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Vol 11 | Issue 05 | December 2024

Striving Towards Wellness:
It is Time to Clean Out The Mental and Emotional Clutter

Cardiology
Raas Inhibition in The Treatment of Congestive Heart Failure

Respiratory
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1. Kirkby Shaw, K, *et al. Vet Med Sci.* 2016;2:3-9.
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Editor's Note

After 10 years I am now finally hanging up my editors' cap for the Vet 360. It has been 10 years of enjoyment but also hard work and deadlines.

Dr Paul van Dam will be taking over as magazine editor, but I will still be contributing and consulting ad hoc, so you will still hear from me.

In this final edition, Dr Izak Venter has written a nice practical article on suturing corneal lacerations. Something we have to be able to accomplish ourselves when referral is not an option. Some simple rules to follow and a magnifying glass!

The cardiology article by Prof Johan Schoeman expands on the everchanging advances in the management of cardiac disease... bringing an old friend, spironolactone, back into the light. You'll need to focus for this one.

And lastly I co-wrote a coughing article with a brand new colleague. A problem he correctly identified on his outpatients' rotation.. proving that the fact that you think and question what is in front of you may be more important than your carry around knowledge. Things must make sense. If they don't, go and look again.

Wishing you all a safe, relaxed and enjoyable festive season.

Liesel

vet 360

VET360 aims to be a leader in the field of continuing veterinary development in Southern Africa by providing veterinary professionals from diverse disciplines with tools to help them meet the challenges of private practice. The magazine aims to make information accessible, both paper and electronic, and provide clinical, business and other veterinary information in a concise form to enable the practitioner to rapidly acquire nuggets of essential knowledge

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Index

Vet360 is a source of current, relevant educational material for practising veterinary professionals in Southern Africa. Published Bi-monthly in hard copy and electronically on www.vet360.vetlink.co.za.

Distribution: Southern Africa, 3 500 copies.

Editor: Liesel van der Merwe

Layout and design: Annalize de Klerk

Publisher and Owner: Vetlink Publications

Other Publications by Vetlink: Vet360 Mobile App, Livestock Health and Production Review, Hooo-Hooo, Equine Health Update

Publisher: Madaleen Schultheiss, madaleen@vetlink.co.za

Advertising Enquiries: AgriConnect
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Striving Towards Wellness:
It is Time to Clean Out The Mental and Emotional Clutter

Retha Watson
(MA Industrial Psychology)

"The most important house to clean is yourself, your own house, which we never do"-Marina Abromovic-

Spring is the symbol of light, of new and fresh sights around us, the sound of thunder and the smell of rain, the glorious sight of blooms and blossoms everywhere.

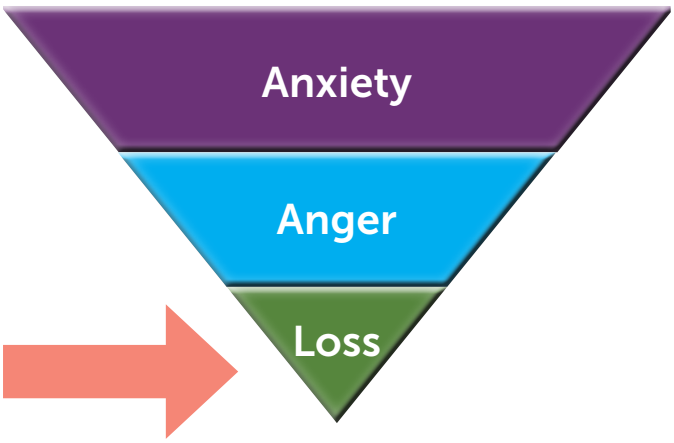
We find ourselves with renewed physical energy, it's time to clean the dusty curtains and carpets, time for decluttering the cupboards, sorting through old papers and sending them for recycling, chucking out clothes that no longer fit. Similarly, we should do the same with our minds and hearts. Clean the old dusty emotions away, sort through old and expired thoughts, chuck out the stuff that does not fit anymore, pack the old, emotional baggage in a box and send it off to the furthest ends of the earth.

In the past few articles, we addressed a few hard topics; suicide and ways to identify risk and warning signs, depression, anxiety, burnout and compassion fatigue, managing emotions and lastly how we manage our relationships.

The veterinary environment remains complex and difficult. We cannot take away the fact that as professionals on different levels, you remain overly committed to your profession and purpose and experience compassion fatigue and trauma as patients die and families grieve for the loss of a family member. You will always be confronted with animal abuse and neglect. That will not change but let us allow for the emotional and mental spring cleaning to take place so that you are stronger to start afresh.

We are sometimes busy with a frustrating menial thing such as driving in traffic, untying an electric cord, struggling to insert your USB, and we are suddenly caught up in a tremendous explosion of anger. It is in times like that, that we need to take a moment for introspection. We need to reflect on where the anger comes from, this simple task was only the trigger event.

Anger and frustration are sometimes just a symptom of something deeper, as the illustration below shows:



Our anxiety and anger are often an indicator of loss and we need to reflect on that loss.

In the practice you experience loss daily. It may be time to say good-bye to the losses of the last year and season for the good of your own psychological well-being. We may also experience loss of a friendship or a colleague due to conflict or a relationship which is not working due to various valid reasons.

The experience of a feeling of loss and actually missing a person, does not mean that we should go back, it just means that we have loved and laughed but it is a chapter closed.

You cannot get rid of old emotions and thoughts if you have not taken stock of what is actually in the cupboards of your mind and heart. What is it that is weighing you down? How many bricks are you carrying in that backpack on your back daily?

I recently presented corporate training where we dealt with body language and the message it portrays to others. One of the useful tips is to take up more space for the subconscious message of self-confidence. We discussed how we should also take up our metaphorical space. We tend to make ourselves small to accommodate and to allow others into our space when we don't really want to. This may cause resentment and frustration.

Continuous bullying and abuse cause long term post traumatic reactions. Negative emotions are a normal part of the episode or experience. In any traumatic experience, an almost knee- jerk reaction is to look for closure, to hear an apology, a request for forgiveness.

It may never happen, but the healing is in that we can let is go and move on. Forgiveness is never the other party's deservedness be forgiven. It is your ability not to allow the power of that trauma to weigh you down anymore or to define you.

Long term financial strain and stress wears us down physically and psychologically. Globally and in this country an economic recession is happening, it is impacting the majority of people and causes several negative emotions.

Concern and worry about a significant other's health, will also cause negative emotions, not to mention the impact on your daily routine and well-being.

Whatever the reason, we must take the time to sit and reflect on where the emotions and thoughts come from. If we have pinpointed the origin, we are halfway in conquering them and to start the spring cleaning process.

We also need to reflect on what present, past and future clutter that needs organising, solid scrubbing and sometimes simply discarding. To actively take stock of the weight in that backpack that is hindering you to move forward more positively and energetically.

Our Past

Not everything in our past is negative. We usually have more good and proud moments than moments of shame, guilt or loss.

- Take stock of the past, what was good and what was bad.
- What are you ready to scrub clean or chuck out? Sometimes we hold onto past experiences like an emotional crutch that keeps us going. We do move forward, but much slower and with a lot of pain.
- What are the positive experiences, something to build on, to work on or to improve on.

- What needs to be actively mourned and buried. It may sound dramatic, but we sometimes need to have a funeral in our minds for specific experiences.
- Say your farewell to the negative, look forwards and upwards.
- Create specific goals that you would like to improve on or celebrate past experiences that were positive and made you happy and content. Sometimes it means rekindling old friendships, taking up past challenges such as running marathons or getting fit, taking road trips or being creative.

Our Present

Our present,everyday is sometimes cluttered with various emotions, winning and losing, joy and loss, good and bad moments, routine and extreme stress. In a previous article, we discussed the fact that life cannot be only positive. Sometimes we are confronted with the negative aspects, however the secret is, what is dragging you down? What is cluttering your life?

- Taking stock once again of your present, the positive. The uplifting should be kept and nurtured. The negative around you should be managed or swept out into the dustbin.
- Relationships that may be toxic or draining? Re-evaluate the impact of that friendship. Re-evaluate the position of the friendship in your life. Has it run its course?
- Disorganisation in your life, not prioritising your goals and objectives is one of the main causes of mental and emotional clutter.
- Aspects in your life that is not in line with your values and beliefs should be re-assessed and re-considered.
- Time consuming dead-ends that we spend our time on and lead to no growth, no energy and no joy – re-evaluate their importance or relevance and throw it out if necessary.
- Destructive habits that cause anxiety and a breakdown in your relationships cause unnecessary stress.
- Over committing yourself to others – stretching yourself too thinly despite a demanding professional occupation with all the emotions that goes with it. No, is a full sentence.
- Take up your space – stop making yourself small for others.
- Take control of your own administration, your relationships. Sometimes the old shirt in the cupboard or the old tablecloth needs good airing and ironing. It may be overwhelming so make a list and tackle it in bite sizes.
- Tackle that conflictive relationship with respect and empathy, listen to hear; understand and not to argue, to resolve the conflict – it is about meeting each other halfway.

Our future

We all know the saying that you have a hundred percent success rate in surviving your worst days. The problem is that we continue to worry about the future, something specific or just a vague undefined niggle.

We worry about things that will probably never happen. Take stock, take control and manage yourself and your mind. Our minds tend to catastrophise the future, learn to trust that everything will work out as it should. Make a list of your future worries, is there something you can take control of now? If not, let it go.

Spring Cleaning Tips

- Our minds hold and believe what we tell it every day, start with positive self-talk.
- Make a list of priorities and tackle it one manageable piece at a time.
- You cannot be everything and everywhere for everybody, let go of the guilt and over-commitment.
- Learn to do relaxation activities, deep breathing, deep muscle relaxation.
- Simplify your life – get rid of the unnecessary.
- Visualise yourself into what you want to achieve.
- Re-evaluate your perceptions and beliefs. Hold on to what is good and let go of that toxic judgements and inner coding.
- Daily manageable cleaning – talk to a trusted friend or a professional.
- Talking about that "backpack you are carrying with you" - take an honest assessment of yourself and the burdens you are carrying.

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Raas Inhibition in the Treatment of Congestive Heart Failure

Prof Johan P. Schoeman
BVSc, MMedVet, PhD, DSAM, Dipl. ECVIM-CA, FRCVS

Abstract

This article is written as a follow-up to the four talks and one webinar that have been presented across South Africa on the role of RAAS inhibition in the management of canine heart disease during October 2024. It presents a review of RAAS activation in congestive heart failure (CHF) and explains the pathophysiology and the recently identified role players; i.e. the various peptides and enzymes of the classical and alternative RAAS pathways. It highlights the maladaptive attributes of chronic RAAS activation and the treatment strategies that should be employed to mitigate these. It also touches on the clinical, radiological and echocardiographic differentiation of the different stages of heart failure. Moreover, it addresses quadruple therapy and drug dosages and combinations for comprehensive RAAS modulation in congestive heart failure. Lastly, it presents a critical and evidence-base review of the latest research and a meta-analysis on the treatment of the various stages of heart disease.

Introduction

The renin–angiotensin–aldosterone system (RAAS) plays a pivotal role during the maintenance of blood pressure and sodium and electrolyte homeostasis. The RAAS is constitutively active with an ability to upregulate in response to various factors such as hypovolemia, hypotension, inadequate sodium intake and chronic sustained sympathetic nervous system activation. Decreased cardiac output leads to acute activation of this system, which is associated with physiologic (sodium retention, water intake and vasoconstriction) and pathophysiologic (hypertrophy, fibrosis, and oxidative stress) responses.

However, in the long run, chronic RAAS activation is maladaptive and its two main end-products, namely, Angiotensin II (AngII) and Aldosterone, that have been shown to play major pro-fibrotic roles. As a consequence, suppression of the RAAS is a major part of the treatment plan in humans and animals with chronic cardiovascular diseases. Yet, current therapies do not always prolong survival, nor do they reduce morbidity (Atkins et al. 2007; Borgarelli et al. 2020; Kvart et al. 2002).

Recent studies have identified redundancy in the RAAS system (i.e. unabated formation of Ang II by other pathways) that may explain the reduction in the benefits of medical therapy. Moreover, therapy contributes to the concomitant and inadvertent suppression of beneficial (vasodilatory) components of the RAAS. Consequently, the development of future drug therapies for cardiovascular diseases will require a better understanding of the changes in the RAAS that accompany disease and, more importantly, the modulation brought about by therapy. This article therefore aims to unpack some of these pathways and provide the clinician with a more granular understanding of the downstream effects of RAAS inhibition or rather "RAAS modulation" as the more correct term.

RAAS physiology

The renin–angiotensin-aldosterone system is made up of angiotensin peptides, enzymes and receptors - the few major ones are illustrated in Figure 1. Angiotensin II is the major effector molecule of this system and it is produced from the degradation of angiotensinogen (AGT) (produced

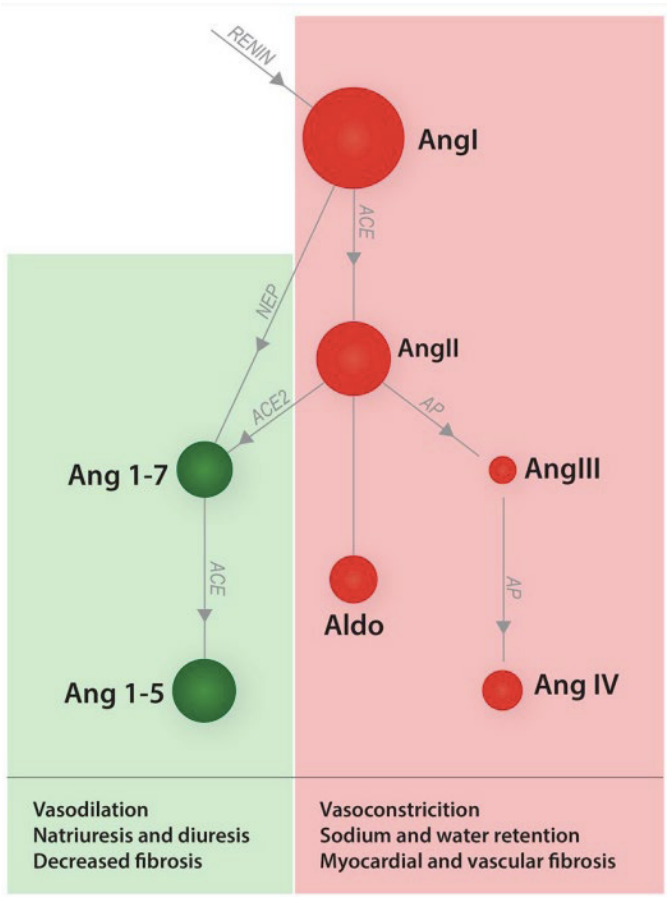

Figure 1: Simplified scheme of the classical (red) and alternative (green) arms of the RAAS. The sizes of the circles are proportional to the concentration of each angiotensin peptide in healthy dogs. Arrows indicate enzymes catalysing the conversion of metabolites. ACE - angiotensin converting enzyme ACE2 - angiotensin converting enzyme 2 Aldo - aldosteroneAng - angiotensinAP - aminopeptidases NEP – neutral endopeptidase

in the liver) to angiotensin I (AngI) by renin (produced in the kidney) and the subsequent degradation of Ang I to Ang II by angiotensin-converting enzymes (ACE). The fluid and sodium retentive, vasoconstrictive, pro-inflammatory and pro-fibrotic effects of RAAS are chiefly mediated by Ang II acting on its angiotensin type 1 receptor (AT1R). AngII ultimately stimulates aldosterone secretion from the zona glomerulosa of the adrenal gland, which potentiates the physiologic and pathophysiologic vasoconstrictive effects of Ang II by mediating the additional retention of sodium, which subsequently increases intravenous fluid volume through osmotic fluid retention.

The angiotensin peptides and receptor combinations that cause vasodilation and counteract the inflammation, fibrosis and apoptosis induced by the classical RAAS, include angiotensin 1,7 (Ang1,7) binding its MAS receptor and AngII binding its type 2 receptor (AT2R) (Santos et al. 2018). In addition, the ACE2 and neprilysin pathways are also responsible for the formation of Ang1,7 from AngI (via angiotensin 1,9) and AngII. The counter-regulatory actions of these angiotensin peptide receptors and enzymes, the ACE2, Ang1,7, MAS pathway and the AT2R have led to this being classified as an alternative pathway, differentiating it from the classical pathway of the core RAAS (AngI, ACE, AngII and AT1R). The RAAS was previously viewed, rather simplistically, as an endocrine system where renin was thought to be released into the blood in a highly controlled manner from the juxtaglomerular apparatus of the kidney and acted on constitutively produced hepatic AGT to form AngI. AngI was then thought to contact ACE on the surface of vascular endothelial cells and get converted to AngII, which acts at its AT1R and AT2R, to induce vasoconstriction and vasodilation, respectively.

The AT1R is the predominant AngII receptor type in adult organisms and is widely distributed, occurring in the vasculature, kidney, adrenal gland, brain and elsewhere. However, it has recently been discovered that enzymes such as ACE, chymase, ACE2, prolylcarboxy-peptidase (PCP), prolyl-endo-peptidase (PEP) and neprilysin also degrade AngII into its various metabolites including angiotensin Ang 1,9, 1,7, 1,5, III and IV. In this endocrine model, angiotensin peptides reach the tissues via the circulation. Yet, local (tissue) formation of AngI and AngII also occur in the heart, kidneys, brain and vascular tissue - all of which markedly amplify the effects of this system.

The enzymes of the RAAS are therefore locally present and, consequently, all angiotensin peptide metabolites of the RAAS are also formed locally in the tissues. It is still likely, though, that most renin originates from the juxtaglomerular apparatus of the kidney and most AGT originates from the liver and are delivered to the tissues via the circulation (Matsuska et al. 2012). Contrarily, tissue production of renin and AGT may mediate some disease processes (e.g., hypertension) independently of the circulating RAAS (Davisson et al. 1999). Moreover, higher AGT transcription levels have been found in cats with chronic kidney disease as compared with controls (Lourenco et al. 2022).

Another source of tissue activation of the RAAS is through the precursor of renin (prorenin), which binds the prorenin receptor, rendering it active, thereby bypassing the rate limiting step (renin production) and initiating the cascade for local generation of angiotensin peptides (Hennrikus et a.l 2018). In addition, the local enzyme environment also affects the overall balance between the classical and alternative RAAS (see Figure 1). Evidence point to the fact that the ideal therapeutic target probably lies in the balance of the classical and the alternative arms of the RAAS.

Following from this, the optimal RAAS modulating drug/drug combination should decrease classical RAAS activity, whilst increasing alternative RAAS activity. Some additional complexity is produced by the fact that the serine protease, chymase, is also capable of catalyzing AngII production from AngI in the tissues, independently of ACE. Hence, chymase would be a very promising therapeutic target if it proves to be a potent mechanism for AngII formation in tissues.

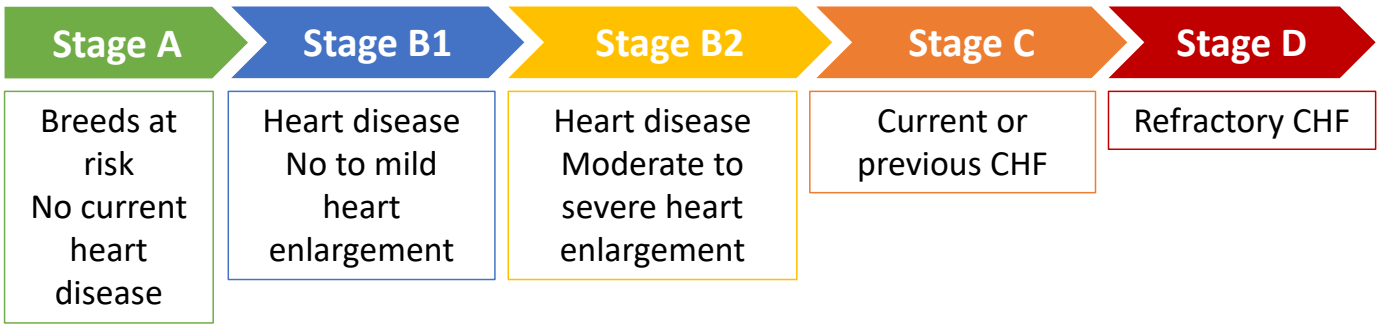

Figure 2: ACVIM Staging of MMVD.

RAAS adaptations in cardiovascular disease

Several studies have quantified the comprehensive RAAS in dogs with naturally occurring cardiovascular disease using the RAS Fingerprint. In a recent study, no differences were found in circulating angiotensin peptide concentrations when dogs stage B1 and B2 myxomatous mitral valve disease, classified according to the American College of Veterinary Medicine (ACVIM) criteria and not on any treatment, were compared with normal dogs (Hammond et al. 2023).However, the balance of the alternative to classical RAAS changed with MMVD progression, as evidenced by a significantly greater alternative activity in stage B2 as compared with normal dogs.

This may have resulted from increased activity of some alternative pathway enzymes or increased cleavage of ACE2 (a major mediator of the alternative pathway) from the tissues, which enhances its presence in the circulation. Once heart failure due to MMVD develops and furosemide therapy is initiated, circulating RAAS activation increases significantly (Adin et al. 2020, Larouche-Lebel et al. 2019).

To complicate matters, studies have shown that polymorphisms in the RAAS occur in dogs and cats (Meurs et al. 2015). These include polymorphisms in ACE, AGT and the AT2R. A polymorphism in the canine ACE gene is the best studied to date and, interestingly, certain breeds, such as Cavalier King Charles Spaniels, Dachshunds, Irish Wolfhounds and Doberman Pinschers have a high prevalence of this variant (Adin et al. 2021; Adin & Hernandez 2023; Meurs et al. 2017).

Yet, there was no difference between dogs with the ACE polymorphism, when compared to wild type dogs, in regard to suppression of ACE activity with ACE inhibitor (ACEi) therapy, although these dogs were shown to have a lower baseline ACE activity. In contrast, a more recent study using RAS Fingerprint analysis showed no difference in RAAS profiles with respect to this polymorphism (Adin et al. 2020).

This discordant finding may have been due to yet another confounder, which is the different methods of determining ACE activity, that have been used in the various studies. In one study, aldosterone breakthrough (ABT) was more common in dogs with the ACE polymorphism, likely occurring via mechanisms other than altered ACE activity or raised AngII levels.

1. **VHS > 10.5**
2. **VLAS > 2.5**
3. **LA:Ao ratio > 1.6**
4. **LVIDD(n) > 1.7**

Figure 3: Radiographic and echocardiographic criteria for Stage B2 heart disease.

The effect of RAAS polymorphisms on long-term cardiovascular outcomes and further evaluation of the functionality of the ACE and other polymorphisms warrant further study. It is likely that genetic variation influences baseline RAAS activity and response of this system to disease progression and pharmacotherapy, which will call for individualised patient care in the future.

Classification of the stages of heart disease

The ACVIM has classified myxomatous mitral valve disease into several stages based on breed predisposition (Stage A), mild cardiac enlargement (Stage B1), moderate cardiac enlargement (Stage B2), onset of congestive heart failure (Stage C) and refractory heart failure (Stage D) (Figure 2).

There are various radiological and echocardiographic criteria that can be used to classify dogs into stage B2 (Figures 3-9) and Stage C & D (Figure 10), with varying degrees of sensitivity and specificity, which are beyond the scope of this article.

Moreover, recent advances in lung ultrasound enables the clinician to detect extravascular lung water at an earlier stage than with thoracic radiography (Figures 11 and 12). This is based on the principle that the mixture of air and fluid creates vertical, laser-like artefacts across the field of view that are known as ultrasound lung rockets or B-lines. Their number indicate the severity of extravascular lung water and their distribution is helpful in determining the aetiology of the problem, i.e. ventral distribution in aspiration pneumonia and perihilar distribution in congestive heart failure.

RAAS suppression in dogs with myxomatous mitral valve disease (MMVD)

Preclinical disease (ACVIM stage B1 and B2)

The usefulness of ACE inhibitors (ACEi) to delay disease progression in MMVD, before the onset of congestive heart failure, has been called into question by the results of several large clinical trials as well as a meta-analysis, that have been unable to show conclusive benefits (Donati et al. 2022). Although one study showed promising results, the difference in the time to onset of CHF was not significantly different between dogs receiving enalapril and dogs not receiving enalapril (Atkins et al. 2007). Other studies clearly failed to demonstrate benefit to ACEi use in preclinical disease in cats (King et al. 2019) and dogs (Kvart et al. 2002).

One explanation for negative or neutral study results of ACEi in dogs is the phenomenon of aldosterone breakthrough, which is defined by high blood aldosterone or AngII concentrations, despite appropriate ACEi dosage (Ames et al. 2017). It was originally thought that the efficacy of ACEi therapy can be measured by the percentage suppression of ACE activity. For, in theory, successful ACE suppression with ACEi should reduce the secretion of aldosterone, because AngII (its major secretagogue) formation is blocked.

Nevertheless, several studies have now shown that the Aldosterone:Creatinine ratio increased after furosemide administration, despite affective ACE suppression with ACEi doses as high as 1 mg/kg q12hours (Ames et al. 2015). Although this phenomenon is widely accepted in humans with heart failure, chronic renal disease and hypertension, the mechanisms remain poorly understood. General causes of ABT are thought to include persistent AngII production by the chymase and other pathways (AngII breakthrough), upregulation of other aldosterone secretagogues such as ACTH and decreased aldosterone metabolism and clearance (Ames et al. 2017).

To address ABT, the DELAY study evaluated comprehensive RAAS suppression using a combination of benazepril and spironolactone (Cardalis®) in dogs with preclinical MMVD. However, this study also failed to demonstrate a benefit regarding time to onset of CHF when compared with benazepril treatment alone (Borgarelli et al. 2020).

Nevertheless, despite a lack of primary end point benefit, the DELAY study found significant reductions in cardiac size and biomarkers in the benazepril and spironolactone (Cardalis®) group compared with the benazepril only group (Borgarelli et al. 2020). Another explanation for neutral study results may be the fact that the RAAS is not activated or dysregulated enough in the preclinical stage of disease. Finally, multiple modifiers of the RAAS are likely contributing to the individual heterogeneity in regard to basal RAAS activity and a response to pharmacotherapy.

In contrast, the EPIC study has shown significant benefit for the use of pimobendan in preclinical MMVD (Boswoord et al. 2016). Median time to primary endpoint was 1228 days in the pimobendan group and 766 days in the placebo group.

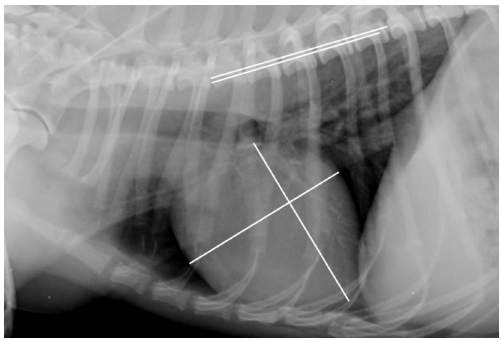

Figure 4: Vertebral heart score (9.9) on right lateral thoracic radiograph of a Chihuahua dog;Length and width of the cardiac shadow are superimposed on the vertebrae starting at the cranial aspect of T4.

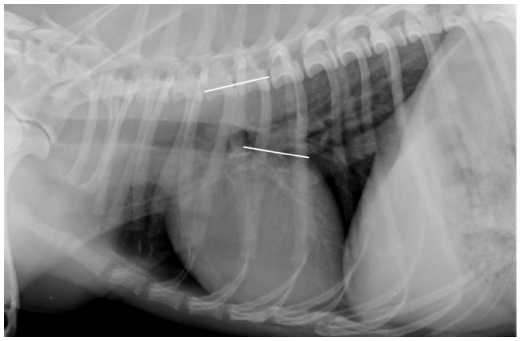

Figure 5: Right lateral thoracic radiograph showing an example of vertebral left atrial size measurement in a Chihuahua dog. For this dog, the vertebral left atrial size was 2.0 vertebrae.

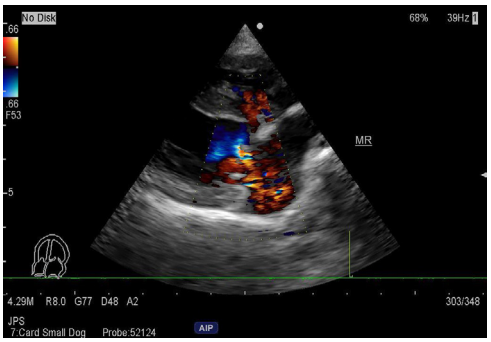

Figure 6: Right parasternal long axis echocardiogram, demonstrating marked mitral valve regurgitation.

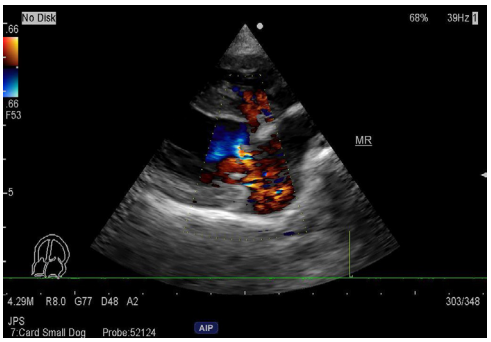

Figure 7: Echocardiogram demonstrating mitral valve thickening, especially the septal leaflet.

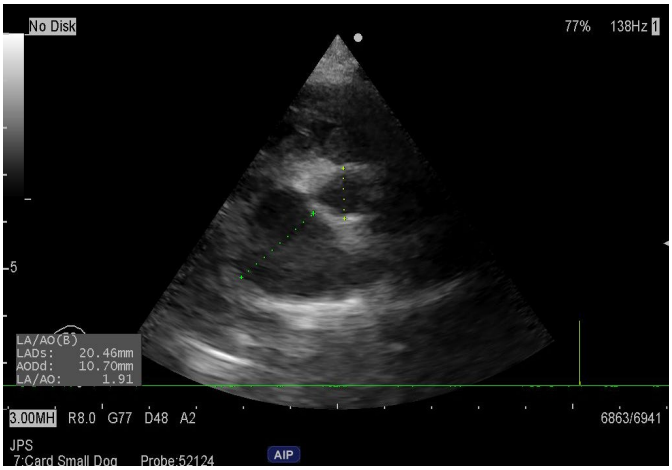

Figure 8: Echocardiographic left atrium/aorta ratio = 1.9.

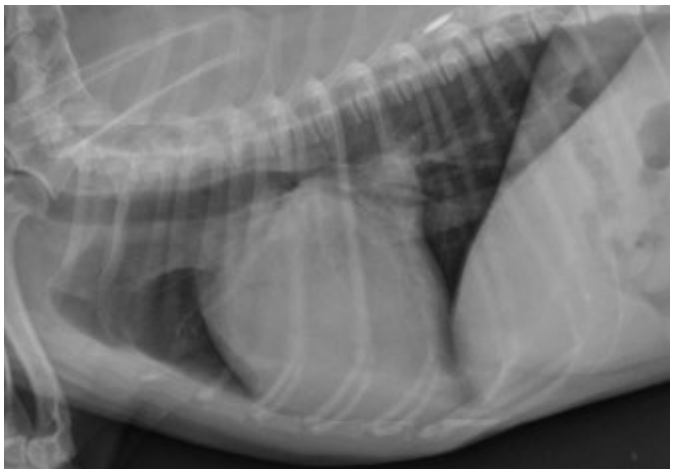

Figure 9: Stage B2: Severe left atrial and left ventricular enlargement; Straightened caudal heart border;Marked tracheal elevation;Pulmonary venous enlargement visible in the cranial lobar vessels.

The hazard ratio for the pimobendan group was 0.64 (95% CI: 0.47–0.87) compared with the placebo group. Dogs in the pimobendan group also lived longer, with a median survival time of 1059 days in the pimobendan group and 902 days in the placebo group. Interestingly, the significant cost of the drug must be weighed up against the 157 days of extended life brought about by the therapy.

ACVIM stage C (Congestive heart failure)

Interruption of RAAS activation is a key strategy in the pharmacological management of congestive heart failure. It is well established that hypotension, decreased cardiac output, chronic sympathetic stimulation and low plasma sodium and high plasma potassium concentrations all stimulate the RAAS.

Clinical studies evaluating ACEi in dogs with CHF demonstrated morbidity and mortality reductions when used in addition to furosemide therapy (Cove 1995, Ettinger et al. 1998, Improve 1995). These studies support the benefit of RAAS suppression in the setting of CHF treated solely with diuretics. Subsequent studies demonstrated superiority of pimobendan over ACEi for the treatment of CHF, but the trial design did not reflect clinical practice, where both medications are typically administered together without a need to make a choice between them (Haggstrom et al. 2008, Lombard et al. 2006).

The BESST study demonstrated end point benefit for CHF dogs given comprehensive RAAS suppression with benazepril and spironolactone (Cardalis®) compared with benazepril alone, which supports the concept that aldosterone breakthrough is important to outcomes (Coffman et al. 2021). Yet, the trial design did not include pimobendan which is typically used together in CHF.

Finally, the recent VALVE study concluded that there was no benefit when adding an ACE-inhibitor (ramipril) in dogs with CHF, if they were already treated with pimobendan and furosemide (Wess et al. 2020). Unlike the COVE, IMPROVE and LIVE studies, the VALVE study included pimobendan, which is now standard of care for dogs with CHF following the landmark EPIC study (Boswood et al. 2016).

However, the VALVE study design used a very high dose of furosemide (a potent inducer of the classical RAAS pathway) and a low dose of ramipril (0.125mg/kg ONCE daily). Moreover, it did not evaluate comprehensive RAAS suppression with an aldosterone antagonist (e.g., spironolactone) in addition to ACEi.

In summary, the most recent ACVIM consensus guidelines on the treatment of CHF advocates for the use of quadruple therapy (furosemide, pimobendan, benazepril and spironolactone) in both DCM and MMVD stage C (CHF). With benazepril and spironolactone that are now available in a single combination tablet (Cardalis®), the number of drugs can be reduced to three instead of four.

References available on request

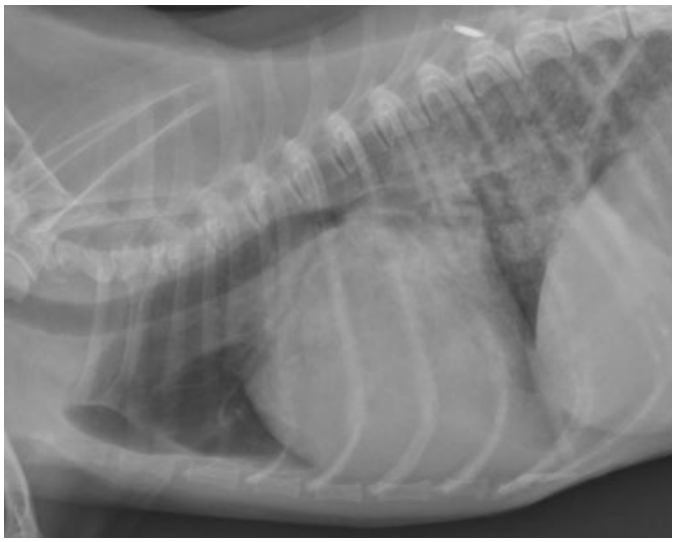

Figure 10: Stage C & D: Severe left atrial and left ventricular enlargement;Marked tracheal elevation;Perihilar and caudodorsal interstitial to alveolar lung pattern.

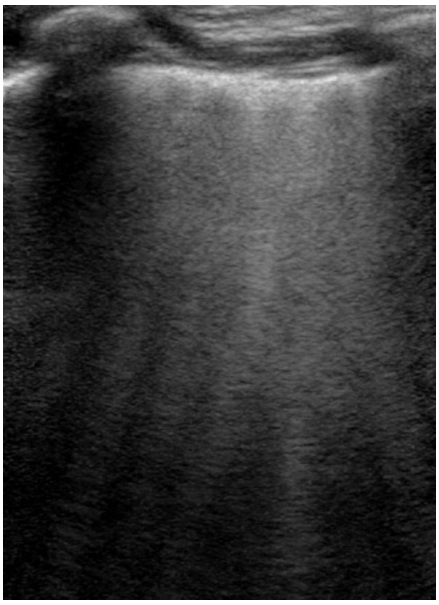

Figure 11: Evidence of moderate extravascular lung water in dogs in Stage C & D MMVD;> 3 B-lines per intercostal space, but the individual B-lines can still be discerned.

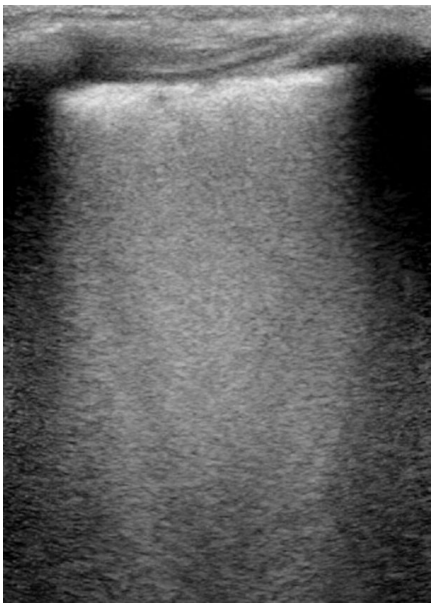

Figure 12: Evidence of marked extravascular lung water in dogs in stage C & D MMVD;Diffuse B-lines, which have become confluent (individual B-lines cannot be discerned); Thickening of the pleural-pulmonary line.

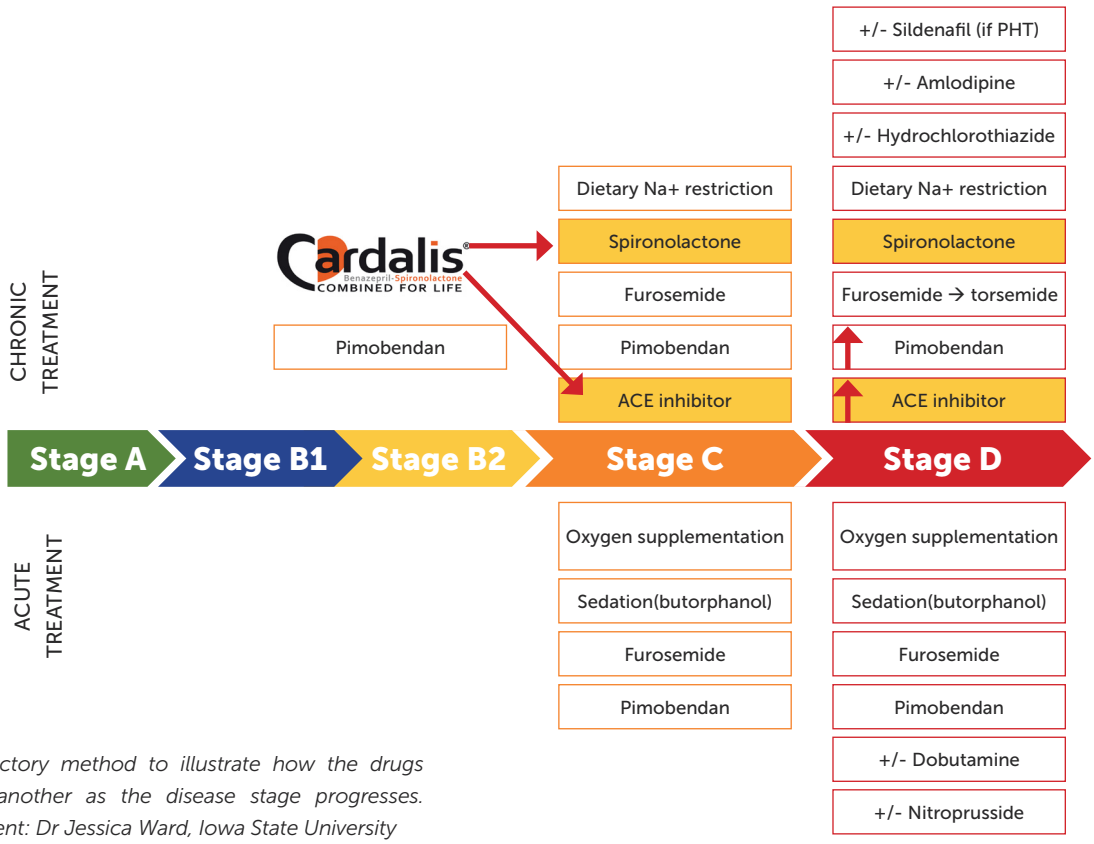

Figure 13: Trajectory method to illustrate how the drugs build on one another as the disease stage progresses. Acknowledgement: Dr Jessica Ward, Iowa State University

CPD Questions

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1. The RAAS is constitutively active with an ability to upregulate. Which ONE of the following factors in UNLIKELY to stimulate the RAAS?

a. Hypovolemia
b. Hypotension
c. Inadequate sodium intake
d. Chronic sustained sympathetic nervous system activation
e. Hypokalaemia

2. Acute activation of the classical RAAS system is associated with physiologic and pathophysiologic responses. Which ONE of the following responses in UNLIKELY to follow activation of this system?

a. Sodium retention
b. Vasodilation
c. Hypertrophy
d. Fibrosis
e. Oxidative stress

3. Which ONE of the following peptides is the major effector molecule of this system?

a. Angiotensin I
b. Angiotensin II
c. Angiotensin 1,7 (Ang1.7)
d. ACE
e. Angiotensinogen

4. Which ONE of the following is NOT part of the alternative RAAS pathway?

a. Angiotensin 1,7 (Ang1,7)
b. MAS receptor
c. Angiotensin II binding its type 2 receptor (AT2R)
d. Aldosterone
e. Neprilysin (NEP) pathways

5. Which ONE of the following breeds has NOT been studies for a polymorphism in the canine ACE gene?

a. Cavalier King Charles Spaniels
b. Chihuahuas
c. Dachshunds
d. Irish Wolfhounds
e. Doberman Pinschers

6. Which ONE of following criteria does NOT fit with ACVIM stage B2 myxomatous mitral valve disease?

a. LA/Ao ratio > 1.6
b. LVIDDN > 1.7
c. VLAS > 2.5
d. VHS > 10.5
e. FS > 43%

7. Which ONE of the following is NOT a cause of aldosterone breakthrough (ABT) during ACE-inhibitor or ARB therapy?

a. Persistent Angiotensin II production by the chymase pathway
b. Upregulation of other aldosterone secretagogues such as ACTH
c. Ang 1,7 binding to its MAS receptor to activate the alternative pathway
d. Decreased aldosterone metabolism and
e. Decreased aldosterone clearance

8. Which ONE of the following statements regarding preclinical MMVD is INCORRECT?

a. Comprehensive RAAS inhibition with Cardalis^{R} was able to delay time to CHF
b. Comprehensive RAAS inhibition with Cardalis^{R} was able to significantly reduce cardiac size
c. Comprehensive RAAS inhibition with Cardalis^{R} was able to significantly reduce cardiac biomarkers
d. Pimobendan is indicated in preclinical stage B2 heart disease
e. Furosemide therapy is NOT indicated in stage B2 heart disease

9. Which ONE of the following statements regarding stage C (current of previous CHF) MMVD is INCORRECT?

a. ACEi and furosemide therapy demonstrated morbidity and mortality reductions.
b. The EPIC trial demonstrated superiority of Pimobendan over ACEi therapy.
c. The BESST study demonstrated end point benefit for dogs given comprehensive RAAS suppression with Cardalis^{R} compared with benazepril alone.
d. The VALVE study concluded that there was benefit when adding RamiprilTM in dogs with CHF treated with pimobendan and furosemide.
e. The most recent 2019 ACVIM consensus guidelines advocates for the use of quadruple therapy (Furosemide, Pimobendan, Benazepril and Spironolactone).

10. Which ONE of the following statements regarding stage D (refractory CHF) MMVD is INCORRECT?

a. Furosemide dosage should be increased or used more frequently.
b. Pimobendan dosage should be optimized or increased.
c. Amlodipine can be added for increased afterload reduction.
d. Sildenafil could be added if exertional syncope and PH is diagnosed.
e. A combination of benazepril and spironolactone (Cardalis^{R}) is contra-indicated.

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When a Cough is Not What it Seems

Dr Liesel van der Merwe*BVSC MMedVet(Med) and Dr Ryan Nel BVSc
*Department of Companion Animal Medicine,
Faculty of Veterinary Science, University of Pretoria

Every effort should be made, in the beginning of a patient evaluation, the ensure that the more advanced diagnostic steps are built on a solid foundation of a good history and a good basic clinical examination

The diagnosis of the specific aetiology of a canine respiratory disease can be challenging for veterinarians. All too often we are faced with hurdles such as trusting in the ability of the owners to differentiate and classify sounds made by their pet and financial constraints limiting imaging and more invasive techniques. A well planned and defined approach is thus crucial.

Similar to a neurological examination, localization of the lesion is the first step (Johnson, 2010). This can be achieved by discussing the different respiratory sounds with the owner, in conjunction with the history, including the time of day the noise worsens, exercise tolerance, and any physical or habitus changes. With this information, veterinarians can gain valuable clues for localization.

Table 1: *Characterisation of respiratory sounds and their region of origin.*

Respiratory noise	Anatomical Localization	How to describe to owners
Cough	Dry: usually Laryngeal or trachea Wet: usually airways and parenchymal	Typical coughing sound (Crucial to differentiate wet from dry)
Wheezes	Associated with airway narrowing, stenosis, or obstruction	High pitched sound usually originating from the laryngeal-trachea area. Could be inspiratory or expiratory.
Stridor	Often associated with laryngeal pathology and BOAS[a] conformational abnormalities.	A rough, vibrating snoring sound originating with inspiration.
Stertor (snoring)	Associated with nasal and nasopharyngeal disease	Typical snoring sound. The vibration of structures in the nasopharyngeal airway with air movement - inspiratory and expiratory.
Gagging	Pharyngeal irritation	A gagging sound in dogs is a retching sound made when a dog opens its mouth wide due to irritation or a blockage in its throat or respiratory system.
Reverse sneeze (paroxysmal respiration)	Any irritation to the nose, sinuses, or back of the throat can trigger an episode of reverse sneezing. Dogs with narrow nasal passages are more commonly affected.	The dog makes a snorting sound and seems to be trying to inhale while sneezing.

BOAS - Brachycephalic upper airway Syndrom

During the diagnosis of respiratory disease, accurate owner descriptions of the sound produced is essential. However, owners may have difficulty differentiating specific sounds their pets produce. With cell phones and videos most owners should be able to record their pet having an "episode". The vet can also mimic characteristic sounds associated with various respiratory pathologies to facilitate localisation or make a similar type of recording. Taking the time to accurately describe and localise the cough, gag or retch will ensure the diagnostic process is on the right track.

Nasal diseases encompass a wide range of conditions, each with potentially overlapping clinical signs. This complexity presents a significant challenge for veterinarians, due to the necessity of having to differentiate the underlying cause based on the limited and often nonspecific signs observed. Traditionally, the approach involves generating a differential diagnosis by considering the shortlist of potential causes associated with each individual sign. However, as this case will illustrate (and as most clinicians are aware), real-world diseases don't always strictly follow textbook presentations. The objectives of this report are to show the importance of starting a respiratory case by correctly identifying the noise!

Case presentation

A 13-year-old castrated male Yorkshire terrier presented with a 3-month history of a progressively worsening cough. The cough was worse at night and when drinking water. The owner also reported that the dog was showing open mouth breathing at times and exercise intolerance. The patient had a history of dental disease and several teeth had been extracted. No history of prior respiratory infections or heart disease was reported by the owner. The dog had also developed a serous nasal discharge that began unilaterally and then progressed to involve both nostrils and was intermittent.

The owner had sought treatment from two different veterinarians, but the cough had only transiently improved. The first veterinarian treated the dog for chronic bronchitis with a course of short antibiotics and prednisolone and the second veterinarian treated the dog for a collapsing trachea with a 2-week course of prednisilone. Response to treatment was suboptimal so the patient was referred to the Onderstepoort Veterinary Hospital for further investigation. We were advised that this case had financial constraints.

On presentation at Onderstepoort Outpatient clinic our focussed clinical examination revealed a not tan healthy appearing alert active patient with a BCS of 4/5. The owner was very distressed at what she perceived as her dog's inability to breathe, especially at night, when he the frequency of open mouth breathing and the frequency of coughing increased. This causing the client to also not get a proper night's sleep. This nasal discharge was not present upon presentation at our practice and lung sounds were normal with no discomfort or cough elicited on palpation of the trachea and laryngeal area. Heart auscultation demonstrated a normal respiratory sinus arrythmia with no murmurs present. Mild periodontal disease was seen on the oral examination. Only the left maxillary canine was present and this tooth showed moderate peroiodontal disease with receding gingiva and exposure of cement.

The first part of our diagnostic approach was to determine from the client and history if this was indeed a cough, or some other kind of upper airway sound, of which there are many. The client did not have any videos of the event, which are generally extremely helpful in cases where clinical signs are intermittent. We thus tried to mimic sounds from the various areas of the upper airway to see if she could recognise any specific sound. It is not that difficult to make the various sounds, although you do have to be prepared

not appear as dignified as usual. The movement of air varies between inspiration and expiration with these sounds as well and the owners can then much more easily recognise the noise their pet is making. By localising the area of the "lesion" in cases where the clinical sign is intermittent and where it is not clear on a clinical examination your diagnostic approach is more targeted. So was the cough indeed a cough? The lesion was localised to the nasopharyngeal area based on owner identification of the sound she was hearing. The open mouth breathing was also determined to be due to obstruction of the nasal passages, as the animal was not cyanotic or in distress at these times and this too was intermittent.

Our initial differentials after localising the lesion to the nasopharyngeal region were neoplasia, Inflammatory rhinitis (Lymphocytic-plasmacytic rhinitis, allergic rhinitis or hyperplastic rhinitis), periodontal disease, a nasal foreign body, nasoharyngeal stenosis or aspergillosis. The last two conditions were considered less likely, and stenosis was ruled out after as the region was visualised during intubation. Nasal radiographs were requested but we were able to obtain a CT scan as a test-run for a new CT machine. The CT demonstrated pathology in the left nasal passage (Fig1). There was loss of bone in the palatine process of the left maxilla around the root of the left canine. The nasal conchae adjacent to this region showed lysis of the conchal cartilage and a soft tissue density, which was poorly contrast enhancing, in the rostroventral portion of the left nasal passage. The radiographic conclusion was a left nasal lesion consistent with rhinitis and periodontal disease with signs of a tooth root abscess the canine and adjacent incisor. The soft tissue density was considered consistent with rhinitis, but a biopsy was recommended to exclude neoplasia.

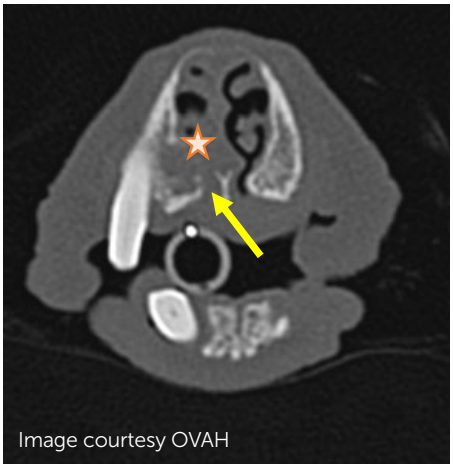

Figure 1: Transverse view or rostral nasal passages of the patient Bruno. The yellow arrow indicated the bone lysis around the root of the left upper canine. The orange star indicates the hyperplastic respiratory epithelium filling the Left nasal passage

Biopsies were taken of both nasal passages without endoscopic guidance. Two biopsy samples of each nasal passage were taken, using the CT scan as a guide to measure the appropriated depth of the lesion. The biopsy of the left nasal passage confirmed purulent rhinitis. The respiratory epithelium showed a severe inflammatory reaction (neutrophils, lymphocytes and some plasma cells) and no bacteria were seen. The respiratory epithelium of the right nasal passage was normal. Our final diagnosis, confirmed by histopathology, was chronic inflammation, not infection, of the nasal passages, secondary to a tooth root abscess of the last remaining maxillary canine and adjacent incisor. We advised the owner that the offending teeth should be extracted. The owner decided to take the patient back to their local vet for further treatment and the removal of the teeth, citing cost reasons. A follow up phone call several months later - the tooth had still not been removed but the clinical signs had resolved on a course of antibiotics and non-steroidal anti- inflammatory drugs.

Differential diagnosis

Discussion – when is a cough NOT a cough!

DISCUSSION

Small breed dogs commonly present with what owners describe as a cough. It is up to the veterinarian to ensure that they do not proceed with the consultation based on an owners' interpretation of a clinical sign. An owner is not trained to make this interpretation. Asking more details about **why** the owner thinks that this is the problem, taking the time and a step back to obtain these seeming inconsequential details, will be invaluable in directing the remainder of the consultation and diagnostic workup. This happens in many scenarios - owners will perceive their dog as being constipated as they misinterpret the presence of tenesmus due to colitis. They may interpret a syncope as a seizure event. It is the duty of the veterinarian to get to the bottom of the clinical presenting sign and not just move on from what the owner diagnoses. This takes a bit of patience but will repay you with a clearer diagnostic route and improved outcomes.

Common causes of in small breed dogs which present with a cough are a collapsing trachea or mainstem bronchi, chronic bronchitis and mitral valve insufficiency with congestive heart failure. The cause of their respiratory issues may be one or possible combinations of more than one of these underlying causes. Effective management may rely on treating more than one problem and balancing the influences of one over the other. Congestive heart failure can be excluded as excluded as a cause of coughing, despite the presence of a heart murmur in a patient, when a respiratory sinus arrythmia is present. This is caused by the heart slowing down in the expiratory phase of breathing when vagal tone is increased. In a patient with CHF with pulmonary oedema causing coughing, the patient is in sympathetic overdrive and show tachycardia with no decrease in heartrate during expiration. Remember the mere presence of a heart murmur is not indicative of heart failure!

As this case demonstrates, it is important to first confirm the actual clinical sign the patient is presenting with. The patient signalment and the cough being worse at night and when drinking water does fit with the suspected diagnosis of both chronic bronchitis and tracheal collapse. These diseases have overlapping clinical signs. (Tappin, 2016) Tracheal collapse presents with a harsh goose-honking cough and although the worsening with exercise and drinking water does fit here, as does the breed, the sudden onset and presence of intermittent open mouth breathing without concurrent severe respiratory distress doesn't fit. The patient also never

showed any cyanosis. Chronic bronchitis was a more likely differential, but accurate diagnosis does require typical radiographic changes and a broncho-alveolar lavage (BAL) with cytological evaluation.

One of the most rewarding diagnostic procedures in patients presenting with a gag or snort or a nasopharyngeal "schnark" is to anaesthetise the patient and evaluate the oral and nasopharyngeal cavity. This cannot be done properly with the patient awake or even just sedated. A light source, a dental mirror, warmed to prevent fogging, and a spay hook to lift the soft palate can be used to evaluate the nasopharyngeal region. This will allow visualisation of masses extending caudally from the nose into the nasopharynx as well as foreign material such a blade of grass, neoplasia in the tonsillar region or laryngeal pathology such as laryngeal paralysis. Trauma from stick injury or eating bones can occur far back in the epiglottal and laryngeal region. Nasal pathology can present with slightly different clinical signs if it occurs more rostrally or more caudally. Disease positioned rostrally in the nasal passages will have a more obvious soft stertor and possible nasal discharge. Disease positioned more caudally into the nasopharyngeal area may present with a more gagging type of sound as discharge and mucous moves into the nasopharynx (post nasal drip) and as proliferative tissue obstructs the passage. The various nasal diseases may also have overlapping clinical signs. In this case the clinical signs and history we obtained did not correlate perfectly with what most literature views as the common clinical signs for our diagnosis (Johnson, 2010).

There were no signs of facial swelling or pain in the area nor a muco-purulent nasal discharge, neither did the owner. A muco-haemorrhagic nasal discharge may indicate a more destructive type of lesions such as neoplasia, aspergillus or lymphocytic-plasmacytic rhinits. A serous discharge is more indicative of inflammation and purulent will indicate infection or an inflammatory response. Inflammatory cells are present in high numbers in immune mediated conditions. Since accurate diagnosis depends on histopathology, obtaining a representative tissue sample is essential. There are three potential methods for sample collection, each with its own advantages and disadvantages. Rhinoscopy-guided biopsy offers a higher chance of retrieving a diagnostic sample but is also the most expensive option and requires specialized equipment (Harris et al., 2014). However, if nasal neoplasia is highly suspected, imaging-guided or blind biopsy can still be effective. To maximise the change of a representative and diagnostic sample multiple biopsies, using the largest biopsy forceps you can for the patient size, should be obtained. (Harris 2014).

Small breed dogs also often have severe periodontal disease. Much more so than large breed dogs. Infection of the roots of molars and premolars will present with swelling and draining sinus tracts under the eye whereas the infected canine teeth roots will affect the nasal passages. Dental disease and its proximity to the nasal cavity can predispose dogs to odontogenic nasal infections (Stepaniuk and Gingerich, 2015). The roots of the upper incisor, canine, premolar, and molar teeth lie very close to the nasal cavity and maxillary sinus, with only a thin layer of bone separating them (De Rycke et al., 2003). This close anatomical relationship means that inflammation and infection in these teeth can easily spread contiguously the nasal cavity.

The mechanism for this spread is explained by the translocation theory (Legert et al., 2004, Stepaniuk and Gingerich, 2015). They theorise that the respiratory mucosa in close proximity to the infected tooth root, becomes inflamed due to the inflammatory process in the tooth root itself. The local innate immunity is thus compromised by the inflammation, predisposing the tissue to infection (Legert et al., 2004). This would explain why in this case there wasn't purulent nasal discharge, but rather a serous discharge due to inflammatory changes in the tissue, but no actual infection. When this Inflammation persists for longer periods of time, as it would with a tooth root inflammation and infection, we start to see hyperplasia of mucosal tissue. The glandular cells within the respiratory epithelium will thus also be more numerous and there will be an increased production of mucus. The chronicity of the tooth root inflammation and infection is clear from the bone lysis on the CT The pathophysiology of hyperplasia is complex, but a simplified definition is that it is an abnormal proliferation of normal mucosal cells (Furtado and Constantino-Casas, 2013). Hyperplasia can be subdivided into simple and complex or atypical; the classification is made by examining architectural crowding and nuclear atypia (Kubyshkin et al., 2016). Simple has been found to be easily resolvable, whereas complex is a greater challenge. Complex or atypical hyperplasia has a higher risk of progressing to neoplasia.

In this patient the chronic inflammation and likely infection of the left maxillary canine had causes bone lysis and chronic inflammation of the bone and adjacent nasal tissue. This had in turn become hyperplastic resulting in an intermittent nasal discharge as well as discomfort causing the gagging and coughing sounds the patient had been exhibiting. By addressing the infected tooth the problem in the nasal passages would be resolved.

Conclusion

The "cough" in this case was caused by an infected maxillary canine tooth root. Clearly, localizing the lesion at the outset of a respiratory case is vital, minimizing the risk of misdiagnosis. Every effort should be made, in the beginning, to make sure the more advanced diagnostic steps are built on a solid foundation of a good history and a good basic clinical exam. Take a little extra time to ask considered questions which show insight rather than just ticking off a list.

Dental disease and sub-clinical infection of the tooth root of the maxillary canine especially, should not be overlooked as an important potential cause of nasal disease in especially small breed dogs. Diagnostic procedures like biopsy and histopathology are crucial for a definitive diagnosis and that this can be performed without access to specialised equipment. These can however be per. Even without access to specialized equipment, a biopsy attempt remains important to be able to perform histopathology.

References are available upon request.

Adapted From: American Veterinarian®, February 2018, pg 14-16

ACVC 2017: Understanding Skin Disease in Cats

By JoAnna Pendergrass, DVM

Although much remains unknown about feline dermatology, one thing is certain: Many skin conditions are related to internal disease.

When it comes to skin disease, cats are not just small dogs. This was a key point from a presentation on skin disease in cats by Candace A. Sousa, DVM, DABVP (Emeritus; Canine & Feline Practice), "Cats are cats," she said, "and their disease processes need to be approached from a cat point of view."

Feline skin diseases may involve a severe underlying illness and require a thorough diagnostic evaluation, including a detailed history and physical examination. Getting a diagnosis generally makes therapy and prognosis "easy,

Unlike internal medicine, veterinary dermatology has limited diagnostic tests (skin cytology, trichogram, biopsy) and analytic tools (Wood's lamp, microscope) available. Trichograms, in particular, are performed more often in cats than in dogs. Veterinarians should charge for each skin diagnostic test, including simple scrapings, Dr. Sousa said, emphasizing that clients should pay for the expert opinion and knowledge. In addition to obtaining a history and performing a physical exam, organizing possible skin diseases into categories can help in making a diagnosis. Categories include allergic, bacterial, congenital/ hereditary, endocrine, fungal, immune-mediated, miscellaneous, neoplastic, nutritional, parasitic, and viral diseases.

After making the diagnosis, Dr. Sousa recommended that veterinarians ask owners what they want to do regarding treatment, "What would you like?" or "What are your goals for your cat?". She then provided details on congenital, endocrine, neoplastic, parasitic, viral, and miscellaneous skin diseases that are unique to cats.

Congenital Diseases

Idiopathic Facial Dermatitis of Persian Cats

Also known as "dirty face syndrome," this chronic disease starts when Persian cats are young and causes black crusts on the face, particularly in the folds and periocular area. Affected cats may traumatize themselves. In addition, ceruminous otitis may be present concurrently. Treatment response is typically poor.

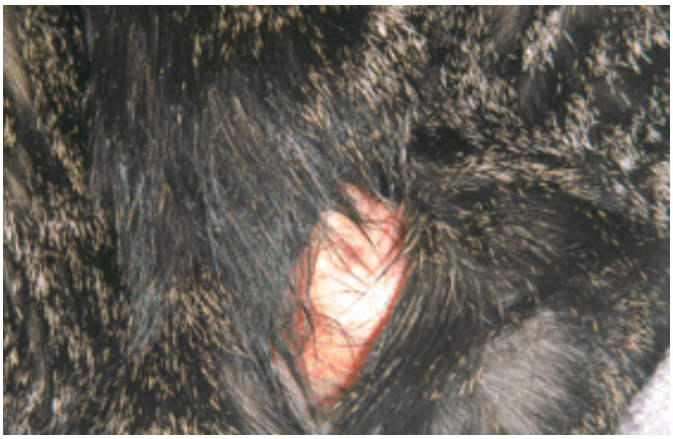

Figure 1: Skin fragility.

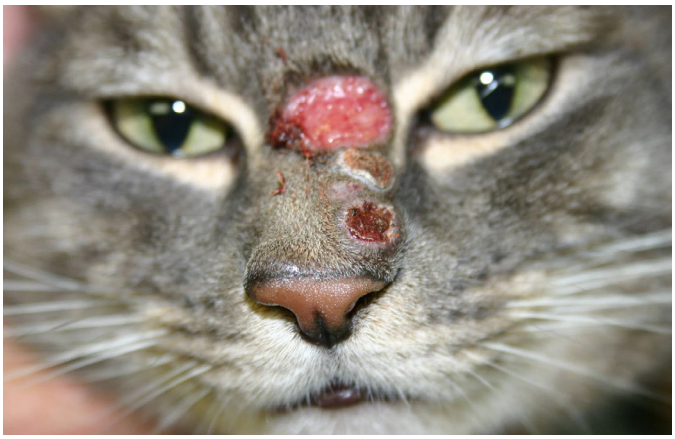

Figure 2: Facial herpes

Proliferative and Necrotizing Otitis

Proliferative and necrotizing otitis is rare, has an unknown aetiology, and typically affects 3- to 6-month-old kittens. Physical examination of the ears reveals large tan or dark coalescing plaques on the concave surface of the pinnae and external ear canals; the ears may also contain comedones. Histopathologic findings include acanthosis, follicular keratosis, and hair follicle outer sheath hyperplasia and keratinocyte necrosis. This skin condition can be treated with topical 0.1% tacrolimus. Oral prednisolone can also be used, but its effectiveness in treating this disease remains unknown. The prognosis is poor.

Endocrine Diseases

Feline Skin Fragility

Typically a marker of feline hyperadrenocorticism, feline skin fragility generally occurs in older cats. With several potential causes (progestin therapy, excessive administration of corticosteroids), this condition results in a collagen deficiency that makes the skin extremely thin. Examining an affected cat requires very gentle handling to avoid tearing the skin. Interestingly, not much bleeding occurs when the skin tears.

Also, unlike in dogs, alopecia is typically not present in affected cats. Biopsies, although not practical because of the skin's thinness and fragility, often show collagen deficiency and severe epidermal, dermal, and follicular atrophy. Dr. Sousa recommended that adrenal function be tested during the diagnostic evaluation. The prognosis is grave, especially if the underlying cause cannot be identified. (Figure 1)

Neoplastic Diseases

Paraneoplastic Alopecia Associated With Visceral Neoplasia

This skin disease has an acute onset and generally affects cats aged 10 years and older. There is complete alopecia of the ventrum; the skin appears shiny, potentially due to stratum corneum exfoliation. Scaly footpads are a hallmark of this disease. Clinical signs (weight loss, anorexia, and lethargy) suggest an underlying systemic disease. Abdominal ultrasound may reveal visceral neoplasia, which is usually a pancreatic adenocarcinoma. Skin biopsy findings are severe follicular and adnexal atrophy; alopecia-associated epidermal thickening can also aid in diagnosis. It is thought that the tumour releases factors, such as cytokines, that cause follicular shrinkage. The prognosis is grave.

Exfoliative Dermatitis and Thymoma Exfoliative Dermatitis

This rare form of dermatitis, secondary to an underlying thymoma, causes scaly and erythematous dermatitis that begins on the head and neck and eventually becomes generalized. The dermatitis is nonpruritic and affects middle-aged and older cats. It is thought that defective thymic lymphocyte selection results in an abnormal immune response to keratinocyte antigens, causing keratinocyte apoptosis.

Histopathology findings include cell poor interface dermatitis (dermatitis at the dermo-epidermal junction), mild hydropic degeneration of basal cells, and keratinocyte apoptosis. This feline skin disease is treatable with surgical resection of the tumour.

Metastatic Bronchogenic Adenocarcinoma

Older cats with lung tumours may develop a condition known as "lung digit." syndrome. Affected cats typically show no signs of respiratory disease before skin abnormalities appear. The tumour metastasizes to the capillaries in the distal extremities and toes, causing an acute onset of haemorrhage in the digits. Without confirmation of a lung tumour, this skin disease may be mistaken for inflammatory pododermatitis. Topical or systemic corticosteroids can be used to relieve oedema and general discomfort, but the prognosis is poor.

Parasitic Diseases

Mosquito Bite Hypersensitivity

This seasonal disease is not that uncommon in cats and is worth knowing about. Mosquitoes may bite thinly haired areas like the bridge of the nasal planum, the pinnae, and footpads, creating immediate and late-phase hypersensitivity reactions to the mosquito salivary antigens. Typically, the initial bite will develop into a wheal within 20 minutes; papules appear in 24 hours and become encrusted within about 48 hours.

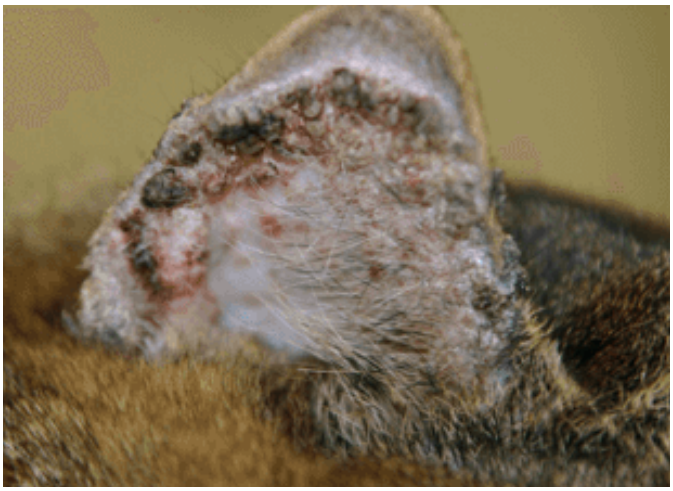

Figure 3: Bowen disease on the inner right pinna.

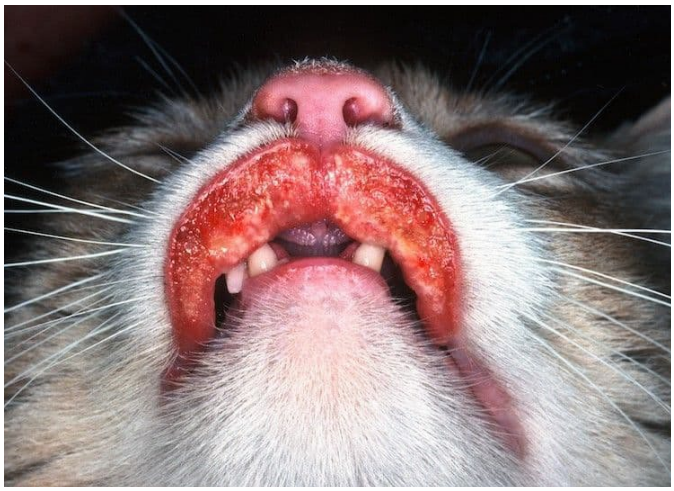

Figure 4 Indolent ulcer

Cats that are chronically affected by mosquito bite hypersensitivity develop lesions such as scaling, alopecia, and pigment changes. Histopathology, which is needed to confirm this skin disease, demonstrates highly eosinophilic dermatitis. (Ed - The nasal planum lesions may appear similar to squamous cell carcinoma is severe. An FNA may also be helpful.) Cats that become hypersensitive to mosquito bites are usually affected repeatedly. Not all cats that get bitten by mosquitoes develop a hypersensitivity reaction.

Management options include confining the cat in a mosquito-free environment and using topical mosquito repellent on areas susceptible to bites (ed - be careful of toxicity). Systemic corticosteroid treatment is indicated for cats that cannot be confined.

Viral Diseases

Feline Immunodeficiency Virus—Related Dermatitis

Cats with either feline immunodeficiency virus (FIV) or feline leukaemia virus may get skin disease. FIV-related skin problems include abscesses, skin and ear bacterial infections, and mycotic infections. Some cats with FIV develop nonpruritic, generalized, papulocrusting lesions with concurrent alopecia and scaling, which are most severe on the head and limbs.

On histopathology, FIV-related dermatitis demonstrates hydropic interface dermatitis and giant keratinocytes. To date, treatment options for this skin disease have not been successful.

Cutaneous Herpes Virus Infections

This condition typically affects older cats (8 to 9 years old) and can cause ulcerative and necrotizing facial dermatitis. (Ed - often with severe pruritic due to trigeminal nerve involvement) The nasal planum, bridge of the nose, and periocular skin are commonly affected.

Histopathologic findings include ulcerated epithelium, intranuclear inclusion bodies in keratinocytes (indicating herpes virus infection), multinucleated giant cells and keratinocytes in the surface and follicular epithelium, and interstitial mixed inflammatory dermatitis with eosinophils.

Systemic antibiotics are indicated to treat secondary bacterial infections, and acyclovir can help treat the viral infection. Corticosteroids can activate the virus and thus are not recommended for treating cutaneous herpes virus infections. Lysine has been proposed as a treatment option but, according to Dr. Sousa, hasn't shown much efficacy.

Human alpha-interferon is another option for treating cutaneous herpes virus infections, but cats eventually develop antibodies to it, making it less effective over time. None of the treatment options will get rid of the herpes virus carrier site within an affected cat's body, Dr. Sousa said. (Figure 2)

Multicentric Squamous Cell Carcinoma In Situ (Bowen Disease)

Bowen disease affects cats older than age 10 years and is triggered by papillomavirus. This slowly progressive disease causes eroded crusted papules and plaques that are frequently found on the head and neck but can also appear on the shoulders and forelegs. This disease is not solar induced and does not metastasize.

To obtain a diagnosis, take a 6-mm punch biopsy of the dermis and epidermis. Be warned that epidermal abnormalities may prevent proper skin healing.

Epidermal dysplasia without basement membrane destruction is apparent on histopathology. Surgical laser excision is the preferred treatment, with radiation and systemic retinoids recommended as adjunctive therapies. Vitamin A therapy may be useful for keratinocyte proliferation. (Figure 3)

Miscellaneous Diseases

Indolent Ulcer ("Rodent Ulcer")

This skin disease has no age, sex, or breed predilection. Well-circumscribed, red-brown ulcers with raised borders usually form on the upper lip. Interestingly,

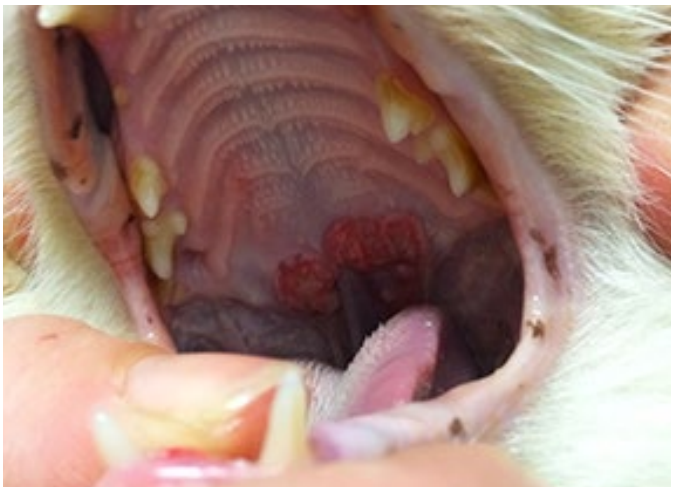

Figure 5: Linear granuloma on the palatine mucousa of a cat

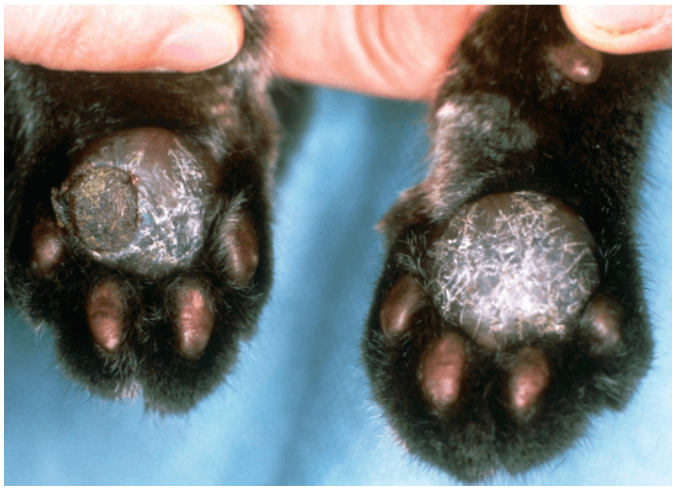

Figure 6: Plasma cell pododermatitis.

ulcers are neither eosinophilic nor granulomatous on histopathology; peripheral eosinophilia may be present. Chronic, excessive licking due to allergic or pruritic stimuli is thought to cause the ulcers. If the ulcers are recurrent or refractory, Dr. Sousa recommended testing for food allergies or flea allergy dermatitis.

Early treatment is best; however, systemic glucocorticoids are often ineffective. Systemic antibiotics and progestational drugs can be useful in refractory cases. Treatments such as cryosurgery and laser therapy are primarily cosmetic, Dr. Sousa said. (Figure 4)

Eosinophilic Granuloma

The clinical description of this lesion varies with location. Eosinophilic granulomas are ulcerated on the lips or within the oral cavity (back of palate, palatal arches) (Figure 3) ; on the caudal thigh, the granulomas are linear, raised, circumscribed, and yellow-to-pink plaques.

On histopathology, eosinophils are attached to the collagen. Linear granulomas can regress spontaneously. Granulomas on the lips or within the oral cavity can be treated with corticosteroids. (Figure 5)

Eosinophilic Plaques

This common feline skin condition also is not associated with a particular age, sex, or breed. Affected cats are extremely pruritic and resort to excessive licking and grooming, causing plaques.

Most often there is an underlying allergic skin disease. The ulcerated lesions are typically found on the abdomen and medial thighs and are well circumscribed, raised, round or oval, and erythematous. Many cats have a circulating peripheral eosinophilia. Consider flea and food allergies. Treatment is the same as for indolent ulcers.

Plasma Cell Pododermatitis

This rare condition has no known aetiology. Initially, the lesions, which frequently affect the central carpal or tarsal footpads, are soft and painless with loss of the surface architecture. Over time, the pads become ulcerated and develop secondary infections, causing pain, lameness, and regional lymphadenopathy. Affected cats are otherwise healthy.

Histopathology provides a definitive diagnosis. Spontaneous regression occurs occasionally, while other cases benefit from systemic glucocorticoid or prednisolone treatment; response begins within about three weeks and peaks within about 3 months. Tetracycline or doxycycline (5 mg/kg twice daily) can also be used. Surgical excision is recommended for ulcerated footpads to improve healing and prevent recurrence. (Figure 6)

Injection-Site Panniculitis

Subcutaneous injections of rabies vaccine, praziquantel, and methylprednisolone acetate have reportedly caused panniculitis in the dorsal cervical region and midscapular area. Occurring several months after injection, the skin reaction can range from inflammation and alopecia to ulceration and fat necrosis.

Disease management can be difficult. Treatment options include intralesional or systemic glucocorticoids, identification and treatment of underlying allergies, and surgical excision.

Self-Induced Alopecia ("Fur Mowing")

Affected cats often present with partial, symmetric, or complete alopecia on areas accessible to licking. Excessive licking may be due to a psychological disorder or underlying allergy.

Trichograms of the distal hair tips will help demonstrate that the alopecia is self-induced. If allergic and inflammatory conditions have been ruled out, medical treatment options—all oral, include phenobarbital (1-2 mg/kg once or twice daily), amitriptyline (5-10 mg/day), and buspirone (0.5 mg/kg/day), which may be also useful for psychogenic alopecia.

Tips for When Using Lidocaine Local Anaesthesia

To remove the sting of lidocaine: add sodium bicarbonate (0.9 mL lidocaine, 0.1 mL bicarbonate) to the local anaesthetic.

To reduce bleeding: prerinsing the syringe with epinephrine to reduce bleeding.

Eosinophilic Granuloma Complex in Cats and Dogs (Proceedings)

October 1, 2011 : By Stephen D. White, DVM, DACVD

Treatment of feline EGC

Traditionally, these diseases have been treated with intra-muscular injections of methylprednisolone acetate (Depomedrol®:UpjohnPharmacia) at 4 mg/kg, given once every two weeks for three injections. The author uses this treatment ONLY IF: the disease has been confirmed by biopsy, there is no evidence of, or no ability to investigate, an underlying cause (especially feline herpes virus dermatitis), and this protocol is only used twice a year at most.

More frequent use of this protocol will lead to the development of diabetes in a very high percentage of cats. If further corticosteroid treatment is needed, oral prednisolone, initially at a dosage of 1 mg/kg q12 h may be used, then tapered to the lowest effective dosage.

In an attempt to avoid corticosteroids, the following treatments have been reported/utilized:

In one study, 4 of 4 eosinophilic granulomas, but 0 of 2 eosinophilic plaques were shown to respond to administration of essential fatty acids (DermCaps®:DVM Pharma¬ceuticals). Dosages approximated the manufacturer's guidelines. These are well-tolerated medications.

Cyclosporine: a good response to a dose of 25 mg/cat was seen in 6 cases of eosinophilic plaque and 3 cases of oral eosinophilic granuloma in one report2 In three cases of indolent lip ulcers, the response was less impressive. Another more recent study confirmed these results with a higher dosage range of 10-12.5 mg/kg.

If dermatophytes are present, fluconazole or itraconazole (10 mg/kg q24 h) should be used – previous anecdotal reports of lip ulcers' responses to griseofulvin may in fact have been due to an underlying M. canis infection.

Clavamox has been reported as effective in the treatment of eosinophilic plaques.

Herpes Virus Dermatitis:

Herpes virus dermatitis is probably under-reported. Persistent ulcerative to necrotizing lesions develop on the face, most typically the nose6 Affected cats often have a history of stress, glucocorticoid administration, or chronic ocular or respiratory disease suggesting this skin disease is associated with reactivation of latent herpes virus infection. However, the respiratory signs may be subtle, and owners should be questioned carefully to determine this facet of the history.

Treatment:

Subcutaneous alpha interferon (Intron A®:Schering; 1 vial contains 3 million units) has appeared beneficial in some cases, sometimes dramatically so. Dosage is approximately 1.5-2 million units/m2 (a 5 kg cat has a m2 body area of 0.29 meters, and thus would receive a dose of 290,000-580,000 units) given sub¬cutaneously three times weekly for at least six weeks. Side effects are uncommon but malaise may be seen. May be difficult to obtain.

Famcyclovir 40mg/kg tid thus a 5kg cat = ½ of a 500mg tablet tid. Effective – now available as a generic.

EchiTab Snakebite Antivenom

History and Introduction of EchiTab Snakebite Antivenom by MicroPharm

Mike Perry
African Reptiles & Venom

MicroPharm, a British biopharmaceutical company founded in 1984, specialised in developing antivenoms and antibodies to treat snakebites and other venomous stings. EchiTab, one of their flagship products, is a polyvalent antivenom specifically developed to treat envenomations from various Echis species (saw-scaled vipers), which are highly venomous snakes found in parts of Africa, the Middle East, and South Asia.

The need for an effective treatment against Echis species arose because their bites are a significant cause of snakebite mortality in Africa due to their potent hemotoxic venom, which can cause severe bleeding and tissue damage. Developing antivenoms like EchiTab was vital to reducing mortality rates in regions where these snakes are common.

Research and Development of EchiTab

MicroPharm's approach to antivenom production involved using hyperimmunised horses. The company would inject these horses with small, safe amounts of snake venom to trigger an immune response. The horses' immune systems produced antibodies against the venom, and MicroPharm would then collect and purify these antibodies to create the antivenom.

For EchiTab, MicroPharm used venom from several species of Echis snakes to create a polyvalent antivenom effective against multiple species. This broad-spectrum approach made EchiTab a valuable medication in areas with diverse viper populations.

Development of EchiTab Plus: MicroPharm and ICP Collaboration

Recognising the need for a polyvalent antivenom that could treat a wider variety of venomous bites in West Africa, MicroPharm partnered with the Instituto Clodomiro Picado (ICP) in Costa Rica. ICP Instituto Picado Clodomiro (ICP), was established in 1970 and became an agency of the University of Costa Rica in 1972, with a link to the microbiology department of that university. This has helped in venom studies to improve antivenoms. ICP antivenoms are well respected in all countries where they are sold. Their production in the 1970s went from 8,000 vials to 112,000 vials in 2016. They have the capacity to meet high demand, which has not been the case with antivenom production in South Africa.

Development and Usage of EchiTab ICP in South Africa in the Veterinary Sector

EchiTab_ICP snakebite antivenom is a vital solution for treating envenomation in animals caused by several dangerous African snakes, including the Puff Adder, Rinkhals, Black Mamba, Snouted Cobra, Mozambique Spitting Cobra, and Cape Cobra. The venom, sourced from African Reptiles & Venom in South Africa, is exported to the University of Costa Rica, where the antivenom is produced under stringent conditions. The final product is imported back to South Africa under Section 21 of the Medicines and Related Substances Act, 1965 (Act 101 of 1965) by Equity Pharmaceuticals. Biofarm, a leading veterinary wholesale distributor of animal pharmaceuticals, distributes EchiTab-ICP nationwide to ensure its availability for animal care.

The antivenom is a welcome addition to the veterinary sector, which has been facing supply shortages. It is available in a 10ml freeze-dried vial, which simplifies storage and transportation as no cold chain is required. By reconstituting the vial with the diluent, veterinarians can administer the life-saving treatment to affected animals via an intravenous administration. The dosage to be administered depends on the severity of the symptoms. With a remarkable five-year shelf life, EchiTab-ICP provides a long-term, reliable solution to snakebite emergencies in the field.

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South Africa. Cell: +27 83 448 8854
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How to Handle Corneal Lacerations in General Practice

Dr Izak Venter
BVSc, MMedVet (Ophthal)
Digital Veterinary Ophthalmology Services
www.dvos.co.za
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Introduction

Corneal lacerations are a common ocular emergency in small animals and equines that, if not managed promptly and appropriately, can lead to significant visual impairment or even loss of the eye.

In dogs and cats they are frequently seen secondary to cat-scratch injuries. In horses, sharp trauma from objects in the environment is most common. Blunt trauma also causes globe rupture, especially in horses.

These injuries can be categorized as follows:

- Partial-thickness lacerations. (Figure 1)
- Full-thickness lacerations: Penetrate the entire corneal thickness and may be further classified by their associated complications:
  - With or without iris prolapse (Figure 2, 3)
  - With or without lens involvement (Figure 4)
  - With loss of intra-ocular contents Figure 5)

While ideal management of corneal lacerations requires specialized microsurgical techniques and equipment, many general practitioners may encounter these cases in their practice.

This article aims to provide practical guidance on assessing the severity of corneal lacerations, performing basic surgical repair techniques, and determining when enucleation may be the most appropriate course of action, particularly in cases where referral to a veterinary ophthalmologist is not feasible.

A common example of a complex corneal laceration that may necessitate enucleation is a full-thickness injury involving the anterior lens capsule.

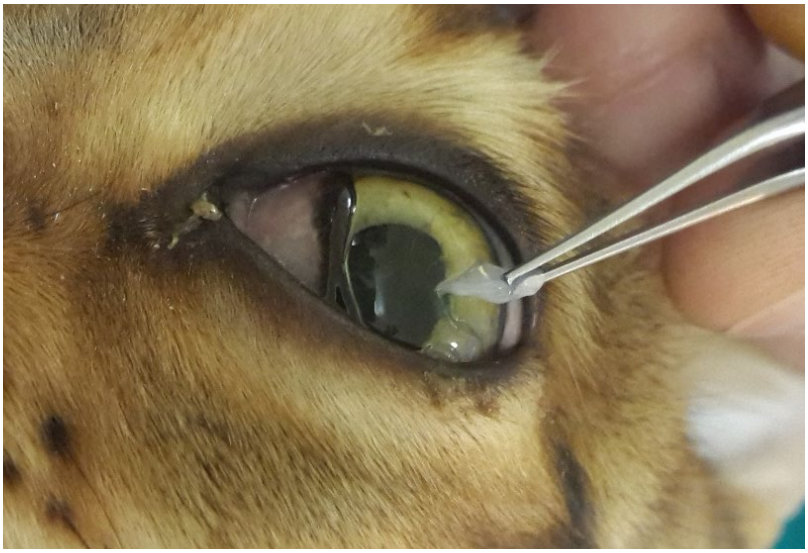

Figure 1: Partial thickness corneal laceration in a cat. See flap of corneal tissue being lifted off the surface.

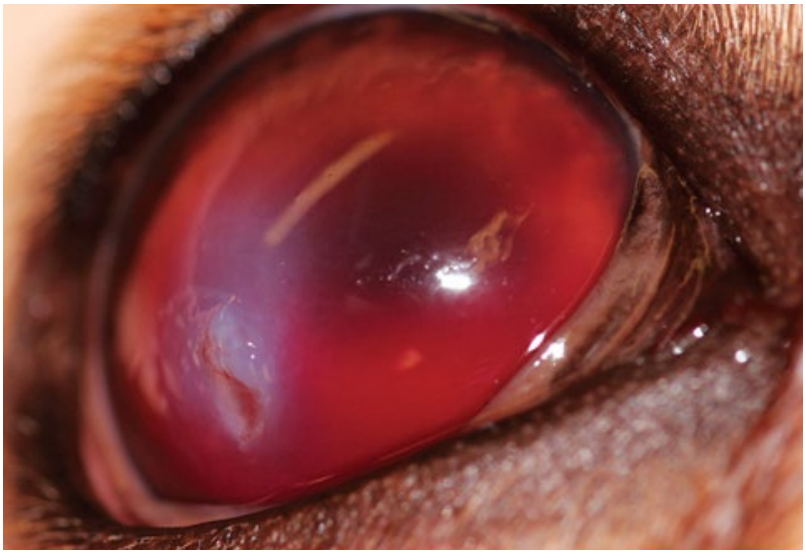

Figure 2: Full thickness corneal laceration. The wound is sealed with fibrin.

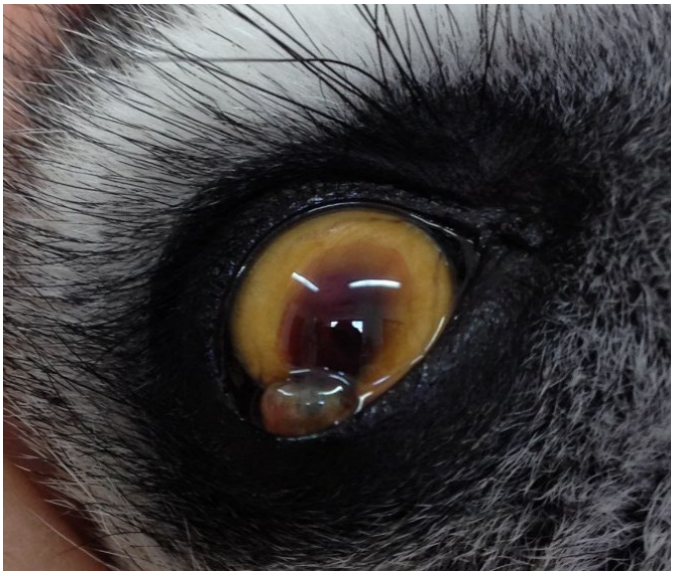

Figure 3: Full-thickness corneal laceration with iris prolapse. Note the secondary changes visible namely severe miosis, corneal oedema, and blood-tinged fibrin in the anterior chamber.

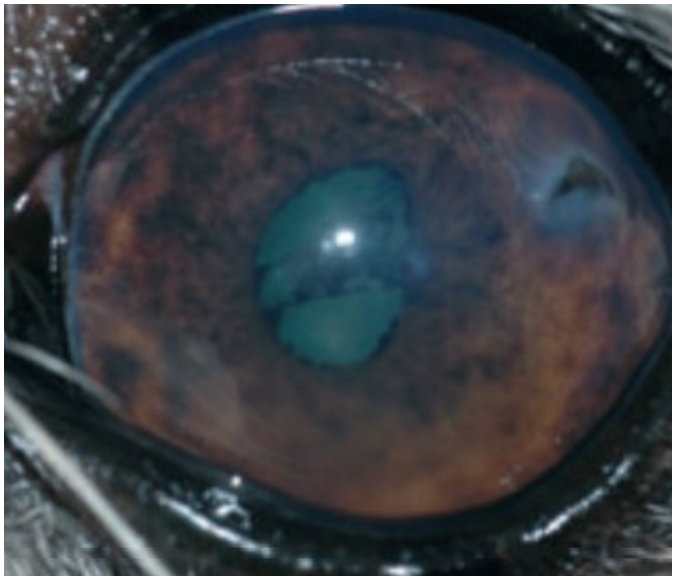

Figure 4: A 2 mm puncture wound in the dorsolateral cornea sealed with a fibrin clot. Plasmoid aqueous, miosis, and lens capsule laceration are visible.
Paulsen ME, Kass PH. 2012. Traumatic corneal laceration with associated lens capsule disruption: a retrospective study of 77 clinical cases from 1999 to 2009. Veterinary Ophthalmology. 15(6):355–368

Prognostic Factors for Corneal Lacerations

The prognosis for successful treatment of corneal lacerations in small animals is influenced by several factors:

- **Species:** Dogs tend to develop more severe post-traumatic uveitis than cats, which can negatively impact visual outcome.
- **Age:** Younger animals (<1 year) are more prone to developing phthisis bulbi compared to older animals.
- **Cause of Injury:** Lacerations caused by organic material (e.g., cat scratches, wood splinters) tend to induce more severe post-operative inflammation and carry a worse prognosis than those caused by clean objects (e.g., metal).
- **Duration of injury:** Prompt surgical repair is crucial. Delayed treatment increases the risk of severe uveitis and poor visual outcome.
- **Depth of laceration:** Partial-thickness lacerations generally have a better prognosis than full-thickness lacerations.
- **Location of laceration:** Axial corneal lacerations, which affect the central visual axis, are associated with a higher risk of visual impairment due to scarring and potential lens involvement.
- **Size of laceration:** Larger lacerations are more likely to lead to severe complications and poorer visual outcomes.
- **Involvement of other ocular structures:** Additional injuries, such as iris prolapse, lens capsule rupture, lens loss, vitreous loss, intraocular haemorrhage, and scleral lacerations, significantly worsen the prognosis for both vision and eye preservation.

Enucleation Considerations

Eyes with extensive corneal lacerations, loss of intraocular contents, and no realistic potential for vision are best managed by enucleation. Before proceeding with corneal repair, it is essential to assess for lens involvement.

Tears in the anterior lens capsule larger than 1.5 mm will inevitably lead to phacoclastic uveitis and require prompt lens removal (phacoemulsification). If phacoemulsification is not feasible, enucleation may be the most appropriate course of action.

Diagnostic Assessment of Corneal Lacerations

To accurately assess the extent of corneal damage and identify any associated ocular injuries, the following diagnostic approach is recommended:

Lens Involvement: If a penetrating injury occurs over the visual axis and the pupil is miotic (constricted), administering a mydriatic agent such as tropicamide can help dilate the pupil and allow for a more thorough examination of the lens.

Ocular oedema or hyphema: In cases of severe corneal oedema or hyphema that obscure visualization of the anterior chamber, ocular ultrasound can be used to evaluate for lens involvement, vitreous haemorrhage, or retinal detachment. These conditions significantly worsen the prognosis for visual recovery.

Pre-operative Medical Management of Corneal Lacerations
Corneal lacerations represent a critical ophthalmic emergency requiring immediate surgical intervention.

While surgical repair remains the cornerstone of treatment, concurrent medical therapy is essential to optimize patient outcomes.

Upon diagnosis, it is imperative to minimize patient restraint to avoid further ocular trauma. The immediate institution of medical therapy should include:

- **Topical Antibiotics:** Prompt administration of broad-spectrum topical antibiotic drops to prevent secondary infection.
- **Systemic Antibiotics:** Systemic antibiotics, such as amoxicillin, are often prescribed to provide additional antimicrobial coverage, especially in cases of significant tissue damage.
- **Systemic Corticosteroids:** Oral prednisolone can be administered to reduce secondary uveitis.

By implementing this comprehensive approach, clinicians can significantly improve the prognosis for patients with corneal lacerations.

General principles when suturing cornea

To ensure optimal healing, the edges of the corneal wound must be precisely aligned. Sutures, regardless of their design, naturally seek the most stable position when tightened. Therefore, correct suture placement is essential for proper wound alignment.Ideally, a monofilament nylon suture (size 9/0 or 10/0) is preferred for corneal suturing due to its low tissue reactivity. However, working with such fine suture material may be challenging for non-veterinary ophthalmologists.

A more practical option is 7/0 polyglactin 910 (Vicryl) with a spatula needle. If this is not available, 6/0 suture material can be used as a last resort. It is crucial to avoid using thicker suture materials, as they can damage the delicate corneal tissue. Corneal sutures should penetrate approximately 90% of the corneal stroma and should be placed at equal depths on both sides of the wound. Full-thickness sutures increase the risk of infection and damage to the delicate corneal endothelium. Superficial sutures only compress a small portion of the wound, leaving the posterior surface unsupported. This can result in a weaker wound and increased post-operative corneal oedema as the exposed, dehydrated stroma absorbs aqueous humor. (Fig 6)

Evenly-spaced sutures in perpendicular wounds ensure proper wound alignment. Unequal suture placement can lead to tissue overlap and irregular astigmatism. (Fig 7). Suturing can be challenging in damaged, swollen tissue. It is essential to include healthy tissue in each suture bite to prevent suture slippage. This is particularly important in cases of keratomalacia.

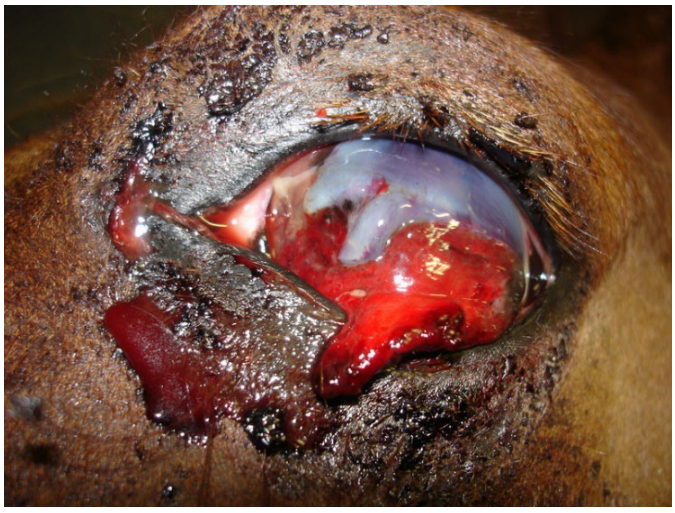

Figure 5: Full thickness corneal laceration in a horse. Due to the extent of the injury, intra-ocular structures are not visible. The prognosis to salvage a globe like this is hopeless.

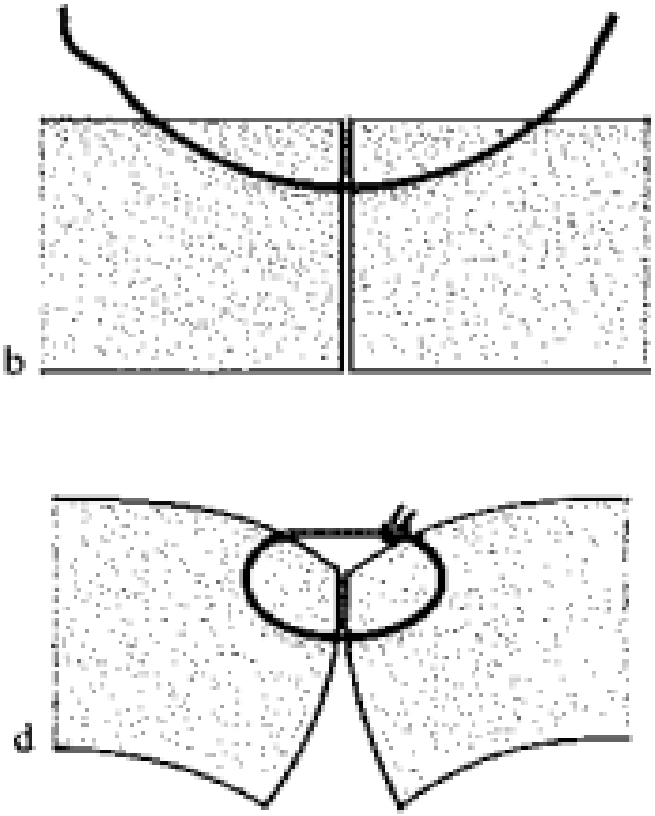

Figure 6 Suture not placed enough leading to an unsupported posterior corneal surface.

Source: George Eisner. Eye surgery. An introduction to Operative Technique.

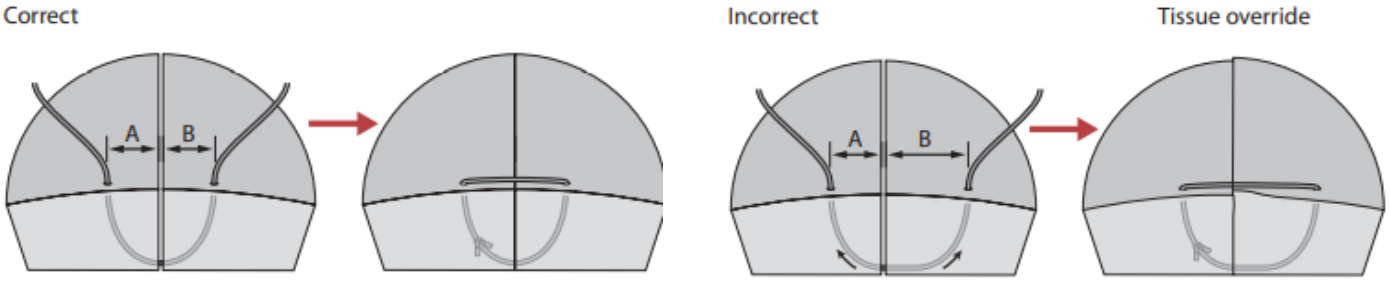

Figure 7: Equal spacing of corneal sutures ensure perfect apposition compared to uneven spacing where tissue override will lead to astigmatism.

Source: Marian S. Macsai (Ed.) Ophthalmic Microsurgical Suturing Techniques

In severe cases, pre-operative treatment with topical antibiotics, oral doxycycline, and topical serum may be necessary to stabilize the cornea before surgery. However, this approach is not suitable for full-thickness corneal lacerations. Sutures bring wound edges together by compressing the tissue within the suture loop. Interrupted sutures create a flat zone of compression within the loop and a triangular zone extending outward on either side. Wound leakage can occur if these compression zones don't overlap enough to keep the wound closed. (Fig 8)

1. Partial-thickness corneal lacerations

Partial-thickness corneal lacerations typically have a good prognosis. Minor, superficial lacerations can be managed conservatively by removing loose tissue under sedation and topical anaesthesia. The wound is then treated as a standard corneal ulcer. Larger, deeper lacerations require surgical intervention under general anaesthesia. The corneal flap is carefully cleaned with povidone-iodine solution. Necrotic tissue is removed, but healthy tissue is preserved. The flap is then sutured with 6-0 or 7-0 Vicryl sutures, taking care not to penetrate the entire cornea to avoid damaging the endothelium and risking infection.

Post-operative care includes topical atropine to reduce intraocular pressure, topical antibiotics to prevent infection, and systemic NSAIDs to manage inflammation. Topical corticosteroids may be initiated after 10-14 days to minimize scarring.

2. Penetrating corneal laceration with/without iris prolapse

Penetrating corneal lacerations with iris prolapse are more serious than partial-thickness lacerations. The iris often becomes trapped in the wound, increasing the risk of inflammation and tissue necrosis. Prompt surgical intervention is crucial to minimize damage. During surgery, the prolapsed iris is assessed. If it has been exposed for more than 12-hours or is necrotic, it is amputated. Otherwise, it is carefully repositioned into the anterior chamber.

The corneal wound is then closed with fine sutures, taking care to avoid damaging the corneal endothelium. In some cases, a conjunctival graft may be necessary to reinforce the wound closure. Postoperative treatment consists of topical and systemic antibiotics, topical mydriatics and systemic NSAIDs or systemic corticosteroids.

Approximately two weeks post operatively a topical corticosteroid can be added to reduce corneal scaring. Postoperative complications are not uncommon and may include corneal scar formation, possible posterior synechiae [Atropine post-op should prevent this], cataract formation, secondary glaucoma and phthisis bulbi.

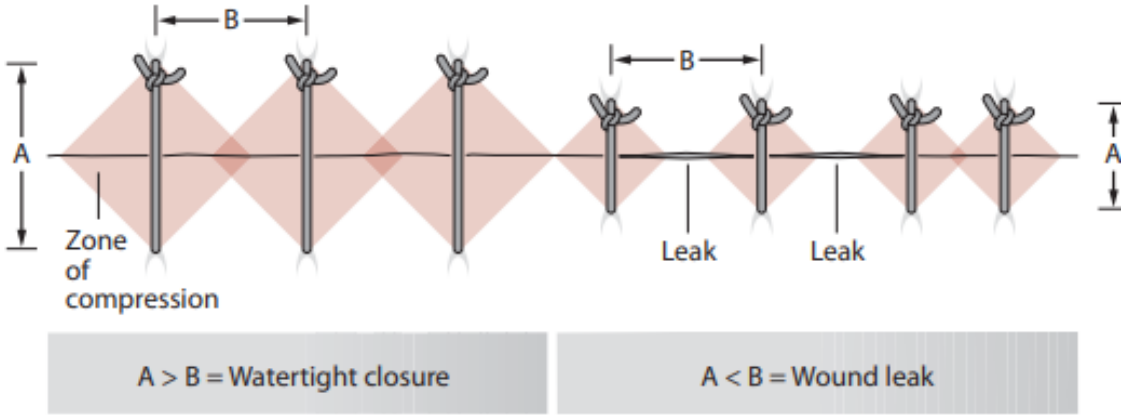

Figure 8: Different sizes of suture bites result in different zones of compression. When the zones of compression overlap, adequate wound closure is achieved.

Source: Marian S. Macsai (Ed.) Ophthalmic Microsurgical Suturing Techniques

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References: 1. Miarikova, M., 2017. The capacity and effectiveness of diosmectite and charcoal in trapping the compounds causing the most frequent intoxications in acute medicine: A comparative study. *Environmental Toxicology and Pharmacology*, 52:214-220. 2. 2009. As many as 1 000 dogs poisoned per week in SA. *Infurmation*. Available at: https://www.infurmation.co.za/home-page/1000-dogs-poisoned-per-week/.

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KL-MA-RESVET360-12