



Centre for Veterinary Education

#### **Control & Therapy Series**

Issue 304 | September 2021







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Veterinary Care of the Pet Bearded Dragon **45** 





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# C&T

Issue 304 | September 2021

#### Control & Therapy Series

Australia's Leading Veterinarian Forum

#### PUBLISHER

Centre for Veterinary Education Veterinary Science Conference Centre Regimental Drive The University of Sydney NSW 2006 + 61 2 9351 7979 cve.publications@sydney.edu.au cve.edu.au

Print Post Approval No. 10005007

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NOTE: Due to cost and space constraints, and the length of many reference lists, these are available for download in the complementary eBook version which is emailed to all members. Alternatively, access the ebooks at

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'I enjoy reading the C&T more than any other veterinary publication.'

**Terry King** Veterinary Specialist Services, QLD

#### Thank you to all contributors

The *C&T* Series thrives due to your generosity. If you're reading this and have been contemplating sending us an article, 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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#### Winners

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## FROM THE DIRECTOR



If I had to choose a word that was experiencing exponential growth in usage at present (ignoring the annoying ones like 'pivot' and 'unprecedented'), it would be 'community'. 'Community transmission' (of course), 'our community', 'community spirit'—the list goes on. It was the first word I thought of when taking a look at the draft of this edition of the *C&T*. Practical tips and new knowledge applied to commonly encountered problems, there's a real sense of a 'learning community' sharing information to improve patient—and clinician—wellbeing.

Tania Sundra's '10 Tips For Performing an Equine Gastroscopy' contains not only a well communicated step-by-step approach, but thoughtfully highlights traps for new players. David Hughes and Rebecca Brady contribute a revolutionary protocol for FIP, with some terrific case examples and before and after photos. Again, practical recommendations are included—like how to stage blood tests to maximise useful information without unnecessary cost.

You may have already seen Richard Malik and Mandy Craig's discussion of the use of Konakion® MM as we pre-released this in email form; many practices have already implemented this approach. Bing Zhu and Lucy Kopecny's contribution will refresh and enhance your understanding of kidney injury (with the considerate inclusion of how and what to communicate to clients). Rachel Korman offers insight into renal infarcts in cats and is featured in our 'Specialists' Corner' column.

Alice Edwards' Perspective on neuro-dietetics is food for thought (see what I did there?!?) and Robert Johnson's article on bearded dragons has so many wonderful photos it has potential for a coffee table book (albeit for a very specific audience).

Happy reading!

Simone

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## 10 TIPS FOR PERFORMING AN EQUINE GASTROSCOPY

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C&T No. 5894

Gastroscopy is the only way to diagnose gastric disease in horses. Our goal should always be to perform a safe, efficient and complete examination of the stomach. The following guide will hopefully provide some useful tips on how this can be achieved.

- In order to perform a complete examination of the equine stomach, the gastroscope should be a minimum of 3m in length. Shorter scopes make it virtually impossible to visualise the pylorus. We utilise a 3.3m gastroscope that is 13mm in diameter (Steris: Endo-i). I prefer the wider diameter as in my experience, it makes navigation to the pylorus much easier than the narrower (8-10mm), more flexible scopes. However, the downside to this wider diameter is that we are unable to gastroscope foals and small ponies.
- 2. The key to a successful gastroscopy is adequate patient preparation. We typically recommend horses are held off feed for a minimum of 12-15hrs prior to the procedure and water removed 1-2hrs beforehand. An automated text message is sent to clients at 3pm the day before to ensure this is clearly stated. Some horses may need to be muzzled to prevent them from eating their bedding or faeces. If owners are transporting their horses to the practice, ensure they do NOT hang a hay net in the float or graze their horses on the lawn whilst waiting for their appointment!
- 3. Prior to the procedure, it is important to ensure all your equipment is working appropriately. There is nothing worse (or more embarrassing!) than having a sedated horse in front of you, only to find that your scope is not flushing properly, or the light source is broken.

- 4. Appropriate sedation is critical. We typically use 0.01mg/kg of detomidine and find this adequate for a complete gastroscopic examination. The procedure should take anywhere from 5-15 mins. We will sometimes add an opiate (butorphanol) depending on the temperament of the horse.
- 5. A competent handler is important. Communication between the handler (passer) and the vet (driver) is even more important. The driver needs to communicate clearly with the passer on what they are trying to achieve e.g., 'please move forward slowly until we get to the lesser curvature'(*Figure 2*). Performing the procedure with someone who is familiar with your technique will make life considerably easier.
- 6. In order to protect the scope from damage and prevent it from retroflexing into the mouth, we always pass a short, 1m piece of outer tubing through the nares. Once the tubing is in place, it is tied back to the halter with some electrical tape to ensure it does not inadvertently travel into the nares during the procedure. We utilise a twitch when introducing the tubing but, once in place, the twitch is removed as most horses tolerate the procedure well.
- 7. The cardia is found at approximately 1.9m from the nares in the average size horse. Once there, we will start to slowly distend the stomach with some air. How much air to use is somewhat controversial, but we will usually distend until the rugae start to flatten.
- 8. Air can be introduced into the stomach in a number of different ways. Some scopes will have very effective air pumps, but most are poor quality, and you will lose considerable time trying to distend the stomach this way. An alternative is to connect a 10L garden sprayer bottle to the biopsy port on the scope with some arthroscopy tubing. This method is cheap, effective and will get the job done.
- Once the stomach is adequately distended, I will always navigate to the pylorus (*Figures 3 & 4*) first for a couple of reasons:
  - i. the pylorus is often the trickiest area to examine and requires the most time; and
  - examination of the squamous mucosa usually requires rinsing of the walls. This adds more water to the stomach, which may make visualisation of the pylorus difficult.

1965

Est.



Figure 1. Gastroscopy is the only way to diagnose gastric disease in horses

10. Once I have inspected all the key areas, and before removing the scope, I will deflate the stomach via a suction pump. Not all vets advocate this, but it is something I have always done. A cheap and easy method to remove air is via an air mattress hand-pump (with a deflate option) connected to the biopsy port. The deflation takes all of 30 seconds and removes any additional air that was introduced.



Figure 2. View of the lesser curvature

Every veterinarian will have their own technique for performing a gastroscopy. If you are only just starting to gastroscope horses, remember that safety is paramount, and a complete examination of the stomach is always the aim. Performing the first few with an experienced veterinarian will be helpful, until which time you develop your own technique. Initially, the procedure may seem daunting but the more you practice your technique, the better and more confident you will become.

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Figure 3. View of the normal squamous and glandular mucosa separated by the margo plicatus



Figure 4. View of the pylorus

## PURPLE MUSH FOR CANKER

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C&T No. 5895

In June 2019, a 10-year-old pregnant mare had a biopsy taken from a proliferative lesion protruding from the frog and adjacent sole, through a defect roughly 2x 4cm. The lesion had been present for many months.

Canker was suspected and confirmed with histology.

#### MACROSCOPIC EXAMINATION

A roughly ovoid piece of firm grey tissue, 35x23x20mm.

#### MICROSCOPIC EXAMINATION

There is proliferative epidermis with marked parakeratotic hyperkeratosis. The epidermis is supported by a dermis which has extensive variably mature granulation tissue. There are several areas of ulceration, neutrophil exudation, and intact areas have keratinocytes with ballooning degeneration as well as serum lakes and groups of neutrophils. Areas of the epidermis are necrotic and are colonised by large numbers of mixed bacteria.

#### SUMMARY

Proliferative, necrotising and neutrophilic pododermatitis with parakeratotic hyperkeratosis and granulation tissue.

#### COMMENT

This combination of histologic changes is suggestive of equine canker. The pathogenesis of canker is poorly understood and there are contributing factors including husbandry, genetic predisposition, infectious agents (spirochetes and anaerobic bacteria) and bovine papilloma viruses have been implicated.

Conventional recommendations for treatment include removal of all over-lying tissue/horn, and application of various topical treatments daily, often for weeks or months.









Canker on the ventral aspect of the hoof of 2 different horses (not this horse) pictured above.

As this horse was 2 hours from town, and pregnant, I looked for alternatives, and decided to try Purple Mush, from Well Horse Products in California.

The active ingredient is resin from *Croton lechleri*—a native tree from South America.

It took a month to get hold of the Purple Mush. It was then applied and bandaged once every 5 days for 4 treatments with no removal of overlying horn. The lesion was reportedly healing well by the time the 4th treatment was applied and has not recurred 18 months later.

The horse's foot after the biopsy specimen was collected, before treatment commenced with 'purple mush'

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## A FELINE INFECTIOUS PERITONITIS TREATMENT PROTOCOL

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C&T No. 5896



David Hughes graduated from the University of Sydney in 2010 and became a practice owner in 2013 at Concord Veterinary Hospital. The hospital has a solid historical affiliation with The Cat Protection

Society of NSW, providing a high feline case load. David and his team have developed an interest in canine and feline internal medicine and ophthalmology. They have so far treated over 20 feline infectious peritonitis cases utilising this constantly evolving protocol.



Rebecca Brady graduated from the University of Sydney in 2012 and immediately went into co-ownership of Concord Veterinary Hospital with David Hughes. She has a strong passion for feline internal

medicine, behavioural medicine and feline welfare. She has gained a keen interest in feline imaging—especially abdominal ultrasound. She finds shelter medicine particularly rewarding which is strengthened by Concord Veterinary Hospital's strong association with The Cat Protection Society of NSW.



Figure 1. A 1-year-old M(N) cat who was originally diagnosed with lymphoma. The cat presented with enlarged mesenteric lymph nodes, enlarged liver, jaundiced, low albumin and high globulin and was subsequently diagnosed with dry FIP—now over 6 months in remission after completing 84 days of remdesivir. He was diagnosed based on his response to remdesivir

Note: Remdesevir has been approved as a drug for IV treatment of COVID-19 by the TGA, and BOVA Compounding have reformulated it for off-label use IV and subcutaneously to treat cats with FIP.

#### Overview

Feline infectious peritonitis (FIP) is a fatal disease in cats whereby the relatively benign feline enteric coronavirus undergoes a mutation within a cat's own immune system—specifically within their macrophages—resulting in a disease known as FIP.

This mutation occurs in roughly 1 in 5,000 cats. FIP can be categorised broadly into two syndromes wet FIP and dry FIP. Wet FIP is characterised by single or bi-cavitary effusions. Dry forms of FIP are much harder to diagnose as symptoms and changes on physical examination can be more subtle. Some of the more obvious symptoms that occasionally occur in cases of dry FIP include jaundice, enlarged mesenteric lymph nodes and uveitis—the disease often mimics lymphoma. FIP generally affects kittens and cats under the age of one; however, it can affect cats of all ages.

#### Diagnosing FIP

The diagnosis of FIP can at times be challenging. The classical symptoms of wet FIP are a high fever non-responsive to anti-microbials, single



Figure 2. A cat with wet FIP presenting with severe jaundice and anaemia

or bi-cavitary effusion, icterus and increased globulins. Cases will often also be anaemic, hypoalbuminaemic and have a significant neutrophilia. The same set of findings can be said for dry FIP in the absence of an effusion—however, with additional clinical signs which may include enlarged mesenteric lymph nodes, enlarged liver, changes to the kidneys on ultrasound, pulmonary granulomas, and central and ocular signs if there is a neurological or ocular component such as uveitis, blindness, and seizures.

Often a presumptive diagnosis of FIP can be made based on the above findings alone. Like any disease, it is up to an individual clinician as to the



Figure 3. Icteric blood after centrifugation from the above jaundiced wet FIP cat. Note how anaemic the sample appears to be also

level of proof they require before commencement of treatment of FIP.

Further diagnostic testing can be carried out in an attempt to confirm the diagnosis of FIP, and some clinicians feel these tests need to be run prior to commencement of treatment. Perhaps a 'happy medium' may be to make a presumptive diagnosis based on symptoms and laboratory findings above, then collect further samples for further analysis (outlined below) but commence treatment whilst waiting for results as some of these results can take up to 7 days.

It is important to note that there is no 100% accurate test for ruling in or out FIP and there will be times when it will come down to a clinician's judgement to diagnose FIP in a feline patient. Often it comes down to a clinician's level of confidence and experience treating FIP.

Further testing, when undertaken, can include:

- Coronavirus PCR on cavity effusion by IDEXX laboratories
- Immunofluorescence assay on cavity effusion by the University Veterinary Teaching Hospital Sydney (UVTHS)
- Immunocytochemistry on FNA samples of tissue aspirates by UVTHS, and
- Immunohistochemistry can be performed on tissue samples collected at ex-lap by UVTHS.

To recap—the common findings with both wet and dry FIP:

- 1. Uni or bicavitary effusion (Wet only)
- 2. High fever not responsive to antibiotics
- 3. Hyperglobulinaemia
- 4. Hypoalbuminaemia
- 5. Icterus
- 6. Non-regenerative anaemia
- 7. Neutrophilia

#### The Concord Veterinary Hospital Treatment Protocol

This article will discuss the use of a drug called remdesivir.

As per previous communication distributed by Richard Malik, one way to 'diagnose' FIP in cats, especially dry forms of FIP, is to trial patients on



Figure 4. Collecting abdominal fluid from a wet FIP patient to submit a sample for immunofluorescence assay to VPDS, SSVS.

3–4 days of remdesivir and if a rapid improvement in clinical signs is seen (reduction in mesenteric lymph node size, resolution of fevers, regaining of appetite) then the diagnosis of FIP is essentially confirmed.

#### What is Remdesivir?

Remdesivir is a broad-spectrum antiviral developed by the pharmaceutical company Gilead. Remdesivir was originally developed to treat Hepatitis C and Ebola Virus; however, it was fast-tracked and given approval in many countries worldwide for the treatment of COVID-19. Its use now in COVID-19 patients is controversial; however, it is showing amazing promise for the treatment of FIP in cats.

#### **Treatment with Remdesivir**

Treatment with remdesivir is for 84 days by subcutaneous injection, so it requires a committed owner or a sympathetic veterinarian geographically



Figure 5. A F(S) Bengal cat with ocular and central FIP before treatment with remdesivir. Note the dilated pupils. This cat was centrally blind prior to treatment

close by. Remdesivir can be sourced readily from BOVA Compounding Pharmacy who ship Australiawide normally overnight. The cost of the drug is significant; the vials are 100mg/vial at \$275/vial inclusive of GST. The final concentration is 10mg/ mL. Needless to say-treatment of FIP by an owner takes a lot of financial and emotional commitment.

#### The First 3 to 4 days

The earlier treatment is commenced, the better the outcome—and this cannot be overstated. There are two options for commencing treatment and like most things in life it often comes down to budget.

#### 1. Subcutaneous Injections

If a patient is stable and eating—subcutaneous injections can be commenced at a dose rate of 10mg/kg and the patient checked daily for the first 3–4 days. This is a perfectly acceptable routine for a well-hydrated patient who is eating, and is the more financially sustainable option.

#### 2.The intravenous Route

If a patient is unwell, or neurological or ocular FIP is suspected, then they should be hospitalised, placed on intravenous fluids, run at a quarter to a half maintenance rates (Hartmann's or similar is fine). The reason for such a low rate will become clear shortly, and remdesivir administered SLOW IV (over 10 minutes is fine) for 3–4 days. We have found if you can maintain your catheter to a fourth day it is great to get a fourth intravenous dose into your patient; however, if there is pain at the cannula site or thrombophlebitis, then 3 days is fine.



Figure 6. The same Bengal some 4 – 6 weeks later after treatment with remdesivir with a normal ocular exam and apparent full vision

The downside to administering remdesivir intravenously is obviously having to hospitalise the patient, the added expense to the owner which is not insignificant and also there is an increased risk (10% risk) of developing or worsening of a pleural effusion with the use of intravenous remdesivir, mostly in wet forms, which then requires close monitoring by either regular thoracic radiography or T-Fast ultrasonography daily or twice daily (we prefer twice daily).

The benefits of intravenous remdesivir are you get a high anti-viral dose to target the virus for the initial 3–4 days, you get to observe your patient and more often than not get to see the fever break in front of your eyes and to see a near moribund patient 'come back to life' and show signs of improvement daily!

Obviously, this is a conversation to be had with each individual owner and for the clinician to make on a case-by-case basis. See below for other considerations before deciding which patients would or would not benefit from intravenous remdesivir therapy.

Cats receiving intravenous remdesivir do not need

to be in a 24-hour facility—remdesivir could be administered intravenously in the mornings so a clinician then has the whole day to observe their patient. We have also heard examples of veterinary hospitals administering the intravenous remdesivir and then sending the patient home for the night with the indwelling intravenous catheter in place so the owner can monitor the cat for the night in case of emergency and to keep costs down.

#### Days 5 to 84

Remdesivir needs to be given at a dose of 8–10mg/ kg subcutaneously once daily for 84 days in total. We would encourage dry forms to be dosed at 10mg/kg (ocular and neurological forms often require 15mg/kg) and wet forms 8mg/kg. In our experience there is a tendency for vets to reduce the dose of remdesivir prematurely. **Do not dose to lean body mass or try and assume what a** <u>kitten might weigh without its effusion</u>—kittens metabolise drugs faster so need a higher dose. Simply dose them for their weight—keep it simple.

Do things to encourage success in your patients for injections. For example; remdesivir is stored in the fridge. However, it is fine for it to come to room temperature before injection; this will help minimise the pain with the injection. We have found 22G needles minimise pain for injection; however, some cats prefer 23G or 25G—it really is a game of trial and error.

Some cats need to come into the vet every day for their injection. Even this shows serious

commitment by the owner! Furthermore, some patients require the use of buprenorphine prior to their injection, especially as they approach the end of the course of their 84 days. Gabapentin orally prior to injection has been trialled with less success but is still worth a try in some cases.

It is worth noting at this point we are often dealing with kittens here – so ensure they are being weighed regularly and their dose of remdesivir is being adjusted upwards accordingly to 8–10mg/kg.

#### Monitoring

Do not fall into the trap of doing blood work on days 1-3 after commencing treatment—nothing will have changed.

Once again, the bloods that are performed depend on the financial limitations of your client. Ideally, we like to perform a CBC and Biochemistry panel (including bilirubin and globulins) at week 4, week 8 and week 12. We are looking for resolution of anaemia, reduced neutrophils, normal albumin and globulin and normal bilirubin.

In theory you could also simply perform a PCV, TPP and a globulin level. This will give you an idea of the anaemia, the colour of the serum to check if your patient is still jaundiced, the albumin and the globulin level by simple calculation.

#### When Does Treatment End?

We get asked this question a lot.

#### Treatment ends when:

- ALL blood parameters are WELL within normal limits including globulins.
- 2. All effusions are absent.
- 3. Lymph nodes are normal
- 4. There are no central/ocular signs.

At this point, we call them `cured'.

#### But How Long Do They Last and How Many Have Died?

It is still extremely early days. The answer is we simply do not know. We estimate the efficacy of remdesivir to be 80-95%. Wet has a better





Figure 7. A M(N) 12-week-old domestic shorthair with dry FIP – they generally just look 'poorly' with a fever that persists

prognosis than dry for example, and neurological has a worse prognosis than non-neurological.

We will only comment on our cases as we have developed our regime. Of some 20 cases—1 case was lost as it presented moribund and unable to be saved, 1 case was lost that had concurrent FIV, the FIP was 'cured'; however, the cat went on to develop mediastinal lymphoma, and 1 kitten was put to sleep for presumed septicaemia. The other cases are at various stages of remission or still in treatment.

It is worth noting—we have 2 cases currently, both dry—where we have been unable to reduce the globulins to a normal level beyond 84 days, so we have had to increase the dose to 15mg/kg, and both are well over 110 days treatment but are doing well. The likely cause for both of these prolonged and `unsuccessful-so-far' cases has been a reduction in dose from 10mg/kg to 6mg/kg. We would advise against this—especially in dry forms.

## 10 Important Points About the Use of Remdesivir:

**THE MOST IMPORTANT POINT TO NOTE:** 10% of cats receiving intravenous remdesivir can develop pleural effusions—especially wet form. Therefore, cats receiving intravenous remdesivir need to have serial radiographs and/or skilled T-Fast ultrasonography on a daily or bi-daily basis. Pleural effusions are more likely if a clinician pushes the intravenous fluid administration beyond maintenance—we prefer to keep the fluid rate below half maintenance.

Figure 8. The same domestic shorthair cat with dry FIP just 4 weeks into treatment with remdesivir

- 2 Many patients will become quiet for 1–3 hours after receiving remdesivir intravenously—the exact mechanism is unknown but do not be too alarmed if this occurs.
- 3 Wet cases: <u>the ABDOMINAL effusion will</u> often get worse before it gets better. This is a good sign. It is recommended to NOT drain the abdominal fluid unless to get diagnostic samples or for urgent therapeutic reasons (e.g. if dyspnoea is severe due to pressure on the diaphragm).
- 4 Globulins will often go up at the first blood test before they begin to come down. Do not get disheartened.
- Many vets are concerned about the reports of elevations in kidney and liver parameters with the use of remdesivir. There have been a few reports of elevations in SDMA and occasional reports of elevation in ALT with doses of remdesivir as high as 15mg/kg that resolved once the dose was resumed to normal. The authors have had 2 cats on 15mg/ kg of remdesivir and has not personally seen elevations in ALT (but have not checked SDMA for various reasons).
- Antibiotics: Often FIP cats have comorbidities with infections which require treatment with antibiotics. Infections such as Mycoplasma are common. Antibiotics such as doxycycline are often a good choice. Other antimicrobialsif indicated -do not appear to be contraindicated.

Η

<sup>7</sup> NSAIDs to bring fevers down are often NOT required with the use of remdesivir.

B The concurrent use of corticosteroids with remdesivir is not strictly contraindicated; however, it is not recommended. If your patient is on corticosteroids we would recommend weaning them as quickly as is safe to do so.

Remdesivir can occasionally sting as an injection, so our clients need our help with respect to teaching them how to administer the drug and pointers on how to minimise the sting (needle choice size, allowing the drug to come to room temperature prior to injection, utilising medications such as buprenorphine prior to injection). The drug itself doesn't appear painful—it is the repeated injections and the amount of liquid that needs to be injected daily.

Often it can be beneficial to a patient to commence a treatment trial of remdesivir to see if a presumptive diagnosis can be made of FIP or whilst waiting for further diagnostics to return from the lab.



#### eBook download C&T 5883 Feline Infectious Peritonitis (FIP)–Now a treatable disease.

Jane Yu, Merran Govendir, Sally J Coggins & Jacqueline M. Norris, Issue 303 June 2021.

#### PLEASE CONTACT US if you'd like to be a part of these trials

Two clinical trials for naturally occurring FIP infected cats have commenced at the Sydney School of Veterinary Science at The University of Sydney and are recruiting Australia-wide:

- one looking at mefloquine, with and without interferon
- the other looking at Remdesivir

#### And/Or you need advice for a suspected or confirmed FIP Case

Jacqui Norris at jacqui.norris@sydney.edu.au Sally Coggins at sally.coggins@sydney.edu.au

## Vale Professor Frazer Allan 1966-2021

By Professor lain Young Dean, Faculty of Science The University of Sydney



It is with great sadness that we record the sudden passing of Professor Frazer Allan, from a heart attack, on Thursday 24 June 2021. Frazer joined the University of Sydney as Head of School and Dean of the School of Veterinary Science in 2018.

Our heartfelt sympathies go out to his wife Leith and his children Sophie, Fergus, Phoebe and stepson Felix. He is also survived by parents, siblings and extended family in Australia and New Zealand.

Frazer brought the worlds of private practice and veterinary education closer together during a distinguished career in the veterinary profession.

Born in Auburn, NSW in 1966, Frazer graduated with a Bachelor of Veterinary Science from Massey University, New Zealand in 1993 and completed a PhD in clinical nutrition. He worked in private practice for many years, honing his skills and developing a vast array of hands-on experience as a successful veterinarian.

From 2004 to 2009, he was Director of the Massey University Veterinary Teaching Hospital. During his time as head of the University's Institute of Veterinary, Animal and Biomedical Sciences (2009 to 2015), Frazer was pivotal in the roll-out of an innovative new globally accredited veterinary curriculum, placing it amongst the top in the world.

He will be greatly missed.

Note: Details of a memorial to commemorate Frazer's life will be shared when they are finalised.

#### Winner Entitled to a CVE\$100 voucher

## 'BILLY' - THE FIRST CASE OF *EHRLICHIA CANIS* DIAGNOSED IN NEW SOUTH WALES

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C&T No. 5897

**'Billy' is a 4-month-old Red Heeler puppy** that presented to Dr Nicole Mace at Moruya Veterinary Hospital in December 2020 (Figure 1). He had recently been adopted by new owners on the South Coast of NSW after spending a few weeks with a foster carer in Sydney following relocation from the Northern Territory. His presenting signs were lethargy, reduced appetite and a 'pot belly' for the previous one week. There was no history of access to rat bait poison or trauma.

Billy was bright during the initial consultation but less bright than expected for a normal Red Heeler puppy. **He had pale pink mucous membranes, was slightly tachypnoeic (respiration rate 28-30) and slightly hyperthermic (rectal temperature 39.5°C). He was noticeably pot-bellied and was tender on abdominal palpation.** The major differentials for Billy at this stage, given his recent travel history, were canine monocytic ehrlichiosis (CME), caused by the Gram-negative intracellular rickettsial bacterium *Ehrlichia canis* and an intestinal worm burden.

Initial in-house blood testing revealed Billy to be anaemic (PCV = 0.11 L/L, reference range [RR] 0.37-0.55; absolute RBC count =  $1.78 \times 10^{12}$ /L; RR 5.50 - 8.50), thrombocytopenic (platelet count 33 x 10<sup>9</sup>/L; RR 165 - 500), and leukopenic due to a lymphopenia (lymphocytes  $0.49 \times 10^{9}$ /L; RR 1.00 - 4.80) and monocytopenia (monocytes  $0.10 \times 10^{9}$ /L; RR 0.20 - 1.50). He had a prolonged activated partial thromboplastin time (APTT) (125 sec; RR 75-105). Abdominal radiographs revealed hepatomegaly and abdominal ultrasound did not detect any free fluid in the abdomen. An in-house faecal float was performed which was negative.



Figure 1. Billy

Serum ( $\geq$  1 mL) and an EDTA blood sample ( $\geq$  2 mL) was sent to the Elizabeth Macarthur Agricultural Institute (EMAI) for *E. canis* antibody testing and PCR testing. In the meantime, a fresh whole blood transfusion was arranged; this increased Billy's PCV from 0.11 to 0.20 L/L (total volume = 160mL of fresh whole blood). Maintenance crystalloid therapy (Hartmann's) was initiated following the blood transfusion, and doxycycline treatment commenced (5mg/kg BID; alternatively 10mg/kg SID could have been dispensed) while awaiting the *E. canis* results.

Billy tested both serologically positive (ELISA result of 1.49; results of > 1.1 are considered positive) and PCR positive ( $C_{\tau}$  value of 33.1), confirming current *E. canis* infection. Blood sent to the Australian Centre for Disease Preparedness (ACDP; previously Australian Animal Health Laboratory, AAHL) by EMAI for confirmatory *E. canis* immunofluorescence antibody testing (IFAT) was positive at a dilution of 1:80.

Doxycycline treatment was continued for four weeks. One week after the commencement of doxycycline therapy Billy's PCV was 0.32 L/L, and at the end of his four week course of antibiotics his PCV had increased to 0.38 L/L. Five weeks after he initially presented, and one week after the cessation of doxycycline treatment, Billy tested antibody negative and PCR negative for *E. canis* at EMAI. Billy's platelet count, however, was still abnormally low at the end of his course of doxycycline (91 x 10°/L), and it took four months for his platelet count to finally return to normal levels (Figure 2, where results below the red dotted line indicate a platelet count of <165 i.e. thrombocytopenia).

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During this time there were concerns about Billy developing the chronic form of CME (see later), or immune-mediated thrombocytopenia secondary to CME, and prednisolone therapy was considered (but not dispensed). Fortunately, his platelet count was normal at his four month check-up without any further treatment.

## Key points that we would like to highlight from Billy's case:

- E. canis is transmitted by the brown dog tick (BDT; Rhipicephalus sanguineus). The BDT requires hot, dry conditions to survive, and does not survive south of about Port Macquarie on the east coast of NSW (www. sciencedirect.com/science/article/abs/pii/ S1877959X19302262). Therefore, E. canis will only become endemic in areas with conditions that suit survival of the BDT; for example the Kimberley and Pilbara regions of Western Australia (WA), throughout the Northern Territory (NT), the north of South Australia, and possibly western Queensland. With current climatic conditions, <u>E. canis will not become</u> endemic in Melbourne, Sydney, or Tasmania.
- 2. However, there is significant movement of dogs from BDT-endemic areas to the rest of Australia. Rescue groups have been set up exclusively for the rehoming of dogs from remote communities in WA and the NT.

A critical question to ask during all consultations, therefore, is 'Has your dog travelled recently?'

- E. canis infection is currently a notifiable disease in all states of Australia. Each state veterinary laboratory is responsible for all testing in that state and a copy of all results is automatically sent to the respective Chief Veterinary Officer (CVO). These results are compiled and national statistics are released quarterly by Animal Health Australia (animalhealthaustralia.com.au/supportingmarket-access/). The latest results available (Q4, 2020) showed that 351 dogs were tested for *E. canis* between October and December 2020, with 102 dogs testing positive (70 in the NT, 31 in WA and 1 in NSW – Billy).
- 4. Despite Billy testing positive for *E. canis* in NSW, Australia's National Animal Health Information Program <u>concluded</u> that `an epidemiological investigation of this case indicates infection most likely originated in the Northern Territory <u>prior</u> to the animal's importation into NSW'. Although Billy was diagnosed with CME in NSW, because of his recent travel history from the NT where *E. canis* is now considered endemic in some areas, he was not considered to be a locally acquired infection. Therefore, local transmission of *E. canis* in NSW is still yet to be documented.
- 5. Since infection with *E. canis* is currently notifiable, all testing for *E. canis* is currently paid for by the federal government from its notifiable diseases budget. Veterinarians can contact their state veterinary laboratory for more information. NSW veterinarians can find more information here www.dpi.nsw.gov. au/biosecurity/animal/humans/ehrlichiacanis/sample-submission-guidelines and the

submission form to be used here www.dpi.nsw. gov.au/\_\_data/assets/pdf\_file/0004/1254595/ *ehrlichia-canis*-surveillance-investigationform-dogs.pdf

- 6. CME has three possible phases of disease: acute, subclinical, and chronic. Severity of disease can vary considerably among dogs. The incubation period for the development of acute disease is about 1–3 weeks, although the chronic form of ehrlichiosis may not manifest until months or years after infection. For more information visit the Primefact sheet www. dpi.nsw.gov.au/biosecurity/animal/humans/ ehrlichia-canis/ehrlichia-canis-pf developed by the NSW Department of Primary Industries. We believe Billy presented with an acute form of the disease.
- 7. Billy's rapid clinical improvement and negative *E. canis* antibody and PCR results one week after finishing a four week course of doxycycline suggested a rapid response to treatment, and hopefully complete eradication of *E. canis* infection. Monitoring response to treatment is not always this simple, however. Given the ability of the organism to sequester in tissues and be absent from circulation, <u>a</u> <u>negative PCR result does not necessarily</u>

indicate that the dog has successfully cleared the infection. Similarly, serology ELISA after treatment is not useful, <u>as antibody titres</u> <u>can be expected to stay high for months to</u> <u>years after treatment.</u> The NT has produced an excellent document to help veterinarians interpret diagnostic results for *E. canis* testing nt.gov.au/\_\_data/assets/pdf\_file/0007/979720/ e-canis-guidelines-for-vets.pdf

8. Dogs at-risk of *E. canis* infection should be on regular tick control all year round. Tick repellent products (e.g. Seresto® tick collar, Killtix<sup>®</sup> tick collar and Advantix<sup>®</sup> Spot-on) are most effective at preventing E. canis infection since they have the fastest speed of kill, and do not require the tick to attach and feed from the dog to be killed, so are therefore recommended for use in individual animals (www.amrric.org/resources/view/ tick-prevention-for-dogs-and-cats/). Chewable acaricidal products (e.g. Nexgard®, Bravecto<sup>®</sup> and Simparica<sup>®</sup>) do not always kill ticks in time to prevent E. canis transmission (parasitesandvectors.biomedcentral.com/ articles/10.1186/s13071-016-1636-9), but are easier to administer en masse, and therefore are recommended for community-wide tick and tick-borne disease risk reduction.

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Est. 1965



Figure 3 & 4. Billy is now living a relatively normal life, although his new owners have reported that he does tire easily with exercise compared to other dogs

9. If possible, ticks (www.dpi.nsw.gov.au/ biosecurity/animal/humans/ehrlichia-canis/ sample-submission-guidelines) should also be collected from the affected dog, placed in a small container (e.g. a urine specimen jar or a plain tube) with 2 mL of 70% ethanol and submitted to the state veterinary laboratory with the collected blood samples ( $\geq$  1 mL serum and  $\geq$  2 mL EDTA blood) for tick identification *and E. canis* PCR testing. If ethanol is not available, submitting ticks dry will also suffice.

## Don't miss the WebinarPLUS & WebinarLIVE! recordings



#### Pain & Behaviour WebinarPLUS (Aug 2021)

Presented by renowned UK behaviourist and world-wide leader in the discipline of veterinary behavioural medicine Dr Sarah Heath BVSc PgCertVE DipECAWBM(BM) CCAB FHEA FRCVS,

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## Small WHAT IS YOUR DIAGNOSIS? SUBLINGUAL LESION IN A CAT

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Randolph Baral BVSc MANZVSC(feline) PhD

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Paddington Cat Hospital 210 Oxford St Paddington NSW 2021 t. +612 9380 6111 C&T No. 5898

#### History

An 11-year-old male neutered Abyssinian cross was presented for hyporexia of two days duration; the cat would go to the food bowl, take a few nibbles, and then stop which was different from this cat's typical enthusiastic eating.

On examination, a 2 cm irregular lesion was recognised rostrally, under the tongue: the lesion was a similar colour to surrounding mucosa with focal creamy-white nodules (*Figure 1*).

The cat's chin was mildly swollen.

The body condition score was a 5/9 and a loss of 220 g in body weight was noted from the visit one month prior. There was evidence of dental disease on oral examination. The heart sounds were normal, and the rate was 200 bpm.

#### Questions

- 1. What diagnostic tests could be considered?
- 2. What is your diagnosis?
- 3. What further diagnostic tests could be considered?
- 4. What is this cat's prognosis?

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Figure 1. Appearance of sublingual mass at presentation



Figure 2. Close up of the sublingual lesion

## 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;<sup>1</sup> 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.



## Small USE OF KONAKION®MM FOR ANTICOAGULANT RODENTICIDE POISONING

#### Richard Malik<sup>1</sup> & Mandy Craig<sup>2</sup>

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C&T No. 5899

At the moment, there is a mouse plague in Western NSW leading to widespread use of long-acting potent anticoagulant rodenticides. As a result of this, secondary intoxication of cats and dogs is common and important. There have been numerous articles in the newspapers and online, the most recent featuring veteran clinician and workaholic, Ross Pedrana – see below.

#### **ABC RURAL**

## Mouse plague causes national critical shortage of vitamin K for poisoned pets

**NSW Country Hour** / By Lucy Thackray and Jennifer McCutcheon

Posted Thu 3 Jun 2021 at 8:05pm



Dubbo vet Ross Pedrana and his dog Digger have both saved poisoned dogs during the mouse plague. (Image supplied)

#### **Source:** abc.net.au/news/rural/2021-06-03/ mouse-plague-leads-to-poison-antidoteshortage/100188764

These cases can be tricky. Traditionally, we have given these patients vitamin K (e.g., Koagulon) by subcutaneous injection, and fresh whole blood or fresh frozen plasma. The problem of course is finding a blood donor or fresh frozen plasma in a timely manner. The reason why vitamin K was given subcutaneously was that the older preparation of Konakion<sup>®</sup> and Koagulon<sup>®</sup> solubilises the vitamin K in polyethoxylated castor oil (Cremophor®—the same stuff that was used to get Alfaxan<sup>®</sup> into solution in Saffan<sup>®</sup> for those vets who were practising before there were smart phones and the internet). The Cremophor<sup>®</sup> is very good at causing anaphylactoid reactions, especially in the dog.



Figure 1: The old formulation of vitamin K solubilised in polyethoxylated castor oil (Cremophor®). Can be given IV, but typically causes anaphylactoid reaction, especially in the dog. So, traditionally given by subcutaneous injection, where absorption is slow. Note IM injections are not recommended in these cases because of the risk of inciting bleeding

However, there is a new formulation of Konakion<sup>®</sup> (Konakion MM) where the vitamin K is solubilised using micelle technology. THIS IS THE WAY TO GO!

Each ampoule contains the active ingredient phytomenadione 10 mg/1 mL in a mixed micelles (MM) solution (the micelles are composed of glycocholic acid and lecithin in an aqueous solution). It is stable in air but decomposes on exposure to light.

#### It is dirt cheap-5 vials for \$15 or near enough.

The human guidelines state: Slow IV injection is reserved for potentially fatal haemorrhage due to overdosage of anticoagulants of the coumarin and indandione series. Konakion® MM should not be diluted or mixed with other injectables except, where appropriate, into the lower part of the infusion set during continuous infusion of sodium chloride 0.9% or dextrose 5%.

The dose in people is 5-10 mg IV. In dogs, a dose rate of 5 mg/kg is often stated, although this is likely unnecessarily high.

K	onakion <sup>®</sup> MM 2 mg	
Lösur Zur Ar Wirks (Vitar <b>2 mg</b> oral/	rå mendung bei Neugetorenen batt Poptomersuiden m K) <b>/0.2 mi</b>	3
2	mg/0,2 ml	
ß	5 fiale da 0,2 ml con siringa per somministrazione orale	Roche

Figure 2: The new micelle formulation of Konakion® designated by the MM suffix. This can be safely given by SLOW intravenous injection

The first report of this micellular formulation was an ECVIM Conference Abstract:

Senzolo M, Gentilini F, Zoia A et al. Evaluation of changes in coagulation times (PT and APTT) after intravenous vitamin K administration in 73 dogs intoxicated with anticoagulant rodenticides. *23rd European College of Veterinary Internal Medicine* – *Companion Animals Congress*, 12–14 September 2013, Liverpool, UK. We understand Dr Erin Mooney and colleagues are conducting a clinical trial of this drug at the University of Sydney, based on their experience with IV administration of older formulations of vitamin K which corrected the clotting abnormalities within 1 hour when given IV. This work was written up recently in the *AVJ*.

We strongly recommend that veterinarians get this new formulation of Konakion MM, so they have the option of IV therapy in cases of acute anticoagulant rodenticide poisoning. Remember to give it slowly over a few minutes. And note that really bad cases will still benefit from the administration of fresh whole blood transfusions or fresh frozen plasma.

Considering the fact that these cases invariably have a large bill, the extra cost of the most appropriate vitamin K product is a tiny price to pay for rapid re-establishment of normal clotting.

#### Reference

ET Mooney, G Agostini, C Griebsch, M Hickey, 18 Mar 2020, Intravenous vitamin K1 normalises prothrombin time in 1 hour in dogs with anticoagulant rodenticide toxicosis, *AVJ*, doi.org/10.1111/avj.12931

SMALL ANIMALS



#### **CASE SERIES**

## Intravenous vitamin $K_1$ normalises prothrombin time in 1 hour in dogs with anticoagulant rodenticide toxicosis $% \left( {{{\left( {{{{{\bf{n}}}} \right)}}} \right)$

ET Mooney,<sup>a</sup>\* G Agostini,<sup>a,b</sup> C Griebsch<sup>a</sup> and M Hickey<sup>a</sup>

Four dogs with anticoagulant rodenticide toxicosis were treated with intravenous vitamin K<sub>1</sub> in lieu of plasma transfusion due to client cost constraints. Two dogs experienced a suspected anaphylactoid reaction, necessitating cessation of the treatment in one dog. Prothrombin time was rechecked 1 h after treatment in the remaining three dogs and all results were within the normal reference range. All four dogs were discharged from hospital within 48 h of presentation. Intravenous vitamin K<sub>1</sub> rapidly reverses the coagulopathic state in dogs with anticoagulant rodenticide toxicosis. It is a viable alternative therapy to plasma transfusion if circumstances preclude its use; however, patients must be monitored for anaphylactoid reactions.

This approach to treatment has persisted over many decades for two reasons. First, there are reports in the veterinary literature that vitamin  $K_1$  does not activate clotting factors – so presumably does not reverse coagulopathy – until three to 48 h following administration by any route, including intravenously which is presumably the fastest.<sup>3,4,9–15</sup> A delay of this magnitude in a markedly coagulopathic patient would pose an unacceptable risk of further haemorrhage to a patient treated with vitamin  $K_1$  alone. Second, vitamin  $K_1$  has not historically been given to dogs IV due to the risk of acute hypersensitivity reactions.<sup>6,9,11,13,15</sup> An experimental study in dogs demonstrated these reactions are actually anaphylactoid in nature and caused by solubilising agents such as polyoxyethylated fatty acid derivatives found in older



eBook download

*C&T* 5216, To Bleed or Not to Bleed? - That is the question... What dose and duration of Vit K prevents canine haemorrhage in rat bait toxicity? Alne Seavers, Issue 266, March 2012

## ACUTE KIDNEY INJURY—WHAT IS IT AND WHAT DOES IT MEAN FOR OUR PATIENTS?

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A 10-year-old Staffordshire Bull Terrier with a serum creatinine of 835 µmol/L, a 12-year-old Cocker Spaniel with a serum creatinine of 675 µmol/L and a 5-year-old Domestic Short Hair (DSH) with a serum creatinine of 555 µmol/L are just some of the patients we have seen over the last few years presenting for vomiting and hyporexia. Before seeking our opinion, their owners had been advised that their pets had end-stage kidney failure and euthanasia was recommended. A year later, all 3 patients are at home, eating well with stable body weights and good quality of life. The cat has a serum creatinine of 180 µmol/L, the Staffordshire Bull Terrier 248 µmol/L and the Cocker Spaniel 150 µmol/L. By chance, none of these patients are on a commercial renal diet or any renal specific medications.

How did we have the audacity to give these owners hope and tell them that there is a reasonable chance for their pets to survive the crisis?

There is no special skill-simply understanding the difference between acute kidney injury (AKI) and chronic kidney disease (CKD).

During veterinary school, we were taught that acute kidney injury is a sudden onset condition and chronic kidney disease a longer-term process. However, many of us never fully grasped why this differentiation mattered and how it would affect diagnostics, treatment, and client education. This article will hopefully elucidate the difference between these conditions and give more veterinarians a starting point for what to do when met with a newly azotaemic patient.

#### The importance of differentiating AKI from CKD

Chronic kidney disease implies irreversible damage to the kidneys. Humans with significant CKD start chronic dialysis while waiting for a kidney transplant. Conversely, acute kidney injury (AKI) is damage to the kidneys that is potentially reversible, depending on aetiology and degree of permanent injury.

To have confidence in a diagnosis of true CKD, one should document that there are structural or functional abnormalities of the kidneys for at least 3 months.<sup>1</sup> Staging of CKD is based on serum creatinine concentrations evaluated on at least 2 occasions in a stable patient. If a patient with stable body weight demonstrates a serum creatinine concentration that changes significantly (more than about 30-40 µmol/L) over a short period of time, it is likely that they are or were suffering AKI. CKD is generally a slowly progressive process. Creatinine is only one marker of renal function. It will decline when muscle mass is decreased, even if renal function is static.<sup>2</sup> Measurement of symmetric dimethylarginine (SDMA) can sometimes help to evaluate change in renal function without being affected by body weight.<sup>3</sup>

The prognosis for AKI is incredibly variable depending on disease aetiology (18% - 75% across many different studies and populations), but the general accepted mortality rate is similar to that in humans-approximately 50%.<sup>4,5</sup> The surviving 50% can expect to achieve either somewhat normal renal function (without the need for specific management) or some degree of CKD (requiring special diets and other medical management). When azotaemia is first discovered, it can be extremely difficult to predict the extent of renal recovery that can be achieved if the patient is managed appropriately and given enough time. An incredibly important concept is that the severity of azotaemia at the time of diagnosis does not predict whether a patient can recover. Many pets with severe azotaemia (creatinine >600 µmol/L) can recover, especially if the cause of disease can be addressed or removed.

Once the cause of AKI has been addressed or removed, renal recovery can still require weeks to months. However, the clinical manifestations of uraemia can result in the death of the patient within a few days or weeks. Our role in AKI management is to bridge that time gap. If we can keep our patients alive and maintain their body in a

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age



Figure 1. Sonographic image of a hydronephrotic kidney secondary to ureteral obstruction in a cat

state that promotes renal recovery, our chances of achieving a good outcome will be higher. Treatment of AKI is aimed at:

- Preventing further injury to the kidneys
- Buying the kidneys enough time to recover while maintaining the body in a state of optimal hydration, volume, nutritional and electrolyte/ acid-base balance. Equally important is prudent use of client finances so that they are able to afford to maintain the pet for the weeks or months that renal function might take to recover.

## The definition of AKI–Why not call it acute renal failure?

By the time a patient is exhibiting persistent isosthenuria, they have already lost two thirds of normal renal function. Azotaemia generally occurs when 75% of renal function is lost. Any AKI progresses through the following stages:

- Risk (the procedure or situation that may incite the kidney damage)
- Injury (harm to the kidneys, but patients may not be azotaemic or have biochemically measurable change in kidney function yet)
- 3. Failure (azotaemic or >75% reduction in GFR)
- 4. Complete loss of kidney function >4 weeks
- 5. End stage disease

Although it is ideal to identify AKI when the injury is occurring, it is most often identified when azotaemia is already present.

AKI Grade	Blood creatinine	Clinical description
Grade I	<140 µmol/L	Non-azotaemic AKI: a) Documented AKI (historical, clinical, lab/ imaging evidence, clinical oliguria/anuria, volume responsiveness) and/or b) Progressive non- azotaemic increase in blood creatinine >26.4 µmol/L within 48h. c) Measured oliguria <1mL/ kg/h or anuria over 6h
Grade II	141-220 μmol/L	Mild AKI: a) Documented AKI and static or progressive azotaemia b) Progressive azotaemic: increase in blood creatinine >26.4 µmol/L within 48h or volume responsiveness. c) Measured oliguria <1mL/ kg/h or anuria over 6h
Grade III	221-439 µmol/L	Moderate to Severe AKI: a) Documented AKI and increasing severities of
Grade IV	440-880 μmol/L	azotaemia and functional renal failure
Grade V	>880 µmol/L	

Figure 2. IRIS grading scheme for acute kidney injury

The International Renal Interest Society (IRIS)<sup>6</sup> has developed the above grading scheme for veterinary acute kidney injury:

Each grade of AKI is further subgraded as either:

- 1. Non-oliguric (NO) or oliguric (O)
- 2. Requiring renal replacement therapy (RRT), commonly termed 'dialysis'

The most important concept to remember from these guidelines is that patients with IRIS Grade I AKI are non-azotaemic. Additionally, a serum creatinine concentration of 141 µmol/L (either due to IRIS Grade II AKI or Stage II CKD) may sometimes be within reference intervals on laboratory results, causing some cases of AKI to be missed. There are some exceptions to using these IRIS based cut-offs, such as breed differences with sight hounds or giant breed dogs who tend to have higher serum creatinine concentrations. Being able to recognise

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and detect an AKI is the first step in providing timely treatment and information to clients about subsequent decisions for their pet.

#### Presenting complaints of patients with AKI

- Lethargy, not quite right
- · Poor appetite
- Vomiting
- · Collapse
- · Polyuria/polydipsia
- No clinical signs, but a hospitalised patient having an increase in blood creatinine exceeding 26.4 µmol/L above their baseline. This may occur after an anaesthetic, or in situations of severe systemic disease and haemodynamic instability (e.g., pancreatitis, sepsis, peritonitis, SIRS, MODS)

To help determine the aetiology of a patient's AKI, the following pertinent medical history should be obtained:

- Any recent anaesthesia or heavy sedation?
- Any recent or chronic nephrotoxic medication: NSAIDs or aminoglycosides?
- Any recent introduction or increased dose of ACE-inhibitors or ARB (angiotensin receptor blockers)?
- · Any nephrotoxin access:
  - o grapes/raisins
  - o lilies in cats
  - o ethylene glycol (antifreeze)
  - o vitamin D (in rat bait, supplements, or psoriasis creams)
- Any access to rodent areas, waterways, farm animals (leptospirosis)?
- Any historical urinary tract infections?
- Before becoming ill, what was the patient's last serum creatinine concentration?
- Was vomiting, regurgitation, or diarrhoea occurring prior to azotaemia?
- If there is PU/PD-duration of signs?
- Any weight loss, and over what period of time?
- When was the last urination and is the patient physically able to pass urine? This is to ensure there is not a urethral obstruction.

#### Key physical examination details

- Hydration status. Unfortunately, changes in skin turgor, tear film, mucous membrane moisture and capillary refill are all very crude measures of hydration. Skin elasticity varies with both age and body condition. Azotaemic animals may have xerostomia (lack of production of tears and saliva) making their mucous membranes and tear film a less reliable representation of hydration. Despite how insensitive physical examination findings may be, the importance of noting these qualities lies in the ability to detect changes once treatments have been instituted.
- Body weight, body condition score and how it has changed since last veterinary visit.
   Sometimes, sudden body weight change (without concurrent change in muscle mass) may provide a more quantifiable hydration assessment. Body weight at time of admission to hospital is essential in all patients, but even more so in patients with AKI because weight changes in as early as the first 4 to 6 hours in hospital can provide critical information as to whether fluid therapy is adequate or even harmful.
- Body temperature. Uraemia resets hypothalamic temperature set point lower so most severely azotaemic patients are hypothermic or have a temperature in the low end of normal.
   In a severely azotaemic patient, a rectal temperature of 38.5°C could be pyrexia.
- Abdominal pain/discomfort, especially over the kidneys
- Bladder size and changes (to assess possible anuria, oliguria or urethral obstruction)

#### Common causes of AKI in Australia

- 1. Infection (e.g., pyelonephritis, leptospirosis)
- 2. Acute inflammation or immune-mediated disease (e.g., glomerulonephritis)
- 3. Nephrotoxin (see medical history questions above)
- 4. Physical causes
  - o ureteral obstruction (urolith, cellular debris, ligation, stricture)
  - o urethral obstruction
  - o trauma
- 5. Vascular or haemodynamic
  - Ischaemic damage including hypotension or poor perfusion under general anaesthesia

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- o Infarction
- Systemic Inflammatory Response
   Syndrome / Multiple Organ Dysfunction
   Syndrome from systemic disease
- 6. Disorders of growth (e.g., neoplasia)

#### Diagnostics for newly azotaemic patients

- A detailed medical history is the most important diagnostic tool, especially finding out when the patient's serum creatinine concentration was last normal. The duration over which azotaemia develops provides direct clues as to whether a patient has an AKI or CKD.
- 2. Urine specific gravity can also be helpful. An inappropriately low USG in the face of severe dehydration or azotaemia suggests true renal disease. However, there are also instances of AKI where a patient's USG may be high, such as sudden bilateral ureteral obstruction (e.g., spey complication, stones). Many nephrologists also question whether dehydration alone is enough to cause azotaemia in a healthy patient with fully functioning kidneys, or whether the azotaemia in dehydrated patients is a true AKI that is easily treated by restoring haemodynamic volume. Due to ambiguity about whether true AKI is occurring in these situations, the term 'volume-responsive' AKI tends to be preferred.
- 3. The combination of urinalysis and bacterial culture can help diagnose cases of pyelonephritis, and glomerular and tubular injury. However, if a client truly has limited finances, then urine culture should be prioritised over urinalysis. Normal urinalysis does not rule out infection. Without a confirmed positive urine culture, empirical antibiotics for 3 to 4 weeks may not only be costly but could worsen appetite for patients that are already in a negative nutritional state. A urinalysis (without urine culture) is rarely going to change therapeutic management for a severely cost constrained client and patient. Even if proteinuria is found, a urine culture should be performed to rule out infection before urine protein creatinine ratio assessment.
- 4. Full biochemistry, including:
  - o liver enzymes including bilirubin (as concurrent hepatopathy may be suggestive of leptospirosis)
  - electrolytes and acid-base status (especially potassium, bicarbonate and blood pH)

- calcium and phosphorous (for medical management and assessment for vitamin D toxicity)
- o blood glucose (to assess whether any glucosuria is appropriate or not)
- albumin, cholesterol (these are markers of liver function and hypercholesterolaemia with hypoalbuminaemia and proteinuria may be associated with nephrotic syndrome)
- 5. Full blood cell count:
  - o non-regenerative anaemia is common in patients with CKD
  - leukocytosis may indicate that an infectious or inflammatory process is more likely and empirical antibiotic therapy while pending cultures may be warranted
  - thrombocytopaenia can occur with immune-mediated diseases or secondary to infectious processes such as pyelonephritis or leptospirosis
- 6. Systemic hypertension can result from AKI or CKD and can worsen renal disease through hyperfiltration effects.
  - 37% of AKI patients are hypertensive at presentation<sup>7</sup>
  - o 81% of AKI patients become hypertensive during hospitalisation<sup>7</sup>
- A resting cortisol may be indicated to rule out Addison's disease, which can present similarly to AKI in dogs. A resting cortisol >55 nmol/L suggests Addison's is extremely unlikely.<sup>8</sup>
- Leptospirosis testing PCR panel (EDTA whole blood as well as urine), ideally before antibiotic administration. Acute and convalescent leptospirosis antibody serology can also be performed.
- 9. A urine protein creatinine ratio (UPCR) should be performed if proteinuria is detected on initial urinalysis and there is no bacterial growth after 24 hours of culture. Proteinuria can be a cause for AKI and if suspected, patients should be offered referral for a discussion about renal biopsy.<sup>9-11</sup> Early referral is prudent because the risk of renal biopsies increase as azotaemia progresses. Early referral for renal biopsy might also improve outcome in cases of immune complex mediated glomerulonephritis.
- 10. The role of abdominal ultrasound in AKI is to exclude obvious structural changes to the kidneys such as masses, cysts, infiltrative disease, pyelonephritis or signs of ureteral obstruction or congenital abnormalities such



Figure 2. Coco a 5-year-old FS Golden Retriever happily undergoing haemodialysis for anuric AKI. After a week of intensive hospital treatment, she recovered. Four years after her treatment, she is still living a normal life with no signs of kidney disease

> as renal dysplasia. It is important to remember that complete ureteral obstruction can be present despite sonographically normal kidneys. If highly suspected, repeat ultrasound examination may be necessary to evaluate changes in renal pelvic dilation over time or for surgical planning.

#### Differentiating AKI from CKD

After acquiring data from history, physical exam and diagnostic tests, veterinarians would be better able to assess whether a patient is more likely to have an AKI or CKD. The following list (*Figure 3*) of characteristics are generalisations that may help differentiate the two.

#### What to tell clients if AKI is suspected

- If an AKI has a known, fixable cause that can be addressed (e.g. pyelonephritis, ureteral obstruction, Leptospirosis), then the prognosis for renal recovery is good although some degree of CKD may remain. However, we cannot predict the degree of CKD any individual patient will be left with—some may not need any management and lead a normal life, while some may need special diets or medication.
- If the cause of AKI is unknown/toxic/metabolic and is still producing urine, then the patient has a 50% chance of renal recovery.<sup>12,13</sup> This can take weeks to months, during which veterinarians and clients together will need to manage the consequences of uraemia (e.g. IV fluids, appetite stimulants, feeding tubes, or renal replacement therapies such as dialysis). We cannot predict whether renal function will improve, how much it will improve and over how long.

AKI	CKD
Patients feel unwell or have sudden development of clinical signs. This is either because their azotaemia has developed rapidly with little time to become accustomed to the uraemic toxins, or because another disease in their body causing the AKI (e.g., pancreatitis) is making them unwell.	Patients are generally feeling well, and azotaemia found on a routine health check. In retrospect, owners may report some polyuria, occasional vomiting or subtle reduced appetite and weight loss.
Good body condition score with only very recent weight loss.	Thinner body condition score or slow progressive weight loss.
Anuric, oliguric or polyuric.	Mostly polyuric.
Normal or large sized kidneys, which may also be asymmetrical or painful.	Small, symmetrical, or irregular, non-painful kidneys.
Glucosuria without concurrent hyperglycaemia is indicative of tubular injury.	Unlikely glucosuric unless diabetic or genetically predisposed (e.g., Basenjis with Fanconi's Syndrome)
Proteinuria of any degree, but AKI secondary to proteinuria should be highly suspected if UPCR > 5.	Generally lower levels of proteinuria between 0.4 and 5
Known recent AKI risk factors.	No recent AKI factors.
Normal potassium or increased potassium	Normal potassium or hypokalaemia
Normal haematocrit	Normocytic normochromic non- regenerative <i>anaemia</i>
Marked hyperphosphatemia relative to the degree of azotaemia	Mild to moderate hyperphosphatemia relative to the degree of azotaemia

Figure 3. Differentiating AKI from CKD

 If the patient is not producing urine or oliguric, severely azotaemic or fluid-overloaded, then the possibility for recovery without short-term renal replacement therapy such as dialysis is low but still possible.

#### Treatment of AKI

Our approach to treating AKI patients is outlined below.

- Discontinue all potentially nephrotoxic drugs (including ACE-inhibitors and angiotensin receptor blockers, unless proteinuria is thought to be the cause AKI)
- 2. Replace any fluid deficit and maintain optimal hydration without overhydration or dehydration. Conversely, if a patient is eating and drinking normally without fluid deficits, hospitalisation and IV fluid therapy may not be necessary. In fact, overhydration decreases a patient's chances for renal recovery by causing interstitial oedema and reducing GFR.<sup>14-17</sup> Fluids are not being used to 'flush the toxins out', rather they are being used to maintain adequate blood flow to the kidneys. In order to achieve an ideal hydration status, monitor body weights regularly (ideally 2-4 times daily) after urination events. The patient's weight should not increase by more than the amount you estimate your patient to be dehydrated. If a patient gains 9-10% body weight in hospital, this could be a sign of oligoanuria or overzealous fluid administration that should be discontinued.
- 3. Address systemic hypertension if systolic blood pressure (SBP) is reliably >170 mmHg
  - Amlodipine 0.1-0.2 mg/kg PO q24h and recheck daily, titrating up to 0.6-0.8 mg kg per day maximum. Higher initial doses may be required if severe hypertension or end-organ damage is present (e.g., SBP >200mmHg and retinal oedema or detachment).

Symptomatic treatment for gastrointestinal tract:

- o Maropitant 1mg/kg IV q 24h or 2mg/kg PO q 24h
- o Ondansetron 0.3-1 mg/kg IV or PO q 12h
- o Esomeprazole 1mg/kg IV or PO q 12-24h if concern for gastrointestinal ulceration
- 4. Empirical antibiotics (if suspect pyelonephritis or leptospirosis)
  - o Ampicillin 22mg/kg IV q 8h while pending



Figure 4. Austin, a 6-year-old MC German Short-haired Pointer who ate a bunch of grapes that had fallen over the fence, developed AKI with a serum creatinine of 880 µmol/L and improved over 2-3 weeks with 2 dialysis treatments. A year later, he is already living a normal life with serum creatinine of 96 µmol/L and USG of 1.030

> urine bacterial cultures will cover many causes of urinary tract infections and leptospirosis. Potentiated beta lactam antibiotics are also an appropriate choice based on index of suspicion for certain bacterial species.

- We do not recommend cefovecin because it will be difficult to know how to dose this for 3-4 weeks in a patient with altered renal metabolism and elimination. Unnecessary use of such a broad spectrum antibiotic may also lead to bacterial resistance.
- 5. Treatment for anaemia support if Hct <25% and non-regenerative or haemodynamically unstable
  - o blood transfusions if haemodynamically unstable
  - o iron dextran 10mg/kg IM if indicated (monitor for intramuscular abscesses or anaphylactoid reactions and do not give this if patient has recently received a blood transfusion)
  - o Darbepoetin 0.45-1 µg/kg SC once a week until PCV >30% and then reduce frequency of administration.
- 6. Nutrition
  - Patients need more than RER (resting energy requirement) for appropriate renal recovery (placement of feeding tubes may be necessary)
  - o Be careful of high fat renal diets in AKI patients as some might have concurrent pancreatitis or gastrointestinal disease

that has either precipitated the development of AKI or has resulted from AKI <sup>18-22</sup>

- Dietary phosphorous restriction and phosphorus binders - aluminium hydroxide (30-100mg/kg per day, mixed with food) is the first choice.
- o Protein restriction if proteinuric.

## Indications for immediate specialist referral or renal replacement therapy ('dialysis')

- 1. Severe azotaemia (creatinine >440 µmol/L)
- 2. Suspected anuria or oliguria
- Severe electrolyte or acid-base derangements such as hyperkalemia or metabolic acidosis
- 4. Overhydration, particularly where anuria or oliguria is present
- Significant uraemic effects on other body systems (e.g., gastrointestinal, neurological, haematologic) where simple medical management has been ineffective

#### Common errors in diagnosing or treating AKI

- Missing Addison's disease, which can present similarly to AKI
- Missing azotaemia on pre-anaesthetic bloodwork for elective procedures (serum creatinine concentration >140 µmol/L, but still within `reference ranges'), inadvertently risking causing an acute on chronic kidney event
- Recommending euthanasia when seeing a markedly increased serum creatinine concentration. Severity of azotaemia is not predictive of recovery
- Giving antibiotics before a cystocentesed urine culture or *Leptospira spp*. PCR has been submitted. Rarely, if there is no urine to collect and the patient is febrile with leukocytosis, it is appropriate to start antibiotics, but urine should be collected as soon as possible.
- Overhydration make sure to monitor body weight changes in hospital closely
- Poor recognition of the role of nutrition adequacy and type for renal recovery. This includes thinking critically about the cause for a patient's AKI. Mild azotaemia is unlikely to cause clinical illness other than polyuria and polydipsia. In a patient who has mild

azotaemia not severe enough to explain their poor appetite and vomiting, there is a high chance that there is concurrent pancreatitis, gastrointestinal disease or other systemic disease that has caused the AKI and clinical signs. In these situations, a high fat renal diet could be harmful by worsening gastrointestinal signs

#### **KEY MESSAGES**

Perform a urine culture +/- *Leptospira* spp. PCR (depending on risk factors, including geographic location) in all newly azotaemic patients before administering antibiotics.

Offer early referral to an internal medicine specialist for assessment of aetiology, abdominal ultrasound examination with a radiologist (depending on owner finances), and recommendations for other diagnostics and development of a treatment plan. Sometimes, this requires hospitalisation, while other times care might be pursued on an outpatient basis. Cases to refer immediately:

- o oliguric/anuric
- o hyperkalaemic
- o dedicated clients who wish to maximise chance for renal recovery
- clients with moderate financial constraints that cannot afford any mismanagement of fluid therapy or low yield diagnostic tests.
   A medicine specialist will be able to have a discussion with these owners about the cost-benefit analysis of diagnostics and treatment and develop the most economical plan to give the patient an opportunity to recover.

Renal azotaemia does not always mean end stage chronic kidney disease.

Renal recovery from AKI takes weeks to months, not just a few days. If we only give a patient a few days in hospital, then we have not given the kidneys adequate opportunity to recover. If patients are not recovering in the first few days, or are worsening, it is important that clients know that renal recovery is still possible. However, if they are not prepared for the financial or emotional commitments of weeks to months of care (which may involve a temporary feeding tube or renal replacement therapy), then euthanasia may be a fair option.

#### **NOTE:** References available in the eBook.

eBook download



C&T 5840, Management of Acute Renal Injury, Matthew Wun & Terry King, Issue 300, September 2020.

## Small DOING A TRICKY SURGERY IN GENERAL PRACTICE

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'BB' is a 9-year-old Domestic Shorthaired Cat who came to me as a second opinion for a rather large swelling in the mid-ventral cervical region.

He had initially presented with a stable weight, normal appetite and thirst. A fine needle aspirate (FNA) concluded:

Sample of high cellularity, thick and moderately haemodiluted, with pools of pale blue-basophilic proteinaceous/mucous debris in the background. Large numbers of epithelial cells are present, grouped in cohesive, dense clusters or small sheets, occasionally with intermixed bright pink stromal/ secretory material. They show cords or occasionally tubuloacinar architecture. The cells appear large, uniform, basaloid, with poorly defined borders, pale cytoplasm, and oval nuclei with finely stippled chromatin and mild anisokaryosis.

COMMENT The cytological findings indicate an epithelial neoplasia, with mild cytological atypia. The cytoarchitecture and the character of the debris in the background suggest a possible salivary origin (e.g. salivary adenoma/carcinoma).

Further diagnostic possibilities would depend on exact location (e.g. thyroid tumour or cutaneous follicular neoplasia). Incisional biopsy with histological examination is necessary for definitive diagnosis.

The primary vet's conclusion was:

`Mass appears to be a salivary gland mass—these are generally adenocarcinomas from mandibular or parotid salivary glands and are very likely to metastasize. Options discussed included referral and CT/surgery to remove the mass although it is very likely to have spread/no action'.

#### Aside: Anatomy counts!

The cytologist needs to know where the mass was to interpret what they are seeing in the stained smears! Their ordering of diagnostic possibilities depends on anatomical location. Tell them precisely. Better still—provide a photo.

The (un-insured) owner did not wish to go the referral route, but equally did not want to leave her cat to die, hence her second opinion with me.

When I saw BB, he had become anorexic and pyretic. Clinical examination revealed a small puncture on the ribcage, and an unrelated cat bite injury was assumed to be the cause of his inappetence. A large mass occupied most of the neck—on the recumbent photos it is clear that it is not salivary in origin, though this was not apparent when the cat was sitting on the consult table.

When seeing a second opinion I always like to `deconstruct', looking at the case afresh, considering the results of previous investigations but not taking into account the previous conclusions. I explain to the client that I am not out to prove the previous vet wrong, I am simply trying to get the correct answer.

I understand that FNAs from glandular structures in the neck are notoriously challenging for cytologists, especially as colloid looks very similar to saliva microscopically. There was a danger here of a 'false anchor' from the initial cytology—a trap for the unwary!

Always take pictures of your unusual lumps and bumps—it may give the cytologist/histopathologist critical insight, and they love the bigger picture. Send them feedback and develop a relationship with them—both parties can learn, to the benefit of the patient.

So, I performed a quick conscious ultrasound and my own fine needle aspirate, finding the same cells as previously from a pretty homogenous structure with some cystic internal content. The fluid aspirated had a slightly mucoid content—not over-stringy, but sufficiently like saliva for me to consider the initial diagnosis to be plausible.



You should always feel able to pick up the telephone and discuss a case with your pathologist, particularly if something in the pathology report does not quite fit with the clinical presentation of the patient in front of you!

I explained the limitations of FNA of such glandular structures, and we discussed the options I felt were available:

The 'proper' route viz. biopsy/CT/histology/surgery at specialist centre (as declined).

- 1. **The in-house 'best' option:** Biopsy, ultrasound to establish margins, X-ray chest for metastases and plan excision in due course if favourable results obtained.
- 2. **The cheap option:** Try to excise lump and be prepared to abandon ship and put to sleep if invasive around important structures.
- 3. **The conservative option:** Do nothing and expect reduced ability to swallow food as mass increases, putting to sleep as required

Until recently the thought of tackling this sort of lump surgically myself would not be a favoured procedure. However, a recent successful op on a slightly smaller cervical lump gave me the confidence to suggest this as an option, ensuring the client understood that I am not a specialist soft-tissue surgeon.

After considerable deliberation, the client opted for the second option. Thoracic rads were all clear, and ultrasound confirmed a nice encapsulated structure, with Doppler confirming the lack of huge blood vessel involvement. In the absence of any firm contra-indications, I decided that I would simply go ahead and excise the lump, buying the cat more time whatever the diagnosis. The client approved.

Surgery was remarkably unchallenging. After a ventral midline incision, I had dissected down onto the mass to get a free working-edge close to the capsule. It was then simply a case of blunt



Figure 1. Ultrasound study of the affected ventral cervical mass. Note the mixed echogenicity



Figure 2. Lateral and ventrodorsal view of the cat prior to surgery to show the size, shape and position of the ventral cervical mass

dissection, teasing all the connected tissue off the mass with my fingers using a moderate amount of force. Occasional careful scissor snips were required, along with 'scissor-opening' to break attachments around nerves and blood vessels – but essentially no sharp surgery other than the initial incision to the mass.

Closure was routine using continuous sutures of 4 metric Monocryl to both the muscle layers and subcutis. Tissue-glue completed the closure. Total surgery time was less than 15 minutes.

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Figure 3. Intraoperative photograph of the tumour being removed, initially by careful blunt dissection

Post-operatively, we readdressed the diagnosis.

Could it be thyroid?—an option I had discounted as the cat's weight was stable at the time of first presentation with its egg-sized mass. Of course, it could have been non-functional—but when we ran bloods post-operatively the T4 was off the scale!!

One-week later BB had put on 1kg in weight!

#### **Histopathology results**

This large mass from the right side of the ventral neck is most consistent with a malignant neoplasm of thyroid follicular cell origin (thyroid follicular carcinoma), based on the presence of apparently invasive growth and degree of mitotic activity. It is possible that some of the cellular and nuclear atypia is secondary to the presence of the reactive fibroplasia and inflammation, but I am still concerned that this is a malignant thyroid neoplasm. The associated lymph node is occupied and effaced by the presence of thyroid tissues, which does not appear to correspond to local extension, and is therefore concerning for the presence of metastatic disease at that site.

Figure 4. The cat at the completion of surgery with a Penrose latex drain to drain the dead space



Unlike the situation in dogs, thyroid follicular carcinomas of cats tend to be indolent malignancies, so full excision can be potentially curative. In some cases, however, the tumours will invade adjacent soft tissue structures via the capsule and vascular invasion can also occur. Metastases to regional lymph nodes (retropharyngeal, mandibular, deep cervical) and distant sites (e.g. lungs) have been reported in less than 50% of cases.

Most often, the prognosis will hinge upon local tumour control and the control of associated clinical signs. Close monitoring of this patient for any evidence of recurrent or more widespread lesions or for any clinical hyperthyroidism would be advisable.

#### Mel D, Finn Pathologists

So, it would appear that we had a massive variably functional thyroid tumour with potential for metastatic spread.

What next? We have the option of scintigraphy to establish if any tissue has escaped the scalpel followed up with radioiodine therapy if indicated. In hindsight, we could have done I131 first off if we had our diagnosis pre-excision, but a lump that size would have necessitated 8 to 9 weeks in hospital in the UK rather than the 8 days needed to knock off any residual thyroid carcinoma tissue.

#### So, what are the morals of this tale?

Firstly, the process! Are we after a diagnosis? Or a practical solution that is right by cat and owner. Are the two mutually exclusive? I say not! My previous cervical lump case was losing weight rapidly through inability to swallow so it was a case of euthanasia or the knife. One short surgery later and the cat was able to eat and rapidly improved.

Figure 5. Photograph of a cut section of the resected mass





Figure 6. Histology of the ventral cervical mass.

- A) Low power view = red arrow is metastatic spread to the adjacent lymph node; green arrow = large central area of necrotic tissues and 'proteinaceous goo'
- B) Red arrows = mitotic figures on high power view
- C) Black arrows = peripherally infiltrative growth of neoplastic cells into the surrounding fibrous capsule. All features suggestive of malignancy

Three months later the cat is doing fine, diagnosis or no diagnosis. Is this case any different?

So, let us look at my prime objectives:

- a) To give the cat an extended comfortable lifeexpectancy if finances permit
- b) The cat should not be subjected to major painful procedures if life expectancy is short
- c) Any surgical procedure is less justifiable (but not excluded) if the disease is advanced i.e. chest full of secondaries

With the referral option out, the only way of achieving these objectives is to `go for it' on the surgical front—with appropriate pre-emptive checking. But I appreciate that not all of us feel happy giving it a go with such an impressive lesion in this area. My message is to consider trying, especially when the alternative is euthanasia.

It worries me greatly that the modern generation of vets find themselves in fear of being sued in case things 'go wrong' and therefore they never get to stretch themselves. I also fear that the corporate entities encourage referral to their own specialist centres for anything more than simple basic cases, denying GP vets the more-complicated 'basic' cases, hindering skill acquisition.

I am fortunate to have spent my early career in the days where pretty-much anything was possible—we didn't have all the technology and drugs of today and it was much easier and less-threatening to `give it a go'. And I guess clients were less litigious and more forgiving if we were good communicators.

Frank discussion with the client is important of course—I was very clear to my client that I had not done many ops like this but was prepared to try. So long as all the options and potential pitfalls are discussed you should be on solid ground, even if you do not know the relative merits of the alternatives. And do not forget the 'ask a friend' option. In the UK, most referral centres are very happy to discuss cases—just do not get overwhelmed by their referral solutions if it does not work for cat and owner. Or post on VIN—or on ISFM forum—or all 3. Do not rush into it, keep a cadaver in the freezer so you can refresh the surgical anatomy knowledge pre-op, and take a deep breath.

It may still go wrong! But you tried, the client will appreciate that—and you will have learned. So much more satisfying!



Figure 7. We all need humour. Especially pathologists (Source: memegenerator.net)

## Specialists' Corner



#### **RACHEL KORMAN** Feline Internal Medicine Specialist

#### What attracted you to this discipline?

I was one of those people that can't remember a time I didn't want to be a vet. I've always loved animals since I was a little kid. My family are medical and I didn't fancy being a human doctor because I loved animals more so being a vet seemed the natural choice! I've been very lucky (and worked hard...) to get into vet school and find my niche in the feline medicine world.

Dr Terry King really inspired me as a student with his bedside manner for pets and their owners and that really cemented to me that small animal practice would suit me. Dr Vic Menrath and Dr Tim Gruffyd-Jones then inspired me to follow the feline medicine pathway.

#### Worst veterinary experience?

Working as a locum in the UK where 'pocket pets' are extremely popular—I had to get through a whole consultation without actually knowing the species of animal I was dealing with. Plus a couple of decent dog bites...

#### **Best veterinary experience?**

That's a hard one to nail down. It sounds corny writing it but really, it happens every time I've had a super sick cat that makes it home, or when we manage to get palliative care right for a patient to spend some extra quality time with their owners.

Another one that does come to mind was a severely neurological young cat—we found some weird radiopaque material in her stomach on X-rays used endoscopy to scope it out and she completely recovered! It was an old 1970's stamp so it probably had some sort of heavy metal material in it!

#### If you weren't a vet, what would you be?

Before starting on my specialist training, I actually tried my hand at being a photojournalist—I love travelling and photography but found I missed being a vet. So now I love being a vet that also travels and does photography!

## RENAL INFARCTS IN CATS—WHAT DO THEY LOOK LIKE AND WHAT DO THEY MEAN?

#### Rachel Korman

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It is not unusual to perform an abdominal ultrasound in a cat and identify changes suggestive of renal infarcts. These are the well demarcated, hyperechoic, wedge shaped lesions seen within the renal cortex, sometimes single, sometimes multiple.

## What do they mean for our patients and what should we do when we see them?

Kidneys are at risk of infarction both due to vascular anatomy (the interlobular arteries supplying the renal lobes have no anastomoses) and the high blood volume passing through the kidneys increases risk for thromboembolism.

In humans, renal infarctions are divided into three categories: arterial, venous and traumatic. Arterial infarctions are further subdivided into complete and subtotal. Clinical findings for humans with acute renal arterial occlusion include abrupt onset of flank pain, nausea, vomiting and fever plus leukocytosis and albuminuria (Lang, Radiology 1967).

Venous infarction is often more insidious, and haematuria is common. A more recent publication identified that people with increased risk for thromboembolism presenting with unilateral flank pain should have increased suspicion for renal infarction (Korzets, Isr Med Assoc J 2002). Haematuria, leucocytosis and an elevated serum lactate dehydrogenase level were strongly supportive of the diagnosis.

Unfortunately, as is often the case in small animals, literature on renal infarction in cats is scarce. However, one useful study retrospectively reviewed 309 cats with renal infarction and



Figure 1. Ultrasound image of a feline kidney demonstrating two renal infarcts, one involving the cranial pole (blue arrows) and one involving the caudal pole (yellow arrows). Note there is also a small volume of retroperitoneal fluid in the patient.



Figure 2. Ultrasound imaging of a feline kidney demonstrating cranial and caudal pole renal infarcts (blue arrows). Note the abnormal bulging appearance of renal capsule outline (orange arrow). Fine needle aspirates were performed of this kidney to exclude the possibility of neoplastic disease



Figure 3. Cats with renal infarcts showed an 8x increased risk of developing distal aortic thromboembolism

291-time-matched controls. Ultrasound identified the renal infarct in 191 cats and necropsy in 118 cats.

Cats between the ages of 7 and 14 years were more likely to have a renal infarct. Clinical signs associated with renal infarction were not evaluated in this study.

Cats with renal infarcts were 4.5 times more likely to have hypertrophic cardiomyopathy (HCM) than cats without renal infarcts and geriatric cats were 8 times more likely to develop distal aortic thromboembolism. Overall, cats with renal infarcts were less likely to have a diagnosis of neoplasia and renal infarcts were not associated with hyperthyroidism.

It is not surprising to see this association with HCM. Hypercoagulability is common in HCM



Figure 4. Right parasternal short axis view at the level of the aortic valves demonstrating increase LA:aorta ratio of 2.5, confirming LA enlargement. Image courtesy Dr Fiona Meyers

associated with endothelial disruption of the cardiac chambers, turbulent blood flow or stasis with platelet activation. This increases the risk of thromboembolism.

Specifically, a small clot probably forms in the left auricular appendage. For reasons that are not always clear, this clot dislodges from the auricular wall, is swept down into the left ventricle and then down the aorta—and for some reason the clot embolises to one of the renal arcuate arteries (rather than proceeding to the aortic trifurcation).

Although an association between infarcts and neoplasia was not identified, I would not exclude neoplasia as a potential cause. Neoplasia has been associated with hypercoagulability, although I would screen for cardiac disease first.

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- 1. I recommend screening via echocardiography for occult cardiomyopathy, as detection and appropriate intervention may help improve survival.
- 2. I discuss the clinical signs of cardiac disease (i.e. no signs to elevated resting breathing rates and altered respiratory effort) and
- Discuss signs associated with aortic thromboembolism (e.g. vocalisation, pain, hindlimb or forelimb paresis).

- If referral echocardiography is not an option, other useful options might include:
  - Obtaining a left atrium: aortic ratio via right parasternal short axis view at the level of the aortic valves [LA:Ao ratio >1.5-1.6 is considered enlarged] via ultrasound
  - Or a ventrodorsal (or dorsoventral) radiograph looking for a left auricular bulge at 2-3 o'clock or valentine shaped heart.

If left atrial enlargement is confirmed, then consider the introduction of anti-thrombotic medication such as clopidogrel (18.75 mg per cat once daily given in a gelatine capsule).



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## NEURO-DIETETICS: MEDIUM CHAIN TRIGLYCERIDES & THEIR SCOPE AS AN ADJUNCT THERAPY FOR CANINE NEUROLOGICAL DISORDERS

Dr Alice V. Edwards BVSc(Hons) BVSc BVBio (CSU) m. 0406 515 213 Petfocus Vetcare Albury NSW 2640 Co-founder Cascade Animal Sciences e. aedwards@cascadevetsci.com

'disease of the brain, characterised by an enduring

experiencing ≥ 2 unprovoked seizures more than 24

Seizures can be further broken down into focal or

occur during sleep or while resting, last less than 5

where the patient's behaviour may be impaired.<sup>5,6</sup> In many epileptic patients, altered consciousness,

autonomic signs, convulsions, and oro-facial muscle

seizures can be difficult to differentiate from other

involvement occur during the ictal period.<sup>6</sup> Focal

Idiopathic epilepsy is reported to affect around

0.6-0.82% of the general canine population, making it one of the most common canine neurological

diseases seen in first opinion practice.<sup>7-9</sup> The average age of onset is between 6 months to 6 years<sup>7.8.9</sup>

and 20 to 60% of dogs will be euthanised or die as a direct result of the disease.<sup>10</sup> It is more common

Labradors due to genetic links.<sup>11</sup> There is, however,

Of relevance to our later discussion on C10's

functionality in both reducing the side effects of

dogs, understanding seizure activity as a result of

hyper-synchronized firing of action potentials in

activity through functioning as an inhibitor.

AEDs and decreasing the seizure frequency in some

neurons is paramount. Most AEDs seek to target the balance between the brain's excitatory and inhibitory

insufficient data in many other breeds that are likely to have a heritable components and further research

among certain breeds such as Golden Retrievers and

generalised events. Most generalised seizures in dogs

minutes and are characterized by a post-ictal period

predisposition to generate epileptic seizures'.<sup>5</sup> Clinically, this definition is appropriated to dogs

hours apart.5

movement disorders.<sup>6</sup>

is needed in this area.<sup>5,11</sup>

#### Introduction

A trove of research in the last decade both in human and companion animal medicine reveals the important but often-overlooked link between metabolic pathways and neurology. Although the literature regarding this axis is broad, this article will focus on how exogenous medium chain triglycerides (MCTs) can act as a low-risk and complimentary tool for treating difficult-to-manage neurological diseases like idiopathic epilepsy (IE), cognitive decline and canine hyperactivity-like behaviors. Indeed, much of the work within the veterinary scientific community in the last decade has been driven by the need for additional clinical tools, as more than two-thirds of dogs are considered resistant to anti-epileptic drugs (AEDs)<sup>.1,2,3,4</sup> In view of MTCs therapeutic potential, particularly decanoic acid's (C10), we will analyse its functions in negating seizure activity, as well as its potential to reduce the side-effects of AEDs, improve behavior in dogs with IE, and improve symptoms of cognitive decline in geriatric dogs.

**Section I.** will provide a brief introduction into IE as a difficult-to-manage disease, and how research that views neurological diseases like IE as a function of metabolic pathways has unlocked new potentials for adjunctive treatments.

**Section II.** will explore MCTs and their proposed mechanisms of action in relation to epilepsy, hyperactivity-like behavior, and cognitive decline.

**Section III.** will outline strategies for clinicians that could compliment traditional pharmacological IE treatment.

#### I. Canine Idiopathic Epilepsy: Current Clinical Practices and The Trends Behind Recent Research

The International Veterinary Epilepsy Task Force (IVETF) defines idiopathic epilepsy (IE) as a



Above: Three fatty acids chemically bonded to a glyerol uint to form a medium chain trigyceride

Figure 1. Molecular and schematic diagram depicting the difference between a free fatty acid and a medium chain triglyceride. Created by Dr. A. Edwards using biorender

dyskinesias<sup>6</sup> have been eliminated. Ideally, a patient's clinical history, signalment and exam are interpreted against their complete blood count, serum chemistry profile, blood lead concentration and urinalysis. **History compilation can be aided by owners filling out a standardised epilepsy questionnaire and presenting smart phone video-footage of any suspected seizure events.** If required, further testing may include analysis of thyroid, bile acids, fructosamine, glucose: insulin ratio, creatine kinase, lactate, abdominal and thoracic imaging, as well as ionised calcium and cobalamin levels.<sup>6</sup> Heavy metals (such as lead), *Toxoplasma, Neospora* and *Cryptococcus* testings, amongst other regionspecific infectious diseases, can also be utilized.

Magnetic resonance imaging (MRI) and analysis of the cerebral spinal fluid (CSF) may be indicated in some patients to help rule out structural abnormalities.<sup>6</sup> Broadly, this may include patients outside the age range of 6 months to 6 years,<sup>12,13</sup> patients that have neurological abnormalities beyond the post-ictal period,<sup>6,14</sup> patients with refractory or cluster seizures, or patients who have experienced a status epilepticus episode.<sup>6,15</sup> The gold standard for diagnosis IE is an electroencephalogram (EEG) scan, although this is generally not available or standard in veterinary medicine.<sup>6</sup>

## Traditional Treatment Approaches: A Need for Additional Tools

AEDs are the most common therapeutic tool for the treatment of IE. While pharmacological treatment alone works well for some dogs, over two-thirds of dogs on AED's continue to experience seizures, with 30% of dogs on two AEDs having less than a 50% reduction in seizure frequency.<sup>1-4</sup> Clinicians and owners are often confronted with balancing the benefits of AEDS with their side effects such as

polyphagia, polydipsia, polyuria, ataxia, sedation, and lethargy, as well as other systemic effects such as drug-induced hepatopathy and hyperlipidaemia.<sup>16</sup> Furthermore, it is well documented that AEDs in humans can impair cognitive function, including learning, attention and memory.<sup>17</sup> In line with this research, Packer *et al* (2016) found that dogs with IE treated with AEDs showed greater impairment in trainability compared with unmedicated dogs.

When examining C10's functionality in both reducing the side effects of AEDs and seizure activity, it is important to understand that seizure activity results from hyper-synchronized firing of action potentials in neurons. Indeed, most AEDs seek to target the balance between the brain's excitatory and inhibitory activity through functioning as an inhibitor.

#### II. MCTs vs Ketogenic Diets: A Subtle yet Important Difference

Studies on the ketogenic diet (KD) as a treatment for IE are mostly in children and focus on dietary induced ketosis<sup>19</sup>, as opposed to exogenous supplementation with MCTs. However, Puchowicz, et al (2000) demonstrated that endogenous ketosis was difficult to achieve in dogs, let alone maintain at a level that would be beneficial for IE treatment. Furthermore, a correlation between blood ketone levels and a reduction in seizure frequency has not been consistently reported,<sup>21</sup> when compared to the reduction in seizure frequency associated through supplementation of MCTs.<sup>22-26</sup> To add, the high-fat, low-protein and low-carbohydrate requirements of a KD contains a myriad of risks and complications such as vomiting, diarrhoea, constipation and pancreatitis.<sup>27</sup> Like any restrictive diet, the KD diet has also been associated with nutrient deficiencies.<sup>28,29</sup> This has paved the way for research into the uses of exogenous MCT supplementation, which, as discussed below, have been investigated in multiple studies with promising results.

#### MCT Absorption, Efficacy and Neurometabolic Mechanisms of Action

Medium chain triglycerides are comprised of a glycerol molecule attached to three fatty acids, with a hydrocarbon chain ranging between 6 to 12 atoms.<sup>30</sup> Unlike long chain fatty acids (LCFAs), they are rapidly oxidised for energy and are liquid at room temperature.<sup>30</sup> The two MCTs of interest for their anti-seizure, behavioural and cognitive enhancing effects, are octanoic (C8) and decanoic (C10) acid.<sup>30</sup> Medium chain triglyceride's have been extensively reported upon in humans in relation to a range of neurological and behavioural diseases. They have been used as a dietary supplement and an exogenous source of energy to aid in the



amplification of endogenous ketosis in humans for a range of ailments such as refractory idiopathic epilepsy in children<sup>19</sup> and improved memory function in Alzheimer's disease.<sup>31,32</sup> Although both exogenous and endogenous ketones have been clinically used for decades, the full extent of their mechanisms are still being investigated.

#### Digestion and Absorption: LCFAs vs MCTs

When long chain fatty acids (LCFAs) are ingested, they stimulate the release of lingual lipase,<sup>33</sup> gastric lipase and pancreatic lipase in the mouth, stomach and small intestines, respectively.<sup>34</sup> This results in the emulsification of LCFAs into emulsification droplets and then further into monoglycerides and free fatty acids (FFAs).<sup>35</sup> Monoglycerides and FFAs are transported with phospholipids and bile salts into enterocytes, where they are re-esterified to form chylomicrons and transported via the lacteals to the thoracic duct, and subsequently around the body.<sup>35</sup>

In contrast, MCTs are absorbed in the gastrointestinal tract and go directly to the liver via the portal vein.<sup>36</sup> where they are metabolised via  $\beta$ -oxidation and the Krebs cycle into ketone bodies, acetate, and carbon dioxide<sup>37-39</sup> or transported as MCTs in the plasma.<sup>40</sup> Compared to LCFA digestion, this direct pathway causes no significant stimulation of pancreatic secretion in dogs.<sup>40,41</sup>

#### MCTs Have Shown Promising Anti-Seizure Effects

A placebo controlled, double blinded, crossover MCT dietary supplement trial showed 71 % of dogs had a reduction in seizure frequency.<sup>25</sup> Furthermore, 48% of dogs had a  $\geq$ 50% reduction in seizure frequency and 14% of dogs achieved complete seizure freedom.<sup>25</sup>



Figure 3. Effect of the mediumchain TAG diet (MCTD) on (a) seizure frequencies per month and (b) seizure days per month compared with the placebostandardized diet (n 21). There were significant reductions in the (a) number of seizures per month (P=0.0195) and (b) seizure days per month (P=0.0216) during the MCTD phase in comparison with the placebo diet. Data are shown as box-and-whisker plots (central lines of the box represent the median, lower and upper limits of the box represent the 25th and 75th percentiles and whiskers represent the minimum and maximum). Twosided Wilcoxon's matched-pairs rank tests were used to compare placebo and MCTD groups

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stimualtion in the presynaptic axon causes the release of glutamate into the synaptic cleft Glutamate diffuses across the cleft and stimualtes AMPA receptors to open An Influx of Na<sup>+</sup> and Ca<sup>2+</sup> ions enter the post synaptic neuron, causing the resting membrane potential to increase Weak electrical stimualtion in the presynaptic axon causes the release of glutamate into the synaptic cleft Glutamate diffuses across the cleft and stimualtes AMPA

> open C10 blocks the AMPA receptor blocking the influx of Na<sup>+</sup> and Ca2+ and the resting membrane potential remans stable

Figure 4: A schematic diagram of AMPA and NDMA receptor interactions with glutamate. Created by Dr. A. Edwards using biorender

Figure 5: A schematic diagram of decanoic acid's (C10) inhibitory action on AMPA receptors. Created by Dr. A. Edwards using biorender

These encouraging results were further evidenced by a recent trial comparing a C8/C10 MCT oil to a control, where the overall seizure frequency and seizure day frequency was significantly lower in the MCT group than the control group.<sup>26</sup> Berk et al (2019) reported a reduction in phenobarbitone (PB) levels that was thought to be associated with its metabolism via the P450 enzyme pathway. Although there are some disparities reported, multiple studies have demonstrated the anti-seizure effects of C10.<sup>22,23,25,26</sup> These findings have not been replicated for the solo use of C8; however, research has demonstrated when C8 is administered in combination with C10 it has enhanced anticonvulsive effects.24

#### MCT Reduces AED's Adverse Effects

Berk et al., (2020) noted an 8% and 10% decline in PB and alkaline phosphatase (ALKP) levels

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respectively in dogs with IE supplemented with C8/ C10 oil.<sup>26</sup> Reductions of PB levels may relate to MCT metabolism via CYP4 isoforms, enzymes within the P450 family.<sup>26</sup> Interestingly, reduction of PB levels were not seen in previous studies with similar total caloric MCT intake in a dry kibble diet.<sup>25</sup> Despite there being a reduction in PB levels, a significant decrease in seizure frequency was observed.<sup>26</sup> In addition, owners reported a significant reduction in AED adverse effects such as ataxia and anxiety, indicating a better tolerance of AEDs.<sup>26</sup> It is conceivable that the Berk et al (2020) study reported a reduction in AED adverse effects for the first time in the literature due to the oil's high concentration of C10 and its dose-dependent nature.

#### MCT Reduces Symptoms of Canine Hyperactivity and **Cognitive Decline**

In children with IE, hyperactivity-like behaviours

are reportedly 5X more prevalent, which included reduced attention span, learning difficulties and impulsive behaviours.<sup>42</sup> Numerous studies demonstrate a decrease in attention deficit hyperactivity disorder (ADHD) like behaviours in people with epilepsy on a KD,<sup>43,44</sup> with similar results being replicated in rodents<sup>45</sup> and dogs, when fed a MCT-enriched diet.<sup>46</sup>

MCT-enriched diets improved 5/6 measurements of cognitive decline within 30 days of commencement, with significant improvement across all measurements upon the conclusion of the trial's 12-week period.<sup>47</sup> Furthermore, similar findings were replicated by Berk, et al (2020), where significant improvements in canine cognitive abilities was observed, as measured by spatial-working memory, problem solving abilities and owner trainability secondary to the use of a C8/C10 MCT oil.48 In view of these clinical results, outlined below are four mechanisms that may help explain MCT (including C10's) functional anti-seizure and cognitive-enhancing properties. It should be noted that these mechanisms are not mutually exclusive, but rather have been used to assist in understanding MCT's various therapeutic effects.

#### MCT's Mechanisms of Action

#### Mechanism #1: Decanoic Acid (C10) as a Non-Competitive AMPA Receptor Inhibitor

Chang *et al.* (2015) demonstrated in studies on rats that C10's anti-seizure effects are explained by its function as a non-competitive AMPA receptor antagonist. AMPA receptors play a key role in the initial depolarisation and attenuation of seizures, whereas their antagonists are known to supress such activity.<sup>49</sup> Once in the portal blood steam, C10 is either metabolised into ketone bodies or transported as a FFA in the plasma.<sup>40</sup> Like ketones, C10 can cross the blood-brain barrier<sup>50</sup> which is paramount for its direct anti-seizure effects.

As shown in Figure 4., glutamate, an amino acid and an excitatory neurotransmitter, is released secondary to depolarisation of the pre-synaptic axon terminal.<sup>51</sup> Upon release, glutamate diffuses across the synaptic cleft, causing the AMPA receptor coupled ionophore to open.<sup>51</sup> This allows for an influx of Na+ and Ca2+ ions to enter the post-synaptic neuron, causing excitation in the dendrite, and in some cases an action potential.<sup>51</sup> As seizures are characterised by hyper-synchronisation and high frequency burst of action potentials, it is clear that AMPA receptor play a pivotal role in their generation.<sup>22</sup>

By contrast, **Figure 5** shows how C10 binds to AMPA receptors, preventing activation by glutamate, and thus the subsequent the influx of Na+ and Ca2+ ions. This means the resting membrane potential remains hyperpolarised and there is therefore a reduced likelihood of the post-synaptic neuron generating an action potential.<sup>52</sup> This effect was demonstrated to be dose-dependent.<sup>52</sup>

Importantly, C10 is not just an AMPA receptor inhibitor but also a non-competitive inhibitor.<sup>22</sup> This is relevant for dogs experiencing seizures, which involve large synaptic release of glutamate, and where drugs that act as competitive inhibitors are likely to be less efficacious. Although C8 is not known to have the same inhibitory effects on post-synaptic



Figure 6. Glutamate is released secondary to depolarization of the presynaptic axon terminal



Figure 7: A high level summary of the conversion of MCFA into ketones and ATP in a hepatocyte. Created by Dr. A. Edwards using biorender

AMPA receptors as C10, <sup>22, 23</sup> it has long been noted to have anti-seizure and sedative effects, which have been demonstrated to increase the chemically induced seizure threshold and may be linked to its metabolism of tryptophan.<sup>53,54</sup>

#### Mechanism #2: Decanoic Acid Improves Mitochondrial Metabolism and Subsequent Inhibitory Neurotransmitter Function

As the brain ages, the pathways converting glucose to ATP for energy begin to degenerate; this is referred to as glucose hypometabolism.<sup>55</sup> This results in mitochondrial dysfunction and a subsequent cycle of energy deficiency for brain cells, increased inflammation, and oxidative stress within the brain.<sup>56</sup> The increase in oxidative stress adds to the vicious cycle of impaired metabolism.<sup>57</sup> As demonstrated in Figure 7 MCT's are directly transported into mitochondria in the liver, where they are converted via  $\beta$ -oxidisation into acetyl-coenzyme A (acetyl CoA) or ketone bodies.<sup>51</sup> The acetyl CoA can be used in the Krebs cycle to produce energy, and the ketones can be transported in the blood stream for use in peripheral tissues such as the brain.<sup>51</sup>Ketones and FFA (MCT esters) are able to cross the blood brain barrier (BBB) and enter astrocytes as shown in Figure 8. Here they can be used in the Krebs cycle or further transported for use in neurons.<sup>51</sup> This alternative source of energy is hypothesised to aid in the stabilisation of neuronal ion gradients, thereby increasing their resistance to depolarisation.<sup>51</sup> Energy from the upregulation of mitochondrial metabolism may also improve the formation of adenosine, an important inhibitory mediator with well researched anti-seizure properties.<sup>30</sup>

#### Mechanism #3: MCTs Have Immune-Modulating and Anti-Inflammatory Effects

C8 and C10 have shown anti-inflammatory

effects mediated via the down regulation of genes associated with inflammatory factors,<sup>58</sup> and M1 macrophage cytokines.<sup>58</sup> Moreover, they enhance the anti-inflammatory effects of non-M2 macrophages.<sup>58</sup>

Further research is needed to explore ketone's exact mechanism(s) of actions in neurological and behavioural diseases; however, the plethora of hypothesis and evidence, points to the likelihood that their mode of actions is multifactorial.

#### Mechanism #4: Decanoic Acid Attenuates Neural Hyperexcitability

Recent research has demonstrated that C10 in vivo, supresses central and peripheral nerve nociception<sup>52</sup> and that it acts as an antagonist to muscarinic acetylcholine receptors.<sup>59</sup> Muscarinic acetylcholine receptors are the predominant cholinergic receptors in the central nervous system and play a major role in the generation of epileptic seizures.<sup>60</sup>

#### III. Vets Need to Lead Advice When It Comes to Dietary Add-ons

Berk *et al.* (2018) stated that vets were rarely the source of information when it came to dietary interventions used to treat epilepsy, with only 17.5% of owners reporting to have consulted their vet.<sup>61</sup> Of the 297 participants surveyed, 45% reported using dietary add-ons for epileptic management. Concerningly, the two most popular sources of information were online owner support groups (38.2%), and owner's own internet research (20.6%).<sup>61</sup> Of the supplements used by the participants, coconut-derived oils were the most popular (77.3%), followed by fish oil (66.9%).<sup>61</sup> This gap in owner-veterinary advice demonstrates an



Figure 8. A high level summary of energy metabolism from ketones and free fatty acids in astrocytes and neurons. Created by Dr. A. Edwards using biorender

opportunity for vet to discuss clinically relevant information with owners, as many of the supplements available online are not formulated for animals, may have minimal benefits and could potentially cause harm.

Regarding the use of Omega-3, it should be noted that while it has shown to be useful for diseases like cognitive decline and arthritis, there is little clinical evidence for it conferring anti-seizure effects in canines. This was found by Matthews *et al.*, (2014) whose study showed that essential fatty acid supplementation did not reduce seizure frequency and severity in dogs.<sup>6</sup> In relation to the mechanisms of action described earlier and the difficulties in inducing canine endogenous ketosis, this is not surprising, as one of the goals of omega-3 supplementation in humans was to aid dietaryinduced ketosis.

## Potential Side Effects: Choosing the Correct MCT Concentration

Generic, unrefined coconut oil typically purchased off-shelf only apportions 60% of its fatty acid (FA) composition from MCTs, and 20% of its FAs from LCFAs. As discussed earlier, LCFAs are a risk factor for both gastrointestinal issues and pancreatitis in dogs. Furthermore, when refined, MCT oil still contains a large portion of lauric acid (C12).<sup>6</sup> Overconsumption of C12 could mean the excessive consumption of unneeded calories, and the MCT oil having lower concentration of C8 and C10.

## How to Incorporate MCTs Safely and Effectively into Canine Diets

Selection of a commercially available MCT-enriched kibble diet vs a C8/C10 oil supplementation should be gauged by the veterinarian on a case-to-case basis.

There were significant palatability issues reported with MCT-enriched kibble diets that contained 9% MCTs.<sup>47</sup> These issues were decreased by reducing the total MCT concentration.<sup>47</sup> In the lower MCTenriched kibble diets, palatability issues caused 7-14% of the dogs to drop out of the studies.<sup>25,47</sup> There were no palatability issues reported in the C8/C10 oil study, which is potentially due to its odourless and tasteless properties.<sup>26</sup> If feeding a commercial dry kibble diet, we suggest following their recommended guidelines.

MCT oil doesn't typically come in capsule-like format for several reasons For example, if it were in a typical fish oil capsule-like format, 5 large size capsules (2.6mls) would be needed for a 25kg dog. This may be challenging to administer, even with food, and I also believe that the process of administering the oil contemporaneously with the owner's dog food achieves two tasks at once and increases the likelihood of compliance.

If supplementing with a C8/C10 oil, then please refer to Table 1, which is generated form Berk *et al.*,(2020) study.

## If Using Oil, does it Matter What Kind of Food a Patient Consumes with C8/C10 MCT?

While the MCT oil studies allowed owners to use their food-of-choice, research to date has not dissected the impact of C8 and C10's efficacy against certain foods that it may be applied to. It is conceivable that C10's functionality as an exogenous excitatory neurotransmitter inhibitor is relatively independent of the canine food the oil would be mixed in with. This bodes well for owners who may have dogs with a sensitive palate or may not want to pay top-end price for some of the select premium canine kibble diets containing MCTs.



Figure 9. A revision of a typical IE treatment framework that incorporates MCT supplementation designed for enhancing overall treatment efficacy

Body Weight (kg)	C8/C10 Oil (mL)
1	1.0
2	2.0
3	2.5
4	3.5
5	4.0
10	6.5
15	7.5 – 9
20	9.5 – 11
25	11.5 – 13
30	13.5 – 15
35	15.5 – 17
40	17.5 – 19
45	19.5 – 20.5
50	21.0 – 22.5
55	23 – 24
60	24.5 – 26.5

#### **Practical Applications for Clinicians**

The IE treatment model above incorporates research of MCT's therapeutic benefits alongside traditional pharmacological treatment. Importantly, MCTs can work synergistically with AEDs insofar that it reduces AED side effects and reduces seizure frequency.

#### **Questions and/or Orders**

If you have any questions about this paper, would like to have a chat or pre-order some 500mL bottles for your clinic please feel free to reach Alice and/or Tim at the numbers or email addresses below:

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#### cascadeanimalsciences.com

**NOTE:** References available in the eBook.

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Table 1. Recommended daily dosage for dogs being supplemented with C8/C10 oil

#### Take Home Points

- MCTs should be considered as a complimentary therapy for dogs with epilepsy, as it may not only just improve seizure control but reduce the negative side effects such as ataxia and AED induced hepatopathy.
- MCTs should be considered for dogs with hyperactivity-like behaviour or cognitive decline.
- 3. Supplementation of MCTs has few potential side effects and generally good palatability.
- MCTs do not cause significant pancreatic stimulation in dogs, due to their direct absorption and transportation to the liver via the portal vein.



After graduating from Charles Sturt University in Wagga Wagga in 2017 (VBio/BVSc), Alice began practising at PetFocus Vetcare, a small animal practice in Albury where she continues to work as a full-time

veterinarian. Her interest in the effects of diet, metabolism, and the brain (neuro-dietetics) was first sparked when looking for adjunctive treatment options for one of the clinic's vet nurse's dog with IE. At the time, they tried to source Purina's Neurocare. However, due its cost and lack-of-availability, she started advising owners to use C8/C10 MCT oil but was faced with compliance barriers, as there were no veterinary options available. The logical step seemed to be to fill that gap and make it easier on both veterinarians and owners to access this very simple but beneficial adjunctive therapy, with an off-the-shelf option. Luckily, her partner Tim has a business background which has allowed her to focus on the clinical aspects of Cascade.

Her research in this area has been driven by her responsibility to be as science-focused as possible. Whilst complex, she believes that it's not out of the GP vets' scope to consider the importance of a patient's metabolism, gut and brain function in relation to overall health and disease.

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## **VETERINARY CARE OF THE PET BEARDED DRAGON**

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Robert Johnson worked in small animal, zoo and wildlife practice in Sydney. He has also worked as a clinical veterinarian at Taronga Zoo. Robert holds a Certificate in Zoological Medicine from the Royal College of Veterinary Surgeons and is a member of the ANZCVS.

His professional interests include clinical practice, particularly reptile medicine and research involving reptilian species of Australia and the South Pacific, and animal welfare. Robert also lectured in reptile and wildlife medicine at James Cook University Townsville. He is the co-author of *A Guide to Health and Disease in Reptiles and Amphibians*, a co-editor of Reptile Medicine and Surgery in Clinical Practice and has published and presented widely on matters reptilian. Robert also served as President of The Australian Veterinary Association 2015-2017. He is also a director of the board of Vets Beyond Borders.

In addition, Robert is the tutor for CVE TimeOnline courses for unusual pets.



Figure 1. Central bearded dragon

#### Introduction

Bearded dragons (Family: Agamidae) are commonly kept as pets both in Australia and overseas (USA, UK, Europe). In decreasing order of popularity, the significant 'beardie' species are the Central bearded dragon, *Pogona vitticeps*, the Dwarf bearded dragon, *Pogona henrylawsoni*, and the Coastal bearded dragon, *Pogona barbata*. Other dragon species that are less frequently kept as pets include the Eastern water dragon, *Intellagama lesueurii*, the Frilled lizard *Chlamydosaurus kingi*, the Central netted dragon, *Ctenophorus nuchalis*, Boyd's Forest dragon, *Hypsilurus boydii*, and the Southern angle-headed dragon, *Hypsilurus spinipes*.

#### HUSBANDRY AND HISTORY

Extensive reviews of bearded dragon husbandry and welfare appear in the literature (Cannon, 2003; Grenard, 2001; Johnson R, Adwick S, 2019). When designing an enclosure for a bearded dragon a dilemma exists between providing a 'naturalistic' habitat with sand as a substrate and an easy to clean, paper substrate that will limit the spread of parasites. The provision of an ultraviolet light source (UVB), day/night cycle and a thermal gradient within the preferred optimal temperature zone is mandatory for good reptile health. Access to unfiltered natural light on a regular basis is also recommended. Behavioural enrichment is necessary. If lizards are kept on paper, gravid females will

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Figure 2. Natural stance of a healthy bearded dragon

require a small tub or box of sand in which to dig. Hatchling and juvenile dragons are obligate insectivores. As lizards mature, they will become more omnivorous in their tastes, eating fruit, vegetables, and some flowers.

**THE PHYSICAL EXAMINATION** (Johnson, 2004) Healthy lizards should have good muscle tone, a 'proud' stance, being able to lift the pectoral region off the ground and respond positively to touch.

Sick dragons may show any of the following signs, with possible diagnoses in brackets—the 'shakes' or twitching (metabolic bone disease [MBD]), size disparity (gastrointestinal parasites, bullying of food and basking sites), stunting or musculoskeletal deformities (MBD), skin lesions (bacterial, fungal dermatitis), listlessness (suboptimal cage temperature, MBD, renal disease, follicular stasis, retained eggs), sudden death (adenovirus, MBD), anorexia (suboptimal cage temperature, parasitism), constipation (overfeeding, renal disease), dehydration (renal disease, high cage temperature), diarrhoea (parasitism, bacterial gastroenteritis), prolapse (MBD, parasitism, systemic disease), obesity, and stomatitis.

Look for trends when assessing body condition and weight in dragons rather than trying to determine an ideal weight. Sex identification can be difficult in young lizards. As dragons mature the sexes can be distinguished by physical and behavioural features. Male bearded dragons have bilateral hemipenis



Figure 3. Male bearded dragon, note the typical hemipenal swelling of tail base and femoral pores

swellings just caudal to the cloaca and a thicker tail base. Femoral pores on the medial side of the hindlimbs are also more prominent in the male.

Male coastal bearded dragons tend to have a black beard. Both sexes will 'head bob' and 'hand wave'; however, the males appear to play a more dominant role. Examination of the oral cavity will detect early signs of stomatitis and gingivitis. Periodontal disease and gingival recession are common in captive agamids.

Routine dental checks and prophylactic therapy are recommended. Juvenile lizards with MBD may suffer from `rubber jaw.'

Oral nematodes may be present in some wild caught agamids (Griffiths *et al.*, 1998; R Johnson, *personal observation*). Unlike snakes, lizards shed their skin in pieces. The underlying 'new' skin should be soft and free of any blemishes. Crusts and ulcers should be investigated. Mites occur less commonly on agamid species compared with large skinks such as blue-tongued skinks (*Tiliqua* spp.). Ticks are common on dragon species housed in outdoor pits or aviaries. Eye conditions including traumatic injuries, blepharitis, hypopyon, hyphaema and pseudobuphthalmos occur (R Johnson, *personal observation*). Rostral abrasion due to 'escape behaviour' occurs more commonly in Eastern water dragons.





Figure 4. Gingival recession and inflammatory stomatitis



Figure 5. Rubber jaw in a dwarf bearded dragon with MBD



Figure 8. Normal appearance of faeces, urates and urine



Figure 9. Oxyurid egg in the faeces of a bearded dragon



Figure 6. Aspergillus blepharitis in a coastal bearded dragon



Figure 7. Venepuncture of the ventral tail vein



Figure 10. Intubation of a bearded dragon

#### SAMPLE COLLECTION & BASIC LABORATORY TESTS

#### Blood

Blood is collected from the ventral tail vein, using a ventral approach with the body of the lizard held vertically to aid venous drainage. Blood parasites, *Plasmodium* sp., *Trypanosome* sp., and microfilaria may be detected in the blood of wild caught agamids. In captive lizards such infestations are usually of no clinical significance and self-limiting due to the absence of an intermediate host. Idiopathic hyperglycaemia and leukaemia (Suedmyer, 1996; Tocidlowski, 2001) have been reported in bearded dragons. Persistent hyperglycaemia associated with gastric neuroendocrine carcinomata in bearded dragons has been reported (Ritter *et al.*, 2009).

#### Lavage

Gastric or cloacal lavage may be used to sample gut contents or faeces. A small amount of warm saline is introduced using a crop needle or soft ended rubber or plastic tube attached to a syringe.

#### Faecal analysis

The same principles used for mammalian faecal analysis apply to reptiles. A direct wet preparation should be carried out followed by a faecal floatation test if the sample is large enough. Acid fast staining of faecal smears for bacteria and *Cryptosporidium* sp. may also be carried out. Any reptile found to have a negative sample should be re-evaluated as transient shedding may occur (Mitchell, 2010).

#### What am I looking for?

Oxyurid nematodes and coccidia (*Isospora* sp.) are common parasites of bearded dragons and frilled lizards (Pasmans *et al.*, 2008; Stahl, 2003). The spirurid, *Abbreviata physignathi* and the heterakid *Spinicauda fluviatica*, sp. nov., are the predominant nematode species inhabiting the gastrointestinal tract of Eastern water dragons, *I. lesueurii* (Jones, 2007; R Johnson, *personal observation*). Spirurids require an intermediate host whereas Heterakoidea have a direct life cycle (Jones, 2007). Cryptosporidium have been identified in a wide variety of reptile species.

#### The Reptile Panel

In general practice the following reptile panel (haematological, biochemical, and faecal) is recommended:

- Full blood and differential count (manual)
  - Relevant biochemical analytes—uric acid (UA), creatinine kinase (CK), aspartate aminotransferase (AST), glucose, calcium, phosphorus, total protein, and bile acids

Faecal analysis for protozoan and nematode parasites, and faecal culture as required. A recent study of the haematology and plasma biochemistry reference intervals in wild bearded dragons has been published (Howard, Jaensch, 2021).

## TREATMENT AND CONTROL OF NEMATODES AND COCCIDIA

## Should a clinically well lizard with faecal oxyurids and coccidia be treated?

Coccidia may be commensal organisms of no pathogenic significance. If a lizard is visibly ill and has a heavy faecal load of coccidia, treatment is indicated. Stahl (2003) uses the term `superinfestation' to describe a higher-than-expected level of parasitism that requires husbandry adjustment or treatment. Bearded dragons are commonly infected with the coccidian parasite Isospora amphiboluri, a species with a direct life cycle. Eggs, larvae, and oocysts will survive in the environment without the presence of an intermediate host (Pasmans et al., 2008). Low level infestations may be controlled by improvements in husbandry practices without resorting to anticoccidial therapy. In a survey of captive reptiles (Pasmans et al., 2008) it was reported that 43.5% of agamids were infested with Isospora sp. and 61.6% with oxyurids.

Fenbendazole (Panacur<sup>®</sup>, 100 mg/mL) at a dose rate of 50-100 mg/kg q 7d, 3-4 doses, is recommended for nematode treatment. A palatable bird wormer (Wormout Gel<sup>®</sup>, Vetafarm, oxfendazole 20g/L and praziquantel 20g/L) is used by the author to `worm' agamids at a dose rate of 0.35mLs/kg body weight, repeated in 14 days. For the treatment of coccidia, sulfadimethoxine is given orally at 50 mg/ kg (or trimethoprim-sulfa at 30 mg/kg), daily for 5 days, then every other day until the coccidia is eliminated based on follow-up faecal examinations (Stahl, 2003). This protocol is used in conjunction with supportive treatment including fluids, soaks, assisted feeding and appropriate husbandry changes (Stahl, 2003). Lizards hosting oxyurid infestations are often asymptomatic. Some sources suggest that they are beneficial, preventing constipation by improving passage of ingesta through the gastrointestinal tract (Telford, 1971).

#### MICROCHIPPING

Microchip transponders are implanted subcutaneously or intracoelomically in the left caudal body.

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#### ANAESTHESIA

A recommended anaesthetic protocol is as follows premedication with morphine at a dose rate of 5 mg/kg IM followed 30 minutes later by intravenous alfaxalone at a dose rate of 5 mg/kg via the ventral tail vein. For prolonged or invasive procedures, lizards should be intubated and maintained on isoflurane/oxygen. Midazolam (0.5mg/kg IM) can be used to supplement other premedications. Patients should be maintained at their POTZ (Preferred Optimal Temperature Zone, 26°C - 29°C with access to a basking site at 40°C) before, during and after anaesthesia (at least until fully recovered).

#### SPAYING

Indications for ovariectomy in the female bearded dragon include pre-ovulatory follicular stasis, chronic egg laying and associated dystocia. The surgical approach is via a paramedian incision, avoiding the midline coelomic vein. Follicles are easily identified, ligating the ovarian vessels with surgical clips or sutures. Muscle and skin closure technique is similar to small mammals using 4/0-5/0 PDS depending on the size of the lizard. Postoperative analgesia is provided with meloxicam (0.2mg/kg SC or PO q 48h) or carprofen (4mg/kg q 48h) for 4 to 5 days.

## COMMON CONDITIONS OF BEARDED DRAGONS

#### METABOLIC BONE DISEASES

The term 'metabolic bone disease' is not actually

a single disease entity but rather a term used to describe a collection of medical disorders that affect the integrity and function of bones.

Metabolic bone diseases (MBDs) comprise the most common disease condition affecting captive amphibians and lizards. In reptiles and amphibians, it is important to distinguish between metabolic bone disease of nutritional origin (NMBD) and renal (kidney) origin (RMBD). The nutritional form is the more common, usually as a result of an improper calcium:phosphorus ratio of the diet.

#### Nutritional metabolic bone disease (NMBD)

The predominant causes of NMBD, also known as Nutritional Secondary Hyperparathyroidism (NSHP), in reptiles are prolonged deficiency of dietary calcium or vitamin  $D_3$ , an imbalance of calcium:phosphorus ratio in the diet (usually an excess of phosphorus), or inadequate exposure to ultraviolet (UV) radiation. The condition is uncommon in snakes as they usually eat whole food items and are therefore more likely to receive balanced nutrition.

#### Vitamin D synthesis

UVB (285-315nm) reacts with the skin. Cholesterol in the skin is then converted to the inactive form of vitamin D, which in turn is metabolised in the kidney and liver to the active form. Vitamin D is used by the body to enable absorption of calcium from the intestinal tract.



#### Captive reptiles

Captive animals may not have adequate exposure to UVB due to several factors:

- UVB does not penetrate Perspex or glass
- Incorrect placement of UV lights
- UVB lights are not replaced as per manufacturers' instructions
- Social hierarchy and domination of basking sites
- Poor husbandry, e.g., UVB supplementation not supplied or inadequate (see Appendix 1), animals not able to thermoregulate adequately
- UV light in the enclosure is not working correctly or producing the wrong type of UV

#### How does MBD occur and what should you look for?

A reduced calcium level in the blood leads to an excessive production of hormone from the parathyroid glands. Calcium is lost from the bones to make up for this deficiency. The result is weakened bones leading to rickets (young animals) or osteomalacia (adult animal). Lizards of all ages can be affected.

#### The effect on nerves and muscles

Calcium deficits lead to nerve and muscle dysfunction—signs seen in lizards include twitching of the digits, tremors, disorientation, and abnormal movement, prolapse of the cloaca, constipation, weakness, paralysis, or seizures.

#### Clinical signs

Other signs include:

- stunted growth-rostral `rounding', small head
- demineralisation of bones—soft pliable bones, especially the mandible
- neurological signs—see above
- musculoskeletal signs—spinal deformities
   including scoliosis, fractures
- fibrous osteodystrophy—thickness and swelling of long bones and the mandible
- prolapse-rectal or cloacal
- · constipation/impaction
- · decreased appetite or anorexia
- weight loss-adults

#### Diagnosis

Diagnosis is based on history, diet (low calcium or suboptimal Ca:P ratio), husbandry factors (inappropriate or no UVB) and typical clinical and radiographic signs.

#### Treatment

Life-threatening signs such as paresis and seizuring are treated first. Calcium gluconate is administered every 6 hours until tetany ceases. After stabilisation, the following protocol is used:



Figure 12. Stunting of the head and body in a bearded dragon with MBD

#### Initial treatment:

- Provision of the preferred optimal temperature zone (POTZ) of the species to thermoregulate and achieve the Preferred Body Temperature (PBT)
- Oral calcium supplementation
  - o Ca glubionate (Calcium Syrup) 10mg/kg twice daily for 1-3 months
  - o Calcium carbonate-sprinkle on food
- Fluid therapy-parenteral fluids may be required if dehydration is significant, and the lizard is not responding well to oral fluids
  - Rehydrate initially using Jarchow's solution (one part, Hartmann's solution: two parts, 0.45% saline + 2.5% dextrose), 20-25mLs/ kg sid
- If not eating assist feeding after rehydration
  - Vegetables, baby foods, avian feeding formulae, Hill's a/d® + Oxbow Critical Care®s, invertebrates
  - o 20mLs/kg/1-2 days
- Soaking in warm water—not full immersion—for 10-20 minutes once or twice daily may aid rehydration.
- Provide a safe environment to avoid injury, and limit handling

#### Long-term treatment:

Husbandry changes should ensure or provide:

- adequate exposure to UVB (see Appendix)
- access to natural light
- appropriate UVB lights
- UV lights are regularly changed
- set at correct distance
- opportunities for basking are available to ALL the reptiles in the vivarium
- food is rich in vitamin D2 and vitamin D3 is available
- appropriate level of calcium in diet (`gut loaded' insects, dusting food items with calcium)
- adequate heat-maintain POTZ and PBT

Causes of renal disease in lizards include dehydration, improper diet (high protein, high vitamin D content), infectious nephritis, trauma, fibrosis, dystrophic calcification, amyloidosis, neoplasia, toxins. Diagnosis is based on clinical signs (listlessness, dehydration, weight loss, muscle atrophy), radiology and ultrasonology. Blood biochemistry may reveal a calcium:phosphorus ratio of less than one and a hypoproteinaemia. Renal biopsy will give a definitive diagnosis (easier in large lizards, e.g., iguanas but more difficult to perform in bearded dragons). Treatment involves the use of phosphate binders-1mL/500g body weight PO bid, of 1g lpakitine® (Vétoquinol) diluted in 9 mLs of water. Supportive care should include fluid therapy, the provision of an appropriate thermal gradient, UVB, a reduction in handling and good quality nutritional support. Antibiosis is indicated if an infectious cause is suspected. Reptiles with renal disease may also have concurrent illness such as gut impaction or pre-ovulatory follicular stasis.

#### Impaction of the Colon (Wright, 2008)

Impaction of the colon, often accompanied by loss of appetite, is a common clinical problem of captive bearded dragons.

#### Husbandry factors

After ruling out NMBD other contributing causes to be considered include inappropriate substrate (e.g., leading to ingestion and impaction), lack of sufficient leafy greens in the diet (e.g., roughage), overfeeding (impaction with chitin), low water intake, vitellogenesis, poor hygiene, recent acquisition or recent changes in housing, over-handling, old age, and a history of trauma.

Depending on the chronicity of the condition, the clinical signs of the lizard may vary. The differentials for a bearded dragon with constipation include



Figure 13. Necropsy of a veiled chameleon with follicular stasis and impaction of the colon

an obstructive mass in the colon (including urates), dehydration, follicular stasis/ dystocia, gastrointestinal parasitism (e.g., flagellated protozoa, amoebas, coccidia), anatomical malformation of the pelvis and spine (e.g., NSHP, trauma), a GIT foreign body, renomegaly/renal failure, an obstructive mass outside the colon (e.g., abscess, tumour), intussusception and neoplasia.

#### Urate Masses (Wright, 2008)

A urate mass within the colon is the most common cause of impaction in bearded dragons. Such a mass should be differentiated from naturally occurring conditions or normal anatomy such as caudal coelomic fat bodies and ovarian follicles. This is easily done by palpation or ultrasound examination. The urate mass is usually firm and cylindrical. More rarely tumours or abscesses also occur. In healthy, well-fed, well-hydrated, active bearded dragons, semisolid urate plugs are discharged at the start of defecation. In lizards suffering from impaction, urates travel in a retrograde direction into the colon.

#### Husbandry factors

According to Wright (2008) captive bearded dragons suffer from 5 key husbandry errors leading to urate obstruction. Points 2–5 decrease the frequency of excretion increasing the length of time that water can be absorbed from the colonic urate fluid.

- Chronic subclinical dehydration. Despite being mainly dry climate reptiles, bearded dragons require continual access to water. Fluid is lost from the lumen of the colon, causing the walls of the colon to contract around the urate fluid, causing it to solidify and adhere to the mucosa.
- 2. Failure to provide a warm enough hot spot in the environment for digestion to occur at a reasonable rate.
- 3. Feeding insufficient quantities of roughage to promote regular excretion.
- 4. Insufficient exercise.
- 5. Overfeeding, leading to obesity and decreased muscle tone.

#### Other factors

- 1. Lizards with a history of NMBD appear to be more at risk of impaction, particularly if they have pelvic or spinal deformities.
- 2. Prior or existing colitis (e.g., may be caused by flagellated protozoans or coccidia).
- 3. Gastrointestinal foreign bodies (e.g., walnut shell substrate, calcium sand substrate).
- 4. Old age, or
- 5. Painful conditions (e.g., hemipenal infections, trauma to the pelvis or spine), all appear to increase the risk.
- 6. Many ovulating females may have constipation issues that disappear after egg laying.

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age



Figure 14. Prolapse of the cloaca and lower bowel in a male bearded dragon

#### Treatment

Rehydrate lizards by soaking in lukewarm shallow water for 30 to 120 minutes. Occasionally parenteral fluids are needed (e.g., Jarchow's solution). Some dragons will excrete on their own during the bath or respond to cloacal stimulation with a lubricated cotton bud. In some cases, an enema may be required, which is administered by inserting a lubricated catheter or crop needle into the cloaca guiding it carefully into the proctodeum. Gently flush with warm water (35-40°C) and obstetrical lubricant (50:50). The volume required varies according to the size of the patient. A volume of 5 to 10 mLs can be administered slowly to an adult bearded dragon. Once rehydrated, pain relief can be administered as required (meloxicam 0.2mg/kg IM q 48hrs).

A direct faecal examination is recommended to assess the parasite load, looking for flagellate protozoa, coccidia and oxyurid nematodes. Examination of the cloaca using an otoscope or endoscope is useful in cases refractory to standard treatments.

Many dragons can be managed as outpatients with an emphasis on correcting the husbandry errors previously discussed. Soaking in warm water (35-40°C) for 30 minutes daily for 7 days and then every 2 to 4 days will help with hydration and may stimulate defaecation. Spray-misting the bearded dragon daily may be adequate in some cases. Any diagnosed underlying conditions should be treated. NSHP should be addressed with supplemental calcium, heat, and UVB lighting. An enterotomy to remove impacted foreign material or an exploratory to assess an impinging coelomic mass may be necessary to resolve the presenting complaint of constipation.

Occasionally bearded dragons will continue to have issues after husbandry corrections and frequent bathing. Some may respond to cisapride (1-4 mg/ kg orally, once a day until defaecating). Assist feeding 3 to 6 mL of a high-fibre diet daily to every 3 days may stimulate defaecation, for example, Hill's a/d<sup>®</sup>: Oxbow Herbivore Mix (Fine Grind)® in a 50:50 mix.

#### Prolapse

#### Hemipenis

Prolapse of the hemipenis often occurs post mating or in conjunction with trauma or metabolic bone diseases (MBDs). Amputation may be necessary if the everted hemipenis cannot be reduced by topical applications of sugar and lubricant. It should be emphasised that the hemipenis does not include a urethra or similar structure and is purely an intromittent organ.

#### Cloaca

Prolapse of the cloaca, bladder (in reptiles with a urinary bladder), lower bowel, or oviduct often occurs in cases of MBD or generalised weakness.

#### Periodontal disease

Periodontal disease and gingival recession are common conditions in bearded dragons. Mitigating factors are poor diet, poor cage hygiene and husbandry practices. Treatment involves dental scaling and topical oral preparations. Occasionally systemic antibiotic therapy is required.

#### Fractures

Limb fractures occur more commonly in young dragons or as pathological fractures in older lizards with MBD (gravid females). Often the limb can be strapped or even glued to the body (FLs) or tail (HLs)

Figure 15. Regular oral examination is recommended





Figure 16. Splinting of a distal limb fracture

with good effect. Long bone fractures in adult lizards can be fixed internally or using external fixateurs; care must be taken as lizard bones are fragile, especially if an MBD is suspected. Radiographs should be taken to check for bone density.

#### ABSCESS

Ideally, resection of the abscess contents and capsule is required. Wounds should be marsupialised or a drain inserted for postoperative flushing, often for several weeks. Enrofloxacin infused poloxamer gel is useful as a slow-release antibiotic treatment. The gel is injected slowly into the wound and replaced as required over a three-to-four-week period.

#### ANEURYSM

Aneurysms have been reported in bearded dragons. Firm but fluctuant swellings appear usually in the head and neck region. The cause is unknown.

#### YELLOW FUNGUS (Johnson et al., 2011)

CANV (*Chrysosporium* anamorph of *Nannizziopsis vriesii*) has been identified as a primary diseasecausing agent in several species of reptiles including bearded dragons.

The CANV is a keratinophilic ascomycetous fungus that causes contagious dermatomycosis in inland bearded dragons (*Pogona vitticeps*). Skin infection slowly progresses to systemic and often fatal disease and the condition has been referred to as 'yellow fungus disease'. Recent molecular analyses of morphologically similar fungal isolates formerly included within the '*Chrysosporium* anamorph of *Nannizziopsis vriesii* (CANV) complex' have led to major taxonomic revisions. *Nannizziopsis* now includes nine species associated with chamaeleonid, gekkonid, cordylid, teiid, agamid, and iguanid lizards; crocodiles; and human hosts (Paré, Sigler, 2016).

#### History and Clinical Signs

There is strong evidence that Nannizziopsis sp.



Figure 17. Splinting of a distal limb fracture

Infections are highly contagious among bearded dragons, especially when husbandry factors are compromised, and the lizards are under stress. More commonly outbreaks in multiple animals occur rather than individual cases within a collection. Skin lesions initially appear as thickened, crusty, oozing lesions. Whole limbs may be affected, and in advanced cases bone and internal organs are involved.

#### Diagnosis

Diagnosis of infection with the CANV is based on typical clinical signs and histopathology and confirmed by culture and sequencing of the fungus. Depending upon the experience of the laboratory technician, culture and identification of the fungus can be difficult. Historically, the CANV has been repeatedly confused with the morphologically similar fungus *Trichophyton* sp.

#### Recommended Treatment for Yellow Fungus

Treatment is aimed at removing the dead and infected tissue which may even necessitate amputation of an affected limb. Antifungal treatment, both topical and systemic, may need to be continued for many months.

- Surgical excision of lesions
- Antifungal therapy (topical and systemic).

## Figure 18. Deep skin infection caused by Nannizziopsis barbata



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Lesions may take weeks or months to regress

- Topical treatment after excision/debridement/ cleaning of affected skin
  - o Miconazole, silver sulphadiazine, dilute formalin baths, iodine-based
- Antiseptics
- Systemic treatment
  - o Itraconazole (5-10mg/kg PO sid) drug of choice for filamentous fungus
  - o Constantly monitor for liver damage (from antifungal drugs)
  - Voriconazole and terbinafine have been identified as possible alternatives to itraconazole in the treatment of yellow fungus disease
- Keep patient at higher end of normal temperature (most isolates of CANV do not grow at 37°C, except for the bearded dragon isolates)

#### Prevention

- Proper husbandry
- Appropriate water quality, good hygiene, optimal humidity, temperature, and diet
- Avoid stressors when possible
- Earlier diagnosis and implementation of a systemic treatment plan is important for a full recovery.

## FOLLICULAR STASIS – THE REPRODUCTIVE TRAFFIC JAM

Pre-ovulatory follicular stasis (PFS) occurs when normally active ovaries develop mature follicles that fail to ovulate. The condition is common in bearded dragons. Surgical treatment of PFS should involve complete ovariectomy. More recently medical treatment of PFS in the green iguana, *Iguana iguana*, has been proposed using the GnRH analogue, deslorelin. Cases of postovulatory stasis (fertilised eggs that 'become stuck') may occur when there is imperfect or abnormal calcification due to the egg not passing along the oviduct. Treatment of postovulatory conditions involves surgical removal of the egg and ovariectomy. Supportive care is often required to treat underlying loss of body condition, dehydration, and infection.

## ADENOVIRUS INFECTION (Hyndman & Shilton, 2011; Doneley *et al.*, 2012)

The first reported association between adenovirus and disease in Bearded dragons was in New Zealand in 1982; since then, it has been widely detected throughout the US and is now considered endemic in that country and Europe. Despite its widespread distribution in the United States, the disease has only been previously reported in individual lizards in Australia until recently. Adenovirus appears to affect mainly juvenile lizards.



Figure 19. Opisthotonos in a bearded dragon due to adenovirus. Photo courtesy Bob Doneley

#### Clinical signs include:

- Failure to thrive compared with unaffected clutch mates
- · Opisthotonos and torticollis
- Fine tremor of the head and sometimes the body

#### Diagnostic testing

- PCR (Tim Hyndman, Murdoch University, Perth, WA)
- Submit cloacal swabs
- Gross pathology—poor body condition with epaxial muscle atrophy. The liver is often small, irregular in shape and pale in colour.
- Histopathology:
  - Liver lesions include hepatocellular disarray with scattered random multifocal to coalescing foci of degeneration and singlecell to small-group necrosis, admixed with aggregates of melanomacrophages, fewer heterophils and lymphocytes, and varying amounts of fibrous tissue
  - Round to oval 5-10µ diameter, smudgy, basophilic, hyaline intranuclear inclusion bodies which marginated nuclear chromatin can be seen in the liver, renal epithelium, oesophageal mucosa, oral mucosal epithelium, gastric mucosa, and pancreatic exocrine epithelial cells
  - Concurrent parasitism including coccidiosis (intestinal and hepatic) and cryptosporidiosis are common

#### Treatment

Supportive care including parasite control, assisted feeding and appropriate environmental control may assist some affected lizards to survive.

#### Prevention and control

A carrier state (clinically normal dragons shedding the virus in faeces) appears to be the most likely source of infection. PCR testing of breeding animals and new introductions should be performed to help prevent and control this disease in a collection. Environmental disinfection can be difficult because of the hardiness of non-enveloped viruses.

*Note*: For an extensive list of conditions commonly affecting captive bearded dragons consult Schmidt-Ukaj S *et al.* (2017).

#### ZOONOSIS

Salmonellosis has been reported as a zoonosis in a wide variety of reptile species (Moffatt *et al.*, 2010).

#### EUTHANASIA

A two-stage technique is recommended for euthanasia (Carmel and Johnson, 2008). Sedation with a ketamine/medetomidine (IM) combination, tiletamine/zolazepam (Zoletil®) (IM), morphine and midazolam (IM) or alfaxalone (IV) followed by intracardiac pentobarbitone is recommended. The agamid heart, as with most lizard species, is more cranial in location compared with mammals and access for injection is easiest via the axilla while the lizard is gently held in ventral recumbency. Freezing of reptiles to induce torpor and subsequent death is unacceptable.

## NOTES ON SUPPORTIVE CARE AND TREATMENTS

#### ANTIBIOTIC THERAPY

Increasingly more data have become available on the pharmacokinetics of antibiotics in reptiles (Eatwell, 2007). More detailed knowledge of the renal portal system of reptiles enables the clinician to select appropriate antibiotics, thus avoiding premature drug excretion by the renal tubules (penicillins) or nephrotoxicity (aminoglycosides). Consequently, penicillins and aminoglycosides should be injected only in the front half of the reptile. Despite undergoing hepatic biotransformation fluoroquinolones do not have a high liver extraction rate and blood levels are similar for oral and parenteral dosing (Holz et al., 1997). Culture, both aerobic and anaerobic, and sensitivity studies will enable the clinician to select the appropriate antibiotic. Dose rates of antibiotics used in reptiles may be determined by considering previously recorded empirical doses, metabolic scaling, and current pharmacokinetic studies, if available (Eatwell, 2007). Certain antibiotics such as fluoroquinolones and aminoglycosides are effective when used as a pulse therapy. Others such as penicillins require a constant plasma level to achieve a therapeutic effect (Eatwell, 2007). Extensive information regarding antibiotic choices and dose rates in reptiles are available in the literature (Hyndman, 2018; Perry, Mitchell, 2019).

#### NUTRITIONAL SUPPORT Oral gavage

Oral gavage should be reserved for reptiles that have not suffered significant trauma, specifically injuries to the head and cranial body. Crop needles or short feeding tubes can be used to deliver alimentation. In some lizards where oral gavage is difficult and potentially stressful for the animal an oesophagostomy tube may be placed.

#### Hand feeding

Recovering reptiles may respond to hand feeding small prey items such as crickets and cockroaches. Water dragons may need to have food items such as crickets and other small insects added to the water bowl rather than be fed on a dry surface.

#### **APPENDIX**

#### UV LIGHT REQUIREMENTS FOR CAPTIVE REPTILES HELD INDOORS (Harlow, 2006)

#### Species requiring UV

All species of lizards from the Agamid (Dragons), Iguana and Chameleon families, as well as all Chelonians (turtles and tortoises) and hatchling crocodilians should have access to UVB radiation. Snakes and all other lizards (skinks, goannas) do not seem to require UVB light to maintain good captive health indoors.

Up to about two weeks without UVB exposure is usually no problem for a healthy reptile; however, many of the reptiles seen in practice are already compromised and may benefit from UVB exposure sooner.

#### **Minimum Exposure**

Ideally a UVB meter should be used to check the output of UV globes. Aim to give those species that require UV the equivalent of one hour's exposure per day at 300-  $500 \mu$ W/cm2 UVB. Therefore, in two hours exposure they would be given  $150 - 250 \mu$ W/ cm2 per hour. See below for hourly rates required to give equivalent total daily exposure:

Hours per day	UV exposure in µW/cm2
1	300 - 500
2	150 – 250
4	75 – 125
6	50 - 80
8	40 - 60
10	30 - 50

Table 1. Hourly rates for UV exposure

1965

Est.

Centre for Veterinary Education |



Figure 20. Regular checks of UV lights are recommended

In Sydney at midday on a clear mid-December day the sun produces 400  $\mu W/cm2$  of UVB and in midwinter it is about 30  $\mu W/cm2$  of UVB.

#### **UVB light Sources**

These are either UV fluorescent tubes or mercury vapour (MV) lamps. UV fluorescent tubes usually have much lower UV output, so need to be mounted much closer to the basking site than MV lamps or be turned on for more hours each day.

A 300 W 'Ultra Vitalux' MV lamp emits 80 - 100  $\mu$ W/cm2 at the 30 cm basking site (through wire lid), while the Zoo Med 'Reptisun' 5.0 UVB 40 W fluorescent tubes give 25 - 30  $\mu$ W/cm2 at 30 cm (unobstructed). To achieve the same total daily UVB exposure 'Ultra Vitalux' are usually set for 3 hours exposure per day, while the Zoo Med 'Reptisun' are turned on for 10 hours per day.

Remember, in those species listed above:

- Even if fed calcium to excess, dietary calcium cannot be properly synthesised (for bone, blood, and other metabolic requirements) in the absence of UVB radiation or adequate heat
- UVB light does not penetrate glass (up to 99% absorbed), most clear plastics or water (~ 50 % loss every 1 cm depth)
- Young, fast growing reptiles require much more UVB than slow-growing adults
- UVB is not utilised until the reptile is within its normal 'preferred body temperature' range, so it is of little benefit having UV lights on in an unheated cage and before a reptile has had a chance to 'warm up'
- Routinely UVB output is measured at the closest basking site; however, in most cases it is usually not known how long each day an individual is actually at that site



Figure 21. An outdoor enclosure providing optimal UV exposure

The actual amount of UVB emitted by a light source decreases with usage, even though visible light does not. The UV output of all globes should be checked at least annually.

- Social hierarchies among cage mates may mean that subordinate individuals are always kept away from the basking site, and thus may get no UV exposure
- UVB radiation drops off rapidly as the distance from the light source increases. See table below

Distance from globe (cm)	Average UVB from a 300W Osram 'Ultra- Vitalux' UV globe (µW/ cm2)
25	400
50	120
75	60
100	30
150	5

Table 2. Level of UVB detected at set distances

NOTE: References and bibliography available in the eBook. ■



Download - a Bearded Dragon Care Sheet by Vet Emma Hall bit.ly/beardeddragon-cs



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