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THE UNIVERSITY OF SYDNEY

Centre for Veterinary Education

Control & Therapy Series

Issue 301 | December 2020

'What's Behaviour Got to Do With It?'

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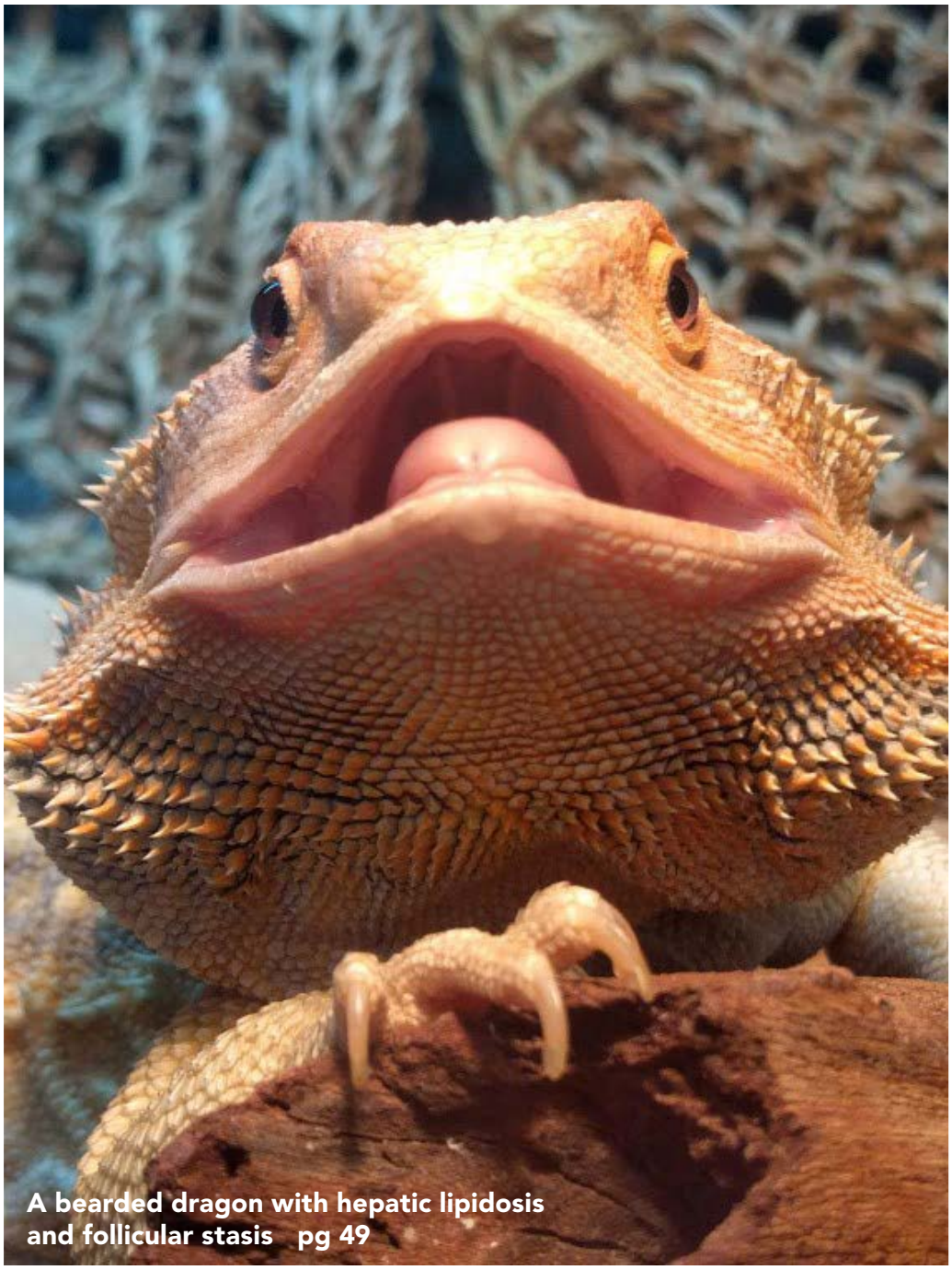

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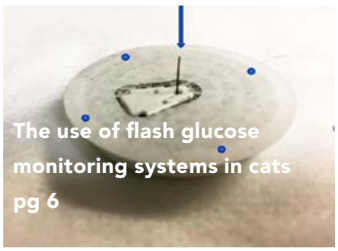

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Issue 301 | December 2020

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Australia's Leading Veterinarian Forum

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The C&T Series thrives due to your generosity. If you're reading this and have been contemplating sending us an article, please don't hesitate.

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

Join in—write up that interesting case

C&T authors agree that it is extremely satisfying to see their articles in print and know they are contributing to veterinary knowledge and animal welfare.

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Major Prize Winner

Winners

Prizes

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* Note: Print has been temporarily suspended due to the impact of COVID-19

FROM THE DIRECTOR

What a weird and challenging year 2020 has proven to be. As if recovering from disastrous bushfires and floods was not enough, to be affected by a global pandemic was something none of us needed. As I write this, Victoria has just emerged from prolonged lockdown and most of the Australian state borders have reopened or will be open again soon.

How many people outside of the medical and veterinary field had even heard of an epidemiologist before the outbreak? Now it is a household word, yet most people have scant knowledge of the principles of epidemiology and risk assessment. As veterinarians, we have been trained in disease surveillance. More than most people, we understand the implications of a virulent disease entering our country. I think we also have a much better understanding of the concept of herd immunity, so to me it has been frustrating reading and hearing the uninformed commentary spruiked by some journalists and commentators about how the disease should have been handled.

It will be interesting to reflect in a few years' time on the pandemic as a whole. Nothing seems more certain to me than the likelihood of further emerging diseases, whether of lesser or greater virulence. Hopefully we will have learnt a great deal from this current experience which will help us in the future.

The CVE has expanded the number of online courses on offer due to the inability to hold face-to-face conferences and workshops. Yet, we are still frustrated by the ongoing uncertainty about when we will be able to recommence in 2021. As soon as we know more, we will let everyone know.

This will be my last editorial as Director of the CVE as I will finish up in this role in March. It has been an absolute pleasure leading the CVE team over the last 14 years and I have enjoyed meeting so many of my colleagues and developing many new and lasting friendships.

I hope that everyone has a safe and enjoyable festive season and that there is no major resurgence of the virus before safe and effective vaccines are released.

Rather than saying goodbye, I much prefer to say *au revoir!*

Dr Hugh White
Director

THE UNIVERSITY OF SYDNEY
Centre for Veterinary Education

What's behaviour got to do with it?

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Let's find out!

Practical – Virtual – Collaborative

15-18 February 2021

Join us on a virtual behaviour event like no other – where one of Europe's most respected specialists in veterinary behavioural medicine Gonçalo da Graça Pereira will be joined by Australia's Kersti Seksel and a highly talented and experienced team of presenters from the fields of medicine, surgery, ophthalmology and dermatology.

cve.edu.au/behaviour

Stellar speaker line-up

Gonçalo da Graça Pereira (keynote)
Kersti Seksel (convener)
Debbie Calnon
Sarah Goldsmid
Dani Hoolahan
Jacqui Ley
Paul McCarthy
Sal Nixon
Isabelle Resch
Grace Thurtell

Large

Winner

REPRODUCTION EXAMINATION OF THE BROODMARE

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C&T No. 5856

The examination of the reproductive tract of the mare is a fundamental procedure that is different in some aspects to the procedure in cattle. It has an elevated level of risk to both the mare and the examining veterinarian and correct procedure is essential for accurate assessment of the reproductive status of the broodmare. It may be a daunting task for young veterinarians but with care and thoroughness can readily be achieved.

Preparation of the mare

It is essential for the safety of both the mare and the operator that the mare is adequately restrained. The level of this restraint will vary with the age and experience of the mare, but it must always be adequate. Young inexperienced mares are unpredictable and may object, often violently to the procedure, particularly to rectal examination. Also, the risk of rectal damage including rectal tears is highest in younger mares, particularly maidens.

Restraint usually includes the use of stocks or a crush. The mare may need sedation and/or the use of a twitch. In the absence of a crush it may be necessary to examine the mare against a wall or gate. In this case the mare is held on her near side by the handlers, one at the head and one at her near side hip holding the tail to one side. Some advocate examining the mare over a gate or over the barn door but this is not to be recommended. Veterinarians have been killed or severely injured by a mare kicking over the door, or going down while the veterinarians' arm is inside the mare.

To young veterinarians first starting out, I recommend they develop the use of their non-dominant arm to palpate the mare. This has a number of advantages; firstly, from a practical viewpoint if the left hand is used around a door with the veterinarian safely to one side, the handlers are on the near side of the mare where she is used to having contact. In mare and foal paired crushes, the foal crush can be on the off side of the mare (handlers are on the near side) so the foal is then in the view of the veterinarian. It is unlikely then for it to pass through the examination area without at least some cursory glance that might draw attention to any early problems.

It is also good to develop sensitivity in the relatively unused left arm. (Some also say it develops the side of the brain used for perception and pattern recognition but I think from my point of view this is conjecture). However in practical terms it does leave the right hand free for manipulation of equipment and machinery such as tuning the ultrasound including recording views, and handling semen and treatments. This is all becoming increasingly important as technology increases.

All of these points become more difficult if you are a right-handed examiner.

The examination

Mare examinations are often described as 'follicle examinations' which is a misnomer as the examination should be of the whole reproductive tract (plus a general assessment of the bodily condition and general health of the mare).

Figure 1. James performing a reproduction examination

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Firstly, a visual appraisal of the perineal region is made. Has the mare got an excessively sloped vulva with loss of perineal tissue—a serious contender for infertility by vaginal contamination? Is the mare likely to be a windsucker that requires a Caslick's procedure? Is there a vaginal discharge or evidence of infection such as coital exanthema?

The rectal examination can then proceed. A plastic disposable glove should be worn and a short latex glove on the other hand is good hygiene practice. The hand with the rectal glove should then be lubricated. It is advisable to liberally apply lubricant to the arm and back of the hand where it will be in contact with the rectal wall, rather than the palm of the hand. Bear in mind that rectal tears usually occur in the dorsal part of the rectum—the part in contact with the back of the hand.

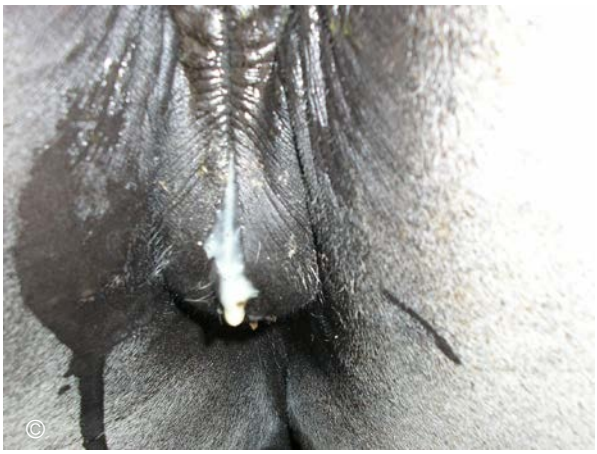

Figure 2. Mare with purulent vaginal discharge

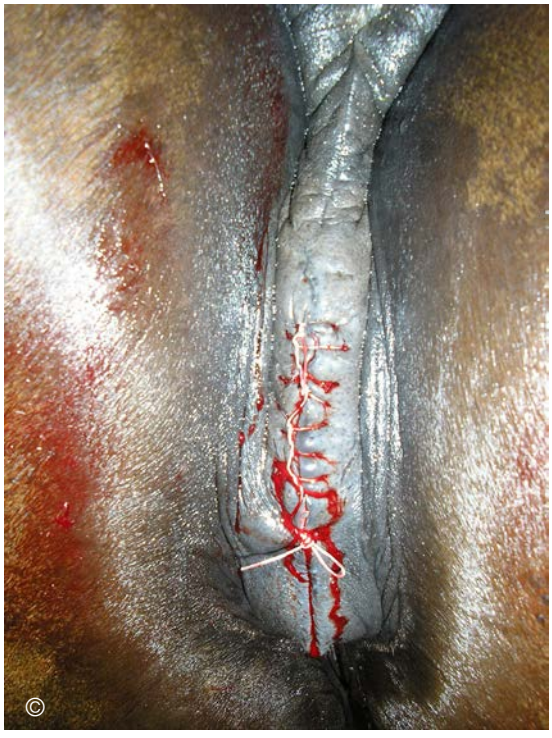

Figure 3. Caslick's operation

In fact, the so-called rectal tears are usually in maidens or young mares and are often a longitudinal split along the roof of the rectum and are associated with tension of the tightening rectum. For this reason, it is essential the mare be relaxed during the procedure and the arm is introduced gently taking as much time as necessary. This procedure is quite unlike that experienced in cattle work.

Having lubricated the back of the hand the rectal ring can be lubricated by rubbing the ring with the back of the hand then introducing the fingers, in a cone shape, gently in to the rectum. This allows the sphincter to relax.

Travelling inwards in a sweeping motion using the back of the hand to expand the rectum and removing faecal balls, the hand can move forward to the brim of the pelvis. At this stage the first assessment can be made. Brushing the side of the hand across the floor of the pelvis, the cervix can be identified. Is it relaxed or firm like a sausage? If the mare is pregnant it might be long and firm like a pencil.

Having identified the cervix it is then quite easy to move the hand forward, palm downwards, to identify the uterus. The uterus of the mare lies across the pelvis, making a 'T' with the cervix. Are you feeling the uterus or a transverse loop of bowel? If it is the uterus then sweeping the hand from side to side with the structure cradled in your fingers will bring you to an ovary at the end of each uterine horn. In maiden or young mares the ovary will be high in the pelvis on each side whereas in older mares it is usually lower, often at the level of the hip.

You have now identified the mare's reproductive tract and can make some evaluations.

In mares in oestrus, the uterus is soft and relaxed. The cervix is also relaxed. In dioestrous, the uterus is firm and the cervix is firm. In early pregnancy the uterus is usually very firm as is the cervix. In pregnancy, sweeping the hand along the uterus using cupped fingers rather than actually feeling or pressing the uterus, a filling or bulge can be identified, that being the developing pregnancy. Don't squeeze but just run it past your fingers to confirm the swelling. You can then estimate the age of the pregnancy by the size of the bulge. Later pregnancies will be further into the abdomen but pregnancy diagnosis is a further subject.

Once you have become competent with these procedures you can be let loose with an ultrasound scanner to confirm your findings. Preferably not before, if you want to get the best of your skills.

Small

THE USE OF FLASH GLUCOSE MONITORING SYSTEMS IN CATS

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Continuous glucose monitors (CGM) are great for hospitalised cats requiring intensive glucose monitoring and for previously diagnosed diabetic patients. These monitoring systems are particularly useful in cats where frequent blood sampling can be difficult and patients can be discharged with sensors for assessment of glucose trends in their home environment.

The FreeStyle Libre (Abbott) is a flash glucose monitoring system which measures interstitial glucose every minute and stores data for up to 14 days. The system has been validated in dogs but not yet in cats (S. Corradini, 2016 Jul-Aug). In dogs, good correlation between interstitial glucose and plasma glucose concentrations has been demonstrated and it was accurate at low, normal and high blood glucose concentrations. The monitor has also been evaluated in 14 dogs with diabetic ketoacidosis (DKA) (Malerba, *et al,* 2020). It demonstrated clinically accurate estimates of blood glucose. Acid-base status, beta-hydroxybutyrate, lactate concentrations and body condition scoring did not influence sensor accuracy.

My experience with this system in cats is positive, but the system has limitations — as would be expected in a monitoring system designed for non-haired, human skin! Sensor application can be tricky, particularly in patients with reduced body condition scores.

In one study of older continuous glucose monitoring systems, placement of the sensor on the lateral chest wall, the dorsal neck or the lateral knee fold positions were compared (Hafner, Lutz, Reusch, & Zini, 2012). Needless to say, the lateral knee fold application was not successful. This preliminary study suggested that dorsal neck placement may be superior to lateral chest wall; however, only a small number of cases were in each group.

I find it easiest to clip a square of fur and apply the sensor to the left or right lateral chest with a small amount of superglue or tissue glue applied at four quadrants on the sensor. The sensor should be placed away from insulin injection sites. Point colouration breeds (e.g. Siamese) may develop a change in coat colour at the site of superglue application.

Once the sensor is in place, no dressings are applied as these seem to contribute to migration of the sensor, shorter reading times and also attract the cat's attention to the sensor. Cats do not appear to be bothered by the sensor and Elizabethan collars are rarely necessary.

The Freestyle Libre sensor does not require additional blood glucose measurements for calibration and the sensor is suitable for 14 days of use. Average sensor time is approximately 5-10 days. Once a sensor has been scanned by a specific reader, it can only be read by that reader. Large amounts of data can be obtained and are a useful guide for assessing the status of diabetic

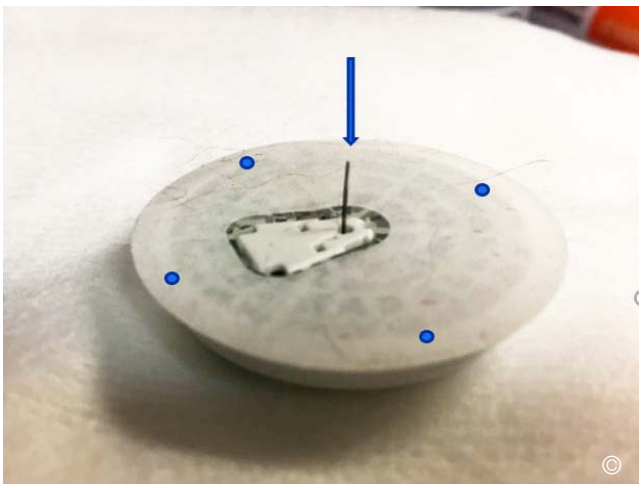

Figure 1. Underside view of a Freestyle Libre sensor. The blue circles represent sites for placement of superglue to hold the sensor in place. The blue arrow denotes the thin, flexible, sterile fibre that sits under the skin.

Figure. 2. Location of the sensor on the left thorax wall. No dressings are applied over the sensor

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patients. It is important to note that aiming for tight glycaemic control based on sensor readings can become problematic and I advise owners not to make dosage adjustments based on readings without discussion with a veterinarian first.

Anecdotally, although interstitial glucose readings appear to calibrate well with blood glucose readings in most cats, there are some cats where discrepancies certainly occur. It is important that additional monitoring tools such as body weight, condition score, appetite and water intake are still used to provide a thorough assessment of the patient before ANY dosage alterations are made.

CGMs are also useful for following trends in non-diabetic patients such as septicemic patients where hypoglycemia is a significant risk and in known diabetic patients undergoing anaesthesia (e.g. dental procedures). There is likely a delay in equilibration between blood glucose and interstitial glucose which is reported to be 9-20 minutes with other systems (e.g. Guardian Real Time). For patients undergoing anaesthesia it is useful to place the sensor 24-48 hours prior to anaesthesia to facilitate readings during the procedure.

In Australia, readers and sensors can be purchased online directly from the wholesaler: https://www.freestylelibre.com

The cost of a sensor is approximately $AUS 95 and a reader is also $AUS 95. Smart phones can also be used as readers.

At our hospital, we stock a number of sensors and readers but advise clients to purchase their own sensors and readers online for longer-term use. We apply the sensors for the owners and interpret the data for a fee. Removal of the sensor is generally not an issue. The cats can be left to remove the sensor themselves, or it can be removed briskly following application of some nail polish remover. Alternatively, if a large amount of glue has been applied, then give the coat time to grow until clippers can be introduced under the sensor to aid in glue removal.

I find sensors invaluable in monitoring brittle diabetic patients and diabetic patients that have a high likelihood of entering remission (e.g. those with concurrent disease such as pancreatitis or who have received diabetogenic drugs such as dexamethasone); however, data obtained must be interpreted together with traditional monitoring tools such as body weight, condition score, thirst and appetite.

eBook download
The impossible diabetic

Figure 3. Sensors are typically well tolerated by most cats

Figure 4. Example of data obtained from a freestyle Libre sensor. The patient received insulin on Thursday evening prior to sensor application. The data demonstrates an initial overswing of interstitial glucose followed by an eventual period of euglycemia and the patient has entered diabetic remission. Note that readings become erratic by Tuesday afternoon and the sensor had stopped working on Wednesday and Thursday.

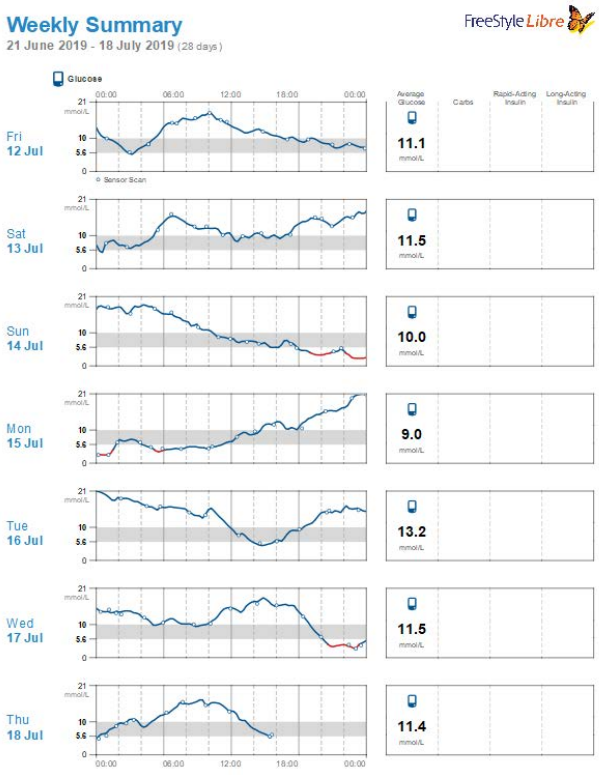

Figure 5. Sensor readings from a newly diagnosed diabetic cat receiving 12 hourly glargine injections at 0700 and 1900. This patient had received an injection of dexamethasone prior to being identified as diabetic and subsequently went into remission. The data identifies periods of hypoglycemia which were asymptomatic and not uncommon with glargine.

MAJOR PRIZE WINNER –
for pulling through a complex challenging case

UVEITIS IN A CAT WITH LIPAEMIA AND PANCREATITIS

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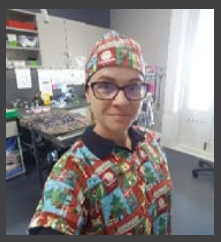
lisajmorrison18@gmail.com

C&T No. 5858



Lisa graduated from Melbourne University in 2003, and went straight into practice working as a small animal vet in small animal and mixed practices in Northern Tasmania and the UK. She enjoys the variety that being a vet can bring to the working day, working with like-minded people, and working with animals (particularly dogs, cats and rabbits). She currently lives with her husband and two children in Launceston, the same town she was born and grew up in. She has worked at the AMC since 2014.

Lilah is a five-and-a-half-year-old female neutered domestic medium haired cat with a nervous disposition. On 11th February 2020, she presented as an afterhours consult. She had likely been inappetant for 4 days and was depressed and lethargic. Lilah had only arrived in Launceston on the 8th February, after catching a plane from Brisbane. Two days prior to her plane trip, she had presented to her vet in Brisbane with a cloudy anterior chamber in the left eye. The right eye appeared normal. The vet diagnosed anterior uveitis and prescribed Prednefrin Forte® eye drops and Vibravet® oral paste. By the following day, the eye appeared completely normal, so the owners cancelled the planned revisit at her Brisbane vet.

On initial clinical exam on the 11th Feb, Lilah was found to be lethargic and dehydrated. She also appeared to be pale and possibly icteric. She was admitted to hospital and placed on IV fluids and given a Clavulox® injection. Blood tests the following day showed increased ALT, hypercholesterolaemia, increased bilirubin, hyperglycaemia, electrolyte abnormalities and a high white cell count. An abdominal ultrasound (performed in-house) showed a homogenous liver, and the pancreas was not readily apparent or visualised.[1]

Treatment was commenced for presumed cholangiohepatitis or hepatic lipidosis. This consisted of IV Hartmann's with added potassium chloride, a one-off dexamethasone injection, feeding of Hills A/d® via an oesophageal tube (placed under general anaesthetic), pain relief (buprenorphine), and antibiotics (Clavulox initially). Three days after admission, Lilah developed a temperature of 40°C, so enrofloxacin was added to her treatment regimen. Her temperature continued to oscillate between 39.2 and 40°C during her hospitalisation. Treatment with glargine insulin was initiated on 15th February, when it was discovered that Lilah had a persistent hyperglycaemia which was worsening (it was 22.7mmol/L on 15th Feb). We also commenced feeding Hill's m/d® mixed with Hill's a/d® (at a 50:50 ratio) and fed through her oesophageal tube at this time.

A week after admission to hospital, Lilah appeared worse overall. She showed only an occasional interest in food, was very depressed and stressed and had a persistent pyrexia. The vet in Brisbane had listed toxoplasma as a possible differential of the anterior uveitis on the 8th February and there was some thought that Lilah's current condition could be due to toxoplasmosis, so the Clavulox and enrofloxacin were stopped and treatment with doxycycline and clindamycin was commenced. She was tested for FeLV and FIV, and was negative for both.

Given Lilah's nervous disposition, it was planned that Lilah would go home to see if she improved when around her owners and familiar belongings. However, as her owners had removalists coming and going, it was decided that this would possibly be more stress for Lilah, and her owners elected for Lilah to stay in hospital until all their familiar furniture had arrived and there would be less disruption to Lilah once she did arrive home.

Maropitant was added to her treatment regimen on 20th February after she vomited overnight. Lilah was due to go home on 21st February, however on in-house bloods on this day, it was discovered that Lilah was severely anaemic, so it was decided to keep her in hospital another 24 hours and retest her haematocrit again the following day.

Lilah went home on 22nd February, after 11 days in hospital, with her feeding tube in place. Her PCV, whilst low on discharge, was showing signs

[1] Editor's comment: A pancreatic specific lipase would have been prudent at this stage – with the benefit of hindsight.

of regeneration and subsequent blood tests showed an increasing PCV.

She was sent home on maropitant, clindamycin and insulin (2 units in the morning and 1 unit in the evening). Clindamycin was continued for a total of 5 weeks. Her feeding tube was removed on the 6th March, and at this stage we also commenced weaning her off her insulin (as blood glucose spot checks showed low blood glucose). Insulin was stopped altogether on 6th April 2020, and the plan was to do a serum fructosamine 3-4 weeks later.

Lilah re-presented to the clinic on the 8th May 2020. Her presenting complaint was 'cloudy eyes again'. She was in good condition otherwise. Her temperature was 38.4°C. The diagnosis of bilateral anterior uveitis was made. Blood was taken for a toxoplasma titre, and it was noted that her blood sample looked very lipaemic grossly. The primary differential diagnosis at that stage was likely lipid anterior uveitis. It was recommended that Lilah be fed a low-fat diet and she was prescribed topical Maxidex and was sent home with clindamycin whilst awaiting toxoplasma titre results—these later came back negative.

Lilah's eyes were clear the next day, however she started vomiting and re-presented to the clinic. Her temperature at re-presentation was 39.8°C. Blood results showed high globulins, ALT, bilirubin, cholesterol and glucose, she had low phosphate, calcium and electrolytes. She was hospitalised and placed on IV Hartmann's and given a Clavulox injection.

The following day her temperature was 40.1°C. An in-house abdominal ultrasound showed an enlarged pancreas and surrounding fat that was hyperechoic, and the liver had the same homogenous appearance as noted in February, leading to a probable diagnosis of pancreatitis with secondary hepatic lipidosis.

On the 11th May a urinalysis showed glucosuria and ketonuria and she was started on glargine insulin again. She was sent home on the 12th May to see if she would eat overnight (being a nervous cat, she has never eaten for us whilst being in hospital, although she ate voluntarily the first day she went home after her initial presentation in February). She did not eat, so was readmitted on 13th May and an oesophageal feeding tube was placed. She had also become

Figure 1. Close up view of bilateral lipid aqueous. For a cat to have this condition, it needs both high triglyceride and/or cholesterol concentrations in blood, and a degree of uveitis (the latter lets the lipid leak into the anterior chamber). Without concurrent uveitis, cats like this will have blood that looks like strawberry milk, but clear eyes. You can also see the lipid in the blood in retinal blood vessels if the anterior chamber is clear. The cause of concurrent uveitis in this case is cryptic.

anaemic again with a haematocrit of around 21%, although red cell indices were normal.

While hospitalised, Lilah's treatment consisted of IV Hartmann's with added potassium chloride, buprenorphine, glargine insulin, maropitant and amoxycillin/clavulanic acid. Feeding via an oesophageal tube was commenced on 14th May with just m/d (due to the lower fat content compared to a/d and Lilah's concurrent hyperglycaemia). After corresponding with Dr Richard Malik, this was swapped to Fancy Feast Salmon variety on 15th May, due to the even lower fat content. Some Fancy Feast was also offered to Lilah on a spoon and she ate it voluntarily. Dr Malik also advised that lipaemia causes haemolysis in cats, and this was the most likely cause of Lilah's anaemia.

Due to Lilah being stressed in hospital, continued improvement in successive blood tests and her owners' being very dedicated and experienced with tube feeding, Lilah was sent home on Saturday 16th May. She has continued to improve, and she and her 'brother' removed her feeding tube between them 10 days after her discharge from hospital. Luckily, she was eating well voluntarily by that stage.

Prior to her second episode of hospitalisation, Lilah had been fed a mixture of a/d, m/d and a 'pet-specific' chicken mince. This was likely a contributing factor to the development of lipaemia and lipid aqueous. Going forward, she will be fed a low fat, high protein diet and all forms of corticosteroids will be avoided wherever possible.

Contents

Figure 2. Same as Figure 1, but from further away.

Figure 3. Lilah the torty with feeding tube in place, obviously feeling under the weather and resting during her convalescence.

Figure 4. Lilah and her 'brother' feeling back to normal. The simple long-term solution was a low fat high protein diet and avoidance of corticosteroids.

Acknowledgement: We would like to thank Dr Richard Malik and Dr Carolyn O'Brien for their help with this case.

HOW DO YOU PROCESS YOUR SURGICAL INSTRUMENTS?

Are there room for savings?

Is your current process sustainable?

Can processes be improved?

Have you ever really looked at the way your practice processes its sterile equipment?

Do you know what the financial, environmental or labour impacts are of these processes?

Your clinic is likely sterilising Surgical Instruments and implants using at least one of the following methods;

- single-use blue wraps
- single-use paper or sealable plastic pouches
- re-usable cloth drapes
- re-usable rigid sterilisation containers

Single-use wraps are inexpensive and commonly available. However, they are designed to be used once and then discarded. They are easily perforated, therefore compromising sterility.

Single-use paper or sealable plastic pouches are also inexpensive and commonly available. They serve the purpose of allowing small quantities of instruments to be contained and sterilised, but are also discarded after each use and are also easily perforated.

Like many single-use products in the clinic, blue wrap and sealable pouches will contribute to a significant percentage of a practice's waste that will end up as land fill. Australians produce approximately 21.6 billion tonnes of land fill each year;[1] however, there are other options to help reduce the waste and the impact it has on our environment.

Re-usable cloth drapes are relatively expensive. Although they are disposed of infrequently, they need to be washed and dried after each use. They can still be perforated and hold contaminated waste in the fibres. Consider the water, detergent, electricity and nursing time required to clean them.

View Primeline ProContainer

Reusable rigid sterilisation containers are typically made of aluminium. While they require a larger upfront cost, there's no comparison in lifespan as rigid containers can last for more than a decade. If you calculate the processing cost per kit, a practice can see savings as soon as year 2 of ownership, simply by comparing to the cost of consumables that would have been used instead.

For more information on Aesculap reusable container systems, visit

- bbraun.com.au/en/products-and-therapies/product-catalog/sterile-goods-management/sterile-supply/sterile-containers.html

or email us at vetcare.au@bbraun.com for personalised assistance.

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SHARING EXPERTISE

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Contents

Small

FAT IN THE BLOOD

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Figure 1. Moneypenny

'Moneypenny' was a one-year-old FN British-shorthair when she presented to me with a severe non-regenerative anaemia (PCV 8%) and was ultimately diagnosed with non-regenerative immune mediated haemolytic anaemia.

She received a typed whole blood transfusion and dexamethasone (0.2 mg/kg IV) and subsequently developed high output heart failure with pleural effusion, requiring needle thoracocentesis. Her anaemia eventually resolved with hefty doses of prednisolone (approximately 2mg/kg BID for 3 weeks before commencing dose reduction) and chlorambucil (2mg per cat every other day). With a gradual taper of medications, she has now been in remission for a number of months. Her similarity to the previous case (C&T No. 5858) was the development of a hypertriglyceridemia and hyperglycaemia, suspected to be secondary to glucocorticoid administration.

In healthy human patients receiving oral prednisolone, there was a significant increase in levels of very low-density lipoprotein-triglyceride (VLDLs), very low-density lipoprotein-cholesterol, high density lipoprotein-cholesterol and apolipoprotein (what a fatty mouthful....). All values returned to baseline within 2 weeks of stopping the prednisolone. (Ettinger Jr Metabolism, 1988).

Some cats (and many dogs, including those with endogenous hypercortisolaemia) seem equally sensitive to corticosteroid effects although there is little information in the literature linking corticosteroid usage to lipid abnormalities (there is more if you are a Schnauzer). One study demonstrated some Burmese cats in Australia had delayed triglyceride clearance compared to other cats (Kluger *et al* JFMS 2009) and this may explain an increased susceptibility to the development of lipid aqueous, pancreatitis and diabetes mellitus. A case report describes a DSH cat who developed transient corneal lipidosis and hypertriglyceridemia following intravenous lipid rescue therapy for permethrin toxicosis (Yuh *et al* Can Vet J 2018). The cat's signs resolved within 72 hours without intervention

After successfully navigating the stormy waters of severe anaemia, heart failure (tri-cavitary effusions) plus thrombocytopenia, **the development of the hypertriglyceridemia and hyperglycaemia just as the patient's anaemia** was starting to improve caused more than just a few grey hairs to develop. There was gnashing of teeth and stamping of feet to say the least.

Clinically, Moneypenny was improving. Although she had developed polyuria and polydipsia (PUPD), this could have been explained by multiple mechanisms in addition to diabetes and after thorough discussions with her owner (who of course was a human medical specialist in immunology) we elected to attempt a more rapid prednisolone taper by the addition of chlorambucil and monitoring

Figure 2. Moneypenny receiving a fresh whole blood transfusion

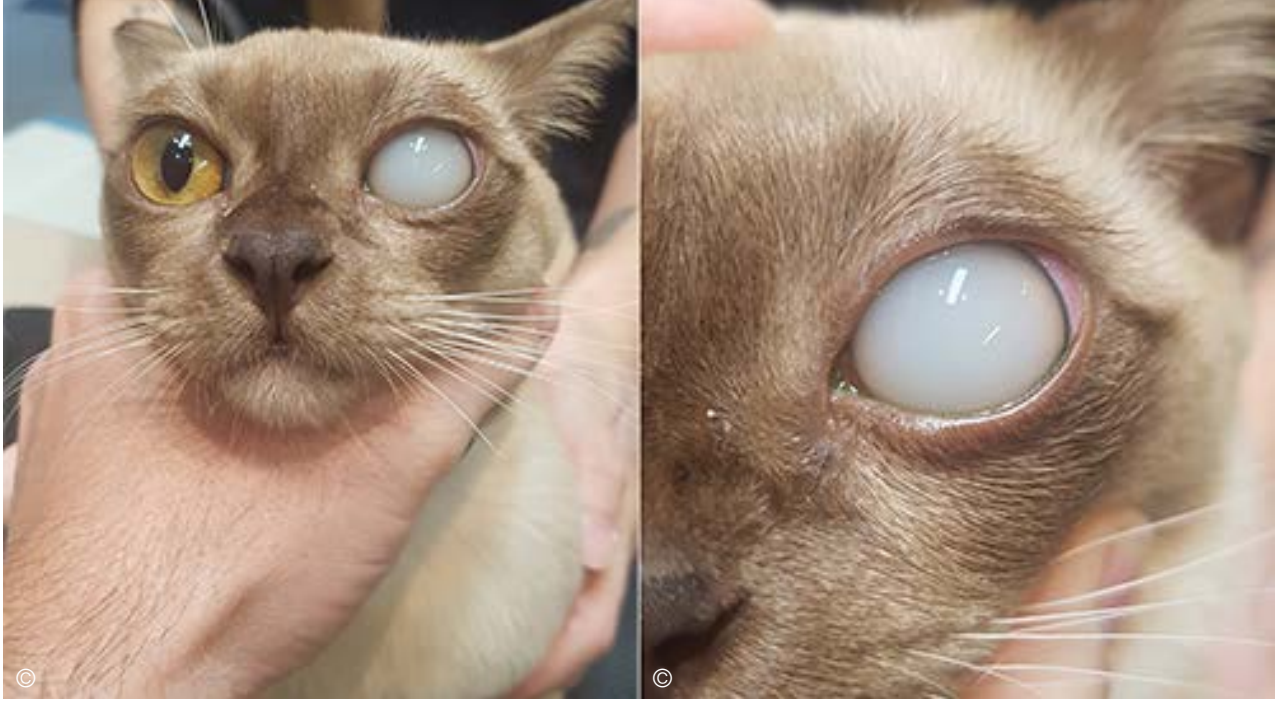

Figure 3. A & B. Burmese cats may have an increased susceptibility to the development of lipid aqueous, pancreatitis and diabetes mellitus

Moneypenny's urine at home via dipstick for signs of ketonuria. We planned to commence insulin treatment if she became ketotic.

As her prednisolone dosage was reduced, her triglycerides returned to normal and the hyperglycaemia resolved; she has remained in remission with her anaemia off all medications for a number of months.

I learnt numerous lessons from this patient:

Firstly, a reminder that anaemia severity at presentation in cats does not predict survival (Korman *et al*, JFSM 2013) and young cats with immune-mediated anaemias can do very well.

Secondly, it is easy for anaemic cats to develop heart failure following blood transfusion and dexamethasone, so close monitoring in post-transfusion period is just so important. Even if patients are discharged, make sure owners monitor resting breathing rates at home.

Thirdly, prednisolone is a drug not to be trifled with—it has so many systemic effects which can be unpredictable. I will definitely be watching triglyceride and glucose levels in any Burmese and British shorthair closely in the future.

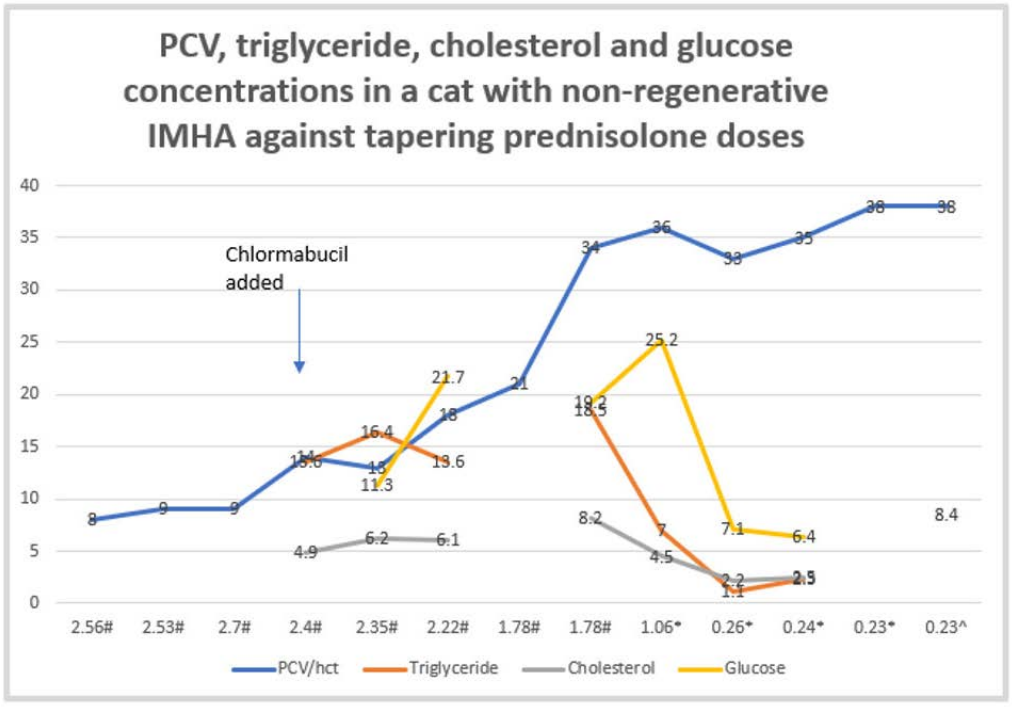

Table 1. Prednisolone doses on the y axis are calculated as mg/kg/day-#twice daily, *once daily, ^every other day. Treatment started with 2.56 mg/kg/day on 14/10/19 and concluded with 0.23 mg every other day 19/8/20.

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Winner

Small

FELINE IRIDIAL MELANOSIS

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In November last year a client who had a cat in for treatment showed me a photo of another of her cats; Lilly, a 2-year-old speyed female Domestic Short Hair. She was worried because over a short period of time, Lily had developed a strange hyperpigmentation on her right eye (see fig. 1). The photo showed a diffuse iridial melanosis which had developed over a period of about 5 months.

At the time, due to financial constraints, she was unable to bring Lily for a consultation, so we had a conversation about the possible differential diagnoses. Firstly, I considered a benign iridial melanoma especially considering Lily`s age. Secondly, we talked about a malignant melanoma. At the time there were no bumps or plaques on the iris, nor anisocoria or eyesight impediments. The difficulty of the diagnosis was discussed with the owner, especially because in many cases a differentiation between the two cannot be achieved with physical examination alone, and sometimes not even with ultrasound. Given the circumstances, the owner was directed to closely monitor the eye.

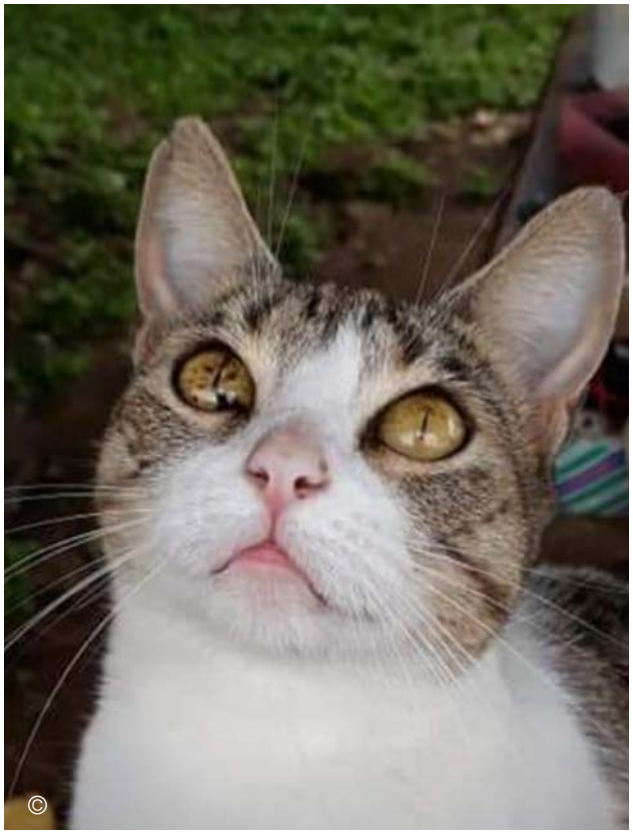

Figure 1. Lily's melanosis on initial presentation

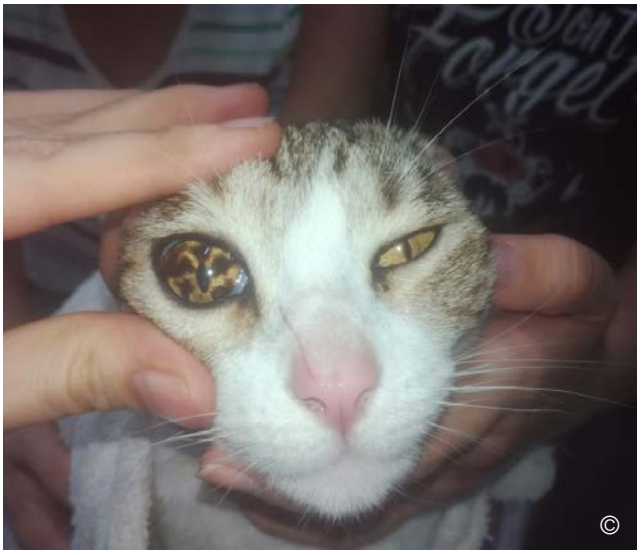

Figure 2. Lily's eye February 2020

In February this year, the owner presented Lily for consultation. She was very worried because the hyperpigmentation had spread throughout almost the entire iris, and the pupil of the affected eye was different from the pupil of her other eye. In the consultation, we verified that the melanosis was affecting the entire iris and that the pupil couldn't contract to the same degree as the one in the normal eye (see fig. 2). I told the owner that I would make some enquiries and that I would call her to discuss a decision. I asked for help in the ISFM forum, whose existence I am so grateful for, and some colleagues discussed with me the possibility of cytology or ultrasound, with a moderate possibility of obtaining inconclusive results.

Given the possibility of having an iridial melanoma—a highly malignant and metastatic tumor, the owner and I decided to enucleate the eye. This is a small clinic—mainly preventive and chronic medicine, so all surgery cases are referred to a hospital. An enucleation was performed, the surgeon put a polypropylene mesh over the socket (see fig. 3) to try to achieve a more aesthetic result. Unfortunately the dorsal aspect of the mesh gave up on day 3, resulting in a semi-sunken appearence. Post surgical outcome was great, pain was managed with meloxicam 0.1mg/kg once daily and tramadol 2mg/kg twice daily for 6 days and amoxicillin-clavulanic 12.5 mg/kg was given twice daily for 5 days. Lily used an Elizabethan collar for the 15 days that the sutures were in.

Figure 3. Polypropylene mesh

Pathology revealed an early stage diffuse iridal melanoma. Due to financial constraints we were unable to access radiographs or ultrasound to search for metastases.

At the time of writing (June 2020) Lily was having a great life and has adapted fully to living with one eye (see fig. 4).

Working within a very small budget is in almost all cases quite stressful, and the risk of taking a normal eye of a young patient was in this case outweighed by the possibility of dealing with a highly metastasic tumor. Gladly in this case we made the right decision.

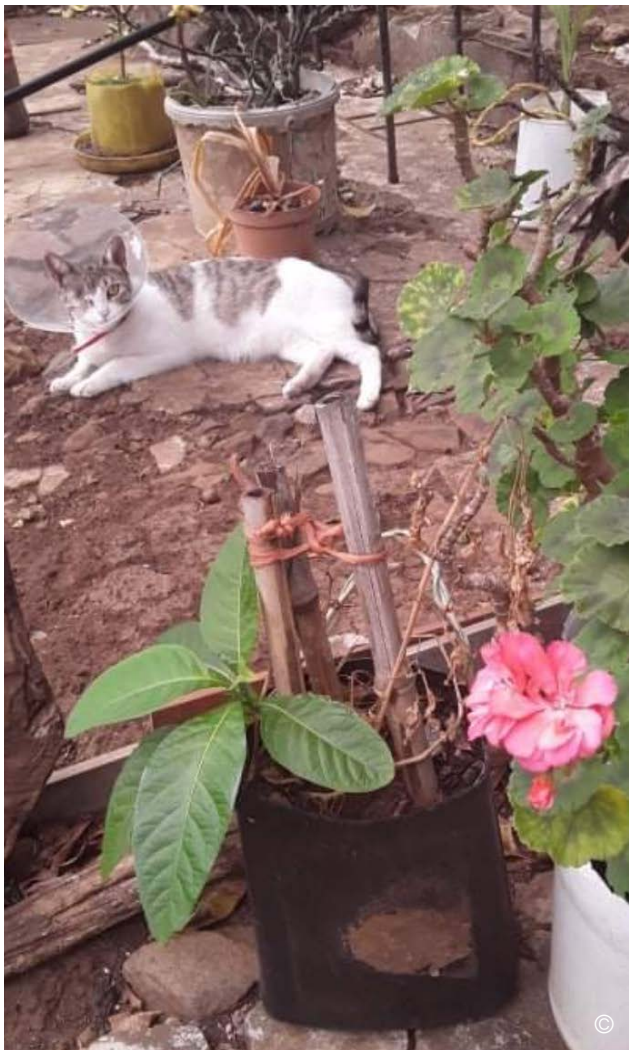

Figure 4. Lily adapting to her new life

Contents

General

RUBBERY NUMBERS: HOW TO MAXIMISE CLINICAL PATHOLOGY INTERPRETATIONS

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INTRODUCTION

When interpreting clinical pathology results, we can have very high confidence when there is a large increase or decrease in the analyte we are assessing, or the result is well within the reference interval. However, we have less certainty if an elevation or reduction is closer to the limits we are assessing in relation to. This is because all clinical pathology results, and the limits we compare them to, have a degree of uncertainty. Having an awareness of what creates these variations enables us to develop strategies to interpret results in light of these inevitable uncertainties.

RESULT VARIATION

Any clinical pathological measurement is an ESTIMATE within a RANGE of possible results. In other words, clinical pathology results are not absolute numbers, as shown in **Figure 1.**

The analytical factors that contribute to the uncertainty of results are analyser variation and intra-individual biological variation. Assessing and minimising pre-analytical factors such as diet, hydration, fasting status as well as collection site and tube selection are beyond the scope of this article. These factors have been reviewed recently[1,2,3] and should be recognised.

Analyser Variation

Repeat runs of the same sample give different results; this variation is known as imprecision. The acceptable amount of imprecision varies by analyte; it should predominantly be determined by the amount of physiological variation of each analyte. All practices should determine the imprecision for each analyte on their in-house analysers and all laboratories should make the imprecision of each analyte publicly available (it should be a source of pride!). Detailed reviews of how to assess imprecision[4,5] and analytical performance goals[6] have recently been published.

Biological Variation

All components measured from body fluids (such as blood or urine) have normal, physiological fluctuations recognised since at least 1970 for human clinical pathology[7] with the first veterinary reports in the late 1980s. There are now more than 50 reports covering >70 measurands across 11 species[8] with more reports since 2011 than the prior 30 years and approximately 5 additional reports each year. The veterinary biological variation website and database (vetbiologicalvariation.org) provides a catalogue and assessment of all measurands (>400) from all veterinary biological variation studies as well as providing guidelines for future studies, standardisation of nomenclature and is hopeful of providing individualised reference intervals in the near future. Biological variation components are constant across age, geography and methodology and values found for healthy individuals are applicable for most (but not all) chronic diseases.[9]

The biological variation components determined in specific studies comprise intra-individual variation (CV_I), between-individual (or group) variation (CV_G) and takes account of analyser variation (CV_A).

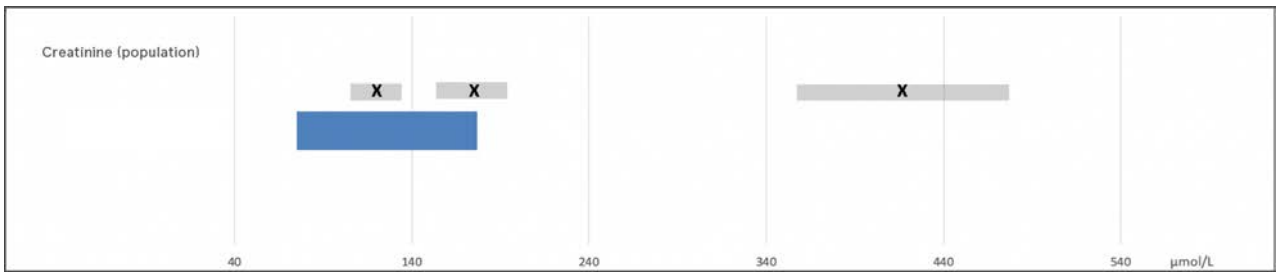

Figure 1. The uncertainty about an individual result is less relevant if a result is well normal or clearly elevated but becomes more important at limits. Blue bar represents population reference interval, grey bars represent uncertainty about individual results (denoted as X).

The following calculations may look intimidating. I am certainly not suggesting that we make such calculations for every clinical pathology result in practice but it is important to know how the ranges I discuss can be determined, i.e. recognise the science behind these statements.

DISPERSION

The range of possible results that each individual result represents is know as 'dispersion'. 'Dispersion' is the calculated cumulative effect of analyser variation (CV_A). and individual biological variation (CV_I); it is typically calculated to a 95% probability.[10]

$$\text{Dispersion (\%)} = 1.96 \times \sqrt{[CV_A^2 + (\frac{CV_I^2}{n_S})]}$$

Where 1.96 is the k-factor for 95% probability and n_s is the number of samples. This requires knowledge of the imprecision for each analyser for each analyte to be assessed and the individual biological variation (data is available for many analytes on the veterinary biological variation website). Note also that dispersion will reduce by running repeat samples so rechecking a result a week later can provide more certainty that a result represents, for example, a true elevation.

Using creatinine as an example,

$$\text{Dispersion (\%)} = 1.96 \times \sqrt{[1.3^2 + (\frac{10.2^2}{1})]}$$

$$= 20.15\%$$

Where 10.2% is the CV_I found on vetbiologicalvariation.org and 1.3% is the known imprecision of an analyser and one sample has been taken.

This means that an individual result of 208µmol/L represents a range of results from 166-250mmol/L. However, rechecking the creatinine (say, a week later) with minimisation of any pre-analytical change reduces dispersion to 14.36% which results in a mean of two results of 208µmol/L represents a range of results from 178-238mmol/L.

The amount of benefit of repeat samples varies by analyte; there is less benefit for analytes with lower CV_I.

If we repeat these calculations for SDMA, using CV_I of 19.9% found for cats in a recent study,[11] and the best scenario imprecision of 4.54% that I found in an, as yet, unpublished study, we find that dispersion is 40.01%. This means that a result of 14µg/dL, at the upper limit of the reference interval represents a range from 8-20µg/dL, so a result of 20µg/dL is required for 95% confidence that there is an elevation above the reference interval. See **Figure 2** (below).

There is a 95% possibility that an elevation represents a true elevation only if the lower level of dispersion is above the limit that a result is being assessed against.

Of course, it is not practical to make such a calculation for every analyte for every blood sample; however, performing the calculation demonstrates how much dispersion is present for each individual result and helps create the awareness of this 'gray area' about results. The calculation might be performed if there is uncertainty whether an elevation is true.

POPULATION REFERENCE INTERVALS

Population-based reference intervals are typically calculated from the central 95% of a normal distribution of healthy individuals (i.e. 2 standard deviations from the mean). Their determination is nothing to do with pathology; they are a probability construct based on the biological tendency to normal distributions (e.g. weight, height).[12] To

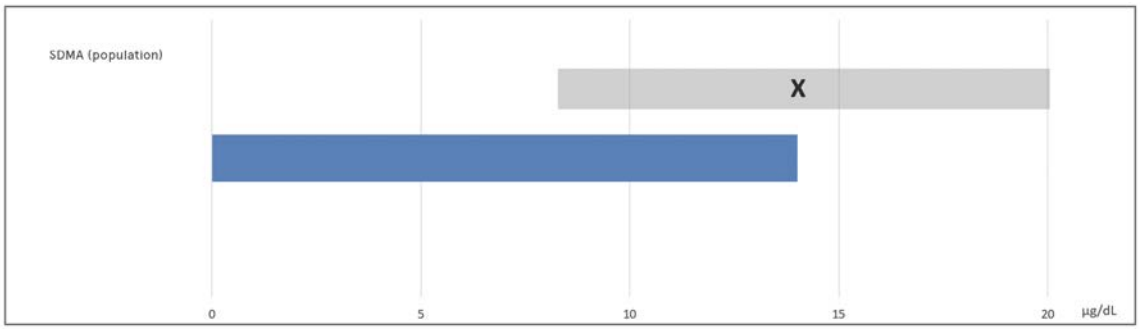

Figure 2. An individual SDMA result of 14µg/dL in a cat represents a range of results from 8-20µg/dL (using imprecision of 4.54% and CV_I of 19.9%). Blue bar represents population reference interval, grey bars represent uncertainty about individual results (denoted as X).

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be correctly determined, population reference intervals depend on subject factors, analytical factors and statistical factors.

Subject Factors

The reference population should represent the population for which the reference interval is to be used. They should be determined for, at least, each species; many practitioners see differences between dog and cat reference intervals on a daily basis (as well as further differences for exotics like ferrets). Breed differences are well recognised[13, 14, 15] such as increased creatinine limits in Burmese and Birman cats, as well as Greyhounds and other sighthounds which also have higher RBC mass and lower neutrophil counts. Even differences based on origin and strain for laboratory animals have been recognised.[16] For some species (e.g, horses and camels), seasonal differences are well described[17,18] and must also be taken into account. Additionally, there can be age-based differences such as increased alkaline phosphatase in growing animals due to the bone iso-enzyme and low PCV and high triglycerides in suckling animals since their milk diet is low in iron and high in fat.[19,20] Further potential differences based on husbandry and diet are possible.

Since population reference intervals are to compare to good health, all individuals tested must be healthy. There is great debate as to what constitutes 'good health' for humans and considerably more difficulty to confirm good health in our veterinary populations. We must also consider geographical differences: are results the same across the world? Are there altitude or temperature variations? We need to be cautious of textbook based or manufacturer provided reference intervals as they have not been created for all populations of individual species across the world. Likewise, universal guidelines and staging systems need to be considered in light of local factors.

Analytical Factors

Pre-analytical considerations are important to create stable sets of results. Patient considerations include fasted or not-fasted; capture and restraint and sample collection site (e.g. jugular vs cephalic vs tail vs ear). Sample handling considerations include anticoagulant used, collection technique and transport and storage times.

Results vary by analyser[21] and method used[22] so reference intervals are only applicable for the analyser they were determined for. Analyser imprecision must meet appropriate performance goals.

Statistical Factors

Non-parametric techniques can be used when >120 subjects have been used to determine the reference interval. With lower numbers, a normal distribution is important and, if the distribution of results is not normal, data must be normalised.

Still Uncertainty

Even when all these factors have been considered appropriately, the resulting reference intervals are calculated with a 90% confidence interval meaning a degree of uncertainty at the reference limits.

HOW WE CAN USE ALL THIS TO IMPROVE

The awareness of dispersion and reference interval confidence intervals enables us to take account of the uncertainty they entail. To be confident of a change, it must be beyond the dispersion range (and the confidence interval of the reference interval). Repeating results often reduces dispersion (but varies by analyte). It is important to know analyser imprecision. We can consider 'individualised' reference intervals (reference change values), however these still have a degree of uncertainty (and there is still dispersion). Above all, we all need to use common sense! Very high increases or decreases are easy to interpret and 'borderline' changes need to be considered as just that: borderline.

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ABOUT THE AUTHOR

Randolph Baral is a primary care veterinarian who has been in a cat-only practice for approximately 25 years. It is not so common for primary care practitioners to contribute to the peer-reviewed literature, yet Randolph has over 30 publications as well as more than a dozen text-book chapters. He achieved his PhD in clinical pathology while continuing in primary care practice. Since his PhD, he has become a world-recognised leader in biological variation; he administers the veterinary biological variation website and has presented about these concepts, and how to use them, at prestigious conferences such as ACVIM and ECVIM. Despite this academic pedigree, Randolph continues to work in primary care, thus straddling the academic/practical divide.

PLEASE HELP US TO CREATE A NOVEL STRESS SCORING SYSTEM FOR CATS

You are invited to take part in a study led by veterinarian Marina Domingues (Internal Medicine Resident, University of Edinburgh (UoE), with Dr Sarah Caney (CEO of Vet Professionals), and supervised by Prof Danièlle Gunn-Moore (Professor in Feline Medicine, UoE).

By sharing your professional opinion, you will be helping us learn more about common stress related behaviours in cats that live at least some of the time indoors. This will help us to create a novel stress scoring system that can be easily applied by owners within their household, as well as aid in the development of future research. This tool will be freely provided to veterinarians across the world to enable them to develop a better understanding of the welfare status of their feline patients.

This Delphi survey consists of two to three rounds of questions. The third round will only be undertaken if it is indicated by the previous rounds.

- Firstly, you will be presented with a list of 34 behaviours potentially associated with feline stress, as per previous publications. However, few of these publications considered cats within their home, which is what we now want you to consider.
- You will be asked if you think that each behaviour can be seen as a consequence of acute (short-term) or chronic (long-term) stress, plus other related questions.
- We estimate that each questionnaire will take you about 15 minutes to complete.

We would much appreciate if you could take the time to complete this survey. If you have any questions about the study, please contact the lead researcher, Marina Domingues via mdoming2@ed.ac.uk - https://www.vetprofessionals.com/site/survey-detail?ID=Cat-Stress

General

COVID TRANSMISSION BY COMPANION ANIMALS?

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In a recent discussion, the incidence of COVID-19 in abattoir workers considered the role of animal transmission in this phenomenon. The following review of information by Sandra Steele about the potential for animals to be involved in the spread of COVID-19 is helpful in contextualising this.

The only cases of infection reported so far in a small number of companion animals (i.e. dogs and cats) have been in association with close contact with COVID-19 positive owners. There have been no reported cases in animals associated with casual contact with infected humans. https://www.vetmed.ucdavis.edu/news/information-animals-covid-19

There have been a small number of studies where a number of animals have been infected experimentally; however, the route of infection and the amount of virus inoculated is not representative of what would occur in a natural setting.
e.g. https://science.sciencemag.org/content/368/6494/1016.abstract

The only animals so far that have been implicated in infection to humans are mink from intensive farms in the Netherlands. These reports involved contact with a significant number of infected animals in crowded conditions. However, the mink were originally infected by an infected farm worker i.e. a reverse zoonoses. Some in-contact farm cats were found to be antibody positive but were not implicated in the spread of the infection.
https://promedmail.org/promed-post/?id=20200525.7375359

https://www.sciencemag.org/news/2020/06/coronavirus-rips-through-dutch-mink-farms-triggering-culls-prevent-human-infections#:~:text=Like%20humans%2C%20infected%20mink%20can,Erasmus%20Medical%20Center%20in%20Rotterdam

A study is being conducted through Washington State University where pets of COVID-19 positive households are being tested to try to determine whether companion animals have played any role in the current pandemic. Understandably, given that the main risk of infection is human-to-human contact, doing surveillance testing on companion animals in these households has required strict infection control protocols for the researchers. This study has been running for a few months and they have not given any alerts regarding a concern about significant disease risk to humans from companion animals.
https://deohs.washington.edu/cohr/covid-19-and-pets-study-caps

As far as I am aware, there are, as yet, no reports of veterinarians who have been infected with COVID-19 by animals they are treating. Considering as a profession they do a number of aerosol generating procedures daily (e.g. intubation, dentistry) one would think that if cats were an infection risk there would have been reports of infection amongst this professional group, especially in the USA or Britain. However, there are many reports of veterinarians in the US being infected by other humans.

Based on what is happening worldwide, epidemiologically the risk of cat to human infection with COVID-19 is far outweighed by the risk of human-to-human infection. In addition, given the temperaments of many of the cats that hang around these sort of establishments, I don't think anyone would be getting up close and cuddly with them.

Sure, it may be worthwhile doing surveillance testing on any cats in these establishments, but that would probably be on the grounds of seeing whether they had been infected by the people who work there.

An excellent resource for anyone who is interested in the current research available regarding COVID-19 and animals is the Worms and Germs blog which is put out by academics at Ontario Veterinary College's Centre for Public Health and Zoonoses.
https://www.wormsandgermsblog.com/

UPDATE

This summary was written in August and the references were current at that time. As is expected with a novel virus, new information is emerging rapidly. Since then, there has been a number of outbreaks in mink farms in Europe with media reports raising concerns about SARS-CoV-2 variants. This link gives an overview of the current information.
Detection of new SARS-CoV-2 variants related to mink
https://www.ecdc.europa.eu/sites/default/files/documents/RRA-SARS-CoV-2-in-mink-12-nov-2020.pdf
Worms and Germs Blog
For current information regarding domestic animals, Scott Weiss has shared some useful information in the Worms and Germs blog - https://www.wormsandgermsblog.com/

Small

SALIVA & TICK PARALYSIS – A POSSIBLE EXPLANATION

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Saliva pooling is commonly an issue in severe cases of tick paralysis, with it becoming thickened and viscous (i.e. somewhere between a fluid and a solid), rarely causing 'mucus plugs', lodged in the laryngeal lumen and seen clearly at necropsy (n = 2; both mild brachiocephalic breeds). These are independent of oesophageal foamy, frothy material, trapped in a dilated and dysfunctional oesophagus (in dogs).

People with neck neoplasia are often exposed to high radiation loads and exacting surgery, to attempt to overcome invasive tissue tracts. Associated collateral dysfunction can be severe, e.g. involving vagus distribution and its impacts in relation to swallowing and/or speaking. Such patients who are unable to form a food bolus (caudal pharyngeal dysfunction), to engage the oesophagus, have to be tube fed (e.g. epigastric, gastrostomy). They too have saliva pooling which (with evaporation) produces thick tenacious material (in the pharynx), necessitating physical removal. (Normal average flow in people is 1-1.5L/day; Anon [2020] ENT Dept, University of Qld, RB Hospital). Incidentally, this saliva flow rate is similar to sweat flow rates in an average environmental space.

Tick dogs are possibly experiencing the same process – i.e. inability to swallow (neuromuscular junctionopathy) so saliva pools (in dependent areas depending on posture) and evaporates, producing potentially obstructive material in the upper respiratory tract. Such material would need clearance to ensure maximal pharyngeal/laryngeal function. The dog's unique cooling technique, mainly by the oral cavity, could also be a species-specific reason for saliva (mostly serous and little mucin; 98% water) to dehydrate, with the associated high airflow rates.

Various publications have projected that this thickening of saliva in affected dogs with tick paralysis could be a drug effect, e.g. attributable to atropine. However, there are so many contra-indications to atropine use in tick paralysis cases – tear flow, cardiac, reflux, urine retention, delayed gastric emptying and dry mucosae. I'm not aware of any substantial evidence based data that proves there is a drug effect or, in fact, what are actual saliva flow rates in TP dogs. In essence, there should be less saliva being produced. So not only is there the chance of toxins (via Ach blockage) effecting saliva secretions, there are no oral (physical or taste) stimuli to encourage a saliva flow. Additionally, general anxiety (of any paralysis) probably encourages sympathetic activity, again potentially reducing saliva flow.

These human (n = 2) observations (while a different cause of swallowing dysfunction) could suggest that evaporation alone could explain the development of viscous saliva, especially in dogs that have a dry 'sticky' mouth/mucosae (possibly suggesting a need for replacement, low maintenance fluid therapy). While posture (e.g. vertical pharynx) in people may encourage pooling, inadequate positioning and poor drainage of saliva in dogs (i.e. neck should be highest point in laterally recumbent cases) may equally cause less gravitational fluid flow and more evaporated deposition, with its associated risks. The two people (so quoted) had extensive neck/face dissection which may have also microscopically affected the autonomic pathways in cranial nerves (7 and 9) innervating the salivary glands, so reducing saliva flow capacity (apart from any 10th nerve issue re swallowing).

Extrapolating data between species is fraught with potential errors. However, looking at a specific dysfunction (irrespective of cause) i.e. tenacious obstructive material in the URT, may give us leads.

With or without autonomic-effecting drugs, it seems that saliva, if not swallowed, will pool and evaporate, creating material that has to be physically removed—directly (by a patient) or indirectly (by Vet. staff).

Ixodes holocyclus paralysis tick
(Photo courtesy of Anne Quain)

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Small

Winner

UNEXPECTED CAUSE OF PYREXIA

Judy Chapman
Gold City Veterinary Clinic
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C&T No. 5864

We run a small animal clinic in north Queensland. Recently I was called in for an afterhours consult with a 8-year-old King Charles Spaniel (female neutered) that presented with what the owner described as 'raspy breathing' that had been getting worse over the last few hours. She even held the dog up to the phone so that I could hear how bad it was! Prior to this, the dog had been in a cage in the back yard with a young pup and had appeared normal.

She presented with neck extension, severe dysnoea and panting. There were no abnormalities on auscultation of the heart. There was a small amount of swelling between the mandible and mucus membranes towards the back of the mouth. The eyes were red (congested scleral vessels) and a bit 'popping out' as the owner described it. The membranes under the tongue were a bit cyanotic. Body temperature was 41.3°C!!

I immediately gave her flow-by oxygen as she resisted a mask and started to cool her down with tap water and wet towels. I then administered IV dexamethasone and IM chlorpheniramine, suspecting some sort of allergic reaction (even though her age didn't really 'fit').

After an hour, her breathing and temperature was nearly normal so I sent her home as I knew that the owner was able to monitor her closely.

Four hours later—in the middle of the night—the owner called saying that the breathing was slowly deteriorating again.

The owner re-presented her to the clinic. Her temp was still normal and the swelling wasn't as bad as previously seen. By then I was desperately trying to get a list of differentials that would fit the clinical signs.

I checked inside the mouth and was running my fingers through the long hair of the neck, behind the ears, when my finger hooked under something.

Figure 1. Patient in distress

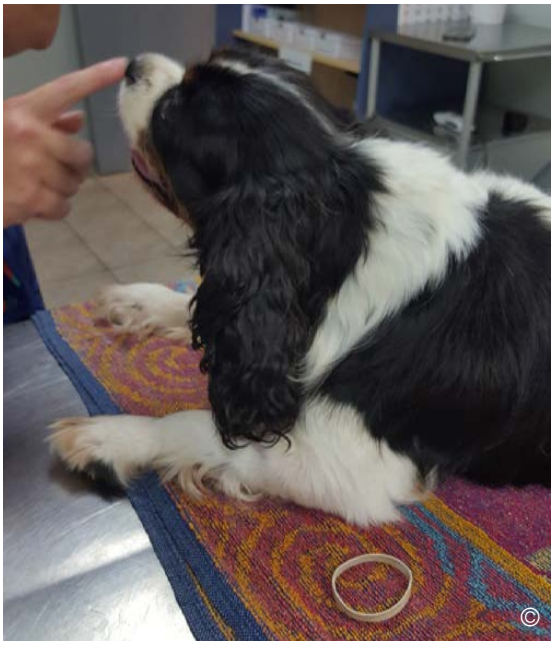


Figure 2. With offending rubber band removed!

There was a rubber band stretched tightly around the dog's neck.

It was removed and the cause of the problem easily fixed. The owner rang her husband to find out where the thick rubber band could have come from. One of her young children had taken the band from the end of the asparagus and for some unknown reason, stretched it over the dog's head.

The thing that really amazed me from this case was the extremely high temperature that the stress and panting had caused when the dog was first presented and rubber band strangulation wasn't on my list of differentials!

Small

MAKE SURE HOLIDAYING PETS 'PHONE HOME'

Dr Aine Seavers
Oak Flats Vet Clinic
58A Central Ave, Oak Flats NSW 2529
reception@oakflats.com.au
C&T No. 5865

As Australians look to holiday more around Australia in the coming months, chances are that means more pets will travel away on holiday with their owners.

To ensure any pet who escapes whilst away on holiday gets to be found quickly and to go home, make sure your pet's details are up-to-date—not just the on the NSW Government registry, but also consider a second level of security by adding your pet's details to an Australia-wide registry.

Australia-wide C.A.R has been around for many years and now for just $5, this register gives that second level of protection to your pet.

At the opposite end of the Holiday trend, or indeed for sole pet owners during COVID-19, make sure your pet-sitters have a HomealonePet card in their wallet, which can be used to contact you if the sitter falls ill.

We have an Alert Card for Pet-sitters and owners (not using NSW Trustee services) free to download and use.

https://www.oakflatsvet.com.au/homealone-pet-double-sided-alert-card-download/

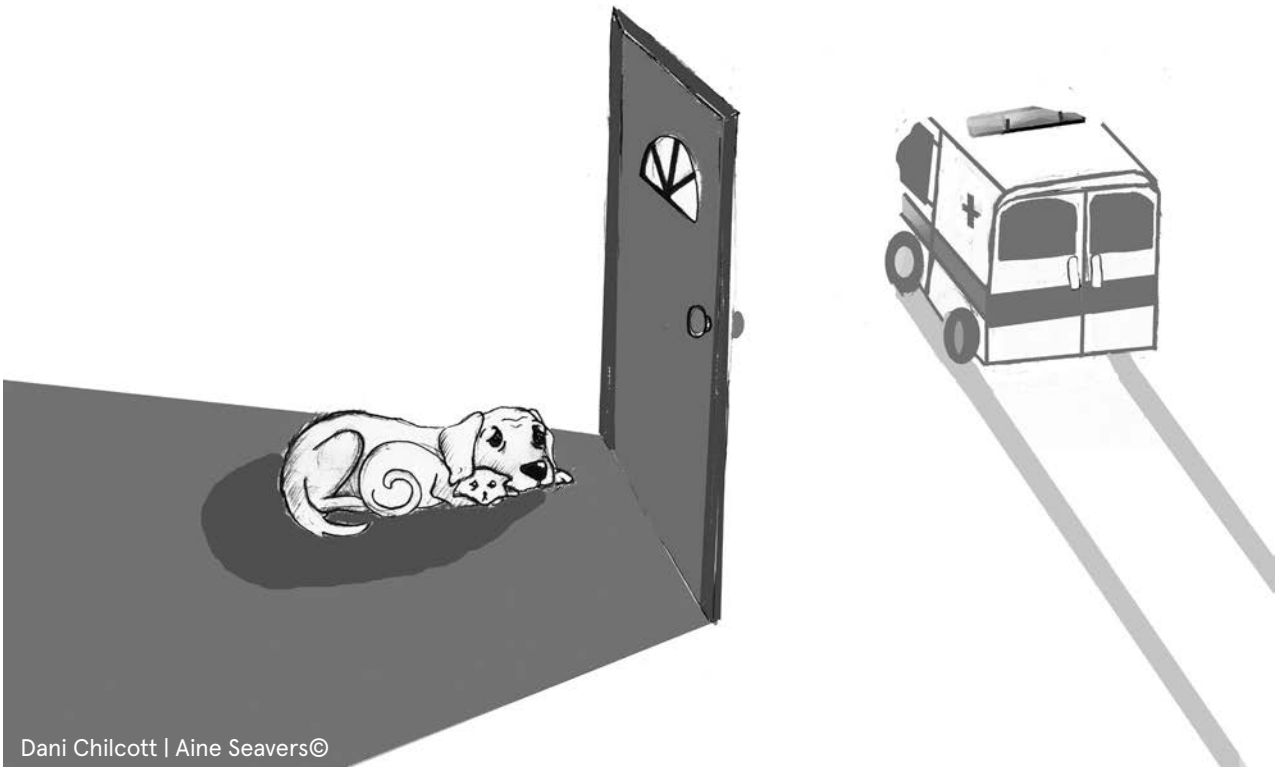
We also have a free poster to download and display in offices and notice boards.

https://www.oakflatsvet.com.au/1185-2/

For the full story behind our HomealonePet Crusade:

https://www.oakflatsvet.com.au/crisis-confronting-pets-owners-paramedics-000-call-outbut-companion-pet/

HOME ALONE PETS: Do YOU have a Plan B?



Dani Chilcott | Aine Seavers©

GASTROINTESTINAL NEMATODES IN DOGS

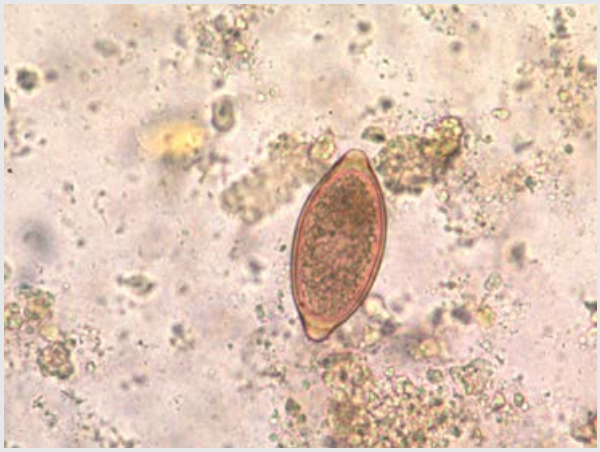
The term "unholy trinity" has been used to describe the three most common soil-transmitted helminth infections in human medicine — ascariasis, trichuriasis, and hookworm infection.[1] In a canine context the "unholy trinity" are roundworm, hookworm and whipworm. Of these, *Trichuris vulpis*, the canine whipworm, is arguably the most frequently overlooked in clinical practice. Is this justified?

A PROBLEM FOR DOGS OF ALL AGES

When it comes to controlling gastrointestinal nematodes in dogs, it is easy to become complacent, particularly when considering older dogs, with gastrointestinal worms usually believed to be more of a problem in puppies. Whilst this is true for hookworm and roundworm, this is not the case for whipworm, with numerous studies demonstrating an increased whipworm prevalence with age.[2,3] The long prepatent period (8-12 weeks), high environmental resilience of shed eggs, and lack of robust age-related immunity means whipworm is a risk for all dogs.[4] Small numbers of whipworm may be tolerated without overt clinical signs, however larger infestations can be associated with significant inflammation (primarily in the caecum, extending into the colon) leading to mucoid or haemorrhagic large intestinal diarrhoea.[5]

MORE COMMON THAN YOU MIGHT THINK

In the most recent national Australian companion animal endoparasite survey, whipworm was the second most prevalent canine gastrointestinal nematode detected, with a prevalence of 1.8% based on faecal flotation.[6] Traditional test methods have been shown to underestimate prevalence by more than 50% compared to faecal antigen testing,[7] suggesting the true prevalence of whipworm in Australia may be 3 to 4%.



PARASITE CONTROL MADE SIMPLE

Whipworm infestations respond well to many routine anthelmintics. Broad-spectrum macrocyclic lactones such as milbemycin oxime and moxidectin are effective, however it is important to consider the dose and route of administration, as products with lower doses may not be effective.[4] NexGard Spectra® (afoxolaner and milbemycin oxime) provides the most complete protection against fleas, ticks, mites, heartworm, and intestinal worms, including whipworm, in a tasty monthly chew.

If you are after more information on parasites and their control, take a look at the some of the resources available on the Boehringer Ingelheim Animal Health Academy (www.animalhealthacademy.com.au). If you are not already a member, use the access code "myAcademy" when first registering. If you are a veterinary nurse and want to develop further in the field of parasitology, sign up for the Vet Nurse Technical Advisor Program in Parasitology. Not only will you become your clinic's parasite expert, it's free to join, provides AVNAT accredited education, and you can also get free NexGard Spectra® for your dog too. Look for the link on the Animal Health Academy for Nurses homepage or speak to your Boehringer Ingelheim Territory Manager.

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Boehringer Ingelheim

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PROIN® CHEWABLE TABS FOR MALE & FEMALE DOGS

New Treatment Option for Urethral Sphincter Mechanism Incontinence

Phenylpropanolamine (PPA) is the primary treatment of choice to restore urinary continence in dogs with urinary sphincter mechanism incompetence (USMI). PROIN® Chewable Tabs provide an easy to use treatment that is registered for use in male and female dogs.

The unique chicken liver favoured chewable presentation of PROIN® creates an advantageous circumstance for the dog and owner. The dog views the treatment as a treat and this makes dosing a much easier and pleasant experience for pet owners.

Ease of dosing is a key factor for veterinarians to consider when dispensing treatment for use at home. If an owner finds it undemanding to follow the treatment protocol, they are more likely to keep the treatment up and thus have a greater chance at success.

Treating USMI successfully is important as it prevents ongoing problems such as urinary tract infections and skin problems due to urine scald. More importantly and disturbingly, a UK based study by The Royal Veterinary College VetCompass™ programme found that USMI was cited as a main reason or contributing cause for euthanasia in 41.6% of incontinent male dogs involved in the study.

Many studies now indicate that urinary incontinence is as prevalent in male dogs as female dogs.

The other benefits in choosing PROIN® as your preferred treatment include:

- Long registered in USA and Canada; supported by many published reports
- Dose rate is 4 mg/kg daily of PPA compared to 3 mg/kg daily of **PPA for syrup presentations**
- Only registered treatment in Australia for **urinary incontinence in male dogs**
- Chewable—chicken liver flavoured—**taste acceptance is at 90%**
- **Dogs can be switched** from other presentation of PPA or stilboestrol without interruption
- Scored chewable tablets—**dispensed in the quantity required for the weight of the dog.**

Side effect in studies report 25% of dogs on Proin® treatment **lost weight.**

Give your owners the best chance at success by providing a treatment option that adds ease of use to its many other benefits.

ausrichter
animal health

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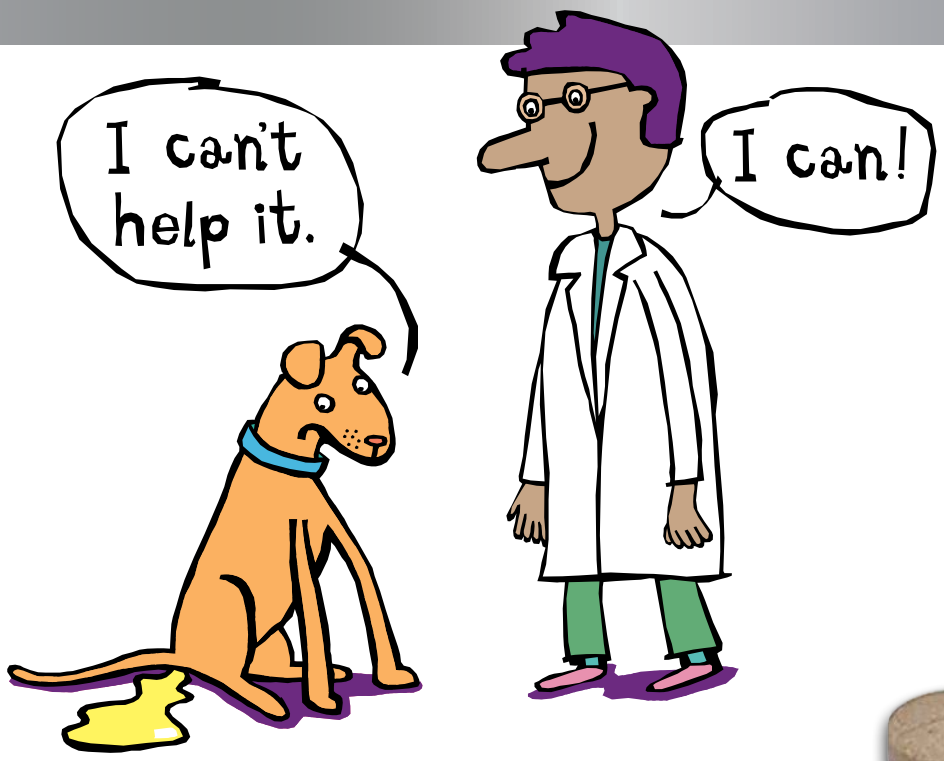
Treating urinary incontinence in dogs is easy with PROIN®

NEW TREATMENT
NEW REGISTRATION



- ✔ Clinically proven for urinary incontinence in **female and male dogs**
- ✔ High dose formulation **for increased efficacy in female and male dogs**
- ✔ Easy dosing **flavoured chewable tablets** – dogs like them
- ✔ **Dogs lose weight** when on PROIN treatment
- ✔ No issues in **switching dogs from other treatments** to PROIN Chewable Tablets
- ✔ Scored tablets for **dosing accuracy**
- ✔ **50mg tablets**, in bottles of 60 or 180.

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AORTIC THROMBOSIS WITH BILATERAL FEMORAL ARTERY EXTENSION IN A DOG

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C&T No. 5866

A 12-year-old, male neutered West Highland White Terrier (11 kg) presented with a 3-week history of progressive hindlimb ataxia and urinary incontinence. At the time of presentation he was on Rilexine® 300mg PO BID for a chronic, recurrent skin infection and a 4 week course of pentosan (Cartrophen Vet® ; 100mg/mL) 0.33mL (a dose rate of 3mg/kg) SC (he had just had his 3rd weekly dose) for osteoarthritis.

On physical exam he was noted to have difficulty walking, with proprioceptive deficits in both hindlimbs (this had deteriorated significantly in the two days prior to presenting), and some mild lumbosacral discomfort on palpation. He also had saliva stained paws due to a chronic, recurrent skin irritation. He has never been tested for specific allergens but is suspected to have an underlying allergic skin condition.

He was admitted for spinal X-rays and an abdominal ultrasound.

The abdominal ultrasound showed an aortic thrombus extending from 1 cm below the renal arteries to at least the level of both popliteal arteries. There was evidence of recanalization but also of a haemodynamically significant stenosis associated with the most cranial portion of the clot within the distal aorta. There were no other significant findings on the abdominal ultrasound noted.

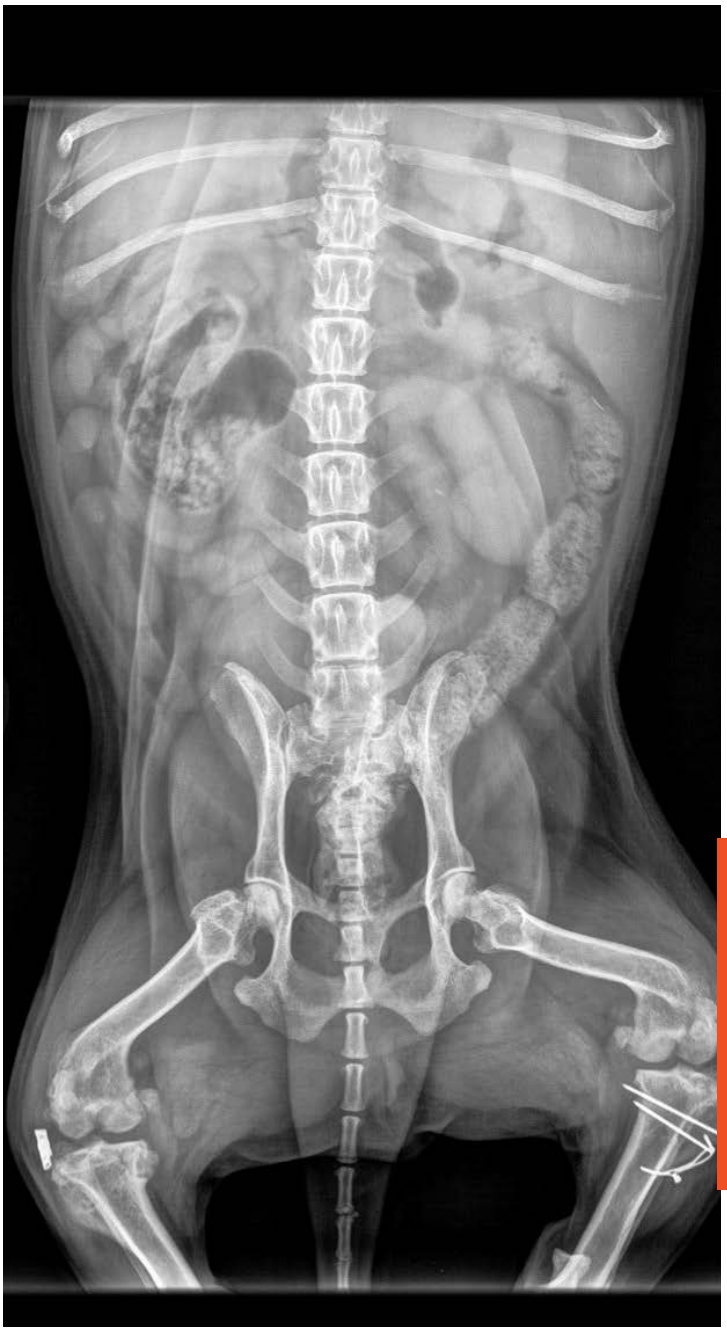

Figure 1. Ventrodorsal spinal radiograph

This aortic thromboembolism was a very unusual finding and it was recommended to both seek the advice of internal medicine specialists for treatment protocols and to look for the possibility of any underlying cause.

After a thorough review of the history of this dog, it was established that a protein losing nephropathy was the most likely cause. There were two UPC (urine protein: creatinine) ratios done in the past 6 months that had both showed elevations of the UPC (see images).

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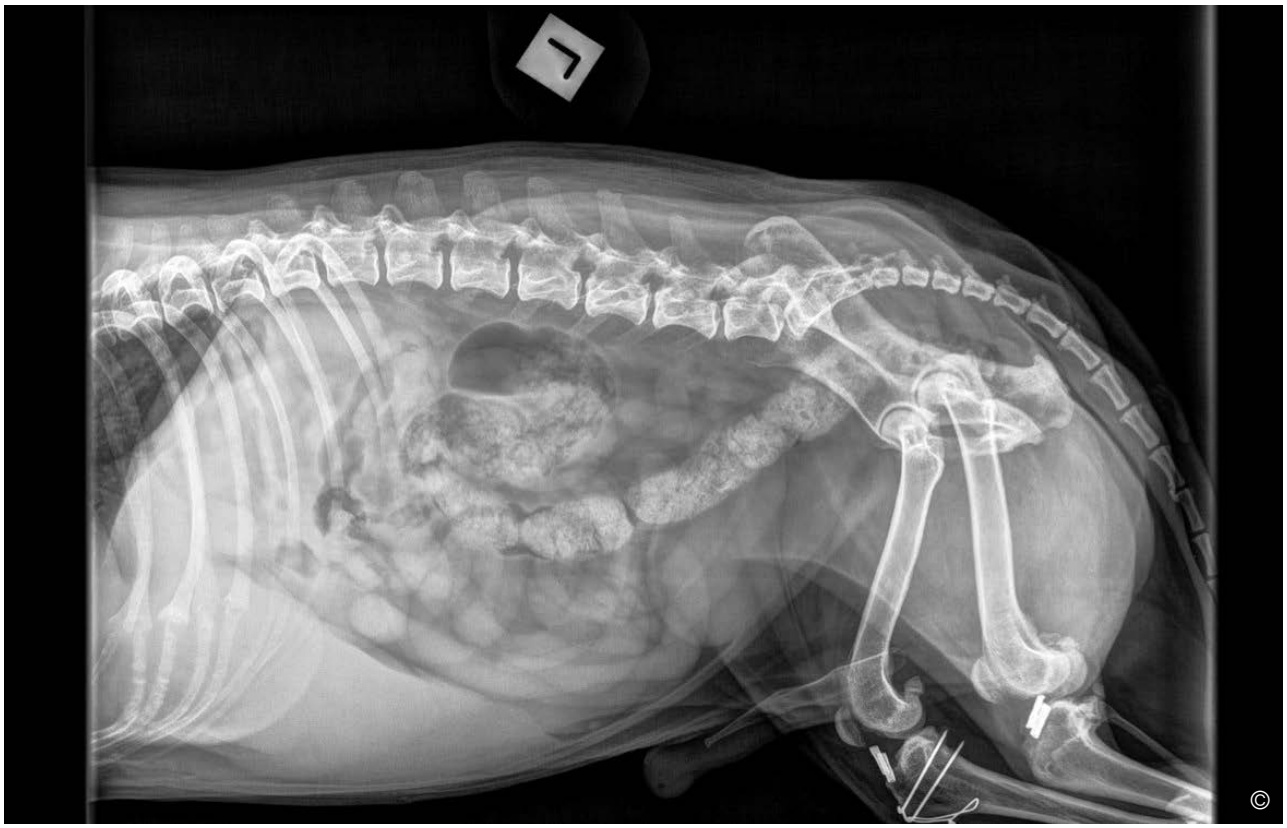

Figure 2. Lateral spinal radiograph

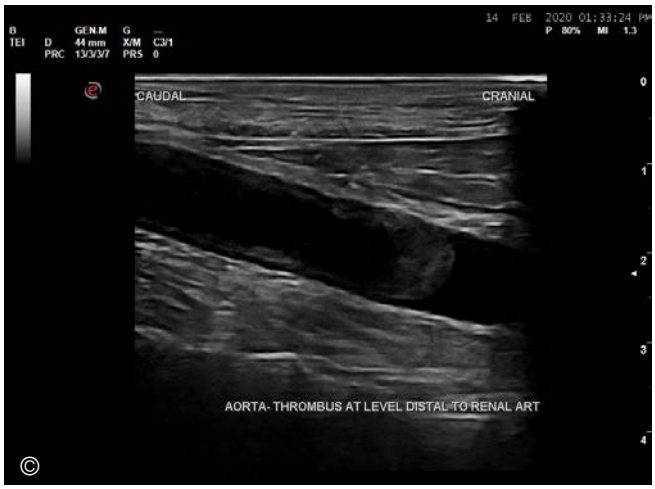

Figure 3. Aortic thrombus 1cm caudal to the renal arteries (caudal structures are on the left side of this image)

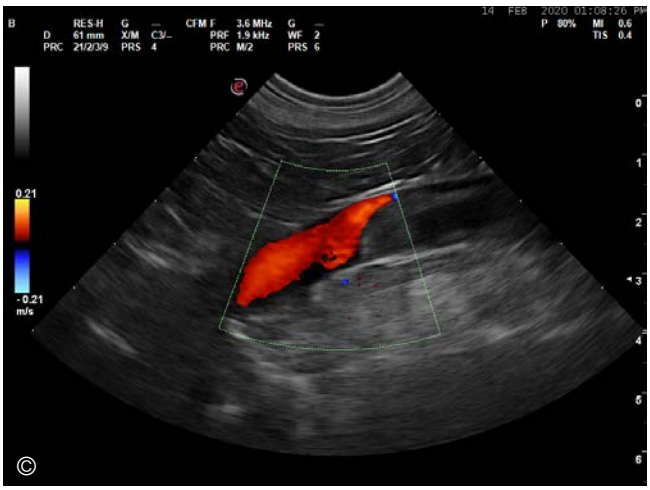

Figure 4. Distal aorta showing perfusion at the level of the thrombus. (In this image and all of the following images except when noted cranial structures are on the left.)

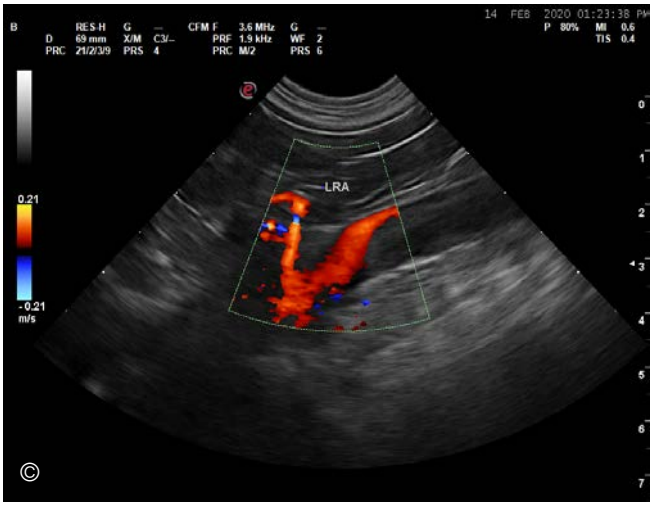

Figure 5. Distal aorta showing the renal artery perfusion in the same image to define the level of the clot

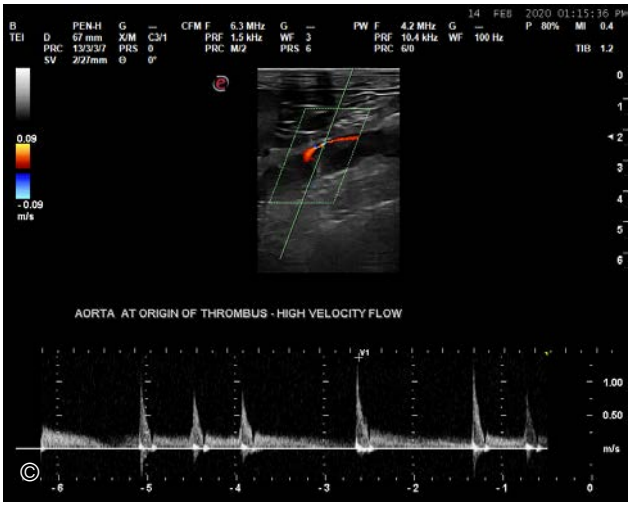

Figure 6. Pulse wave Doppler image showing the significant stenosis associated with the proximal clot

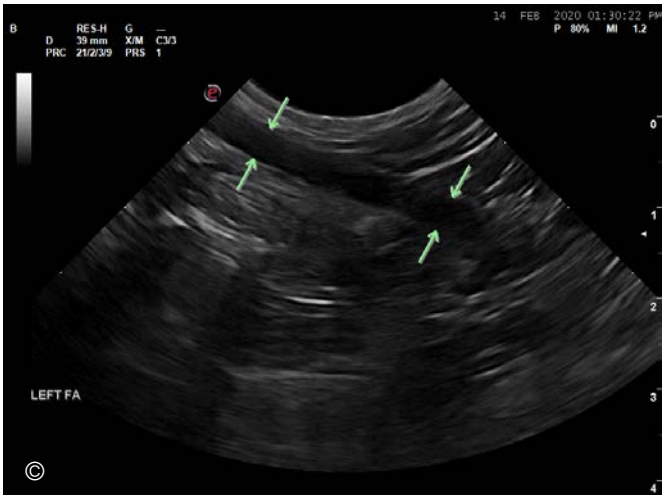

Figure 7. Clot extension into the right External Iliac artery (EIA)

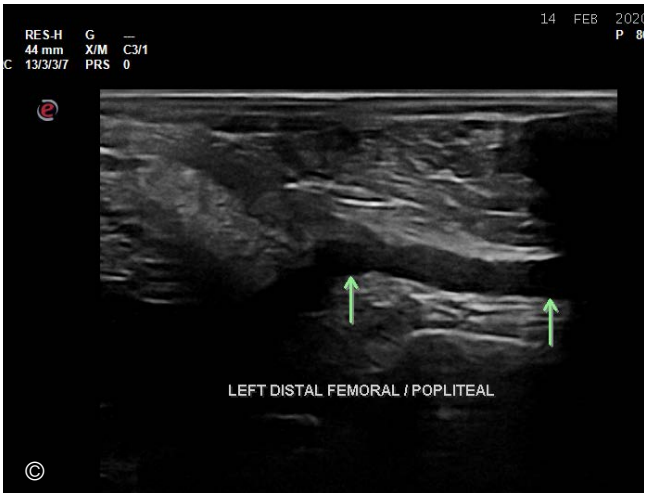

Figure 8. Clot extension down to the level of the popliteal artery (in this image caudal structures are on the left side)

Urine Protein: Creatnine - Canine			
TEST	**SEPT 2017**	**FEB 2020**	
Creatnine Urine (#)	2.63	4.23	mmol/L
Protein Urine (#)	2.7	2.7	g/L
Protein Creatnine Ratio	9.0[1]	5.6	

The guidelines for interpretation of these results provided by the lab where testing was undertaken are summarized below:

`...IRIS guidelines for interpreting proteinuria in stable, chronic, renal insufficiency are as follows:

UPC < 0.2 = Non-proteinuric (NP)
UPC 0.2 – 0.5 = Borderline proteinuric(BP)
UPC > 0.5 = Proteinuric (P)

Primary glomerular disorders may cause a very high UPC ratio (> 3 and often > 5), whereas primary tubular disorders generally cause more modest increases in the UPC ratio (generally < 3)... `

Based on consultation with several internal medicine specialists, the dog is currently on:

- Clopidogrel 3mg/kg PO SID and

- Rivaroxaban 0.5-1mg/kg PO SID

Rivaroxaban is one of a relatively new class of oral anticoagulants referred to as NOAC's (Non-Vitamin K Oral AntiCoagulants) that work on the clotting cascade pathway (see a very detailed diagram within reference article 2 for this clotting pathway). Specifically, rivaroxaban works by inhibiting factor Xa[2] and is recommended to be used in both the treatment and the prevention of clots (venous or arterial).

Clopidogrel is a more widely recognized, antiplatelet medication that has been used to prevent clot formation in human medicine for some time. More recently it has been used in veterinary medicine for the same purpose.

The rationale behind this treatment was therefore to both aid in the dissolution of the clot (through the use of rivaroxaban) and to attempt to prevent the extension of the clot (through the use of both clopidogrel and rivaroxaban). Their use in combination has been shown to have a low potential for bleeding.[2]

A follow up scan was scheduled for 3 weeks post initial scan but a full body CT was performed instead, as 3 weeks post the diagnosis of the large aortic thrombus, a right forelimb mass had been removed and diagnosed as a sebaceous cell carcinoma. This had subsequently recurred and the CT was done to determine any spread, both locally and more distally .

The part of the report that refers to the aortic thrombus is shown below:

`...There is a large luminal filling defect within the lumen of the abdominal aorta. This filling defect extends cranially to the level of the L3-L4 articulation. This area extends caudally to the level of the aortic bifurcation. There is a filling defect associated with the cranial abdominal portion of

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the left iliac artery. The lumen of the abdominal aorta is not completely occluded by this filling defect. This filling defect is most consistent with a thrombus and measures up to approximately 8-9mm in diameter... `

This report highlights the resolution of aortic thrombus extension into the femoral arteries and R iliac arteries, as well as only a partial occlusion of the aorta and L iliac arteries being noted.

This is consistent with the patients improvement—he is no longer dribbling urine, has no proprioceptive deficits noted and is walking almost completely normally.

Discussion

Diseases that result in hypercoagulable states (the thrombophilias) can cause thrombus formation within the distal aorta. These include cardiac disease, hyperadrenocorticism, IMHA, DIC, sepsis, PLE and PLN. An underlying cause is often not determined.[1, 3]

Aortic thromboemboli are less commonly seen in dogs than cats and more often seen in middle-aged to older larger breeds of dog than younger dogs and smaller breeds of dog.[1]

In cats, cardiac causes are the commonest underlying reason for aortic thromboemboli, whereas in dogs it is most commonly a non-cardiac cause (such as IMHA, sepsis, neoplasia, hyperadrenocorticism or glomerular disease).[2] In dogs, unlike in cats, aortic thrombosis of the distal aorta often extends into the arteries of the distal limb, leading to progressive hindlimb dysfunction.[4]

Most dogs present ambulatory but with varying degrees of hind limb dysfunction.[4] It is not uncommon to have them present with exercise induced hindlimb ataxia / paresis.[1]

Dogs that present with more chronic signs have a better prognosis than those that present acutely affected.[3]

Dogs have a better prognosis than cats, with over half surviving until discharge and many living for extended periods after this, with a gradual improvement in symptoms noted. Recurrence is possible, even if the inciting cause has been treated.[1]

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SOLVING THE PROBLEM OF OWNER COMPLIANCE WHEN TREATING CANINE OTITIS EXTERNA

ADVERTORIAL

The successful treatment of canine otitis externa traditionally relied on long courses of multi-dose medication administered by owners to their dogs at home. However, long courses of home treatment can make compliance and accurate dosing difficult to achieve.[1,2] A survey found that most dog owners find twice daily administration for up to 14 days difficult, or even impossible.[1,3]

Another study revealed that up to 80% of owners give the incorrect dose when administering ear products to their dog.[4] This becomes more challenging when dogs become 'ear shy' or stressed, which is often further exacerbated by repeated administration throughout the course of treatment. Owners may also feel guilty that their pet is distressed, and cease treatment too soon.

86% of owners prioritise reducing stress for their dog when treating ear infections.[1]

Furthermore, even owners that believe they can administer ear medication may not always want to. 72% of dog owners would prefer a single dose otitis externa treatment administered by the vet, including those that don't mind administering treatment to their dog at home.[1]

Neptra® is an effective new treatment option for otitis externa that ensures owner compliance.[5,6]

Effective:

- Treats acute otitis externa in dogs associated with mixed infections of susceptible strains of bacteria (e.g. *Staphylococcus pseudintermedius, Streptococcus canis)* and yeast *(Malassezia pachydermatis)*[5]
- Contains broad-spectrum antibiotic florfenicol and anti-fungal terbinafine[6]
- Has a potent anti-inflammatory and anti-pruritic effect, with the 'soft' but potent glucocorticoid mometasone furoate,[5] for rapid and welcome relief

Straight forward:

- The first otic solution to be administered in one single dose by the vet, eliminating uncertainty around owner compliance.
- Provided as a 1 mL pre-measured dose, suitable for all breeds and sizes

For complete control over compliance in the treatment of otitis externa[5], visit www.elanco.com.au/contact and find out more.

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Have you heard?

There's an easier way to treat otitis externa.

Convenience & compliance in one dose – administered by you.

Anti-bacterial (Florfenicol)

Anti-inflammatory (Mometasone furoate)

Anti-fungal (Terbinafine hydrochloride)

NEPTRA®

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5TH COMMANDMENT OF VET MED: THOU SHALT NEVER FORCE AN ANIMAL TO FIT A BLOOD TEST RESULT

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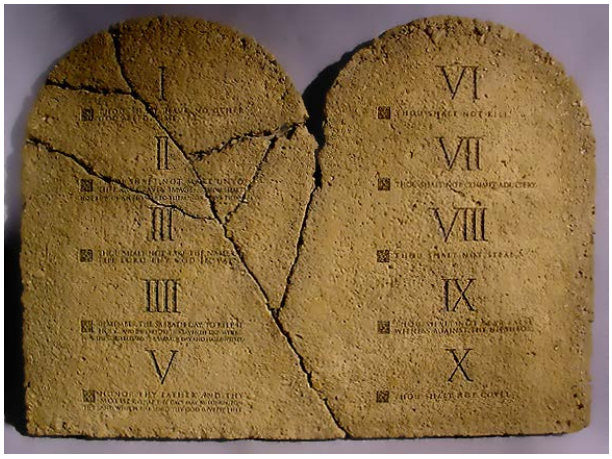
C&T No. 5867



A 20 kg fit 9-year-old dog presented late one evening to the clinic, already on 72 hours of gabapentin and meloxicam. The paramedic and nurse owners, suspecting a relapse of the intervertebral disc disease (IVDD) the dog had suffered 2 years previously, had restarted the left-over drugs from that episode.

The day the dog was presented to our clinic was also the first birthday since the owner's 17-year-old, high-achieving son had taken his own life. Needless to say, everyone's emotions were in over-drive.

On presentation the dog was ambulatory, mildly resented abdominal examination but reacted more strongly on the right-side lumbosacral area where muscle spasm was easily noted. Given IVDD can cause elevation in pancreatic lipase (as good a co-diagnostic tool for IVVD as some imaging procedures); the mild abdominal pain still fitted the picture of IVDD. The dog resented neck movement and had his tail tucked under in a C shape which also suggested IVDD issues. A laser session was run, but the dog continued to decline.

The owners returned the next day and reported a loss of appetite, loose stools covered in mucous and vomiting of about 5 days duration—all against a backdrop of a general slow decline over the last 10-14 days, not just the last 72 hours...

The dog was admitted to hospital and his temperature was now >40°C. A full pancreatic FBC and her biochemistry profile was conducted prior to commencing her IV fluids, along with meloxicam, maropitant and Clavulox injections. IV Ranitidine as a slow infusion was added when faecal scoop (per rectum collection of faeces) produced a stool sample suggestive of digested blood (melaena).

After 3 hours, her temperature was nearly back to normal, she was brighter and responding to the sounds of her favourite squeaky toy being unwrapped—a toy she always received after a visit to the vet.

The 4-hour post-hydration in-house biochemistry was abnormal, but not abnormal enough for what was still a very ill dog. Abdominal palpation revealed an odd fluid wave, which felt like 'balls' of fluid ping-ponging around. Abdominocentesis was negative. Abdominal radiographs revealed a fragment of bone in an odd place.The client did not feed the dog bones routinely, so could say with certainty this yard-contained dog had last had a bone 2 weeks ago.

Pathology Results

Hb	207 g/L	(115-180)
RCC	8.6 $x10^{12}$/L	(5.0-8.0)
Hct	0.59	(0.37-0.55)
MCV	68 fL	(60-74)
MCH	24 pg	(20-25)
MCHC	354 g/L	(310-360)
Plat	178 $x10^9$/L	(200-900)
Band	0.2 $x10^9$/L	(< 0.1)
WBC	7.8 $x10^9$/L	(6.0-14.0)
Neut	6.3 $x10^9$/L	(4.1-9.4)
Lymp	1.1 $x10^9$/L	(0.9-3.6)
Mono	0.2 $x10^9$/L	(0.2-1.0)
Eos	0.0 $x10^9$/L	(0.1-1.2)
Baso	0.0 $x10^9$/L	(< 0.2)

Red cells : Normal
White cells : Occasional Band forms
Platelets : Large platelets +
Note: Manual platelet estimate performed.

Contents

Fasting status - Random		
Sodium	138 mmol/L	(140-155)
Potassium	4.7 mmol/L	(3.8-5.8)
Chloride	99 mmol/L	(100-120)
Bicarbonate	18 mmol/L	(16-24)
Anion Gap	26 mmol/L	(15-25-74)
Urea	19.7 mmol/L	(2.5-9.0)
Creatinine	125 umol/L	(40-140)
Serum Glucose	3.4 mmol/L	(3.5-6.7)
Biliruben	9 umol/L	(< 11)
AST	60 U/L	(1-80)
ALT	20 U/L	(< 80)
GGT	< 5 U/L	(< 6)
Alkaline Phosphatase	309 U/L	(1-120)
Protein	48 g/L	(55-78)
Albumin	18 g/L	(22-36)
Globulin	30 g/L	(25-40)
Albumin/ Globulin Ratio	0.6	
Calcium	2.03 mmol/L	(2.00-2.80)
Phosphate	2.29 mmol/L	(0.80-2.00)
Creatine Kinase	563 U/L	(< 401)
Amylase	951 U/L	(< 2400)
Lipase	223 U/L	(1-70)
Cholesterol	8.2 mmol/L	(3.6-8.8)
Triglyceride	1.9 mmol/L	(0.2-1.7)

**Abnormal values in red*

Alarm bells rang; no bone should still be in the alimentary tract 14 days later. Despite the clinical improvement and surprisingly unremarkable in-house biochemistry (for such an ill dog), we went straight to exploratory laparotomy.

If ever there was to be a medal awarded (sadly posthumously), to an omentum—it is this patient.

The perforation site, where the fragment had sliced almost blade-like through the intestinal wall to finally become free in the abdominal cavity, would have been an easy site to suture. The omentum had tried to wrap around and seal that wound up, along with wrapping omental tissue around the loose fragment to stop the bone doing more damage. Alas, the bone had also sliced through such a vast tract of the intestines that the damage and the peritonitis was not survivable. The omentum had trapped the thick exudate and the pus into pockets—hence the reason why the first abdominal tap was negative.

How did this dog not only survive this long but function at 80% capacity for those 14 days?

How can you have litres of fluid and 200mLs of frank pus inside an abdomen and yet only have a WBC count of 7.8?

A horse vet told me the neutrophils flock into the peritoneal fluid, leaving the horse's blood level low in peritonitis cases, but does this happen in dogs? In this case the WBC was not even low, it was normal. I suspect a lot of the 'wrong' signals from blood tests occur because the omentum is so good at trying to patch up perforations.

One of the main aims of this submission is to highlight the 5th Commandment of Vet Medicine

Nothing in the bloods of this chronic case revealed the true extent of the dog's suffering and eventual poor prognosis.

If I was pushing the cause of Pattern Recognition in the Sept C&T—this C&T message is 'Never forget the Art of History Taking and of trusting your hands and your own gut instinct.'

It does not matter if the bloods look 'ok-ish'. If you know the pet and you get the twitchy, itchy annoying voice in your head that something is still wrong—even if the owners have not paid for it—do additional testing yourself like X-rays and u/s.

We euthanased this dog on the table with the owners present, which is why there are no photos of the graphic lesions we were faced with. Taking photographs was not appropriate given the owners now considerable stress, self-blame, and grief.

For the first time in 6 months, I broke Covid protocol (though we were all wearing masks) and wrapped up the collapsed woman lying against the clinic wall (from around her back) and held her tight for 10 minutes as she sobbed and told me life was too hard to continue with, she was done. Given she herself had 2 horrendous accidents before and after her son took his own life, had been retrenched at the start of Covid from her nursing job of 17 years and now had this self-blame, one could understand her total exhaustion.

Hopefully, holding her and sharing some of the gift of Irish spiritual non-religious beliefs and 'colourful' phrases in how to view the world and its events helped lift some of her pain. It opened up for her a chain of positive thoughts and memories she could recount and acknowledge. She, in turn,

eventually recalled funny positive stories that we could all smile about. With the owners having good qualified professional counsellors in their lives up to this point, my hope is they can get past this loss. Knowing C&T will be publishing this story has already given them some comfort and closure.

The second aim of this article is to highlight the need for vets to give full disclosure advice when they recommend raw meat and bones

Up to six months ago, the mother had never fed bones to her pets. They were fed Hill's dry dog food and lived very long lives, 17-18 years. Six months ago, the owner took advice from elsewhere and started adding bones.

The owner did everything right; she fed only large fresh raw bones from young animals, human consumption grade, fed with meat on to get the dental floss action of the flesh on the teeth, fed under supervision to a calm dog and removed after 20mins to reduce the risk of enamel damage from constant gnawing. The bones were then put in the bin to reduce the drive to dig holes and bury them and hence dig up later and get food poisoning.

Despite all the extra safety feeding guidelines above that I insist on with my clients, when they insist on going against my advice not to feed bones: ***This dog died a preventable death from a food-induced injury that was not survivable.***

To make matters worse, a neighbour rang later to offer support to the owners but suggested it could not be the bone as it must be safe to feed raw bones as dogs in the wild eat them....

Thankfully, the owners having a medical background, saw and understood the intestinal trauma, saw the vet surgeon excise the fragment in front of them and recognised it as the raw bone they fed. No diversionary thoughts could be fed to them as to the real cause of death being other than what it was—the raw bone.

Why on earth and Where on earth does the suggestion come that a wild animal never suffers a slab fracture nor ever dies from an internal injury or toxicity from raw bones?

If pet dogs are allegedly no different to wild (in this argument) what extra 'powers' does a wild animal have that make it bullet proof to any injury or risk? None.

The wild animal does not have super powers, which is probably why very few of them live to 17 years; whereas the majority of our pet animals now hit 17 years without any effort.

- Natural does not mean safe.
- Nature kills and is a killer.
- Ebola, Salmonella, Typhoon, Volcanoes etc are all 'natural' but they are also natural born killers.

Some 20 years ago an unknown breeder in the USA sent me this message after reading my tirade against the feeding of bones:

'Dear Dr Aine: I wished my vet had told me what you have shared today; I would have preferred my dogs above ground with funky breath, than pearly teeth six foot under'.

This phrase seared in my soul and fired me up for many years to make a stand: to allow pets, who have evolved alongside man, to share the same advances in diet that man has evolved to enjoy. The dogs sat by the campfires and evolved alongside man to eat the cooked meat on offer. We need to face that fact and not pigeon-hole modern pets into primitive dietary constraints.

I was not taught a skerrick of nutrition at Uni so it is not an excuse to say *'Oh there is not time to teach nutrition at Uni.'* Learning only really starts when you qualify and get out in the real world.

When you recommend something—for sure embrace the positive aspects—but make sure you know how your advice could go wrong and alert your clients to the risks.

How many vets, who actually advise raw bones, even take the time to give even some of the advice given to this client to reduce risk?

The advice list above is not even the full extent of the safety advice we give clients—and we are not the ones recommending this raw diet!

Most clients who feed raw are shocked when we advise them on risk management for raw diets—very few of the vets advising raw are also advising on risk mitigation.

Whilst life had exhausted some of my passion to fight for pets to be allowed to evolve nutritionally, this case is on the side of the angels and has fired me back up.

Vets need to upskill owners in the risks and benefits surrounding any and all foods fed, be it raw, cooked, premium, or pet shop. Only then will pets be safe.

eBook download
Pattern recognition comment courtesy of Aine Seavers

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CRYPTOCOCCUS GATTII IN THE NOSE OF A CAT

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Oscar is a 14-year-old male neutered domestic shorthair cat that presented as a second opinion from a neighbouring clinic.

Oscar's first presentation to a veterinarian in Cairns was soon after moving from Sydney in early 2019. He was suffering from sneezing and a weeping right eye. It was thought perhaps he was suffering from 'cat flu'-like symptoms and was placed on several courses of antibiotics with limited success. In later 2019 his right eye began to 'bulge' and it was decided to enucleate the eye in hope of curing the sneezing, which it did not. He then went back for revision surgery and 50% of the cartilage was removed from his right nostril in an attempt to curb the sneezing.

Oscar first presented to our clinic in February 2020 as a second opinion case. The mass on his nose had grown back and was causing severe irritation and the mass was actively bleeding due to self-trauma. Oscar also had very loud stertor and the owner was concerned about his welfare.

On presentation, Oscar had a large ulcerated bleeding mass in place of his planum nasal, and it was difficult to differentiate discrete nares. The bridge of his nose was also swollen, with the swelling more distinct on the right side; however, the bulk of the mass resided on the left side. His right eye was absent due to previous enucleation and it appeared to have healed well.

Quality of life was discussed with the owner, and because we were unable to attain the previous clinical records after several attempts, an estimate was provided to the owner to take a biopsy for histopathology as well as run pre-anaesthetic bloods. Oscar was prescribed gabapentin at 10.8mg/kg BID until the owner could book in for surgery (2 weeks later).

On the morning of surgery, biochemistry and haematology were all within normal limits. He tested negative for FeLV and FIV. Oscar had lost 600 grams since his previous consultation.

Figure 1. Appearance of Oscar's nasal planum on presentation

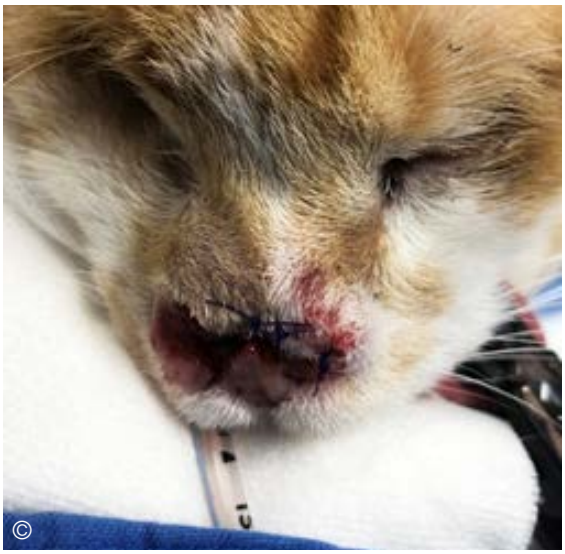

Figure 2. Post biopsy

Anaesthesia was routine, with a 0.015mg/kg ace/0.3mg/kg Methone premed, Alfaxan induction and IVFT at 5mL/kg/hr. Prior to any biopsies, the procedure of impression smears was shown to a new graduate veterinarian, and at this point it was suspected that Oscar may have been infected with *Cryptococcus spp*. The impression smears and fine needle aspirates (see Fig. 3) showed classic round yeast bodies with a distinct clear border.

Because of Oscar's age, and the previous suggestion of cancer, we proceeded with biopsies for histopathology, however we attached photographs of the patient and impression smears with a note to pathologist about our suspicion of cryptococcus. Four punch biopsies were taken from the lesion and a swab was taken for culture and susceptibility if histopathology was indicative of cryptococcus.

Regional lymph nodes were aspirated which showed normal cell populations and no evidence of spread of the yeast.

Dental radiographs were also performed to see the extent of previous curettage.

Oscar was sent home with oral meloxicam and a fentanyl patch for analgesia. He recovered well.

Histopathology results confirmed our clinical and cytological suspicion of cryptococcosis, revealing pyogranulomatous inflammation with numerous intralesional yeasts that exhibit narrow-based budding.

Dr Richard Malik was contacted for current advice on treatment of feline nasal cryptococcus. Oral fluconazole was recommended. Cryptococcal antigen testing via latex agglutination was discussed with Richard, and as the owner was willing, it was suggested to be a good way to monitor response to therapy, especially if the case wasn't going well.

Oscar was seen 3 days post biopsy as he had poor appetite and had lost a further 300g. He was given 150mLs of fluids S/C and prescribed mirtazapine for appetite stimulation. The owner was also advised to warm any food to encourage eating. Bloods were taken for Cryptococcal antigen via latex agglutination (LCAT) which returned a reactive titre of 8192. He was started on Fluconazole at 12.5mg/kg BID orally.

Dr Mark Krockenberger at the Faculty of Veterinary Science (University of Sydney) was contacted and he very kindly cultured samples provided prior to any fluconazole. *Cryptococcus gattii* was cultured from nasal swabs.

During March, Oscar's owner was contacted via phone due to the COVID-19 restrictions in place. Medication was initially rejected by Oscar, however the owner persisted and he was now taking the medication well.

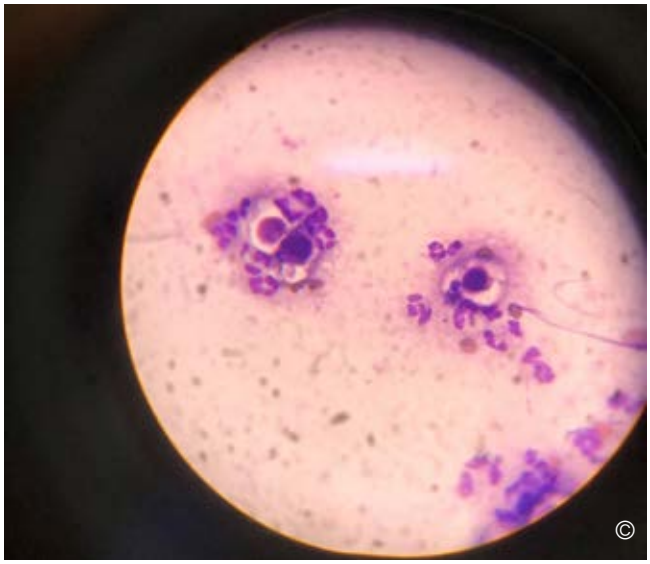

Figure 3. Cytology showing round yeast bodies with a distinct clear border

A revisit in early April showed good improvement in Oscar's condition. His wounds were no longer actively bleeding and he had also regained any weight that had been lost prior to the procedure. An impression smear showed neutrophils only.

In June 2020, Oscar returned for another recheck which showed excellent progress of his condition. He no longer had any open wounds and the swelling on the bridge of his nose had resolved. Breathing was normal and the owner was very happy with the course of treatment thus far.

Oscar is still on fluconazole and we will continue treatment for a further 3 months (3 months past clinical signs being present), at which point we will repeat the LCAT and hopefully stop the fluconazole.

This case highlights the importance of using basic skills (such as impression smears) to help guide clinicians in their decision-making process. It also highlights the importance of diagnostic testing such as histopathology in any case that is not responding to repeated attempts of a cure.

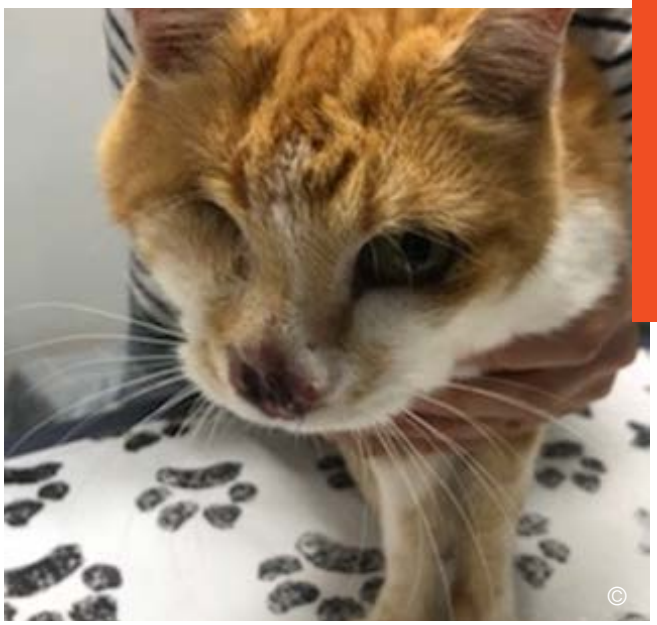

Figure 4. Oscar at his June recheck

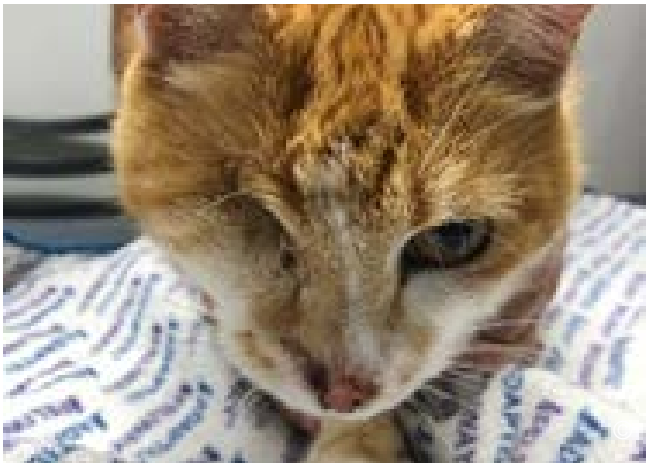

Figure 5. Oscar at his 4 month post initial presentation check

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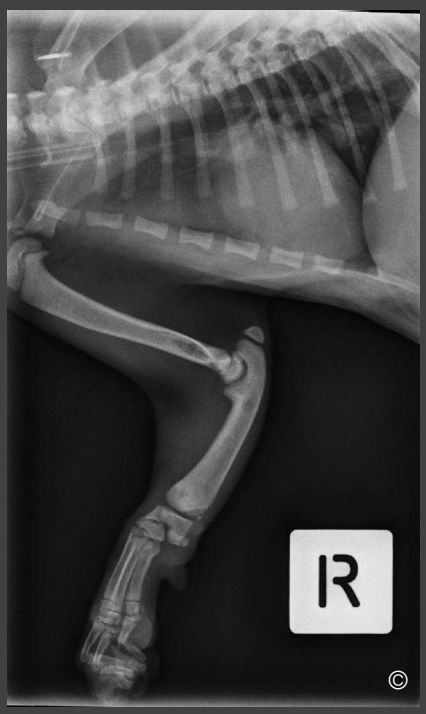
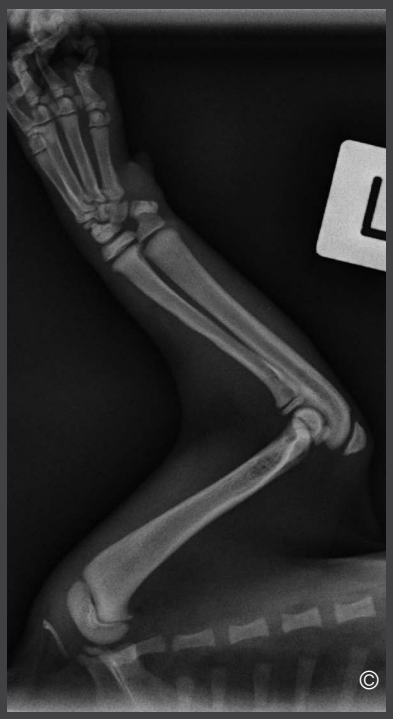
WHAT'S YOUR DIAGNOSIS?

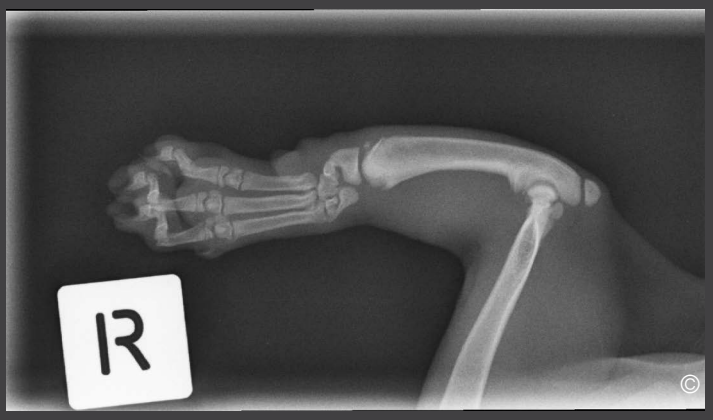
C&T No. 5869

Dom is a male neutered 11-week-old kitten DSH kitten who has some difficulty jumping but otherwise behaves like a normal kitten.

What is your diagnosis and how would you confirm the diagnosis and manage the case?







PLEASE SEND US YOUR ANSWER

Please email your answer to cve.enquiries@sydney.edu.au before 1 February 2021. The best answer will be published in the March issue; the winner will receive a $100 credit prize.

Photos courtesy of Jenny Storaker.

Visited the Mental Wellbeing Hub yet...?

Thanks to everyone who contributed to, sponsored, and bought copies of the wonderful *Vet Cookbook*—a true collegial collaboration.

Proceeds from sales contributed to funding last year's sold-out *Mental Wellbeing for Veterinary Teams Symposium.*

Missed out? Recordings from this event, and other great resources supporting your mental wellbeing, are now available FREE to everyone involved in the veterinary profession at the Mental Wellbeing Hub.

Please spread the word!

cve.edu.au/mental_wellbeing_hub

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Winner

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HAND (HUMAN) REARING OF KITTENS— PURR-PET-UATING PSYCHOLOGICAL TORTURE

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Dr Kim Kendall has been messing with feline minds since last century. While she obtained her Membership in Cat Medicine from the ANZCVS early on, she realised that much of cat medicine, especially feline cystitis and even abscesses, have their root cause in a failing behavioural strategy, so she then studied for and obtained the ANZCVS Membership in Veterinary Behaviour.

In the meantime, her cat clinic in Sydney had evolved into a *de facto* kitten adoption centre, and with the experience of homing over 5,000 cats and kittens, she and her staff were called on to anticipate and also resolve the conflicts between the humans and kittens in their new homes. Kittens, in their early years, are remarkably consistent in their behavioural and other requirements, and eventually patterns emerged. In researching solutions, it became apparent that kittens—from birth to about 18-months-old—need a feline mentor, be that their mother, a sibling or other kitten, or even an unrelated male who is prepared to lick them and teach them about the world. People are not a substitute for a feline friend. Occasionally a dog will do — at least they'll do the licking.

These days, Dr Kendall's passion is to prepare people to provide the best home they can for their feline friend. And she's very pushy about it. From the heart, backed by science.

With COVID-19 EVERYBODY is locked indoors and it has been causing mental distress in more than just cats! Hard way to learn a simple lesson about cats?

For the first couple of years in my clinic, I used to accept that people wanted one cat and they wanted to keep it indoors.

Then I started boarding these cats that were kept as single, indoor cats, adopted at 8-weeks-old or less, and it became clear that they make up a disproportionate percentage of the attack cats. I learnt slowly, but I finally figured out that the cats who were completely confined indoors, alone or with a dog, became very poor boarding candidates because they were the ones who would throw themselves at the front of the cage when anyone walked by and lunge out to scratch and bite when you try to feed them or clean the cage.

So what are the situations where we are getting these deranged cats from?

The trend has become to take young kittens away from their mothers, and usually from their littermates. I think it starts—and is continuing—with a misrepresentation of 'socialisation'. Kittens need CONTACT with people between 2-7 weeks old (20 MINUTES A DAY IS ENOUGH! not ISOLATION and SMOTHERING). There are a lot of people that like to rescue and foster kittens, and then they give them away. However, I found I couldn't just give the kittens I homed out away and then just ignore the problem, because I wanted the people back as clients. So I had to actually listen to the stories and go through them with the owners. And at the start, I got injured trying to figure out these cats.

Who is concerned with these cats for the next 15 years? There's really nobody in the list of stakeholders in the kitten adoption industry who is taking responsibility for mental and physical health of kittens in their new homes and on their progress to adulthood. The RSPCA has free telephone behavioural advice, but no follow up from what I can gather. Most vet practices will home out kittens, but it is actually the nurses who typically take charge of the upbringing and homing out of the kittens. It is an effort for the vet to even deal with the medical issues of rescue cats and kittens (download CVE article re squitty Kitties below which covers most of the medical issues), and since vets in general don't want to talk to owners (endlessly, for free), it is the nurses who do the homing out. Most of the time I had kittens and cats available and looking for homes so I had a fairly structured method of homing out kittens and made an effort to retain the owners as clients. The kittens were my marketing opportunity. My standard practice is to follow these kittens and their owners for more than a month.

At the Cat Clinic we put in place some structure for getting the kittens into their new owner's houses and having them and the owner adjust, become a happy household, and then come back to us for routine vet care AND boarding AND grooming.

Sixty percent of the new owners stayed and became my clients, and I've seen them through all the variations on a theme. Sometimes the cats go out and get run over—you know, the statistics—that one in three kittens that goes outside doesn't make it to its first birthday and another one in three doesn't make it to the second birthday (ref Dr Kersti Seksel and repeated findings on analysis of cat age statistics). People don't like finding their feline friend squashed. So they want to keep them indoors. In that case I tell them it's MUCH better to have **two kittens and a scratching post and keep them inside their whole life.** Or only let them out when you are home—you are more interesting than anything else, so they won't go far. But you still need 2 kittens if you are going to lock them up for 8 hours per day and keep them in at night.

Stray orphaned kittens Grey, Blackie and Fluff.

I teach people when they get a kitten that there are two things you need to teach them—to clip their claws and to take a pill. If you can do those two things to any kitten, particularly if you can teach it before the kitten becomes 16-weeks-old, then taking pills and having your nails clipped without struggle becomes the default.

These days I've got all of these 15, 16, 17-year-old cats who can take their medication and live longer. It makes a huge difference to the effort and cost involved in treating any medical conditions but particularly the geriatric, complex issues. Pills are so much cheaper than the transdermal creams, liquid potions and so on. I think I have I finally discovered why people want to treat old cats. It's a pain, it's medication twice a day, but that effort is worth it so that they don't have to learn a new cat's rules. It's hard changing cats. It's harder to change horses midstream but it's hard to change cats any time.

Should cats be confined?

- The British behaviourists say no, every cat should go outside.
- In NZ, only 8% of cats are kept indoors.
- In Australia;
  - 23.8% are totally indoors (highest likelihood of mental issues if kept alone, with owners working).
  - 16.4% have an enclosure—probably works to increase variability in their life, but depends on whether the enclosure is just a balcony.
  - 5.6%—indoor and outside on a harness—I think these are equally at risk if they are an only cat.
  - 16.3%—Supervised outdoor access[3]—they are probably lying—the cat gets out and they go look for it to bring it back in?—But better than a harness (it gives the kitten / cat choices if nothing else and that is the important thing).

Cats with free access or who are outdoor all the time have a high risk of death while young, but if they get old then they are emotionally more stable as single cats. I have seen 'solo indoor cats' improve their—for want of better words—agoraphobia / anger management / reactivity once let outside even if it is only for the day.

23.8% of indoors only cats is a lot of cats who are having to deal with developmental and deprivation issues because very few are coming from pet cat mothers whose kittens get given out to neighbours at 10-12 weeks old. At least 20% are going out through the foster care / shelter systems.

I get bitten twice a year and it's usually when I'm pushing one of those—for a better word—deranged, deprived, desperate, whatever you want to call them cats. I don't believe such cats are genetically feral. I have met some who are—who resisted parental raising to become a reclusive scratchy biter.

I can only put that down to genetics. You eventually become your mother, don't you?

Who needs a mum when you've got a foster carer?

What if a kitten is hand-raised? Dr Kersti Seksel says as a BROAD generalization, to euthanase anything that has not had a 'mother' or other cats around until they were at least 8-10 weeks of age. Cats need to have either their real mother or a foster mum—i.e. a well-behaved adult cat to learn from as well as other members of their own species. In fact, Finnish authors discovered weaning after 14 weeks reduces aggression problems. I used to say that 6 weeks is your cut off but I think I'd go up to 12 weeks at least, like a lot of breeders do. You're getting into trouble if you don't have a mum. I can only put it down to licking bums, because people do actually stroke the kittens.

Here's a little cat poem:

THE ONLY ONE

WHO WILL LICK YOUR BUM

IS YOUR MUM

The kittens (Blackie and Fluff) some months later in their new home

Blackie and Fluff playing in their new home

Grey and Senior Cat

But there is something magical in cat spit. After doing a caesarian, you can fluff up little babies and blow dry them but then mother wakes up, has three licks and the little thing goes from daggy and sad to fluffy and happy. What is in that stuff?

Is a litter mate enough to make up for being orphaned?

Well the research (from 1959 and 1980) says it's actually pretty good! It shows that with a littermate up until 12 weeks old there is some stability so if you see a singleton hand-raised kitten then maybe don't put it through any more trauma, euthanasia may be a better option than a lifetime of mental distress. This is not the worst thing that can happen to a cat.

Are we still treating cats as little dogs? Well, actually dogs are little baby cats. Dogs are stuck in neoteny—they remain socially immature (compared to wolves) and they have a genetic need for human company. But are cats little dogs? Well no, cats grow up to be independent. Dogs are all just babies, they just never grow up.

Now, back to basics—learning from your mother is most efficient. Anybody who has done anything in the behaviour field knows that Skinner began with studies of cat behaviour. The original experiments were with cats watching another cat, and they learn a novel task quite efficiently. But if they watched their mother solving a problem, then the kittens learned even quicker.

So what is true about social isolation?

So I asked Dr Google about social isolation of kittens. The articles were published a long time ago because you certainly are not allowed to do the things to cats and kittens that you could back then.

Back in 1959[1] they did some pretty horrific things (read it for yourself):

https://awionline.org/lab-animal-search/seitz-p-f-d-1959-infantile-experience-and-adult-behaviour-animal-subjects-ii-age

The essential experiment was done by Seitz, and the experimental model involved leaving kittens with their mother until they were 12-weeks-old. They had 6 pregnant cats and they culled the litters when they were born to 3 per mother. At 2-weeks-old they took one kitten from each of the 6 mothers and they put one each inside a little box. The attendants fed them and they wiped the bums of ALL the kittens, because obviously the 2-week-old orphans were not going to survive without that. They didn't lick their bums but they did wipe their bums. At this stage all the kittens were handled similarly, with or without mothers. Once the isolated kittens were lapping milk and feeding on their own they left all of them completely alone. They weren't touched at all. Then at 6-weeks-old they removed another from the mother and its littermate, and put it into isolation. So now there's a mother and 1 kitten (singleton), and from 6 weeks old there are two in their isolation boxes. **They all have some behavioural derangements because it does turn out you need a litter mate to learn some social rules as well as a mother to lick your bum.** Finally that last kitten got put into a box at 12-weeks-old and none of them met any other cats then till they were 40-weeks-old (9-months-old). There was a control set of kittens raised with mothers and littermates in an interesting environment, and those were used later to test the reactions of the isolated ones.

They fed them all, they weighed them each day. But they had a minimum of handling; so from 12-weeks-old everybody was left unhandled by humans till they were 9-months-old and then they were tested in various apparatuses.

At 9-months-old, they then got daily interactions with cats again, but only when they got tested. So from being left alone in boxes full time, they were let out and made to climb trees and scratching posts and do all of that sort of stuff.

The experiment is actually ongoing. Every time something is born at the zoo and the mothers reject it because maybe they weren't raised properly, which we know is pretty well certain, it's an ongoing cycle and the results are similar to the experiment. The zookeepers do make a big effort to try to keep the babies at least with conspecific mates. In wildlife rescue there are very good statistics on how many actually go back to the wild effectively. Going back to the wild and mating effectively is an even more interesting statistic. And if you go into the agricultural statistics, one of the main ways men on farms get killed is by the orphan poddy-raised intact males: the stags, the bulls, the stallions. You know, leave the man with his testicles and he'll get in trouble sometime. But particularly if he has not had his mother to tell him what to do.

Back to the isolation experiment set up. Group I, removed from their mothers at 2 weeks, had to be handled 5 times a day to be fed. Therefore, all the kittens were handled 5 times a day until 6 weeks old. Then at 6 weeks old everyone stopped being handled by people. The experimenters didn't know about the effects of early contact that is useful in socialising kittens to humans, but it was to an extent fulfilled because of these ones who were handled until 6-weeks-old. They didn't know about that at all in 1959, and **what we know now is that the handling that improved rat, dog and cat stress responses was because of the extra licking the babies got when they were put back with their mother, not the contact with the humans or in some way getting used to human smell.** Back to licking again.

The conclusion essentially was that all the isolated animals were all pretty deranged after not being handled since 6-weeks-old and being all by themselves from 3 months as a minimum. And when they met each other they did different weird things. Some climbed and some didn't. Some sat in boxes and some didn't. **The least deranged were the kittens separated from mum at 12 weeks but who had a littermate.**

What I wondered was at what point did socialising kittens from 2 weeks old get translated into isolating them from conspecifics from 2 weeks old?

I've read a lot of the old material, and it says that the socialising time for kittens is between 2 and 10 weeks old. They're not talking about having only humans around, and humans randomly grabbing the kittens and talking to them. In the socialisation experiments they were talking about, handling the kittens 20 minutes once a day and putting them back with their mother was more than ample. But that's the stuff that's trotted out now—that it is a good idea to be getting rid of mum and keeping the kitten—and keeping the kitten away from other cats is going to make them bond more strongly

Contents

Blackie, Grey and Fluff

to humans. No, actually, it's going to make them chronically anxious, and unable to adapt.

There were some more studies in 1980[3] and they decided that you don't need a mother; they had the mother anesthetized for every feeding session. And they had other mothers who were not lactating—actually they did everything you can imagine to sort out the data. And the results came out: **you don't need a mother BUT you do need some other kittens in your environment. If you have other kittens to grow up with, then you're friendly or indifferent but you're not hostile to other cats later on.**[2]

These days, with the fostering and adopting of rescue kittens, you often can't have a mum, and the rescue situation is that the kitten supply is random, but there is always another kitten or two around – and they should live together. The worst they are going to share is their viruses and their fungi. Those they can get over if they are fed well enough. There is local research that has identified the group of people who are bringing the potential adoptee kittens in. If you can only educate those people, that those kittens would be great if they could stay with their mothers in a family home, until they were 12 weeks old then they would be much better cats for their whole life. But someone needs to say don't bring them in until they're 12 weeks old. Get home, feed those kittens, feed that mother, bring them in when they're 12 weeks. I think you just have to be firm.

Again, back to first principles – it is simple. Two kittens and a scratching post.

What is a good scratching post?

Will curtains do? I think the cats need to get their shoulders into it. You want to allow the artwork and shredding to stand alone. If an owner says `oh I want to replace the scratching post'. I tell them: `but that's a work of art in progress, that's not appropriate. You're taking away so much effort; please don't do that. The cat might even pee on something if you do. It's Okay to just leave it. You don't even need to refurnish it. It's not to look good to you; it's a lot of good messages to the cat.'

On the other hand, you can let the cats outside. The isolation effect is diffused if the cat gets outside and makes choices. They often get killed. But the British postulate that it's better for a cat to go outside and have a short, happy and natural life, than it is to have a long life indoors, potentially in isolation and not feeling well mentally. That way the cats go outside, they climb trees, they chase other people, other cats, or they get chased by them, and they pee on things. And I think a lot of these early isolated, hand raised cats are going to be the ones that come inside to pee in the house because there's too much information out there. Would it be better to keep them indoors all the time because they can't deal with the outside variability? But then when they get stressed indoors because of a change of routine, if they are lucky-ish they get put on Clomicalm or Prozac—often they just get banished to the outside or rehomed or euthanased. Maybe life can't be normal for a cat raised in isolation. Take your choice—for a damaged cat, life will always be difficult.

I do tell clients to let the cat outside but just insure it for the first two years of life to make sure you can afford to repair it. But I don't think the insurance companies want it presented that way. Alternatively, introduce it to a new feline friend before six months, or at least before it's 18-months-old. There are variations on a theme, but I tell people it is best before six-months-old to get another cat—that way they haven't forgotten cat language. Littermates are best even if they've both been handraised.

Those are my kitten pairing parameters and I've tested it over time and they seem to work. My nurses and I got interested in some data collection and we did some temperament testing to see if you can find the kitten that was bomb proof. But we found that even the best cat ever could be turned into a complete ballistic nightmare if it was kept isolated. It just went nuts if its energy was not directed appropriately and it didn't have another feline to teach it cat manners.

Then we did some research on cats that came boarding and assessed their behaviour.

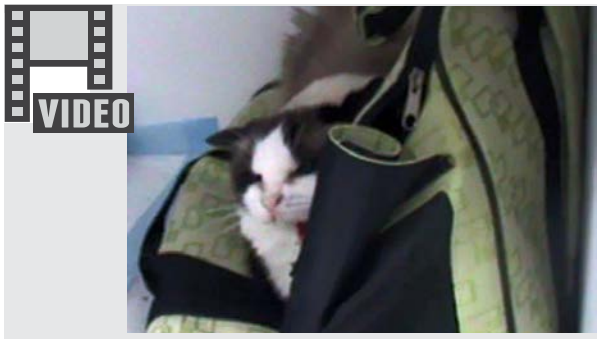
I went back and I looked at the data and it turns out that the rating was enormously influenced by whether there were two cats or not in the household.

The individual cat effect was overridden by the number of friends that it had. The research started by being about the personality in the testing, but it showed the environmental influence more strongly.

One of the key studies that I finally came across regarding this issue was the one conducted by Paul Hemsworth who did a study on pigs and he had pigs who had a genetic fear of humans, pigs who liked humans and had no genetic fear of humans, which equates to the cat's boldness, friendliness and shyness. Then he got stockmen. Some stockmen liked pigs and understood pigs, and the other bunch didn't. He then did the crossover study. It turned out that the friendly pigs with the good stockmen could have anything done with them and they produced well. You put the 'fear of humans' pigs with the unhelpful stockmen and it was a nightmare; with starving piglets everywhere. And then, in the crossover where you had the people-friendly pigs with the bad stockmen—disaster—not as bad as the genetically fearful, but pretty bad. If you are a bad stockman, you can make a pig dislike you. And then, if you put a good stockman with the fearful pig, the stress reaction was still doubled and they weren't as good as the friendly lot, but they were better than the friendly pigs who were screwed up by the bad stockmen.

So what you do in the first year of life overcomes your genetics. And pigs generally don't live long enough to become their mother.

Watch the video of Brick the 7-year-old with multiple staff members' blood on his claws



Watch this video of Olivia – unreformed even after getting a friend cat (Chloe) when she was 6-months-old

Watch this video of Snowy

Early desexing and behavioural issues affecting young cats

Have you wondered why those gorgeous orphaned kittens, lovingly handraised by your softest hearted nurse, seem to become freakish feline terrorists as they progress through their lives? This video explains why.

eBook download
Perspective No. 79 – Purrr...spective - cementing the human-feline dyad early

eBook download
C&T No. 5009 Squitty kitties

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Small

UROLITHS IN MUNCHKIN CATS

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C&T No. 5871

Munchkin cats are a dwarf short-legged cat that was first developed in 1991 and recognised by the International Cat Association (TICA) as a breed in 1994. The name 'munchkin' derives from writer L. Frank Baum's diminutive inhabitants of Munchkin Country, originating in the 1900 novel, *The Wonderful Wizard of Oz*. The breed was advocated by Dr. Solveig Pflueger, a show judge and geneticist.

The Munchkin is not yet recognised as a breed in all regions, although it is becoming increasingly popular in Australia and in some Asian countries.

The dwarfism gene in Munchkin cats is autosomal dominant and lethal if homozygous (affected kittens dying *in utero*). To avoid homozygous matings, Munchkins can be outbred with an unaffected cat usually a domestic shorthair, to produce heterozygous affected Munchkin kittens. Normal-legged offspring can also result from a Munchkin-non-Munchkin pairing. Munchkins are potentially more genetically diverse than many other breeds and the regular out-crossings have generally been regarded as protective.

The short legs observed in Munchkins were said historically to result from achondroplasia, a genetic disorder associated with dwarfism. More recent work from Missouri and Germany has suggested the breed is attributable to a genetic variant that causes one of the bone morphogenic proteins to be abnormal. In addition to short legs, the disorder can result in an enlarged head, undersized jaw, thickened joints, and a curved spine. Munchkins have a higher incidence of lordosis and pectus excavatum and are at a higher risk of developing severe osteoarthritis.

The Sydney School of Veterinary Science (SSVS) in conjunction with the Veterinary Nutrition Group, and with assistance from Dr Carolyn O'Brien and Dr Richard Gowan, has identified a cluster of cases of xanthine urolithiasis in Munchkin cats thought to be due to an inherited defect in purine metabolism.

All the affected cats (Figure 2) have been between 5 and 18 months-of-age and bred in Australia. The cats presented with urethral and/or ureteral obstruction, dysuria, and acidic urine. Urinary tract ultrasound may show cystitis (bladder wall thickening), bladder and renal pelvic 'sand' and renal pelvic dilation if ureteral obstruction is present.

Urate and Xanthine crystals form as by-products of purine metabolism, vital for energy production in the body. Urate crystals can arise secondary to the presence of a portosystemic shunt, or in association with a metabolic deficit in purine metabolism. Xanthine is only known to be associated with deficits in purine metabolism.

Both urate and xanthine crystals form amorphous aggregates, which create a sand-like sediment in the renal pelvis and bladder (Figure 3) and can appear morphologically similar. The crystals are often small and difficult to observe (Figure 1) but may form larger uroliths when combined with other urine precipitates. Cystocentesis or free catch urine samples may frequently only obtain very small amounts of crystal, despite sediment being visible on sonographic examination. Definitive diagnosis may need to be made on analysis of urine metabolites.

The SSVS of Sydney is looking to identify if these xanthine crystalluria cases can be traced back to a single mating or is now associated with dispersal of the abnormal gene amongst the wider Munchkin breed population. We are commencing genetic testing of the affected cats and would be interested in including any affected or unaffected Munchkin cats.

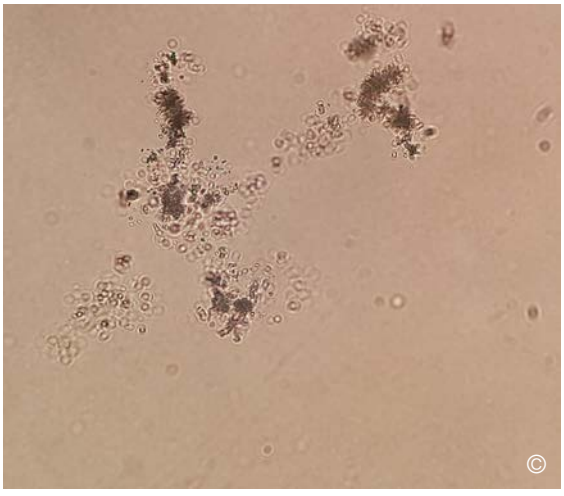
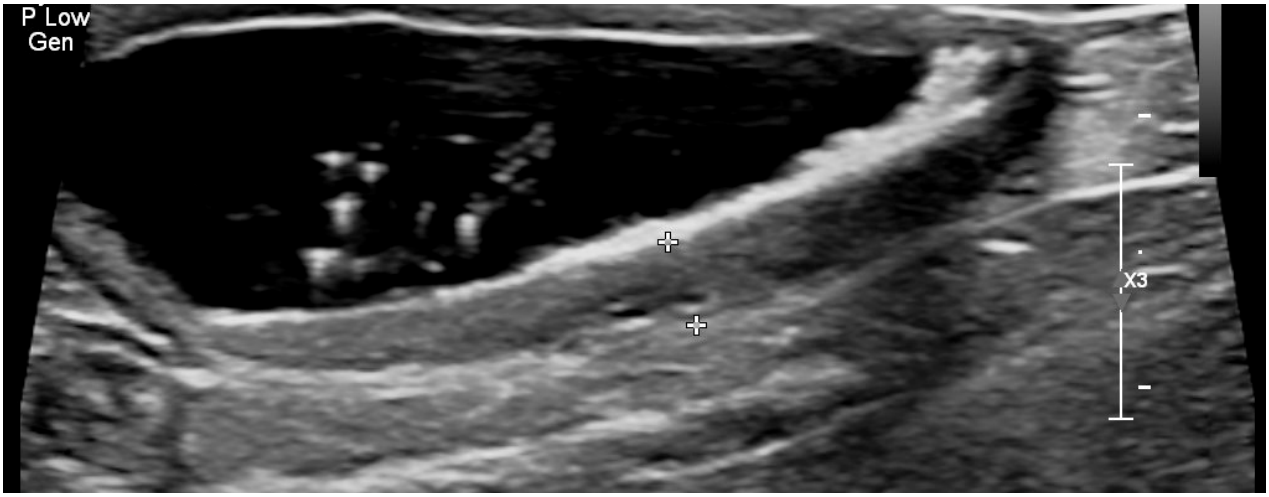


Figure 1. Xanthine crystalluria from affected Munchkin cat



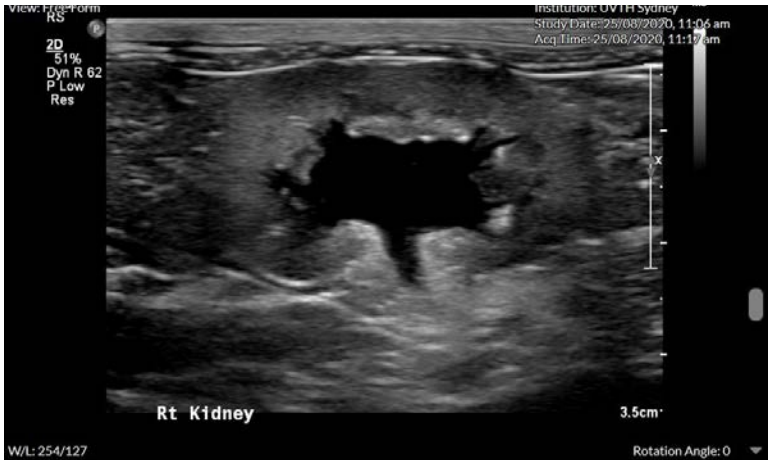


Figure 2. (above) A Munchkin – a type of autosomal dominant dwarfism. Note the disproportionate shortening of the appendicular skeleton.

Figure 3. Urinary bladder of an affected Munchkin cat with thickened bladder wall and hyperechoic sediment within the lumen.

Figure 4. Renal pelvic dilatation in affected Munchkin cat with ureteral obstruction.

CAN YOU HELP US?

If you have any cases of Munchkin cats with urolithiasis, urethral or ureteral obstruction, azotaemia or inappropriate urination we would ask you to contact Dr Emily Pritchard, Resident in Feline Medicine, UVTHS, SSVS at Emily.pritchard@sydney.edu.au, phone 02 9351 3437 or fax 02 93517436. Emily is conducting her research in collaboration with her supervisors Dr Lara Boland, Professor Clare Wade and Dr Bianca Waud.

Contents

Reptile

A BEARDED DRAGON WITH HEPATIC LIPIDOSIS AND FOLLICULAR STASIS

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C&T No. 5872

Figure 1. Amber

Amber is a 2 ½-year-old female 490g central bearded dragon.

She is kept in a 120cm x 45cm x 60cm glass tank, with a basking spot temperature of 35-39°C, a cool end temperature of 23-27°C, and a T5 12% UVB tube. She is currently fed 3 times a week, with a mix of greens and 2 insects (e.g. crickets, wood cockroaches) once a week. As a juvenile, she was overfed with insects and became obese. She became progressively less active, then, in January 2019, at 1 year and 3 months of age she began showing reproductive behaviour (hyperactivity, digging, and increased frequency of defecation). At this time her weight was 585grams and her body condition was 7/9.

Ultrasound confirmed a fatty liver, hyperechogenic and homogenuous, and a pre-ovulatory follicular stasis. An ovariohysterectomy was performed. Amber received Hartmann's 10mL sq and meloxicam 0.2mg/kg IM and was premedicated with Ketamine 10mg/kg and Medetomidine 0.1mg/kg. The premedication provided adequate sedation for intubation. Amber was intubated with a non-cuffed tube of 1.5mm and maintained with isoflurane at 1.5% and oxygen 0.5L/min. She was ventilated throughout the surgery. During surgery, the liver had a generalised tan appearance and hepatic lipidosis was diagnosed. To determine whether these liver changes were due to Amber's active reproductive status or pathological hepatic lipidosis, the ultrasound examination was repeated in April 2019. The liver appeared again diffusely hyperechoic and hepatic lipidosis was confirmed. Amber was started on milk thistle at 0.05 mL SID (30 mg/kg) and her diet was restricted by further reducing her intake of insects. The appearance of the liver was monitored periodically by ultrasound, and biochemistry was performed but no abnormalities were detected. The hyperechoic appearance of the liver did not change and Amber remained lethargic. She was started on SAMe (Denosyl 90mg tablets) at 45 mg/kg in October 2019, and continued on milk thistle, but her behaviour did not improve.

In March 2020, a liver biopsy was taken via coelioscopy and submitted for histopathology. During surgery, the liver appeared diffusely pale and the edges were slightly rounded. Diffuse, marked hepatic lipidosis with mild multifocal fibrosis was diagnosed on histopathology.

Pathology Results

Quadruplicate sections of both specimens were evaluated histologically and showed minimal to mild-moderate crush artefact, depending on the specimen. Changes in the sections were similar and were described as one, Hepatocytes are diffusely markedly swollen with one to multiple variably sized and distinct colourless cytoplasmic vacuoles that commonly displace nuclei to the periphery. One of the two specimens has multiple thin bands of fibrous connective tissue that extend from the capsular surface into the parenchyma, separating contiguous hepatocytes; less than 10% of the sectional surface is affected by this change. There is a focal tiny (100µm diameter) aggregate of bare lobulated nuclei located subjacent to the capsule that is not associated with any overt change in the immediately

surrounding hepatocytes (possible extramedullary haematopoiesis). There are small numbers of clustered melanomacrophages in the sections (within normal limits for a reptilian liver).

Diagnosis

Hepatic lipidosis, diffuse (within both submitted specimens), marked, with mild multifocal fibrosis (one of the two specimens).

Comments

In reptiles, the collection of lipid droplets in hepatocytes does not always reflect a disease process and it may be influenced by multiple factors including sex, species, season, and nutritional status. However, when the entire cytoplasm of hepatocytes is distended with lipid and is observed in more than 20% of the parenchyma, then a diagnosis of hepatic lipidosis is warranted.

Hepatic lipidosis is a common metabolic derangement in captive reptiles, and it is particularly common in bearded dragons. Overfeeding, lack of exercise, and chronic hyporexia/anorexia are the most common causes of hepatic lipidosis in captive reptiles. This metabolic derangement is considered 'moderate' when 80% of the liver is affected and 'severe' when the entire organ is involved. Hepatic lipidosis was a diffuse change in the submitted specimens and if they are representative of liver elsewhere, then these changes are considered of sufficient severity to result in significant hepatic dysfunction. These findings should be interpreted in context with clinical impression and history.

Pathologist: Shannon Donahoe DVM PhD DACVP Veterinary Pathologist

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Treatment

After receiving the histopathology results, Amber was started on Ursodeoxycholic Acid (Ursofalk 10mg/kg po bid), 10-15 drops of Liv52 (Himalaya) PO SID, 0.05 mL Aminoplex PO SID, and continued on SAMe. She was also started on subcutaneous fluids at a rate of 10 mL/kg of Jarchow's solution (2 parts 0.45% NaCl and 2.5% glucose to 1 part Hartmann's solution) q2 days. Milk thistle was discontinued as it appeared to have no effect.

Amber's behaviour has not changed very much since her treatment for hepatic lipidosis began. She has a good appetite, although she has gradually eaten less at each feeding over time. She remains mostly inactive and lethargic inside her tank; however, she is still very active and alert when taken outdoors.

She also has had intermittent diarrhoea for about 6 months which has been attributed to her hepatopathy, as her faecal floats have been negative. She is given probiotics when needed.

On the 6th of June the ultrasound examination was repeated with slightly less echogenicity found compaired to the initial ultrasound.

On June 18th Amber seemed to start moving more. She is still eating and drinking well.

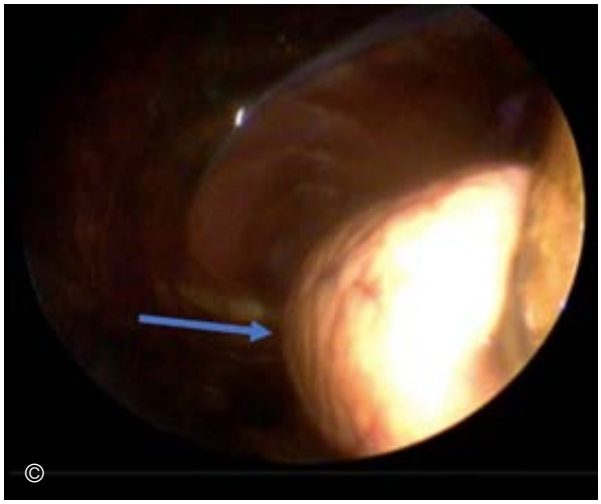

Figure 2. Endoscopy liver (blue arrow)

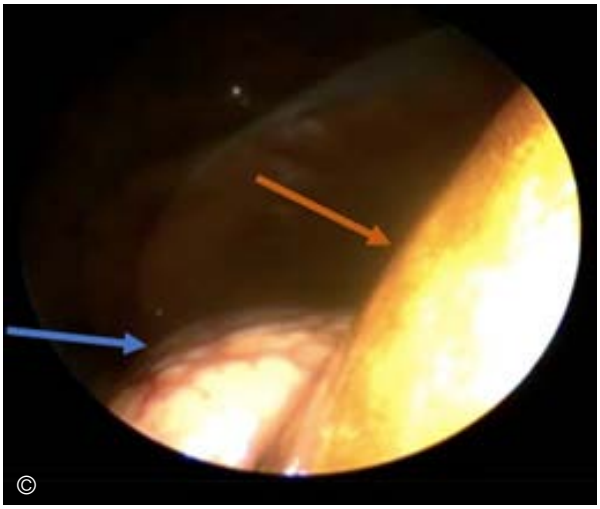

Figure 3. Endoscopy Liver (blue arrow), fat body (orange arrow)

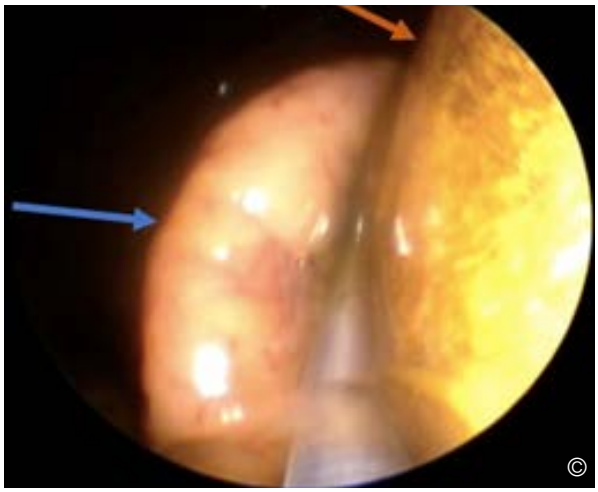

Figure 4. Endoscopy liver (blue arrow), fat body (orange arrow)

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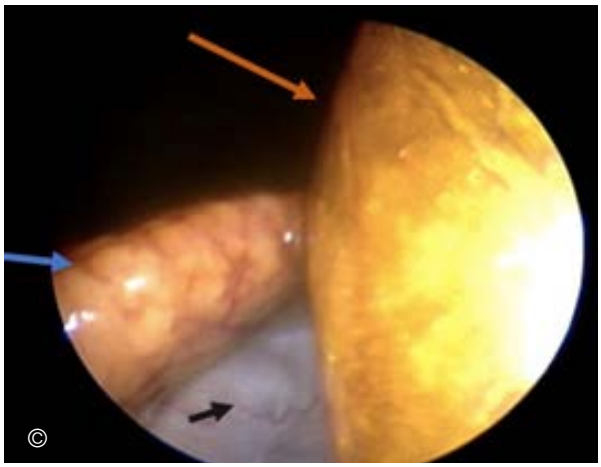

Figure 5. Endoscopy Liver (blue arrow), Fat body (orange arrow), Stomach (black arrow)

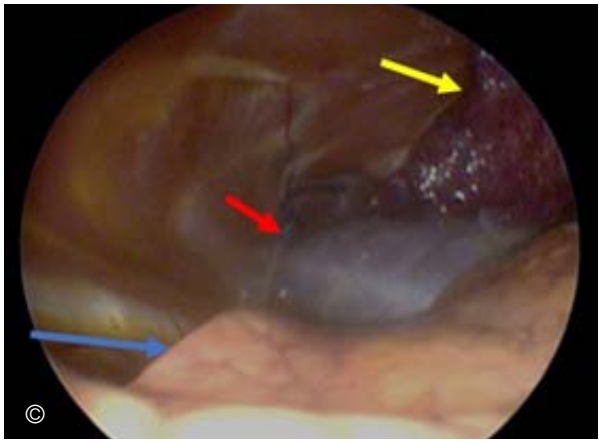

Figure 6. Endoscopy: Liver (blue arrow), heart (red arrow), lung (yellow arrow)

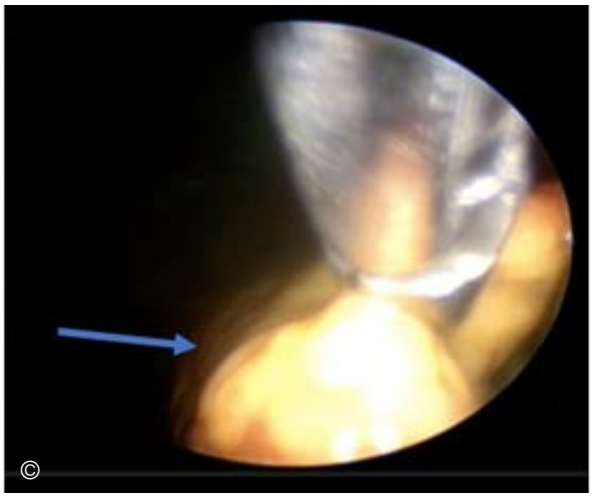

Figure 7. Endoscopy: biopsy of the Liver (blue arrow). Storz Vetpack 2.7mm rigid endoscopy with working channel

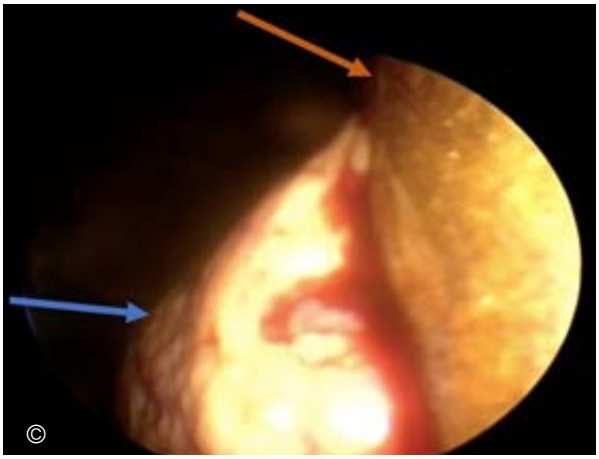

Figure 8. Endoscopy: liver (blue arrow), fat body (orange arrow) after biopsy sampling with minor bleeding

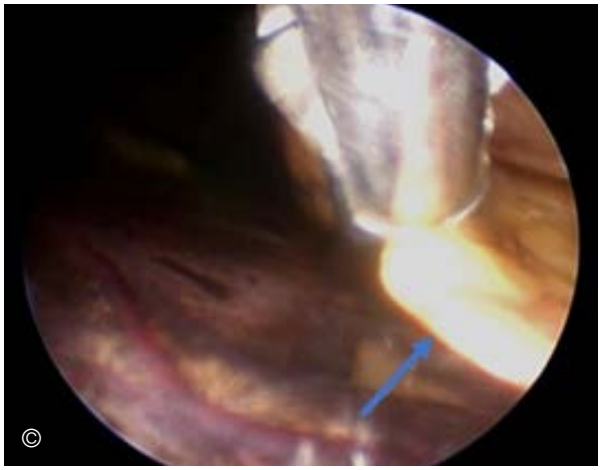

Figure 9. Endoscopy: liver (blue arrow) during sampling

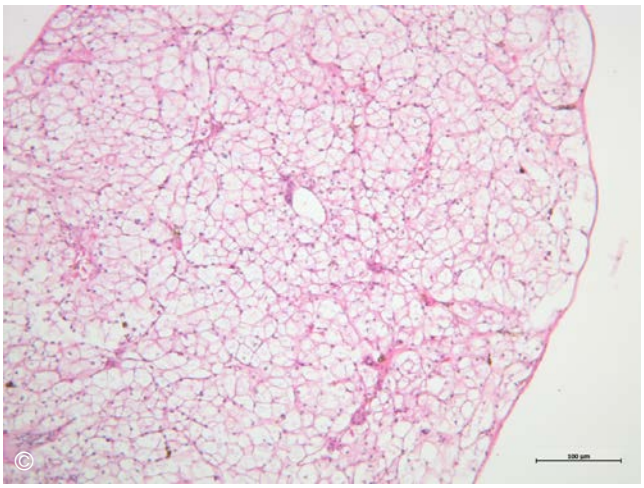

Figure 10. Liver Histopathology (Haematoxylin & Eosin). This is a low magnification view of one of the liver biopsies. Diffuse marked vacuolation of hepatocytes is evidenced by the pallor of the section. The spaces (lack of staining) represent fatty accumulations within hepatocytes that have been removed by tissue processing. Also note that the hepatocellular swelling has reduced the prominence of the hepatic sinusoids.

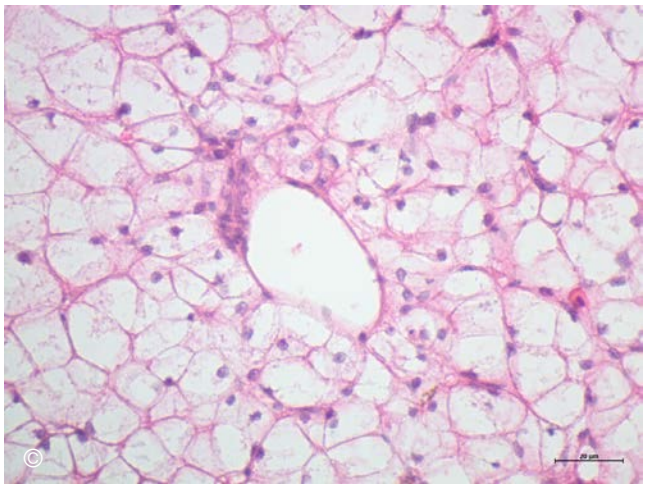

Figure 11. Liver Histopathology (Haematoxylin & Eosin). This is a high magnification view of hepatocytes around a central vein. Note the marked vacuolation of the hepatocytes, with the frequent peripheralization of the hepatocyte nucleus. Frequently there are numerous vacuoles in each cell. The appearance of these vacuoles is consistent with that of fat vacuoles rather than any other cause of cellular vacuolation (hydropic change or glycogen). Note the marked hepatocellular swelling, obliterating the hepatic sinusoids.

Comments courtesy of

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Liver disease in bearded dragons

Liver disease in bearded dragons or reptiles in general can have many aetiologies. It can be caused by bacteria, protozoa or atadenovirus infection.[1]

The most common cause without an infectious origin is hepatic lipidosis which is caused by a high-fat diet based on insects, such as waxworms, mealworms, crickets and woodies, and a low exercise lifestyle.[2] Long term treatment is often required. The compliance of the client is crucial for the success of the therapy as the patient is often anorectic for a long period; it may take years.[3] The treatment is extrapolated from dog and cat medicine as no evidence of a successful treatment has been published.

Milk thistle is one of the most common hepatoprotectors used in exotic medicine, but also used is SAMe, Ursodeoxycholic Acid, LIV52 and carnitine.

Pre-ovulatory follicular stasis

Normal follicular development in reptiles starts with the vitellogenesis, the formation of yolk in the liver. The estrogens then stimulate the liver to convert the adipose tissue and dietary fat into vitellogenin. The follicles can reach up to 100 times their size and the liver enlarges dramatically and becomes yellow.[4] Such was the appearance of Amber's liver during the surgery. An interruption of this process leads to pre-ovulatory follicular stasis and retained follicles fail to undergo regression. The ethiology of follicular stasis is multifactorial. Husbandry related, behavioral or endocrine disorders are reported as cause[4]. In this case no husbandry disorder could be verified. In the common anole *(Anolis carolinensis)* the absence of a displaying male led to incomplete egg shell formation. If the follicles persist long enough, they will become necrotic, which can lead to coelomitis. It is commonly reported in captive lizards.

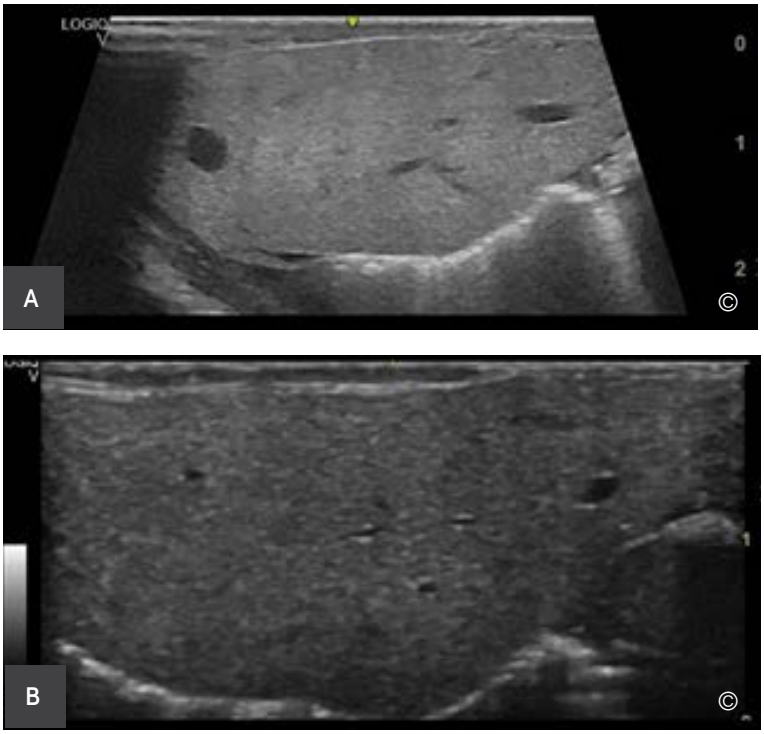

Figure 1. Ultrasonography
A: severe hyperechogenic heterogenuous liver
B: hyperechogenis heterogenuous liver with disseminated hypoechogenic areas.

Endoscopic images courtesy of Lorenzo Crosta, A/Prof, PhD, Dipl ECZM (Zoo Health and Management), Avian, Reptile and Exotic Pet Hospital

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GERIATRIC MEDICINE IN COMPANION ANIMALS

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Wendy is currently performing research on lower back pain in dogs and cats, and osteoarthritis. She is also developing a physiotherapy program for companion animals at the Uni as well as fundraising for a Physiotherapy and Geriatric Care Centre for Companion Animals.

This review will discuss some of the more common conditions of geriatric dogs and cats: cognitive dysfunction, sarcopenia, and lower back pain.

Introduction

Approximately 30-40% of small animals presenting to companion animal hospitals are considered senior or geriatric (Fortney 2012). Geriatric refers to the medical care, diseases and problems of old age as defined by Merriam Webster - https://www.merriam-webster.com/dictionary/geriatric

With so many of our patients reaching an older age, having a systematic program for their care is a necessity. Regularly scheduling wellness examinations is the first step, as well as screening for organ function using bloodwork, urinalysis and imaging such as ultrasound (Fortney 2012). The relative age, breeds and sizes of companion animals is important as it determines what conditions will commonly occur and at what age an individual is likely to develop these conditions (Table 1). Owners can be made aware of their pet's relative age so that they can understand that chronic conditions in dogs and cats may progress much faster than in people, therefore more rigorous treatment and monitoring are commonly required (Fortney WD. 2012). Geriatric medicine has been evolving for the last 30 years. What we now know can improve the quality of life for geriatric pets as well as increase their life span.

Actual Pet Age (years)	Feline Relative Age (years)	Canine Relative Age by Mature Body Weight (kg)			
		0-9	10-23	24-55	>55
3	28	28	29	31	39
4	32	33	34	38	49
5	36	38	39	45	59
6	40	42	44	52	69
7	44	46	49	59	79
8	48	50	54	66	89
9	52	54	59	73	99
10	56	58	64	80	
11	60	62	69	87	
12	64	66	74	94	
13	68	70	79		
14	72	74	84		
15	76	78	89		
16	80	82	94		
17	84	86			
18	88	90			
19	92	94			
20	96				

Table 1. **Human-Pet Age Analogy Chart**

The relative age of adult cats and dogs, yellow boxes in Table 1 indicate mature or senior ages and the blue boxes indicate geriatric ages. Adapted from Fortney WD. Implementing a successful senior/geriatric health care program for veterinarians, veterinary technicians, and office managers. Vet Clin Small Anim 2012(42)823-834.

Geriatric Years

The beginning of the geriatric age is defined as the loss of physiologic reserves including declines in internal organ function, musculoskeletal regeneration, special senses function and immune responses. Frequent assessment by the veterinarian and discussion of the animal's behavior with the owner are important in that early intervention during the development of chronic disease can prolong the animal's life (Dewey *et al* 2019, Sordo *et al* 2020). Assessments of the animal's housing, diet, exercise, mentation and specific conditions that may affect the particular breed of the pet (Willems A *et al* 2017) can be discussed with the owner and any changes that are needed begun as soon as possible. Several owner questionnaires are available to help determine whether the animal's behavior is due to pain, stress or cognitive decline such as the Canine Cognitive Dysfunction (CCD) Rating Scale and the CCD questionnaire (Rofina *et al* 2006, Salvin *et al* 2011).

In dogs, regular assessment of patients for emerging conditions can begin at 7 years, or earlier in giant breeds (Hoskins and McCurnin 1997, Davies 2012). Approximately 30% of adult dogs over 7 years-of-age have an abnormality that is undetected by the owner, but which can be found by the veterinarian (Joubert 2007). Obesity is one such condition that is common in adult dogs, with up to 39-54% of adult dogs currently obese or

Figure 1: A geriatric patient

overweight. One in 5 adult dogs has osteoarthritis and 62% of dogs have an orthopedic abnormality (Willems A *et al* 2017). Half of adult dogs have gingivitis and up to 18% have subcutaneous masses that may be benign or malignant (Willems A *et al* 2017). Once the dog reaches 10 years of age or older, geriatric mental decline becomes more common and up to 60% of geriatric dogs have canine cognitive dysfunction (Fast *et al* 2013). Less common abnormalities of older dogs include chronic kidney disease (1%), bacterial cystitis (4%), hypothyroidism (1%), or neurologic abnormality (15%) such as intervertebral disk disease. Approximately 13% of geriatric dogs have age related loss of muscle mass and strength or sarcopenia (Willems A *et al* 2017, Pagano *et al* 2015).

In cats, aging and decline can be insidious and difficult to determine based on observations from

	LIFE STAGE	AGE OF CAT	HUMAN EQUIVALENT
 TIGGER - 3 months	**Kitten** Birth to 6 months	0 - 1 month 2 - 3 months 4 months 6 months	0 - 1 year 2 - 4 years 6 - 8 years 10 years
 SUGAR - 13 months	**Junior** 7 months to 2 years	7 months 12 months 18 months 2 years	12 years 15 years 21 years 24 years
 ROSIE - 3 years	**Prime** 3 to 6 years	3 4 5 6	28 32 36 40
 NEMO - 8 years	**Mature** 7 to 10 years	7 8 9 10	44 48 52 56
 GEORGE - 13 years	**Senior** 11 - 14 years	11 12 13 14	60 64 68 72
 CHINAROSE - 16 years	**Geriatric** 15+ years	15 16 17 18 19 20 21 22 23	76 80 84 88 92 96 100 104 108

Table 2. How old is your cat?

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Figure 2. Cognitive decline

the owner. Over 30% of cats aged 10 years or older have chronic kidney disease (Lulich *et al* 1992) and cats with degenerative joint disease have an even greater incidence of renal disease (as many as 68% of cats with degenerative joint disease also have chronic kidney disease, Marino *et al* 2014). Radiographic evidence of osteoarthritis has been reported in as many as 82% of cats >14 years of age, however, clinical signs are much less frequent and only 30% of geriatric cats have moderate to severe osteoarthritis (Slingerland *et al* 2011). Dental disease, hypertension and hyperthyroidism also increase with age in cats (Bijsmans *et al* 2015, Sordo *et al* 2020). Hyperthyroidism prevalence has been reported as 14% in senior cats. Diabetes mellitus was not a common finding with only 0.7% of older cats affected (Sordo *et al* 2020). The incidence of cognitive disorder in geriatric cats is unknown, although as many as 36% of cats show behavior changes that are not related to an underlying medical condition and 50% of cats >15 years old display alterations in activity and increased vocalization (Gunn-Moore *et al* 2007). Unlike dogs, cats tend to lose weight, muscle mass and body condition score as they age (Bellows *et al* 2016). With the loss of 100g of body weight, the risk of death increases by 6.4%, with every 100g of lean body mass lost risk of death increases by 20%, and for every 100g of fat loss, the risk of death increases by 40% (Perez-Camargo *et al* 2004). Sarcopenia occurs in aging cats as well as dogs, although the incidence is currently unknown. The increasing energy demands of older cats may predispose them to sarcopenia which can most easily be seen and palpated along the dorsal spine of cats (Bellows *et al* 2016a).

Cognitive Function

In dogs, declines in cognitive function begin at approximately 7 years-of-age and in cats, at 10 years-of-age (Landsberg *et al* 2010). Canine cognitive dysfunction (CCD) has not been found to have an association with breed, sex, body weight, or reproductive status, but does increase dramatically with age and has an earlier onset in dogs with idiopathic epilepsy (Schutt, *et al* 2016, Salvin *et al* 2011, Nielson *et al* 2001, Azkona *et al* 2009, Packer *et al* 2018).

Dogs older than 8 years were followed for 2 years and 33% of dogs with normal cognitive status progressed to mild impairment, and another 22% of dogs with mild impairment progressed to having clinical cognitive dysfunction during that time (Schutt *et al* 2015). Clinical signs in both dogs and cats can be summarized: disorientation, altered interactions with owners, sleep/wake cycle reversal, house soiling, and altered activity levels (DISHA, Nielson *et al* 2001, Golini *et al* 2009, Landsberg *et al* 2010). Some dogs and cats will have increased activity or perform a behaviour repetitively, while others will have a decrease in activity levels. An alteration in appetite as well as increased anxiety may also occur in cases of cognitive dysfunction.

Diagnosis is most commonly based upon the clinical signs and findings on MRI. The most common differential diagnosis is frontal lobe neoplasia but organ disease (liver, renal) or pain from spinal/musculoskeletal systems are also possible causes. In the future, serum amyloid A in blood may be a method of diagnosis as it was found to be significantly increased in dogs with CCD compared to older dogs without or with mild cognitive dysfunction (Schutt *et al* 2015).

Treatment is aimed at slowing down the degenerative process which is thought to include inflammation as well as neuron loss. Medium chain triglycerides (MCT's) provide an alternate fuel source for the brain and can decrease clinical signs in CCD cases where the brain's ability to utilise glucose as an energy source is impaired (Pan *et al* 2010, Oulhaj *et al* 2016). The dosage for dogs is 250mg/kg daily for one week then 500 mg/kg daily for one week and if tolerated, thereafter. MCT gelcaps or soft gels can be purchased at health food stores. These doses are approximate as the published dose is 5.5-9% of food intake daily, on a dry matter basis. Most dogs do not like coconut oil and will not eat food with greater than 6.5% MCT in the diet therefore capsules can be given to reach the needed level of 6.5-9% of the diet on a dry matter basis (Manteca 2011, Pan *et al* 2018).

There are specialised diets for dogs with signs of reduced cognitive ability, instead these are also recommended in dogs >7 years-of-age for prevention: Hill's Prescription Diet b/d Brain Aging Care and Purina One SmartBlend Vibrant Maturity 7+ Formula. Omega 3 fatty acids are recommended as well for canine and feline cognitive disorder for their anti-inflammatory effects and protection from neurodegeneration with the added benefit of reduction in osteoarthritic signs and progression

(May and LaFlamme 2019). Supplementation of the diet of dogs with S-adenosylmethionine (SAMe) is recommended for its antioxidant benefits (May and LaFlamme 2019). In a blinded clinical trial, dogs supplemented with SAMe (18 mg/kg/day SAMe tosylate) had decreased mental impairment, increased activity and improved awareness (Reme *et al* 2008). A combination of nutrients with antioxidants and mitochondrial cofactors improves cognition function in dogs and cats and may be more effective than single nutrients alone (May and LaFlamme 2019, Vauzour *et al* 2017, Pan *et al* 2013). In cats, SAMe is recommended for cognitive decline at 100mg orally per day (Landsberg *et al* 2018).

Cognitive enrichment slows the progression of CCD in dogs and cats and may be incorporated into the daily life of a geriatric pet. Social interaction with others improves function as well as regular exercise, while new toys can be very helpful (Landsberg *et al* 2018, Landsberg *et al* 2012, Landsberg *et al* 2011, Schutt *et al* 2016). Pharmacologic therapy may be instituted to reduce the severity of the clinical signs. Selegiline is a monoamine oxidase B inhibitor used for the management of canine cognitive disorder (Campbell *et al* 2001). Its effects result in increased catecholamine release and dopamine production which in turn improve cognition in dogs (Dewey *et al* 2019). Selegiline has been used in cats with cognitive dysfunction and is reported to reduce vocalisation and repetitive activity, while improving affection (Landsberg 2006, Campbell *et al* 2001, Ruehle *et al* 1995). Propentofylline (xanthine derivative) may reduce dullness and lethargy in dogs and has been used in cats with reported positive results (Parkinson *et al* 1994; Gunn-Moore *et al* 2007).

Sarcopenia

Generalised skeletal muscle loss/atrophy or sarcopenia is common in older dogs and cats as they need more protein in their diet than younger individuals to maintain lean body mass (Cupp *et al* 2008, Laflamme 2005, Pagano *et al* 2015).

As dogs and cats age, protein synthesis declines and protein (as lean body mass) turnover increases (Richardson and Birchenall-Sparks 1983, Wannemacher and McCoy 1966). This results in an overall loss of lean body mass. In dogs and cats, this is associated with increased mortality (Adams *et al* 2016, Cupp and Kerr 2010). From the age of 10 to 15 years, cats loose approximately one third of their lean body mass and in both dogs and cats, increased protein intake resulted in the least loss of lean body mass (Perez-Carmargo 2004b, Cupp and Kerr 2010, Lawler *et al* 2008). For geriatric dogs, the recommendation is a mostly fish-based dog food (kibble) that is

Figure 3. Watch this vidoe of this game to enrich k9 cognition

25-30% protein, and 14-18% fat (on a dry matter basis, Kealy *et al*. 1992; Laflamme 2018, Wakshlag 2010).

Weight loss is common in older cats, in addition to loss of lean body mass. From the age of 10-12 years onward, cats require an increased energy intake (due to reduced digestion of fat) to maintain their body weight. Increasing the frequency of feeding to geriatric cats by offering more frequent meals and increasing water intake can help reduce overall weight and lean body mass loss as well as increasing protein intake (Bellows *et al*. 2016). Healthy geriatric cats require >5 grams high quality protein per kg body weight per day (Churchill 2018). For geriatric hyperthyroid cats, up to 40% of the calories may need to be a high-quality protein (Petersen 2018). In geriatric cats with renal disease, phosphorus restriction is indicated as well as closely monitoring the high-quality protein intake which may need to be restricted/reduced in azotemic cats (Petersen 2018, Churchill 2018).

Omega 3 fatty acids are recommended for geriatric dogs and cats to maintain lean body mass and should be an important part of the diet of dogs beginning from age 7 years and cats beginning at age 10 years (Hall *et al*. 2016, Smith *et al* 2011). In dogs, Omega 3 fatty acids and other nutrients may slow geriatric renal insufficiency and the development of sarcopenia resulting in longer life (Hall *et al*. 2016). Omega 3 fatty acids may abate the pro-inflammatory state that occurs with aging in cats as well as modify the progression of osteoarthritis and loss of lean body mass (Larsen and Farcas, 2014). Closely monitoring a geriatric's weight, body condition and muscle mass scores is essential to keeping them active and healthy.

Lower Back Pain in Dogs and Cats

Lower back pain is, most commonly, a slowly progressive deterioration of the lumbosacral disk that progresses to disk protrusion and in some cases instability of the lumbar-7 and sacral-1 spinal segment, hypertrophy of associated ligaments, osteoarthritis, and spondylosis resulting in narrowing of the spinal canal and foramen with compression of the lumbar-7

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nerve roots (Worth *et al*. 2009, Jeffery *et al*. 2014, Bergknut *et al*. 2012). Lower back pain is common in humans with a lifetime prevalence of 70%, and in non-chondrodystrophic dogs may be as common as 55% (Bergknut *et al*. 2012, Andersson 1999). It is most commonly diagnosed in dogs >25kg body weight and in dogs of middle to older age (beginning at >5 years of age, Danielsson and Sjostrom 1999, Suwankong *et al*. 2008). In middle-aged to older cats (approximately 9 years or older), 34% may be affected with degenerative changes found on radiographs (Kimura *et al*. 2020).

Clinical signs in dogs and cats can be variable with many animals not showing signs of lameness (Jones *et al*. 2013, Worth *et al*. 2009). Instead, alterations in behavior are common such as reluctance to work, reluctance to jump (climb furniture or stairs), incontinence, stumbling, reluctance to be touched, or increase in hiding behaviour (Jones *et al*. 2013, Danielski *et al*. 2013, Overall 2003). Physical examination of both dogs and cats for lumbosacral spinal pain, pain on extension of the hips, or pain on dorsiflexion of the tail is recommended in middle aged to older dogs and cats (Bebchuk 2017, Jones *et al*. 2013, Danielski *et al*. 2013). Progression of disk and spinal deterioration results in signs of weakness or paresis of the hind limbs, knuckling or ataxic hind limb gait, lowered tail carriage, crouched stance, and hind limb lameness, if nerve root compression is present (De Risio *et al*. 2000, De Decker *et al*. 2014). Dogs and cats may present with acutely worsening signs after jumping up or down from vehicles/furniture/obstacles or following exercise due to reduced blood flow to the cauda equina nerves (intermittent claudication) from spinal canal stenosis (Danielski *et al*. 2013, Harris and Dhupa 2008). Diagnosis of lumbosacral disease is based upon imaging and clinical signs with computed tomography or magnetic resonance imaging the preferred methods of diagnosis (Gamble *et al* 2020, Danielski *et al*. 2013, Dewey 2008). Radiographs are useful to identify instability if other modalities are not available by imaging in the mediolateral view of the lumbosacral spine with the hind limbs in full flexion and then in full extension (Da Costa and Samii 2010).

Treatment of lower back pain remains controversial in cats, dogs and humans (Worth *et al*. 2009, Silva *et al*. 2020, Danielski *et al*. 2013). In animals with no previous history of lower back pain and minimal neurological deficits, medical management may be instituted with rest for 4-6 weeks, analgesics such as non-steroidal anti-inflammatory drugs, gabapentin, and physiotherapy such as photobiomodulation, neuromuscular stimulation and/or aquatic therapy (De Decker *et al*. 2014, Giudice *et al*. 2019, Dewey 2008). For animals with recurrent signs, mild neurological deficits, or co-morbidities that make surgery unavailable, epidural injection of methylprednisolone may be performed (Janssens *et al*. 2009, Gomes *et al*. 2020). Epidural steroid injection improves the condition of dogs in 84% of cases, although 77% relapse within 6 months (Gomes *et al*. 2020). Surgical decompression is recommended in dogs and cats with progressive clinical signs, continued neurological deterioration, incontinence, failure of medical or epidural management, and in any working or sporting dog (Worth *et al*. 2019). Surgery may involve only dorsal or foraminal decompression of the cauda equina and lumbar-7 nerve roots or it may also include decompression and stabilisation with implants, or decompression, stabilisation and distraction (Gomes *et al*. 2020, Worth *et al*. 2018, Danielski *et al*. 2013, Worth *et al*. 2019). There is a trend toward improved outcome, reduced recurrence of clinical signs, and improved quality of life with surgical management versus steroid epidural and reports of excellent outcomes following surgery range from 47% to 93% (Danielski *et al*. 2013, Gomes *et al*. 2020, Worth *et al*. 2009). Which surgery is performed is still under debate and may depend on the extent of pathology found on CT scan or MRI.

Conclusions

Cognitive dysfunction, sarcopenia and low back pain are conditions that often develop slowly and insidiously over time and many owners may not recognise these conditions as they see and interact with their pets every day. Regular assessment and examination of dogs and cats once they reach 7 to 10 years of age is vital for early treatment and management of these conditions. Regular assessment of the signs of these conditions by the veterinarian can improve and prolong the life of geriatric pets.

References: *Wendy has supplied an extensive reference list. Download here >>*

21-year-old cat with CRI has SC fluids
(Photo courtesy of Anne Quain)


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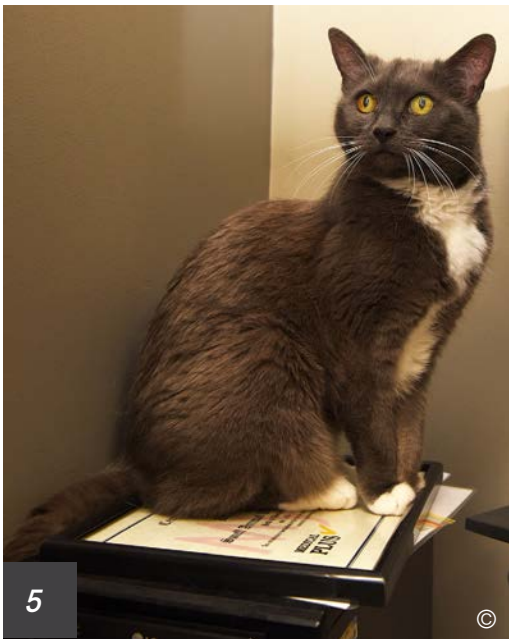
2 ©


3 ©


4 ©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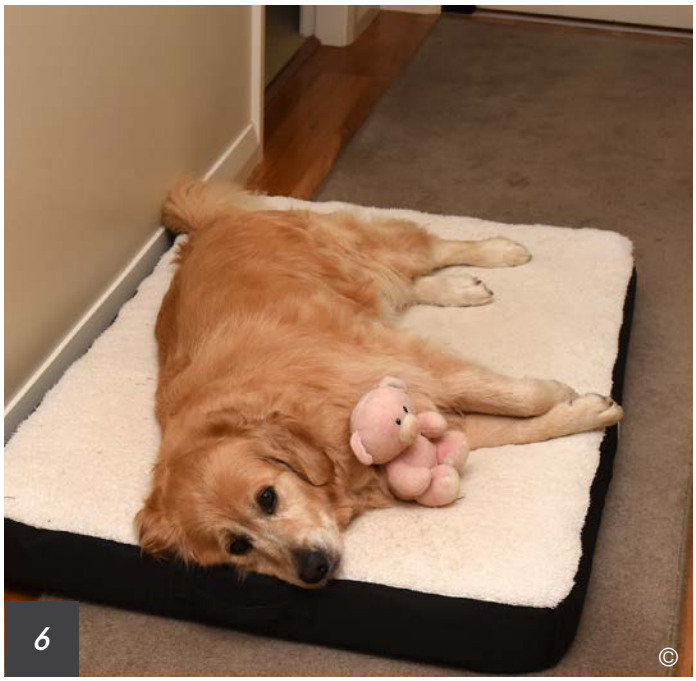

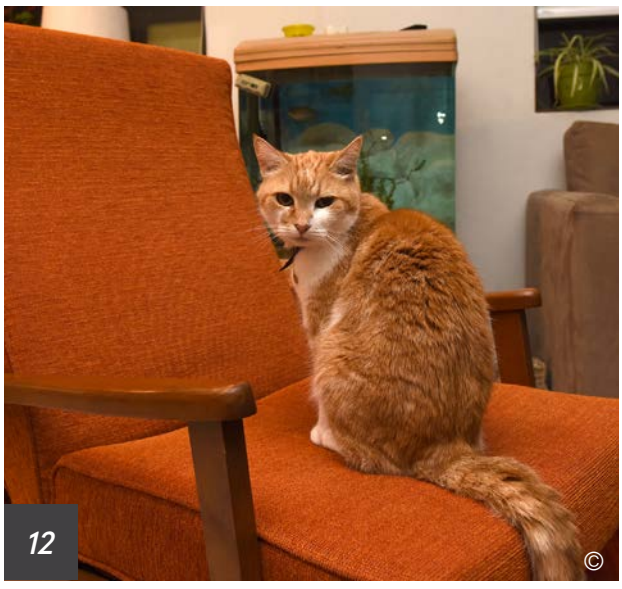

Senior pets courtesy of Anne Quain

1. Mr Bits reflecting
2. Macy Grey
3. Mr Bits having his eyes cleaned
4. Bosca in front of the fire
5. Biscuit - almost 20 years old
6. Skye going to bed for the night
7. Bosca in bed
8. Bella on the sink
9. Michael drinking from a glass
10. Mr Bits taking a pill
11. High sided litter tray for a senior cat
12. Twiggy on a chair

RUSSELL KEITH DICKENS

15/11/1928 – 22/9/2020

Russell Keith Dickens, a much beloved citizen of Blacktown and a treasured member of the Australian veterinary profession, died recently at the age of 91. Russ, likely Australia's oldest practising veterinarian, devoted his rich life to the betterment and advancement of his family, his community, the veterinary profession and animals. A man of incredible energy, he will perhaps be best remembered by the veterinary profession for his unwavering commitment to animal welfare and his dedication to advancing knowledge and understanding of Australia's wildlife. To his family and the Blacktown community he will be most remembered for his wisdom, wit, generosity of spirit and caring nature.

Born at Westmead in Sydney's West, Russ was the youngest son of Russell and Jessie. He was educated at Westmead Public School and then Parramatta High School where he excelled scholastically and at a variety of sports. It was these early years that instilled a sense of community and duty in Russ, which ensured that he never turned his back on his cherished western Sydney. He first attended the University of Sydney in the School of Dentistry but quickly became disillusioned with the prospect of looking down people's mouths for the rest of his life. He then took a position in the McMaster Laboratory at the University of Sydney investigating infectious diseases of animals. Obviously, this work struck a chord with Russ and, despite the prospect of spending the rest of his life looking into the mouths of animals, he subsequently entered Sydney's Faculty of Veterinary Science in 1948.

Life in the Veterinary School was hard going, but Russ managed to blend his scholastic activities with representing the Faculty in both Soccer and Rugby Union as well as playing rugby for the Parramatta Two Blues. In 1953, Russ completed his studies and graduated on the 27th of January 1954 with unbounded enthusiasm for the future. His final year valedictory mentioned that this graduate, possessing a subtle and often unsuspecting wit, had a future treating poodles and parrots. However, that was too limiting for Russ and he opened an all species practice, first operating from his family's home at Westmead and then in the 1960's from a purpose-built veterinary practice, 'Hillside', at Blacktown.

In those early days practice life was difficult for Russ and his wife and main support, Lorina. The day began well before dawn when Russ would service the large farm community ranging from Parramatta to the Hawkesbury River, especially the large dairies that operated along the Windsor Road and out to Freeman's Reach and Richmond. Often the day on the road ended after sunset, but then Russ would return to base and treat the myriad of pet animals brought to his door, sometimes operating past midnight. However, Russ embraced this challenge, even when his children Jenny, Helen, Sue and Rowan came onto the scene and his time became even more precious. As the practice expanded, Russ was able to employ more veterinarians to service Blacktown and the surrounding communities and his life should have become less hectic. However, his natural inquisitiveness and drive to serve people and animals meant that his enormous energy was channelled into other challenges and pursuits. These included furthering the knowledge and understanding of Australian wildlife, particularly koalas; championing animal welfare; and serving the Blacktown community in an official capacity as Councillor and Mayor.

In the 1960's, Russ had developed a strong interest in Australian Wildlife because of his care of animals in wildlife parks in Sydney, especially at Featherdale Wildlife Park and Koala Park. He quickly learned that little was known about the illnesses and treatment of these animals, particularly koalas. This frustration and desire to understand led him to enrol in a higher degree in the Veterinary Faculty at the University of Sydney. He was likely the first practising veterinarian to undertake a higher degree in koala diseases and he focussed on the rapidly developing disciplines of haematology and biochemistry in veterinary diagnosis. The road was long and full of distractions because of his growing prowess and demand as a koala expert, but he was awarded his MVSc in 1975. However, his legacy to koala health continued to grow as he played a key role in establishing the Australian Koala Foundation, as well as training and

Contents

mentoring future generations of koala researchers and practitioners. The current Koala Diseases Hub at the university of Sydney owes much to the 'grandfather' of koala health.

In 1992 Russ was awarded the Medal of the Order of Australia (OAM) for his services to Animal Welfare. Always an advocate for improving welfare for all animals, he had served on the Westmead Hospital Animal Ethics Committee for many years, but perhaps his most significant single contribution was ensuring that stray animal control in the Blacktown Municipality was of the highest standard in terms of animal welfare. In particular, Russ had an inherent love for dogs, perhaps the reason why he became a pathfinder for Canine Medicine specialization through sitting and passing examinations in the discipline in 1978 and operating as an examiner for several years for the Australian and New Zealand College of Veterinary Scientists. His last faithful dog, Cullen, was by his side towards the end.

Russ' lifelong loyalty to the Blacktown community was not only expressed through his veterinary work but also through his challenging work as a Councillor for Ward 3 from 1981-2016 and as Blacktown Mayor from 1987-1988. He worked tirelessly for his constituents and strove to improve the lot of all citizens of Blacktown. For this community work and for all the veterinary contributions, Russ was selected for an Alumni Award (Community) by the Faculty of Veterinary Science, University of Sydney in 2013. This was especially apt as Russ had been a long-term active and generous alumnus of The University of Sydney. His support and generosity were also extended towards the veterinary profession having been an active member of the Australian Veterinary Association since graduation. At a time in life when he could have chosen to take advantage of discounted life AVA membership, he declined; rather continuing to work full time at Hillside veterinary practice until earlier this year.

100 years Faculty dinner in 2010

It is with great sadness that we say farewell to this quietly spoken, wise and witty man who left an indelible mark on his family, his colleagues, his community and the Australian veterinary profession. If a man judges the worth of his life on what he achieves for others, then Russ can rest contented on a life worthy of remembrance and continued admiration. May we all follow his example.

Robert Johnson & Paul Canfield AM

Vale Russell Dickens
Lifelong member and supporter of the CVE/PGF, speaker at PGF conferences and contributor to the C&T Series.

In practice

2020 DISTANCE EDUCATION

Congratulations to the class of 2020!

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.

Australian IVAS Veterinary Acupuncture

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Beth Campbell, New Zealand
Angela Cannell, QLD
Victoria Chan, VIC
Ishtar Chan, NSW
Kin Yan Chow, NSW
Sue Collins, VIC
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Jennifer Eades, QLD
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Beijie Xu, NSW

Cardiorespiratory Medicine

Tutor: Niek Beijerink

Witchanon Dontrisanor, Thailand
May Lyn Lim, Malaysia
Marietjie Malherbe, VIC
Mick Muresanu, NSW
CHOO YIN TAN, Malaysia
Catherine Veciana, Spain
Tan Wan chin, Malaysia
Chung Hei Andrew Yang, Hong Kong

Clinical Neurology

Tutors: Simon Platt, Laurent Garosi

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Rosemary Bensley, NSW
Anne-Marie Buden, VIC
Luis Chambel, Portugal
Shirley Chow, WA
James Davies, South Africa
Sael Finney, VIC
Aiste Gradeckiene, Lithuania
Triona Kelly, United Kingdom
Lan-Hsin Kuo, Taiwan, Province of China
Wen Chi Lin, Taiwan, Province of China
Hilary Ling, NSW
Chih Yu Lo, Taiwan, Province of China
Roy Mac Gregor, Costa Rica
Samantha Maerker, Mexico
Jonathon Nott, NSW
Susanne Rodekohr, Mexico
Wasan Tangphokhanon, Thailand
Susan Tomlin, New Zealand
SUN CHUAN YANG, Taiwan, Province of China

Clinical Pathology

Tutor: Sandra Forsyth

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Rachel Griinke, QLD
Carol Ann Harte, Ireland
Melanie Hills, QLD
Marje Kuipers, United States
Jovene Lee, SA
Rebecca Welsh, QLD

Diagnostic Imaging: Abdominal

Tutor: Zoe Lenard

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Nan Choisunirachon, Thailand
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Jess Divall, ACT
Alister Douglas, SA
Anita Elise Edwards, NSW
Jaclyn Fung, Hong Kong
Charlotte Gibson, New Zealand
Nicole Hawken, VIC
Jemima Jones, NSW
Sedick Khisa, Kenya
Ryan Lane, NSW
Mário Bruno Magalhães, Portugal
Sarah Mason, WA
Alexandra McClelland, QLD

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Elizabeth Parsons, NSW
Kerri Renshaw, WA
Michelle Starr, SA
Chutimon Thanaboonnipat, Thailand

Diagnostic Imaging: Musculoskeletal

Tutor: Sarah Davies

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Ting Hey Denise Chan, Hong Kong
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Kevin Kang, WA
Sedick Khisa, Kenya
Scott Lackenby, NSW
Richard Lucas, WA
Zoe Manning, VIC
David Oswald, SA
Kerri Renshaw, WA
Steven Sath, VIC
Chutimon Thanaboonnipat, Thailand
Charmaine Verloop, SA
Shelley Wo, NSW

Diagnostic Imaging: Thoracic

Tutor: Robert Nicoll

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Siti Nur Asyikeen, Malaysia
Susan Carroll, QLD
Nan Choisunirachon, Thailand
Su Mi Choung, NSW
Ross Evans, QLD
Charlotte Gibson, New Zealand
Amy Graham, QLD
Hilary Hu, NSW
David Hughes, NSW
Sedick Khisa, Kenya
Wen-Meng (Monica) Liu, NSW
Alexandra McClelland, QLD
Kheng Boon Oon, VIC
Michelle Starr, SA
Chutimon Thanaboonnipat, Thailand
Shelley Wo, NSW
Jordan Zec, QLD

Emergency Medicine

Tutors: Trudi McAlees, Sandra Forsyth, Yenny Indrawirawan

Kate Chambers, NSW
Agus Efendi, Indonesia
Delwyn Fenby, New Zealand
Isabella Gomez, VIC
Cally Hammond-Tooke, VIC
Zhiyou Lin, VIC
Bernadine Mamaril, Singapore
Lisa McRobert, VIC
Lo Pin Hsun, Taiwan, Province of China
Sitapa Pluempreedee, Thailand
Jessica Russell, VIC
Lehgarubini Shanmuganathan, Malaysia
Dinaesh kumar Sivananthan, Malaysia
JANYAPORN THANKANSAKUL, Thailand
SU YI DA, Taiwan, Province of China

Feline Medicine

Tutors: Carolyn O'Brien, Jessica Quimby, Katherine Briscoe, Katie McCallum, Lara Boland, Myles McKenna, Rachel Korman, Sally Coggins, Samantha Taylor, Sarah Spencer, Sheila Wills, Wayne Mizon

Shayda Ahkami, United States
Mireia Balliu, Spain
Magdalena Berndt, Ireland
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Dana Snell, Canada
Silvana Sosa Portugal, Spain
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Narisara Suparatpinyo, Thailand
Sansanee Taksinoros, Thailand
Apinya Tangtanasakun, Thailand
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Minette Venter, Hong Kong
Catherine Vetter, United States
Carolina Vilches, United Kingdom
Lindsay Villalobos, Costa Rica
Harriet Wilkinson, United Kingdom
Brian Wood, United States

Internal Medicine: A Problem Solving Approach

Tutors: Jill Maddison, Sue Bennett

Kitanjali Batliwalla, VIC
Wietz Botes, Hong Kong
Conor Brand, SA
Terry Chew, SA
Katrina Couttie, VIC
Anna Coyne, QLD
Meredith Crowhurst, VIC
Lara Day, VIC
Kate Daymond, NT
Lisa Di Vitto, VIC
Anita Dukes, VIC
Kellie Farraway, QLD
Christina Felty, NSW
Alison Hayward, WA
Louyen Heng, SA
Casey James, QLD
Soon Siok Michelle Low, WA
Irene Mitry, VIC
Dawn Ong, Singapore

Brody Pullens, VIC
Chelsea Ridl, New Zealand
Steven Sath, VIC
Ilke Sen, VIC
Craig Stanghon, QLD
Caitlin Sullivan, QLD
Catherine Sutton, QLD
Felicia Tam, NSW
Caitlyn Tremble, NSW
Pascal Urlings, SA
Angeline Wong, Singapore

Internal Medicine: Keys to Understanding

Tutors: Darren Merrett, Steve Holloway, Jen Brown

Arissara Belle, Thailand
Suthasinee Boonyindee, Thailand
Giulia Cerone, VIC
Lok yan Chin, Hong Kong
Gail Chong, Singapore
Tsz Wan Natalie Chow, Hong Kong
Gareth Dunkerley, New Zealand
Chloe Friend, NSW
Caitlyn Gallagher, NSW
Kirsten Gasser, New Zealand
Tiarni Johnston, QLD
Jeyamani Kuppusamy, Malaysia
Hannah Ling, TAS
Philippa Mason, SA
Gail McDowell, NSW
Suphat Soeratanapant, Thailand
Pong Techapongtada, Thailand
Kanokphan Vongchaisri, Thailand
Sonja Vorster, QLD
Pacharawan Wanaroonwong, Thailand
Michael Yazbeck, NSW

Medical Oncology

Tutors: Peter Bennet, Katrina Cheng

Michael Hanna, VIC
Clare Jackson, WA
Lan-Hsin Kuo, Taiwan, Province of China
Rosemary Soh, VIC
Jeremy Wearne, Hong Kong
Sheree Zirafi, VIC

Ophthalmology

Tutors: Robin Stanley, Marnie Ford

Daphne Ang, Singapore
Fiona Burch, TAS
Ruth Dupingay, Philippines
Manaporn Kasornsri, Thailand
Tipsuda Krawkijprasert, Thailand
JULALUK KRUAFU, Thailand
Hoi Wun Lee, Hong Kong
Carlos Medina Torres, QLD
Gillian Sim, ACT
Nicholas Smith, Hong Kong
Arthit Sriart, Thailand
Chutimon Sunthawatrodom, Thailand
Margot Walton, VIC

Ruminant Nutrition

Tutor: Paul Cusack

Ben Ashton, NSW
Matthew Boon, VIC
Mary Chirgwin, SA
James Dawson, WA
Tamara Freitas, QLD
Susan Goodland-Smith, QLD
Eve Hall, NSW
Lauren Harrison, VIC
Jo Hoad, NSW
Rachael Holmes, VIC
Catherine Kreutz, WA
Katherine Lang, VIC
Bridie Luers, WA
Aaron Matheson, VIC
Simon McPhee, NSW
Carolyn Newberry, NSW
Sarah Rodwell, QLD
Deborah Scammell, SA
Kate Smart, NSW
Lyndell Stone, NSW
Jessie Ward, VIC

Surgery

Tutors: Chris Tan, Mark Newman, Katherine Steele, Wendy Archipow

Laree Alexander, New Zealand
John Barrett, SA
William Berry, NSW
Samuel Biddle,
Alex Browning, QLD
Leo Amor Buguina, Singapore
Enoch Chan, Hong Kong
Chloe Cheung, Hong Kong
Kaiser Dawood, NSW
Frieda De Jager, QLD
Nurul Hawadah, Malaysia
Thomas Hawthorn, NSW
Tess Holm, SA
Mary Hong, NSW
Margriet Johnson, NSW
Sarah Leber, VIC
Chloe Lefebvre, NSW
Rhys Loudon, QLD
Iris Ma, Hong Kong
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Adam McCosker, NSW
Sarah Mo, Hong Kong
Jennifer Nesci, SA
Sawinee Poon-iam, Thailand
Christopher Stott, VIC
Allison Tedesco, ACT
Adam Windel, NSW
Chi Wai Wong, VIC
Sacha Woodburn, NSW
Viola Wynne-Hoelscher, VIC

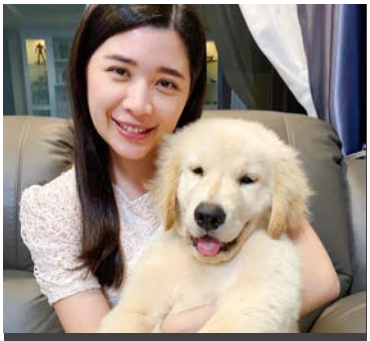
DE WINNERS: DE SUPER EARLY BIRD & EARLY BIRD DRAWS

The prize is a CVE $1,000 voucher each towards any CVE course/product valid for 2 years.

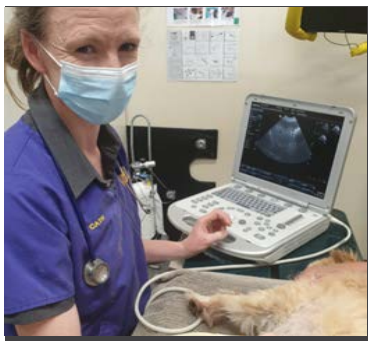
Congratulations to Nisakorn & Cath!

Enrolling before the Super Early Bird + Early Bird DE deadlines meant that all registrants made substantial savings, secured their places in the course of their choice and entered both draws.

** Nisakorn is the 44th staff member from the practice to enrol in a CVE Distance Education course.*



SUPER EARLY BIRD WINNER
Dr Nisakorn Songaksorn from Thonglor Pet International Hospital* (Thailand) is enrolled for Ophthalmology DE.



EARLY BIRD WINNER
Cath Rampton from Eastwood Vets (VIC) is enrolled for Internal Medicine: Keys to Understanding DE.

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