Issue 296 | September 2019

C&T

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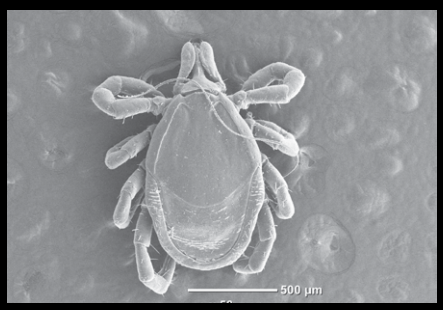

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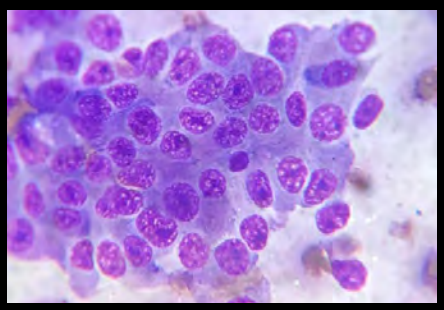

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From the Deputy Director

As I write this, it is national RUOK? Day. In this profession we often find ourselves swamped and sometimes overwhelmed by the volume of work and level of responsibility we shoulder every day. I was listening to a podcast on the way in to work and it spoke of the importance – and sometimes the awkwardness – of asking someone, 'are you ok?' It could be the lifeline someone needs to realise they are not alone.

Being aware of the potential psychological costs of this career is one thing; safeguarding oneself against it is another. But we are eager to make change: as evidenced by the fact our *Mental Wellbeing for Veterinary Teams Symposium* has completely sold out. Funded by the proceeds of the *Vet Cookbook*, even the wonderful sponsors have embraced the spirit of the occasion by forgoing promotional displays in order to accommodate more participants. For those of you that can't attend, we will make content readily available following the event.

Last week I attended the *Robert Dixon Memorial Animal Welfare Symposium* which this year was a discussion of the application of the One Welfare framework to companion animal feeding. This concept is based on the understanding that animal welfare, human wellbeing and environmental sustainability are all inextricably linked. The symposium was a lively discussion about the ethics, welfare implications and future of what we feed our companion animals, including how our relationships with our pets have changed over time and what our species' preferences may be in the coming decades.

We are more acutely aware than ever of the interconnectivity of all forms of life on the planet and the impact our decisions and preferences have on more than just ourselves. If you are interested in exploring the One Welfare concept further, including discussions of how different disciplines can collaborate to explore solutions to some of the most pressing challenges of our time, you might like to attend the second international One Welfare Conference in October: cve.edu.au/conference/one-welfare-conference-ii.

We achieved an interesting milestone here at the CVE in the last few weeks: a new record for the highest number of hits on a posted video. It was Aine Seavers' clip of the rescue dog showing marked 'humping' behaviour. Like me, you probably can't wait to find out the resolution to the situation – you'll find it on page 13.

If that's not enough for you, we have an update on the Sydney Leptospirosis outbreak and some fascinating case studies from which you may just glean a pearl or two. We also pay tribute to Professor David 'The Prof' Hutchins, whose legacy lives on in so many of his students. Finally, Dr Paul Nisselle's '12 tips to reduce the chance of diagnostic errors' on page 17 is an absolute gem – worth warming up the laminator for.

Be entertained, stay informed. Happy reading.

Simone Maher

Deputy Director

29
Dec 2019

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Dr Susan Little of Canada is once again headlining another Valentine Charlton Feline Medicine Conference. If you missed her in 2016, now's your chance! She will be joining esteemed local presenters with the program yet to be finalised – watch this space!

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> ❝ *I enjoy reading the C&T more than any other veterinary publication.*
>
> Terry King, Veterinary Specialist Services, QLD

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

The C&T is *not* a peer reviewed journal. We are keen on publishing short pithy practical articles (a simple paragraph is fine) that our readers can immediately relate to and utilise. And the English and grammar does *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.

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Aine Seavers..... p13

BEST VISUALS

Caring for wildlife under threat Elliot Carr p23

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Suprelorin® implant in a dog with concurrent hyperadrenocorticism and diabetes mellitus
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The hidden insurance of epididymal sperm
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Tibia fracture repair in an alpaca cria
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Vale Associate Professor David Robert Hutchins: an influential life

Professor David Hutchins was a teacher and clinician who was a true inspiration to his students and colleagues and a byword for veterinary excellence. Meg Brownlow has written a beautiful tribute interspersed with a gallery of wonderful images which chronicles his veterinary career and legacy.

'The Prof'

They might not remember much but most of them, even those who were only interested in small animals, will remember 'The Prof'. This man stood out from the crowd. He was slight in stature but huge in the effect he had on students. He demonstrated an extraordinary diagnostic ability across species, a discerning eye to pick the most subtle of clinical signs, an agile, flexible and energetic mind which could always think outside the square, and the dogged tenacity to persevere when things did not go right. He had a wisdom, a certain philosophical approach and said on many occasions 'the mark of a man is how he copes with his failures rather than his successes'.

On behalf of the veterinary profession, we offer our sincere condolences to Assoc Prof Hutchins' family, friends and colleagues.

Mentor, astute observer, gifted clinician, heroic surgeon, innovator and leader

Text and Images Courtesy of Meg Brownlow—From All of Us

Figure 1. Prof didn't mind getting his hands dirty—it was all about the animal. Here he is examining this foal for meconium retention.

eBook download:
Read the tribute in the eBook

Authors' views are not necessarily those of the CVE

Sydney Canine Leptospirosis update

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C&T No. 5761

Background

Leptospirosis is caused by a motile aerobic spirochete bacterium of the genus *Leptospira*. In its most severe form, it causes vasculitis leading to tissue injury such as acute kidney and/or hepatic disease in dogs and importantly is a zoonotic disease. For *Leptospira* serovars affecting dogs, rodents are the usual reservoir hosts and shed leptospires in their urine.

Dogs can become infected by contact with urine directly from reservoir hosts, bite wounds from infected rodents or ingestion of infected tissues (ratter), or indirectly via contaminated water or soil[1]. After infection there is a phase of bacteraemia lasting about 10 days leading to vasculitis, organ ischemia and invasion of the kidneys and liver with resultant shedding of leptospires in the urine[2].

Leptospirosis should be suspected in dogs with non-specific clinical signs (lethargy, vomiting, diarrhoea, haemorrhages, conjunctivitis), consistent clinicopathological abnormalities (azotaemia, increased liver enzymes, glucosuria) and risk factors (contact with rodents, exposure to contaminated environmental water)[1]. Clinically healthy dogs can shed some serovars of leptospires without clinical signs, posing a potential zoonotic risk[3-5]. Similarly, cats are thought to be potential silent shedders[2].

Diagnosing leptospirosis can be difficult. Diagnosis can be achieved by PCR on blood and/or urine collected and submitted concurrently BEFORE antibiotic treatment and by serology (microscopic agglutination test, MAT) which is required to determine the serovar. However, PCR results can be negative after just one dose of antibiotics (penicillins or doxycycline). Therefore, a negative PCR result does not exclude the diagnosis in a dog that has been treated with antibiotics before taking blood and urine.

Acute and convalescent (after 7-14 days) MAT titres should be tested, where a 4-fold increase in titre for individual serovars is consistent with infection[1]. Unfortunately, convalescent titres can often not be tested if the patient dies acutely.

Prevention of leptospirosis is achieved by limiting contact to sources of infection (rodents, contaminated water) and vaccination[2]. There are no peer-reviewed publications reporting clinical cases of canine leptospirosis in Sydney and therefore, dogs in Sydney are not routinely vaccinated against leptospirosis.

In Australia, vaccines against *L. interrogans* serovars Copenhageni and Australis are available[6], although the latter is only under limited permit. In 2004, Copenhageni was the most prevalent serovar in NSW, identified in 5/10 seropositive dogs from animal shelters[6], and the vaccine currently available for vaccination in the Sydney area (Boehringer Ingelheim vaccine (Protech C2i) covers serovar Copenhageni only.

The recommendation is to give two doses 2-4 weeks apart, from 6 weeks of age. Annual revaccination is recommended. The vaccine may offer some cross-protection within the same serogroup but this remains uncertain. The vaccine is likely to be effective in protecting against clinical disease caused by serovar Copenhageni but may fail to prevent infection or organism shedding.

Most dogs with clinical leptospirosis will require intensive care and ideally should be referred to a specialist clinic. Initial treatment involves administration of IV penicillin derivatives (ampicillin, amoxicillin). This should be commenced as soon as samples have been taken for diagnostic testing, and before results have returned from the lab.

This will prevent further replication and shedding of leptospires, however, will not clear the infection. Therefore, as soon as it is possible to give oral medication,

a two-week course of doxycycline (10mg/kg SID) is recommended to ensure that the infection is cleared from the kidneys. Further treatment involves supportive care including IV fluids, antiemetics, gastroprotectants, pain medication, nutrition and potentially fresh frozen plasma if disseminated intravascular coagulation (DIC) or systemic inflammatory response syndrome (SIRS) are suspected. In anuric or oliguric patients diuretics (frusemide, mannitol) might be indicated.

Importantly, leptospirosis is a zoonosis. Therefore, suspicious cases should be isolated. Ideally, a urinary catheter should be placed to avoid contamination of the environment as well as to monitor urine volume output. It is imperative to avoid contact with urine and to wear appropriate personal protective equipment (PPE) including gloves, impermeable gowns, a face mask and glasses (Figure 3&4). Isolation is usually maintained until the dog has been treated with IV penicillin derivatives for at least 72 hours.

Sydney leptospirosis 'outbreak'

From May to August 2019 there have been 5 PCR-confirmed cases of canine leptospirosis in the Inner city (Surry Hills and Glebe). Prior to that there were three PCR-confirmed cases—two in 2018 and one in December 2017. Two dogs were known ratters. The serovar involved was identified in 2 dogs (serovar Copenhageni), yet it remains unclear if other serovars could be involved and if the current vaccine is effective.

While it is thought that the recent outbreak could be due to major construction occurring in Sydney, and therefore increased exposure to rats and contamination of subterranean water, the current source of infection involved in the outbreak is unknown.

Mortality rates of 20-48% have been described in the literature[7,8] however in the current outbreak all dogs died or were euthanised due to a poor prognosis. It is unclear why a 100% case fatality rate is seen in the current outbreak. All dogs had hepatorenal involvement. At least 3 dogs died because of acute pulmonary haemorrhage syndrome (diffuse bleeding into the lungs), one dog was euthanised because of diffuse haemorrhage including a sublingual haematoma which resulted in upper airway obstruction and one dog was euthanised because of seizures.

'Leptospirosis consensus'

On the 14th of August, practitioners convened at a 'Leptospirosis night' organised by Geoffrey Golovsky from Vet HQ, Double Bay. Invited were Small Animal Specialist Jody Braddock from Sydney Veterinary Emergency & Specialists, Small Animal Specialist and Senior Lecturer Christine Griebsch from the University of Sydney and Gavin Harper from Boehringer. After discussing what is currently known the following recommendations were made:

The geographical areas of concern are centred on Surry Hills

Conservation biologist Professor Peter Banks has suggested that rats are unlikely to travel beyond 3km (very unlikely beyond 5km) from this area and carry *Leptospira* (see figure 1).

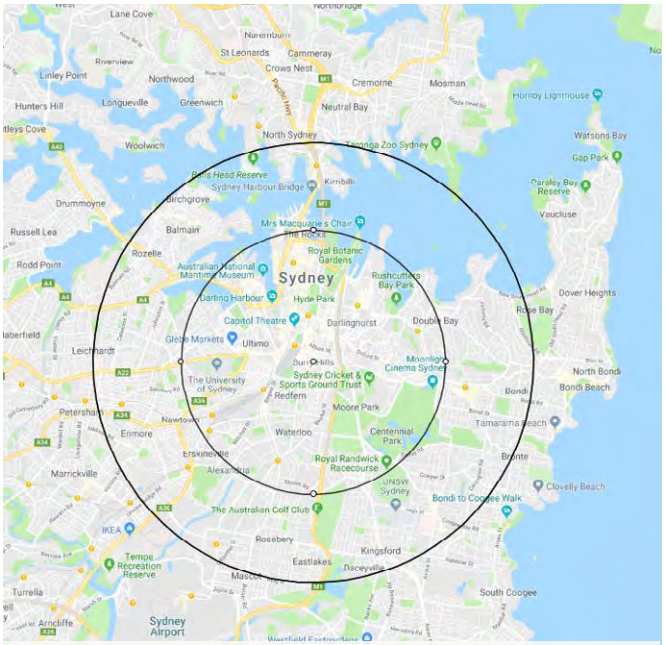

Figure 1. Map of Sydney with circles indicating 3 and 5 km radius of Surry Hills.

The advice is to vaccinate all dogs that live or visit within the 3km high risk area. We also recommend vaccinating dogs that eat rats or have significant rat contact. Please note this includes Centennial Park.

It should be remembered that infection can occur by:

- Contact of the bacteria across intact mucus membranes (ingestion), abraded (cut) skin
- Contact with infected urine or soil, water, food that has been contaminated with infected urine
- Bite wounds (infected rodents)
- Ingestion of infected tissues (ratter)

There is a zoonotic risk, therefore it is important, especially if you are immunocompromised, to vaccinate your pet.

The vaccine is given twice—with an initial injection and a booster 2 to 4 weeks later.

Current research

The University of Sydney has started a research project to look into the current outbreak. The research team involved includes Christine Griebsch, Senior Lecturer and Specialist in Small Animal Medicine (project leader), Jacqui Norris, Professor of Veterinary Microbiology & Infectious Diseases, Michael Ward, Professor of Veterinary Public Health & Food Safety, Peter Banks, Professor of Conservation

Biology, Claire Wylie, Senior Lecturer in Evidence Based Practice and Nicolle Kirkwood, Senior Registrar in Primary Care.

Aims are to:

1. Investigate the geospatial distribution of the recent outbreak of canine leptospirosis in Sydney;
2. Determine the clinicopathologic, molecular and serological characteristics of the recent outbreak of canine leptospirosis in Sydney;
3. Conduct a pilot regional seroprevalence study of Leptospira serovar exposure in healthy dogs and in-contact dogs/cats;
4. Devise evidence-based guidelines to promote awareness of canine leptospirosis in Sydney;
5. Facilitate an epidemiological investigation of risk factors for leptospirosis status in dogs in Sydney.

Information about this project has been sent to veterinarians in the wider Sydney area, however, the aim is to extend the project to veterinarians in NSW. To participate in the study, owners have to provide written consent (consent form) and veterinary clinical data regarding the geographical location of the dog, some information about the lifestyle, including which parks are frequented and exposure to rats or stagnant water, must be provided (Leptospirosis Veterinary Questionnaire). The aim is to collect blood (EDTA and serum) and urine from healthy dogs that have not been vaccinated against leptospirosis before. Samples can be stored in the fridge for up to a week or can be frozen. Ideally, the serum should be separated and transferred into a different plain tube shortly after blood collection and urine should ideally be stored in small urine tubes. Collection of samples can be organized by contacting the University of Sydney (univet@sydney.edu.au). Similarly, veterinarians who are interested in participating can request forms via this email.

Veterinarians who have a confirmed or suspicious case are asked to contact the University of Sydney immediately (ideally via phone). If possible, a postmortem examination should be performed after consent from the owner has been obtained in deceased dogs. The University of Sydney can organize transport of dead bodies to the pathology service. The postmortem exam will be free of charge for the owners.

This research project will enable us to identify the causative serovar and begin to investigate the epidemiology of this outbreak, both of which are essential for an effective preventative plan. Identifying the serovar is of utmost importance as the vaccine used to vaccinate dogs in Sydney's Inner City after the recent outbreak is covering serovar Copenhageni only. Should other serovars be involved, development of a new vaccine or import of vaccines from North America or Europe covering other serovars might be necessary. Future studies will also involve characterisation of the reservoir (environmental sampling and sampling of rats) and serological response to vaccination in dogs.

No cases of human Leptospirosis in the current outbreak

After meetings and discussion with human medicine infectious disease specialists, the NSW Public Health Unit and the NSW Department of Primary Industries, we can confirm that there have been no cases of human leptospirosis associated with the current outbreak in dogs.

Figure 2. Free vaccination event in Ward Park organized by the City of Sydney.

Vaccination events

The University of Sydney has participated in free Leptospirosis vaccination events for homeless people and pensioners organized by Pets in the Park (Darlinghurst) and the City of Sydney (Ward Park, Surry Hills). Volunteers included students, nurses and veterinarians from the University of Sydney and other clinics in Sydney (including Vets on Crown, My Vet Animal Hospital Waterloo, Asquith Veterinary Hospital) including the Head of School and Dean Frazer Allen (Figure 2).

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Useful links

https://theconversation.com/explainer-what-is-leptospirosis-and-how-can-it-harm-us-and-our-pets-120221

https://theconversation.com/curious-kids-where-did-rats-first-come-from-121307

Figure 3. Dog with acute Leptospirosis, urinary catheter in place.

Figure 4. Isolation area for a dog with acute Leptospirosis. Students wearing appropriate PPE.

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Suprelorin® implant in a dog with concurrent hyperadrenocorticism and diabetes mellitus

Steven Metcalfe, Caitlin Hargan & Jia-Wen Lim

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C&T No. 5762

Champ is an 8-year-old castrated Maltese cross who presented in March 2017 for lethargy and alopecia but without observed polyuria/polydipsia (drinking 350-400mLday). He weighed 8.8kg and had a BCS 3/5.A referral laboratory blood profile revealed a normal T4, CBC and biochemistry changes—stress/steroid-mediated leukogram and mild hepatopathy—suggestive of hyperadrenocorticism (HAC).

A low dose dexamethasone suppression test demonstrated inadequate cortisol suppression with a baseline cortisol of 188nmol/L, 4-hour 58.5nmol/L and 8-hour 146nmol/L. Ultrasonographic appearance of his adrenal glands revealed bilateral hyperplasia. The diagnosis of HAC was confirmed. Champ was started on 2mg/kg of compounded trilostane[b] once daily in food. This dose was later increased to 2mg/kg BID, resulting in small improvements in his alopecia and clinical condition. He lost some weight (8.7kg) and his water intake remained normal at 400-500mL/day.

Champ developed diabetes mellitus (DM) in October 2017. He experienced weight loss (7.7kg) and profound polyuria/polydipsia (PU/PD) (drinking >1L/day). It is well documented that elevated serum cortisol levels contribute to the development of insulin resistance. HAC therefore predisposes to the development of DM. One theory for why some dogs with HAC will go on to develop DM is due to pancreatic β-cell exhaustion secondary to compensating for insulin resistance.[1]

Champ displayed progressive, significant insulin resistance and persistent PU/PD (signs absent at initial presentation). His water intake fluctuated between 700mL and up to 1L/day. By January of 2018, he was receiving insulin doses greater than 2 IU/kg with no evidence of Somogyi effect and his weight continued to reduce to 6.3kg, which was a BCS 2/5. He was also requiring large doses of trilostane (8mg/kg BID) to control his HAC. There were no other identifiable factors that could have led to his observed insulin resistance and poorly controlled HAC. Interestingly, Champ also developed hypersexual behaviours and began to hump his owner. This made us question if he also had hyperandrogenism (not tested), in addition to the hypercortisolism despite having undergone juvenile castration.

Recent communication with Dr Michelle Kutzler, whose research explores the link between sustained elevations in luteinising hormone (LH) and the effect this has on different disease processes, led us to question the refractory nature of Champ's condition and the potential mechanisms behind it. Champ was castrated at 6 month-of-age and there is accumulating data that demonstrates that this leads to prolonged and sustained elevations in LH due to a disruption in the negative feedback mechanism. LH receptors are located throughout the body in non-gonadal tissue including the urinary bladder, thyroid, bone, lymphoid tissue and adrenal cortex.

There are reports of LH-dependant hypercortisolism due to expression and activation of functional LH hormone receptors in both women[2,3](and ferrets[4]).In humans this typically results in bilateral adrenal enlargement, however, in ferrets unilateral adrenal tumours have been reported. This process leads to LH-induced hypersecretion of glucocorticoids by the adrenal glands.

In sterilised ferrets HAC typically presents as hyperandrogenism (although there are reports of hypercortisolism) and this is a commonly reported condition. LH receptors are present within the adrenal glands and in affected sterilised ferrets the LH receptors are functional. It is believed that after sterilisation HAC arises from chronic stimulation of the adrenal glands, primarily by LH[5]. (When HAC ferrets are administered a GnRH agonist this results in an increase in plasma concentrations of adrenal androgens. Conversely, healthy ferrets do not exhibit this increase). GnRH agonists are commonly used to treat HAC ferrets as prolonged GnRH receptor stimulation results in desensitisation and a resulting reduction in LH production.

There is a significant increased risk for sterilised dogs to develop HAC [6]. With this information, we trialled the use of a GnRH agonist implant in Champ to see if we could reduce and resolve his need for medication. The Suprelorin® 6 (4.7mg Deslorelin)[c] implant was inserted in September 2018. His water intake at this time was 600mL-1L/day and his weight had increased to 7.4kg. Within one month hair re-growth was apparent as well as a reduction

in his polyuria/polydipsia to 500-550mL/day. He also lost a small amount of weight and was BCS 2.5/5 at 7.1kg. Continued clinical improvements and complete regrowth of all hair was observed despite no other changes being made to his therapy. These improvements continued until he experienced a hypoglycaemic crisis in March of 2019. He presented seizuring and had a blood glucose of 1.0mmol/L. At this point in time he weighed 6.8kg and was receiving 1.6 IU/kg insulin BID. In response to this crisis his insulin dose was reduced to 0.7 IU/kg BID and trilostane dose was reduced to 2mg/kg over the course of 8 days. He was still hyperglycaemic with a nadir of 31.9mmol/L. He was maintained on this insulin dose for 8 more days and had a nadir of 12.4mmol/L at the end of this period. The insulin dose was then increased to 0.9IU/kg BID and he has been clinically stable since then. It remains unknown whether this clinical decline was due to Somogyi effect or if the suprelorin implant had reduced his insulin requirement.

By the end of that month, in an interesting turn of events, Champ re-presented with anorexia, vomiting, and lethargy. His electrolyte results found a low Na:K ratio, and on CBC an absence of lymphopenia (and eosinophilia). This indicated he was experiencing a mineralocorticoid crisis. At this point in time Champ weighed 6.6kg and his water intake was 350-400mL/day. At the time of writing he has stopped all trilostane medication and is receiving 5mg of cortate[d] once daily and has returned to his previously stable clinical state.

Time will tell whether Champ will revert from being Addisonian to again relapsing back to HAC and he requires ongoing monitoring and blood work. However, his dramatic apparent clinical improvement that correlated with the insertion of the GnRH agonist, raises many questions and provides evidence that LH-dependant hypercortisolism due to expression and activation of functional LH hormone receptors may exist in dogs. Further investigation may find the use of GnRH agonists as useful adjuncts in the treatment of HAC in sterilised dogs.

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a. *Stenlake Compounding, Level 1, 76 Spring Street, Bondi Junction NSW 2022*

a. *Virbac Australia 361 Horsley Road, Milperra NSW 2214*

a. *Aspen Pharmacare Australia Pty Ltd, 1st Floor, 34-36 Chandos St, St Leonards NSW*

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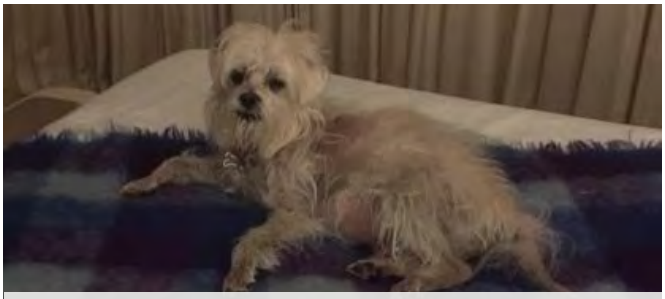

Figure 1. Champ prior to Suprelorin implant.

Figure 2. Champ one month after Suprelorin implant.

Figure 3. Champ three months after Suprelorin implant.

eBook download:
Read C&T No. 5083 'Ferrets' about a ferret treated for hyperadrenocorticism with a suprelorin implant by Kim Kendall.

Authors' views are not necessarily those of the CVE

Comment courtesy of Peter Howe

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This is a great case study presented by Dr Metcalfe. He raises the important topic of the aetiology and alternative treatment for hyperadrenocorticism (HyperA).

I do not purport to be in any way an expert on hyperA, but I have developed a great interest in this entity over the past 20 years, and more especially since I studied the endocrinology of menopause and its clinical manifestations in humans. The latter mimics in many ways hyper A in our canine patients.

The very high incidence of hyper A that we see today would initiate large scale enquiries if it were seen in other species! It does of course occur in other species, such as the horse, but at lower incidences. Given that neutered dogs are over-represented in the cases seen, at least in our clinics, I have long held the opinion that neutering is associated with hyperadrenocorticoidism.

All our neutered animals are hypergonadotrophic because of their agonadism. They have excessively high LH levels and as Dr Metcalf points out there are LH receptors on the adrenal glands. They are in the bladder sphincter and elsewhere as well. That there is a cause and effect relationship between high serum LH levels and the hypercortisolemia of hyper A is, I believe, supposition, but a reasonable supposition I would suggest. Steroid synthesis is pushed from synthesis of a balance of aldosterone, dihydrotestosterone, oestrone and oestradiol steroids and skewed towards 17-OH-progesterone and cortisol.

Luteinizing Hormone (LH) receptors have been reported to be present in the adrenal cortices of humans and other mammalian species, as well as in the form of ectopic LH receptors in tumours of non-adrenocorticotropin (ACTH)-dependent Cushing's syndrome patients. They have also been reported in human adrenal glands of pregnancy associated ACTH-independent Cushing's syndrome. In the case of the latter, the receptors are probably stimulated by HCG (Human Chorionic Gonadotrophin).

Thecal metaplasia of the adrenal gland with elevated gonadotrophin levels is well recognized by human endocrinologists. The burning question is whether the LH responsiveness of the adrenal glands occurs only with neoplastic processes or does it also occur as a normal process post-menopause in humans or post-gonadectomy in animals. We might also ask does an elevated LH level stimulate adrenal steroid production in conditions such as polycystic ovary syndrome in humans or in the hypergonadotrophic agonadism state following neutering in animals?

ACTH is the major stimulator of adrenal steroid production, cortisol and dehydroepiandrosterone (DHEA). It has been demonstrated that production of both of these latter steroids can be stimulated by the highly elevated LH levels in post-menopausal women in their chronic hypergonadotrophic hypogonadic state where cortisol, LH, E2, Sex Hormone Binding Globulin (SHBG), serum insulin, testosterone and dehydroepiandrosterone (DHEA) levels were assayed.

We too have had good responses to GnRH antagonists in selected canine HyperA cases. In all cases the owners have been resistant to daily dosing with trilostane and regular ACTH-stim monitoring and the associated costs.

Interestingly the associated hepatopathy in all cases, showed marked improvement as measured by a decline in liver enzyme levels, whereas in our hands with trilostane and mitotane we do not invariably see this.

GnRH agonists reduce LH levels by a desensitising block but there are other means of achieving the same aims. Hormone replacement therapy, oestriol and stilboestrol for example, in cases of urinary incontinence, reduces LH levels.

We also use melatonin. The exact way in which it suppresses LH levels is not fully understood (and in some species it does the reverse). Newer formulations of ultra-long acting melatonin currently in development promise to give us further tools to control the hypothalamic-pituitary axis. **We have had several cases of canine hyperA managed by melatonin therapy with quite good results.**

Hyper A is certainly frequently encountered in small animal practice but perhaps we do not always recognise it. Is a decline in collagen quality and resultant anterior cruciate rupture in our medium to large breeds associated with hypergonadotropism? And what of bone neoplasms and mast cell tumours in our larger breeds? Perhaps ultimately we might also link increased risk of dental disease and some skin conditions to hyperA. I have long considered Hyper A to be a reproductive endocrinology problem.

Dr Metcalfe's well-presented case study exemplifies our notions on the subject and I believe he is to be congratulated for bringing it to our attention and hopefully raising some debate on the subject.

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Fear of car travel in dogs

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Situational fear—fear of car travel

Travel anxiety or fear associated with travel is a poorly defined disorder in dogs not least due to the confounding factors of excitement, territorial behaviour and motion sickness causing signs similar to fear[1, 2]. The fear of travel may be a primary behavioural disorder relating to the fear of the car itself or the experience of travel (noise, balance, space reduction etc.), or it may be a classically conditioned fear due to the unpleasant experience of motion sickness nausea or a fear of the destination such as the veterinary clinic[2].

Emotional conflict may occur because the dog is excited about the destination but fears the act of travel itself. This conflict then results in a heightened state of arousal and anxiety[2]. Fear may be due to a previously bad experience or lack of experience (habituation) during the sensitive period of development[1, 2]. Assessing the potential relevance of all these factors in each individual case is necessary in order to determine how to treat the problem. Behavioural signs associated with travel anxiety are vocalisations, restlessness, hiding, licking, panting, trembling, salivation and vomiting[1,2].

Motion sickness results from a conflict of information received from the visual and vestibular sensory systems[2]. The body can learn to tolerate this and so animals may 'grow out of it' although given the potentially learned fear component, it is important not to rely on this alone and additional treatment modalities are recommended[2].

The nausea and vomiting element of the experience can be treated pre-emptively, with a potent anti-emetic such as maropitant (8mg/kg PO) [3](NB. This is much higher than the usual dose of 1mg/kg used for acute vomiting) and its non-sedating effects are advantageous if the dog is to participate in an activity at the destination where sedation is not desirable. Antihistamines can have a sedative and anti-emetic effect on dogs due to their central anti-cholinergic action. Its anticholinergic activity may enhance urine and faecal retention if soiling of the car is a concern. Diphenhydramine hydrochloride is a frequently used medication (2-4mg/kg PO)[4] for this purpose. Pheromones could also be used in conjunction with other medications, management and behavioural modification.

Uncoupling classically conditioned stimuli such as not just travelling to a veterinary clinic or groomer prevents fearful associations to be made with the travel[2]. Counterconditioning using familiar toys and blankets as well as treats during travel can be utilized though care must be made not to reward the fearful emotion[2]. Providing adequate restraint and placing the dog on the floor of the car can also reduce the sense of lack of control of the situation[2]. It is extremely important that a desensitisation program be instituted to habituate the dog to the presence of the car and being in the car both while stationary and when moving.

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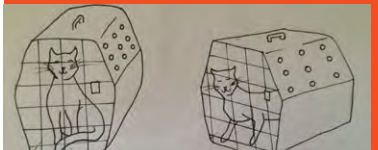

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For example C&T No. 5600 Cat carriers & visiting the vet: Turn cat transport into a positive experience from Aine Seavers & Dani Chilcott (Issue 286 - Mar 2017).

Answer to C&T No. 5746 'If you could only do one intervention/test—what would that be?'

Aine Seavers

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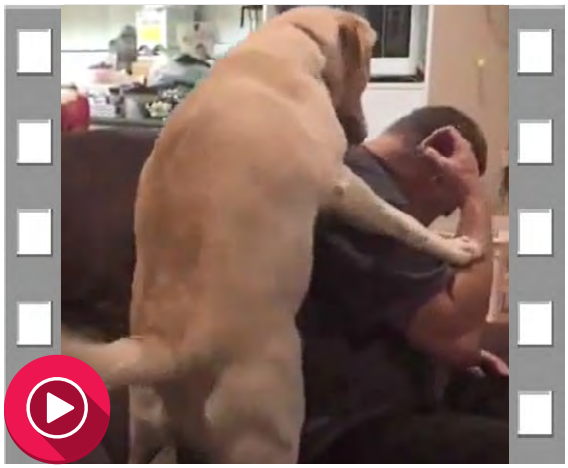

C&T No. 5764

Answer: Empty Anal Sacs!

History

The patient is a female Labrador, purchased at 8-weeks-old and basically locked in a run set down the side of a house.

She received no intellectual stimulation, no human interaction other than food and water put in the run and hence developed zero communication skills. She was subsequently rescued by my clients at around 11 months-of-age. The dog lacked features of neoteny usually associated with the Labrador retriever breed. Anecdotally, I have found that box-headed phenotype to be an early warning of behaviour issues and of an aggressive temperament.

Her new owners have been amazing; they have worked so hard and have achieved miracles with this dog.

Medications

Clomipramine (highest dose as prescribed by Elsewhere Vet: a dose to which she has responded well and been left on).

She had an adverse reaction to Trazadone and was either 'stoned' or 'manic' on it, so I had that drug stopped. This allowed her to settle down and to begin to learn appropriate ways to interact with her humans.

Complaint

After three months in her new home, she was doing well, then suddenly began to madly hump the male owner 24/7; to such an extent that neither the dog nor the owners had slept for 3 days.

The advice from Elsewhere Vet was: add yet another behavioural medication and instigate a series of new training steps for the dog.

I asked the owner to hold off everything until I did one intervention with the dog. I also asked for a home video of the issue as well.

Background

For 'behaviour concerns', pets our clinic has different formats for consultations: what I call our 'Pace and Space' approach.

This approach *is not about all the things we can do, but about doing only what we should or really need to do.*

These consults are ultra-short and occur outside our normal consultation hours, so that the clinic is less busy. This dog has been trained to a 'Hug-Hold-Release' interaction from her owner at home as practice for when I need to handle her.

In the clinic, I decide what I need to do from a distance. Owners do the loose Hug. Then I give the Hold command. The owners hold the dog close and feed as I quickly handle the dog. I give the Release command and the dog gets a treat simultaneously.

Treatment

The dog came in: fed biscuit while on the weighing scales, up on consult table, fed another biscuit, Hugged, Held, I emptied two HUGE hot anal sacs, biscuit given and then we sent her back out to the car ASAP.

The dog went home. Her owner reported that the dog slept for 14 hours, woke up, had some food, went back to bed and slept until the next morning.
Over a year later and she has not done it again. For the price of consultation, and with no drugs, the problem was fixed.

Dogs are more than the sum of their primary condition.

A behaviour concern dog, who suddenly exhibits a new odd behaviour, is not necessarily behaving badly, but rather may be signalling a new medical issue.

ANAL SACS

Anal sacs, from a canine medicine point of view, are an amazing and fascinating part of the body that just oozes clinical information to help treat so many conditions.

I have had dogs with behavioural disorders, 'startle' behaviour as if someone booted them in the rear end, alleged 'seizures', lameness and paralysis signs, aggression to people, manic mounting of another dog and food allergy dermatology patterns for which the cause and/or the evidence lay in the anal sacs.

With any humping, you have to empty anal sacs first.

Otherwise, missing it leaves the client unimpressed when they seek a second opinion from another vet who fixes the issue in 3 minutes flat. I have had that happen to me as the second vet so often that it is stressful for me, as I know it will cause the client to be irate with first vet which is something I hate to have happen.

If I can make all vets do this first for all humping dogs, then everything is covered, no harm done and either fixed or ruled out.

Why do anal sacs make dogs hump?

I don't have the definitive answer. I think the anal sac contents or a particular bacterium must have an oestrogen-like smell.

30% of entire bitches in one study had anal sacculitis issues 1-3 weeks post oestrus which is interesting in itself. (If the bitch is entire and there is a temporal relationship to issues post-oestrus, I take it as another sign to desex this dog).

- When another dog, desexed or otherwise suddenly begins to hump its canine mate regardless of the gender of either dog, I have the humped dog as well as the humper dog brought in. Often the problem trigger is the anal sacs of the humped dog.
- If the dog humps items, whilst I do initially empty their anal sacs, most times it is not the sacs that are at fault. Dachshunds, in particular, like to hump anything shorter than themselves be it a shoe, another dachshund, a bed edge or a toy. I leave them to it.
- If the dog humps a human, in addition to doing the dog's sacs, I have the humped human have their doctor do a full check-up of blood pressure, glucose etc.

Dogs live in a world of scent and are used to flag hypoglycaemia, cancer or epilepsy issues in humans. The owner above was checked out and had no issues.

Another male owner was checked on my recommendation when his Basenji developed odd issues suddenly, and only with the male owner. The client turned out to have the earliest ever detected start of a haematological neoplasia that normally requires strong chemotherapy and has poor survival. Because we discovered it so super early, a tolerable course of chemo has given the owner 15 years remission.

So: Never ignore when 'sane' dogs suddenly exhibit 'manic' behaviour

- Every single itchy dog, from foot chewer to ear rubber, gets their anal sacs emptied and assessed by me. The additional behaviour of: nibble then 0-15 sec hold in a fake bite motion rather than a mainly licking -chewing pattern, raises the index of suspicion that we should be looking at anal sacs.

A series of papers by Clive Halnan in *Journal of Small Animal Practice* from many years ago linked experimental simulation of blocking the anal sac ducts with dermatological symptoms which subsequently resolved when the simulated impaction was resolved.

A previous paper by Halnan, in same journal, linked blocked anal sacs ducts in dogs with symptoms in ears, facial skin and feet, which then resolved when the anal sac impaction resolved. Some of those cases in the first paper, we would now classify as having atopy, but a portion would stand alone in an impaction-pruritus pairing. Halnan's second (direct research based) paper, wherein they ligated ducts and/or inoculated with culprit bacteria, produced results that supported the clinical observations in the previous paper.

- If the clinical dermatology signs are arising from food intolerance/allergy, you will often see issues in the ear/s but also in the anal sacs.

I love the situation where clients intensely wonder why a dog scoots regularly for no reason (i.e. no impaction, no tapeworms, no vulvo vaginitis- no hessian beds) thus inferring that you the vet are failing in your job. This is where anal sac expression is your friend.

You can get owners to confess to dietary indiscretions when you gaze knowingly at the anal sac secretion on your glove, then look up at the owners and ask: "Ok: what Else have you been feeding, besides what you told me?" 'I am seeing way more than the foods you have listed so far

showing up in these anal sacs…. someone needs to fess up now.'

The client's guilt- usually male owners- becomes so great that they can't cope and blurt out that they like to feed the dog the remains of their home-made sausage madras curry... or other bad foods choices to that effect!

In the curry case I asked the owners how THEIR rear ends felt after said curry? They agreed that *region of their own was a tad more inflamed than when not eating said curry.* Yet the dog is somehow supposed to have an iron anus with inbuilt cooling system.

Odds & Ends

I have a few other canine references I have used over the years, where you scale from 1-5 based on consistency, colour, texture and smell.

Another interesting link is that the microorganisms in the mouth determine the microorganisms in the anal sacs. I can't cite the paper, around 2006, but it is surprising how often a foetid mouth when cleaned, will decrease the frequency of hind leg and toe/foot chewing.

Tips

- I have 'at risk' breeds (spaniels as part of their immune disorders, Basenji, German Shepherds on wrong diet, smaller 'teeth-on feet' dogs who need regular emptying of sacs) who really benefit from having their owners 'heat-pack' the dog's anal area 2-3 days before a visit.
- This makes many sacs so much easier to empty, which makes it less painful for the dog to have done.
- I use a ton of lubrication to do it and if there is any hair there, I will shave a narrow area off first as catching the hairs with the clippers as you go in makes them arc up.
- I never let owners hold them for this; the owner's body language is all wrong and tense and so the dogs panic; nurses always hold the pet, then the dog can escape back to mum afterwards.
- The ventral skin of the tail can be really sore from scooting so it is good to check first that the tail skin is not raw before you go move the tail away to get clearance to the anus.
- If I have to shave a lot just because the dog is hairy or because the area is a mess, I do warn the owner in advance that the shaved dogs will behave oddly when we put them back down on the floor, that the dog is not hurt but rather is disconcerted by getting 'air on their rear' now that the matts are removed. The odd 'bum-down, bum-up' act stops in a few minutes.
- I used to use the Vet Malacetic wipes to clean down the anal area afterwards but they are no longer available. Whilst I hate using human topicals on dogs, I have found none react to a once-off wipe down in-clinic with a *'Wet-Ones Be gentle Hypoallergenic' wipe from Coles*. The wipes really do clean the dog down fast; and deodorise your room and your clinical waste bin as an added bonus.

Diet

In Halnan's paper, over 50% of the dogs had no biscuit in their diet, just all meat which produced a strap like faeces not conducive to emptying anal sacs. I fix my anal sac patients by addressing their diet, reducing the allergen load and upping the fibre content of the diet.

Questions

1. Has anyone used the new in 2019 Apex generic Panalog in anal sacs?
2. Has anyone used Osurnia in the sac?
3. Canaural?
4. How do vets find Glanex?
   https://www.sacex.com/
5. Anal Adenocarcinoma/SCS:
   Over 30+ years, I rarely ever see one; although I am always alert for it.

   From my first year of practice I have always checked/emptied anal sacs on almost all dog patients most visits (within reason i.e. not if the dog at clinic weekly for something or monthly).
   I have done so in different countries around the world, so I am not staying within genetic or familial lines re disease risk etc.

   By removing an inflammatory infected focus/trigger very early on in the piece, am I inadvertently having a preventative effect on the expression of a neoplasia?
6. ANAL SAC CALCULI 2019: I have JUST had my first-ever case of what could only be described as Anal Sac 'Calculi: solid discrete creamy 'struvite/crystals' mixed into a not too odorous pale-yellow semi-clear liquid. I have never seen this 'crystal' mixture come out of an anal sac ever…

   I have seen hard black impacted grit as you start, but not a tumbling cascade and not this light color and irregular shapes with minimal odour. This crystal-calculi scenario has never been reported before. I had never seen it before nor have any vets I have asked. The only unique aspect of the case was this dog was on Apoquel (not by me) and this was the first anal sacs I have emptied in a patient on Apoquel. Watch this space….

In Summary

Anal sac issues are one of the great impersonators of both behaviour problems and dermatological conditions: we need to be addressing those before any more drugs and interventions are loaded onto the dog, and at more cost to the owner.

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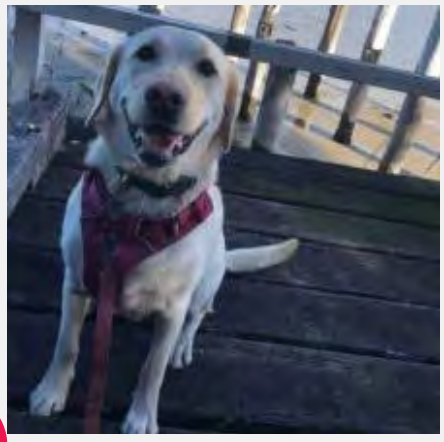
Editor's note

Thank you very much to all our members who took the time to write a reply to Aine's question.

Whilst no-one suggested emptying the anal sacs, we had many thoughtful replies and Aine has chosen James Harris as the winner of the closest answer and he is entitled to a free DVD from the CVE video library.

Luna the Labrador in the video is a reformed character and is leading a wonderful life with her dedicated owners Follow the link below to see her daily routine.

oakflatsvet.com.au/a-day-in-the-life-of-a-rescued-adult-dog/



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12 tips to reduce the chance of diagnostic errors

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C&T No. 5765

'More is missed by not looking than not knowing'

12 tips to reduce the chance of diagnostic errors:

1. Don't jump to conclusions based on what's common or what you've recently experienced, or because you don't have time to think it through.
2. Avoid confirmation bias—keep an open mind. Don't explain away symptoms or signs that don't fit your presumptive diagnosis.
3. Use diagnostic timeouts – think again, with fresh eyes, before closing down the diagnostic process.
4. Think of the worst case. Is there anything else serious that could be going on?
5. Be aware of your reaction to the patient, and it's owner.
6. Be systematic in history taking and examination, and don't cut corners.
7. Don't skip the physical examination because you think the x-ray or echo or blood test will make the diagnosis for you.
8. Ask yourself why an event happened. For example, the patient has an acute attack of asthma which needs treatment, but what brought it on?
9. Apply Bayesian theory. Read the article at kevinboone.net/bayes.html or spend 10 minutes watching 'Bayes Theorem—Explained Like You're Five'. Warning: Could make your brain hurt!
10. Learn about Occam's razor. The common adage, 'If you hear hoof beats, think horses, not zebras' is usually—but not invariably—correct, because zebras exist. In medicine, Occam's razor translates as diagnostic parsimony. If the patient's presentation can be explained by the existence of one rare condition or two co-existent common conditions, then you probably have one zebra, not two or three horses. Now, the contrary view is expressed in Hickam's Dictum, where patients can have as many diseases as they please. Hickham could be right, so at least test for a zebra, just in case.
11. If you're learning, slow down. You're under pressure to work quickly. You should feel greater pressure to work safely.
12. Admit your mistakes, learn from them, then move on.

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Rectal exams: a simple screening test for a silent killer—canine apocrine gland anal sac adenocarcinoma

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Why write about rectal exams?

When my sister-in-law's dog, Sandy, an 8-year-old male neutered Labradoodle was diagnosed with end-stage metastatic apocrine gland anal sac adenocarcinoma (AGASAC) and sadly, had to be euthanased, I decided to look at this cancer in more detail.

Early detection through regular rectal exams in healthy patients

The rate of dogs diagnosed incidentally with AGASACs is >50% and the smaller the size of the tumour, the better the prognosis.

A routine rectal exam as part of a clinician's exam is a simple, cheap and effective screening test for this cancer and while the majority of general practitioners are not doing it routinely at every annual health check, perhaps they should.

Prognosis is significantly better for dogs with smaller AGASACs that have not metastasised, so early detection through regular rectal exams allows earlier treatment and better overall outcomes for our patients.

Aside from assessing the anal sacs properly, a rectal exam also allows assessment of the prostate gland (males), urethra (I have detected a urethrolith this way in a female dog), medial iliac lymph nodes, pelvic bone structure, anal tone, rectal mucosa and stool consistency. What other 20 second test can assess so many things with no equipment other than a gloved finger and some lube?

If the anal sacs are full then it is preferable to express the anal glands in order to thoroughly palpate the glands. The owners often want this done anyway, particularly if their dog is displaying scooting behaviour or a bad smell. I would recommend expressing the anal sacs in the treatment room with a nurse restraining the patient, and not the consultation room due to the unpleasant lingering smell it produces. I've worked for one vet clinic that had a rubbish bag in the freezer for rancid excretions to prevent the vet hospital from smelling bad –highly effective!

Interestingly, as one oncologist pointed out, many AGASACs are initially identified by the groomer who often perform anal sac expression as part of their grooming service. If the groomer can identify these tumours early, then so should we.

The only way to detect an AGASAC early enough to allow a surgical cure is if you go looking for it.

When should I do a rectal exam?

Rectal exams may be unpleasant (for vet, owner and dog) and as clinicians, we have a duty of care to perform a thorough health check. When the reasoning behind why we should perform a rectal exam is discussed with the owner, they are usually happy to do this routinely.

It's important that health check standards are discussed with vet team members so that there is a consistent message being delivered to dog owners. This can be achieved with a check list or health report that includes rectal exam.

One oncologist quoted a mysterious 'wise person' who once said "There are only two reasons not to do a rectal exam 1) you have no finger or 2) the animal has no anus."

Making rectal exams more pleasant for the dog

Most dogs that will tolerate their rectal temperature being taken will tolerate a rectal exam fairly readily.

Having a nurse or a family member place one hand under the dog's tummy to prevent them sitting down during a rectal exam is really helpful as if there is movement during the exam, then it can be painful. A consult support nurse can also be utilised here.

Options to distract the dog:

- Lots of pats and attention/reassurance from the owner
- Lick mats
- High value treats

Things to remember:

- Keep finger nails short
- Lots of water soluble lubricant eg K-Y Jelly
- Avoid pulling the dog's tail up too high as this can be uncomfortable

The Fear Free program has a module on co-operative care has further tips on pet participation during examination at www.fearfreepets.com

Fearful patients

There are some occasions where it is simply not appropriate for the vet to perform a rectal exam:

- Puppies
  - Avoid rectal exams unless absolutely necessary during the fear period (8-10 weeks)
  - During the fear period, puppies show stronger fear reactions and retain their fear of an object or person, which could make future vet visits unpleasant
  - Consider palpating the anal glands externally in puppies, unless scooting behaviour seen
- Fearful dogs
  - Rectal exams are important but if it is going to be dangerous for the vet or detrimental to the mental health of the dog, then perhaps reconsider
  - Ensure approach is slow, gentle and with predictability rather than surprising the patient
  - Psychotropic medication such as gabapentin, clonidine or trazodone prior to vet visits may enable this without impacting the mental health of the patient
  - Sedation may be required if a patient cannot be examined while conscious as vaccination should not be carried out without a meaningful physical exam
  - Use opportunities where fearful patients come in for a procedure such as dentistry or radiography to perform a rectal exam
  - Consider waiting until the dog is 5 years old before routine rectal exams are performed, keeping in mind that while AGASACs are rare in dogs < 5 years old, it is still possible so should be on your radar
  - EMLA cream can be effective as a local analgesic but requires 20 mins to be effective, so the cream would need to be applied right at the start of the consultation in order to work

Document it

Record your rectal exam findings in the patient's record including descriptions such as 'anal sacs easily expressed, NAD per rectum' so that a baseline normal is determined for that patient to compare to.

Recheck it

For any case where there is a deviation from that patient's normal but you are uncertain about whether there is a mass present, have a colleague palpate the glands too for a second opinion. If there is any doubt, repeat the rectal exam in 2 weeks.

What about cats?

Most oncologists agree that a rectal exam at a routine health check in a conscious cat is not required (nor safe) as AGASACs in cats are rare (0.5% of all feline skin neoplasms). There is no harm, however, in using the opportunity of an anaesthetic for another procedure to do a quick rectal check in cats too. If the cat is displaying clinical signs suggestive of AGASAC then sedation to evaluate the rectum and anal sacs is important.

Facts about canine apocrine gland anal sac adenocarcinomas (AGASACs)

Clinical signs

- There may be no clinical signs until late in the course of disease
- Perianal swelling
- Excessive scooting and licking are less commonly seen
- Mass detectable per rectum
- Metastases to local lymph nodes (eg medial iliac, hypogastric, sacral) can cause further clinical signs including:
  - Tenesmus
  - Constipation
  - Flattened stools
  - Urethral obstruction
  - Hind limb lameness
  - Hind limb oedema

- PU/PD, anorexia, vomiting, lethargy, hind limb paresis (secondary to hypercalcaemia)

Paraneoplastic hypercalcaemia is present in up to 90% of affected dogs with both total and ionised calcium being elevated due to the production of parathyroid hormone-related protein (PTHrP) by the cancer cells.

Diagnostics

- Biochemistry screen may reveal hypercalcaemia, hypophosphataemia, azotaemia (secondary to hypercalcaemia-induced renal injury)
- Cytology via fine needle aspiration readily differentiates AGASAC from non-neoplastic anal sac diseases such as anal gland sacculitis or abscess
- Abdominal radiography may reveal sublumbar (medial iliac) lymphadenomegaly as a caudodorsal soft tissue opacity causing ventral deviation of the colon
- Thoracic radiographs may reveal pulmonary metastases in 2-13% of cases at time of diagnosis
- Abdominal ultrasonography is more sensitive in detection of suspected lymph node metastases which occurs in approximately 50-70% of dogs with AGASAC at diagnosis (particularly medial iliac lymph nodes)
- Other less common metastatic sites include spleen, bone, pancreas, heart and mediastinum
- CT of the thoracic and abdominal cavities has advantages over radiography and ultrasonography but cost and availability can be a factor

Signalment

- Mean age at diagnosis is 10.8 years
- Breeds predisposed include English cocker spaniel, English springer spaniel, German shepherd dog, dachshund, Alaskan malamute and golden retriever

Clinical staging

Clinical staging is essential given that up to 80% of cases have evidence of gross metastases at the time of presentation. This includes assessment of local tumour size and determining if metastatic disease is present and should ideally include either thoracic radiography (3 views) and abdominal ultrasonography or thoracic and abdominal CT scans if available. Enlarged lymph nodes should be aspirated and assessed for tumour cells if accessible or removed at time of surgery and sent for histopathology.

Hypercalcaemia

For severe paraneoplastic hypercalcaemia (uncommon), medical management may be required prior to surgery including:

- Prednisolone therapy (0.5-1mg/kg/day)
- IV fluids eg 0.9% NaCl to reverse the hypercalcaemia-induced ECF volume contraction
- Frusemide to increase renal calcium excretion following fluid therapy

Surgery

Surgery is the initial treatment of choice for dogs with non-metastatic AGASAC and when there is metastasis limited to the regional lymph nodes. The recommended technique is closed anal sacculectomy to reduce risk of local tumour recurrence.

Metastatic medial iliac lymph node resection is indicated when there is hypercalcaemia and/or tenesmus. The parathyroid hormone producing cells causing hypercalcaemia are present at all tumour sites so affected lymph nodes need to be removed to achieve normocalcaemia.

Radiation therapy

Radiation therapy can be considered for inoperable cases of AGASAC and affected medial iliac lymph nodes. There is a risk of side effects to important structures in the vicinity including colon and bladder. Palliative radiation therapy results in a statistically better median survival time.

Chemotherapy

There is no chemotherapy protocol developed so far that is better than surgery alone. Dogs with localised, non-metastatic AGASAC do not require adjunctive chemotherapy, particularly if tumour size is < 2.5cm diameter. It is recommended for cases that have metastasised to the medial iliac lymph nodes or for

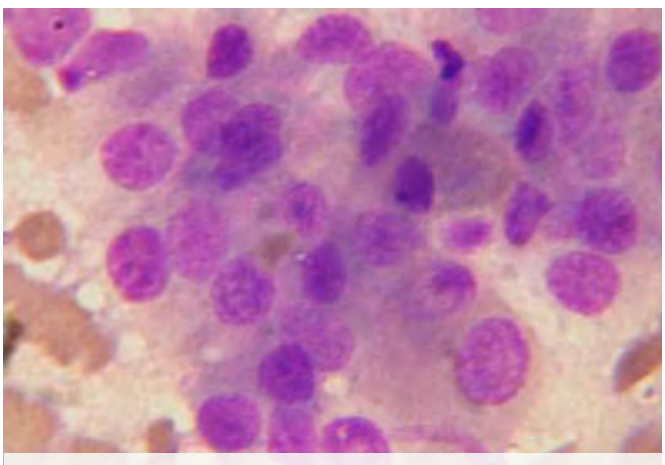

Figure 1. Cytology from an AGASAC. Picture courtesy Jennifer Blair.

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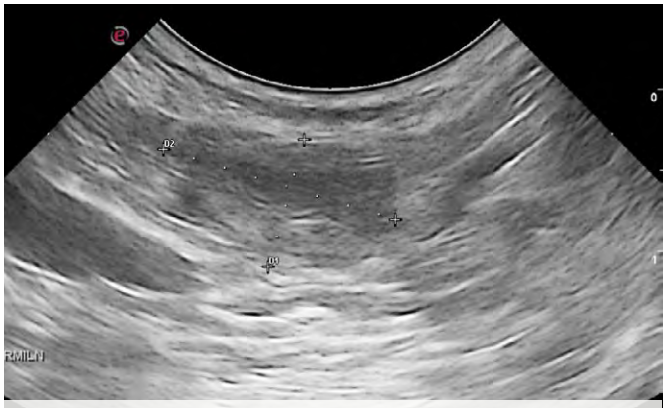

Figure 2. Picture courtesy of Dr David Collins, Northside Veterinary Specialists. Ultrasound image of enlarged medial iliac lymph node from a dog diagnosed with AGASAC.

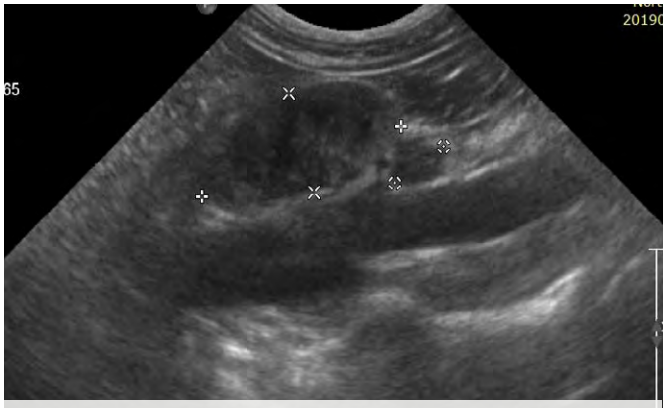

Figure 3. Picture courtesy of Dr Sarah Davies, Veterinary Imaging Associates. Ultrasound image of enlarged medial iliac lymph node from a dog diagnosed with AGASAC.

palliative treatment in unresectable tumours and/or distant metastatic disease. Chemotherapeutic drugs shown to have some activity against AGASAC include cisplatin, carboplatin, actinomycin-D and toceranib.

Prognosis

Negative prognostic factors include large tumour size (> 10cm), hypercalcaemia, distant metastases eg lungs and not proceeding with surgical excision of primary tumour and affected lymph nodes.

The presence of medial iliac lymph node metastasis is associated with a worse prognosis if the lymph nodes are left intact. The outcome markedly improves if the metastatic lymph nodes are excised.

For dogs with non-metastatic AGASAC the median survival time was 1612 days for surgery alone. The recurrence rate is approximately 20% after anal sacculectomy.

The association between small tumour size and a favourable prognosis if surgery is undertaken in this early stage, highlights the importance of early detection through routine rectal exams for all dogs and has been my main drive for writing this article.

In Summary

I did a search of all confirmed cases of AGASACs at Turramurra Vet Hospital and we have had 5 dogs diagnosed since the beginning of 2015.

I've certainly placed a lot more emphasis on the importance of a complete physical examination, which includes a rectal exam, even when there are no associated signs to suggest a problem. The fact remains that when detected early, this disease is potentially curable and those dogs can go on to live a normal life span. This compares tragically to the cases presenting in the advanced stages of the disease where the chances of a good outcome are greatly reduced.

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Caring for wildlife under threat

Elliot Carr

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C&T No. 5767

Cikananga Wildlife Centre is Indonesia's largest conservation breeding, and multi-species rescue and transit centre for wildlife threatened by the illegal wildlife trade. The illegal wildlife trade is continuing to grow across the world, and centres like ours provide a key function in assisting governments to deal with the seemingly endless animals confiscated and rescued from the trade.

Everyone needs to be multi-functional to care for 71 species

Currently we are caring for over 500 individuals of 71 species—and as you can imagine it is a huge mission to meet not only the nutritional, housing and behavioural requirements of so many different and unique species let alone manage the diversity of medical issues. Our small clinic currently has only two full-time vets (figure1); who are assisted by our other staff and interns—no one at Cikananga is ever relegated to one role—everyone needs to be multi-functional! Luckily, we have the support of some world class facilities such as Chester Zoo, Wildlife Reserves Singapore and the Wanicare Foundation who are on hand to provide financial and technical support.

We operate 24/7 with diagnostic equipment shortfalls

Our rescue team, local government departments and partner NGOs confiscate animals from across the region. Sadly, emergency medical treatments are often the area where we lose the most battles. This is primarily due to having minimal diagnostic tools on-site. For diagnostics we rely heavily on external sources for example, the closest X-ray machine is located in a human hospital about two hours drive away and blood tests often take weeks to return results. Our vets do a phenomenal job despite overwhelming obstacles. Confronted with these challenges (we never say problems around here!), they work tirelessly to deliver the best care and treatment. Animals arrive at our centre after travelling huge distances with illegal traders (Indonesia is made up of over 17,000 islands stretching the width of Australia) to trade hubs such as Jakarta. During this time they have endured huge stressors of capture, and transport. They arrive to us malnourished, dehydrated and often with horrendous wounds, and other issues which we cannot see or diagnose quickly. Whilst we save many, the numbers lost during, or immediately after transport are great. The illegal wildlife trade is horrible and needlessly cruel, and this is without even considering the irreversible ecological damage it causes to natural environments. Just last month we lost an endangered Javan Langur, also known as a leaf monkey. This individual was stable for a few days, but without serology and rapid response diagnostic equipment, we lost her suddenly, only finding out after her death the horrendous internal injuries and bleeding she had sustained. We are constantly looking for sponsors to help us address diagnostic equipment shortfalls.

Figure 1. Wahyu Hananto (Head vet), Inge Tielen (Conservation Manager) Cikananga Team Member Yvette Anderson.

Figure 2. Loris whose teeth had been cut with nail clippers.

The need for critical emergency dental care

Slow Lorises—the 'adorable' YouTube sensations—are heavily traded throughout Asia, and even abroad to Europe and the States where they are destined to become improperly cared for pets. Interestingly, lorises have specialised glands near to their elbow which they lick when threatened and, when the toxic protein is activated with their saliva, it produces a powerful toxin that can cause intense pain, and even anaphylactic shock in some people. Many traders incorrectly think this toxin is released from their teeth like snakes, this misconception means many lorises have their canines cut out with nail clippers (figure 2) to make them 'less harmful' and 'safer' pets to the ignorant. As you can imagine this causes immense pain, and irreversible damage to the lorises. Traded in the thousands, many never even make it to a point of sale. On arrival, our veterinary team undertake critical emergency dental care, but with the severity of the injuries caused by traders, and limited equipment, this means in most cases complete dental extraction is undertaken (figure 3), meaning these animals cannot be released back into the wild.

Figure 3. Loris dental extraction.

It is hard not to anthropomorphise

In 2016 we received two orphaned Sumatran Orangutans. We feared the first, Rosi, would die from malnutrition and exhaustion. For two days the team worried as Rosi slept continuously, only reluctantly being woken up for milk. Femi was discovered literally inside a parcel being sent via mail on a public bus coming from Sumatra. It was touch and go for at least a month. Both were horribly traumatised. It is hard not to anthropomorphise when you look into the eyes of an ape, let alone an infant so young. Their mothers, refugees from forests that have been slashed and burnt for the insatiable human demand for unsustainable palm oil, wander into plantations and villages looking for food to keep producing milk for their young babies, only to be slaughtered, and their children sold into the pet trade.

The forests are silent

In traditional Javan culture, to be considered a 'man' it is said you need a house, a wife, a horse, a weapon, and surprisingly, a caged song bird. This tradition is still alive,

Figure 4. Javan Warty Pig.

Authors' views are not necessarily those of the CVE

Figure 5. Femi.

Figure 6. Released Javan Loris.

Figure 7. Southern Cassowary.

Figure 8. Javan Green Magpie.

Figure 9. Slow loris in Sanctuary.

Figure 10. Rosi.

Figure 11. Crested Serpent Eagle in rehabilitation.

Figure 12. Black Winged Myna.

Figure 13. Femi.

and an exceptionally popular hobby. Like rural shows in Australia, Javanese people show their song birds at festivals and competitions, vying for prize money and ribbons for the best song. If you drive along any road in Java, go into any marketplace, the homes and streets are lined with beautifully ornate and expensive cages, with a single bird kept inside for their song. Every person who keeps a song bird loves their bird genuinely; caring for it, spending what little money people have on it and giving it a beautiful cage and the best food. No one can doubt people's passion for this hobby. However, whilst the streets are alive with the songs of birds, the forests are silent. With over 280,000,000 people in Indonesia (yes 280 million), the demand for birds is phenomenal, and cultural shifts in attitudes are difficult.

Thank you

On 24 August 2018, Yvette posted a request on the CVE's Facebook page asking if we could donate absorbable suture material. We called Peter Darge, Business Unit Manager for B. Braun Australia Pty Ltd in Sydney, who was very keen to assist this worthwhile project. Pete couriered the supplies in time for Yvette to deliver them in person to Cikananga Wildlife Center.

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SHARING EXPERTISE

The rarest of the rare

In 2007 Cikananga started a captive breeding centre for the rarest of the rare. The Javan Green Magpie (figure 8), not seen in the wild for 10 years, hangs on with a known global captive population of just over 60 individuals, half of which live at Cikananga. The Rufous Fronted Laughing thrush (figure 14), has just 40 individuals remaining, also over half live with us. The West Javan Black-winged Myna (figure 12) disappeared in 2018 from its last known locations, now presumed extinct in the wild and the forests silent. And the aptly named Javan Warty Pig (figure 4), whose 'warts' on its face swell during breeding season to attract the female's attention, lives in the largest captive insurance population in the world within Cikananga. For our team, this is a humbling responsibility. Everyday our veterinary team is caring for the rarest of the rare, where every decision on an individual potentially impacts an entire species.

Inspiration to continue our work

However, in a world that seems to be continuously full of the depressing and the 'is there any hope?' news from the

Figure 14. Rufous Fronted Laughing Thrush.

natural realm, we do have our successes that inspire us to continue to our work.

We work closely with our partners to release critically endangered Javan lorises back into the wild, populating and protecting areas where they have been exhausted. We have halted the extinction of the Javan Green Magpie, Rufous Laughing thrush, Black-winged Myna and Javan Warty Pig. We have started a local bird watching club to inspire the next generation to keep birds flying free. Cikananga has huge success with releasing eagles (figure 11) and owls back to the wild, with local communities acting as guardians for these animals, and these animals are even seen breeding and benefiting local people, allowing naturally functioning ecosystems around their farms to decrease rat and mice populations, and increasing harvests. The Southern Cassowary (Figure 7)—the same species we find in Cape York in Queensland—is also found in Papua. Whilst in Australia traffic incidents and insatiable land clearing for houses along the coast threaten their existence, in Indonesia it is again people wanting an exotic pet that sets alight the illegal wildlife trade. Cikananga has instigated a world first and now preparations are underway to fly the flightless birds home. Rescue centers and zoos across the country are inundated with cassowary—the initial program has identified over 45 candidates. Programs like this not only afford animals a second chance, and repopulate areas exhausted of wild populations helping to restore ecosystems, they allow for local communities to benefit, feel pride in the attention

Figure 15. Rescuing Javan Leopard from human animal conflict in Cireunghas, Sukabumi.

Authors' views are not necessarily those of the CVE

their wildlife receives, and in turn we hope changes the next generation to protect their assets, not ransack them for short term gain. Our Javan Leopard Mitigation Program provides second chances for critically endangered Javan leopards—with an estimated 250 wildlife individuals left—every single one counts. We operate a rescue team who rescue leopards from conflicts with human populations (figure 15)—an ever-growing issue on an island of 140 million people in an area just over half that of Victoria. For those animals for which the past horrors cannot be corrected, we provide sanctuary for life. Sun bears, Bornean orangutans, Siamangs, Gibbons, turtles, crocodiles and cockatoos are provided with the lifetime of care not afforded to them before.

And Femi and Rosi, the two traumatized infant orangutans?

This year they flew back to Sumatra to start their rehabilitation back into the tropical forests of Sumatra as young confident animals ready to take on the world.

Proactive and dedicated individuals always make a huge difference

Yvette Anderson, a full-time clinical pharmacist, masters student and mother of three young nature-loving boys went on a life-changing tour of Borneo in early 2018. Yvette was struck by the harsh and sobering reality of wildlife issues in Asia. Rather than sink into the pit of despair or ignorance like so many, she got to work. Despite her already hectic life, Yvette signed on to fundraise for 'Free the Bears', and 'The Australian Orangutan Project'. And for Cikananga, she set about contacting medical companies such as B -Braun and hospitals—both human and animal—seeking donations to help rescued and critically threatened wildlife. In a matter of months, she raised AU$4,000 worth of equipment to help Java's wildlife This equipment sourced by Yvette—microchips, surgical tools, anaesthesia equipment, medicines and hundreds of other hard to get items in Indonesia—has made the most significant difference to the wildlife in our care, and in turn conservation efforts through our wildlife release programs. One extremely busy but inspired individual found time to seek help for wildlife and her actions have made a huge difference to our centre.

Like many of the NGOs operating throughout the developing and even developed world, we run on the support of donors, partners and volunteers like Yvette to help us fight the constant threat from the illegal wildlife trade. However, this isn't a call for how harsh the reality is and how bleak the future is—rather it is to highlight what can be achieved when a small dedicated group of people puts their skills and tireless efforts to into actions for wildlife.

Keen to help?

If you would like to donate your time or assist to provide much-needed equipment and supplies to the centre, please contact Cikananga team at cikanangawildlifecenter@gmail.com

Figure 16. Education programme – bird watching club.

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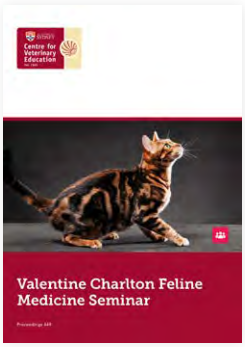

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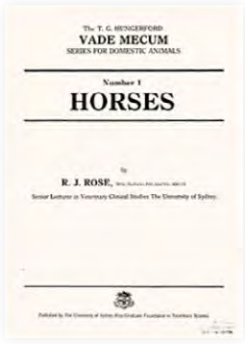

Vade Mecum

The hidden insurance of epididymal sperm

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When presented with the unexpected loss or euthanasia of a valuable breeding male, preservation of genetic material is not often at the forefront of conversation. However the ability to harvest semen and thus genetics via the storage site of sperm, the epididymis, makes it a discussion worth having.

Sperm maturation

Sperm of all species undergo a maturation process starting in the testicular tubules and working their way to the epididymis. In dogs this process takes 62 days and in horses 55 days. Sperm cells that have made their way to the tail of the epididymis, whilst not as fertile as ejaculated sperm, now have fertilising ability; they have acquired progressive motility, the cytoplasmic droplet has migrated and the acrosome region has matured. With sperm in this region known to make up 61-66% of all extragonadal sperm reserves in stallions, this storage vessel has a significant number of sperm available for harvest (up to 50 breeding doses in stallions depending on quality).

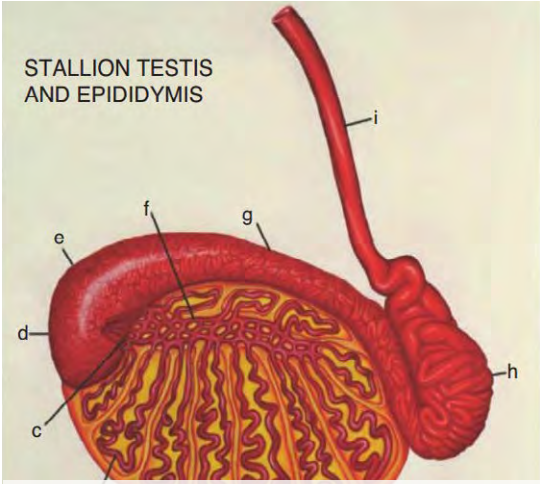

Figure 1. Mckinnon et al 2011.

Initial castration and shipping procedure

Remember that the tail of the epididymis (h) is firmly attached to the caudal pole of the testicle by the proper ligament of the testes and to the vaginal tunic by the ligament of the tail of the epididymis (Figure 1). Therefore only a few modifications to a routine castration are required to transport the epididymis to the laboratory.

Castration can be performed immediately prior to euthanasia under GA or as soon as possible following the sudden death of the stud. The testes are obtained by routine open castration, with three major 'modifications' to note:

1. Identify and ligate the ductus deferens as proximally as possible to minimise loss of sperm
2. Minimise handling of the tail of the epididymis to prevent traumatic injury
3. Ligate or clamp the vasculature to prevent contamination of the epididymal sperm with blood

Following castration testes should be cleaned and rinsed with warmed isotonic saline to remove blood, tissues and contaminants. Testicles should then be wrapped in swabs for placement in an esky containing frozen ice bricks. It is important that there is no direct contact between the two, with tea towels separating them usually the most practical option. This is essential, as the factor known to be most detrimental to sperm health during transport is an abrupt

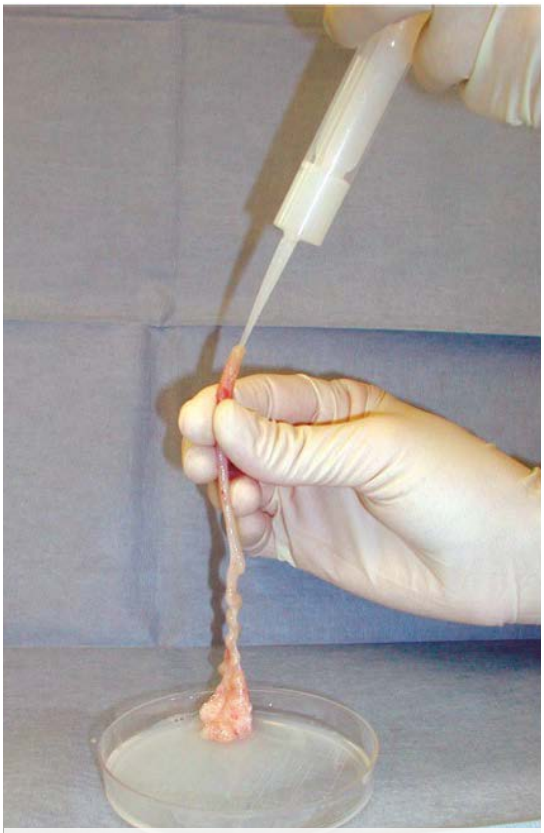

Figure 2. Eichelberger et al 2007.

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temperature change. If access to an 'Equitainer' or semen shipment box is available, this is preferred.

Whilst shipment to the processing laboratory should occur ASAP, it has been demonstrated that delays in processing up to 24h in the horse and 48h in the dog did not exert a negative effect on post-thaw motility of the sample (James et al. 2002, Ponglowhapan et al. 2006).

Laboratory processing

There are a couple of recognized techniques for harvesting epididymal sperm upon arrival to the laboratory, including the retrograde flush and float-up techniques. Both begin with careful dissection of the tail of the epididymes and ductus deferens from the testes having placed clamps on the proximal ductus deferens and at the junction of the body and tail of the epididymis. At VRC we utilise the retrograde flush technique, which involves flushing the tubular lumen with semen extender via a catheter after removing the distal clamp on the epididymis.

The sample is then processed similarly to an ejaculated semen sample, frozen and stored indefinitely in liquid nitrogen at -192˚C. A test straw will be thawed to assess how the sperm have tolerated the freeze-thawing process, giving us an idea of quality of the sample after withstanding the rigours of processing.

Insemination & conception

Due to the decreased fertilisation ability of these sperm cells precise timing of insemination is required in both the bitch (via progesterone testing) and mare (via rectal ultrasound examinations or Advanced Reproductive Technologies including intracytoplasmic sperm injection [ICSI]). Pregnancy rates are significantly lower compared to fresh, cooled or frozen thawed semen, hypothesised to be a result of reduced motility and morphology, decreased capacitation capacity and/or acrosome react, absence of natural seminal plasma within the collection or likelihood of presenting situations involving inflammation or pyrexia and thus impacting on sperm health. However, with careful breeding management the best chance can be given to propagate valuable genetic material that is otherwise unobtainable.

Conclusion

Harvesting and cryopreservation of sperm from the tail of the epididymis provides a last resort for studs suffering unexpected injury or loss of life and should only be recommended for animals of fertile breeding age (i.e. dogs <6yo). Should the desire to store semen from individuals be known it should always be recommended to ensure a collection is performed at a young, fertile age (i.e. 2yo in the stud dog). However knowledge of this technique is key in providing clients with all options at a time when unexpected losses are faced.

Epididymal sperm harvesting is offered by Dr Jasmin Hyatt at Veterinary Reproduction Consultancy, Orange NSW.

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The value of in-house cytology

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This is a collaboration between Chris, who supplied the images, and Mark the comments.

The gross image shows a variably pigmented (likely ulcerated), well circumscribed lobulated oral mass in the buccal mucosa. There is clearly a subcutaneous or dermal mass at the angle of the jaw. This is at the position of the mandibular lymph node and is likely to be lymphadenomegaly.

General approach

The general approach to a mass lesion like this would be to consider a pathological process that results in cells accumulating in a tissue to form a mass. The two pathological processes, to consider therefore, are inflammation and disorders of growth (proliferations). Chronic inflammation, including granulomatous inflammation, can result in large numbers of inflammatory cells being recruited into a tissue with very limited evidence of the cardinal signs of acute inflammation. A disorder of growth (neoplasia) is more likely. The gross image appears to be a mass that is likely to have a firm consistency and its position is also not supportive of a degenerative condition like a cyst or sialocoele, or a circulatory disturbance such as focal haemorrhage.

So, the most likely pathological process present is a disorder of growth but the possibility of a chronic focal inflammatory process cannot be completely excluded.

Within Disorders of Growth, we would need to examine the mass microscopically in order to confirm whether it is a disorder of growth and then to try to determine whether it likely to be a malignant proliferation or a benign proliferation.

The primary differential to rule out in the diagnostic process would be malignant melanoma. Given its site and clinical appearance this is possibly the highest on our list of potential diagnoses. This is probably the differential that you would want to rule out or confirm primarily because of the poor prognosis of these cases. Some interventional therapeutics around autologous vaccines have been attempted.

Malignant melanoma can have various microscopic appearances, including round cell, epithelial cell or spindle cell morphologies, sometimes all within the one tumour.

On appearance, I would not completely exclude the possibility of an epithelial proliferation such as squamous cell carcinoma, or even potentially a round cell proliferation such as lymphosarcoma. From the gross appearance, I think a round cell proliferation is less likely. Other spindle cell proliferation seems less likely but an histiocytic proliferation could not be excluded at this stage. Histiocytic malignancies can have spindle cell and round cell manifestations.

The possibility of granulomatous inflammation is less likely but not impossible as a foreign body response or similar.

The easiest next step in investigation would be cytological examination of the mass. A fine needle aspirate biopsy could be useful, and preparation of a smear (squash prep or a spray prep) is likely easiest, as long as you get good exfoliation of cells.

You may consider taking a surgical biopsy at the same time,

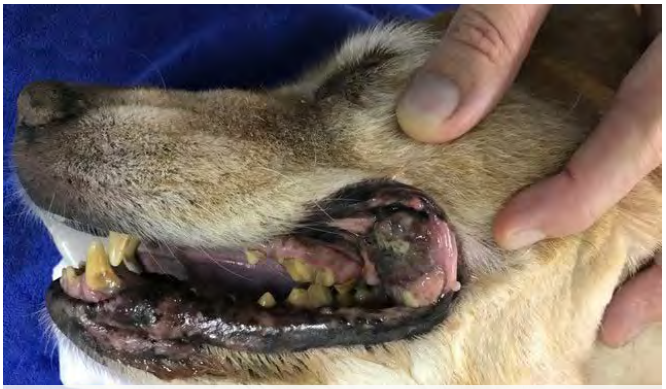

Figure 1. Oral mass in buccal mucosa.

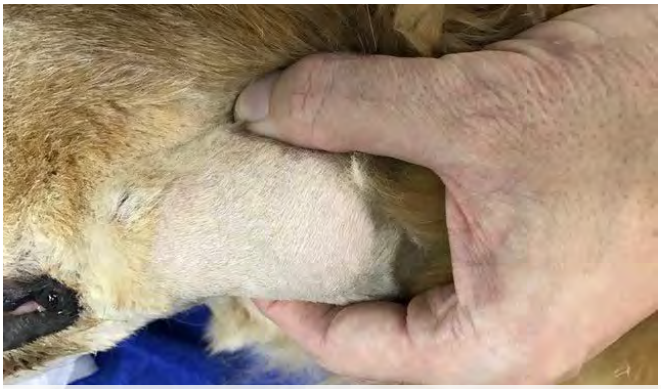

Figure 2. Lymphadenomegaly.

if you have the animal anaesthetised anyway to collect the FNA. When you do a surgical biopsy, consider doing an imprint smear cytological preparation to increase the likelihood of getting a diagnostic specimen. You can hold the surgical biopsy in case cytological examination is non-diagnostic and then submit to the laboratory.

A fine needle aspirate of the local lymph node should be performed at this stage to gather information about staging if it does in fact turn out to be a malignant disorder of growth.

Other diagnostic interventions that may be considered at this point may be imaging to assess potential metastasis, particularly to the lung fields.

Cytology

Description: The cytological image (Figure 3) shows good nucleated cell recovery with minimal blood contamination in the field. The cell recovery is characterised by a group or clump of cells. The cells have medium to large round irregular nuclei with clumped chromatin and frequently large prominent nucleoli. There is mild to moderate anisokaryosis with nuclear moulding evident. The cells have a moderate to abundant pale blue to grey cytoplasm occasionally containing fine blue cytoplasmic granules in low abundance.

The cytological image (Figure 4) shows a part of the slide in which there is substantial background blue grey material (likely ruptured cytoplasm) with scattered coarse and fine blue granules throughout and numerous naked nuclei of similar appearance to the previously described cells. Centrally, is a large cell with a medium sized round nucleus with dispersed chromatin and a large central nucleolus. The cell has abundant cytoplasm containing numerous fine to course blue-grey-green granules (likely melanophage).

Interpretation

If the image is representative of the mass, it confirms the presence of a disorder of growth. The clumping nature of the cells is suggestive of an epithelial proliferation but does not exclude a melanocytic or histiocytic proliferation. The presence of the blue-grey-green cytoplasmic granules at low abundance is supportive of the cells in fact being

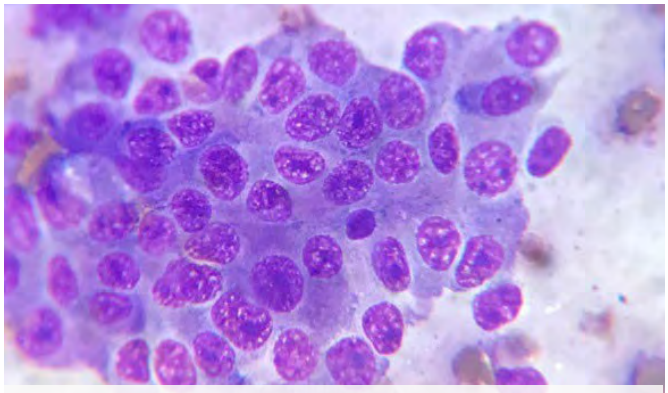

Figure 3. Cytology from FNA of oral mass.

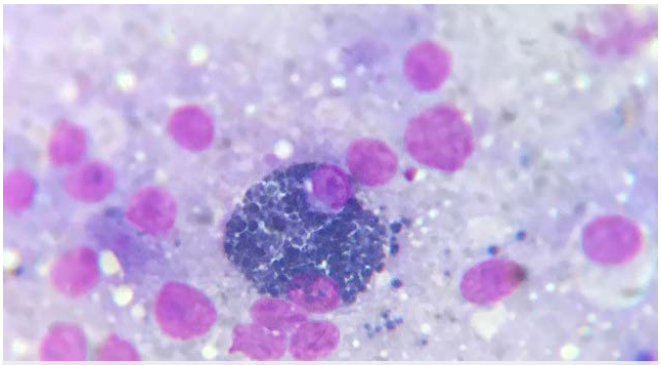

Figure 4. FNA of mandibular lymph node showing melanin granules in melanophage.

melanocytes (melanin pigment). Amelanotic melanomas will produce little melanin and it is the presence of melanophages accumulating the pigment from dead tumour cells that may be one of the only clues that the neoplastic cells are actually producing melanin. It is possible that the likely melanophage is actually simply a coincidental finding and is just a normal melanophage associated with a pigmented mucosa. Given the presence of the proliferation and light presence of granules in the neoplastic cells, this is less likely. The pigment does not appear to be haemosiderin (which should be more blue on staining with DiffQuik), so an erythrophagocytic histiocytic malignancy seems unlikely.

Conclusion

Likely oral malignant melanoma.

Hypocalcaemia complications post an unplanned bilateral thyroidectomy

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Thor is a 13-year-old male neutered Domestic Short Hair that lives exclusively indoors with 2 other cats. He first presented to The Cat Clinic Hobart in January 2016 for over grooming, largely due to stress. He had previously had multiple recurrent episodes of this throughout his life.

His owner was not concerned and was not interested in treating him for this condition. He weighed 5.29kg and had a body condition score of 5/9. He was started on Hills Science diet C/D® Stress dry food. One month later his owner reported that he has been exclusively on the diet and was possibly a little better.

Twelve months later Thor presented for weight loss. He weighed 4.45 kg and had a body condition score of 4/9. His appetite was increased (in fact he was dying of starvation according to his owners), his over grooming had resolved but he was showing signs of arthritis. His clinical examination was unremarkable other than a heart rate of 240 bpm. A thyroid goitre was not palpable at this time.

Blood was collected for a Total Annual Health Profile (TAHP). The results showed mild elevations of his ALP, AST and ALT. His T4 was > 150 (10-60) nmol/L. The remainder of his blood results were unremarkable. Treatment options for hyperthyroidism were discussed with his owner, and radioactive iodine was recommended. Thor's owner initially started treatment with carbimazole 5 mg BID. As sometimes happens, the next blood test that was performed on Thor was 6 months later at a medication checkup. He had gained weight and was now 5.33 kg. His appetite was good (no longer increased), his over grooming remained resolved and the remainder of his clinical exam was unremarkable apart from his heart rate being 200 bpm. He continued to show signs of possible arthritis according to his owner.

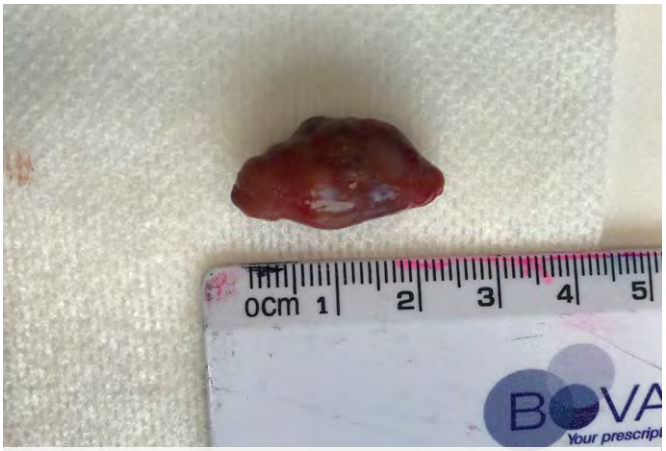

Figure 1. Left thyroid gland removed at first surgery.

Thor's T4 was 24 (10-60) mol/L. His SDMA had increased from 10 µg/dL to 16 µg/dL (0-14). Other than that his bloods were unremarkable. We started him on daily oral meloxicam for his arthritis.

Thor returned to the clinic 7 months later for repeat bloods and a checkup for ongoing medication. His owner reported that in the last couple of weeks he was not as content as he had been previously. He was still on carbimazole 5 mg BID and oral meloxicam 0.25mg once daily. His clinical examination was unremarkable and his heart rate was 140 bpm. Thor's weight remained stable at 5.30 kg. Thor's T4 was < 10 nmol/L (10-60). We reduced his carbimazole to 2.5 mg BID and booked him in for repeat bloods in 4 weeks time.

Four weeks later we repeated Thor's bloods. He had lost 400g in four weeks, his blood pressure was 200 mmhg on doppler (5 readings) and he now had a palpable large thyroid goitre. We started amlodipine at a dose of 0.625 mg SID and booked him in to recheck his blood pressure in 7 days time. His T4 was again > 150 (10-60) mol/L. His liver enzymes were mildly elevated, and his SDMA was 8 µg/dL (0-14). His owner did not want to persist with medical management and opted for a thyroidectomy. Thor also had a small lump on the left side of his chin that she requested we remove at the same time.

At the time of the surgery Thor's weight had reduced a further 280g. His blood pressure was 180 mmhg (doppler: average of 5 readings) although he was not as relaxed as the last time it was taken. Thor received an ACP/ Methodone IM pre-med and alfaxalone induction and Isoflurane maintenance. His GA was unremarkable and he recovered quickly.

The thyroidectomy was routine with a midline ventral neck approach. The thyroid gland was very large and cystic and I was concerned about neoplasia so opted not to auto

transplant the parathyroid tissue back into the surgical site. The mass/cyst on the left hand side of his cheek was also removed in its entirety at the same time.

Post operatively Thor received buprenorphine, oral meloxicam and thyroxine at a dose rate of 0.2 mg SID for 7 days, then 0.1 mg SID for a further 7 days. Thor was discharged the day after surgery and booked for a post-operative check 7 days later.

At post-op checkup Thor had gained 140g and was very bright and happy. He continued on with oral meloxicam (0.225 mg SID) and amlodipine (0.625mg SID). He was scheduled for a repeat blood pressure in 4-6 weeks and to consider starting a course of Zydax (pentosan polysulphate sodium) injections as his owner was still concerned about possible arthritis.

Thor presented 2 months later for repeat clinical signs of hyperthyroidism. He had not had repeat bloods post his initial thyroidectomy and we had not checked his blood pressure in this time either. Thor again had lost 200 g in weight and was again ferociously hungry. His owner was quite disheartened and was considering euthanasia due to a perceived poor quality of life. His owner thought for the first 3-4 week post thyroidectomy he was amazing but in the last 3 weeks he had really declined. His clinical examination was unremarkable apart from a large thyroid goitre that was palpable. The goitre was at least the same size as the previous goitre that was removed 2 months earlier.

As we had kept the initial thyroid gland in formalin we sent this for histopathology and his bloods were again repeated. Histopathology diagnosis was Thyroid gland neoplasm (carcinoma suspected) and the cheek lump was a Basal cell tumour (apocrine gland). His blood results were again consistent with Hyperthyroidism (T4 > 150 nmol/L (10-60)). At this stage I contacted Dr Richard Malik for advice as I wrongly assumed that this meant a poor prognosis for Thor. Dr Malik suggested that Thor simply now had disease within the other thyroid gland and we should treat them as independent diseases and that most feline thyroid carcinoma cases do very well. Dr Malik also commented that large thyroid goitres or thyroid carcinomas rarely responded to medical management.

Thor's owner was happy to proceed with a second thyroidectomy for Thor. The biggest concern this time was that I knew I had not preserved the left parathyroid and issues with his calcium regulation and levels post operatively could complicate his recovery.

Thor again received an ACP/methodone IM pre-med, alfaxalone induction and isoflurane maintenance. His general anaesthetic was unremarkable and he recovered quickly. His thyroidectomy was routine and I ensured that I dissected out his parathyroid gland and auto transplanted it back into the muscle at the surgical site. I was not able to preserve the blood supply as I was concerned about leaving any thyroid tissue that could potentially result in another thyroid carcinoma.

Thor's surgery was performed first thing in the morning and his ionised calcium checked at 4.30 pm (6 hours post-surgery) on an in-house iSTAT machine. A pre surgery ionised calcium was not performed. His post-op ionised calcium was 1.01 mmol/L (1.20-1.32).

Thor's intravenous fluids were changed to NaCl 0.9% with 10% calcium gluconate added and a CRI commenced at 2.1 mg/kg/hr. of Ca gluconate. He was sent to the after-hours veterinary emergency centre for overnight monitoring where he was maintained on this intravenous fluid regime. He also received Buprenorphine IV q 6-8 hourly, Maropitant IV slowly q 24 hourly, thyroxine orally 0.2 mg q 24 hourly, amlodipine 0.625mg q 24 hourly and was started on oral calcium carbonate tablets (approximately 37.5 mg). Thor was kept in the ICU for closer monitoring of signs of hypocalcaemia. He was stable overnight and discharged the next morning back to our clinic for ongoing monitoring.

His ionised calcium was repeated the next morning. It was 1.09 mmol/L (1.20-1.32). He was still clinically normal with no signs of hypocalcaemia evident. He was eating normally and bright. We kept his calcium gluconate CRI going at a rate of 2.1mg/kg/hr and continued oral calcium carbonate tablets 37.5 mg crushed in food three times daily, oral thyroxine 0.2 mg q 24 hourly. We also started oral calcitriol.

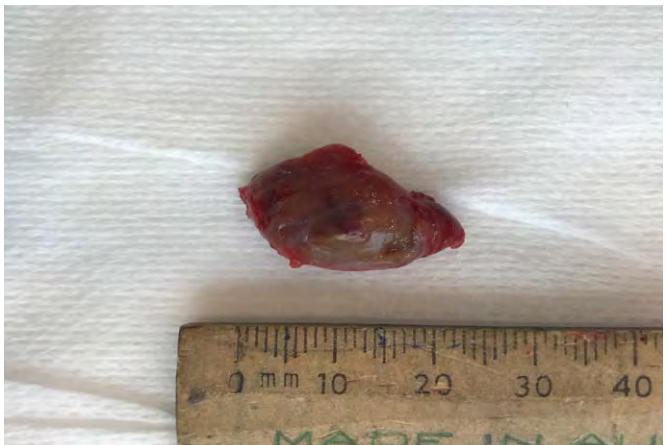

Figure 2. Right thyroid gland removed at second surgery.

Figure 3. In house i-STAT machine for measuring ionised calcium.

Caclitriol dosing was complicated (0.01-0.02 µg/kg divided daily and given twice daily). Thor required treatment immediately so compounding was not an option initially. In the end we obtained 0.25 µg capsules and used diabetic insulin syringes to draw up the contents of the capsule and gave him 0.025 mLs orally on his food twice daily. His owner knew that this was not accurate but was happy to proceed. Thankfully Thor continued to eat well and took all his medication mixed in his food.

Thor spent the night again at the after-hours veterinary emergency centre for close monitoring and they began reducing his calcium gluconate CRI with the plan to stop it the following morning providing he was still asymptomatic. When Thor returned the next morning his calcium gluconate CRI was stopped, his IV catheter was removed and his ionised calcium was repeated. His iCa was 0.92 nmol/L (1.20-1.32). Thor was discharged later that day as he continued to show no signs of hypocalcaemia, was taking his medication well and appeared ready to go home. His owner gave us an update the next morning to say he was a little unsettled, every time he went to rest he would get back up again. Eventually he went to sleep but his owner thought she observed him twitching. She did not feel it was necessary to bring him back into the clinic.

Thor presented to the after-hours veterinary emergency centre 4 days post thyroidectomy at around 10 pm at night. He had fallen off the sofa at home and had a 10 second seizure. His owner was again considering euthanasia but thankfully one of the after-hours veterinarians talked her around and explained that Thor could still expect a good quality of life once his calcium regulation was better controlled and that this was not completely unexpected. Thor's ionised calcium was 0.76 mmol/L (1.1-1.4). They started him back onto a 10 % calcium gluconate CRI at a dose rate of 2.1 mg/kg/hr. He responded quickly and after 1.5 hours his ionised calcium was 1 mmol/L (1.1-1.4). At 5 am his ionised calcium was 1.18 mmol/L (1.1-1.4), he had been stable and comfortable overnight and his vital signs remained within normal limits. Thor returned to our clinic in the morning.

We increased Thor's oral calcium carbonate to 100 mg TID along with the calcitriol to 0.04mLs (4IU) BID orally. Thor was markedly improved and we again reduced his calcium gluconate CRI and weaned him off. Thor was discharged at the end of the day. The following day Thor refused his medication which his owner considered a good sign. His owner's next update was 2 days later. She reported that he was bright, eating very well, was no longer lethargic and there were no further signs of twitching or seizure activity.

We repeated his ionised calcium 2 days later: Our iSTAT machine showed an error for his ionised calcium so we sent his blood to our local human laboratory. The result we got back was 0.81 mmol/L. They do not have a reference for cats. Given the delay in testing the sample we assumed that his actual ionised calcium was probably higher than this result. We made no changes to his medication and planned to repeat his iCa in 7 days. His creatinine was 112 mg/dL (21-141) and his urea was within normal limits.

The next iCA result was performed on our iSTAT machine in house and was lower again: 0.75 mmol/L (1.20-1.32). Thor was gaining weight however and was very bright. His owner's biggest concern for Thor at this stage was that he was showing increased signs of arthritis and so we started him back onto oral meloxicam once daily. We also increased his calcitriol dose by 1 IU twice daily 0.05 mLs or 5 IU). His creatinine was 118 mg/dL (21-141) and his urea was within normal limits.

Ten days later we again repeated his iCa. Thor had again gained weight and his owner was happy with his progress. She had ceased the use of the oral meloxicam due to Thor vomiting blood and her main concern was still his arthritis. Thor was eating well and doing everything normally. She had made no other changes to his medication. His iCa was 1.23 (1.20-1.32) mmol/L. The decision was made to keep all medications the same for the next month. We went to monthly testing due to costs. His creatinine was 187 mg/dL (21-141) and his urea was within normal limits.

Thor presented 4 weeks later. He had lost 500 g in weight. He was miserable, he had a poor appetite and his sleeping routine had completely changed. His owner was still concerned about his quality of life and whether he was sore with arthritis. His iCa was 1.36 (1.20-1.32) mmol/L. We made the decision to stop the oral calcium gluconate tablets completely at this stage and discussed considering a course of Zydax (Pentosan polysulfate sodium) injections for his arthritis. His creatinine was 353 mg/dL (21-141) and his urea was within normal limits.

One week later Thor returned again with further weight loss, increased vomiting and hyporexia. He was very clingy with the owner at home and again his owner considered if we should persist and was concerned about his quality of life. We repeated his iCA which was higher again at 1.38 mmol/L. His creatinine was 353 mg/dL (21-141) and his urea was within normal limits. We reduced his Calcitriol dose by half and scheduled him for a repeat iCA in 7 days.

Seven days later our iSTAT machine had another error so his sample was sent to our local lab again. His iCa was 1.31 (no reference range available). This was lower than the result the week before but still too high. We did not get a crea/urea result at this visit. We reduced his calcitriol to once daily every other day. Thor had gained 320g in a week. He was obviously sore in the consult room and his owner agreed to start a course of Zydax injections. We planned to repeat his iCa at the end of the 4 week Zydax course. At the 3rd Zydax injection we stopped the Calcitriol.

Thor has completed his Zydax course. His weight is back up to 4.73 kg. We intend to repeat his bloods and his blood pressure at his next visit which is scheduled in the next month. Apart from Zydax and amlodipine he is on no other medications.

Figure 4. The gorgeous Thor.

Without the help from Dr Richard Malik I think that Thor might have had a very different outcome. I am not sure had I been able to preserve the parathyroid on the first thyroidectomy if things would have been different? Given that his other thyroid gland was affected within a couple of months maybe it would have made no difference but I will certainly ensure I auto transplant the parathyroid in all future thyroidectomies. I also do not know if radioactive iodine would be useful due to the dose limit that our facility in Tasmania is able to use. The fact that Idexx no longer gives us an actual value when the result is >150 mmol/L makes it more difficult to differentiate between adenoma and carcinomas. In Thor's case we did not know he had a suspect carcinoma until we got the results of the histopathology back. I think compounding the calcitriol in future cases would allow much more accurate dosing, although we did get there in the end and the human tablets were very cost effective. Without our after-hours veterinary emergency clinic I do not think that I would attempt a bilateral thyroidectomy given the potential for severe post op complications. Our iSTAT machine, when it was working, was invaluable in this case as ionised calcium must be measured immediately after collection. It also allowed us to monitor his creatinine which was shown to increase when he became hypercalcaemic.

We would like to acknowledge the help of Dr Richard Malik from the CVE. As I said, the outcome would most likely have been different had he not given us direction throughout this case.

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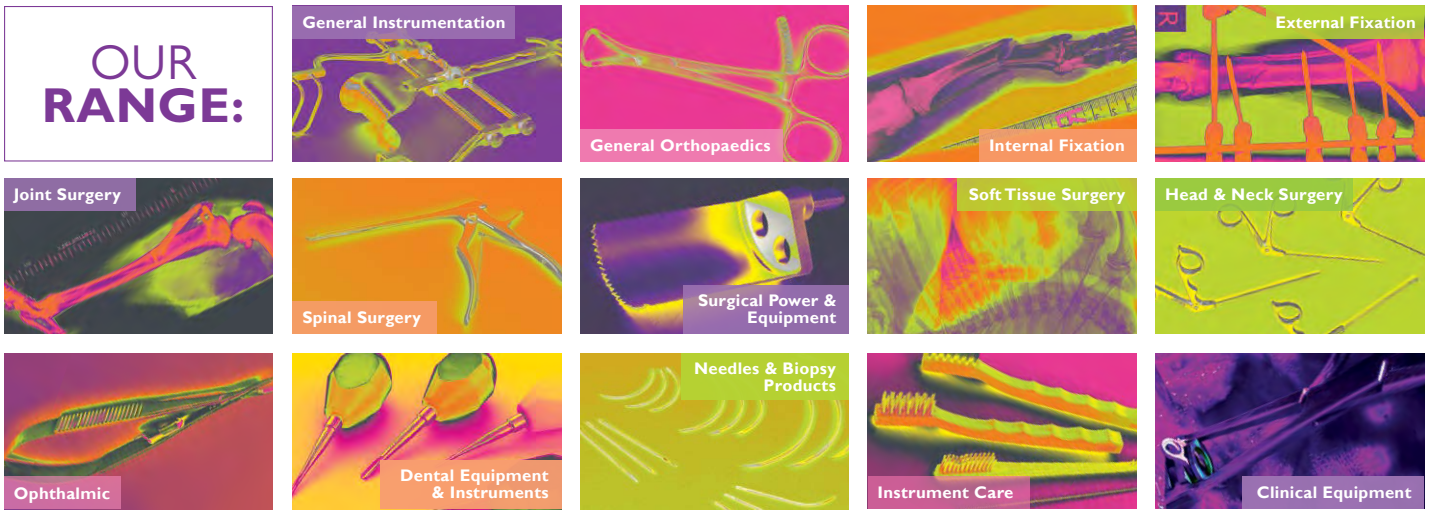

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A diversity of salivary holocyclotoxins provides a plausible explanation for multisystem organ dysfunction in cases of tick paralysis due to *Ixodes holocyclus*

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The recent report using transcriptone analysis to identify 19 holocyclotoxins (HTs) from the Eastern paralysis tick *Ixodes holocyclus* and submission to GenBank (ncbi.nlm.nih.gov/pubmed/23193287) of 21 nucleotide sequences related to HTs by Zoetis, Australia (WO 201408724 A1)[1] underscores the diverse toxin profile elaborated by this ectoparasite. These findings provide scope to better explain the often-puzzling array of clinical signs of multisystem organ dysfunction observed in cases of tick paralysis that are not readily defined by a single or narrow toxin profile.

Indeed, in addition to the generalised ascending lower motor (LMN) weakness that characterises this disorder, other signs may include bladder dysfunction, prolonged QT interval with altered T wave morphology, cardiac diastolic dysfunction, mydriasis and asymmetrical focal neurological deficits such as anisocoria, Horner's syndrome, unilateral facial paralysis and unilateral absence of the cutaneous trunci reflex.[2,3]

Neurophysiological evidence for blockade of potential molecular targets by different HTs to delineate a number of these effects has been described. Blockage of voltage-gated calcium channels of presynaptic motor nerve terminals by HT1, HT3 and HT12 likely underlies the pathophysiological mechanism for the signs of generalised LMN weakness [4] that was first suggested by earlier studies on phrenic nerve-hemidiaphragm preparations of mice paralysed by nymphal Ixodes holoculus[2]. Further, effects are variable, as shown by investigations in neonate mice were HT1, HT2 and HT3 each produced mild signs of paralysis of slow onset, HT4 induced severe and rapid paralysis and a combination of HT1, HT2 and HT3 resulted in more rapid and severe paralysis than each HT individually.[1]

Similarly, findings in a rat model demonstrating blockade of outward rectifying potassium channels in cardiac and vascular smooth muscle, by as yet unidentified HTs, provides a plausible explanation for many of the cardiovascular effects seen in affected cases.[5]

Krishnan et al described the case of a 45-year-old man with unilateral upper limb weakness, lasting for at least one month after a bushwalk in eastern Australia, that involved all muscles innervated by median and ulnar nerves, and was due to a tick in the lateral aspect of the left axilla. Nerve conduction studies showed persistent conduction block in the left median and ulnar nerves at the level of the axilla and nerve excitability studies detailed significant abnormalities in the duration of the relative refractory period and refractoriness; both markers for abnormal nodal transient sodium conductance in motor axons.[6] These findings parallel observations of unilateral facial paralysis and unilateral loss of the cutaneous trunci reflex in a proportion of dogs and cats with tick paralysis[3] and raises the possibility that specific HTs may cause local conduction block by blockage of voltage-gated sodium channels, in much the same was as local anesthetics work by focally blocking voltage-gated sodium channels.[7] In such cases, the site of tick attachment was either directly over, or in the region of, branches of the facial nerve innervating affected facial muscles or the caudal axillary location of the lateral thoracic nerve innervating the cutaneous trunci muscle and recovery of function was prolonged, lagging behind that of recovery from the generalised signs of tick paralysis.[3]

Research is yet to be undertaken to elucidate the pathophysiology underlying signs of bladder dysfunction, mydriasis, anisocoria and Horner's syndrome observed in selected cases of tick paralysis.[2,3] However, it is plausible that ion channels subserving neural transmission in pre and/or postganglionic elements of the neural innervation to the bladder, oculosympathetic and/or

oculoparasympathetic pathways are also molecular targets for specific HTs.

Thus, the possibility that multiple channelopathies can be induced by differing HTs or combinations of HTs, is likely to provide new and exciting avenues for future research to better understand the mechanisms underlying multisystem organ dysfunction in tick paralysis due to *Ixodes holocyclus*.

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SMALL

Seasonal timing of more severe canine cases of tick paralysis

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C&T No. 5772

The variation in case severity during a season has been a confusing entity. Some veterinarians (feedback based on a series of lectures over several years on the east coast) felt that the most severe cases are at the beginning of their season while others felt they occurred at other times, during their own local season. In theory the most severe cases should be at the start of the (local) season when ticks are most viable (and accessible to pets) and animals least immune from any previous tick exposure.

However each practice is unique in its animal draw area, its season start time and in their clients' animal exposure potentials. Ticks however, can be wind-blown and can locate high in vegetation; thus they can effectively 'arrive' in a bandicoot-free area. Ticks can also be 'carried' by many different species. Usually the most viable ticks are more likely acquired while pets are exposed to cooler, moist areas (e.g. shady creek bank) where the temperature / humidity 'microclimate' factors are the most favorable for tick viability.

Based on many prolonged observations of their movements, ticks tend to move up vegetation until they get to an end point. For example up a trunk to a cross branch and then out to the tip of leaves (bushes) or up grass and out to smallest tip. They sense gravity so will keep walking 'up' until a 'cross branch' is found. They then walk laterally until they get to an end where they stay still (in their gene, down-regulated state) until CO_2, heat, vibration etc. ensues or when a (collecting) white sheet is dragged over them (for grass) or placed around the trunk and the bush shaken. They will continue to move upwards if no cross branch is found; being tracked to high (metres) levels in vegetation.

When do the most severe cases appear? Allowing for the perceptions of the first case exposure of maybe 'inexperienced' staff and the practice 'gearing up' to its local tick season, the worst cases, proportionally, occur at the start of the season. There are many more general cases later in the season but, proportionally, fewer severe cases so the most probable perception is more severe, proportionally, earlier in the local season.

A local clinic[1] recorded (over two successive seasons; pilot followed by final study) the case severity over time based on the NMJ scoring system of 1 to 4, (with 4 being unable to right, 3 not able to stand and 2 not able to walk). Sales

[1] Manly Road Veterinary Hospital, 219 Manly Rd, Manly 4179; Supported by a Merial Study Grant

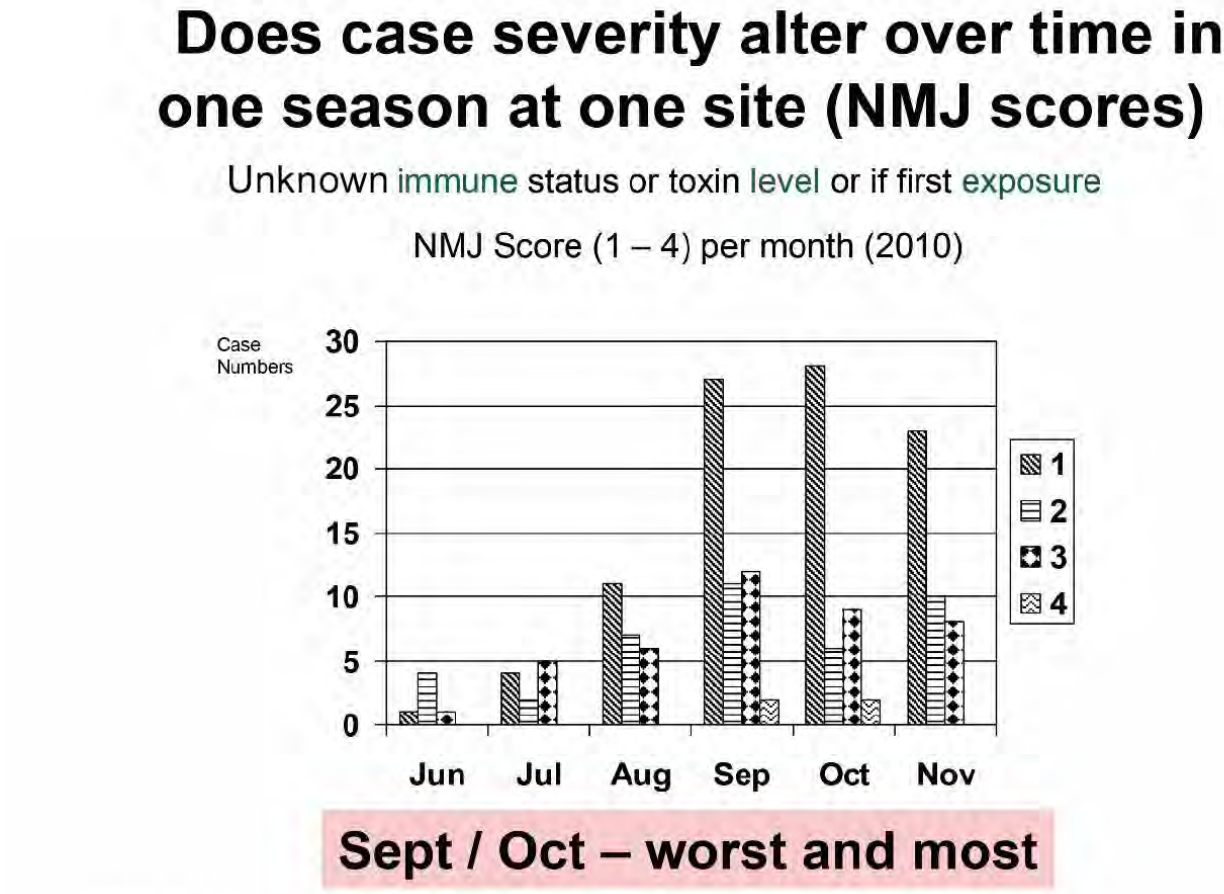


Figure 2.Case severity according to season.

of TAS (tick anti-serum) were used to check computer case data to ensure all possible completed data sets were included. The defined season (where a lot of quickly-progressive cases, e.g. from stage 2 to 4, suggested a 'hot' season) lasted 5 months (data for June was also collected but too few cases were seen) and overall mortality (excluding euthanasia cases) was just under 5%.

A total of 227 cases were available for analyses. Of the 24 dogs euthanased[2], overall costs were the primary issue in 6 cases, whereas clinical deterioration was the reason in 17. Most were in Sept / Oct with 14 of the deterioration group. Of those that died in the hospital (n=10), most did so in Sept / Oct (n=7).

Of 16 cases given TAS only and discharged (with appropriate documentation), 13 claimed overall costs as the issue and one dog could not be hospitalized due to behavioural issues.

Looking at severity (NMJ score stage 3 +) and timing, July to November (Fig.1.), most cases occurred in the Sep. to Nov. period as did more severe cases (stages 3 + 4 but mostly 3). July and August had 11 (28%) of 40 cases (no stage 4, 29 stages 1 + 2) whereas Sept., Oct. and Nov. had 25 (18%) of 138 (stage 4 = 4; 105 stages 1 + 2).

So more cases occur in September and October as do more severe cases. However, proportionally, more severe cases occur in the July—August period.

In general the worst cases should occur when the ticks are 'most viable' and the 'host' most susceptible. However this scenario could occur at 'any time,' as some microclimates ensure tick viability over time and the first exposure / season (when the host is most vulnerable) could be at the start of the overall local tick season or perhaps less so, the chance 'walk by the creek' contact of host and viable tick at another time during that season. The heat and dryness of summer however has an overall effect on tick viability, so cases lessen (in number and severity) as increasing heat and reducing humidity come into effect over time.

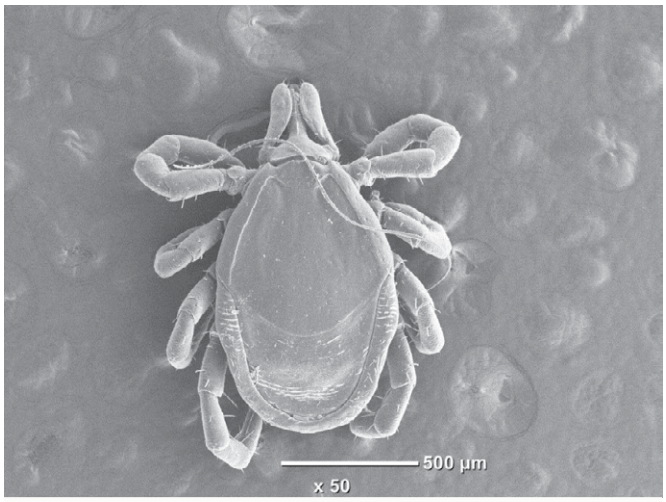
Mortality is determined by the toxin release rate into the host and by the host's susceptibility (passive and active immunity), coupled with the 'best known' therapy to date. Only two drugs have been verified to effect mortality i.e. TAS, and antibiotics when used in severe cases i.e. stage 4 (Merial Study, 2008, n = 506). Oxygen supplementation, anaesthesia and assisted ventilation are not tick specific and need to be evaluated (hopefully under a random, controlled and blinded protocol) in the light of other critical cases that have similar pathophysiology to tick paralysis / toxicity in the same species. Ventilation is vital when indicated but no other tick paralysis-specific therapies have yet been shown to alter mortality.

As with botulism, once toxin is bound to' target cells', effecting e.g. NMJ function, TAS is believed to have no beneficial effect on that bound toxin. As severity is linked to the toxin supply rate and subsequently the total bound toxin load, the sooner TAS is in circulation the better.

[2] Data missing in one case

Toxins so far identified (Toxin Res. Lab., Uni Qld) are not all sensitive to TAS and not all are associated with NMJ effects.

As the TAS reaction has very low mortality, it is much more important to have circulating TAS and not to spend time (delaying its neutralizing effects) by taking a long time setting up e.g. complicated infusion modalities. The TAS reaction is dose / time dependent and rapid i/v dose can induce a detectable reaction in about 85% of (dog) cases. In fact very rapid i/v TAS can cause severe cerebral signs (Schull D., PhD, Uni Qld). However, capturing toxins (still in circulation) with i/v TAS is the issue at hand, thus (maximally) avoiding peri / post vascular escape of toxin, moving to binding sites within various tissues, causing more severe disease.



Scanning electron microscope photograph ixodes holocyclus *nymph. Image courtesy Shona Chandra and Jan Slapeta.*

Image courtesy Jan Slapeta.

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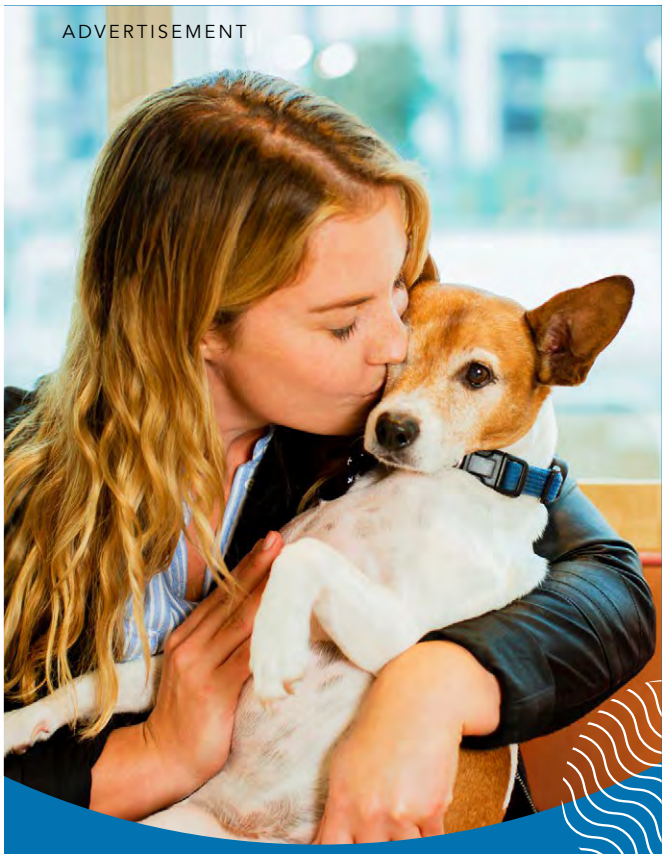

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Persistent pupillary membranes in a Burmese cat

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Leo is a 7-month-old chocolate Burmese, who we welcomed into our family with his sister Lucy, in March this year.

At his third vaccination and check-up, our vet noticed he has what looks like 'bilateral persistent pupillary membranes with no adhesions.' Interestingly, this went unnoticed at his two previous vaccination check-ups.

After this appointment, we made contact with Animal Eye Services in Brisbane by email and provided them with these photos. Our biggest concern was if his eyesight would be affected as he gets older. They advised; 'persistent pupillary membranes are generally a very benign anomaly of development in utero (he would have had them since birth) and would not be expected to cause him any problems in the future'.

We also contacted our breeder as we thought it would be of interest to them. They thought it was fascinating, as they have never seen this before in over 30 years of breeding and showing pedigree cats.

Leo is a much loved member of our family and as our daughter describes, has a 'special sparkle' in his eyes.

Figure 1. Leo.

Figure 2. Leo with persistent pupillary membranes visible in normal light.

Figure 3. Leo

Figure 4. Appearance in bright daylight.

Comment courtesy of Jeff Smith

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Veterinarians need to be able to recognise PPMs and their significance when examining the eyes of young animals. Prior to birth, vascular channels exist within the anterior chamber of the eye. These nutrient bearing vessels will ordinarily atrophy prior to, or soon after birth, leaving no trace.

On occasion, vascular remnants may be recognisable within the anterior chamber as fine pigmented opal strands. The strands may be individual or they may coalesce to form a web-like structure. The strands attach to the iris surface, the lens surface or the inner surface of the cornea.

Where iris-to-lens or iris-to-cornea attachments occur, the attachment sites may be opaque through interference with the normal surface structure.

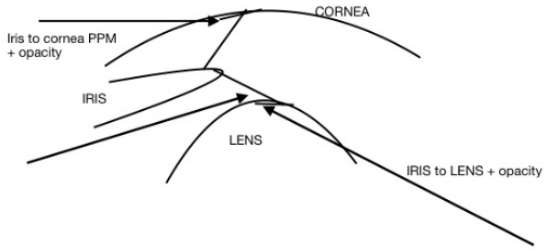
In such cases, focal cataract or focal white spots in the cornea or both may be seen.

The areas of opacification may be large and interfere with vision however as the eye grows with age, the cloudy areas remain the original size and so become relatively smaller and less interfering.

PPMs tend to disappear by 4-6 months of age but their attachments sites remain affected.

Repeated appearance of PPMs in breeding stock should evoke suspicion of a genetic basis.

A genetic basis has been proven in Basenjis.



The use of acupuncture as adjunctive modality to treat symptoms of intervertebral disc disease in a Beagle

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Introduction

Intervertebral Disc Disease (IVDD) in dogs is one of the common neurological conditions seen in practice[1]. It is caused by displacement of part of the intervertebral disc, often located in the thoracolumbar and cervical region, causing pain and neurological deficits. Most disc lesions (85%) occur in the thoracolumbar region (between T11-T12 and L2-L3)[2], with 50% of all disc lesions occurring at the thoracolumbar junction between T12-T13 and T13-L1[3]. Treatment options include medical and/or surgical approaches. Regardless of the treatment performed, physical rehabilitation is an important part of the recovery and acupuncture may be used as an additional modality for pain relief and to enhance recuperation[4].

Disc displacement/ herniation is caused by disc degeneration and can be classified as Hansen Type I or Hansen Type II. Hansen Type I is typically seen in chondrodystrophic breeds[3, 5, 6]. Degenerative change in the discs of these breeds can begin as early as 8 months and continues until 2 years-of-age[7]. The peak incidence for at-risk breeds is between 4 and 6 years-of-age[8]. Normal collagen fibres in the nucleus pulposus (NP) are replaced with less elastic hyaline fibres which increase mechanical stress on the annulus fibrosus (AF) as intradisc pressure changes. The collagen fibres of the AF fail and degenerative NP is released into the neural canal. Disc material is extruded into the spinal cord causing haemorrhage, inflammation and oedema.

Hansen Type II is usually seen in older large breed dogs 5-12 years-of-age. Fibrous collagen slowly increases in the NP, increasing pressure on the AF[4, 6]. As the AF bulges, it places pressure dorsally, compressing the spinal cord leading to a slow focal myelopathy.

Acupuncture has been shown to be effective in the management of IVDD though its analgesic effect, preventing muscular atrophy, strengthening muscles, reducing spasticity and enhancing sensory awareness[9, 15].

In particular, electroacupuncture[i] integrated with western medicine is effective in managing thoracolumbar IVDD[10]. The local and systemic effects of acupuncture are thought to be due to the release of neurotransmitters (endorphins, dynorphins, serotonin, norepinephrine, dopamine), an increase in blood circulation to regulate inflammation and growth factors, changes in cell signalling and reduction in hyperalgesia and allodynia in patients with chronic and neuropathic pain [11, 12, 10, 13].

Murphy's Story

Murphy is a 10 ½ year-old Beagle. Murphy had previously been diagnosed with IVDD in his cervical region at C5-C6 in July 2016. Decompression surgery (ventral slot) was performed and Murphy recovered well.

Figure 1 Murphy.

In January 2019 Murphy's owner noticed that he was walking oddly with his hindquarters swinging 'side-to-side'. On clinical exam there was slight ataxia in both hind limbs and slight lumbar pain on spinal palpation. He had normal CP and superficial pain in both hind limbs (HL) and tail. There was significant panniculus reflex along his whole spine. He was unable to turn in a tight circle and was resistant to backing up. He was given a meloxicam injection and oral meloxicam to take home.

Five days later Murphy was represented for HL pain and ataxia. He had not responded to meloxicam. During clinical examination there was no ataxia or proprioceptive deficits in the HL. HL reflexes were normal. There was marked pain on extension of both hips and pain response on the caudal thoracic area and at L1 to L3.

Differential Diagnosis:

IVDD (+/- osteoarthritis)

Hip Dysplasia (+/- osteoarthritis)

Radiographs:

Lateral and VD views of the thoracic and lumbar spine and hips revealed collapsed intervertebral disc space at T11-12 and spondylosis T12-T13.

He also had bilaterally thickened femoral necks (L>R) and osteophytic lipping on caudal acetabula.

Initial Treatment

Murphy was diagnosed with suspected Grade 1-2 Thoracolumbar IVDD (T11-T12), Hansen Type I. It was recommended Murphy be confined and his movement restricted for 6 weeks. His referring vet prescribed gabapentin and to continue with meloxicam.

Two months later Murphy's owner felt he was still in some pain and favouring hips and left HL despite the continued gabapentin and meloxicam treatment. There was pain on palpation of T11-T12 and the lumbosacral junction. There was restricted extension of the left hip, and the quadriceps in both hind limbs were very tense. Mild kyphosis in the thoracolumbar area was noted. Acupuncture was recommended as a treatment option.

Treatment with acupuncture

Pain and ataxia (mainly LHL)—6 weekly sessions of acupuncture—needle, laser[ii] and electroacupuncture.

Points selected: BL40, ST 36, Bai Hui (master point back and waist), BL 60 ('aspirin point'), BL54, GB 30, GB 29, BL 67 (ting), SP6 (damp), LI11 (wind-heat). Local bladder points corresponding with suspected IVDD lesion.

Needle—Bai Hui, BL 11, GB 30, BL 54, GB29

Figure 2. Murphy receiving acupuncture (needles in local bladder points and Bai Hui) and laser acupuncture (using the Pointer Plus handheld device at BL40).

Figure 3. Murphy receiving acupuncture needles in local bladder points, BL 11 and Bai Hui.

Local bladder points (T11-T13)—needle and electroacupuncture

Laser and electroacupuncture combination (TENS) BL40; ST 36; BL 60; BL 67, SP6, LI 11—as the patient preferred to stand and not be tightly restrained during therapy, it was decided that a needle-less option would be more suitable for these areas with thinner skin and in the distal limb.

Needle and electroacupuncture—trigger points in trapezius and latissimus dorsi muscle on right side and therapeutic ultrasound therapy.

How is Murphy doing today?

Murphy responded immediately and stopped gabapentin and meloxicam after the first treatment from that point onwards. He had a complete resolution of clinical signs at the 4th week session. He displays even weight bearing on both HL at rest, walks (slow and brisk), circling and the kyphosis has resolved.

He is currently on a weight loss plan and a pentosan polysulfate (Cartrophen, Biopharm) injection course. It has been recommended that Murphy continue with regular acupuncture sessions (fortnightly- monthly) depending on recurrence of symptoms.

Conclusion

Murphy is an example of an older patient with a few orthopaedic problems, having previously had a hemilaminectomy in his cervical area, evidence of early arthritis in his hips and spondylosis T12-T13.

It is common in these patients to have pain on palpation with muscular hypercontraction elsewhere, associated with an over-compensation for the weaker limb(s), back and muscle atrophy. Addressing this referred pain is just as important as treating the condition, to balance and strengthen all the muscles of the patient.

Figure 4. Pointer Plus (TENS and laser acupuncture handheld device) and Ultravet Therapeutic Ultrasound

Acupuncture and its different modalities are very useful in addressing the pain associated with IVDD. Electroacupuncture can be used specifically to target muscles to prevent disuse atrophy as well reduce spasticity[9].

Laser and TENS acupuncture are viable options for patients that are adverse to needles, in patients that may be non compliant to sitting still for 20 minutes or in areas where needling is difficult.

Acupuncture is a useful additional treatment modality for rehabilitation and pain management of patients with intervertebral disc disease.

Figure 5. Intelect Vet Machine (Electrotherapy, Therapeutic Ultrasound and Low Level Laser) and Seirin Acupuncture needles.

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i. Electroacupuncture involves application of a gentle electrical current to several acupuncture points to produce a more vigorous and prolonged stimulation to the needles. The electrical current can be applied to the acupuncture needles at either a high or low frequency, to produce different effects on systemic neuromodulation (10).

i. Laser (photobiostimulation), instead of a needle, is used to stimulate the acupuncture points (14).

Congenital peripheral vestibular disease in a Domestic Short Haired cat

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A 21-day-old female intact domestic shorthaired kitten presented to a kitten nursery operated by a private shelter in the Northeast United States. An intake examination was performed including a Wood's Lamp test for dermatophytosis and an in-house SNAP FIV/FeLV test which were both negative. She was scheduled to begin vaccinations and deworming when she reached four weeks of age.

The kitten weighed 127g, had nasal congestion with no appreciable nasal discharge, and had severe, bilateral chemosis. An intention tremor was noted once. She was started on Terramycin® (oxytetracycline) ophthalmic ointment BID x 10 days and doxycycline at a 10mg/kg PO SID dose x 10 days for a presumed upper respiratory infection (URI). She was bottle fed feline milk replacer, was eating well, and was euhydrated. During the course of treating the kitten's URI, warm compresses were applied to the eyelids and nasal aspiration via bulb syringe was utilised as needed. The patient developed bilateral mucopurulent ocular discharge and when the chemosis subsided it became apparent that her eyes were phthisical and that she was blind bilaterally.

At 34-days-old she was eating canned kitten food on her own with milk replacer supplementation. The URI was clinically resolved. A left head tilt and circling to the left was observed. Occasionally, she demonstrated head excursions where she intermittently tilted her head to the left, back, and moved her head around with exaggerated movements. No obvious aural discharge was noted, but otitis media and/or interna were suspected so the patient was placed on enrofloxacin at a 5mg/kg PO SID dose x 10 days. Three days later she developed significant mucopurulent nasal discharge. Nebulisation with gentamicin ophthalmic solution in distilled water BID x 3 days and doxycycline at a 10mg/kg PO SID dose x 14 days were initiated. At 42-days-old the left eye developed purulent discharge and erythromycin ophthalmic ointment BID x 7 days was added. Her appetite remained hearty as she continued to wean off of milk replacer. At 50-days-old the mucopurulent nasal discharge continued, the right eye developed partial ankyloblepharon, and the left ocular discharge resolved. The left head tilt and occasional circling were still present and a clindamycin course at a 10mg/kg PO SID dose x14 days was added to allow for a broader spectrum of bacterial coverage. A nasal swab was submitted for culture and sensitivity testing and returned as an *Enterococcus* species and an *E. coli* species which were both sensitive to amoxicillin. While the results were pending, the nasal discharge decreased dramatically in volume and became primarily serous in nature. The kitten was started on amoxicillin at a 22mg/kg PO TID dose x 10 days and was sent to a foster home for continued care where the URI resolved. The patient returned to the kitten nursery for medical rechecks and scheduled vaccinations as needed.

At 5 months-of-age the patient returned to the kitten nursery from her foster home for boarding. She had an excellent appetite, was playful, and her phthisical eyes were quiet. She displayed no evidence of URI and while no head tilt or circling was appreciated at the time of examination, her head excursions were still present. The deciduous maxillary canines were still intact. She developed diarrhoea with hematochezia so she was dewormed with fenbendazole at a 50mg/kg PO SID dose x 5 days and was given Bene-bac gel®(prebiotics and naturally occurring microorganisms) 0.5g PO q3 days for 6 days. A Canine Parvovirus Antigen Test (in-house Parvo SNAP test) was performed to screen for Panleukopenia and was negative. She was sent back to the foster home and the diarrhoea resolved.

At 5.5 months-of-age the kitten presented to the shelter's adoption center for a recheck, as the kitten nursery was no longer operational for the season. The foster family reported that they recently had a litter of kittens in the home that tested positive on an in-house Parvo SNAP test and were treated for Panleukopenia. The kitten was asymptomatic and up to date on FVRCP vaccinations, but because of the possible exposure an in-house Parvo SNAP test was performed and was negative. It was also determined that she was deaf because she repeatedly did not react to loud sounds. She returned to the foster home to complete a 14-day quarantine for panleukopenia monitoring.

After the quarantine, the kitten returned to the shelter's hospital for bilateral enucleations to prevent any future complications associated with the previous ocular

Used with permission.

infections, ovariohysterectomy, retained deciduous teeth extractions, a nasopharyngeal polyp check, skull radiographs, and an otoscopic examination. Pre-operative I-STAT Chem 8 and PCV/total solids were within normal limits. A repeat in house FIV/FeLV SNAP test was negative. The procedures were performed, and she recovered uneventfully.

The polyp check and otoscopic examination were unremarkable and skull radiographs revealed a subjectively mildly thickened left bulla so a course of enrofloxacin at a 5mg/kg PO SID dose x 6 weeks was initiated for possible otitis media/interna. She developed moderate mucoid nasal discharge with moderate audible congestion so the enrofloxacin she was already prescribed was also used to treat the URI and nebulisation was added. The kitten went back to the foster home and the URI resolved quickly.

Six weeks later a surgical consultation was pursued to further assess the ongoing left head tilt and determine whether any surgical interventions such as a ventral bulla osteotomy were indicated. It was determined that a true head tilt was not present, although when stimulated the patient did tilt her head to the left and caudally, and she moved her head around in both directions with exaggerated motions (see Figure 1).

When gently wrapped in a towel and not stimulated the head remained straight. No circling was observed. The prior skull radiographs were reviewed by the surgeon and the left bulla was found to be unremarkable and not thickened as thought previously. A repeat otoscopic exam by the surgeon was not consistent with middle ear disease.

An internal medicine consultation was also pursued, which included input from a neurologist at a neighboring specialty hospital. The internist documented similar findings on examination but noted that the kitten would preferentially circle to the left. An intention tremor with a dorsal and ventral head bob was noted when the kitten was offered a treat. Despite this, she continued to be playful and was observed playing with a toy in her cage. A biochemistry panel, CBC, and Toxoplasmosis titers (IgG and IgM) were run and were all normal. An MRI and CSF tap were recommended, but they were unable to be pursued within the means of the shelter.

Based on the neurologic anatomic localisation, the primary differential diagnosis was bilateral peripheral vestibular disease, due to a presumptive congenital vestibular syndrome with the possibility that there could be another congenital anomaly in the brain contributing to clinical signs. The kitten was started on cefpodoxime at a 10mg/kg PO SID dose for 8-12 weeks for penetration into the CNS to empirically treat otitis interna.

Omeprazole at a 1mg/kg PO SID dose was recommended to decrease CSF production, in the event that intracranial hypertension was causing intermittent cervical dorsal hyperextension and/or if she had a congenital condition such as hydrocephalus.

Given that the kitten had a good quality of life, was comfortable, and was well adjusted, she was put up for adoption with the conditions that she was to be adopted by a dedicated family without very young children since she was blind and deaf and that the family consider taking her to a neurologist for further evaluation. Very shortly after she was made available for adoption, the kitten was happily welcomed into a family who was aware of her medical history and was dedicated to caring for her.

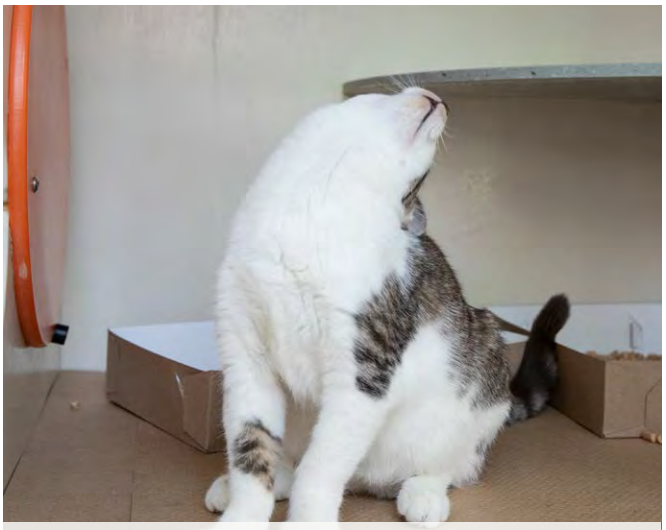

Figure 1. The kitten displaying exaggerated head movements. Used with permission.

Discussion

Many of the medications used in this patient were prescribed in an off-label fashion. When weighing the benefits and risks of use, it was deemed that aggressive treatment with them outweighed the possible risks as young kittens can decompensate rapidly without timely intervention.

The vestibular system is responsible for maintaining balance and it is comprised of the central vestibular system located in the brain stem and the peripheral vestibular system which is located in the inner ear. It stabilises the eyes in space so that the visual image is maintained, and it dictates the position of the head in space so that the body remains stable[1]. Signs of vestibular disease usually include rolling, falling, head tilts, circling, nystagmus, positional strabismus, and asymmetrical nystagmus. Central vestibular disease is associated with brainstem lesions which present as postural reaction deficits, such as paresis or loss of general proprioception. Central involvement can also manifest as altered mentation and cranial nerve V-XII deficits. Vertical nystagmus is usually associated with central vestibular disease. Abnormalities of the peripheral vestibular system occur due to disorders of the inner and middle ear. While middle ear disease alone doesn't produce vestibular abnormalities, usually middle ear disease progresses to inner ear disease which causes peripheral vestibular signs[1]. A distinguishing feature of peripheral vestibular disease is that a patient's strength or general proprioception is not diminished. Bilateral peripheral vestibular disease can occur occasionally, and it is more commonly seen in cats. It is characterized by lack of a head tilt or nystagmus, and an absent oculocephalic reflex (physiologic nystagmus or the 'doll's eyes' reflex). These patients may crouch low and fall to both sides and they usually have wide lateral head excursions from side to side to attempt to stabilize visual fixation[2].

Neurological examination usually leads to lesion localisation of either the peripheral or central vestibular system. Skull radiographs, otoscopic examination, myringotomy, bulla ultrasound, MRI, CSF analysis, and CT are all modalities that can aid in further assessing vestibular disease. Brain stem auditory evoked responses (BAER) testing can be utilised to assess auditory function[2].

Causes of central vestibular disease include congenital malformations, hypothyroidism, thiamine deficiency, neoplasia, infectious and inflammatory diseases (such as feline infectious peritonitis, toxoplasmosis, and cryptococcus), aberrant parasitic migration, brainstem trauma, metronidazole toxicity, and cerebrovascular disease[1,2]. Causes of peripheral vestibular disease include congenital malformations, hypothyroidism, neoplasia, otitis media/interna, naso and otopharyngeal polyps, idiopathic vestibular disease, inner ear trauma, and ototoxicity[1, 2].

Animals with congenital vestibular disease usually have clinical signs that are apparent at or soon after birth and some signs may resolve spontaneously. Affected patients can usually compensate for the dysfunction. Deafness or other congenital malformations are variably associated with this type of vestibular disease[2]. In this kitten's case, it was reasonably assumed that she was deaf since birth but given the challenges of diagnosing deafness in neonates and very young kittens, it was not appreciated until she was older. A BAER was not performed prior to her adoption, but it could be done for confirmatory purposes. An intention tremor was noted only twice in this kitten's medical history, and it could be supportive of a brainstem lesion such as cerebellar hypoplasia. As it was only seen twice, it is unlikely to be cerebellar hypoplasia or is a very mild case of it. Nystagmus, which can still be present in blind animals, was never noted on exam and given she was blind it was not possible to test her oculocephalic reflex. Despite the observed tendency to sometimes circle to the left, her deafness, lack of head tilt, and pronounced head excursions are most consistent with bilateral peripheral vestibular disease of a congenital nature. Further diagnostics such as advanced imaging with CSF analysis would be useful in determining and confirming contributing factors to her presentation.

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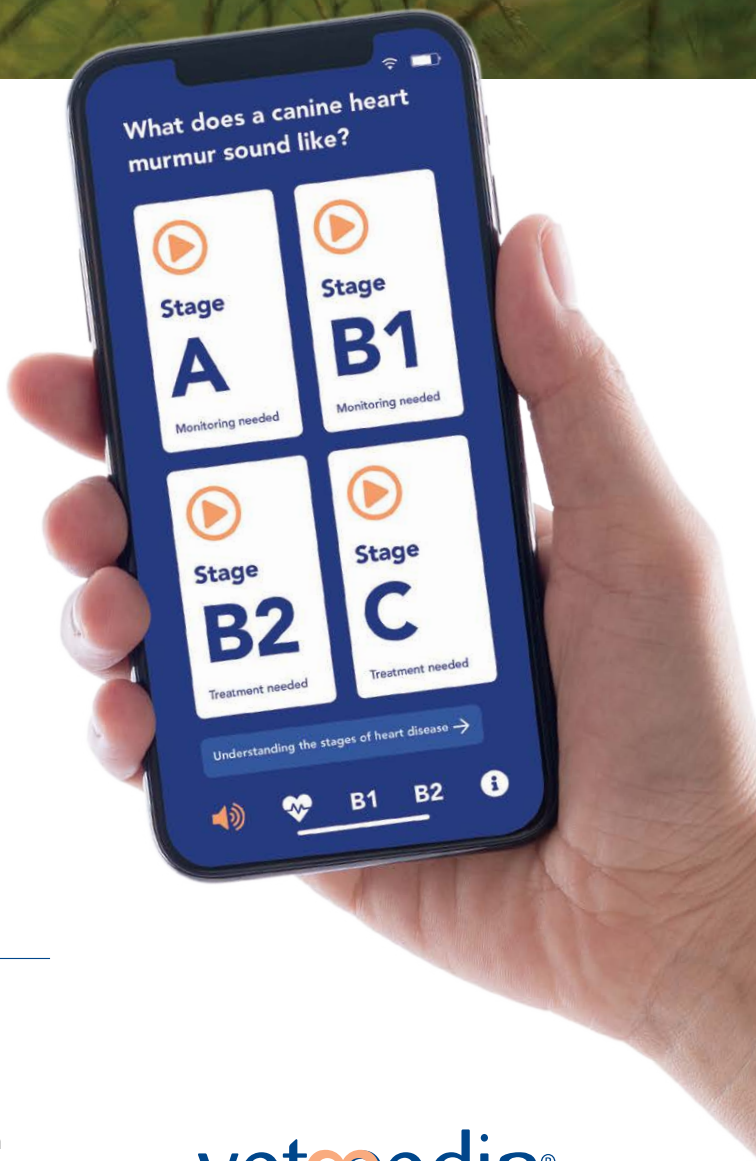

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Unusual mouth and tongue lesions in sheep in Barossa Valley area South Australia

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Introduction

Mouth lesions and ulcerations have been detected in sheep at routine inspections from time to time and these are an important differential diagnosis for some serious exotic diseases such as Foot and mouth disease and Bluetongue. A study in Victoria *(DPI Victoria 2006)* described 27 different conditions that were associated with mainly ulcerations or nodules, but lesions on the tongue of sheep are rarely reported.

In this case, the producer reported that one of his three mobs of merino ewes with lambs at foot appeared to be 'frothing at the mouth', one ewe had died and ~ 20% of the flock appeared to be affected, he was concerned about losing more animals and requested an investigation. Since serious exotic diseases were a possibility, PIRSA subsidised the investigation and laboratory costs.

Serious exotic diseases were ruled out and a diagnosis made of an unusual parasite- *Gongylonema* spp. The affected ewes appeared to improve after treatment with antibiotics and anti-parasitic drench, and no further cases were reported.

History

An experienced sheep producer contacted me in late June 2018 to report that he had noticed sheep frothing at mouth the day before. Approximately 40/240 3-year-old ewes with lambs at foot were affected and occasional cases in two other mobs of ewes were seen. The ewes had been vaccinated with '3 in 1' (a commonly used vaccine against two clostridial diseases and 'Cheesy gland' e.g. Glanvac® 3—Zoetis) prior to joining, and possibly treated for internal parasites with an oral ivermectin drench. The affected ewes seemed to be in poorer body condition that other ewes and appeared to have profuse white froth and saliva dripping from the mouth. The sheep were being fed some barley as a trail feed, some supplementary hay and pasture was of poor quality and fairly short with moderate faecal contamination. No obvious weeds, toxins or heavy metal contamination was apparent and there was plentiful water available.

Ten affected ewes were examined, all were bright and alert but with poor body condition (1-2 / 5), a moderate pyrexia (39.3- 40.1°C), and they all had white frothy saliva in the mouth but there were no respiratory signs or coughing. One sheep had multifocal, coalescing, discoloured, yellow adhesions across the tongue but all the others tongues were normal with no evidence of blisters, wounds or similar. There was no lameness or inflammation in feet, no interdigital lesions or coronary band lesions. One sheep was sacrificed for a full post mortem examination and a full range of samples submitted. There were no gross lesions on the tongue, oesophagus or abomasum of this animal and no parasites were observed in the intestines.

Most severely affected sheep were treated with Alamycin 1 mL/10kg (300 mg/ mL oxytetracycline Norbrook Laboratories) and Metacam 1 mL/20kg (20mg/ mL

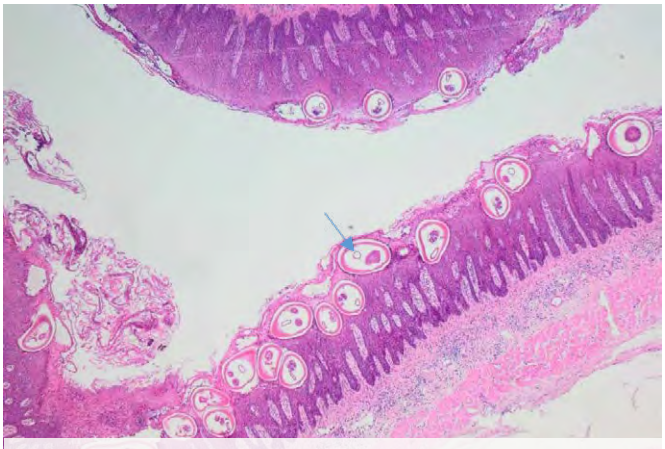

Figure 1. Cross section of worms seen in tongue tissue (blue arrow).

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Meloxicam, Boehringer Ingelheim) and faeces for a faecal egg count were collected. I advised the producer to move the sheep to a fresh paddock and offer hay as well as barley to rule out any chance of rumen acidosis as cause.

The affected sheep appeared to recover, and there were no further cases reported.

Results from Laboratory tests

The final diagnosis from histopathology was alimentary nematodiasis. Foot and mouth disease, vesicular stomatitis and Bluetongue tested negative from samples sent to AAHL. Further detail from the report is quoted below:

'The most significant findings are seen within the tongue and oesophagus of this sheep: there is evidence of a mucosal hyperplasia, parakeratosis, erosion and ulceration and mixed neutrophilic (suppurative) and eosinophilic inflammation associated with numerous nematode parasites that are burrowing through the mucosal squamous epithelium. This is likely to represent a severe infection with *Gongylonema spp.*

Gongylonema is a rare spirurid nematode parasite of sheep and goats (and various other mammalian species) found worldwide. *G. pulchrum* (worm is found within the tissues of the oesophagus, where the parasite burrows through the cornified layer of the mucosa forming serpiginous tracks. The worm has an indirect life cycle, with dung beetles or other insects (e.g. cockroaches) acting as intermediate hosts. The final host becomes infected

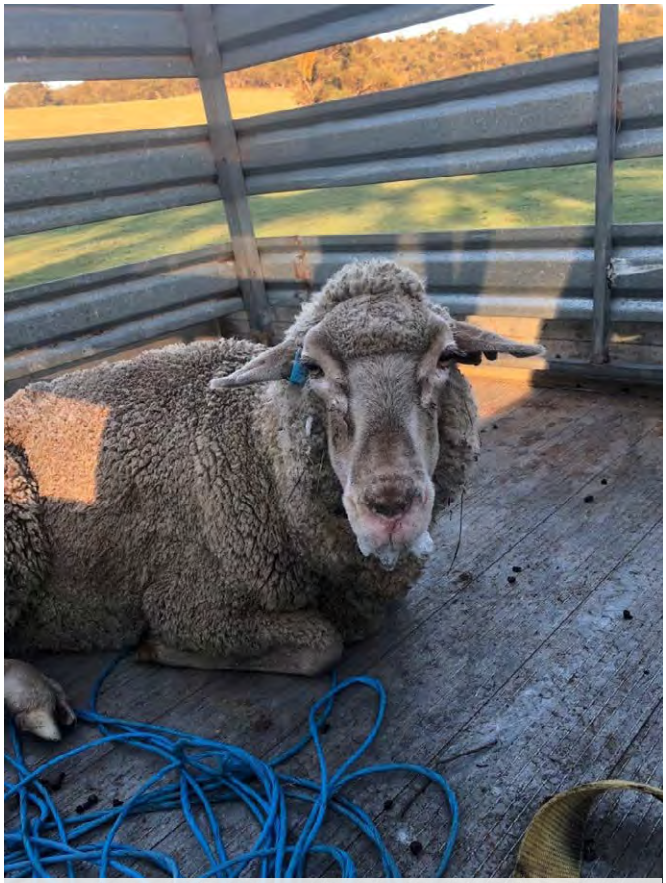

Figure 2. A salivating, unhappy ewe.

Figure 3. Heavily grazed pasture.

Figure 4. .

when ingesting carrier beetles or directly (e.g. on pasture), or indirectly through contaminated feed. This parasite is normally an incidental finding and of little clinical significance. I have occasionally seen one or two worms as an incidental finding in the oesophagus of ruminants, without any obvious inflammatory response. However, in this case, there are large numbers of worms within the epithelium and their presence is clearly provoking a severe inflammatory reaction, complicated by secondary bacterial infection.

It could be that there is a hypersensitivity reaction stimulated by parasite migration through the mucosa. The upper alimentary lesions are presumably responsible for the hypersalivation and perhaps the poor condition (e.g. pain associated with feeding or swallowing?) Mild pyrexia might be due to the secondary bacterial infection.'

The faecal worm egg count was low—185 eggs per gram.

Interpretation

This is a most unusual case and it is difficult to find cases reported in Australian literature of this parasite. Nieberle & Cohrs (1966) describe *G pulchrum* as 'a whitish threadlike filarial worm 4- 14.5 cm long found free in the oesophagus or coiled in the epithelium, apparently without causing

any disturbances'. References from Iran in experimental infections report that mild inflammation may occur on the tongue and oesophagus approximately 10 days following ingestion.

Discussion

This case could easily have been an emerging, new or an exotic disease, and oral lesions in sheep are very rarely reported in SA.

Occasionally producers report cases of 'vomiting sheep' where a greenish liquid is seen dribbling from the mouth, and under investigation, this may result from dentition issues, foreign bodies, drenching injuries, acidosis or undetermined cause. However, in this case obvious and dramatic symptoms were observed and it is pleasing that the producer sought professional advice immediately.

Seasonal conditions in SA have been difficult with reduced pasture growth in autumn and winter leading to livestock under nutritional stress. In this case ewes under maximum nutritional demand may have been grazing closer to the ground and consuming intermediate hosts (dung beetles etc. or larvae directly), where in better seasons they may not have.

Treatment of the affected sheep with antibiotics, anti inflammatories and anthelmintics appears to have been effective in this case. However, 2 months later some ewes from the same property were found dead with lesions attributable to pneumonia, hypocalcaemia and intestinal parasitism.

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Mycoplasma hyopneumoniae infection in a pig

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Introduction

Respiratory disease is a common occurrence in commercial pig production and of major concern for animal health and economic losses due to reduced daily weight gain, reduced feed efficiency, medication costs and fatality[1].

The host-pathogen-environment axis is very important to consider when diagnosing and treating respiratory diseases. Housing, management systems e.g. All-In/All-Out versus continuous flow[2], and vaccination all play a crucial role in how respiratory diseases manifest on farm.

Mycoplasma hyopneumoniae is one of the most common respiratory pathogens across the swine industry, with high morbidity and low mortality[1]. It causes primary respiratory diseases but it is also related with immunosuppression and a breakdown in host defences predisposing pigs to secondary respiratory infections. The term enzootic pneumonia is given to the syndrome when a pig infected with *Mycoplasma hyopneumoniae* develops a secondary infection with *Pasteurella multocida* Type A[3]. However, secondary infections can occur with a range of respiratory pathogens, the most common being *Actinobacillus pleuropneumoniae, Pasteurella multocida, Streptococcus suis, Haemophilus parasuis and Bordetella bronchiseptica*[1].

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Typical gross pathological findings of an uncomplicated *Mycoplasma hyopneumoniae* infection include dark purple to grey rubbery consolidation of cranioventral aspect of lung lobes with catarrhal exudate[1]. Histologically, it is characterized by an interstitial pneumonia with infiltration of lymphocytes and macrophages cuffing airways and blood vessels. As time progresses there is hyperplasia of alveolar epithelium and submucosal glands. When secondary infection occurs, such as in enzootic pneumonia, extensive exudate in the alveoli and airways can be observed and the inflammation becomes more suppurative with bacteria being present[1].

Case report

A 3-year-old, large white cross sow presented with fever, increased respiratory rate, dyspnoea, anorexia and agalactia three days after an uneventful farrowing. The animal did not respond to antibiotic therapy (Tylosin, Oxytetracycline and Ceftiofur crystalline free acid). Other animals in the shed had started showing respiratory signs such as coughing and increased respiratory rate.

Since the animal was not responding to treatment it was euthanized on welfare grounds. At the post-mortem examination, a marked, diffuse, interstitial pneumonia with mild fibrinous pleural adhesion and pleurisy was diagnosed (Figure 1). The lungs were characterized by multifocal, marked areas of consolidation throughout all lobes, most severe in the cranioventral region. There was a moderate amount of white froth throughout the trachea and main stem bronchi. There was also a small amount of yellow to white, semi-opaque, thick fluid in the thoracic cavity and some fibrinous pleural adhesions to the lung.

Histologically, surrounding bronchi, bronchioles, blood vessels and infiltrating the submucosal space there were aggregates of lymphocytes, plasma cells, macrophages with fewer neutrophils (Figure 2). Bronchus-associated lymphoid tissue was hyperplastic. Bronchial and bronchiolar epithelium were lost or attenuated and devoid of cilia. The lumina of bronchi and bronchioles contained an amphophilic material (mucus) admixed with sloughed epithelial cells and degenerate inflammatory cells.

PCR from fresh lung tissue was positive for *Mycoplasma hyopneumoniae* and negative for Porcine Circovirus Type 2. There was no growth in aerobic bacterial culture.

Discussion

The necropsy and histological findings indicate that there was an interstitial pneumonia with pulmonary oedema. *Mycoplasma hyopneumoniae* was detected by PCR in the lung, however bacterial culture and Porcine Circovirus Type 2 PCR were negative. In this case the gross and histological findings cannot solely be explained by *Mycoplasma hyopneumoniae* infection and the lesions are more likely to be attributed to a secondary bacterial proliferation. The lack of bacterial growth in aerobic culture may be related to the antibiotic therapy.

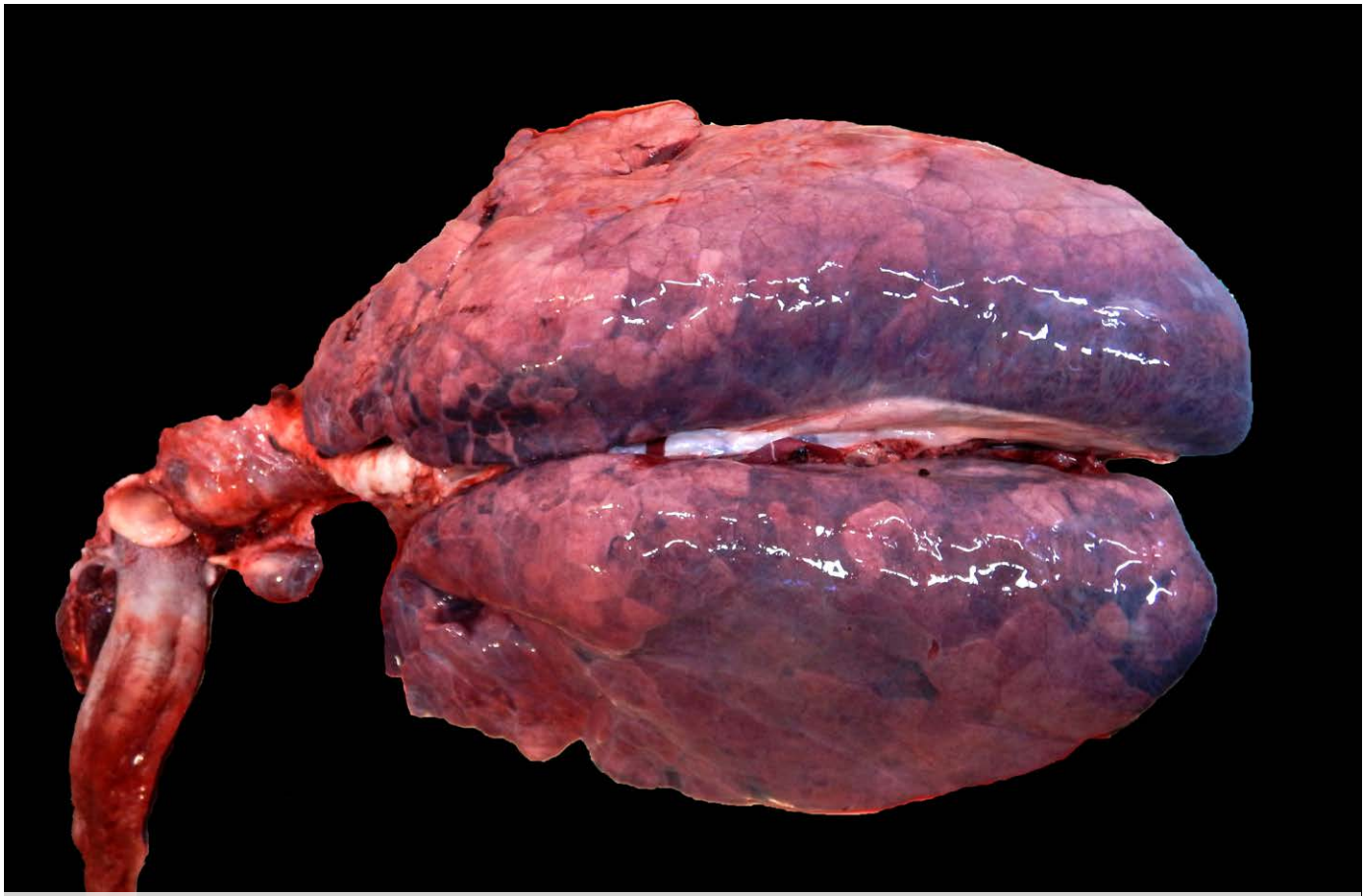

Figure 1. Marked, diffuse, interstitial pneumonia with mild fibrinous pleural adhesion and pleurisy.

Transmission of Mycoplasma hyopneumoniae is usually by direct contact from pigs or by aerosol over short distances. It adheres to ciliated cells in the trachea and bronchi[4]. This binding results in ciliostasis, clumping and loss of cilia, with loss of epithelial and bronchial goblet cells[5]. Mycoplasma cell membranes contain superantigens that induce polyclonal proliferation of lymphocytes aggregating around airways and blood vessels. They also suppress the alveolar macrophage phagocytic response, rendering pigs more susceptible to secondary bacterial pathogens[4].

Consideration of the host-pathogen-environment axis is crucial to managing Mycoplasma infections in farms. Mycoplasma is an insidious pathogen that can begin to produce clinical signs within two weeks after infection. In herds where Mycoplasma is endemic piglets are usually infected within the first three to four weeks of life unless they are weaned early[2]. This eventually spreads throughout the grower and finisher population and causes a chronic non-productive cough and reduced weight gain[1]. Given that older pigs are more likely to be infected due to increased chance of exposure, segregating age groups in an all-in/all-out system is an important factor in reducing horizontal transmission and therefore increasing performance[2]. It also decreases the stress on animals by not mixing different age groups together. Regardless of the management system in place it is also ideal to reduce any environmental factors that can potentiate respiratory diseases (i.e. good ventilation, reduce dust). Host factors can be manipulated to reduce infection by mycoplasma vaccination, and in Australia an inactivated vaccine is commercially available. While the vaccination does not give impervious protection against mycoplasma it has been shown to reduce clinical signs and even improve daily weight gain[6].

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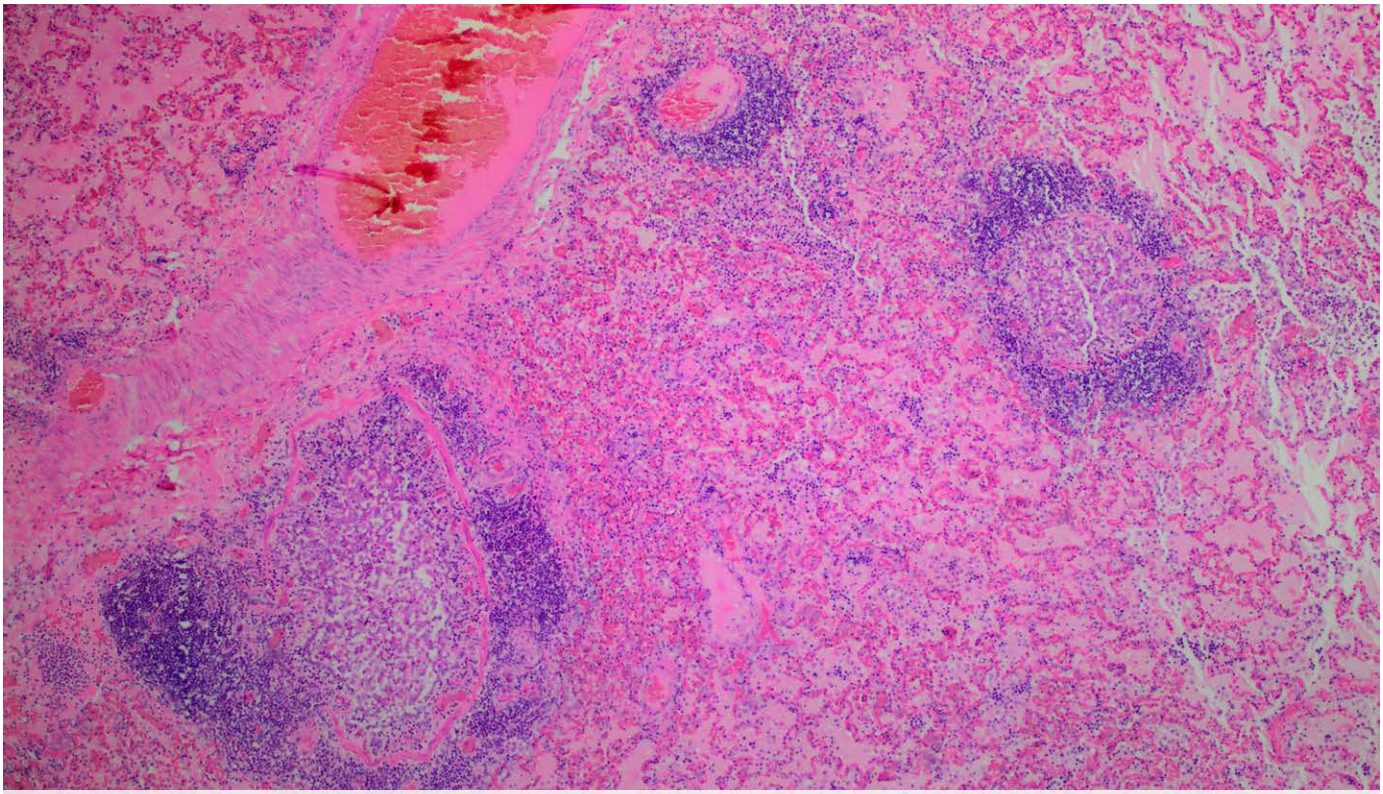

Figure 2. Histologically, surrounding bronchi, bronchioles, blood vessels and infiltrating the submucosal space there were aggregates of lymphocytes, plasma cells, macrophages with fewer neutrophils.

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Tibia fracture repair in an alpaca cria

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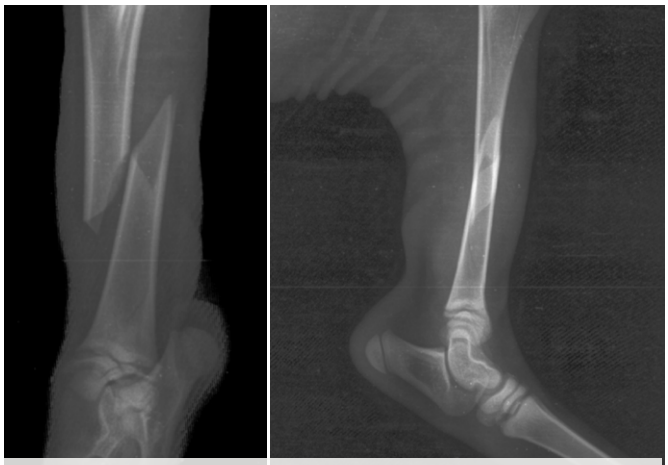

Figure 1. Radiograph of left tibia fracture.
Image: Dr Phil Sharman/Casino Veterinary Clinic.

I was fortunate to observe a fascinating surgery during my pre-clinical placement at Casino Veterinary Clinic, NSW. This article is based on my observations of the surgery, and the clinical record written by Dr Phil Sharman.

'Stallone', the 16-day-old alpaca cria (6.8kg) was found in the paddock with his left leg 'swinging in the breeze' and was presented to Casino Veterinary Clinic. Upon palpation, a fractured tibia was suspected. *The Glove Box Guide to Alpacas* was consulted to determine drug dosages for alpacas and Stallone was administered 0.12mL butorphanol IM (10mg/mL) and 0.2mL meloxicam IM (5mg/mL) (Vaughan 2017). The limb was radiographed, which confirmed a spiral mid shaft tibial fracture (Figure 1).

The limb was splinted while the owner considered the options: plating in-house, casting in-house, referral, or euthanasia.

Referral option was dependant on whether the nearest specialist hospital (in Queensland) would operate on an alpaca cria. Of the in-house surgical options, internal fixation was the most desirable due to improved chance of healing with internal fixation. In contrast, casting the limb posed the risk of non-union, as immobilising Stallone's stifle joint would be difficult.

The owner decided to give Stallone a chance and opted for the internal fixation. I had the enjoyable task of holding Stallone until surgery time (Figure 2).

At 5pm on Friday afternoon, preparation to plate Stallone's tibia started! The right mid-jugular vein was clipped and a 20G catheter placed and intravenous fluids (Hartmann's Solution) started at 60mL/hour. Stallone was induced with 1.2mL Alfaxan®; he was still a bit light for intubation so was topped up with 0.4mL Alfaxan®.

Intubation proved tricky due to the shape of the larynx; after some fiddling, a size 4 ET tube was inserted. Isoflurane was started at 3%, then turned down to 2%. The left hind limb was clipped from fetlock to inguinal area, scrubbed and the lower limb wrapped in 'Vetwrap'. The right hind limb was bandaged loosely in flexion to keep it out of the surgical field—alpaca cria are very 'leggy'! (Figure 3).

Dr Phil Sharman and Dr Laif Mearns scrubbed in for

Figure 2. DVM3 Pre-clinical Placement Student Frances Zewe holds Stallone prior to surgery to plate his fractured tibia.
Image: Shania Carter/Casino Veterinary Clinic.

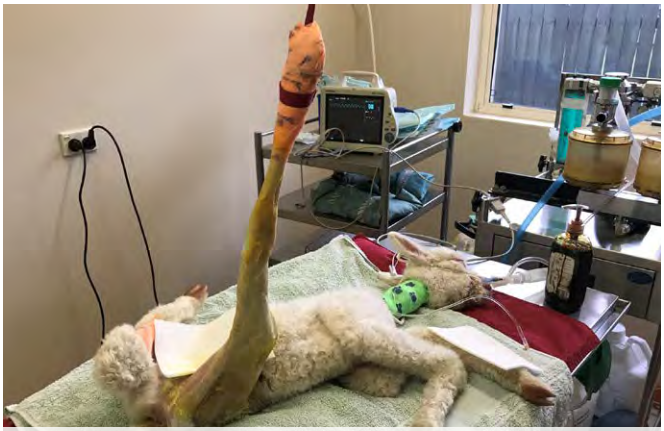

Figure 3. Stallone prepped for surgery.
Image: Frances Zewe.

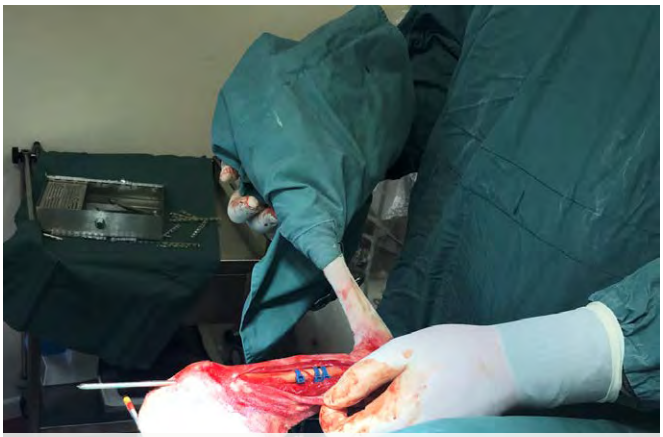

Figure 4. The tibia is stabilised with 'Bonebinders' and an intramedullary pin inserted.
Image: Frances Zewe.

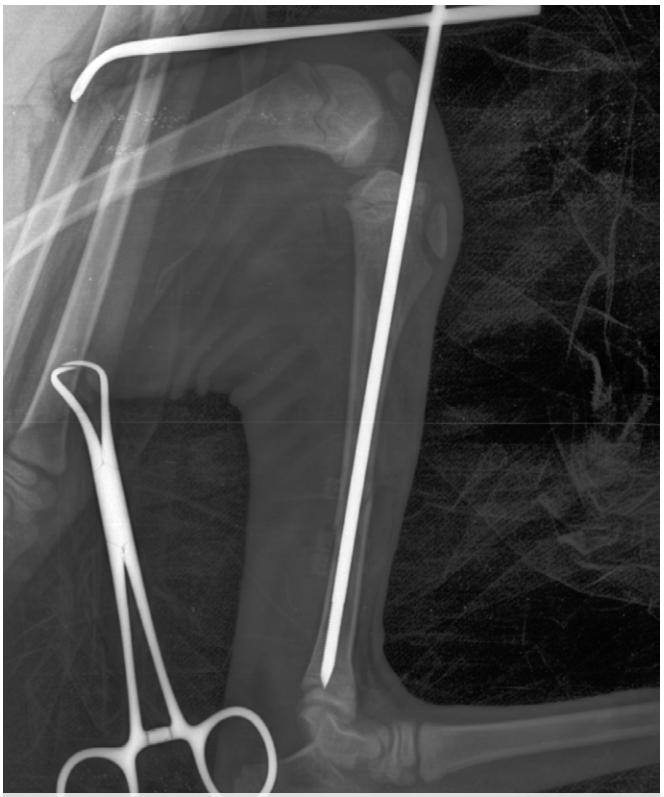

Figure 5. Radiograph to confirm placement of intramedullary pin.
Image: Dr Phil Sharman/Casino Veterinary Clinic.

Figure 6. Stallone recovering under the care of Dr Laif Mearns (left) and Dr Phil Sharman.
Image: Frances Zewe.

Figure 7. The morning after surgery.
Image: Dr Phil Sharman/Casino Veterinary Clinic.

Figure 8. Stallone gingerly bears weight on his left hind limb.
Image: Dr Laif Mearns/Casino Veterinary Clinic.

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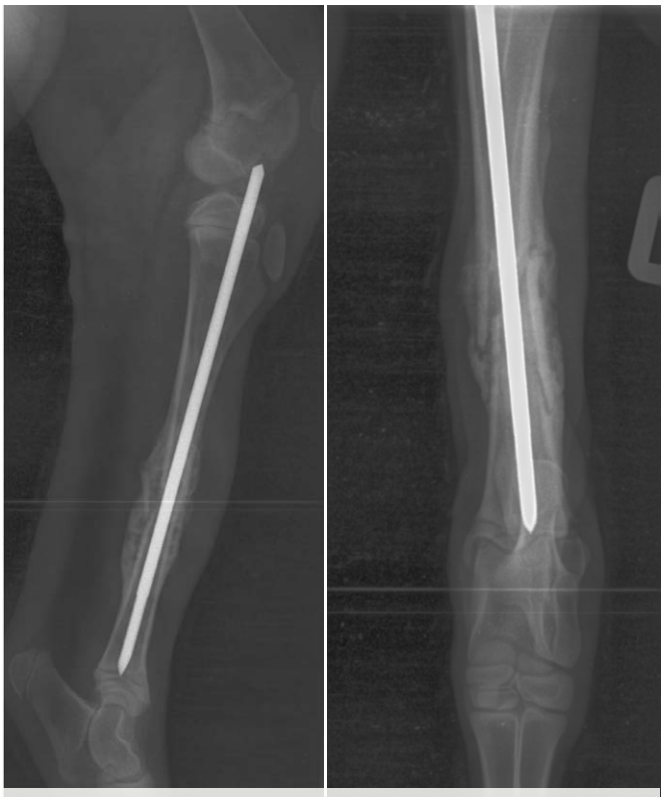

Figure 9. Radiograph of Stallone's hind limb to assess the fracture site. Image: Dr Phil Sharman/Casino Veterinary Clinic.

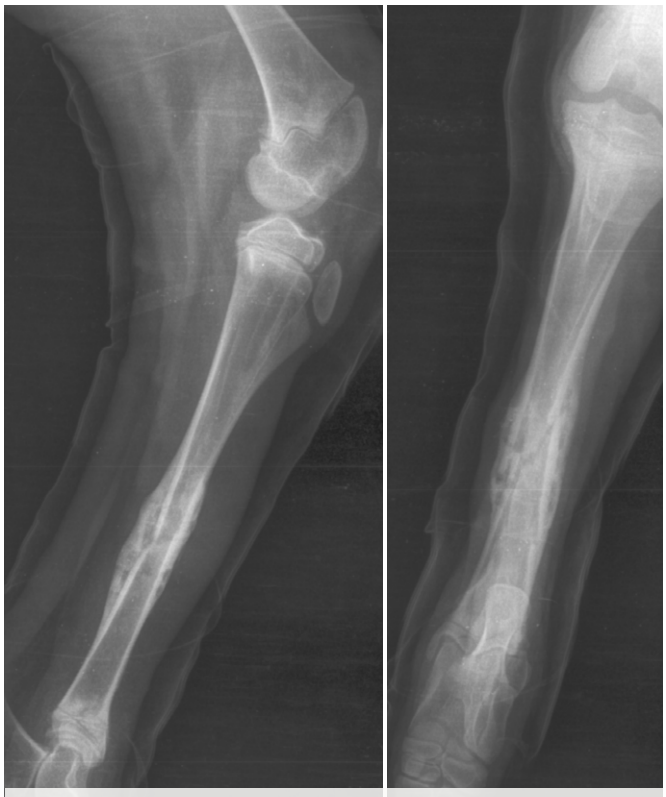

Figure 10. Radiograph of Stallone's hind limb after removal of the pin. Image: Dr Phil Sharman/Casino Veterinary Clinic.

surgery. An incision was made along the medial tibia and a periosteal elevator was used to elevate tissues and reveal the fracture. The first job was to re-align the fracture, however, while attempting to do this, a long vertical shaft of lateral distal tibia fractured. This added complexity to the situation and meant a new plan for Stallone had to be devised.

Plan A was to seat a cerclage wire below the bone cortical level to allow continuation of the plate and screws, but the surgeons were not able to get the bone fragment stable enough. The use of lag screws was ruled out as they would interfere with plate placement.

An alternative (Plan B) involved pinning, rather than plating, the bone. The lower fragment was stabilised with 2 x 3.5mm 'Bonebinders' (absorbable PDS cable ties) and a 5/32 intramedullary threaded/trocar pin was placed in retrograde fashion. The fracture was re-aligned and clamped with bone holders, and a third 3.5mm Bonebinder placed across the initial main fracture (Figure 4).

The intramedullary pin was seated into the distal tibial segment, then rotated a further 4 turns. The wound was flushed with soluble penicillin. Tissues were closed with 0 PDS simple continuous suture and the skin closed with 0 Nylon simple interrupted suture.

Radiographs of the repair were taken prior to the pin being cut as short as possible (Figure 5). The repair was stable with good alignment. Impingement of the stifle joint in full extension was noted but this was not problematic with a normal range of motion. 1mL Engemycin IM (oxytetracycline 100mg/mL) and 0.07mL Temvet IM (buprenorphine 300µg/mL) were administered and Stallone moved to the recovery room.

RECOVERY (December 14)—6pm onwards.

Early in recovery Stallone was hypothermic (rectal temperature 35.6°C) and unable to maintain oxygen saturation on inspired air; he was left on oxygen and warmed with a forced air warming blanket and a hot water bottle on top of blankets (Figure 6).

Phil was on call that night and had a busy night with Stallone. The primary concern was ensuring adequate ventilation; the ET tube was kept in as long as practical with positive pressure ventilation applied in response to shallow respiration.

After the ET tube was removed Stallone was having difficulty inspiring air as his nostrils closed on inhalation and he was unable to mouth breath. Nasal catheter insertion was attempted but the resulting struggle resulted in the drip being disconnected. After re-connecting the drip and delivering fly by oxygen he settled and was breathing well by himself.

THE DAY AFTER (December 15)—1-day post-surgery—sent home.

After a 'rocky' night Stallone was ready to go home (Figure 7). He was sent home with 5mL of Engemycin® 100 (1mL daily IM) and 10mL meloxicam dog oral (1mL daily PO) and the instruction to keep him confined on a non-slip

surface. The owner was advised to remove the bandage in 5 days (or change earlier if soiled) and to have the sutures checked in 10 days.

FOLLOW UP 1: DECEMBER 2018 (December 24)—10 days post-surgery—suture check.

Stallone visited the clinic for a check up on Christmas Eve. The sutures needed a little more time, so the owner was advised to remove at home, after Boxing Day. Stallone was gingerly bearing weight and otherwise healthy.

FOLLOW UP 2: JANUARY 2019 (January 10)—27 days post-surgery—radiographs of fracture site.

The new year arrived and so did Stallone's next check-up. He was admitted on 10th January for radiographs which revealed a firm callus forming at the site (Figure 9). The plan was to send Stallone home to further confinement for two weeks then remove the pin.

FOLLOW UP 3: JANUARY 2019 (January 24)—41 days post-surgery—pin removal.

Two weeks later Stallone made another trip to the now-familiar Casino Vet Clinic. Radiographs revealed a still-visible fracture line, but an extensive, firm callus (Figure 10). Stallone was prepared for surgery—he now weighed 11.7 kg, almost doubling his mass during his 6-week recovery period—and the end of the pin located and removed. The leg felt firm at the fracture site which was great news for Stallone and his owner.

Stallone's recovery from anaesthesia was markedly improved from his initial surgery. This improvement was attributed to the placement of an intra-nasal oxygen tube at extubation and a nurse supporting him in sternal recumbency. Stallone was given 0.5mL meloxicam SC and 0.5mL Engemycin SC (oxytetracycline 100mg/mL) and sent home with meloxicam and the advice to take it very steady for two weeks after pin removal.

FOLLOW UP 4: FEBRUARY 2019 (February 3)—10 days post-pin removal.

A phone call to the owner revealed that Stallone was slightly favouring his good leg, but otherwise doing well.

FOLLOW UP 5: July, 2019—Dr Phil Sharman reports that Stallone continues to grow well with no lameness.

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Comment courtesy of Professor Andrew Dart

Director of the Research and Clinical Training Unit and a Registered Specialist in Equine Surgery
Sydney University School of Veterinary Science

In this *Control & Therapy* series there is a nicely written article on 'Tibia Fracture Repair in an Alpaca Cria' by a third year DVM student Frances Zewe undertaking her preparatory clinical placement (PCP). PCP students are just entering their more clinically focussed years and are being exposed to what can be achieved in practice compared to their experience in the University. This often opens up a much broader perspective of what the veterinary profession can offer.

South American Camelids (SACs) are very common in Australia and were first imported in any numbers the 1980s. There was very little known about this species and there has been a steep, but rapid, learning curve. Practitioners are seeing more and more alpacas, and as values have dropped, there are many patients out there with sentimental rather than commercial value. So, as practitioners have become more comfortable with the species, and clients have economic constraints, referral is becoming less common and many procedures are being performed in practice.

SACs are quite stoic animals, generally sensible and quiet and make good orthopaedic patients. Crias in particular are light weight and have active bone so are suitable for most fixation methods. Internal fixation with plates and screws always offers the gold standard, however external fixation, pins and cerclage wires and casts with or without transfixation pins offer acceptable alternatives if they are used appropriately. These techniques have all been reported in the literature with comparisons of methods, outcomes and complications, albeit numbers are small. This case highlights some of the challenges and rewards of orthopaedic repair in alpacas.

The initial choice of open fixation of a closed tibial fracture was safe and appropriate to achieve good reduction, stability and early return to function. Venous and airway access, fluids and pre-emptive analgesia were sensible aspects of the pre-surgical plan.

The importance of addressing body temperature during medium to longer term procedures is important and were highlighted in this case.

As with many orthopaedic procedures there were challenges with the planned fixation. A Steinman pin and cerclage were chosen as an alternative approach when a third fragment of bone complicated the original fixation

technique. Lag screws and an external fixator could have offered another alternative. Alpacas wear these well. Silicone tube filled with methyl methacrylate or even 'Kneed It', a steel polymer repair compound from Bunnings, can be used as the stabilising bars. This simplifies the equipment required, the application and the costs.

The alpaca in this case developed a substantial callus which suggests the fixation was not entirely stable, however also highlights and reminds us that young animals, particularly those that are compliant, have a great potential for fracture healing. This seems to be a feature of alpacas and crias in general.

This article provides a very nice example of what can be achieved by experienced practitioners drawing on and modifying the general principles of veterinary practice and applying them to the problem presented. Increasingly our students see specialist practice in the University Hospitals. While this has a place, it is important as graduates they appreciate that specialist centres add another treatment option but it should not limit what can be achieved in private practices. It is important they are not deterred from developing the skills to offer a range of treatment options to their clients.

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Reply to: Why is it so hard to find a vet? C&T No. 5724 Issue 293

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C&T No. 5779

I hope I may be permitted to offer some comments and raise some questions about this issue from a retiree's perspective.

I graduated from Sydney University 51 years ago and after 8 years of Department of Agriculture service, started a practice in Narrandera with my wife.

My cohort graduated with full knowledge that we were embryonic with a lot to learn and that the diagnostic aids promoted at university were unrealistic in the real world. Also that our duty was to provide the best veterinary service we could to the public.

In the 1960's and 70's new practices appeared in every sizeable town in the Riverina mostly run by sole practitioners or 2-3 vet businesses—all fulltime positions and all providing after hours. All thrived but now that ageing workforce are unable to find staff or buyers for their practices.

In our case we found a buyer in 2006 who leased our premises until downsizing and moving to smaller premises a year ago—now offering only a part time service with, I believe, no after hours or large animal service.

We have tried all available avenues to find a veterinarian to take over the premises and restore full-time mixed practice service but our efforts have not been fruitful. Those parties interested have expressed concern at high start-up costs but I would suggest that a start-up with no good will cost, leased premises and the ability to lease basic equipment should not be overwhelming.

So my question is—what has changed the attitude of graduates?

The medical and dental professions have similar problems but pharmacy seems to have less. There is a constant stream of new tradies starting their businesses in Narrandera and they thrive. I think this refutes the theory that we have a generational problem. It does raise the question of the difference between a TAFE and a university education influencing entrepreneurship.

Is the burden of HECS fees the difference?

I'm too old to know the intricacies of HECS repayments but thought repayments were tied to income.

Perhaps regional towns are perceived as poverty stricken back-waters. We relished our move from Sydney, our children were born here, educated to high school near here and CSU is only an hour's drive. The town has all normal services and amenities, housing is affordable and commute times to work and sport are measured in minutes. The town has not boomed but has weathered many droughts and downturns successfully and is moving ahead.

I seek reassurance that our problem does not stem from our educators. I note that another correspondent refers to access to emergency after hours services being regarded as 'gold standard' by teaching hospitals and suspect that gold plated diagnostic aids has ballooned since the 1960s—which leads to angst about starting costs. It may be relevant that the only parties expressing interest in coming to Narrandera are CSU graduates.

Another correspondent refers to disappointing attitudes by employers and is obviously not thrilled with salaries. I would suggest that starting your own practice solves both issues.

I have no idea of universities' attitudes to self-employment in regional towns as a sole trader or to mixed practice as an enterprise. Could it be that our graduates emerge with no guidance on basic business principles or with an inclination to restrict their professional lives to small animals or even specialising further?

I mourn the fact that our profession appears to be losing our principal reason for existence which used to be providing veterinary services to animal owners wherever it is viable to do so.

I regret that veterinarians are missing the opportunity to practice their profession in friendly welcoming communities where their skills are appreciated, they are able to earn the respect they deserve and they are able to earn the income they deserve.

eBook download:
C&T No. 5724 Issue 293. 'Why is it so hard to find a vet?' & multiple replies

Replies to multiple C&Ts:

1. **C&T No. 5751 Ocular ouch: Clerapliq**
2. **Perspective No. 143 Ethical dilemmas**
3. **C&T No. 5757 Acute renal injury in a cat called Asterix**
4. **C&T No. 5755 Severe sunburn in three dogs presenting to veterinary clinics on the Atherton Tablelands of Far North Queensland**

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C&T No. 5780

Clerapliq. I have just completed the excellent TimeOnline course 'Practical Ophthalmology' but had never heard of Clerapliq until now. A feline ulcer or keratopathy responds well to Hylo Forte. It is not an analgesic but contains hyaluronidase which aids feline corneas to heal quickly perhaps. I shall be getting Clerapliq in. Another good Aine tip!

I am lucky to have a very 'on the ball' optometrist who told me about Nova Tears, made by the same company that makes Hylo Forte and it is a very fine oil that covers the tear film and prevents evaporation of same in dry eye.

Ethical dilemmas. This is a gem. I refer to an extract in the State veterinary regulator's latest communication; it mentioned two vets who rehomed two cats after having been presented for euthanasia. They obviously had a moral dilemma putting healthy animals to sleep, and my opinion, for what it is worth, is good on them. But 'their failure to euthanase when requested was considered "improper"'.

We also do our very best to rehome the convenience euthanasias. Euthanasia is very hard on all the staff, even if we know we are dealing with terminally ill and usually very elderly patients often known since a pup/kitten. Even through the client's grief we are thanked for being so kind.

We will not euthanase a young healthy animal. But we have had great success in getting the animal in question to be signed over to us for rehoming, and of course do not charge for euthanasia. Having a regulator investigate, then caution and fine, for doing what was in their mind the right, moral thing (our number one job as vets is the welfare of animals) is a massive stress. Killing healthy animals is not good for their welfare, in my opinion. These might be two vets who change career because of this. So there is a conflict between the regulators and the vets in this case. Dr Fawcett maybe could comment on this dichotomy:- moral values versus bureaucracy paperwork.

Asterix. A great article, a Goanna track type article by a coal face vet, without references just like Tom* would have wanted. There is a paragraph, where the author talks of the 'correct' way, what we might call the 'Gold Standard'. Not using this could cause worry about litigation or falling foul of the regulators. There are cases where funds are non-existent, you do your best, improvise, and whatever the outcome the client is so grateful.

Severe sunburn. An excellent article on sunburn causing severe third degree burns. There is somewhere in the C&T archives an article written by a vet who uses Rose Hip oil on wide old scars in dogs. I have told clients about it and they have also got old scars to shrink up.

**Tom Hungerford, the first Director of The CVE who was the originator of the* C&T Series.

eBook download:
C&T No. 5352. Malicious freezing of a kitten—Rosehip Oil for Burns Issue 273, Dec 2013

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Reply to C&T No. 5755 Severe sunburn in three dogs presenting to veterinary clinics on the Atherton Tablelands of Far North Queensland

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C&T No. 5781

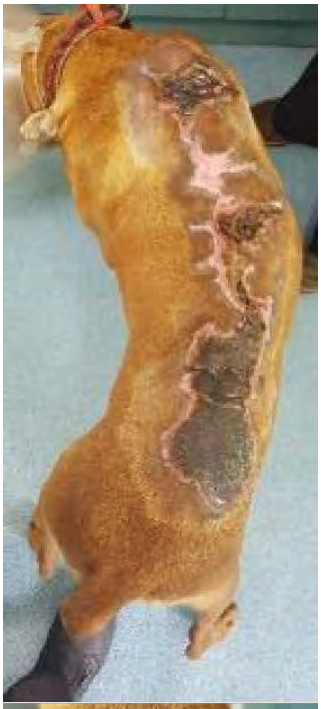
The photos reminded me of 2 'possible' cases in Dachshunds (this breed was more common then), that presented with similar 'skin and fat necrosis' (no histology) between 1974 and 1976, to the Bundaberg Vet Clinic.

We were not aware of dorsal thermal necrosis (DTN) but the history (of one case) was associated with the dog lying (back first) parallel to a horizontal bar heater in the lounge. It was presumed that the increased localised skin temperature lead to subcutaneous necrosis in a darker skinned (hair and/or skin), coloured dog.

Treatment was routine for such lesions for that time period —analgesia was probably not used as much as it is now—fewer such drugs were available at that time.

Recovery was prolonged, cases being eventually seen by all clinicians in this mixed practice, over time. Most of us (approximately 70 %?) are visually oriented and these 2 cases may well not have been recalled except for the characteristic dorsal images so presented.

Again from memory alone, these 2 cases looked more like the images on page 31, case 2, Figure 6, days 27 and 28 of the DTN paper. (see figure 1)



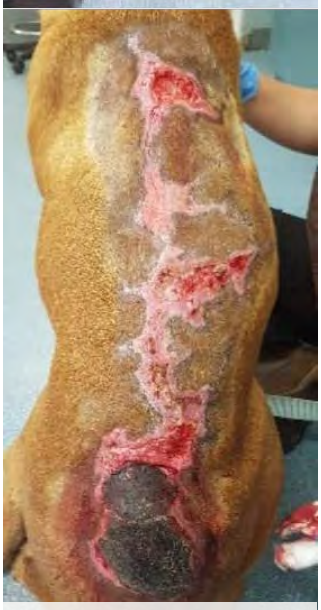


Figure 1.

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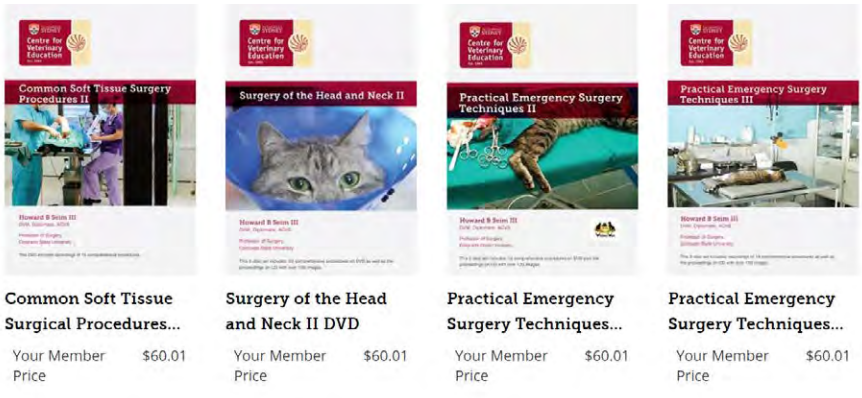

Pathology for practitioners: feline gastrointestinal disease

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This paper provides an outline of talks delivered at the Wanaka Veterinary Conference, Edgewater Resort, Wanaka in August 2016 (www.vetsontour.com.au). Sessions on feline gastrointestinal disease discussed biopsy strategies, histologic diagnosis of lymphoma, and grading schemes for inflammatory disorders, using illustrative case examples.

Endoscopic vs full-thickness biopsies?

Biopsy method selection is ultimately influenced by multiple factors including clinical signs and risk factors for invasive procedures, primary differential diagnoses, access to specialised equipment and expertise, and cost. Both endoscopic and full thickness biopsies are associated with various advantages and shortcomings. Many pathologists strongly advocate for the former on the basis that these samples provide more complete histologic information with relatively fewer complicating artifacts of collection and tissue processing (crush, poorly oriented sections).

Full thickness samples enable examination of all layers of the intestinal wall, and are thus critical to the diagnosis of lesions localised within the submucosa (e.g. lymphangiectasia in dogs) or the muscularis (e.g. smooth muscle tumours, feline gastrointestinal eosinophilic sclerosing fibroplasia [fig. 1], myenteric ganglioneuritis). They may also be necessary in order to access the distal small intestine and ileum when these are beyond the reach of the endoscope. Nevertheless, procuring full thickness biopsies is a more invasive undertaking, and typically only one or two biopsies from each segment of the intestine are collected. For extensive or diffuse infiltrative lesions this may be sufficient; however, limited sampling may be a disadvantage when lesions are patchy or multifocal.

Endoscopy, by contrast, is minimally invasive and enables relatively rapid sampling from multiple sites. It also provides the endoscopist with an opportunity to visualise the intestinal lumen and assess the integrity of the mucosa. The accuracy of diagnoses made from endoscopic biopsies is heavily dependent on the quality of the samples evaluated[1].

Poor sample quality dramatically reduces diagnostic sensitivity, which can only be mitigated by increasing the number of samples. In some cases, the number of samples required for accurate diagnosis from marginal samples is so high as to be unreasonable – in order to recognise crypt lesions in marginal canine duodenal samples with 99% confidence, more than 20 samples are required[1]. The relative importance of sample quality varies with site, species and lesion: in feline duodenal and gastric mucosal lesions characterised by 'moderate cellular infiltrates,' the quality of samples has been found to be less important than for other lesions (e.g. villus blunting, mild infiltrates) and differences in the number of samples required for accurate diagnosis from inadequate, marginal or adequate biopsies were not statistically significant.

Ideally, multiple samples should be collected to allow for one or two marginal tissue samples without hindering diagnostic sensitivity. One study suggests that obtaining 6 marginal or adequate feline gastric or duodenal samples should give 99% confidence of identifying a significant lesion[1]. In order to minimise handling of fragile samples during tissue processing (in order to avoid potential crush artifact), submit your samples in CellSafe (or similar) biopsy capsules.

The quality of a specimen is influenced by both endoscopic technique and sample processing[2]. Inadequate specimens

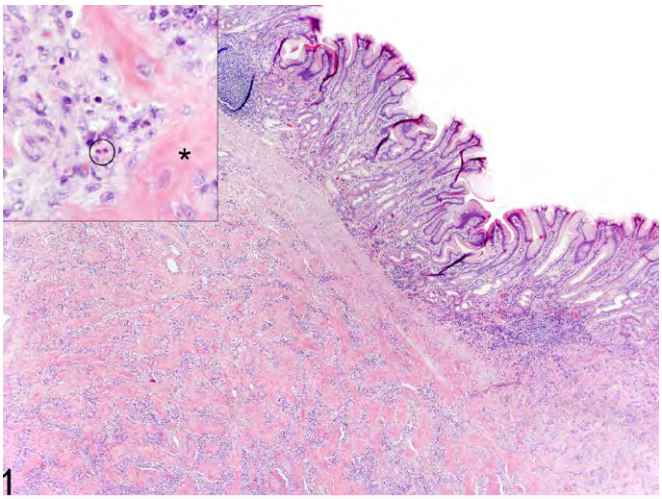

Figure 1. Feline gastrointestinal eosinophilic sclerosing fibroplasia, stomach, cat. The submucosa is expanded and effaced by arborising trabeculae of bland, sclerotic collagen. The mucosa is spared and diagnosis requires histologic evaluation of the gastric wall, which would not be represented in endoscopic biopsies. High magnification inset: Dissecting collagen trabeculae (*) are accompanied by a moderate infiltrate of eosinophils (circled) with fewer lymphocytes and macrophages. Haematoxylin and eosin (HE).

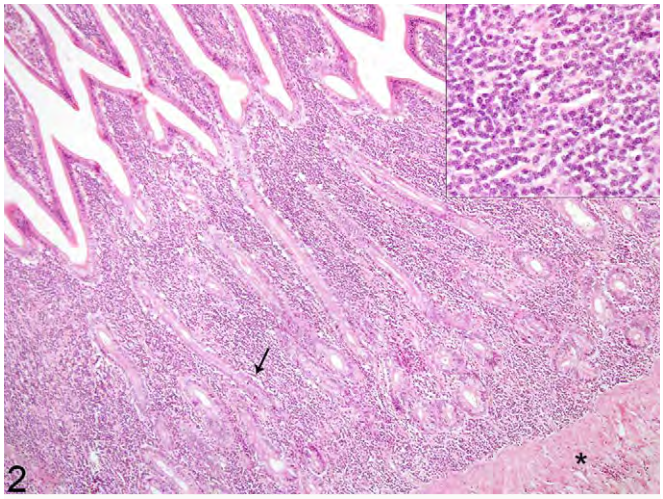

Figure 2. Small cell, low grade lymphoma, jejunum, cat. Mucosal architectures is mildly disrupted by sheets of monomorphic neoplastic round cells that expand the lamina propria, displacing crypts (arrow), which are irregularly spaced. The infiltrate is confined to the mucosa, sparing the underlying muscularis mucosae (asterisk). High magnification inset: Neoplastic cells resemble small T lymphocytes, with scant cytoplasm and small (7um diameter), deeply basophilic nuclei, minimal variation in cell and nucleus size and shape, and rare mitotic figures. HE

may be non-diagnostic because of poor sampling technique or clumsy orientation of samples within the paraffin block. It is reasonable to contact your pathologist with a request to reposition your samples if you believe the histologic description of your sample quality is inconsistent with the specimens you submitted. In fact, it is reasonable to contact your pathologist before you have even submitted a biopsy – if you are not sure which approach is best suited to a particular case, it is worthwhile calling your pathologist, who will after all be examining your sections.

Distinguishing IBD from lymphoma

Distinguishing feline inflammatory bowel disease from intestinal lymphoma is a diagnostic challenge for clinicians and pathologists alike. Histologic examination of intestinal biopsies is often critical to making a definitive diagnosis, however, distinguishing inflammation from neoplasia may be complicated by concurrent inflammatory bowel disease, the patchy distribution of lesions, and by the limitations of the biopsy specimen.

The largest population of lymphocytes in the body resides in the mucosal-associated lymphoid tissue (MALT), which is largely populated by CD3 positive T lymphocytes. The majority of feline intestinal lymphomas are T cell in origin and have a predilection for the jejunum, occasionally affecting multiple segments (fig. 2)[3, 4]. By contrast, B cell neoplasms tend to arise within the stomach and ileocaecocolic junction, and lesions are often multifocal (fig. 3). Collectively, the most commonly implicated sites in feline alimentary lymphoma are the ileocaecocolic junction and jejunum[5]. Biopsy of multiple sites, including the distal jejunum and ileum, are therefore indicated when alimentary lymphoma is suspected.

Neoplastic T cells are often cytologically indistinguishable from reactive infiltrates (fig. 2), and pathologists rely on the pattern of neoplastic infiltration to make a diagnosis of lymphoma, including epitheliotropism, mucosal effacement and transmural invasion. The cytomorphology of neoplastic cells and pattern of infiltration are important prognostic factors. Small-cell type T cell lymphoma is associated with prolonged survival time (up to 28 months), compared with large-cell type T cell (1.5 months), and B cell lymphoma (3.5 months)[4]. Lymphoma confined to the mucosa is associated with a more favourable prognosis (median survival time 29 months), compared with transmural lymphoma (1.5 months). Note, demonstrating transmural invasion (fig. 3) is not possible in endoscopic biopsies, in which only the mucosa is represented.

Immunohistochemistry (IHC) is a critical adjunct tool in the diagnosis of lymphoma in any site. Labelled antibodies against lineage-specific markers are utilised to distinguish between neoplasms derived from T cells (CD3 expression) and B cells (CD79a and Pax5 expression). Using IHC in conjunction with histopathology increases diagnostic accuracy and in many cases is critical in differentiating IBD from lymphoma, particularly early in the course of disease progression. Important prognostic information can additionally be obtained simply by determining the cell of origin (T vs B cell lymphoma), and by demonstrating the pattern and extent of the neoplastic infiltrate.

More recently, molecular assays for lymphocyte clonality (PCR for Antigen Receptor Rearrangements – PARR) have provided more acute diagnostic sensitivity. These assays evaluate the diversity of T cell receptor genes and those in the B cell immunoglobulin heavy chain variable region, determining whether lymphocytes infiltrates are clonal (neoplasia) or polyclonal (reactive). Studies using a combined approach have demonstrated that feline intestinal T cell lymphoma is easily misdiagnosed as inflammation using either histopathology alone or with additional IHC[4]. A diagnostic algorithm that employs stepwise use of histopathology, IHC and PARR significantly improves diagnostic accuracy and prognostication[6, 7]. Importantly, PARR must be interpreted within the context of histologic changes and IHC findings, and is not a stand-alone test; interpretation of results in consultation with an oncologist is advised. PARR is now available through most commercial diagnostic laboratories and can be performed on formalin fixed biopsy material or paraffin embedded sections.

Lymphoplasmacytic enteritis

Even after lymphoma has been excluded, interpreting the significance of non-neoplastic infiltrates within the gastrointestinal mucosa remains problematic. Often the greatest diagnostic dilemma is in determining whether a cellular infiltrate lies within the spectrum of normal, which remains poorly defined. Multiple grading and classification schemes have been proposed over the decades, rarely improving concordance between pathologists.

Most schemes classify changes according to the prevailing inflammatory cell type and any significant morphological

changes, for example *lymphoplasmacytic enteritis or ulcerative colitis*. In 2008, the WSAVA Gastrointestinal Standardisation Group published a classification system in order to define multiple morphological abnormalities at four anatomical sites: the gastric body, gastric antrum, duodenum and colon[8]. This system has provided pathologists with a framework for distinguishing normal from abnormal and prescriptive criteria for grading the severity of various lesions. Nevertheless, correlating histologic changes with clinical findings remains problematic and it is presently not possible to confidently predict the severity of clinical signs on the basis of histologic findings[9].

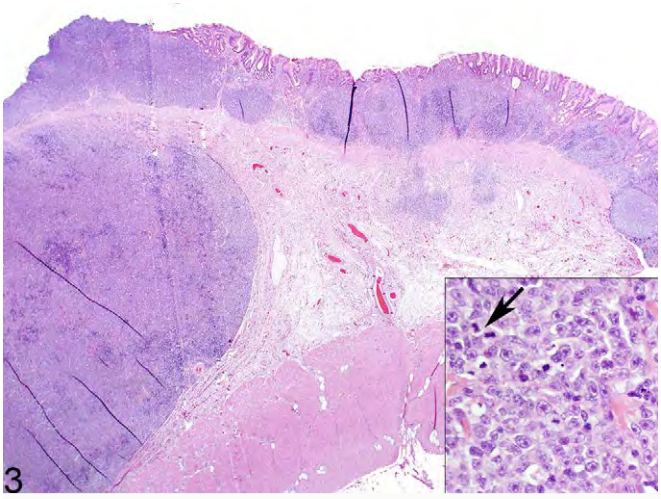

Figure 3. Large cell, high grade lymphoma, stomach, cat. The pyloric mucosa is markedly disrupted by a poorly demarcated infiltrate of neoplastic round cells that extends into the underlying submucosa. Confirming transmural invasion, which has implications for prognosis, requires full thickness biopsies. High magnification inset: Neoplastic cells have scant cytoplasm, large (15-20um diameter) round to slightly indented nuclei and a single distinct, centrally located nucleolus. Mitotic figures are frequent (arrow). Immunohistochemistry confirmed the B cell origin of neoplastic cells in this case (not shown). HE

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