THE UNIVERSITY OF SYDNEY
Centre for Veterinary Education
Professional Development Leaders

C&T

CONTROL AND THERAPY SERIES

December 2012 ISSUE 269
Australia's Leading Veterinary Forum

Feature Article
How I manage Tick Paralysis

'How I manage Tick Paralysis' author Rob Webster pictured with 'Kimba'

We want YOUR ticks!

Part 3: Wildlife Flashcard Series - Turtles, Lizards and Snakes

Centre for Veterinary Education
Professional Development Leaders

C&T
CONTROL AND THERAPY SERIES

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COVER IMAGE
Pull your head in boys!
Saw-shelled Turtle courtesy of Mimi Dona

Christmas Closedown
2012 - 2013

CVE's last working day will be Friday 14 December 2012 and we will reopen on Wednesday 2 January 2013.

Here we are at the end of another year and once again we have another great issue of C&T, packed full of useful information which will keep everyone reading and thinking well into the new year.

In this edition, we have a list of those people who have completed CVE Distance Education courses and who were successful in the Australia and New Zealand College of Veterinary Scientists (ANZCVS) Membership and Fellowship exams in 2012. Congratulations to all of those successful candidates, which included for the first time 13 from Europe who sat for the Medicine of Cats membership exams. In Europe there is no equivalent qualification available to study only felines, so the ANZCVS has established an arrangement with the International Society of Feline Medicine where European candidates can sit their written and oral exams in Europe.

Once again we have 2 fine articles contributed by Heather Shortridge, who is becoming one of our most prolific young authors and surely must be an inspiration for those of you who have lots of interesting stories to tell, but have not yet made the time to do so. Why not take a leaf out of Heather's book and put pen to paper, or tap on the keyboard over the holidays, so you too can contribute to a future issue of C&T?

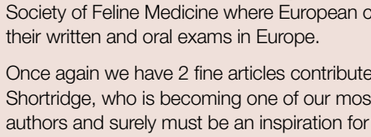
As usual there is a broad range of interesting articles, from horses and goats, to tips and tricks with turtles and reptiles, articles about flea control and chronic cystitis in dogs. There is something for everyone – how many out there know what a plastron is?

Finally, we have a lengthy Perspective section on Tick Paralysis. This follows on from Part 1 of a round table discussion in the last September issue, which in turn followed on from other articles in the C&Ts of September and December 2011. If you have been using our ebook format you will not have any trouble following the simple links to these earlier discussions.

Rob Webster, Kath Briscoe, Fiona Campbell and Rick Atwell have all contributed to Part 2 of this Perspective and we are indebted to them and our earlier contributors to Part 1 for the time and effort that each person has taken to offer their views on such a complex and challenging disease. No doubt there may still be unanswered questions, but I challenge anyone who reads these articles to say they have not been both stimulating and thought provoking.

As this is the last edition for 2012, it is fitting that we thank once again all our contributors, both regular and infrequent, as well as Lis Churchward and Richard Malik, who spend many hours on each issue checking copy and seeking feedback from both the authors and others with a special interest in a particular field.

All of us at the CVE wish each and every one of you a safe and happy time over the Christmas and New Year period and hope that you have a happy, healthy and prosperous 2013.



Hugh White BVSc MVSc MACVSc
DIRECTOR

A love affair with CVE's Distance Education

JANET CRIDLAND (AUGUST 2012)

Since 1981 I have averaged a conference every second year with all but 3 being CVE and its predecessor PGF. The conferences have always had excellent speakers and have been valuable but the DE component of CVE is the most useful and exciting for me.

I did my first DE course in 1991: **Anaesthetics with Jacqueline Grandy**, which began my love affair with distance education. Back then though, the assignments were hand written, contact was by phone and there were no uploads from computers! Since then I have done Cardiac Medicine with **Russell Mitten, Internal Medicine with Jill Maddison, Oncology with Peter Bennett, Surgery with Wing Tip Wong and Glenn Edwards.** As well, I have done a **TimeOnLine Course: Animal Welfare** and a few evening seminars.

My experience of DE has always been fabulous with wonderful course content, exceptional references and support materials. As the years have gone by I have been impressed with the continued relevance and accessibility for working practitioners. DE courses have been tutored by not only leaders in their field but people with excellent teaching ability and understanding of GP vets needs. The embracing of technology has been an enormous aid, but I have to tell you that the access to the wealth of data available via the www does not always make assignments easier – but it does make you learn even more than you thought there was to learn.

Every time I start a DE course I am excited by the prospect of new knowledge and attitudes. I am always particularly pleased when suddenly I understand the whys behind the things we think we know. The flush of new knowledge powers the pursuit of more knowledge.

The supportive information and the fine tuning and hands-on skills that have been provided in every DE course I have done with CVE have provided very special extras that give a fabulous completion to each of the courses. After completing a DE course I found myself re-enthused with an absolute determination to keep current with my new area of extended knowledge.

CVE 2013 SHORT COURSES

With a wide range of conferences, workshops and online courses, you can confidently choose the CVE to provide you with the professional development you seek to become a better practitioner and ensure the continuing success of your practice.

To register your interest or for further information, including prices and programs, visit **www.cve.edu.au/calendar** or email **cve.events@sydney.edu.au**

CONFERENCES & SEMINARS

2013

11-14 Feb	**Small Animal Ophthalmology Conference** Robin Stanley, Chloe Hardman, Kelly Caruso, Cameron Whittaker and Anu O'Reilly	Sydney
17 Mar	**Wound Management & Reconstruction** Arthur House	Launceston
14 Apr	**Hot Topics in Feline Medicine** Vanessa Barrs	Perth
28 Apr	**Looking Down the Microscope** Paul Canfield	Adelaide
5 May	**Diabetes** Linda Fleeman	Melbourne
2 Jun	**Hot Topics in Feline Medicine** Vanessa Barrs	Canberra
24-27 Jun	**Cardiorespiratory Conference** Niek Beijerink, Nick Russell, Mariano Makara and Richard Woolley	Melbourne
13-14 Jul	**Approaches to Avian & Exotics** Bob Doneley and David Vella	Sydney
13 Oct	**Diabetes** Linda Fleeman	Brisbane
27 Oct	**Looking Down the Microscope** Paul Canfield	Port Macq.

TIMEONLINE COURSES

TimeOnline courses are delivered wholly online, giving you the flexibility to study when and where you wish and complete your course at your own pace. For more courses and further information visit: **www.cve.edu.au/timeonline**.

18 Feb - 17 Mar	**Dermatology** Linda Vogelnest
25 Feb - 24 Mar	**Marine Wildlife** David Blyde

WORKSHOPS

The following workshops will be available in 2013.

Basic Echocardiography	**23 March**	Dubbo, NSW
Advanced Echocardiography*	**24 March**	Dubbo, NSW
Hip & Stifle in the Dog	**18 or 19 May**	Perth, WA
Canine Rehabilitation Introductory Workshop	**18-19 May**	Sydney, NSW
Canine Rehabilitation ICU Masterclass*	**20 May**	Sydney, NSW
External Fixators	**19-21 July**	Melbourne, VIC
Basic Echocardiography	**24 August**	Townsville, QLD
Advanced Echocardiography*	**25 August**	Townsville, QLD

* Prior learning will be required to attend this workshop.

Listed dates for all short courses are subject to change. Please refer to the Events page of the CVE website, **www.cve.edu.au**, for the latest updates, full programs, prices, etc.

TimeOnline

Connect – with a professional group with common interests.
Validate – your knowledge and achieve better patient outcomes.
Engage – with current knowledge and know you are offering best practice.

Time – at a time convenient for you
Online – office learning or wherever

7 NEW courses coming in 2013

Visit **www.cve.edu.au/timeonline** OR

Email your enquiries to **cve.timeonline@sydney.edu.au**

Coming in 2013 – TimeOnlinePLUS

Terms and Conditions and Policies of the CVE can be accessed through the CVE website.

Congratulations to our former CVE DE Participants (Australasian & European) who were successful 2012 candidates for Australian College exams.

Fellowship in Veterinary Oncology		
Amy	Lane	05 Medical Oncology
Membership in Animal Reproduction		
Stuart	Mason	04 IMKU
Membership in Avian Health		
Alan	Warner	09 Avian
Ruth	Barrett	13 IMPS
Membership in Medicine of Cats		
Agnieska	Zoltowska	11 Feline Medicine
Audra-Lynne	Turner	09 Feline Medicine
Belinda	Kays	11 Feline Medicine
Caroline	Blundell	10 Feline Medicine
Caroline	Carlton	09 Feline Medicine
		10 IMKU
Clare	Meade	10 Feline Medicine
Elanor	Atkinson	11 Feline Medicine
Emma	Buckley	11 Feline Medicine
Jessica	Nevile	10 Ophthalmology
		11 Feline Medicine
Kay	Weller	09 Feline Medicine
Lisa	Bennett	11 Feline Medicine
Nathalie	Dowgray	10 Feline Medicine
Peter	Trinder	09 Feline Medicine
Rebecca	Francis	11 Feline Medicine
Sally	Coggins	09 Feline Medicine
Teija	Viita-Aho	10 Feline Medicine
Vanessa	Milborn	11 Feline Medicine
Wayne	Mizon	09 Feline Medicine
		07 IMKU
		10 Diagnostic Imaging
		11 Sonology
Membership in Medicine of Horses		
Nicholas	Bamford	09 Equine Medicine
Membership in Small Animal Medicine		
Amanda	Taylor	11 IMPS
Barbara	Jameson	10 IMKU
Isabella	Gartrell	02 Ruminant Nutrition
		10 IMPS
Joanna	Pilton	11 Feline Medicine
Nicole	Cook	09 IMKU
Membership in Small Animal Surgery		
Anthony	Payten	11 Surgery
Christopher	Livingston	11 Surgery
Fiona	Coghill	06 IMKU
Fui	Yap	11 Surgery
Leah	Bonnette	10 Surgery
Winnie	Shih	09 Surgery
Membership in Surgery of Horses		
Anthony	Doherty	05 Equine Surgery
Holly	Lewis	07 Equine Medicine
Membership in Veterinary Behaviour		
Fiona	Warton	10 Behavioural Medicine
Glenn	Tobiansky	03 Emergency Medicine
Heather	Chee	11 Behavioural Medicine
James	Sabin	08 Behavioural Medicine
Katherine	Lindsey	11 Behavioural Medicine
Kim	Kelly	09 Behavioural Medicine
Sophie	Nielsen	11 Behavioural Medicine
Susannah	Wood	09 Behavioural Medicine
Tina	Chen	10 Behavioural Medicine
Trepheena	Hunter	11 Behavioural Medicine
Membership in Veterinary Emergency and Critical Care		
Anna	Macnaughtan	09 Surgery
Helen	Wilson	09 Surgery
		11 Emergency Medicine
Sarah	Purcell	10 Surgery

IMKU = Internal Medicine: Keys to Understanding
IMPS = Internal Medicine: A Problem Solving Approach

We also congratulate all 2012 CVE DE Participants. Due to space constraints, we will publish the names of our 2012 DE graduates in our upcoming March 2013 issue.

Don't Miss This: CVE Ophthalmology Conference, University of Sydney

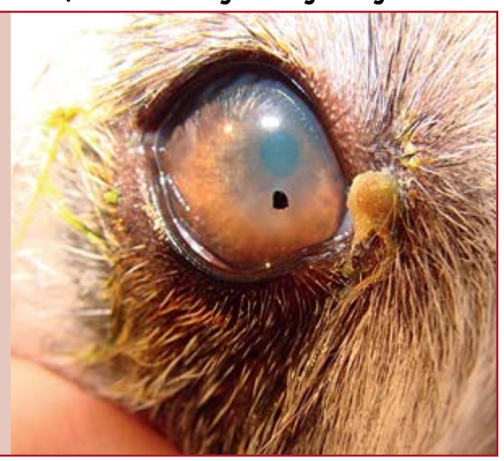
What do you see?

There are ***four*** problems with the eye of this Terrier. A black lesion has appeared in the ventroaxial (central) cornea. This is consistent with a small corneal foreign body (FB). These foreign bodies are often small, and sit on the cornea, almost like a suction cup onto the cornea.

What else? The cornea is slightly hazy – corneal oedema from corneal inflammation. Miosis – small pupil. Corneal disease causes the reflex down the axon to the iris, resulting in miosis (and in some cases hypopyon as well). Ocular discharge, mucoid to mucopurulent.

How would you manage and treat these problems? What other issues need to be considered? Was the mucoid discharge the result of irritation from the FB, or was it from a dry eye? Too often in practice we just reach for the antibiotics, a symptomatic treatment rather than considering what could be underlying cause and or predisposing factors.

Learn more about cases such as these and how to better approach and manage them at the CVE Ophthalmology Conference coming up on 11-14 February 2013 at the University of Sydney.



Thank you to all contributors

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

Winners

Major Prize
Entitling the recipient to one year's free membership of the CVE
- **Robert Webster: How I manage tick paralysis**

CVE Publication Prize Winners
- **Vanessa Milborn:** Sourcing a reasonably priced Doppler blood pressure monitor
- **Angela Phillips:** Difficult breathing in a young Ragdoll cat
- **Kellie Seres and Peter Launders:** Paracetamol toxicity (and foreign body ingestion!) in a kitten

Winner of Best Film Clip
Mimi Dona for clips demonstrating restraint of:
- Turtle
- Eastern Water Dragon
- Blue Tongue Lizard
- Spencers Monitor
- Snake Venomous
- Snake Non-Venomous

Are YOU making the most of the C&T Series?

To view: Mimi's film clips, previously published C&Ts pertinent to the articles in each issue or rollover X-rays or images to enlarge them, you need to read our complementary e-book version.

Look for these symbols

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

Contact **cve.membership@sydney.edu.au** or call Jacqui Kennedy and receive your Login and Password details. Then visit **www.cve.edu.au/candtebook** which allows you access to this current issue in e-book format and the 4 prior issues (Dec 2011, March 2012, June 2012 & Sept 2012).

You don't have to be a CVE Member to contribute to the Series.

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

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

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

Hindlimb amputation of, and anaesthetic adventures with, a Saanen Goat

C&T No. 5265

Heather Shortridge
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E. heather@armidalevet.com.au

'Goatis' a 4-month-old Saanen Buck kid presented at our clinic for hindlimb amputation. Goatis had a fracture through the right metatarsal bone. His owner had been splinting the leg, but the fracture opened up and Goatis was not going well. The owner was going to euthanase Goatis herself; however, she found he was too cute to go through with it!

On presentation, Goatis was 15.6kg, bright and alert, with an infected open fracture of the right hindleg. Goatis was premedicated with ACP/methadone and a 22 gauge catheter placed in the cephalic vein in the left foreleg. Anaesthesia was induced with 3mL of alfaxan IV and a size 7 cuffed tube was placed, with the assistance of a laryngoscope. At this point, Goatis (much to our surprise) sat up and began chewing the tube! Goatis was extubated – fortunately the tube was intact. Goatis was re-anaesthetised with alfaxan, re-intubated with a 7.5 tube, and the cuff inflated. To our shock, Goatis regurgitated a little, then sat up and began chewing again! Following prompt re-extubation, we noticed that Goatis' fluids were going subcutaneously into his left leg. We removed the catheter from the left forelimb and placed a new catheter in the right cephalic vein. At this point we re-induced Goatis with aquafol, and then maintained him on isoflurane for some time before attempting intubation again. This time, Goatis remained asleep while he was reintubated and attached to gas. Surgery itself was much more unremarkable than the anaesthetic. A mid-femoral amputation was performed, much as would be carried out in a similarly sized dog, and muscle was sutured over the end of the femur. There was some surprising blood spatter when the femoral artery was nicked, however the vessel was ligated repeatedly with 2-0 visorb and no further bleeding was noted. Keeping Goatis asleep was the main challenge, and for most of the surgery, isoflurane was maintained at 3%. Additionally, Goatis had a shallow fast respiration of around 40 breaths per minute throughout surgery. Goatis was kept on a heat mat during the surgery and received Hartmann's Solution throughout surgery and postoperatively. He was given depocillin peri and post-operatively, and given subcutaneous butorphanol and meloxicam on waking up. Goatis was woken up under a Bair Hugger and woke up well, standing up within an hour of surgery, and he went home the following day on depocillin bid and bute paste sid. On the last update received, Goatis was getting around very well on 3 limbs.

This was an interesting case and highlighted the challenge of anaesthetising more unusual patients!

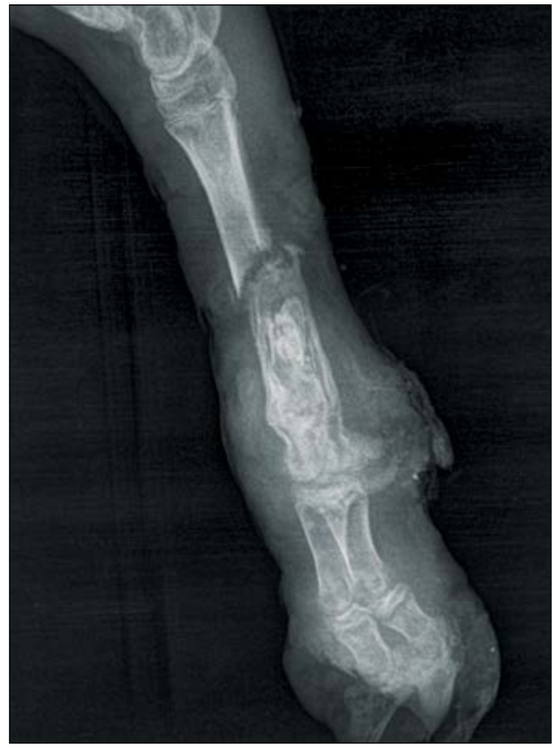

Figure 1. Lateral radiograph demonstrating fracture through the metatarsal bone.

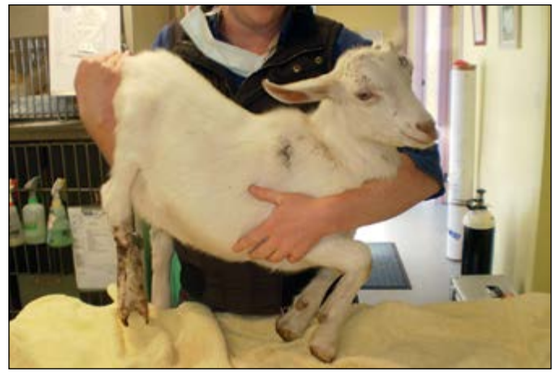

Figure 2. Goatis on the prep table – to some extent we just handled him as though he were a 15kg dog.

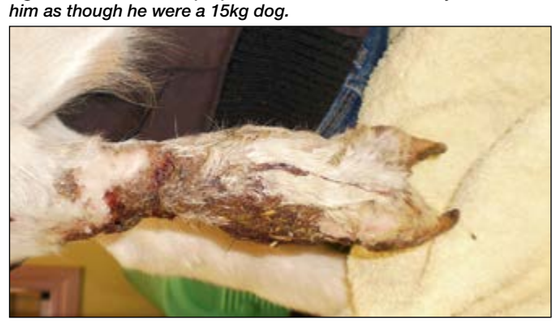

Figure 3. Infected leg with open fracture.

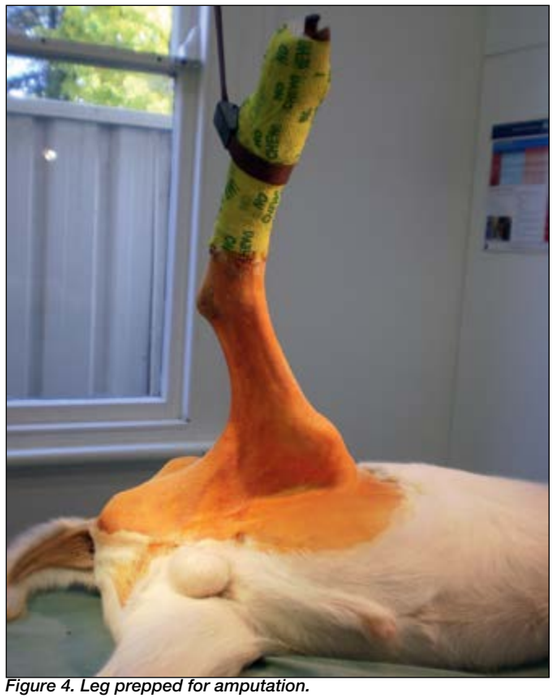

Figure 4. Leg prepped for amputation.

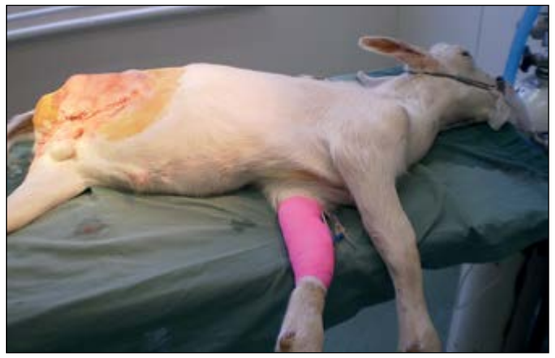

Figure 5. Post-operatively but before extubation.

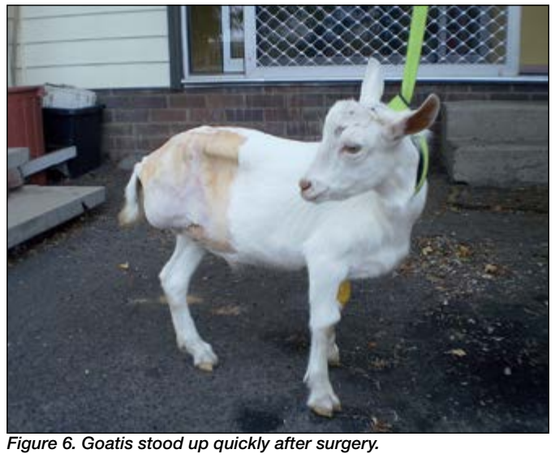

Figure 6. Goatis stood up quickly after surgery.

Figure 7. The patient and author the day after surgery.

CALL FOR MORE C&TS ON LARGE ANIMALS

We need more articles on Large Animals – Horses, Cattle, Sheep, Goats, Pigs, Llamas, Alpacas, Deer etc to ensure the C&T Series remains broad and interesting. So if you see an interesting Large Animal case, please get your digital camera/mobile phone out and send the article to us complete with coloured images and videos where possible. Now that we have the facility to produce the C&T Series in e-book version we want contributors to use it.
A picture tells a thousand words, a video –?

Images courtesy of Scott Reid. (Far left, top & bottom: Karisa Reid and friends, 'Basil' and 'Rosie', 'Rose' & 'Thorn', 'Collie' & 'Sasha' and 'Laura')

Equine splint bone removal under field conditions

C&T No. 5266

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While Equine orthopaedic surgery may bring up images of specialist centres, there are a few procedures which can, if necessary, be performed in the field. Splint bone resection is one such procedure. Hickory, an 8-year-old Stockhorse, initially presented for a non-healing sore swelling of the left foreleg.

Radiographs were taken which revealed that the lateral splint-bone was fractured. (Figure 1)

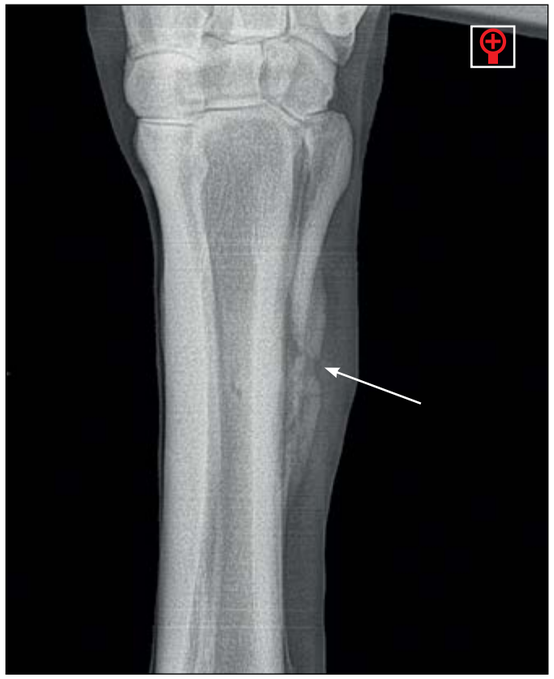

Figure 1. Anteroposterior radiograph of the affected leg. The fractured splint bone is highlighted by an arrow.

The swelling had been present, and the horse sore, for some time. Referral was offered however the owners opted for resection in the field (we are quite far from referral facilities).

The surgery was scheduled for a sunny morning. Hickory was pre-medicated with 1mL butorphanol, 5mL xylazine 100, and 1mL acepromazine given in the left jugular vein. The leg was clipped while Hickory was standing. (Figure 2)

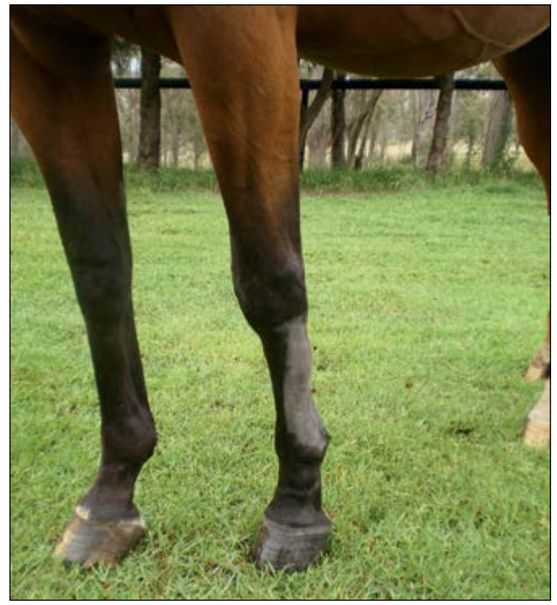

Figure 2. The leg clipped under sedation, prior to surgery.

Anaesthesia was then induced with 10mL ketamine combined with 1mL diazepam. A 14 gauge catheter was then placed in the jugular, so Hickory could be maintained under anaesthetic using 'triple drip' (guaifenisin + xylazine + ketamine), given concurrently with several litres of Hartmann's solution. The 'field drip stand' is always a help in these situations. (Figure 3)

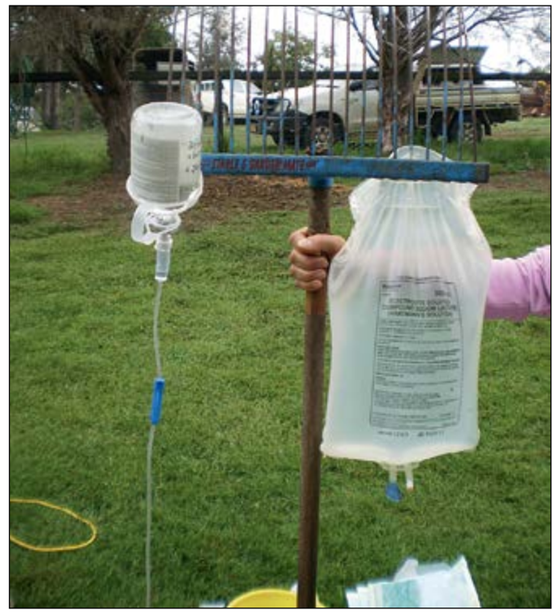

Figure 3. 'Field drip stand'!

Hickory's leg was elevated and the surgical site scrubbed and draped, to create a sterile surgical field. (Figure 4) ▶

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

Figure 4. Prepped and ready for surgery.

A vertical incision was made over the fracture site and the incision deepened to the level of the periosteum. (Figure 5)

Figure 5. Incision over fracture site.

An osteotome and hammer was used to sever the interosseous ligament. The osteotome was also used to create an amputation site 2cm proximal to the affected area.

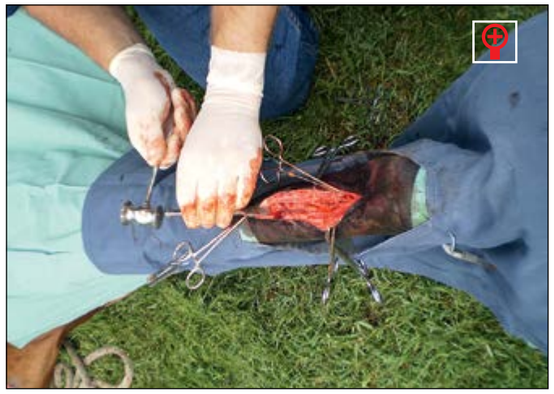

Figure 6. Using the hammer and osteotome.

In some cases the splint bone can be removed in one piece, however in this case, there were numerous fragments to be removed. (Figure 7)

Figure 7. Splint bone removed as several fragments.

All fragments,and abnormal tissue was removed, and the surgical site was lavaged with one litre of normal saline.The surgical site was closed and the limb bandaged. (Figure 8)

Figure 8. Limb bandaged after surgery.

Hickory was treated perioperatively and postoperatively with gentamicin and penicillin. Hickory was also given tetanus toxoid and anti-toxin, and intravenous flunixin before waking her up. Hickory woke up fairly ataxic, which is not uncommon with guaifenisin anaesthesia. The rest of Hickory's recovery has been unremarkable, so this has been a very satisfying case.

WINNER OF BEST FILM CLIP

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

Part 3: Wildlife Flashcard Series

Turtles, Lizards & Snakes

C&T No. 5267

This series is the result of collaboration between Mimi Dona & Dr Michael Pyne of Currumbin Wildlife Sanctuary Veterinary Hospital.

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

‹e-book› Film clip courtesy of Lincoln Williams
www.fotomedia.com.au

FRESHWATER TURTLES

Video demonstrating Turtle restraint

Be Aware:-

- Turtles head back to the same breeding grounds therefore must be released in the nearest body of water to their found location. If releasing before cold weather is expected, provide heat and do not feed for at least 2 weeks prior to release. This will allow hibernation to commence with an emptied digestive track.
- Always radiograph during breeding season to confirm if gravid.
- Turtles can be misidentified as marine turtles; the difference being marine turtles have flippers.
- Care should be taken when providing warmth as they are ectothermic and their body temperature is influenced by their surroundings.
- Lack of sunlight and the ability to bask may result in shell and bone deformities.
- Juvenile turtles receive no parental care and should be housed separate to their parents.

Handling

- They can bite and scratch; a towel or thick gardening gloves can assist when handling larger or more active species. They will settle down once restrained.
- A fall can lead to a damaged shell; always restrain over a table and have a firm grasp when handling the more active patient.
- When capturing grip both hands on the edge of the carapace (top shell) behind the front legs and in front of the hind legs. They can still reach you with their hind claws in this position.
- Hold smaller species at the rear of their shell with your thumb on top. For larger species place your spare hand flat underneath on its plastron (lower shell) and rest your elbow on the table.
- If they have a hind shell fracture or injury, hold using a towel at the back of the plastron and at the top of the carapace (upper shell).

Figure 1. A Brisbane River Turtle being restrained with a towel due to a shell fracture.

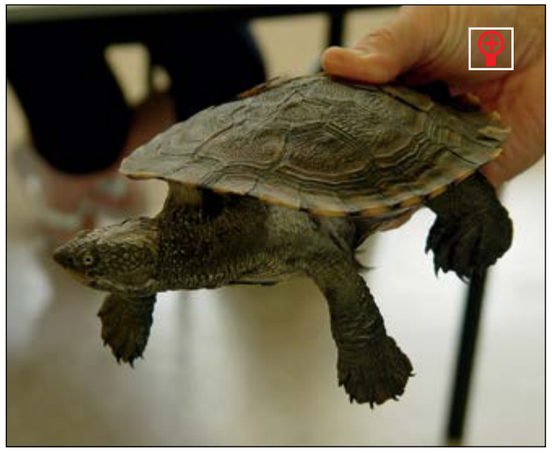

Figure 2. A Saw-shelled Turtle being handled at the rear of its shell for an assessment

Housing the sick or injured turtle

- Preferred enclosure temperature is approximately 26˚C.
- Ideally, heat should be provided in a controlled environment (vivarium/tank) warming them to their preferred body temperature (PBT). This can be provided with a temperature controlled room, aquarium heater and/or basking light. Alternatively, for a short term in hospital, a heat pad can be placed under a box or plastic tub; only sit under one half of the enclosure.
- For temporary housing you do not have to keep turtles in water; wet towels or daily soaking are sufficient. House in a smooth sided ventilated tub with newspaper or towels to line the bottom and a secure ventilated lid. ▶

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- If dehydrated or debilitated keep housed on wet towels and cover the enclosure to create a hide.
- Long term they will require UV light. The best alternative to sunlight is artificial ultraviolet lighting.

Figure 3. A temporary set up with warmth being provided by a heat pad.

Figure 4. Turtles can be dry docked.

Emergency diet

- Australian turtles are basically carnivores, although some short neck varieties will eat some vegetable matter.
- They will only eat and defecate when in water; the water should be warmed to their preferred temperature approximately 23℃ to 30℃.
- Turtles can go weeks without food if hydrated or being given fluids/baths and not emaciated.
- They can be offered a mixture of celery tops, dark lettuce, spinach, aquatic plants, live insects, fish, shrimp, worms, yabbies and mince.
- Tube feeding with the use of Hill's a/d™ may be valuable in some cases.
- Calcium is important in turtles' diets; especially the long-term patients with shell damage.
- Pet stores stock frozen turtle food that provides a balanced meal and easy alternative in hospital.

Assessment under anaesthetic

- Intermittent positive pressure ventilation (IPPV) is required throughout the procedure due to the inability of turtles, once anaethetised, to move their limbs, which assists in inducing inspiration.
- An important anaesthetic monitoring aid is a Doppler blood flow monitor.
- Turtles rarely ever regurgitate and fasting is not required.
- Warming the patient up prior to anaesthesia assists in speeding up the onset of anaesthesia and recovery.

Gaseous

Using a gaseous anaesthetic via a facemask or chamber in turtles is generally not successful. Intubation can instead be performed under manual restraint if unable to give an intravenous anaesthetic.

Once intubated maintain on Isoflurane at 1.5 – 2% with an oxygen flow rate of 1 L/min.

Injectable

Alfaxan CD RTU 6 – 9 mg/kg.

Intravenous anaesthesia induction with isoflourane maintenance is the preferred anaesthetic method.

If required it can be given I/M at 10-15mg/kg; the disadvantages are the onset of anaesthesia and recovery time is a lot slower.

Intubation

Uncuffed endotracheal tube or catheter tip; some species are too small for intubation.

Larynx is very cranial in the mouth and difficult to see until it opens during a breath. Use long tipped cotton buds to assist in opening the mouth and exposing the glottis. Insert the endotracheal tube with the aid of an anaesthetic spray and tie in with micropore tape.

Recovery

Reptiles are slow to recover (15 – 90 minutes). Ensure heat is given throughout the procedure to aid in a faster recovery. Recovery will also be aided by swapping the oxygen to an ambi bag that blows room temperature air.

Figure 5. Intermittent positive pressure ventilation (IPPV) is required throughout the procedure.

Figure 6. A Bair Hugger providing warmth whilst under anaesthetic.

Fluid Therapy (subcutaneous preferred route)

It is important to remember to warm the fluids being administered. Mix one part Hartmanns and two parts 0.45% sodium chloride and 2.5% glucose. Give 3 % of body weight, no more than 5% of bodyweight in fluids should be given at any one time.

Preferred routes for medications and fluids therapy

- Subcutaneous – administered in loose skin just cranial to hind legs.
- Oral – for small doses give via a syringe or stomach tube for larger volumes.
- Intravenous – ventral tail vein.
- Intracoelemically – the right lower quadrant of the abdomen.

Euthanasia methods

Injection of sodium pentobarbitone can be administered either by intravenous or intracoelomic routes. Confirm with a Doppler blood flow monitor.

- If administering by intracardiac or intracoelomic, the turtle must be anaesthetised first.

LIZARDS

Video demonstrating Eastern Water Dragon restraint

Video demonstrating Blue Tongue Lizard restraint

Video demonstrating Spencers Monitor restraint

Beware

- Skinks/legless lizards and geckos have the ability to drop their tail in self-defence; avoid handling any lizard by their tail if you are unable to identify them.
- Legless lizards can be misidentified; all lizards have external ear openings unlike snakes.
- Lizards are ectothermic; body temperature is influenced by their surroundings. Care should be taken when providing warmth, which is required when rehabilitating sick or injured lizards.
- Lizards are capable of defending themselves using either: their mouth/teeth, claws, tail or a combination of these.
- Some lizards are arboreal and will easily escape from their enclosure if not covered.
- Juvenile lizards receive no parental care.

Handling

- Goannas and lace monitors are best restrained with large thick welding or gardening gloves. Hold away from the body by the neck and tail, and include the hind legs (when possible). Secure the end of the tail between your body and the examination table, or your legs. These are potentially dangerous and will require an experienced handler.
- Dragons are best restrained with a towel. Watch for the claws and hold the head and body just before the hind legs or restrain the legs holding them back with the tail.
- Skinks, legless lizards and geckos can lose their tails; always handle above the tail region. ▶

Figure 7. A Lace Monitor being restrained.

Figure 8. An eastern water dragon being restrained with a towel to avoid its spikes.

Figure 9. An Eastern Blue-Tongue Lizard being manually restrained.

Authors' views are not necessarily those of the CVE

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Housing the sick or injured lizard

- Preferred enclosure temperature = 28˚C to 32˚C
- Ideally heat should be provided in a controlled environment (vivarium) warming them to their preferred body temperature (PBT). This can be set and maintained with a temperature probe that connects to a basking light, heating up only one half of the enclosure so they can cool down if required. Alternatively, for a short term in hospital, a heat pad can be placed under a box or plastic tub; only sit under one half of the enclosure.
- For temporary housing use a smooth sided tub with newspaper or towels to line the bottom, a hide (box, newspaper, leaves, bark), a bowl of water for soaking in and a ventilated enclosed lid for arboreal species.
- Long term they will require UV light; the best alternative to sunlight is artificial ultraviolet lighting.
- Dragons require higher humidity levels; this can be achieved by spraying the enclosure frequently using a spray bottle.

Figure 10. A blue tongue lizard enclosure.

Figure 11. Hides have been temporarily provided with paper towels, leaves and newspaper.

Emergency diet

- Australian lizards diets are varied: Skinks = omnivorous, Dragons = insectivorous/omnivorous, Monitors = carnivorous and Geckos/Flat footed lizards = insectivorous.
- Only offer food once rehydrated and maintained at a warm temperature.
- Adult lizards can go several days without food if hydrated, being given fluids and not emaciated.
- Lizards can be offered a mixture of fruit, live insects and dog kibble (soaked) as a temporary diet.
- If not eating, force feed or give Hill's a/d™ in a syringe; but first try seeing if they will lap it from a bowl. This can also be used for feeding via stomach or esophagostomy tubes.

Assessment under anaesthetic

- Intermittent positive pressure ventilation (IPPV) is required throughout the procedure.
- An important anaesthetic monitoring aid is a Doppler blood flow monitor.
- Warming the patient up prior to and during anaesthesia assists in speeding up the onset of anaesthesia and recovery.
- Lizards rarely ever regurgitate and fasting is not required.

Figure 12. Anaesthetic induction box.

Figure 13. Tubes are being used to help prevent perforation of the endotracheal tube.

Gaseous
Intubation can be performed under manual restraint if unable to give an intravenous anaesthetic. Alternatively place in an anaesthetic box at 5 % induction. This can take some time so a chamber is preferred over a facemask.

Once intubated maintain on Isoflurane at 1.5 – 2% with an oxygen flow rate of 1 L/min.

Injectable
Alfaxan CD RTU 6 – 9 mg/kg – (I/V).

If required can be given I/M at 9-15mg/kg; the disadvantages are the onset of anaesthesia and recovery time is a lot slower.

Intubation
Uncuffed endotracheal tube or catheter tip; some species are too small for intubation.

Use long tipped cotton buds, or for larger species shoelaces to assist in opening the mouth, taking care not to damage the teeth. Insert the endotracheal tube with the aid of an anaesthetic spray and tie in with micropore tape, or shoelaces in larger species. Keep the long cotton tips in to help prevent perforation of the endotracheal tube.

Recovery
Reptiles are slow to recover (15 – 90 minutes). Ensure heat is given throughout the procedure to aid in a faster recovery. Recovery will also be aided by swapping the oxygen to an ambi bag that blows room temperature air.

Fluid Therapy (subcutaneous preferred route)

It is important to remember to warm the fluids being administered. Mix 1 part Hartmann's Solution and 2 parts 0.45% sodium chloride and 2.5% glucose. Give 3% of body weight; no more than 5% of bodyweight in fluids should be given at any one time.

Preferred routes

- Subcutaneous – administered in loose skin auxiliary and inguinal regions.
- Oral – given via a syringe or stomach tube for larger volumes.
- Intravenous – ventral coccygeal vein.
- Intracoelomic – paramedian, midventral site.

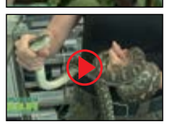
Euthanasia methods

Injection of sodium pentobarbitone can be administered either by intravenous, intracardiac or intracoelomic routes. Confirm with a Doppler blood flow monitor.

- If administering by intracardiac or intracoelomic, the lizard must be anaesthetised first.

SNAKES

Video demonstrating Snake Venomous restraint



Video demonstrating Snake Non-Venomous restraint

Be aware:-

- All snakes admitted should be regarded as potentially dangerous until its true identification is determined. This can be achieved by consulting an expert.
- Sea snakes are venomous and can be identified by their flattened, oar-like tail.
- Care should be taken when providing warmth as they are ectothermic and their body temperature is influenced by their surroundings.
- Snakes seek heat; never leave exposed heat sources in the enclosure as this can cause serious burns.
- Snakes are escape artists; ensure containers are sealed well or leave snakes tied in a pillow case/calico bag.
- Juvenile snakes receive no parental care and should be housed separate to their parents.

If an unidentified species has been presented in a pillowcase or tub then place in a plastic container, label and call your local snake handler or care group for advice.

Figure 14. Consult an expert to handle venomous or unidentified snakes.

Figure 15. Snakes can escape - ensure containers are sealed well and also leave tied in a pillow case/calico bag.

Handling

- Be aware they bite and have backward facing teeth; if you are bitten avoid pulling the snake off. To remove, squeeze together the corners of the snake's mouth or place some liquid soap in its mouth. If still attached, push the head forward to release – not backwards – to avoid further damage to yourself and the snake.
- Minimise handling if a snake has just eaten to avoid regurgitation.
- Minimise handling if a snake is opaque (cloudy eyes visible prior to shedding), as they will have reduced sight making them more likely to strike.
- For safety reasons and to support the snake's body weight, 2 people should handle any snake over 1.5 metres long.
- Handling techniques vary depending on the size and demeanour of a snake. Large, passive, non-venomous snakes can be held loosely around the neck, with the body coiling around your arm or waist to help support its body weight. Alternatively, for a firmer grip you can place your fingers on top if its head and your thumb and little finger underneath for grip.
- Venomous snakes must be held by trained specialists. The technique used is a pistol grip, with the thumb on top of the head and the fingers curled under the chin. ▶

Figure 16. Venomous restraint method.

Figure 17. A carpet python restrained for assessment.

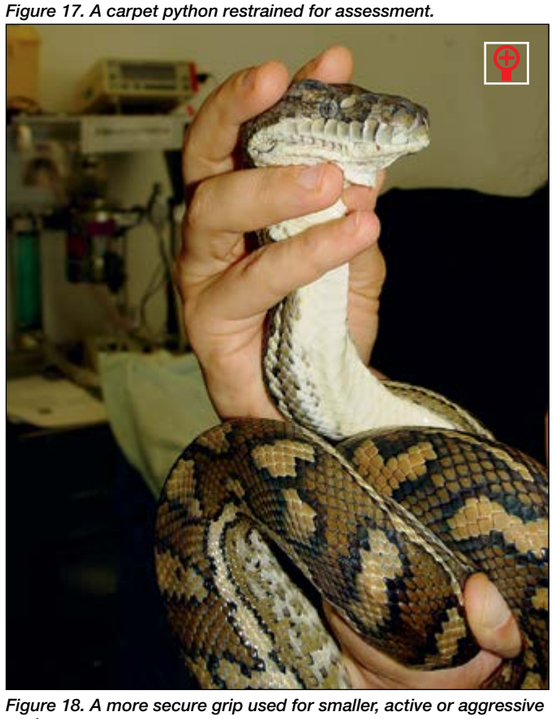


Figure 18. A more secure grip used for smaller, active or aggressive snakes.

Housing the sick or injured snake

- Preferred enclosure temperature is approximately 28˚C to 32˚C.
- For temporary housing, use a smooth sided ventilated tub with newspaper or towels to line the bottom, a hide box (empty syringe box works well), furniture to climb on and use for shedding, a bowl of water for soaking in and a well-sealed ventilated lid. To avoid stress, always cover the box.
- Ideally, heat should be provided in a controlled environment (vivarium) warming them to their *preferred body temperature* (PBT). This can be set and maintained with a temperature probe that connects to a basking light, heating up only one half of the enclosure so they can cool down if required. Alternatively, for a short term in hospital, a heat pad can be placed under a box or plastic tub; only sit under one half of the enclosure. Snakes seek heat; never leave exposed heat sources in the enclosure as this can cause serious burns.

Figure 19. A temporary set up using a heat pad to warm one half of the enclosure, and an empty glove box as a hide.

Emergency diet

- Australian snakes, with the exception of blind snakes, are carnivorous. Depending on the species their diet can vary and include: mammals, frogs/tadpoles, other reptiles or fish.
- Snakes can go months without eating; if in good body condition it is best not to feed snakes whilst on treatment and being handled regularly due to the risk of regurgitation.
- Always feed defrosted (not cooked) dead mice or rats; never live prey items.
- Feed by using tongs (a heat sensing python may mistake your hand for food!) – presenting it in front of them with movement; this can take up to 20 minutes.
- If in poor body condition and injury does not allow them to feed they can be fed Hill's a/d™ via a stomach or esophagostomy tube.

Assessment under anaesthetic

- Intermittent positive pressure ventilation (IPPV) is required throughout the procedure.
- An important anaesthetic monitoring aid is a Doppler blood flow monitor.
- Warming the patient up prior to and during anaesthesia assists in speeding up the onset of anaesthesia and recovery.
- Snakes should be fasted prior to anaesthesia to prevent regurgitation.

Gaseous

Intubation can be performed under manual restraint if unable to give an intravenous anaesthetic. Alternatively, you can place in an anaesthetic box at 5% induction.

Once intubated maintain on Isoflurane at 1.5 – 2% with an oxygen flow rate of 1 L/min.

Venomous snakes

The experienced handler will aid them to slide into a similar sized modified clear tube; this can then be connected to the anesthetic machine at 5% induction.

Injectable

Alfaxan CD RTU 6 – 9 mg/kg – (I/V).

If required can be given I/M at 9-15mg/kg; the disadvantages are the onset of anaesthesia and recovery time is a lot slower.

Intubation

Uncuffed endotracheal tube or catheter tip; some species are too small for intubation.

The glottis sits rostrally on the floor of the mouth just caudal to the tongue sheath. Use long tipped cotton buds to assist in opening the mouth and exposing the glottis, being careful not to damage teeth. Insert the endotracheal tube with the aid of an anaesthetic spray and tie in with micropore tape; be careful not to damage any scales when removing. Keep the long cotton tips in to help prevent perforation of the endotracheal tube.

Snakes can become very flaccid during surgery; to assist with moving either strap to a long board or tape their head on a clipboard to reduce the chance of the endotracheal tube being pulled out.

Recovery

Reptiles are slow to recover (15 – 90 minutes). Ensure heat is given throughout the procedure to aid in a faster recovery. Recovery will also be aided by swapping the oxygen to an ambi bag that blows room temperature air.

Fluid Therapy (subcutaneous preferred route)

It is important to remember to warm the fluids being administered. Mix 1 part Hartmann's Solution and 2 parts 0.45% sodium chloride and 2.5% glucose. Give 3% of body weight; no more than 5% of bodyweight in fluids should be given at any one time.

Preferred routes

- Subcutaneous – administered in thin loose ventral skin.
- Oral – given via a syringe or stomach tube for larger volumes.
- Intravenous – ventral tail vein below cloaca; count down 10 scales to avoid the hemipenes if a male.

Euthanasia methods

Injection of sodium pentobarbitone can be administered either by intravenous, intracardiac or intracoelomic routes. Confirm with a Doppler blood flow monitor.

- If administering by intracardiac or intracoelomic, the snake must be anaesthetised first.

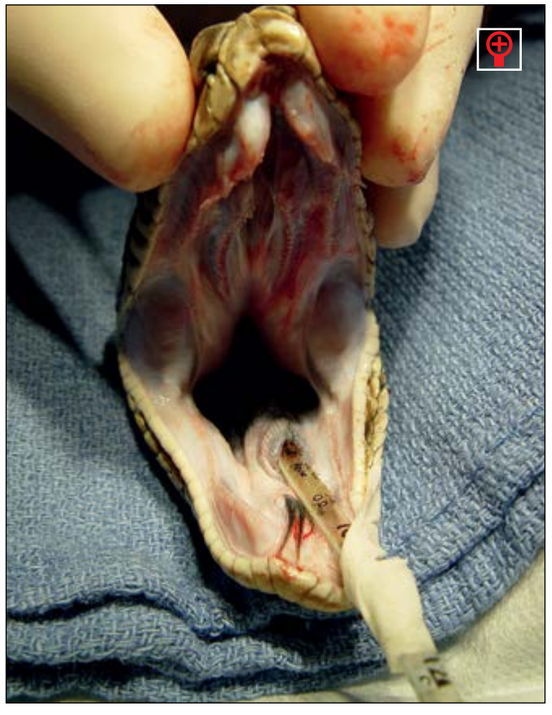


Figure 20. (top left) A snake tube. A venomous snake being anaesthetised.
Figure 21. (bottom left) An ambi bag assisting in providing warm oxygenated air post surgery.
Figure 22. (right) The glottis sits rostrally on the floor of the mouth just caudal to the tongue sheath.

Re-published here courtesy of Vetnostics, North Ryde

ADRENALS: What you won't find in a textbook

C&T No. 5268

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Part 1: Signalment – Hyperadrenocorticism (HyperA)

Part 2: Clinical Signs – Hyperadrenocorticism (HyperA)

Part 3: Routine clinical pathology – Hyperadrenocorticism (HyperA) – (Cushing's Disease)

PART 4A: ADRENAL FUNCTION TESTS

Basal Cortisol

1. The gold standard method for cortisol measurement is radioimmunoassay (RIA). Radioimmunoassay (RIA) results are accurate to 10 nmol/L. However, most laboratories today measure cortisol by chemiluminescent assays as these avoid the radiation hazards of RIA. Low and low normal cortisol concentrations cannot be measured accurately by some chemiluminescent assays (Russell *et al* 2007). The chemiluminescent assay used at Vetnostics was validated against RIA for concentrations <100 nmol/L, using the blood samples from Russell's study (2007) on which another assay was found to be unreliable. There was excellent correlation and no statistically significant difference demonstrated between the Vetnostics assay and RIA (Foster, unpublished data).
2. Prednisolone, cortisone and fludrocortisone all cross-react with cortisol assays. It is difficult to ascertain accurate withdrawal times; however, it is safe to use the following:-

   - prednisolone: 36h
   - cortisone acetate: 12h
   - fludrocortisone: 24h

   It is widely stated that fludrocortisone does not cross-react with the cortisol assay as it is a mineralocorticoid but that is not the case (Tate 2004) with one RIA cortisol kit insert reporting up to 33% cross-reactivity (GammaCoat Cortisol kit). Dexamethasone does not cross react with the cortisol assay. The withdrawal time for long-acting corticosteroid preparations depends on the actual corticosteroid used and is also likely to vary in individual dogs.

Hyperadrenocorticism (hyperA)

1. Basal cortisol concentration has no place in the diagnosis of hyperA unless it happens to be greater than the upper level cut-off for a post-ACTH stimulation test cortisol.

Hypoadrenocorticism (hypoA)

1. Basal cortisol can be helpful as a rapid check for the possibility of hypoA in a collapsed/ill dog although the gold-standard for diagnosis of hypoA is still an ACTH stimulation test. Only 1 study has looked at basal cortisol concentrations in suspected Addisonian patients (Lennon et al 2007). The abstract states that a result >2 µg/dL (56 nmol/L) excluded hypoA (provided there were no cross-reacting corticosteroids) and that if results were ≤2 µg/dL, no conclusions could be drawn. However, what wasn't included in the abstract was the information that only 2/110 dogs with non-adrenal illness had a basal cortisol of ≤1 µg/dL (28 nmol/L), giving basal cortisol concentration a specificity of 98.2% in ill dogs (specificity of ≤2 µg/dL was only 78.2%). Given that the assay used in the study had 1 µg/dL (28 nmol/L) as its lower level of measurement accuracy, we have no information as to whether the 2 'false positives' were 27 nmol/L or <10 nmol/L (the usual cut-off for RIA). **A cortisol concentration of <10 nmol/L is highly likely to be indicative of hypoA in a collapsed/critically ill dog.**
2. Relative adrenal insufficiency is a syndrome in which there is insufficient secretion of cortisol in relation to an increased demand during periods of stress, particularly in critical illnesses such as sepsis and septic shock. It is defined as a transiently inadequate response to exogenous ACTH. Differentiating relative adrenal insufficiency from true glucocorticoid deficiency would not be possible using basal cortisol. It may even be difficult with an ACTH stimulation test and sometimes serial testing is required.
3. ACTH stimulation test results from cases of iatrogenic hyperA can mimic those from spontaneous hypoA cases. Topical, aural and ophthalmic preparations can all have systemic effects and result in iatrogenic hyperA with failure to stimulate in response to ACTH administration. Prednefrin® Forte eye drops, for example, are not infrequently associated with iatrogenic hyperA. Even drugs such as budesonide, which theoretically act locally with minimal systemic effects, can significantly affect ACTH stimulation tests and Vetnostics recently noted ACTH stimulation test results of <10 nmol/L pre and post-stimulation in a dog being treated with a standard dose of budesonide. The ACTH stimulation test returned to normal after withdrawal of the budesonide.

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WINNER

Sourcing a reasonably priced Doppler blood pressure monitor

From the DE Feline list serve

(Readers are advised that this correspondence occurred in July 2011 so prices may not be current although the tips certainly are.)

Q. Vanessa Milborn

Has anyone got any good tips on where to buy a good but well priced doppler BP monitor in Australia?

A. Amanda Lugsdin

Given the current exchange rate you are far better off investigating eBay and PARKS MEDICAL in the US direct. The model you would be looking for is a Parks 811-B. Your eBay search needs to be **.com NOT .com.au** and the unit will most likely come with a pencil probe (which is not what you want).

That's where Parks Medical comes in handy:
www.parksmed.com/products/?page=3.php

An infant flat probe is best for vet use and the size in MHz will depend on the Doppler unit (8.0-9.7 MHz). Probes are about US$120 + postage which generally starts at approximately US$30. You then need to source cuffs (again check eBay or your regular suppliers) and a sphygmomanometer.

ERKA equipment is available in Australia from various equipment suppliers and retails for around $1,900.

It was a bit of effort but I have sourced everything for just under AU$500 including postage which I think is a bargain. I purchased the parks Doppler 811B on eBay recently for US$160 + postage. Conversely, you could order everything through Parks Medical and you would be looking at approximately US$900 + postage and also have a guarantee of good service and working condition.

Don't under-estimate eBay though! You can get lucky if you do your research and ask the sellers appropriate questions, but as always 'buyer beware'.

The only other thing you might need is a power adaptor converter from US to AUS.

Feedback from Sarah Caney – DE Feline Tutor

Very impressive work and very helpful! I can't think that there would be any other way of getting a BP kit for less than AU$500 – brilliant work!

In the UK hospitals are another source worth checking:
http://hospitalmart.org/index.asp

Update from Vanessa Milborn

After researching the available products, we ended up purchasing a VetDop2 Doppler system including all accessories, 6 cuffs and training DVD for US$1,039 plus freight. At the time the Australian dollar was strong which worked in our favour. I have been really happy with the unit and can take BPs stress free in the consult room in most cats.

The supplier was V-Med Technology **www.vmedtechnology.com**

Comment courtesy of

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Measuring blood pressure is a VITAL part of feline medicine – hypertension is common in association with renal disease and uncontrolled hyperthyroidism. Hypertensive retinopathy is the most common cause of blindness in our practice. Every cat that has renal disease needs to have its blood pressure measured. With prices as low as they are now for blood pressure measuring equipment there is no excuse to not have this facility available. I love eBay and it can be a wonderful source of equipment – but you do need to know the details of what you are after; sometimes it is better to pay the extra to know you are getting what you need to have. Also consider the value of your time that you spend searching through heaps of listings. I have only ever used the Parks 811 units to measure blood pressure so I can't comment on other methodologies.

With a little practice anyone can get blood pressure readings from most cats. It is rare that they are too fractious to allow a good reading. The Parks units last a really long time if you look after them – just be careful to treat the probes gently, especially where the wires leave the sensor part that goes on the cat. Don't bend the wires at that location any more than you have to as almost every probe that I have had fail has done so as the wires have broken here. Make sure the wires aren't rolled tightly when they are being packed up and store the entire unit/probe/sphygmomanometer in a plastic container to reduce the chance of accidental damage to the probe (e.g. crushed in a closing draw).

WINNER

Difficult breathing in a young Ragdoll cat

C&T No. 5269

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History

'Chloe' was a 1½-year-old Ragdoll cat that was presented to our clinic mid morning in a collapsed state. The owner had been away for the weekend and a friend had been coming in to feed the cat. The cat was a totally indoor cat whose most recent history of illness was mild 'cat flu' 2 months earlier.

The owner noted that for the last couple of days prior to going away the cat was more 'sooky' than usual, but otherwise seemed fine.

She didn't eat the night before when the owner got home. Her normal diet is one of the premium cat food brands. She had no known access to any toxins within the house. Immediately prior to presentation she was reluctant to move, 'breathing up' and urinated where she sat.

Physical Findings

On presentation Chloe was weak, unable to stand and soaked in urine. Her body condition score was 5/9 on the Purina scale.

She was obvious tachypneic, with short shallow breaths. Heart sounds were audible bilaterally but markedly reduced and she had crackles in all lung fields. She was extremely pale, almost white. Her heart rate was >200 and her respiratory rate was >60. Abdominal palpation was unremarkable and her peripheral lymph nodes palpated normally.

A nasal 5 French feeding tube was passed into the nasopharynx and she was supplemented with nasal oxygen 100mL/kg/min. Intravenous access was established and she was started on maintenance intravenous Hartman's, 10mL/hr.

Investigation

Full bloods were collected for hematology, serum biochemistry and retroviral tests. In house PCV was also run pending bloods results to determine whether the pallor was a perfusion deficit i.e. poor cardiac output, anemia or both.

The PCV was 0.46L/L indicating perfusion deficit rather than anaemia.

Chloe was then taken straight to radiology. She was radiographed without sedation.

On the initial radiographs, (11.16am), there is marked opacity across all the lung fields. The outlines of the cardiac silhouette are not clearly visible. Initially there appeared to be a solid opacity dorsal to the tracheal bifurcation and one immediately ventral to the cardiac base. These opacities seemed more solid that the rest of the lung fields. Alveolar opacity suggestive of pulmonary oedema was quite generalised and there was some leafing of the dorsocaudal lung lobes. A fluid line appeared to run up the caudal cardiac apex. The liver extended well beyond the costal margins.

At this point pulmonary oedema and some pleural effusion seemed most likely but I was also concerned about the 2 more solid opacities. My main differentials were infiltrative neoplastic disease, severe cardiac disease or, lower down on my list, an infectious cause.

Treatment

Frusemide was given intravenously (2mg/kg IV), nasal oxygen was maintained as was maintenance Hartmann's Solution. Pleurocentesis was preformed but only 30mL of clear transudate was recovered.

Over the next few hours Chloe's color started to improve, her respiration rate dropped to 44 and the depth of her respiration improved. She was brighter and able to move in and out of the litter tray and around the cage.

Chloe was re X-rayed 6 hours after the initial X-rays (Figure 2). Pulmonary oedema was still present but markedly reduced. Most of the leafing had resolved and it was now apparent that the 2 'solid opacities' were resolving. The cranial cardiac border was still not clearly defined.

Frusemide (2mg/kg) was repeated and nasal oxygen and intravenous fluids were maintained. The liver was not as caudally displaced beyond the costal arch. Chloe was now pink. She was still dyspneic but significantly improved.

Bloods were back from Idexx Laboratories. She was FIV and FeLV negative. Hematology was within normal limits. Red cell and white cell morphology was normal. On biochemistry glucose was elevated 16.0mmol/l (3,2-7.5). Despite being more than 2 x normal range it was suspected this was only a stress response. Interestingly creatinine kinase (CK) was high 1243 which I suspected of being perfusion related.

Chloe was maintained on the pre-mentioned supportive care overnight.

The next day 7.30am Chloe was brighter, although still weak. She has pulled out her nasal catheter overnight but was still pink and had a respiration rate of 36. She still had pulmonary crackles bilaterally but less than the previous day. Her heart rate was 204. Given the improvement with frusemide and supportive care underlying cardiac disease was suspected. A lateral X-ray was taken, edema was still present and some of the leafing had returned. A further 40mL of fluid was collected via pleurocentesis.

Echocardiograph

Chloe then had an ultrasound carried out by Dr Graeme Allan of Veterinary Imaging Associates.

ECHO- VIA- The left atrium is larger than the right (right=12mm diam, Left= 16x19mm). No signs of LA thrombi. On the M-mode there is thickening of the intraventricular septum and the posterior wall of the left ventricle, (both 6.7mm thick in diastole). The systolic thickness were within normal range, (IVSs 9.1mm, PWs=8.2mm). The left ventricular contractility (LVSFx) was 40 and 60%, based on LVIDd=9=11mm, LVIDs= 4-6mm. There was no detectable regurgitation across the mitral valve.

Interpretation: Failure of diastolic relaxation, most typical of restrictive cardiomyopathy.

Chloe's color remained pink, her respiration improved but she remained floppy. I was concerned I may have upset her electrolytes with diuretic therapy so I repeated them. I also noted her high CK at admission. Her electrolyte results were: Na 132, Cl 96 and K 3.0mmol/L.

Thus, 20mmol of potassium chloride was added to the 1 litre Hartmann's Solution and Chloe was started on oral potassium supplementation, potassium gluconate (1 tablet bid) and regular force feeding of Hill's a/d® and premium tinned food which she accepted quite readily. Frusemide was maintained, but at 5mg sid.

Over the course of the day Chloe's strength increased and by late afternoon she was moving around her cage quite freely. She was also started on Fortekor® 2.5mg sid.

By the next morning Chloe was eating, moving around her cage and was very bright. Her electrolytes were re-checked, Na 139, Cl 101 and K 3.3. By now the owners extended themselves beyond their limits (we had already covered some of the monitoring costs ourselves). As Chloe was eating and breathing well and the owners were at home with her I felt she would be OK to be discharged with a re-visit in 3 days. She went home on Fortekor® 2.5mg sid, Frudix® 5mg sid and Hypokal 1 bid.

She re-presented 4 days later as arranged and the owner reported she had been doing extremely well at home. She was active and breathing normally according to the owner. Her color was pink and on auscultation her heart sounds were clearly audible. Her lung fields were not perfect but much clearer. Her respiration rate was 36 in consult and heart rate 196.

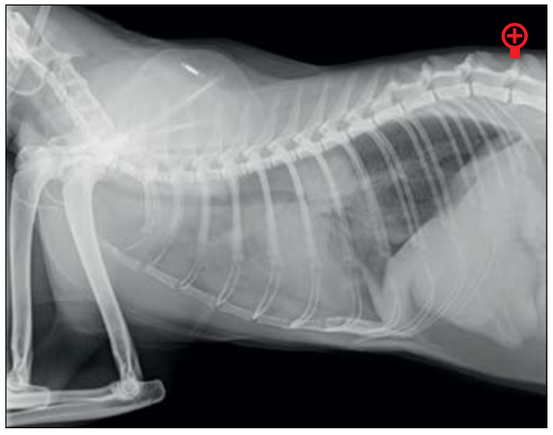
Her electrolytes were repeated. The results were Na 161, Cl 123 and K 8.3, (there was some lysis in the serum sample and whilst I did not ignore the hyperkalemia, it is possible that cell lysis had contributed to this level). Her Hypokal® was ceased and she was sent home to continue on the Frudix® and Fortekor® sid.

On follow up conversation, Chloe appears to be doing well at home, although the owners realise that her life span is likely to be markedly reduced.

Restrictive cardiomyopathy is characterized by impaired diastolic ventricular filling, normal or decreased ventricular diastolic volume. These cases are hard to classify and Ettinger suggests that many can only be called unclassified cardiomyopathy.

Ettinger suggests ACE inhibitors can be considered for, 'blunting neurohormonal activation and reducing ventricular remodeling.' Because of her electrolyte disturbance following aggressive frusemide I have started her on 5mg sid but this may need to be titrated upwards and electrolytes closely monitored.

Post script: It will be 2 years come Jan 13th 2013 and Chloe continues to do well on the current meds.



FLEA TREATMENTS

Frontline Resistance

C&T No. 5270

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To read previous articles on this subject

Fleas and Fipronil C&T No.4582 Michael Strakosch Issue **237**, Dec 2004

Fleas and Fipronil – Reply to C&T No. 4582 C&T No.4583 Merial Australia Pty Ltd, Issue **237**, Dec 2004

Fleas and Fipronil – Reply to C&T No. 4582 (No 1) C&T No.4619 Christine Tindal, Issue **238** April 2005

Fleas and Fipronil – Reply to C&T No. 4582 (No 2) C&T No. 4620 Max Fargher AND Comment from Merial Issue **238** April 2005

Around mid 2009, I had a chat to our drug representative (Rep) about Frontline® (Merial; fipronil) and the fact that it didn't seem to be working against fleas anymore and that we felt that resistance had developed. We usually use the 'Frontline® Spot On' every 2 weeks for tick paralysis prevention (though this is now an off-label recommendation). We had also found that it appeared less effective with the addition of Methoprene.

The Rep had several responses to our comments:-

- Incorrect application of the product was causing the problem, or
- Other animals from neighbours were bringing back the fleas, or
- Clients were seeing the early stages of the fleas before Frontline® had killed them. It takes 24-48 hour to kill a flea and you can then see the earlier stages prior to death. He said that this means that 8-10 fleas on an animal was acceptable and that the product was working.

Trying to explain to clients that the product is still working even though the animal has fleas and that it can take time for fleas to be eradicated takes a lot of time! So, if fleas are a real concern, I push Advantage® (Bayer; Imidacloprid) and use something else for the tick problems.

We still find that the Frontline® spray works but is harder to apply regularly to cats; they smell it and run.

The Rep did explain that if we were concerned about resistance and a client was interested they would send someone to go through their problem and find a reason for the Frontline® not working. He told us that, at that time, there was no evidence that there is resistance to fipronil. ▶

Authors' views are not necessarily those of the CVE
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Pondering flea treatments worldwide...

C&T No. 5271

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Funny how the preferred flea treatments vary across continents... Elanco tell us that their tablet Comfortis® (spinosad), which has been launched in the UK this year, is by far the market leader in the US. In the UK Frontline® (fipronil) spot-on and its generic equivalents are by far the market leader. However, Advocate® (imidacloprid + moxidectin) is making an attempt to catch up, partly on the back of the spread of *Angiostrongylus vasorum* in the UK, as Advocate® is licensed to prevent and treat angiostrongylosis, and Bayer are heavily marketing nationally that everybody needs to worm their dogs monthly with Advocate® now.

Merial recently launched a new spot-on in the UK called Certifect®, which is Frontline® Combo with added amitraz, that they claim will be more effective at killing ticks. Ticks can be a problem in some parts of the UK but less so than in Australia – the climate is cooler and we do not have tick paralysis to worry about here!

There are so many very effective flea products now it is hard for a general practice to stock them all – for years now we have used Frontline® Combo (fipronil + methoprene) as our first-line recommendation and Advocate® as the second line, but we now stock the Comfortis® tablets as well. Comfortis® is undoubtedly very effective at killing fleas, and does so quicker than Frontline, but we decided not to use it as our first line because it is more expensive than Frontline® and it makes about 1-in-20 dogs vomit.

Using Frontline® and then Frontline® Combo over the years, we had not noticed a problem with fleas until the last couple of years when we do seem to have had more clients having trouble with persistent flea infestation despite apparently using Frontline® or Frontline® Combo regularly and applying it correctly. I do not think that this is due to fipronil resistance, for a combination of reasons. First, the number of these problem cases remains a tiny minority of those using these products and, interestingly, seems to be limited to cats (usually multi-cat households) but not dogs, at least in our practice. Second, even Merial's competitors confirm Merial's advice that there is no evidence of fipronil resistance in UK fleas. Third, just a few weeks ago I watched a webinar (admittedly sponsored by Merial!) by 'Dr. Flea,' US flea expert Dr Mike Dryden, in which he indicated that there was no evidence for fipronil resistance in fleas in the US – where there is arguably greater selection pressure for resistance than there is in the UK – and that all of the leading flea products are very effective. Fourth, UK veterinary dermatologists don't seem to have strong feelings about the relative efficacy of the different leading flea products – if they do, they seem to keep it to themselves! ***(Editor's Note:** Watch out for a Comment from Australian dermatologists in the upcoming March 2013 Issue 270 C&T Series.)*

A number of environmental factors – for example, temperature and humidity both outside and in the client's house, and the animal's interaction with wildlife or with other local pets that may or may not be treated – influence flea numbers and the progression of an infestation. In a typical UK summer, it may take 3-8 weeks for flea eggs to become adults, so it is not surprising that fleas often still appear on a treated pet for several weeks after treatment in the face of an established infestation. When I sell flea treatments to clients, I explain that, even though the products we sell are very effective at killing fleas, there is no treatment or combination of pet and home treatments that will immediately completely eliminate their home's flea infestation.

More generally, while pet fleas currently appear not to be resistant to fipronil, several other insect species definitely are, including cockroaches, planthoppers and rice stem borers; imidacloprid resistance has become a problem for its use as an agricultural pesticide in several countries including the US; and there is evidence of spinosad resistance in agriculturally significant flies and moths.

Comment courtesy of:

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Insecticide resistance develops when a closed population of insects is repeatedly exposed to a chemical that kills the susceptible insects, leaving tolerant insects alive to reproduce and pass on the pre-existing genetic traits that reduced their susceptibility to the pesticide in the first place. Importantly, resistance does not occur as a result of an insecticide causing a genetic mutation in individual organisms, rendering them suddenly less susceptible to that product.

The development of resistance requires several factors including pre-existing genetic traits, the ability to propagate and transfer the resistant trait to the next generation and insecticide usage patterns that provide a high enough selection pressure to outweigh any fitness cost that the resistance trait imparts to the insect.

Fleas exist in an open system i.e. they are not confined to one place and a significant proportion of the population lives on untreated animals and is not exposed to pesticides at all, maintaining a reservoir of susceptible genes. For the ubiquitous cat flea (*Ctenocephalides felis*), refugia include the fleas living on hosts such as untreated pets, strays and feral animals, rodents and native wildlife. There is also a wide array of flea control products derived from different classes of insecticides available for use on pets, therefore fleas on treated animals are likely to be exposed to a variety of insecticides. Taken together, there is in effect, low selection pressure driving the development of resistance in *C. felis* populations.

For resistance to develop, organisms with resistance traits need to be able to reproduce and pass these traits on to subsequent generations. FRONTLINE PLUS combines an adulticide [fipronil] with an insect growth regulator [(S)-methoprene]. The incorporation of an insect growth regulator prevents the development of flea eggs, larvae and pupae and stops resistance characteristics being passed on to the next generation, further reducing the probability of resistant *C. felis* populations developing.

In Australia, Merial has conducted extensive investigations into persistent flea infestations within households and surveyed pet owner habits and attitudes to flea control. Poor compliance, a lack of understanding of flea ecology (effect of climatic conditions on emergence and reservoir hosts on re-infestation) and unrealistic expectations of flea product performance attributes remain the prevailing causes of apparent product failure. These findings are consistent with Dr Michael Dryden's conclusions following 20 years of research into the biology, ecology and control of fleas. Resistance in *C. felis* as a cause of modern flea control product failures has not been documented in the field anywhere in the world.

Merial has a dedicated helpline to assist veterinarians, nurses and pet owners with product related enquiries. We are happy to provide customer support to achieve better flea control outcomes via our toll free number, **1800 808 691**.

Outcome of the survey for fleas on dogs and cats in Australia

(C&T Series Issue 257 December 2009)

C&T No. 5272

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Fleas are ubiquitous and a year around problem in most coastal regions of Australia. Many different species have been recorded from dogs and cats in the past including cosmopolitan fleas – *Ctenocephalides f. felis* (cat flea), *C. canis* (dog flea), *Pulex irritans* (human flea), *Echidnophaga gallinacea* (stick fast fowl flea), *Nosopsyllus fasciatus* (rat flea), *Spilopsyllus cuniculi* (rabbit flea) and few other species including endemic marsupial fleas (Table 1).

In 2010, we investigated the traditional wisdom that the majority of 'fleas' on dogs and cats are by default *C. f. felis* (cat fleas), holds true in the 21st century (*Control & Therapy Series* – 257 December 2009, p. 43-44 e-book 1 2). In early 2010 we received large number of fleas from dogs and cats from many enthusiastic veterinarians across Australia.

We received 1,279 fleas from NSW (716 fleas; 127 from 27 dogs, 318 from 33 cats, and 271 from 90 unspecified dog/cat host), VIC (66 fleas; 62 fleas from 13 dogs, 4 fleas from 3 cats), QLD (233 fleas from 3 dogs), WA (258 fleas from 21 unspecified dog/cat host) and 6 fleas without geographical location from one cat. Our sampling was further supported by Merial Australia, which donated additional flea specimens to the grand total of 2,530 fleas from 291 cats and dogs (151 dogs, 69 cats and 71 uncategorised dogs or cats) from 5 states of Australia. The large majority of fleas were from coastal areas along the eastern coastline (Figure 1). After sorting through the fleas and inspecting all individual specimens submitted to us we came to the conclusion, that, indeed, the cat flea is the dominant flea. The great majority of fleas were the cat flea (n=2,500); 30 were stick fast fowl fleas found on companion animals in WA (Figure 2). The outcomes of this identification study, complemented by DNA sequencing confirming presence *C. f. felis*, are now published in Veterinary Parasitology [1].

It came as a relative surprise that we did not encounter the dog flea (*C. canis*), a close relative to the cat flea (Figure 2). This result does not mean that the dog flea is 'extinct' because we have been able to recently detect this species on foxes. Generally, however, the dog flea is more of a specialist for fox and dog, rather than the cat flea which is a so called 'generalist' (i.e. not a host-specific parasite), flourishing on cats, dogs, rodents, marsupials etc. Clearly, this generalist behaviour of the cat flea is more suited for our urban situations. It has also been previously reported that the dog flea is absent from the tropics, contrary to the cat flea which is distributed across all states. It was not surprising to have the majority of flea specimens from costal regions as fleas do not appreciate the drier environments of inland Australia.

We would like to express our gratitude to all the veterinarians that submitted fleas. This study highlights the potential that such collaborative work between the veterinary practices, university and industry has to address current issues in the veterinary profession.

Thank you.

Jan Šlapeta

References:

[1] Šlapeta J, King J, McDonell D, Malik R, Homer D, Hannan P and Emery D (2011). The cat flea (*Ctenocephalides f. felis*) is the dominant flea on domestic dogs and cats in Australian veterinary practices. *Veterinary Parasitology* 2011 Aug 25;180(3-4):383-8. Epub 2011 Apr 4

Figure 1. Maps of Australia and greater Sydney (inset) indicating locations of recorded fleas surveyed (colour-coded for host - blue: dog; yellow: cat; pink: unknown/dog/cat). Maps courtesy of Google Maps.

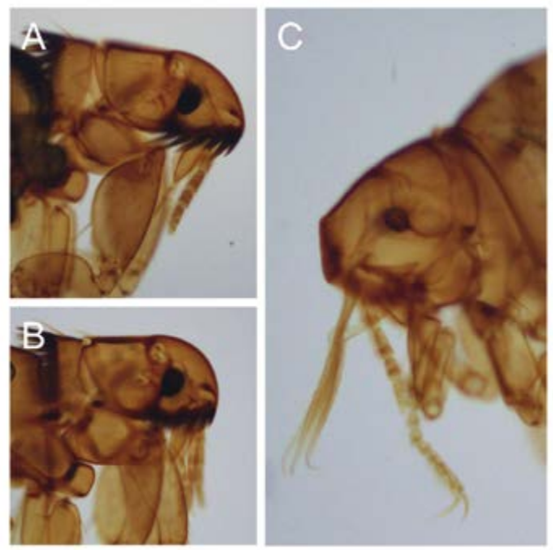

Figure 2. A* – Ctenocephalides felis *(cat flea). B* – Ctenocephalides canis *(dog flea), note the round forehead. C* – Echidnophaga gallinacea *(stickfast fowl flea), note absence of ctenidia (comb).

Table 1: Review of flea species recorded from dogs and cats in Australia

Flea species	World distribution	Host range in Australia	Distribution in Australia
Ctenocephalides f. felis	Cosmopolitan	Mainly on introduced mammals (dog, cat, mice, rats), also from native rodents and marsupials	Introduced, throughout Australia.
C. canis	Cosmopolitan	Predominantly on introduced canids (dog, fox).	Introduced, south eastern Australia, not recorded from the tropics.
Pulex irritans	Cosmopolitan	Human and animals within the household (dog, cat, fowl, mammals, marsupials).	Introduced, throughout Australia.
Nosopsyllus fasciatus	Cosmopolitan	Predominantly on introduced rats, accidentally on native rodents, rabbits, cat and dog.	Introduced, throughout Australia.
N. londiniensis	Cosmopolitan	Predominantly on introduced house mouse, accidentally on introduced cat, native and introduced rats, and marsupials	Introduced, throughout Australia.
Leptopsylla segnis	Cosmopolitan	Predominantly on introduced house mouse, accidentally on introduced cat, native and introduced rats, and marsupials.	Introduced, throughout Australia.
Xenopsylla cheopsis	Cosmopolitan	Normal host is introduced rat, also accidentally other introduced and native rodents.	Introduced, in and near large ports in Australia, no record from Tasmania.
Echidnophaga gallinacea	Cosmopolitan	Birds (fowl), introduced (dog, cat) and native mammals, marsupials, often nest inhabiting hosts.	Introduced, continental Australia, no record from Tasmania.
E. myrmecobii	Australia	Marsupials, marsupials (possum), mammals (rodents) and introduced mammals (rabbit, dog, cat).	Native, continental Australia, no record from Northern Territory and Tasmania.
E. perilis	Australia	Native mammals and marsupials, introduced mammals (rabbit, cat, dog).	Native, throughout the southern half of Australia, no record from Tasmania.
Spilopsyllus cuniculi	European	Rabbit, also accidental other hosts (cat, hare)	Deliberately introduced in 1968.

Chronic cystitis driving me crazy

C&T No. 5273

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'Rosie', a 14kg 7-year-old female desexed Cavalier King Charles Spaniel (CKCS) was presented on 25/11/10 for 24 hours of pollakiuria and stranguria. Physical examination was unremarkable other than a 3/6 heart murmur, loudest over the mitral valve, and a small firm bladder. Urinalysis (UA) revealed: USG 1.037, pH 6.5, 2+ protein, 4+ RBCs, negative for glucose, ketones and bilirubin. Ultrasound revealed a small bladder with a thickened wall. No stone or mass was visible. A tentative diagnosis of bacterial cystitis was made and she was treated with amoxicillin-clavulanic acid 250mg (18mg/kg) PO bid and we ordered in Royal Canin Urinary Dry Food® for her.

On 2/12/10, the owner reported that the frequent squatting had stopped. Repeat UA on a free-catch specimen revealed: pH 7, 1+ protein , 3+ RBCs negative for glucose, ketones and bilirubin. The antibiotic course was extended another 10 days and the urinary food was supplied. The owner was warned of the potential for underlying neoplasia not readily visible on the ultrasound as it seemed strange the haematuria was persisting.

On 9/12/10, repeat UA revealed: pH 5.5 (urinary acidification diet), trace protein, 4+ RBCs , negative glucose, ketones and bilirubin. Urine was submitted for culture and susceptibility (C&S) but unfortunately I ticked the wrong box and only a sediment exam came back. Scanty disrupted neutrophils and erythrocytes (mild pyuria and haematuria) were noted; no neoplastic cells were seen. Rosie finished her 3 week course of antibiotics and prescription food and was fine for 7 weeks before the symptoms relapsed.

On 1/2/11, UA revealed persistent proteinuria and haematuria (2+ and 4+ respectively) and USG > 1.050. Repeat ultrasound revealed a small bladder with 1cm thick bladder wall. No stone, sediment polyp nor neoplastic growth was seen. A free catch sample was sent for C&S as I was reluctant to do a cystocentesis for fear of seeding an unidentified neoplasm.

Rosie was again put on Amoxiclav® 18mg/kg bid PO pending results and again appeared to respond quickly. A light growth of *Proteus mirabilis* sensitive to all antibiotics tested, including cephalexin and amoxicillin-clavulanic acid, was cultured. Urine pH was 6 and 2+ calcium oxalate crystal was identified. A total course of 6 weeks of Amoxiclav® at the previous dose and frequency was prescribed. Maybe I just didn't go for long enough the first time?

On 25/2/11, whilst the dog was on antibiotics, a repeat free catch urine sample revealed persistent haematuria and proteinuria with a USG of 1.050 and pH of 6. Options given to the owner included referral to a specialist, repeat ultrasound with us, repeat C&S and/or plain radiographs; we radiographed Rosie only 6 months prior for thoracolumbar pain (spondylitis) and couldn't see anything on the radiographs retrospectively. The owner elected to finish the antibiotics and see what happened.

Only a few days after finishing the 6 week course of Amoxiclav® acid, the cystitis signs were back with a vengeance (see Figure 1).

Figure 1. Photo of gross haematuria with clumps of calcium oxalate crystals readily visible.

Poor client compliance was dismissed as a possible explanation as compliance appeared good (they seemed to come back at the right time for more tablets). It was decided to repeat the C&S and sediment exam and repeat the plain radiographs. I was considering contrast radiographs to highlight any structural anomaly I was missing. Plain radiographs revealed normal kidney size and shape and no liths. Repeat bladder ultrasound revealed a very thickened bladder wall. Repeat sediment cytology revealed a marked pyuria, marked bacteriuria, mild crystalluria and haematuria. A heavy growth of *Proteus mirabilis* was again cultured.

At this point I think the otherwise calm and patient owner was going to get a second opinion elsewhere and I thought I was going crazy failing to sort out a simple cystitis. I discussed all the results with Sue Foster and Doug Hayward at Vetnostics and it was suggested that a VBA be done to check for the likelihood of hyperadrenocorticism. I wasn't certain this would be helpful since the dog wasn't polyuric, polydipsic or isosthenuric/hyposthenuric and didn't look Cushingoid but Sue pointed out that sometimes Cushingoid dogs just present with urinary tract infections.

The owners consented to a haematology and serum biochemistry and VBA and FBC and all results were normal except ALP 190U/L (1-120) and albumin 41g/L (22-36). I spoke to the owners about the results but after Googling 'Cushings disease' they sheepishly asked if giving their dog prednisolone could have been the problem. It turned out that they had been giving 0.5mg/day daily for 4 months before weaning and stopping for 2 months. The male owner takes this low dose long term to help with his rheumatoid arthritis and thought it would help Rosie's osteoarthritis. Surely this low dose wouldn't be a problem?

On 30/3/11 we did an ACTH stimulation test and found: T=0 82 nmol/L (30-100) and T=1 399 nmol/L (200-400 nmol/L). At this time, she wasn't iatrogenic hyperadrenocorticism but borderline for spontaneous hyperadrenocorticism. It was decided to continue with the amoxicillin-clavulanic acid and do a repeat C&S this time via cystocentesis.

On 18/4/11 a cystocentesis urine sample was collected and for the first time there was no haematuria nor any bacteria cultured. It has been decided to finish this 6 week course of antibiotics and stop.

On 8/5/2011 only10 days after finishing her second 6 weeks course of antibiotics her symptoms were back again with a vengeance. I wanted to cry and I'm sure so did the clients. A cystocentesis and urine collection was again performed and again *Proteus mirabilis* was cultured susceptible to everything. Rosie was started back on the amoxicillin-clavulanic acid 18mg/kg orally bid and 3 days later a LDDST was performed.

This test showed conclusively that she didn't have spontaneous Cushing's disease as both the T=4 hours and T=8 hours showed cortisol levels of <10.

At this point we have decided to change to amoxicillin to reduce the ongoing expense and go with 12mg/kg bid to finish this 6 week course then reduce to sid at night only to try and prevent a relapse.

I have never had to deal with such a nasty relapsing cystitis but Sue assures me that they are well documented and do happen and are better to treat than the recurrent ones where a different bacteria is cultured each time.

I'd like to say a big thank you to Sue and Doug at Vetnostics for being fantastic in helping me handle this case. Many times they calmed me down and helped me work through it and I'm very grateful.

Postscript: 'Rosie' is still on amoxicillin at night and to date has not suffered a relapse.

Editor's Note: *Watch out for a Comment on this article (in our upcoming March 2013 Issue 270) courtesy of Mary Thompson, University of Queensland, whose PhD was on this topic.*

Investigation of anatomic characteristics and heritability of canine portosystemic shunts

Clinical study at ARH

C&T No. 5274

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Portosystemic shunts (PSS) may be congenital or acquired, intra-hepatic or extra-hepatic. They allow blood from the portal venous system (draining the gastrointestinal tract, pancreas and spleen) to enter the systemic circulation (vena cava or azygous veins) without passing through the liver. This results in reduced afferent blood supply to the liver, and thus inadequate hepatic development. Other sequelae include reduced clearance of ▶

endogenous and exogenous toxins, retinculendothelial dysfunction, altered fat metabolism and if severe, progressive liver failure. Clinical signs vary depending on the size of the shunt and include lethargy, gastrointestinal dysfunction, lower urinary tract signs (uroliths, urinary tract infection, stunted growth, poor body condition, recurrent infection, coagulopathy and hepatic encephalopathy. Intrahepatic PSS are most commonly reported in large breeds of dogs while extrahepatic shunts are more common in medium and small breeds.

Congenital PSS result from abnormal persistence of connection(s) between the vitteline and cardinal venous systems. They are reported more commonly in pure breed dogs and their crosses, with certain breeds being over represented (Cairn Terriers, Dachshunds, Miniature Schnauzers, Labrador and Golden Retrievers, Maltese and Australian Cattle dogs). Intrahepatic PSS are familial in Irish Wolfhounds. Genetic predisposition and heritability of intrahepatic PSS is strongly suspected, given the overrepresentation in certain breeds and the increased occurrence in some families of dogs.

The surgery department of ARH is currently conducting a study investigating the genetics and anatomy of portosystemic shunts, following on from the work of Geraldine Hunt conducted at the University of Sydney. Affected animals are diagnosed using conventional clinical assessments, pre- and post- prandial bile acid determinations and rectal ammonium chloride tolerance testing, the 2 biochemical tools most sensitive and specific in demonstrating functional shunting. We then use computer tomography (CT) angiography to anatomically define the shunts to aid in surgical planning (Figure 1). Blood is also being collected for a planned genome wide association study aimed at characterising key gene loci. Such studies are already underway overseas; however, we wish to concentrate on breeds that are more common in Australia than elsewhere, viz. Maltese (Figure 2), Australian Cattle dogs and Miniature Schnauzers. The long term plan is to identify dogs at increased risk of producing shunts using classical genetics and eventually a PCR test, to decrease the prevalence of this condition from pedigree dogs.

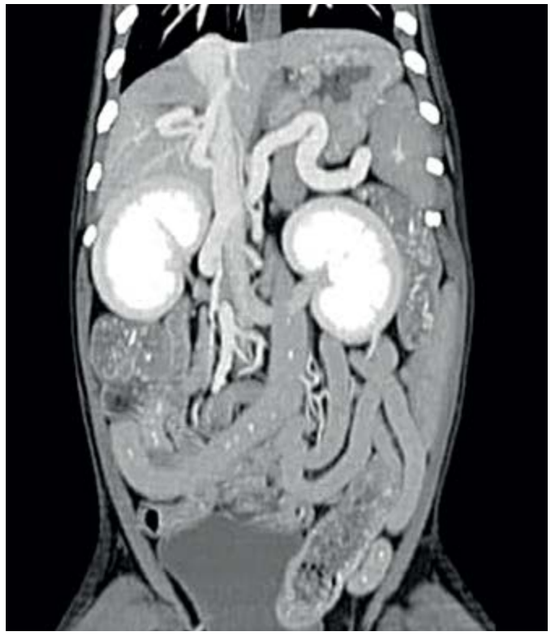

Figure 1. Computer tomography (CT) angiography aids in anatomically defining the shunts to aid in surgical planning

We welcome referral of any patients with suspected or diagnosed portosystemic shunts. If you would like to discuss your case further please contact Dr Arana Parslow (Small animal surgical resident) on 9758 8666 or **a.parslow@arhvets.com.** Even if owners are not interested in referral, we would be interested in obtaining blood specimens from dogs shown definitively of having a shunt and can arrange for samples to be collected from your clinic.

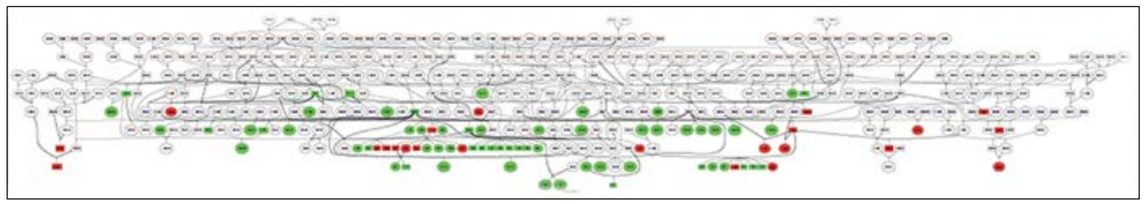

Figure 2. A genome wide association study aimed at characterising key gene loci in Maltese dogs.

Sample : contents from CVE, Small Animal Endocrinology 2012, Proc. No. 401 Page 1 Page 2

‘Gonzo’, a bonzer case

Sudden-onset inspiratory stridor in an old cat

C&T No. 5275

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‘Gonzo’, a 12-year-old desexed female domestic shorthair, was presented after hours at the Animal Emergency Centre Canberra for sudden onset dyspnoea. Other than a history of lower urinary tract problems, Gonzo had no previous health problems. She had been heard coughing for 2 or 3 days, as if to bring up a furball, which was unusual for her. That night she had eaten dinner and fallen asleep normally. Several hours later she woke her owners with a noisy and laboured respiration.

On physical examination she had an elevated temperature (39.8˚C), a heart rate of 200 beats per minute (BPM) and respiratory rate of 40 BPM. Her oral mucous membranes were pink but there was a pronounced respiratory stridor. The rest of the examination was unremarkable.

A cephalic catheter was placed in case there was a sudden decompensation, and she was sedated subcutaneously with acepromazine, methadone and diazepam at 0.1mg/kg each. Once she showed signs of sedation, conscious thoracic radiographs were taken without signs of distress on her part.

They showed an intra-tracheal radio-opaque foreign body lodged at the level of the bifurcation into the mainstem bronchi.

We discussed the options with the owners. As our centre does not possess an endoscope and the local options were not available on an Easter long week-end, the possibilities included referral that very night to a specialist centre in Sydney, or an attempt by us to retrieve it blindly. The owners understandably did not want to attempt a 4 hour drive to Sydney with a dyspnoeic cat, so we prepared to deal with it ourselves, having made sure they were aware of the risks involved.

We prepared long otic alligator forceps as well as one meant for endoscopic work but that we felt would be too large for a feline trachea.

Gonzo was induced with alfaxan-CD® intravenously until she was deep enough to probe with the otic alligator forceps while held head down. This was unsuccessful. Our next plan was to use suction on the larynx gently and then repeat the action. Lastly, we would intubate her trachea and use hyperinflation while holding her head down. None of these procedures moved the object when rechecked radiographically.

Our next resort was to use the larger gauge forceps. Before doing this we took several radiographs lining it up with the object. As it was overlying the cat, it understandably looked huge next to her trachea but turned out to be an easy fit. We still couldn’t retrieve or even feel the object.

She was allowed to recover from the anaesthetic, while held head down and gently coupaged in the hope she would cough on recovery. No such luck.

The next option being a thoracotomy, she was given an injection of dexamethasone (0.5mg/kg IV) while the owners considered all the risks involved, especially considering her age. In the end they still wanted it attempted, even given the guarded to poor prognosis and the cost of the procedure.

It is a matter of debate whether she saw her life flash before her eyes but she then had a coughing fit and was able to breathe without difficulty... A radiograph confirmed that the offending object was now in the stomach! (Figure 2 White arrow denoting it)

She was given prophylactic antibiotics, bronchodilators and kept overnight for observation. She was discharged the next day, smooching and purring normally.

On a search of the literature, particularly in the Veterinary Information Network, there were indications that such events are associated with poor dentition. A loose tooth is suddenly aspirated as it dislodges from its alveolus. Since Gonzo is over 12-years-old, and the object was of bone density and irregular, this seems logical.

I would like to thanks Dr Shannon West, a regular locum at the AECC, and Dr Vicky Saye, a surgeon at the Canberra Veterinary Hospital for their inputs in this case.

Postscript: Since writing this article, Dr Shannon West is now a permanent veterinarian at AEC and the practice has an endoscope.

Editor’s Note: *Perhaps the easiest way to deal with this type of scenario is to pass an embolectomy catheter or a cat Foley catheter (lubricated with a little KY Jelly®) **past** the foreign body, until the catheter is in a main stem bronchus. You then inflate the balloon and gently pull back the catheter. The foreign body is then pulled back out of the trachea. Ideally this is done using fluoroscopy or with DR radiographic assistance.*

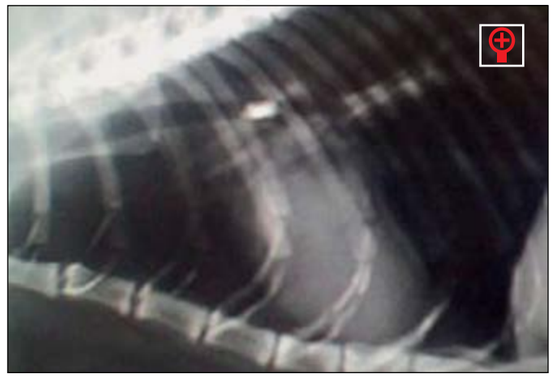

Figure 1. Lateral radiograph of the cat’s chest.

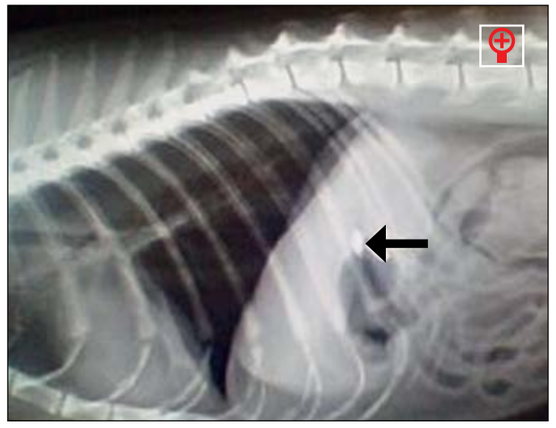

Figure 2. Foreign body in the stomach.

Authors’ views are not necessarily those of the CVE

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Useful mnemonics from (pre PC) college days...

C&T No. 5276

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Which cranial nerve is | sensory (S), | motor (M) | or both (B)?

[Each word of the mnemonic corresponds to the cranial nerve of the same number]

1	2	3	4	5	6
Some	Say	Marry	Money	But	My
7	8	9	10	11	12
Brother	Says	Big	Bonds	Make	More

Everyone knows the oh oh oh mnemonic but for those of the other (drinking!) persuasion:

For the 12 cranial nerves (olfactory, optic, ocolomotor, trochlear, trigeminal, abducens, facial, vestibulocochlear, glossopharyngeal, vagus, accessory, hypoglossal), we learned:

'On Old Olympus Towering Tops, A Finn And German Viewed Some Hops'

My recollection from Prof W.P.B. McAleer's (Physiology, Biochemistry) class (1960) is:

The bile of the liver saponifies fat
Helps to absorb and emulsify that
Aids putrefaction and purges a bit
The bile of the liver helps you to 'defecate'!

OR the pre-PC version we learnt: from Prof. Charles Burke-McAleer, 1960 (supplied for C&T by Phil Rogers, Ireland)

The bile of the liver saponifies fat
Helps to absorb and emulsify that
Saves putrefaction and purges a bit
and Stercobilinogen colours the s**t!

Beta-blockers: main contraindications, cautions -ABCDE:
Asthma
Block (heart block)
COPD
Diabetes mellitus
Electrolyte (hyperkalemia)

What's Your Diagnosis?

Answer to C&T No. 5253 (Sept 2012, Issue 268)

C&T No. 5277

Ursina Schiltknecht – DE Feline 2011 participant
E. ursinakas@hotmail.com

Figure 1. Flaccid paralysis in the chin and salivation problems.

Q. What could be the reason for an acute onset of these symptoms?

A. Ursina reports that there was spontaneous complete remission after some days and she believes the cat had idiopathic mandibular neuropraxis/neuropathy.

WINNER

Paracetamol toxicity (and foreign body ingestion!) in a kitten

C&T No. 5278

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'Alice', a 17-week-old domestic short hair kitten weighing 1.8kg, presented to our clinic for having swallowed a 10-cent coin the previous evening. On presentation, Alice was cyanotic (even her nose and ears were blue) with increased respiratory rate but no obvious dyspnoea. X-rays (see Figures 1 and 2) showed several metal opacity objects present in the stomach/cranial abdomen. Alice was immediately given oxygen therapy (in our 'purpose built' oxygen chamber – Figure 3) and placed onto IV fluids, which gave us a hint as to another possible problem. Her blood was very dark which, combined with the cyanosis, led us to suspect methaemoglobinaemia. Further questioning of the owners ascertained that Alice could also have eaten a 500mg paracetamol tablet.

Paracetamol toxicity can lead to methaemoglobinaemia and hepatotoxicity as the paracetamol conjugates with glutathione through a cytochrome P-450 oxidase pathway forming toxic metabolites. When these products are eliminated from the body, the glutathione stores (which in cats are low to begin with) are depleted. The toxic metabolites cause feline haemoglobin to be oxidised to methaemoglobin. This oxidisation also leads to Heinz body formation in red blood cells and can cause haemolysis as well as hepatic cell damage. Methaemoglobin is incapable of carrying oxygen resulting in severe cyanosis and a chocolate brown colour to the blood. Signs of toxicity appear 1-4 hours after ingestion and persist for 12-48 hours with death occurring 18-36 hours post ingestion if treatment is not implemented.

The aims when treating paracetamol toxicity are decontamination (by induction of emesis, gastric lavage and activated charcoal – if seen soon after ingestion), replenish glutathione stores, convert methaemoglobin back to haemoglobin and prevent or treat hepatic necrosis.*

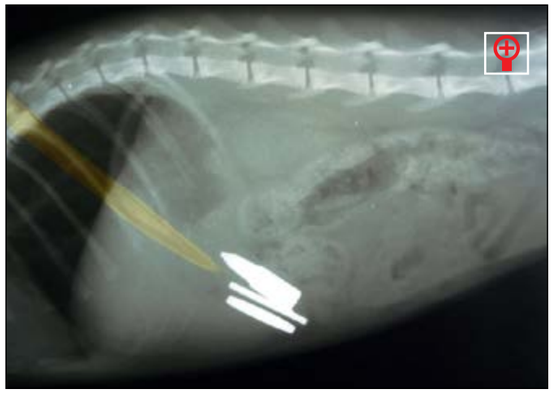
Emergency specialist colleagues (and VIN) advised us to administer N-acetylcysteine ('NAC'; Figure 4; trade name is Mucomyst®** as it is also used as a mucolytic) but warned that Alice had a very poor prognosis. NAC provides an alternative substrate for conjugation with the paracetamol metabolites thus acting to enhance elimination and reduce glutathione depletion. NAC enhances endogenous production of nitric oxide, dilating hepatic vessels and improving hepatic blood flow, and can reduce the extent of methaemoglobinaemia through its action as a free radical scavenger. Premedication with an antihistamine is advised as there is some potential for anaphylaxis. The loading dose rate is 140-280mg/kg slow IV*** (ideally within 16 hours of ingestion) then 70mg/kg IV every 4 hours until a total of seven doses have been given. NAC was sourced from the local hospital pharmacy and administered, along with Vitamin C (for anti-oxidant effect; 30mg/kg IV every 6 hours). VIN also suggests the use of S-adenosylmethionine (glutathione precursor) and cimetidine (reduces paracetamol metabolism by cytochrome P-450) but we did not have access to these medications.

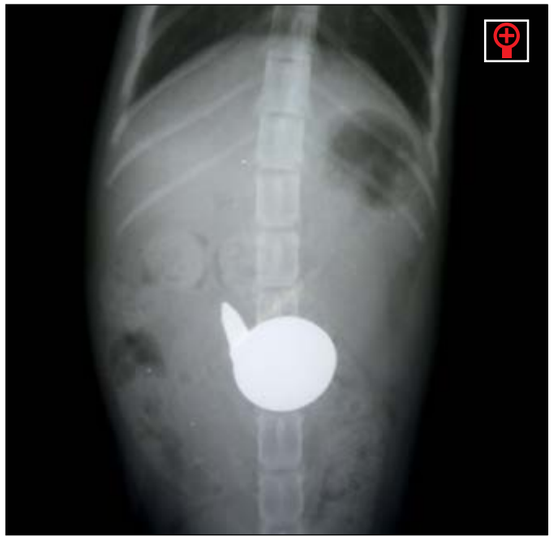
Alice remained in the oxygen chamber for 3 days (Thursday; Friday; Saturday) during and after medical therapy. She remained bright and alert throughout treatment and was eating well. Blood tests to assess her renal and hepatic function showed high potassium (6.6; 3.8-4.6), low chloride (114; 115-123), low calcium (1.49; 1.75-2.50), elevated alkaline phosphatase (101; 5-80), alanine aminotransferase (106; 5-80) and creatine kinase (741; 50-300) but were otherwise normal. PT and APTT were normal. Haematology was normal with no evidence of oxidative cell damage.

On Tuesday morning (Monday was a public holiday), Alice was bright and her mucous membrane colour was normal so we proceeded to surgery. We followed our routine anaesthetic protocol (acepromazine/methadone premed; Alfaxan induction; isoflurane maintenance) with the addition of intravenous cephazolin. A gastrotomy removed a 10 cent coin, an ALDI trolley token and a screwdriver bit for a drill. All abdominal organs appeared normal (despite the foreign bodies being inside Alice for almost a week) and Alice recovered well from the surgery. Two days later she went home.

Unfortunately we didn't get any photos of the offending items but the photo of Alice taken several months later (Figure 5) when she came in for her spey sutures to be removed shows she was fully recovered, if a bit wary of her trips to the vet.

Hopefully, this case shows that it is possible (with lots of hard work) for non-specialist/non-emergency practices to save cats with paracetamol toxicity. The fact that Alice survived paracetamol toxicity AND subsequent gastrointestinal surgery shows just how resilient our feline friends truly are – even when they are very little.





Figures 1&2 above - Lateral and ventro-dorsal abdominal radiographs showing metallic objects in the stomach.

***Editor's Note:** Some cats benefit from a transfusion of fresh whole typed blood after implementing the other treatment suggestions.*

****Editor's Note:** Note: Aine Seavers advises that Mucomyst® and Pardale-V® are now no longer available in Australia. However, a new commercial and very reasonably priced replacement acetylcysteine product of different concentration (800mg/4mL x 10 vial) is available through CH2 (Clifford Halem Supplier) and also from your local human Pharmacy. For further acetylcysteine information re dose, availability, concentration, routes of use etc, refer to or*

'Paracetamol poisoning: Was the Poison in the Purse or Was the Poison in the Purse?' (Persp. 87 SA Poisons, June 2011).

To justify a 10-vial purchase, Aine uses acetylcysteine not only for the rare Panadol® poisoning cases but also as a topical eye ulcer treatment in dogs and in rabbits and cats with snuffles so there are plenty of reasons to validate keeping the vials on the clinic shelf for ready access. Watch this space in our March 2013 Issue 270 to find out exactly how to use acetylcysteine topically.

****Editor's Note:** Some authorities recommend that some NAC be given orally as a larger proportion of the dose goes to the liver via the portal venous system.*

References

Veterinary Information Network, Inc. (www.vin.com) – Associate Database – Acetaminophen Toxicity; accessed 30/09/2010.

Figure 3. Our 'purpose built' oxygen chamber. Rollover to read: Malik R. Apr 2002. An inexpensive cat induction chamber. C&T No. 4397, Issue 223, *C&T Series*.

Figure 4. N-acetylcysteine (Mucomyst®) is also used as a mucolytic).

Figure 5. 'Alice' – fully recovered.

Authors' views are not necessarily those of the CVE

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CALL FOR ASSISTANCE

We Want YOUR Ticks!

Victoria McCarl, Tiziana Beninati, Graeme Brown,
Richard Malik & Nathan Lo
Faculty of Veterinary Science
School of Biological Sciences
The University of Sydney

The pathobiology group at the University of Sydney is currently embarking on studies to determine how extensive tick-borne infections are in Australia. This is following on from the ground-breaking work by Graeme Brown that the rickettsial bacterium *Anaplasma platys* is present in Brown Dog Ticks (*Rhipicephalus sanguineus*) from most parts of Australia[1].

The research project is focused on pathogenic and symbiotic bacteria of Australian ticks. In particular, we are interested in looking for novel and/or well understood *Rickettsia, Anaplasma, Borrellia* and *Ehrlichia spp.* in *Ixodidae*. Rickettsiae are known to be a cause of tick transmitted zoonoses worldwide. Recently, new rickettsiae have been discovered in ticks from Australia. These bacteria may be the cause of rickettsioses in humans and possibly animals[2,3]. Our aim is to check if ticks collected from domestic and wild animals could be vectors of bacterial pathogens. We have also discovered novel bacteria present in ticks, including *Midichloria*, which are of unknown pathogenicity[4].

We are interested in analysing ticks from the Sydney and Brisbane areas, and also from fringes along the east coast of Australia where *Ixodes* ticks are endemic.

PLEASE HELP BY SENDING US TICKS THAT YOU ENCOUNTER ON CATS, DOGS AND WILDLIFE

All ticks at any stage of engorgement are of interest to us. Please indicate the animal from which they were recovered, the date (year and month), and the **postcode** of the region.

Ticks can be easily stored in plastic vials or glass jars in either 70-100% ethanol or acetone. Methylated spirits will do, although the 5% methanol contained in the solution can sometimes interfere with PCR testing. **We can provide vials and ethanol/acetone and would arrange pick up from your clinic.**

We can also analyse live ticks. They can be stored in a jar or sterile urine container that contains a little cotton wool on the bottom to which a few drops of water are added. Jars should be sealed with a lid with a few pinholes in it. The ticks should be kept at room temperature and can live for weeks. They may even survive in sealed containers (no fresh O_2) for weeks. Be aware that these ticks can climb out of vertical smooth sided glass and plastic jars.

Veterinarians will be fully acknowledged for any results obtained from the samples you provided.

Samples should be sent to:

Dr Nathan Lo, PhD
School of Biological Sciences, Macleay Building A12
The University of Sydney NSW 2006
T. +612 9036 7649 F. +612 9351 4771
E. nathan.lo@sydney.edu.au

References

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3. Owen H *et al.* 2006 Oct Detection and identification of a novel spotted fever group rickettsia in Western Australia. *Ann N Y Acad Sci.*;1078:197-9.
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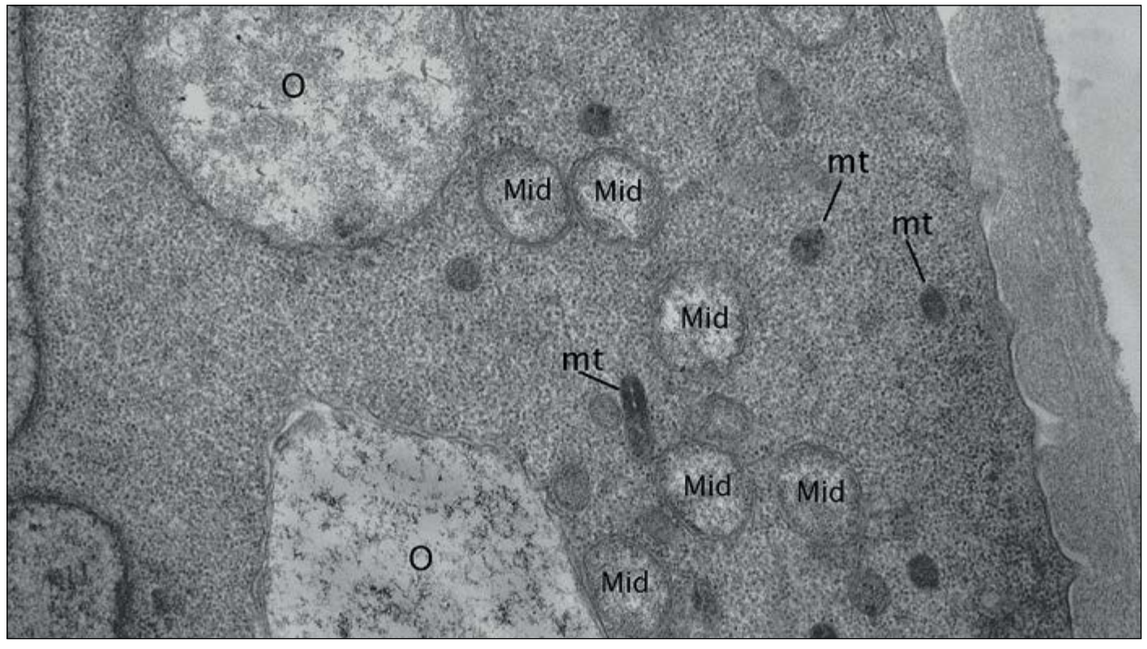

Figure 1. Transmission electron micrograph showing a developing oocyte from the ovary of an adult female I. ricinus. M. mitochondrii *(mid) is present either in the cytoplasm. Mt = mitochondria. Large unidentified bacteria are indicated by the letter O (Photomicrograph courtesy of Luciano Sacchi).*

Perspective 93

Round Table Discussion - Part 2

Replies to Tick Paralysis in the cat
C&T No. 5193 by Frank Gaschk, Dec 2011, Issue 264

Multiple Authors
Rob Webster, Kath Briscoe,
Fiona E. Campbell & Rick Atwell

Thank you to the multiple authors who generously provided these comments for Part 2 of our roundtable discussion.

New members/readers can read Part 1 in our e-book version.

Download Part 1 from Issue 268, September 2012.

Unsure how you access the e-book version?

The e-book is available to all CVE members. You just need your Username and Password from your IMIS account to access the e-book section of our website, which hosts each current e-book issue and the previous 4 issues. If you are unsure of your Username and Password, please call Jacqui Kennedy or email Jacqueline.kennedy@sydney.edu.au and she will advise you what they are. Then simply login to www.cve.edu.au/candtebook

Figure A. Ixodes holocyclus *(paralysis tick) in water (courtesy of Anne Fawcett)*

Figure B. Ixodes holocyclus *(paralysis tick) showing scale (courtesy of Anne Fawcett)*

MAJOR WINNER

Rob Webster and Jessica Webster 24/08/2012

Dr Rob Webster BVSC (Hons)MACVSc (emergency and critical care)

I graduated from UQ in 2000 and went straight into emergency and critical care. I loved the challenge, the pace, and the great people I worked with and have never left. I practice in the Southern Brisbane and Gold Coast corridor and manage many primary accession cases of tick paralysis, as well as referral cases requiring critical care.

In August 2012 I completed a 6 year training program in emergency medicine and critical care supervised by Professor Steve Haskins, a pre-eminent specialist in our field whose primary interests are respiratory physiology and mechanical ventilation techniques. During the training program Prof Haskins has helped my practice (The Animal Emergency Service) develop effective and logical critical care strategies for patients with tick paralysis. This has become my major clinical focus and research interest.

My goals within the profession are to understand the pathogenesis of tick paralysis, and to develop satisfying career paths for emergency vets. I live on the Gold Coast with my wife Julia, my daughters Zoe (23 months) and Jessica (2 months), 3 dogs, 5 parrots, 6 turtles and a few fish… ▸

Comment No. 1 courtesy of:

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Q&A (Qs by Richard Malik/As by Rob Webster as per the format in Part 1)

1. Have you read the Ilkiw papers?

Yes, they give valuable insight into the progression of respiratory dysfunction, respiratory pattern, and cardiovascular changes in the natural disease process. They are the only controlled experiments conducted on dogs with tick paralysis. There were limitations because of the invasive monitoring equipment and small numbers but I feel the criticism in the more recent literature is excessive.

The major difference I see between Ilkiw's dogs and our clinical patients is that only 1 dog in her series of experiments aspirated (it deteriorated quickly and was withdrawn) whereas we see bronchopneumonia and aspiration pneumonia very frequently in our patients today.

2. Do they treat any tick paralysis patients for heart failure? If so, how come we get away without treating them?

In answer to this question, Fiona Campbell doesn't treat clinical patients with tick paralysis. We (AES, VSS) never treat tick patients for heart failure.

Fiona proved the existence of a cardiac component to the tick toxin. On a large series of clinical patients she demonstrated significant pulmonary venous dilation. She sampled alveolar fluid and determined it was low protein, then concluded that pulmonary oedema is due to left sided heart failure.[1] She is the first to admit that tick patients lungs do not 'flood' like the typical decompensated mitral regurgitation dog. **The congestion and oedema is there (confirmed in multiple post mortem studies including mine).** The questions we need to ask are: How clinically relevant is the congestion and oedema, in the setting of such a multifactorial syndrome? Secondly, are we certain that congestion and oedema is secondary to left sided heart failure?

Unfortunately, we clinical vets like to treat first and ask questions only after repeated failures. While Fiona interprets the clinical implication of her research cautiously, the rest of the veterinary community is drying out every dog/cat with tick paralysis using repeated furosemide, and the resultant azotaemic/dehydrated critical patient becomes more difficult to manage.

I read Karina's comment as a plea for more reasoning, similar to that which I have thought of writing many times. I do not think I have ever managed a tick paralysis patient for fluid overload.

I do not know how to interpret the pulmonary congestion and oedema seen at post mortem in a clinical setting, because I am not sure of the mechanism behind it. Starling's Law states that it must be due to increased capillary hydrostatic pressure or permeability (low oncotic pressure would be easily recognised). Fiona's work showing the oedema fluid is low in protein is consistent with increased hydrostatic pressure.

3. What about measuring pulmonary wedge pressure and cardiac output?

I thought the easy way to resolve this would be to perform a controlled experiment like Ilkiw's monitoring wedge pressure throughout the progression of disease. Basically elevated wedge pressure suggests increased left atrial pressure as a consequence of left sided heart failure. Unfortunately there is a real issue with obtaining ethics approval for this sort of project.

We measured capillary wedge pressure in 2 dogs when Steve Haskins was here last and the pressures were normal, but they were isolated measurements in dogs without severe respiratory distress (both were 2b).

We are currently looking at doing a study like this using a Swan-Ganz pulmonary artery catheter continuously and frequent echocardiography by offering free treatment to owners without sufficient resources in exchange for participation in the study. I am not yet sure if we will get ethics approval to conduct such studies.

The problem with either approach is that even if wedge pressure is elevated, does this definitively point the finger at left sided heart failure?

Necropsy studies have shown that the congestion and oedema seen in tick paralysis is widespread. It has been reported in spleen, liver, gastrointestinal tract, myocardium, and CNS.[2-4] This cannot be all due to left sided heart failure. Other possibilities are:

- If oedema is high protein (which seems unlikely), then a syndrome of endothelial leakage is probable, and could be attributed to an as yet unknown toxin.
- If the oedema is low protein (more likely) the increased capillary hydrostatic pressure could be due to (a) bi-ventricular heart failure, or (b) some form of post capillary venular 'spasm'?

I don't know how this could be resolved at our current level of understanding.

4. Have you done careful echocardiographic examinations?

I have really been trying to get our cardiologists (Brad Gavaghan and Fiona Campbell) to try and non-invasively measure left atrial pressure. They tell me that there is too much variation in the normal measurements, and the techniques do not have the precision nor accuracy to be used in a scientific study to prove/disprove a hypothesis. They reckon wedge pressure is still our best way of estimating left atrial pressure.

5. Do you use CPAP or PEEP?

Peep of 3-5 cm H_20 is used in all ventilated tick patients. The aim is to maintain the trans-pulmonary pressure above the critical closing pressure. This avoids repetitive opening and closing and shear injury to the alveoli; keeps us in the steeper portion of the pressure/volume curve for the lung allowing greatest change in volume per unit change of pressure; and works as a recruitment technique to prevent atelectasis allowing lower inspiratory pressures to be used.

Higher PEEP is a component of lung protective ventilation strategies for patients with severe parenchymal disease.

CPAP is a useful weaning mode when the patient maintains some respiratory drive. Some of our clinicians use it, some don't. I typically just disconnect the patient at the end of my individual weaning strategy, but there is an article there in itself[5].

6. Why do you do so many arterial blood gases - why not capnography?

Capnography and pulse oximetry are the real time monitors we constantly use on ventilator patients to assess the adequacy of ventilation (capnography) and oxygenation (pulse ox) but we use frequent arterial blood gas analysis to validate these measurements and make changes to ventilator settings. PaO_2, is more accurate than a pulse ox (which has inherent problems/variability due to probe sensitivity and interference) and it is sensitive enough to evaluate lung function by looking at PF ratio on enriched oxygen, and alveolar to arterial oxygen gradient on room air. For example, an SPO_2 of 98% on your pulse oximeter (while on FiO_2 of 1) could reflect a PaO_2 anywhere from 150mmHg to 550mmHg (a threefold difference in oxygenating ability). The patient with PaO_2 of 550 has normal lungs while the patient with PO_2 150 has severe pulmonary disease yet the SPO2 will not show any difference.

Capnography is really useful. The end tidal CO_2 is a bit lower than $PaCO_2$ but works as a reasonable estimate of PCO_2 and the adequacy of alveolar ventilation. You need to be a bit careful in interpretation if a patient is intubated but not ventilated – sometimes the tidal volume is so reduced that the sample is not really alveolar gas, and artificially lower. Again, we validate the measurement with an arterial blood gas analysis.

In addition to measuring $PaCO_2$ and PaO_2, the arterial blood gas gives blood pH, base excess, bicarbonate level, electrolytes and lactate, all of which are valuable in managing a critical patient on life support. You can monitor a ventilated patient using only pulse oximetry and capnography but frequent blood gas analysis can help you do it much more effectively.

7. Do you have many cats that just need an endotracheal tube and not ventilation, like with a laryngeal problem?

Yes.

8. Why do cats and dogs have an end-expiratory grunt?

Prof Rick Atwell can provide the best explanation. It is to do with the patient's involuntary adoption of the respiratory pattern most advantageous to gas exchange given their particular pathology. I think in his explanation, the noise is due to the opening of the glottis after expiring against a closed glottis in an effort to open the small airways.

9. Have you not treated any where you actually see oedema fluid come back out of the tracheal or the endotracheal tube?

No oedema fluid, only pus in some horrible cases

10. Do you think the tick anti-toxin (TAS) works - is there any evidence?

I think it works, but we can always repeat Ilkiw's work in cats to prove it. The more severe the paralysis, the longer the signs last despite Tick anti serum and a logical inference is that the anti-toxin does not neutralise toxin which has already bound to the neuromuscular junction.

Local effects like palpebral paralysis always last heaps longer (2-3 weeks) and TAS doesn't seem to have any effect on them. We often send patients home with a temporary tarsorrhaphy because of palpebral paralysis long after they are walking, eating and drinking.

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Rob with a Shi Tzu called 'Kimba' after 36 hours of mechanical ventilation, and a tracheostomy. Kimba was discharged successfully after 174 h hospitalisation at Gold Coast AES/VSS hospital.

Comment No. 2 courtesy of:

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How I manage severe tick paralysis

Introduction

Administration of tick anti serum (TAS), and removal of embedded ticks are the 2 most important aspects of treating **tick paralysis**, but severely affected patients ultimately die from **respiratory failure,**[1] and this needs to be managed objectively to improve the chances of successful treatment.

The separation of Tick Paralysis (TP) and respiratory failure is a key concept in critical care of affected patients. The neuromuscular syndrome can result in multiple causes of respiratory failure including neuromuscular failure,[2] pulmonary oedema,[1, 3] aspiration pneumonia,[4] and upper airway obstruction.[5] Management of severely affected dogs and cats with tick paralysis is oversimplified by attempts to develop standard protocols. The heterogeneity of respiratory failure that develops in these patients requires an individualised approach which involves diagnosing the specific cause, so that effective management can be implemented.

Animals with stable respiratory function should be managed to avoid stress. It is important to minimise intervention which may precipitate deterioration of breathing. Some patients may be more suited to management in their home environment, but the clinician must weigh up the problems of monitoring the patient inherent with this approach. Respiratory failure must be managed aggressively if it occurs, because that is what ultimately kills the patient. Patients either fail to breathe sufficiently to support life because of advanced neuromuscular paralysis, or they become unable to oxygenate because of pulmonary parenchymal disease (pneumonia, pulmonary oedema). If you keep an animal breathing long enough, they almost all survive tick paralysis syndrome.

I advocate close monitoring of respiratory function in animals with tick paralysis and early, goal oriented measures when necessary to support respiration and sustain life until eventual recovery of the syndrome. This paper examines the individualised treatment of patients after the administration of tick anti-serum. There is an overview of general nursing techniques and supportive care, followed by an in depth discussion regarding the management of respiratory failure. For additional information regarding initial management of tick paralysis please refer to the articles by Prof Rick Atwell,[6] and Dr Dan Schull,[7] which review common approaches to management of dogs and cats, respectively. The Animal Emergency Service tick paralysis protocol gives a stepwise approach to initial management.

GENERAL NURSING CARE

Asepsis

General hygiene is essential in the handling of any critical patient. Hand-washing between patients is mandatory, and more extensive measures are taken when dealing with anaesthetised or intubated patients and those with an indwelling urinary catheter. These patients should be handled by the minimum number of personnel, and disposable gloves should be used.

Endo-tracheal tubes, urinary catheters, and tracheal suction catheters should be sterile when used. ▶

Intravenous catheters should be placed in an aseptic manner, and checked at least once daily for any signs of phlebitis. A catheter should be replaced if there is pain detected at the site, hyperaemia, or difficulty injecting fluid.

Bedding and Positioning

Soft bedding and frequent re-positioning helps avoid prolonged pressure to the weight bearing surfaces. Susceptible pressure points include the greater trochanter of the femur, the shoulder, and the olecranon of the ulna. Patients should be maintained in sternal recumbency angled slightly to one side. The position is altered every 4 hours to reduce pressure on the elbow. When it is not possible to position in sternal recumbency, changing sides frequently becomes more important because of the greater degree of atelectasis of the dependant lung fields. Oxygenation may limit attempts to re-position a patient. Where lung disease is severe, patients may only tolerate a limited number of positions, and frequently become hypoxaemic when this is altered.

Oral care

Oral care becomes extremely important when a tick patient is intubated. Oro-pharyngeal ulceration and lingual swelling will develop quickly if oral care is not prompt and meticulous. The clinician should also be mindful that most cases of hospital acquired pneumonia in human patients are caused by pharyngeal microflora,[8] and this could also be significant in tick patients, given the high incidence of bronchopneumonia and aspiration pneumonia which occur in severe cases (Webster unpublished). Intravenous tubing should be used as tube ties because it is non-absorbent (compared to gauze ties), the mouth should be kept slightly open to reduce pressure on the tongue and should be rinsed using sterile saline and dilute chlorhexidine daily to maintain hygiene. Gentle tooth brushing with an extra soft tooth brush may also be beneficial. Glycerin or Paw-Paw ointment can be used to keep the tongue moist, and wrapping it in moist swabs is also useful. Pulse oximeter probes may also cause pressure necrosis to the tongue or buccal mucosa and should be re-positioned every 2-4 hours.

Bladder care

Urine retention is a common problem when managing recumbent patients with tick paralysis. The consequences are bladder atony and/or urinary tract infections. If the bladder is easily expressed, this can be performed 4 hourly onto absorbent padding. Weight changes of the padding can be used to monitor urine output. When the bladder is difficult to express, a urinary catheter is placed using aseptic technique and is used to keep the bladder empty and monitor output. The urine sediment is examined daily for development of urinary tract infection (UTI) after placement of a urinary catheter. Where sediment examination is suggestive of UTI the catheter is removed (or replaced in an aseptic manner), the urine is cultured and broad spectrum antibiotics are commenced. Antibiotic therapy is modified depending on urine culture and susceptibility results. Urinary catheters are removed as soon as practical in the recovery period.

Nutrition

Megaoesophagus,[9] laryngeal paralysis,[10] and choking[5] are all well reported in dogs affected by tick paralysis and probably all predispose to aspiration pneumonia. Most animals with tick paralysis are kept nil-by-mouth until neuromuscular recovery, when a supervised feeding trial is performed before gradual re-alimentation. Unfortunately, critical patients may be unable to eat for an extended period especially if anaesthetised or mechanically ventilated, and there may be a beneficial effect to feeding that outweighs the risk of aspiration pneumonia.

There is not yet any research supporting either extended nil-by-mouth periods or early enteral or parenteral feeding in patients affected by tick paralysis. Experience and research into other critically ill patient groups suggest that early feeding may significantly improve prognosis.

In our practice feeding commences on day 3 or 4 of fasting after admission to hospital. In most instances we feed through a naso-gastric tube in order to bypass the oesophagus, which also allow us to check for residual food in the stomach prior to the next feeding. The draw-back to a naso-gastric tube is reduction in the effectiveness of the cardiac sphincter which may predispose to gastro-oesophageal reflux. Standard feeding guidelines are to commence feeding 1/3 of the basal metabolic requirement on day one, increasing to 1 x resting energy requirements on day 3.

Figure 1. Ventilation corner at Gold Coast AES/VSS hospital.

Parenteral nutrition remains a viable (albeit expensive) option for tick paralysis patients.

Fluid therapy

Patients unlikely to resume oral fluids or food within 24 hours should receive intravenous fluids to maintain homeostasis and replace losses from the gastrointestinal and respiratory tract. The most common fluid choice in our practice is a balanced crystalloid such as lactated ringers solution (Hartmann's Solution) with supplemental potassium where indicated. Fluid therapy should be tailored individually and carefully tailored to the patient's needs using clinical examination, bodyweight changes, packed cell volume and total protein (PCV/TP), and blood gas analysis to monitor hydration levels. In critical patients requiring anaesthesia and/or ventilation, central venous pressure is an additional estimation of pre-load which can be monitored to help avoid excessive fluid administration. Maintenance fluids are probably sufficient in most patients with tick paralysis, and I am inclined to administer slightly below this level in most instances due to the possibility that fluid overload could contribute to the development of pulmonary oedema.

Clipping/shaving

Anecdotally, the patients where a second (or more) tick(s) is present but not found until several days after initial treatment are at a higher than usual risk of death from the syndrome. The 'missed tick' can also be a public relations nightmare especially when the patient is moved to or from a referral hospital and the tick is located by a second veterinarian. This can be avoided by open discussion with owners regarding the fallibility of tick searching, but it is essential to do everything possible to ensure every tick is located and removed.

There are different strategies for killing or removing additional ticks **in dogs not cats** including application of an acaracide top-spot or spray (Advantix®/Frontline®) or through bathing in an acaracidal shampoo.

In my practice every dog with hair longer than a greyhound is clipped, bathed, and searched at least 6 hourly by technicians. We have gradually got more aggressive with tick searches over the 12 years I have worked in the practice because of ongoing issues with missed ticks, despite other efforts to kill or dislodge them. We now only have to deal with client complaints about the aesthetic appearance of their pets!

Tick clipping is usually done under the minimal sedation required to alleviate patient anxiety. This is often achieved using ACP 0.02mg/kg and butorphanol 0.3mg/kg. When anaesthesia is required alfaxan is used to effect. A cuffed endotracheal tube is maintained in every anaesthetised dog although cats are often given O_2 by face-mask. (Interestingly we do not see aspiration pneumonia in this species).

Eye Care

Animals with tick paralysis are susceptible to exposure keratitis and superficial ulceration because of reduced palpebral reflexes. In many cases this occurs as part of the generalised neuromuscular weakness and resolves with the other clinical signs. This is different to local paralysis of the eyelids which may occur when the site of tick attachment is close to the eye. In these cases prolonged eyelid paralysis may persist for at least 2 weeks after discharge from hospital. Superficial corneal ulceration can quickly progress to keratomalacia and a 'melting ulcer'. Perforation and loss of the eye is possible.

All paralysed patients should receive eye lubrication as part of their general nursing care. Cellulose-containing drops such as Celluvisc® administered 2-4 hourly may prevent desiccation of the globe and maintain the pre-corneal tear film. Careful examination of the cornea should be performed by a veterinarian at least once daily. If ulceration is detected on ocular examination or through corneal flourescin staining, antibiotics are warranted. In our practice we add Tricin® ointment 8 hourly to the existing 2 hourly lubrication.

If a stromal ulcer develops, anti-collagenase therapy is initiated. Autologous plasma drops are administered hourly while the patient is hospitalised. The drops are made by removing the plasma from blood collected in EDTA after spinning in a centrifuge for 3-5 minutes. The drops are refrigerated in 1mL syringes with the syringe in current use kept on the animal's chart for quick access. When plasma and Celluvisc® drops are due together, they are separated by 5 minutes, and the Celluvisc® is administered last. When the patient is eating, doxycycline 5mg/kg BID is administered. This is often used as a medication for home use after discharge.

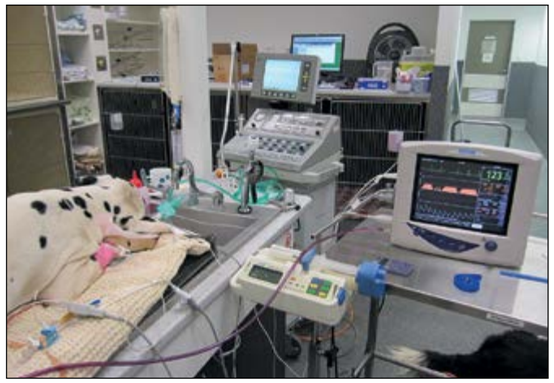


Figure 2. 'Patch', a 6-year-old Dalmatian, was successfully discharged after 132 hours of mechanical ventilation.

Tarsorrhaphy is performed only for patients with complete lack of a palpebral reflex due to local tick attachment. The lateral third of the eyelid is apposed using a single mattress suture of 3/0 or 4/0 non-absorbable monofilament (Novafil®). The eyelids are not clipped before this procedure to avoid contamination with minute hair fragments. Topical medication may still be administered into the palpebral fissure.

Stromal exposure necessitates topical drops rather than ointment as the ointment causes pain on administration and is irritant to the stromal tissue. It is important to select an antibiotic which is effective against *Pseudomonas*. We use ofloxacin (Ocuflox®) drops 6 hourly for these patients.

Comment: *ofloxacin 0.3% (3 mg/mL).*

Note: *Thank you to Dr Denise Brudenall MA VetMB CVR MVS (Eye Care for Animals) for contributing to the 'Eye Care section' of this article.*

Regurgitation and vomiting

Emetic events are common in patients affected by tick paralysis. Historically, regurgitation was thought to be the sole cause of these events due to oesophageal dysfunction. Clinical experience suggests that vomiting is also common because patients may exhibit abdominal contractions prior to emesis, projectile expulsion of ingesta occurs and bile staining is not uncommon. It is logical to infer that reducing the incidence of vomiting and/or regurgitation may reduce the propensity to aspiration pneumonia.

A multi pronged approach to vomiting/regurgitation involves the use of anti-emetics, pro-kinetics, and gastro-intestinal protectants. ▶

Metaclopramide can be used intermittently or as a continuous rate infusion for a pro-kinetic effect, an anti-emetic effect and because of its effect to increase tone of the lower oesophageal sphincter.

Omeprazole and ranitidine can be used to reduce gastric acidity and potentially reduce reflux oesophagitis.

Maropitant is also an effective anti-emetic.

None of these medical therapies have been studied for their effect on emetic events in tick paralysis patients, and there is not yet any evidence to say that reducing emetic events will correspond with reduction in aspiration pneumonia.

Basic physical strategies to reduce regurgitation and/or vomiting have involved nil-by-mouth until recovery from the syndrome.

There is a potential benefit from early airway protection when there is reduced ability to protect the airway and simultaneous regurgitation or vomiting by inducing anaesthesia and placing a cuffed endotracheal tube. This is an invasive and potentially expensive step for veterinary patients, but has a role in the management of humans affected by tick paralysis and similar lower motor neurone diseases.[11-13]

RESPIRATORY ASSESSMENT

To recognise deterioration in a TP patient frequent respiratory assessment is required. This involves physical examination and whichever diagnostic tools are available. The deteriorating patient will exhibit one of 3 types of breathing:-

- **Hypoventilating** patients have reduced respiratory rate and effort. Patients with respiratory rates below 20 and advanced tick paralysis are likely to be exhibiting severe enough hypoventilation to warrant treatment. Quantifying hypoventilation involves either capnography to measure end tidal CO_2 ($ETCO_2$), or blood gas analysis (preferably arterial, but venous is acceptable; it should be about only 5mmHg higher than $PaCO_2$). The critical limit we use for PCO_2 (arterial or venous) is 60mmHg. Levels higher than this indicate severe hypoventilation and mechanical ventilation is required. Some animals will survive much higher PCO_2 levels than 60mmHg, but this is unpredictable. Respiratory acidosis is the pathological problem associated with hypoventilation, and this can be assessed using the blood gas analysis along with the CO_2 level.
- **Restrictive breathing patterns** which involve increased rate and work of breathing are consistent with pulmonary parenchymal disease. TP patients are known to develop pulmonary oedema, and aspiration pneumonia. Hypoxaemia often occurs, but the patient will work very hard to prevent this, and **may respiratory arrest due to fatigue while the SPO_2 level is adequate**. The level of hypoxaemia can be quantified using arterial blood gas analysis or pulse oximetry. The critical limits for these measurements are: SPO_2 >90% and PaO_2>60mmHg. Management of hypoxaemia and increased work of breathing involves oxygen supplementation initially. Mechanical ventilation is required should this not be effective in maintaining adequate oxygen **and** reducing the work of breathing to a sustainable level.
- **Upper airway obstruction** occurs frequently in TP patients especially brachycephalic animals and those intubated for a prolonged period. The classic clinical signs of stridorous breathing and inspiratory dyspnoea may not be apparent if the patient is showing marked paralysis. Additional clinical signs include gagging, retching, regurgitation, and anxiety. The suspected upper airway obstruction can be confirmed if the signs resolve after placement of an endotracheal tube. Management of upper airway obstruction involves maintaining an endotracheal tube under anaesthesia until the signs resolve or placement of a tracheostomy tube.

Complexity

Management of 1 component of respiratory failure can be straightforward, but it is typical for a patient with severe TP to have contributing problems from the upper airway, the pulmonary parenchyma and have marked hypoventilation as well. Despite its paramount importance, physical examination of the respiratory system in patients with TP may not clearly identify the underlying abnormalities. This is often attributed to the fact that respiratory failure may be caused by multiple concurrent mechanisms, and because the clinical signs of respiratory disease may be masked by the generalised weakness and central respiratory depression which occur with TP. Hypoxaemia may be present due to ventilation perfusion mismatch in the lungs, neuromuscular paralysis or a combination of the two. Stridor may not be heard if a patient with upper airway obstruction cannot inspire vigorously because of muscle weakness. Similarly, inspiratory crackles may not be auscultated when a patient cannot inspire deeply. In summary, patients with respiratory failure because of TP may adopt a rapid shallow (restrictive) breathing pattern which diminishes the classical physical signs of respiratory disease. Each patient requires a different life support strategy which should be developed and monitored by repeating the physical assessment and arterial blood gas analysis.

Figures 3 to 5. 'Heidi', a 12-year-old Miniature Daschund, was successfully discharged from hospital after receiving mechanical ventilation for 62 hours.

Respiratory support strategy

Assessment → Intervention → Re-evaluation

The strategy of managing respiratory failure in tick paralysis involves close monitoring of respiratory function, and intervening when respiratory failure is detected. Any increase in respiratory effort, altered breathing pattern, or reduced oxygen saturation using pulse oximetry should prompt the clinician to evaluate respiratory function more thoroughly by assessment of arterial blood gas. Oxygen therapy should be implemented if the animal is hypoxaemic (PaO_2 < 60 mmHg) and mechanical ventilation should be instituted if the animal is hypoventilating ($PaCO_2$ > 60 mmHg) or if the animal does not respond to oxygen therapy. Increasing the fraction of inspired oxygen (FiO_2) is the first method of managing hypoxaemia. There are several ways that this can be accomplished.

Increasing the fraction of inspired oxygen

i. Flow-by and Mask oxygen

High flow oxygen administered by holding a tube 2-4cm from a patient's nose can significantly increase FiO_2. Studies performed in anaesthetised dogs of different sizes (15-29kg) using 2L/min flow rate, improved FiO_2 at the tracheal bifurcation to a mean of 37.2%. When the oxygen was concentrated using a closely fitted face mask, the FiO_2 measured using the same methodology reached a mean of 46.5%.[14] Efficacy of these techniques is adversely affected by uncooperativeness of the patient (some respiratory distressed animals will not tolerate the face mask). This technique requires high flow rate and some patient restraint so it is generally only used for short term management while planning a more suitable method of long term support. An Elizabethan collar with the opening covered with plastic wrap or a plastic-bag hood may also be used as a short-term method of increasing the FiO_2. One study showed FiO_2 of 40% with 1L/min flow rate,[15] and another obtained 95% FiO_2 with 300mL/kg/min flow rate.[16] These patients were healthy and anaesthetised so caution is needed in extrapolating the results to clinically affected TP patients. This technique is more suitable than mask oxygen for long term use. Exhaled carbon dioxide can diffuse through the plastic or can vent through the inevitable leaks in the system and rebreathing should not be a problem. Use of a mask or a hood will reduce the heat-losing efficiency of panting, and can lead to patient discomfort or hyperthermia, both of which increase oxygen demand and are undesirable. The inside of the mask or hood will become very humid with the animal's exhaled water vapour and the patient's tolerance to it may be time limited.

ii. Nasopharyneal oxygen

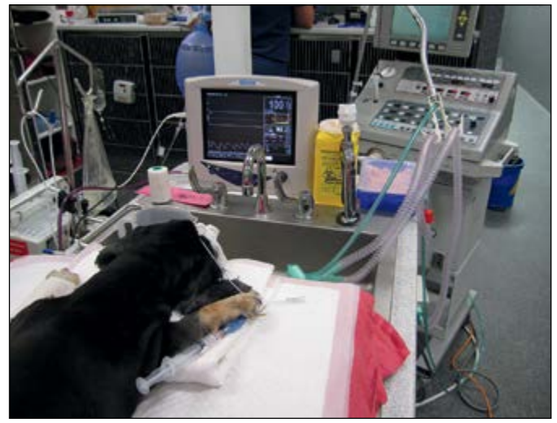
Nasal O_2 is a very effective method for improving FiO_2 over the short to medium time frame.[15] A nasal catheter is placed,[17] which permits delivery of oxygen at relatively low flow rates into the nasopharynx. The resultant FiO_2 is proportional to the flow rate administered and the breathing pattern of the animal.[18] After placement of the tube, patient discomfort is minimal and most tolerate the nasal tube well. Inspired oxygen can be further increased by placement of bilateral nasal lines. Median FiO_2 of 0.6 was achieved in healthy dogs using bilateral flow rates of 100mL/kg/min.[18]

iii. Cage oxygen

Commercially available oxygen cages are effective at delivering high inspired oxygen concentrations and controlling humidity and temperature. Unfortunately they are expensive and demanding on space within a hospital. Another draw-back is the loss of FiO_2 whenever the door is opened. Homemade-oxygen cages can be created using impervious cage doors or cabinets and high flow oxygen or human infant humid-cribs (incubators). Oxygen concentration within one of these chambers can be monitored using an inexpensive oxygen analyser (Pro O_2 oxygen analyser, Nuvair, Oxnard CA). The author (Rob Webster) commonly uses high flow oxygen in a hospital cage covered by plastic food wrap which achieves FiO_2 between 40-52% using 30 L per minute oxygen flow. (*Webster, personal communication*) **Note:** Soft smaller oxygen cages that only need 10L/min – which you can generate using an oxygen concentrator – are also available.

iv. Trans-tracheal oxygen

High FiO_2 can be achieved by delivering a relatively low flow of oxygen directly into the trachea. This can be performed using a silicone feeding tube inserted into an endotracheal tube or tracheotomy tube. A technique for percutaneous administration of intra-tracheal oxygen has been described where a large gauge intravenous catheter is inserted into the trachea between 2 adjacent tracheal rings.[19] When this method was evaluated in healthy awake dogs the FiO_2 achieved with a flow rate of 100mL/kg/min was equivalent to intranasal administration of oxygen at 200mL/kg/min. The authors suggested starting with a flow rate of 50mL/kg/min, then adjusting it guided by pulse oximetry and blood gas analysis.[19] This technique (with minor modification) was described in the management of 2 dogs with TP and respiratory failure using flow rate of 50mL/kg/min with good clinical outcomes.[20]



Figures 4 to 5. 'Heidi'.

Airway management

Airway protection by placing a cuffed endotracheal tube has been reported to prevent aspiration in humans with dysphagia and neuromuscular paralysis. It is commonly employed as a supportive treatment in patients when airway integrity is compromised,[21] before blood gas abnormalities suggest respiratory failure.[12] There are several case reports of humans with tick paralysis who were intubated to protect the airway without requiring mechanical ventilation.[11] Small animal patients with TP may develop megaoesophagus,[9, 22] laryngeal paralysis,[10] 'choking'[5] and regurgitation during their treatment, and those patients dying of tick paralysis often have histological evidence of aspiration pneumonia (*Webster unpublished data*). Given the clinical utility of this technique in human patients with similar disease processes, it is possible that aggressive early airway management may reduce aspiration in small animal patients with TP.

The author has adopted early intubation in patients with TP for airway protection when there is gagging, choking, or regurgitation combined with any evidence of laryngeal dysfunction, upper airway obstruction, or aspiration. General anaesthesia is induced (see guidelines below), and a snug fitting, cuffed endotracheal tube is placed. Intra-tracheal oxygen is administered by introducing a suitably sized sterile feeding tube (3.5, 5, or 8 Fr Argyle feeding tube, Tyco Healthcare, Mansfield MA) through the endotracheal tube to the level of the thoracic inlet and the required flow rate is determined by monitoring oxygen saturation or PaO_2. Although early airway protection has been anecdotally useful in these patients, there are no veterinary studies to support the strategy.

Editor's comment: *Mike Fitzgerald used to manage some cases like this by using a suction catheter to evacuate the fluid accumulating in the dilated oesophagus.* ▶

MANAGEMENT OF SPECIFIC LUNG DISEASE

Aspiration Pneumonia

Aspiration pneumonia is commonly encountered in dogs with severe tick paralysis and a single aspiration episode may cause dramatic deterioration in an otherwise stable patient. Animals with tick paralysis may develop megaoesophagus, laryngeal, and/or pharyngeal dysfunction which predispose to development of the condition. There may not always be a history of regurgitation or vomiting, as silent aspiration is possible. Remember, TP cases have a weak cough, so normal clearance mechanisms of protection are not operating!

When pulmonary disease is considered likely on the basis of physical examination or blood gas analysis, thoracic radiography is required. The typical aspiration pattern consists of a lobar, broncho-alveolar pattern with a propensity for right cranial and right middle lung lobe involvement. (Figure 6)

Management of pneumonia involves appropriate antibiotics and respiratory support. Oxygen therapy is used to maintain SPO_2 >90% without excessive respiratory effort. If oxygen supplementation does not resolve hypoxaemia and lessen the respiratory effort, ventilation is warranted. The survival probability for a dog ventilated for hypoxaemia for parenchymal disease is significantly lower than for hypoventilation alone.

Antibiotics should consist of agents with activity toward the likely flora of the upper gastro-intestinal tract and adequate penetration of lung tissue. Our typical regime involves either ampicillin, or cephalosporin with a fluroquinolone for Gram negative cover.[23] Alternate suggestions include a potentiated penicillin (either amoxicillin/clavulanate or ticarcillin) or 3rd generation cephalosporin. Ideally, antibiotic choice should be based on culture and susceptibility data. There is a study underway to evaluate the most common organisms associated with pneumonia in patients with tick paralysis.

Physical therapy involves short walks if ambulatory; frequent repositioning if not, and sternal positioning where severe paralysis is present. Patients are nebulised using saline or water for 10-15 minutes 4-5 times daily, and coupage is performed immediately afterward. The need for airway humidification to assist mucociliary clearance can't be overstated, so careful fluid therapy is also a mainstay for these patients.

We attempt to prevent aspiration using nil-by-mouth and pro-kinetic medication. In recent times we have adopted early airway protection by inducing anaesthesia and placing a cuffed endotracheal tube when a patient is felt to be at risk of aspiration. Patients considered at risk have repeated regurgitation, or gagging episodes, and those require pharyngeal suction to remove excessive secretions.

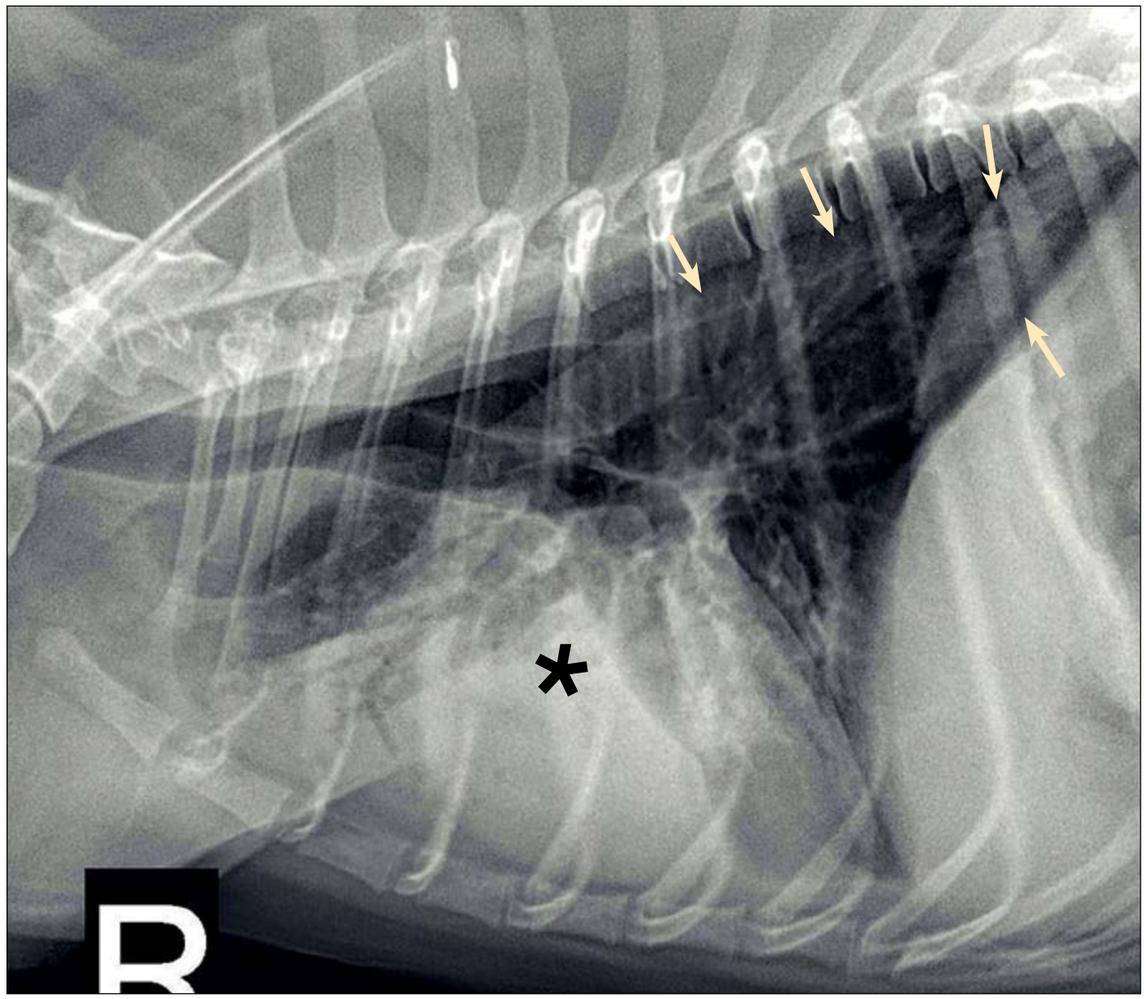


Figure 6A. Lateral thoracic radiograph of a dog with tick paralysis, megaoesophagus (arrows) and suspected aspiration pneumonia (*)

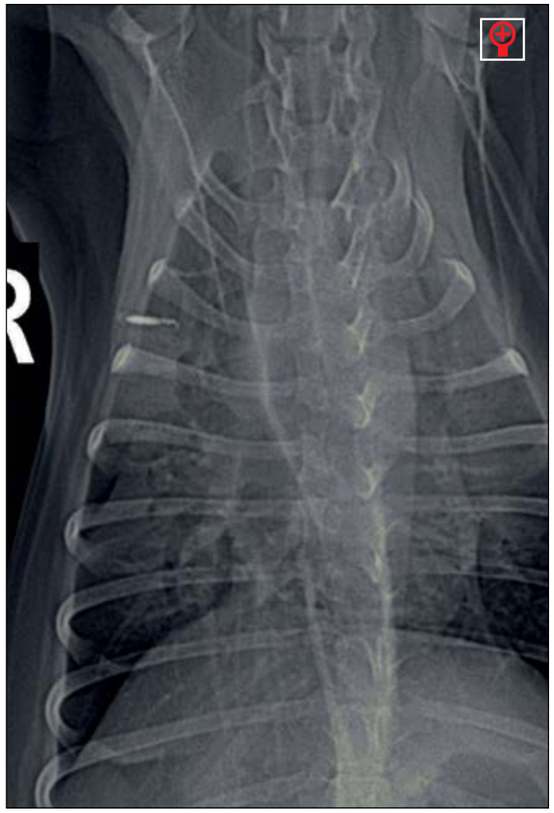


Figure 6B. VD radiograph of the same patient shown in Figure 6A.

Pulmonary Oedema

Pulmonary oedema has been recognised in several experimental studies of tick paralysis, but it is potentially over-emphasised in the management of patients affected by tick paralysis exhibiting respiratory distress. Radiographs of patients with respiratory failure from tick paralysis are often more consistent with aspiration pneumonia than pulmonary oedema (lobar distribution of an alveolar pattern most prevalent in the cranioventral and right middle lung lobes). We never see thoracic radiographs from dogs with tick paralysis where there is a pronounced hilar distribution of interstitial or alveolar opacity as seen with left sided congestive heart failure. Similarly, clinical measurements do not suggest increased central venous pressure (as measured with a central line in many patients) or increased pulmonary capillary wedge pressure (we have only measured this twice) in tick patients. Both of these pressures would be expected to increase if pulmonary oedema is being caused by left sided congestive heart failure. Although pulmonary oedema is recognised experimentally, it remains to be shown how relevant it is in the clinical patient, and whether it is caused by heart failure or another mechanism e.g. neurogenic pulmonary oedema

The main problem with widespread diuretic use based on the suspicion of cardiogenic pulmonary oedema, is that the side effects can be significant. These are worsened if respiration fails to improve after administration of a diuretic, and the dose is subsequently increased or repeated. While a single dose of furosemide may be beneficial if pulmonary oedema secondary to left sided heart failure is present, we frequently see the situation where repeated large doses of diuretics are administered to a patient where there is little clinical support apart from worsening respiratory distress. This leaves a patient (who may have aspiration pneumonia) in a worse situation because dehydration and azotaemia (and to a lesser extent: hyponatraemia and ▶

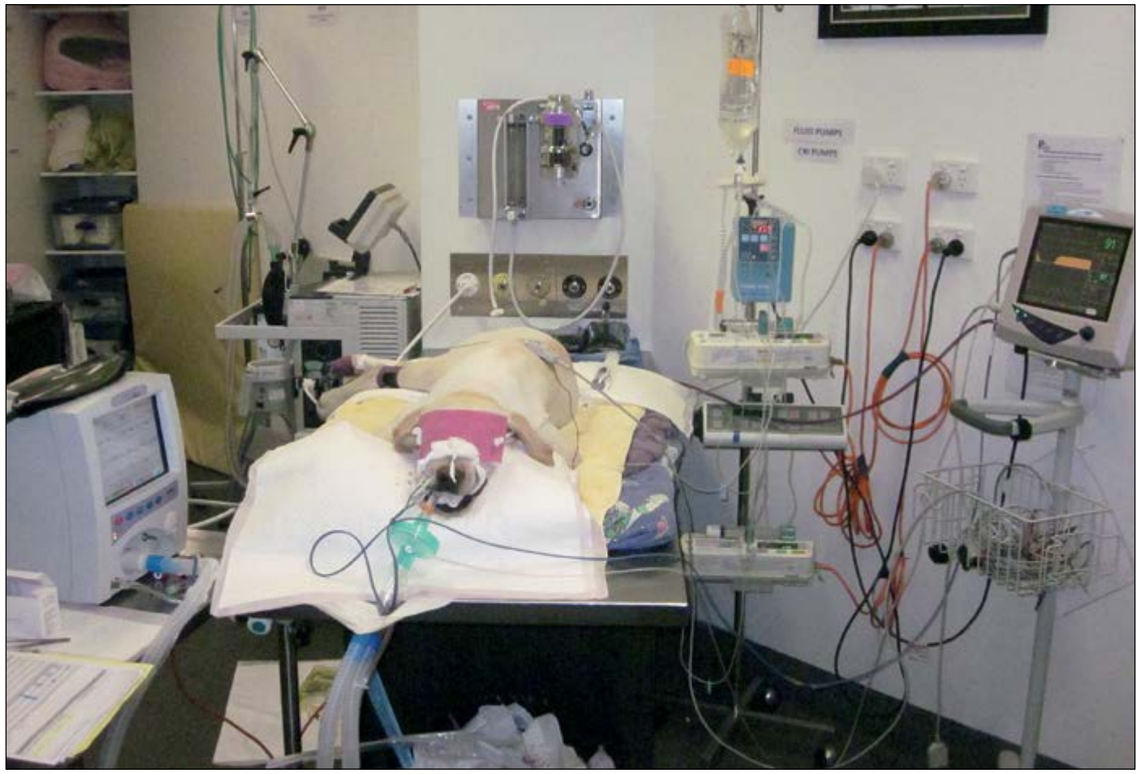


Figure 7. Labrador receiving mechanical ventilation for respiratory failure associated with tick paralysis.

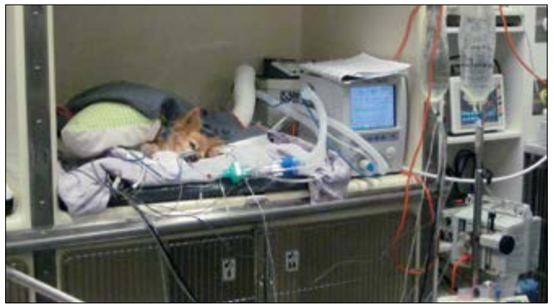


Figure 8. Chihuahua receiving mechanical ventilation for respiratory failure associated with tick paralysis

hypokalaemia) compound the original problem. Rather than repeating a medical therapy which has not worked the first time, deteriorating respiratory function requires a thorough diagnostic work-up (thoracic radiography) and appropriate supportive care (oxygen therapy or mechanical ventilation).

I do not use furosemide in the clinical management of tick paralysis in dogs or cats because I have seen it do more harm than good. Experimental and clinical studies are needed to further evaluate the contribution of pulmonary oedema to respiratory failure in tick paralysis patients, and the potential mechanisms by which it develops.

Despite the issues of diuretic treatment of patients with respiratory distress from tick paralysis, it is important to be mindful that dogs affected by tick paralysis develop a degree of pulmonary oedema, and determine the fluid therapy plan with this in mind. Bodyweight changes should be tracked closely, and central venous pressure can be monitored in the critical patient; both in order to avoid inadvertent fluid overload.

VENTILATION

Ventilation is indicated in 3 circumstances:

1. **Hypoventilation with severe hypercapnia: $PaCO_2$ >60mmHg.**
2. **Hypoxaemia not responding to oxygen therapy: PaO_2 <60mmHg** (ideally, an animal requiring FiO_2>0.6 to relieve hypoxaemia should be ventilated due to the increased risk of oxygen toxicity with high FiO_2).
3. **Unsustainable work of breathing:** If the patient looks like their breathing effort cannot be maintained for the medium term (at least 48 hours).

Ventilation Strategy

We ventilate using minimal pressures and FiO_2 necessary to maintain the patient just above the cut-offs listed. The minimally invasive settings are used because improving the patient beyond these end points will do no further good, and will increase the likelihood of ventilator induced lung injury. The description of our ventilation strategy follows:-

- **Pressure Control**
  We set an inspiratory pressure rather than a particular tidal volume. The patient's lungs may be severely injured, and therefore not cope with an arbitrary volume. We initiate ventilation with minimal pressure, and confirm whether we are achieving adequate ventilation by using the blood gas to evaluate the $PaCO_2$. Tidal volume is the dependant variable and will be different in every patient due to the compliance of their lungs.
- **Synchronised Intermittent Mandatory Ventilation**
  The timed breaths can be synchronised to the patient's respiratory effort to reduce fighting the ventilator, and also reduce the level of anaesthesia required to maintain ventilation. If the patient tries to breathe near the timing for the next mandatory breath, they will trigger the ventilator to deliver a pressure controlled breath.
- **Pressure Support**
  Sometimes the patient is breathing really rapidly – about 30 breaths per minute. If these all triggered ventilator breaths we would cause hyperventilation which will affect cerebral perfusion. Instead of triggering a breath each time, or having to breathe against the ventilator, pressure support supplements these breaths to a smaller magnitude than the mandated breaths and provides improved patient comfort.
- **Positive End Expiratory Pressure (PEEP)**
  We maintain some pressure in the ventilator circuit at the end of expiration. This keeps alveolar units open, which firstly prevents injury due to repeated opening and closing (shear injury) but also allows us to use lower inspiratory pressures and maintain adequate ventilation. Using higher PEEP and lower inspiratory pressure is a key component of lung protective ventilation strategies which are required to ventilate the sickest lungs.
- **Weaning from Mechanical Ventilation**
  This method of ventilation is basically a constant weaning process where we gradually reduce the ventilator breaths, pressure, and FiO_2 while monitoring the patient's blood gas analysis to ensure we do not drop below the minimally acceptable cut offs.

When the patient is down to minimal ventilator support, we disconnect them from the ventilator but maintain intra-tracheal oxygen therapy. When the patient has shown that they can maintain acceptable blood gas levels without working too hard off the machine, the next step is to wake them up.

- **Tracheostomy**
  Frequently a patient can cope without the ventilator, but de-compensates when extubated and changed to intra-nasal O_2 therapy. This is very suggestive of upper airway obstruction, but these patients do not typically exhibit loud stridor or obviously exaggerated inspiratory effort. I suspect these patients have relative laryngeal paralysis which is accompanied by swelling, and reduced functional reserve capacity to compensate for the problem. The lack of ability to generate negative thoracic pressure on inspiration prevents us hearing and seeing the 'classical' signs of upper airway obstruction.

  These animals can be maintained under heavy sedation or anaesthesia with an endotracheal tube, but the only option for waking them up is to place a tracheostomy tube. We often choose this step because we reason that the patient is more likely to have complications while under anaesthesia, the tube through the larynx may contribute to swelling of the larynx, and there is more cost associated with maintaining anaesthesia than the tracheostomy tube.

  A tube is placed surgically using a well described technique, and maintained until the patient is walking well when it is removed and monitored closely for 12-24 hours further.

  We have also used tracheostomy in some tick paralysis patients (especially brachycephalic animals) where we find that upper airway obstruction is the key cause of their initial dyspnoea. The typical course of events is that we anaesthetise the patient because they are not coping with sedation and nasal oxygen, but find that they maintain their blood gas without requiring artificial ventilation. This patient will sometimes have a tracheostomy tube placed soon after anaesthesia is induced and then woken up shortly afterward.

CONCLUSION

Tick paralysis is a life threatening illness with significant mortality of severe cases despite treatment. Many of the patients which survive this toxicity do so with standard treatment using tick antiserum. Improving the survival rate beyond its current level requires respiratory life support implemented at exactly the right time and performed with sufficient care and skill not to injure the patient further. This treatment modality requires close attention to monitoring the patient, and a standardised approach to determine the most effective way to support respiratory function.

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Critical care of cats with tick paralysis

Rob kindly wrote this 'supplementary' article at short notice in reply to CVE's request that he write a section specifically on cats for publication in this issue.

Introduction

My rationale for managing cats and dogs with tick paralysis is identical. In mildly affected patients, tick anti-serum (TAS) should be administered and embedded ticks removed. Severely affected patients require support of respiratory function until they recover from neuromuscular paralysis.

The differences in managing cats with severe tick paralysis are primarily because respiratory failure associated with the toxicity manifests differently in this species. There are also inherent differences in feline physiology and temperament which require a somewhat differing approach from canine patients.

Apart from emphasising the need to administer TAS as safely as possible, remove embedded ticks, and avoid any undue stress to the patient, I will omit the initial management protocol from this discussion. There are numerous similar protocols and I see no evidence to support one over another. Optimising the management severely affected patients receive after the administration of TAS may yield greater improvements in survival than debating the initial treatment protocol itself.

Let's assume that the patient we are dealing with is the one described by Dr Vera Pickering in C&T 268: *'Once severely affected with respiratory issues it is going to be hard to deal with and serum is only useful if there is still a tick on the animal pumping in toxin.'*

Pathophysiology of respiratory failure in cats with tick paralysis

Cats don't seem to aspirate like dogs with tick paralysis, and pulmonary parenchymal disease is rare. When they develop respiratory failure from tick paralysis it is usually due to hypoventilation, which can be from severe flaccid paralysis or due to laryngeal obstruction. While this form of respiratory failure is straightforward to manage if it is recognised, establishing the diagnosis of severe hypoventilation requires either blood gas analysis or end tidal CO_2 measurement, neither of which is used frequently in the monitoring of tick patients[1].

Hypoventilation on room air causes hypoxaemia which is easy to recognise: cyanosis, low oxygen saturation (pulse oximeter) or low PaO_2 (arterial blood gas analysis). Hypoventilation also causes respiratory acidosis (severe hypercapnia) which is harder to recognise because there are no 'classic' clinical signs. Although hypoventilation might be suspected because of low respiratory rate, or shallow thoracic excursions, it can only be confirmed by measuring end tidal CO_2 measurement or PCO_2 (venous or arterial).

The hypoxaemia in these cases is very responsive to oxygen therapy and increasing the FiO_2 to 30% (easily achievable with cage oxygen or nasal oxygen) will abolish hypoxaemia in most cases. The dramatic improvement in SaO_2 often gives the impression that the problem has been resolved but unfortunately this may not be the case. Respiratory acidosis remains unchanged and may lead to death of the patient.

While some patients will survive with elevated PCO_2, others will die suddenly from vasodilation and cellular dysfunction which occur due to severe acidosis. The highest PCO_2 measured at AES on the Gold Coast was 126 mmHg with pH of 6.8. The patient died shortly afterward. Respiratory acidosis needs to be recognised early to enable effective management.

Venous blood PCO_2 is only about 5mmHg higher than in arterial blood ($PaCO_2$). For this reason the venous blood gas analysis can be used to confirm hypoventilation even though it is not useful in determining the oxygenating ability of the lung. **We use venous blood gas in feline patients to monitor adequacy of ventilation and pulse oximetry to monitor for hypoxaemia because arterial blood gas collection is stressful to cats** and arterial catheters may only be maintained for short periods. ▶

Respiratory support strategy for cats

Assessment →Intervention →re-evaluation

Any patient with hypoxaemia (SPO_2 <90%) or unsustainable work of breathing receives oxygen therapy as the initial management strategy. In general these patients are placed in an ad-hoc oxygen cage made of a plastic tub, a Vetario ICU cage (as mentioned by Kim Kendall in C&T 268) or a glad wrapped hospital cage. I also recommend buying a cheap oxygen monitor (Pro O_2 oxygen analyser, Nuvair, Oxnard CA) to allow rational selection of flow rate. For example, our glad wrapped hospital cage (with no gaps) requires 2 x 15L/min oxygen flow lines to maintain FiO_2 about 50%.

After instituting oxygen therapy we reassess the work of breathing and collect a venous blood gas analysis. If PCO_2 is greater than 60 mmHg or the work of breathing is still unsustainable the next step is anaesthesia and intubation.

Anaesthesia is induced and a sterile endotracheal tube placed and cuffed (only inflated just sufficiently to seal the airway). Intra-tracheal oxygen is provided using an argyle feeding tube and flow rate of 50-100mL/kg/min. When a steady plane of anaesthesia has been achieved the patient parameters (work of breathing, PCO_2 and SPO_2) are re-assessed once more. If SPO_2 <90%, PCO_2 >60mmHg, or the work of breathing is judged to be unsustainable mechanical ventilation is indicated.

Mechanical Ventilation

The goals of mechanical ventilation have been described in the accompanying article. The ventilator merely maintains minimally acceptable blood gas levels until the patient can achieve them with a sustainable effort.

Summary of Intervention steps

1. Oxygen therapy→ 2. intubation → 3. mechanical ventilation→4. intubation→5. oxygen therapy→ 6. recovery

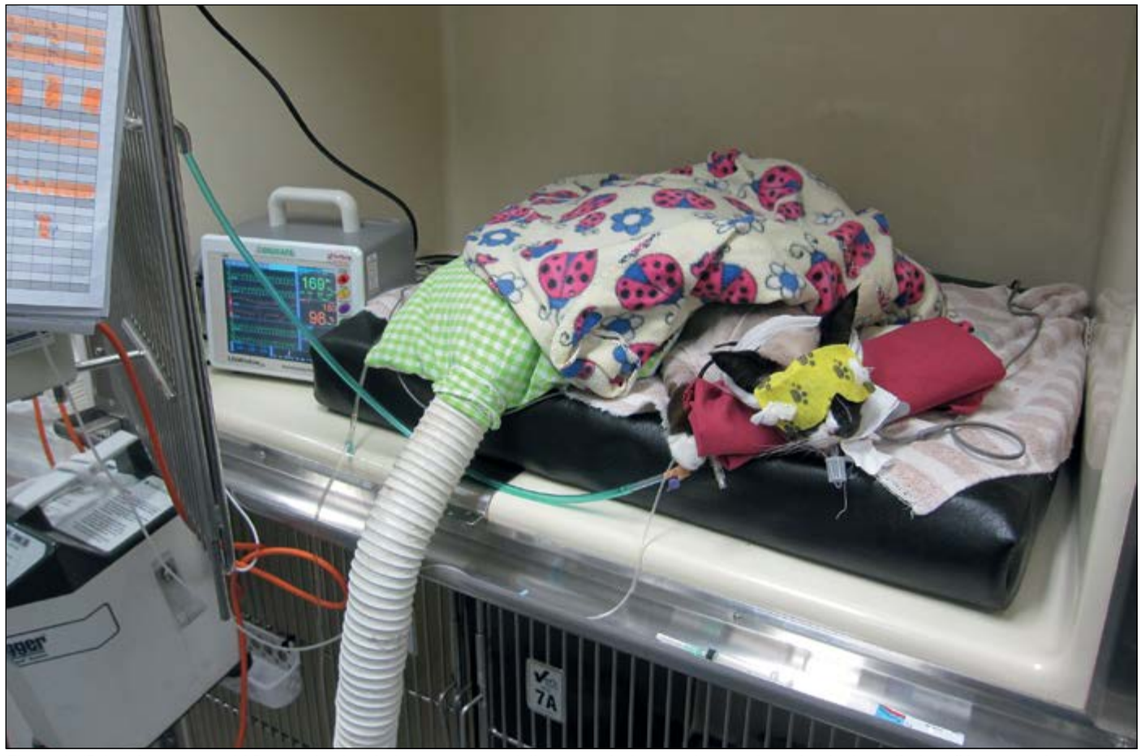

Figure 9: 'Prince', a 1-year-old Domestic shorthair, was successfully weaned after 144 hours of mechanical ventilation. (He was re-homed by an AES nurse after his owners failed to return to collect him!)

While there is no research to support the commonly held belief that TAS has no effect on bound toxin, this hypothesis explains why severely affected patients do not improve after administration of TAS. The recovery from neuromuscular paralysis may take a 'long time' (3-5 days +), the prognosis remains favourable if ventilation or intubation can be maintained. We often have patients which need to be put back on the ventilator several times, and sometimes cats do well intubated but decompensate quickly when extubated (presumably due to laryngeal paralysis). I avoid tracheostomy tubes in cats. The smaller tracheal lumen precludes placing a tube with a removable insert making cleaning more difficult, and also predisposes to blockage with airway secretions. When cats can't be extubated I generally maintain the ET tube and light anaesthesia until this is possible.

Conclusion

Cats have a good prognosis with appropriate supportive care. Our experience suggests that hypoventilation is the most common cause of respiratory failure (about 80% of cases, article submitted for publication), and that the prognosis for survival is good if support is continued long enough (83% from the same retrospective study). Some cats in our practice have required quite protracted ventilation, one of which was ventilated for 144 h and hospitalised for 360 h before discharge. The challenges in managing severely affected cats are recognising hypoventilation early enough to successfully intervene, then being able to continue supportive care long enough for recovery despite the inevitable financial limitations of owners and difficulties of staffing for 24 h patient care.

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Firstly, I'd like to thank Dr Gaschk, Dr Graham and the others who commented for raising a timely and topical point of discussion – what is the 'best' treatment for cats with tick paralysis? So little is published in the peer-reviewed literature regarding this topic, yet so much anecdotal evidence, and so many personal opinions are provided it is no doubt difficult and daunting for a recently graduated vet, or indeed vets moving from areas where paralysis ticks do not reside to areas where they do, faced with their first case of tick paralysis in the cat (or, indeed, the dog!).

In considering the treatment of feline tick paralysis, the topics which I feel are important to note are:-

1. As stated so frequently, 'cats are NOT small dogs'

We've all heard that more than enough times, but in tick paralysis (as in so many other diseases) it certainly rings true. In dogs, tick paralysis presents most commonly with an ascending lower motor neuron (LMN) paralysis, in severe cases involving the respiratory musculature, and occasionally presenting with local paralysis (e.g. facial nerve paralysis) but in cats this 'typical' ascending paralysis is, in my experience, the exception and not the norm. By far and away the majority of feline tick paralysis patients I have managed have presented for assessment of respiratory distress, with only mild hind limb or forelimb paresis. Those patients that have presented with paresis have infrequently followed an ascending pattern, with some cats having only a single limb affected, and some cats apparently having a descending LMN paresis. Typically, the respiratory distress exhibited by feline tick paralysis cases is characterised by an expiratory dyspnoea +/- an expiratory grunt.

2. Sedation

Wherever possible, I avoid acepromazine in tick paralysis patients. HOWEVER, I do think that provision of sedation is of great benefit to most cats with respiratory distress. My preference is to use butorphanol 0.2mg/kg IM or IV. I find this provides adequate sedation, provided that other stressors are kept to a minimum (i.e. place the patient in an oxygen box, attach a pulse oximeter to allow measurement of heart rate and SpO_2 with minimal handling, house the patient away from the sight and sound of dogs, minimise handling). As is so often the case with cats in respiratory distress, placing the patient in an oxygen-rich environment to allow time to settle can be the difference between life and death. Cats in respiratory distress are one stressful event away from respiratory arrest and should be handled with kid gloves. If all you can do for the first hour or two of treatment is to provide oxygen, then so be it. So often we feel the need to obtain IV access, perform full tick searches, full physical examinations, clip the patient, apply the Frontline spray and give the tick anti-serum (TAS) all within the first 5 minutes of entering the hospital, but in actual fact, the degree of stress caused by these events may be the cat's undoing.

3. Administration of TAS

TAS should be administered to all affected patients. My preference is to administer the TAS (usually 0.5-1mL/kg diluted 1:2 with 0.9%NaCl or sterile water for injection, warmed slowly to room temperature) via slow IV injection, using a syringe driver to administer over half an hour. Whilst the IP route can be used, administration via this route is no less likely to cause an adverse reaction than the IV route. Indeed, if an anaphylactic/anaphylactoid reaction does occur an IV injection can be stopped, appropriate therapy administered, and the TAS restarted at a slower rate or in a more dilute form. Absorption from the intra-peritoneal route cannot be 'stopped' once the drug is administered and may, in theory, provide a greater risk. I know many clinicians who have used either the IP or IV route and who will all swear by their own technique. In my experience, adverse reactions are rare. I tend not to pre-medicate with a corticosteroid or anti-histamine, but do have the 'crash cart' easy at hand should it be required. Additionally, a member of nursing staff is assigned to the patient for the duration of the administration of TAS and patient vital signs (including HR, RR, body temperature, CRT, mucous membrane colour and demeanour) are monitored every 5-10minutes for the duration of TAS administration. Placement of the IV catheter is achieved once the patient has been sedated with butorphanol and provided an oxygen-rich environment. Sometimes this takes a number of attempts (first round = clipping of the fur over the vein, application of EMLA cream, second round = preparation of the skin, etc) but in almost all cases it can be achieved. Provision of oxygen by face mask during the placement of the catheter can also help to avoid decompensation.

4. Supportive care

So frequently overlooked and yet so critical to patient wellbeing, supportive care is in my opinion a key contributor to the success or failure of management of feline tick paralysis cases. My recommendations are:-

a. Ensure adequate padding and bedding to prevent pressure sores in the non-ambulatory patient and minimise ventilation-perfusion mismatch in the lungs. Where possible keep the patient in sternal recumbency as this will facilitate improved ventilation. Nasal oxygen catheters can be placed if oxygen supplementation is required.

b. Use absorbent, plastic-backed pads ('puppy training pads' or 'incontinence pads') placed under the hind quarters to allow easy recognition of when a patient has urinated or defecated, and to keep bedding clean and dry. In the absence of an indwelling urinary catheter, these pads can be weighed (on paediatric or kitchen scales) to monitor urine output. In the event that a patient loses voluntary control of urination, frequent manual expression (3-4 times daily) or placement of an indwelling urinary catheter should be performed to avoid over-distension of the bladder.

c. Intravenous fluid therapy at maintenance rates and monitoring the 'ins and outs' should be performed. In patients with intercurrent disease e.g. chronic kidney disease, diabetes mellitus, it is important to remember that maintenance requirements are greater than in the patient without these conditions due to the underlying inherent polyuria. Twice daily monitoring of body weight can also help to determine whether we are maintaining adequate hydration in our patients. Remember, too, that cats become depleted of potassium rapidly when they are not eating, so supplementation of fluids with potassium is necessary.

d. Ensure adequate body temperature is maintained – avoid over- or under-heating the patient. ▶

e. If tolerated by the patient, passive movements of the limbs may assist in reducing limb stiffness.

f. Withhold food and water until a strong gag reflex is present.

g. Monitor closely for signs of aspiration pneumonia, which in my opinion is a major cause of morbidity in these patients. Whilst I don't routinely perform thoracic radiographs at the time of admission of patients with tick paralysis, an argument could be made that this should be routine to evaluate for evidence of aspiration or megaoesophagus.

5. Pulmonary oedema, ticks and frusemide

I cannot think of a single case of tick paralysis (in a dog or cat) that I have managed that has developed cardiogenic pulmonary oedema which did not already have underlying cardiac disease (and in those cases I suspect it was due to overzealous fluid administration). I am aware that pulmonary oedema secondary to tick paralysis is reported to occur but, like Dr Graham, I don't think we see this clinically. In cats, cardiogenic pulmonary oedema can assume many radiographic appearances, but my first concern in feline tick paralysis patients that have radiographic evidence of pulmonary disease is for aspiration pneumonia.

6. When to ventilate? Simple – sooner rather than later.

The reason for the majority of deaths of tick paralysis patients is ventilatory failure, which I think is likely secondary to exhaustion and likely hypercapnoea. If the respiratory difficulty is severe and/or not relieved by oxygen supplementation, mechanical ventilation should be considered. Ideally, arterial blood gas analysis should guide this decision (PaO_2 <60mmHg or $PaCO_2$>50mmHg = ventilate) but is frequently not available. Unfortunately, SpO_2 is often not useful as it is hypercapnoea and not hypoxaemia that is the major concern. Most frequently, we ventilate with IPPV, and only use PEEP if IPPV is not adequately maintaining oxygenation or appropriate CO_2 levels. CPAP is used by us during the recovery phase, usually once the patient is off the ventilator.

7. Clipping, tick searching and application of Frontline

Yes, yes and yes! Find and remove the tick when possible, clip the patient when safe to do so (use butorphanol if needed) and perform multiple tick searches a few hours apart (we usually have at least 4 tick searches, performed by different people).

8. Client expectations

As we all know, it is most frequently a lack of, or mis-, communication that leads to disappointed clients. In tick paralysis cases this seems to happen more times than in other diseases, perhaps due to the occasionally emergent nature of the condition. I always advise my clients at the time of admission of the patient that: Firstly, I expect the situation to get worse for the first 24hours after administration of the TAS and not better. Remember, the TAS only binds unbound toxin. The toxin that is already bound has to be released and degraded before the patient will improve. Secondly, I discuss the possibility of requiring ventilation and the costs associated with ventilation (in the order of $2000-3000 per day). This is especially important in patients that present already in an advanced stage of respiratory compromise. Thirdly, I discuss the expected period of hospitalisation and the costs associated with that hospitalisation. And whilst many may think this a bit dramatic, I also discuss the fact that tick paralysis can be a killer – it is certainly the minority that die from this disease, but it does happen. Murphy's Law states that the one time you don't mention this, it will happen. Don't be the one to make that mistake.

Comment No. 5 courtesy of

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I was interested to read Karina Graham's comments in C&T 2011. To my mind, there may be a few explanations for her observation that diuretic therapy for pulmonary oedema is rarely necessary for tick-paralysed dogs.

Firstly, there is a possibility that dogs are exposed to variable doses of the *I. holocyclus* cardiac toxin due to regional differences in its constituents. One electrophoretic study, which morphologically identified ticks from a small geographic area as *I. holocyclus*, found marked genetic diversity and concluded that *I. holocyclus* is a species complex. This supports other reports that the toxin of this tick varies with geographic origin, and perhaps between seasons and within season as climatic changes alters toxin production of the feeding tick such that the tick toxicosis may have some degree of regional distinctiveness. Having said that, here in SE QLD our emergency folk treat large numbers of tick-paralysed dogs and similarly avoid the routine use of diuretics.

Secondly, and perhaps more importantly, the cardiac toxin may rarely precipitate severe pulmonary oedema; and mild pulmonary oedema alone is not acutely life-threatening. For otherwise normal dogs with primary heart disease, mild pulmonary oedema becomes life-threatening because the underlying disease that precipitated it persists and the oedema worsens over a period of usually days and sometimes weeks. But for a dog with tick paralysis whose pulmonary capillary pressure is only transiently elevated, mild pulmonary oedema may spontaneously resolve when pulmonary venous pressures normalize without becoming life-threatening and without the need for diuretic therapy. Furthermore, mild pulmonary oedema may easily be missed in a tick-paralysed patient with dyspnoea attributable to a variable contribution of respiratory muscle paralysis, upper airway obstruction and aspiration pneumonia.

Thirdly, mechanical ventilation may provide sufficient non-specific therapy to obviate the need for diuretics in tick-paralysed dogs with transient mild, and even more moderate, pulmonary oedema. In human patients, acute and acute-on-chronic cardiogenic pulmonary oedema can be caused by a variety of transient insults including dietary indiscretion and medication non-compliance and treatment with mechanical ventilation is a well-recognised therapeutic approach which produces immediate preload, work of breathing and after load benefits and optimises arterial oxygen saturation until oedema resolves. Perhaps in tick-paralysed dogs, mechanical ventilation facilitates resolution of mild, or even moderate, pulmonary oedema that may go unidentified because arterial blood gas alterations with aspiration pneumonia and pulmonary oedema are comparable and radiographs may be insensitive for the detection of mild oedema and difficult to interpret in dyspnoeic dogs with the potential for more than 1 pulmonary insult.

Assessing a tick-paralysed dog's potential for pulmonary oedema is hugely challenging. Measurement of pulmonary capillary wedge pressure (PCWP) via pulmonary arterial catheter is the gold standard but the technique is invasive, insensitive to small changes, has poor repeatability and catheterisation itself has the potential to influence the pressures it aims to measure. In addition, for dogs with tick paralysis the measurements risk being influenced by variable changes in intra-thoracic pressure associated with respiratory muscle paralysis, and also with mechanical ventilation. Ilkiw floated pulmonary artery catheters in non-ventilated tick paralysed dogs in the mid-1970's. Data acquisition was complicated by technical problems and, if my memory serves me, the only 2 dogs with complete hemodynamic data sets had divergent values; and this was experimentally-induced tick paralysis where confounding variables were minimised! Estimating left atrial pressure via echocardiography utilising tissue Doppler imaging and colour m-mode (e.g. E:Ea, E:Vp or E: IVRT) is an alternative but is unlikely to have any predictive value for an individual dog and from a research standpoint would require large numbers to overcome inaccuracies associated with scanning dyspnoeic dogs exposed to variable doses of the cardiac toxin. Furthermore, acquisition of these echocardiography measurements requires specialised skills and in order to be detected, any toxin-induced change must be greater than the intra-observer variability of the echocardiographer.

In vivo, tick toxin prolongs relaxation of isometric left ventricular papillary muscle preparations before any K+ channel blockade effects, suggesting a toxin-induced alteration in calcium cycling/sensitivity. If this same direct effect occurs in vivo then isovolumic relaxation should be retarded elevating left ventricular diastolic pressures with the potential to precipitate cardiogenic pulmonary oedema. Ilkiw's catheterisation studies did involve micromanometer evaluation of the left ventricle but no indices of diastolic function were reported and repeated invasive measures of isovolumic relaxation (-dP/dt, Tau etc) would provide valuable information. Unfortunately, pulsed-wave Doppler evaluation of mitral inflow assesses only the filling phases of diastole and, as an indicator of left ventricular diastolic function, is too crude a technique to prove useful in dogs with tick paralysis where transmitral flow will be altered by many factors including variable loading conditions, heart rate, and altered intra-thoracic pressure. Rather, comprehensive echocardiography examination utilising newer techniques of tissue Doppler imaging (TDI) and speckle track imaging (STI) assessing all 4 diastolic phases is indicated to most appropriately assess diastolic function in tick-paralysed dogs.

Ah, so many challenges! So, my 2 cents worth; we need to minimise any biological toxin variability and isolate the cardiac portion of the toxin of genetically-confirmed *I. holocyclus* salivary glands. (Unfortunately, my memory is that University of Queensland's Paul Alewood ran HPLC for us over 10 years ago and found our extract to be frustratingly complex with multiple potentially toxic constituents – Rick Atwell may have more updated information). Administration of aliquots of this 'pure' and isolated cardiac toxin (or engineered analogues of it) to experimental dogs would then minimise variability in clinical change and avoid confounding effects of respiratory muscle paralysis, upper respiratory tract obstruction, aspiration pneumonia etc. Documentation of PCWP via pulmonary catheterisation, micromanometer catheterisation of the left heart, coupled with comprehensive advanced echocardiography imaging (including TDI and STI) and then we'd be in a lot better position to know what exactly it does to the canine heart in vivo. In the meantime, I'd agree with Karina that diuretics are unlikely to be life-saving if mild transient pulmonary oedema is as bad as it gets for most dogs. But, if coupled to ventilatory failure with respiratory muscle paralysis and/or aspiration pneumonia, even mild pulmonary oedema could be the straw that breaks the camel's back, and we are wise not to discount the role of diuretics.

Comment No. 6 courtesy of:

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So much energy has been used to disprove previous poorly-interpreted findings – most of these are based on errors of interpretation, too few cases, our innate bias, our inefficient memories (we recall mainly special 'good and bad' cases) and on a 'good idea from a person who should know' (but has no proof). Examples of such errors are: therapeutic hypothermia – no benefits (Fearnley PhD University of Queensland, Cooper PhD University of Sydney); systemic hypertension – (phenoxybenzamine) not proven; tick squeeze – impossible for toxin to escape; chemicals and tick irritability – no proof; extrapolating to cats from dog data – unscientific and illogical; most drugs and host survival – no proof except TAS and antibiotics (in severe cases); tick twist to R or L – both OK; head out – there is no head, ears, eyes etc – only lots of sophisticated receptors and a mouth (attached to a gut which is then replaced by a uterus); tick pluck danger (dogs) – no proof (See notes *AVP* 37:120 and March 2011) and a clinical lag and deterioration period – recent data (Merial 2008) suggest that most cases do not have a pronounced lag phase before a clinical response is seen. All the recent clinical tick and PhD data was completed in a large well equipped 24 hour hospital and with a 32 cage (controlled) research facility for induced-disease studies.

ECHO C&T

(C&T No. 5251: Reply No. 1 by Karina Graham - Sept 2012, Issue 268, pg 17)

If you use Echo on several (acute?) cases you would firstly need case control cases, simultaneously, to control for all the variables that exist in a practice setting (maybe not believed until one sees the errors of n = too few for any assumptions). Based on an ACVSc lecture on emergency clinic cases in SE Qld and N. NSW, clinical pulmonary oedema (PO) and overt CHF occurred in 5% (4+1) and 1%, respectively, of such a population (>100 cases, ACVSc, critical care section (Vet. Spec. Serv. [VSS] data Brisbane); approx. '08 – '09? – I have asked the author for this data to be published).

So (assuming 'several' is <10?) to do only several cases would be unlikely to prove (just on numbers alone) or disprove PO and CHF. There is also the prevalence of such cases in general tick toxicity (TT) cases versus 'sicker' or unusual TT cases, referred to emergency +/- medicine clinics. I well recall both my excitement and ignorance in getting a great result (PDA – attempted medical correction – ∝1978) in 1 animal but then finding totally, unacceptable side effects in the next 4 dogs (so an 80% side effect rate). So the echo data, even with in-flow and other technology, (interestingly tissue Doppler has not become a major diagnostic modality in the largest human cardiology group in Qld) is unlikely to see such cases, especially if they do not show obvious pulmonary (alveolar) signs (excluding toxin-induced hyperpnoea), thus enhancing the pre-test probability of 'seeing' such myocardial defects.

I am also always concerned that some operatives (not those necessarily referred to in the tick C&T articles) use echo without the full knowledge of the error rates involved, the abbreviated, human heart-based formulae, the difference in pulmonary vein (PV) anatomy of people and dogs, flow alignment (especially with only small anticipated changes in m/sec), the probable need for a breed-based normal range of values, the 'unable to hold breath' factor – meaning multiple recordings and averaging, to avoid the dynamic effect of intra-thoracic pressure changes (ITPC) on pulmonary venous flow. Compare ITPC from an obstructed upper respiratory tract (URT) or lower respiratory tract case to a simple slower breathing, normal TT, or to a 'fast shallow' breather ▶

– often the sign of the lung's last attempt to maintain O_2 levels and, as such, a critical prognostic indicator of failure, in both people and animals. (I am the first to say let those with more expertise do the echo and let me sort out all the first principle issues – I have only to watch a medical thoracic technician 'do their thing' to realise my own deficiency!)

There is no doubt that a diastole defect exists in the TT cases as studied (Campbell PhD University of Queensland, plus other published sources) – this has been seen (ACVSc – above) clinically (PO + CHF) and at necropsy of ICU cases, where pneumonia and PO were the 2 (almost equal) causes of death/severe-disease euthanasia (without any 'too quick' i/v barbiturate effects on the lung) (*Pers Comm* R. Webster (2012) VSS study – *AVJ* submission). It is seen rarely in general practice, but presents with a congested, DCM-like acuteness with an equal short-term recovery, the classical sign of TT-induced CHF. The heart is (in TT) unusual as even pre-tick TAS will not stop toxin attaching. It will detach over 1 – 2 days (Campbell PhD University of Queensland) and so the CHF abates with the loss of toxin. However this proposed hypothesis is unproved – it is suggested to be new receptor replacement as, like sharks' teeth, receptors are on a production line of regular replacement (hypothesis = maybe true but as of yet is unproven; theory = fact is proved, usually need twice proven and from – different sources, different modalities! But modern general use of the word 'hypothesis' seems to imply proof!).

Campbell's work also looked at 40 general TT dogs in which she showed there was no systemic hypertension (SH). SH was an understandable assumption (Ilkiw PhD University of Sydney) based on the use of Greyhounds and their unique blood pressure (BP) control (see *AmJ of Physicol* + GH and BP 1986? See author), with heavy intra-thoracic instrumentation, and the use in a small group of animals (without BP recording) of a so called anti-hypertensive (phenoxybenzamine), that is actually now known to induce reflex hypertension. In fact the systemic hypertension story did not gel as such a condition takes a long time to induce any lung disease (in people and animals), such as the reported TT pulmonary congestion (*Pers. Comm.* J. Neilson 1998). Hence an earlier *AVP* 'point of view' on the cardiac confusion of TT (*AVP* 24:155). It was this hypothesis that lead into the subsequent cardiology studies where Campbell documented evidence of cardiac dysfunction, based on PV/artery ratios (suggestive of PV hypertension as per the interpretation of similar changes in other cardiac diseases), echo changes, pharmacological and physiological *in vitro* preparations and toxicology studies (K+-Ca++ interplay). In addition to this was the work of Day and Schull (*AVP* 33:86) where 75 dogs and 16 cats with general TT were X-rayed (but not the more severe cases, where simply rolling from DV to lateral can cause respiratory arrest!). This was in some ways a 'double-blinded' study as the X-ray interpreter did not know case severity or that TT cases could have PV dilation. (She started to see PV dilation and wondered why, to (eventually) be told that was seen in TT and so expected). So there is an unusual TAS/TT/cardiac story, there is cardiac dysfunction (based on data from clinical to laboratory to echo to X-ray), it is reversible and short lived, can be covert (as cardiogenic PO) and will self-correct provided the PO is not too severe and the 'spasm' (i.e. the lack of relaxation) resolves (CC Blockers – Campbell – were used in pharm. preps. to relax the spasm of failed dystolic 'expansion/filling'). However the frequency of CHF could be low (<I%) based on existing frequency data. In a recent trial of 506 TT cases (Merial 2008 unpublished) no overt CHF was seen in cases, from North Qld to Victoria, but there were many PO cases. It is also possible that cardiac clinical expression is based on individual dog susceptibility and maybe locality (geographically) dependent and therefore may not be a feature of TT in selected areas (see DNA below).

So canine cardiac (diastolic) dysfunction does exist based on many data sources: echo, X-rays, clinical, (∝ 4% referral cases), PV dilation (∝ 30% general cases), pharmacol. and PCV toxicol. preparations, $K^+ \rightarrow Ca^{++}$, QT prolongation, arrhythmia, lung histology and noradrenalin levels (but not cardiac muscle structural 'enzyme' changes). It also may explain why ACP use has had no bearing on mortality – if systolic dysfunction was involved there would have been some correlation between ACP use and outcome, in > 1000 dog cases (Merial 2001 + 2008 data) i.e. the vasodilator, reduced afterload effect of ACP (in some cases with high doses) should have affected outcome. However with diastolic disease afterload reduction has no such effect (this is not direct proof just adds weight to a consistent story).

The toxin may also vary as does DNA data (Vankan DNA lab – University of Queensland – *Int. J. Parasit*.) – so ticks differ significantly over distance (makes sense with the hindrances to moving of tick populations and small (main host) environmental transit distances) and it is therefore possible that 'one body system TT cases' (see below) may have different prevalences in different areas e.g. Vic to North Qld (but there is no proof of this toxin variability – just a valid assumption on the basis of other toxins' variations with DNA variability). The toxin will most likely be a cocktail (when analyses are completely done) and the proportions or percentage of various components may be the varying factor re DNA and geography. The 'disease' does vary between areas (Merial unpublished) so it is logical to consider that different toxin profiles may exist in different areas.

It also seems that TT affects some individual animals in unusual ways – some just get 1 system disease – e.g. CHF, oesophageal dilation (Malik, n = 3, *AVP*), 'asthma-like' signs (in cats) or have delayed recovery or residual issues e.g. urethral obstruction (usually male), weakness (some data to say heavier dogs have prolonged morbidity [Merial, n = 506, 2008]), persistent regurgitation, or blinking, tear and pupil issues. On the other hand recovery and discharge is not a full recovery e.g. dogs go home with residual oesophageal dilation, repeatable exercise-induced weakness (Atwell unpublished 2007) and rarely, 'found dead' cases, believed to be due to residual QT issues and induced terminal ventricular trachycardia (Campbell PhD).

So far as the data that are available (e.g. Day & Schull) cats do not get cardiac disease nor, due to species-specific anatomy, the obvious megaeosaphagus seen in dogs, with and without clinical signs, both in hospital and after discharge (*AVP* 31:75). TT cases have been seen (in cats) with other cardiac disease e.g. HCM (*AVP*; 34:32) but the lesion was well established and did not resolve. The only drugs proved to work (Merial 2008, n = 506) are TAS (all brands were of equal effect), and antibiotics (especially in NMJ 4th stage, high respiratory VAS). No other drug use (as recorded by 42 practices) affected mortality. Selective drug use is still indicated e.g. anxiety and anti-anxiety therapy (cats especially), diuretics and overt PO (but only when they are indicated and only while the indicated signs exist). TAS reactions (Schull PhD University of Queensland) are not stopped by any drugs. It is not seen if TAS is given by slow i/v infusion over 20 mins (but do not delay starting TAS as toxin needs to be neutralised before receptor attachment (upsetting K+ channel functions) and (possibly more important?) before vascular transit to the TAS-inaccessible, extra vascular space. Rapid TAS use (i/v) will induce clinically evident reactions in over 80% of dogs but anaphylaxis occurs much less (no valid data to give a percentage). Although (in theory) more cats than dogs should be affected by canine sera, feline anaphylaxis is not common either. However (with our biased psychology) we recall every shock experience, especially if it had a poor outcome. The TAS reaction is easily induced and Type I needs to be proved to be sure of the cause of an animal's reaction with the use of TAS. In general TAS reactivity is more likely and is associated with excess TAS/time and this particular animal's susceptibility (to sera of any sort!)

However, unlike TAS, a very severe Type I event will not be stopped by any drug (cats- Litster - PhD University of Queensland; people – *Proc. Eur. Res. Society* 2011 Portugal) and those that survive may have 'never been meant to die'; the volume of human (can we extrapolate?) anaphylactic cellular secretions equals the volume of the spleen and severe shock is simply too advanced by the time the secondary responses (hair, GIT, HR, RR etc) to profound canine hypotension are evident. This is not saying not to resuscitate (fluids, O_2 etc) but it does say that some Type I cases will not recover and with good reason (based on very recent human mast cell studies).

Re Type I and therapy there are few data on what works or not works except the PhD of Litster in cats. Species-specific, anti Type I mediator therapy should help but how often and when it is to be given, and how much is needed to successfully neutralise the acute shock is not known (i.e. based on appropriate data). Based on the only clinical cat data (C&T report, Brisbane Cat Clinics) available, the suspected Type I rate is low (about 1%) with TAS use (n = 200+, i/v and i/p TAS groups). (See below).

Type I outcomes are based on the amount, the rate and route of allergenic exposure and on the host cellular response, especially mast cell release (MCR); this can be used to both diagnose (Type I) and gauge the level of MCR, by testing for MC elastase levels released with the other MC mediators.

In some areas ticks can be seen all year round (e.g. Upper Brookfield, Brisbane). This is understandable as survival is dependent on the micro climate of the tick. So avoiding dryness (they are very susceptible to dehydration) and excess wetness (fungal growth on 'cuticle') will ensure a longer survival while waiting for a host. However older ticks are less active and feed with less enthusiasm compared to recently caught, younger (e.g. not stored) ticks (based on multiple, long-stored, wild-caught ticks – they are visibly less viable). Toxin is not secreted if the tick does not feed – there is a repeating cycle – salivate-feed-expand, salivate-feed-expand etc so a large (normal) tick has salivated (intoxicated), repeatedly, to get that much blood.

Based on the assessment of > 1000 detailed cases (Merial 2001, 2008) it appears that clinical disease and outcome are based on the toxin supply rate (not just tick numbers, size etc), the host immunity (e.g. large viable normal tick on mild, recovering case suggests some host immunity), and on host susceptibility (K+ - Ca++ toxin effects) and disease expression (which organs and severity). One of the most lethal factors is animal fatigue (well established in people with similar disease, Fearnley PhD University of Queensland) and we may well make this worse by excessive handling and chronic disturbance in hospital, all increasing O_2 needs in an already compromised respiratory system (paresis/paralysis, acid base issues, dehydration, airway obstructive factors, 1^O to (?) 2^O fatigue, pulmonary oedema, aspiration pneumonia, hyper and hypothermia, poor chest and neck positioning, anxiety, hypoxaemia, hypercapnea). Anything that increases the respiratory effort (activity or anxiety or guarded responsiveness) is also increasing O_2 needs, using up already reduced muscle reserves and therefore more closely approaching fatigue for these overworked, faster recycling, respiratory muscle units. (see later)

Early TT in cats (based on n = 12 caged trial animals) is seen as an unusual weakness – cats will (repeatedly) walk slowly, 3 - 5 paces, then squat (= fatigue) with pelvis well under a more straightened thorax-abdomen axis. Their cry is altered, they may be doll test positive (forced chest compression with induced URT stridor, Atwell tick seminars, 2001) and they will have wheezes, representative of distal obstructive airway disease (DOAD) (not 'asthma' which, in natural (?) cases does not comply with the airway reversibility factor (there are 4 such factors of human asthma). In fact most feline asthma papers are from one laboratory and author, (with induced, allergic DOAD). This feline TT wheeze is not reversible with standard dilator products (atropine, beta 2 adrenoreceptor agonists, adrenaline – small pilot trial data only) based on altered breathing and/wheeze intensity.

Signs relate to bound toxin (i.e. $K^+ – Ca^{++}$) and the rate of clinical onset is determined by the rate of toxin passing out the vascular compartment and into tissue. In C&T articles I'm not sure what acute (sudden, severe?) tick paralyses implies as the cases are better classified with the NMJ 1 - 4 status and using VAS (of 42 vets/506 cases); for overall toxicity, respiratory signs and paralysis. Compared to the old respiratory score (now disproved twice, > 1000 cases) which is neither progressive or prognostic (as so many respiratory signs are not uniquely disease specific), the 3 VAS systems correlate well with the neuro muscular junction (NMJ) signs re disease progression and prognosis and are highly predictive of outcome, as are some very specific individual clinical signs (Merial data 2001, 2008; CE series – '09,'10,'11).

As it has been clearly stated re diuretics in dogs (not cats), they are not routinely used and only used once, and then wait and see if crackles are determined to be due to PO and it is then significantly affecting the TT case. It is more likely to occur earlier (in the case progression) whereas the other cause of crackles, pneumonia, is more likely to be later in the TT case, especially if there are those loud URT noises* with attempts to clear the pharynx-larynx areas (*that terrible loud groan, come retch-like sound as the animal uses its limited clearance mechanism to move thick tenacious material from the extreme sensitivity of the larynx/pharynx 'choke' URT areas). I am not aware of the PO occurrence with URT obstruction i.e. rate of PO and its extent, and with what degree of URT obstruction in TT or for other URT causes i.e. the frequency of association and the level of respiratory tract obstruction (URTO) required to induce PO. Dehydration needs to be assessed prior to diuretic use but, if the lung is wet its decongestion should take precedence! However, the use of diuretics in pneumonia are definitely contra-indicated so crackles have to be disease-verified. The only indicated heart (basic) therapy would be diuretics and maybe CCB (Verapamil®) but only in confirmed CHF cases. (This is based on little hard data i.e. a small series of cases in various practices as reported to the author). The data on anti-arrhythmic therapy, if truly needed, has again low case numbers and using the drugs normally used (i.e. there are no TT drug trials).

Frontline® can be used directly on ticks (small pilot trial AVP) but the kill rate is slow. Almost any dewaxing agent (methylated spirits, kerosene, turpentine, alcohol) will kill ticks as they are so susceptible to dehydration – the dewaxing leads to acute H_2O loss. I'm not aware of any trials that say one product is associated with less severe TT but (as TT is so unpredictable, in both presentation and progression) it is so easy to misinterpret results or support a personal bias without a controlled, randomised, blinded, prospective large number trial). (See data below).

Periactin® probably has a role in cats re clot breakdown (see original aortic embolism *JSAP* 1980's) and perhaps anaphylaxis, but Litster's PhD clearly showed if Type I is too severe (i.e. rate and route of allergen exposure and mast cell release etc) no drugs will reverse (so late) in this acute disorder. Even having the drugs prior to induced Type I does not stop progression as the mediator volumes are probably just too large.

While weaning off ventilation can be associated with respiratory muscle 'non-use' issues and rapid (comparatively) 'functional-atrophy', the most common form of fatigue is the disease itself – the progressive loss of motor units in TT and the faster (and so necessary) recycling of fewer and fewer, not-completely-rested motor units leads to primary physiological fatigue. (Secondary histologically-evident fatigue damage can occur in neuro muscular diseases). Primary fatigue can be restored to some extent e.g. by the rest (Fearnley PhD) induced by very light anaesthesia (low dose barbiturate) with appropriate support (intubation, O_2, lung appraisal, hydration status etc). ▶

'Salivation' in TT has not been analysed to see if it is a truly increased salivation rate and/or reduced capacity to clear material by swallowing, as in some other NM disorders. Similarly the cause of corneal lesions and whether it is reduced tear flow, poor or absent blink or exposure to bedding (or a combination) has not been resolved (See *AVP* March 2011). (Saliva volume could also be directly affected by TT and by some drugs!)

Local paralysis (tick above eye and unilateral loss of 'blink') is a different pathophysiology to general TT cases. In people there can be a similar local-only paresis/paralysis. The implication is that in such cases the tick toxin supply rate is low and/or it is trapped locally by some immune process. Why such local paralysis occurs or takes so long to recover is not known but is seen in other localised NM diseases. (Editor's Note: Dogs, cats and humans with local tetanus can take longer to recover than cases with generalised tetanus). It is therefore unlikely that systemic TAS would alter this sort of uniquely localised case, especially once the toxin is bound. It (i.e. single site) bears some resemblance to single organ disease e.g. CHF, oesophagus, urethra etc but both hypotheses have to be verified by detecting toxin levels, seeing local toxin attachment/release, 'toxin' receptor density in different dogs/organs and perhaps different toxin components in different geographic areas. So the perceived 'no PO in Sydney cases' (needs a n = 100 case, lung X-rays – Echo study e.g. as Day and Schull *AVP* 38:86) could be explained by a low level of PO (i.e. you need to look for it in lots of cases?) or a local tick toxin that does not act as does tick toxin cases in SE Qld and northern NSW, where both cases and necropsy results confirm PO and where controlled studies have proved a wide range of cardiovascular issues (in dogs and less so in N. Qld flying foxes).

Fluid overload can occur easily in cats (University of Georgia PhD studies 1980) so PO in cats could be i/v fluid excess (?) and/or URT obstruction (but as above what degree of URTO is needed and for how long before PO occurs, and in what percentage of URTO cat cases). In an (n = 12) cat intoxication study (with 5 ticks/cat) there was no evidence of cardiac disease or of cardiogenic PO but NMJ 4 cases were induced and treated. (None of the recent University of Queensland PhD data have involved cats so nearly all recent data are dog based). The most dramatic event in the cats was the early URT anxiety (usually with paw tapping?) and profound paralysis, with early onset cat-unique paresis (see above – gait etc) and DOAD ('asthma'). I would guess in an average tick season some cats are thought to be 'asthmatic' and return the next day with a more detectable, more expressed TT.

However, both dogs (and cats) with 5 - 10 ticks still take 3 days to induce signs (in multiple studies [see *AVP* March 2011] with controlled environments with caged animals). This appears to be the time taken (not proved for *I. holocyclus* but for other *Ixodes*) for gene up-regulation to produce salivary volume and proteins; tick feeding capacity; gut expansion, enzymes and absorption; Fe transit etc. Once in place feeding and saliva-toxin secretion continues, in a pulsed manner, with body expansion based on blood absorption from a feeding site, which is kept viable by various anti-platelet, anti-clot, anti-inflammatory, local anaesthetic etc agents in tick saliva. Lack of 'growth' indicates poor feeding (blood intake for some reason) and by association, poor toxin supply contained in the lower saliva flow rate. Any product that does not kill a tick but makes it ill, (with the associated poor growth rate), should reduce toxin supply rate as the lowered viability makes it less able to feed properly. (See viability above).

Mortality in 506 cases (Vic to N. Qld) was 6% (10% to 0%) from 42 practices with their own tick protocol (Merial 2008). Other data in approx. 100 cases at an emergency clinic was 4%. Mortality is based on many factors and could be expected to vary between area, seasons, individual case loads and levels of practice competence – especially at seeing what TT aspects are present in each case and minimal interventional nursing with appropriate referral, especially for ventilation medicine.

For cats and TAS (i/v, i/p) – only one study (n= about 200) reported in C & T where there was no statistical difference for each process, based on a low mortality (approx. 1%). The cats' unique fluid path from the abdomen probably means TAS i/p gets into i/v compartment reasonably quickly (faster than the dog as per RSCHF and the fluid handling of cats and dogs [Atwell 2012 submitted]). However, in such a trial the same batch of the same TAS should be used and probably n = 300+ cases would be needed as the expected death rate (anaphylaxis not TAS reaction) would be of the 1% order. Books will speak of using various protective agents (see earlier) but there is little data to support what we are told to do. Litster (PhD) clearly showed in cats that no so-called protectants worked in an experimental anaphylaxis study. It would therefore seem wise to avoid 'insult', by i/p and not i/v, and slowly rather than faster! (but for both it is probably only a 1% factor!).

Until we can measure TAS protective antibody levels and levels of toxin in each case, a precise TAS dose rate is impossible to know (as no large scale trial has been published on dose re severity or dose re species or dose re toxin or host weight – there is an essay on this topic produced by Merial). TAS effective 'clinical' half-life is not published and the levels of extra vascular (not accessible to TAS) pre-bound toxin are not known. However, the possible levels in this extravascular area help to explain sudden deterioration of cases, especially in seasons when ticks are very viable and feed well. The more severe the case, the more of the toxin is bound and not TAS accessible, explaining why 'double' TAS dosing or a 2nd TAS dose (e.g. on day 1) does not alter outcome (as most is bound). Similarly, TAS at the tick site has no effect (as the i/v TAS will neutralise newly arrived site-toxin) (*AVJ* 2001). The toxin flow rate (to i/v) is not known but everything says TAS should be used as soon as possible i.e. straight away to help avoid more 'spill' into the extra vascular space, so avoiding possible neutralisation. The more unbound toxin escaping to this extra-vascular space the more the case will deteriorate (Gympie cases, 2010 season).

I would gladly further support the facts above with the more detailed sources. The sequence was in response to several C&T articles provided by the Editor. Other related areas may need to be covered, as well, but the thrust of the above was based on these Q & A areas of TT. I should add that I have tried to ensure appropriate authorship to the above data. I have mentioned only one acaricide based on a direct question of that product and on having performed the small study to confirm its effect. I am indebted to the funding from Merial to allow me (and so this profession) to gain so much insight into the practice-based study of over 1000 TT cases (dogs) in 2001 and 2008, involving (with much personal gratitude) over 80 practitioners and their staff and clients.

Re therapeutics and TT – while we all have our own ideas we probably need to become more evidence-based orientated and need to be able to support (from various sources) our judgements re drugs and disease evaluation, species cross-over issues and the complicated severity of some TT cases. Even if we have seen (without proof) that a certain drug(s) seems to help a case, it is illogical to assume such drug efficacy without a single 'one drug at a time' trial, with similar TT cases in a cross-over study – to try to avoid the complexity of variable case types and multi-therapy while trying to say that this one particular drug worked! Everything comes back to numbers (of cases) and single drug-use studies to verify effectiveness in a wide range and large number of TT cases, before we can be sure of our (unique) ideas!

Editor's Note: *Part 1 & 2 of this roundtable discussion have mainly focussed on Tick Paralysis Treatment and we invite readers' replies and comments on the articles published. As well, in our next issues, we are keen to publish on Tick Paralysis Prevention and therefore invite submissions from readers/members on effective prevention strategies that work for both cats and/or dogs. We welcome contributions from all readers, not just CVE Members. Please email articles to* ***cve.publications@sydney.edu.au***

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