histopathology report described: Streams of neutrophils and vacuolated macrophages extending through proliferating fibrovascular tissue, surrounding aggregates of yeasts 15-30 µm diameter with a large clear capsule. The cytological changes were diagnostic for cryptococcus *(Figure 4)*

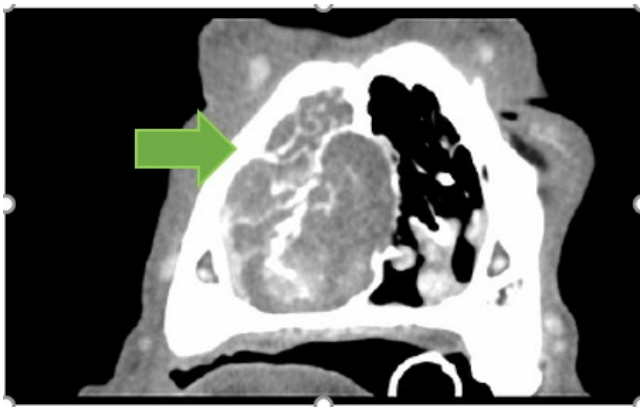


Figure 2. Transverse view of the nasal cavity showing the right nasal mass

Based on the diagnosis of nasal cryptococcosis, a LCAT was performed to confirm the diagnosis and for monitoring efficacy of subsequent treatment. The LCAT pre-treatment was significantly elevated. The patient was treated with a combination of weekly Amphotericin B subcutaneous injections for 12 weeks and oral fluconazole indefinitely. While receiving treatment with amphotericin, renal parameters were monitored due to this antifungal having the potential to be nephrotoxic.

During the course of treatment, the nasal signs resolved and the LCAT decreased but remained elevated necessitating continuation of Fluconazole after 12 weeks. Unfortunately, the patient was lost to follow-up after this period so continued serial monitoring of the LCAT was not possible.

Discussion

This case highlights the importance of performing a complete workup with cases even when imaging findings are supportive of a specific diagnosis. Imaging is a very useful diagnostic modality in our armamentarium, but it is not a panacea: It can tell us there is an aberration but not definitively what that aberration represents. Therefore, a cytological or histopathological (the latter favoured with certain diseases) should always be pursued where feasible. A comprehensive work up is especially important when the clinician is at odds with an aspect or aspects of the case. For example, in this case it was deemed peculiar (albeit not impossible) for a patient of this age to have nasal neoplasia. Based on a literature review by Mortier and Blackwood, 2020; the median age for canine neoplasia is 10 years of age.

Nasal cryptococcosis is an uncommon mycotic rhinitis in dogs, with this fungus more commonly affecting cats, and dogs being more commonly affected by nasal aspergillosis. There are 2 species of cryptococcus that affects animals: *Cryptococcus gattii* and *Cryptococcus neoformans*. In this patient, culture and or molecular diagnostics were not performed to determine the biotype of the species.

Treatment for nasal cryptococcus can be prolonged and require significant financial and time commitment by the owner. Monitoring of the LCAT is recommended to guide length of treatment; however, some animals require several months of treatment and others may indefinitely require anti-fungal medication.

Some clinicians recommend surgical debulking of the fungal granuloma prior to initiating systemic anti-fungal therapy. However, this is a relatively invasive procedure and as far as the author of this article is aware, the addition of surgical debulking to the therapeutic plan Vs systemic therapy alone, has not been evaluated to determine if treatment success rate is increased with surgery.

In conclusion, canine nasal cryptococcus should be on a clinician's radar in dogs presenting with nasal signs, especially when a nasal mass is documented.

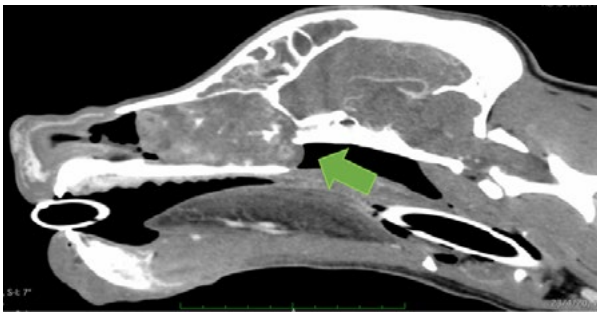


Figure 3. Sagittal view of the nasal cavity showing the nasal mass extending into the nasopharynx and occluding the meatus

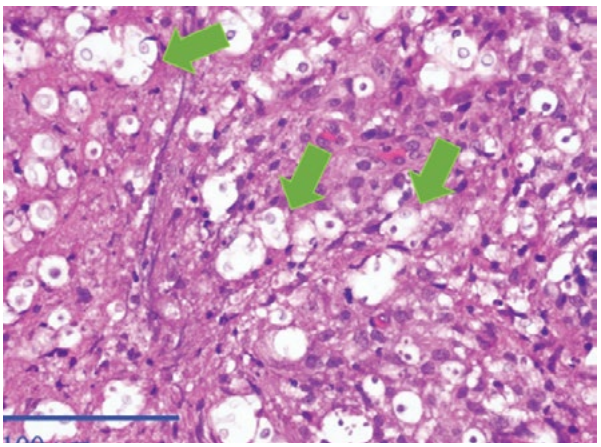


Figure 4. Nasal histopathology slide from another dog diagnosed with nasal cryptococcus.

Image extracted from: Gan, Z, *Israel Journal of Veterinary Medicine*. 2012, Vol 67. (4)

Editor's Comment

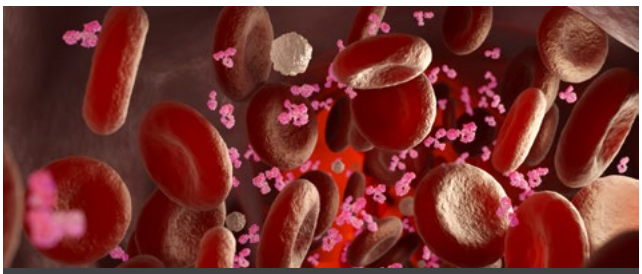
What an interesting case! Normally cryptococcosis in dogs is not well constrained, and rapidly spreads from the nasal cavity to the CNS, eyes and regional lymph nodes and nearby tissues. Almost all cases of cryptococcosis in dogs with nasal involvement can be diagnosed with a deep nasal swab, as the organism multiplies by budding, and the sp *blastoconidia* exfoliate readily into nasal exudate. Stated another way—it's much easier to diagnose than aspergillosis.

In a case like this where the CT shows very solid focal disease—a so called cryptococcoma (tumour-like granulomatous mass lesion), it opens the door to another avenue for treatment. INTRALESIONAL amphotericin B is highly effective in a situation like this. It's just a matter of using the CT to define the size of the mass and instil 0.5 mg/kg of stock amphotericin B (10 mg/mL) into the centre of the lesion. For a Staffordshire bull terrier of 20 kg—the dose would be about 10mg or 2mL.

Using a 3mL syringe luerlocked to a 23- or 25-gauge needle—insert the needle into the middle of the mass and slowly inject the 2mL into the centre of the mass. This could be done at the end of the CT study, combined with 500 mLs of Hartmann's or Plasma-Lyte subcutaneously to make sure the dog remains well hydrated.

You get an intense reaction with quite an immediate reduction in the size of the lesion. You then move onto biweekly subcutaneous infusions, as per the article ◆

eBook download
C&T No. 5698 Treatment nasal cryptococcus in a dog



Call for Cases

ACUTE KIDNEY INJURY AFTER NSAIDS

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It is well known there is a potential relationship between acute kidney injury (AKI) and the administration of NSAIDs such as meloxicam, carprofen and robenacoxib. Indeed, I have been told that that up to 20-25% of current complaints to the NSW Board of Veterinary Practitioners involve NSAIDs.

I am part of a group of clinicians interested in obtaining data on dogs and cats that have developed Acute Kidney Injury (AKI) after prudent use of NSAIDs.

We would like to better understand how and why this can occur despite following current best practice and the manufacturer's recommendations.

It appears to be that despite the safety data available from studies of normal cats and dogs, when these drugs are used in the field, problems can occur that cannot be predicted from what we know about the pharmacology of these useful and effective agents.

My colleagues and I wish to gain a better understanding of this via post marketing surveillance (pharmacovigilance).

We need your help, please!

If you or your colleagues have seen cases like this, could you please write us a short e-mail summarising the case, or provide us with the medical records for affected animals so we can analyse them further?

Thank you.

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Small

MELIOIDOSIS IN A PERSIAN CAT

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C&T No. 5952

Ollie, a 4-year-old Persian Cross cat, first presented for 'sore eyes' in late August of 2020. Ollie had been owned by the previous tenant of an apartment block and the new owner of the apartment took over ownership of Ollie when he moved in. Ollie spent time inside and outside the apartment, located in Cairns.

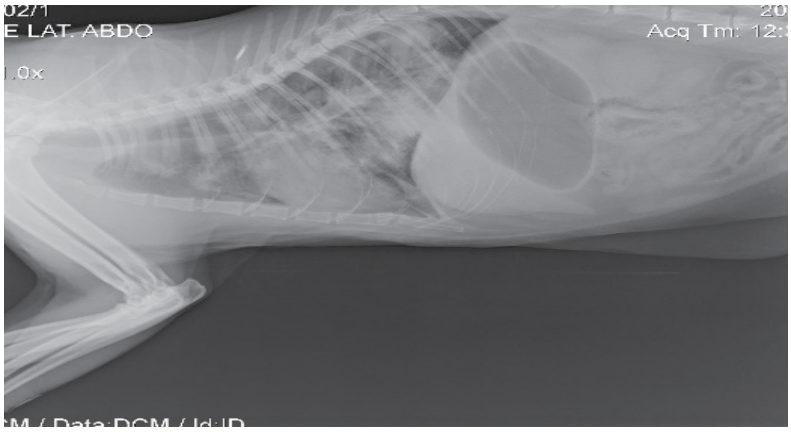
Vaccination and parasite control were unknown. He had been otherwise well. Ocular examination revealed clear corneas, no discharge, bilateral 3rd eye lid elevation covering 1/3rd of the eye, both eyes being held open comfortably, no blepharospasm or irritation, normal pressures in retrobulbar spaces when applying pressure to eyes, pupils of appropriate size and symmetry, normal pupillary reflexes. Fluorescein stain was negative and no foreign material was noted behind the third eyelids after application of local anaesthetic. The rest of his physical exam was unremarkable.

The owner declined further work-up at this stage as Ollie was otherwise well and a presumptive diagnosis of 'Haws-up' syndrome was suspected. Ollie was given a full intestinal wormer in case of any parasite burden that could be causing the symptoms.

Ollie then presented in early January for broken nails. He had gone missing for a period of time and only just returned to the owner. On physical examination Ollie had lost approximately 500g, his mandibular lymph nodes were moderately enlarged, he was pyretic (40.7°C), and his eyes appeared normal. Almost all of his nails were broken off down to the quick. An FIV/FeLV test was performed and was negative, he was wormed with an intestinal wormer and given Convenia SC as well as meloxicam orally. His owner declined full bloods at this stage and was happy to check for his response to treatment.

Four days later Ollie presented with severe dyspnoea. Over the preceding four days he had deteriorated at home and was now off his food and water. His presenting temperature was 39°C. The owner agreed to bloods, chest radiographs and stabilization.

Figure 1 A & B. Ollie on initial presentation.



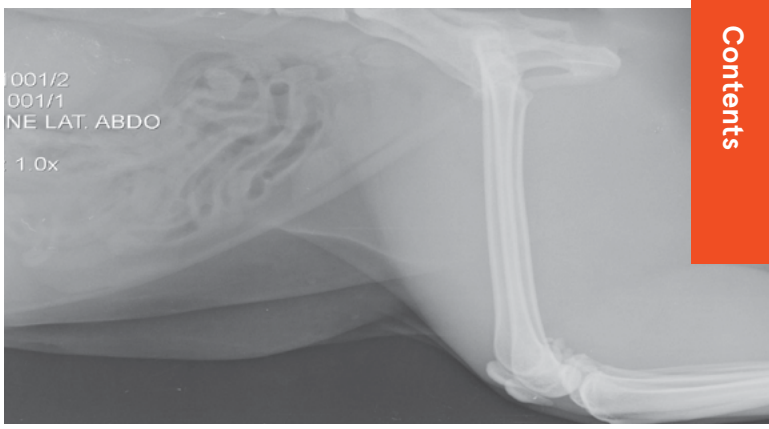


Figure 2. Chest radiograph above showing severe mixed alveolar pattern. Lateral abdominal radiograph on the right.

Bloods showed:

- hypoglycaemia (1.65 (4.11-8.84))
- hypocalcaemia (0.64 (1.95-2.83))
- hypalbuminaemia (19 (22-40)),
- increased total bilirubin (34 (0-15)), hyperphosphataemia (2.65 (1-2.42))
- non-regenerative anaemia.

All white cell lines were decreased. A lateral chest x-ray was taken and showed severe mixed alveolar and bronchial pattern with some consolidation.

The owner elected humane euthanasia but agreed to a post-mortem for a definitive diagnosis. Necropsy examination revealed multiple non-pigmented smooth nodules, ranging in size from 4 mm in diameter to 35 mm in diameter in the lung tissue. No foreign bodies were found. There was serosanguinous fluid within the chest cavity. Samples were sent for both histopathology and culture.

Histopathology revealed severe acute to subacute fibrinosuppurative bronchopneumonia, with intralesional bacilli and the culture revealed *Burkholderia pseudomallei*.

Burkholderia pseudomallei is a soil-dwelling bacterium endemic in tropical and subtropical regions worldwide, particularly in Thailand and northern Australia. It infects humans and other animals and causes the disease melioidosis. It is also capable of infecting plants.

It is unknown whether Ollie contracted the disease when his nails were damaged or earlier than this, although the first presentation with his eyelids could have been very early in the disease. We are very thankful to the owner for allowing us to do further testing on Ollie. Although we often see melioidosis present in non-healing skin wounds of dogs, this is the first time we have diagnosed it in a cat.

Editor's comment: Practitioners in the north of Australia do see this disease, and it is seen throughout SE Asia, and I understand there was an outbreak in Cairns last year. Carolyn O'Brien wrote up a cat imported from Southeast Asia that developed the disease at the end of quarantine, while Helen Parks wrote up a few cases seen in the far north of Australia. The disease can be successfully treated. Generally, *B pseudomallei* is susceptible in vitro to some third generation cephalosporins, carbapenems, chloramphenicol, tetracyclines, trimethoprim/suphonamide (TMP-SMX), amoxicillin clavulanate and ticarcillin clavulanate. Based on recent trials, the current recommendation for initial therapy of acute melioidosis in humans is an intensive therapy phase with intravenous ceftazidime or a carbapenem (imipenem or meropenem), with or without TMP-SMX, given for at least 10days or until definite signs of a response. Bacterio-static antibiotics, including tetracyclines and chloramphenicol are inferior in acute cases. Intravenous therapy is followed by an eradication regimen of orally administered TMP-SXT with or without doxycycline, given usually for 8 to 12 weeks to prevent relapse. Finally, granulocyte colony stimulating factor therapy may be helpful in the setting of melioidosis septic shock with combination antimicrobial therapy. ◆

Go to eBook to download.
Further reading:
Parkes HM, Shilton CM, Jerrett IV, *et al*. Primary ocular melioidosis due to a single genotype of *Burkholderia pseudomallei* in two cats from Arnhem Land in the Northern Territory of Australia. *Journal of Feline Medicine and Surgery*. 2009;11(10):856-863. oi:10.1016/j.jfms.2009.02.009

General

CAN GUINEA FOWL PROTECT PETS FROM SNAKES?

Veterinary Editor

C&T No. 5953

I have always wondered what the best way is to prevent dogs and cats being bitten by snakes. Ultrasound emitting devices you hammer into the ground are available, but I am never sure they actually work. In my hands—they do not. So, what can we do, apart from keep dogs and cats exclusively in doors?

A friend—Frank Santori—provided a possible answer. Guinea fowls!!

In addition to providing excellent pest control in the garden, Guinea fowl make magnificent snake guards.

Their preferred strategy is to make a large amount of noise to deter snakes; if this does not work, they work as a team to gently persuade the snake off the premises (*See YouTube video below*). If teamwork and intimidation does not achieve the desired result, they have anecdotally been known to kill snakes.

One guineafowl breeder she said she would see up to 10 snakes a season in her house yard and two of her dogs had been bitten and saved at the vets. Since getting the guineafowl a few years back, she barely sees a snake a year—this year there have been none, and the dogs have not been bitten since. She has 70 guinea.

The guinea get about 95% of their food from free ranging. She feeds them a treat late in the afternoon, they all run back into their enclosure, and she closes it for the night. French White Millet is the favoured treat and they will come from anywhere for it. It's to be used as a treat only, just enough for them all to have a mouthful. She uses scratch mix, mash and sometimes adds in meat bird grower as a protein treat.

Guineas may not be for everyone though; they can be extremely loud and of course are no good if your dog eats them. They are also highly sociable and you should have a minimum of 4-6 in the flock. ◆

Figure 1. Guinea fowl grazing happily
Watch: https://www.youtube.com/watch?v=1dr2piBoQJU

Figure 2. Guinea fowl escorting snake off the premises
Watch: https://www.facebook.com/watch/?v=563004664497312

Small

CATS & UPPER RESPIRATORY TRACT ISSUES IN TICK PARALYSIS

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C&T No. 5954

Compared to the dog, cats have a relatively smaller glottis and are, therefore, mathematically more susceptible to Upper Respiratory Tract (URT) blockage issues. They are more susceptible to stress and anxiety 'attacks' in general, e.g. 'flea fits', and are very sensitive to laryngeal stimulation of many sources, (especially associated with intubation and anaesthesia)—as is documented with people; mucus, fumes, dust, etc.[1] often being associated with resultant laryngeal 'spasm.' (Spasm is apparently what you feel when 'something goes down the wrong way'; you lose your normal voice, feel a choking-like sensation and your eyes often water, along with feelings of discomfort and susceptibility, etc.)

With Tick Paralysis (TP), cats change their phonation (vocal folds, etc.), lose their capacity to purr (larynx) and their ability to swallow (vagus nerve, etc., complexity). Saliva also becomes more sticky as it perhaps dehydrates.[2] Early TP changes are also associated with wheezes (on auscultation), producing similar sounds to feline obstructive airway disease. Such wheezes are also seen in early dog TP cases (data from n=506 TP cases), suggesting lower airway dysfunction is present as well.

In summary, cats have a relatively smaller glottis (compared to dogs), a super-sensitive larynx, are prone to anxiety-associated URT issues, probable airway changes and have lost, to some extent, laryngeal adductor and abductor muscle function (with TP). (It is not known if all laryngeal muscles are equally affected in TP, both during the initial clinical onset period and/or with increasing clinical severity).

However, the severity of these URT TP issues seem to wane with time. Perhaps this is some form of acceptance (especially if left alone and not constantly disturbed), the progression of TP or the effects of any ongoing sedation. Historically, cats were given 'sedation, TAS & left alone.' Do they all need a fluid line? Do they need close-in, regular physical examination when more distant 'head/neck/chest/breathing observations' would detect deterioration versus more close-in regular interference, especially if known to be 'vet-clinic-anxious' cats.

So, if there is clinically apparent loss of URT muscle function (voice, etc.), why do cats develop such URT-associated 'fits of anxiety'—they often appear to be attempting to clear their URT with head elevation, rapid tongue and jaw movements, as well as scratching at their neck. Is it simply the 'fear of choking'[3] with sticky saliva, unclearable debris, etc., with a 'known to-be sensitive' larynx? Alternatively, do they trigger adrenergic or non-adrenergic/non-cholinergic (NANC) pathways to over-ride TP-induced, post-synaptic, muscle flaccidity? While there are four (two mainly) adductor muscles and one abductor muscle—again, are they all equally affected or is there a functional bias to either adduction or abduction? If the abductor is more affected, that would comparatively induce severe glottic obstruction (air flow being roughly inversely proportional to radius to 4th power), whereas weakened adductor muscles may cause relatively less glottic airflow reduction. However, is the cat, with its very efficient O_2/Hb dissociation, more able to cope with URT spasm and obstructed airflows?

The basic question is then—if TP has induced laryngeal paresis/paralysis, especially of the abductor muscle, how can the cat then produce a

1. Human Laryngeal Spasm – Various websites

2. R. Atwell (2021) – opinion piece – C & T, University of Sydney

3. Rabies patients & water testing – people fear the perceived potential for choking (DPI Rabies Video; pers com Zimbabwe Rabies Facility)

profound, clinically-suggestive 'spasm' (confirmed by direct laryngeal visualisation and the physical difficulty with subsequent intubation, oxygenation, etc.)?

It must be caused by non-TP-affected nerves (see above), associated with intact NMJ function and/or other forms of chemical intervention, e.g. there are at least 20 neurotransmitter/enabler chemicals present in the mucosae of the mammalian larynx (a major study area re basic URT physiology).[4]

So, is the cat TP-URT anxiety presentation due to loss of basic protective abduction (with loss of voice, purring, swallowing, etc.), along with the presence of 'hard to move,' sticky, dried-out laryngeal debris, inducing a choking sensation?; or is it these factors, plus an anxiety-induced spasm of adductor muscles, via different neural pathway(s) (e.g. NANC,) stimulating effectively-normal, post-junctional muscle tissue (which is not directly affected by any TP issue re pre-junctional, Ach-blocked, functional NMJ-opathy)? So, the TP presynaptic paresis is overridden by laryngeal-sensitized, choke-induced 'spasm,' employing different neurotransmitters (not effected by TP toxins) inducing effectively normal muscle function; i.e. muscle is normal and TP-reduced function (by Ach blockade) is now not applicable as the tissues are now stimulated by other (non-TP affected), transmitter-receptor, nerve-muscle pathways. Does this now explain the clinical paradox of progressive TP paresis and profound 'tightly-closed-glottic spasm'?

If all things are equal, then the glottis is well equipped to be closed, with associated adductor/abductor muscle activation. Considering the feline way of killing and the more dagger-type teeth, basic protection of the glottis would be essential if any arterial penetration in the prey's neck occurred, with open-mouth-grasping of the prey.[5] Without such efficient and immediate glottic protection, pressurized arterial (prey) blood could enter the glottis. (Dogs in a similar position tend to crush the carotid area leaving S/C bleeding points in the neck, along the route of the carotid artery). In support of this protective glottic status, does the cat's lower haemoglobin oxygen affinity (and higher tissue dissociation) enable more efficient oxygen usage and so, longer functional glottic closure may be possible?

So, in TP, the feline larynx may be very much sealed and perhaps any anxiety/stress occurrence encourages such closure (as does local debris) as confirmed by concurrent or subsequent URT observations in association with generalised paresis/paralysis.

References

1. Human Laryngeal Spasm – Various websites
2. R. Atwell (2021) – opinion piece – C & T, University of Sydney
3. Rabies patients & water testing – people fear the perceived potential for choking (DPI Rabies Video; pers com Zimbabwe Rabies Facility)
4. Web research – laryngeal (mammalian) mediators
5. Atwell clinical note AVP 2011

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4. Web research – laryngeal (mammalian) mediators

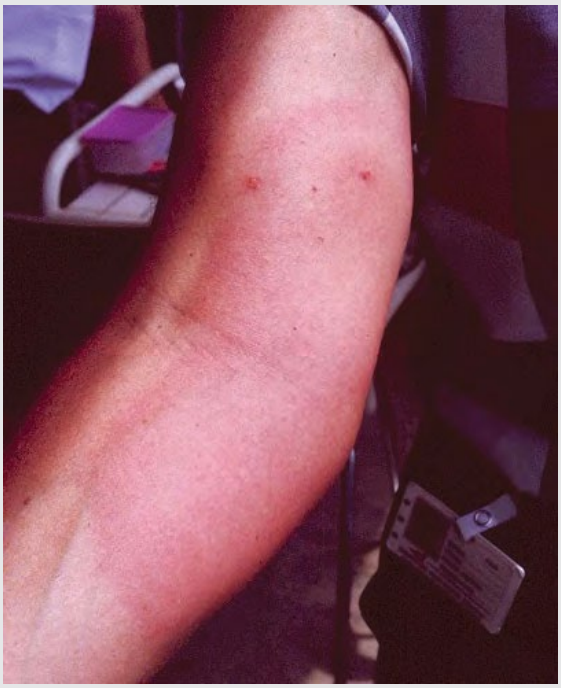
5. Atwell clinical note AVP 2011

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

FOR EXAMPLE, HAVE YOU HEARD OF RED MEAT ALLERGY?

Prof Sheryl van Nunen (Royal North Shore Hospital) and Emeritus Prof Paul Canfield (University of Sydney) contributed this fascinating article in September 2014, C&T NO. 5416.

C&T NO. 5416 is available in the complementary eBook version; the url is emailed to CVE Members.



Winner

Entitled to a CVE$100 voucher

Small

FLEA-INDUCED ACUTE RESPIRATORY DISTRESS

Gabby Lawson
The Cat Clinic Hobart
150 New Town Road
New Town TAS 7008
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C&T No. 5955

Gavin is a 4-year-old male neutered Domestic Shorthaired cat that previously lived an indoor-outdoor lifestyle on a property in southern Tasmania. Last spring, Gavin's owner contacted the clinic as she was concerned about his sudden strange behaviour and breathing which was described as open mouth panting. An emergency appointment was scheduled, and she was advised to bring him straight to the clinic.

On arrival to the clinic, I opened Gavin's carrier and he came out immediately and was friendly and interactive. Fortunately, he was not in respiratory distress, and was actively exploring the consultation room and even jumping in the sink!

His owner reported that he was completely normal first thing that morning and had eaten his breakfast as usual. He had been inside all night and went outside mid-morning, returning 15 minutes later, and then accompanied his owner into the home office. He initially sat on the office chair, and then jumped to the top of a cabinet and looked like he had a spider web on his face. He then started panting, flicking his ears, and seemed very itchy and agitated. He was described as licking his paws and rubbing his ears, and had his eyes closed.

Video 1: Gavin showing signs of acute respiratory distress at home

In consult, Gavin was calm, friendly and happy to be handled. On physical examination he was found to be in perfect body condition, weighing 5.2kg with a body condition score of 4/9, his mucous membranes were pink and moist, no abnormalities were found on oral examination, HR 180BPM regular and no murmur detected, and he had normal chest sounds and respiratory effort. His temperature was not taken as he was much too busy and active. The only abnormality detected was that he was flicking his ears and intermittently licking his back. During the examination, I found a dead flea on his pinnae, and then more on his head and neck area. I then noticed that his blanket was covered in dead and dying fleas.

Video 2: Dying fleas in consult

Gavin had an indoor-outdoor lifestyle and lived on a small property on the outskirts of Hobart in an area with many lifestyle blocks. Gavin's owner had applied Bravecto® Plus topical spot on (for cats >2.8-6.25kg) treatment approximately 10 days ago. Given the large number of fleas detected, the clinical presentation, and Gavin's lifestyle and outdoor access, I suspected that he may have stuck his head down a rabbit burrow that morning, and the clinical signs he was experiencing were due to the excitement phase of dying fleas as the Bravecto®Plus took effect. Gavin was treated with a subcutaneous injection of 0.6mg Dexamethasone, and his owner advised to keep him indoors for close monitoring, but I suspected there would be no further issues. Ongoing strict external parasite control was also recommended.

Bravecto® Plus spot-on contains the active ingredients moxidectin and fluralaner. Moxidectin is a member of the milbemycin group of macrocyclic lactones with the mode of action based on the binding of ligand-gated chloride channels. This leads to an increased membrane permeability of nematode and arthropod nerve and/or muscle cells for chloride ions and results in hyperpolarization, paralysis, and death of the parasites. Fluralaner is a member of the antiparasitic class of isoxazoline-substituted

benzamide derivatives and acts antagonistically on arthropods ligand-gated chloride channels (GABA-receptor and glutamate-receptor). Fleas do need to bite and feed to ingest fluralaner and can feed within 5 minutes of infestation. Fleas are usually killed within 12-24 hours during the 3-month treatment period.

Figure 1: Gorgeous Gavin in consult

Having previously used Capstar® (nitenpyram) for those cases with high flea burdens and witnessed cats becoming very irritated 20-30 minutes after dosing as the fleas go through an excitement phase as they die, I assumed that this was the case with the Bravecto® Plus and the high flea burden that Gavin was acutely infested with. After speaking to the technical vet at MSD Animal Health there haven't been any other reports of such a reaction. Adverse reactions to this family of chemicals are rarely observed and are typically seen in the period post administration of the product such as vomiting, diarrhoea, lethargy, inappetence, and neurological signs.

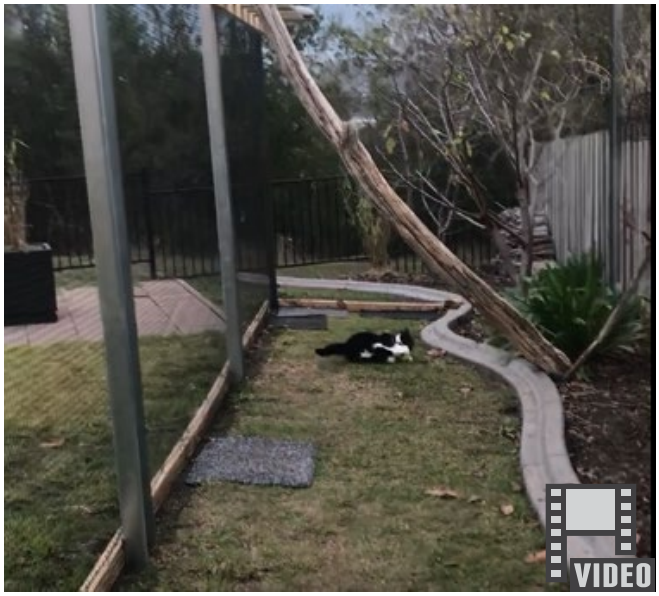
I recently saw Gavin for his annual vaccination and health check. His owner reported that he recovered very well from the flea incident and had no further issues. Since the last visit, a large cat enclosure has been built for Gavin, and he now lives a contained lifestyle, enjoying his walks on a harness and games in his enclosure. Gavin was clinically well other than being in a slightly less perfect body condition now that he wasn't so busy spending his days looking for bunnies!

This was an unusual presentation of a cat in acute respiratory distress and a much happier consult than the other usual outcomes of cats in respiratory distress!

Thanks to Gavin's owner for providing images & videos and Dr Jess Parry from MSD Animal Health.

Figure 2. Bravecto® Plus topical spot on for cats

Figure 3. Gavin in the 'front room' of his cat enclosure. (Image supplied by owner)



Video 3. Gavin enjoying games in his cat enclosure (Video supplied by owner)

Resources

Bravecto® Plus for cats

339698_R2.pdf (bravecto.com.au)

Capstar® (nitenpyram)

H???? P056485.indd (capstarpet.com)

Dryden *et al.* In-home assessment of either topical fluralaner or topical selamectin for flea control in naturally infested cats in West Central Florida, USA *Parasites & Vectors* (2018) 11:422

https://doi.org/10.1186/s13071-018-2995-1

Ranjan *et al.* A single topical fluralaner application to cats and to dogs controls fleas for 12 weeks in a simulated home environment, *Parasites & Vectors* (2018) 11:385
https://parasitesandvectors.biomedcentral.com/articles/10.1186/s13071-018-2927-0

Rohdich *et al.* Field effectiveness and safety of fluralaner plus moxidectin (Bravecto® Plus) against ticks and fleas: a European randomized, blinded, multicenter field study in naturally-infested client-owned cats, *Parasites & Vectors* (2018) 11:598
https://parasitesandvectors.biomedcentral.com/articles/10.1186/s13071-018-3175-z◆

Small

SKINSUITS FOR THE RASHY DEVON REX & SPHYNX

Sue England

t. 0400 110 040 (sms only)

e. devilrex@optusnet.com.au

C&T No. 5956

The 'skin-suit' was created while caring for my own Devon Rex cats who suffered with skin disease and skin cancer.

I spent much time patterning a skinsuit that would help stop them being able to lick and scratch their skin, breaking open new spots and causing further damage.

The bonus is that the skinsuit allowed me to apply topical ointment under the skinsuit without the cat being able to lick it off.

The skinsuits I make have a T-shirt-like fabric that has a 4-way stretch. It's thin but supportive with a calming effect. It fits firmly to the skin much like leggings. It is sewn with a reverse hem to avoid stitching touching the skin and causing further irritation.

As they are custom-made, they can be made to cover front and or back legs with a variety of leg lengths.

It a little tricky for me to get a photo of the pattern I use as I adjust it with each cat so the pattern warps in measurements, but I have included a photo that is freely available online that is very similar to mine, it works a treat!

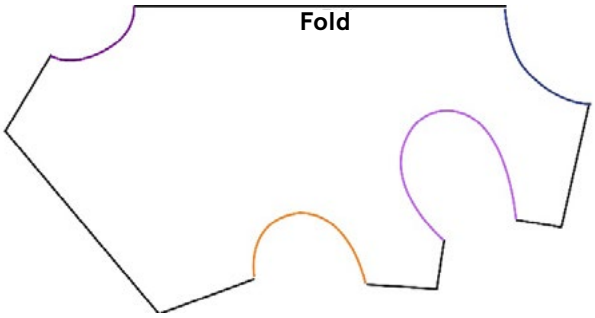
If someone cannot access a skinsuit they can easily buy a baby onesie size 0000 or a little bigger for the more generously proportioned—surprisingly they fit cats rather well.

The cat can wear them for an hour or two while absorbing a topical cream or they can be worn all day provided owners supervise.

Most importantly, the skinsuit helps stop that barbed tongue getting to the skin and wreaking havoc!

If you need my help to prepare a skinsuit, please feel free to contact me.

I ask a small fee to help cover materials used.

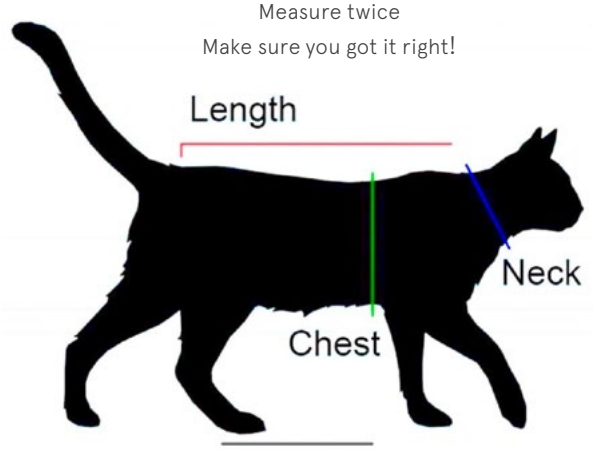


Provide measurements in centimetres

Measure once

Measure twice

Make sure you got it right!



COMMENT COURTESY OF RACHEL KORMAN

Co-Tutor for Feline Medicine Distance Education course

Cats being a wise species, they are less tolerant of body dressings as a general rule and there is never a 'one size fits all' for cats. For cats with skin fragility syndromes (e.g. hyperadrenocorticism) we have used suits from https://www.bromellidogs.com.au/. These suits are made to measure so clients have to carefully measure their cats to make sure they fit well. For other conditions, we tend to use soft Elizabethan collars or newborn baby onesies!

Minimising harm and maximising benefit associated with Elizabethan collars
Dr Anne Fawcett & Yustina Shenoda
C&T No. 5847

COMMENT COURTESY OF ANNE QUAIN

Lecturer Professional Practice

Skin suits and Elizabethan collars are non-pharmacological methods of preventing feline self-trauma.

We surveyed owners of cats and dogs worldwide who had worn an Elizabethan collar during the previous 12 months (n=434) (Shenoda *et al*. 2020). Of these, 77% reported that their companion animal had a worse quality of life when wearing the collar. They reported that collars interfered with activities including drinking, playing, toileting and grooming. Changes in feline behaviour reported by owners included agitation, 'freezing', altered posture or gait, inter-cat conflict, and difficulty getting in or out of the cat door.

Skin suits may prevent self-trauma while avoiding some of these problems (e.g. cat door issues). For the right cats (i.e. those who can be coaxed into appropriately fitted clothing without injury to themselves or others, and whose behaviour is not altered negatively by wearing a suit), these skin suits can be a better alternative to Elizabethan collars.

I have recommended them for cats whose wound healing would be complicated or delayed by self-trauma. In my experience, cats adapt to them well.

The design features of the skin suits that I think are most helpful are:

- Customization to ensure fit (reduced likelihood of 'wardrobe malfunction' e.g. cats getting a limb out of a sleeve, or into the wrong sleeve/neck)
- Thin, breathable fabric
- The reverse hem to avoid abrasion of the skin

I agree with the author that cats wearing clothing should be supervised to ensure prompt intervention in case of misadventure, though I suggest the same with Elizabethan collars. Clothing should be cleaned regularly.

It is critical that the underlying cause of self-trauma is addressed, as affected cats will continue to suffer from pruritis or pain, and self-trauma may recur when the suit is removed.

Reference

Shenoda, Yustina, *et al*. (2020), '"The Cone of Shame": Welfare Implications of Elizabethan Collar Use on Dogs and Cats as Reported by their Owners', *Animals*, 10 (2). ◆

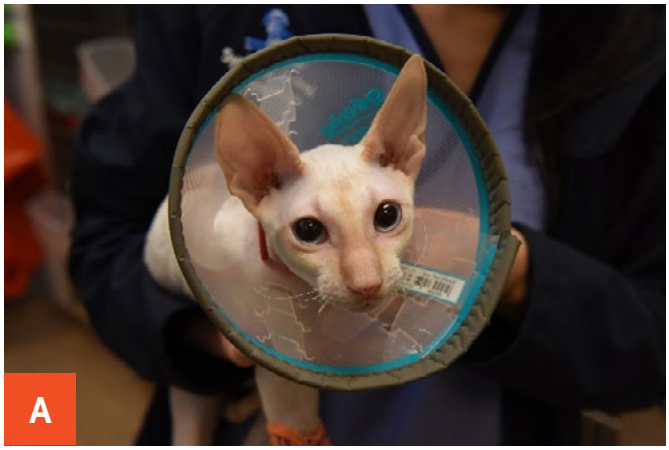


Photo A, B and C: Kittens wearing Elizabethan collars
Photo courtesy of Anne Quain

Elizabethan collar in the bin
Photo courtesy of Anne Quain

Winner - Best Visuals

Entitled to a digital video or DVD from the CVeSHOP

Small

OCULAR MISCONCEPTIONS

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C&T No. 5957

1. Breed is not important

WRONG!
Breed predisposition is very important in veterinary ophthalmology.
Most of the eye problems—apart from trauma are breed-related.

Always check the breed predisposition lists in all purebred dogs and their primary crosses.

For the Animal Eye Care breed predisposition list go to cve.edu.au/Common/Uploaded files/CT/Breed-Predisposition-to-Eye-Disease.pdf.

Veterinary Editors' comments: Almost all diseases in dogs are breed related, including many immune-mediated diseases and cancers; this is the problem with our owned dog population—it is essentially a population of pedigree dog hybrids, compared to cats where 80% of individuals are genetically diverse crossbreeds.

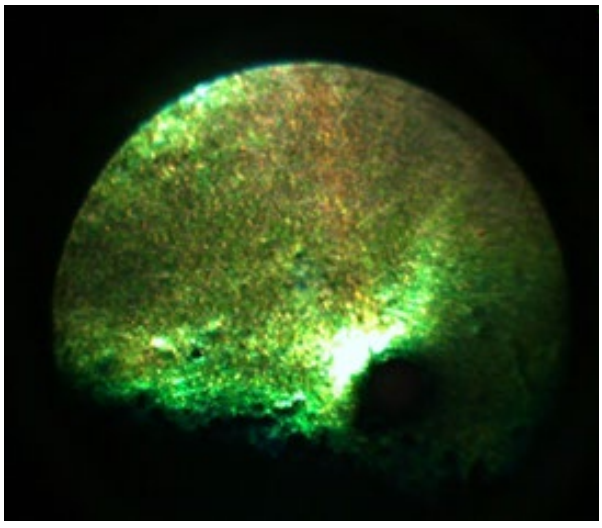


Figure 1. Fundoscopic appearance of advanced PRA. Note absence of any retinal vessels.

Some Eye Diseases that are breed related

- Progressive retinal atrophy (PRA)—often patients present with poor night vision (Labradors, Australian Cattle Dogs, Poodles, Cocker Spaniels, Labradoodles).
- Lens luxation—always think of lens luxation in any terrier with a sore eye.
- Glaucoma—increased intraocular pressure (IOP)—Bassets, Cockers, Poodles, Maltese, Golden Retrievers

Beware of a one-eyed Pure-Bred dog! One eye may have been removed because of glaucoma pain—the other eye will be predisposed to glaucoma!

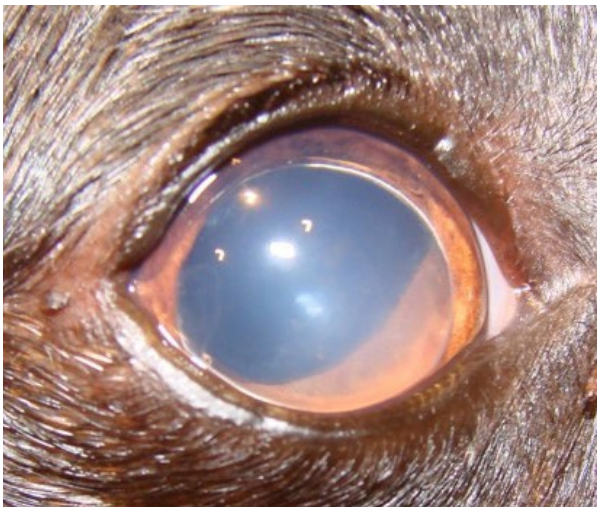


Figure 2. Lens luxation—Always think of lens luxation in any terrier with a sore eye. Beware of a one-eyed Terrier! One eye may have been removed because of pain—the other eye may be developing lens luxation!

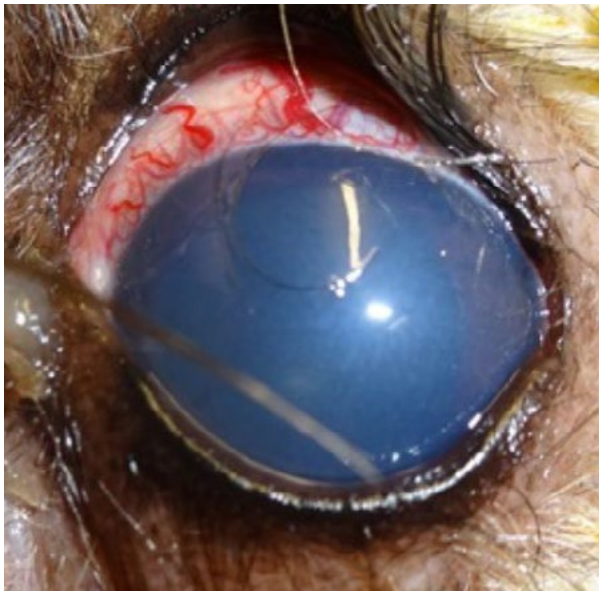


Figure 3. Glaucoma

2. Schirmer Tear Tests are not needed as dry eye is pretty obvious

WRONG!

- One of our golden rules at Animal Eye Care is to always do a Schirmer Tear Test (STT) in all cases of corneal disease, conjunctival disease, and ocular disease.
- Dry eye can easily be missed, it does not always present with the classic clinical signs of a thick mucoid discharge that dries and crusts on the eyelids.
- Remember in dogs to always relate the clinical signs to the STT. If the eye looks dry and the STT is not too bad, consider treating the eye anyway. Some eyes are dry because the tear quality is low. Cyclosporin and tacrolimus will help these cases by increasing the tear quantity and also improving tear quality.
- Remember in cats they usually do not get the classic mucoid discharge that dogs do. Consider using Hylo-Forte® eye drops for about 6 weeks in all cats that have had viral keratitis and/or conjunctivitis.

STT values

- Normal STT for dogs is > 15 mm wetting/minute
- Normal STT for cats > 9mm wetting/minute

Figure 4. KCS-classical features of dry eye syndrome

3. Oral NSAIDs are good enough

WRONG!

- Ocular trauma is common, and in many cases, oral cortisone is a better choice than oral NSAIDs! In some cases, this can cause vision loss, and other uveitis induced sequelae.
- Many cases of ocular trauma are treated with oral NSAIDs, but in severe cases, the oral NSAIDs are simply not enough to treat the uveitis and minimize potential damage to the eye.
- At Animal Eye Care, we prefer to use oral prednisolone for most cases of ocular trauma. In very severe cases we will also use epibulbar subconjunctival cortisone injections. We lose most eyes to uveitis rather than infection and or slow wound healing.

When not to use oral prednisolone:

i. Infections in the cornea—keratomalacia, corneal and or scleral lacerations that have not been sutured,

ii. The animal is otherwise unwell or diabetic.

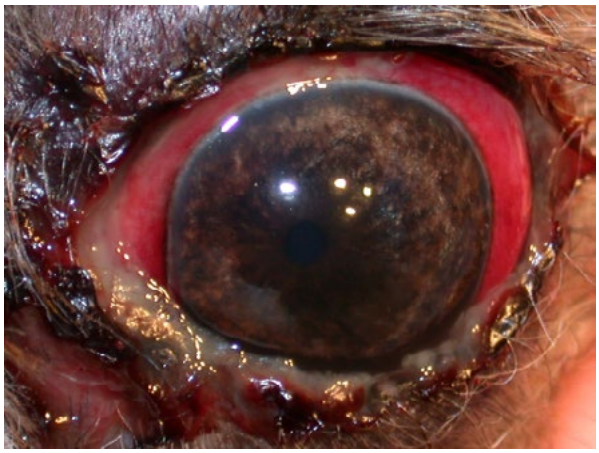


Figure 5. Ocular trauma: note the scleral haemorrhage and the small pupil

4. Using ointment to treat ocular trauma

As previously discussed, uveitis and ocular trauma cases need to be treated with anti-inflammatories. In most cases we should use oral anti-inflammatories—usually oral prednisolone rather than oral NSAIDs for severe cases of uveitis and ocular trauma.

When you see a case of uveitis or ocular trauma consider using a potent topical anti-inflammatory -e.g., Maxidex® or Prednefrin® Forte eye drops rather than an ointment that contains an antibiotic and hydrocortisone. These ointments simply are not strong enough.

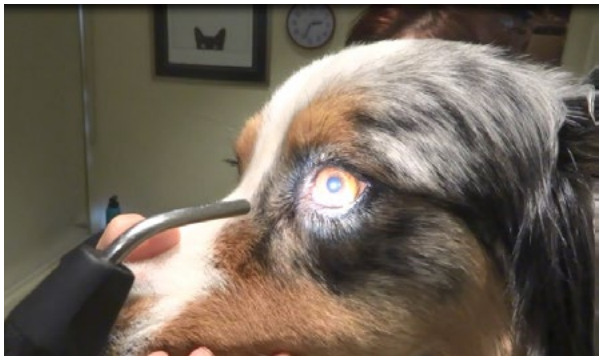


Figure 6. PLR testing—Always use a bright focal light sourcePLRs are way too basic to be useful for me

Topical anti-inflammatories obviously must not be used when the cornea is ulcerated. Use caution if the cornea is inflamed, or if the blink response is reduced after ocular trauma, or if the STT is less than 15mm wetting/minute. These cases are more likely to ulcerate.

5. PLRs are way too basic to be useful for me

WRONG!

We find pupillary light reflexes (PLRs) very, very useful in our practice.

The PLR can be affected by a number of factors.
Slow and incomplete PLR can be caused by:

i. Weak focal light—make sure that you use a bright light source.

ii. Nervous patient—adrenaline release will create a slow and incomplete PLRs.

iii. Iris atrophy—older Poodles and other breeds can have slow PLRs due to iris atrophy.

iv. Retinal disease—PRA and retinal inflammation.

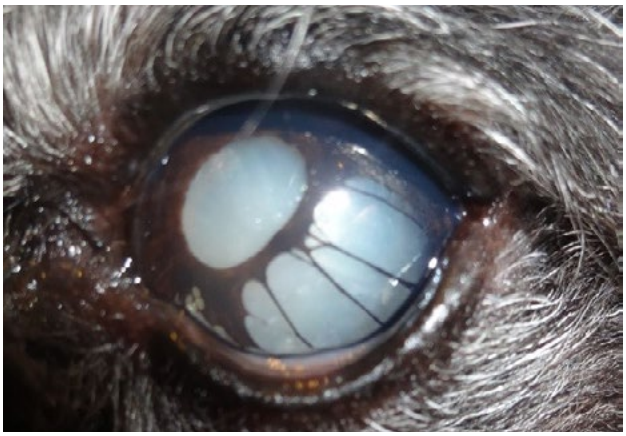


Figure 7. Iris Atrophy causing polycoria—multiple pupils—highlighted by the cataract

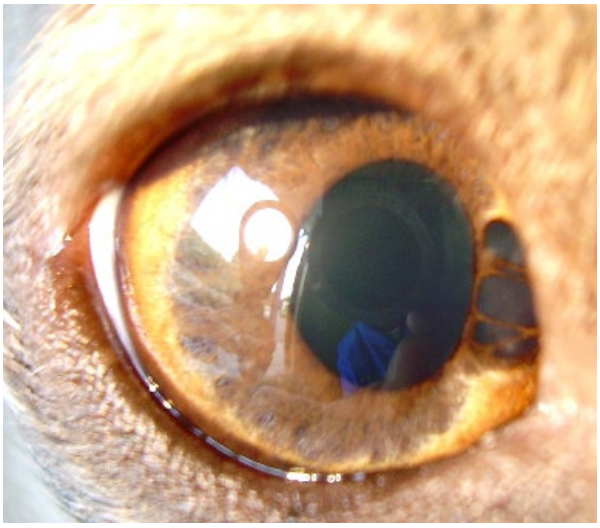


Figure 8. Note the iris atrophy medially—this dog would have a slow PLR

6. When dealing with a red eye, do not bother with the PLR

WRONG!

- At Animal Eye Care we always do a PLR in all eye cases and especially in red eyes.
- A dilated nonresponsive pupil and a red eye in a purebred dog is likely to be glaucoma. We suggest measuring the IOP in all red eyes, blue eyes and whenever you suspect glaucoma.
- We also recommend measuring the IOP in all cases of uveitis. The IOP is usually low, and this would confirm the diagnosis. Also, as the uveitis improves the IOP should increase. Again, check the IOP, if the eye looks better but the IOP is low the medication should be continued.

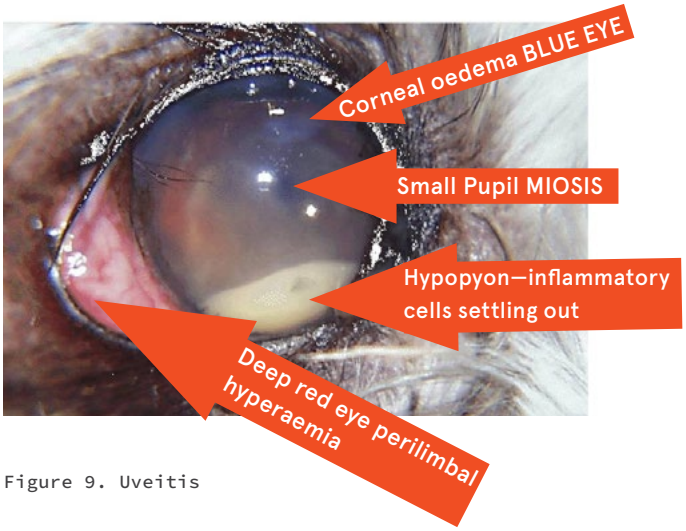


Figure 9. Uveitis

7. Use the largest possible suture when doing cherry eye surgery

WRONG!

- Cherry eye surgery can be a challenge especially in large breed dogs. In problem cases that have had previous surgery we often find that large sutures have been used. We also find large knots on the inside of the third eyelid that cause corneal ulcers.
- At Animal Eye Care we normally use 6/0 Vicryl for most cherry eye surgeries. We use a modification of Morgan technique.
- For large breed dogs over 25 kgs we often place 3 inverted simple interrupted sutures to help hold (and reinforce) the bulbar aspect of the third eyelid. We do this before closing the mucosal pocket that is created to replace the prolapsed third eyelid gland.
- We usually do a continuous suture to close the mucosal pocket. You can either bury the starting and finishing knots into the mucosal pocket OR you can place these knots on the eyelid side of the third eyelid, and then pass the suture into the pocket. Remember to include some of the connective tissue under the conjunctiva as you close the conjunctiva.

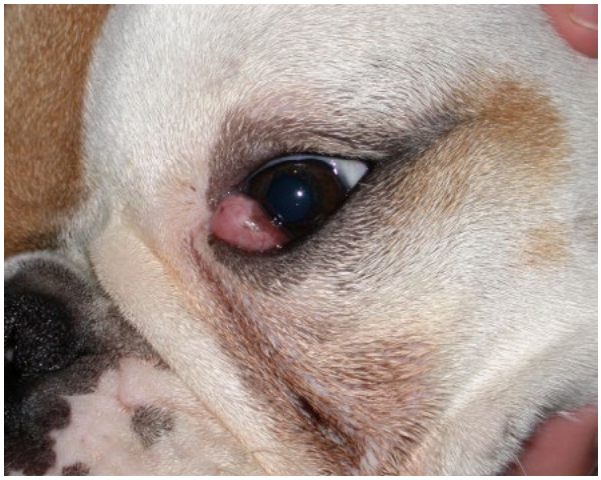
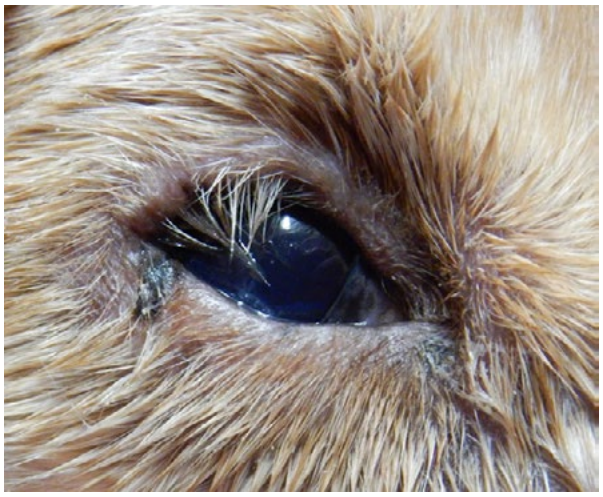


Figure 10. Cherry eye

8. Eyelid surgery is easy

Eyelid surgery can be tricky, and it is very important to use the right surgical equipment and also the right sutures. Unfortunately, from time to time we see eyelid surgeries where inappropriate sutures have used.

- Entropion surgery, eyelid tumour removals Use soft, fine, absorbable suture—e.g., 5/0 Vicryl or 5/0 Maxon. These sutures do not need to be removed, so the ends can be cut short. Some like to use 4/0 Vicryl Rapid sutures. In some cases, these sutures dissolve out too quickly.



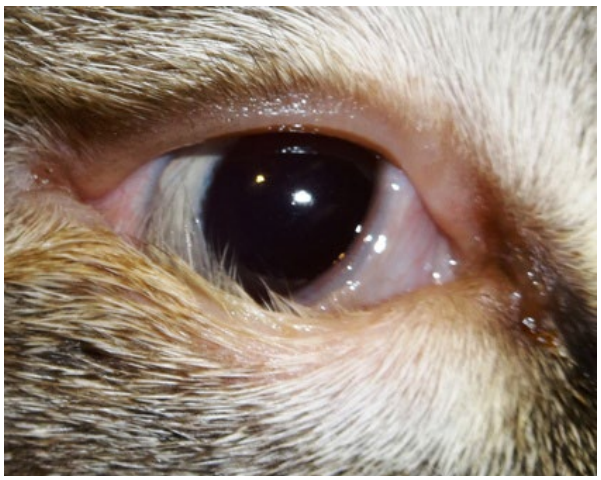


Figure 11A and B. Two entropion patients.

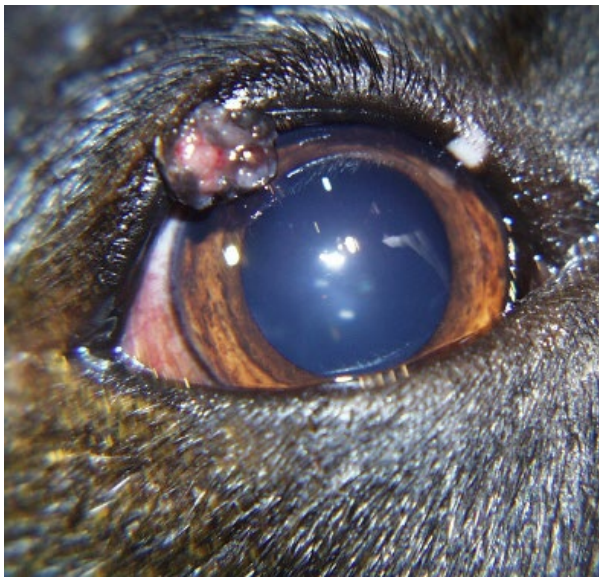


Figure 12. An eyelid tumour—trickier to remove than you might think.

- If the entropion is associated with a corneal ulcer and blepharospasm—consider placing lateral temporary tarsorrhaphy (TT) suture at same time as entropion correction. The TTs can help protect the cornea and can make the eye much more comfortable. We would use 5/0 to 4/0 nylon suture for the TTs, and we would remove these TTs (temporary tarsorrhaphy) sutures after 10 to 14 days.

9. Topical NSAIDs can be used on corneal ulcers

Topical NSAIDs such as Acular® and Voltaren® eye drops can be very useful in Veterinary Ophthalmology.

There is a common misconception that topical NSAIDs can be used on corneal ulcers.

This is WRONG!

At Animal Eye Care we see lots of melting corneal ulcers, corneal perforation, and delayed corneal healing with the use of topical NSAIDs.

DO NOT USE TOPICAL NSAIDs on an active corneal ulcer or in cases when the cornea could ulcerate.

We recommend oral NSAIDs and topical and oral antibiotics in all cases of corneal ulceration.

10. When to use a topical NSAID

At Animal Eye Care we use topical NSAID drops e.g., Voltaren® or Acular® eye drops for:

1. The treatment of Lens induced uveitis (LIU)—e.g., cataracts and also for lens rupture.
2. To treat vascular keratitis, and also to reduce vascularization/fibrosis once an ulcer has healed.
3. Uveitis in cats in dogs and cats.
4. This can be very useful in cats as topical cortisones may reactivate a latent Feline Herpes Virus (FHV-1) keratitis.
5. To prevent post-operative inflammation after cataract surgery. ◆

Further Reading
C&T No. 5601 Entropion in Eyes, Robin Stanley, Issue 286 March 2017

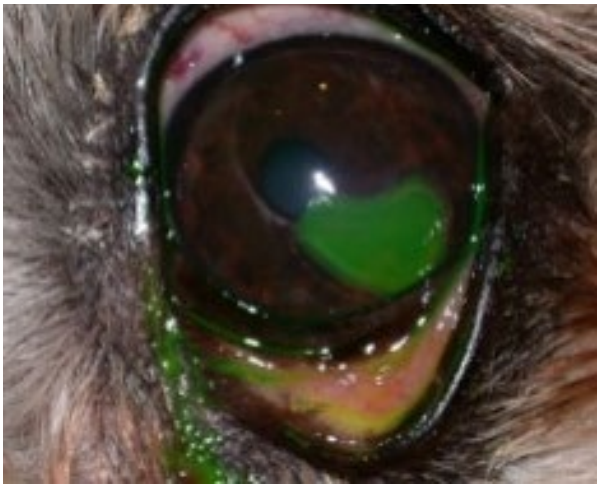


Figure 13. Corneal ulcer. Note the uptake of fluorescein.

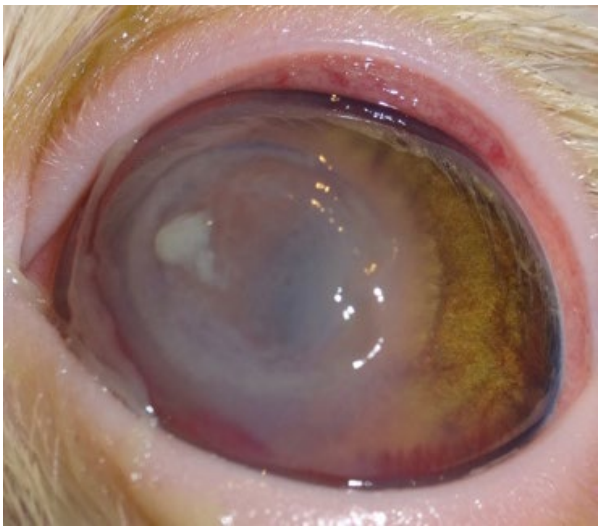


Figure 14. In this case, there is a stromal corneal ulcer with some purulent discharge (arrow). You can see blood vessels ventrally. Topical NSAIDs should not be used on this eye.

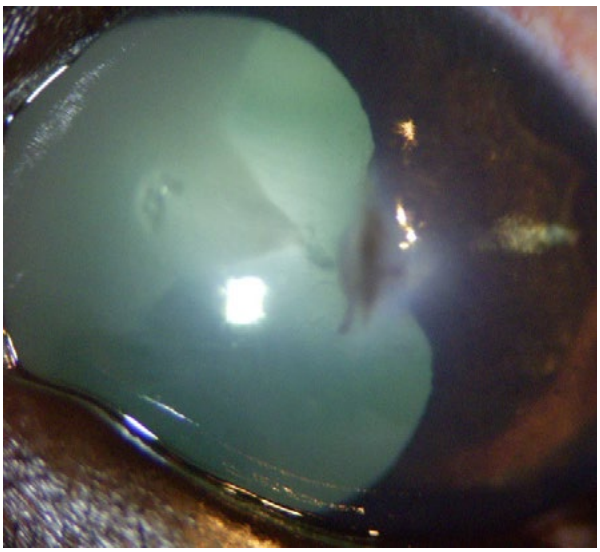


Figure 15. Lens rupture in a dog after a cat scratch

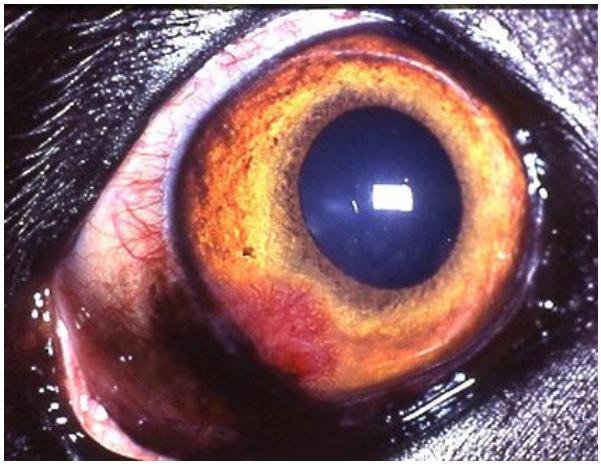


Figure 16. The ulcer in the ventrolateral cornea has healed with blood vessel—no dye uptake

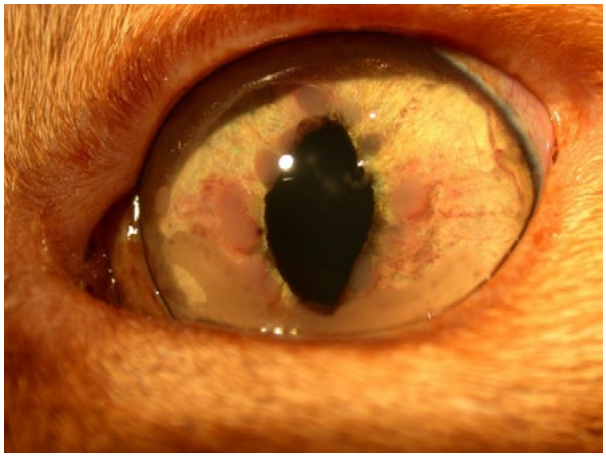


Figure 17. Fibrinous uveitis in cats

Winner

Entitled to a CVE$100 voucher

WildLife

BLINDNESS IN SOME EASTERN GREY KANGAROOS

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C&T No. 5958

Introduction

Environment and SA Water staff noticed what appeared to be an increasing number of Kangaroos affected by blindness and staggering.

Staff who are employed in culling operations regularly have to shoot badly affected kangaroos, on welfare grounds. The syndrome appears to have spread to another Reservoir area near Myponga in the past few months, and there was a concern that the condition may be infectious, and a desire to have a diagnosis to determine what actions may be taken, if any, to prevent spread.

Although a syndrome of blindness in kangaroos has been observed and described in the late 1990s[1], this syndrome appeared slightly different, in that kangaroos of all ages and sexes seemed to be affected, and affected animals had bluish or discoloured eyes.

History

Management at the SA Water catchment described their concerns, observations and request for an investigation, in early July 2022. I visited the catchment area on the afternoon of 14th July and accompanied some officers and a qualified shooter to an area where cases might be observed. We observed groups of 10-20 eastern grey kangaroos, possibly 100 in total, and in that time observed 2 cases.

Affected kangaroos were:

- Of any age (mature) and sex
- The animals appeared ataxic—stumbled and fell to the side when running, and when they got up demonstrated uncoordinated gait
- Advanced cases waste away
- All cases have 'blue discolouration' of the eye/ cloudy eyes
- Ears droop and they do not look well
- Kangaroos can still hear, and there is not total blindness.

I was shown videos of cases, and an advanced case in a large adult male eastern grey was shot, and I collected blood, brain and eyes from this case for laboratory submission. There appeared to be a keratitis/scar on the L eye (possibly a fight/ scratch), and the right eye had subtle opacity—possibly a uveitis. In observing this animal ante mortem, it appeared affected by a Central Nervous System (CNS) disorder, rather than blindness.

There is abundant *Phalaris sp* pasture in this area (mostly young and short) that appears to be heavily grazed. *Phalaris tuberosa* has been implicated in CNS disturbances in kangaroos and other species in the past.[2]

In late September 2022, a further 2 wallabies were sampled from another National Park area about 30km away, with similar symptoms. These wallabies had very similar gross and histopathological changes, and it was reported that there seemed to be more of these cases this year at that sight than previously.

Results summary

Samples submitted to the lab included fresh and fixed brain, a swab from the cranial cavity, and both eyes. Ocular fluid samples were normal for calcium, magnesium, urea and hydroxy butyrate—indicating that nervous signs were unlikely to be metabolic in nature.

Swab grew a plant pathogen, probably a contaminant.

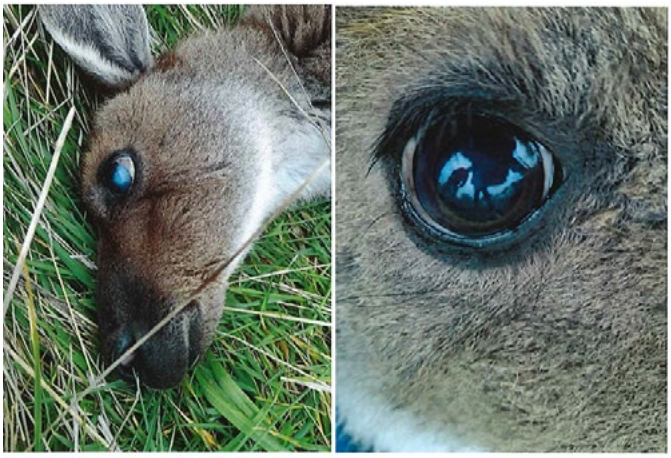

Figure 1. L eye has slight scar on surface, R eye appears 'cloudy' internally

Diagnosis

Dr Effie Lee BBioMedSc (Hons) BVSc (Hons) MPhil MANZCVS (Pathobiology)

Brainstem and spinal cord: Neuronal pigmentation with neuroaxonal degeneration and Wallerian degeneration compatible with Phalaris toxicity.

Eye: Conjunctivitis and scleritis, lymphoplasmacytic, multifocal, minimal; with glaucoma and cataract

Comments

Brainstem and spinal cord histopathology of perinuclear brown granular pigment in neurons, with neuroaxonal degeneration and mild lymphoplasmacytic meningoencephalitis, is compatible with Phalaris toxicity. The intramyelinic oedema in the white matter tracts is typical of exposure to high levels of ammonia and could suggest toxic impairment of urea-cycle enzymes.

Cataract is a common sequel to inflammatory-associated changes in the eye. Glaucoma would cause the iris to bow forward and adhere to the cornea (iris bombe); clinically, the eye would be painful rather than blind in the absence of RPE and optic nerve pathology. The perivascular lymphoplasmacytic infiltrations in the conjunctiva and sclera suggests probable immune mediated response to antigenic stimulation (e.g. Bacterial, viral, chemicals, foreign proteins) or initiating traumatic events. Possible viral diseases affecting eyes of kangaroos (e.g. Wallal, Warrego, and Herpes) cannot be excluded in this case.

In general, Phalaris toxicity causes a stagger syndrome associated with the ingestion of *Phalaris aquatica* species containing toxic alkaloids. Characteristic findings involve storage of perinuclear green-brown granular pigment within neurons of the brain stem nuclei, spinal gray matter and dorsal root ganglia, and in macrophages of cerebral spinal fluid. Typically, there is concurrent damage to descending motor tracts characterized by Wallerian degeneration of the spinal cord and brainstem. Clinical onset of staggers syndrome may be delayed for several months after exposure to toxic plants. Nervous signs can persist for two months and have been known to manifest up to five months after animals have moved off Phalaris.

Discussion

Orbiviruses of the Wallal and Warrego serogroups were isolated from kangaroos affected with blindness in a major epidemic in south-eastern Australia in 1994 and 1995 and extending to Western Australia in 1995/96. Histopathological examinations showed severe degeneration and inflammation in the eyes, and mild inflammation in the brains. In affected retinas, Wallal virus antigen was detected by immunohistochemical analysis and orbiviruses were seen in electron microscopy. There was serological variation in the newly isolated Wallal virus from archival Wallal virus that had been isolated in northern Australia.[1]

In this case there was some concern that observed blindness in these kangaroos may have been a reappearance of these viruses; however, it appears that in this case Phalaris grass toxicity is responsible. Further tests are pending in this case to detect viruses, if present.

This kangaroo may not have been typical of previous cases but consistent observations were made that the eyes appeared to be opaque or discoloured in most other cases- and this was not the case here.

Environment and SA water staff supplied two further wallabies from another nearby Water catchment area on 20/9/2022. These animals were reported as suffering very similar symptoms to the kangaroo earlier described, with the comment that `there have been a lot of these this year, but not many seem to die from it'. Histopathological changes in these two wallabies also confirmed *Phalaris* toxicity.

Phalaris toxicity has been documented previously in Kangaroos in SA[2] in 2014 and elsewhere in Australia[3] but it is helpful to investigate unusual syndromes in animals where they occur. Some reasons for this include:

- Confidence in the public that animal welfare and health is being cared for in National Parks, particularly in close urban areas
- Investigation adds to the pool of published information that builds over time
- Investigations may assist in a diagnosis that might reduce impacts, or therapies may be available in some cases

References

1. PT Hooper, RA Lunt, AR Gould, AD Hyatt, G M Russell, J A Kattenbelt, S D Blacksell, LA Reddacliff, P D Kirkland, R J Davis, P J Durham, AL Bishop, J Waddington Epidemic of blindness in kangaroos--evidence of a viral aetiology *Aust Vet* J 1999 Aug;77(8):529-36
2. B Bacci, PL Whiteley, M Barrow, PH Phillips, J Dalzielc and CM El Hagea Chronic phalaris toxicity in eastern grey kangaroos *(Macropus giganteus) AVJ* 92, No 12, December 2014 ◆

Large

WHAT IS YOUR DIAGNOSIS?

PATCHY ALOPECIA IN A MATURE HORSE

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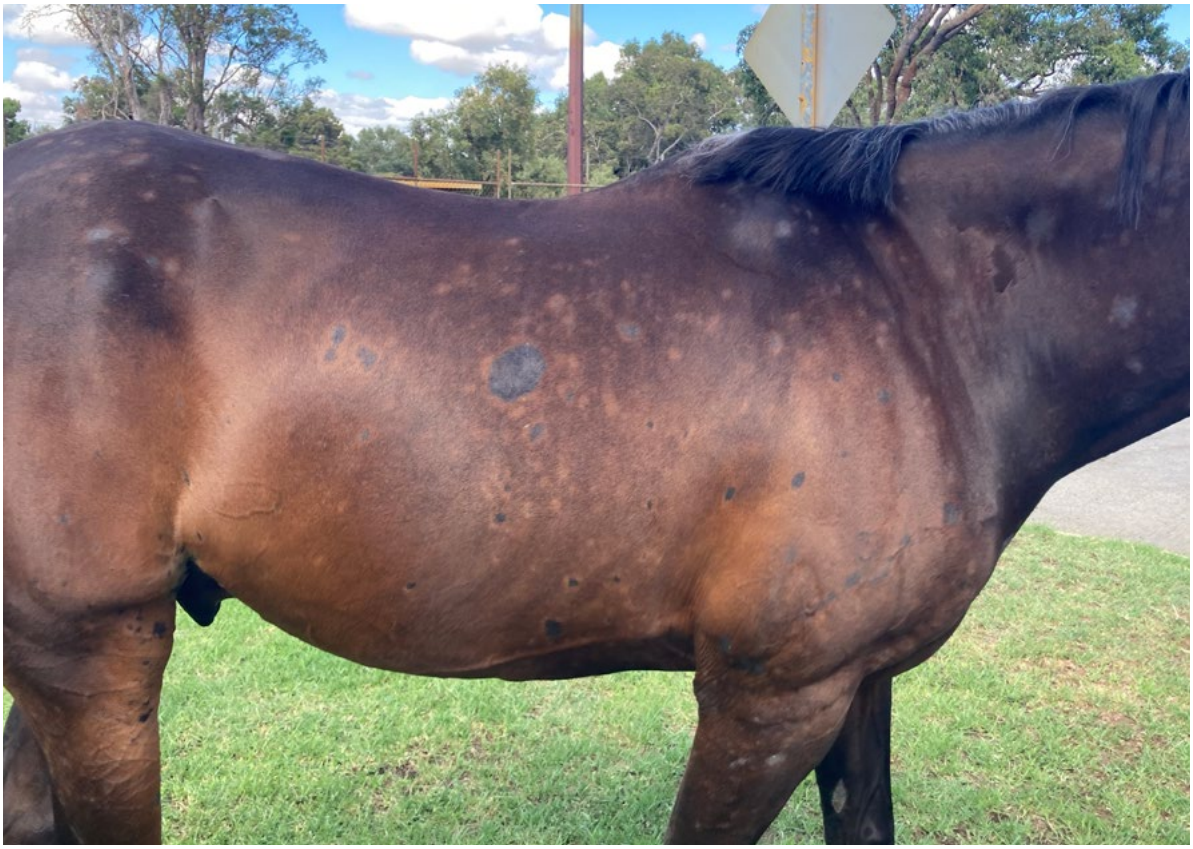
C&T No. 5959

Thanks to Dr. Janet Littlewood MA PhD BVSc DVR DVD MRCVS

An 18-year-old Arabian gelding presented for non-pruritic, patchy areas of alopecia. Lesions initially occurred on the ventral neck, then progressed to involve the trunk, limbs, rump and face. There were no other dermatological abnormalities noted; the horse was systemically well.

- What is your tentative diagnosis?
- How would you investigate the patient?
- How would you treat this case?



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Winner

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Large

A CASE OF CLOSTRIDIAL MYOCARDITIS IN AN ANGUS STEER

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In September 2021, I was called out to a property just outside of Singleton in the NSW Hunter Valley to an ataxic yearling Angus steer. Upon arrival to the property the steer had just died. There was no supplemental feeding on this property, with the property being a small non-improved pasture-based system. All animals had been drenched recently (unknown product), there was no history of other sudden deaths on the property, but the farmer mentioned that his neighbours had lost a few cattle recently.

All cattle on the property had apparently been vaccinated with at least 2 initial doses of Ultravac®7in1 vaccine and were re-vaccinated annually. This steer was confirmed to have been vaccinated twice as a calf.

There was no history of cloudy weather so nitrite toxicity was unlikely; there was no obvious bloating, subcutaneous emphysema/crepitus (black leg), external haemorrhage (anthrax *vs* trauma), jaundice, mucosal pallor, diarrhoea or any external lesions that would explain the sudden death. The steer was found in a paddock with a creek running through it so Green Cestrum toxicity was on my list of differentials.

On post mortem examination, the steer was in good body condition with no external abnormalities. The heart was the only obviously affected organ. There was a serosanguinous fluid with fibrin clots in the pericardial sac and black streaks of presumed necrosis on the myocardium (Figure. 1). Given the sudden death of the animal, a clostridial disease was suspected and organ samples were sent away for histopathology and culture.

The steer had died from a 'severe necrotising myocarditis consistent with *Clostridium chauvoei* infection'. This is a rare visceral form of *Clostridium chauvoei* infection (which usually manifests as the classic myositis commonly referred to as 'Black Leg') and the EMAI pathologist seemed excited to have diagnosed it!

There is a Brazilian case study on clostridial myocarditis, but this bull had not been vaccinated for any clostridial disease (Casagrande et. al). The farmer immediately revaccinated all his animals for clostridial diseases and to my knowledge has not had any more sudden death cases.

Reference

Casagrande, R.A., Pires, P.S., Silva, R.O.S., Sonne, L., Borges, J.B.S., Neves, M.S., Rolim, V.M., Souza, S.O.D., Driemeier, D. and Lobato, F.C.F., 2015. Histopathological, immunohistochemical and biomolecular diagnosis of myocarditis due to Clostridium chauvoei in a bovine. Ciência Rural, 45, pp.1472-1475.

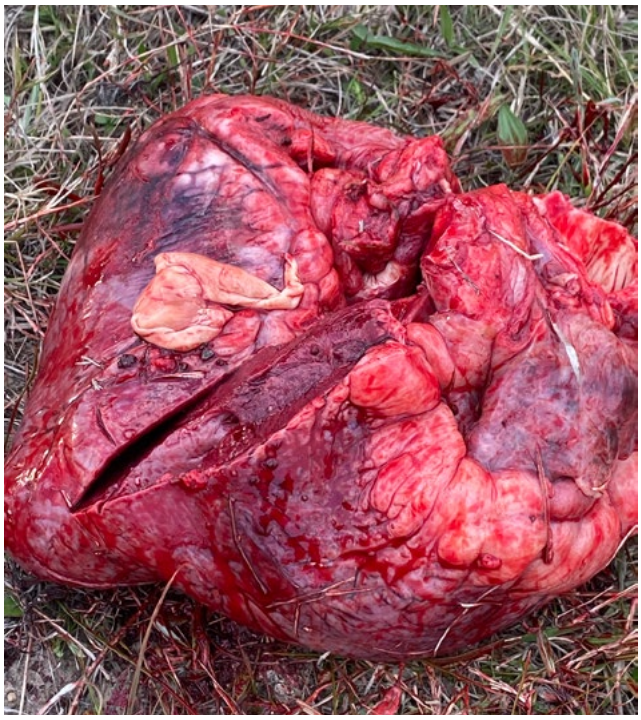

Figure 1. Myocardial necrosis of the steer heart

COMMENT COURTESY OF

Mark Krockenberger
Professor of Veterinary Pathology
Sydney School of Veterinary Science

This is a fascinating case of an unusual occurrence. A case report in the *Veterinary Record* in 2003 by Uzal *et al* about an outbreak of clostridial myocarditis in 7-month-old Herefords in the Patagonia region of Argentina, also drawing on the literature from Gastonbury *et al* 1988 (sheep) and Helman *et al* 1997 (cattle),

suggested that stressful events (yarding) and subsequent hypercortisolaemia and/or release of catecholamines may have been sufficient to cause sufficient physiological changes in the myocardium to stimulate resting spores of *C. chauvoei* to germinate, resulting in clostridial myocarditis. Rain events and increased proliferation in wet soils were also speculated upon as predisposing to the cases in the case report by Uzal *et al* (2003).

I wonder if our recent wet weather in combination with stressful events could have combined to result in this unusual occurrence?

Reference

Glastonbury JRW, JE Searson, II Links, LM Tuckett (1988). Clostridial myocarditis in lambs. *Australian Veterinary Journal* 65:208-209

Helman G, RD Welsh, EI Stair, RW Ely (1997). Diagnosing visceral blackleg as a cause of sudden death in cattle. *Veterinary Medicine* 92:914-918

Uzal, FA, M Paramidani, R. Assis, W Morris, MF Miyakawa (2003). Veterinary Record 152: 134-136 ◆

General

PASTEL PETS

Heather Crisp
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Heather is an animal-loving retired art teacher who began working in pastel during lockdown. She is able to capture an animal's unique personality, and has discovered her dream job creating portraits of very special pets. She mostly focuses on dogs but also draws other animals and landscapes, usually working from a photograph. ◆

General

HOT WATER BOTTLES

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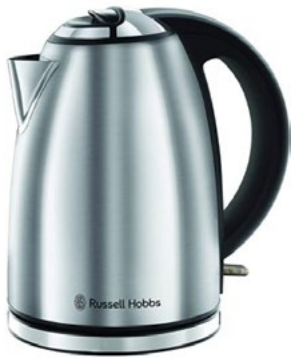
C&T No. 5961

During a nine year study[1] with a cohort of human burn victims (n=85), 19% had been burnt by direct contact with hot water bottles (HWB), 48% with split bottles and 33% by spilt hot water, when filling etc. Why are they used, why does heat and light physical pressure help?—Apparently it is to do with stimulating larger diameter nerves fibres, which 'closes off pain gateways' in the spinal cord—so altering pain perception in the brain.

However, associated burns can be extensive and require long therapy periods e.g. surface area effected averaged 3.07% and length of hospitalisation was a mean of 28.9 days, both issues having a wide range of values.[1]

Veterinary clinics use various forms of hot packs, as do people with their pets. Occasionally animals are burnt, especially if they are unable to move and heat sources are in direct contact with their skin.



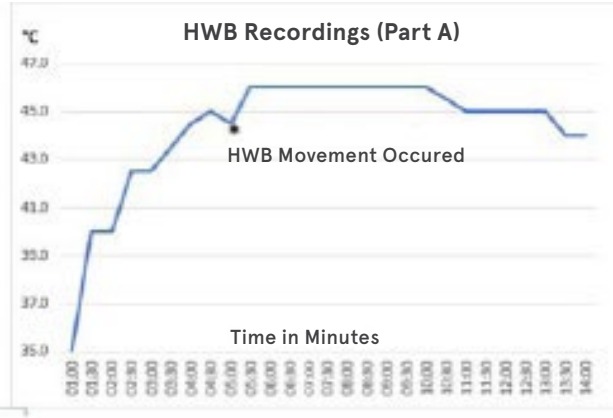
Outlined below is what happens 'heat-wise' with an uncovered and covered HWB, when recording temperatures over time—both from the bag, the environment and via the slightly ribbed surface of a traditional rubber HWB.

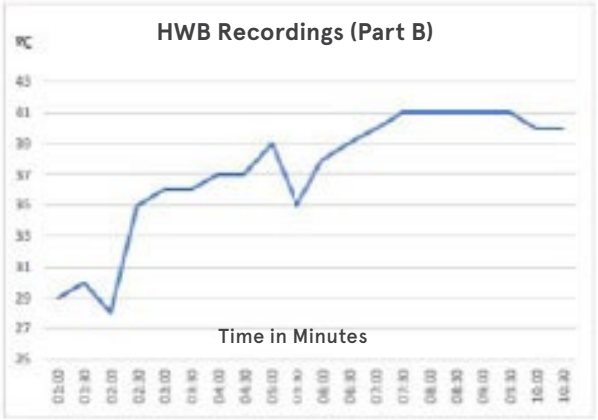
All temperature recordings (Parts A & B) were rounded to the nearest 0.5°C. Recordings occurred every 30 seconds using a large wall clock with a clearly visible second hand. Water was boiled in a standard kitchen kettle. The water temperature, as it was poured into the HWB from the kettle was >50°C (beyond thermometer capacity). Recordings continued until temperatures started to fall.

Once sealed, the HWB was placed directly (Part A) on the min/max "up-market" thermometer (which was laying on a cold inert marble surface) and readings were subsequently recorded. Room temperature was also recorded. It was hoped this would represent an uncovered ribbed HWB directly placed on e.g. a paralysed animal.

The room temperature was consistent at 20°C, the HWB water temperature was 44°C at conclusion (Part A Graph) and the thermometer temperatures peaked at 46°C over 9 consistent recordings. At the 20 min. mark (off graph) the temperature was still 44°C. The temperature rose from 35°C to 46°C over 9 recordings, which continued up to the 9.5 min. mark.

This process was repeated (Part B), but with the HWB wrapped (8 folded layers) in an older, thinner cotton pillowcase. The same procedures were repeated. Room temperature was 29°C (2 hours after Part A) and during 20 recordings, the temperature reached 41°C (over a 2.5 min segment), before starting to fall at 10.0 mins.





Dips at 2.00 and 5.30 were associated with spilt hot water (or to the operator) due to residual water in the crown of the HWB after filling. This may have affected the range of temperature?

This pilot data suggests that an uncovered HWB could easily burn the skin (with 46ºC direct contact) and with temperatures above or at 44ºC for 21 continuous 30 sec. recordings. Even when covered, 41ºC may cause some harm over a 2.5 min. period.

To ensure that injuries do not occur, close observational care is required, especially in animals that can't move away from direct heat, especially if an uncovered HWB is applied.

Reference

JABIR, S. *et al* (2013) *Plast. Surg. Intern.* Vol. 2013 - Article ID 736368

** Many human hospitals have banned the use of hot water bottles.* ◆

Small

ANAESTHESIA, HEAT LOSS & WHAT IT MEANS FOR OUR PATIENTS

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C&T No. 5962

Hypothermia is a very common complication in general anaesthesia and it is associated with multiple complications. The risk of hypothermia can be minimised in most cases and, when this cannot be prevented, the animal can be actively warmed. This short review covers:

- the mechanisms of heat loss
- when the loses tend to occur, and
- some considerations about prevention of heat loss and active warming.

Homeothermic animals can maintain the body temperature within a tight range regardless of the environmental temperature. In summary, temperature-sensitive cells located in the hypothalamus detect the core body temperature and instigate the heat production/retention processes. But why do animals lose more heat during anaesthesia? That is a question that has multiple angles:

Animals lose heat via convection, radiation, evaporation, and conduction. Due to this, it is important to control the environment around our patient. We cannot prevent the radiation (from the skin) or the convection (movement of air around the skin), but we can minimise them, using reflective blankets (also known as shock or space blankets) and covering and wrapping all the available part of the animal's body. Conduction losses (for example to the cold metal table) can be prevented by the use of an insulated mattress or the table can be covered with a reflective material, so we also prevent radiation in that part of the body. Evaporation is a very important loss during anaesthesia as our patients breathe very dry oxygen coming from the cylinder. Oxygen generators are slightly better, but they also produce oxygen with a low humidity level (certainly below the room air humidity). As the gases are delivered directly to the trachea without passing through the nasal cavity, the humidification must be done by the trachea (and this is an organ not prepared for it), so this is done at a great cost of heat (latent heat of evaporation). For this reason, it is recommendable to use heat and moisture exchangers (HME) to minimise this loss.

Additionally, during general anaesthesia, the central nerve system is depressed, so the temperature sensitive cells in the hypothalamus are less responsive than during normal conditions. The patient is not moving, so there is no contribution from skeletal muscle to the body heat, and metabolism is low, so heat from organs such as *the liver is also relatively low.*

Hypothermia is a general depressant. Enzymatic activity is decreased, and this might lead to a relative overdose or longer duration of drugs. Eger and others (1965) demonstrated a reduction of 30% in the minimal alveolar concentration of halothane with a body temperature of 33ºC. And there are multiple studies reporting similar effects with different volatile anaesthetics and species.

This depression effect might be responsible for the increase in mortality associated to hypothermia that was observed by Gil and Redondo (2008).

In two multicentre studies, one in dogs and one and cats, they observed that more than 80% of dogs and 97% of cats suffered hypothermia during general anaesthesia. Some interesting information from this study suggests that the duration of the procedure, the reason for anaesthesia (being abdominal and orthopaedic surgeries) and ASA

Scores over III out of V were associated with lower body temperatures at the end of the anaesthetic. The body temperature before premedication was a positive prognostic indicator (higher temperatures were associated with better temperatures postoperative). Another interesting fact from these studies was that, in both species, the core temperature dropped by more than 1ºC between the basal (prior to premedication) measurement and the induction time. This points out a crucial phase during the anaesthetic procedure that is usually neglected.

It is undoubtable that warming the patient is the way forward when the animal has lost core temperature, but this warming is not without risk. In people, skin temperatures over 43ºC have been associated with burns (Martin 2017) and there is a well established time-temperature risk (44ºC skin temperature over 3 hours resulted in thermal skin insult)(Moritz and Henriques, 1947). The veterinary literature is not as detailed, but there are plenty of reports of skin burns associated to warming devices (especially those without an upper temperature control) and even a case series (Dunlop *et al*, 1989).

In summary, prevention is key in the prevention of hypothermia. This prevention would allow a higher core temperature and consequently if external warming is needed, this will not have to be aggressive (which intrinsically decreases the risk of skin burns).

References

Dunlop CI, Daunt DA, Haskins SC. Thermal burns in four dogs during anesthesia. Vet Surg a1989;18:242-246.

Martin NA, Falder S. A review of the evidence for threshold of burn injury. Burns 2017;43:1624-1639.

Moritz AR, Henriques FC. Studies of thermal injury: II. The relative importance of time and surface temperature in the causation of cutaneous burns. Am J Pathol 1947;23:695-720

Gil L, Redondo JI. Canine anaesthetic death in Spain: a multicentre prospective cohort study of 2012 cases. Vet Anaesth Analg 2013;40(6):e57-67. ◆

All Creatures Great and Small ... these Franciscan Sisters Love Them All

Article contributed by Dr Stephanie McEwan, California

Contents

Feline Chronic Kidney Disease

Clinical Trial

e. jwhite@sashvets.com

Chronic kidney disease is one of the most common health conditions in older cats.

It affects the majority of cats over the age of 10, and is a contributing factor to early mortal and reduced quality of life.

The SASH Clinical Research Team are currently enrolling cats for a chronic kidney disease trial. Research into this area can help to significantly improve outcomes for the many cats suffering from chronic kidney disease.

Costs will be covered by SASH and cats will be sent back to their normal GP for routine care.

Please contact Joanna White.

Find out more and read this article in the eBook.

Small

LEPTOSPIROSIS UPDATE 2022

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C&T No. 5963

This summary is to give you an update on leptospirosis cases seen in July 2022 only.

To date, 60 cases of canine leptospirosis have been confirmed in NSW with the first reported case in 2017, two cases in 2018, eight in 2019, seven in 2020, ten in 2021 and a surge in case numbers in 2022. Diagnosis was based on the presence of typical clinicopathological findings and the presence of positive PCR in blood (n=2), urine (n=2), blood and urine (n=8), a single high antibody titre ≥1/800 (n=10) or seroconversion (n=4). The case fatality was 31% (5/16). Five dogs were euthanised due to severe renal failure. Two dogs had been vaccinated with the currently used vaccine containing *Leptospira interrogans* serovar Copenhageni (Protech®C2i, Boehringer Ingelheim) and both of those dogs recovered. Fifteen dogs had not been vaccinated against leptospirosis. Microscopic agglutination testing (MAT) was conducted in 14/17 dogs. In twelve dogs serovar Australis was thought to be the likely causative serovar based on MAT titres ranging from 1/50 (n=1), 1/100 (n=2), 1/800 (n=2), 1/1600 (n=4), 1/3200 (n=1) to 1/6400 (n=2). One dog had equally high titres to serovars Australis and Copenhageni (1/1600). In one dog MAT was negative likely due to insufficient time for seroconversion. Cases were located in the following suburbs: Bayswood (n=2), Jervis Bay(n=1), Old Erowal Bay (n=5), Sanctuary Point (n=2), Sussex Inlet (n=1), Tomerong (n=2), Woollamia (n=2) and Vincentia (n=1). All of these are located in the LGA of Shoalhaven. We are working with the manufacturer of a limited permit *Leptospira* serovar Australis vaccine (Treidlia BioVet Pty Ltd) to acquire approval by the AVPMA to use this vaccine in dogs living in and visiting the LGA of Shoalhaven.

A further hot spot has been Newcastle with five reported cases between April and June. Diagnosis was based on typical clinicopathological findings and the presence of positive PCR in blood (n=1), urine (n=2), blood and urine (n=2) and/or seroconversion (n=1). The likely causative serovars were Copenhageni (n=1), Pomona (n=1) and Icterohaemorrhagiae and Zanoni (n=1). One dog died and one was euthanised due to severe renal failure. None of the dogs were vaccinated.

In May and September two cases were reported in the LGA of Wollongong. For one dog from Figtree, serovar Australis was the causative serovar. For another dog from Kembla Grange, no MAT was performed and the causative serovar could not be determined. Both dogs recovered.

In July a dog from Potts Point was reported as a case. The causative serovar was Copenhageni and the dog recovered. The vaccination status was unknown.

In June a case was reported in Tuggerah. The causative serovar was Copenhageni. The outcome of the dog was not reported and the vaccination status was unknown.

In April, one case was confirmed in Ingleside, Northern Beaches. Sadly, this dog was euthanised. The causative serovar could not be determined, likely due to insufficient time for seroconversion. The dog had received its first leptospirosis vaccination one day prior to onset of clinical signs. Also in April, a case was reported from Burradoo near Bowral. **This dog had positive titres of 1/800 to serovars Copenhageni, Icterohaemorrhagiae and Djasiman and recovered.** The dog was not vaccinated against leptospirosis.

In March, a case was confirmed in Marrickville. The dog had access to a backyard with rats. The dog was euthanised and on postmortem exam multiple intussusceptions were found—a known complication of leptospirosis. Again, the causative serovar could not be determined, likely due to insufficient time for seroconversion. The dog was not vaccinated against leptospirosis.

In addition to the cases reported in NSW, one dog was reported in Jerrabomberra in Canberra in June. This dog was euthanised due to anuric renal failure. No MAT was performed in this case so the

causative serovar could not be determined.

We strongly recommend vaccination against leptospirosis for dogs living in the Newcastle and South Coast areas or in any area if the dog is in contact with rats or other rodents. Vaccination in other geographic locations with confirmed leptospirosis cases should be considered and discussed with clients. For dogs that live in and visit the Shoalhaven LGA, vaccination with Leptospira serovar Australis should be considered (if this vaccine is approved for use in dogs in this area). We appreciate that there is a current shortage of the widely used vaccine Protech® C2i from Boehringer Ingelheim containing serovar Copenhageni. For concerned owners unable to complete a primary vaccination course or the annual booster vaccination for their dog due to this shortage, we recommend providing the following advice to clients:

> *Risk mitigation methods are the most important measures to prevent leptospirosis infection. Contact with sources of infection should be limited. This includes limiting contact with, swimming in, and drinking out of, stagnant water and avoiding contact with possible reservoir hosts such as rodents and farm animals, which can be achieved by fencing and rodent control. Similarly, contact with leptospirosis infected dogs should be avoided. In endemic areas—especially during leptospirosis outbreaks—close dog-to-dog contact like doggy day care and boarding in kennels should be reconsidered.*

The University of Sydney is continuing to investigate leptospirosis cases to determine which serovars are causative and if there is any specific source of infection which can be identified.

In a subsequent study we are investigating the immune response and its implication for the diagnosis of natural infection after vaccination with the currently available monovalent vaccine containing *Leptospira* interrogans serovar Copenhageni.

Leptospirosis may be suspected in any dog with:

- Nonspecific clinical signs like lethargy, vomiting and diarrhea, which can precede more obvious clinical signs like icterus
- Azotemia
- +/- hyperbilirubinaemia, elevated liver enzymes
- +/- glucosuria

Important information to ask:

- Is there any contact with rats?
- Is there any contact with stagnant water (e.g. ponds)?
- Which area is the animal from?
- Has there been any travel into areas in which there have been reported cases (Annandale, Ashfield, Balmain, Bayswood, Burradoo, Cardiff Heights, Cheltenham, Cooks Hill, Crows Nest, Darlinghurst, Elanora Heights, Erskineville, Figtree, Firefly, Glebe, Horsley Park, Ingleside, Jervis Bay, Kembla Grange, Lurnea, Marrickville, Medowie, Newcastle, Newtown, Old Erowal Bay, Paddington, Potts Point, Redfern, Sanctuary Point, Speers Point, South Coast, St Georges Basin, Sanctuary Point, Surry Hills, Sussex Inlet, Tomerong, Tuggerah, Trunkey Creek, Vincentia, Waterloo, Woollamia)?
- Of special importance are the movements of the dog in the 30 days prior to developing clinical signs.

In suspicious cases we recommend the following:

Collect urine **and** EDTA blood samples **BEFORE** giving antibiotics—and send to IDEXX or Vetnostics for PCR – if you obtain a positive result, please inform us about the case and request the laboratory ships leftover samples to us after obtaining client consent—these will be useful for further research.

Collect a serum sample—send to IDEXX or Vetnostics for antibody testing (this will help to identify the infecting serovar). If there is a high index of suspicion of leptospirosis but the PCR is negative, it is important to perform another titre 2 weeks later to determine whether there has been seroconversion. Similarly, in confirmed cases of leptospirosis a follow up titre will be helpful to determine the causative serovar.

Ensure appropriate PPE (gloves and gowns) are worn when handling the animal, as leptospirosis is a zoonotic disease.

Start treatment with IV fluids and antibiotics. IV penicillin derivatives such as ampicillin or amoxicillin are recommended initially, however, these will not clear the organisms from the kidneys. To clear the infection, oral doxycycline (5mg/kg BID or 10mg/kg SID) should be given for 14 days once the patient can tolerate oral medication.

The animal should be isolated from other animals and only be handled with appropriate PPE. We currently recommend isolation for 72 hours following the commencement of antibiotics. Ideally a urinary catheter should be placed to monitor urine output and avoid contamination of the environment with urine.

Due to the zoonotic risk of leptospirosis the owner(s) should be advised to seek medical advice.

We kindly ask that you report any suspicious cases to the UVTHS

And request you obtain and store **serum, EDTA and urine samples** if you can for us (please separate serum, use small urine tubes if possible and freeze samples if stored for >1 week—if storage time is less, we can come and collect the samples or organize a courier). If you have a deceased dog which is a confirmed or suspicious case and the owners have given consent for a post-mortem examination, please do not freeze the body—contact us and we will organize collection of the body. If you have a high index of suspicion for leptospirosis however the client is financially constrained, please contact us and send us the history and blood results for the patient. **We have a small amount of research funds available to cover costs for leptospirosis testing in those cases.**

In-contact dogs should be treated with a 14-day course of doxycycline. If possible, and after obtaining client consent, please collect whole (EDTA) blood, urine and serum from these in-contact dogs before starting doxycycline. This will help us to assess if in-contact dogs are infected without having clinical signs (silent shedders) or have been exposed to leptospirosis without being infected. Please contact us and we will provide you with an appropriate submission form. **The costs for testing in contact dogs will be covered by us and we will inform you about the results.**

Similarly, if you vaccinate dogs against leptospirosis in the Newcastle area or in the South Coast area, we would appreciate if you could collect the following samples before the dog's initial vaccination (only in dogs never vaccinated against leptospirosis):

- Serum tube
- EDTA tube (whole blood)
- Urine sample (can be free catch)

Please process the samples as outlined above and notify us so that we can collect these samples from your clinic.

If you have any suspicious cases or have samples to collect or if you want to discuss a case, please contact Dr. Griebsch christine.griebsch@sydney.edu.au. ◆

Distance Education Early Bird Winners for 2023

Congratulations to:

The Super Early Bird Winner (30 June):

Annie Yun Han Chen of Koala Park Veterinary Surgery, QLD, Australia.

The Early Bird Winner (31 October):

Briar Morton, locum veterinarian in NSW.

Annie and Briar are both enrolled in the Internal Medicine: A Problem Solving Approach Distance Education course in 2023 with tutors Jill Maddison and Sue Bennett.

400+ veterinarians from around the world enrolled in our premium Distance Education courses in 2021

Still time to enrol in 2023!

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2022 DISTANCE EDUCATION

Distance Education is demanding and requires dedication and commitment, especially when juggling study commitments with work and family. Thanks to all our participants for a rigorous yet rewarding year of continuing education.

—CVE Tutors & Staff

Anaesthesia & Analgesia: Fundamentals

Tutors: Christina Dart, Eduardo Uquillas

Stephanie Nga Ching Kam, Hong Kong
Herbert Bruinenberg, Netherlands
Geraldine Pearson, NSW
Yvonne Van Der Veek, NSW
James Le, NSW
Genevieve Lee, NSW
Jeff Howe, NSW
Allen OGrady, QLD
Stacey O'Regan, QLD
Yun Jin, QLD
Michele Goh, SA
Emma Kennare, SA
Daniel Lawrence, SA
Mark Reeve, SA
Xinyi Eunice Chan, Singapore
Tiyaton Tuksaranupong, Thailand
Anchisa Chaloempornpong, Thailand
Kunal Nagaich, VIC
Clare ABeckett, VIC
Darrell Gust, VIC
Yuni Lin, VIC
Kyla Ballard, VIC
Samantha Cheetham, VIC
Alison Simmons, VIC
Shirley Chow, WA
Philippa Simms, WA

Behavioural Medicine

Tutors: Kersti Seksel, Debbie Calnon, Jacqui Ley, Sally Nixon, Isabelle Resch

Ryan Johnstone, Ireland
Eimear Geary, Ireland
Heidi Hiu Wa Hon, Hong Kong
Prerna Vaswani, India
Natalia Kopylova, Russia
Jennifer Tseng, New Zealand
Cynthia Cricri Vera, NSW
Caitlin Mcquarrie, NSW
Rachel Nightingale, NSW
Joanne Cheney, NSW
Chloe Friend, NSW
Peta Macarthur, NSW
Chiao-Fang Chen, NSW
Arielle Giles, NT
Caroline Kerr, QLD
Kirsten Krogh, QLD
Katherine Loring, QLD
Pooja Bahal, QLD
Karen Smith, QLD
Emma Ruck, SA
Elin Lindell, Sweden
Claire Rhodes, VIC
Annie Tao, VIC
Tessa Poot, VIC
Corrie Pinkster, VIC
Jade Brown, VIC
Laura Vissaritis, VIC
Anna Petty, VIC
Chantelle McGowan, VIC
Christine Hou, VIC
Nicole Hillier, VIC
Zara Meer, VIC
Rupert Baker, VIC
Debbie Prattley, New Zealand
Ping-Yin Wang, Singapore

Clinical Pathology

Tutor: Sandra Forsyth, Karen Jackson

Gwen Shirlow, ACT
Sujit Seshadri, India
Juliana Zardo, Denmark
I-Te Lu , Hong Kong
Nicola Kynoch, NSW
Catherine Le Bars, NSW
Jenny Yan Lam Chu, NSW
Charmaine Frith, NSW
Jess Liddiard, NSW
Teri Bellamy, NSW
Ellie Moore, NSW
Christiana Willenborg, NSW
Keisuke Harada, QLD
Daniel McEvoy, QLD
Yvette Ellen, QLD
Krysten Lee, QLD
Claire Taylor, QLD
Lucilla Pratt, QLD
Josephine Hartono, QLD
Geoff Brown, SA
Lyndsay Smith, SA
Vanessa Sabio, Singapore
Tyler Wu, Taipei
Ruth Gore, VIC
Georgia Hocking, VIC
Kelsey Nigbor, VIC
Wanran Luo, VIC
Karlien Penning, VIC
Suki Cheung, VIC
Lesley Fantin, WA
Hannah Broadhurst, WA
Stephanie Liow, WA
Amy Khoo, WA
Priya Tharasi Sukunan, Malaysia
Liu Dongtai, Singapore
Heidi Hiu Wa Hon, Hong Kong

DERMATOLOGY

Tutors: Ralf Mueller, Sonya Bettenay & Stefan Hobi

—Advanced

Andrea Holthaus, Germany
Margot Tong, NT
Nutjira Sawatmongkol, Thailand
Sutapa Loyawatananan, Thailand
Marisa Saributr, Thailand
Korntanat Samattasinwanit, Thailand
Chaniporn Srisakdi, Thailand
Wiwitsana Saengphoem, Thailand
Kathy Gillies, VIC
Shanna Harris, WA

—Infectious Skin Disease

Carrie Tay, NSW
Andrew Herron, NSW
Ella Massy-Greene, NSW
Margot Tong, NT
Joanne Mclaughlan, QLD
Darren Burgess, SA
Apinyawat Naksittiwong, Thailand
Emma Bruce, TAS
Ubonpan Kansap, Thailand
Wiwitsana Saengphoem, Thailand
Korntanat Samattasinwanit, Thailand
Kathy Gillies, VIC
Sutapa Loyawatananan, Thailand
Marisa Saributr, Thailand
Chaniporn Srisakdi, Thailand
Nutjira Sawatmongkol, Thailand
Jiraya Chaiprapa, Thailand

—Pruritic Skin Disease

Emma Feeney, ACT
Andrea Holthaus, Germany
Claire Choe, NSW
Carmen Ali, NSW
Margot Tong, NT
Olivia Heggie, QLD
Joanne Mclaughlan, QLD
Ubonpan Kansap, Thailand
Korntanat Samattasinwanit, Thailand
Kathy Gillies, VIC
Sarah Shannon, VIC
David Dalton, VIC
Arissara Paksookchai, Thailand
Sutapa Loyawatananan, Thailand
Marisa Saributr, Thailand
Chaniporn Srisakdi, Thailand

DIAGNOSTIC IMAGING

—Abdominal

Tutor: Zoe Lenard

Charlotte Prowse, ACT
William Baird, AUC
Wansida Keastisakthavorn, Thailand
Amelia Yap, Singapore
Peter Rourke, NSW
Levente Palfi, NSW
Wan Shan Choi, NSW
Eamon Grattan-Smith, NSW
Cyndi To, NSW
Sandra Hodgins, NSW
Maddy Rose, QLD
Teagan Rainford, QLD
Rutendo Mukandi, QLD
Carla Allison, QLD
Ka Nam Law, QLD
Chloe Oxford, QLD
Garwai Phan, SA
Jace Koh, SA
Alexandra Carey, SA
Rebecca Kelly, TAS
Notthasuang Sutthipattananggul, Thailand
Jenny Andrew, VIC
Anneliese McKinley, VIC
Vivian Chan, VIC
Madeline Robinson, VIC
Soon Siok Michelle Low, WA

—Musculoskeletal

Tutor: Xander Huizing

Marcin Wolski, Poland
Lucy Kirton, NSW
Peter Rourke, NSW
Levente Palfi, NSW
Elizabeth Parsons, NSW
Ingrid Trommelmans, NSW
Natasha Pesce, NSW
Tanja Elisabeth Kleideitert, NSW
Maddy Rose, QLD
Teagan Rainford, QLD
Rutendo Mukandi, QLD
Jenny Ji, QLD
Carla Allison, QLD
Rava Tsing, QLD
Brooke Burnett, QLD
Ross Evans, QLD
Olivia Dan, QLD
Miguelita Prinsloo, SA
Rebecca Kelly, TAS
Notthasuang Sutthipattananggul, Thailand
Anneliese McKinley, VIC
Nicole Hawken, VIC
Meg Redenbach, VIC
Alison Simmons, VIC
Soon Siok Michelle Low, WA

—Thoracic

Tutor: Belinda Hopper

Lin-En Chen, New Zealand
Scott Doyle, NSW
Leonie Kwok, NSW
Alison Logan, NSW
Wan Shan Choi, NSW
Levente Palfi, NSW
Peter Rourke, NSW
John Coles, NSW
May Lyn Lim, Malaysia
Maddy Rose, QLD
Teagan Rainford, QLD
Karishma Dahia, QLD
Amy Russell, QLD
Chloe Oxford, QLD
Ka Nam Law, QLD
Rutendo Mukandi, QLD
Carla Allison, QLD
Ng Ching Ching Sheryl, Singapore
Jen-Chao Ho, Taiwan
Notthasuang Sutthipattananggul, Thailand
Atijit kongchun, Thailand
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Jenny Andrew, VIC
Lynda Bonning, VIC
Justin Burns, VIC
Anneliese McKinley, VIC
John Houghton, WA
Vishant Prembaj, WA
Kristy Denmead, WA
Soon Siok Michelle Low, WA

Feline Medicine

Tutors: Carolyn O'Brien, Jessica Quimby, Katherine Briscoe, Katie McCallum, Lara Boland, Myles McKenna, Rachel Korman, Sarah Spencer, Jane Yu, Ashlie Saffire

Ana Quintana, United Kingdom
Louise Hales, ACT
Kar Yee Jade Ng, ACT
Alice Kermond, ACT
David Iñiguez Sadurní, Spain
Malgorzata Ruszczyk, United Kingdom
Laurence Graf von Galen, Germany
Joanna Szymanska, United Kingdom
Natalia Mohr, United Kingdom
Kaarin Vekman , United States
Adrienne Verayo, United States
Valentina Pentimalli, United Kingdom
Sandra Holmström, Finland
Katie Knapp, United Kingdom
Iines Savolainen, Finland
Paola Ferrè, Italy
Maria Teresa Bonomo de Juan, Spain
María Rodríguez Sánchez, Spain
Kellie Holmstrom, United States
Christina Larsen, United States
Verónica Nieto Martín, Spain
Sigrid Rokahr, Germany
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Emma Pilkington, NSW

Nara Zhou, NSW
Peter Mackenzie-Wood, NSW
Erika Gladman, NSW
Fernando Elias, NSW
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Megan Bredhauer, QLD
Katja Storm, QLD
Winniesa Pi Chit, Malaysia
Mafalda Ribeiro, Portugal
Margarita Chankova, Bulgaria
Shin hui Chiu, Taiwan
Marta Del Arca Jabal , Spain
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Seyfan Lederman, TAS
Sara Thomas, United States
Oona Miuku, Finland
Elisa Nurmela, Finland
Megan Tucker, VIC
Pamela Oppenlander, VIC
Noam Lang, VIC
Stewart Greedy, VIC
Yvette Berkeley, VIC
Terri Wilks, VIC
Sam Balson, VIC
Clare Thomson, VIC
Charles Banks, VIC
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Emma Dunne, Ireland
Vanessa Marin Cucala, Spain
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Catherine Rae, United Kingdom
Dalya Livy, United Kingdom
Noelia Sorribes,
Katrin Rohde, Germany
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Viphavee Trisaksri, Thailand
Catherine Leighton , United Kingdom
Pemika Vipabusabakorn, Thailand
Yehuda Aji, Indonesia
Maria Gonzalez Ruiz , United Kingdom
Kirsten Davies, United Kingdom
Kousuke Nogi, Japan
Violet Azzopardi, Malta
Tsz Wing Jessica Wong, Hong Kong
Tsz Wa Li, Hong Kong
Maria Macarena Sanchez Martel, United Kingdom
Adriana Wawrzyniak, Poland

Internal Medicine: A Problem Solving Approach

Tutors: Jill Maddison, Sue Bennett

Matthew Ng, Hong Kong
Aileen Wong, NSW
Kaytlyn Playford, NSW
Rachael Long, NSW
Stephanie Cochrane, NSW
Monika Wilton, NSW
Yvonne Goh, NSW
Timothy Chan, NSW
James Torode, NSW
Courtney Turner, NSW
Helen Marks, NSW
Tamsin Paxton, QLD
Samantha Coomes, QLD
Emma Santin, QLD
Ching Wai Law, SA
Leah Dornin, SA
Courtney Swanson, SA
Elizabeth Hendricks, VIC
Qiao Yoke Tan, VIC
Nikita Sanal, VIC
Alison Tudor, VIC
Yan Wing Ho, VIC
Sharon Chi-Wah Tsim, VIC
Joanne Watkins, VIC
Estelle Goldsworthy, VIC
Rachael Harfield, VIC
Eleanor Windle, VIC
Asha Duggan, WA
Jessica Collier, WA
Ching yee elsa Lee, Hong Kong
Sara Oka, Singapore

Internal Medicine: Keys to Understanding

Tutors: Darren Merrett, Steve Holloway, Jen Brown

Lalinthip Manatpreeprem, Thailand
Dhurka Nirthanakumaran, NSW
Olivia Chin, NSW
Photcharaphan Maneetong, Thailand
Pieter Theron, QLD
Felicity Banks, QLD
Leo Immelman, SA
Eleanor Owen, SA
Kristy Junker, SA
Jaimie Le Page, SA
Joy Wee, Singapore
Tanamon Poppinit, Thailand
Krittanut Kanittakul, Thailand
Sheree Zirafi, VIC
Stefanie Smith, VIC
Stephanie Cumming, VIC
Katie Gaut, VIC
Zhihan Xue, VIC
Adrian Coghill, VIC
Jia-wen Lim, WA
Kartiyayini Sinathurai, Malaysia
Michelle Ong, Singapore
Wallis Chan, Hong Kong
Sarmila Rajendran, Malaysia
Run Sakulsirajit, Thailand
Curtis Croucamp,
Kim Man Tang, Hong Kong

Clinical Neurology

Tutors: Laurent Garosi & Simon Platt

Barbora Löfflerová, Czech Republic
Peenicha Subchanakul, Thailand
Nurul Hawadah, Malaysia
Ingrid Trommelmans, NSW
Yi-Hsien (Iris) Lin, NSW
Karin Woldring, NSW
Penny Dobson, NSW
Robin Tsz Chun Kwok, QLD
Kalyarat Phonnongkun, Thailand
Punnasee Ubol, Thailand
Napat Bundao, Thailand
Jennifer Hamm, United States

Ophthalmology

Tutors: Robin Stanley, Marnie Ford

Jonathan Chin, Hong Kong
Neennara Pattarapanont, Thailand
Emma Fouracre, NSW
Simone Brown, NSW
Natalie Hock, NSW
Amy Bird, NSW
Emily Boshammer, QLD
Jalal Thompson, QLD
Lisa Butler, QLD
Nicole Tapp, QLD
Stephanie Ortega, SA
Jae Chung, SA
Riana Fitzpatrick, VIC
Trish Holyoake, VIC
Eliza Rose, VIC
Ebonnie Forster, VIC
Dave Warren, WA
Tanchanok Chountragoon, Thailand

Pasinee Sakulchatwut, Thailand
Kulsiri Prakanrattana, Thailand
Athicha Srisutthakarn, Thailand

Practical Oncology

Tutors: Peter Bennett, Katrina Cheng

Fiona Starr, ACT
Tospol Rujipimolkit, Thailand
Carlos Coto, Costa Rica
Helen Kwan, Hong Kong
Michaela Keen, NSW
Hannah Smith, SA
Madeleine Baird, SA
Ginnie Liew, Singapore
I-Hsuan Teng, Taiwan
Lalita Suwantana, Thailand
Stephanie Middlemast, VIC
Jane Tiernan, VIC

Ruminant Nutrition

Tutor: Paul Cusack

Dione Howard, NSW
Olivia Thiris, NSW
Luke Ingenhoff, NSW
Bronte Clair Sutton, NSW
Robert Hayward, NSW
Jocelyn Todd, NSW
Trinity McNicol, QLD
Elizabeth Erasmus, QLD
Alex Boileau, QLD
Abigail Guthrie, QLD
Jennifer Cook, SA
Molly Kalman, SA
Christina Johnson, TAS
Evelien van der Geest, VIC
Gemma O'Reilly, VIC
John Jardine, VIC
Hayden Morrow, VIC
Michael Doyle, VIC
Katrina Martin, VIC
Peter Nugroho, WA
Michylla Seal, WA

Surgery

Tutors: Chris Tan, Mark Newman, Bronwyn Fullagar, Wendy Archipow

Wai Lun Alan, Hong Kong
Titus Prasetya, Indonesia
Michelle Tabisz, NSW
Tiarna Barclay, NSW
Louisa Poutsma, NSW
Jeremy Peiser-Oliver, NSW
Leo Lee, NSW
Abbie McEwen, NSW
Miguel Pajate, NSW
Flora Wong, NSW
Ashleigh Smith, NSW
Rachel Payne, NT
Pauline Pierson, QLD
Gustav Dippenaar, QLD
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Tsz Ching Cheung, SA
Victoria McRae, SA
Faustina Niap, SA
David Anderson, SA
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Uday Singh Karki, VIC
Sin McGuane, VIC
Elita Frazer, VIC
Yuanliang Liu, VIC
Annie Carty, VIC
Sijun Pan, VIC
Loredana Pregnolato, VIC
Jessica Kenworthy, VIC
Yehua Qiu, VIC
Tamryne McNair, WA
Henry Chung, WA
Fiona Hapelt,
Brian Yiu, Hong Kong
Kate Anderson, New Zealand

Congratulations to DE participants who passed the ANZCVS exams in 2022

Medicine of Cats

Charles Banks
Chloe Cheung
Diana Crispe
Erika Gladman
Rachel Tsang
Jacqueline Victor
Nara Zhou

Medicine of Cats UK

Jacqueline Cole
Carla Cruz
Lucy Fleming
Yvonne Lambach
Raquel Martinez-Vega
Natalia Mohr
Maria Macarena Sanchez Martel
Nina Schiotz
Carolina Vilches Romo
Harriet Wilkinson

Small Animal Dentistry and Oral Surgery

Lana Robertson

Small Animal Medicine

Renea Barrett
Zhan Hong (Terry) Chew
Carmen Chui
Robert French
Simone Hardinge
Tiarni Johnston
Clare Koh
Catherine Rampton
Chi Yan Jenny Shiu
Michael Yazbeck

Small Animal Surgery

Sarah Austin
Samuel Biddle
Marina du Preez
Karmen Fong
Theresa Holm
Charlotte Krisanski
Valerie Martinot
Jennifer Nesci
Jerrold Tan

Veterinary Behaviour

Nicole Chan
Zoe Devine
Naomi Graffin
Nicola Martinson
Carol Mayes
Hannah Sherry
Diana So

Veterinary Practice (Small Animal)

Vivien Tam

Veterinary Radiology (Small Animal)

Alice Birckhead
Bree Cashmore
Kimberly Si Min Lim
Gordon Lye
Jessica Milne
Robert Pertzel
Shelley Xue Ni Wo

B BRAUN
SHARING EXPERTISE

DIRTY WOUNDS? WE HAVE THE SOLUTION!

Wounds can vary widely in how they present, from straight forward through to complex, challenging us at every turn. Wounds may be superficial, gaping full thickness or punctures that go deeper than we can see, but no matter the depth of the wound, if it is not clean it cannot heal.

What is Biofilm?

Biofilm forms when bacteria adhere to surfaces by excreting a thick, glue-like substance known as the Extracellular Polymeric Substance (EPS). This substance forms a protective layer, where the bacteria are no longer free to move (planktonic) but adhere to the wound bed. New bacteria are produced, and the colony grows under the protection of the EPS. Biofilms are often difficult to detect visually, but they delay wound healing due to the protection they provide to the bacteria in the wound bed.[3]

The Solution

Traditional wound cleansing with saline and water is ineffective at removing coatings and debris in many wounds, especially complex biofilms.[4] Prontosan® Irrigation Solution and Prontosan® Wound Gel/Gel X are specifically indicated for the prevention and removal of biofilms. Prontosan® contains two key ingredients: Betaine and Polyhexanide.

Betaine is a gentle surfactant which is able to disturb, penetrate, clean and remove biofilm and wound debris.

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The unique combination of Polyhexanide and Betaine have a double effect on the wound bed to create a wound environment optimal for healing.

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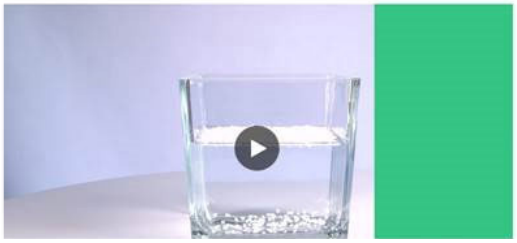
How it works?



There is a plastic granulate on the surface.

ADVERTORIAL

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