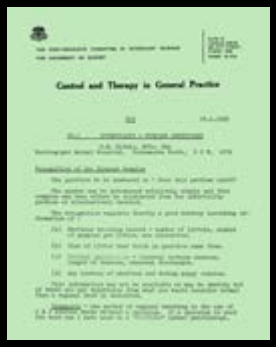
C&T

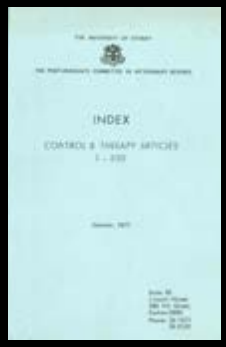
SPECIAL ANNIVERSARY ISSUE

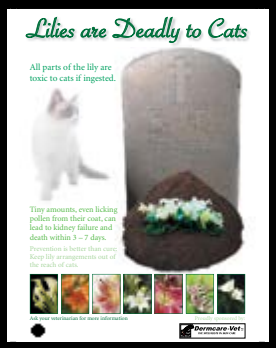
Issue 294 | March 2019

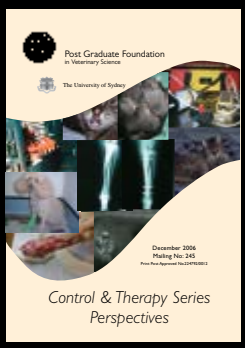
Centre for Veterinary Education

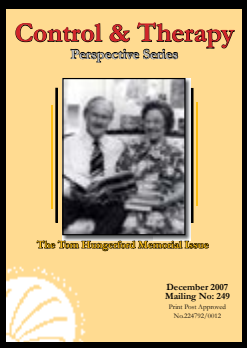
Control & Therapy Series

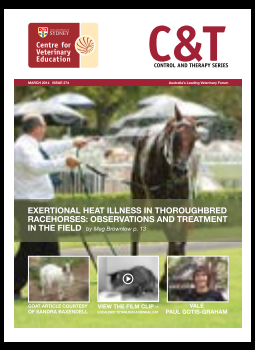












50 YEARS OF VETERINARY ALTRUISM IN ACTION

C&T

Issue 294 | March 2019

Control & Therapy Series

Australia's Leading Veterinarian Forum

Centre for Veterinary Education

PUBLISHER
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3,000 – CAB audited as at Sep. 2018

Audited Media Association of Australia cab

Contents

From the director

2019 has not started well for many of our colleagues, with severe flooding in far North Queensland, extensive bushfires, heat waves and a worsening drought affecting much of the continent. Signs of a slowing economy are starting to be acknowledged by even the most optimistic forecasters, which means that on top of the natural disasters there is little to look forward to in the immediate future.

Many younger Australians have never lived through a major economic downturn, given that we did not suffer during the GFC nearly as badly as much of the western world. Unfortunately, a stagnant or shrinking economy has a negative impact on all types of veterinary practice, on top of the adversity imposed by climatic aberrations. When times are tough, discretionary spending is reduced and unfortunately this can impact on many routine visits to the veterinarian.

Having been through this situation in regional mixed practice several times during my years as a principal, it was soon realised that rather than sitting around waiting for it to rain and for the economy to pick up, time can better be spent working on aspects of the business which are often neglected when everyone is flat out. Time can be well spent on improving your HR and business systems, stock control, assessing the usefulness and lifespan of older equipment, getting essential repairs and maintenance completed, taking overdue leave and analysing your client relationships, so that when things start to pick up, you and your team are ready and rearing to go.

Another factor adding insult to injury is the impact of a shortage of veterinary locums across the country. Despite graduating more vets each year than the UK, we have the paradox of a workforce shortage, which means that many practices are finding it hard to send vets on CPD courses or to accommodate annual leave requests. Thankfully, with our affordable TimeOnline courses, the CVE offers a broad range of subjects which can be taken without leaving the practice and enable people to stay up to date with the latest information.

For those who can get away and long for some in depth learning, the June Melbourne conference will set you and your practice up to handle emergencies both medically and surgically, with two entertaining and very informative speakers from the UK—the husband and wife team of Sophie Adamantos and Mickey Tivers, backed up by high calibre local speakers. See the CVE website and emails for further information. This course looks like being a corker!

The feedback survey after the recent cardio-respiratory conference held in Sydney was extremely positive, which was self-evident during the conference, so for those of you CVE members who were unable to attend, look out for the recordings of the lectures as they become available in the CVE video library. As usual we asked for suggestions of future conference topics, and there were numerous requests for practical neurology. Guess what? The CVE holiday destination conference will provide exactly that, with 4 days of practical neurology to be held in Cairns in September.

Hopefully I will see you at one of these conferences, in the meantime we can only hope for the end of the drought and the speedy recovery for those practices impacted so severely by the recent massive floods in Townsville and surrounds.

Hugh White
Director

Calendar

Conference | **Seminars**

MELBOURNE

Critical Care in Internal Medicine & Surgery Conference
Mon 17 - Thu 20 June 2019 +
Masterclass Fri 21 June 2019

CAIRNS

Neurology Conference
Mon 23 - Fri 27 September 2019

SYDNEY

One Welfare Conference II
Mon 14 – Tue 15 Oct 2019 +

Four-Words, Forwards! (Mental Health Workshop)
Wed 16 Oct 2019

Clinical Pathology
Wed 5 June 2019

CANBERRA

Cat Catch-Up: Update Your Feline Medicine Skills
Sat 25 May 2019

BRISBANE

Oncology Seminar
Fri 30 August 2019

Theory & Practice

SYDNEY

Dentistry
Fri 12 – Sat 13 April 2019

Ophthalmology
Fri 25 – Sat 26 October 2019

Workshops

SYDNEY

Basic Anaesthesia
Sat 27 April 2019

Practical Ultrasound Intensive Short Course
3,4,6,7 June 2019

Basic Echocardiography
Sat 22 June 2019

Advanced Echocardiography
Sun 23 June 2019

Hip & Stifle
Sat 27 July 2019

Bone Plating
Sun 28 July 2019

Stress Free Surgery Workshop
Sat 24 August 2019

Rabbit & Rodent Dentistry
Sat 19 October 2019

2019

March

Su	Mo	Tu	We	Th	Fr	Sa
31					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
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April

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28	29	30				

May

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June

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July

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14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30	31			

August

Su	Mo	Tu	We	Th	Fr	Sa
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4	5	6	7	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29	30	31

Workshops

Avian Orthopaedics & Beak Repair
20 October 2019
Canine Soft Tissue Surgery
Sat 26 October 2019

Feline Soft Tissue Surgery Made Easy
Sun 27 October 2019

Advanced Anaesthesia
Sat 2 November 2019

Soft Tissue I Workshop
Sat 2 November 2019
Hip & Stifle

TOWNSVILLE

Back to Basics: Diagnostic Ultrasound
Sat 10 – Sun 11 August 2019
Sat 9 November 2019

TimeOnline

Backyard Chickens
Mon 1 - Sun 28 April 2019

Demystifying ECGs
Mon 8 April - Sun 5 May 2019

Essential Wellbeing & Coping Skills for Veterinarians
Mon 15 April - Sun 12 May 2019

Respiratory Failure
Tue 23 April - Mon 20 May 2019

Small Animal Nutrition
Mon 6 May - Sun 2 June 2019

Feline Emergencies
Mon 13 May - Sun 9 June 2019

Practical Ophthalmology
Mon 20 May - Sun 16 June 2019

Avian Anaesthesia & Analgesia
Mon 3 - Sun 30 June 2019

Equine Lameness
Mon 3 June - Sun 14 July 2019

Shock & Fluid Therapy
Mon 17 June - Sun 14 July 2019

Goat Medicine & Husbandry
Mon 8 July - Sun 4 August 2019

Canine Abdominal Ultrasound
Mon 15 July - Sun 11 August 2019

Rabbits & Rodents
Mon 12 Aug – Sun 8 Sept

Heart Murmurs & Coughing in Dogs & Cats
Mon 5 August - Sun 1 Sept 2019

PodcastPLUS

Clinical Reasoning in Veterinary Neurology
Thu 28 March - Thu 4 April 2019

Dealing with the Emergency Cat
Thu 25 April - Thu 2 May 2019

Avian Radiology
Thu 30 May - Thu 6 June 2019

Update on Drugs in Behaviour Medicine
Thu 27 June - Thu 4 July 2019

Salmonella - Cast it Out!
Thu 25 July - Thu 1 August 2019

Adrenal Diseases in Cats
Thu 29 August - Thu 5 Sept 2019

Calendar Key

- Conference
- Seminar
- Workshop
- TimeOnline
- PodcastPLUS
- Theory & Practice

THANK YOU TO ALL CONTRIBUTORS

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

Terry King, Veterinary Specialist Services, QLD

Follow Tom Hungerford's 'goanna track to success'...

Since its establishment in 1969, the *C&T Series* has been a unique forum:

> ***"... 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..."***
>
> Tom Hungerford

Thank you to all contributors

...and more C&T articles and Perspectives are needed.

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

Winners

MAJOR PRIZE WINNER

Control of feline dermatophytosis in a shelter with use of mycoparasite Pythium oligandrum *& vaccine*
Martina Načeradská p46

BEST VISUALS

A selection of infectious diseases of companion animals housed in shelters (with a focus on cats)
Mark Westman, Simone Maher & Richard Malik p54

CVE PUBLICATION PRIZE WINNERS

'How does a vet find a new disease that no one has seen before?' Marshall Thornton p15

*A Sooty Owl (*Tyto tenebricosa*) with aspergillosis & A Powerful Owl (*Ninox strenua*) with* Haemoproteus *infection* Charlie Carter p7

Anaesthetic protocols for dogs on anti-anxiety medications Gretta Howard p24

Cryptic presentation of nasopharyngeal lymphoma in a cat Moira van Dorsselaer p33

PRIZES

Major Winner: a year's free CVE Membership
Best Visuals: Digital video or DVD of your choice
Winner: A CVE proceedings

Visit vetbookshop.com to peruse our list of titles.

Contents

A study investigating the use of the hand-held infrared thermometer to identify horses with high skin surface temperatures as an early detection method for exertional heat illness in thoroughbred racehorses: a study at the racetrack

M A Brownlow & T Cole

Racing Australia

e. mbro8605@uni.sydney.edu.au

C&T No. 5725

Abstract: Thoroughbred racehorses perform exercise at maximal intensities and typically become hyperthermic due to the huge amount of metabolic heat that is produced during racing. Despite a thermoregulatory mechanism which performs efficiently in most circumstances, certain weather conditions can compromise the horse's ability to dissipate heat.

Early detection of exertional heat illness (EHI) in thoroughbred racehorses can be difficult because signs are often vague and the measurement of rectal temperature as an indicator of hyperthermia is not practical. Best practice in the treatment of EHI in horses, as in humans, centres on early detection, rapid assessment and aggressive cooling. Research in humans has shown that EHI is manageable when recognized early and appropriate treatment provided.

The aim of this study was to investigate infrared measurement of skin surface temperature as an aid in the early detection of EHI. A high skin surface temperature (HSST) >39°C in the immediate post-race period reflects the combined interaction of exercise-related metabolic heat production, physiological adjustments such as increased skin blood flow and sweating, together with environmental influences specifically high ambient temperature. HSST is associated with a reduced core-to-skin temperature difference, which retards heat transfer from the deep body tissues to the body shell and therefore hinders heat dissipation. Identification of horses with HSSTs can prioritize them for rapid cooling and curtail possible progression to EHI.

Click the link below to read the full article as space constraints prevent us from publishing the article in full in this print edition. For new readers who have missed Meg's excellent series, all articles are available in CVeLibrary.

eBook download:
Read Meg's full article here.

Contents

Thank You!

- To all the veterinarians, veterinary nurses, veterinary technicians and allied professionals who have contributed articles to this unique and much-loved quarterly veterinary forum since its inception in 1969 until the present day.
- To those vets who ensured that the C&T lived on by supporting the production of this valuable publication through their membership dues over the last 50 years.
- And finally, to Dr Tom Hungerford, the inspiration behind the C&T Series who also coined the term '*Follow the goanna track to success*' meaning once you master a skill, don't rest on your laurels but proceed to tackle the next—the philosophy which underpins the CVE's provision of continuing professional development.

Innovative Vet—Keen Educator

Tom Hungerford was a force of nature and an inspiration worldwide. As his obituary in the *Sydney Morning Herald* stated:

'By the time he retired in 1987, his five-day courses led by eminent veterinarians, workshops and various publications were used in 53 countries. He started a revolution in continuing veterinary education, which won international recognition.'

To read the obituary in full, and also the last written contribution from Tom to the CVE, C&T No. 4428 Origin of the C&T Series by Tom Hungerford (Issue 229, Feb 2003) please go to the eBook version of this issue.

Also included is classic Tom in full flight answering criticism of the C&T: Control & Therapy Criticism… (We welcome criticism), Issue 255, Jun 2009.

For 19 years during his tenure as the Director, Tom fought off efforts to 'introduce criticism, proof reading, a pean of survey to scrutinise edit and "uplift" the Control & Therapies. This would be their death knell.'

eBook download:
Read Tom's last C&Ts here

Last word from Tom...

> A man/woman wrapped up in himself makes a very little bundle, OPEN UP, share your knowledge and treasure

50 YEARS OF EVOLUTION

In pictures, as depicted on our anniversary cover

It begins with the original 'Hungry Tom's green notes' and shows the progression of the series.

Vets & contributors provide our cover images

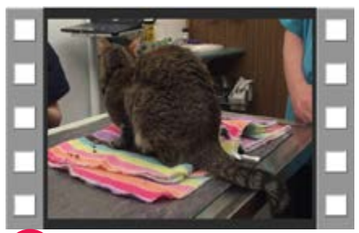
As we proudly proclaim, the C&T is written by vets for vets. Likewise, our contributors have provided all the wonderful images that have graced our covers since we went colour—a tradition we are keen to maintain, so please get your cameras out next time you see an interesting case.

Complementary eBook

Established March 2012, Issue 266

Producing a complementary eBook version in addition to print allows for the inclusion of multimedia as well as the facility to produce images in full screen. The eBook is emailed to all CVE Members quarterly while the back issues are saved here: www.cve.edu.au/control-and-therapy. Watch the 3 Videos in this issue in the eBook version.

C&T No. 5731 CVE South Africa Field Trip, p. 20



C&T No. 5733 Using a warm table to 'freeze' a cat into bliss and prevent actinic skin disease, p.23

C&T No. 5738 Cryptic presentation of nasopharyngeal lymphoma in a cat, p. 33

Reading the C&T for the first time?

Brief definitions

C&Ts: Ideally, short, pithy, practical articles ranging in length from a few lines to a page or two, hopefully accompanied by colour images, videos, graphs, X-rays, hand-drawn diagrams, tables etc. They should be something you can read and put into practice today.

Perspectives: Generally lengthier and more technical or theoretical.

We want to hear from you!

Is there one C&T or Perspective that is of particular importance or significance to you? Perhaps an article that revolutionised your approach to a particular disease or method? Or a particular issue that stands head and shoulders above the rest? Or maybe an article that you contributed that affected you in a profound way…? We'd like to hear what the C&T means to you and why you value it.

The best reply will win a voucher for CVE$1,000!

The 3 runner-up comments will each win a voucher worth CVE$250

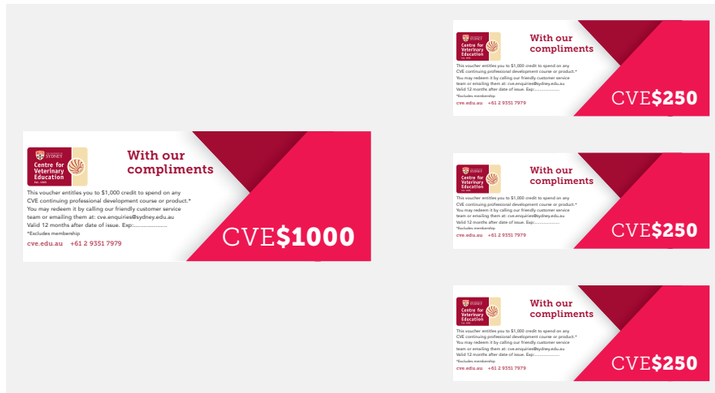
Submissions are open until 30 October 2019 with the winners announced in the December 2019 issue.

We look forward to your feedback!

Thank you.

Please send your reply to:

Joanne.Krockenberger@sydney.edu.au no later than 30 October 2019. It may be that the C&T or Perspective you nominate has stood the test of time and should be reprinted so that newer veterinary graduates can benefit from its wisdom.



AVIAN

Owls:

1. A Sooty Owl (*Tyto tenebricosa*) with aspergillosis

2. A Powerful Owl (*Ninox strenua*) with *Haemoproteus* infection

Charlie Carter

Southern Highlands Veterinary Centre

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t. (02) 4868 1310

e. charlie@southernhighlandsvets.com.au

C&T No. 5726

Comments Courtesy of Bob Doneley BVSc FANZCVS (Avian Medicine)
CVE Tutor for Backyard Chickens TimeOnline course.

Background:

'HigherGround Raptors', located in the Southern Highlands, was started and is owned and run by Peg McDonald. The facility rehabilitates sick, orphaned and injured raptors and owls.

1. A Sooty Owl (*Tyto tenebricosa*) with aspergillosis

Allied organisation Australian Raptor Care and Conservation Inc (ARCC Inc) is a not-for-profit organisation dedicated to furthering and sharing our knowledge with regard to the rehabilitation and release of injured and sick raptors and owls. ARCC Inc is also dedicated to furthering our knowledge of these wonderful birds.See www.australianraptorcareandconservation.com.

HigherGround Raptors has a purpose-built circular flight aviary modelled on the flight aviary at Abu Dhabi Falcon Hospital, but adapted for the climatic conditions found in the Southern Highlands of NSW. Peg McDonald has travelled to the Falcon hospital four times and completed 16 weeks training in raptor care and treatment.

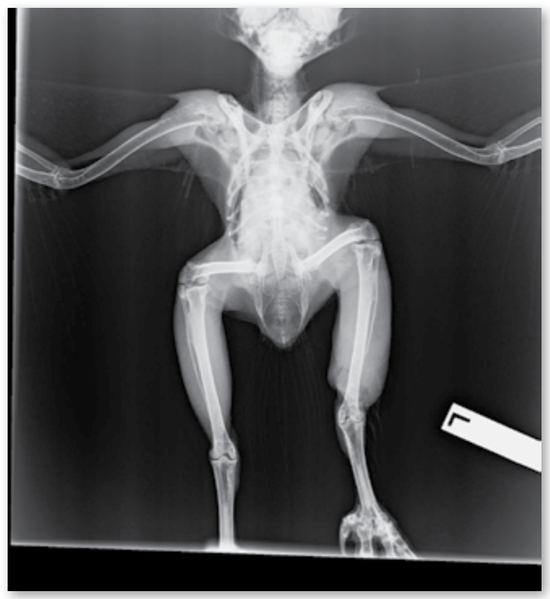
She also has successfully rehabilitated and released over 1,000 raptors/owls over the past 30 years.

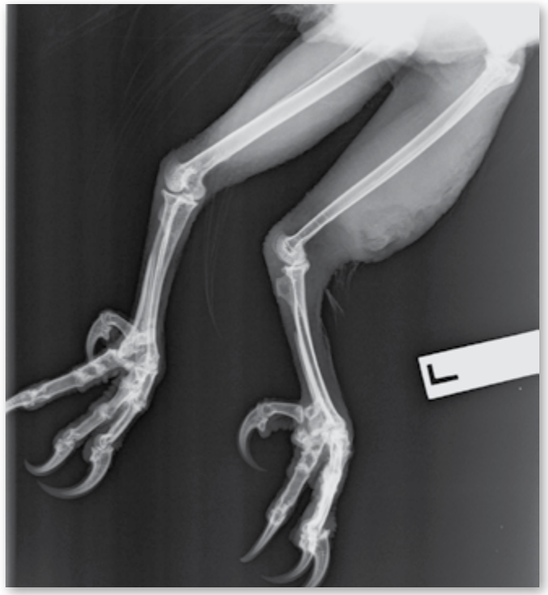
The aviary is 100m round and between 6-8m high. It attaches to two traditional rectangular aviaries which feed into it when birds are deemed ready for more appropriate exercise.

Southern Highlands Veterinary Centre provides *pro bono* veterinary services to this wonderful charity.

Sooty Owl (*Tyto tenebricosa*)

This owl presented as a second opinion in November 2016 with a chronic wound on the left distal tibiotarsus with a large amount of scar tissue.





BLOOD RESULTS	ARCC Sooty Owl 003	Barn owl reference intervals
Haematocrit	0.33	0.35-0.55
WCC (x10^9/L)	23.1	2.7-40.0
Heterophils (x10^9/L)	15.5	0.5-22.0
Lymphocytes (x10^9/L)	5.8	0.3-15.2
Monocytes (x10^9/L)	1.6	0.1-2.0
Eosinophils(x10^9/L)	0.0	0.1-8.1
Basophils (x10^9/L)	0.2	0.1-1.1
Serum Glucose (u/L)	8.2	9.7-22.0
Bile Acids (umol/L)	2	<35
Urea (mmol/L)	0.9	0-7.1
Urate (mmol/L)	0.10	0.23-1.90
AST (U/L)	109	82-303
Total Protein (g/L)	39	20-46
Albumin (g/L)	13	8-24
Globulin (g/L)	26	6-27
Amylase (U/L)	425	
CK (U/L)	3315	165-7708
Cholesterol (mmol/L)	5.9	3.1-8.8
Calcium (mmol/L)	2.57	
Phosphate (mmol/L)	1.79	
Triglyceride (mmol/L)	0.4	0.9-6.9
Serum Glutamate Dehydrogenase GLDH (U/L)	3	

[2] *Raptor Medicine, Surgery and Rehabilitation, David E Scott 2nd edition*

It weighed 1,270 grams and was thus determined to be a large female.[1] There was reduced range of motion in the left intertarsal joint (by approximately 40 degrees).

Examination was performed under general anaesthetic, she was masked down with 4-5 % isoflurane then maintained on 2% isoflurane, bloods were collected and radiographs taken.

There was no evidence of osteomyelitis (bone infection) in the left leg on radiographs. Her white cell count and heterophil count was within the normal range. She was started on half an Amoxyclav (amoxicillin/ clavulanic acid) 250mg twice daily and also oral meloxicam at a dose rate of 0.5mg per kg twice daily.

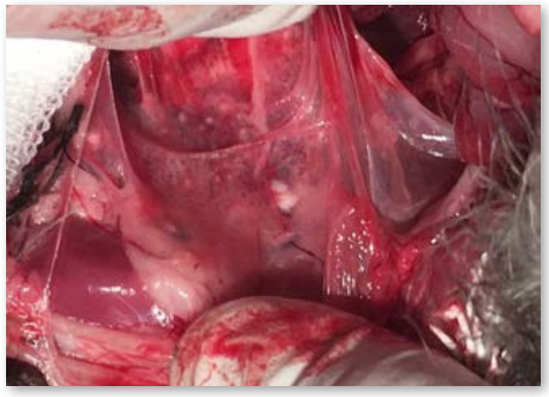
BOB DONELEY'S COMMENT:

The accepted dose of meloxicam for birds is actually 1.5mg/kg q12h. There may be some species variation, but I use the higher dose in all raptors without a problem.

Prior to admission here the Sooty Owl had been kept on organic material in a box for one week, then moved to a carer who maintained her in a large box with shade cloth over the top and a soft towelling base to facilitate clean wound healing for approximately four weeks.

She had been maintained on a suitable diet consisting primarily of rats and mice, and this was continued with feathered quail added once weekly.

Eight days later she was re-examined under general anaesthetic. A large portion of necrotic material was



removed from the wound on left leg and the wound was flushed and the skin edges debrided and closed with 3-0 polydioxanone (absorbable suture material).

Following surgery she was started on clean quail and chicken and housed in the intensive care unit. The oral antibiotics and meloxicam were continued for another 5 days.

Following suture removal she was moved to a small 3x3x4m aviary to allow her access to sunshine and fresh air without a lot of movement, flying and bouncing. She had been eating well, but following transferral into the small aviary she was reluctant to feed for three days, and then a very faint noise was detected when she was breathing. There was no open mouth breathing.

It was decided to monitor her closely and move into intensive care again, but by the next day her condition deteriorated rapidly and she died suddenly.

A post mortem examination was performed and this revealed she had a pale mottled liver and 1 to 2 mm white granulomas in the right lung. Ovarian structures were also identified confirming her sex. The lung and liver tissue was sent for histopathology at Vetnostics.

The histopathology results revealed chronic granulomatous pneumonia and hepatitis and fungal hyphae were found in the liver. This was consistent with aspergillosis infection. Unfortunately, poor husbandry, particularly housing the owl on damp organic matter is very likely to have resulted in ***aspergillus*** fungi colonising the lungs.

Bedding should not be organic (newspaper, straw, shavings). *Aspergillus fumigatus* is found commonly in the environment. It is also saprophytic meaning it grows in decaying organic matter, and this is the reason that organic bedding should be avoided at all times. It is also opportunistic and is more likely to infect birds with a compromised immune system[1].

Clean intact towels that can be changed regularly are ideal. The bird should be kept in a well ventilated area to ensure regular exchange of fresh air. Birds should also be removed from their enclosure before cleaning due to the risk of inhaling fungal spores that become airborne during the cleaning process.

BOB DONELEY'S COMMENT:

Many raptor and owl species are prone to aspergillosis, especially in the stressful conditions of captivity. Many avian vets routinely start them on terbinafine (Lamisil®) 10mg/kg q24h or itraconazole 10mg/kg q24h as a prophylactic therapy.

Reference:

1. Debus 2009 *Australian High Country Owls*, Jerry Olsen, CSIRO publishing 2011 page 307
2. David E Scott *Raptor Medicine, Surgery and Rehabilitation*, 2nd edition

2. A Powerful Owl (*Ninox strenua*) with *Haemoproteus* infection

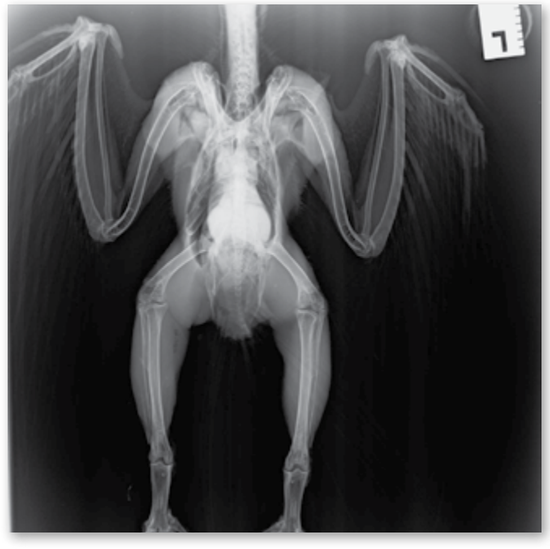
A Powerful Owl presented from the Milton area on the South Coast of NSW. He was found by a member of the public who lived in an area surrounded by forest.

This owl was observed sitting/lying on the ground and becoming progressively weaker over a period of five days, during which time the member of the public tried to contact an appropriate rescue group.

On presentation he was extremely thin and very weak. He was also ataxic and unable to walk. He was presumed to have suffered some sort of traumatic event resulting in him being unable to fly and thus hunt. His weight was 1,070 grams (females average 1,250 grams and males 1,450 grams.[1]) and given his current starved state he was likely to be a larger male.

It is important to note that Powerful Owls (along with other Ninox Owl species —the Rufous and Barking Owls) exhibit normal sexual dimorphism where the males are larger than the females. Reversed sexual dimorphism, where the females are larger than the males occurs in other owl species such as the Sooty owl and Masked owl.[2]

He was examined under anaesthesia and blood was collected from the cutaneous ulnar vein. NB :**no more than**



1mL per 100g body weight should be removed from avian patients (1% body weight) which is normally not an issue for raptors and owls due to their size. Blood is collected using a 25 gauge needle and 3mL syringe (usually 1mL) and placed in a 1mL lithium heparin tube. A blood smear is also made at the time of collection.

Radiographs were unremarkable and there were no other findings on physical examination. Fifty mLs of warmed Hartmann's solution was given under the skin prior to recovery from anaesthesia.

Interestingly his blood results revealed he had a very mild anaemia with *Haemoproteus* parasites present on approximately 5% of his red blood cells. He also had elevated muscle enzyme (CK) due to muscle damage (see Table below).

BLOOD RESULTS	ARCC Powerful Owl 004 27/2/2018	ARCC Powerful Owl 004 26/3/2018	Barn owl reference intervals
Hct	0.34	0.39	0.35-0.55
WCC (x10^9/L)	11.3	11.1	2.7-40.0
Heterophils (x10^9/L)	9.4	8.1	0.5-22.0
Lymphocytes (x10^9/L)	0.8	2.1	0.3-15.2
Monocytes (x10^9/L)	0.0	0.1	0.1-2.0
Eosinophils(x10^9/L)	0.2	0.1	0.1-8.1
Basophils (x10^9/L)	0.9	0.7	0.1-1.1
Serum Glucose (u/L)	18.1	16.4	9.7-22.0
Bile Acids (umol/L)	10	6	<35
Urea (mmol/L)	2.2	2.2	0-7.1
Urate (mmol/L)	0.28	0.27	0.23-1.90
AST (U/L)	631	475	82-303
Total Protein (g/L)	41	44	20-46
Albumin (g/L)	17	21	8-24
Globulin (g/L)	24	23	6-27
Amylase (U/L)	1328	1731	
CK (U/L)	11361	6621	165-7708
Cholesterol (mmol/L)	7.7	8.3	3.1-8.8
Calcium (mmol/L)	2.4	2.44	
Phosphate (mmol/L)	1.35	0.57	
Triglyceride (mmol/L)	1.2	0.7	0.9-6.9
Serum Glutamate Dehydrogenase (U/L)	6	4	

[4] *David E Scott* Raptor Medicine, Surgery and Rehabilitation, *2nd edition.*

Haemoproteus is spread by biting insects such as the *Culicoides* midge.[3] Treatment can be attempted using chloroquine and mefloquine,[4] but as obtaining these medications is difficult and given that less than 10 % of red blood cells were infected (meaning the infection was less severe) we elected not to treat the infection in this patient. It was also very likely that this infection was not this owl's primary problem and was more likely an incidental finding.

BOB DONELEY'S COMMENT:

Many wild birds have *Haemoproteus* in their erythrocytes. Occasionally they can be problematic, but they are usually an incidental finding.

He required intensive treatment to ensure his survival. Peg McDonald started him on Vetafarm Spark, giving 10mLs via tube feeding every couple of hours.

After 12 hours, egg yolk was added to the Spark mix and he was fed every 4-5 hours. After 24 hours, blended chicken meat to puree consistency was added to the Spark and egg yolk mix—feeding around 18mLs every 4-5 hours. After 36 hours hand feeding commenced using clean chicken breast.

This regime saw him eating solid food faster than a more typical patient, but he was at high risk of starvation and required a faster progression onto solid food.

Once the ataxia resolved after 10 days he was allowed to feed unassisted. He was provided with clean chicken and quail meat. Owls can't digest bones and fur as their proventriculus produces much less acidic stomach acid, so it is important to provide clean meat whilst they are convalescing. Feeding food items with bones and skin will require the owl to work harder digesting this food and is less desirable in a hospital situation.

In fact, raptors like goshawks have stomach acid 20 times stronger than owls meaning they can digest bone, whereas owls can't digest bone and need to pass bones and fur as casting.

BOB DONELEY'S COMMENT:

I agree with these comments. The conventional wisdom is not to feed until the owl has cast after the last meal. This is not suitable in a situation like this, as intensive assisted feeding is required.

One month later and the Powerful Owl was doing very well. He had gained weight and was showing no signs of ill health. He was re-examined under anaesthesia and blood was collected to check the status of the *Haemoproteus* infection. His weight had increased to 1400 grams.

The blood results showed his red cell count had increased to within the normal range (see table left).

It was now very difficult for the pathologist to locate *Haemoproteus* organisms, meaning the infection had almost completely resolved without specific treatment, as his health had improved and he gained weight.

He was released in the same area he was found after one month in care.

If you would like to follow our work, check out the HigherGround Raptors and Australian Raptor Care and Conservation Inc Facebook pages.

If you can assist us in any way please feel free to contact Charlie via charlie@southernhighlandsvets.com.au

Acknowledgments

- Peggy McDonald
- Stephen Yeomans, Sue Jaensch, Doug Hayward and all the team at Vetnostics for their ongoing support in allowing us to monitor blood biochemistry and haematology in these beautiful birds
- Tony Gestier, Vetafarm
- Dr Margit Muller, Abu Dhabi Falcon Hospital
- Stephen Debus
- Jenny Packwood
- Dave Harker
- Melanie Barsony

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2. Jerry Olsen, *Australian High Country Owls*, CSIRO publishing 2011 pages 109-110
3. Margit Gabrielle Muller, *Practical Handbook of Falcon Husbandry and Medicine*, Nova Science Publishers, 2009
4. David E Scott *Raptor Medicine, Surgery and Rehabilitation*, 2nd edition

Figure 2. Powerful owl prior to release.

eBook download:
C&T No. 5631 Australian hobby in care.

Who was your most memorable patient and why?

Niels Pedersen

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C&T No. 5727

When I was an intern at Colorado State University in 1967-1968, we received a litter of Chihuahuas with severe ringworm.

I worked hard to clear up the ringworm so that I could find them homes. However, I overestimated the demand for these little dogs, and we were reaching the point of having to put them down. At the same time, I had an older client who had a hunting dog with an incurable brain tumour. This dog was this man's whole world, and I knew that he would take its death hard. So after I euthanased his old dog, I sat him and his wife down and presented him with one of these puppies, carefully explaining that it would soon be put down for lack of a home. He became angry, threw the puppy back into my arms, and stormed out.

No dog could replace his old dog! His wife came back a minute later, said she knew he would change his mind, and asked if she could take the puppy. Six weeks later, she phoned. The puppy had grown fond of her husband, but he was not reciprocating. A few weeks later, she said that her husband would let the dog sit on his lap occasionally. After a few more weeks, she informed me that her husband and the Chihuahua were inseparable. From this experience, I learned that people go through a grieving period, and only when their grief is spent, will they open their hearts to a new pet.

Rabbit image courtesy of Anne Fawcett

Members | Readers are directed to search the CVeLibrary for articles or chapter proceedings dealing specifically with grief e.g. Trepheena Hunter contributed C&T No. 5261: Dealing with grief: The owner's relationship occurs with the individual, regardless of the species (Issue 268, Sept 2012).

Veterinary care of bats and Australian Bat Lyssavirus

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C&T No. 5728

Anne Fawcett's C&T No. 5705 was very relevant as we approached the summer season, when flying fox feeding activity and numbers are at their highest. The frequency of reported interactions between bats and people will probably continue to increase, as urbanisation and development results in closer contact with wildlife. All bats are vulnerable to a range of anthropogenic threats, such as roost disturbance and habitat destruction. Thirteen of Australia's bat species are now listed as threatened under our national conservation legislation, including the grey-headed flying fox.

The relatively recent establishment of grey-headed flying fox colonies in Victoria means that these charismatic and intelligent animals are frequent patients in Zoos Victoria's wildlife clinics. Injuries suffered by bats often are severe, and require immediate veterinary attention to relieve pain and suffering.

Veterinary intervention is required following bat interactions with humans and pets:

- Clinical assessment and treatment of injured bats: insectivorous bats and flying foxes. Entanglement in fruit netting is a major cause of hospital admissions of grey-headed flying foxes in Melbourne, and these animals may have devastating soft tissue trauma of the wing/s and oral cavity (Scheelings and Frith, 2015).
- Humane euthanasia of a bat for testing, following a report of possible ABL exposure (bite or scratch to pet or person).
- Providing advice for pet owners following bat-pet contact: dog attack, as described in C&T No.5705, or cats that have presented an insectivorous bat to their owner.

Within Zoos Victoria's veterinary clinics, animal care staff follow these protocols when handling bats:

1. They must have completed the pre-exposure rabies vaccination course, and their rabies neutralising antibody titres must be adequate when checked. Titre checks are performed every two years. The initial course of pre-exposure rabies vaccine is provided free of charge to Australian wildlife handlers working in a volunteer service in Victoria.

2. Staff members that are involved with capture, restraint and/or assessment of bats must have received adequate training in restraint and safe handling of bats, and must be wearing mandated personal protective equipment (PPE) during procedures.

When handling flying foxes, a long-sleeved shirt or jacket of heavy material (with both sleeves buttoned at the wrist) and long pants must be worn. We use 19" Protective Arm Sleeves (Hexarmor® AS019S) to prevent scratches that may occur during restraint. These are manufactured for industrial puncture protection and are washable. In addition, two layers of gloves are worn: protective gloves (Hexarmor®or leather or deerskin handling gloves) with a nitrile or latex underglove must be worn by all staff members involved with handling a conscious flying fox. Once the bat is safely anaesthetised, the outer gloves can be removed and nitrile or latex gloves are worn throughout the procedure. Glasses/goggles and surgical mask or face shield are worn when there is risk of exposure to bodily fluids or injury from a conscious bat.

When handling insectivorous bats, a double layer of disposable gloves is worn, with the outer glove being nitrile.

Figure 1: Hexarmor® protective sleeves will provide protection against bites and scratches and are easy to wear. Nitrile gloves are worn throughout the procedure. During conscious restraint and anaesthetic recovery, additional leather/deerskin/Hexarmor® gloves are also worn, as these are the periods when there is greatest risk of bite injury.

Note that *PPE is worn even when the handler is fully vaccinated.* When handling dead bats, direct handling of the animal is avoided by wearing gloves and/or wrapping in a towel/jumper. Appropriate care must be taken if there is any risk of puncture wounds from material entangling the animal (e.g. barbed wire). Needle stick injuries pose a similar risk—do not recap needles.

Euthanasia

The most frequently-used method for induction of anaesthesia is restraint for administration of gaseous anaesthetic agents by facemask. Lethabarb is then administered IV via the saphenous (interfemoral) vein in the uropatagium, or the cephalic vein. If the bat is contained in a transport box, you can adapt the box for use as an induction chamber to induce anaesthesia with minimal handling. Intramuscular administration of medetomidine/ketamine combination may also be used to anaesthetise an animal prior to euthanasia. Forty mg/kg tiletamine/zolazepam squirted directly onto oral mucous membranes may immobilise flying foxes in situations where handling is extremely dangerous—this method is not recommended for routine procedures, as anaesthetic recovery is prolonged and variable (Heard 2014).

Possible ABL exposure cases:

C&T No. 5705 outlines the approach required when a case of possible ABL exposure is reported in NSW. Each state animal health authority produces recommendations that will assist veterinarians involved in these cases. In our experience, such cases can have very complex histories, and several people may have had contact with a single bat before it is presented at the veterinary clinic. In Victoria, state authorities do not yet have a formal, co-ordinated system for transportation of bats and/or bat tissues

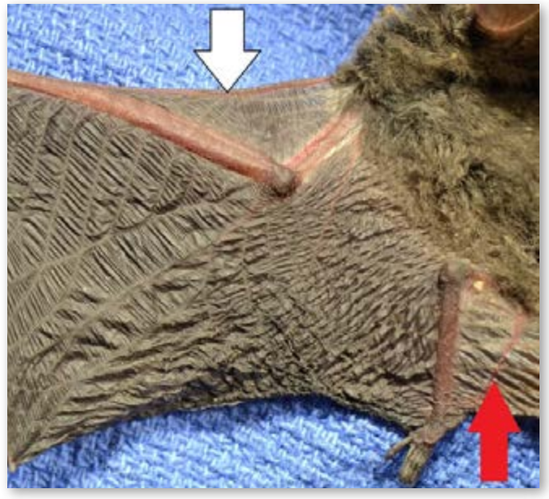

Figure 2: The lesser long-eared bat Nyctophilus geoffroyi *is an insectivorous bat, here in dorsal recumbency. The location of the cephalic vein (white arrow) and saphenous vein (red arrow) is similar in all bats.*

Figure 3: The grey-headed flying fox, Pteropus poliocephalus, *in dorsal recumbency. The location of the left cephalic vein (white arrow).*

Figure 4: Grey-headed flying fox, dorsal recumbency: the location of the right saphenous vein (red arrow).

between veterinary clinics and laboratories, and we rely on the goodwill and hard work of rabies-vaccinated wildlife carers, veterinary pathology couriers, and the state's veterinary pathologists and animal health officers to make the system work.

In all possible exposure cases, the bat must be tested for ABL, therefore submission of tissues to the relevant government laboratory must occur as quickly as possible. Often the bat is alive following these interactions, and so must be transported to a veterinary clinic for euthanasia and submission of fresh tissues for testing. When ABL testing is required, the entire body is submitted on ice (double-bagged, sealed in an esky that is clearly labelled as a potential biohazard, with submission documents

attached to the outside of the esky). Submission documents must include contact details of any person that was bitten/scratched, as this paperwork will also notify the state's health department about the case. This may become important in cases where the patient fails to attend a doctor.

Vaccinated wildlife carers should avoid bites or scratches by wearing appropriate PPE, but usually they do not do so, choosing instead to assume that they are adequately protected following rabies vaccination. However, when vaccinated carers report bites/scratches, health authorities may recommend post-exposure vaccination regardless of their serological status (Merritt *et al.* 2018). Bite/scratch prevention avoids unnecessary post-exposure vaccination of carers, and reduces the number of healthy bats that must be euthanased for testing following handler injury.

Unvaccinated people that have been bitten or scratched must seek medical attention immediately. In these cases, the post-exposure vaccination course usually is commenced immediately after the injury occurs. Post-exposure treatment for ABL is provided free of charge by the Department of Health and Human Services (DHHS) in Victoria. DHHS will also provide appropriate post-exposure vaccination advice to a patient's medical practitioner. It is the patient's doctor that provides vaccination advice to the patient, including results from testing of bat tissues, when managing the case (in Victoria, AAHL will report these results to DHHS, and DHHS will liaise with the doctor). DHHS clinicians assess each case and determine if and when post-exposure vaccination will be given.

When a domestic pet may have been exposed, state veterinary officers provide post-exposure vaccination/monitoring advice to the pet's veterinarian: in Victoria, we will contact the Disease Watch hotline (1800 675 888) to report incidents, and seek their advice in each case. AUSTVETPLAN's disease strategy for ABL is currently under review.

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Additional information and fact sheets may be found at wildlifehealthaustralia.com.au

eBook download:
C&T No. 5705. Possible Lyssa Virus Exposure in a Siberian Husky.

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joanne.krockenberger@sydney.edu.au

WINNER!

GENERAL

How does a vet find a new disease that no one has seen before?

Marshall Thornton

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C&T No. 5729

This a very brief version of events: I thought I could have discovered a new disease, possibly zoonotic from flying foxes.

There were 9 dogs in a yard over which flying foxes were flying—dropping faeces, urine, and dead foetuses in the yard, as the client's house was close to their roost in Cessnock.

Test, I hear you say.

All 9 dogs developed a cough, two died, the husband also developed a cough and was apparently quite unwell. The concept of One Medicine / New Zoonotic Disease—all these were running through my mind!

We only had clinical interaction with two of the dogs. The first died in spite of treatment. The second was treated with everything I could think of, had clinical pathology performed and then an autopsy. The pathology specimens were sent to Elizabeth MacArthur Agricultural Institute (EMAI) after talking to a pathologist there—they were worried this was an unknown zoonosis.

The EMAI pathologist's reports were inconclusive. I argued that EMAI was a Government lab and should be looking for new disease causative agents, but apparently they only look for what they already know, especially notifiable diseases. They do not try to look for what they do not know.

So who looks for diseases we do not already know about?

RESPONSES

1. Siobhan Mor

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From my perspective, and even before further laboratory investigation, I would still think the scenario should —as a first step—be reported to the Animal Disease Hotline (1800 675 888). This would engage government veterinarians in the risk assessment process and usually they are better informed than private vets in terms of who to engage (public health unit, wildlife coordinator etc.) But there are some legitimate hurdles in this scenario as it involves a companion animal species, which doesn't really fall under the remit of government unless there is a clear threat to the livestock industry and/or public health (much to the frustration of private vets).

This is a *bona fide* gap in disease surveillance (lack of companion animal health surveillance) – and it is common throughout the world. In Australia, there is the National Significant Disease Investigation program (NSDI) (www.animalhealthaustralia.com.au/what-we-do/disease-surveillance/national-significant-disease-investigation-program) which pays a subsidy to private vets to investigate significant disease events; private vets should know about this though I suspect this is still largely directed to livestock investigations. Then there is the question of lab capacity and whether state labs have the capacity and remit to investigate unknowns in companion animals. I suspect in the end this kind of scenario falls to researchers to investigate (in universities or government labs), but the entry point for a private practitioner to engage with that side is probably not very clear.

2. Keith Eastwood

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This is an interesting topic and one that has challenged our (human) public health unit in the past when we've been involved with animal health situations such as a kangaroo die-off. Early notification is an important starting point so that agencies can determine whether they have a role

in the response or are, at least, able to monitor progress. Pursuing an emerging disease, per the scenario provided, would depend on the circumstances and explores the boundary of surveillance and research – a likely area of contention. Pragmatic issues such as lead agency, cost-sharing and media reporting have already been mentioned but are crucial and not always easily resolved.

It is not possible to have a conversation involving One Health incidents without considering how much easier incidents like this would be managed through a national communicable disease control centre. Imagine if the incident described by Marshall occurred on the border of two states!

3. Karrie Rose

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Investigating novel pathogens can be a very expensive and lengthy process and the current biosecurity systems do seem to focus on the notification and management of known animal diseases. In addition to the programs that Siobhan has outlined, there is a NSW Wildlife Inter-Agency Group, chaired by DPI and involving EPA, OEH, Health, LLS, WHA and the Registry. These meetings are held quarterly to address the management of emergent wildlife disease events. Discussions to define a lead agency and determine who will pay for the costs for emergency/emerging wildlife disease events are ongoing. The NDSI program is fantastic to enable the early stages of a disease investigation and WHA have done a great job to ensure that it is easily accessed. Those funds often range between $500 to $3,000 which is a very, very small fraction of the money expended on any investigation.

In relation to practical advice to practitioners, contacting WHA and the Registry are also good options if wildlife are affected (not for domestic animals though).

- Outside of the given scenario, I would tend to contact experienced wildlife carers to determine whether a disease syndrome in wildlife was common or likely to be something new. Whenever experienced and trusted carers say this is new, there is good value in conducting an investigation.
- Generally, we recommend an investigation whenever there is a mass mortality event (5-10 or more unexpected mortalities), signs or findings consistent with a notifiable disease (tubercles, foot or oral ulcers), animals with unexplained emaciation or neurological signs, or signs of potential infectious disease (enlarged lymph nodes, polyarthritis, discharges, respiratory distress).
- Think of Occupational Health and Safety/Personal Protective Equipment (PPE) before first contact. We've just recently had some cases where we have been incredibly pleased that we were replete with PPE and had no students attending animal handling and necropsies of some severely emaciated wild animals.

4. Kate Wingett | Senior Veterinary Officer

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An excellent area of working across sectors to assess and mitigate risks. The advice from Siobhan is spot on in regards to contacting the Emergency Animal Disease Hotline on 1800 675 888 to report such cases. The NSW Biosecurity Act 2015 and subordinate legislation require mandatory reporting of biosecurity events, an unusual mortality event would be considered a biosecurity event. Actions taken in any one event will be based on a risk assessment performed by the relevant stakeholders, including NSW Health and Office of Environment and Heritage when appropriate. As mentioned by Siobhan and others, NSDI funding, WHA and The Registry are all excellent avenues open to private practitioners to collaborate when investigating such events.

Managing disease outbreaks and public health incidents that occur at the human and animal health interface

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C&T No. 5730

Introduction

Incidents such as animal die-offs, zoonosis clusters, chemical spills, emerging diseases and a host of other events impact on human, animal and environmental health. As such the response can be complex and involve multiple agencies and organisations that rarely work together at the coal-face. The following article identifies some of the issues and challenges and is taken from a presentation delivered at the 2016 One Health EcoHealth Conference, Melbourne and co-authored by Paul Freeman, Tiggy Grillo, Tony Merritt, Peter Massey and David Durrheim.

Understanding One Health

In a simplistic fashion, One Health may be considered as the area of intersect between various disciplines usually confined to human, animal and environmental health.

However, in reality One Health is complex and responding to a One Health incident may involve multiple agencies plus a host of stakeholders such as the RSPCA, Wildlife Health Australia, councils, interest groups, local veterinarians, land management groups, environmental bodies, community organisations etc. The geographical jurisdiction of these groups rarely coincide so an incident affecting a large area may impact multiple branches and organisations in other states and territories. Additionally, other disciplines such as the social sciences may have relevance.

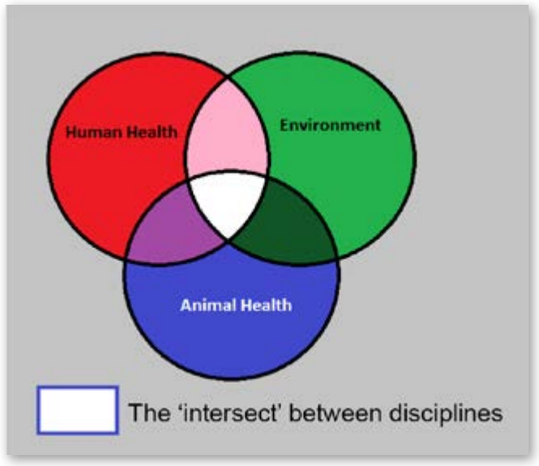

Figure 1: One Health?

Managing One Health incidents

Coordination of expertise and inter-departmental collaboration is important. In some circumstances the lead agency is obvious, however, in other situations responsibility may be blurred and leadership may become an issue. In a recent joint exercise (Tamworth, 2017), 7 organisations were invited to meet and discuss bat-related environmental issues with each group asked to nominate the lead agency. Six different suggestions were provided and no group stepped forward themselves.

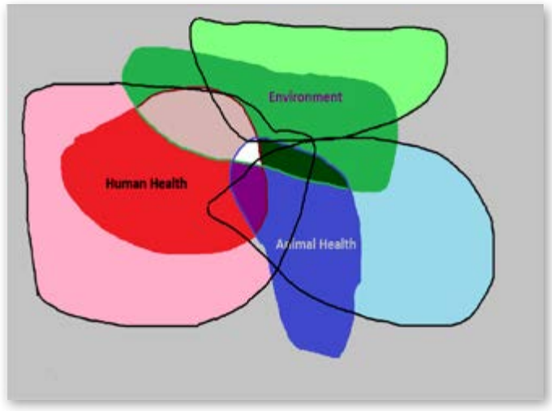

Figure 2: A more realistic description of One Health.

Table 1: One Health response process
Surveillance: disease or event notification
Establishing the lead agency
Organizing a response team
Allocating responsibilities
Sharing workload, costs and activities
Combining skills, networks and resources
Interventions
Report, recommendations and actions

Figure 3: Agency priorities (Graphic courtesy Dr Siobhan Mor).

Table 2: Possible One Health incidents
Zoonotic clusters e.g. anthrax, Q fever, psittacosis
Commercial animal outbreaks e.g. avian influenza
Animal die-offs (sentinel events)
Food safety e.g. salmonella contaminated melons
Events of unknown aetiology
Animal contact e.g. brucellosis
Environmental contamination
Severe weather events
Others

Key to the entire response process is prompt event notification. The alerted agency is obliged to inform all affected parties, a responsibility which inevitably exposes flaws in the communication path. Once the lead agency is determined an investigation team is assembled and responsibilities allocated. Issues of sharing workload and costs rely on good will and (hopefully) prior experience working together, but offer opportunities for tension.

Contention may also arise from agencies with contrasting resource capacity, financial and personnel. Also priorities may not align. Even events involving zoonoses cannot be presumed to be common territory as diseases acquired from animals often have little impact on livestock (although they are important for farmers, vets, hunters and others in contact with animals).

Preparedness

In some of the incidents noted in Tables 2 and 3, the One Health involvement is obvious, but in others it is less evident but nonetheless important. For instance in the 2007 Australian equine influenza outbreak there was no direct human risk but impact on income and stock/pet-loss, export implication etc. placed a significant mental health burden. The outbreak provided opportunities for a One Health response through sharing staff, expertise and providing surge capacity (resulting in cooperation between the Department of Primary Industries and New South Wales Health).

The value of preparedness, collaboration and network-development in 'peace time' is important to ensure a cohesive response when One Health incidents are identified. This can be achieved through encouraging regional One Health Networks that bring together professionals from relevant disciplines. There are many opportunities to work together before the inevitable incident arises, such as research and surveillance projects, joint exercises, regular meetings, cross-education opportunities, collaboration on policy development and development of factsheets.

Conclusion

Unwelcome interactions between humans, animals and the environment resulting in health emergencies are inevitable and demand multi-agency intervention. Preparedness through regular networking, joint exercises and other collaborations can only be of benefit and should be encouraged.

Table 3: One Health incidents experienced in northern NSW since 2007
Influenza: equine, avian, human, swine
Q Fever clusters
Brucellosis (B. suis): pig shooters, dogs, families, litters
Wildlife die-offs
Cryptosporidiosis in livestock and farmers
Kunjin in horses
Anthrax in cattle
Bats, Australian Bat Lyssavirus risk and bat camps
Weather events: 'east coast lows', bushfires, floods, drought

CVE South Africa Field Trip

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C&T No. 5731

The CVE's South Africa field trip was an incredible time. Two full weeks were spent at Dinokeng Game Reserve, a 22,000 hectare reserve an hour from Johannesburg, observing wildlife which included numerous species, as well as conducting various surveys to collect data to help WEI (Wildlife Ecological Investments) manage the reserve from a conservation perspective. We experienced so many fabulous moments.

In the first week we were involved in remote anaesthesia using a dart rifle on 4 lionesses to insert Desloren (GRNH antagonist) implants to prevent pregnancy to manage the population and prevent mating between siblings. This included hands-on work taking blood samples, faecal samples, external parasites (ticks), hot branding, applying and monitoring a pulse-oximeter, auscultating breathing and heart beat and minor surgeries on open wounds. The next day we were involved in darting a male lion to replace his radio collar with new batteries to enable tracking his movements and those of the accompanying females. Similar samples were taken from him as well. There was much unexpected excitement when the male suddenly growled and staggered to his feet with most of us close by early in the procedure. He did not rouse fully and we were able to move away (some too quickly) to allow the wildlife veterinarian to administer extra anaesthetic to continue the work. We wanted to dart the female as well to renew her Desloren implant but couldn't. However, now her companion is collared it should be much easier to locate and anaesthetise her to renew her implant.

Game drives involved identifying all mammals larger than a scrub hare along a 10-km transect, including age, sex and GPS coordinates. Interesting species included white and black rhinoceros, impala, kudu, nyala, blesbok, red hartebeest, wildebeest, eland, cape buffalo, warthog, waterbuck, vervet monkey, jackal, mongoose, cheetah and of course lion, elephant, giraffe and zebra. For the vegetation surveys we were divided into two teams, one identifying and measuring (height, grazing damage, fire damage) all the grass species within multiple plots, the other collecting similar data for woody plants over 0.5 m high. The data are used to monitor the populations and estimate the reserve's carrying capacity to avoid damaging the ecosystem.

The CVE's trip to South Africa was a truly unique opportunity for veterinarians to employ some of their veterinary skills in an authentic wild African environment. The group loved every minute of their time at Dinokeng Nature Reserve, and some are making plans to return next year with friends and family.

Watch the video in the eBook:
vimeo.com/307956574

Figure 1: Lecture time with WEI's Lozanne Van Sittert.

Figure 2: Enjoying the view from the back of the bakkie (Afrikaans for ute!).

Figure 3: Juvenile and adult white rhino.

Figure 4: CVE's South Africa Field Trip 2018.

Figure 5: Monitoring the heart rate of the anaesthetised male.

Figure 6: Dinokeng's elephants enjoying the cool of the waterhole.

Figure 7: Darted male lion being carried to the shade of a tree to have his radio collar replaced.

Figure 8: Cape Buffalo.

Figure 9: Cheetahs.

Figure 10: Young male lion.

‘What makes a horse good?’ & 'What makes a good horse?’

The evolution of modern equine practice in Australia

RR Pascoe, GH Hazard, WP Howey

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C&T No. 5732

During the enlightened ‘60’s a few real scientists began to fully examine these questions.

The Evolution begins post WWII

Modern equine veterinary practice began with the increasing number of veterinary graduates immediately after the end of World War II. Prior to this period, in most States, only small numbers of veterinary surgeons derived the bulk of their income from horse practice.

Director Dr Tom Hungerford Heads the PGF

The initial impetus began around 1965 with the Sydney Post Graduate Foundation (PGF) in Veterinary Science courses.

These were usually held over a week, expanding from one to a number occurring throughout the year under the very capable directorship of Dr Tom Hungerford who invited overseas lecturers from the USA and England to present cutting edge lectures on horses, covering the areas of Equine Medicine, Surgery and Reproduction.

These courses which attracted veterinary surgeons from Australia and NZ were hugely successful as they excited a great thirst for knowledge which was difficult to obtain due to the lack of published scientific articles with interest in the horse and its problems. Many firm friendships developed at these courses and helped with the dissemination of ‘new’ knowledge.

The PGF hosts the J D Stewart ‘Equine Diseases’ course in 1970

Driven by the icon Dr T G Hungerford, it was held at Sydney University, and was one of the first PGF courses purely for horses. The keynote speaker was Associate Professor of Pathology at the University of Pennsylvania Dr Jim Rooney DVM. Dr Rooney had autopsied over 900 horses and was a revelation! He was supported by A. M. Bain, P. E. Sykes, D. R. Hutchins, T. K. Bell, Hugh McL. Gordon, R. R. R. Pascoe, J. D. Steel, M. Robinson, R. H. C. Penney, L. H. Larsen, P. Fallon and J. G. McLean.

Figure 1: Horse operation 1914. Equine Surgery was very much a ‘grass lawn’ adventure with poor, ineffective anaesthetics, almost no analgesics and no tranquillizers.

Interested in reading more? Go online to the C&T Ebook.

eBook download:
Read this fascinating article complete with archival images in the eBook.

Search the CVeLibrary to access all the PGF’s equine proceedings e.g.:

- Proc No. 4 Equine Practice (Nov 1968)
- Proc No. 7 Equine Diseases (Feb 1970
- Proc No. 25 Equine Surgery (May 1975)
- Proc No. 33 Equine Lameness (May 1975)

Access to the CVeLibrary containing 50+ years of proceedings, C&Ts, Perspectives and Vade Mecum is a major member benefit.

Thanks to Bill Howey, former PGF Director, for supplying the article (first published in the Australian Veterinary History Record, *Mar 2009) and to the University of Sydney Veterinary Science Archives for the images.*

Using a warm table to ‘freeze’ a cat into bliss and prevent actinic skin disease

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C&T No. 5733

Given the preferred ambient temp of cats being so high (32-38°C), I now have a variety of removable heat packs wrapped in fleeces (the latter is the preferred cat substrate according to a behaviour study) on my consult table. (See C&T No. 5626 ‘Comfortable ambient temperatures in elderly cats’, and C&T No. 5639 ‘Reply to 5626: ‘Sun drunk cats.)

The heat plates transform handling—the cats realise the heat pack is there, sit on it and they then don’t move. You can do so many things and the cat doesn’t care because they are in a heat bliss. One grumpy old cat, who came to the clinic in a cat box hand-carried from home by the owner on a cold day, was so enamoured of the heat pack that he dug his nails into the wheatgerm version and we could not separate him from it. He hugged it to his chest like a baby and would not let go. The packs are small and almost disposable (they are made by a client; see C&T No. 4891 figures 1-4) so he went home with it. The owners say the heat packs have transformed his behaviour.

We also use the ‘SnuggleSafe’ heat discs which have removable covers and are wipeable. I am now suspicious that all those drawings/illustrations over the centuries of cats curled up are actually cats freezing cold; when given the chance, cats stretch out and soak up heat and lie extended out in the sunshine. They usually only curl up tight if they are cold.

Now, when clients bring a cat to the clinic in their car, the nurses also ask them not to sit the cat cage on the back seat directly in the flow of the car's air-conditioner. We ask them to set the carrier behind a seat and cover it.

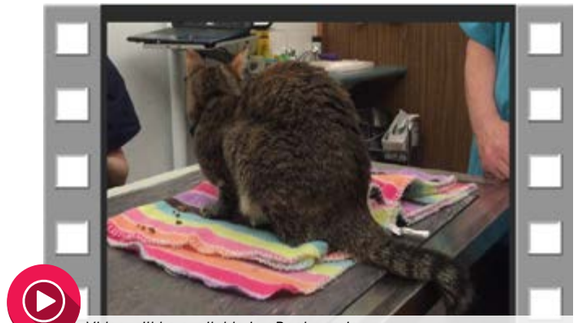
Re actinic-induced or solar-induced skin disease

How many of our cat-owning clients understand, or even know, that if a cat’s preferred ambient temperature is 30-38°C, then failing to provide appropriate non-solar sources of heat indoors pushes the cat to seek out and remain in sun-heated areas for longer than is safe/healthy?

Keeping a cat indoors, and away from windows, etc—especially in an air-conditioned house but then failing to provide access to heat sources may keep the cat safe from actinic damage but sets up a cascade of stress and uncomfortable living, similar to how we humans would feel living at 10-14°C.

I use the heat packs on the table to show how obsessed the cat is with getting warm, and how that endangers the pet. Not just the usual perils of hiding under car engines and getting into warm tumble dryers but also, while seeking warmth through extended window-baking, they are getting lots of damaging rays as well.

If skin cancer is a reflection of imbalance/overexposure to damaging sun rays in the environment, then not addressing all aspects of the environment leaves treatment underprovided. We prescribe UV shirts, cox2 drugs, Aldara etc but we need to get the owner to understand the ‘why’ and the ‘how’ actinic lesions occur in the first place, so that the owner understands and is on-side and committed from day 1.



Video will be available in eBook version

eBook C&T downloads:

No. 4891 Medical white coats—more versatile than you think

No. 5626 Comfortable ambient temperature in elderly cats

No. 5639 Reply to 5626 Sun drunk cats

Figure 4: SnuggleSafe heat pad.

WINNER!

Anaesthetic protocols for dogs on anti-anxiety medications

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C&T No. 5734

Hospitalisation and anaesthesia for a dog on oral anti-anxiety medications including tricyclic anti-depressants (TCAs) e.g. clomipramine or selective serotonin re-uptake inhibitors (SSRIs) e.g. fluoxetine, needs careful pre-planning and management to help reduce the stress of both hospitalisation and after care. Stress can have negative effects on both the welfare of the patient while in hospital and the outcome of surgery in the post-operative period.

I have put together a protocol which I use for patients on anti-anxiety medications requiring a general anaesthetic. The protocol is designed for dogs undergoing routine procedures such as dentistry, desexing or minor surgery not requiring extensive use of opioid medication post-operatively. There are many different options, but after discussion with veterinary anaesthetists and veterinary behaviourists, this is my approach:

Regular medication

Patients who have been prescribed SSRIs or TCAs need to continue the medication as before.

Example 1: If the patient takes fluoxetine in the evenings, ensure that the dog receives the dose the evening before the surgery and the owner brings his medication along in case the dog needs to stay in overnight.

Example 2: If the patient takes clomipramine twice daily and is due for a dose the morning of the surgery, time the evening dose to be given a little earlier than usual (e.g. 5pm) and give the morning dose with a tablespoon of food 12 hours later (i.e. 5am). A small amount of food given early in the morning 6 hours before surgery is acceptable.

Do not abruptly discontinue the behaviour modification drugs pre or post-surgery, as this can be detrimental to their welfare.

Admitting the patient

Consider a later admission for highly anxious patients so that the wait time between admission and surgery is reduced. This may mean the owner takes a day off work to allow for a suitable admission and collection time.

Avoid 'double-handling' the patient unnecessarily by administering the pre-med in the consult room with the owner present where possible.

Premedication

I use one of the options below:

1. Acepromazine (ACP) 0.025mg/kg + methadone 0.25mg/kg IM; or
2. Medetomidine 0.003mg/kg + methadone 0.25mg/kg IM

There is no evidence to suggest that doses of acepromazine (ACP) between 0.01-0.03mg/kg alter the seizure threshold in dogs.

Ideally give the pre-med into the neck muscles with a 25 gauge needle as it tends to be absorbed faster when given in this region. These combinations should be given in the same syringe, rather than 2 separate injections.

Anaesthesia

Alfaxan® 2mg/kg (0.2mL/kg) IV for dogs then isoflurane/oxygen maintenance. I tend to 'top up' my anaesthetics with small increments of Alfaxan® rather than increasing the isoflurane above 2%.

Intravenous fluid therapy at 5mL/kg/hr for cardiovascular support for the first hour, then reduce to 2.5mL/kg/hr (maintenance) is warranted for all patients on SSRIs or TCAs undergoing anaesthesia. (In our practice we use it routinely for all patients).

Blood pressure monitoring is worthwhile in these patients to check for hypo or hypertension intra-operatively and manage as required.

Post-operative pain relief

For patients having a 'rough' recovery (vocal or painful) then medetomidine 0.003mg/kg IV can be given immediately (if not used in the pre-med),

Repeat methadone 0.25mg/kg subcutaneously at 4 hours after the pre-med dose was given.

Non-steroidal anti-inflammatory drugs are a good option for post-operative pain relief in these patients.

There is no concern about the short-term use of NSAIDs and behaviour modification drugs (SSRIs or TCAs) in dogs. The risk of gastrointestinal ulceration is something that is monitored for in people but not a contraindication for combining the two classes of medications and has yet to be recognised as an adverse interaction in dogs.

For canine patients unable to tolerate NSAIDs, then paracetamol at 11mg/kg PO SID could be an alternative option.

Serotonin syndrome

Serotonin syndrome is a toxicity caused by excess serotonergic stimulation in the central and peripheral nervous system.

There is a risk of serotonin syndrome when SSRIs or TCAs are combined with certain medications for extended periods or if a dog receives an extremely high dose of their SSRI or TCA (e.g. overdose).

Clinical signs include:

- Autonomic signs—diarrhoea, mydriasis, tachycardia, tachypnoea, hypertension, fever
- Neuromuscular signs—hyperreflexia, myoclonus, tremors, rigidity, seizures, respiratory muscle compromise
- Altered mentation—agitation, confusion, disorientation, vocalization, excitement

Serotonin syndrome is unlikely to occur for routine procedures where opioids are used at standard dose rates in the immediate post-operative period; however, for patients receiving high dose fentanyl constant rate infusions (CRIs), it needs to be monitored for closely. It is best to avoid post-operative tramadol or fentanyl patches for patients on SSRIs or TCAs.

Drugs to be aware of that may contribute to serotonin syndrome (serotonergic agents) include: fluoxetine, clomipramine, opioids, mirtazapine, metoclopramide, trazodone. There is an extensive list of these on VIN and treatment of serotonin syndrome in the conference proceedings from IVECCS 2017, '*Serotonin Syndrome*' by Dr Lauren Harris.

Practitioners should be aware of the clinical signs to look out for when preparing to anaesthetise a patient on a long-term SSRI or TCA medication.

Continued overleaf...

Additional anti-anxiety medications

For particularly anxious patients for the perioperative period, I would consider adding in:

- Trazodone 2.5-5mg/kg PO SID
- Clonidine 0.01mg/kg (more readily available than trazodone which requires compounding)
- Gabapentin 10-20mg/kg PO BID - TID
- Zylkene supplementation

For complex cases where a patient is clinically unwell and on multiple medications requiring a general anaesthetic, it is worthwhile consulting with a veterinary anaesthetist pre-operatively.

Gentle handling

When admitting patients with an anxiety disorder:

- Use a low level cage, preferably in a quite separate room from other pets/people
- Gentle handling techniques recommended
- See www.fearfreepets.com and www.drsophiayin.com for further information
- Southwest Veterinary Symposium 2018 conference proceedings article titled "Fear-Free Gentle Control and Handling Techniques" by Dr Debbie Martin is available on VIN
- Try to discharge the patient as soon as possible once recovered and stable to avoid unnecessary long stays in hospital

ADDITIONAL COMMENTS

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The only variation we have on this protocol is where the owner gives the normal dose late at night. (We have owners who work odd hours so pilling times can vary so say 10pm is the normal pill time).

In these cases, the dog is fasted overnight—then admitted at 8.30am—sedated in the consult room—and once sedated, taken from owner and operated on so the dog is back and awake in kennel before the effect of the previous night's meds have worn off; this avoids the double handling that Gretta so rightly highlights.

We now have a Behaviour Surgery Day where we reverse our clinic procedure and ops get done first and consults start later. On Behaviour Op days the clinic is almost cathedral quiet early in the morning and the anxious dogs and owners appreciate this.

We always stagger each surgical admittance by 20 mins so no dog goes in the kennel first but waits with its owner until sedation kicks in and is then taken and operated on; this helps keeps most dogs in the 'green light' zone. We do this for all surgeries (learnt the hard way in the UK doing a lot of Pit-bull and Rottie ops that avoiding the spike of anxiety getting such dogs out of a kennel 2 hours after they were in it all alone and in the 'red zone' makes for safer handling and smoother general anaesthetics).

We also do dummy runs in the weeks before elective surgeries on behaviour cases. This is where owner and dog come in as if for the op—they arrive, go into the consult room, then the owner and the dog walk to kennels and the dog goes into the run with owner. Both dog and owner get an appropriate edible treat and then both leave. This means the dog has a good first experience of the run and when it wakes up in it, it has some idea of where it is and that nothing bad happened last time. Post op, the owner comes to collect it from the run after all paperwork and payments processed so the dog goes straight out and home. This routine makes the return visit for suture removal (if not dissolvable) so much less stressful for both dog and for the vet.

SIMONE MAHER

Simone has joined the team as Deputy Director.

Simone's background is primarily in shelter medicine, having most recently been Chief Veterinarian of Animal Welfare League NSW, where as well as running a busy private clinic she treated myriad surrendered, seized and injured stray animals.

Following graduation from Sydney University in 2000, Simone started her career as a new graduate at the RSPCA's Yagoona clinic. From here she had a foray into media, writing weekly columns for the Sun Herald, monthly articles in lifestyle and children's magazines and was concurrently the resident vet on a Channel 9 lifestyle program.

Simone loves many aspects of general small animal practice: soft tissue surgery, building rapport with elderly clients, program delivery in disadvantaged communities, shelter medicine and the odd goat castration. She is excited about the prospect of building and expanding relationships and networks within the CVE and the wider veterinary community. Simone is interested in innovative and engaging ways of delivering education; loves a good practical joke (as long as she's the instigator); being outside; raiding her father-in-law's wine cellar and travelling of any kind.

Hypoglycaemic coma associated with intestinal lymphoma in a cat

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C&T No. 5735

An 8-year-old female neuter British Shorthair was presented in hypoglycaemic coma with seizures. She had a several year history of persistent diarrhoea and her diet consisted of supermarket dry food only.

Clinical pathology revealed absolute neutropenia, hypoglycaemia and likely sepsis. Insulin levels were (appropriately) undetectably low on a paired blood sample, and abdominal imaging (see below) showed no nodule or mass suggestive of insulin-secreting neoplasia.

Accordingly, the hypoglycaemia was presumed to be a manifestation of sepsis. She was stabilised with IV fluids, dextrose and antibiotics and abdominal imaging was performed. Abdominal ultrasound imaging showed spectacular jejunal thickening, including thickening of the muscularis layer. Maximal jejunal thickening was 3.4mm. Intestinal biopsy was offered but declined. Inflammatory bowel disease and diffuse small cell lymphoma were considered the most likely diagnostic possibilities, but small cell lymphoma was suspected due to the severity of clinical presentation and the sonographic changes.

Treatment was started with prednisolone 5mg sid and chlorambucil 2mg EOD.

She was also supplemented with subcutaneous cobalamin (B12) injections for the first 6 weeks of treatment and monthly thereafter, as well as Hills i/d diet® (canned).

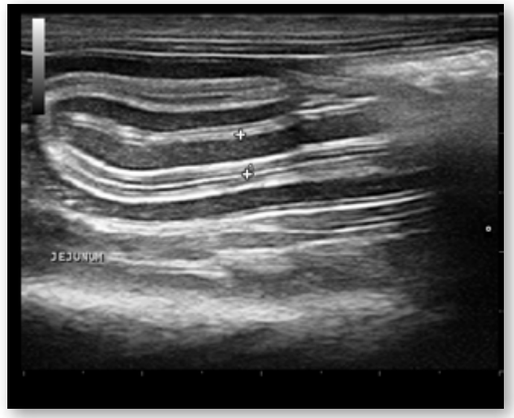

Figure 1: Abdominal imaging prior to treatment.

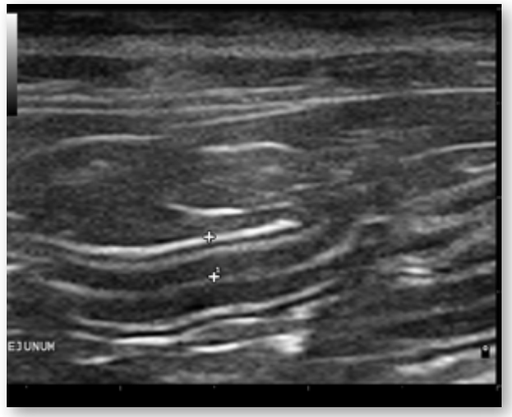

Figure 3: Repeat abdominal ultrasound showing reduced thickening of jejunal wall.

Figure 2: Before treatment.

Figure 4: 6 months into treatment. Weight gain from 2.6kg to 4.5kg.

Reply to C&T No. 5720 Alleviation of thermal strain after racing in the thoroughbred racehorse with the use of a cooling collar

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C&T No. 5736

I was totally captivated by this article.

What a clever, scientifically-based (cutting edge info re carotids and heat stress) utterly simple and effective device for horses. This is true 'Tom's paddock/Goanna Track' stuff where great veterinary ingenuity using simple and affordable concepts has the potential to ripple out across the paddock to the dog in the kennel as well as the horse in the stable.

So, can we translate this collar/carotid concept to small animals?

I am hoping I can translate that idea to heat-distressed canine hospitalised patients, as all I would have to do is fill my intravenous fluid bag heat pack covers (C&T No. 4891) with an icepack filler and, voila, my canine heat-distressed patients now get ice on their carotids. We had long tubular heat/cool packs to put in the groin and the axillae (Figs 1-4 C&T No. 4891) but had not thought about the strap-on carotid ice pack concept.

Preventative Cooling Collars are sold for dogs. I noted them in my simple 'Cooling Tips for Hot Dogs' blog post (C&T No. 5649) but have not directly promoted their use as the current models cover only about a 1/3rd of the neck. They are somewhat heavy and cumbersome,

so mostly only of use as an in-home cooling aid for non-emergency cases.

Now my new concern (having read this whole equine cooling article in full; a rare thing for me to do as a small animal vet) is whether the whole carotid cooling concept actually works in dogs at all? There are interspecies variations in carotid blood supply and presence (or not) of rete.

The work on carotid cooling in man has been extrapolated superbly to horses where similar anatomy ensures it is effective.

Can we claim the same effect in dogs/cats given the differences in their anatomy?

I would love an anatomy guru to comment on this so we can work out possible success or not.

Two other thoughts:

1. Given that we apply rugs to horses to assist them to acclimatise when we move them to colder pastures, should we be applying cooling collars to horses to assist them when we transport them to hotter climes?

2. Can this device be used in the ill pyrexic or injured horse?

Note: Aine has updated her client handout featured in C&T 5649. Please follow the link bit.ly/cool-pets-aine

Editor's Note: Meg is working on a cooling collar for racing greyhounds and we await the results with great interest.

eBook download:
C&T No. 5649 'Simple ways to keep pets cool and safe in summer'

Cooling interventions for dogs with the use of a cooling collar?

Comment on C&T No. 5720, Dec 2018 Alleviation of thermal strain after racing in the thoroughbred racehorse with the use of a cooling collar by M A Brownlow

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C&T No. 5737

What an absorbing read, this latest writing for C&T from Dr Brownlow (actually her series C&T Nos. 5376 and 5377 as well as Perspective No. 137), and while there are many differences in the way athletic horses overheat ('exertional heatstroke' from strenuous physical exercise) compared to our mostly less active canine companions, there is much we can learn from this fantastic treatise of Dr Brownlow.

May I also put in a plug for the excellent C&T No. 5649 by Dr Aine Seavers who shares her Client Handout titled 'Simple ways to keep pets cool and safe in summer—a client handout'.

You only have to google 'cooling collars for dogs' to see a host of collars that are reputed to be cooling, some with ice gel and some exerting their effect by water evaporation—to my knowledge, there hasn't been any studies to support or not support their use in the prevention and treatment of heatstroke in dogs. The horses' long neck makes it a better candidate for long collars that one would assume improves efficiency of the cooling mechanism by more contact with the overheated cervical vasculature. The variable depths of cover for the targeted vasculature from the surface seen in obesity, and short thick necked brachycephalics as examples, would be likely to make heat exchange more problematic with these devices. Similar trials to those being done in horses would need to show potential or real benefits for us to have any confidence in recommending such devices. Despite the paucity of evidence of benefits in dogs, the concept looks very sound and I, for one, look forward to hearing more of Meg Brownlow's excellent research in horses, and perhaps someone will be able to emulate this in dogs also —performance dogs as athletes with comparatively longer leaner necks would be the ideal candidates to study this, I imagine.

The reason a companion canine develops heat-induced illness usually is multi-factorial leading to the inability to dissipate heat (Johnson, SE *JVECC* 2006, 16 (2), 112-119). It is a consequence of exposure to a hot and humid environment ('classical') or from voluntary strenuous physical exercise ('exertional') or severe, uncontrolled tremors or seizures (Bruchim, 2016).

Exogenous predisposing factors that decrease heat dissipation:

- Confinement and/or poor ventilation - decreased conduction, convection, radiation, and evaporation
- Increased humidity - decreased evaporative heat loss
- Water deprivation - decreased blood volume that leads to decreased cutaneous vasodilation and cooling

Endogenous predisposing factors that decrease heat dissipation:

- Lack of acclimatisation - decreased neurohormonal responses; acclimatisation (a time dependant process) engages adaptive changes, both behavioural and physiological, that enhances the individual's ability to handle excessive environmental heat
- Brachycephalic anatomy - inadequate ventilatory capacity
- Obesity - decreased heat dissipation and decreased ventilation (increases the body's natural thermal isolation)
- Cardiovascular disease - decreased cardiac output
- Neurological/neuromuscular - altered thermoregulatory function; decreased ventilatory capacity
- Age (geriatric, or the very young) - poor acclimatization, compromised cardiovascular response, and deficient voluntary control

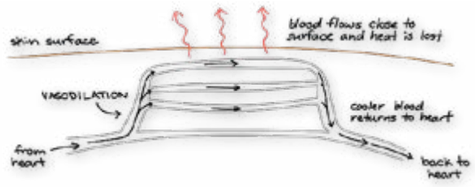
- Hair coat and colour - darker coats absorb more heat; thicker coats decrease radiation and convection
- Prior occurrence of heatstroke—affects the thermoregulatory centre in the preoptic zone which is responsible for heat sensation and dissipation
- Body weight (dogs >15kg are overrepresented in clinical cases)—large breed dogs are predictively more at risk of developing heatstroke, especially exertional heatstroke, implicating the ratio between body size and surface area as a crucial factor in heat dispersement during heat stress (Bruchim Y *et al, JVIM* 2006; 20(1):38-46)
- The 'rapid heat shock response' is an adaptive mechanism that is entreated directly to fight heat stress; it ropes in the production of HSPs (heat shock proteins) which are cytoprotective (Howowitz M, 2002). Bruchim's 2014 prospective study in military working dogs showed a compelling positive correlation between very significant enhancement of aerobic power and physical performance with the enscription of increased HSP72 mRNA; there was also a profound rise in serum HSP72 levels in these dogs at rest and after strenuous exercise (Bruchim Y *et al J Appl Physiol* (1985), 2014;117(2):112-118). HSP72 concentrations in serum have been advocated as a prognostic indicator in heat stroke (Dehbi M, 2010). Bruchim Y *et al, J Comp Pathol* 2009;140(2-3):97-104 assessed HSP72 concentrations in 30 dogs with classical heatstroke and found improved outcomes with more rapid recovery of the levels while hospitalised. Conclusions from both these researchers were that HSP72 is a reliable biomarker in heat stress as well as being protective and aiding improved recovery.

Dogs pant when they're hot but it's not the only way they regulate body temperature. Thermoregulatory strategies that have adapted for the dog involve behaviour, anatomy, and physiology.

Heat loss mechanisms include such behavioural strategies as postural changes, seeking shade or at least a cool environment, and grooming (in the cat).

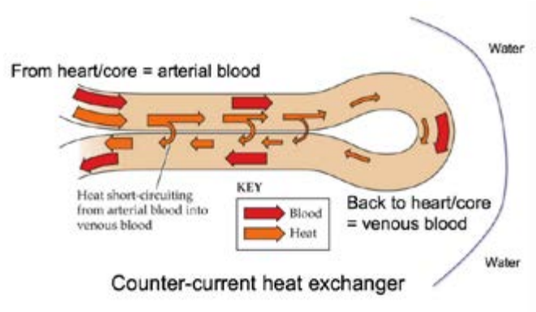
Dogs also have body structures and physiological responses that control how much heat they exchange with the environment:

1. i. Circulatory mechanisms, such as altering blood flow patterns.
   The body's surface is the main site for heat exchange with the environment. Controlling the flow of blood to the skin is an important way to control the rate of heat loss to—or gain from—the surroundings. In endotherms, warm blood from the body's core typically loses heat to the environment as it

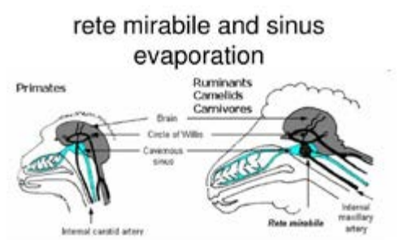


passes near the skin. When needing to lose heat, vasodilation increases blood flow to the skin and helps the animal lose some of its extra heat to the environment. In dogs, it is said that over 70% of total body heat loss is dissipated through radiation and convection from the body surfaces (peripheral vasodilation).

ii. Many mammals have countercurrent heat exchangers, circulatory adaptations that allow heat to be transferred from blood vessels containing warmer blood to those containing cooler blood, usually reducing heat loss.



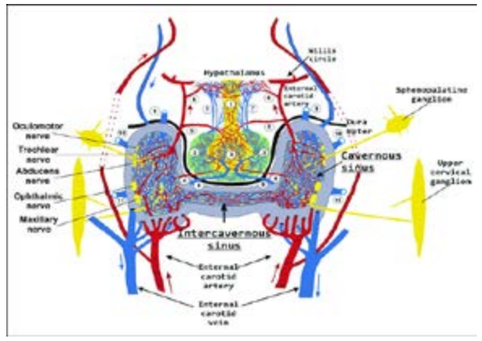
2. Insulation, such as fur and fat.
3. Evaporative mechanisms, such as panting and sweating become more important in maintaining normothermia as the environmental temperature rises to close to body temperature. There is a large surface area for water loss from the nasal turbinates which are essential in evaporative cooling; hypersalivation improves this evaporation efficiency. However, high humidity (>35%) reduces evaporative competence, and evaporation is effectively annulled when environmental humidity reaches 80%.



Brain (site of thermoregulation) remains cooler than body during heat stress

In dogs, evaporative cooling from panting combined with a countercurrent heat exchanger (the rete mirabile) helps keep the brain from overheating.

Although written on racehorses suffering exertional heat stress, there are some very relevant quotes, and paragraphs in Dr Meg Brownlow's recent review of the equine cooling collar for us small animal practitioners, and I iterate some of these below for our interest:



Selective Brain Cooling (SBC)

'In the dog, the temperature of the cerebral arterial blood is the major determinant of brain temperature. The panting carnivore can keep the temperature of the brain below that of the body during times of heat stress. This is achieved by a special arrangement of the carotid artery called a rete which is a compact network of intertwined arteries that lie submerged within the cavernous sinus at the base of the brain. The rete structure allows constant conductive heat exchange between the incoming "hot" arterial blood and the exiting venous blood which has been cooled by evaporation from respiratory surfaces. The presence of the rete structure in those species allows brain and body temperature to dissociate, particularly when conditions are hot and during exercise-related thermal stress. This lowering of brain temperature below arterial blood temperature has been referred to as selective brain cooling (SBC).'

'The basic cooling principle of a rete is that the blood flow is slowed and there is a relatively large surface area for heat exchange to take place between "hot" and cool blood.'

Cooling the Neck Region in Humans

'Cooling collars have been used in human athletes (Tyler and Sunderland 2011a; 2011b) and in the military (O'Hara et al., 2008). The intended purpose of these devices in humans was their use during sporting competition or battlefield exercises to maintain performance.'

'Subjectively, neck cooling in humans was found to more effectively alleviate heat strain than cooling the same surface area of the trunk (Shvartz 1976). Palmer and colleagues (2003) also found that cooling the neck region in humans during high intensity exercise attenuated the rise in brain and core temperature and improved the perception of physical effort and thermal strain. Participants were able to tolerate higher rectal temperatures and higher heart rates when their neck regions were cooled compared to when they were not cooled. It was documented that application of ice packs to the lateral surface of the neck could reduce brain temperatures between 0.2°C and 0.5°C (O'Hara et al 2008; Palmer et al 2003; Gordon et al 1990).'

'Further to these effects and most importantly, it has been shown experimentally that cooling the wall of the carotid artery induces dilation, which has significant upstream effects, increasing blood flow and perfusion of the brain (Nybo et al 2002; Zhu 2000; Godon et al., 1990). Mustafa and Thulesius (2002) demonstrated experimentally that heating the carotid artery (as might occur with exercise induced hyperthermia) elicited a vasoconstrictive response in proportion to temperature elevation, which resulted in decreased cerebral blood flow and directly contributed to cerebral ischemia, which is a major factor in the pathophysiology of heatstroke. These authors concluded that cooling the neck region and thereby the cerebral supply vessels represented a promising therapeutic strategy in that it might eliminate or diminish vasoconstriction and trigger an additional vasodilation, effectively reversing the cerebral pathophysiology associated with exercise-induced hyperthermia.'

Cooling collar mechanism of action

'The mechanism of action of the cooling collar is yet to be determined. Some studies in humans suggests that cooling collars may cool the blood in the carotid arteries as it passes to the brain and if the brain is kept cool, tolerance to elevated core body temperature may be extended and signs of heat illness more easily controlled. Others suggest that the collar just acts as a 'heat sink' removing heat from the skin, decreasing body temperature and reducing the perception of hyperthermia by the animal and thus reducing thermal strain as an effect simply of cooling skin thermoreceptors in the area of the horse's neck. It is also quite possible that the collar is cooling venous blood in the jugular vein. This implies that any effect on the brain is a consequence of a decrease in the temperature of the blood coming up from the heart and in this instance the collar might be inducing general body cooling and secondarily cooling the brain. The carotid arteries supply the brain with the majority of its blood flow. If the collar can cool the blood flowing in the carotid arteries there might be two positive effects: firstly, if the temperature of the blood going to the brain is reduced, heat will be lost from the brain simply by convection because of the temperature differential; and secondly, cooling the arteries might also cause them to dilate, increasing blood flow through the brain.'

Conclusion

'The cooling collar may be a promising therapeutic strategy for exercise-induced hyperthermia in all sporting horses and in particular thoroughbred racehorses. The supposition is that if the brain can be kept cool, tolerance to elevated deep body temperature might be extended

and signs of heat illness may be more controllable or entirely negated. It must be emphasized that the cooling collar is only recommended for use as an adjunct to whole body targeted cooling and does not replace it.'

References/Quotes from references from Dr Brownlow worth reading by canine enthusiasts

'Cooling of carotid artery preparations induced a reversible graded vasodilatation and decreased or abolished the effect of vasocontractile neurotransmitters. The effect of local hypothermia could increase cerebral blood flow...' (Mustafa S & Thulesius O, *Stroke*. 2002; 33:256-260.)

'Many of the cooling methods and devices detailed in the literature are impractical for use in the field. Future research should focus on cooling technologies that are practical in the battlefield and have sustainable cooling effects…….' (O'Hara R, Eveland E *et al, Military Medicine,* 173, 7:653, 2008)

'Influence of the carotid rete on brain temperature in cats exposed to hot environments' Baker MA, J. *Physiol* (1972), 220, pp.711-728. This full paper is worth reading .

'Blood supplying the brain appears to be cooled via a countercurrent heat exchange with cool blood draining the nasal mucosa (in the carotid rete)' (Taylor CR & Lyman CP, *American Journal of Physiology*, Vol. 222, No. 1, January 1972

'In alert, resting dogs, the brain is warmer than arterial blood in the common carotid artery. When dogs run, brain temperature drops, despite a sharp rise in carotid blood temperature, and is maintained 1.3°C below carotid temperature during exercise. This brain cooling apparently results from countercurrent heat exchange between warm arterial blood supplying the brain and cool venous blood draining the nose and mouth. The heat exchange occurs in the arteries at the base of the brain, which form a rudimentary carotid rete in the dog, and is greatest during exercise, when respiratory evaporation is at a peak. In animals with a carotid rete, the brain is protected against overheating during the severe thermal stress of exercise.' (Baker MA, Chapman LW, *Science* Vol 195, Issue 4280, February 1977)

'The largest single source of arterial blood supplying the brains of dogs and cats comes from the maxillary artery over an anastomotic ramus leading to an internal rete mirabile enmeshed in the cavernous sinus'... 'Extensive extracranial arterial anastomoses located in the orbit, retia mirabilia and neck musculature are significant and make it difficult to render the brain ischaemic.' (Gillian LA, *Am J Ana*t 1976 Jul; 146(3):237-53

'The rise in brain temperature during tracheostomy breathing increased with increasing ambient temperatures. Brain temperature always rose more than carotid arterial temperature during tracheostomy breathing in warm environments, suggesting that carotid arterial blood is cooled on its way to the brain in dogs which are breathing through intact respiratory passages…' (Baker MA, *et al, Respiration Physiology* Vol 22, Issue 3, December 1974)

eBook download:
C&T No. 5720. Alleviation of thermal strain after racing in the thoroughbred racehorse with the use of a cooling collar.

WINNER!

SMALL

Cryptic presentation of nasopharyngeal lymphoma in a cat

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C&T No. 5738

Sassy is a 3-year-old neutered female Oriental. She presented to the original veterinarian on 25/05/2018 for anorexia. She had a 5 day history of vomiting shortly after eating and had lost weight. Clinical examination revealed no abnormalities. The clinician took a conscious lateral radiograph which he thought demonstrated food in the stomach although the rest of the intestines were empty. He recommended paraffin oil and was investigating the option of endoscopic exam.

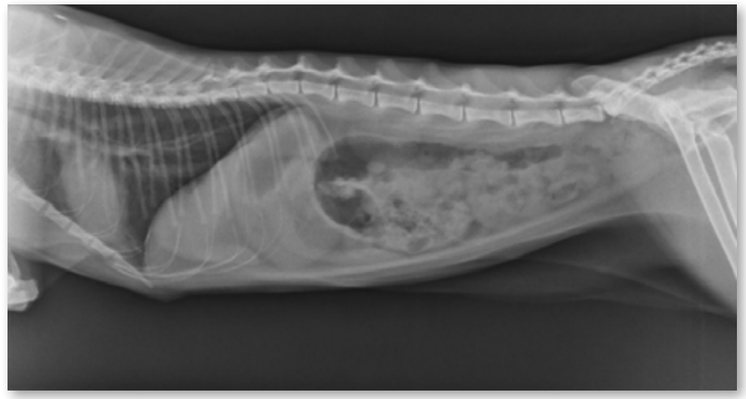

Figure 1: Lateral abdominal radiograph taken on initial presentation. Note air in the colon possibly suggestive of earlier aerophagia.

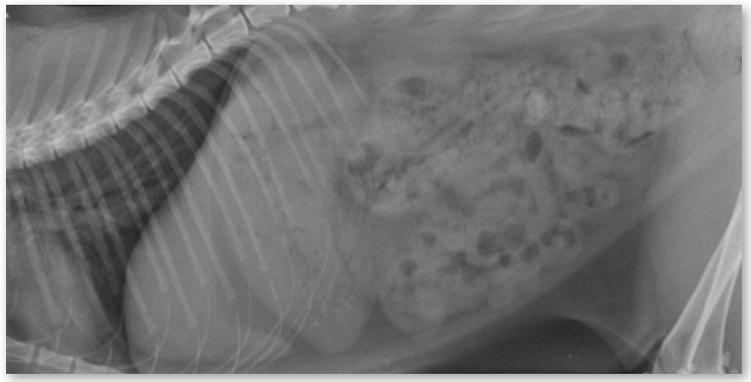

Figure 2: Radiograph taken 8 days later.

Sassy presented to our clinic 5 days later. Her owners had stopped all her dry food and were feeding her boiled chicken, sardines and paraffin oil. They were advised to give her 6 mLs of paraffin oil 4 times per day. They were concerned that she had started to make a 'gurgling' noise during respiration and she was slightly more laboured in her breathing. They also thought she had lost nearly 2 kg in weight. They mentioned that Sassy had not defecated for 5 days. According to her owners she had not vomited since her initial visit to the original veterinarian as he had given her something to stop vomiting, but this was not recorded in her clinical notes.

On first clinical examination she had a BCS of 3/9, weighed 3.6 kg and was bright and alert. She had a strong gag reflex and cough with gentle tracheal pressure. Her intestines felt thickened but no obvious mass was palpable. She was purring which made thoracic auscultation difficult to assess for a wheeze and there was no obvious stertor or stridor. She had no discharge from her nose. I did not think that Sassy was laboured in her breathing and nor did she have increased effort. She was purring, however, so a respiratory rate assessment was not possible.

We sedated Sassy (using Zoletil® 5mg/kg IM) with the aim to collect bloods for a total annual health profile (Idexx), take thoracic and abdominal radiographs and to do an abdominal ultrasound. Lateral abdominal radiographs looked similar to the previous veterinarians radiographs taken 5 days earlier. It appeared that she possibly had something within her stomach, almost empty intestines and small amounts of faeces at the pelvic canal. Thoracic lateral radiographs were unremarkable, with a VD difficult due to her conformation and incomplete sedation.

On abdominal ultrasound I noted that her intestines were thickened (0.3 cm), she had multiple prominent lymph nodes and her stomach was filled with something, mostly likely food and maybe a fur ball (see images). I noted that either a lot of food or liquid was moving through her intestines or they appeared hyper motile on ultrasound. The remainder of her abdominal ultrasound examination was unremarkable.

Urinalysis results were unremarkable.

Figure 3: Sassy. Watch the video in the eBook showing respiratory stertor.

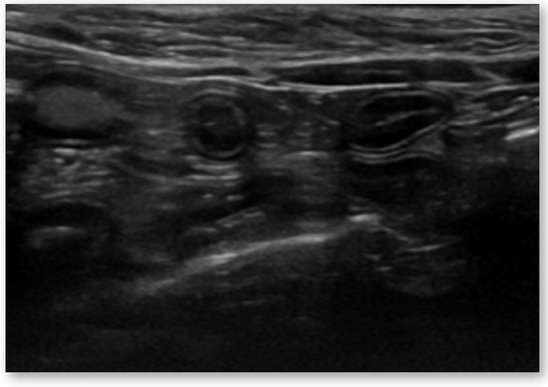

Figure 4: Abdominal ultrasound-note thickening of the intestinal wall.

I placed Sassy on Hartmann's (7mls/hr.) with 20 mmol/L KCl added and gave her intravenous maropitant (1mg/kg IV SID), metoclopramide (1 mg/kg IV BID) and started Doxycycline (Vibravet®) paste (25mg SID). She was not overly interested in food but was left with a variety of wet diets in her cage overnight. The reasoning behind the Vibravet® paste was because I was concerned that some of the paraffin oil may have gone into her respiratory tract when her owners were dosing her orally. Apparently she did not like it and it did tend to go everywhere.

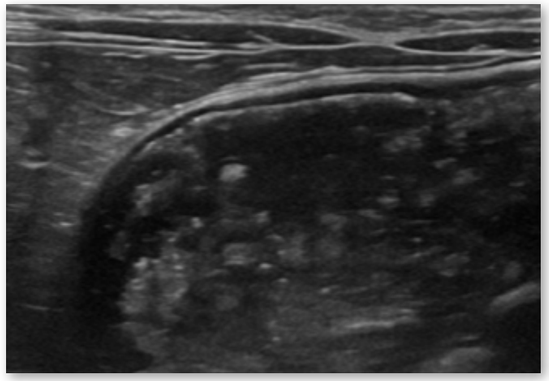

Figure 5: Abdominal ultrasound.

Figure 6: Faeces containing lots of fur.

Overnight Sassy ate all the wet food that was left for her, she had urinated but had not produced any stools. Her heart rate was 192 bpm, her respiratory rate was 36 bpm and she had a temperature of 38.1. She was bright and affectionate and interested in more food. We continued with her IVFT, fed her only wet food with MiraLAX®(polyethylene glycol 3350) (1/4 teaspoon) added and maropitant and metoclopramide IV at the same dose as the previous day. No increased respiratory effort, stertor or stridor were noted.

On day three Sassy produced a large semi formed stool which appeared to have large amounts of fur in it (see images). She was bright and affectionate. Her appetite was excellent and she had again eaten all the food that was left for her. Her medication remained the same. She had gained 70 g in weight. We repeated the lateral abdominal radiograph which showed that she still had something within her stomach but more stools within the large intestine. We planned to send her home the next day if she continued to eat well and produce stools. Her heart rate was 200 bpm, her respiration rate was 40 bpm and

temperature was 37.9°C. Again it was not noted that she had increased respiratory effort and no stertor or stridor were noted.

Day four Sassy was started onto oral maroptitant (1.06mg/kg, SID), oral metoclopramide (0.66 mg/kg, BID), Vibravet paste (25mg SID) and I continued with MiraLAX 1/4 teaspoon SID in wet food. Her appetite was excellent, she was eating 2-3 sachets of Whiskas 'Oh so fishy' per day and was very bright and alert. She had gained 150 grams since first presentation. We sent her home on her oral medications with the plan to see her back in 2 weeks for a weigh-in and depending on how she was going ,possibly repeating her abdominal ultrasound to take a look at her stomach again in 6-8 weeks' time.

Her owners phoned back 3 days later (6/6/18) to say she was eating well, had been defeating normally but they were concerned that she was still a little wheezy and possibly a bit snotty. Two days later her owners called again to report that she had gone slightly off her food, was very snotty and almost sounded obstructed in her nose. In particular her left nostril. They also thought she was a bit laboured in her breathing. They thought that she was like this prior to coming to our clinic the first time. Sassy was last vaccinated 12/5/16, but was a completely indoor cat with no history of boarding. The other household cat was completely normal. We dispensed another course of Vibravet paste (25 mg SID) as the last course was just about to end.

Six days later (12/6/18) Sassy's owners brought her back because she had gone off her food again, despite appearing to be hungry. Her owner thought that she was having trouble swallowing. According to her owners she had defecated only twice in the last 2 weeks. They had continued with the Vibravet paste but had stopped the Maropitant and Metoclopramide tablets for reasons they could not explain. She was also struggling to drink water and would gag and gurgle after attempting. They described a purulent discharge from the left nostril that waxed and waned and did not think that the antibiotics were making any difference at all.

On clinical examination **she had an obvious loud upper respiratory stertor** when breathing in (see video). She was much more subdued in her demeanor and had marked weight loss. Sassy now weighed 3.46kg. She was mildly dehydrated and her heart rate was slow at 140 bpm and her respiratory effort was increased. She became easily distressed with minimal examination. She had no discharge from her nares at this point.

I performed an in house smear exam of a cotton tip placed into her left nostril. There was a lot of squamous cells but no obvious bacteria and was pretty unremarkable. We started Sassy back onto intravenous fluids (Hartmann's with 10mmol KCl/500 mLs) at a rate of 8.6 mLs/hr. I gave her intravenous buprenorphine (0.015 mg/kg IV) and gave her Mirtazapine (1.8mg/cat) orally. It was my plan to GA Sassy the next day when she was better hydrated to

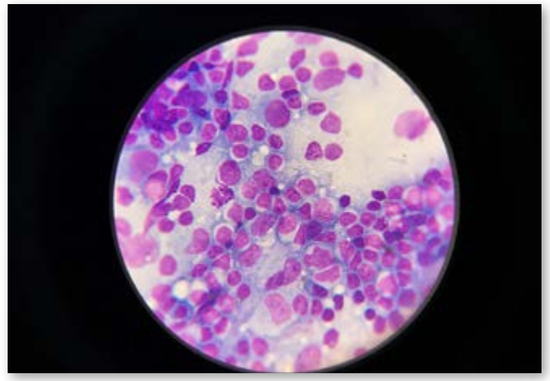

Figure 7: Cytology from nasopharyngeal tissue flushed out by vigorous hydropulsion.

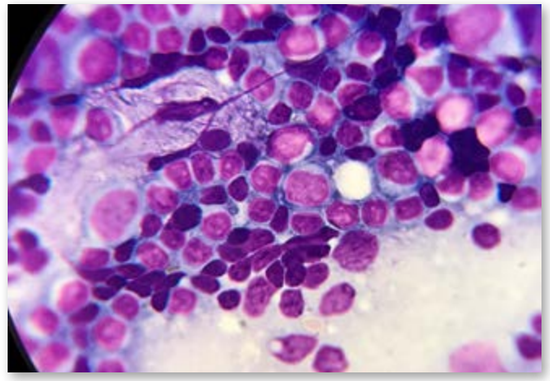

Figure 8: Nasal cytology: There is high cell recovery with the field of view dominated by medium to large round cells with scant to moderate blue cytoplasm consistent with the appearance of medium to large lymphoid cells. There are few scattered small lymphocytes and other cells present. The specimen appears consistent with nasal lymphosarcoma. The histology of the case confirmed this diagnosis.

radiograph, scope, flush and biopsy her nose depending on what we found at each step of the way.

Overnight Sassy had eaten all her food and was much more alert and affectionate, as she had been in the past. Obvious loud upper respiratory stertor was still present.

I used ACP (0.03 mg/kg) and methodone (0.3 mg/kg) as a premed intramuscularly. I induced her with Alfaxalone (1mg/kg IV) and intubated her with an un-cuffed size 3.5 ET tube. Her anaesthetic was unremarkable. We initially took nasal radiographs which were unremarkable. I also took a lateral skull radiograph to look for a pharyngeal polyp. With my limited experience, I scoped up the back of her nose to view her choana. On the left side I thought I could see some whitish lumps but there was a lot of blood which made examination difficult. There was no obvious polyp. I then packed the back of her throat with cotton swabs tied together & flushed her left nostril only with 10 mLs of warmed saline. When I was removing the swabs there was a lot of blood and mucous along with **a small piece of**

whitish pink tissue. We repeated the process but this time we got a large piece of the same tissue. Before placing this into formalin I wiped the piece of tissue onto a glass slide and set it aside. We repeated the process a third time and again got more small pieces of tissue. All were placed in the formalin pot.

Lastly I used nasal biopsy forceps and took 3-4 tissue samples from her rostral nasal passages.

Sassy recovered uneventfully from her procedure and anaesthetic. She was eating again within the hour and bright and affectionate again.

On examination of the glass slide I had stained using Diff Quik I was very concerned that Sassy may in fact have nasal lymphoma. The sample had numerous cells which look to me to be possible lymphocytes of varying sizes. (See images of in-house cytology)

On discussion with her owners we decided to send the two samples from her nose for histopathology.

Sassy was discharged the next day to her owners. She was much less laboured and noisy with her breathing. She was eating well and had regained 100 g in weight. She was still extremely bright and affectionate. She had defected normally again.

I sent her home on oral Mirtazapine (1.8 mg/cat SID), transmucosal buprenorphine (0.01mg/kg BID) and Vibravet paste (25mg/cat SID). Sassy was easy to medicate orally and it was my intention to get her as well as possible should her owners consider chemotherapy should the diagnosis of nasal lymphoma be confirmed.

Histopathology did confirm the diagnosis of nasal lymphoma from the pieces of tissue that were flushed out of the back of her nose. Interestingly the nasal biopsies taken blindly from the rostral nasal cavity came back with a diagnosis of chronic mild lymphoplasmacytic rhinitis.

Sassy is a complicated case because she presented initially for anorexia, vomiting and weight loss. It was not until she returned the second time that we had heard her noisy breathing or saw signs of a nasal discharge and stertor that we investigated her for nasal discharge. To be honest, I was thinking that she possibly had an inflammatory polyp and nasal lymphoma was not on my list of differentials. At her initial visit she most definitely had something within her stomach (confirmed on both radiographs and ultrasound) and thickened intestines and prominent lymph nodes which could have all explained her anorexia and weight loss. It was not until she re-presented with obvious stertor that we diagnosed her nasal lymphoma and this could have been completely missed and treated inappropriately had we not flushed her left nostril with saline and collected the material on the swabs.

Sassy lives over 2 hours from our clinic and due to a number of family factors and finances her owners have chosen not to consider chemotherapy despite the potential for a very good outcome. I am hoping that they may reconsider as she is one of the sweetest cats we have had the pleasure of treating.

I would like to acknowledge that Dr Richard Malik assisted me multiple times (via email) through the diagnostic and treatment process of Sassy, assisting with keeping me on track and keeping an open mind to the possible differential diagnoses.

BELATED CONGRATULATIONS!

The Investiture at Government House on Wednesday 5 September 2018. Dr Bill Howey (left) is presented with his medal from NSW Governor David Hurley.

Dr Bill Howey awarded Order of Australia Medal (OAM)

Dr Bill Howey, Director 2000 to 2002 of the CVE then known as the Post Graduate Foundation in Veterinary Science or PGF, was announced a recipient of the Order of Australia Medal for service to veterinary science in the Queen's Birthday Honours list on 11 June 2018.

Referring to himself often as a '10 pound Pom', Bill has dedicated himself to the veterinary profession and public service.

Many of our members and readers will know Bill through his long association both with the PGF and the Scone Equine Group. Those with a love of history and keen interest in equine medicine will enjoy visiting Bill's website http://sconevetdynasty.com.au

See page 22 to read a short précis of an article co-authored by Bill: The evolution of modern equine practice in Australia

The impossible diabetic

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C&T No. 5739

You know the scenario— the scraggy polydipsic cat that needs sedation or restraint for blood collection, and your heart sinking when the glucose hits the 20's. Sorry mum/dad, your cat really isn't a suitable cat for insulin injections never mind the monitoring blood tests. Euthanasia beckons.

But hang on, why is the cat so freaky? Nasty cats don't exist—frightened, abused, or anxious cats do, so why don't we address this in order to give the cat some peace and to permit medication and monitoring?

As a slight aside I reckon that a high proportion of our cats are held hostage to their emotions and may lead tortured lives due to fluoxetine (Prozac) deficiency—but that is another story for another time.

So enter Tabitha, a proteinuric chronic renal disease (CRD) cat who is content and happy at home with mum, but has never been a cuddle cat and who reacts violently to strangers in the house—and at the vets. Her diagnosis was during a 30 degree heat-wave (that's hot for the UK!) so she was immediately admitted for fluids and confirmation of diabetes. Blood glucose was 15 mmol/L with 2+ glycosuria, SG 1020 (previously 1010). Bloods were sent for fructosamine, and urine was collected overnight into Katkor (non-absorbent litter) for short term differentiation of stress hyperglycaemia. Fluoxetine was commenced 7mg *per os* along with gabapentin 25mg BID. Continued glycosuria the following morning was the prompt for commencement of glargine 1unit BID.

With a more sedate Tabitha the following evening, the owner was instructed on how to inject, and watched me administer the insulin. The following day she managed it herself—a major achievement in everyone's eyes. Glycosuria persisted but the fructosamine came back as 340—indicative of high non-diabetic or reasonably controlled diabetic! Had Tabitha become diabetic so recently that there had not been enough time elapsed for a high reading to develop?

Urine glucose actually increased to 4+, so insulin was increased to 2 units BID. A week after initial diagnosis, urine glucose was still 4+, BG 13-15mmol/L and fructosamine was repeated—and had dropped to 303!

With hypoalbuminaemia and hyperthyroidism excluded it was concluded that Tabitha may be one of the cats for whom fructosamine is not helpful. Ear pricks performed by our nurse in the home environment confirmed continued mild hyperglycaemia of 14-15 mmol/L—eliminating, in my eyes, the possibility of renal tubular leakage of glucose in a non-diabetic patient.

The question then was: how were we to monitor Lily's blood glucose when urine is glucose-free. We had switched her diet to minimal-carb high-meat, and had obvious concerns of hypoglycaemic episodes if she went into diabetic remission. One thought was to reduce the insulin until glycosuria returned and then drop the dose by one unit.

Enter Freestyle Libre, a human diabetic monitoring device recently introduced and taking the world by storm. A 35mm disc with a central wire is applied to the skin with the wire bathed in interstitial fluid. Whilst it does not measure blood glucose there is excellent correlation with a time-lag between blood and interstitial fluid levels. Measurements are taken every 15 minutes on a rolling 8 hr recording period, with a sensor lasting for 14 days. At the end of this period it ceases to work. It can be purchased with a reader but can also be used with an iPhone 7 or later, or Android 5 or later, obviating the need for the reader. The device costs around AU$92.50 and the reader a similar sum.

There is a blue-tooth device called 'linkblucon' ambrosiasys.com which is an extra device which attaches to and which trebles the size of the sensor and allows real-time remote monitoring. Not really an option, nor indicated, for our kitties!

A major consideration: once the device has been paired to a phone it cannot be changed, so don't test it on your own phone unless you want to surrender it to your client for 2 weeks!

The Libre device comes with its own applicator. The skin is cleansed, wiped with alcohol and allowed to dry (without blowing on it) and the applicator is pressed onto the skin. Whilst the device is self-adhesive, a tacking agent can be used for extra security (Torbot Skin Tac™). Once applied there is a narrow fabric fringe which can be superglued to the skin. Discussions on VIN mention the occasional need for body-bandages.

Application of the reader went without incident, and we lowered the Gabapentin over the first week, coming off it in

10 days, at which point we started reducing the fluoxetine dose to 5mg with a view to reducing it to the minimal effective dose. The initial heavy sedation not only permitted us to perform all the tasks we needed but also gave the owner the confidence to learn the technique in a compliant patient. The cat quickly took to being injected while she was eating and we aim to have her off anxiolytics, if possible.

So far the device is working a treat, and the ease of getting glucose curves means that hourly ear-pricks or foot pad pricks can potentially be relegated to the history books for any diabetic never mind the tricky ones.

So for the next unmanageable case bring on the gabapentin and Prozac, and the Freestyle Libre!

Applying Freestyle Libre

1. Clip the chosen area—back of neck or shoulder. My latest cat was dehydrated and emaciated; sub-cut fluids prompted a useful thought, creating a soft cushion to which the patch could be applied! This will make it much easier to apply it to the base of the neck.

2. Cleanse the clipped area with alcohol.

3. Apply Skin Tac™ or equivalent if you use an adhesive agent—otherwise the patch is self-adhesive.

4. The dispenser and applicator are 'mated' and the applicator is pressed firmly onto the chosen site.

5. Spots of cyanoacrylate glue can be applied to the exposed fringe.

Post script

This article was written in our first flush of enthusiasm for the device. Whilst the latter doesn't wane there are certainly a few important considerations.

Application

Lateral chest gives a flatter base.

Some cats seem irritated—either by the device, or where the glue is used. Application of 'soft-nails' will reduce self-trauma; alternatively a neck sock will protect.

Removal

Geriatric cats often have very thin skin and a firmly adhered device needs very careful teasing off to avoid tearing the skin, especially at the glue points. An adhesive remover such as Detachol can help here.

We have had devices stop working—I think because the probe comes adrift from the skin; I think this is an unavoidable hazard in thin skin.

Technology

One aged couple struggled with the technology, simple as it seems to us.

The constant recording means that lows are clearly identified and this can create great client anxiety. The lack of any clinical signs of hypoglycaemia associated with these 'lows' is interesting and deserves further consideration.

Case end—result

Tabitha is now in diabetic remission with no insulin required. She remains on low dose fluoxetine as this has had beneficial effects on her psyche.
She is now hyperthyroid!

Figures 1: The freestyle Libre device.

Figures 2: The device comes with its own applicator.

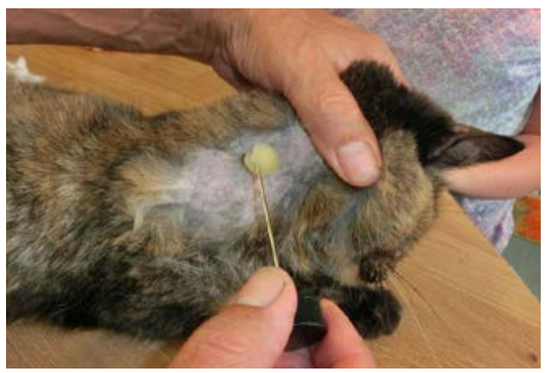


Figure 3: Cleanse the clipped area with spirit. Apply Skin Tac™ or equivalent if you use an adhesive agent; otherwise the patch is self-adhesive.

Figure 4: The applicator is pressed onto the skin.

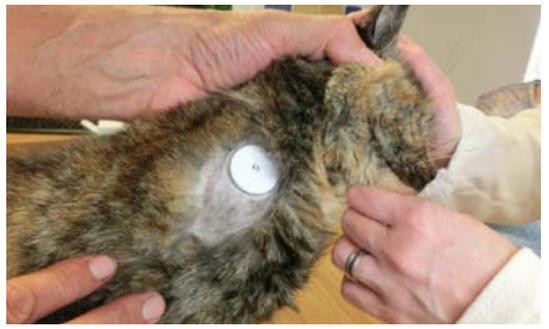
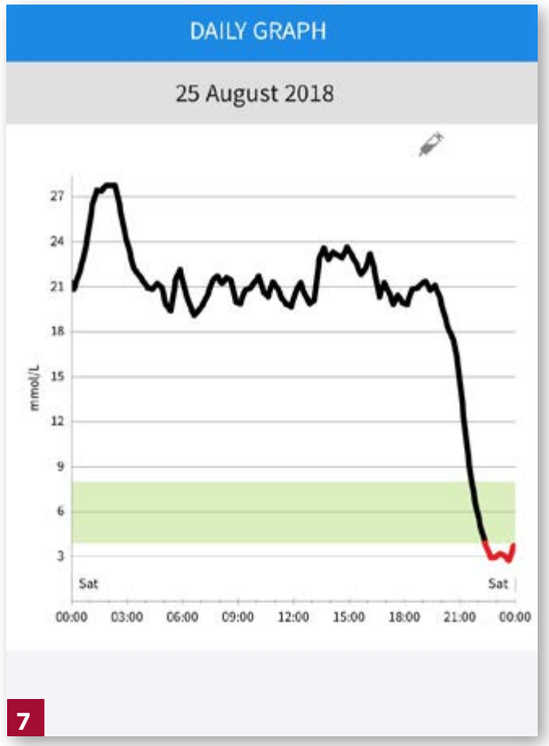
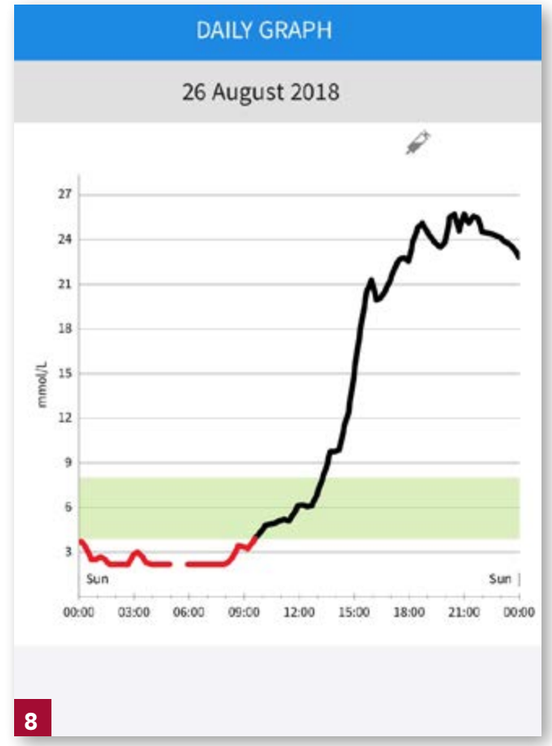


Figure 5: Freestyle Libre attached over area raised by sub-cutaneous fluids. Note narrow fabric fringe.

Figure 6: Fabric fringe can be superglued to the skin.

Figures 7 & 8: 24 hours of readings.





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Problem Solving in Internal Medicine Conference (Feb 2018)

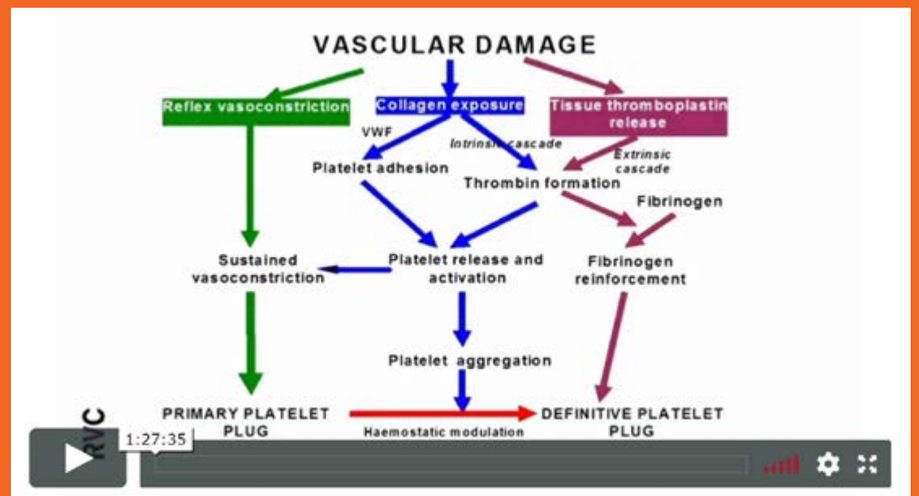

Image from The Problem-based Approach to the Anaemic or Bleeding Patient presentation by Jill Maddison

Each month in the CVeNews we advise which videos from our major conferences have been uploaded for viewing by Members in the CVE Video Library.

This conference provided a review and update across a variety of internal medicine topics using a logical, problem-based approach to key clinical issues. Videos available:

Jill Maddison BVSc DipVetClinStud PhD FACVSc SFHEA MRCVS

- Clinical Reasoning in Clinical Practice: Making the most of the initial consultation
- Assessing the Patient with Vomiting & Diarrhoea: When should I worry?

Caroline Mansfield BSc BVMS MVM PhD MANZCVS Dipl. ECVIM-CA

- Diagnosis & Management of Canine & Feline Pancreatitis in the 21st Century
- Understanding Inflammatory Bowel Disease Caroline Mansfield What's new and what does it mean for my patients?

David Church BVSc PhD MACVSc MRCVS presents:

- Coughing, Sneezing & Dyspnoea
- A Logical Approach to Polydipsia and Polyuria
- Updates in Diabetes Management in Cats and Dogs - Is there a 'best' insulin?
- Hypercalcaemia – Ignore at your peril

Niek Beijerink DVM PhD Dipl. ECVIM-CA (Cardiology) presents:

- Diagnostic Tools in Cardiorespiratory Medicine – What, when and where?
- Cardiac Therapeutics – Choosing the best drugs for dogs presented with heart disease
- Cardiac Therapeutics – Choosing the best drugs for my feline patient

Note: Members must log into the CVE website to access the 250+ videos available.

cve.edu.au/video-library

How would you treat this case: 8-year-old female neutered Kelpie with vaginal bleeding?

Elizabeth Thrift

Northside Veterinary Specialists
335 Mona Vale Rd, Terrey Hills NSW 2084

C&T No. 5740

An 8-year-old female kelpie who had been desexed presented to the veterinary clinic 8 months later with haemorrhagic vulval discharge. She was prescribed antibiotics. When rechecked almost one month later, the owner reported the discharge had largely stopped. However, blood was noted on vaginal examination.

She presented to me for further assessment. An abdominal ultrasound was performed with the following results:

1. The left adrenal is characterised by a prominent caudal pole containing a well demarcated hyperechoic rounded silhouette. Given the morphological features, adrenal hyperplasia is suspected. Other possibilities include small myelolipoma or adenoma.

2. Caudal to the right kidney and in association with the retroperitoneal adipose tissue, there is a rounded well demarcated hypoechoic structure/nodule without visualization of hilar region measuring 1.0cm by 0.5-0.7cm. No visualization of cystic regions. Small right retroperitoneal nodule. Possibilities for this lesion include lymph node, granuloma, residual ovarian tissue.

3. The uterine stump is prominent. No visualization of uterine horns. No evidence of underlying uterine pathology at this stage.

Her Anti-Mullerian Hormone (through Vetnostics) was elevated at 0.9 (<0.6 for a neutered female).

Taking all these findings, the working diagnosis was ovarian remnant syndrome (ORS), possibly with stump endometritis or early pyometra.

COMMENT COURTESY OF

Dr Fiona Hollinshead BVSc(Hons) PhD MANZCVS DipIACT Registered Specialist in Small Animal Reproduction

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I agree, ORS would be first on my list of differentials with a recent history of ovariohysterectomy (OVH) and then a bloody vulval discharge 8 months later. However, it is important to rule out all other causes for a bloody vulval discharge such as vaginitis, vaginal neoplasia, foreign body, uterine stump pyometra (this is often a sequelae to ORS), exogenous oestrogen exposure, coagulopathy and urinary tract infection. A complicating factor would be if she had a granulosa cell tumour which is something to consider given her age.

Further diagnostics would initially include a vaginal cytology if the bitch was still showing signs of heat or oestrous. This is a simple, cheap tool that provides very important information on the presence of oestrogen as well the possibility of an inflammatory process also occurring (i.e. pyometra). Greater than 50% cornification/keratinisation of the vaginal epithelial cells indicates oestrogen is present. The most likely source of oestrogen is ovarian. Personally, I find little value in measuring blood oestrogen concentration unless it is part of a stimulation test. Measurement of progesterone can also help determine if ovarian tissue, specifically luteal tissue, is present. Concentrations greater than 2ng/mL are indicative of ovarian tissue presence. However, progesterone will only be detected if the bitch is in dioestrous (2 month period following oestrous).

Measurement of LH (lutenising hormone) can be used as part of a GnRH stimulation test. However, it can also be helpful as a one-off measurement. If LH is high (>1µg/L) this indicates no ovarian tissue is present unless the blood sample happened to be taken at the time of the LH surge. If LH concentrations are low (<1µg/L) this indicates the presence of ovarian tissue and negative feedback. The advantage of LH is that it can be measured at any stage of the cycle, unlike oestrogen when the bitch needs to be in pro-oestrous or oestrous or progesterone when the sample needs to be taken during the 2 month period that the bitch is in dioestrous. However, not all labs offer this assay especially as the gold standard is to use radioimmunoassay (RIA) for a quantitative result. There are commercially available semi-quantitative canine specific LH kits (Witness, Synbiotics) which have recently shown to be helpful in diagnosing ORS cases (Alm and Holst 2018).

AMH is only produced by the granulosa cells of small growing pre-antral follicles. It is not produced by the adrenal glands. Therefore, AMH can be used at any stage of the bitch's cycle to detect the presence of ovarian

tissue; making it the first test I run in a suspected ORS case. However, it is important to ask the lab which AMH assay they are using. There are two commercially available AMH assays that have been reported for use in canids. There is a human-specific ELISA assay (Gen II ELISA) by Beckman Coulter which is the most commonly used assay in veterinary laboratories. Take care when interpreting results from this assay for bitches. I do not believe this human-based assay cross reacts well with canine antibodies which makes the sensitivity of this assay in detecting the presence of ovarian tissue variable, particularly in prepubertal animals (Place et al., 2011). I suspect the BD assay was used in this case with the low total AMH values reported. Therefore, positively, the elevated AMH result is strongly supportive of the presence of ovarian tissue in this case. However, if the AMH has been low and below the reference value I would be running additional tests (LH) as discussed above. Interestingly, this human based assay is highly sensitive for the detection of ovarian tissue in queens (Axner and Strom Holst, 2015). The other commercially available AMH assay is made by ANSH and is a canine-specific ELISA assay. It is for this reason that I think it has greater sensitivity and accuracy in the detection of ovarian tissue presence regardless of the bitch's age (Hollinshead et al., 2016). It is my assay of choice for measurement of AMH in bitches and confirmation of OR.

Ultrasound detection of an OR is generally very difficult. In many cases it is an exercise of finding the needle in the haystack, so your ultrasonographer has done a fantastic job! I think this would also be the case for MRI…but I have no experience with this.

Unfortunately, definitive diagnosis and treatment is an exploratory laparotomy.

Either as part of a diagnostic stimulation test or to induce lutenisation /ovulation of the follicles present on the OR tissue prior to surgery, either GnRH or hCG can be given IV. Luteal tissue is pinkish/reddish (like a corpus luteum) and generally easier to identify than microscopic follicles. It is also safer to perform surgery under the influence of progesterone than oestrogen. Progesterone concentrations should increase > 2ng/mL after administration of GnRH or hCG or if the bitch is left to spontaneously luteinise the follicles on the OR. Accompanying this rise in progesterone, is a change in vaginal cytology from 100% cornified/keratinsed vaginal cells to <50% cornification and a cessation of vulval bleeding and swelling.

Technically ORS is caused by surgical error but I think it is very important that a surgeon is not 'blamed' for this occurring. Two studies in 1991 and 1995 showed no correlation between ability of the veterinarian and ORS incidence and both studies concluded the occurrence of ORS is not related to the ability of the surgeon. 'Seeded' (not ectopic—that is another debate) ovarian tissue is often found in the ovarian pedicle, in the remaining broad ligament (this is more common in queens due to the anatomy of the ovarian bursa) or in the abdominal cavity. Resecting the ovarian pedicles and any remaining broad ligament is important in these cases as these are the most common sites that ORs are found. Even if no ovarian tissue is grossly visible submitting for histopathology is critical—to confirm the OR has been removed and ensure all ovarian tissue has been taken out. The surgeon also needs to examine the omentum, all abdominal viscera (especially liver, spleen and kidneys) and the abdominal wall as although uncommon, revascularisation of a dropped piece of ovarian tissue is possible.

Exploratory lapratotomy and removal of the OR is the treatment of choice. However, given this bitch's age, non-invasive medical management is attractive but unfortunately can be unrewarding, frustrating and in some cases cause additional complications. A 12 month Suprelorin implant is a non-invasive option which is appealing for many cases BUT when used in older bitches it can result in unpredictable and undesirable side effects such as persistent oestrous. Given this bitch's older age this side effect is a possibility if this treatment option was taken. Long term use of a short acting progestagen at very low daily dosage to suppress her heat is an option also but there are a number of potential medical side effects, especially when used long term and in an older bitch. Also, she would need to be out of heat before starting or the risk of a stump pyometra occurring would be high. There are not many other medical options. Mibolerone (anabolic steroid) is cost prohibitive in Australia and NZ and long term would pose health concerns also. If she is left to cycle every 6-8 months it is not only inconvenient for the owners but she is at risk of developing a life-threatening stump pyometra and the risk of mammary neoplasia is also increased.

Unfortunately, in my opinion, exploratory laparotomy is the best treatment of choice for this bitch. I know this is not ideal in an older bitch with owners that have financial concerns. But long term, if successful, it will be the best choice for her health and long term client satisfaction!

ORS is not easy to treat and in many cases not easy to diagnose. Management of the client and their expectations/understanding of this surgical complication is important.

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Reply to: Why is it so hard to find a vet?

C&T No. 5724 Issue 293

1. John Baguley

Registrar, The Veterinary Practitioners Board of NSW

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C&T No. 5741

The object of *Veterinary Practice Act 2003* is to regulate the provision of veterinary services for a number of purposes including to promote the welfare of animals and ensure acceptable standards are met by veterinarians.

Within the constraints of its legislative functions, the Veterinary Practitioners Board (Board) is able to assist the profession in relation to this apparent shortage of veterinarians in a number of ways.

Firstly, it is possible for a veterinarian who does not possess the qualifications for full registration in NSW to be granted limited registration. Limited registration is granted for a specific purpose including to assist with development of practical skills to pass the National Veterinary Exam (after passing an English test and series of multiple choice questions) or where there is an identified need and no person capable of being granted full registration is available who has the necessary qualifications or experience to fulfil the role.

Limited registration is typically granted with specific conditions, including working under supervision, working only in a specific area and for a specific employer. Limited registration is generally only possible for up to one year.

For further information please review the Board policy Requirements for Limited Registration available from our website (Resources, Policies).

Secondly, the Board is able to assist the profession through data collection and dissemination. This has been limited to some extent in the past due to the lack of some data being collected (for example full time or part time employment) and the Board's database itself (it was not initially designed for this purpose).

Based on currently available data the Board has recently provided the following publications which may assist further discussion of this issue:

1. Where are the new graduates? (see Board-talk December 2017)
2. Number and movement of veterinarians in NSW (2018 AGM presentation)
3. Annual Report (see Statistics Section for each year)

The Board has made a number of changes to the database in recent years and is conducting a major revision of the database this year. As a result, more specific information will be available from the Board in the future to assist with workforce planning.

Finally, all Boards have agreed to assist the Australian Veterinary Association with dissemination of the Australian Veterinary Workforce Survey every 2 years and with reporting results from these surveys. This survey was specifically designed to assist universities, the profession and other stakeholders with workforce planning.

The Board will continue to assist the profession, universities and veterinary professional associations such as the Australian Veterinary Association in relation to this issue aligned with its legislative functions where possible.

2. Frazer Allan

Head of School and Dean
Sydney School of Veterinary Science
University of Sydney

C&T No. 5742

The most important role performed by veterinary schools is to produce graduates with the day one competencies required of the veterinary profession and expected by the public. This is a role that the Sydney School of Veterinary Science (SSVS) takes seriously.

Where there is an issue, such as the inability to attract veterinarians to rural Australia, it is the role of veterinary schools to work with all stakeholders (practitioners, the veterinary practitioners' boards, industry groups, the public at large, the AVA, and the State and Federal Governments) to firstly understand why this is occurring and then propose solutions.

As John Baguley has outlined in his response, the VPB can assist on the supply side through the use of limited registration provisions. The VPB can also provide data to inform good decision-making.

Veterinary schools have potential levers that they can pull but these have a medium- to long-term impact given that it takes a minimum of four years to produce a veterinary graduate. Schools can influence both the curriculum (and hence the student experience) and the student cohort through the setting of admissions criteria. The relative influence of these levers, compared to, say, the student experience when in rural practice, peer to peer influence, students' perceptions of veterinary life after graduation and a host of other variables, is currently unknown. We are about to embark on a study at the University of Sydney to try to unravel this fundamentally important question.

With respect to the curriculum, the views of the profession were taken into account when the new DVM program was created (noting that the first cohort of Sydney DVM graduates cross the stage in March this year). The School has provided students with greater exposure to large animals, with the aim of better preparing students for rural practice. We have doubled the hands-on classes involving horses and cattle. Compared to the BVSc program, classes in cattle pregnancy diagnosis have increased threefold. Students now undertake hands-on production animal classes each semester of the course in order to provide continuity of learning and exposure, and most importantly to build their confidence in dealing with these species. The number of sheep classes have increased and now incorporate two intensive training days at a large, University-owned commercial sheep and beef property, Arthursleigh.

Rural experiences are integrated in the DVM through compulsory rural placements. Students are required to complete half of their pre-clinical placements in a rural practice as well as at least one of their final year elective extramural placements. DVM students now enter final year with much more confidence in production animal handling, compared to BVSc students, and have opportunity to choose a placement in a rural setting of their choice. Together, this should foster a positive rural experience for students.

With respect to admissions criteria, the University offers the Early Offer Year 12 (E12) Scheme for domestic students coming from a school listed on UAC's Educational Access Schemes S01E list (https://www.uac.edu.au/assets/documents/eas/eas-S01E-school-list.pdf). These Schools are amongst the most economically disadvantaged or geographically isolated, with many rural schools represented. All successful applicants are guaranteed a place, provided:

- They meet the E12 ATAR (which for BVB/DVM is 5 points lower than students not applying through this pathway); and
- They meet admission requirements (commitment statement).

These students also receive a $5,950 scholarship.

During the selection process, postgraduate entry DVM students with a rural background also receive a selection advantage compared to those from an urban background.

Finally, the School would be delighted to support initiatives that rural practices wish to be involved with to increase exposure of DVM students to opportunities in rural practice. I have seen scholarship programs and 'Careers Days' work well in New Zealand. In the case of the latter, practices have the opportunity to mix and mingle with students and talk to them about the opportunities that exist.

In summary, the rural shortage is likely to be multifactorial and SSVS absolutely wants to play its part and help with a solution. We need to make changes in an evidence-based way or in the absence of evidence, ensure that mechanisms are in place to monitor what we do when we make changes.

3. Anonymous

C&T No. 5743

I have just been reading with interest C&T No. 5724: Why is it so hard to find a vet?

I graduated from Charles Sturt Uni in 2015 and am in job number 3—not something I'm particularly happy about but at least the first two jobs I could not have continued on with for much longer than I did. As a new graduate I was set to work in a 3-vet practice growing to 4 vets and supposedly 'new grad friendly' with plenty of support, but which ended up being down to 2 vets and mostly sole charge work within 6 months of starting. Any suggestions I had for how to improve how things were being run were listened to at the time but not heard, and ultimately my love for my job was waning rapidly. I left the first job for a variety of reasons, but would have preferred to stay on if things could have changed for the better.

I then moved to job number 2, an internship role predominantly focusing on dairy cattle but with some small animal work too. As an intern I was told that I would get full support, that I would not be expected to do any sole-charge work that was out of my comfort zone and that an experienced vet would always be available to help as required. Unfortunately I was again disheartened to hear a disgruntled senior colleague on the phone, particularly during the calving season, saying that I just had to deal with the situation as it presented itself, or on a couple of occasions they just went out and did the job themselves without taking me as it was 'easier that way'. This was during a period for me where I spent 3 months on call every weekend, with no time off during the week. So I was sufficiently exhausted and although learning plenty, I just wanted for an experienced set of hands or eyes at some of these call outs.

Suffice to say I looked for a new job at the end of my 12 month contract despite being offered ongoing work with that clinic. Now in job number 3 back in mixed general practice I am back doing a similar job to what I was doing as a new grad, although now with at least another vet to discuss cases with if required, either in person or over the phone. However, there have been numerous times in this role that I have considered other options—either locum work or work outside the vet profession—simply due to the fact that the opportunities for improvement in skill base are not readily available in this job. Essentially I feel now as though I'm in a role where I can only improve slightly from where I am, and most times when I requested certain CPD courses/workshops the bosses will say 'no, you won't need to know things that in-depth, I'll just find some old course notes from when I did a course and then you'll be fine'.

In response to the comment that vet students going through courses these days are from privileged backgrounds—that's certainly not what I would consider was the case with my graduating class. I spent 9 years at uni, first gaining a BSc, then going on to do vet. I have a hefty loan and with living costs and vet salaries as they are I won't be paying that off in a hurry. So unfortunately from my experiences I can understand why there are large numbers of inexperienced vets moving towards locum work—at least they might feel then that they are making some progress in life!

While I understand the great cost that taking on new grads is to a veterinary business (particularly a private practice), the first few years are so critically important for developing sound basic skills—not just clinical and surgical skills but hearing from senior vets about communication, dealing with really difficult cases, mental health awareness etc. Our profession is now under more pressure from the public to do more and offer more as clients become better educated about whatever ailment their animal may have, so providing appropriate support to junior vets, at least in my opinion, is critical to retaining them in the profession—even if, 'dare I say it', a female vet decides to have children 5-10 years into her career!

I don't have any answers to this dilemma, but agree that there ought to be some more brainstorming and/or research done regarding how many vets are retained in clinical roles and, if not, what their reasons are for diversifying or leaving the profession altogether. Maybe there are just so many non-clinical roles now that the number of graduates still isn't enough, once international students have been accounted for, heading into professional roles. Or maybe there just aren't enough practices available to support graduate vets and give them a good career grounding, so they take any new opportunities when they are offered. Volunteer work for 8 months, overseas travel, and a yoga course sure does sound pretty good, but definitely not an option for most new grads I would have thought!

It's definitely a challenge from both ends, new grad and boss, to get things right. I think sometimes the biggest issue is that we have bosses who have so many things going on that they can't just be there as a vet and/or mentor to support their colleagues. And in many of the situations I've been in it's the nurses that lose out too, unless the head nurse is well trained and is taking care of that side of the business. I have friends who have gone through internships with large franchise companies and they loved their first few years out—they get heaps of support and CPD provided by senior vets across all the branches, which also means they have access to help from people with a large number of special interests too. A support program like that, or something akin to how graduate doctors start out in industry may be beneficial. But there are still plenty of vet practice owners who are of the opinion that being dropped in the deep end is the best way to start out in practice. It's that attitude that would be nice to see gone from the industry—I don't think it does any good for our profession at all.

MAJOR WINNER!

PERSPECTIVE NO. 141

Control of feline dermatophytosis in a shelter with use of mycoparasite *Pythium oligandrum* & vaccine

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Abstract: *Pythium oligandrum* (PO) is a mycoparasite widely used in the treatment of fungal infections in plants important for agriculture. Several studies have been published in the past 15 years about the use of PO in the treatment of dermatophytoses in humans and a few about the treatment of dermatophytoses in animals (cats and guinea pigs). A study was performed in a cat shelter where an outbreak of an infection of *M. canis* was present. Severely ill and really shy cats were treated topically with PO; healthy cats were vaccinated with inactivated vaccine (autovaccine made directly from the strain cultivated at the laboratory from this shelter). Treatment with PO was successful in 87% of the cats treated by PO and 100% successful in the vaccinated cats. No life-threatening side effects were observed. Two years after the time of treatment there were not any cases of dermatophytosis observed in the cats in the shelter or in adopted cats. The results of this study suggest that the treatment of cats and the environment with PO can be useful in the control of dermatophytosis in shelters or multi-cat households with sub-optimal conditions.

Introduction

Dermatophytosis is a fungal infection, widespread throughout the world, which affects the superficial layers of the skin, hair and claws, as the dermatophytes produce enzymes which enable them to utilize keratin from the keratinised portion of the epidermal tissue. It is one of the most commonly diagnosed zoonoses in cats. It is quite a significant disease in animal shelters, because of environmental contamination and the easy transmission of spores.[1-3] Many species of the filamentous fungi, belonging to the genera: *Microsporum*, *Trichophyton* and *Epidermophyton*[1-4], have been recognized. Molecular studies have shown that all dermatophytes are closely related genetically to each other.[4,5]

The most often diagnosed cause of dermatophytosis is *M. canis* which is a typical zoophilic dermatophyte. Some of the infected cats may be asymptomatic, e.g. cats living in a contaminated environment. Animals living in a contaminated, humid environment; young, pregnant, immunocompromised, or old animals[1,4,6,7] and Persian heritage cats are predisposed to the infection.[8]

Dermatophytosis can be diagnosed by Wood´s Lamp examination but the effectiveness of the examination is only about 50%—*M. canis* strains fluoresce, but scales, debris, bacteria or topical medicaments can produce false positive results.[1,2,6,9] An easy and rapid procedure is a direct microscopic examination of the hair and scale, but this should be carried out by an experienced diagnostician.[1,2,6,10] The method of choice for diagnosing is fungal culture, because it enables identification of both genus and species.[1,2,6,11]

It has been documented that the disease is self-limiting and healthy cats could heal spontaneously. Treatment is still recommended in order to limit the spread of the infection to other animals and humans[1,4,6] and to reduce dissemination of highly resistant spores to the environment. There are many reports on successful treatment protocols for catteries, shelters and for cats with experimental infections based on a daily or pulse administration of a systemic antifungal therapy, application of topical antifungal solutions and home hygiene recommendations. None of them is without side effects[12,34].

Only a few studies report on vaccination with killed vaccine,[35,43] but vaccination could be also a therapeutic option for treatment of clinical signs of dermatophytosis in otherwise healthy animals.[35] Some studies report good efficacy in treatment,[38, 39] some studies even 100% efficacy.[40,43] There are two commercial vaccines available for treatment in Czech Republic, one for cats[39] (Biofel M, ÚSKVBL registration No. 97/044/01-C) and one for dogs[38] (Biocan M, ÚSKVBL registration No. 97/065/01-C).

Biological control of soil-borne pathogens by the mycoparasite *Pythium Oligandrum* (PO) is well known[44,46] and has been demonstrated in a number of studies on plants important for agriculure[47,66]. Recently, studies utilizing the mycoparasitism of PO in dermatophytosis therapy in humans[67,71] and some case reports of the same in animals[72, 73] described a good therapeutic effect, while no side effects were recorded at all. Recently *in vitro* studies documented mycoparasitism of PO.[74, 75] This fact is very important for groups of animals containing individuals in a variously compromised health status such as those in animal shelters.

Open field questionnaire among Czech veterinarians showed that the treatment with PO was comparable with the conventional local therapy.[76] In an open field study performed on 25 guinea pigs with dermatophytosis, 100% of the fungal cultures were negative after the PO treatment.[77]

Materials and Methods

The study was carried out in an animal shelter in the Czech Republic, which did not have any established treatment approach to the management of dermatophytoses. Before our study, only cats with clinical signs of the ringworm disease were treated—there were no systematic measures to deal with dermatophytosis in the shelter during the six years of its existence. The shelter and the shelter staff participated in the study with informed consent.

Cats treated in the study

There were 94 cats and 3 dogs in the shelter at the time of the first collection of the samples for fungal culture. These cats were divided into groups in the shelter according to the places in the house. Group 1 (middle-aged, relatively healthy cats No. 1-34), Group 2 (quarantined cats No. 35-46 and 93-94), Group 3 (healthy queens and new-born kittens, middle-aged tomcats, No. 47-64), Group 4 (young cats, shortly after treatment of *Giardia* and *Cryptosporidia* infection, No. 65-72), Group 5 (very shy, healthy middle-aged cats, No. 73-84), Group 6 (old and chronically ill cats on renal diet, No. 85-92). (Fig. 1) Because of the failure of full funding of this project during the study, only a reduced number of fungal cultures could be performed at the end (Day 92). No cats were euthanased in order to conduct the study (e.g. due to difficulty handling).

From all cats in the shelter, 35 were included in the study. Inclusion criteria were: presence in the shelter for the whole time of the study, ability to collect fungal culture samples at the end of the study. In one case where a cat could not continue the vaccine therapy, it was switched to the PO treatment but not included into the study group (cat No.17). It needed treatment for inflammation in the right carpal joint, just one week after the first dose of vaccine—this cat was treated with antibiotics and NSAIDs and the condition resolved in three weeks.

From the 6 cats with lesions, fur samples for trichoscopy were also collected at the same time (Day 1 and Day 92). One of the cats included in the study was long haired and the rest were domestic shorthair cats. Cats were divided into two treatment groups according to their health status. Group POT (*Pythium oligandrum* treatment) consisted of 18 cats—very old or severely ill or living in group where

MARTINA NAČERADSKÁ

MVDr. Martina Načeradská, PhD., MANZCVS. (feline medicine)

Martina finished her PhD on Research on *Pythium oligandrum* and Dermatophytes in 2017 (with huge support from Richard Malik and David Lloyd) and in 2018 passed the ANZCVS exam in Feline Medicine and became a MANZCVS in feline medicine.

Her favourite topic in veterinary medicine is feline medicine, particularly internal medicine and dermatology, behaviour and dentistry and she has 3 cats, two of which are rescues.

Martina loves to be first! ☺

Her first clinic was the first 'Cat Friendly' accredited clinic in the Czech Republic and her current clinic was the first to earn the ISFM Cat Friendly Certificate in 2019. She and colleague Katka Horáčková set up the Czech Feline Veterinary Association two years ago and her dream is to assist in setting up excellent veterinary medicine education in Prague.

She is involved in owner education about animal welfare and health – speaking internationally, and successfully campaigned with others to ban fur farming in the Czech Republic. Her current project is working towards banning puppy farming. Watch the preview of a documentary on this topic: www.facebook.com/PedigreeDream/

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Martina's full bio is available in the eBook

FIP (feline infectious peritonitis) occurred. Group VT (Vaccination treatment) consisted of 17 young or middle aged clinically healthy cats.

Housing

Cats were housed in a family house (Fig.1), divided into 6 groups inhabiting 6 separate rooms or runs. Cats in Group 3 were kept in two smaller groups in an external run separated completely from the house. Group 2 was housed next to the garage away from the main building. Except for Groups 2 and 3, the shelter area was open to visits from members of the public, guided by the shelter staff, but everyone had to wash their hands with a disinfectant (Sterilium) after touching the cats. The house was equipped as a normal family home (carpets, wooden furniture, wooden wall facing) and inhabited by the owners of the shelter at the same time—the people were sharing the living space with the cats, including the sleeping room and kitchen. The house was kept very clean, carpets were vacuum cleaned twice daily, all smooth surfaces were regularly washed by disinfectant, and walls were painted every 3 months. However, there were problems with humidity in the walls of the house, the house was heated to 24°C during the cold period of the year and fungicides (Sodium hypochlorite 5%) were used regularly to prevent mould on the walls.

Cats were allowed to run freely in the given rooms using cat scratchers and toys as in a normal home, except for cats No. 27 and 35, who were treated for acute illness (No. 27 with chronic renal disease (CRD) treatment; No. 35 in re-convalescence after severe injury of spinal cord, suffering from UTI; and they were placed individually in large wooden cages.

The owners were advised to change the conditions in the shelter immediately, but they were not able to do it due to a lack of financial resources (an NGO fully dependent on donations). Besides the cats, 3 large guard dogs lived outside the house in the garden.

Clinical examination and fungal culture

On Day 1, a clinical examination of all cats and dogs was performed. Samples for fungal cultures were taken by a cotton swab set Fungi Quick, by rubbing the whole body of the animal. In 6 cats with visible lesions on the skin, samples of fur for trichoscopy were collected from the affected areas. The follow up sampling for fungal culture and the clinical examination of all animals was performed on Day 92 in the same way as on Day 1.

Treatment protocol

As a means of reducing *M. canis* in the environment, the walls and all the furniture were also treated by spraying the suspension of PO (in a special formula for use on walls, Bio Repel—Silica, *Panicum miliaceum*, PO, registered by Czech Republic Ministry of Health), used following the producer's instructions periodically. This process was proposed as a preventive procedure to be periodically carried out also in the future.

Pythium oligandrum treatment group (POT group)

The effervescent tablets with PO (Ecosin 3g tbl. Institute for State Control of Veterinary Biologicals and Medicines (ÚSKVBL), registration No. 057-09/C in the Czech Republic) were dissolved in two litres of lukewarm water (about 34 °C) and left to activate for 30 minutes. The fresh suspension was immediately applied onto the cats by a sprayer; this was easily applicable to the very shy cats, too. The fur of every cat was completely covered with the suspension after the application. Then, the cats were left to dry naturally. Although the producer of PO preparation recommended only 3 weeks of the therapy, we decided to continue for 6 weeks in total, according to our previous experience.[72,73]

The spray was applied once in 24 hours on the scheduled days: the 2-day blocks of application were followed by a 3-day pause between Days 52 and 73. Then, the suspension was applied in single pulses twice a week until Day 86.

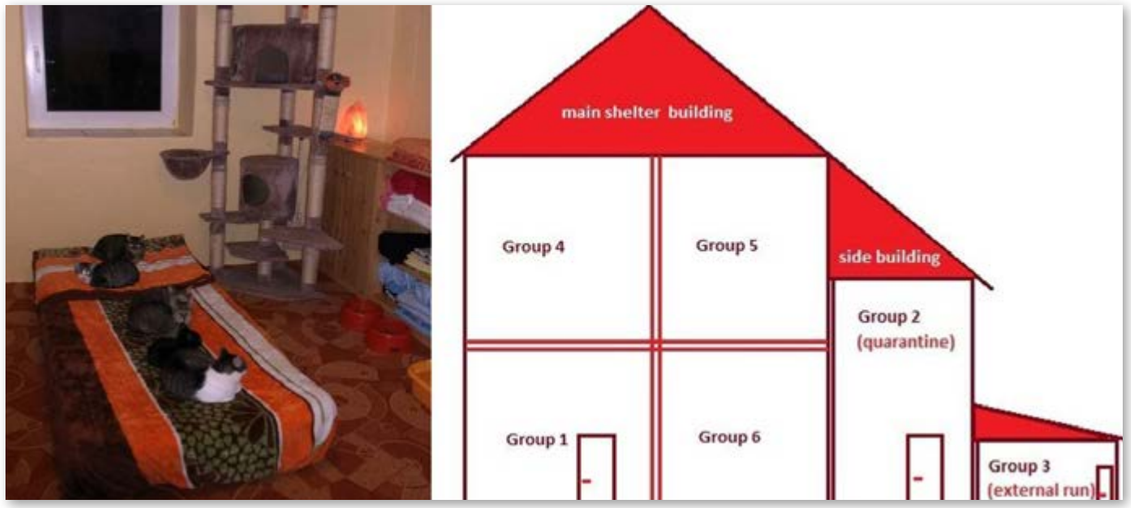

Figure 1: Example of the interior of the shelter (Group 5) and the scheme of the cat housing.

Inactivated vaccine treatment group

The inactivated autovaccine (SEVAX 0412, ÚSKVBL No. 56/2012, 1mL pro toto) was first administered SC to two young, healthy, easily manipulated cats on Day 44 (cats No. 9 and 18). These cats were carefully observed for possible adverse effects and their temperature was measured daily for a week. As there were no changes in their state of health, the vaccine was given to the other cats as scheduled.

Figure 2: Cat No. 35 which tested positive for dermatophytosis (a) before treatment and (b) after treatment.

The autovaccine was applied to the 15 other cats of VT group on Days 51, 65 and 79. The cats No. 9 and 18 also received the following vaccines one week earlier. All the cats were examined carefully before the application and observed in detail on the following 2 days.

Monitoring in the shelter

Cats were observed at least twice daily by the owners of the shelter. All the cats were observed and clinically examined by a vet every 14 days during the treatment. All changes in the clinical state of the cats were noted.

Fungal culture

M. canis was cultivated on a solid culture medium (Sabouraud dextrose agar OXOID CZ s.r.o. Thermo Fisher Scientific, Czech Republic). After optical growth control, a microscopic check of the strain was performed. Then, 5-10 mL of saline was applied to a Petri dish and the culture was re-suspended and transferred into the bottle with a culture medium for dermatophytes. The cultivation in liquid media was carried out at 25°C and took 8 days; the bottles were shaken for 1 hour every day. During this time, the growth and purity of the dermatophyte were checked on Day 4. At the end of the cultivation, the growth was checked microscopically and the samples were applied to solid media to check on the growth.

Data analysis

Data were analysed with use of STATISTICA 8.0 programme.

Results

The study was carried out between June 2012 and September 2012; the 2-years clinical follow up continued until September 2014. At the beginning, 35 cats were included in the study. After the therapy, fungal culture was performed on Day 92 on these 35 cats.

In September 2014, the total number of cats present in the shelter was 76, from which 18 cats, which were included in the study, were still present in the shelter. This illustrates the fluctuation in the numbers of animals in the shelter. No cats exhibited any clinical signs of ringworm disease.

Initial fungal culture

Before the proposal of the treatment, the status of dermatophytosis in the shelter was examined in detail. Every cat and dog present and even the shelter owner were tested for two species of the *Microsporum* genus (*M. canis, M. gypseum*) and for *Trichophyton spp.*

Among the cats from the study, there were 22 cats positive for *M. canis*, from which were on Day 1 originally 15 cats from POT group and 7 cats from VT group. Two of them from POT group remained positive (Table 1 and 2).

Table 1: Summary of the data from cats POT group

Cat No.	Age/yr	Sex	Breed	Note	MC Day 1	CE Day 1	Trich	Health status	side effects	MC Day 92	CE Day 92	1.9.2014
4	2	M	DSH		+	-	ND	CURT	-	-	-	R
7	2	F	DSH		-	-	ND	CGIT	-	-	-	D
8	3	F	DSH		-	-	ND	CRF	-	-	-	D
11	7	M	LH		+	Ears	+	OK	-	-	-	R
27	15	M	DSH		-	Dors.body, neck	-	CRT	-	-	-	R
29	12	F	DSH	shy	+	Tail	+	AURT	-	-	-	S
30	18	M	DSH		+	Head and neck	+	DM	+	-	-	D
33	3	M	DSH	shy	+	-	ND	AURT	-	+	-	S
35	2	M	DSH		+	dors.nasi, limb	+	Spinal cord injury, UTI	-	-	-	S
36	3	F	DSH		+	-	ND	CURT	-	-	-	R, D
38	3	M	DSH	shy++	+	-	ND	CURT	-	-	-	D
39	15	M	DSH		+	-	ND	OK	-	-	-	R
40	3	F	DSH	shy	+	-	ND	Ca. in mouth	-	-	-	D
41	14	F	DSH	shy	+	-	ND	CRF	-	-	-	D
42	2	M	DSH		+	-	ND	OK	-	-	-	S
43	1	M	DSH		+	-	ND	OK	-	-	-	R
72	1	F	DSH		+	-	ND	OK	-	-	-	S
88	15	F	DSH	shy	+	-	ND	CRF	-	+	-	D

Abbreviations: MC—fungal culture; + positive; - negative; CE—Clinical Examination, DSH—domestic short hair; LH—long hair; R—rehomed; S—in the shelter; D—died; OK—healthy; AURT/CURT—acute/chronical upper respiratory tract problems; CGIT—chronical non-infectious intestinal inflammation; ARF/CRF—acute/chronical renal failure; UTI—urinary tract inflammation; Ca—carcinoma, ND—not done

Table 2: Summary of the data from cats VT group

1	3	F	shy	-	-	ND	-	-	-	S
3	2	F	shy	+	-	ND	-	-	-	S
5	2	F		+		ND				R
6	1	F	shy	+	-	ND	+	-	-	S
9	2	M		+	-	ND	-	-	-	S
12	4	F		+	-	ND	+	-	-	R
13	2	F	shy	-	-	ND	-	-	-	S
14	8	M	shy			ND	+			S
15	10	F	shy	-	-	ND	-	-	-	S
16	2	M	shy	-	-	ND	-	-	-	S
18	1	F		+	-	ND	-	-	-	S
19	4	F	shy	+	-	ND	+	-	-	S
25	3	F				ND				R
28	4	M	shy+	-	-	ND	-	-	-	S
31	5	M	shy+	-	-	ND	-	-	-	S
32	3	M	shy+	-	-	ND	-	-	-	S

Abbreviations: MC—fungal culture; + positive; - negative; CE—Clinical Examination; DSH—domestic short hair; ND—not done

Trichoscopy

In addition to the fungal culture, a trichoscopy was performed on cats with clinical signs. The aim of this was to compare the easy, traditional diagnostic method with the laboratory results in order to distinguish whether this, as a method of first choice for many vets, is reliable enough for diagnosing dermatophytosis. Five of the cats exhibited suspect lesions on the skin: Cat No. 11—ears; No. 29—tail; No. 30—head and neck; No. 35 dorsum nasal region and paw were found positive in the trichoscopy and also on culture; another, Cat (No. 27—dorsal body and neck) was examined, but no dermatophytes were seen and the fungal culture was negative, too. Still, all skin lesions healed during the treatment. For illustration, see Fig. 2.

Clinical findings and treatment responses

The complete scheduled treatment was possible in 35 cats. After the treatment, all of the cats were clinically examined by the veterinarian. In view of the fact that the literature often describes the risk of vaccinated cats becoming carriers of the infection without clinical signs, [1, 36] samples for fungal culture at Day 92 were taken from all of the vaccinated cats.

Four cats from VT group (No. 6, 12, 14 and 19, Table 2) showed mild side effects, such as upper respiratory tract signs 2 days after vaccination. (Table 2). But these symptoms disappeared spontaneously within 2 days. The result of fungal culture and clinical examination was negative (i.e. clear of dermatophytes) for all the vaccinated cats (Table 2).

In POT group 18 cats treated with Ecosin. Detailed information about those cats and their health status is displayed in Table 1. The only cats remaining positive after the treatment were one old neutered female (No. 88), which suffered from CRF and died 3 months after the treatment—this cat was regularly treated weekly with the PO suspension; and one young neutered male (No. 33) which suffered from an acute upper respiratory tract infection at the time of the Day 92 testing. This young cat was first further treated with Ecosin and after recovering from the respiratory problems, it was additionally vaccinated on the same course as the other cats, *i.e.* 3 consecutive applications following each other in 2 weeks. There were no severe adverse effects observed during the PO therapy. Still, the cat No. 30 (old diabetic cat) suffered from fur loss after the first 2 courses of the PO treatment, but this problem was only temporary and resolved itself spontaneously within 3 weeks.

Long-term clinical follow up

A long-term follow up was carried out for 2 years after completing the study and was based on the reports of clinical signs by the shelter owners and the vets. All of the cats and dogs present in the shelter were regularly checked by a veterinarian every 6 weeks until September 2014. There were no reports of any clinical manifestations similar to ringworm disease in the shelter in cats, dogs or the owners until 2018. Moreover, there were no reports about a recrudescence of the disease after rehoming of any of the shelter cats—the shelter owners collect information about the state of health of every rehomed cat from the new cat owners, including photographs of the animal in the new environment, during the first weeks after the rehoming and they sometimes even visit the new homes of the cats.

Discussion

Originally, the dermatophytosis treatment study in the shelter was designed as an eradication study, including substantial changes of the cat housing and husbandry. We intended to carry out a statistical study of PO and vaccination treatments with a control group of cats conventionally treated by itraconazole.[1,2,6,7,12,15,18,23,26,28,34]

With respect to the complicated situation of the NGO running the shelter and also to the low rate of completely healthy cats eligible for the conventional treatment, the intended study had to be changed. Finally, the design of the study was far from ideal, but still we were able to apply the treatment by vaccination to the healthier cats as described above and to apply the PO therapy to the cats with high morbidity. For these old and/or chronically or acutely ill cats and also for the extremely shy ones, this was the only acceptable treatment modality.[72,73] In the literature, there are recommendations to euthanize such cats at the beginning of a dermatophytosis eradication process, [14, 27,31] but this was morally unacceptable for the shelter owners.

While some of the studies stated that the cats which were vaccinated against *M. canis* become life-long carriers of the disease or develop immunocompromise,[1, 36, 37] we did not observe anything like this in our study; our results are consistent with other recent studies.[38,43] As all of the 17 vaccinated cats were clinically and mycologically negative after the treatment, there was no need for a statistical analysis. Still we have to admit, that we cannot quantify the contribution of PO to this state, while PO was regularly spread all over the environment and it actively chases the pathological fungi.[44,46]

In the POT group of 18 cats, treated by PO, it is clear immediately, that no statistically correct conclusions can follow from there, because the number of treated individuals is too low for any rigorous statistical testing. Despite the fact that we have no exact evidence, we can intuitively evaluate these results with great reliability. We judge from our data that treatment by PO is useful especially for cats which were diagnosed with comorbidities. The treatment of positive cats was successful in 87% of the group (16/18 cats) and all 100% of the negative cats remain negative after the procedure.

Although this pilot study could be realised only to a limited extent, the valuable experience that was collected still suggests that such treatment, although carried out imperfectly and in a very difficult environment, can have the desired effect. After the end of the therapy, we proposed to the shelter owners a set of measures to be taken to prevent a new outbreak of the disease, which is being gradually implemented in the shelter's daily practice. Surprisingly, the suppression of the occurrence of clinical signs of ringworm disease persists even two years after the Day 92. This result was reached due to the combination of several new routine methods applicable even to severely ill cats—every new incoming cat is sprayed with the PO suspension as soon as possible and also the environment is regularly treated with the suspension of PO in a special formula for use on as a part of periodical maintenance.

Furthermore, the housing of the cats is being significantly improved by removing the carpets, the redundant wooden wall facing and some of the furniture. The carpets are being replaced by easily washable surfaces (ceramics) and the wooden quarantine cages have already been replaced by completely washable metal ones. The previously imperfect quarantine room has been divided into two rooms, one for newcomers and ill cats with contagious diseases and one for convalescents after surgery. The quarantine rooms are completely washable and it is easy to disinfect them. Furthermore, there are no more kittens accepted to the shelter building at all, they are all kept in a separate home-like shelter building (deposit). The improvement in the housing conditions contributes to the prevention of a repeated outbreak of dermatophyte disease in the shelter.

Although we cannot describe our results as a study of the eradication of the disease, because we were not able to perform as many follow up fungal cultures as necessary for this conclusion,[1,2,7,14,19,23,28,33] the long term clinical observations did not show any further outbreak of the condition. This is a great success in the given conditions, even comparable to the results of systemic itraconazole therapy or a local combined therapy published elsewhere.[1, 2, 6, 7, 12,15, 18,23, 26,34]

We would like to emphasize that the original housing conditions in the shelter at the time of onset of the study were comparable to those at the homes of typical cat owners, but loaded by the high number of cats and their poor state of health typical for the rescued cats deposited in shelters.[7, 14, 28, 29] Concerning the compliance of the shelter owners, it did not much differ from that expected in individual loving pet owners. The vaccine can be considered an option for healthy animals and we found out that it worked well without any life-threatening side effects. But taking into account the shelter reality, *i.e.*, the lack of the funds for laboratory examinations, presence of many severely ill or very shy animals and high rotation of the cats, the easily administrable treatment with PO can be considered as a satisfactory, efficient and easy way to treat and prevent dermatophytoses.

Conclusions

The results of this study suggest that vaccination against *M. canis* with an autovaccine tailored to the given cat can be a safe option for the treatment of healthy cats. But still, treatment with *Pythium* mycoparasite has a great advantage in its applicability to shy or health compromised cats and thus this can be a reasonable first choice treatment for the shelters. The greatest advantage over the traditional therapeutic approach[7, 14, 28, 29] is the fact, that the ill and shy animals do not need to be euthanased in order to eradicate signs of the ringworm disease in the shelter conditions, and therefore the moral issues of the rescuers are not in conflict with the treatment.

Acknowledgements

The University of Veterinary and Pharmaceutical Sciences Brno is acknowledged for financial support of the fungal cultures on Day 92.

Conflict of interest

The authors do not have any potential conflict of interest to declare.

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A selection of infectious diseases of companion animals housed in shelters (with a focus on cats)

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Shelter medicine provides challenges that are unique due to dealing with large numbers of stressed animals housed in a confined space, usually at a high population density. It's even more complicated since many of the animals are young and at increased risk of developing infectious diseases because their passive and acquired immunity are in a state-of-flux.

A key challenge for shelter vets is minimising the risk associated with the development of infectious diseases. Failing that, shelter vets need to know how to best manage infections when they arise, in a timely and even preemptive fashion. Our focus in this Perspective is contagious diseases generally encountered uncommonly in privately-owned animals but observed quite frequently in sheltered companion animals in Australia.

We do this with the aim of informing clinicians who predominantly focus on private practice but perform some shelter work (e.g. assisting local rescue groups and pounds). We also include a short section on vaccination, since the higher stress levels and increased pathogen exposure encountered by shelter animals compared to privately-owned animals demands for some modifications to general vaccination philosophy and guidelines.

1. Feline herpesvirus-type 1 (FHV-1)

FHV-1 is the most common respiratory pathogen isolated from cats and kittens, both with and without overt feline upper respiratory tract disease (FURTD). One Australian study of privately-owned cats found a FHV-1 prevalence of 21% (98/462) in cats with URTD using a PCR assay.[1] While there are no published reports to date of FHV-1 prevalence in Australian shelters, an overseas study investigating several Californian animal shelters found that FHV-1 prevalence was as high as 41% in one shelter sampled.[2]

The most common manifestation of FHV-1 infection in cats is sneezing with serous nasal and/or ocular discharge (Figures 1 & 2). Less commonly, but worth remembering, is that FHV-1 infected cats can present with eosinophilic facial dermatitis. An article by Malik *et al*. (2009) includes representative photographs of cats with FHV-1 associated dermatitis for readers interested in learning more about this condition (Figure 3).[3]

Undoubtedly stress and overcrowding play a huge role in sheltered cats developing FHV-1 infection, and recovering from FHV-1 infection, whether it be a new infection or recrudescence of a latent infection (where the virus hides away in the trigeminal ganglion). Consequently, **treatment of the overall situation is just as important as treatment of the infection itself**. Ideally, infected cats showing clinical signs should be removed from the shelter and fostered into low-stress households. If this option is not available, infected animals should be isolated in a quiet, low-stress section of the facility, and some people like to spray bedding with feline facial pheromone (Feliway™) to help de-stress the patients.

Figure 1: Cat with severe FHV-1 infection displaying prominent and severe ocular involvement.

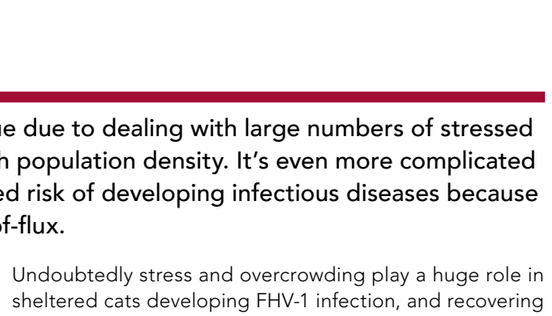

Figure 2: A range of presentations for cats with FURTD.

'Portal-mania' is a phenomenon sweeping the shelter medicine world; this is the installation of portals to convert one cage into two cages, connected by a 'port' or tunnel (Figure 4). This allows cats to have separate toileting and eating/drinking areas, which has been shown to reduce stress levels and therefore the incidence of FURTD (Figure 5). The Koret Shelter Medicine Program at UC Davis has some great online resources for clinicians interested in reading more about the prevention and holistic treatment of FURTD. Appropriate disinfection protocols and personal protective clothing for staff are of course a mandatory component of case management. There is also a recent trend for minimalistic cleaning of cages (e.g. only cleaning the litter tray and replacing food bowls without cleaning the whole cage) to reduce stress and to leave important marking scents secreted by cats, with a total cage clean out and disinfection occurring at the end of a given patient's stay (terminal clean).[4]

Figure 3: Herpetic dermatitis in the cat. Lesions tend to be located close to the eyes or nares. For some bizarre reason, the pathology is dominated by eosinophils.

Bacterial co-infection (secondary invasion) with FHV-1 is common and may include *Mycoplasma* spp., *Bordetella bronchiseptica, Chlamydia felis* and a variety of obligate anaerobes. Multiplex PCR testing is commercially available in Australia and will detect nucleic acid from viral and bacterial pathogens commonly implicated in cases of FURTD.[5] Both amoxicillin-clavulanic acid and doxycycline have been used to treat FURTD with similar efficacy[6]; our personal preference, however, is to treat with doxycycline monohydrate at 10mg/kg SID (with food or just before a meal) to cover possible secondary *Mycoplasma* infections and to avoid the stress of twice daily pilling. Treat for 21 days if you suspect *Chlamydia* infection (cats with chlamydiosis usually present with severe chemosis and purulent ocular discharge, often affecting one eye first, and then spreading to the other eye).

Recently, famciclovir has been used successfully in many cases of FHV-1 infection; this is an anti-viral drug that was developed for the treatment of α-herpesvirus infections in people (e.g. 'cold sores') and has demonstrated clinical efficacy in cats infected with FHV-1. Our current recommended dosage for famciclovir is 30-40 mg/kg every 8-12 hours based on serum pharmacokinetics (and saturation of the hepatic conversion of famciclovir to penciclovir), even though David Magg's group still recommends 90mg/kg BID-TID,[7] which most people find prohibitively expensive. Based on some unpublished research in Australia we have been involved with, we do NOT recommend 'blanket' prophylactic treatment of all cats presenting with FURTD with famciclovir; instead we advise it to be used early and regularly, but only in cats presenting with clinical signs likely associated with FHV-1 infection (i.e. where nasal and/or ocular signs are prominent, rather than oral cavity disease with ulcers more suggestive of infection with calicivirus). Cats with prominent FHV-1 associated ocular

Figure 4a: Photograph courtesy of Anastasia Klose, *AWL NSW.*

Figure 4b: 'Portal-mania', the joining of two cages by a connection to create a toileting area and an eating/drinking area for hospitalised and sheltered cats. This reduces the incidence of stress-related infections like FHV-1.

Figure 5: 'Cat Condo' en suite with separate eating/drinking, toileting and sleeping areas. Photograph courtesy of Anastasia Klose, *AWL NSW.*

disease including corneal ulceration may also benefit from cidofovir drops given twice daily (sourceable from good compounding pharmacies, e.g. BOVA).

FHV-1 SUMMARY:

1. FHV-1 infection is more common in stressed shelter cats than in pet cats
2. Treat the stress as well as the infection
3. Use a combination of doxycycline and famciclovir treatment in FHV-1 infected cats
4. Add cidofovir eye drops if ocular disease is especially severe.

2. Virulent systemic feline calicivirus (VS-FCV)

Virulent systemic feline calicivirus (VS-FCV) infection was first described in the literature as a focal outbreak affecting cats in northern California in 1998.[8] Although not widely cited, the syndrome might actually have been first recorded in Australia in 1972 by Daria Love and Keith Baker in a case cluster of kitten mortalities, with isolation of calicivirus from lung and brain tissue as well as mouth ulcers. Since these seminal reports, it has been reported in many countries including Australia, with a recent relatively mild outbreak occurring in Canberra in early 2018.[9]

One of the pioneers of the shelter medicine movement, Dr. Kate Hurley, reported an outbreak affecting 54 cats in the west Los Angeles area that occurred in 2002 with a mortality rate of 40%.[10] This outbreak was particularly concerning as it demonstrated the interplay between shelters and private veterinary clinics. It was a reminder that private clinicians not working in shelters also need to be on the outlook for cases of VS-FCV, despite it being a disease that usually arises in group-housing situations (e.g. shelters and research colonies). In Hurley's report, three different veterinary clinics were presented with cases of VS-FCV following a suspected outbreak at a local rescue facility that housed around 40 kittens and cats at the time of the outbreak. Some of the treated cats did not have direct contact with sick animals but were likely infected due to fomite transmission via owners who had other cats being treated in hospital or were technicians at one of the three clinics. This was an unfortunate demonstration of the environmental survivability of FCV (up to 1 month under ideal conditions) and the need for vigilant barrier nursing and disinfection protocols in veterinary hospital and shelters. **We favour accelerated hydrogen peroxide for disinfection after preliminary cleaning, since it is inoffensive to staff and straightforward to use.**

In addition to common signs of upper respiratory FCV infection (e.g. anorexia, tongue ulceration, nasal discharge and dyspnoea), veterinarians should also be aware of these additional signs which may be evidence of the more pathogenic VS-FCV:

- High fever (as high as 41.40°C)
- Severe respiratory distress
- Facial and/or limb oedema *(Figure 6)*
- Ulcerative dermatitis, crusting and alopecia of the face, ears and feet ('paw and mouth disease'; Figure 7)
- Limping
- Ocular discharge and/or conjunctivitis
- Icterus probably due to viral pancreatitis
- Sudden death.

Definitive ante-mortem diagnosis of VS-FCV is not yet possible, although this is an area of active research. Swabs of lesions are PCR-positive for FCV. Treatment of cats with VS-FCV infection is mainly supportive (intravenous fluids therapy, analgesia and antibiotics to cover secondary bacterial infections). Treatment with feline interferon-omega (rFeIFN-ω) is helpful[11] (Virbagen™, manufactured by Virbac Animal Health). Drugs used to treat norovirus infections in people might eventually prove useful for the clinical management of cats with VS-FCV. Core vaccination with commercial vaccines is generally not protective against VS-FCV.

VS-FCV SUMMARY:

1. Both shelter and private veterinarians need to be on the lookout for outbreaks of VS-FCV in cats and kittens
2. Mortality rates during outbreaks are high (up to 67% reported)
3. F3/F4 vaccination is NOT protective
4. Treat early, treat aggressively (e.g. add rFeIFN-ω if possible), and ensure staff practice barrier nursing and follow strict hospital disinfection protocols.

Figure 6: Cat with severe subcutaneous facial oedema referable to VS-FCV. This cat was diagnosed and treated by Rachel Korman at Veterinary Specialist Services, Queensland with assiduous symptomatic and supportive therapy including nutritional support using an oesophageal feeding tube.
Photograph courtesy of Rachel Korman.

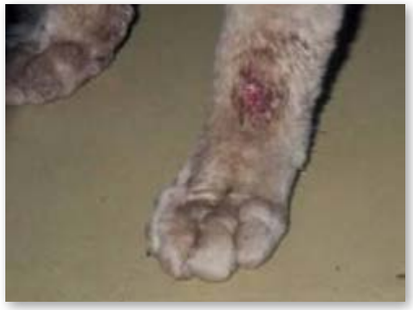

Figure 7: The ulcer on the leg of this cat is a result of a conventional FCV infection. The cat also had the well-recognised ulcers on the surface of its tongue. Licking the forearm transferred virus to the skin—causing ulceration there, what Margaret Sabine once called 'paw and mouth disease'.

3. Coccidiosis

Coccidiosis, caused by *Cystoisospora* spp. (called *Isospora* spp. in older literature), is relatively uncommon in privately-owned pets but is commonly detected in shelter animals (particularly puppies and kittens). Palmer *et al.* (2008) performed a national Australian study of gastrointestinal parasites in dogs and cats and found *Cystoisospora* spp.[12] in 10% of shelter kittens/cats sampled, but in only 1-2% of privately-owned cats and dogs tested. The figure of 10% in shelter animals is reminiscent of some unpublished research we performed with Professor Jan Slapeta in Sydney in 2012 on shelter animals. Coccidiosis is so common in some shelters in Australia that affected rescue organisations routinely treat all animals awaiting adoption, and some breeding kennels routinely treat all puppies in the third or fourth week of age[13], with a single dose of the anti-protozoal drug toltrazuril (see below).

Detection of *Cystoisospora* spp. is relatively straight forward with routine saturated salt faecal flotation and microscopy. Look for organisms about half the size of nematode eggs; some may appear in the distinctive sporozoite stage (two nuclei contained within the one sporocyst wall; red arrow in Figure 8).

Note: Currently available multiplex faecal PCR panels do NOT test for *Cystoisospora* spp. So, you will miss them unless you do a faecal float!

The off-label treatment of choice for coccidiosis in small animals is toltrazuril[14], which comes in a formulation for piglets or chickens called Baycox™ (Bayer Animal Health). In confirmed cases of *Cystoisospora* infection, administer a once-of dose of 20-30 mg/kg (dogs) or 20mg/kg (cats).[15] Some people give this dose every day for 3 days, to ensure adequate dosing (the formulations are not palatable; the pig formulation is better to give than the chicken formulation). Some authorities recommend re-treatment after 3 weeks to cover the possibility of re-infection via acquisition of oocysts in the environment. We recommend bathing kittens and puppies (e.g. using Malaseb™, Dermcare), although any pH neutral shampoo will do) after treatment to remove any contaminated faeces that might be stuck to the fur and then placing them in a disinfected cage to prevent re-infection.

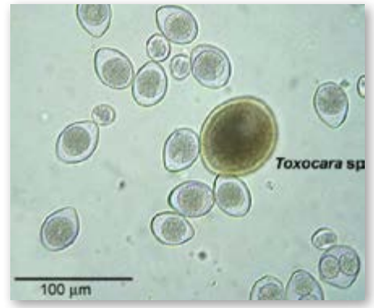

Figure 8: Faecal flotation showing a single Toxocara *sp. egg and numerous* Cystoisospora felis *eggs.*

COCCIDIOSIS SUMMARY:

1. Coccidiosis is common in shelters, particularly in young animals
2. Don't forget the basics - perform a saturated salt faecal flotation on any shelter animal with diarrhoea to look for nematode eggs and *Cystoisospora* organisms, before considering more expensive diagnostics like a faecal PCR panel!
3. Routine anthelmintics do NOT cover coccidia spp. - treatment is with toltrazuril (Baycox™).

4. Giardiasis

Like coccidiosis, giardiasis is relatively uncommon in privately-owned pets but is common in shelter animals (particularly dogs). Palmer et al. (2008) found 14% of Australian shelter dogs sampled were infected with *Giardia* spp.[12] (versus 6% of privately-owned dogs), and 3% of Australian shelter cats sampled were infected with *Giardia* spp. (versus 1% of privately-owned cats). Interestingly in this study, no dogs with giardiasis displayed clinical signs of gastrointestinal disease, highlighting that infection with *Giardia* spp. can often be asymptomatic. The unpublished research we performed in Sydney in 2012 on shelter animals with Jan Slapeta found a similar overall prevalence of giardiasis and of predominantly sub-clinical infections in dogs and cats. Environmental decontamination is a very important part of preventing this disease spreading in shelters, and good design of facilities which make them easy to disinfect and keep clean is important for both prevention, and to prevent cross contamination once disease occurs (Figure 9).

Giardia organisms are very difficult to detect with routine saturated salt faecal flotation and microscopy. Instead, **a faecal antigen point-of-care kit is the easiest way to diagnose giardiasis in-clinic. Multiplex faecal PCR panels DO include *Giardia* spp. and are another way to detect infection.**

Even though many cases of giardiasis are asymptomatic, we still advise treatment because of the possible public health risk to owners (some assemblages of *Giardia* are host-specific for dogs and cats, while others are potentially zoonotic). Under some circumstances *Giardia* can cause significant small bowel and/or large bowel diarrhoea (sometimes hemorrhagic) in cats

and dogs, both acute and chronic, and sometimes signs can be severe.

Treatment for giardiasis is usually with fenbendazole (Panacur™, Coopers Animal Health) at 50 mg/kg SID for 5 consecutive days (Plumb's Veterinary Drug Handbook). Fenbendazole at this dose will also remove any nematode infections (including roundworm, hookworm, whipworm and lungworm), and consequently fenbendazole can be used as a routine anthelmintic in dogs, cats and pocket pets (as it is in the UK, with registered companion animal products licensed for this purpose). However, fenbendazole is only effective against some tapeworm infections (effective against *Taenia* spp. but not *Dipylidium caninum*, the flea tapeworm), and therefore praziquantel should be added whenever a cestode infection is suspected or confirmed. Panacur™ is highly unpalatable as the large animal drench preparation, and if treating many animals with fenbendazole it can be well worth investing in fenbendazole tablets from a quality compounding pharmacist (e.g. BOVA). It's best not to use metronidazole as a treatment for giardiasis since the dosage required to clear *Giardia* has the potential to cause neurotoxicity. One of us uses concurrent metronidazole at a conventional dosage (10-15 mg/kg twice daily for 5 days) to treat the dysbiosis and inflammatory bowel disease which commonly occurs secondary to *Giardia* infection.

For cats not amenable to treatment with fenbendazole for 5 days, a single dose of secnidazole (which can be ordered from BOVA compounded to a suitable strength) at a dosage of 30 mg/kg and repeated 3 weeks later, has demonstrated excellent efficacy for clearing giardiasis in treated cats.[16] Jim Euclid drew our attention to this drug. We have used secnidazole successfully in several *Giardia*-infected cats.

Veterinarians should also remember that co-infection in cattery and shelter cats with *Tritrichomonas foetus* and Giardia is common. In one American study, over one-third of cats (14/36) infected with *T. foetus* were also infected with *Giardia*. Diagnosis of *T. foetus* infection is via a wet preparation of very fresh faeces (very quick and easy, but not that sensitive), by InPouch™ culture or by qPCR testing, and requires off-label treatment with a pigeon medication called ronidazole for eradication of the infection.[17] Ronidazole has been associated with serious neurological side-effects in some cats and therefore should be used with caution; current dosage recommendations are 20-30 mg/kg SID for 14 days in cats and 10mg/kg SID for 14 days in kittens. For cats co-infected with *Giardia* and *T. foetus*, we recommend first clearing the *Giardia* infection with a single dose of secnidazole, waiting a few days, then commencing ronidazole therapy. Prebiotics and probiotics have a place in management of these cases, and it's not uncommon to have a protozoan-induced inflammatory bowel disease which takes a while to resolve. So be patient after therapy and persist with highly digestible diets.[18]

eBook download:
C&T No. 5313 'Secnidazole as a one-dose for Giardia'

TOP TIP: As a side note, one of us (SM) uses 'magic mince' (beef mince + Protexin™ + psyllium) as a miracle cure for persistent kitten diarrhoea with unknown aetiology. It has revolutionised our treatment of these patients, so consider trying if you have any similar cases!

GIARDIASIS SUMMARY:

1. Giardiasis is common in shelters, including animals without signs of gastrointestinal disease
2. Detection of *Giardia* spp. requires antigen testing with a point-of-care kit or qPCR testing (i.e. routine faecal flotation will often miss this parasite)
3. Treat with fenbendazole or secnidazole
4. In cats, look for co-infection of *Giardia* spp. with *T. foetus*, and treat with ronidazole as appropriate, but sequentially and NOT simultaneously.

5. Dermatophytosis (Ringworm)

Dermatophytosis is a nightmare for cat shelters to manage. Infected cats (Figures 10 and 11) need to be isolated during treatment (usually for 6-8 weeks). Fungal spores can survive in the environment on broken hair shafts for months or even years, making outbreaks of ringworm in shelters a huge risk. There are few things more heartbreaking as a shelter veterinarian than the euthanasia of otherwise healthy cats with a treatable skin disease to reduce to risk of spread of this dermatophyte. *Microsporum canis*, a zoophilic dermatophyte, is the normal culprit, although occasionally the geophilic (soil dwelling) organisms *Microsporum gypseum* and *Trichophyton mentagraphytes* are responsible. The diagnosis and treatment of all three organisms is much the same.

Screening cats with a Wood's lamp is quick and easy (Figure 12), but unreliable (not all isolates glow apple green) and highly affected by the skill of the operator. In addition, some cats with dermatophytosis are asymptomatic and therefore very difficult to identify. In a study of 273 stray cats in Italy, which reported a dermatophytosis prevalence of 6% (many of which were asymptomatic), 20/20 cats declared infected based on Wood's lamp testing were found to be uninfected when hair was cultured (i.e. all 20 positive results were false-positives).[19] This result was a reminder that the only way to accurately diagnose dermatophytosis in cats is by fungal culture, although PCR may offer another option in the foreseeable future. Many shelters have started to routinely perform fungal culture on all incoming cats using sterile toothbrushes, and this can be done cheaply if fungal culture plates are bought in bulk and some basic fungal identification training is completed. The authors would like to acknowledge the efforts of Dr. Denise Wigney at the University of Sydney - over the past five years Denise incubated and interpreted the fungal results for thousands of rescue cats in the Sydney area, all in her own time and for no personal gain.

Treatment of dermatophytosis was recently covered by Richard Malik and Mark Krockenberger in an excellent perspective of *Control and Therapy*. In summary, veterinarians

Figure 9: Dog kennels of good design. Note selection of surfaces with a view to easy cleaning. This makes prevention of diseases spread by the faeco-oral route far more practical. Photograph courtesy of Anastasia Klose, AWL NSW

eBook download:
Perspective No. 134 'Antifungal therapy in companion animals—a practical approach'

treating ringworm need to isolate infected animals for the duration of treatment, and a treatment regime should consist of both a systemic medication (itraconazole or terbinafine) and twice weekly bathes (Malaseb™ in Australia; often overseas lime-sulphur formulations are used for bathing cats, but such preparations are not available in Australia). Itraconazole (Sporanox™) is our preferred treatment for feline dermatophytosis at a dosage of 5mg/kg SID in the food. We do not like using compounded formulations of itraconazole due to variable and usually low bioavailability. Terbinafine (Lamisil™ and generics) is an alternative treatment at 10-20mg/kg BID, although its effectiveness is possibly slightly less than that of itraconazole therapy. Lamisil™ ointment is a convenient adjunct to treat severe focal lesions. One of us (SM) uses systemic terbinafine in cats >2 kg for cost reasons and twice weekly drenches with enilconazole (Imaverol™) in a 'rose sprayer' instead of dipping, due to the high stress associated with dipping, as well as disease control issues associated with moving cats in and out of cages.

An animal with dermatophytosis should not be cleared from infection until it has returned two negative fungal cultures two weeks apart.

There is new and interesting work from Martina Načeradská in the Czech Republic about using a saprophytic mycoparasite[20] to control dermatophyte infections in catteries, and there is some interest in developing and utilising vaccines against *M. canis*.

Fortunately, canine dermatophytosis is a relatively rare occurrence in shelters (just as it is in privately-owned dogs).

DERMATOPHYTOSIS SUMMARY:

1. Be alert for ringworm in cats with and without clinical signs of skin disease in shelters
2. Ideally screen all incoming cats for ringworm by fungal culture
3. Treatment of cats with ringworm should consist of isolation, systemic medication (itraconazole or terbinafine) and bathing

6. Feline leukaemia virus (FeLV)

Many Australian clinicians with whom we speak claim to rarely see FeLV infection in their private patients nowadays, and some have even ceased to test for it regularly. We agree that the prevalence of FeLV in Australian cats has probably reduced over the past thirty years, as has also been the general trend in Europe.[21] Note that FeLV is still quite prevalent in many parts of Asia and South America; for example, a recent study found 12% of privately-owned cats presenting to an emergency hospital in Malaysia were infected with FeLV. However, over the past five years we have seen enough cats with FeLV to say with confidence that it is still out there (Figure 13).[22] Australian veterinarians need to keep it on their list of diagnostic possibilities and be on the lookout if doing any work for local shelters or rescue organisations.

Of concern for us, in terms of spread of FeLV infection, are small rescue organisations that cannot afford to test all incoming cats for FeLV infection, and who practice group-housing without routinely vaccinating against FeLV. We have assisted two such organisations in Sydney, both with around 40-50 cats housed in a single facility, that had outbreaks of FeLV infection (including in some cats older than 10 years-of-age). The problem is amplified if FeLV-infected cats are rehomed to new owners before the shelter realises they are infected—it is these cats that Australian veterinarians in private clinical practice might see in the setting of FeLV-related disease, even if not specifically doing work for a shelter or rescue organisation. In passing, we would like to thank Martine Perkins and Andrea Harvey who have helped us track down these FeLV cases back to the rescue facilities they came from.

Due to the low FeLV prevalence in Australia (less than 2%), veterinarians testing for FeLV antigen with a point-of-care test kit need to beware of false-positive results[23], and **always confirm a positive in-house FeLV result with FeLV qPCR testing.** An excellent resource for referring to when testing for FeLV infection can be found on the ABCD website.[24]

We have been involved with the management of many FeLV-infected cats, and, despite the generally poor prognosis

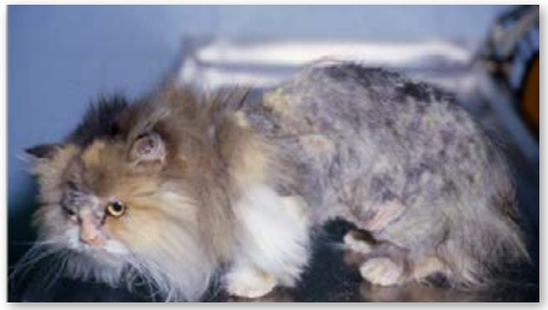

Figure 10: Severely affected long-haired cat with Microsporum canis. Photograph courtesy of Kim Conyers.

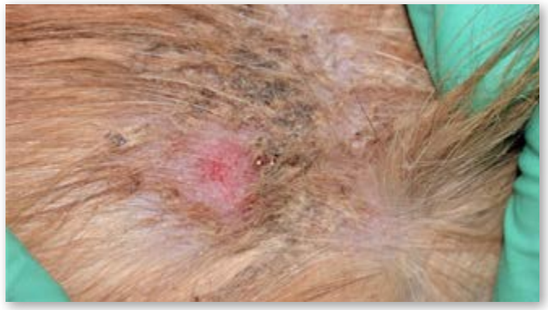

Figure 11a & b: More feline lesions due to M. canis. Photograph courtesy of Kim Conyers.

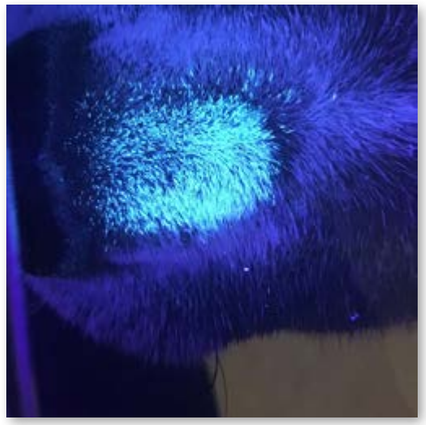

Figure 12: Dog with M. canis *lesions fluorescing under the Wood's lamp. Remember to let it heat up for 10 minutes before use! It can be a useful screening tool, but mycological culture is more reliable. Photograph courtesy of Kim Conyers.*

reported in the literature (a common figure used is that 90% of cats with progressive FeLV infection will die within three years of diagnosis, usually due to development of lymphoma, leukaemia or severe aplastic anaemia), **some will remain healthy for many years**. Our advice to veterinarians, therefore, is to treat the animal, not the statistics (i.e. a poor prognosis should not be used as a reason for euthanasia, if owners are willing to treat and agree to house the cat indoors to stop the spread to other cats). We are currently overseeing a medication trial for FeLV-infected cats in Australia using a novel drug manufactured in Malaysia that has shown some early promising results; if anyone is interested in finding out more please email mark.westman@sydney.edu.au. We are also trialing a treatment protocol for FeLV-infected cats using a triple drug approach that has been a highly effective antiretroviral therapy for HIV-positive patients; this protocol requires committed owners with good financial resources, as the drugs are expensive.

FeLV SUMMARY:

1. All incoming cats to a shelter or rescue facility should be screened for FeLV infection using a point-of-care antigen test kit (with confirmatory PCR testing performed on any positive samples)
2. If FeLV testing all incoming cats to a shelter or rescue facility is not possible, cats should be housed separately (or only mixed with cats vaccinated against FeLV)
3. Veterinarians should consider FeLV infection for any cats presenting with leukaemia, lymphoma or anaemia, particularly younger cats and kittens.

7. Feline immunodeficiency virus (FIV)

Like FeLV infection, FIV infection is commonly screened for by the larger rehoming organisations and shelters in Australia, and less commonly screened for by the smaller rehoming/rescue organisations. We performed the largest seroprevalence survey in Australia to date in 2011-2013, finding 15% of cats older than 2 years-of-age with some level of outdoor access were FIV-infected, while 6% of cats in a large shelter in WA were FIV-infected.[25] The American Association of Feline Practitioners recommends that all new cats entering a shelter should be tested for FIV (and FeLV) infection prior to rehoming.[26] We recommend all incoming cats older than 6 months-of-age, and kittens born to FIV-positive queens, should be FIV and FeLV tested.

FIV vaccination, which currently is only available in Australia, New Zealand and Japan, interferes with the diagnosis of FIV infection by some commercially available point-of-care antibody test kits[27] (e.g. SNAP Combo™ and Abaxis Rapid™). Two other FIV test kits, Witness™ and Anigen Rapid™, are generally not affected by FIV vaccination and are able to differentiate FIV-vaccinated and FIV-infected cats (unless the cat has received a primary course of FIV vaccination within the past 6 months[28]). For this reason, we recommend all

shelters screening for FIV infection should be using Witness™ or Anigen Rapid™ FIV test kits, since incorrect diagnosis of FIV infection by other FIV kits may result in unnecessary euthanasia or restrictions being placed on the cat for rehoming. To date, these are the only test kits that have been independently reviewed for performance in FIV-vaccinated cats, and we would therefore advise against using any other test kit for FIV screening until rigorous scientific evaluation has been performed. A helpful algorithm for determining FIV infection can be found in Figure 4 of this open access paper.[27]

Kittens born to FIV-positive queens can be FIV tested using an antibody test kit from as early as 12 weeks of age, although kittens that test FIV-positive should be retested 1-2 months later or confirmed by PCR testing in case of a false-positive result from persistence of circulating maternally-derived antibodies.

As clinicians would already be aware, the prognosis for a FIV-infected cat is much better than the prognosis for a FeLV-infected cat. In fact, although FIV-infected cats are approximately 5-6 times more likely to develop lymphoma than FIV-uninfected cats[29], if kept in low stress households

Figure 13: Multiple, firm, nodular subcutaneous lesions in a cat with feline sarcoma virus (FeSV), a result of recombination of FeLV with host proto-oncogenes. Photograph courtesy of Andrea Harvey.

many FIV-infected cats may live relatively normal lives.[30] Within stable households (i.e. minimal or no inter-cat aggression), FIV transmission between cats is rare.[31] Despite this, if considering rehoming a FIV-infected cat to a household with a FIV-uninfected cat, we recommend vaccinating the other cat against FIV. However, since the FIV vaccine only has 56% effectiveness, we would in addition recommend retesting the FIV-vaccinated cat for FIV infection annually to ensure no FIV 'vaccine breakthrough' has occurred in the preceding 12 months.[32]

FIV-infected cats should be individually housed while at the shelter, or only housed with other FIV-infected cats, to stop FIV transmission in stressful group-housing situations. A separate FIV facility away from FIV-uninfected cats is unnecessary, since normal cleaning and hand washing protocols will eliminate the risk of FIV transmission. Barrier nursing is also unnecessary since FIV is not transmitted by oral ingestion of FIV-laden saliva. It is good practice to not mix food and water bowls between FIV-infected and FIV-uninfected cats. However, since FIV is easily inactivated by most disinfectants due to its fragile envelope, routine washing in hot water with detergent will kill the virus.

FIV SUMMARY:

1. Shelters should screen for FIV infection using Witness™ or Anigen Rapid™ FIV test kits
2. Shelters that rehome FIV-positive cats should house them individually while at the shelter, or only group-house them with other FIV-positive cats
3. Shelters should recommend FIV vaccination of FIV-negative cats to be co-housed with a FIV-positive cat, and recommend annual retesting of FIV-vaccinated cats to detect vaccine breakthroughs
4. Shelters should help educate the public about FIV transmission and the prognosis for FIV-positive cats.

8. Feline panleukopenia virus (FPV)

Feline panleukopenia re-emerged as a clinical disease in Australia in 2014 after 30 years of absence in companion animal practice.[33] Caused by a (proto)parvovirus, kittens and incompletely or unvaccinated young cats are most at risk of infection. Canine parvovirus (CPV) has also been implicated in causing disease in cats in other jurisdictions. Cats can also be asymptomatic carriers of CPV[34] and therefore may be important reservoirs for the maintenance of infection in cats and dogs, which is important to remember when designing housing, cleaning and disinfection protocols in shelters and veterinary clinics.

Like its canine counterpart, feline parvovirus replicates within rapidly dividing cells. Enterocytes in crypts are damaged leading to severe gastrointestinal signs. Lymphoid tissue and bone marrow are also affected (hence the leukopenia), as is CNS tissue in cases of *in utero* infection. It is one author's experience (SM) that most kittens present with peracute illness. Within a 12-hour period, a kitten looking vaguely 'flat' can progress to severe illness or even death. Pyrexia and inappetence are commonly seen, followed by or associated with vomiting and watery diarrhoea. Within a single litter

of kittens, the full spectrum of clinical signs may be seen from asymptomatic infection to sudden death. In-house CPV antigen tests are routinely used for diagnosis of FPV infection. These must be interpreted in the light of clinical signs and not used as a screening test if modified live virus (MLV) vaccines are used, as these may cause a false-positive result for several weeks post-vaccination. Diagnosis can also be obtained using multiplex faecal qPCR panels.

Transmission is predominately via the faecal-oral route, although the virus may be shed in any body secretion. Since FPV is incredibly stable in the environment, fomites play an important role in transmitting infection. The virus is inactivated by bleach or, as one of the authors prefers (SM), activated hydrogen peroxide. **Kittens are particularly at risk in the maternally-derived immunity gap** when maternal antibodies are too low to provide passive immunity, but high enough to interfere with vaccination. Since the resurgence of the disease, many shelters have instituted a protocol of initial vaccination from 4 weeks (younger than this may cause cerebellar signs), then every 2-3 weeks until 18-20 weeks of age.

Treatment of FPV infection, as with CPV infection, is mainly supportive. Restoration of fluid balance, intravenous antibiotics due to the potential for bacteraemia and septicaemia are mainstays of therapy, and in some cases transfusion of typed fresh whole blood from well-vaccinated cats of the same blood group can be life-saving due to oncotic support and anti-parvovirus antibodies as well as red cells (Figures 14-16). Treatment with feline interferon-omega (rFeIFN-ω; Virbagen™) is likely beneficial.[35] However, despite intensive care and treatment, mortality rates with FPV infection remain considerable.

FPV SUMMARY:

1. Highly contagious, environmentally resistant virus
2. Vaccinate early and often in a shelter environment
3. Be mindful of the role of fomites in transmission and quarantine all kittens with unknown clinical histories
4. Use chlorine bleach or accelerated hydrogen peroxide for disinfection

9. Vaccination in the shelter setting

Vaccination is a key component of overall disease prevention strategies in any shelter environment. Vaccination guidelines for shelters vary from those for privately-owned animals due to inherent epidemiological factors which increase the risk of an animal in a shelter becoming infected with a preventable disease. For a more detailed discussion of vaccination guidelines in shelters, we recommend this helpful review by Richard Squires.[36]

Our advice to shelters is simple: Vaccinate often, vaccinate for longer and vaccinate using live vaccines.

Vaccinate often. Animals should be vaccinated with a core C3/F3 vaccine as soon as they enter the building (unless the animal is sick or injured), and dogs should also have an intranasal or intraoral 'kennel cough' vaccine administered at the same time. Note that this does NOT mean 2, or 4, or 8 hours after admission. **Animals need to be vaccinated within an hour of entry**. Vaccines should be stored correctly and reconstituted at the time of vaccination (i.e. not made up and stored in the fridge for later use). Animals then need to be re-vaccinated 2 weeks later. Puppies and kittens should be vaccinated as early as 4 weeks-of-age and then, ideally, fortnightly until 20 weeks of age (if still being housed at the shelter).

Vaccinate for longer. Despite the 'early finish' label claim by some vaccine manufacturers, we recommend vaccinating all shelter animals with a C3/F3 vaccine every 2 weeks until 20 weeks of age, as per the 2015 World Small Animal Veterinary Association (WSAVA) Vaccination Guidelines.[37]

Figure 14: Point-of-care blood typing kit using immunochromatography methodology.

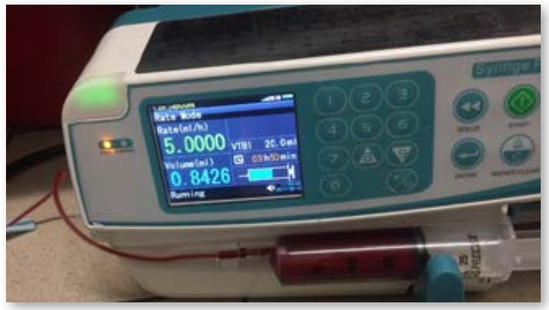

Figure 15: Giving a feline blood transfusion using a syringe driver. This permits slow and accurate rates of blood administration, and if you collect blood into 25 mL syringes preloaded with citrate phosphate dextrose, there is no double handling of blood, as it goes straight into the syringe driver warm and ready to administer.

Vaccinate using live vaccines. This recommendation relates to the parvovirus component of core vaccines (i.e. CPV/FPV), since modified live vaccines have been demonstrated to produce a faster onset of immunity, and more effective immunity[37] compared to killed adjuvanted vaccines. This is of particular importance in Australia due to the recent re-emergence of FPV.[33] The live versus killed vaccine debate is more ambiguous when it comes to FHV-1 and FCV infections, since there is some evidence that vaccination with a killed vaccine may produce a more effective response in cats against FHV-1 challenge.[38] However, given the dire consequences of FPV outbreaks in shelters (as well as in the community), and the relative treatability of FCV and FHV-1 infections compared to FPV, we recommend the use of live vaccines in a shelter setting.

Finally, it is helpful to remember that a single dose of either a live or killed vaccine is likely to produce long-lasting immunity in adult cats[39] (i.e. without a second dose being administered), which is extremely useful for cats that can only be vaccinated once (e.g. if too fractious to be handled again, or as part of trap-neuter-release programs).

Figure 16: Panleukopenia kitten receiving a blood transfusion. Photograph courtesy of David Hughes, Concord Veterinary Hospital.

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