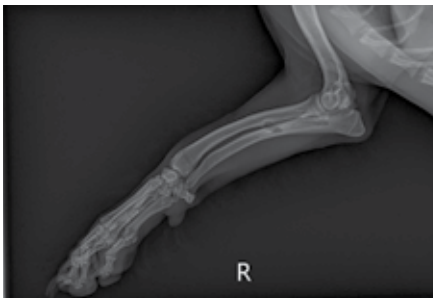
C&T

Issue 293 | December 2018

Centre for Veterinary Education

Control & Therapy Series



Danièlle's top tip

p45

C&T

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Control & Therapy Series

Australia's Leading Veterinarian Forum

Centre for Veterinary Education

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Contents

Happy holidays!

The CVE closes on Friday 21 December 2018, reopening on Tuesday 8 January 2019.

From the director

Here is the fourth and final C&T for 2018, which I am sure will provide interesting holiday reading for many of you. In true C&T tradition, this edition is packed with a wide variety of articles, including two more on heat stress in horses, written by Meg Brownlow, building on the articles on this topic she has contributed in the recent past.

We have included a monograph based on a talk given by omnicompetent country practitioner John Dooley to our Recent Graduate seminar twelve months ago. We believe John's talk touched on a lot of truisms and provided some excellent advice for graduates in general practice, especially recent graduates. John has been a mentor to many younger members of the profession for his whole career and his down to earth approach to practice is as applicable today as it was thirty or more years ago.

There is also a provocative article questioning why it is so hard to fill veterinary positions in general practice. This seems to be a recurring situation that has been raised with me by vets from all over the country. The problem does not appear to be restricted to regional or remote locations, and flies in the face of the large cohort of new graduates from the 7 Australian vet schools. Many practice owners in busy small animal practices are facing the same problems of finding employees, even when there are flexible working arrangements, no after-hours work and a location that would once have been considered highly desirable. I have asked Jo Krockenberger to continue to explore this issue in future C&Ts in order to better understand the ongoing shifts occurring within the profession.

The CVE is pleased to announce that we have appointed a veterinarian as Deputy Director, commencing in January 2019. Dr Simone Maher will bring a younger, vibrant, female approach to the CVE leadership team and hopefully will enable us to better connect with the younger members of the profession. We will have more on Simone's background and experience in the first C&T for 2019.

For those of you who have been impacted by the severe drought ravaging so much of eastern Australia, we hope that you and your clients receive a reprieve in the coming months despite the El Nino effect and that the drought does not persist much longer. Having spent most of my life in mixed practice I know only too well the impact that drought has on your clients as well as practice, and how long it takes everyone to recover. We at the CVE wish you well and hope that Christmas brings you some joy and relief from your everyday worries.

I hope you enjoy this C&T over the festive break and look forward to seeing you at conferences and events in the new Year. Don't forget that you and your team will benefit from continuing professional development and that in 2019 the CVE will remain the premium provider of high quality continuing veterinary education, starting with our annual distance education courses and the Sydney February Cardio-respiratory conference.

Hugh White
Director

Calendar

Conference

SYDNEY

State of the Heart Cardiorespiratory Conference
Mon 18 – Thu 21 Feb 2019 + Workshops x2 Fri 22 Feb 2019

One Welfare Conference II
Mon 14 – Tue 15 Oct 2019 + Four-Words, Forwards! (Mental Health Workshop)
Wed 16 Oct 2019

MELBOURNE

Critical Care Internal Medicine and Surgery - Mon 17 – Thu 21 June 2019

CAIRNS

Neurology Conference
Mon 9 – Thu 12 Sep 2019

Seminars

SYDNEY

Dentistry Theory & Practice
Fri 12 – Sat 13 April 2019

Practical Ultrasound Intensive Short Course
Mon 15 – 19 July 2019

Ophthalmology: Theory & Practice
Fri 25 – Sat 26 October 2019

Hands-on Workshops

SYDNEY

Basic Echocardiography
Sat 23 February 2019

Advanced Echocardiography
Sun 24 February 2019

Back to Basics: Diagnostic Ultrasound
Sat 16 March 2019

ADELAIDE

Hip & Stifle
Sat 23 March 2019

Bone Plating
Sun 24 March 2019

TOWNSVILLE

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

2018 - 2019

December

Su	Mo	Tu	We	Th	Fr	Sa
30	31					1
2	3	4	5	6	7	8
9	10	11	12	13	14	15
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January

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February

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March

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April

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28	29	30				

May

Su	Mo	Tu	We	Th	Fr	Sa
			1	2	3	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	

Hands-on Workshops

SYDNEY

Soft Tissue
Sat 26 – 27 October 2019

Hip & Stifle
Sat 9 November 2019

Bone Plating
Sun 10 November 2019

QUEENSLAND

Soft Tissue
Sat 2 – 3 November 2019

TimeOnline

Prevention & Treatment of Neonatal Calf Disease

Dermatology

Dental Bites for the Enlightened Practitioner

Backyard Chickens

Demystifying ECGs

Essential Wellbeing & Coping Skills for Veterinarians

Respiratory Failure

Reptile Medicine

Rabbits and Rodents

Small Animal Nutrition

For full list of TimeOnline 2019 topics and dates visit cve.edu.au

Distance Education

Beef Production Medicine

Behavioural Medicine

Cardiorespiratory Medicine

Clinical Pathology

Dermatology

Emergency Medicine

Feline Medicine

Internal Medicine: A Problem Solving Approach FULL

Internal Medicine: Keys to Understanding

Ophthalmology

Surgery FULL

Thoracic Imaging

Musculoskeletal Imaging

Abdominal Imaging

Clinical Neurology
Important course date:
1 Feb 2019 - DE program start!

PodcastPLUS

Dental radiology: A beginner's guide to radiographic interpretation
28 February – 7 March 2019

Clinical reasoning in veterinary Neurology – How to become more confident with neurology patients?
28 March – 4 April 2019

Calendar Key

- Conference
- Seminar
- Hands-on Workshop
- Distance Education
- TimeOnline
- PodcastPLUS
- CVE closed

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

BEST VISUALS

CVE PUBLICATION PRIZE WINNERS

PRIZE ENTITLEMENTS

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.

WINNER OF BEST VISUALS + SMALL

Mandibular symphyseal fracture using acrylic Splint

Pete Coleshaw

Jaffa's Health Centre for Cats
Salisbury, United Kingdom

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C&T No. 5707

Repair of mandibular symphyseal fracture using cerclage wires has a number of potential complications including poor stability from insufficient wire tension to gingival damage and discomfort from excessive tension. Acrylic splinting is an easy way of achieving rock-steady apposition without any risk of iatrogenic damage.

The principle is to oppose tissues with monofilament suture, tensioning the repair with successive figures of 8, stabilising with dental acrylic (Protemp).

Reduce fracture, having ruled out further fractures of mandible.

Clean teeth, then acid-etch the enamel to ensure good adhesion of the acrylic.

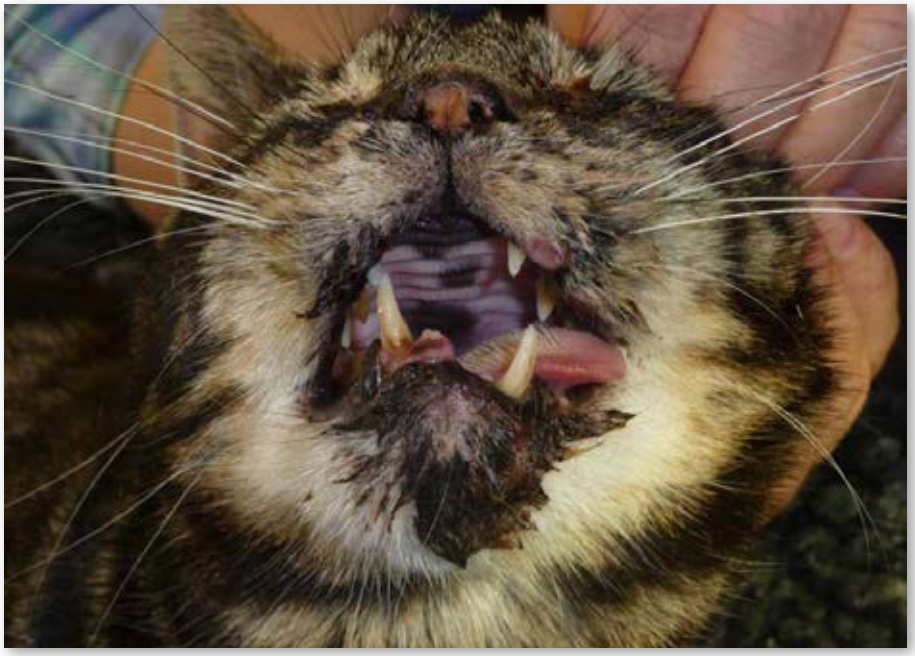
Once applied, allow to work for 10 seconds or so then wash off thoroughly ensuring none goes in the mouth – it's concentrated phosphoric acid!

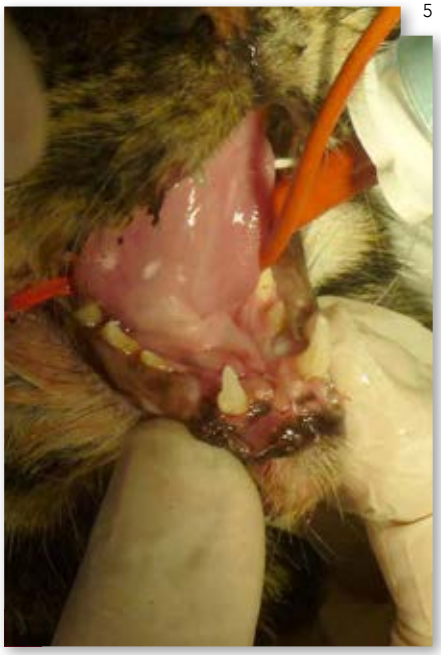
Blow dry with the air-feed of the dental unit, and apply a drop of bonding agent. Blow this to a thin microfilm which is then light-cured.

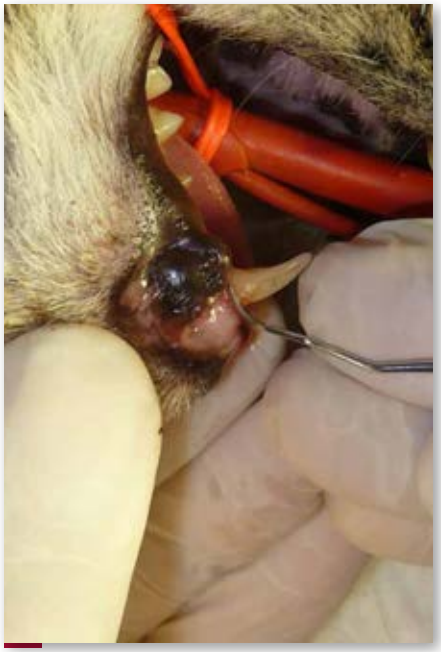
Although you can manage without this step, stop the sutures slipping off when tying between the teeth by applying a small drop of light-cured dental composite to the lateral aspect of each canine. Alternatively you could use rapid epoxy or a tiny blob of epoxy putty.

Tie around the tooth using whatever monofilament you have on the shelf. Successive wraps/knots can tighten the repair, allowing minor changes before achieving the final position. Try to make sure the sutures don't cut into the gums. Extubate and ensure good alignment. Intubate again.

Apply dental acrylic onto the suture framework avoiding excessive product on the buccal aspect to enable normal occlusal. You need to work very quickly as setting time is rapid – have a play first to get a feel for the material, its flow properties, and speed of setting. A Teflon spatula assists in moulding where needed. It normally takes on its own form (you will understand when you have used the







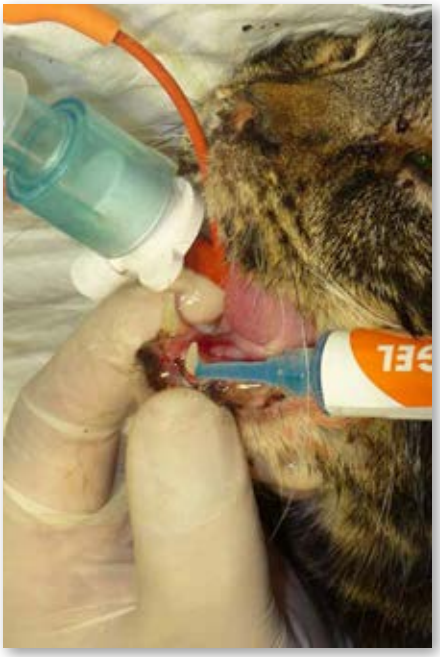


Figure 1. Mandibular symphyseal fracture

Figure 2. Reduce mandibular fracture

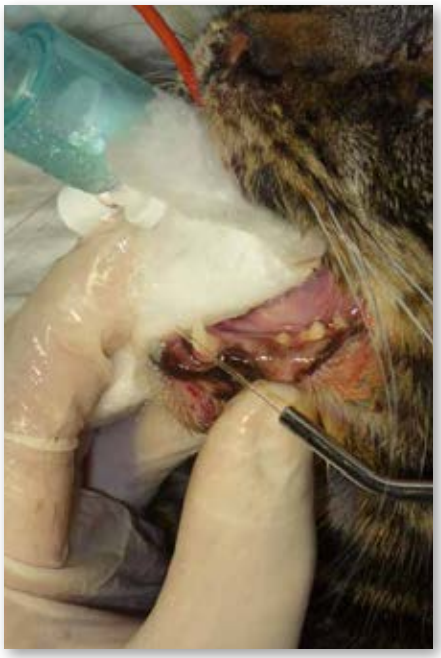
Figure 3. Clean teeth

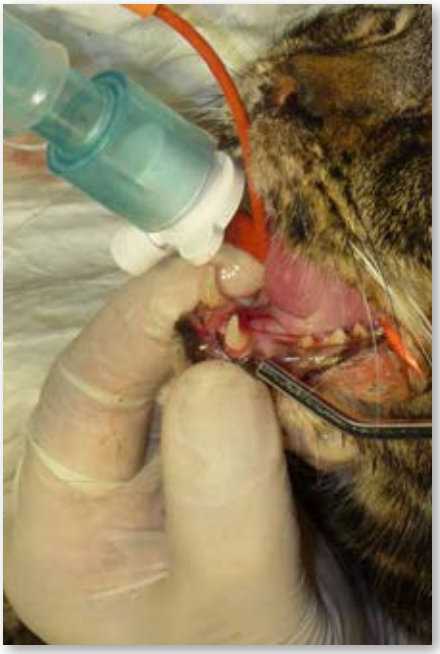
Figure 4. Acid etch enamel

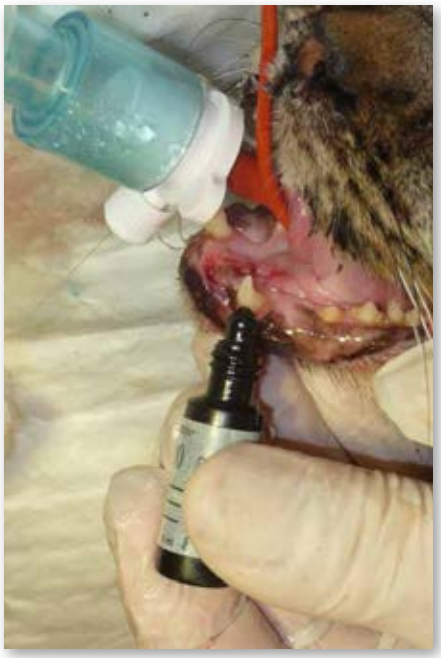
Figure 5. Rinse thoroughly

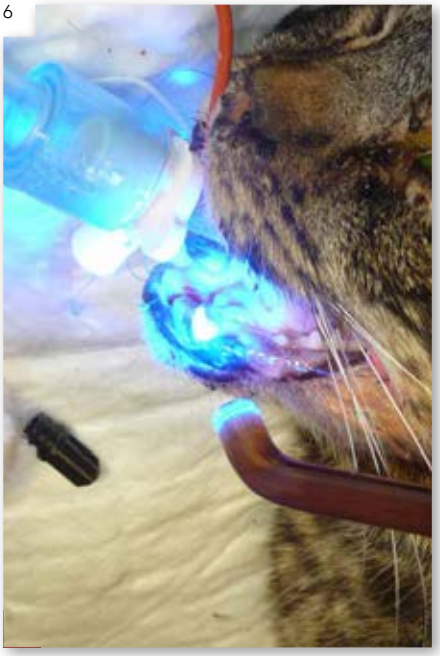
Figure 6. Dry

Figure 7. Apply a drop of bonding agent









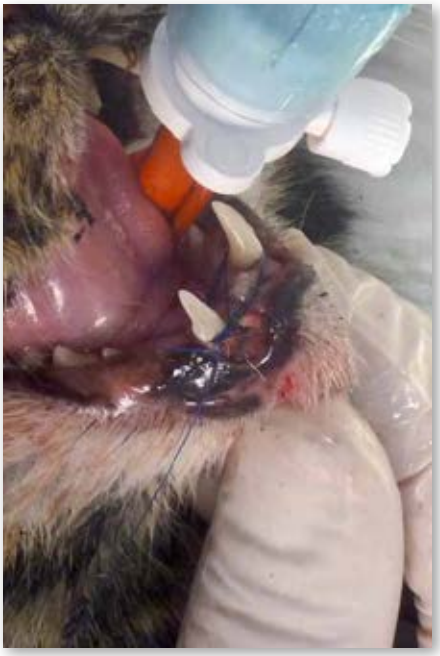


Figure 8. Blow dry bonding agent to a thin microfilm and light cure

Figure 9. Apply a small drop of light-cured dental composite to the lateral aspect of each canine

Figure 10 Use monofilament suture to wrap around canines

Figure 11. Dental acrylic applied to suture framework

Figure 12. Remove excessive product to enable normal occlusion

stuff) but some persuasion may be needed – in particular try to keep it clear of the gums.

6-8 weeks later under general anaesthesia it can be removed by sectioning with a dental bur and crunching/teasing off the dental arcade.

eBook video: Watch Pete apply an acrylic splint to stabilise a mandibular symphyseal fracture.

eBook download:
C&T No. 5685 Pete Coleshaw, My Life as a Vet, Issue 291, June 2018

Can you trust your in-house clinical pathology analyser?

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C&T No. 5708

Randolph Baral has been in private feline practice for over 20 years. He has published widely about and achieved his PhD on clinical biochemistry, including assessing in-house analysers.

When veterinarians hear that I have assessed the performance of in-house clinical pathology analysers, the first question I am typically asked is either:

- 'Is X analyser better than Y analyser?' or
- 'Which analyser is the best?'

I'm not sure what readers expect but there's no easy yes/no answer!

When choosing a new analyser, or assessing the performance of your current analyser, the most important question should be: **'Can I rely upon the results?'**

There are two ways to assess analysers and, ideally, both should be done.

1. precision (repeatability)
2. bias (variation from a known standard).

All in-house analysers show bias from commercial lab analysers (which also show bias between each other). This means you can't directly compare results from one analyser to another....so a comparison to results determined at a commercial lab or on a prior in-house analyser is not possible.

You could interpret that to mean that precision is more important to assess since that is considering the analyser in isolation.

Precision (repeatability):

Precision is assessed by running the same sample on multiple occasions. It is typically done at labs with 'artificial' quality control solutions (QCM) (usually bovine or human serum that has been spiked with analytes to get high enough concentrations of them all) but the best to use is pooled plasma.....it is typically not done at all in practice!

We have used commercial QCM but also collect excess plasma over a working week and pool it. We recently assessed a new analyte and spent a week collecting 2 pools (normal cats and cats with renal disease so we would have two concentrations to assess); at the end of the week, I ensured they were mixed (gently up down multiple times with 12mL syringe), then divided into 0.6mL aliquots and froze them all; the following week, my staff got out one of each frozen aliquot each morning, and, when defrosted, ran the sample. In this case, we ran one for the new analyte and one other as a control (in this case creatinine) to ensure there were not unforseen issues with the analyser.

This process should be done EVERY TIME you get a NEW analyser and ANNUALLY. Yes, there is a cost; build it into your lab testing pricing (it's not that much extra per test when you spread that cost over a year).

How many tests are good enough? I think 20 is ideal. We do 20 at a new purchase and 10 annually (i.e. needs to be of EACH analyte so full profile). In the current assessment, I could only get enough for 12 (and you don't want to take too long to collect samples as you won't be certain about how stable the plasma is beyond about 5 days).

There is also a difference in testing on consecutive days (between day) or all on the same day (within day). I prefer between day as that will give you an idea of day to day variation (within day gives less variation).

To actually assess precision from your results, I use Excel....put the daily (or within day) results across a row and at the end of the row, determine the mean and standard deviation, then divide SD by mean and you have coefficient of variation....and that % is your measure of precision. I have attached a screenshot of a chart from our recent study that has the formulae on it already. I have left the cursor on one of the standard deviation cells so you can see the formula.

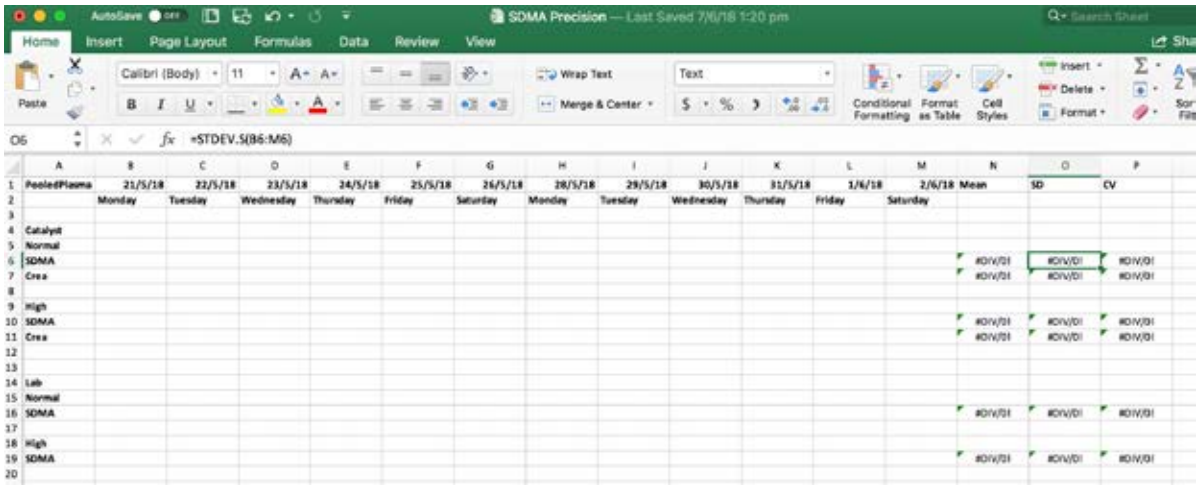
Continued...

The formulae for the first rows are:

- Mean = AVERAGE(B6:M6)
- Standard Deviation = STDEV.S(B6:M6)
- Coefficient of Variation = Standard Deviation/ Mean (so =O6/P6)

(You can copy and paste the formulae and Excel is smart enough to adapt to whatever row number you are up to).

Feel free to adapt it to your uses.



What is good enough precision? The accepted ideal standard in human clin path (and I just cowrote a review for *Vet Clin Path* that echoes this) is half of the known biological variation of each analyte.....which means half of the known physiological, in vivo variation.....which should be taken from the median values on the charts at http://vetbiologicalvariation.org/

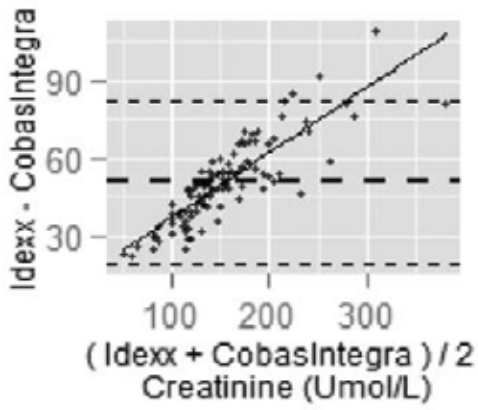
Having analyser variation (precision) at no greater than half individual variation (biological variation) means that the total variation in any sample contributed to by the analyser will be no greater than 12%. (If anyone is REALLY interested, respond to CVE and I can write a piece about the maths for that).

Why is the acceptable precision percentage different for different analytes? The differences relate to how tightly controlled the various analytes are in the body. Consider if you demonstrate precision of 10%: 10% variation in a result (just due to the analyser) would not make much difference to a bilirubin sample (so 7µmol/L might be as low as 6.3µmol/L or as high as 7.7µmol/L) but consider sodium where a result of 155mmol/L with 10% variation might mean a result as low as 139.5mmol/L or as high as 170.5mmol/L.

Bias (difference between analysers):

Bias, the variation between analysers, is assessed by checking results (from the same blood draw) on the new analyser and a known standard. These are best compared by Bland-Altman plots (i.e. assess difference between 2 samples from same draw compared to mean of the 2 samples) over a range of concentrations. I feel the difference is best assessed as % difference when assessing multiple analytes so there is a single basis of comparison but little difference if assessing just one analyte at a time. The main questions you want to assess are: how great is the difference between the 2 and does that difference vary by concentration, meaning the amount of difference varies with concentration being measured (i.e. proportional bias). How great a difference is acceptable can be debated – maybe it doesn't matter if you consider the analyser a closed system....NO proportional bias is ideal (but again may not matter if a closed system). All of this assumes that the comparison analyser (usually a commercial lab) is correct, and e.g. creatinine is often BETTER on in-house equipment which uses enzymatic method to determine compared to Jaffe method typically used at labs (more labs are going over to the enzymatic).

This figure below shows that the mean difference between this in-house analyser and the commercial laboratory is approximately 50µmol/L (bold dashed line) but the bias is proportional with the difference between analysers varying from about 25µmol/L at a concentration of 50µmol/L (lower left of figure) to a difference of about 100µmol/L when the concentration is about 300µmol/L. None of this is to say that either analyser is 'right' or 'wrong'. They are each true within their own 'universe', you just can't compare results between the two analysers.



The upshot:

If you want to be able to trust your in-house analyser, the most important assessment to make is to check precision as outlined above. If you are not satisfied with the results, I recommend speaking to the manufacturer about how it can be improved. Checking bias is ideal to do but unlikely to affect your day-to-day results (unless you routinely assess results from both an in-house analyser and a commercial laboratory).

SMALL

What caused that sudden swelling?

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C&T No. 5709

I had a Chihuahua present with a swollen left upper lip. It was agitated and dead against a clinical examination so I agreed with the dog. I tentatively diagnosed a probable insect bite and gave it 1mL Betamox and 1mL 2% Dex with the instructions to ring back if it did not improve.

I told the owner that I have covered everything except snake bite. Whereby the owner said she had never seen a snake around her place but she did live near a nature reserve.

The next day the dog was presented very depressed and passing some blood in the urine. The lip was easily turned over and 2 fang marks were found on the inside.

My area is near a river and Red Belly Black Snakes (RBBS) are quite common; I even had one little fellow in my kitchen 10 years back. I am pretty certain I lost my Jack Russell to a RBBS a couple of years ago on a long weekend so I always have a bottle of Tiger Snake (TS) and RBBS antivenom available in-house these days, even though it is expensive.

Bloods were taken and the results (see below) came back and added weight to our clinical diagnosis. There was a a non-regenerative or pre-regenerative anaemia (HCT 0.23 L/L; reticulocyte count 80x10 9/L hyperbilirubinaemia (12 µmol/L) and increased acitivities of AST (1770 IU/L), ALT (107 IU/L) and CK (15683 IU/L). The serum protein was normal so it was thought that the anaemia and hyperbilirubinaemia could be due to acute haemolysis. With this CK and AST being so much higher than the ALT, it was likely that all 3 represented very severe muscle damage. Both were potentially consistent with red-bellied black snake envenomation

2mL 2% Dex was given 10 minutes before the antivenom. Ross Sillar (Northern Serum Laboratory) the tick guru has always said you don't treat the size of the dog only the amount of venom/symptoms. So the full 10mL vial was given slowly IV and put it in an oxygen tent.

Three hours later the dog was on its feet and barking. The recovery was uneventful over the next few days.

Hindsight Post Mortem

Without a witness, a snake carcass or fang marks, a snake bite is easily initially missed and identification of snake species an educated guess.

Compared with insect stings, snake bites are rare in suburbia.Blood in the urine within 24 hours or less of the bite and a very depressed dog should get the bells ringing if it is a Tiger Snake (TS) or Red Bellied Black Snake (RBBS).

In this case a good dose of ACP and examination of the inner lip would have confirmed the diagnosis immediately but if bitten elsewhere it could be a shave job.

Horses bitten by RBBS usually lie down for 3 days and recover uneventfully. Joe Levy, a Master Farrier, put me onto this little gem. I have seen 2 of my wife's horses with

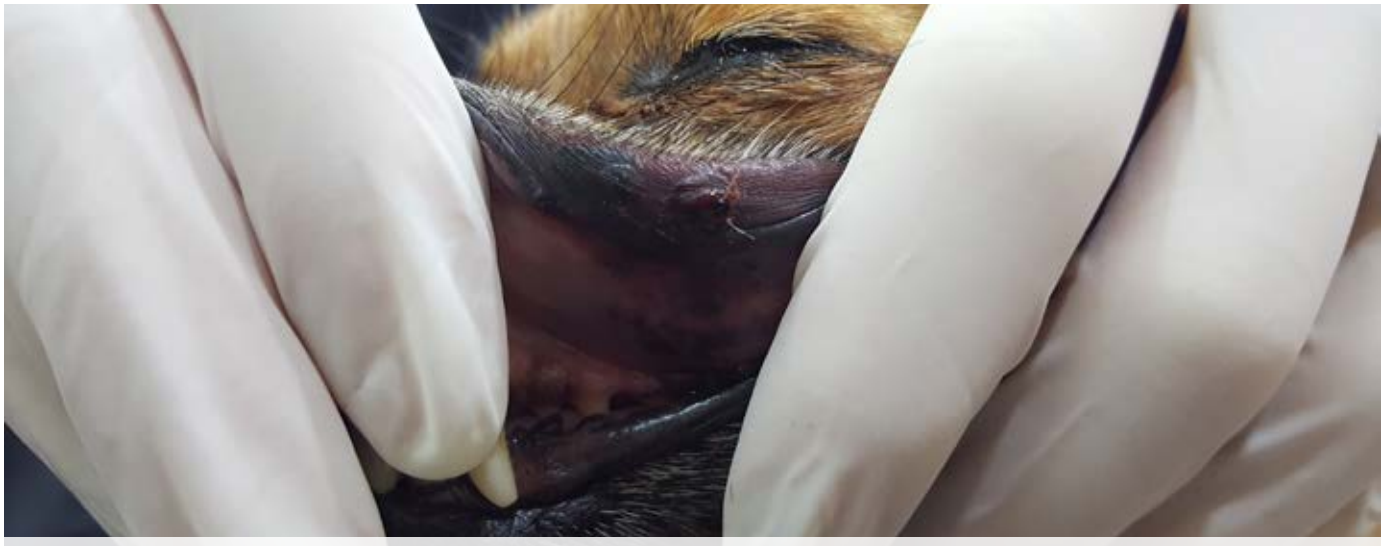

Figure 1. Fang marks visible on mucosa

these symptoms. It appears humans basically get the horse treatment these days and no RBBS antivenom.

Dog fatalities probably depend on size and the amount of venom but, after observing the effects of TS/RBBS antivenom, I would strongly recommend its use.

Bloods, though not necessary with a witness or carcass, are extremely useful to confirm a diagnosis without a witness.

VETNOSTICS

Hb	100 * g/L	(115-180)	WBC	18.6* x10^9/L	(6-14)
RCC	3.2 *x10^{12}/L	(5.0-8.0)	Neut	16.6*x10^9/L	(4.1-9.4)
Hct	0.23*	(0.37-0.55)	Lymph	1.5x10^9/L	(0.9-3.6)
MCV	72 fL	(60-74)	Mono	0.2x10^9/L	(0.2-1.0)
MCH	31*pg	(20-25)	Eos	0.2x10^9/L	(0.1-1.2)
MCHC	435*g/L	(310-360)	Baso	0.0x10^9/L	(<0.2)
NRBC	5/100 WBCs				
Plat	300x10^9/L	(200-900)			
Retic	2.5*%	(<2.1)	Band	0.2*x10^9/L	(<0.1)
abs	80x10^9/L				

Red Cells:	Polychromasia +
	Occasional Nucleated red blood cell.
White Cells:	Leukocytosis +, Neutrophilia +
	Occasional toxic changes, Band forms.
Platelets	Normal

NOTE : WBC parameters corrected for presence of NRBC.
NOTE : Manual PCV performed. Manual platelet estimate performed.

Fasting status	Random	
Sodium	145 mmol/L	(140-155)
Potassium	3.8 mmol/L	(3.8-5.8)
Chloride	106 mmol/L	(100-120)
Bicarbonate	14*mmol/L	(16-24)
Anion gap	29*mmol/L	(15-25)
Urea	6.3mmol/L	(2.5-9.0)
Creatinine	50µmol/L	(40-140)
Serum Glucose	3.7mmol/L	(3.5-6.7)
Bilirubin	12*µmol/L	(<11)
AST	1770*U/L	(1-80)
ALT	107*U/L	(<80)
GGT	<5U/L	(<6)
Alkaline Phosphatase	34U/L	(1-120)
Protein	69g/L	(55-78)
Albumin	31g/L	(22-36)
Globulin	38g/L	(25-40)
Albumin/Globulin Ratio	0.8	
Calcium	2.06 mmol/L	(2.00-2.80)
Phosphate	1.45mmol/L	(0.80-2.00)
Creatine Kinase	15683 *U/L	(<401)
Cholesterol	3.5*mmol/L	(3.6-8.8)
Triglyceride	1.2mmol/L	(0.2-1.7)
Haemolysis	4+	

Case Study: Large (well, massive) urolith in a 7yo mare

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C&T No. 5710

History;

This mare had been expressing prolonged 'oestrous' behaviour noted by the owners, with increasing frequency and intensity. For a couple of months prior to my being called to examine this mare, the owners had been intending to bring her to the clinic for ultrasound and further investigation, however, this had just not happened. Looking even further back, this mare was seen at a pony club event to urinate a puddle of urine that was visually blood tinged to the owners. No further blood tinged urine had been noted.

I was called early on a Monday morning to come and visit this mare, as she had been displaying symptoms of discomfort with increasing severity over the past week. These symptoms had escalated particularly overnight and the mare was now down and very unhappy. The owners didn't know if she had colic (but she was eating and passing manure), was in labour (but was definitely not pregnant), or was trying to urinate (but would pass some urine at times).

Clinical presentation;

The mare was standing upon my arrival and was eating hay. She was not pawing at the ground or trying to lie down. She was not sweaty, but was covered in mud where she had been laying down. Her demeanor was a little dull. T 37.4°C, HR 60bpm, no arrhythmias, gut sounds normal, respiratory sounds normal. Mucous membranes were pale but with CRT<2s. Muscle twitching was persistently present in the dorsal quadrants of the abdomen on both sides. Urine staining and scalding was present down the insides of the back legs. The mare was straining, but was passing small amounts of both urine and manure.

A vaginal exam was made (with the intention to follow onto rectal if this was unremarkable), which revealed a hard urolith just within the urethra.

The mare was sedated with 0.5mL dormosedan and 0.5mL butorphenol IV. Some progress was made towards manually extracting this stone; however, the mare objected to complete removal and it was deemed unsafe to continue in this way. The decision was made to perform a general anaesthesia. This was achieved via 5mL xylazine 100mg/mL and 8mL ketamine 50mg/mL. Manual extraction was achieved with relative ease and was not especially traumatic to the tissues. The urethra was well dilated now, and I could manually examine the bladder via the urethra vaginally. No further stones were palpable.

The mare recovered from the anaesthetic without complication. Trisoprim 480 was administered 25mL IM SID 4 days.

The stone was a palm sized calcium carbonate urolith, measuring 8 x 6 x 5cm.

Discussion;

This mare had been predominantly pasture fed, the pasture contained a high proportion of kikuyu. Kikuyu is a high oxalate pasture, the oxalates binding preferentially to the calcium making its bioavailability very low. The calcium is then excreted in the urine, resulting in a calcium carbonate precipitate developing within the bladder. This mare needed to be removed from that pasture (a rye pasture was available), and needed to be fed a diet which is conservative in calcium levels. Anecdotally, acidifying the urine will help reduce the likelihood of recurrence, however products such as ammonium chloride, ammonium sulphate and vitamin C need to be fed in quantities that are not usually tolerated from a palatability point of view. The diet will be supplemented with humidimix, which contains some ammonium chloride, but the added salts will hopefully also encourage increased water consumption and urine dilution.

One could think that this mare had been growing this stone for more than 18 months when the episode of haematuria was noted by the owners.

Also anecdotally, rates of recurrence seem to be low.

The owners of the mare described this mare as being significantly better the following day.

COMMENT COURTESY OF GLENN SHEA

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I guess not surprisingly, there are few data on the diameter of the horse urethra. Passing a urolith this diameter could only happen in a mare – the male urethra couldn't accommodate anything more than about a cm wide at absolute maximum (and even less once you got to the urethral process at the apex of the penis – more like about 3-5mm at full diameter).

For the mare urethra, I'd normally consider it to be about 1-2cm diameter when normally distended during the passage of urine. This fits the statement in Sisson & Grossmann's *Anatomy of the Domestic Animals*, which states 'its lumen is sufficient to easily allow the introduction of the finger', but then introduces the potential for further dilation: 'it is, however, capable of remarkable dilatation if sufficient care and patience are exercised in the process' (I guess like a hand-size urolith being pushed along it over several hours!). Unlike the male urethra, which is constrained caudally by the ischial arch, and in the penis by the corpora cavernosa dorsolaterally, and the urethral process at the glans, the wall of the mare urethra consists of softer tissues: lots of smooth muscle and a richly vascular submucosa (thought to be at least partially erectile). Take it slowly, and you can stretch smooth muscle a lot!

Fluoroscopic-guided endoscopic removal of screw foreign body in a chicken

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History

Elkie is my own 12-month old Isa Brown free-ranging chicken that loves to eat anything and everything. Over four days Elkie became progressively inappetant, lethargic and stopped laying. Elkie's normal diet consisted of layer pellets, lots of green vegetables and any other kitchen scraps that came about. Being an Isa Brown chicken my initial differential diagnoses were reproductive tract disease; specifically egg binding, egg peritonitis or uterine neoplasia, with gastrointestinal diseases such as crop impaction, endoparasites or foreign body considered less likely.

Initial work up

Physical exam – Elkie weighed 1.9kg, she was in good feather condition, had a dull demeanor, her comb was notably paler than it had been in preceding weeks, she had normal cardiac and thoracic auscultation and was normothermic. Digital cloacal exam was unremarkable.

Radiographs – radiographs were the first test performed given the primary differentials of an egg-based disease. Radiographs showed a metallic screw in her proventriculus but were otherwise unremarkable.

Blood testing – full haematology screening was unremarkable. Zinc serum testing was performed, in case the screw was zinc based and had been leeching into her gastrointestinal tract. The results took two days to come back and were normal.

Telephone consultation with Dr Hamish Baron (Avian and Exotic Veterinarian) deemed that the metallic screw was likely clinically significant (given an otherwise unremarkable work up) with primary concern of pain and potential for perforation if left in place.

Treatment

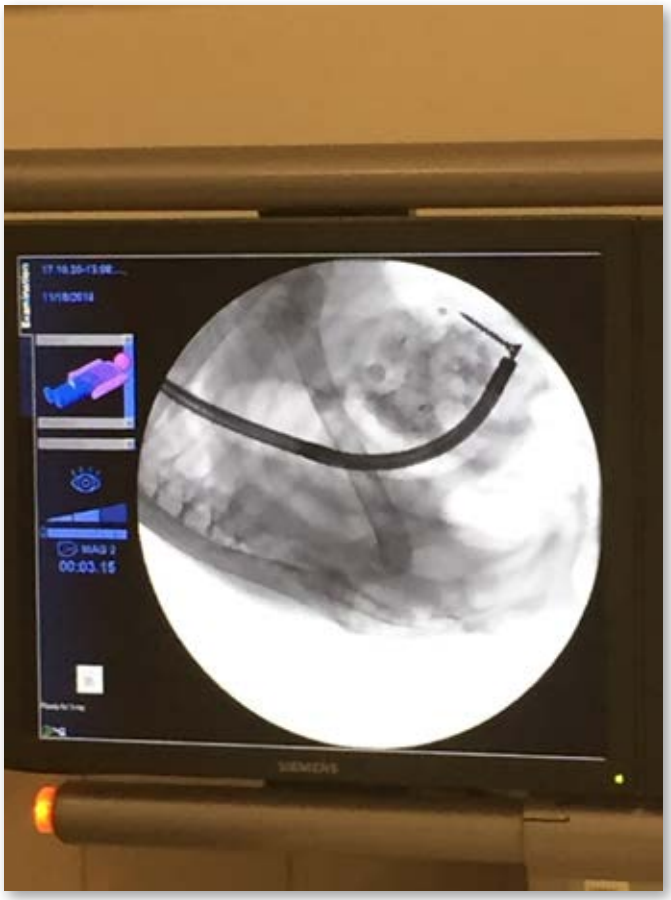
Elkie was placed on meloxicam 1mg/kg PO for analgesia. She started eating again the next day. Five days later she was anaesthetized for endoscopy. Elkie was fasted for four hours, with her crop palpably empty at this time. Premedication consisted of midazolam 2mg/kg and butorphanol 1mg/kg intramuscularly. This provided excellent sedation. A 24 gauge intravenous catheter was placed into right medial metatarsal vein. Anaesthesia was induced with alfaxalone to effect (total dose of 15mg/kg). Induction quality was stormy with a phase of excitement (wing flapping and dorsoflexion of neck) occurring before muscle relaxation. Intubation was performed with an uncuffed 3.5 endotracheal tube. Anaesthesia was maintained with isoflurane in oxygen. A multiparameter monitor was used for monitoring of pulse oximetry, end-tidal carbon dioxide and non-invasive blood pressure. A Doppler probe was also placed over the wing artery. Elkie maintained spontaneous respiration during the procedure. Intravenous fluids (Hartmanns solution) were administered at 5mL/kg/hr.

Dr Christine Griebsch and Dr Amanda Taylor (two small animal clinicians taking on their first chicken patient) undertook the task of fluoroscopic-guided endoscopy to remove the screw. Success was achieved after 100 minutes of anaesthesia. This is a particularly

Figure 1. Elkie following induction of anaesthesia with alfaxalone and intubation with a 3.0 uncuffed endotracheal tube

Figure 2. Monitoring of anaesthesia was performed with pulse oximeter placed into comb, doppler placed over wing artery, non-invasive blood pressure cuff on limb and capnograph

Fig



long anaesthetic for a chicken, however Elkie made an unremarkable recovery. She received meloxicam 1mg/kg SC and butorphanol 2mg/kg IM post-operatively.

Follow up

Elkie was back at home in the arms of her owner that evening. She resumed normal behaviour including normal eating within four hours post-operatively. She did not require any further medications.

COMMENT ON FLOUROSCOPIC SCREW RETRIEVAL OF A HEN COURTESY OF

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The vets here have done a remarkable job and should be congratulated. Just a couple of pointers:

1. I'm not a fan of alfaxalone in birds. The stormy induction does not surprise me at all. I will usually mask induce after sedation, and then intubate. If I need an IV induction I will use medetomidine-ketamine (equal volumes of each in the same syringe, starting with a combined dose of 0.2mL then given incrementally to effect)
2. Other options for retrieval could have been:
    a. Ventricululotomy via a midline coeliotomy. A nightmare approach given the thick muscular wall of the ventriculus in a chicken
    b. Endoscopic approach via the mouth or via the crop through an ingluviotomy
    c. Flushing the ventriculus can often wash out material but I would be very concerned doing that with a pointy screw in there
    d. A flank approach into the proventriculus could also have been used, but is subject to wound breakdown (although the chicken proventricular wall is pretty thick compared to parrots
3. 100 minutes is not too long for a bird anaesthetic so long as the patient receives:
    a. Thermal support – keep them warm!
    b. Cardiovascular support – IV fluids
    c. Respiratory support – I always manually/ mechanically ventilate anything under for more than 10 minutes

On the whole, a great job very well done and I'm just a little bit jealous!

Congratulations to DE tutor Bob Doneley who is the first Australian to win the prestigious TJ Lafeber Avian Practitioner of the Year Award in 2018. Bob's Backyard Chickens TimeOnline course is one of the most popular and runs again on 1 - 29 April 2019.

Malignancy associated neutropaenia in a cat

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C&T No. 5712

Dexter was an 8-year-old DSH who was rehomed from a shelter many years ago and then belonged to my vet nurse. Dexter was a healthy cat up until 18 months ago.

At that time, Dexter presented with triaditis and responded well to treatment. Initially he had a mild regenerative anaemia but all further investigative tests performed were negative (Haemotropic Mycoplasma etc.).

At this stage, an in-house FeLV test was weakly positive but FIV was negative. We sent this to IDEXX where the PCR was positive but ELISA was negative. This is consistent with a so-called regressive infection. Throughout this entire process he had severe neutropaenia. (His levels got as low as 0.1). I spoke to Bruce Mackay at the time, it was a possibility that FeLV was the aetiology and immunosuppressive doses of prednisolone were given which improved the neutrophils count.

Dexter did well on prednisolone and was otherwise clinically well; however, every time we tapered the prednisolone dose he would develop neutropaenia, suggesting the neutropenia was secondary to something (e.g. FeLV). There were no signs of anaemia during this period.

Around October 2017 I was in contact with Mark Westman at the Sydney School of Veterinary Science because of his research interest in feline retroviral disease and specifically the pathogenesis of FeLV related disease and the efficacy of diagnostic testing. Mark ran multiple FeLV tests and found that while Dexter was initially positive with in-clinic screening tests (Anigen Rapid and SNAP Combo), he was later negative on Anigen Rapid. PCR at the University of Sydney was interpreted as negative (testing was negative in three of the quadruplicates), although the possibility of regressive infection was not completely excluded.

We were happy to conclude that Dexter was FeLV negative at this stage.

Dexter was still clinically well at this time and so we decided to try cyclosporine as a treatment for immune-mediated neutropenia. This was going well until around January. We did a trough cyclosporine level at the end of January and the levels were excessively high at around 2000. Soon after, Dexter presented with pyrexia,

regenerative anaemia (PCV 20) and neutropaenia despite the immunosuppressive doses of cyclosporine. He had small bowel diarrhoea prior to presentation for about 5 or so days. He was placed on clindamycin (just in case of toxoplasmosis). Initially he improved for a few days but then relapsed. We discontinued the cyclosporine just in case severe sepsis was the aetiology of the regenerative anaemia. Faecal PCR returned as only positive for coronavirus, Haemotropic mycoplasma PCR was repeated and was negative, Coombs test was also negative.

Abdominal ultrasonography was normal. Chest radiographs were normal. Biochemistry was performed twice in this period of time and no abnormalities were detected.

Abdominal ultrasonography was repeated: hypoechoic regions were evident that I suspected to be possibly mesenteric lymph nodes. A small amount of free fluid was evident around this region.

Due to the presence of jaundice, a FNA was taken of the liver and this region. The liver sample came back as 'lymphocytosis' and the abnormal hypoechoic region returned as having large lymphocytes within the sample.

At this stage, we were worried about lymphoma versus FIP. Dexter was too unwell for an exploratory laparotomy and euthanasia was elected.

A post mortem was performed immediately post euthanasia. The liver was engorged with an increased lobular pattern, the kidneys looked a bit abnormal to me with prominent division by surface vessels and slight yellow discolouration. Intestines appeared pale but otherwise seemed normal. Lymph nodes were not dramatically enlarged, no free fluid was noted. The lungs were brown in colour and heavy. This cat was not dyspnoeic at euthanasia, respiratory rate was around 20/min. No pleural effusion was present within the chest cavities.

The following were submitted for histopathological examination: liver, kidneys, spleen, mesenteric lymph node, duodenum, jejunum, colon, ribs (to assess bone marrow), lung and heart.

HISTOLOPATHOLOGICAL FINDINGS (as reported by Dr John Mackie BVSc PhD FACVSc DACVP, QML Vetnostics) and performed at no charge because of everyone's interest in this challenging case.

Liver: Marked expansion and effacement of portal areas by dense sheets of neoplastic lymphoid cells. These cells are medium sized to large with 2-5 mitotic figures per high power field. On immunohistochemical staining, these cells are positive for CD3 and negative for Pax5, which is consistent with T cell lymphoma.

Spleen: Multifocal infiltration by sheets of neoplastic lymphoid cells, similar to those described in the liver.

Neoplastic lymphoid cells are not evident in the other tissues examined.

DIAGNOSIS:

T cell lymphoma (liver and spleen).

COMMENTS:

Regarding the clinical history in this cat, immune mediated disease may occur in a subset of lymphoma cases which might explain the neutropaenia.

There is no evidence of FIP infection.

Our working hypothesis is that the cat had a regressive FeLV infection that caused provirus to effect a clone of neoplastic lymphoid cells which were affected by a 'second hit' to generate T-cell lymphoma in the marrow which eventually disseminated widely.

Further tests on the tumour are 'a work in progress' in the hands of Mark Westman and Jacqui Norris.

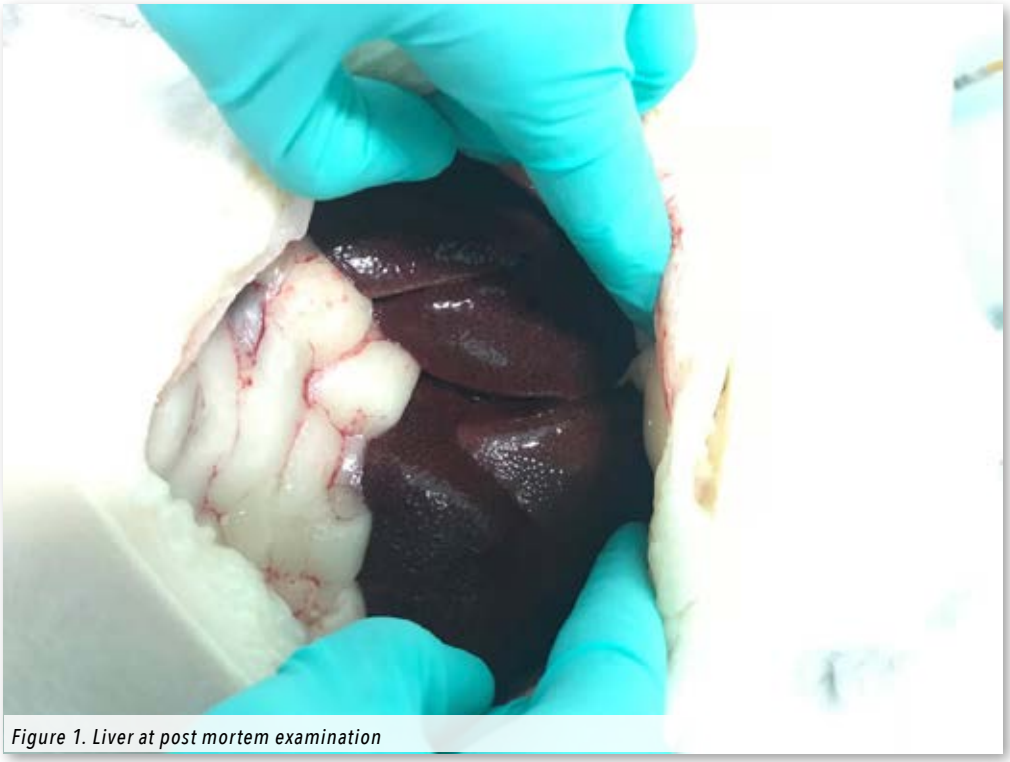

Figure 1. Liver at post mortem examination

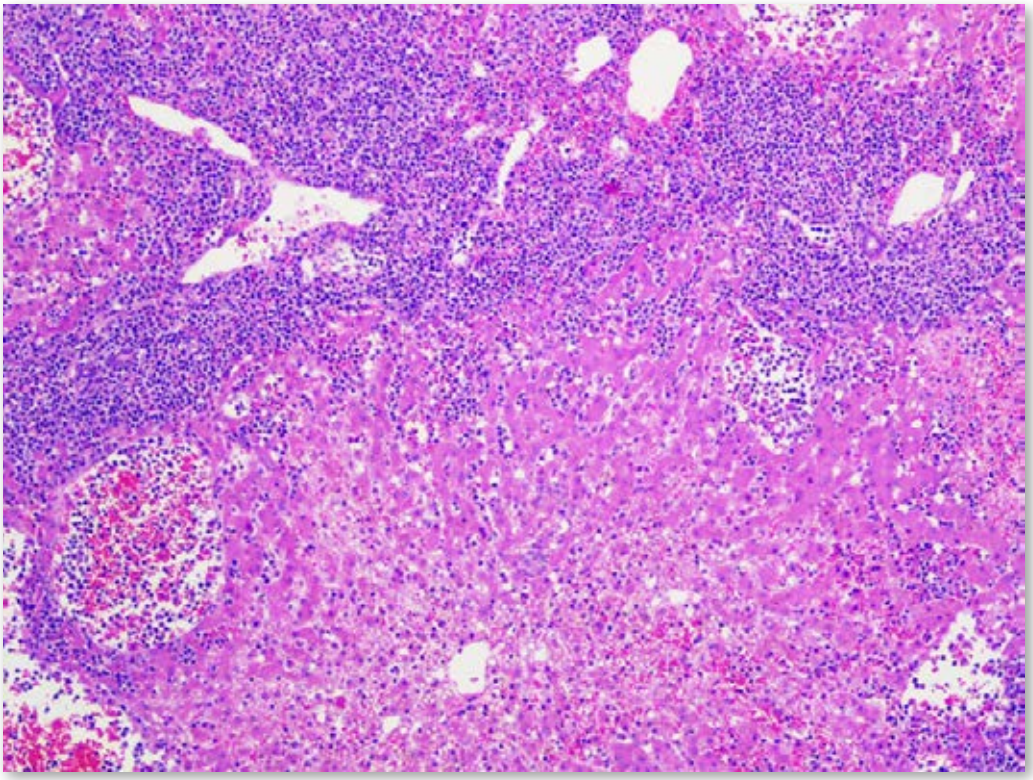

Figure 2. Liver: There is expansion and effacement of portal areas by sheets of neoplastic lymphoid cells, which also infiltrate sinusoids to a lesser extent. There is lipid vacuolation of centrilobular hepatocytes. Original magnification x10

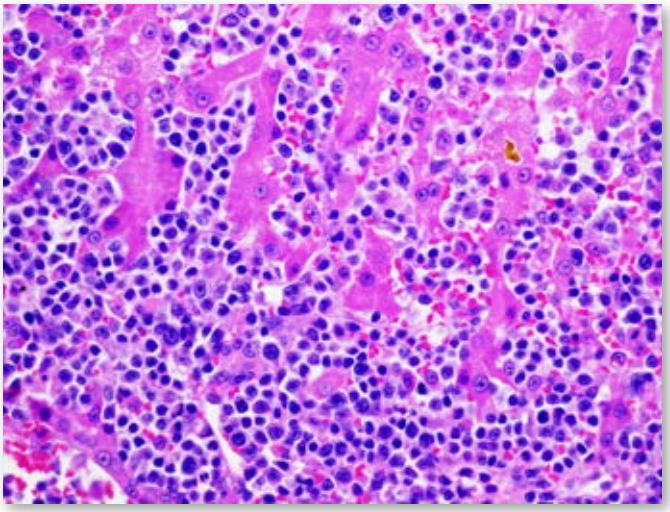

Figure 3. Liver: Neoplastic lymphoid cells are medium sized to large. There is patchy canalicular cholestasis. Original magnification x40

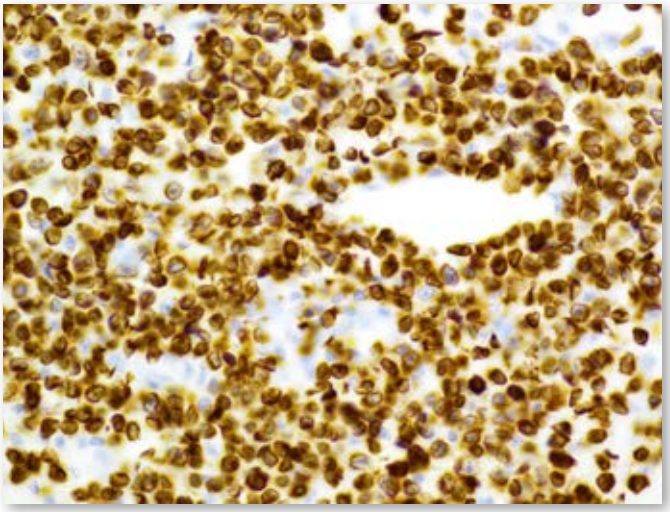

Figure 4. Liver: On immunohistochemical staining, neoplastic cells are positive for CD3, which is consistent with T cell lymphoma. Original magnification x40

Replies

REPLY TO C&T NO. 5699
(ISSUE 292 SEPTEMBER)

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Regarding 'What happens in my back yard at night?'

How about confining the cat to the house and or a cat enclosure. They are available, not terribly expensive and would prevent the problems you are having. Cats do not get bitten or hit by motor vehicles. Also talk to your neighbours and explain the benefits of confining their cats.

WINNER + SMALL

Raw chicken diet for faecal incontinence

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C&T No. 5713

Fergus is a 12 month old Male neuter domestic short hair cat. He was adopted from a local cat shelter with another male cat. Limited history of the cats is known except that they had been kept outside in an outdoor run. The shelter had vaccinated both cats and they had been desexed as adults 2 months prior to his first consultation.

Fergus's new owner brought him to see me due to what she described as faecal incontinence. According to his owner he would drop small faecal balls all over the house up to as many as four times per day (see figure 1). The faecal balls were formed and approximately 1cm in size. Fergus only knew about them when they would fall out. One time the owner even found one on the other cat!

Both cats had excellent appetites and were fed a combination of wet and dry commercially available grain free cat food. At the time of initial presentation Fergus weighed 4.03 kg. He had a body condition score of 4/9. Clinical examination was unremarkable. His anal area looked grossly normal and I chose at this stage not to perform a rectal exam.

Having had success in the past with other diarrhoea cases that I could not control with regular treatment options I decided to trial Fergus on a raw chicken diet. The challenge was not getting him to eat the chicken but convincing his vegetarian owner to consider it.

To remove some of the challenges for his owner I spoke with her butcher about mincing an entire chicken carcass (raw), including bones & skin & dividing it into 200 gram portions that she could defrost on a daily basis and feed him.

His owner phoned me 6 days later to report that his stools were completely formed and he had no incidence of faecal incontinence in the past 4 days. He was happily eating the chicken and she said he was behaving as normal.

Fergus came back for a revisit 2 weeks after his initial consultation. He was bright and very affectionate. He was eating raw chicken only and had gained 220g in weight. He had had one episode of a loose stool after accessing the other house hold cat's food. Since going back onto raw chicken his stools were formed and regular. He had no further incidences of faecal incontinence.

At this consultation I continued Fergus on his raw chicken diet (whole minced carcass) and got his owner to add in raw chicken drumsticks or wings for his dental health. By this stage his owner had also started the other household cat on raw chicken. To assist the owner with Fergus stealing dry food from Winter, her other cat, we allowed Fergus a tablespoon of Delicate Care Skin & stomach dry food per day.

Fergus continues to be faecally continent and is thriving on an almost 100% raw chicken diet.

Figure 1. Faecal ball – often defecated randomly around the house

eBook download:
C&T No. 5487 Moira van Dorsselaer 'Trials, Tribulations & Triumphs - setting up your own practice'

Sporotrichosis in a young cat

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C&T No. 5714

Dymphna was born and raised in the Netherlands. In 2003 she finished her veterinary degree in Utrecht, the Netherlands. After this she worked for 12 years in small animal practices, developing a keen interest in feline internal medicine and dentistry.

After travelling the world for many years, she decided in 2015 to move to a different country, closer to her other passions: diving and underwater photography. After applying for a job on Skype, she started work in Singapore. This has been challenging as diseases and owners are very different to the Netherlands, but she has learnt a lot and is still learning every day.

This describes a Sporotrichosis case in a young cat in Singapore.

Gypsy, a 1-year-old female domestic shorthair, came to us in December 2017 for a check-up after being found in a forested part of Singapore. She was slightly skinny, tested positive for fur mites (*Cheyletiella*), was FIV/FeLV negative and had a mild yeast otitis externa. No dermal masses were noted. She was treated with Revolution® (Zoetis), ear medication and dewormer.

Three weeks later the owner noted the development of subcutaneous masses on the left thoracic region and left lateral elbow which also had an ulcerated wound with purulent discharge and cellulitis. Her demeanor seemed subdued.

Basic biochemistry and CBC were unremarkable, and a toxoplasmosis screen was performed which came back negative. She received her vaccination and the wound was treated with cleaning and sterile honey.

Towards the end of January she presented for review of the masses. According to the owner, the cat was becoming more and more subdued and had developed softer stools. As the cat had been licking the left lateral elbow wound a lot, that wound had become bigger and had begun to granulate. The left thoracic mass had swollen subcutaneously but had not opened up. She was spayed at this time and given Convenia® (Zoetis) injection for the skin infection.

Gypsy had a very long and poor recovery after surgery, taking multiple days to start eating again.

Early February Gypsy returned with more dermal masses and non-healing wounds, she was more lethargic and not gaining any weight. There was a third mass in between the toes of her left front leg, the left elbow mass had another small satellite lesion next to it and the left thoracic mass had opened up and had purulent discharge. No nasal or facial masses were present.

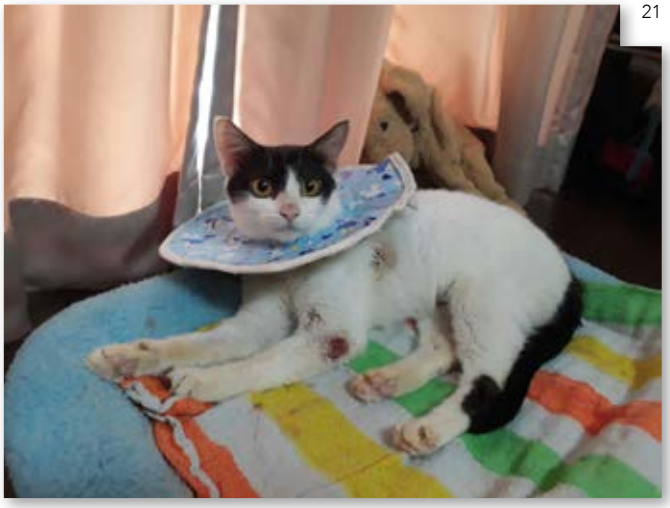
In-house cytology was performed on the purulent exudate from the thoracic wound, which revealed a neutrophilic inflammation with intracellular bacteria and big round vacuolar cells were seen.

Figure 1. Gypsy with the primary lesion on left elbow

As PCR for Cryptococcus would take several weeks to get results, we decided to send off for cytology, with the plan to do PCR and possible histopathology if cytology was non-diagnostic.

We started amoxy-clavulate for the secondary bacterial infection, together with wound cleaning and bandaging.

Figure 2. Primary thoracic and elbow lesions after shaving and cleaning



Figures 3 and 4. Showing development of further lesions

The pathology showed a cigar-shaped intracellular yeast, more consistent with histoplasmosis or sporotrichosis than cryptococcosis. To differentiate between them, we did a fungal test on the sample. The result came back: *Sporotrichosis schenckii.*

Following the ABCD guidelines we started itraconazole 10 mg/kg once daily *per os*, we dispensed the generic itraconazole available in our clinic. We advised the owner of the zoonotic and infectious nature of Sporotrichosis, and to keep Gypsy indoors and away from the other cats. We also warned the owner of the potential hepatotoxicity of itraconazole treatment and we therefore recommended checking ALT levels monthly.

Sporotrichosis schenckii is a dimorphic saprophytic fungus which can infect many mammalians. The fungus exists as a hyphal or mycelial form at environmental temperatures below 37°C and as a yeast form at body temperature. *S. schenckii* is found worldwide in soil, wood, living plants and decaying plant material. Infections are caused by traumatic inoculation of contaminated soil or organic material.

While on the generic itraconazole, more lesions appeared on the tail and other legs. The previous masses did seem to have less purulent discharge on itraconazole and amoxy-clavulate. On in-house cytology the intracellular yeast persisted, but the intracellular bacteria had gone.

In March Gypsy had started sneezing with blood. Famciclovir was dispensed in case of herpes rhinitis.

On review mid-March more masses had started to appear around the ears and face, these seemed more painful and had purulent bloody discharge. There was no improvement in the bloody sneezing. Gypsy also developed a limp on the right hind leg. Orthopedic evaluation showed a painful swelling around right stifle, no other abnormalities. No medication for limping was given at this time as the owner was concerned about the potential for hepatotoxicity due to NSAIDs.

No elevation in ALT activity was noted.

We decided on the advice of specialists to change the generic itraconazole to Sporanox® itraconazole capsules, where the owner was counting the beads for accurate dosing. Fluconazole was not available in Singapore and the Sporanox liquid was a lot more expensive.

With Sporanox the sneezing reduced and the nodules seemed to be drying up but still more nodules appeared on the face and toes. The old masses were dry but all the new masses had discharge. The famciclovir was stopped as the sneezing was more likely due to the sporotrichosis than herpes.

We considered sending the fungal culture off for susceptibility testing as we suspected an azole resistance, but this was not available in Singapore. It was available in Texas but was declined by owner due to cost.

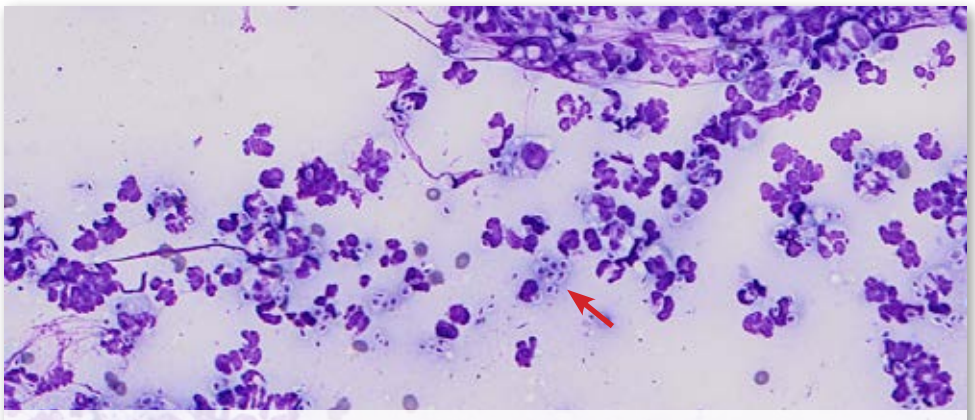


Figure 5. Cytology from thoracic wound showing intracellular and extracellular cigar shaped yeast form (arrow)

As the Sporanox® did not produce good enough results, we researched other possibilities. Amphotericin B given intralesionally was effective in 2 case reports, but the side effects are nephrotoxicity. Ketoconazole was available in Singapore and effective in case reports but has

Figure 6. Lesion digit left foreleg, not healing and still having discharge on the itraconazole

Figure 7. New lesion arising on left hind leg while on generic itraconazole

Figure 8. Lesion on face

COMMENT FROM ASSOCIATE PROFESSOR MARK KROCKENBERGER

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Sporotrichosis is an interesting disease, with a wide distribution globally. It is caused by the Sporothrix species complex, including S. *schenckii* seen in this case in Malaysia but also sporadically in Australia. The excellent presentation of this case demonstrates the classic presentation and cytopathology of the clinical disease.

It is an important differential to keep in mind, particularly in view of the zoonotic potential of this group of organisms. The cat has been shown to be a particularly excellent source of infection for people, with very high burdens of organism shed from ulcerated skin lesions. Another interesting feature of this case is that nasal cavity disease is not infrequently noted in cases caused by another member of this species complex (*S. brasiliensis*) seen in South America. Epidemiology of feline sporotrichosis varies quite dramatically globally, with sporadic disease caused by *S. schenckii* seen in South East Asia, Australia and North America, and epidemic disease caused by *S. brasiliensis* seen in South East Brazil.

The disease should be on the radar of clinicians on the east coast of Australia, as we have had a few cases surface quite recently.

a higher risk of hepatotoxicity. Terbinafine was reported to be effective in many cases - the major side effect is nausea.

After discussing with owner, we decided to add on terbinafine 30 mg per cat once daily *per os* and topical terbinafine cream while monitoring appetite.

In April the masses were getting smaller and all drying up. Gypsy still had a great appetite, ALT was still normal.

By the end of April, all the facial, ear and toe lesions had disappeared. There was no more sneezing or limping. The 2 original masses still had slight superficial skin changes, but no more open wounds and the other lesions had resolved. The fur was getting better overall, and she started to gain weight and became bright and happy again.

By the end of May there were no more lesions to be found and no more systemic symptoms. As per ABCD guidelines we advised continuing medication for 1 month after clinical resolution of symptoms.

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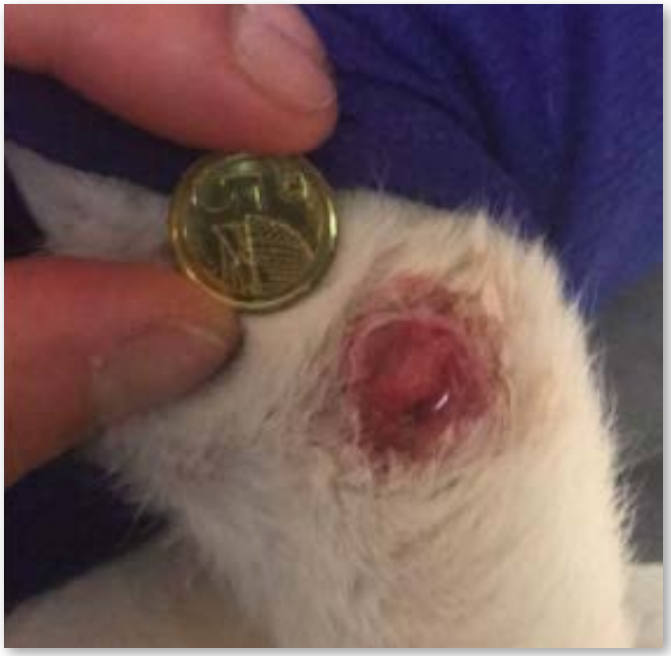

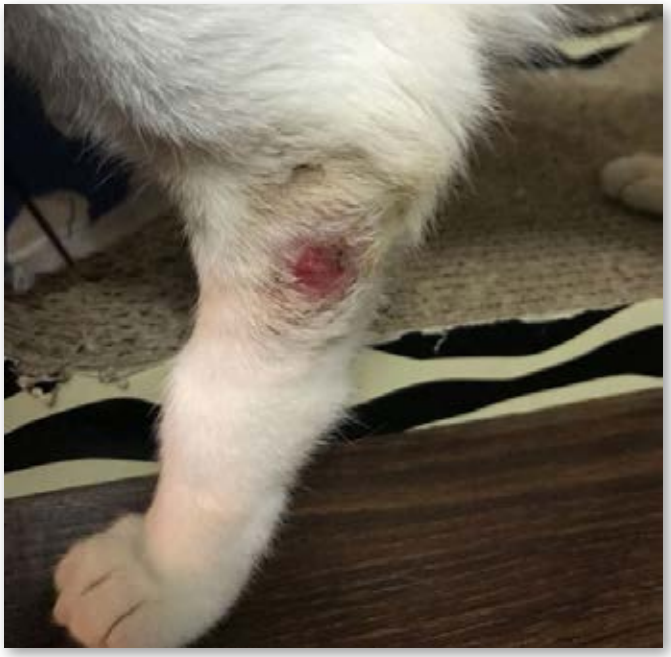

Figure 13. Gypsy returned to good health

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Perspective No. 134 Antifungal therapy in companion animals – A practical approach, Richard Malik & Mark Krockenberger (Issue 288, Sept 2017)

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(By Rob Webster, Proc No. 411 Emergency Conference)

Radio-Ulnar ischaemic necrosis in a 5yo Bichon-Frise X

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C&T No. 5715

Bella a 5yo FN Bichon-Frise cross presented on the 3.4.18 for acute right forelimb lameness grade 3 out of 5. Bella lives on a property and had previously presented for grass seed abscesses within the last 12 months. Physical exam revealed marked soft tissue swelling on the caudolateral aspect of the mid-proximal antebrachium with a harder swelling underneath that seemed congruous to the bone. No wounds were evident but there was significant pain on palpation of this area. There was no response to full range of motion movement in all joints of the forelimb, and no other signs of injury or trauma were found. Temperature was 39.1°C. Permission was given for sedation and radiographs.

Bella was sedated with a combination of 0.01mg/kg medetomidine/0.1mg/kg midazolam/0.1mg/kg butorphanol IM and lateral and craniocaudal views of the right thoracic limb were taken and also of the left forelimb (LF) for comparison. The Right Foreleg radiographs showed a curious and rather neat lytic 'hole' (Figure 2) in the proximal diaphysis of BOTH the radius and ulna, seeming to cross the bone neatly. There was some mild sclerosis on the border of the lytic area. The bone didn't appear particularly reactive around the lesion in that it wasn't fluffy, or 'moth-eaten' so I was a bit stumped as to what this was, and if it was indeed the cause of the lameness. Bella was sent home that day with meloxicam 0.1mg/kg for 5 days and clindamycin 10mg/kg in case of osteomyelitis whilst I delved further into the mystery.

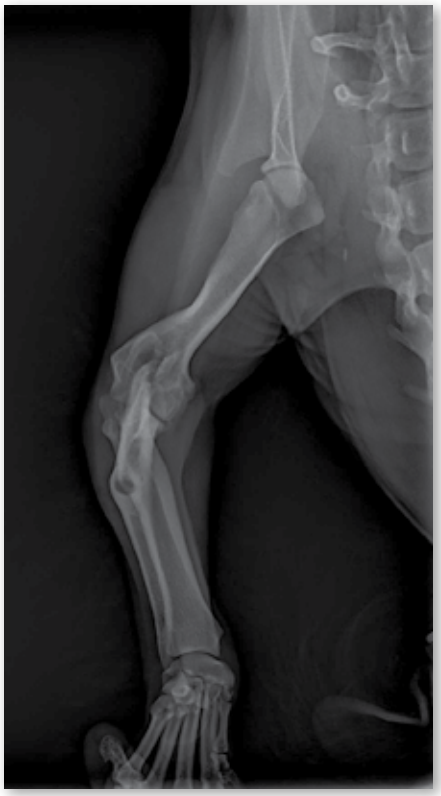
Radiographs were forwarded to a rather learned colleague who I am forever indebted to for his continuous help throughout my career (you know who you are!), who suggested a biopsy and repeat radiographs. Bella's lameness resolved completely within 5 days, and the soft tissue swelling disappeared. Temperature was also normal. However 2 weeks later on the 16.4.18 the bony swelling was more obvious on palpation, though not painful, and repeat radiographs indicated the 'hole' was growing! It was now apparent that a pathological fracture was a definite risk given the little remaining bone in the unaffected cortices, especially of the ulna.

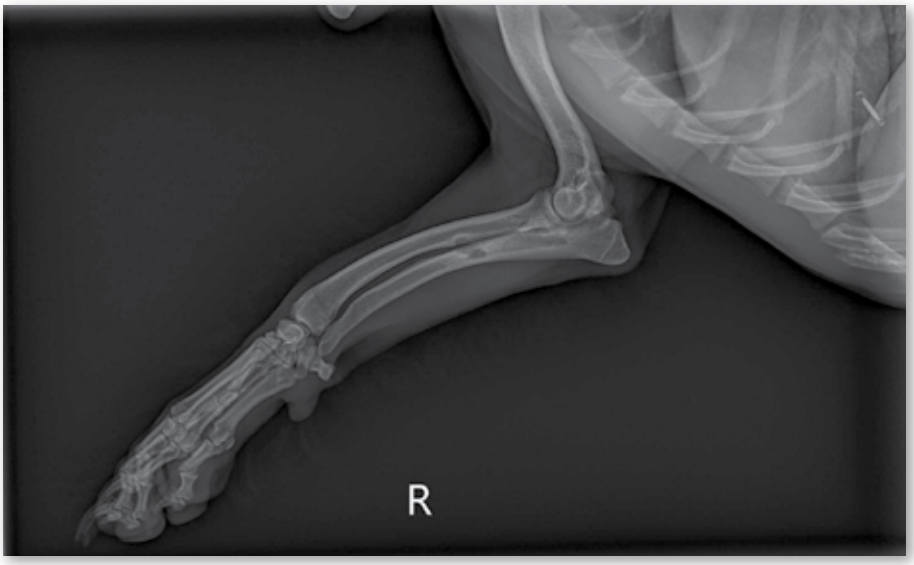
Given we didn't know the cause, the owner consented to biopsy. And on the 19.4.18 Bella was admitted for bone biopsy. Pre-anaesthetic bloodwork was normal, and Bella was sedated with a combination of ACP/Methone SQ and induced with Alfaxan IV. Maintenance was isoflurane and oxygen and fluid support was provided from sedation to post-operatively. Cephalothin IV at 20mg/kg was given at induction. A lateral approach was taken and 3 small biopsies obtained using a 20g spinal needle as we thought the Jamshidi had a greater chance of causing an iatrogenic fracture. The incision was sutured with 3/0 MonoQ and Bella's recovery was uneventful after a short course of meloxicam.

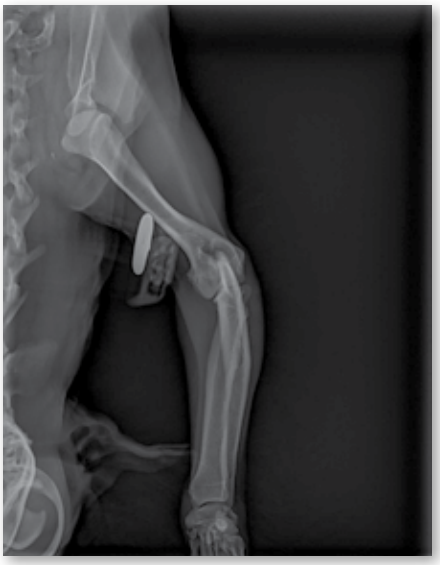
Biopsies were sent to Vetnostics in Sydney where unfortunately the results were inconclusive. However, Dr Rolfe Howlett, senior pathologist at Vetnostics had rounds with the radiologists at Veterinary Imaging Associates and kindly requested the radiographs which we sent down for him and his colleagues to assess. He was kind enough to send back a suggestion for RUIN (radioulnar ischaemic necrosis) and requested 8 week follow-up radiographs. In the meantime Bella continued to be lameness-free and merrily recovered from surgery, only having to have her suture line partially glued back together after licking her suture line a tiny bit open on day 5 post-op.

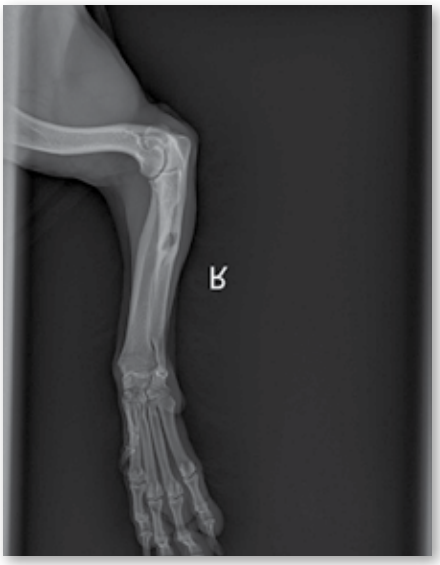
RUIN seems to be poorly reported in the literature. The only paper I can find is from the *Veterinary Radiology and Ultrasound* journal 2016 59:1:E7-E11. This paper describes Bella's case almost to a tee (bar the pathological fracture part) and leaves me with no doubt as to what Bella's radiographic diagnosis is. Ischaemic necrosis is described in carpal bones, and associated with medullary tumours or genetic disease such as Legg-Calves-Perthes disease, but other than the aforementioned article I can find none within the long bones of the forelimb. RUIN seems to be secondary to some kind of interruption to the blood supply of the radius and ulna, both of which have nutrient foramens in the proximal third of the diaphysis (though on opposing sides). Interruption of the supply leads to ischaemic necrosis of both bones. However, we are still left with the inciting cause?? On original presentation Bella had significant soft tissue swelling over the proximal and lateral antebrachium, and a slight pyrexia. One could think that this was the cause of the RUIN, but given we never found the cause of the soft tissue pathology and treated empirically (with excellent response) then we are still left wondering.

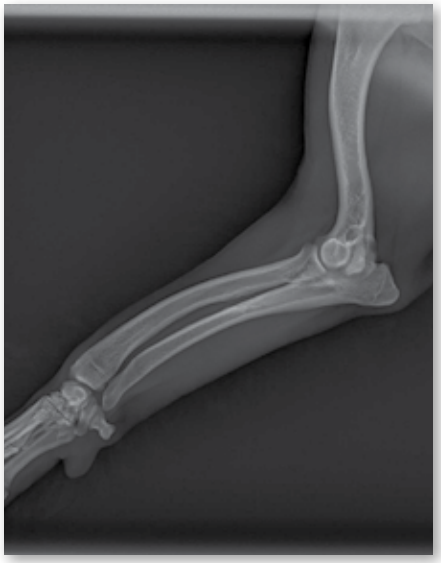
Even though there appears to be a dearth of information on RUIN, I don't doubt that it has been presenting in various dogs for a long time, but maybe only now is becoming recognised as a definite syndrome? Either way, it turned out to be a very interesting case. Here is the link to the article: https://onlinelibrary.wiley.com/doi/pdf/10.1111/vru.12448

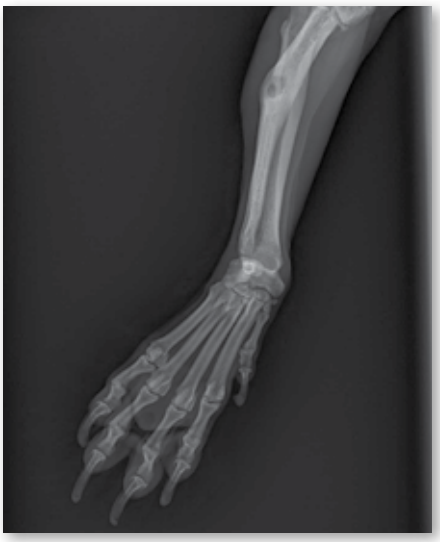


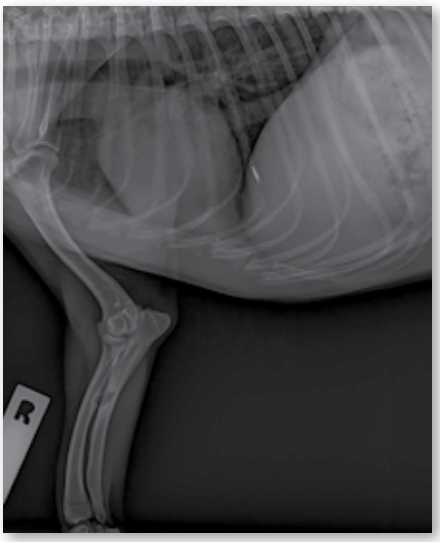












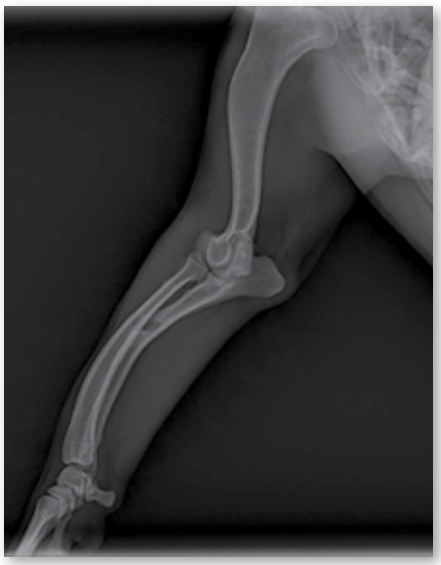


Figure 1. Right Foreleg AP view (3.4.18)

Figure 2. Right Foreleg lateral view (3.4.18)

Figure 3. Left Foreleg lateral view (3.4.18)

Figure 4. Left Foreleg AP view (3.4.18)

Figure 5. Right Foreleg AP view (16.4.18)

Figure 6. Right Foreleg lateral view (16.4.18)

Figure 7. Right Foreleg AP view (30.5.18)

Figure 8. Right foreleg lateral view (30.5.18)

COMMENT ON RUIN CASE REPORT COURTESY OF:

Sarah Davies BVSc MS DACVR
Veterinary Imaging Associates, Sydney
www.online-vets.com

Right forelimb - Initial radiographs show well demarcated small regions of lucency in the caudal cortex of the radius and adjacent ulna in the region of interosseous ligament attachment and the sites of the nutrient foramina. A zone of increased bone opacity extends proximally and distally from the site of lucency in the ulna. A thin line of increased bone opacity surrounds the area of lucency in the radius. There is concurrent forelimb angular deformity with lateral angulation of the forelimb distal to the carpus. Angular deformity is likely breed/conformation related. Osteophytes are present on periarticular margins of the right elbow, indicating degenerative joint disease.

Radiographic findings for the right forelimb appear similar over the follow-up radiographic studies.

Conclusions/comments:

Radiographic findings in this case are consistent with those reported with radioulnar ischaemic necrosis (RUIN), as reported recently in the provided citation (Imaging Diagnosis-Radiography and Computed Tomography of Radioulnar Ischemic Necrosis in a Jack Russell Terrier. Schmid et al., 2016 VRUS). The exact cause of ischemic necrosis remains unknown but it is thought to be a primary vascular lesion. There could be an association with insertional desmopathy of the interosseous ligament. The lesion is thought to be self-limiting and we would typically not recommend biopsy or surgical intervention. As the lesion repairs extensive new bone formation can be a prominent radiographic feature.

Sarah tutors the Musculoskeletal Imaging DE course www.cve.edu.au/de/musculoskeletal-imaging-1

GENERAL

Tick prevention in 2018

Rob Webster BVSc FANZCVS

Animal Emergency Service

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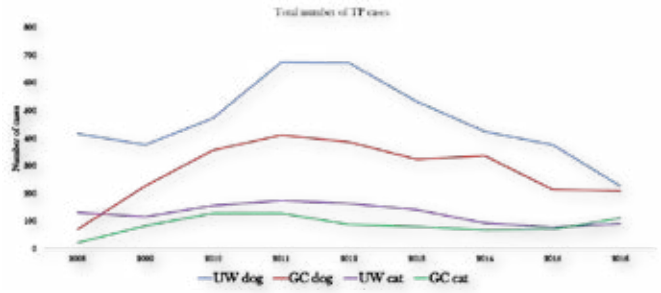
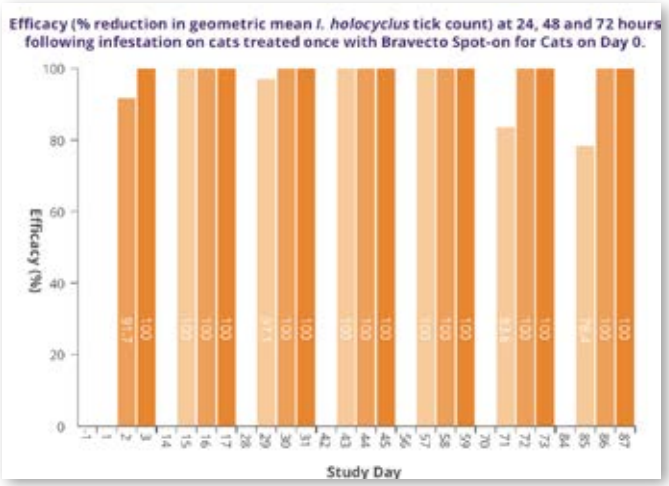
C&T No. 5717

The introduction of canine isoxazoline products in 2014/2015 lead to decreases in the number of dogs treated for tick paralysis in all of the Animal Emergency Service East-Coast practices in 2015, 2016, and 2017 (2017 data not shown on graph). These are really promising results. The significant decrease in case numbers is also supported by anecdotal reports from veterinarians, declining anti-serum sales, and reductions in insurance claims for tick paralysis patients.



Feline cases of tick paralysis have remained constant with a slight increase in line with the increased overall patient numbers.



I hope that the introduction of Fluralaner top-spot for cats will lead to significant decreases in feline cases. The efficacy of the product and duration of action are impressive.

Increased adoption of Isoxazoline products by pet owners may be assisted by enthusiastic veterinary endorsement, but this must be carefully considered. Isoxazolines have received negative anecdotal feedback from consumers, and have also been subject of an alert released recently by the United States FDA cautioning about the risk of adverse neurological events after administration of this class of drugs. The warning states that while the FDA is satisfied of the safety of these drugs in the majority of animals, veterinarians should make individual treatment decisions after reviewing patient medical history, and in consultation with their owner.

I believe almost all dogs & cats in tick areas in Australia are suitable patients for isoxazoline administration. The risk of death or suffering of a patient by not administering an effective product is higher when considering the impact of paralysis ticks than any other Australian parasite. Isoxazolines present the best chance of reducing the impact of this parasite on our pets and patients, and we should consider them the first line treatment for susceptible patients.

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Read Tick C&Ts & Perspectives by Rob and other contributors here.

Canine Leproid Granuloma Syndrome and histiocytoma in an aged Bulldog

Wendy Mashado

Wendy's Mobile Vet Service

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C&T No. 5716

Ruby lives in a rural area which is serviced by vets visiting just once a fortnight. She is a 7-year-old female neutered Bulldog.

Her owners presented her on 2/5/17 after she developed two masses on the pinna of her left ear. The larger of the masses at the base of her ear flap had been present for a little more than a week, the smaller mass had only been present for a few days. Ruby was clinically normal in all other ways, the masses were not red, hot or painful. The skin was normally haired. The larger one was about 3cm, ovoid shape, 1.5cm high, the smaller mass was less than 1cm. They were well demarcated and quite firm.

These lesions didn't follow any usual pattern that I could recognise. I believe the owner's history to be reliable and so a FNA was done immediately. I also sent off an aspirate of the mass in transport media looking for bacteria. She was left with amoxicillin/clavulanic acid 'just in case' because it would be two weeks before I was back again.

A slide was stained with Diff Quik and looked at 'in-house'. It looked like a granulomatous reaction but still didn't make much sense to me. (I completely missed the negative staining bacilli. This was very obvious when I had a second look.) The already stained sections were sent to Vetnostics. The report received as below for the cytology along with the culture and susceptibility results of the aspirate.

Microscopic examination

Across the smear examined findings revealed moderate to marked red blood cell contamination accompanied by a mild to moderate increase in the number of mixed inflammatory cells present comprising numerous neutrophils (some of which appear degenerate) and macrophages (some of which contain increased numbers of negatively-staining bacilli within their cytoplasm). No other obvious cellular detail was evident other than occasional small mature lymphocytes and scant eosinophils.

Diagnosis

Mild to moderate active chronic inflammation.

Comment

Cytological findings here are consistent with an underlying infectious process most likely mycobacterial infection (canine leproid granuloma syndrome).

Microbiology report - veterinary culture

Few leucocytes

No organisms seen

Culture: Scant growth of Org 1: *Staphylococcus pseudintermedius*

Susceptibility:

Ampi/Amoxicillin R Marbofloxacin S

Cephalexin S Enrofloxacin S

Clindamycin S Sulpha/Trimeth S

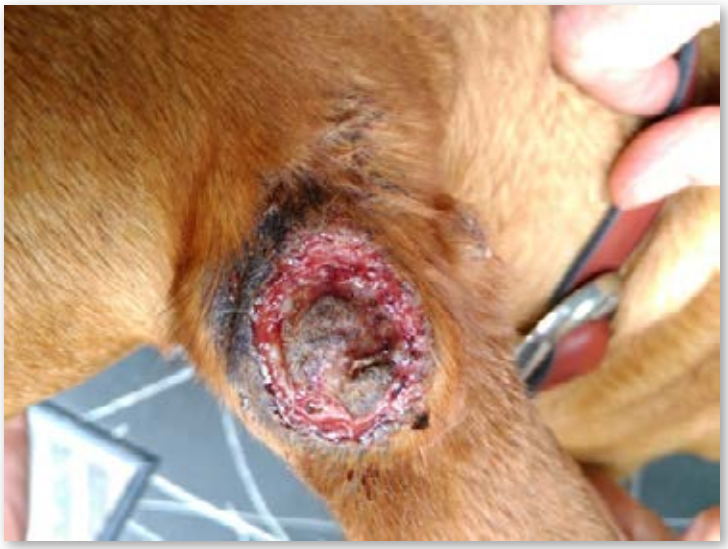
Tetracycline S Clavulan/Amox S

Cefovecin S

No anaerobic bacterial pathogens isolated.

I started her on Cephalexin 600mg bid after I had repeated the FNA to get sufficient samples for PCR testing. This confirmed Canine Leproid.

Figures 1 and 2. Showing adjacent lumps



Granuloma Syndrome, report below:

Mycobacterial PCR result:

CLGS = Canine leproid granuloma syndrome.

Mycobacteria DNA DETECTED by polymerase chain reaction.

Mycobacterium sp. *CLGS identified by sequence analysis of the PCR product.*

This result shows the presence of Mycobacterium sp. *CLGS DNA but does not necessarily indicate presence of viable organisms.*

I examined Ruby again on the 30/5/17. The lesions were now 3.8 cm and 1cm in size and the skin over both lesions was eroded.

I then contacted Dr Malik for his advice. I have included my email as it illustrates my thought process in my ignorance of this condition.

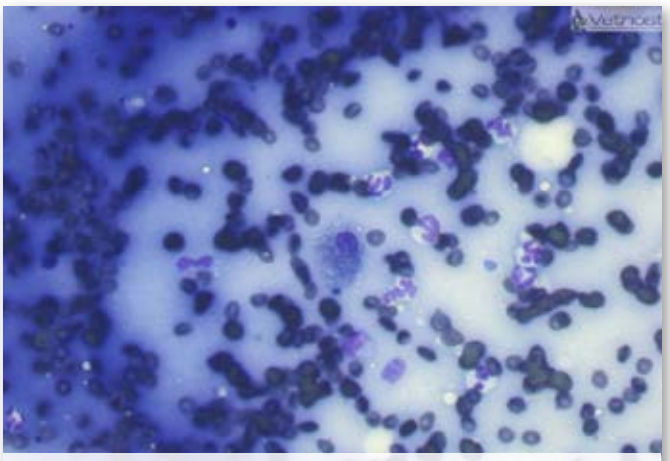


Figure 3. Cytology from F.N.A. showing presence of scattered large mononuclear cells with abundant blue cytoplasm containing negatively stained bacillary elements

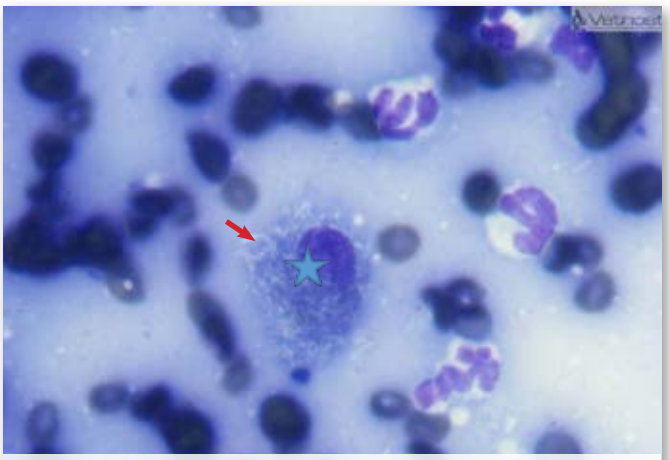


Figure 4. The star labels a macrophage and the arrow point out a negatively staining filamentous intracellular components consistent with the appearance of mycobacteria

eBook download:
C&T No. 4485. Malik R, Canine Leproid Granuloma Syndrome (canine leprosy), Issue 232, Sept 2003.

Dear Dr Malik,

I would love to have your advice on treatment options for Ruby. Ruby is a 6-year-old female Bulldog owned by clients from Mallacoota, coastal far eastern Victoria, in Bairnsdale ulcer area. I saw Ruby 4 weeks ago on one of my fortnightly visits to Mallacoota. She had a 1cm raised ovoid mass about 3cm long on the base of her ear flap. It had been present about 1-2 wks. A smaller mass was developing adjacent to this <1cm, raised, spherical. The skin was intact. The dog was fine, the lesions weren't irritating her in any way. I did an FNA that day and sent it off. George Reppas suggested Canine Leproid Granuloma and so a PCR was performed which confirmed this. Since then the entire surface of the larger lesion has ulcerated, sensitive to cephalexin so she has just been started on this.

My questions are:

- Do we give this lesion time to regress?
- OR do we start heavy duty medication?
- OR is surgery an option? I think a clever surgeon would be able to do a transposition flap. What sort of margins would be required if this is an option?

I would really appreciate any insight you can give me.

Dr Malik's replied as below, very promptly!

Good to think of DDx *M. ulcerans* in this location. I have seen far worse. They ulcerate when they are about to get better. The combination of rifampicin and clarithromycin is no longer expensive.

Rifampicin 10-12 mg/kg ONCE a day.

Clarithromycin 250 mg twice a day (or Pradofloxacin at the label dose).

Stop the cephalexin, stop medication if dog goes off food. Measure ALP and Alt every 7-10 days if possible. You can get both from a local chemist. Use something topically e.g. sulphasalazine, eye ointment with gentamicin.

Most get better. You don't need surgery. There is usually no scar.

As a result of this I started Ruby on rifampicin 250mg sid, clarithromycin 250 mg bid, (not available at our little local pharmacy).

Flamazine was used on the lesions which were now raggedly ulcerated. All other treatment was stopped.

11/07/2017: Ruby was eating well and behaving normally. We were not able to do a blood test until she had been on treatment for almost a month. By this time the larger of the lesions was much smaller, (about 1 cm across) and flatter, with a smaller ulcerated centre. The smaller lesion had not changed much.

All values, FBC and VBA, were within normal limits.

24/08/17: Ruby was still eating well and behaving normally. The larger of the lesions was now quite flat, the area of ulceration 2 mm, the lesion towards the tip of ear was looking dry, some surfaces had mild fissures, still 5 mm, raised, round.

Blood was taken. This time Ruby was showing a leucopenia and neutropenia. All biochemistry values were normal:

Hb 157 g/L (115-180) WBC 5.1* x10^9/L (6.0-14.0)
RCC 6.5 x10^12/L (5.0-8.0) Neut 3.1* x10^9/L (4.1-9.4)
Hct 0.45 (0.37-0.55) Lymp 1.6 x10^9/L (0.9-3.6)
MCV 70 FL (60-74) Mono 0.2 x10^9/L (0.2-1.0)
MCH 24 pg. (20-25) Eos 0.3 x10^9/L (0.1-1.2)
MCHC 349 g/L (310-360) Baso 0.0 x10^9/L (< 0.2)

Red cells: Normal.
White cells: Leukopenia +, Neutropenia +.
Platelets: Platelet clumps ++,
Platelets Clumped: Numbers appear adequate on film.

Treatment was continued for another month but a further blood test was scheduled for 2 weeks. This time there was a mild increase in ALP 172 U/L (1-120).

Ruby was seen again in November. By this time she had been off all medication for about a month. The hair on her pinna was thin over the area of the largest lesion and the subcutaneous tissue felt thickened.

4/01/18: Ruby presented this time with a lesion on the ventral surface of her tail about 2/3rds of the way down. The owner only noticed this when she was seen to be licking at it. This was a round, 1.5cm, raised, with a crater-like, ulcerated centre with rounded edges. The tissue was soft. It was sore. Ruby did not like anyone near her tail.

An FNA was taken from this lesion, producing a lot of bleeding.

I couldn't see any areas of negative staining in macrophages or neutrophils but it's funny how the confidence wanes after missing some pretty obvious negative staining. There were mixed inflammatory cells, lymphocytes, plasma cells, large round cells with pale cytoplasm, looking like histiocytes. I could not see infectious agents. This lesion was sore (not usual for a histiocytoma). The decision was made, for a number of reasons, to amputate the end of her tail.

Histopathology came back as a histiocytoma.

There is a pattern (illness script) to be recognised with CLGS. They occur most commonly in short haired dogs, often in Boxer/Bulldog breeds. They are frequently found on ears (especially on the dorsal ear fold), around the face and less commonly on extremities. They grow rapidly and they are not painful.

SMALL

Second case of nasal myiasis in an urban-living domestic cat

Aine Seavers

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C&T No. 5718

In March of 2008, a 6-year-old semi-feral cat was presented with a several month history of episodes of odd and unexplained behaviour, most often triggered when the cat was walking along the darker areas of his house especially the lower floor hallways. The cat would suddenly either stop to hiss and spit at nothing in particular and/or stop walking along normally to suddenly twitch and skin roll, then sit and furiously rattlesnake shake the end of his tail. At that point the cat would commence laboured breathing: first open mouth panting then closed mouth breathing with nasal snorting that was akin to the sound of a human trying to clear a blocked nostril.

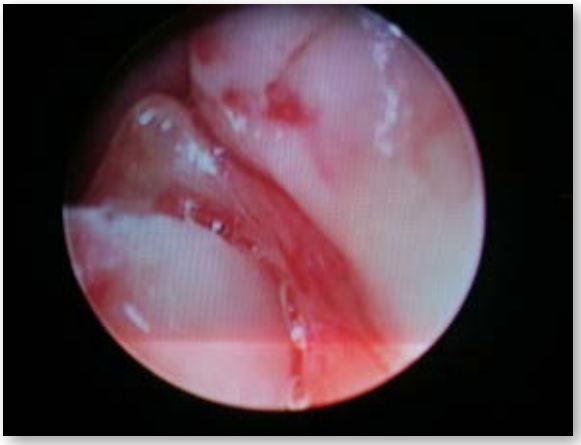

Figure 1. Arrow points to nasal bot

Figure 2. Rhinoscopic image (Image courtesy of Sarah Webb)

The cat was clinically normally when presented to the clinic but was unmanageable in the consult room. We admitted the cat out the back of the clinic for further observation. The kennel lights were turned down and the cat left to adjust to his surroundings. After a short time, the cat commenced hissing and twitching and I observed several dry creamy oval shaped objects suddenly appear at the nares, slowly working their way forward and outwards. As these objects emerged from the nostrils the cat settled down. The objects were dry, not mucoid or dripping exudate and had the appearance of a flattened sesame seed. There were no signs on the nares or mouth that the cat had attempted to lick or groom the nasal area nor any sign of a chronic nasal discharge. The object on the right moved right out of the nares protruding straight out - not stuck to the ventrum of the nares; the left 'object' came halfway out then retreated into the nares again. The objects were photophobic and withdrew rapidly with camera flash. Differential diagnosis was a migratory aberrant nasal parasite of unknown origin!

The cat was unapproachable for injections and unpillable at home so we chose Advocate®, given its broad range of parasitical activity against many of the life-stages of endoparasites. The owner applied Advocate® Day 1, Profender® Day 7, Advocate® Day 10, Provender® Day 21. (At recheck one month later the cat was normal and remained so until it died many years later.)

I contacted Richard Malik on Day 7 who promptly raised the possibility of Bots and alerted me to Sarah Webb's case (Webb & Grillo, *AVJ* Nov 2010). Both Sarah and Richard were extremely helpful in providing additional information and direction in this case.

Back at the time this case first presented-2008, Bayer Australia had no information on the use of Moxidectin® for Bots but an Irish Vet colleague gave me data on moxidectin use overseas for the infestation in sheep. Ivermectin, Abamectin, Moxidectin and Closantel are registered for use against nasal bot. Ivermectin is not successful in some cases. Capstar could in theory work and of-course in 2018, we have Bravecto either as a chew or a topical version. Because of the migratory aspect/ suspicion, I would tend to still chose Advocate and have Bravecto as a Plan B.

The cat lived in an urban environment about 200km from the any sheep. However, at least 2 of the families living on the street had weekend hobby farms in sheep country and would return with fleeces and carcasses. The cat frequented all of these gardens on a daily basis. For the remainder of his life he was treated monthly with Advocate® as a precaution. There had been zero improvement in his feral temperament.

Unlike the Canberra case there was minimal inflammation noticed in this chronic presentation, but the most striking feature of this cat's condition was the forced expiration; something this cat did share with Sarah Webb's case. Practitioners both rural and urban need to be aware of

this condition and add it to the list of differentials for the sneezing or snorting cat.

Back in 2008, Feline Hyperaesthesia Syndrome was not on my radar but in the ensuing years, when shown videos of this affliction on other cats, I often thought of this Nasal Bot cat and the potential for wandering, migrating parasites to trigger sudden unexplained bursts of manic twitching and neuroitch-pain etc as seen in Idiopathic Feline Hyperaesthesia Sydrome.

Some Information on the Nasal Bot itself.

- The adult is a hairy, yellowish, bee-like fly about the size of a common horse fly active during summer and early fall.
- Larva: 20 to 30 mm long/larvae in the nostrils of sheep move up the nasal passages to the nasal/ frontal sinuses for 8 to 10 months.
- Development of the 1st instar larva may be delayed for 1 to 9 months to assist over the wintering cycle.
- Larvae pupate in the soil for 1 to 2 months depending upon temperature. Adults may live as long as 28 days.

Sneezing and a mucopurulent nasal discharge can be seen as a marker for the presence of the parasite. Humans report a stinging painful reaction. Rarely, secondary bacterial spread from mucosa to meninges may occur. Eye infections can occur in humans.

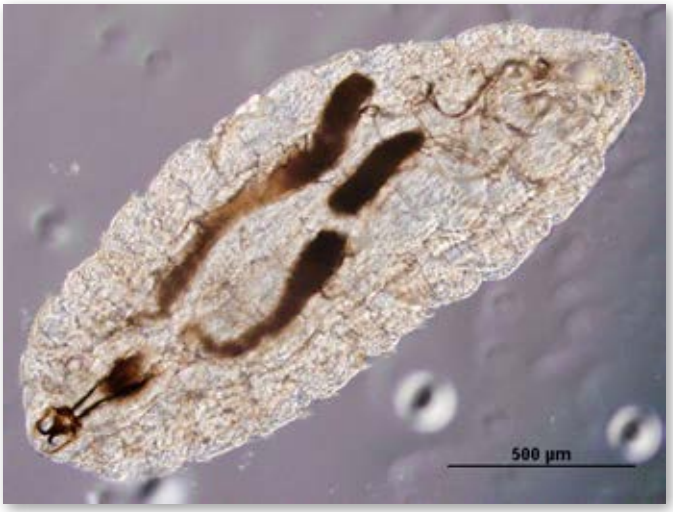

Figure 3. Larva of Oestrus ovis *recovered following saline irrigation of the nasal cavity of a cat*

Image courtesy of Sarah Webb

'Pre-pill cortisols' for trilostane monitoring

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Recommendations for trilostane monitoring have recently included the possibility of pre-pill cortisol measurement as a replacement for a routine ACTH stimulation test performed at the estimated peak effect of trilostane.

Use of pre-pill cortisol monitoring offers substantial cost benefits, potentially making trilostane treatment more affordable and attractive to clients. Whilst this is an enticing proposition, we need to carefully examine the science behind this recommendation.

Firstly, pre-pill cortisol monitoring entails assessing drug efficacy at a time when the drug's effects are likely to be minimal or negligent and would thus seem to be of questionable logic.

Secondly, it involves measurement of a hormone, well documented for its fluctuations in healthy and diseased dogs and known to range from 1-58 nmol/L (median 21.5 nmol/L) in healthy well conditioned calm dogs (Foster 1998; Foster 2011).

Thirdly, one cannot directly compare cortisol measurements between laboratories and between different cortisol assays (Graham 2017; Macfarlane et al 2016) so using fixed 'cortisol' values worldwide is not scientifically valid.

Lastly, the source of the recommendation is one paper (MacFarlane et al 2016) which was funded by Dechra Veterinary Products (UK), the manufacturer of trilostane.

The 'Pre Pill Cortisol' Study

The study was not without limitations including:

1. that dogs with adrenal and pituitary dependent hyperadrenocorticism receiving once or twice daily trilostane were all included as one group
2. that the unvalidated survey used was developed from an 'ad hoc survey of practising veterinarians'

using an unvalidated weighting of the questions by the authors

3. the authors choice of optimum monitoring test efficacy was then based on a Receiver Operator Characteristic (ROC) curve maximised for specificity of optimal cut-offs to reduce the likelihood of unnecessary dose increases, potentially favouring less rigid control of hyperadrenocorticism and less cost for owners

4. the study used ACTH stimulation testing performed 3 hours after trilostane dosing, based on a pilot study by Griebsch *et al* (2014). This study assessed basal cortisol measurements in 9 dogs (not post ACTH stimulation cortisol measurements). Data was not normally distributed and the graphical representation of geometric data (mean and dispersion factor) demonstrated considerable overlap at most time points. Thus 3h post-trilostane ACTH stimulation testing itself is questionable, especially given the peak effect variability demonstrated so neatly in another study (Bonadio *et al* 2014). The authors failed to acknowledge the possibility that the reason why measurement of post-stimulation cortisol 3h after trilostane dosing was not useful, *was that it may not have been the optimal time for testing* for trilostane efficacy.

5. dogs with excellent control of hyperadrenocorticism in the study actually had a median ACTH -stimulated cortisol of approximately 60-70 nmol/L (extrapolated from the box and whisker plot of Figure 1 in the paper) not 130 nmol/L, the figure used as a cutoff in the ROC curve. Based on thousands of observations using the Centaur Advia assay at Vetnostics (not directly comparable), a post-stimulation cortisol of 60-70 (in an ACTH stimulation test performed 4-6h after trilostane) would be associated with good control and a post-stimulation result of 130 nmol/L would be associated with poor control of hyperadrenocorticism.

The upshot, with these limitations, was that pre-pill cortisol measurement performed better than an ACTH-stimulated cortisol of 130 nmol/L in an ACTH stimulation test performed 3 hours after trilostane.

Whilst the study was interesting, some of the limitations (including difficulty of extrapolation of these results to other laboratories and other assays) were actually acknowledged by the authors and it should have been regarded essentially as a pilot study. Instead, pre-pill cortisol monitoring was almost immediately endorsed by the European Society of Veterinary Endocrinology in a Consensus Statement. The survey questionnaire for this Consensus Statement[1] required assessment of seven separate statements regarding hyperadrenocorticism and cortisol measurement, a number of which would be unarguable, but one at least was quite controversial (pre pill cortisol monitoring). Survey participants then only had 2 options: 'endorse' or 'absolutely cannot endorse' the Consensus Statement in its entirety. It is possible (perhaps likely) that participants who agreed with 5/7 or 6/7 statements may not have chosen 'absolutely cannot endorse' based on objections to only one or two statements.

A study presented as an abstract after the ESVE Consensus Statement (Sieber-Ruckstuhl et al 2017), compared two pre-pill cortisol measurements in trilostane-treated hyperadrenococorticoid dogs and found 30% disagreement in cortisol measurements taken one hour apart. The presenter (Sieber-Ruckstuhl) stated that both cortisol measurements were susceptible to stress, and that stress was difficult to define or assess in individual dogs. Despite this, the authors of this study appeared to also support pre-pill monitoring. Their study also received funding from Dechra Veterinary Products (UK).

After these studies, pre-pill cortisol monitoring was then incorporated into the monitoring recommendations of Dechra Veterinary Products (UK)[2] using information from both studies. Interestingly, whilst the recommendations do incorporate the pre-pill cortisol recommendations for select patients (e.g. unstressed dogs), treatment decisions are then actually largely based on clinical assessment, suggesting the uncertainty of this monitoring method.

RECOMMENDATIONS

1. I believe that pre-pill monitoring cannot be justified based on the scientific evidence available

2. I do not recommend pre-pill monitoring.

3. If owners are cost constrained then the options are:

   a. do the 1h post-stimulation cortisol only (to reduce the cortisol costs) in an ACTH stimulation test 4-6h post trilostane

   b. use the minimal amount of ACTH possible (1-5 μg/kg aqueous Synacthen IV if testing at exactly 60 minutes after ACTH) and freeze the remainder in appropriate aliquots (aqueous Synacthen can be stored frozen for 6 months)

[1] See https://www.facebook.com/theESVE/posts/project-alive-2017-agreeing-language-in-veterinary-endocrinology-has-entered-the/887032924806681/ Accessed 23rd August 2018

[2] See: https://www.dechra.co.uk/therapy-areas/companion-animal/endocrinology/canine-hyperadrenocorticism/vetoryl-monitoring-1?utm_source=directmailing&utm_medium=link&utm_campaign=PreVetorylCortisolSuperPage. Accessed 23rd August 2018

c. change to a cheaper drug with less onerous monitoring, namely mitotane! This is still a very good drug for treating hyperadrenocorticism and actually the preferred treatment of some endocrinologists. We must not forget this very efficacious and long-proven treatment of hyperadrenocorticism.

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LARGE

Alleviation of thermal strain after racing in the thoroughbred racehorse with the use of a cooling collar

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Abstract

When thoroughbred (TB) racehorses perform strenuous exercise, a large amount of heat is released from muscular contraction and will add heat to the core. This heat will be transferred by direct conduction to adjacent tissues and by convection via the blood to the rest of the body. The temperature of the blood perfusing all tissues, including the brain will be elevated which might place the animal at risk of exertional heat illness. It has been well documented that the brain in all species is extremely vulnerable to heat, with reversible disturbances in cerebral function starting at temperatures of 41°C (humans), a level common in race-horses during maximal exercise. The clinical signs of heat illness in TB racehorses are neurological, acting along a continuum from mild to severe, each level corresponding to a specific effect on the brain. Treatment for exertional heat illness has been described and involves whole-body cooling with ice-cold water. This article presents the development of a cooling collar as an adjunct to that process. The exact mechanism of action of the cooling collar is unclear and without experimental studies remains conjectural but whatever the mechanism of action the clinical effects are most positive with significant reduction in thermal strain as evidenced by decreased preceived levels of distress and a more rapid return to normality in terms of heart and respiratory rates. The cooling collar warrants further investigation as a useful therapeutic strategy for racehorses competing in hot and humid conditions as an adjunct to the cooling process.

Introduction

Strenuous exercise in the heat is not only a challenge for the musculoskeletal and cardiovascular systems but also for the brain. As the body core and arterial blood temperatures rise the cerebral temperature rises in parallel, with the attendant risk of thermal injury to the brain. (Cabanac 1993; Nielsen and Nybo 2003; Mitchell et al. 2006). Although hyperthermia has effects on all body organs, brain function is particularly vulnerable to heat (Sharma and Hoopes 2003). During strenuous exercise in the heat, a complex physiology leads to disruption in cerebral heat balance. Firstly, there is an exercise-related activation of the brain resulting in increased levels of metabolic heat production and secondly, there is impaired heat removal due to a significant reduction in cerebral

blood flow (Nybo et al. 2002; Nybo and Secher 2004). The latter has been attributed to a hyperthermia/exercise induced hyperventilation and consequent hypocapnia and the overall effect is heat storage within the brain (Nielsen and Nybo 2003; Nybo 2007). Because the blood that perfuses the brain removes most of the metabolic heat produced, brain temperature is related to the rate of blood flow through the brain and the temperature of the blood supplying the brain. In the animals which have been most thoroughly studied (monkey, cat, sheep and dog) the temperature of the cerebral arterial blood was found to be the major determinant of brain temperature (Baker 1982: Zhu et al 2006).

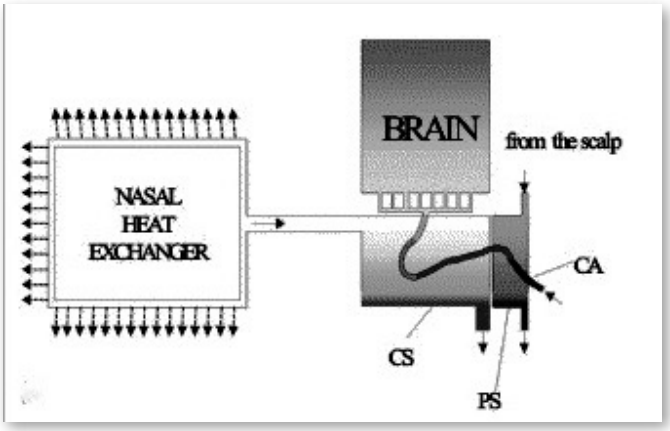


Figure 1: Adapted from Caputa (2004) with permission. Diagrammatic representation of blood supply to the brain of the horse. CA – carotid artery, PS – petrosal sinus, CS- cavernous sinus. Nasal Heat Exchanger represents the upper respiratory system of the horse; the arrow to the cavernous sinus (CS) is the angularis oculi vein.

The thoroughbred racehorse is an exceptional athlete, galloping at very high work intensities of about seventy kilometres per hour but over relatively short distances. It has been calculated that the heat produced can possibly elevate core body temperature by about 1°C per minute, resulting in a possible elevation to 42°C if heat is stored and not dissipated (Hodgson et al. 1993). This is usually not a problem when horses are raced in thermoneutral climatic conditions but when adverse hot and humid weather conditions predominate, heat dissipation is affected and levels of exertional heat illness may become become apparent (Brownlow et al 2016).

The controversy of selective brain cooling (SBC) – a mechanism for lowering brain temperature in response to exercise-induced hyperthermia.

A mechanism for dealing with the hot brain in certain species.

Species with a carotid rete

Many species of mammals — artiodactyls (pigs, camels, deer, sheep, cattle antelopes and others) and panting carnivores, can keep the temperature of the brain below that of the body core during times of heat stress. This is achieved by a special arrangement of the carotid artery called a rete (Zhu et al. 2006; Baker 1982). This 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. In one species of antelope, the Thomson's gazelle, a 2.7°C gradient between brain temperature and trunk temperature was observed (Taylor and Lyman 1972). This lowering of brain temperature below arterial blood temperature has been referred to as selective brain cooling (SBC).

Species without a carotid rete – the horse and human

In both humans and horses there is no carotid rete. Blood is supplied to the brain through the internal carotid arteries which enter at the base of the cranial cavity where it passes, unmodified, through venous sinuses containing reservoirs of venous blood. One of these is the cavernous sinus, through which the internal carotid artery passes before forming the circle of Willis and then dividing to form the cerebral arteries which perfuse the whole brain tissue (Levine, 2008).

In species without a rete the brain temperature and body temperature tends to change in parallel with each other and this has obvious consequences for the brain when exercise induces hyperthermia. Over the years there has been and continues to be considerable debate about the relative physiology of horses and humans, as to whether a mechanism for selective brain cooling exists. SBC was documented to occur in horses (McConaghy et al., 1995) but more recently other researchers (Mitchell et al., 2006) have questioned that finding, along with the role of the guttural pouch in SBC as suggested by Baptiste and colleagues (2000). Mitchell et al., (2006) argued that SBC probably did not occur in any equid and, from an evolutionary standpoint, compared to species with a rete, horses were not designed to run at high speed for long periods and that in their natural state probably responded to exercise in the heat by rapidly initiating robust sweating, and terminating the exercise activity as soon as possible (Fuller et al., 2000; Maloney et al., 2002). Thoroughbred racehorses do not have that option.

Caputa (2004) has diagrammatically represented the blood supply to the brain of the horse (see Figure 1). The paired carotid arteries pass up the neck and divide into internal and external branches. The internal carotid artery (CA) enters the petrosal sinus (PS), then forms an S-shaped curve and enters the cavernous sinus (CS) before piercing the dura mater to form the circle of Willis, from which arise the cerebral vessels. 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'

Figure 2. Horse after racing on a very hot day. Note the dilation of the angularis oculi vein transporting cooled blood from the respiratory evaporative surfaces to the cavernous sinus at the base of the brain.

and cool blood. It is obvious that a single carotid artery within the cavernous sinus would probably not have this capacity and therefore the potential for heat exchange between artery and sinus could only be small (Jessen 1998; 2001).

The role of the upper respiratory passages as a powerful heat exchanger – the horse as a 'capable panter'?

It is probable therefore that the horse has little capacity for selective brain cooling.

The horse does, however, have a powerful nasal heat exchanger in the form of its upper respiratory tract passages, and although not universally recognised, the horse is in fact a 'capable panter' under certain conditions of exercise-related heat stress. This translates to the ability to cool blood using the large mucosal surface area of the upper respiratory tract. There are two possible routes for the cool venous blood returning from the nasal mucosa: one is via the angularis oculi vein which is shown in Caputa's diagram (Figure 1) entering the cavernous sinus from the respiratory tract; the other (not shown) is via the facial vein which flows into the jugular vein and thence into the systemic circulation (Jessen 2001). The question is, does this mechanism provide any real brain cooling under conditions of exercise-induced heat stress and hyperthermia? The prevalence of exertional heat illness with neurological manifestations in those countries where horses race in adverse weather conditions would suggest that it may not (Brownlow et al 2016).

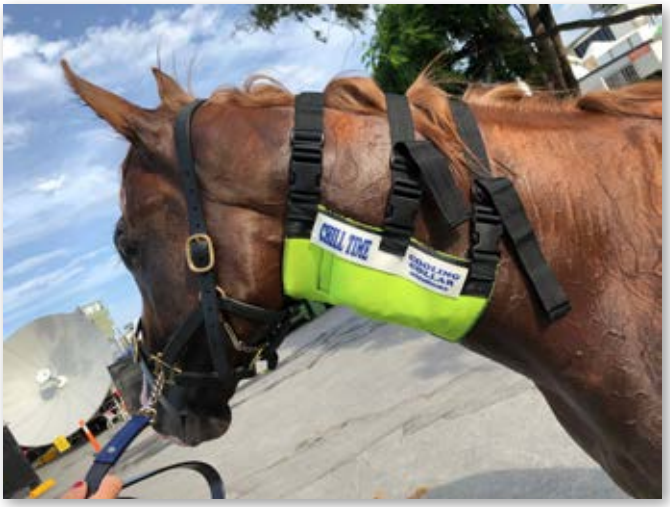
Development of a cooling collar as an adjunct to whole-body cooling

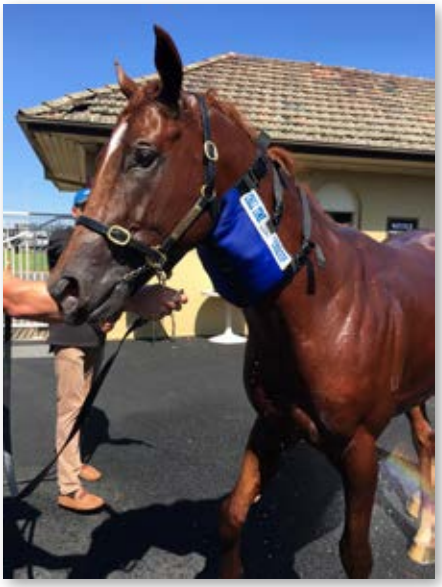
Clinicians faced with EHI symptoms in any sporting horse, but in particular racehorses, recognise that cooling and cooling as quickly as possible is the cornerstone of effective treatment. A useful dictum for treatment is 'early recognition ; rapid response'. Targeted cooling involves concentration of an iced-water stream onto the major vessels of the head, neck and major veins of the legs because these will circulate cooled blood around the body. Cooling really 'hot' horses to reduce their core body temperature usually takes ten to fifteen minutes; they will then usually be walked and re-present with a rebound hyperthermia within another ten minutes. Re-cooling is an accepted part of the process (Brownlow 2014).

It would appear that the only practical method clinicians can help to cool the brain of the 'hot' horse is to cool the skin surface and by convection/conduction cool the blood. Whole-body cooling achieves that to some extent but rebound hyperthermia occurs consistently. A cooling collar was designed as an adjunct to the whole-body cooling process. The aim was to position a pouch with a relatively large surface area on either side of the neck: this is packed with crushed ice, creating a 'heat sink' effect around the artery, adjacent vein and surrounding tissues. The long neck of the horse is well suited to a collar and the very superficial position of the artery and vein in the upper third of the neck provides an easy target for cooling (see Figures 3a and 3b). Three elastic straps strategically located allow a 'one size fits all' design and provide adequate pressure around the horse's neck to hold the collar in place and this has been found to be extremely well tolerated in most horses. Although the collar can accommodate commercially available chemical cold packs, research has shown that ice-filled packs have a greater cooling capacity and remain colder for longer. The use of ice maximizes the heat sink capacity (McMeeken et al.,1984: Phan et al., 2013).

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. However,





Figures 3a and 3b. Showing the cooling collars in place on horses that have just raced in very hot and humid weather conditions.

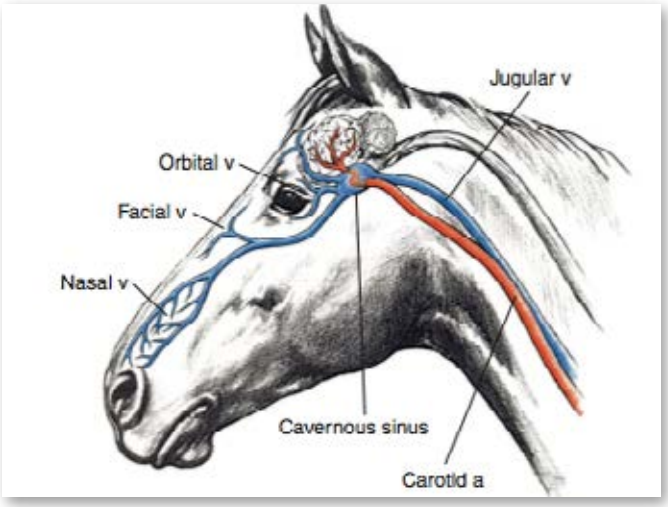


Figure 4. The anatomical arrangement of the brain and associated arteries and veins (with permission, McConaghy 1993).

in the thoroughbred racehorse or sporting horse the sole purpose is to aid in the recovery process. 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 EHI.

Discussion

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. The author has been using a cooling collar over the recent summer period in eastern Australia to manage 'hot' horses after they race in the heat. The cooling collar is placed on the animal as soon as possible after a race, followed by whole-body targeted cooling with iced water until skin temperature is considered acceptable, usually 33°C. The horse is then walked and the collar left in place for up to

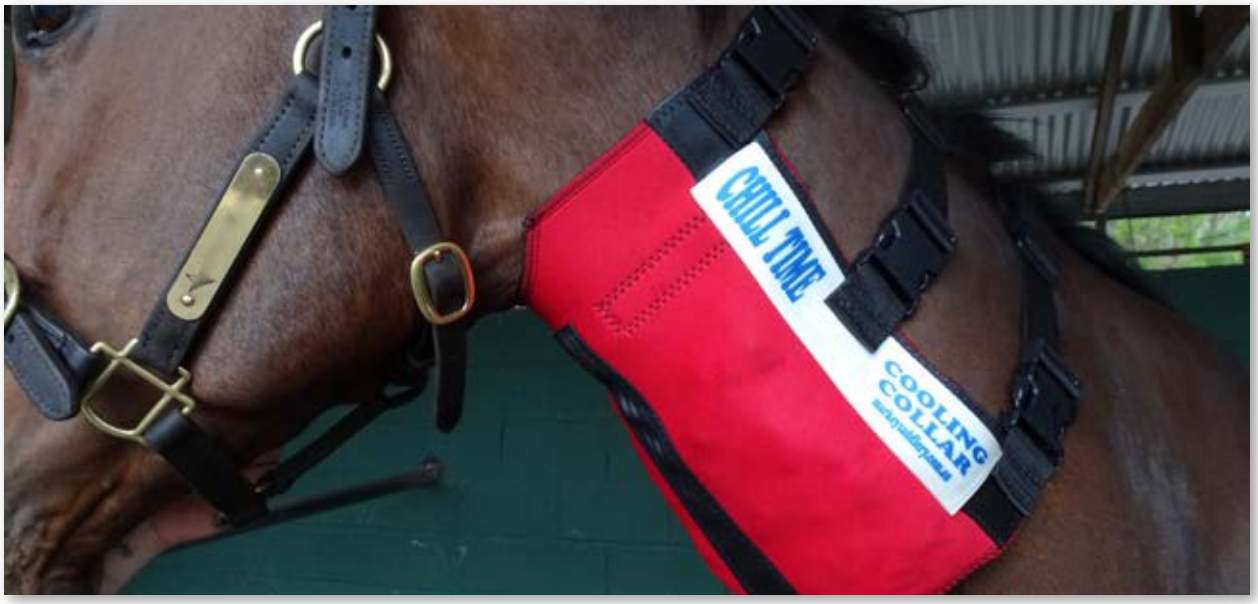


Figure 5. The collar is made from neoprene. The inside surface has bi-lateral pockets sealed with Velcro that hold the ice in place and are separated by a median strip that runs centrally down the ventral neck. The attachment straps are elastic with Mojave side-squeeze interlocking clips and Sliplok slides for adjustment. This horse has not raced but the collar is filled with ice that can be seen projecting above the contour of the collar. Note that there is no patent on this collar. The author, has no pecuniary interest in this product but has spent more than months in the design and development of the collar before using it at the racetrack in the summer of 2017-18 – which has been particularly hot !

fifteen minutes. Rebound hyperthermia does not seem to occur with the cooling collar in place because cooling appears to be sustained and the perceived level of distress substantially improved. Rectal temperatures were not measured during this process due to risk to handlers.

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.

The use of the cooling collar in the management of 'hot' racehorses has in the author's view been beyond expectation in alleviating the clinical manifestations of thermal strain. This article is an attempt to discuss its possible mechanism of action. If the climate warms, performance horses may encounter adverse weather conditions more commonly and if so, any device that cools hot horses should be considered an important part of the management strategy. The welfare of our horses being the absolute priority, it is concluded that further research into the mechanism of action of cooling collars is warranted.

Acknowledgement: Associate Professor Dr. Shane Maloney, School of Human Sciences, The University of Western Australia for his valuable editorial assistance and advice.

The cooling collar referred to in this article is manufactured by Markey Saddlery, Australia.

Reference

http://www.markeysaddlery.com.au/products

Strenuous exercise and environmental heat loads — how the weather imposes heat stress on the racing thoroughbred

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Introduction

The official race day veterinarian is often faced with responsibility for the welfare and management of thoroughbred racehorses competing in adverse weather conditions, particularly the heat and humidity of eastern Australia. The media and welfare organisations have become more vocal in questioning policy and protocol regarding racing horses in the heat, so it is most important that veterinarians have an understanding of how the weather imposes heat stress in order that valid assessments of risk can be made. This article can be extrapolated to encompass all equine sporting activities.

Strenuous exercise and the thermal environment provoke separate and largely independent physiological strains

The mechanism by which environmental heat impacts all animal species is complex and although often considered to be the sole product of temperature, in reality it is the result of interaction between temperature, radiation, wind and humidity. In addition to this, athletic species, namely, the human, greyhound dog and the racehorse produce large quantities of metabolic heat in response to strenuous exercise, which drives hyperthermia. In the thoroughbred racehorse, however, the situation is quite unique. It runs at maximum intensity over very short distances and generates a prodigious amount of energy, much of which is released in the form of heat. During a race there is no active thermoregulation; most of the heat is stored within the body, which raises the core temperature. Hodgson and colleagues[1] have estimated that if all the metabolic heat was stored the core temperature of the racehorse could increase by about 0.8°C per minute. Assuming a starting point of 38°C this suggests that core temperature could approach or exceed 40°C, and muscle temperature would be even higher after two or more minutes of racing. This accumulation of metabolic heat is characteristic of racehorses during competition (see Figure 1), and in the immediate post-race period thermoregulatory processes must begin in earnest to dissipate that heat load if core temperature is to be controlled. If this is not achieved there are consequences for the health of the animal, which have been described previously.[2]

Cooling depends on the thermal environment

Metabolic heat that reaches the skin must leave by radiation, convection and/or the evaporation of sweat. Exchange by these processes is governed by four physical characteristics of the environment: **thermal radiation, air temperature, humidity and wind velocity.**[3,4] In cool weather horses will generally dissipate their accumulated metabolic heat into the surrounding air most efficiently, but high air temperature and humidity not only impede the heat dissipation process but can add to the metabolic heat load. This restriction of heat transfer does not reflect a shortcoming in the animal's thermoregulatory mechanism but rather a limit in the capacity of the environment at that point in time and place to dissipate the heat load[5].

THE HEAT BALANCE EQUATION[6]

$$S = M \pm (R + C) - E \pm W$$

S = storage of body heat (+ for net gain)
M = metabolic heat production (always +)
R and C = radiative & convective heat exchanges (negative for loss or positive for gain)
E = evaporative heat transfer
W = work performed

Figure 1. The heat balance equation shows the integrated relationships between metabolic heat production and the environmental variables influencing heat transfer. There must be a dynamic balance, so that heat transfer into the body and heat generation must be balanced by heat output from the body. If heat generation and inputs are greater than heat outputs the body temperature will rise, and conversely if heat outputs are greater the body temperature will fall. The environmentally driven heat transfer mechanisms of radiation, convection and evaporation play a vital role in either increasing or decreasing levels of heat storage within the body.

How metabolic heat from the core is transferred to the environment — the processes of convection, radiation and evaporation

Heat transfer between the body and the environment occurs along temperature and water vapour pressure gradients (or differences), flowing from high to low, through three independently acting physical processes. These are convection, radiation and evaporation, each of which in a cool environment

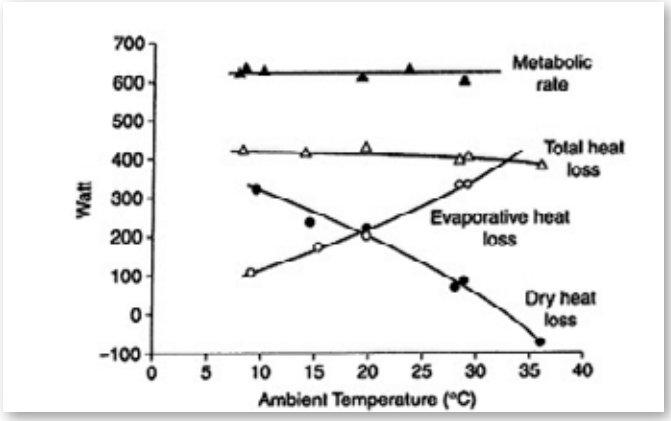


Figure 2. The relative contributions of dry (convective, radiative) and evaporative heat exchange to the total heat loss vary with air temperature. As the ambient temperature increases, the gradient for dry heat exchange diminishes and evaporative heat loss becomes more important. When ambient temperature approaches or exceeds skin temperature, evaporative heat exchange must account for virtually all heat loss. [6,7] (with permission from Dr. Michael Sawka).

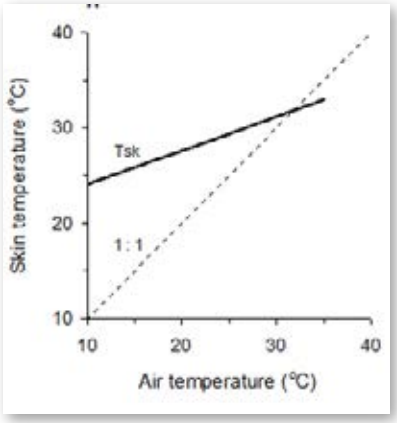


Figure 3. The relationship between skin temperature and air temperature, and how the skin-to-air temperature difference determines the potential for convective heat exchange. At air temperatures below 35°C the skin temperature (represented by the solid line) is higher than air temperature, so that heat is lost through convection. When air temperature exceeds skin temperature, convection adds to the metabolic heat load (heat gain). Zero convective heat exchange is represented here as a 1:1 relationship between skin temperature and air temperature: heat loss occurs above the dotted line, heat gain below [5] (from Brotherhood, 2008 with permission from the publisher).

contributes to heat loss (see Figure 2). Exercising in the heat, however, is the ultimate challenge to thermoregulation and for horses racing in such conditions evaporative sweating is the only means by which heat can be dissipated efficiently. Whether adequate evaporative cooling can be achieved depends, firstly, on sufficient sweat production and, secondly, whether the environmental variables operating at that particular time will allow sufficient evaporation of the sweat produced. This evaporative capacity of the environment is referred to as Emax [5].

Convective heat transfer is referred to as 'dry' heat loss and is entirely dependent on environmental conditions. **The skin-to-air temperature difference determines the potential for convective heat exchange.** [8] When horses race in cool conditions (15-20°C) their skin temperatures (30-33°C) are greater than the air temperature (*see Figures 2 and 3*), and convection and radiation alone will dissipate much of the heat load. However, at higher air temperatures (>35°C), even though the skin tends to be hotter, the skin-to-air temperature difference is smaller and convection is reduced, so that heat loss becomes increasingly dependent on the production and evaporation of sweat. Convective cooling becomes negligible at air temperatures around 36°C and beyond this point convective heat exchange reverses and heat is added to the body instead of being lost. The rate of convective cooling is also **profoundly influenced by air movement** over the skin surface.[5,8]

Radiative Heat Transfer

Outdoors, heat gain due to thermal radiation (R) occurs through direct exposure to the sun, plus reflected and re-radiated heat from surroundings that are hotter than the animal's skin. Radiant heat will be at its maximum in full sun on a hot clear day reaching a peak close to 12:00 hours and can impose a substantial quantity of heat on an animal body depending upon cloud type. Under a full solar load there are substantial radiative heat gains from the sun and nearby hot surfaces, and it is quite apparent that the black bitumen paving popular in race-day stall areas is responsible for the re-radiation of substantial heat on very hot days. It has been estimated that solar radiation may increase skin temperature in humans by 1.5 to 2.0°C;[6,9] and similarly, heat gains from solar radiation of up to 15%[11] have been recorded in horses during exercise in hot, sunny conditions. Radiative heat gain is also associated with coat colour, implying that animals with dark-coloured coats will gain more heat from solar radiation than animals with light-coloured coats.[10] Mean radiant heat (MRT°C) is a meteorological assessment of radiant heat level and during hot days in the summer months readings of 60° to 70°C are commonly observed, compared with 15° to 20°C during winter.[12]

Evaporative Heat Transfer

The production and evaporation of sweat is considered to be the most effective biophysical mechanism counteracting hyperthermia in humans and horses.[13,8] It is the principal avenue for heat loss during exercise in most environments and the only means of dissipating heat if air temperature exceeds skin temperature, but it is a mechanism that works best in conditions of low humidity. Evaporation is a two-step process involving the phase transition of sweat on the skin surface from liquid to vapour followed by the diffusion of vapour into the surrounding air. The driving force for evaporation is the difference in water vapour pressure (WVP) between the saturated skin surface and the air, which diminishes with increasing humidity. Sweat visibly dripping from the skin is a sign that evaporation is not taking place efficiently

and the body is not being adequately cooled.[5] Sweat evaporation is determined by air movement (wind speed) and water vapour pressure (absolute humidity).

Absolute humidity (Pa) is the water vapour pressure in the air and directly affects evaporation. **Relative humidity (RH%)**, although commonly referred to, is the most misunderstood and misused humidity variable[14]. Usually expressed as a percentage, it is the ratio between the water vapour present in the air, and the theoretical saturated vapour pressure at the same temperature. Put simply, RH% represents the extent to which the air is saturated. Scrutiny of its calculation, however, reveals that whilst the numerator depends on humidity the denominator varies with air temperature. This means that relative humidity can change quite markedly while the air's actual moisture content (the number of water vapour molecules) remains constant. RH is highest when the air is close to saturation, which usually happens in the early morning and in winter. Thus, RH is diurnal typically declining throughout the day as it is inversely proportional to ambient temperature. Yet, absolute humidity and thence the evaporative drive often remains near constant.

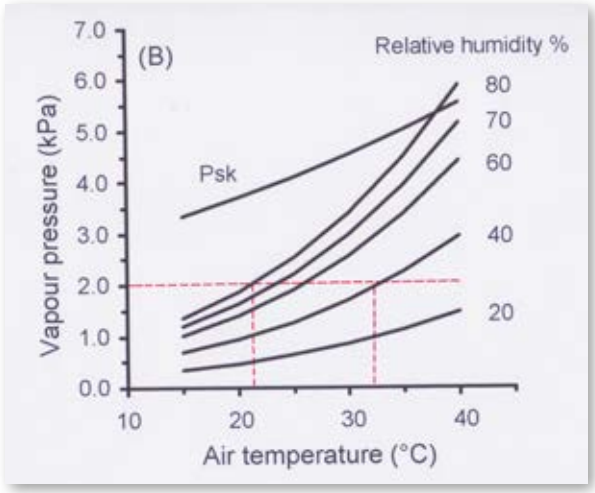
Because RH depends on both humidity and temperature, its use is limited in a heat stress study where the objective is the specific impact of individual variables. Relative humidity is also not directly related to the evaporative capacity of the environment (see Figure 4), in contradistinction to absolute humidity, so is not a reliable guide to the potential for heat stress.

Humidity and wind speed dictate the evaporative capacity of the environment.

The limiting factors to sweat evaporation are air flow over the skin and absolute humidity. The relationships between air temperature, relative humidity and absolute humidity are shown in Figure 4.[5]

In stable weather, water vapour pressure (Pa) remains fairly constant throughout the day, while relative humidity is often high in the cool of the morning and then decreases as the day warms up. This decrease in RH% in the middle of the day when racing is usually staged does not mean that evaporative conditions have improved, but rather that the saturated vapour pressure (theoretical maximum) has increased with rising air temperature. Nevertheless, if relative humidity levels are high it is an indication that the evaporative capacity of the environment is probably restricted and that horses will face difficulties with cooling.

Wind speed has a significant effect on evaporation because it modifies the horse's immediate thermal environment. By dispersing the hot air and vaporised sweat close to the skin it increases the rate of convection and evaporation and thereby accelerates heat loss from the body. However, air movement can be quite unpredictable, varying in time, direction and from location to location. A sudden wind drop can transform an uneventful race day into one on which horses become heat affected. It is also apparent that some areas on racetracks are shielded from the wind; for instance, horse stall areas tend to be characterised by '*wind still*' conditions and in effect have their own microclimate, with different and changeable evaporative conditions compared to the more open racing and parading areas. Low wind speed severely compromises cooling and will predispose to heat stress conditions in the hot summer months. In contrast, moderate to high wind speeds create good conditions for evaporative cooling despite high levels of absolute humidity.[12]



Psk is the saturated vapour pressure at skin surface (varies with skin temperature)

Figure 4. At constant air temperature, absolute humidity (vapour pressure) increases with increasing relative humidity, but at constant vapour pressure relative humidity decreases as air temperature rises. For example: Ambient vapour pressure of 2.0 kPa (red dotted line) corresponds to 80% RH at 21°C and 40% RH at 33°C.

Discussion

Strenuous exercise imposes a significant metabolic or internal thermal load. The capacity of the horse to dissipate that heat without sustaining a progressive elevation in core body temperature is inextricably linked to the thermal environment. The term '**thermal compensability**'[7] **defines this interaction between the body and the environment**. If the environment is considered to be uncompensable it is quite likely that hyperthermia will result, with the possibility of thermal strain and the manifestation of exertional heat illness. It is important to appreciate that in regard to horses **not all sports activities are equal**. During thoroughbred racing the large muscle groups responsible for locomotion produce large amounts of heat whereas, endurance and dressage activities result in lesser levels of heat production. Hodgson (1993) has documented varying levels of heat production from different equine athletic events as follows:- 400 (kJmin-1) for endurance; 500 (kjmin-1) for eventers and 1250 (kjmin-1) for thoroughbred racehorses respectively. Thus, metabolic heat production is substantially higher for the thoroughbred racehorse.

How **the weather imposes heat stress** can be divided simplistically into two different categories. Firstly, hot

days with ambient temperatures up to 40°C and high radiant heat loads and, secondly, days of moderately high temperatures with high levels of absolute humidity. Superimposed on these conditions is the effect of wind speed. For the purpose of this article heat-affected horses are described as 'hot' with skin temperatures as determined by infrared thermometry > 39°C and display emergent signs of CNS dysfunction.

KEY POINTS

ENVIRONMENTAL CONDITIONS AFFECT BOTH CONVECTIVE AND EVAPORATIVE HEAT TRANSFER MECHANISMS

Convection depends on the temperature gradient (or difference) between the skin and air. Heat is lost below air temperatures of about 33°C but will be gained at higher temperatures.

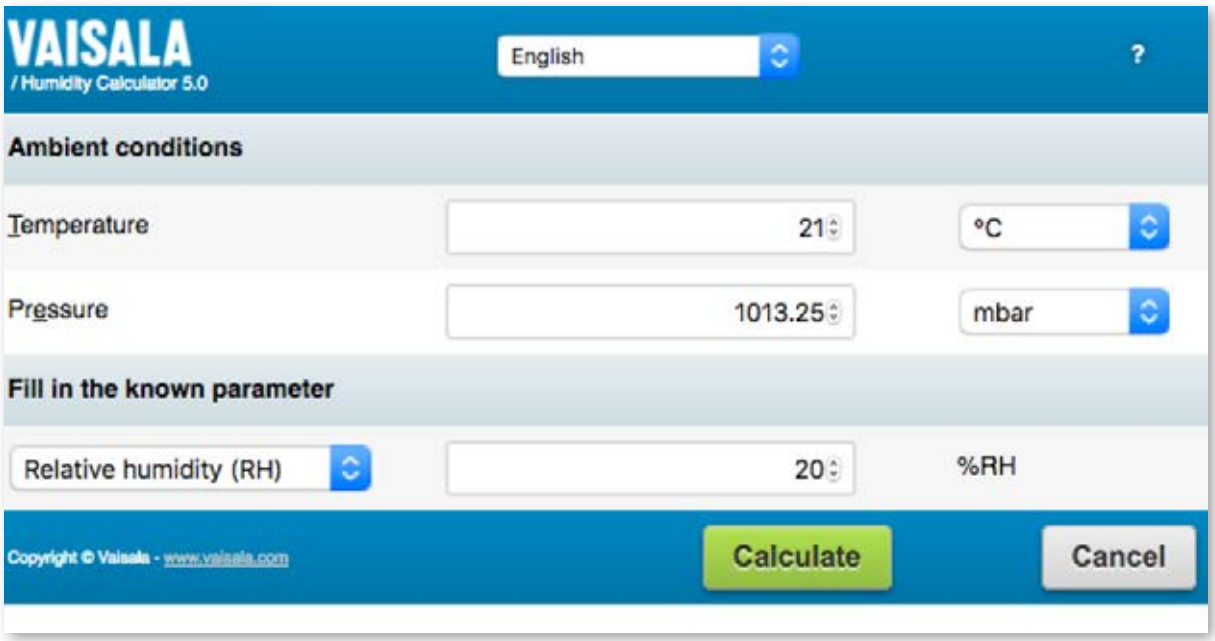
Evaporative cooling results from the vaporisation of water at the skin surface and its dispersal into the atmosphere. The process depends on the difference in water vapour pressure (kPa) between the body surface and the ambient air.

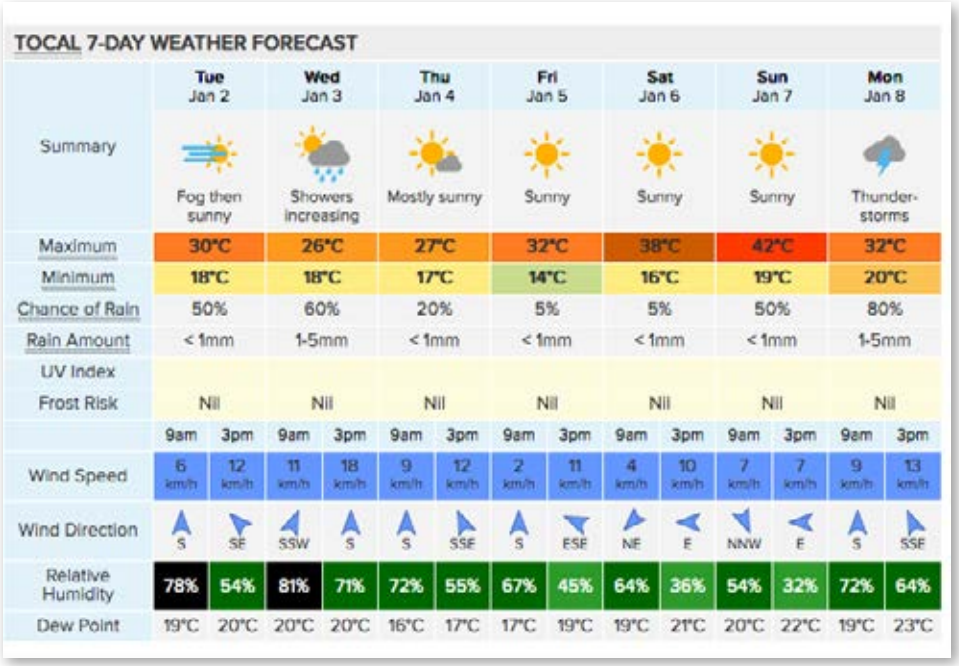
The maximum evaporative potential of the ambient environment is referred to as Emax and is largely determined by air velocity and absolute humidity. Most importantly it is the evaporation, not the production of sweat that allows the body to cool.

The most difficult question is, how hot is too hot?

Very high temperatures (>38°C) act to increase skin temperature, which alters the relative contribution of convective and radiative heat transfer, possibly initiating heat gain from both sources. Physiologically, there is increased skin blood flow and the initiation of sweating. At this point the only mechanism for significant heat dissipation is evaporative sweating with some additional heat loss via the respiratory tract. External radiative heat gains may add to the thermal load produced from racing, making hyperthermia more likely in the recovery stage. In the author's experience, thoroughbred racehorses tolerate 'dry' heat quite well, and very hot days (39 to 41°C) in NSW are not usually accompanied by very high humidity. Of 23 hot days monitored by the author where horses were heat affected, 6 days were categorised as hot, ranging from 36.5°C to above 40°C, with a mean relative humidity of 30.0% and a mean absolute humidity of up to 1.9 kPa. The key determinant of clinical severity was wind speed. Wind speed below 1.0 metre per second created the most difficult conditions, but at wind speeds above 2.5 metres/second horses coped well. The latter were actually dry coated because their sweat had evaporated efficiently and their heart rates were within the same limits as horses recovering on thermoneutral days, that is, they were not showing clinical signs of thermal strain.

The second adverse weather category comprised moderately hot days but with higher levels of humidity, and many horses in this group were significantly heat affected. There were 17 days with temperatures ranging from 29.2 to 35.0°C, the majority being in the low 30's. Relative humidity was between 50% and 80%, but most importantly absolute humidity was generally above 2.50 kPa (*absolute humidity levels can be calculated from the VAISALA humidity calculator 5.0 (see Appendix 1* for link). Once again, the most critical factor was the wind speed. If it dropped below 1.0 metre per second conditions were most difficult, with most horses requiring aggressive cooling. Horses in the recovery phase after racing were dripping with sweat and displaying signs of thermal strain, with elevated heart rates and a panting type of respiration. This was because evaporation was restricted and they were not cooling.





TOCAL 7-DAY WEATHER FORECAST

	Tue Jan 2	Wed Jan 3	Thu Jan 4	Fri Jan 5	Sat Jan 6	Sun Jan 7	Mon Jan 8
Summary	Fog then sunny	Showers increasing	Mostly sunny	Sunny	Sunny	Sunny	Thunder-storms
Maximum	30°C	26°C	27°C	32°C	38°C	42°C	32°C
Minimum	18°C	18°C	17°C	14°C	16°C	19°C	20°C
Chance of Rain	50%	60%	20%	5%	5%	50%	80%
Rain Amount	< 1mm	1-5mm	< 1mm	< 1mm	< 1mm	< 1mm	1-5mm
UV Index							
Frost Risk	Nil	Nil	Nil	Nil	Nil	Nil	Nil

	9am	3pm	9am	3pm	9am	3pm	9am	3pm	9am	3pm	9am	3pm	9am	3pm
Wind Speed	6 km/h	12 km/h	11 km/h	18 km/h	9 km/h	12 km/h	2 km/h	11 km/h	4 km/h	10 km/h	7 km/h	7 km/h	9 km/h	13 km/h
Wind Direction	S	SE	SSW	S	S	SSE	S	ESE	NE	E	NNW	E	S	SSE
Relative Humidity	78%	54%	81%	71%	72%	55%	67%	45%	64%	36%	54%	32%	72%	64%
Dew Point	19°C	20°C	20°C	20°C	16°C	17°C	17°C	19°C	19°C	21°C	20°C	22°C	19°C	23°C

Example of an internet weather site: Gives hourly predictions for required weather variables. Note the diurnal variation to relative humidity levels.

Conclusions

For the veterinarian anticipating a possible hot day for racing it is important to monitor the predicted weather conditions using one of the many internet weather sites (see Appendix 2). A typical race day encompasses 4 to 5 hours in the afternoon and predictably ambient temperature tends to increase as the day progresses with peak values obtained between 14:00 to 16:00 hours. On the other hand radiant temperatures peak closer to 12:00.

Establish whether it will simply be hot, or hot and humid, and most importantly get an idea of the wind speeds that might prevail on the day. Your risk assessment will depend upon that information. Relative humidity measurements should be used with caution and with an understanding that RH% varies as a function of both water vapour content and air temperature, so that RH% may rise at the end of the day even though the air's absolute water vapour pressure remains the same. **It would appear that absolute humidity and wind speed are the most important determinants of the incidence of heat illness in thoroughbred racehorses** (Brownlow – unpublished observations). Monitoring weather conditions on the day is also recommended - how to do this will be the subject of a further article.

The worst prediction is for a day with moderately high temperatures (33.0 to 35.0°C) and high levels of absolute humidity (>20.0 kPa). **These conditions tend to be underrated by all concerned, and remember, wind speed is critical.** The weather is also unpredictable and can change abruptly. A morning breeze might completely disappear by mid-afternoon; or cloud cover may be lost, making conditions more difficult.

From the above discussion it is apparent that blanket 'cut-off' points for temperature won't work. This is because physiological strain is individually determined and depends on the complex interaction of the horse's metabolic heat production and weather variables such as humidity, cloud cover and wind speed. Nevertheless the veterinarian must be '**prepared in advance**' during the summer months. If a difficult day is suspected, you must put in place advanced contingency plans, in consultation with the stipendiary steward responsible for the race meeting. Generally, the risk to horses on the warm and humid days will be under-estimated and temperature alone is not a reliable guide for risk assessment. Cooling devices, a continuous supply of ice and extra personnel are mandatory to cope with 'hot horses'. As always the welfare of horses is the absolute priority.

APPENDIX:

Step 1: ***Access weather information****. There are several websites which are useful to predict weather conditions in advance. These are the Bureau of Meteorology* www.bom.gov.au/nsw/forecasts/sydney.shtml*. Scroll down to MetEye™ and put in your specific location. Record the projected wind speeds, temperature and relative humidity levels as provided. Another good weather site providing detailed information for seven days ahead is weatherzone* www.weatherzone.com.au/nsw/sydney/detailed-forecast*. Enter racetrack location to receive specific data. Weatherzone provides information on projected temperature, radiant heat, cloud cover, wind speed and relative humidity and is also available in graph form.*

Step 2: The following link provides access to the VAISALA humidity calculator 5.0. which can be downloaded to mobile phone devices so that absolute humidity levels can be easily obtained at race meetings.
http://go.vaisala.com/humiditycalculator/5.0?utm

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eBook download:

Perspective 126. M Brownlow, The management of 'Hot' Throughbred racehorses after a Strenuous Excercise in Hot and Humid Conditions. A Physiological Approach, Issue 283 June 2016.

Perspective 136. M Brownlow, How do Thoroughbred Racehorses Cope so Effectively with the Physiological Challenges of Strenuous Excercise in Hot Humid Conditions? Observations from the Race Track. Issue 289, Dec 2017.

Perspective 137. M Brownlow, Cooling Interventions for Thoroughbred Racehorses an Overview of Physical Heat Transfer Mechanisms & Practical Considerations, Issue 289, Dec 2017.

Answer to 'What is your diagnosis?'

C&T No. 5698 (Issue 292, Sep 2017)

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C&T No. 5722

A 3-year-old male German Shepherd presented for nasal crusting and depigmentation. No other skin or mucocutaneous lesions were present. There were no signs of systemic disease. He was eating, drinking, urinating and defecating normally.

Questions

- What are the diagnostic possibilities?
- How would you investigate this patient further?

Answer

An ulcerated wedge of right nostril was biopsied and submitted for histopathology.

Diagnosis Mycotic dermatitis with intralesional cryptococcosis.

Pathology Summary

Histopathology revealed an inflammatory infiltrate in the dermis dominated by macrophages and neutrophils with numerous intralesional yeast cells with large negatively staining halos, consistent with *Cryptococcus* spp.

Bloods were collected to measure the LCAT titre (latex cryptococcal antigen agglutination test) to ascertain whether the infection was more likely to be localised or systemic.

Treatment Protocol

Day 1. Collect bloods for renal monitoring and L cat. Infusions are made from one diluted syringe of amphotericin that has been frozen thawed and is injected through a filter into a single bag of 500mL 2.5% glucose, 0.45 % NaCl. This bag must be warmed to between 60 and 70 degrees in a warm water bath.

For each treatment we used two bags, one bag to be given subcutaneously on either side of his chest.

The Lcat is to be repeated in 6 months and the renal enzymes monitored each month.

The treatments were to be repeated at least twice per week for 3 months or until complete resolution of the lesions.

Gloves and face-masks with eye protection should be worn and administering and handling the amphotericin

The main side effects are sterile abscesses and kidney disease.

L cat: Cryptococcal antigen (REMEL) latex agglutination titre positive @ 1:64- low positive result suggested that it was primarily a localised, not a systemic disease.

In view of these results, after 2 weeks of twice weekly administration we changed the treatment protocol to once weekly intralesional injections i.e. 2.5mL amphotericin B diluted in 5mL 5% glucose, warmed to 65 degrees for 10 minutes and injected intralesionally Renal enzymes were monitored prior to each treatment.

By week 5 of treatment all crusting had resolved and previous lesions were contracting. Two further weekly intralesional injections were given.

Treatment then transitioned to 400mg of fluconazole twice daily.

Three months later there was no sneezing or nasal discharge. Scarring had reduced significantly and repigmentation was occurring.

L cat levels were repeated L cat 1:8.

Maintain fluconazole for another 6 months.

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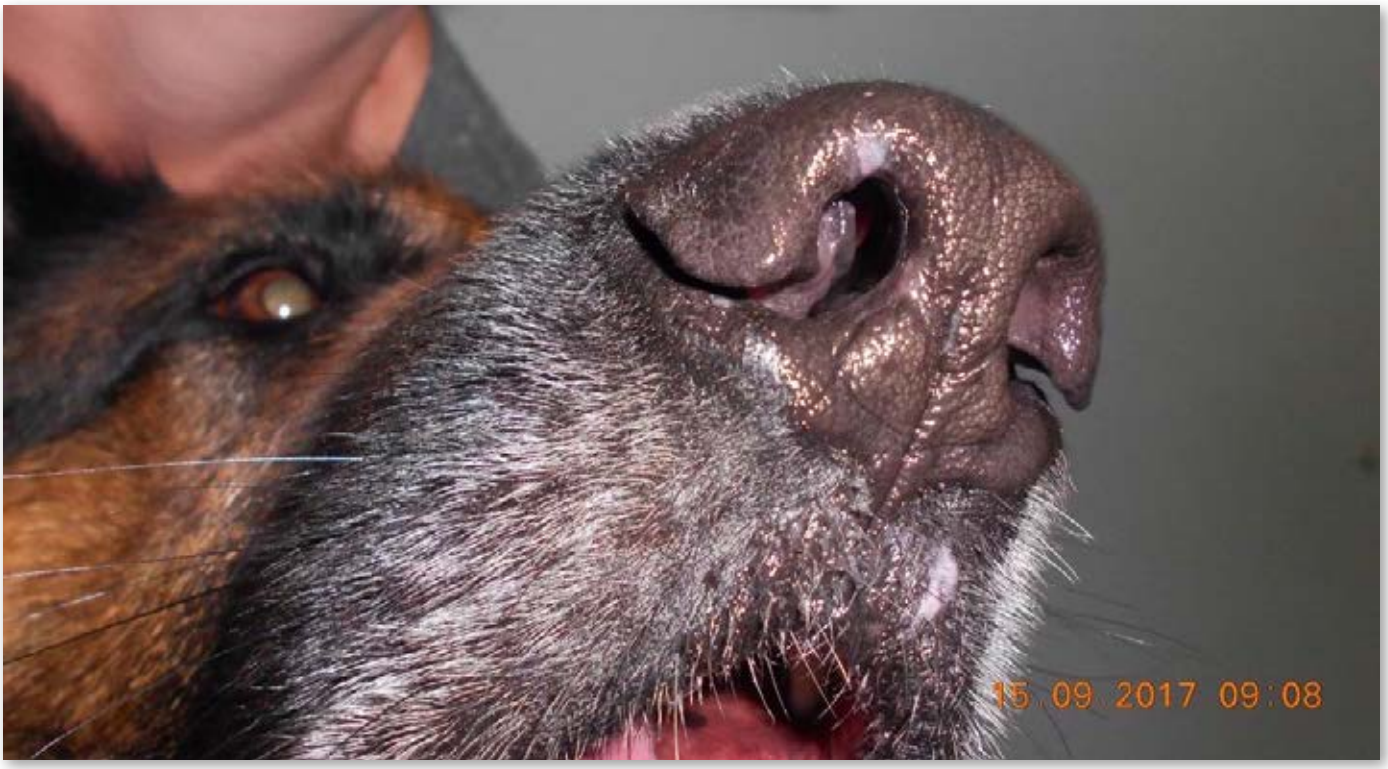

Danièlle's top tip

Danièlle Gunn-Moore

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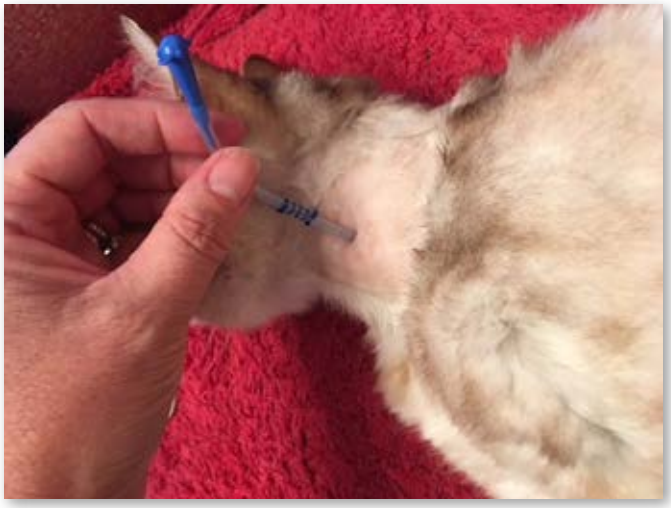
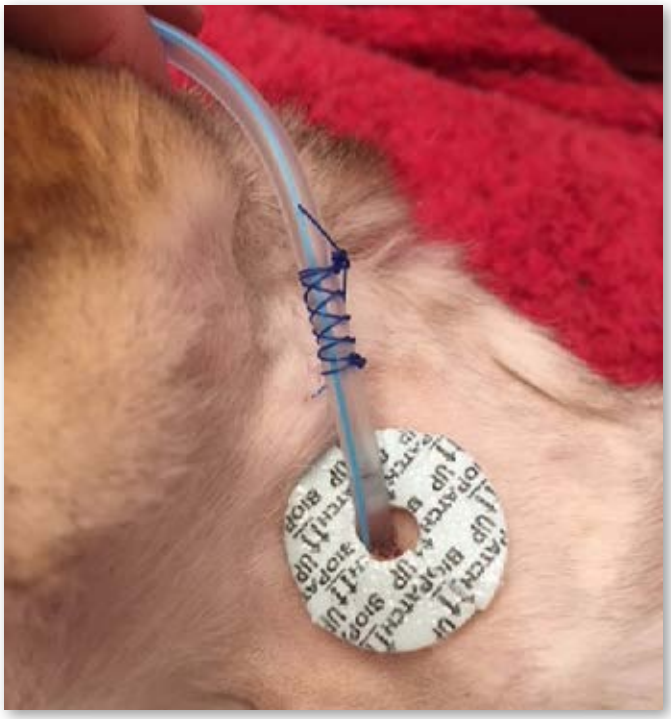
C&T No. 5723

We attach oesophagostomy tubes with 2 finger trap sutures to reduce the risk of the tube slipping or being pulled out. If you suture a little away from the O-tube entry site it is easier to clean the site and change dressing. We place the second suture into the periosteum behind the ear so the tube sits really comfortably, cut off short and plugged, that way you can stop the dressings pretty quickly and the cat can get on with its everyday business.



This patient (fabulous Cassius) needs to wear a soft dressing every day to stop his scratching – so we now only place a single stitch into the periosteum away from the wound – he is more comfortable this way and it is very easy to change his dressing.

He has a Biopatch placed round it to reduce recurrent infections (this kitty has an oesophageal diverticulum so biofilms wick up to the O-site). The stitch is through the wing of the atlas so it does not move or pull out. He does tend to scratch (because of the recurrent infections, plus neuropathic pain from probable hypervitaminosis A – he has been nil by mouth for 5 years and some commercial diets in the UK are >10 the max concentration of vit A allowed in the US – but still legal in the UK – I was very upset) so he wears a little collar over it



Hasn't anyone mentioned that before...?

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Wingham and Valley Vets

Wingham NSW 2429

Cartoons courtesy of Dr Robert Johnson

Participants at the 4th Recent Graduate Survival Seminar held in November 2017 heard from an excellent range of vets happy to share their experiences to help the next generation in the workforce. John's presentation was quintessential Australian: years of practical experience in mixed practice distilled down and presented in an informative and humorous manner. Read on...

I welcome the invitation from the CVE to make a contribution to this event by passing on to Students and Recent Grads some food for thought to help smooth your path, and help keep you out of trouble while you are learning to flourish in the Profession.

I do have a warning for you, though – do not accept anything which I have to say, at face value – it is simply the product of my own experience, not of yours. Over time though, I expect that some of my thoughts may ultimately prove to be valid in your own experience.

I would like to open with a poem!
This is written by Glenn Colquhoun who is a New Zealand GP, and published by Ginninderra Press.

John graduated from the University of Sydney in 1975. He has been in mixed-species practice since then, mainly in the Manning Valley. In January 2011, his very successful Wingham Vet Clinic merged with the Valley Veterinary Hospital to become Wingham and Valley Vets. John has long enjoyed the challenge of being competent across all species – friends who are single-species specialists say their jobs are much simpler!

> She asked me if she took one pill for her heart
> and one pill for her hips and one pill for her chest and one pill for her blood
> how come they would all know which part of her body they should go to.
> I explained to her that active metabolites in each pharmaceutical would
> adopt a spatial configuration leading to an exact interface with receptor
> molecules on the cellular surfaces of the target structures involved.
> She told me not to bullshit her.
> I told her that each pill had a different shape and that each part of her body
> had a different shape and that her pills could only work when both of these
> shapes could fit together.
> She said I had no right to talk about the shape of her body.
> I said that each pill was a key and that her body was ten thousand locks.
> She said she wasn't going to swallow that.
> I told her they worked by magic.
> She asked me why I didn't say that in the first place.

It is about communication

Assess your audience, and choose your language and vocabulary to suit that target. This is a very important consideration in Consultations, for Discharge instructions, and for provision of ongoing care. You are all highly educated young people, but the sooner you learn how to communicate effectively with each of your clients, the better. Effective communication has a direct effect upon many a patient outcome.

I believe that for many of us, ***we do the work we do for the People, even more than we do for Animals.***

Hence, developing effective communication techniques is essential.

It may be useful to think of a Consultation or other discussions with clients as a theatrical performance – so learn how to work with a script, which may be of your own devising. Learn how to use light and shade in your delivery, in order to effectively emphasize points.

Learn how to choose terminology which is appropriate to the client's level of education, and appropriate to their emotional state at the time, perhaps.

Learn when to shut up, and not say anything—stop digging when you are in a hole. That can sometimes be a very effective method of communication.

It has been a bit of an exercise putting the material for this presentation together – most of my most useful advice crops up while one-on-one with a student while driving back from attending a case, and things arise from there. I am a master of the self-induced spontaneous digression, so I suppose that I should extend an invitation to you to interrupt me if you so wish.

Having employed a fair few new grads, from a variety of Vet Schools, and having hosted a lot of students, I thought it useful to approach some of those people for feedback as to what if anything they remember of things that I said to them or showed them which ultimately proved to be true or useful to them – so I sincerely thank those people.

So you have your job – what now?

Be aware that being a Boss is stressful – stress comes with the territory when employing new Grads. The Boss will have Duty of Care responsibilities – to the Patient, to the Client, to the Business, to his or her Domestic setup, to Himself | Herself, and to You the New Grad – and some Bosses bear this burden more lightly than do others.

Your role is to learn how to become as productive as possible, as soon as you can.

Videos: CVE members are reminded that Recent Graduate Survival Seminar recordings and others are available in the CVE Library

The best way to do this is to concentrate like hell – focus your mind – bring method and mindfulness to everything you do in your work.

Equally importantly, I encourage you to bring that same approach to all of your non-work activities.

Be very aware of the risk of becoming a dull boy or girl. In my opinion, you will become a happier individual if you do not apply every waking moment to your work. You will also be a more interesting work colleague for everyone else.

An allied thought is this:

I recommend not approaching your need to see progress in yourself, with any sense of rush. Cultivate in yourself the recognition that once in the workplace, your progress is inevitably constant – hour by hour, day by day you will be making progress – and learn to recognize that fact.

To assist this, I think that it is ideal to have a regular booking with a senior person in your workplace – e.g. the boss or another senior clinician – to discuss your progress. The recommended frequency for these discussions will vary depending upon personalities, but it may only require as little as 10 minutes per week, and some weeks will require no discussion at all.

It is useful to rapidly develop a clear understanding – at both an intellectual level and an emotional level – that ***you will not solve every case at its initial consultation.*** If an animal needs to be presented again, it is not your fault!

I think that for many of us this is a confidence and Self-esteem issue – but it is a good one to unburden yourself of, as soon as you can.

Some cases simply need to evolve – so develop the confidence to tell clients this. This becomes part of your own script, and learning this will be a step towards taking some pressure off yourself.

As a tip in dealing with the revisit case:

I have found it a useful habit to ***NOT RE-READ the previous case notes*** –whether they are your notes or from one of your colleagues –prior to carrying out the clinical re–examination.

Train yourself to approach the patient as if it's your first examination of them – first double-check the case history with the client, then carry out the Physical examination, ***and then*** re-read the previous case notes.

This process should go some way towards reducing bias in the second assessment.

As a digression, I hold the view that we should not charge a reduced fee for such revisit consultations – it defies logic

to be probably putting in even more intellectual effort etc than we did originally, stressing about what we 'must have missed' i.e. viewing it as an omission or error on our part that the patient has not yet turned the corner. This latter attitude arises from, and is evidence of, the Self-esteem Cringe which pervades parts of our industry.

I am not including in this the consultations for purposes of rechecking wounds, etc. But in any case, it makes no sense to me to ever charge less than the primary consult fee for repeat examinations on unsolved cases.

Always be very careful to not diagnose what the patient is booked in for!

I think that for inexperienced and under-confident clinicians, this is a very real risk! Making such an error is much easier to do when you are inexperienced in everything you are doing. For example – a patient booked in for Arthritis.

So: always get a proper history

Ask what it is that the patient is doing at present which is different, or has stopped doing, or is doing in a different fashion to previously. Once you are very clear on the History, move to carrying out a thorough clinical examination.

Having a patient booked in for 'Arthritis' does not absolve you of the responsibility for carrying out a complete physical examination and finding a set of diagnostic indicators.

We have been given a wonderful scientific jargon, with terminology for every detail we will ever need to record, so I commend to you the value of using said terminology. Provided that you use the terminology accurately, then all of your records and communications will be delightfully clear and unambiguous, especially to your colleagues.

However, I must caution you ***to NEVER ACCEPT*** under any circumstances the client's terminology for describing the patient's signs – if you do so, then this way may lie acute embarrassment! I wish to really ram this point home.

Consider this:

Sniff, Sniffle, Sneeze, Snort, Reverse sneeze, Splutter, Gag, Choke, Cough, Wheeze. These are some of the terms which you may apply to describe sounds made by a patient. **But please be warned—**

The client's use of this terminology may be rather different from your own!

The solution: Ask the client to reproduce the sound! You may be a bit coy about asking this, but get over it! Most clients are fine with being asked – I do not remember ever having my request knocked back.

I think that you will find that a client will be very accurate with making the sound. The only complication will occur when the client has company – who may say 'No – it wasn't like that – it was more like... (makes the noise)…' So, getting the firsthand audio of the sound simply increases the reliability of the information.

Then: apply your own terminology to the sound, and carry on with your thinking! The reason that I make this point is that in this particular scenario, getting the History right may make the difference between performing nasal endoscopy, or doing thoracic radiographs – quite an important distinction.

So do not rely on Client terminology!

Take on board also, both intellectually and emotionally, that you cannot save them all. Sometimes a patient will simply be too damaged, and it would not matter what level of resources was to be provided, death would still be inevitable. Sometimes Financial constraints are a fact of a client's circumstances – this is not your fault either. Rarely only will loss of a patient be through any fault of commission or omission on your part – even as new grads, most of you have excellent knowledge, and sufficient experience to take sensible decisions. So loss of a patient is not a reflection on your ability, or your compassion, or your lack of experience. ***But you simply have to learn to live with these inevitabilities!***

Do a very thorough clinical examination!

This is good for the Patient, good for the Client, and ultimately good for the Practice and its Economics. A good thorough physical examination does not take long. Work out and develop your preferred examination protocol or sequence as soon as you can – many of you will have done this already, I know.

Having a fixed process helps the brain interpret and recognize abnormalities – ***your unconscious brain*** reacts much more quickly to picking up on variations from the expected, if it is used to receiving sequenced inputs. So work out your preferred sequence for examining each species, and stick to it!

In the first instance, carrying out a good thorough P/E delivers to the client an immediate very strong ***Perception of Value.***

Generally speaking, the clinical examination is a process over which you mostly have significant control – except where patient behaviour precludes – so do your Clinical assessment really well!

When a new client says to me 'That is the most thoroughly that any vet has ever examined one of my animals …',

my immediate sense is that I have provided a very good Perception of Value, and that if we manage things well around this, this perception should be transferrable to the Practice as a whole.

So do it – be thorough – you will never regret it!

Be prepared to leave the Consult room to look stuff up. I don't think that it is too dishonest a manoeuvre to make up some excuse if you feel the need to – but be prepared to act to inform yourself, if need be.

I encourage you to develop a well-rounded philosophical view about what constitutes doing good work.

(Coolongolook story:)

One afternoon earlier this year, our Receptionist came to me and asked me to take a call. The caller had a problem with a bovine, is a long distance away, and has declined the offer of a visit. My discussion with the client reveals that he has an 18mo bull, kept tethered on grass and moved about. The bull is the sole animal. There were no observed abnormalities yesterday – the bull was observed on his feet.

Today he has diarrhoea with blood and slime, discharge and some mucus from his eyes and mouth, and is quote 'crying his eyes out'. He reportedly had the same signs 6-8 weeks ago, and came good after a deworming, as the sole intervention. He is today so weak that he has to be propped up to achieve sternal recumbency.

I proffered the opinion that regardless of the Differential Diagnosis, the prognosis is likely to be less than 50%. Client then said 'Tell me Mate – is this like a Priest thing?' 'In what way do you mean?' asked I. He replied 'If I tell you something, will it stay between just us?' 'Of course!' I replied.

He said 'I have a 100 year-old Animal Remedies book, so I read it, and bought some whisky, and gave him two stubbies-full of that.' I checked re stubby volume – 375mLs each, so almost 800mLs of whisky. He continued – 'The book advised giving him Castor oil; I didn't have any of that, I only had olive oil – so I gave him one stubby-full of that. And I also gave him about a litre of lemonade.'

I asked him whether swallowing had appeared competent – although I did not use that exact phrasing. He answered in the affirmative. I advised as follows: I said that the lemonade was OK, should do no harm. I advised giving no more oil. I advised that whiskey in that quantity may risk causing an insult to the liver, which would not help the evident bowel problem. I advised that the patient as described would require IV fluids and other maintenance, and that prognosis is very guarded. I advised that regardless of his not wanting us to visit, I did not think it worthwhile for him to drive all the way in to the clinic to pick up any 'on-spec' medication. I then advised that in the circumstances, I really did not have anything else to offer him or the patient.

He replied 'No mate – that's fine – I feel so much better after talking to you – thanks. Bye!'

I wish to make this point to you – we do work for People, who come in all shapes and sizes and cultural packaging.

On some days, something like the event just described may be the best work you do on the day – unsophisticated, not at all glamorous, but it made someone feel better!

Similarly, as a keen young vet you are presented with an itchy terrier – named 'Ivan'.

Client is a young mother, 2 infants at foot, and clearly pregnant again. You are having to be careful to not step on one child, while the other is swinging off the leg of the consult table. You diligently commence to develop rapport by asking some general background questions – she tells you that her husband is working FIFO mining, is away for much of the time, and is tired when at home. She is on good terms with 'Shirley Next Door', who sounds like a very good neighbour for her to have.

You collect a thorough History, and proceed to examine Ivan. Erythematous and mildly lichenified pinnae, saliva-stained feet, mild lip fold and chin skin changes, erythematous ventrum. You develop your differentials, collect your samples, do your microscopy, and compose your treatment plan.

A choice of Miticides, 4 weeks course of Cephalexin, shampoo, Fatty Acid supplement, setting up food trial, Apoquel etc. You have it all covered, because you are a highly educated young vet, and really keen to make as much difference to animal health as you can. You provide it all, plus loads of written material to back up your recommendations, and make a booking for a revisit in a couple of weeks' time. You even help by carrying all of the meds to the car, and helping her to put the kids in.

The client gets home, and Shirley Next Door sticks her head over the fence, and asks 'How'd you go at the Vets?' The client comments upon how nice you were, how caring and knowledgeable you seemed, and how encouraging you were about the prospects of improving Ivan'sskin condition.

But, she says to Shirley 'I have been given all of these things to do, and I just don't know how I am going to manage to fit them all in!' The chances are that compliance will prove to be a real problem here – one of the kids gets sick, and everything else has to be let slide in consequence.

An alternative approach may be:

Same thorough history, and thorough clinical examination – simply because this is how you always do things. Same diligent microscopy. Work out your ideal treatment plan.

Then stop, and look at the situation in its entirety.

What are your available resources here? Explain to the client that Ivan's clinical status is likely long-term to be at best manageable, rather than curable. Make sure that she understands this. Tell her that management will require long-term sustained effort, then ask her what patient management procedures she feels she will be able to take on and stick to pretty reliably.

Point out that you can see clearly how much responsibility she already has, and acknowledge that you understand how much of her time this must take up.

Then you make your treatment decision, in partnership with her! The best case scenario will at times be simply providing the client with a bottle of well-chosen shampoo, to be used at appropriate intervals. You may perhaps ask whether her neighbour may be able to assist with shampooing at times, to ensure that the 10-minutes lathering period is complied with.

In this scenario, client goes home, and Shirley Next Door asks how she went. Client details how great you were, and how understanding of her circumstances. Client asks Shirley whether she could give her a hand at times, but says that she reckons she can stick to the recommended shampooing program.

On some days, that second scenario may well be your best work – getting things right for people, and all being well making something of a difference to the patient too. I would encourage you to learn and understand that that is OK. It is how things work in the real world – there are loads of things beyond your control, and your mission is to do the best which you can, in the prevailing circumstances.

Get very good at euthanasia

This is often what we need to be best at.

Talk to clients in advance about the procedure and process – is it their first experience of Euthanasia? And, especially if it is their first experience, talk them through what happens, and what may happen. Then ask whether they have any questions prior to moving into the task.

Look at all of your logistics first! How is the patient lying, what is the access to your preferred vein, etc? If you apply a tourniquet, will you be able to get at it to release it? Work out all of the ergonomics, and be as well set up as you possibly can, prior to attempting venepuncture.

Remember that some patients who may not have moved significantly for hours or even days, may still react non-usefully to having a needle prick their skin.

For Sunday morning on call jobs, when you have no nursing assistance, you may be presented with a very elderly skinny and partly demented cat, with euthanasia indicated and required. This class of patient can be difficult to handle without skilled assistance, especially as they typically start pawing at you constantly whenever you do anything at their front end.

A useful tip: most of these such patients can be toned down a bit by simply placing a towel over their front end, and encouraging them to remain in lateral recumbency. You can manually raise a kidney to an immediately subcutaneous position, and quickly with your other hand slip half a mL of Euthanasia solution into the kidney. This can be very surreptitiously done, and usually works very quickly. I usually tell a client that I have a 'special spot' for giving the drug.

Other stuff:

Be aware of the Cascade effect

This is much more relevant to L/A work in the field, and typically having less in the way of trained assistance than when in the clinic.

What I mean by Cascade effect is: What if this very unlikely thing happened as a result of what you are doing, then what might happen in consequence of that development? I think that this cascade is three-tiered i.e. what if that happened, then that happened, then that happened? I think that you need to plan large animal procedures this way. Remember also Murphy, who especially with L/A work lurks everywhere.

In fact, it may be best to consider this concept as the Murphy Cascade potential.

As an example, cancer eye story: You are in your new job, and the Boss finally has enough confidence to send you out on a farm call by yourself, at some distance from the Practice – say 45 minutes' drive away. The job is to attend a cow with a likely Eye cancer, and to pregnancy test 25 cows.

So off you set. When you arrive you are very relieved to find that the facilities are very good – modern crush, roof over it, etc. The cow with the eye problem has been drafted off and is in the feed-in yard at the top of the race. It is clear to even your inexperienced self that her eye is likely to require ablation. The cows for testing are in an adjacent yard.

So you think to yourself: 'That's good, I will do the clean job first – I would be really concerned about whether I could clean myself up thoroughly if I got manure on myself prior to doing any eye surgery.' You then get the cow baled up; she proves somewhat flighty, so you decide to give her just enough sedation to steady her up (choice of drugs is not a necessary detail for this story), you put

gloves on, and competently inject into her tail vein – keeping hands nice and clean.

You then move back to the head, shave the surgical field, apply your skin prep.

You start to inject Lignocaine at appropriate sites, and then the cow starts to wobble a little bit, and goes down. 'That's a bit of a bugger', you think – you check that her respiration is not impeded by the position in the bale gate – all is fine. The client asks you whether the cow went down because some of the injection you were administering – i.e. the Lignocaine – went into the wrong place. This takes you slightly aback – you know that this is probably not the reason why – so you quite reasonably suggest that it is more likely to have been an effect of the sedation, while pointing out that with the dose you gave her it should not have happened.

You find that you still have access to the surgical field, so you continue your prep. You then proceed to ablate the eye, although this is difficult because you have to operate on your knees – but this is OK – you are young and supple!

You then administer whatever other drugs you think are indicated, and clean up really thoroughly – it is so important to create a good impression!

Then you and the client make conversation, which rolls along nicely for a while. You give the cow a bit of stimulation every so often, but 30 minutes later she is still showing no sign of rousing. You nervously check her breathing pretty frequently, while trying not to appear anxious.

Beyond this time, you are starting to wonder how much longer this is going to take. The client has run out of questions to ask about your background prior to coming here to work, and is getting a bit twitchy – mainly because the day is heating up, and he has a yard full of cows standing in the sun waiting to have access to the crush so you can pregnancy test them.

Eventually you have to call it quits – the cow is still down – there she sits, moderately dopey still, and going nowhere – so you pull the plug. You apologize for the way things have worked out, and head for home with your tail between your legs.

From this point on, the story may vary in its details.

You may or may not have copped a blast from the client for your perceived incompetence. By the time you get back to the clinic, the client has been on the phone to your boss to fill him or her in. The client may or may not have been abusive to the Boss.

The client may or may not wish to ever have you on the place again, but in any case the client books the Boss to come out at some stage and pregnancy test the cows. This will be an uneconomical job for the practice, because the Boss will decide that to charge travel for the second trip would be a waving a red rag at a bull – so the boss will have to do 1.5 hours driving at no charge, and the sole fee will be for about half an hour of professional time.

So, in the field, think everything through in advance of committing to an action.

In that example, look at the day, look at the job – consider these 'What Ifs?' – then decide what gets the priority – in this case the 'Many' should get the priority over the 'One' – for reasons which I hope are obvious. So don't get hung up about your capacity to thoroughly clean your hands and arms after doing preg testing!

- **Remember that as the attending Clinician, you are in charge – in charge of everything – it is all your responsibility.**
- **Consider Patient safety, Client safety, Your safety.**
- **Always make very diligent efforts to Control the Controllable.**

How far from that tree/fence/building are you going to anaesthetize that horse – what are possibilities for occurrences during the induction process, and during the recovery process?

Some more general specific points:

Symmetry is your friend – always use it to your advantage. Lymph nodes, eyeballs, palpebral fissures, muscle masses, head tilt, lip droop, nostril position, ear positions.

To improve the acuity of one of your senses, shut another sense down. E.g. if it is safe to do so, close your eyes while auscultating with your stethoscope, or when palpating.

When intubating an airway – placing an endotracheal tube – do not develop tunnel vision: develop the awareness that you are passing through and beyond a whole lot of anatomy while you are carrying out this

procedure, in order to place the tip of the endotracheal tube through the laryngeal os – so make a point of examining this anatomy as you go past!

Hard palate, palatal aspect of upper teeth, tongue and ventral to tongue – the day you fail to look under a dog's tongue may be the day when you miss the squamous cell carcinoma lurking there – it happened to me many years ago.

Pharynx, tonsils, soft palate, epiglottis, arytenoid cartilages. Then intubate. Looking beneath the tongue takes 2.5 seconds, doing the complete assessment takes approximately 6 seconds. It's time well spent.

Learn about canine scrotal ablations – the indications for this, and techniques. Learn how to assess the canine scrotum very critically, in advance of operating on it. You will only need to deal with one case where ablation should have been done, to learn that this particular piece of advice is absolutely worth accepting at face value!

A scrotum the size of a cricket ball, covered with painful cracking and possibly necrosing skin, and a patient who seemed so nice prior to castration now wanting to repel you at all cost.

If you are wondering how to handle such a case, my advice is 'Very Gently'.

Prior to canine castration, in every case, assess the scrotum – is the skin thin, and how will the scrotal tone be once the scrotum is empty? Always ask yourself – in every case - should I or should I not ablate this scrotum? If you find yourself undecided, then always opt for the affirmative. You will never be wrong, provided that you leave sufficient skin to complete the closure.

Anal sacs: I think that anal sacs get a bum rap! I think that too often our fingers go into canine anuses without adequate justification.

When a patient is presented for anal sac evacuation, do a thorough examination of the skin, at least. Examine the pinnae, the feet, the chin, the lip folds, the vulva and the perineum. Look for subtle changes, and don't hesitate to do tape preps – including of perianal and perineal skin. In those patients upon whom you discover Malassezia on the perianal skin, regular use of an appropriate shampoo may greatly reduce the requirement for digital disruption in the future.

While on the subject of anal sacs – when expressing them, use a cross-handed technique i.e. left hand for Right sac, and vice-versa. Years ago I discovered a new grad causing a great deal of patient discomfort by inadvertently blocking the duct opening while squeezing – advising a cross-hand technique solved that problem, and it is much more ergonomically satisfying. It does double the glove requirement, but I think it is worth it.

"We've been using the digital system for years."

Ambidexterity is very easy to cultivate and develop – think of all the things you do with a dominant hand: washing your hands, scrubbing your hands, brushing your teeth, drying your toes, putting your legs into pants. Think about how many repetitions of these basic manoeuvres you do. If you take it on, I promise that you will notice a big difference in both strength and coordination within 3 weeks. This development will be of general usefulness, but will also be good for your Brain.

Always read previous history for the patient in front of you.

Look for trends in weight etc. Ask client whether the patient fully recovered from the previous presenting complaint, even if that was 12 months or more ago – there will be times when that bit of information will prove very useful.

You can diagnose HyperAdrenocorticism this way – the patient may have been presented previously for ear disease, dermatopathy, bacterial cystitis, mobility disorders etc each on separate occasions, so a history review may prove very useful.

I know that you all have young eyes, but always use a bright light and magnification – it is never inappropriate to do so! You may be surprised by what turns up.

Eye injuries in horses especially: seek the opportunity for an examination in low light. Either drag your heels in getting to the job, or go out as last job for the day, to try to organize some subdued light. Get things in your favour, so as to assist in not missing details. Alternatively, do not be afraid to book a second appointment soon after the first, to permit a more comprehensive assessment.

Remember – you are in charge of the case!

Horses: develop the confidence to refuse to perform lameness examinations on uneven surfaces. I am not talking here about patients 4/5 lame on one leg, but those where things are way more subtle.

Why submit yourself to unnecessary pressure? Work on having the confidence to tell a client that a venue is unsuitable, and that another venue will need to be arranged.

Dogs: Remember that the hip bone is connected to the thigh bone, and the thigh bone is connected to the knee bone, and the knee bone is connected to the leg bone, and the leg bone is connected to the ankle bone, and the ankle bone is connected to MANY FEET BONES, and the feet bones are connected to a nail bed and a claw/nail.

So: do not take radiographs on spec of the upper limb, for a negative finding, to try to find a lesion whose presence you have not been able to demonstrate clinically, ***WITHOUT first examining the distal limb.*** You may discover later that the patient has an interphalangeal joint lesion, or a phalangeal or metacarpal/tarsal lesion, or an atypical Pododermatitis, or a Paronychia, or a partly-avulsed but non-displaced nail.

My point: examine all of the relevant anatomy! If there is lameness, examine the complete limb, in complete detail. And do not omit to palpate the popliteal lymph nodes, and compare them using the Symmetry to which I referred previously – if the LN on the lame leg is enlarged, it behoves you to examine the distal limb in very fine detail, before you spear off and radiograph the upper limb!

Periodontal elevators: this instrument can make or break you! They are like 'the little girl who had a little curl right in the middle of her forehead' - when they are good they are very, good, but when they are bad they are HORRID!

Regardless of one's level of experience, this is potentially a very dangerous implement.

Try to ensure that you have access to good ones – and do whatever it takes to keep them sharp, by which I mean with an edge which is correctly angled. There are correct and incorrect ways to sharpen them – I would encourage having sharpening done professionally.

While preparing this presentation, I was provoked to contemplate quite a bit of stuff. One of my contemplations was specifically about Periodontal elevators.

I have decided that these are such a make or break instrument that I would encourage new grads, when attending a job interview, to examine the practice's Periodontal elevators – their state may be a useful indicator about the practice's attention to detail generally. This may be an extrapolatable indicator for many other aspects of the practice.

If you are in a practice already, it may almost be worth going on strike for – because you do not have much of a fall-back position available to you if you have shot an elevator into the deeper structures of the patient's head, due to the elevator's not having an edge which is engageable in the periodontal ligament.

The risks from using dodgy gear are too great.

In other words, when you are in a practice, attention to some of these details not only serves to protect you, but will in an ideal world provide Leadership to those around you.

Other stuff

When speying cats and dogs, always double-check that you have removed BOTH OVARIES and an appropriate amount of uterus – I have some stories about such omissions – always double check prior to closing the abdomen.

Similarly, when performing Caesarean sections in all species, check from horn tip around to horn tip, and check the uterine body and the vagina, to ensure no foetus is left therein – it has happened – I have had new grad employee experience with both of these procedures.

A big point: **do not worry about getting to become quicker at doing things**. Be mindful – just concentrate as closely and fully as you can – work on simply becoming very good at every various little thing you do, and ***IMPROVED speed will follow*** as surely as night follows day.

Applying mindfulness will result in giving you a capacity to break processes down into their component parts.

For example: closing an abdomen – you need to ensure that you are bringing your complete attention to the placing of every suture, whether you are using an interrupted or a continuous pattern. Do not dwell on how far along the closure you have come, or how far you have to go – simply focus on every suture in its turn. Ultimately, you will find that there is no room to place another suture – hence, the closure is complete!

I guarantee you that if you use your mind in this fashion, skill levels and speed will accrue rapidly!

You will not know if you don't look! You will all have heard

that before, and it is surely true. It is simply part of being thorough.

Be thorough, be determined – keep asking yourself 'What am I missing here?!' The correct answer may not in fact be the obvious one e.g. the cat may prove to be FIV +ve – but is that the cause of the clinical problem right now?

Always take the correct number of radiographic images for the presenting problem or for the relevant anatomy. You should not need me to tell you this – just do it.

Be prepared, especially for L/A calls. Do a bit of research based on the client *description* noted on the appointment schedule; look information up, ask some advice in advance from a more experienced associate.

Do a check-list of stock and equipment in the car before you set out. This will ALWAYS be time well spent – especially if you share a work car with a colleague. E.g. for a calving, talk yourself through the procedure in advance: I will arrive, I will assess the general health and the gait and mobility of the calving cow herself, I will assess degree of straining - so Epidural required or not?

Therefore, do I have sufficient fuel for the round trip, do I have Lignocaine, Lubricant, Clenbuterol, chains, traction device, etc etc?

Having controlled the Controllable, stuff will still come at you out of left field, because we cannot control the Uncontrollable.

So, learning about how to do great work is great, but is not as nearly as great as showing courage in the face of adversity. Learn how to stand up and take responsibility for each of your outcomes, and be prepared always to investigate further, if you can – so that you can learn from the situation.

For example, if circumstances appear appropriate, seek permission to carry out an autopsy to assist your learning – but then perform it fully, comprehensively and thoroughly.

In my opinion there is rarely much benefit to be had from such a procedure as a 'partial autopsy' – consider how much you may miss – not only in the cadaver with which you are dealing, but by way of building up more 'normal' to be applied to future cases. Lay the cadaver out, open it in accordance with standard techniques, appraise the full field of view, and proceed from there.

Be prepared to seek advice from Specialists – we are very fortunate to have ready access to many people who donate us their time. So don't take them for granted – send a Thank-You card – remember that these people are not simply sitting around waiting for you to phone in with a cry for help!

Humour: this is a biggie! Humour tends to save us, often.

It's so much more positive than whingeing and complaining. It's not unethical IMO to have a laugh about patients and clients, provided that we do it with love and kindliness.

Only lately I dealt with a client who expressed concern that the dog was at risk of developing 'severe emancipation'...

My son suggested to me that the dog might be about to slip its collar...

Job seeking

Number 1 tip – BE YOURSELF – relax into this.

Be prepared to relate to your job prospect at an emotional level, because getting a position is about the 'Vibe'. As an employer, what may well matter most to me is 'how will this person fit into our group?'. So just be yourself.

Look for evidence that the Boss/Practice has an interest in helping you develop as a person and as an individual – as well as a clinician. Look for evidence that the employer displays a genuine understanding of the challenges faced by new grads.

One of my items of feedback from a former new grad employee was that I had suggested to her that I was OK with her pulling up to enjoy the scenery for a few minutes while on the way back from farm calls. On balance I would expect that that process would be more likely to have only positive outcomes for all concerned – i.e. for the employee and for the Practice.

Look for evidence that the Practice has a well-thought-through induction process for new grads.

Sniff the wind – do the nurses and receptionists appear to have a good rapport between themselves?

Dare I say – Check the state of the Periodontal elevators!

Be prepared to leave a bad job e.g. where you feel after a decent period of giving it your best that you do not fit in, or that you find certain aspects of the situation simply intolerable. The decision to leave can only ever be your own and should be based upon your feeling compelled to be somewhere else, and while knowing that you have given things a good crack, and not simply quit and run.

You will need confidence that you will be able to find a position which is better for you, and confident that the fact of having left the previous job will not count against you in the future.

The risk attached to remaining in a position which you find noxious is that the detrimental effects may prove so demoralizing that you leave the profession.

Some thoughts in summary

I read the following about Leadership recently.

> Leaders must pursue change because they believe in their hearts that it is the right thing to do. It will help them become a better leader, team member, family member – and by extension improve the lives of people in their immediate orbit. It will help them live the values that they believe in. Getting better is its own reward.

A quote from Buddhist monk Thich Nhat Hanh:

> If while washing the dishes, we think only of the cup of tea which awaits us, thus hurrying to get the dishes out of the way as if they were a nuisance, then we are not 'washing the dishes to wash the dishes'. What's more, we are not alive during the time we are washing the dishes.
>
> In fact, we are completely incapable of realizing the miracle of life while standing at the sink. If we can't wash the dishes, the chances are we won't be able to drink our tea either. While drinking the cup of tea, we will only be thinking of other things, barely aware of the cup in our hands. Thus we are sucked away into the future – and we are incapable of actually living one minute of life.
>
> So don't do any task in order to get it over with – resolve to do each job in a relaxed way, with all of your attention. Enjoy and be one with your work.

My final remark – which is the thread running through all of the afore-mentioned – is: just be thorough – it will never let you down.

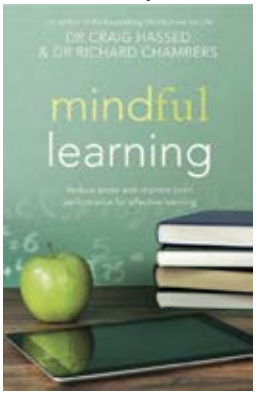
Thank You for reading.

Postscript:

Having listened to the combined presentation by James Bennett and James Carroll (at the Recent Graduate Survival Seminar) I agree with a fair bit of what they said, but with regard to After-hours cases, there are obviously distinct demographic differences.

In our practice, we encounter very few jobs which are not justified as emergencies. The range of true emergencies is the same as for most mixed-species practices – Ixodes intoxication, Calvings, colics, dog/cat trauma fights/ MVAs etc; swellings which were observed by client yesterday and are much bigger today (hence a justifiable emergency case IMO) and which prove to be abscesses; snakebite, haemorrhage due to ruptured splenic neoplasm, pharyngo-oesophageal obstruction (Choke) in horses...

A useful book is ***Mindful Learning*** *by authors Craig Hassed and Richard Chambers. Craig Hassed is, amongst other things, a GP and an Educator at Monash University's Medical faculty.*



'Why is it so hard to find a vet?'

Mario Viscardi

Ballantrae Drive Veterinary Clinic
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C&T

I have been speaking to a number of vets around my area and got some 'gossip' from others down the south coast.

The general feedback, including from myself, is that we cannot get vets to work in general practice, in particular full time, followed by part time and locums.

I have advertised through an agency, and in the last 2 years not one single vet has replied to the ad!

Kookaburra locum agency has sent me an email informing me that I could apply for an overseas vet if cannot get an Australian one.

Two years ago I had a Danish graduate who had an excellent CV, was very competent and tried the COPQ test twice, first time failed the pig session, passed all the other ones, was told to redo the whole thing, she paid $3000 for it, redid the test, failed again, total cost $6000.

She is married to an Australian, went back to Europe, and started a successful career as a vet there.

Now I am told that I could ask her to come here and work with a special visa for up to 5 years.

In the meantime I could employ a vet from countries where the degree is not up to standard?

The last 2 locums I was lucky enough to have for a week each had somewhat limited experience, but would not accept anything less than $60 an hour—one has 1 year experience and cannot do surgery past castrations without supervision.

They told me that they prefer locuming as the pay is much better and they can pick and choose where and when to offer their services.

Bottom line is, if they keep doing that, is very unlikely that they'll improve their skills.

Also, one had to be paid as wages, that means fill up an employment form, pay superannuation, follow through with the accountant for group certificates, the Supa fund etc. If you want to be a locum, you should have a business number and charge your fees through that and add whatever for the missed superannuation. Tried to explain that but got nowhere.

So, my questions are:

- What happens to the hundreds of new graduates each ear?
- Why so many do not want to start a proper career in the job they chose?
- Has this anything to do with the high number of female graduates? (Dear oh dear, I dare ask the question)

I heard many complaints from the PC group re unfairness when male workers are a majority, dead silence now.

I do need to understand though, because an increasing number of us are finding life as a vet really tough.

Are Universities or the Vet Surgeons' Boards aware of this situation?

And if yes, what are their plans to ease this block?

Comments courtesy of:

1. Gail Versluis

Southern Tablelands Veterinary Hospital
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e. southerntablelandsvets@gmail.com

We have been advertising for a replacement vet for over 18 months with very little response. We have employed quite a lot of new graduates over the years but in recent years we have had very little interest in the position from new grads. We are in a rural city, small animal only and doing our own afterhours. We have a new purpose built clinic with good facilities and support staff. So some good points and some not so as far as a new grad would look at it. Looking at the numbers of available jobs we are not the only ones struggling to attract applicants.

Why is this when the numbers graduating have increased?

One theory is that new grads are in no rush to work as they are now all from privileged backgrounds (as they are the only ones that can afford to go to vet school) and therefore don't need to put themselves out to actually work.

Another theory is that the reality of the work is also putting them off working as a vet in GP practice as they go into the course with unrealistic views on what the job is about.

I can give you an example of a new grad who was doing a tour of Eastern Australia looking at different practices advertising for a new grad. We talked to her, she had been

graduated for over 8 months, had only done volunteer work as wasn't ready to commit to full time work yet. She was at that time about to travel overseas to do a yoga course for several months, after that she would then consider working as a vet somewhere. Admittedly only one case and may not reflect majority of new grads' attitudes.

Are there numbers on:

- How many new grads actually end up in practice?
- How many are registered compared to the numbers graduated?
- Educational background i.e. How many come from public schools compared to private or selective schools?

The high number of full fee paying students from overseas may also be contributing to some of the problems as these students don't usually end up in practice in Australia so is this artificially inflating the number of new grads or has this been taken into account?

As to what could be done, perhaps the change to the DVM may already have changed perceptions of new grads? Time will tell for that.

Is there enough bias to select students who may have come from less privileged backgrounds? With selection based on both marks and interviews? As per Wagga Wagga.

Is there a place for some kind of sponsorship/cadetship with financial support whilst at vet school with an obligation to go into the workforce upon graduation?

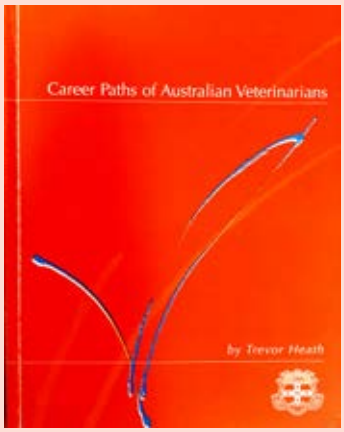
2. Mark Baldwin, Veterinarian

Over the past 10 years 1 have been employing veterinarians, there has been a noticeable move from full-time employment to either part-time or locum. This is, in my opinion, due to a number of factors:

1. Most veterinary school intake is from city-born or overseas students. This has led to a reluctance to work full-time in either regional or outer metro areas. No graduate vets want to work after hours as the majority of city practices have access to after-hours emergency / overnight care. This bias is very much encouraged by the teaching hospitals. It is 'not the gold standard' to operate any other way.
    - Large veterinary groups are constantly needing short-term locum stints thus escalating hourly rates. The dearth of locums has led to very inexperienced vets being able to negotiate exorbitant rates and not want/have the need for full-time employment.
    - A vast majority of graduates are now female.This is an increasing trend over the last 10 years, the last figure I saw was over 80%. This has led to a loss of experienced 5-10 year vets to the industry completely due to child rearing, or a preference for a part-time return to the work-force.
2. The intake into the veterinary science course is heavily biased to academic achievement over practical skills (students are accepted purely on marks with no interview [except at CSU Wagga] and very little onus on previous practical /interpersonal skills).
3. An increasing number of graduating vets are moving away from practicing into either academic research or working in industry roles.
4. A generational change in employment: veterinarians graduating now do not envisage working at a practice for a number of years then working their way to partnership/ownership. The increase in large group activity has left very little aspiration to own a practice. The option to partnership is rarely available, so locum work is more attractive than slogging away with no prospect of advancement past 'senior employee'.
5. The locum rates available will exceed even a senior veterinarian's wage and has far less practice management responsibility.
6. There really are not any good sites /agencies that actively source or promote veterinarian employment; Kookaburra is merely a list that you 'hope' people look at.

Editor's Note: In 2001 the CVE published *Career Paths of Australian Veterinarians* by Emeritus Professor Trevor Heath from the University of Queensland. It was based on a longitudinal study of veterinary students who commenced study at the UQ in 1985 and 1986 and who completed 5 questionnaires over the next 15 years as well as other questionnaires completed by the profession. It covered many of the questions raised by Mario, Gail and Mark. Perhaps it is time for another comprehensive study to address the important points raised in Prof Heath's book?

Four-Words, Forwards! (Mental Health Workshop)16.10.19
After 5 years in the workforce, when questioned about stress and burnout, 80% of recent graduates felt that veterinary work caused them significant stress. Most who had changed from enthusiastic graduate to disillusioned veterinarian could point to stress-inducing problems involving lifestyle, support, reward or recognition that had quenched their idealism and diminished their energy and commitment.



2018 DISTANCE EDUCATION

Congratulations to the class of 2018!

Distance Education is demanding and requires dedication and commitment, especially when juggling study commitments with work and family.

Congratulations to you all for successfully completing a vigorous but rewarding year of continuing professional development.

CVE DE Tutors & Staff.

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Simone Herbert
Gregory Ireland
Rachel Kent
Craig Simon

Medicine of Cats
Wietz Botes
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Lan-Hsin Kuo
Anne-Marie Moody
Rachel Nugent
Leah Puk
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Jonathan Carruthers

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Adi Frisch
Xiaojia Lee
Caroline Romeo
Ailsa Rutherford
Benjamin Stewart

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Veterinary Radiology (Small Animal)
Katrina Garrett
Jason Lenord
Phanuel Mponda
Catherine Walsh

Veterinary Radiology
Travis Jayson

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* Reference Studies: World Health Organisation. (2009). *WHO Guidelines on Hand Hygiene in Health Care; First Global Patient Safety Challenge Clean Care is Safer Care.* Geneva, Switzerland. World Health Organisation Press.

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